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Pulmonary Disability in Asbestos Workers

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Asbestosis has been described in detail in numerous published articles and texts. While it is true that cases of far-advanced asbestosis may have severe pulmonary disability, relatively few of the people exposed to the fiber develop the disease. Therefore, it is the intent in this paper to mention asbestosis only briefly and then to outline various other pulmonary disabilities seen among asbestos workers.

The word "asbestos" is generally used to describe several fibrous magnesium silicates which are different in their chemical composition and physical properties. The most important types of fibers are chrysotile, amosite, crocidolite, anthophyllite, actinolite, and tremolite. Total world production of all fibers last year amounted to slightly more than 1,500,000 tons. Approximately 95% of the fibers produced were of the chrysotile variety, 3% crocidolite, and 2% amosite. Deposits of various types of this mineral are found in many countries, but the largest mines are located in Canada and Africa.

Asbestos fibers are highly resistant to heat and acids. They have great tensile strength and large surface areas. Because of these properties as well as their filamented structure, industrial use of these fibers throughout the world is increasing. The textile industry has used them for many years to produce blankets, clothing, threads, ropes, tapes, braided tubing, and filters. In recent years, however, there has been an increasing use of asbestos in the insulation, building, and friction-material trades. In addition, the fibers can be found in paper, wallboard, shingles, pipe covering, floor tiles, brake linings and brake blocks, cements, putties, and plastics.

It should be noted that the facts presented here apply to those persons who have been exposed only to asbestos fibers and to no other dusts. As previously indicated, industry today is finding many new uses for the fibers when they are mixed with other dusts. It is an established fact that when asbestos fibers are mixed with silica, diatomaceous earth, or other potentially toxic dusts, the pulmonary changes resulting from the inhalation of these mixtures are not typical of asbestosis. The x-ray pattern may be different, the clinical course changed, or the susceptibility to intercurrent infection increased or decreased. Thus, in making a diagnosis of occupational pulmonary disease, it is highly important to obtain a detailed occupational history, so that asbestosis, silicosis, or mixed pneumoconioses can be differentiated.

The statements and observations reported here concern several thousand men and women employed in the asbestos industry in Canada and the United States. In this industry the various mining, milling, and manufacturing operations create some dust containing asbestos fibers. If the fibers up to 50μ in length are inhaled continually in sufficient quantities over a period of several years, a typical pulmonary fibrosis will develop. It has been demonstrated that this fibrosis is due not to the chemical but rather to the mechanical action of the fibers.[1] The asbestos fibers are deposited in the terminal bronchioles, initiating a tissue response which coats the fiber and eventually produces what is known as the asbestos body. This appears to be a defense mechanism of the lung. Numerous asbestos bodies can be found in the

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sputum of persons who have had only short and sporadic exposure to the dust. These persons are healthy and have no demonstrable signs or symptoms of asbestosis. Therefore, it seems more appropriate to use the term "asbestos" bodies rather than "asbestosis" bodies, signifying exposure to the fibers but not necessarily indicating disease.

If increasing quantities of the fibers are continually inhaled, the tissue reaction progresses, and a generalized, diffuse fibrosis gradually appears throughout the lower lobes of the lungs. With additional exposure, this

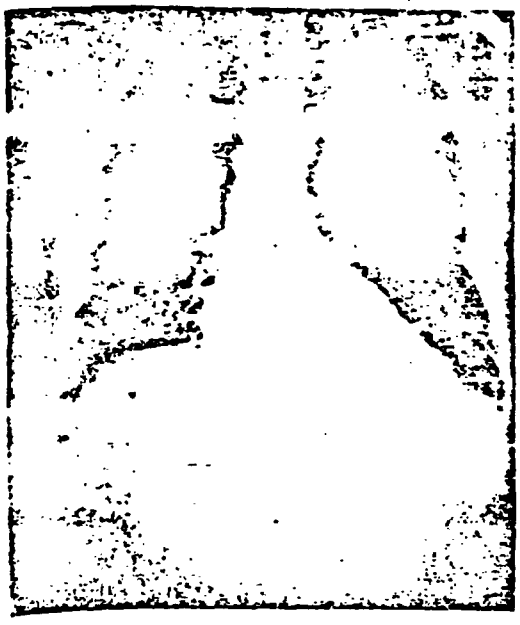


Fig. 1.—Early asbestosis.

fibrosis will spread to the other lobes, eventually causing respiratory embarrassment and finally cardiac failure.

The pulmonary fibrosis resulting from prolonged inhalation of asbestos fibers will produce a typical x-ray pattern. In early, or first-stage, asbestosis (Fig. 1), the x-ray shows a fine, diffuse, homogeneous infiltration throughout both lower lung fields. It should be noted that this infiltration is bilateral, that it is generalized at both bases, and that the nodular conglomerate patterns of other pneumoconioses, such as silicosis, are not seen in asbestosis. There is a considerable amount of pleural reaction associ-

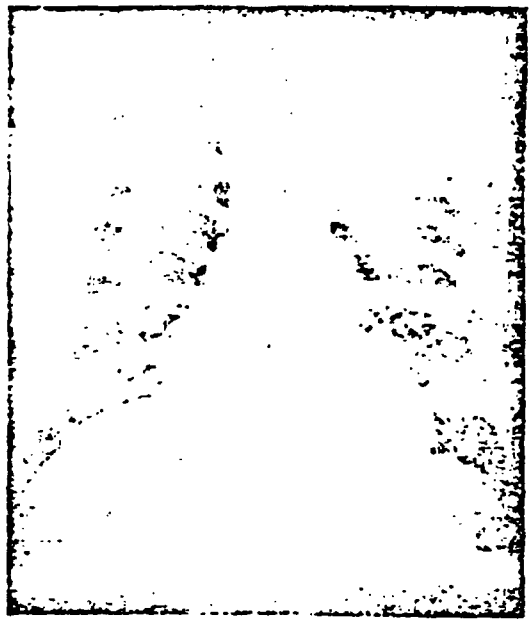
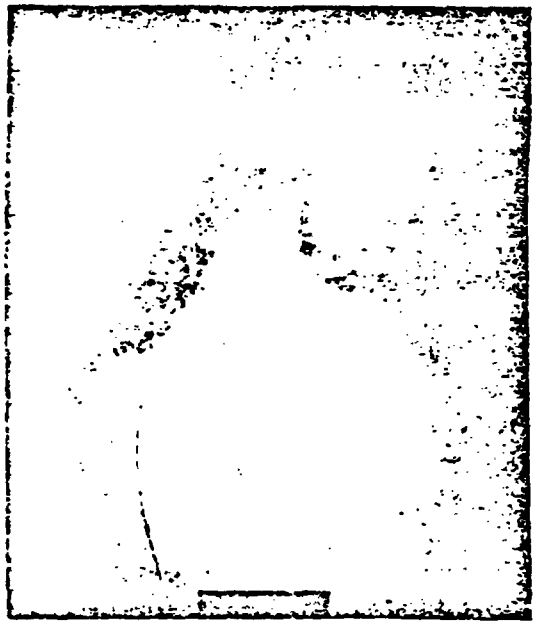


Fig. 2.—Moderately advanced asbestosis.

ated with this disease, which may account for the "ground glass" pattern which has been used to describe the typical x-ray picture.

In moderately advanced, or second-stage, asbestosis (Fig. 2), the infiltration has increased but still is confined to the lower lung fields. The "ground glass" pattern is

Fig. 3.—Far-advanced asbestosis.



more apparent, and the heart borders are becoming indistinct or shaggy. There is some irregularity of the diaphragmatic outlines and beginning obliteration of both the cardiophrenic and the costophrenic angles.

In far-advanced, or third-stage, asbestosis (Fig. 3), the infiltration still is homogeneous and bilateral, has spread to the middle and possibly the upper portion of the lung fields, but the apices remain clear. The cardiac outline is almost completely obliterated, as are the domes of the diaphragm and the costophrenic sulci. With this picture in mind, it is advisable to reiterate the observations of many physicians, namely, that the x-ray picture should never be used to estimate the presence or the extent of impaired pulmonary function or disability. Many cases with x-ray evidence of third-stage asbestosis have been known to carry on their usual work and live fairly comfortable lives for several years. On the other hand, no case of definite disability has been seen unless there was the typical x-ray pattern.

There is no typical clinical picture for asbestosis. The disease is insidious in its onset and slowly progressive with continued inhalation of the fiber. There is a gradual increase in cough and expectoration, some anorexia and weight loss, then slowly increasing dyspnea. Cyanosis and clubbing of the fingers are rare findings. There is evidence that asbestosis will not progress after exposure ceases, but this seems to be true only if the worker does not develop an intercurrent pulmonary infection.

It will be shown that asbestos workers are not predisposed to develop more intercurrent pulmonary infections than are found in other workers. However, when an acute pneumonitis develops in the presence of an established asbestotic fibrosis, the infection is slow to heal, relapses are frequent, and the patient may be more susceptible to subsequent pulmonary infections. While it is true that the disease is slow and insidious in its onset and that people with advanced asbestosis may lead relatively normal lives, eventually the heart begins to fail, and death from cor pulmonale rapidly follows.

Gregoire* has reported that pulmonary function studies on asbestos workers have shown that the chief physiological problem is that of a "tight" lung. The vital and maximum breathing capacities are lowered, expansion of the lung is difficult, and arterial oxygen saturation of the blood is diminished in some cases, indicating an impairment of gas transfer through the lung. Diffuse obstructive emphysema, so commonly seen in silicosis, is not apparent in asbestosis. These pulmonary function studies are of importance in the proper diagnosis of pulmonary fibrosis and the estimation of pulmonary disability. Of more importance is the fact that these tests can often help the clinician in directing the treatment of the case. Unfortunately, there are too few persons who are properly qualified today to carry out these tests and interpret the results.

An x-ray survey was made of one group of 708 employees working in an asbestos mill where the ore was dried, crushed, separated and graded, packed, and then shipped. Operations in this plant required the employees to rotate through various jobs; hence it was impossible to relate any x-ray changes to a particular job or to a specified dust concentration. At the same time it could be assumed that all members of the group had been exposed to varying concentrations of the dust. The chest x-rays of these employees were divided into three broad groups: (*a*) essentially normal lungs; (*b*) marked linear exaggeration (1-2) but no typical pattern of asbestosis, and (*c*) definite asbestosis.

Table 1 shows that of the 708 employees studied 649, or 91%, had normal x-rays. This is of interest because 204 employees, or 29% of the total group, had 10 or more years of service, and 2 men actually had worked more than 40 years in the dust.

Table 2 indicates that 52 of the 708 employees showed a marked increase of all peribronchial markings, although none had defi-

*Gregoire, F.: Pulmonary Function Studies in Men Exposed for Ten or More Years to Inhalation of Asbestos Fibers, read before the Seventh Saranac Symposium, 1952 (unpublished).

TABLE 1.—*Employees with Normal X-Rays*

Exposure, yr	1-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49
Employees, no.	258	151	94	40	44	16	5	2	1	1

nite asbestosis. Inasmuch as the majority of these men had essentially normal x-ray films early in their employment history, it is assumed that most of the later increased lung markings were associated with their subsequent dust exposures. Further indication of the length of exposure necessary to develop x-ray changes is seen when it is noted that 69% of this group had 10 or more years of exposure.

Table 3 shows that of the 708 employees 7 had developed definite x-ray evidence of asbestosis. These men exhibited various stages of pulmonary involvement, but all were working steadily at their accustomed jobs with no signs of disability. It is of interest to note that none had developed x-ray evidence of asbestosis with less than 20 years of exposure.

Frequently it has been stated that it takes from 5 to 10 years of exposure to develop asbestosis. The survey reported here concerned mill employees. It is possible that other operations might have different exposures with other disease experience. Factors which might influence this experience are not only the length of exposure or the concentration of dust in the air but also the fact that there is probably an individual susceptibility to the development of pulmonary fibrosis.

Medical literature has given considerable attention to occupational pulmonary disease, but very little has been reported on the occurrence of nonoccupational respiratory disease among those employed in the dusty trades. The following survey was made in order to determine the incidence of nonoccupational respiratory disease in another group, of 1561 men and women, working in the asbestos industry.

A review of absentee records indicated that no valid conclusions could be obtained from this source, because the reason for absence was given by the employee himself. Sickness often was used as an excuse to cover short absences for a variety of personal reasons.

It then was decided to study the claims submitted for sickness and accident insurance. All employees in the survey participated in a plan operated by an independent insurance company. Indemnification was made only after the nature of the illness had been certified by the treating physician.

The study included claims submitted over a three-year period for such illnesses as the common cold, sinusitis, pharyngitis, grippe, bronchitis, pneumonia, asthma, and pleurisy. Occupational respiratory diseases and pulmonary tuberculosis were excluded in the report.

Table 4 shows that of the 1561 employees in the survey group there were approximately equal numbers in dusty and nondusty occupations. Of all claims filed for respiratory diseases, 45% were for employees with dust exposure. Clinical observations for many years had given the impression that a dusty occupation in itself would not predispose a person to more nonoccupational respiratory disease than would a dust-free job. This survey shows that the rate of disease over

TABLE 2.—*Employees with P-2 X-Ray Readings*

Exposure, yr	1-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49
Employees, no.	5	11	8	10	11	4	1	1	0	1

TABLE 3.—*Employees with Asbestosis*

Exposure, yr	1-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49
Employees, no.	0	0	0	0	5	1	0	1	0	0

a three-year period was approximately the same in the two groups. In addition, there was no appreciable difference in the duration of illness in either group.

Animal experiments and clinical observations have shown that asbestosis does not predispose a person to the development of pulmonary tuberculosis, nor does it aggravate an apparently healed tuberculous lesion. In two isolated one-industry towns in the Province of Quebec where asbestos was mined and processed, the incidence of tuberculosis over a period of many years was no greater than in other isolated towns with comparable populations but without a dusty

Fig. 4.—Electron micrograph, chrysotile asbestos: × 4000.

trade.† In addition, the incidence of tuberculosis among asbestos workers was lower

Fig. 5.—Electron micrograph, amosite asbestos: × 4000.

than that among the general population of these mining towns.

Conflicting opinions and different reports make it extremely difficult to confirm or deny conclusively the causal relationship of asbestosis and carcinoma of the lung. Too often a common conclusion is drawn from observations and experiences with different racial groups, living in different parts of the world under variable socioeconomic conditions and working in diverse occupational exposures. To these variables should be added the fact that there are various types of asbestos fibers.

Fig. 6.—Electron micrograph, crocidolite asbestos: × 4000.

† Parrot, P.: Personal communication to the author.

TABLE 4.—*Nonoccupational Disease Experience*

Employees, total no.	1,561
Employees with dust exposure	807
Employees with no dust exposure	754
Claims for respiratory disease	27
Claims for respiratory disease with dust exposure	12
Claims for respiratory disease with no dust exposure	15
Average length of absence with dust exposure, wk.	3.8
Average length of absence with no dust exposure, wk.	3.1

Badollet ‡ has reported that there are several different types of asbestos fibers used by industry today, and these fibers have different physical and chemical properties. Figures 4, 5, and 6 are enlarged electron micrographs of three types of fibers used most commonly in industrial processes. The long, soft, and silky chrysotile fibers are found chiefly in Canada, while the amosite and crocidolite fibers which are shorter, stiffer, and brittler come from Africa.

Canadian experience with asbestosis has been limited to the chrysotile fiber. The majority of industrial processes in the United States use this fiber, but in recent years there is increased use of the amosite and crocidolite fibers. The British and European industries use greater quantities of these harsher fibers. Therefore, in trying to clarify the causal relationship of asbestosis and bronchogenic carcinoma, many variable facts should be clearly identified, especially the type of fiber used and whether or not other dusts were present in the industrial and environmental atmosphere.

SUMMARY

Respiratory disease experience among several thousand male and female asbestos workers in the United States and Canada is reported. Of all workers exposed to the fibers, very few develop asbestosis.

Asbestosis is insidious in onset and progresses slowly with continued exposure, causing respiratory embarrassment and cardiac failure.

Physiologically, asbestosis is the problem of the "tight" lung. Expansion of the lung is difficult, and there is impaired gas transfer through the lung. Diffuse, obstructive emphysema is not common.

There is a typical x-ray pattern which cannot be confused with other pneumoconioses.

An x-ray survey of 708 employees in a milling operation showed that the majority had normal x-ray patterns, that 10 or more years of exposure were necessary to produce x-ray changes, and that no cases of asbestosis were found who had worked less than 20 years in the dust.

The incidence of nonoccupational respiratory disease was not increased, nor was the illness more prolonged among workers exposed to asbestos dust than among nonexposed workers.

Asbestosis does not predispose to the development of tuberculosis, nor does it aggravate an apparently healed lesion.

There are several reasons for different opinions expressed concerning the relationship of asbestosis and bronchogenic carcinoma. Differences in asbestos fibers are noted.

‡ References 2 and 3.

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