tomy is still in its infancy. Postoperative observations may be changed as the survival period increases, which, if it does, will alter the surgical indications and the selection of cases. Close cooperation between laboratory investigators, clinicians and neurosurgeons is imperative in order to make the best selection of cases for surgery and to evaluate accurately the results obtained, whether they are failures or successes.

ASBESTOSIS

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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 Hoffman[1] called attention to the possible harmfulness of asbestos dust in 1918, 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 again in 1934 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 Merewether.[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 uncomplicated 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 Cummings[4] at Saranac Lake, N. Y., commenced animal experimentation with asbestos in 1928 and reported their observations in 1931. These two authorities in the United States and Gloyne[5] in England have described the pathology. While silicosis is predominantly parenchymatous, asbestosis is mainly interstitial; nor is asbestosis 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 nineteen cases. The first case noted was in 1924, there were two in 1927, one occurred in 1931, and the rest have occurred since 1933. The diagnosis was supported by autopsy in only six. In one of the six the primary cause of death was carcinoma, in another glioma 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.

Asbestosis complicated by heart disease was given as the cause of death in seven cases. There were no autopsies in these seven and pulmonary tuberculosis was diagnosed in several by the attending physician, so perhaps too much weight cannot be given to these certificates.

Diabetes and pulmonary tuberculosis were also mentioned in the remaining six cases, in which there were no autopsies. All nineteen patients were males and with one exception all were white.

In our studies of asbestos mines and fabricating plants,[6] the clinical picture of asbestosis was milder than that of silicosis. To be sure, the individual patient with marked asbestosis will greatly resemble the individual with silicosis. There is the same dyspnea on exertion, the same dry cough, and the more or less indefinite physical signs elicited by the stethoscope. The patient with asbestosis is apt to have clubbed fingers—not usually seen in silicosis—and he is apt to be pasty faced and even show a slight cyanosis, while the silicotic patient is apt to be fairly robust looking. Of course, in each instance I refer to patients whose disease is not complicated by infection.

We did not find in communities in which asbestos was mined or fabricated the familiar picture of disability and tuberculous infection so characteristic of hard rock mining communities. Our observations were supported by the statements of physicians practicing in these communities. All the patients with asbestosis that we detected were, with one exception, working steadily at their trades. In only one case did we find evidence of active tuberculosis and that diagnosis was based only on the roentgen appearance. Several showed healed tuberculosis. Gardner and Cummings in their reports called attention to the difference between the action of asbestos dust and silica dust in relation to tubercle infection in experimental animals, and their observations tend to bear out our clinical study.

In all, our clinical data are based on 126 physical examinations of asbestos workers, all of whom had more than three years' exposure and who were selected at random. Sixty-three of these presented a roentgen appearance which we thought indicated a pneumoconi-

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osis, but the symptoms were indefinite and inconclusive. We called these cases first stage. Four presenting evident pulmonary symptoms and corroborative roentgenograms, we termed second stage. Of these sixty-seven patients, twenty had been exposed more than ten years and thirteen more than fifteen years. We are still continuing our asbestos studies and hope to secure additional 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 asbestosis 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 "mattress 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 disabling asbestosis will be rare.

As in silicosis, the diagnosis centers on the roentgenogram. 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 my 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 asbestosis with a silicosis foot rule.

The x-ray appearances are not clear cut or distinctive as in silicosis and do not lend themselves to ready grouping into progressive stages. There are less evident pathologic changes in these films and the shadows are finer, more granular and softer than in silicosis. The asbestosis film gives the impression of ground glass, and there is no nodulation 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 shadows may occupy the major portion of the lung. We noticed frequently 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 ramify along with the bronchioles.[7]

It is possible that the x-ray appearance of asbestosis may not be distinctive of this disease alone but uniform 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 speak 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 presumptive 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 tissues 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.[8] Some observers believe that, when these asbestos bodies appear in the sputum in clumps, they indicate actual disintegration of lung tissue.

Dr. Miller[9] of the United States Public Health Service describes the technic that he employs in the intraperitoneal 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, the second by dusts that might cause pulmonary fibrosis, and the third as the typical reaction of silicosis. Recently Miller[10] 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 fibrosis type as distinct from the proliferative reacting dusts which cause nodular fibrosis. This assumption, as you know, has not been proven by corresponding animal experiments with the same dusts, but results from observation of pathologic material from autopsies. . . . I believe that the gross behavior of asbestos in the tissues has further strengthened the value of our classification of dusts and the intraperitoneal 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 closely and intimately associated with asbestosis as with silicosis. The answer to these and other problems resulting from exposure to silicate dusts demands further study both in the field and in the laboratory.

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