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A REVIEW OF OUR PRESENT KNOWLEDGE OF PNEUMOCONIOSIS, BASED UPON ROENTGENOLOGIC STUDIES, WITH NOTES ON THE PATHOLOGY OF THE CONDITION*

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THE word "pneumoconiosis" is of Greek origin, and implies a condition of the lungs arising from the inhalation of excessive quantities of dust. The fact that damage to the lungs may be wrought by the inhalation of certain dusts has been recognized for many centuries, but the exact type of dust or dusts responsible for the pulmonary changes and the nature of the pathological processes induced are of comparatively recent knowledge. Systematic roentgen-ray studies, painstaking pathological investigations following autopsies and abundant animal experimentation throughout the world during the past ten or twelve years have led to a very exact understanding of this most interesting condition.

America is far behind many other countries in the methodical study of pneumoconiosis and the practice of preventive measures. Comparatively few systematic investigations have been carried out. In 1916, one of us, in conjunction with Miller and Landis, instituted clinical and roentgenological investigations of the dust workers in a large number of dusty industries, and our preliminary results were published in 1918.[1] These studies have been carried on intermittently ever since.[2] The United States Public Health Service and the Bureau of Mines have been carrying out a routine study of dusty occupations, especially mining, for even a longer period. Their first report including roentgen-ray observations was made by Lanza and Childs[3] in 1917. Jarvis and his coworkers[3-14] have contributed much valuable data from their studies of the granite cutters of Barre, Vermont. The experimental work of Gardner,[15-25] Permar[43] and others has added much to our knowledge of the pathological processes involved in pneumoconiosis. We can be justly proud of Miller[27-32] for the wonderfully clear knowledge he has imparted to us of lung histology, and particularly of the lymphatic system, upon which to base our conception of pulmonary pathology. Notwithstanding all these and many other American contributions, the amount of work accomplished in the study of pneumoconiosis in this country cannot compare with the extent to which the condition has been studied abroad, especially in South Africa, Australia and Great Britain. Naturally, in the latter country, especially, the concentration of dusty industries has been a definite aid to all observations. In those countries not only

* Thirteenth Annual Meeting, American Roentgen Ray Society, Washington, D. C., Sept. 22-25, 1925.

diaphragms. There were no symptoms referable to the appearance. On investigation they found she had worked for three years handling and helping in printing of cigarette packages on which there was considerable bronze work. The material was used in liquid form and was frequently spilled and dried, leaving a fine dust. After a day's work she could taste metal and her sputum was said to be brownish. We are familiar with this case and can vouch for the above statements.

In connection with metal industries we have examined a few stove plate moulders and foundry men whose lungs have showed well-marked second stage changes after a number of years' occupation. We have regarded the appearances as most likely due to sand.

BRICK WORKERS

The constituents of brick are suitable for the production of pneumoconiosis. Clays contain hydrated aluminum silicate with more or less sand or free silica and a few other ingredients. Sand is a necessary constituent of brick clays and may constitute quite a high percentage. Fire brick mixtures usually contain more sand than the clay for building bricks, and the constituents are often mixed dry.

Middleton[38] reported silicosis in 18 silica brick makers. Purdy[41] quotes Collis[40] as stating that almost pure silica occupations like silica brick making have a high mortality from tuberculosis, while with workers in mixtures of silica and clay as used in ordinary bricks, the mortality is very low. We have had no personal experience with pneumoconiosis in brick workers. One man who had worked continuously as a brick maker for forty years was recently examined by us and no changes of a silicotic nature were found in the lungs. On the other hand, we have been told by other brick workers that many of their companions who had worked in the industry for a long time were obliged to stop because of lung troubles. We have been informed of one individual whose occupation was the replacement of fire brick in a steel mill furnace and who showed clinical evidence of advanced pulmonary fibrosis after many years of such occupation. The dust was always very dense and he had worked longer at the job than any other man he knew, all the others having been obliged to stop because of lung troubles.

CEMENT WORKERS

Anyone who has seen a cement factory in operation knows that it is one of the most dusty of all places, and yet the industry cannot be regarded as a hazardous one from the standpoint of pneumoconiosis. There are several distinct occupations in this industry, especially those of coal handling, rock grinding, cement grinding and the packing and handling of the finished product. With Miller and Landis[1] we examined 20 cement workers several years ago. Five of these showed possible first stage changes and the other 15 definite second stage appearances. The earliest second stage changes were found in a packer after seven years occupation and were slight only. Of the men longest at work, a cement grinder showed slight second stage nodulation after eighteen years and a kiln room man presented very definite nodules after nineteen years. All occupations showed some changes. One would expect the greatest effect among the rock grinders, although the rock contains but a comparatively small percentage of free silica. We cannot, of course, tell what changes might have been found had any of the men been working thirty or forty years. The second stage appearances were variable, some of the nodular shadows being small and discrete and others "soft" and comparatively large.

ASBESTOS WORKERS

This is another very dusty industry. Asbestos is a magnesium silicate, and the rock that is crushed contains more or less free silica, to which the workers may be exposed all the way through the process of manufacture to the finished product. Cook[40] describes asbestos as a calcium and magnesium silicate, with 40 per cent of free silica among other constituents. This percentage no doubt varies in different localities. Cook reported one asbestos worker who died of a pulmonary condition

after twenty years' occupation. The roentgenograms showed extensive pulmonary fibrosis. Macroscopically the lungs were densely fibrosed and showed caseation and cavities, and microscopically tubercle bacilli were found in caseated areas. From our own experience we would regard this as largely a tuberculous case.

With Miller and Landis[4] we examined 17 asbestos workers, 2 of whom showed first stage changes and the other 15 definite second stage appearances. Of the men longest at work, one after seventeen years' occupation showed very definite diffuse, "soft" spots throughout both lungs, and another showed about the same appearance after fourteen years' occupation. Very slight nodular shadows were found in one man after only two years' occupation. Most of these second stage cases showed also well-marked first stage appearances still present, indicating a persistence of free drainage hilumward. In all instances the nodular shadows were characteristically "soft" and varied considerably in size.

CARBORUNDUM

We have been unable to obtain any definite information in regard to the condition of the lungs of carborundum workers. Holmes,[46] who investigated the industry at Niagara Falls, N. Y., regarded the dust as injurious to the lungs, and the reason why workers were not affected to any great extent was because of the labor turnover. Few laborers remained at work sufficiently long to acquire serious damage. He described the entire industry as exceedingly dusty and a dust analysis showed that the number of small particles were far beyond a safe limit. The shaving rooms where the abrasive wheels were cut down before going to the baking kilns showed the greatest hazards.

Gardner[45] described the carborundum plants as very dusty; and 75 per cent of the particles were 1.5 microns or less in size, and therefore suitable for cellular disposition. By animal experimentation he found that carborundum dust reached the tracheobronchial lymph nodes and did produce fibrosis there. He was unable to find a true fibrosis in the lung parenchyma, but if extensive inhalation tuberculosis was developing, a decided pulmonary fibrosis resulted. He concluded that carborundum dust was harmless to the normal lung.

In the manufacture of carborundum, a mixture of sand and coke is fused in an electric furnace. The resulting product is a crystalline and amorphous silicon carbide (SiC), which is said to be silica free. After leaving the furnace the crude product is crushed and ground. In making abrasive wheels and stones the particles are held together by some substance like porcelain, then shaved or cut down and fired in a kiln. The substance holding the carborundum particles together is likely to contain silica, which may be set free when the wheels or stones are used for abrasive purposes.

LIME DUST

Nicholson[48] states that the inhalation of lime dust in the Derbyshire region does not produce any extensive lung fibrosis. It is soluble in the body fluids. Roentgenograms showed a diffuse mild fibrosis probably due to a small amount of free silica present. Purdy[41] in his review of the literature on silicosis found that dusts like plaster of Paris and lime stone were regarded as devoid of all harmful action on the lungs. We have had no experience in the examination of lime stone workers.

MISCELLANEOUS INORGANIC DUSTS

We found second stage appearances in one charcoal burner of many years' occupation, but the exciting factor in this individual is unknown.

One flint grinder in a sand paper works showed advanced third stage changes after ten years' occupation (Fig. [illegible]).

One worker in a plaster works showed third stage fibrosis.

One man who had worked in a wallpaper factory running a wallpaper machine for six years showed second stage appearances.

We have been informed that pneumoconiosis has been observed among jute workers, and also in cotton mill workers, especially those who open the bales. No doubt there is a certain amount of free

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