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Pulmonary Asbestosis*

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THE manufacturing of asbestos products in the last few years has assumed a somewhat greater importance in the industrial world. Although the total number of workers in asbestos mills is probably far smaller than in many other lines of trade, their health is of paramount importance. The conditions surrounding the greater proportion of the employees constitute a distinct and serious industrial hazard, and often sufficient devices for protection have not been provided. It is doubtful if any single employee in certain departments of these mills can possibly escape some damage to his respiratory system because of the unavoidable inhalation of asbestos dust. Naturally, the longer the service of an employee, the more certain is more or less extensive pulmonary damage.

Asbestos is composed principally of magnesium silicate in crystalline hydrated form, always found with other minerals, particularly iron and magnetite, and usually contains about 2.6 per cent free silica. The rock is blasted in open quarries, being afterward crushed and pulverized, although often the pulverizing is done at the mill where the finished product is produced. At the mill the fibers are mixed with other fibers, usually cotton, then carded and spun by processes very similar to those employed in the production of cotton goods. In all of these processes a considerable quantity of asbestos dust is produced. Cooke states that the asbestos fiber microscopically consists of two different elements, (1) the glistening material which constitutes the bulk, and (2) black opaque angular particles. The dust contains these particles and minute granules. Analysis of samples showed that the dust with the greater quantity of these black particles contained the largest amount of iron. As the dust contained 18.4 per cent of iron and the finished product only 0.1 per cent, Cooke concluded that the black brittle parts of the fiber are the cause of the dust, and the danger to the health of the workers. Since the asbestosis bodies found in the sputum of disabled workers have been found to have cores which appear to be asbestos fibers around which has been deposited some kind of material from the living tissues, the iron-containing particles cannot be the *sole* cause of the condition. In fact, Gloyne, in 1929, showed that the core of the "asbestosis body" is an asbestos fiber.

The "dusty trades" for a long time have been considered inimical to the health of employees. In spite of this it has only been within the last 10 years that "pulmonary asbestosis," a disability resulting from working in one of the dustiest of trades, has been to any appreciable extent recognized. The first recorded case of "asbestosis" was reported by Dr. H. M. Murray at Charing Cross Hospital in 1900, and his diagnosis was supported by necropsy findings. The man was the only survivor of 10 who had worked in the carding room of an asbestos mill 10 years

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and expansion is very much restricted. The percussion note over the whole chest is higher-pitched than normal except in the apices where the note is frequently resonant, probably because of compensatory emphysema. Although invariably bilateral, the impairment of resonance is more marked on the right side. The physical signs on auscultation are diminished to feeble breath sounds, prolonged and roughened expiration, and either dry crackles, moist rales, or both, usually over the lower half or two-thirds of the lungs. On the whole, the physical signs are quite similar to those found in uncomplicated silicosis, fibroid tuberculosis, or atrophic pulmonary emphysema. There are no definite physical signs distinctive of the condition. Signs are supposed to develop in less than 10 years' exposure, but they are frequently discovered within 2 years. The shortest time in my series of cases, from exposure to the development of symptoms and physical signs, was 18 months. The continuously progressive cases often develop extensive bronchiectasis, or engorgement of the right heart with progressive heart failure as a terminal result. Bronchopneumonia is frequently a serious complication.

The X-ray films show a somewhat distinctive picture. According to Merewether,[4] if films of asbestosis subjects be compared with those of victims of silicosis, the amount of disease in the former will be underestimated, because of inherent differences in the underlying pathology. The nodular distribution of the fibrosis in patients with silicosis is not often found in asbestosis. In the earlier stages of asbestosis the films present what has been frequently called a ground glass appearance. Later there is more definite mottling, but the lesions are never as dense as those of silicosis; consequently, it is difficult to divide the progress of the condition into stages. Stereoscopic films show that the ground glass appearance is caused by the development of a very fine fibrosis, and this is almost invariably seen to be much more extensive in the basal portions of the lungs.

Evidence of pleuritic involvement is often indicated by obliteration of the costo-phrenic angles, and flattening or peaking of the diaphragm. Often the diaphragm is indistinct in outline. The films sometimes show some thickening of the parietal pleura. The outline of the heart shadow is often "shaggy," the degree depending on the length of time of exposure to the dust. Occasionally calcareous deposits are seen not only around the hilum of the lung, but scattered in the parenchyma as well. On the whole, in well advanced cases of asbestosis, X-ray films do not show the amount of pathological change that one would expect from the clinical signs and symptoms. Even the most severe cases do not show such definite X-ray evidence as is seen in silicosis, although the area of involvement is usually somewhat extensive.

During the past 3 or 4 years I have collected 15 cases of asbestos workers who have been referred to our chest clinic because of symptoms referable to the respiratory system. With one exception, these have had a diagnosis of pulmonary tuberculosis. Invariably their principal and most distressing symptom has been dyspnea, in combination with slight or more severe cough, lassitude and progressive although gradual loss of weight.

Having had occasion some years ago to familiarize myself with the conditions in asbestos factories, I have since been intensely interested in determining the extent of the industrial hazard in this type of work. For that reason I have attempted to obtain X-ray films of the lungs of all asbestos workers who have been referred to the clinic because of respiratory symptoms. Of the films taken, only 3 have failed to exhibit

some definite evidence of the condition called pulmonary asbestosis. Although this series is comparatively small, it seems reasonable to conclude that this condition is far more frequent among asbestos workers than is usually supposed, in spite of the increased attention paid to the condition in the past 4 or 5 years.

Case histories with the X-ray films of 3 patients may be of interest. All of the cases are still living, although totally disabled. I have not been able to demonstrate asbestosis bodies in the sputum, which should not be absolutely necessary for a diagnosis when the X-ray film shows unmistakable pathology, and the physical signs indicate more or less widespread involvement.

Case I—D.S.W., white male, age 64 years. Came under observation Jan. 8, 1929. His complaint for 6 months had been cough and dyspnea, combined with anorexia and loss of weight. He had worked for 18 months in the card room of an asbestos plant with no protective devices. He had considerable cough, although the amount of sputum was not large and was negative for tubercle bacilli. There was a history of only a very slight rise of temperature at any time. There was slight hoarseness at times. The expansion was noticeably deficient over the whole chest and the resonance was slightly impaired over the lower lobes. The breath sounds were prolonged anteriorly over the mid-portion of the lungs, and, posteriorly, from the middle of the scapula to the base. Over the latter region there were many dry crackles and some rales.

The X-ray films showed considerable thickening around the hili. The heart outline was shaggy, and both costo-phrenic angles were obliterated. The diaphragm was irregular and indistinct in outline. The lower two-thirds of the lungs were hazy and infiltrated with fine fibrous lines, and over the left base there was a considerable area of fairly heavy mottling.

This patient was put on complete rest for 3 months, during which he gained 30 pounds. At this time (4 years after becoming incapacitated) he is working as night orderly in a tuberculosis sanatorium, but his cough and dyspnea show no improvement. X-ray films show no clearing of the condition. He suffers at rather frequent intervals with chest pains, and about 3 months ago had an attack of fibrinous pleurisy with a rise of temperature to 102°.

Case II—C.A.B., white male, age 73 years. Patient appeared at clinic June 13, 1931, with cough and distressing dyspnea; had lost a few pounds, and had the appearance of a very ill man. He had worked practically continuously in the card-room of an asbestos plant for 6 years, but had been compelled to quit work about 6 weeks before on account of extreme weakness and shortness of breath.

The paroxysms of dyspnea, even on the slightest exertion, were severe. Talking apparently required considerable effort. Expansion was much deficient over the whole chest, the intercostal spaces being drawn inward on inspiration. Resonance was impaired over the lower two-thirds of both lungs, and the expiration was prolonged and roughened from the apex to base on both sides. There were dry and moist rales anteriorly from the 3rd rib downward and posteriorly on both sides from apex to base. The breath sounds were considerably diminished over both lower lobes posteriorly.

X-ray films present a fairly typical picture of asbestosis. The right costo-phrenic angle is obliterated and the right leaf of the diaphragm is irregular in outline and pushed by adhesions. The left costo-phrenic angle is partially obliterated. The lung fields present the ground glass appearance. Around both hili are numerous apparently heavily calcified areas, and there are several such small areas scattered through the parenchyma of the lung. Stereoscopic examination of the films shows a fibrotic involvement of both lungs which is much more extensive over the lower lobes. In both bases there are fairly extensive areas of heavy mottling.

This patient, 2 years after the condition was first recognized, is still living, but his symptoms have in no way improved, and he remains totally incapacitated. The eventual fatal result by progressive cardiac failure appears to be inevitable.

Case III—R.G.J., white male, age 31 years, was admitted into the sanatorium on April 21, 1930, with a diagnosis of pulmonary tuberculosis. His complaints were lassitude, loss of weight, and cough, which symptoms had been gradually growing worse for 1 year. He had lost about 30 pounds, and, on account of weakness and shortness of breath, had been compelled to quit his work about 4 weeks before. His daily temperature, he stated, had not exceeded 100° F. He had worked in the card-room of an asbestos mill, in which there were some protective devices, for about 4 years.

time as a heavier shadow. The heart is more shaggy in outline, the diaphragm is fluttered on both sides, and there is apparently increased retraction of the lower ribs. Although the patient appeared to have remained in good physical condition, the X-ray films indicate some increase in pathology. This man is still living, but remains incapable of continuing remunerative employment for any length of time.

These cases are typical of the condition found in each patient of the series, and, from the symptoms and X-ray findings, it seems justifiable to assume that the exposure to asbestos dust is the cause of their total and permanent disability. That exposure to the inhalation of this dust for even a comparatively short time is a definite and serious industrial hazard, has been too frequently indicated to be open to doubt. The fact that the condition when once acquired is permanent and more or less rapidly progressive is most important from a public health viewpoint. It also seems to be the consensus of opinion, not only among writers on the subject, but also among the asbestos workers, that the protective devices now in use in many plants are most inadequate.

*The compensation laws in North Carolina do not list this disability as entitling the worker to proper remuneration, and I presume that legal status obtains in other states. In England, in 1931, the Asbestos Industry Regulations were made legally mandatory. These Regulations require the substitution of wet methods for dry, the enclosure of dust producing machines, the substitution of enclosed mechanical methods for hand conveyance and for dusty hand work generally, and the installing of exhaust drafts at dust producing points. In the same year it was also made obligatory for workmen in asbestos plants to appear for periodic medical examinations as a means of preventing or arresting the development of asbestosis.

If any similar regulations have been adopted in this country I am not familiar with them. Asbestos factories produce various necessary and valuable articles of commerce, and are, consequently, important parts of our industrial system. For that reason, if for no other, the workers should be provided with every facility for the protection of their health, so that they may earn a livelihood for themselves and families, for at least an average period of time, at the occupation in which they are most skilled. Unfortunately, every worker handicapped by pulmonary asbestosis becomes almost invariably physically incapable of engaging in any occupation, and many of them become an added expense to the welfare agencies of the community. Although the number of asbestos workers is much less than that in many other industries, their occupation is extremely hazardous, and they are amply justified in expecting whatever protection it is possible to give them. Furthermore, the fact that efficient protective devices in this industry, in spite of the added expense, will effect a substantial financial saving, is becoming more apparent. The workers themselves are becoming informed of the danger to health, and many civil suits for damages against factory owners are the result.

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