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A REVIEW OF PNEUMOCONIOSIS

FURTHER ROENTGENOLOGICAL AND PATHOLOGICAL STUDIES*

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IN 1925, at the request of the Editor of the *American Journal of Roentgenology and Radium Therapy*, we reviewed the roentgenological and pathological aspects of pneumoconiosis in a paper presented before the American Roentgen Ray Society and published in the Journal[90] of that year. A more complete presentation of the subject was made in a monograph[91] published in 1926. The condition has aroused a surprising amount of interest throughout the civilized world in the short time that has elapsed since then, and especially in this country. Papers have appeared in great numbers, further experimental work has been done and numerous investigations have been carried out, especially here, by federal and state health bodies in connection with many industries. For these reasons it has seemed pertinent to present a further and up-to-date review of the subject to the body of roentgenologists.

This increasing interest in the subject is a recognition of the great importance of the condition of pneumoconiosis as a more or less necessary risk of commercial development in the progress of civilization during recent years. This has led to the enactment of adequate compensation laws and regulations governing the adoption of safety measures intended to eradicate or modify the essential dangers of occupations by many countries, notably the South African Union, Australia, New Zealand, Great Britain, Germany and one of the Canadian Provinces. In this country we have no federal laws directly applicable to the enforcement of controlling measures or compensation. In only a few states is pneumoconiosis a compensable disease. Some states enforce regulations directed toward protection of workers. It is a condition which is all too prevalent in America without adequate oversight, but largely because of the geographical extent of this country and the scattering of the more or less dangerous dusty industries, there is a lack of knowledge of that prevalence and its seriousness.

As the roentgenological examination is the most accurate and satisfactory method of detecting the condition from its earliest to its most advanced stages and of studying its progress and its incapacitating effects, it behooves us as roentgenologists to be familiar with all of the phases of the disease, and not solely with the roentgenographic appearances with which all of us are more or less familiar. We must play a large part in the education and the publicity which are so much needed to overcome the ignorance still existing, even on the part of employers and employees. We will be called upon for our part in the program leading to the enactment of satisfactory compensation laws which are sure to follow in time. In the meanwhile, civil suits are increasing in number and we must train ourselves in order that we may be able to testify, when called upon, honestly and justly for the benefit of all concerned.

It has been our endeavor in this presentation to avoid as far as possible any repetitions from our previous publications, and references to them will be made only when it may be necessary to better explain the newer phases of the subject.

NOMENCLATURE

While it still seems probable that silica (SiO_2) is usually the active fibrosing agent in the condition which we recognize as essentially a pulmonary fibrosis, adherence to the general term of pneumoconiosis still seems advisable just as much now as at the time of our previous presentations of the subject. Some other dusts undoubtedly

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bestosis is ample reason for the presentation of a general résumé of the entire industry, as well as the pathological, clinical and roentgenological aspects of the condition.

The dust evolved at the asbestos mines contains a moderate amount of silica. The harmful products of the asbestos factories are almost entirely silicates. Heffernan[42] believes that silicates such as asbestos undergo changes in the lungs quite like silica, only slower, and that asbestosis is a true silicosis. This is, of course, not proved.

The product was mined and the asbestos fibers were separated from the mineral and employed in the making of indestructable fabrics several centuries B.C. Attention is called by Cooke[17] to a cremation cloth described by Herodotus 450 B.C. Materials made of it have been used ever since then, but the most rapid development of the commercial industry has been during the past two or three decades.

Nature. Asbestos is a peculiar fibrous and crystalline mineral composed almost entirely of silicates, mainly of hydrate magnesium silicate, with varying admixtures of those of aluminum and iron. The general term is applied to all silicate minerals which have a fine fibrous structure that can be split up into long, flexible fibers for the purposes of spinning and weaving. The fibers will split up in a longitudinal direction almost indefinitely. The minerals differ in their exact constituency, especially as to the amount of iron and aluminum, in different countries in which they are found and mined. Much of the asbestos used in Great Britain and the United States is mined in Canada. Cooke[17] gives the following chemical composition of asbestos:

	per cent
Silica	40.87
Magnesia	41.50
Iron oxide	2.81
Aluminum	0.70
Water	13.55

The Industry. At the mines, the mineral is blasted, crushed and finely ground in machines. The crushed dust is then passed over vibrating machines. The heavier rock particles are separated by gravity and the asbestos fibers are aspirated out by vacuum fans into special chambers. The crude asbestos is then shipped to the various factories. All of these procedures are carried out at the mine bases because of the economy in shipment of the lesser bulk of the crude asbestos.

The mining part of the industry is attended by the elimination of a large quantity of dust, which contains silica as well as silicates. Pneumoconiosis is found among the workers. Very little has been written about the condition of these men. Recently we have had the opportunity of examining the roentgenograms of 58 men from one of the Canadian mines, some of which showed pneumoconiosis. As the mineral rock contains as high as 60 per cent silica, the mine worker's condition is due largely to this directly, although probably not entirely. The raw product which goes to the factories contains very little free silica.

The most dusty occupations in the industry are to be found in the factories, in which the work is done in inclosures where the asbestos is refined and is finally spun and woven into the various materials with which we are familiar. During all of the factory processes, a large quantity of fine dust is set free, but in varying amounts in the different ones. Most factories, at least in Great Britain, have devices for removing much of the dust.

Early Case Reports. The first report of a death resulting from asbestos dust inhalation was presented by Murray,[38] in 1900. The man worked in a factory for ten years, mostly as a carder, and was the sole survivor, before his death, of 10 other men who started work with him. The autopsy on this case revealed pulmonary fibrosis. Photomicrographs of sections of the lungs showed large spicules of asbestos, at least they were so regarded at that time.

The second case reported in medical literature was presented by Cooke,[16] in 1924. It was again reported by him in detail in 1927.[17] This individual stopped his

work in a factory after eighteen years of intermittent occupation. The cause of death was probably asbestosis and superimposed tuberculosis. We referred to this case in our previous contribution,[91] in 1926, as largely a tuberculous one, especially as it was the first available autopsy report in the literature. In view of the many deaths which have been reported since then, we must now concede that this death was probably the result of both conditions. Naturally, Cooke was the first to describe the unusual and characteristic asbestos bodies in the lungs, and the authoritative description of them was made in his second report of this case in 1927. These bodies will be described in detail later on.

The second paper and third public report was presented by Oliver,[87] in 1927. He reported the cases of two women who had died as a result of asbestosis after working thirty and eighteen years respectively. Neither individual had evidence of tuberculosis. It is interesting to note that the second of these cases stopped work after eighteen years' occupation, and did not develop definite symptoms until four years later.

The next report was by McDonald,[68] in 1927. He referred more fully to Cooke's case, and reported an additional one which died. He thought it reasonable to believe that in the former one the tuberculosis was a superadded infection. His own case did not have tuberculosis. In addition to the two types of asbestos bodies first described by Cooke, he added a third, all three of which are now recognized.

Seiler,[101] in 1928, reported another case who had worked twenty-two years in various capacities in asbestos factories, and presented the typical symptoms of the advanced case. No tubercle bacilli were found in the sputum. The roentgenographic appearances seemed not to have been unusual.

Stewart,[103] in 1928, reported two deaths among asbestos mill workers in South Africa with autopsy findings. The first man worked only one year and died of miliary tuberculosis. The lungs showed slight fibrosis in addition to the evidences of the tuberculosis. The second case worked two years in the mill and died. The physical signs suggested tuberculosis with fibrosis. Autopsy showed a moderate degree of fibrosis with nodules, but quite cellular, and not like the usual silicotic nodules. There was no evidence of tuberculosis. The various asbestos bodies were found in both cases. These cases are of interest first, because both presented definite evidences of asbestosis after one and two years' work respectively, and also the various bodies characteristic of the disease, and, secondly, for the reason that the second case presented the terminal symptoms and died of the condition after only two years' occupation.

Wood and Page,[130] in 1929, reported autopsy findings in the case of a woman who died of bronchopneumonia after nine years' work in a factory. There were no evidences of tuberculosis. The lungs showed diffuse fibrosis and thickened pleura. Numerous asbestos bodies were found in the lungs, but not elsewhere in the body. Wood and Gloyne,[129] in 1930, reported the autopsied cases up to four. Tuberculosis was found in three of these cases, and in two, bacilli had been found in the sputum before death. Haddow,[36] in 1929, reported on the deaths of Oliver's two cases, and on two others. He stated that the certificate of the last one, March, 1928, brought about the publicity and official recognition of the condition. Since then, the workmen's compensation act of Great Britain has been extended to include asbestosis.[93] Haddow states that one of Oliver's patients had tubercle bacilli in the sputum.

Simson[103] quotes Hoffman[48] as reporting 13 deaths in 1918 and Collis,[12] 5 deaths in 1910. These were in health reports, and will be referred to again under predisposition to tuberculosis.

Mereweather and Price,[73] in their recent comprehensive report on the asbestos industry state that approximately 2,200 individuals are exposed to practically pure asbestos dust in the factories of Great

Britain. These authors examined 363 individuals among the workers. Of this number, 230 had been employed less than ten years, and only 21 over twenty years. All but one of the 363 individuals had been working at the time of the examinations. Ninety-five, or 26.2 per cent, showed definite evidences of fibrosis, due apparently to asbestos dust solely. Their table of the time incidence of the condition is of interest:

Years at Work	Cases Examined	Showing Fibrosis	Per Cent
0 to 4	89	0	0.0
5 to 9	141	36	25.5
10 to 14	84	27	32.1
15 to 19	28	15	53.6
20 and over	21	17	80.9

This table does not apply to any one group of workers in the factories. The authors have collected also, outside of these statistical cases, records of 10 deaths resulting from asbestosis, 9 of which were verified by autopsy and the tenth by repeated clinical and roentgenographic examinations. Nine of these deaths occurred from 1927 to 1929. The exposure of the 10 cases varied from nine to twenty-four years.

At the time of this writing, Lynch and Smith[67] (1930) are among the latest extensive contributors to the subject, and their report has been the first one in this country, at least in medical literature, to deal specifically with the subject. One of their cases, a negro, died as a result of a gunshot injury after working twenty-eight months in a mill during a period of three years. His lungs showed definite interlobular, perivascular and peribronchial fibrosis, and also the various asbestos bodies. The second case, also a negro, died of lobar pneumonia after four and a half years of continuous work in a mill. His lungs showed everywhere a definite increase of young fibrous tissue, in addition to the various asbestos bodies. Reference will be made to other cases examined by these authors in the description of asbestos bodies. It is interesting to compare the findings in these cases and those of Simson with the clinical table of Merewether and Price. It is quite possible that protective measures played an important part in the incidence of the condition, as shown in the report of the last named authors.

Appearance of Asbestos Dust. Cooke[7,18] has described the microscopic appearance of the components of asbestos dust in somewhat the following manner:

(1) Small fragments of asbestos fiber. The bulk of the fiber is translucent and glistening, with here and there within it opaque particles and minute black granules. These appear to be actually parts of the fiber, but their appearance suggests different chemical composition and physical characteristics.

(2) Translucent spicules split off from the fibers.

(3) Black, brown and blue particulate matter. The black particles are iron. They are few in the finished asbestos product but are numerous in the dusts of the carding rooms. The iron in the finished product was only 0.1 per cent, whereas in the dust of the carding rooms it was 18.4 per cent.

Asbestos Bodies. This is, perhaps, the most interesting feature of pulmonary asbestosis. The various bodies found in the lungs of asbestos workers have been described in detail by numerous authors, notably by Cooke,[16,17,18] McDonald,[68] Simson,[104] Gloyne,[30] Merewether and Price[73] and Lynch and Smith.[67] The description given by Cooke[18] of the various asbestos bodies is as comprehensive as that of any other author and is abstracted as follows:

(1) Enormous amounts of fine granular pigment like that seen in the asbestos dust fiber. Much of this may be carbonaceous.

(2) Large or moderate sized angular particles, 10 to 360 microns in size, corresponding to the black, brown and blue particulate matter found in the dust. These particles are found in fibrous and necrotic areas, singly or in clumps. Because of their size, they might occlude small bronchi.

(3) Almost complete absence of the very

of course, as in any fairly well advanced case of pneumoconiosis. The most unusual feature of the condition seemed to us to be the fact that in both sets of roentgenograms the appearance seemed to begin more often on the *left* side than the right and progressed more on that side. This is the reverse of what is the rule in all other occupations we have studied. Merewether[72] states that it begins on the right side, as one would expect, and the cases we observed may have been examples of the exceptions which are occasionally observed in



FIG. 9. Pneumoconiosis in an asbestos worker. Male, aged forty-three, employed for seventeen years and a superintendent at the time of examination. There are two predominant types of fibrosis in this case—perivascular-peribronchial-lymph node and fairly advanced interstitial. The latter is indicated by the general haze toward the bases, and more on the left side. Note the partial obliteration of the left heart border. The size of the cardiac shadow is due to the old technique of many years ago. Diaphragm excursion ½ inch each side, ordinary respiration, and 1⅜ inch on forced respiration.

any industrial pneumoconiosis. We noted the same tendency to obliteration of the left cardiac border as mentioned by Wood, because of the diffuse fibrosis and the prominent trunk shadows. Wood states that the late stage may or may not show consolidations. Wood and Gloyne[109] state that the ground glass appearance in late cases on careful analysis resolves itself into a fine mottling and linear shadows which, toward the base, may look like cobwebs.

It is evident that the nature of these changes in the lungs requires the best type of roentgenograms for their portrayal. Also, the seriousness of what may seem to be minor changes to the uninitiated requires a roentgenoscopic study of diaphragmatic movements to make one realize the actual state of disability present. It is evident also, as Merewether states,[72] that an opinion as to the degree and intensity of an asbestosis fibrosis based upon a comparison of roentgenographic changes with those shown in standard silicosis films will be an underestimate.

Of the 363 cases examined by Merewether and Price, 133 had roentgenographic studies. Of these, 52 were said to have presented appearances of diffuse fibrosis and 22 were suspicious.

We have had the opportunity recently of examining in consultation the roentgenograms of 58 men employed at the mines in many occupations and during periods varying from eight months to forty years. Of this number, 15 showed definite evidence of pneumoconiosis, one was doubtful and 42 showed no lung changes. The longest worker showed a moderate nodular stage of the condition. The shortest times in which definite changes were indicated were four years for one first stage case, five years for another and five years for one early second or nodular stage case. The worst case showed a diffuse third stage fibrosis after thirty-one years' occupation.

Asbestos mine dust is not comparable to that of the factories for many reasons. Some silica is present, but there is not the great concentration of asbestos dust in the mine as compared to the factory. From our own experience so far, we would not regard the hazard at the mine as very great.

Prognosis. It seems to be the rather general opinion that the prognosis in pulmonary asbestosis among factory workers is to be regarded as grave without adequate protection, in a large number of cases

among those who work where there is great concentration of dust. The condition is apparently progressive even after cessation of occupation. Merewether and Price state that with continued exposure to high concentration dust fibrosis may be fully developed in from seven to nine years and death may result in about thirteen years. With less concentration, fibrosis may not be fully developed for fifteen, twenty or twenty-five years. Inhalation of dust in high concentration results in a more marked degree of fibrosis in a shorter time than when the concentration is lower.

Predisposition to Tuberculosis. The condition of pulmonary asbestosis being a new child in the pneumoconiosis family, a great amount of interest centers around the incidence of tuberculosis. Among the early case reports referred to in the beginning of this occupational section, pulmonary tuberculosis was reported as an accompanying condition in a few instances. Cooke's case had advanced tuberculosis. Simson reported one case which died with tuberculosis and one without evidences of the infection, He thought that we could not as yet formulate any definite opinion as to the frequency or the predisposition. Simson quoted Hoffman[48] as reporting 13 deaths among asbestos workers, 3 of whom had tuberculosis. He also quoted Collis[12] as reporting 5 deaths from tuberculosis among less than 40 workers at a factory. Wood and Gloyne[129] referred to 3 cases showing evidences of tuberculosis at autopsy. Haddow[46] observed many asbestosis cases and among them 4 died. He did not believe that the condition predisposed to the infection, at least not as a rule. Merewether and Price stated that asbestosis differs from silicosis in the distribution of the fibrous tissue, in more rapid development, in roentgenographic features and possibly in a *lessened susceptibility to tuberculosis*. Merewether[77] states that out of 3[illegible] cases examined, evidence of active tuberculosis was found in three. He thinks that while there is no outstanding susceptibility proved, he does not agree that the question has been finally settled as yet.

Compensation Laws. Legislation in many countries and states provides for compensation for silicosis, which is a condition produced by silica. Asbestos is a silicate, and if we are to regard asbestosis as a silicosis, as has been suggested but not proved, the latter condition is only indirectly caused by the asbestos. Certainly the legal status of asbestosis as silicosis has not been established. In some instances asbestosis has had to be made a compensable disease.

Hard Rock Mining. This group of workers comprises the largest engaged in any single hazardous industry throughout the world, provided the rock is of a siliceous nature. The large amount of dust evolved in the various processes of mining is always dangerous unless adequate protective measures are adopted. The drillers and those working with them or near them are prone to be the most exposed. If the fine dust is allowed to escape, it remains suspended in the air for hours. Blasting always stirs it up. Without protection, the usual typical symptom-complex and the roentgenographic appearances develop early and progress rapidly, and the workers are predisposed to tuberculosis. More has been written upon the hazards of this industry than those of any other. Numerous writers have been extensively quoted elsewhere. Many more or less similar industries and their hazards have been described in detail, such as the drilling in coal mines, granite cutting and sandstone quarrying.

The most detailed descriptions of hard rock mining have emanated from South Africa through individuals associated in medical work or other ways with the Rand gold mines. These mines are so often referred to in the literature that a short abstract of the description of the region and of the character of the rock may be of interest. Cluver[10] states that the first gold mining in South Africa started in the Transvaal in the 70's, and the first gold was taken from the Witwatersrand in 1885.