PLAINTIFF'S EXHIBIT

MET-878

Reprinted from The Journal of the American Medical Association
August 19, 1933, Vol. 101, pp. 583 and 584

ETIOLOGY OF SILICOSIS

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The Committee on Pneumoconiosis and the Committee on Standards of the American Public Health Association, at a joint meeting in November, 1932, adopted the following definition of silicosis:

> Silicosis is a disease due to breathing air containing silica (SiO_2) characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present) and by characteristic x-ray findings. The disease is divided arbitrarily into first, second and third stages for convenience of description and possible compensation purposes.

I wish to make clear that in this discussion we are concerned with true silicosis and not with other forms of pneumonoconiosis due to the inhalation of dusts containing combined silica in the form of silicates or of organic dusts. Numerous dusts will produce a pneumonoconiosis demonstrable by x-rays; but just as these other dust diseases of the lung differ in their exciting cause, so do they differ in the pathologic changes that characterize them. Silicosis is an important disease because it is widespread in industry, and, because, as a result of the peculiar susceptibility to tuberculosis which it induces, it is attended by a high rate of mortality.

DUST

The primary cause of silicosis, then, is free silica, inhaled in the form of dust. Extensive research and experimentation have clearly demonstrated that this

Read before the Section on Preventive and Industrial Medicine and Public Health at the Eighty-Fourth Annual Session of the American Medical Association, Milwaukee, June 14, 1933.

dust, in order to achieve its harmful effects on the lung tissue, must be in a fine state of subdivision; that the dust particles must, in fact, be less than 10 microns in their greatest diameter. Most of the dust penetrating the alveoli is less than 5 microns in diameter.[1] It is, therefore, a dust invisible to the unaided eye, and its estimation, in any specific instance, depends on a definite technic of dust sampling and counting. In this country that technic has been defined and standardized by the United States Public Health Service.[2] Dust concentrations are measured in terms of million particles per cubic foot.

Silicosis is a disease that develops slowly — very slowly as a rule, most frequently over a period as long as ten years. It may develop in two years, perhaps even less. The rate of development is dependent largely on the dosage of silica, and this in turn is determined by three variables—the amount of dust in the inspired air, the amount of silica in the dust, and the extent of exposure. Obviously, exposure to a concentration of forty million particles per cubic foot, containing 90 per cent silica, for eight hours a day, day in and day out, will produce silicosis much more rapidly than the same degree of exposure for two days a week or daily exposure to ten million particles containing 40 per cent silica. Actually, as these factors may and do vary in different localities, so do they mold the clinical picture. This accounts for the occasional dogmatic and varying statements made by observers familiar only with the conditions prevalent in their own vicinity. It has been claimed by some observers that certain substances, which may be present in silica-containing dust but not in combination with the silica, may advance or retard the development of silicosis. The presence of clay and other inorganic substances has been stated to prevent the development of silicosis,[3] while in other instances certain alkalis are credited with greatly speeding up silicosis, so that it may develop into a full blown case with fatal termination in the space of months rather than

1. McCrae, John: The Ash of Silicotic Lungs, Pub. 3, South African Institute for Medical Research, 1913; General Report of the Miners' Phthisis Prevention Com. S. A. 1916, appendix 7, pp. 131-134.

2. Katz, S. H.; Smith, G. W.; Myers, W. M.; Trostel, L. J.; Ingels, M., and Greenburg, L.: Comparative Tests of Instruments for Determining Atmospheric Dusts, U. S. Pub. Health Bull. 144, January, 1925.

3. Hefferman, Patrick: Exposure to Silica Dust Without the Occurrence of Silicosis, J. Indust. Hyg. 8:481 (Nov.) 1926.

years.[4] Further study is needed to evaluate these observations.

INFECTION

The distinguishing characteristic of silicosis is the extraordinary susceptibility to tuberculosis which it induces in its victims. Years ago, Dr. Summons[5] of Bendigo, Australia, stated that the gold miners there who contracted silicosis died of tuberculosis and that observation has been confirmed in every part of the world where silicosis is prevalent. In my own experience I can recall but one patient who died from his pulmonary fibrosis. When a tuberculous infection is implanted on a silicotic lung, it intensifies and accelerates the formation of fibrotic tissue, and the pathologic vicious circle thus set up may continue for some time before the infection becomes clinically manifest. In other words, tuberculosis is more than a terminal infection in silicosis.

Studies made at the Picher Clinic by the United States Bureau of Mines and the Saranac Laboratory have shown that infection also occurs in silicotic lungs, as a result of a rather complex symbiotic union of the ordinary anaerobic bacteria of the upper respiratory tract, principally fusiform bacilli and Vincent's spironemes. Research into this phase of silicosis is now being carried on at the experimental laboratories of the Bureau of Mines in Pittsburgh and at the Saranac Laboratory. Thus, the principal etiologic factor in silicosis is silica in the form of dust, with the added factor, in a great majority of all but early cases, of tubercle infection, with the rôle of other infections not as yet clearly established.

CONTRIBUTING CAUSES

I do not believe that individual susceptibility plays an appreciable part in the etiology of silicosis in normal

4. Chapman, E. M.: Acute Silicosis, J. A. M. A. 99:1439 (April 23) 1932. Kessler, W. H.: Silicosis in the Abrasive Powder Industry, J. Am. Pub. Health A., December, 1931, p. 1391. Kilgore, E. S.: Pneumonoconiosis: An Unusually Acute Form, J. A. M. A. 99:1414 (Oct. 22) 1932; discussion, July 9, p. 166. Silicosis: Acute Cases Caused by Cleaning Powder, Bull. 541, U. S. Bureau of Labor Statistics, p. 353.

5. Summons, W. E.: Report of Miners' Phthisis submitted to Committee of the Bendigo Hospital, Bendigo, Victoria, 1907; quoted in Silicosis, Records of the International Conference at Johannesburg, August, 1930; International Labour Office, Studies and Reports, Series F, No. 13, Geneva, 1930, p. 303.

0110

individuals. Pre-existent pulmonary diseases, especially tuberculosis and pneumonoconioses, other than silicotic, tend to hasten and aggravate the development of silicosis. I have observed that coal miners who presumably had anthracosis and who then went to work in hard rock metal mines succumbed to silicosis much more rapidly than their fellow workers. The published reports of the Picher Clinic bear this out.[6] Age undoubtedly has an influence on the development of silicosis. The Picher studies show, for 8,000 men examined, that whereas all the patients with first stage silicosis had worked an average of thirteen years, those who had started mining after the age of 40 had worked an average of only 7.83 years. Nasal obstruction or other conditions causing mouth breathing are naturally credited with increasing the incidence of silicosis. The Picher reports do not show much definite relationship between the incidence of silicosis and previous diseases, but this was possibly in part due to the difficulty in securing accurate histories. Syphilis, however, definitely enters into the picture. All the evidence tended to show that men with a 4 plus Wassermann reaction developed silicosis more quickly than the Wassermann negative group, and their disease ran a more rapid course.

The influence of race on the incidence of silicosis is probably of minor importance, except perhaps as it may involve resistance to tuberculosis. There is some recent evidence which tends to show that American Negroes contract silicosis in a shorter time with a quicker fatal termination than do white men; but this may be but a reflection of a higher tuberculosis ratio, a higher syphilis ratio, or both.[7]

No correlation could be established in the Picher studies,[6] at least, between good or bad living conditions and the incidence of silicosis. Occupation, so far as it involves exposure to siliceous dust, is of the utmost importance. Silicosis exists only as an occupational

6. Sayers, R. R.; Meriwether, F. V.; Lanza, A. J., and Adams, W. W.: Silicosis and Tuberculosis Among Miners of the Tri-State District of Oklahoma, Kansas and Missouri: I, for the Year ended June 30, 1928, Technical Paper 548, U. S. Bureau of Mines, Washington, D. C., Department of Commerce, 1933. Meriwether, F. V.; Sayers, R. R., and Lanza, A. J.: II, for the Year ended June 30, 1929, Technical Paper 552, U. S. Bureau of Mines, Washington, D. C., Department of Commerce, 1933.

7. Everett, F. R.: The Pathological Anatomy of Pulmonary Tuberculosis in the American Negro and in the White Race, Am. Rev. Tubere. 27:411 (May) 1933.

disease and has been identified with metal mining for centuries. The introduction of pneumatic tools for drilling, cutting or chipping siliceous rock has occasioned a world wide distribution of silicosis, especially in mines, quarries, tunnel construction and stonework of various kinds. Sandblasting involves a most serious hazard and foundries and potteries contribute their quota of cases, as do processes involving grinding and vitreous enameling; the use of artificial grinding wheels has greatly diminished the hazard formerly associated with the use of natural stone wheels. Many other industrial processes, too numerous to list, involve exposure to siliceous dust.

Silicosis is a widely spread occupational disease and its peculiar relationship to tuberculosis, the difficulties of diagnosis, and the extreme difficulty associated with efforts for prevention and control, all combine to make it a disease of major importance. For this reason, I desire to emphasize most strongly the necessity for the precise recording of the patient's occupation, both by physicians and hospitals, especially in cases which present pulmonary symptoms. Not merely the patient's present but also his previous occupations should be ascertained.

Recent investigations and studies have revealed various types of pneumonoconiosis which tend to confuse further both diagnosis and prognosis. This makes imperative an accurate marshaling of all the etiologic factors in the consideration of any case in which the inhalation of silica dust is concerned.

0120