

ASBESTOSIS

PLAINTIFF'S EXHIBIT

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NEW YORK

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Asbestosis is a pneumoconiosis caused by the inhalation of asbestos dust. It is distinct from silicosis in its pathology and clinically. Whether asbestosis will remain a distinct form of pulmonary dust disease or will prove to be of a type common to a number of dusts remains to be seen. So far, it is the only pneumoconiosis, other than silicosis, that has received any considerable amount of study from pathologists, clinicians and industrial hygienists.

Whereas silicosis has been recognized for many centuries, asbestosis is a newcomer. Asbestos (Canadian) is a hydrated magnesium silicate containing no free silica but about 44 per cent of combined silica, 43 per cent magnesium, nearly 13 per cent of water, and traces of iron and nickel. While asbestos was known to the ancients, the fabricating of asbestos on a large scale is comparatively new; it received a tremendous impetus from its use in connection with automobiles and as an insulating material and a heat resistant for a great variety of mechanical purposes.

While Hoffman¹ called attention to the possible harmfulness of asbestos dust in 1916, it was not until February 1927 that asbestosis was, so to speak, officially recognized in this country by the filing of a disability claim for workmen's compensation in Massachusetts. The claimant was a foreman in the weaving department of an asbestos plant, and the claim was upheld by the Massachusetts Industrial Accident Board. This was twenty-seven years after the first fatal case was reported in England by Dr. Montague Murray. In 1910 and

¹Read before the Section on Preventive and Industrial Medicine and Public Health at the Twenty-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., June 12, 1915.
²I. Hoffman, T. L.: Silicosis from Beryllium Dust in Derry Trade, Bull. 231, U. S. Department of Labor, June 1915.

again in 1933 a fatal case was reported in England, and in 1928 the British Factory Department conducted an investigation and enacted laws for the protection and compensation of the worker against this hazard. The whole subject in England has been summarized by Metcalf.² At the present time, as nearly as can be estimated, there are about 12,000 individuals employed in the chief asbestos plants in the United States, of whom 10,000 might be exposed to asbestos dust.

In 1927 a fatal case of unreplicated asbestosis was reported to the Medical Society of South Carolina,³ and since then, including this case, there have been eleven fatal cases reported in the United States, eight uncomplicated and three complicated by tuberculosis. These reports, together with the fact that asbestosis figured in the extraordinary occupational disease litigation that has spread over this country, resulted in both laboratory and field studies of this new hazard. Gardner and Cummings⁴ at Saranac Lake, N. Y., conducted animal experimentation with asbestos in 1928 and reported their observations in 1931. These two authorities in the United States and Chyngue⁵ in England have described the pathology. While asbestosis is predominantly pneumoconiosis, asbestosis is usually interstitial; it is asbestos characterized by the nodular formation so distinctive of silicosis.

A search of all the death records on file in the Metropolitan Life Insurance Company revealed that asbestosis had been given as a cause or contributing cause of death in only thirteen cases. The first case noted was in 1921, there were two in 1927, one occurred in 1931, and the rest have occurred since 1931. The diagnosis was supported by autopsy in only six. In one of the six the primary cause of death was carcinoma. In another pneumonia of the brain, and in another pulmonary tuberculosis. In the other three asbestosis was given as the primary cause, with cardiac failure as the contributing cause. Some of these cases were reported in the literature.

2. Metcalf, E. M. At a Symposium on Asbestos, Toronto, Ontario, Canada, the (Dec) 1931, (the 1932) 1933.

3. South Carolina Medical Society, N. C. Asbestosis, Am. Rev. Tuberc. 53:131 (1931).

4. Gardner, E. H., and Cummings, J. E. Studies on Experimental Pneumoconiosis. V. The Asbestos Pneumoconiosis. J. Exp. Med. 52:131 (1931).

5. Chyngue, M. The World Asbestos and Silica Industry, London 1931, 1932, 1933, 1934, 1935, 1936, 1937, 1938, 1939, 1940, 1941, 1942, 1943, 1944, 1945, 1946, 1947, 1948, 1949, 1950, 1951, 1952, 1953, 1954, 1955, 1956, 1957, 1958, 1959, 1960, 1961, 1962, 1963, 1964, 1965, 1966, 1967, 1968, 1969, 1970, 1971, 1972, 1973, 1974, 1975, 1976, 1977, 1978, 1979, 1980, 1981, 1982, 1983, 1984, 1985, 1986, 1987, 1988, 1989, 1990, 1991, 1992, 1993, 1994, 1995, 1996, 1997, 1998, 1999, 2000, 2001, 2002, 2003, 2004, 2005, 2006, 2007, 2008, 2009, 2010, 2011, 2012, 2013, 2014, 2015, 2016, 2017, 2018, 2019, 2020, 2021, 2022, 2023, 2024, 2025, 2026, 2027, 2028, 2029, 2030, 2031, 2032, 2033, 2034, 2035, 2036, 2037, 2038, 2039, 2040, 2041, 2042, 2043, 2044, 2045, 2046, 2047, 2048, 2049, 2050, 2051, 2052, 2053, 2054, 2055, 2056, 2057, 2058, 2059, 2060, 2061, 2062, 2063, 2064, 2065, 2066, 2067, 2068, 2069, 2070, 2071, 2072, 2073, 2074, 2075, 2076, 2077, 2078, 2079, 2080, 2081, 2082, 2083, 2084, 2085, 2086, 2087, 2088, 2089, 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We called these cases *first stage*. Four presenting evident pulmonary symptoms and confirmative roentgenograms, we termed *second stage*. Of these sixteen patients, twenty had been exposed more than ten years and thirteen more than fifteen years. We are still continuing our selection studies and hope to secure substantial information on the time element in development and the tendency and rate of progress.

One feature that has impressed us is that the British investigators found asbestos more severe and more menacing than we did. This difference may be more apparent than real, but it is possible that the English factories may be more dusty than ours. There were not available any comparative dust counts, but this impression is based on their reports and on statements made to myself and my colleagues by persons familiar with the English conditions. One process, described in the British reports as "nutless making" and stated to be extremely dusty, does not appear to have a counterpart in this country. In both countries, energetic steps have been taken to control the dust hazard in asbestos plants, so that it is probable that further cases of disfiguring asbestosis will be rare.

As in siliconosis, the alveolus centers on the emphysema. However, the whole matter of attempting to interpret these films and correlate them with the clinical evidence, if any, is difficult and elusive. If, in our studies, we had found a more clear-cut and severe type of pneumoconiosis with marked symptoms and disability as well as a distinctive roentgen appearance, such as our colleagues and myself had been accustomed to find in our previous investigations of silicosis, our task in attempting to make a positive diagnosis and estimate the extent of the disease would have been easier. There is no doubt that, especially in the beginning, we were handicapped by endeavoring to evaluate pneumoconiosis with a silicosis food rule.

The x-ray experiments are not clear cut or distinctive in silicosis and do not lend themselves to ready extrapolation into progressive stages. There are less evident systematic changes in these films and the shadows are finer, more granular and softer than in silicosis. The, therefore, more homogeneous film gives the impression of ground glass, and there is no resolution with the consequent tendency of the nodules to coalesce and give dense opaque areas in the films. The distribution of the shadows is

somewhat different, occupying the lower third of the lung, except in far advanced cases, when the shadow may occupy the major portion of the lung. We undeniably recognize the well marked outline of the interlobar septum on the right side. We also noticed that a number of films in cases of asbestosis showed enlarged hearts, and this might be expected when one considers that the pathologic process tends to constrict the pulmonary blood vessels as they tangle along with the bronchioles.

It is possible that the x-ray appearance of asbestos may not be distinctive of this disease alone but unique in appearance with pneumoconiosis due to other silicate dusts. Much more investigation and study of roentgenograms of industrial workers exposed to all sorts of silicate and other dusts are needed before it will be possible to reach definitely on this, the most important phase of the diagnosis of pneumoconiosis.

One thing is certain. The utmost care and patience are needed to elicit the occupational history of the patient and to correlate this and the clinical picture with the roentgenogram before a diagnosis of asbestosis is justified. As noted in relation to other occupational health hazards, there is an all too frequent tendency to make a diagnosis of a specific occupational disease because of presumed or actual exposure without the corroboration of other essential factors to a correct diagnosis. One is not justified in making a diagnosis of asbestosis any more than of silicosis in the presence of a roentgenogram showing no distinctive pulmonary pathologic changes.

Associated with exposure to asbestos dust is the occurrence in the sputum and pulmonary tissues of a peculiar formation known as asbestos bodies. These asbestos bodies have been described by a number of observers and are due apparently to the action of the dust on the asbestos fiber. Their exact significance is doubtful, but it is commonly agreed at the present time that they are not diagnostic of pulmonary fibrosis and indicate merely that the individual has been exposed to asbestos dust.² Some observers believe that when these asbestos bodies appear in the sputum in

J. Jackson, J. R., by direction of McCann, W. S. 1 Electric Avenue,
Cincinnati, Ohio; and Gray, W. T. The Estimation of Ferrous
Iron in Iron Ore by the Potassium Dichromate Method. 190
928.

P. Siegmundson's Wood, W. H., and Chapin, S. M. Preliminary
Report on the Forest Resources of Alaska. 1900.

clumps; they indicate actual disintegration of lung tissue.

Miller⁹ of the United States Public Health Service describes the technique that he employs in the intratracheal injection of finely divided dusts in a state of suspension. Miller defined three types of reaction: absorptive, inert and proliferative. The first is produced by relatively harmless or inactive dusts, they are dusts that might cause pulmonary fibrosis, and the third as the typical reaction of silicosis. Recently Miller¹⁰ described the effects of the injection of three varieties of asbestos; namely, chrysotile, crocidolite and amosite. Chrysotile is Canadian asbestos and, as previously stated, is largely magnesium silicate. Crocidolite and amosite contain only a small quantity of magnesium, containing instead iron silicate in approximately the same quantity.

The three types of asbestos produced the same type of reaction; namely, the one described by this investigator as inert. He states:

We have been assuming that the dusts producing this inert reaction cause pneumoconiosis of the diffuse fibrotic type as distinct from the proliferative reacting dusts which elicit nodular fibrosis. This assumption, as you know, has not been proven by corresponding animal experiments with the same dusts, but results from observation of pathological material from autopsies.

I believe that the gross behavior of asbestos in the lungs has further strengthened the value of our classification of dusts and the histopathological test as a means of determining the harmful dusts by its clinical correlation.

Much work remains to be done before asbestosis is spoken of as authoritatively as is silicosis. In the meantime, asbestos plants are being cleaned up and the dust is being controlled. This, together with the smaller number of persons employed, implies that there will probably never be the wealth of clinical material that has been available in silicosis. It is by no means certain that asbestosis progresses as does silicosis after withdrawal from dust exposure, nor does infection seem to be as clearly and intimately associated with asbestosis as with silicosis. The power to there and other problems resulting from exposure to silicate dusts demands further study both in the field and in the laboratory.

Metropolitan Life Insurance Company.

⁹ Miller, J. W., and Smith, B. J. The Pathological Response of the Pulmonary Tissue to the Inhalation of Foreign Bodies, *Pub. Health Rep.*, 46:109 (Jan. 1931).

¹⁰ Miller, J. W. Personal communication to the author.