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AN X-RAY STUDY OF THE LUNGS OF WORKMEN IN THE ASBESTOS INDUSTRY, COVERING A PERIOD OF TEN YEARS[1,2]

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DUST inhalation is a subject in which we have been particularly interested for the past ten years. Our approach to this problem has been through the medium of the x-ray. During the past five years, the dust hazard in many industries has become a serious economic factor, for the individual workman as well as for the corporation and insurance company. In some instances the amount of alleged disability among the workmen, from dust, had reached such proportions that insurance companies became insolvent, and the industrial plants were forced to close for lack of compensation insurance protection.

During this period, we have had the opportunity of studying the chests of over three thousand workmen exposed to a dust hazard in their regular occupation. This group included various types of dust hazards—mining and manufacturing—and from various parts of this country, east of the Rockies. From this group it has been our privilege to observe with the x-ray the changes in the chest which may result from more or less continuous exposure to various types of dust, such as silica, talc, marble, coal, asbestos, heavy metals, such as iron and brass, and various organic dusts, such as wood fiber.

In this rather cursory paper it is our purpose to record our observations and conclusions based on an x-ray study of one type of dust hazard, namely, asbestos dust. The disease condition resulting from the inhalation of asbestos dust is that form of pneumoconiosis known as asbestosis. We shall comment on this disease, as an economic problem, as it concerns the insurance companies and the industrial accident boards, and also as a disease condition in the individual, its pathology and prognosis as observed by us through the x-ray.

In 1928, we began the intensive study of those exposed to the asbestos hazard in one of the large industrial plants in Massachusetts, where brake lining was manufactured. The hazard created was principally asbestos, with less than 30 per cent silica. Dry weaving was first used and then changed to wet weaving. This change was brought about by the fact that in 1928 and 1929 there were numerous cases which showed symptoms, referable to the lungs, characterized by increased difficulty in breathing on exertion, cough, sleeplessness, and loss of weight. Of all the cases we examined in 1928 and 1929, our records show that in 12 cases x-ray examination revealed changes in the lungs, upon which we based our diagnosis of asbestosis. These 12 were at once put on compensation or given a lump-sum settlement and have been under observation for seven years, throwing considerable light on the natural progress of the disease. From about 1928 to 1933, we routinely examined those cases suspected clinically of asbestosis. By 1933 the insurance premiums had pyramided to 15 per cent of the payroll, and the carriers were rather indifferent to the taking on of this risk. Finally, one of the large companies decided that before they took over this risk, they would have examined, clinically and by x-ray, all of the employees of the plant, totaling approximately eight hundred. The majority of these workmen were examined by us immediately at the plant. There was a very small number of the total employees whose conditions we diagnosed as asbestosis. Out of this number that we either suspected or diagnosed asbestosis, there were three cases that were definitely advanced. These three cases were of interest because they did not show

[1] Read (in part) by invitation at the Twenty-fourth Annual Meeting of the Radiological Society of North America, at Pittsburgh, Nov. 28-Dec. 2, 1938.

[2] This is one of a series of papers contributed by friends and former pupils of I. Seth Hubbard, M.D.

any of the usual clinical signs; and would t, on account of the high wages they re earning, change their occupations. hese three men continued to work until e plant was closed, in 1936. One of the ree became a litigant later. The other o disappeared into other industries and e not recorded as claiming disability to te.

A survey of this type naturally revealed any interesting lung conditions aside m asbestosis, such as aneurysm, early aled, active and chronic tuberculosis, art abnormalities, and many cases of sidual changes from previous lung disses.

The plant was eventually sold to a competitive company and was immediately losed. Within a short time after the closg of the plant, 90 workmen brought in ims for disability due to asbestosis. hese 90 cases were examined by us by x-y and we made a positive diagnosis of bestosis on 12, with three additional ses questionable. About 13 per cent of he positive x-ray diagnoses, in this group 90, claimed symptoms of asbestosis. The insurance carrier for this competitive mpany immediately placed these 12 sed either on compensation or they were ven lump-sum settlement in excess of 50,00?. The diagnosis was based upon he clinical and the x-ray examinations, incipally the x-ray. There were left 78 ses claiming disability, but their physical nd x-ray examinations were negative. f this group of 78 claiming disability, but ithout physical or x-ray evidence of sbestosis, 30 came to trial. There is, in ur city, the Legal Aid Society which urnishes able counsel for the plaintiffs, as ell as competent medical experts. A very apable attorney was chosen for these ases, and the late John B. Hawes, Jr., D., as the medical expert. He, in turn, xamined these cases both clinically and y x-ray. On these 30 cases he made a ositive diagnosis. Under our Statutes of ndustrial Diseases of the State of Massausetts is the so-called "Panel Doctors," group of three, one of whom is a roentgenologist. Under our State Law their diagnosis is final. They had the opportunity to examine 10 of these 30 cases. Their reports in no instance definitely stated asbestosis, but their findings were reported as, "consistent with," or "a slight degree of," or "the probability of" asbestosis. It was obvious in reading their reports that they were not depending on x-ray findings alone, but were giving the benefit of the doubt to the workmen, in many instances being influenced by the history of exposure. The x-ray films of these cases which were diagnosed as positive by the Panel Doctors, we would have called negative. This illustrates the great difficulty in interpreting an x-ray film, as to the presence or absence of lung changes due to asbestosis. In many instances it is a matter of personal opinion, and the value of an opinion is in a large measure in proportion to the roentgenologist's training and experience in this specialized field.

The late John B. Hawes, Jr., M.D., who was one of our distinguished specialists on tuberculosis, always forcefully expressed his opinion regarding an individual who worked in a dust hazard, claiming that exposure for three years or more made the individual a potential case of asbestosis. He so expressed himself when these cases came to trial. We all felt that at least 80 per cent of his positive diagnoses depended upon history of exposure rather than subjective or objective physical signs.

We will not attempt in this paper to take up the question of physical diagnosis, but we will say that it is our firm conviction that at the present time the only method of positive diagnosis is the x-ray, and that aside from the history of exposure there are no clinical pathognomonic signs of the disease. The clinical facts on which Dr. Hawes based his diagnosis and prognosis of asbestosis were as follows: (1) history of exposure; (2) difficulty in breathing on exertion; (3) cough; (4) rapidity of heart action on exertion, and (5) that asbestosis was a progressive disease, once contracted, whether the individual remained in the industry or was removed.

In addition to these clinical observations of Dr. Hawes, it is interesting to note that in our experience a large number of patients showing a definite x-ray evidence of asbestosis, give a history of coughing up blood. The amount of blood varies considerably, usually no more than blood-streaked sputum, occasionally a substantial amount of pure blood. We have speculated considerably as to the etiology of this symptom. It has been observed for some time that workers in asbestos dust are prone to the development of areas of irritation on the hands. In many instances, these lesions develop into open ulcers which are extremely difficult to heal. Microscopic examination of some of these ulcer bases has demonstrated the presence of asbestos fibers embedded in the deep layers of skin. A total removal of the ulcerative tissue containingt he asbestos fibers results in prompt healing of the lesion. It is not impossible that a somewhat similar condition may develop in the trachea or bronchi of the lungs, with a superficial erosion of the mucous membrane, due to the mechanical irritation of the asbestos fiber and the symptom of hemorrhage, so commonly observed, is due to small bronchial ulcers.

It has also been observed that asbestos dust like silica and some of the other dusts seems to have a selective action. In other words, certain individuals seem sensitive to the asbestos dust and others are immune. It is a very common experience to find two individuals working in the same concentration of dust for the same length of time and same type of work, and only one developing asbestosis; the other remains in perfect health. No satisfactory explanation has been made of this apparent idiosynerasy. It is possible that the explanation may be a simple one, namely, the method of breathing. The man who habitually breathes through his nose, with all the protective mechanism which Nature has furnished to prevent dust hazard, would have much less chance of developing injury to his lungs than the man who habitually breathes through his mouth and does not receive the benefit of the protective organs of the nose and nasal pharynx.

As a side-light on the question whether or not asbestosis is a progressive disease, I

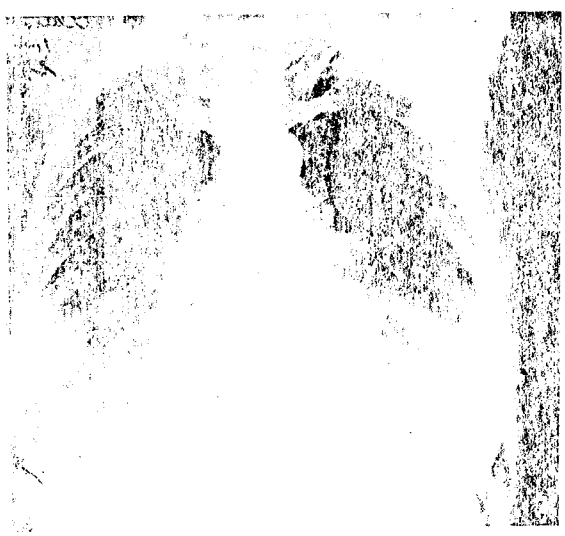


Fig. 1.

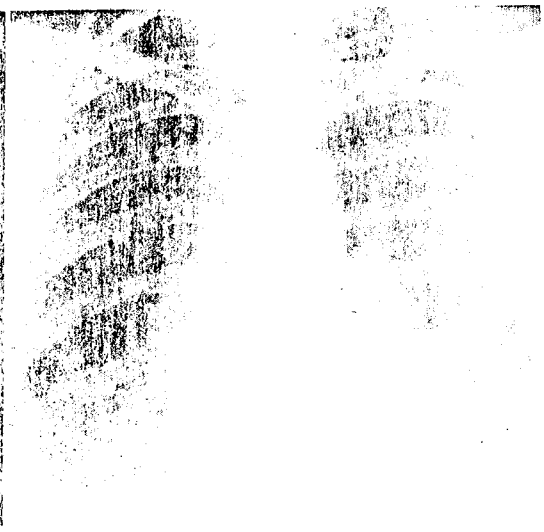


Fig. 2.

Fig. 1. Case 1. Mrs. A., examined impartially in 1931, was negative; 1936, positive. Intermittent employment from 1919 to 1929, from 1931 to 1934.
Fig. 2. Case 2. Mr. D., more than six years' continuous exposure.

would now like to refer to the 12 cases previously mentioned, that were diagnosed as positive in 1928 and 1929, given compensation, and discharged. At one of the hearings they were re-called by the insurance

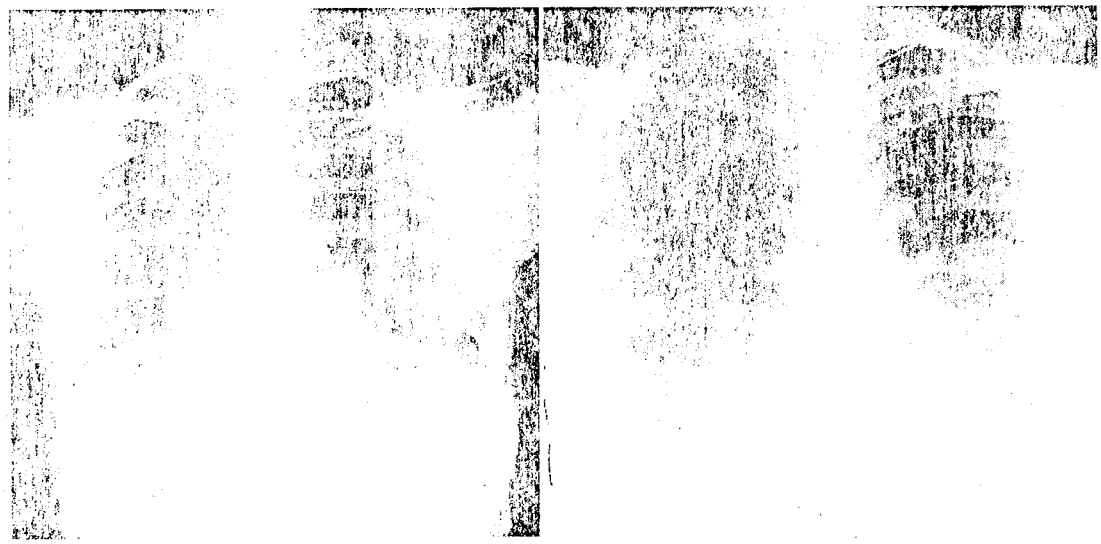


Fig. 3. Fig. 4.

Fig. 3. Case 4. Mr. S., more than six years' intermittent exposure.
Fig. 4. Case 1. Mr. V., more than six years' exposure.

company to illustrate that these 12 who had been found positive by both clinical and x-ray examination were, by their own testimony, free of all symptoms, and, except for several women in the group, were doing laborious work in their new occupations. If, according to Dr. Hawes, asbestosis is a progressive disease, once contracted, these individuals should have been in the same condition as in 1928 and 1929, or much worse. It would be logical to assume that eight or nine years away from the industry, without symptoms, gives us very definite evidence that this disease, provided we were correct originally, is not a progressive one, at least in this particular industry.

It is evident that if a history of exposure to asbestos dust is eliminated, a positive clinical diagnosis of asbestosis cannot be made. In other words, there is no one physical finding or group of objective signs which are pathognomonic of asbestosis. We are of the opinion that in x-ray examination we have the best and perhaps the only method of diagnosis of asbestosis at the present time.

The x-ray reveals the actual tissue changes in the lungs. In a general way these changes are much less extensive, as compared with silicosis or anthracosis. We find little parallel between the extent of the visible pathology and the severity of the clinical symptoms. It has been impracticable to classify the disease into different stages. All of our positive cases have shown about the same amount of lung involvement.

The theoretical "early" or "incipient" asbestosis, a stage before visible lung changes are present, cannot, of course, be diagnosed by the x-ray. On the other hand, we are of the opinion from our general observations, and it is the opinion of several clinicians who have studied these cases, that asbestos dust will not give symptoms until there has been sufficient lung-tissue change to be visible on an x-ray film. The average uncomplicated case of asbestosis gives rather consistent and characteristic x-ray findings.

In the first place, there is an increased density in the lung-fields. This increased

density is characterized first by bilateral distribution, and, secondly, by its being limited to the lower lobes of both lungs. The quality of the shadow is characterized by a homogeneous quality and is described

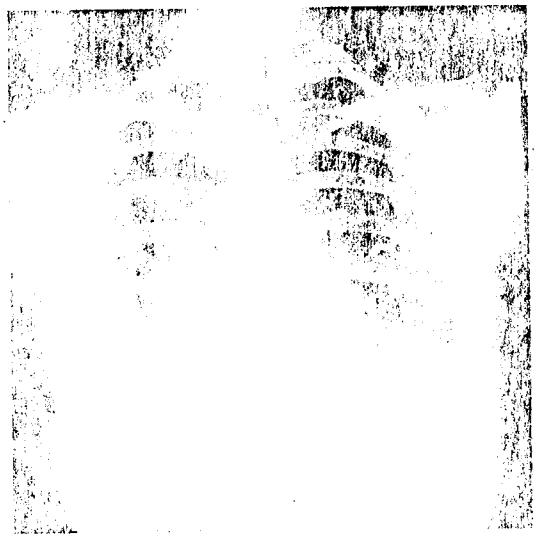


Fig. 5. Case 5. Mr. H., more than six years' exposure.

in the literature as a "ground-glass" or "smoky" appearance. The microscopic changes in the lung show that we are dealing with an almost interstitial fibrosis with areas of atelectasis of the alveoli and smaller bronchioles. There is an absence of the gross nodulation seen in silicosis. What nodulation there is has a fine, evenly distributed quality. There are no characteristic changes about the root shadows. Some cases are associated with hilum fibrosis and glandular enlargement, but many cases of asbestosis show no variation in the appearance of the hilum shadows. Often there is some accentuation of the bronchial shadows. There is frequently found a peculiar appearance of the bronchial tree in both lower lobes, in that it presents a rather laminated appearance. This peculiar arrangement of the bronchial shadows is apparently due to localized areas of emphysema scattered throughout the lower lobes. The microscope tells us that the basic pathologic change is the deposit of a protective element about the asbestos fiber, rather than a destructive process of the lung tissue. The asbestos fibers appear to be surrounded by a substance uniform in character and having the appearance of connective tissues. It would appear from a study of the slides that the fiber is innocent of producing any pathologic disturbance, except an impedance to the air in this region of the lung. In other words, in asbestosis we are dealing primarily with an atelectasis due to the mechanical blocking of the alveoli and bronchioles by the protecting fibers about the asbestos fiber. This peculiar diffuse microscopic fibrosis, associated with atelectasis and microscopic areas of emphysema, is the type of change that produces the so-called "ground-glass" appearance on the x-ray film.

In the second place, asbestos dust produces changes in the pleura, the most constant finding being an accentuation of one or more interlobar pleural septa. The microscope shows that fibrosis associated with the asbestos fibers is prone to involve the pleural covering of the lung. The demonstration of pleuritic adhesions of both lower chests, with obliteration of the costo-phrenic angles, is a rather constant finding in uncomplicated asbestosis.

In the third place, the x-ray shows the usual signs of emphysema. This emphysema, of course, is not pathognomonic of asbestosis but when accompanied by other x-ray indications, can be considered of a confirmatory nature. This evidence of emphysema is best demonstrated by the limited excursion of both diaphragms and the increased radiability of the lung tissue.

In the fourth place, we have noted in the cases of asbestosis certain changes in the contour of the heart shadow which we feel is somewhat causally related to the process in the lungs. Other writers have put considerable emphasis on the dilatation of the right auricle and ventricle, as evidence of asbestosis. It is our opinion that dilatation of the right side of the heart is seen in a higher percentage of asbestosis cases than in other forms of pneumoconio-

sis, particularly silicosis. There is a theoretical possibility that the type of interstitial fibrosis seen with the asbestos dust is such as to produce a mechanical interference with the circulation in the perialveolar capillaries and that this embarrassment in the pulmonary circulation results in some hypertrophy or dilatation of the right side of the heart. We have not observed any demonstrable dilatation of the pulmonary artery.

There are several points which are of importance in the differential diagnosis between asbestosis and other forms of pneumoconiosis, and also between asbestosis and other shadows, due either to faulty technic or to variation in the lung markings from other conditions not connected with the dust hazard. It would seem unnecessary to mention faulty photographic technic. It is our belief that many erroneous x-ray diagnoses of asbestosis have been made on x-ray films which were either under-exposed or over-exposed. The physical qualities of the individual should also be taken into consideration. A woman with large breasts will give an x-ray picture which may have all the characteristics of lung changes due to asbestosis. The fact being, however, this variation in density is due to chest-wall structure, rather than to changes within the chest cavity.

There are certain points of importance in the differential diagnosis of asbestosis and silicosis.

(1) The increased density in asbestosis is confined to the bases; in silicosis it is primarily found in the central portion of both lungs and frequently the bases are clear.

(2) The nodulation of asbestosis is the fine granular-type areas; in silicosis the nodulations are coarse and in the advanced stages they become conglomerate, producing areas of apparent consolidation.

(3) The pleura is more likely to be involved in asbestosis than in silicosis. The same is true of the hypertrophy of the right side of the heart in asbestosis. In our experience this heart condition is rarely seen in silicosis. In this particular industry which we have studied, the percentage of silica, where it was found in the dust tests, was less than 30 per cent, and we have not been bothered by a combination of asbestosis and silicosis. It is conceivable

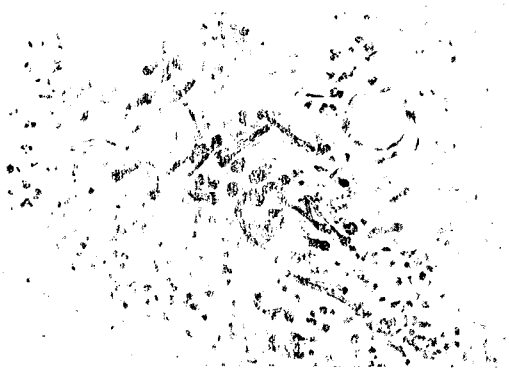


Fig. 6. Photomicrograph of section of lung showing asbestos bodies. (Courtesy of Liberty Mutual Insurance Company.)

in certain industries, and perhaps in the mining of asbestos, we may have a dust hazard of asbestosis with a high percentage of silica. In the advanced cases of pneumoconiosis due to this double hazard, it might be fairly easy to determine from the x-ray the amount of disease due to the silica and that due to the asbestos fiber, but in the average case an accurate interpretation of pneumoconiosis due to both asbestos and silica would be a difficult procedure.

In this series of cases we have found no case of complicating tuberculosis. From our experience we are of the opinion that asbestosis is not prone to the development of tuberculosis, and we have had no case in which there was evidence of asbestosis lighting up a pre-existing tuberculosis. The differential diagnosis between asbestosis and tuberculosis is reasonably easy. The diseases have little in common, as far as their x-ray appearance is concerned. This is not true in cases of silicosis complicated by tuberculosis. It is sometimes difficult to make a differential diagnosis between asbestosis and certain conditions which produce congestion of the lower lobes of the lungs or accentuation of the

bronchial shadows in the lower lobes. Chronic passive congestion associated with heart lesion might well simulate conditions of asbestosis. The differential diagnosis in such an instance depends considerably upon the clinical history. Chronic bronchitis or bronchiectasis might produce a picture somewhat confusing with asbestosis, but neither of these conditions usually shows the characteristic "ground-glass" increased density over the lower lobes.

CONCLUSIONS

(1) It is our opinion that every industry with an asbestosis hazard must be a special study in itself. All asbestos mining and manufacturing cannot be classified in one group as to the dust hazard. The percentage of silica in the asbestos dust must be taken into consideration. The method of manufacturing, whether it is the dry or wet method, is of importance.

(2) We are of the opinion that the disease, asbestosis, is not a progressive disease after the individual has been removed from the dust hazard.

(3) There is no evidence in our study of asbestosis for the past ten years that asbestosis has any causal relation to tuberculosis.

(4) It is our opinion that the final diagnosis of asbestosis must depend mainly on the x-ray evidence, that there are no clinical findings pathognomonic of asbestosis, and that an amount of asbestos dust in the lungs, sufficient to produce symptoms, will give visible evidence of tissue changes on the x-ray film.