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PULMONARY ASBESTOSIS: INCIDENCE AND PROGNOSIS*

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WITHIN a comparatively short time the occupational disease, pulmonary asbestosis, has become a matter of considerable importance, not only to manufacturers of asbestos products, but also to the workmen engaged in the industry. That the serious industrial hazard of this type of work has not previously received sufficient attention is becoming more apparent. The manufacture of asbestos products has increased more than four-fold in the last 20 years, and hence, because of the greater number of workers exposed, and the more frequent recognition as a pathological entity of the resulting pulmonary condition, the subject of asbestosis has rapidly assumed greater importance.

It is indicated that the owners of asbestos plants, and the workers themselves, are beginning to realize that exposure to asbestos dust is a serious occupational hazard, and it also is apparent that these workers *must* be protected against the hazard as effectively as is possible. That this protection can be made complete seems very doubtful. It seems to be the opinion of engineers that the most effective types of suction apparatus now in use will remove a maximum of not over 90 per cent of the dust, but the remaining 10 per cent may be definitely injurious. In 1916 protective devices of the suction type were placed in a certain asbestos mill, and yet 15 years later cases of asbestosis with fatal termination were reported among workers from this plant. Since the most efficient type of protective machinery known apparently does not completely protect, although the number of workers affected is probably somewhat reduced, and the time required for the production of an advanced type of disease is probably lengthened, the problem becomes a matter for serious consideration.

In the case of any industrial employee presumed to be physically handicapped by any condition or disease, supposedly caused or aggravated by conditions under which he has worked, there enters the question of compensability. Pulmonary asbestosis has not as yet been widely recognized as a compensable disease in states having laws governing the compensation of workers. If such cases are viewed as compensable under the Industrial Compensation Laws, it will be necessary to decide the degree of disability in each individual in order that proper rating may be made.

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Practically all writers on the subject are agreed that the principal symptom of the condition is a varying degree of dyspnea, and that the physical signs are those found in fibroid tuberculosis. Hence a definite diagnosis cannot be made by a physical examination alone. The x-ray film, however, is distinctive, in that it shows a picture of pathological changes in several particulars different from those found in the x-ray films of any other respiratory abnormality. The presence of the condition can be demonstrated by the x-ray film alone, but the degree of disability cannot be estimated unless the symptoms and physical signs are also considered. The dyspnea is usually out of proportion to other symptomatic manifestations, and is the one symptom which, in the more advanced cases, precludes any form of muscular exertion. For that reason, an asbestotic victim is unable to sell his labor on the open market, and must be rated as totally disabled.

I have seen no statistics in the literature in regard to the incidence of asbestosis among the workers in the industry. It would seem to be a matter of some importance to determine the average number of employees who may be now, or who are likely to become, disabled by the condition. I have lately had occasion to review the x-ray films of 151 workers in asbestos mills. Of these, fifty-two showed definite evidence of asbestosis in varying degrees. Of the 151, eighty-three had worked in asbestos for periods varying from 4 to 20 years, and in this group were found fifty-one of the positive cases, a percentage of 59.3. There was only one case with definite x-ray evidence of asbestosis who had worked less than 4 years in the industry. Of the 52 workers with evidence of asbestosis only four had worked less than 5 years, forty-eight having been employed for 5 years or more. There were eleven with an advanced type, no one of whom had worked less than 8 years. One of these advanced cases had been exposed to asbestos dust for 8 years, two for 9, and one each for 10, 11, 12, 14, 15, 16, 17 and 20 years.

Unfortunately I have no record of the length of service of these employees in the various departments of the mills, all of which are unquestionably dusty, but it is agreed that the carding room is the dustiest, and the weave room probably next. It is presumed that the dustier the work, the more likelihood there is that pathological changes in the lungs may result. It is noteworthy, however, that some employees work in asbestos plants for years without suffering any pathological changes in the lungs, as far as can be detected in the x-ray films.

In this series of 151 employees, ninety-nine showed no positive evidence of asbestosis in the films. Five had worked in asbestos for 6 years, three for 7, three for 8, four for 9, six for 10, one for 11, and one for 15 years. All the others had worked for periods varying from a few months to 5 years. Since the percentage of those in the series who were affected was so high, it seems remarkable that so many could be entirely free from the condition even after years of exposure. Since this process is non-infectious, the term "physical resistance to disease" is not applicable. It is true that muscular development avails

nothing as a protection against the disease, since well developed men are as frequently affected as those not so well developed. It is also possible that some individuals may be susceptible even though exposed to low concentrations of dust.

It is the opinion of some writers that the inhalation of asbestos dust and the consequent production of asbestosis has a tendency to exacerbate old tuberculous lesions. This opinion is apparently not universal. Merewether (1) says "from a review of 42 fatal cases of confirmed asbestosis it appears that asbestosis . . . is less frequently accompanied by tuberculosis than is silicosis." Gardner and Cummings (2) state that "primary tuberculous infection is influenced only to a limited degree by inhaled asbestos." Egbert (3), in an analysis of 28 cases of pulmonary asbestosis with fatal termination as reported in the literature, found that tuberculosis was present in only 6 cases of the 28, and in only 3 instances was death due primarily to tuberculosis.

It is universally agreed that silicosis does tend to exacerbate old tuberculous lesions. In fact more or less rapidly progressive tuberculosis is considered the most frequent and most serious complication of silicosis. Boisliniere (4) says that silica is the only phthisis-producing dust. In observations for several years I have been unable to find evidence that the inhalation of asbestos dust, or the condition asbestosis itself, has any tendency to render active old, apparently healed tuberculous lesions. In this series of 151 workers there were three who had a definite asbestosis in addition to an apparently healed tuberculosis. One had been working in asbestos for 10 years, another for 4 years, and the third for 2 years. There was no evidence in the films that the presence of asbestosis had yet had any tendency to activate the tuberculous lesion. The films of 23 workers showed evidence of tuberculous infection without any pathological changes indicating asbestosis. Of these, eleven showed healed childhood type tuberculosis, but none of these had worked longer than 15 months in asbestos. Two workers showed healed childhood type tuberculosis with no asbestosis, one of whom had spent 8 years in the work and the other 6 years. There were 5 films showing apparently arrested adult type disease with no asbestosis. These films were of employees who had spent the following periods in this work: One each for 4, 6, 7, and 9 years; and one, no term of service given. There was 1 case with an apparently healed and calcified miliary tuberculosis with a history of only 8 months' service in the industry.

In the 151 films there were only four which, from an x-ray standpoint, were diagnosed as probably active tuberculosis of the adult type. One of these had been in service for 5 months, and one each for 15 months, for 6 years, and for 10 years. Since 1 case had worked only 5 months and another 15 months, it would seem improbable that this type of work could be the cause of their active disease, particularly as the films of both cases indicated a chronic type of disease and apparently not an acute exacerbation. There remain for consideration 2 active cases with 6 and 10 years' service respectively. Agreeing that

the work in asbestos may have aggravated their pulmonary condition, 2 cases of active tuberculosis in 151 workers seems a decidedly low percentage, if this type of industrial occupation has any tendency *per se* to exacerbate old tuberculous lesions. Tuberculosis of an active type may be found quite as frequently in the routine examination of workers in any industry, regardless of whether or not the occupation be dusty.

Case I. The x-ray films of one man who has worked approximately 10 years in asbestos illustrates the fact that employment in this industry even for a long time does not tend always to re-activate old tuberculous lesions. The first film, taken April 2, 1930, shows some fine fibrosis on both sides from the 6th to the 9th posterior ribs, thickened interlobular pleura with some involvement of the diaphragmatic pleura, and a slightly "shaggy" heart outline, these pathological changes indicating a probable asbestosis which is not very far advanced. In addition to this condition the film shows a tuberculous infiltration in the right apex, which appears to be in a quiescent state. The second film, taken May 3, 1935, indicates that the tuberculous lesion still remains inactive, although a period of 5 years has elapsed since the previous film was taken. This man left the asbestos industry for a while during this period, but returned to it about 2 years ago.

One of the most important problems in the consideration of pulmonary asbestosis is whether or not the disease is progressive, even after an individual handicapped by it ceases to work in the industry. Information on this question is of considerable moment in rating the eventual degree of disability of a worker, either in the assessment of damages in civil suits or in claims under State Compensation Laws.

Sparks (5) states that the disease is apparently progressive, not even the cessation of exposure to the dust checking its spread. Wood and Gloyne (6) state that once asbestosis bodies are found in the sputum the course of the disease appears progressively downward, and cessation of exposure then does not check the spread.

Many writers consider that silicosis is a progressive disease, regardless of whether or not there has been cessation of exposure. However, Sayers (7) says that "a man suffering from simple silicosis generally improves when removed from the dusty atmosphere and placed in suitable surroundings." Boisliniere (4) says: "Silicosis by no means always progresses after the hazard has ceased, nor does it always produce tuberculosis."

It is reasonable to assume that the eventual effect of the inhalation of a dust which sometimes contains as much as 99 per cent silica might be somewhat more severe than that caused by a dust which contains a maximum of only 2.6 per cent. It seems to be true, however, that the fibrosis in asbestosis is produced by a much shorter exposure than is the rule in silicosis. Gardner and Cummings (2) say: "The particles and elongated fibers of asbestos dust do not, during this period (2 years and 5 months), penetrate so deeply into the air passages of the lung as do the other dusts previously studied. The major portion is held up and phagocytosed in the lumina of respiratory

bronchioles and the alveoli given off from their walls." Since the condition is not an infectious process, it should not continue to progress on removal of the causative factor. Gardner and Cummings (2) state that the asbestosis body and the production of fibrous tissue in the lung is caused by hydrolysis of asbestos fibers in the tissues. Theoretically, then, extension of the pathological process should continue until all asbestos fibers remaining in the lung are hydrolyzed, which may require a considerable time.

Several of my cases seem to indicate that the rapidity of progress of the fibrotic change in the lungs after cessation of exposure is dependent to some extent on the amount of involvement which occurred before the individual ceased work. To illustrate this point, consideration of additional x-ray films of 3 cases reported in 1933 may be of interest.

Case II. D. S. W., white male. Ceased work in card room of asbestos plant in January, 1929, after working only 18 months. The first film was taken June 13, 1931, and shows a fairly extensive fine fibrosis, a "shaggy" heart outline and some involvement of the pleura. The second film was taken on March 8, 1935, approximately 4 years later. Comparison of the two films indicates that in the 4 year interval the densities at the hila have increased noticeably, the pulmonary fibrosis has extended toward the apices, and the hypertrophy of both the right and left heart has increased. Symptomatically, the dyspnea has noticeably increased within the last year. The man has not been exposed to asbestos dust since January, 1929.

Case III. C. A. B., white male. Quit his work in the card room of an asbestos plant in June, 1931, after having worked continuously for 8 years. The first film was taken June 13, 1931; the second film, January 30, 1935. The first film shows a bilateral fibrosis over the lower two-thirds of the lungs, the usual "shaggy" heart outline and adhesive pleurisy. This case was diagnosed well-advanced asbestosis. The last film shows an extensive progression of the condition, the fibrosis having extended into the apices, and also become more dense. In spite of the fact that this man has had no exposure for a period of 3½ years, the condition has steadily become more extensive. At the time this is written he has begun to show symptoms of progressive heart failure, and a fatal termination seems inevitable.

Case IV. R. G. J., white male. Quit his work in the card room of an asbestos plant in April, 1930. The first film was taken April 23, 1930, and shows fine fibrosis and mottling over the lower lung fields with some "shagginess" of the heart outline and pleural adhesions. The next film, taken September 2, 1931, 16 months after the first film, shows some increase in the pulmonary fibrosis. This man, under rest treatment, improved his general physical condition materially. He was advised not to return to work in an asbestos plant, but he did not heed this advice because he felt that he must make an attempt to care for his family. He started to work again in May, 1933, and continued until October, 1934, when he was forced again to leave his job. The final film, taken August 30, 1934,

indicates extensive progression of the fibrosis well up into the apices, and a definitely hopeless outlook. In fact this man is beginning to manifest the physical signs and symptoms of progressive cardiac failure, which, in my opinion, is the terminal result most frequently met with in pulmonary asbestosis.

The rate of progress of the condition Case II, and the extensive progress Case III, even though there was cessation of exposure in both cases, nalifies the statement of Wood and loyne (6) that "once asbestosis odies are found in the sputum the urse of the disease appears progresely downward." Asbestosis bodies re not found in the sputum of se II, but were found in that of se III. The sputum of Case IV s at first negative for the bodies, t they are present since the condition become far advanced. Incidenly careful examination of the spun of cases with definite asbestotic olvement in the lungs frequently s to exhibit asbestosis bodies.

The high percentage of pulmonary estosis (34.4) found in this series 151 workers, indicates unquestiony the need for more complete tection of employees in asbestos s. Sufficient evidence has been duced to prove that the inhalation sbestos dust is productive of serious airment of health. In fact, the im of asbestosis, as a rule, eventu- becomes totally disabled from ging in any form of labor. The prominent and lasting symptom his disease is dyspnea, and this t of all proportion not only to physical signs found in the lungs, also to the pathological changes depicted in the x-ray films. It is this symptom which very frequently precludes any form of physical exertion. Furthermore, even in those cases in which the x-ray films show a negligible increase in the pathological process over a period of years, there is no indicated improvement in the dyspnea. Serial x-ray films indicate that the condition is slowly progressive even when exposure has been discontinued for several years. The prognosis for extension of life after cessation of exposure in the cases in which involvement is not far advanced is encouraging, but the hope for amelioration of the dyspnea is not encouraging.

An industrial worker is entitled to every protection that may safeguard his health, so that he may earn a livelihood for himself and family for at least a reasonable period of years in the work in which he is most skilled. If he is prevented from continuing in such work because of impairment of health through no fault of his own, he is entitled to some remuneration for his loss of earning power. That protection of asbestos workers has been woefully lacking in the past has been definitely shown. It is imperative that such protection, as nearly complete as possible, be provided by mill owners. Efficient protective devices will be far less expensive in the final check up than the aggregate of numerous claims for compensation and frequent damage suits. That complete protection can be afforded by the devices in use at the present time seems to be somewhat doubtful, but workers are entitled to the highest type of protection which the engineers familiar with the hazard can provide.

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