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PNEUMOCONIOSIS: A STUDY OF 379 CASES

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A survey was recently conducted (while the author was on duty at Veterans' Administration Facility, Sunnyside, N. Y.) to determine the relative incidence of pneumoconiosis in beneficiaries of the Veterans' Administration. The data for the study consisted of questionnaires, formulated by the chief, division of postgraduate instruction and medical research, which had been sent to and executed at field stations, in relation to beneficiaries who had been exposed to occupational dust hazards; radiograms of chests of affected beneficiaries; and, in a few instances, tissues from affected beneficiaries who had died.

A total of cases so collected from the field stations of the Veterans' Administration was 379. The beneficiaries in this series had been variously occupied: As miners of metal ores (lead, copper, gold, silver, iron, quartz), coal (anthracite and bituminous) miners, brickyard laborers, tunnel laborers, rock drillers, street cleaners, laborers on various types of construction work, automobile factory employees, blacksmiths, grinders, stonecutters, cement workers, railroad switchmen, machinists, sandblasters, pottery workers, quarrymen (limestone, sandstone, granite, marble, slate, etc.), laborers on stone crusher, tobacco-factory employees, bricklayers, silk-mill employees, steam-engine firemen, asbestos workers, molders, grain-elevator employees, textile workers, steel-mill employees, stone masons, grocery dealers, farmers, aluminum melter, clerk, tile presser, iron workers, foundrymen, carpenters, metal polishers, glass workers, smelter workers, and powder-factory employees. In a few cases no occupational history was given.

Modern study of dusty occupations has led to the rather definite conclusion that there are only two forms of dust which really cause pulmonary disease, namely free silica (SiO_2) or silicon dioxide and asbestos (magnesium silicate). Talc, also a silicate of magnesium, is closely allied chemically to asbestos and is under suspicion. As yet, however, it has not been definitely proven to be a factor in the production of lung disease. Coal-dust causes pigmentation (anthracosis) and occasionally slight fibrosis, but this has been proven to be relatively harmless from a pathological standpoint. Dust from the various minerals commonly mined is not considered a factor in the production of the disease. The silica-bearing rock encountered in mining is the source from which the disease is contracted. Workers in the cement industry, although exposed to dust, are not affected, because the materials which they handle contain little or no silica. Most of the various clays used in the pottery industry are innocuous, and there is some reason to believe that some of them modify or even inhibit the development of silicosis. Coal dust is also believed by some observers to modify or inhibit the progress of the disease. It

should not be understood from this that there is no dust hazard in the pottery industry, because certain employees in this industry are subjected to high concentrations of silica-bearing dust, especially the grinders, polishers, and furnace men. Dusts from hay, grain, flour, etc., encountered on farms, in grain elevators, mills, and bakeries constitute no real hazard so far as the production of chronic lung disease is concerned, although they may set up an acute bronchitis. They would also probably aggravate an existing tuberculosis. The same may be said of the dusts encountered in jute and textile mills. Road and street dust cannot be regarded as hazardous, as the amount of silica in such dust is very small and in low concentration.

After study of the data submitted, 171 cases of this group were classified (either definitely or probably) as having lung disease due to dust inhalation, either uncomplicated or complicated by infectious disease. The histories in many of these cases were rather inadequate, and, without a detailed history, it is difficult to form a definite opinion in the individual case. For instance, a man may have worked in a mine for many years and still have had little or no exposure to disease-producing dust. A history of this type of case should show not only where he worked, but what he did in detail, how long he did it, and to what form of dust he was exposed. In other words, the production of disease by dust depends upon the form of dust, plus its concentration, plus the time of exposure to it. Some of the films submitted were of poor quality and the lesions were not as sharply outlined as they should have been. However, the general average of the films was quite good.

The characteristic lesions in the lungs in silicosis are fibroid nodules varying in size from 2 to 5 mm. in diameter. These are discrete in the early cases, tending to become confluent in the far-advanced cases. In the extremely far-advanced cases these nodules tend to lose their individual identity in dense masses of fibrous deposit. These cases are almost invariably associated with infectious disease, usually tuberculosis.

The cases studied were classified as:

1. Silicosis, first stage (S1): Showing accentuation of the lung markings with fine discrete generalized nodulation.
2. Silicosis, second stage (S2): Showing marked extensive nodulation, still discrete, the individual nodules being generally larger than in the first stage.
3. Silicosis, third stage (S3): Showing very extensive nodulation with large nodules tending to become confluent.
- 4, 5, and 6. Silicosis, first, second, and third stages, plus infection: These cases were of the same types as 1, 2, and 3, with definite evidence of accompanying infectious disease.
7. Silico-tuberculosis (S TB): Far-advanced cases in which the individual nodules had lost their identity in dense fibrous masses of infiltration. These are considered the resultant of silicosis combined with tuberculous infection, but it is impossible to determine a separate background for either process.
8. Cases without definite evidence of silicotic involvement, but with definite evidence of lung disease, the appearance of which indicates a background of infection only.

9, 10. Fibrosis: Cases showing no nodular deposits, with slight or well-marked accentuation of the lung markings. This condition cannot be regarded as characteristic of silicosis; it is often present in cases of respiratory infection, acute or chronic, in passive congestion due to cardio-renal disease, or in other conditions. These linear shadows are also subject to normal variations in the adult chest.

11. Asbestosis: This condition has been termed "silicatosis" in contradistinction to silicosis. Early cases of asbestosis are difficult to recognize as such. The radiographs usually show only minor changes indicating an interstitial fibrosis, similar in appearance to those cases in classifications 9 and 10. The more advanced cases show what has been described as a "ground glass" appearance; that is, a diffuse haze over the lower lung fields, together with a marked accentuation of the lung markings, but with little or no nodular deposit. This haze may be so dense as to obscure the cardiac borders and the domes of the diaphragm.

12. Negative: Cases in which there was no evidence of lung disease.

The following table shows the incidence of industrial lung disease according to the occupations represented:

	S1	S2	S3	S1-Inf.	S2-Inf.	S3-Inf.	S TB	Asb.	Total
Miners, metal ore ¹	11	7	1	11	20	7	15		72
Miners, coal ¹	22	3	1	13	7	4	7		57
Granite workers	1	1		3	1	1	2		9
Rock drillers				2	1	5			8
San blasters					2	1	1		4
Stonecutters		1			1		1		3
Pottery workers					2	1			3
Stone crushers	1			1					2
Molders	2								2
Bricklayer, furnace							1		1
Smelter	1								1
Stone mason				1					1
Asbestos miner								1	1
No industrial history ²				1	3				4
Carpenter ³						1			1
Aluminum molder ⁴								1	1
Sheet-metal worker ⁴					1				1
Total	38	12	2	32	38	20	27	2	171

¹ It will be noted that there is a preponderance of miners shown in this table. Hard-rock miners constitute the largest group of workers in dusty occupations. These could be subdivided into smaller occupational groups if more information were available.

² Although there was no industrial history given in these cases, the radiographic appearance was quite typical of silicosis and they were so classified.

³ The industrial history of these cases is open to question. It is believed that more detailed histories would have furnished more appropriate information.

⁴ Aluminum molders wear large asbestos aprons and gloves while working over the crucibles. The molten metal splatters on these, thus releasing considerable quantities of asbestos dust. In the rooms where a number of these men are at work there is probably a considerable concentration of this dust. It also seems likely that the friction of these garments incidental to the work would be an added factor in the release of asbestos particles.

Diagnosis.—The importance of a detailed occupational history cannot be emphasized too strongly. The symptoms exhibited by cases of uncomplicated silicosis are few and often absent. Cough and expectoration are not troublesome. In the far-advanced cases there may be dyspnea, anorexia, loss of weight, and cyanosis as a terminal manifestation. The same may be said of physical signs. Decreased resonance of various degrees may be elicited in the more-

advanced cases, together with signs of emphysema. The dearth of evidence of disease shown by symptoms and physical signs is striking when compared with the extent of disease shown by the radiograph. X-ray study is the most definite and satisfactory method of diagnosis. This, together with a carefully taken history, is absolutely necessary in making the diagnosis and in following up the individual cases. The great hazard in these cases is an intercurrent acute respiratory infection, such as pneumonia or influenza. In the cases complicated by tuberculosis it is often difficult to differentiate the shadows of the two diseases. Often, cases of tuberculosis will show nodular deposits very similar to those of silicosis; the appearance of the shadows of each disease may also be modified by the other. In these cases detailed histories are even more important, and the cooperation of the clinician and the roentgenologist is indispensable. Of course in these cases the symptoms, physical signs and laboratory findings of tuberculosis are present.

Of the 171 cases shown in the table, 52 or about 30 percent were classified as uncomplicated silicosis or asbestosis; 38 of these were in the first stage, 12 in the second stage, and only 2 were classified in the third stage. A post-mortem examination of one of the last cases showed a far-advanced silicosis without evidence of infection. Nevertheless, the pathologist, who was one of the reviewers of this series of cases, felt that there was probably a background of infection. If this case had not come to autopsy, it would have been classified as silico-tuberculosis. The remaining 119 cases all showed evidence of complicating infectious disease, more or less extensive and in most cases probably tuberculosis.

While this is not an extensive series of cases and no definite conclusions can be drawn from it, it is believed that it agrees with most observers in that it supports the belief that disease of the lungs due to dust inhalation increases the susceptibility of those organs to tuberculosis, and that this susceptibility is increased in direct ratio to the extent of the industrial disease. The reviewers are of the opinion that the number of cases of industrial disease in this series is very small, considering the number of cases of pulmonary disease coming under the observation of medical officers of the Veterans' Administration. Only 379 cases were considered as potential cases, and nine of these were submitted in duplicate from different facilities. This review has shown that there is a need for more careful and detailed histories in cases of this type. Medical officers should familiarize themselves with this disease; they should inform themselves of the various sources of disease-producing dust, and should know what types of occupations are hazardous from this standpoint.

In the study of this series of cases, the writer was associated with Dr. Leroy U. Gardner, pathologist and director of the Saranac Lake Laboratory for the Study of Tuberculosis, and Dr. Homer L. Sampson, roentgenologist, Trudeau Sanitarium, both of Saranac Lake, N. Y. A composite opinion of the three reviewers was appended to each questionnaire, after careful study of the available data. These reviewers were much interested in this study, and wish to express their thanks to the officials of central office, and to the medical officers of the various facilities, who cooperated in furnishing the data.