FILE NAME: Brakes (BRK)

DATE: 1940

DOC#: BRK007

DOCUMENT DESCRIPTION: Journal Article - Clinical Studies in Asbestosis - American Review of Tuberculosis

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#8

PLAINTIFF'S Depo. Ex. No. 8
Date 6/13/84 by JHL

THE AMERICAN REVIEW OF TUBERCULOSIS

OFFICIAL JOURNAL OF
THE AMERICAN TRUDEAU SOCIETY

VOLUME XLI
JANUARY-JUNE, 1940

PUBLISHED MONTHLY
AT MT. ROYAL AND GUILFORD AVENUES, BALTIMORE, MD.
BY THE NATIONAL TUBERCULOSIS ASSOCIATION

R Depo. Ex. No. 14
Date 4/29/83 by AAC

CLINICAL STUDIES IN ASBESTOSIS[1]

MOSES J. STONE[2]

Among the newer diseases, which are a byproduct of our industrial age, asbestosis has come to occupy a fairly prominent position. The diagnosis of this form of pneumonoconiosis, its clinical course, the effect it produces on organs other than the lungs as well as its various complications and sequelae are still very much in the controversial stage. The marked development in the industrial use of asbestos has resulted in an increase in the number of people exposed to this occupational hazard. This, together with the greater interest developed by medical men and legislature boards in industrial disease, has produced a great impetus in the study of this subject. The literature on this subject is still rather meagre, and only recently has the hazardous nature of exposure to asbestos dust been recognized and studied.

Asbestos is a hydrated magnesium silicate, the composition of which varies with the sections from which it is mined. Most of the asbestos used in the United States is Canadian crysotile containing approximately 43 per cent magnesium, 13 per cent water and traces of iron and nickel. To be sure, asbestos is not a new mineral. Known to the Romans, who mined it from the Alps and from the more remote Urals, the mineral was mentioned in the writing of Herodotus and the second Pliny. Marco Polo spoke of its use by the Tartars.

Only within the last decade, has the subject of asbestosis really received the attention of the medical world. Prior to 1924, there is but one recorded case of disease due to the inhalation of asbestos dust. This was recorded in 1900 by Montague Murray in the Charing Cross Hospital Gazette. The first complete description of this disease entity appeared in 1927 when Cooke (1) and McDonald (2) reported 2 cases of asbestosis and discussed the histological changes found in the lung. Hoffman (3) was the first American to focus attention on the magnitude of the asbestosis problem in the United States. In 1930 Mills (4)

[1] Presented at a session of the Clinical Section at the 35th annual meeting of the National Tuberculosis Association, Boston, Massachusetts, June 28, 1939.

[2] 520 Beacon Street, Boston, Massachusetts.

offered the first pathological report published in the United States. In the same year, Lynch and Smith (5) reported their findings on asbestosis bodies.

An interesting feature of asbestosis is the finding of the asbestosis bodies in the lung as well as in the sputum. Gloyne and others described the asbestosis body as a core of asbestos fibre surrounded by iron containing deposits. It is golden yellow in color and does not stain with ordinary histological stains but becomes a brilliant blue when treated with potassium ferrocyanide. The bodies are slender, elongated, segmented structures with bulbous ends. The substance of the body appears homogeneous except for the centre linear thickening which is the asbestos fibre. The essential features of the pathological changes appear to be pleurisy, marked diffuse fibrosis with contraction of the lungs, and the presence of the asbestos fibres and the asbestosis bodies. Uniform thickening of the visceral pleura is found with varying degrees of pleural and pericardial thickening. The fibrosis extends into the apices with obliterating layers of fibrous tissue surrounding the bronchioles. The pulmonary endarteries are surrounded by fibrous tissue and may show thrombosis. The interlobar and intrapleural connective tissue is thickened. Numerous alveoli may be obliterated by fibrous tissue, others may be emphysematous, while still others are filled with leucocytes and alveolar cells. Occasional giant cells, not to be mistaken for tuberculous giant cells, are found. There are asbestosis bodies in the lumina of the bronchioles, in the alveoli and in the fibrous tissue. An amorphous brown pigment, often phagocytized by the alveolar cells, is scattered throughout the lung. On section the trabeculae stand out in the fibrous network and may present evidence of septic bronchitis, bronchiectasis, bronchopneumonia or lobar pneumonia as the terminal events. Lynch (6) and Gloyne (7, 8) note that the presence of asbestos fibres in the mouth and nose are indicative of exposure but not necessarily of disease. Essentially the presence of asbestosis bodies indicates tissue response to dust.

Since 1930 much has been written on this subject both from the clinical and public health points of view. Many controversial points, such as the action of asbestos dust on lung tissue, the relationship of asbestosis to infections, especially tuberculosis, and the effect it has on the circulatory system, etc., still remain that demand the attention of investigators.

This report is based on a study of 180 patients who were examined by me in collaboration with the late Dr. John B. Hawes, 2nd. The patients

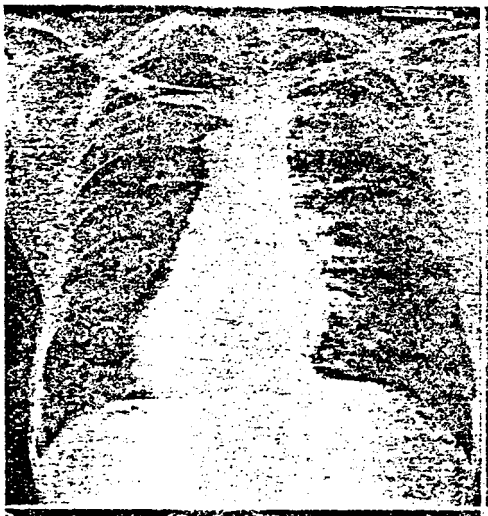

FIG. 1

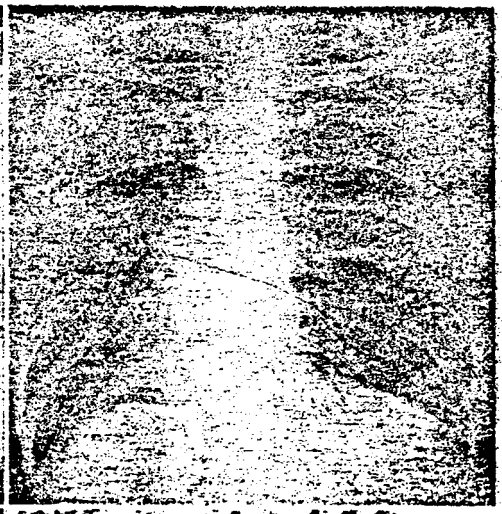

FIG. 2

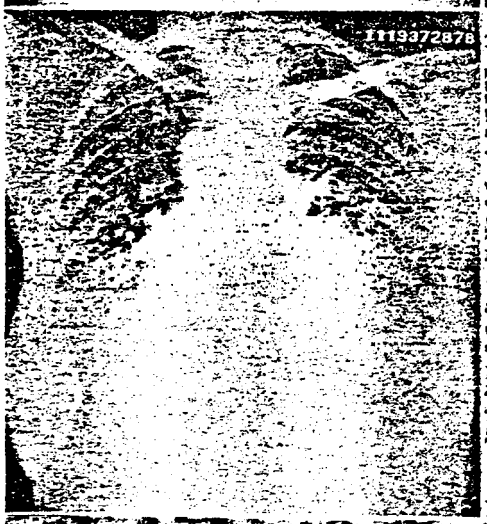

FIG. 3

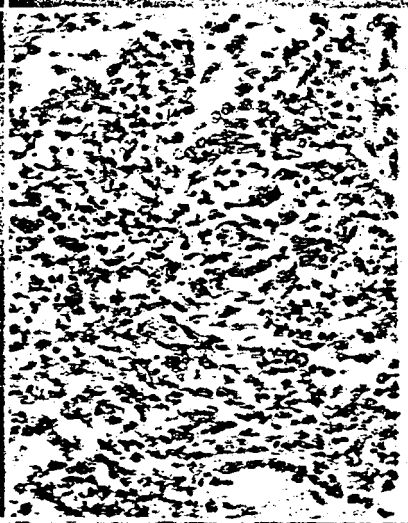

FIG.

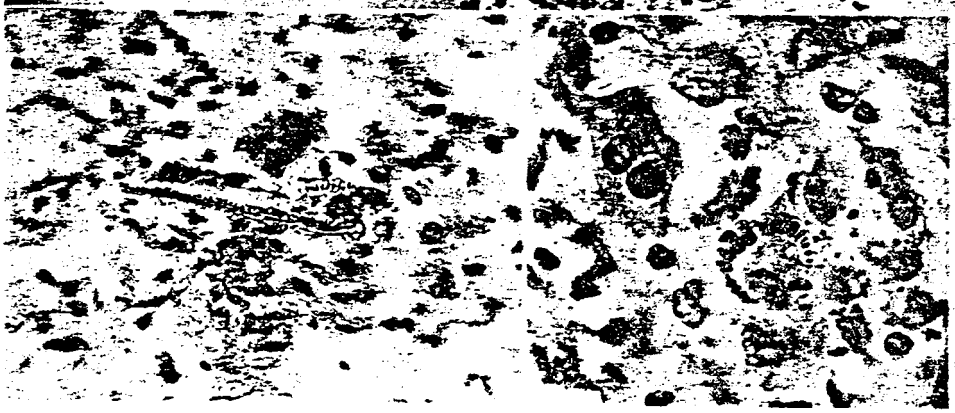

FIG. 5

FIG.

FIGS. 1–6

had been employed for three years or more in a factory manufacturing asbestos brake lining for automobiles and worked in the carding, spinning or weaving rooms. Many of them were engaged in the more dangerous work of crushing the crude asbestos. The great majority of them had been employed from five to fifteen years, and many had worked for more than twenty-five years. The cases came to our attention through disability claims and were given complete physical examinations during the years of 1936 and 1937. None were employed in asbestos factories at the time or since their medical examinations.

CLASSIFICATION

Of the 180 patients, 32 were diagnosed as negative, although they claimed disability. X-ray film as well as physical signs failed to show any abnormalities that could be ascribed to asbestosis. The remaining 148 patients were classified as follows:

Stage I—78 patients
Stage II—54 patients
Stage III—16 patients

A patient was classified as stage I when there was definite limitation of chest expansion (less than 2 inches) in addition to roentgenological evidence of increased lung markings. It must be admitted that frequently this interpretation must be somewhat arbitrary, as it is often quite difficult to draw the line between normal and exaggerated lung markings. Unlike silicosis the early fibrotic changes are very indefinite and only after considerable experience, careful standardized X-ray technique and proper history, can a diagnosis of early asbestosis be made, and even then not with any degree of certainty. In stage II were included those

FIG. 1. Stage I. Patient worked for four years as an inspector in a plant manufacturing asbestos brake-lining. Two years prior to the examination he noticed lack of energy and strength, also some cough and expectoration. There was only slight dyspnoea on exertion.

FIG. 2. Stage II. Patient worked for thirteen years as an inspector in a plant manufacturing asbestos brake-lining. He has had dry cough for seven years which has continued on about the same; also dyspnoea on the slightest exertion.

FIG. 3. Stage III. Patient has been exposed to asbestos dust for twenty-five years. He has marked dyspnoea and has to sit up on a stool at side of bed and sleeps in that position. Unable to lie down. Chest expansion, one inch.

FIG. 4. Asbestosis bodies found in the lungs of patient who died of pulmonary tuberculosis. Note the various sizes and shapes of these bodies, as seen with the low power lens.

FIG. 5. Asbestosis body. High power magnification

FIG. 6. Another view of the asbestosis bodies (high power)

patients who had definite symptoms and whose X-ray films revealed definite evidence of pulmonary fibrosis. In stage III were included those patients who had both definite symptoms as well as roentgenological evidence of marked pulmonary involvement.

LENGTH OF EXPOSURE

Number of Cases	*Years of Exposure*
47	4 or less
56	4–9
40	9–14
25	14 years and over

RELATIONSHIP OF LENGTH OF EXPOSURE TO PATHOLOGICAL CHANGES IN THE LUNGS

Of the 47 patients who were exposed four years or less, 25 showed no definite pathological changes; 14 were classified as stage I and 8 as stage II. Fifty-six patients worked four to nine years. Of these, 36 cases were classified as stage I, 12 were considered as stage II or fairly marked asbestosis and 8 were found to be suffering from advanced or stage III asbestosis. Of the 40 patients who were exposed ten to fifteen years, 14 were classified as stage I, 20 as stage II and 6 as stage III. Twenty-five patients were exposed for over fifteen years. Of these, 9 showed the early pulmonary changes of stage I, 14 were classified as stage II and 2 as stage III. The average length of exposure of the stage I patients was eight years; stage II, ten years; stage III, eleven years. While there is but slight evidence that the degree of fibrosis is apt to increase with the length of exposure, there are other factors, such as intercurrent infections and other constitutional factors, that play a part in the development of fibrotic changes in the lung structures.

Since all patients were seeking compensation on account of disability, many of the complaints were undoubtedly exaggerated. On careful questioning, however, one could evaluate the symptoms fairly accurately. The outstanding symptom in all the patients was dyspnoea. Many also complained of tightness in the chest. Of the 54 patients in stage II, 32 complained of dyspnoea on slight exertion, tightness in the chest, cough and general fatigue. In addition to the above symptoms, 10 patients also complained of marked loss of weight. All of the 16 patients in stage III complained of dyspnoea, inability for any sustained effort, cough, expectoration, tightness in the chest, anorexia and loss of weight.

PHYSICAL EXAMINATION

The chest findings were mainly those of basal fibrosis with accompanying emphysema at the apices. On the whole, physical findings were quite meagre, and were not in proportion to the symptoms given by patients. The outstanding physical sign was diminution of chest expansion. Many cases showed evidence of dulness at the bases with hyperresonance near the apices. In the majority of cases auscultation disclosed prolongation of the expiratory phase with some high-pitched dry crackling râles. Moist râles were found only when there was definite evidence of associated bronchitis or bronchiectasis.

X-RAY EXAMINATION

X-ray examination constitutes the most important single procedure in the diagnosis of asbestosis. Those who have examined many cases of silicosis will find the X-ray interpretation of asbestosis extremely difficult. Early X-ray diagnosis of asbestosis is still in the realm of conjecture and should be undertaken only by those who have had the opportunity to examine many such cases.

In the early stages there is only a slight relative increase in density in the lower zones producing a filmy, hazy appearance of the bases. The shadows are much finer, lighter, and have a granular appearance, rather than the nodular or patchy type of infiltration found in silicosis. The apparently small amount of lung involvement in the early stages makes the X-ray evaluation rather difficult. As the disease progresses, signs indicative of stage II are seen. There is an increase in density in the lower lung zones, the diaphragm becomes indistinct in outline and shows, on roentgenoscopic examination, limitation of motion. The costophrenic angle is obliterated by thickened pleura. A fine lace-like network of fibrosis of interstitial or perivascular form rather than a definitely parenchymatous type is seen. The characteristic chest X-ray film shows granular or "ground-glass" appearance with more or less obliteration of the usual linear pulmonic markings. This is localized in most cases in the midlung region and bases. The bronchovascular markings are increased and often pericardial and pleural thickenings are noted. Many cases in our series showed elevation of the diaphragm. In stage III, because of the frequently associated bronchitis and bronchiectasis, the parenchymatous changes become more marked.

TUBERCULOSIS IN ASBESTOS WORKERS

The still debatable question of the relationship between asbestosis and tuberculosis has been discussed by numerous authors. This study was mainly undertaken to determine the incidence of tuberculosis among asbestosis workers. Since many patients do develop pulmonary fibrosis, their resistance to pulmonary infections is greatly decreased. Most of the English investigators, among them Stewart (9), found marked increase of tuberculosis infection among asbestos workers. He states that there can be no question that pulmonary asbestosis predisposes to tuberculous infection of the lungs. His opinion is also held by Ellman (10) who found an increasing incidence of tuberculosis in persons exposed to asbestos dust. Donnelly (11) refutes these above statements and finds no definite relationship between asbestosis and tuberculosis. In fact Merewether and Price (12), finding only 3 cases of tuberculosis among 374 cases of asbestosis, indicated that a lessened susceptibility to tuberculosis existed in such cases.

Out of 180 films taken in our series of cases, 9 showed evidence of parenchymatous tuberculous infection; two of this group had active tuberculosis and 7 showed inactive or healed lesions. In our series of 16 cases of advanced asbestosis, only one had active tuberculosis. One other active case of tuberculosis had only moderate asbestosis (stage II). Of those patients who had inactive disease, 5 had moderately advanced asbestosis (stage II), and all had been exposed to asbestos dust for more than ten years. Thus we are not impressed with tuberculosis as being a serious complication of asbestosis. We find that bronchitis, bronchiectasis and bronchopneumonia are more frequently associated with asbestosis than is tuberculosis. Indeed we feel that, because of the frequency of tuberculosis in silicosis, many patients with bronchitis or low grade bronchopneumonia were erroneously diagnosed as being tuberculous. Only one person in our total series has subsequently developed active pulmonary tuberculosis.

HEART STUDIES

One hundred and fifty patients were studied to ascertain the cardiac involvement, if any, in cases of asbestosis. Heart measurements were done by Dr. George Levene of the Massachusetts Memorial Hospitals. Ninety of the 150 patients studied, or 60 per cent, showed prominent pulmonary vessels. This prominence was apparently either due to en-

gorgement or perivascular fibrosis. The transverse diameter was increased in 50 cases, or 33 per cent, after excluding cases of organic heart disease, such as hypertension, coronary sclerosis and rheumatic heart disease. Thus it was felt that asbestosis was apparently the cause of the increased transverse diameter in 33 per cent of our cases. Thirteen cases, or 8 per cent of our total series, had right-sided hypertrophy. A study was then carried out to determine the heart findings in 56 cases classified as moderately advanced (stage II) and advanced (stage III) asbestosis. Of these 56 cases, 35, or 62 per cent, showed prominence of the pulmonary vessels; 25, or 44 per cent, showed an increase of the transverse diameter (7 cases of the latter group were excluded because of associated heart disease). In the remaining 18 patients, or 32 per cent, the enlargement of the transverse diameter was associated with asbestosis.

Apparently then the heart and the pericardium are affected in asbestosis much more so than in any other type of pneumonoconiosis. The cardiac outline is obscured by the superimposed increased fibrous tissue of the lung with advancing degrees of asbestosis. The pleuropericardium, the pleura of the lower lung fields and the diaphragmatic domes become thickened and the usually sharply demarcated heart contours are blurred. The marked emphysema and pulmonary fibrosis of stages II and III may cause rotation or lateral displacement of the heart. Unless roentgenoscopic examination is carried out, the sagittal roentgenograms alone will not convey a true impression of cardiac size or shape. In such cases lateral views together with roentgenoscopic examination are recommended to clarify the diagnosis. The roentgenographic appearance of any stage of asbestosis associated with heart disease may be greatly accentuated by passive congestion. As a result of our studies, roentgenoscopic examination is recommended to determine enlargement of the heart shadow and alterations in the cardiac silhouette. This is of special importance in cases of asbestosis associated with hypertension, mitral stenosis and cor pulmonale. It is our belief, therefore, that cardiac embarrassment is of utmost importance in the causation of early symptoms. It is evident that the dyspnoea noted early in this disease is of cardiac or circulatory rather than of pulmonary origin.

FOLLOW-UP STUDIES

We had an opportunity to reëxamine 13 patients of this group from two to three years after their first examination. Two of this group were

originally classified as stage III; 8, as stage II, and 3, as stage I. These patients were carefully studied in The Clinic for Cardiac Research of the Massachusetts Memorial Hospitals by Drs. George Levene, William Duncan Reid and Maurice A. Lesser. The vital capacity was affected in all cases, being from 50 to 75 per cent below the normal, calculated on the basis of height and weight. X-ray examination revealed definite progression of fibrosis in the two cases that were originally classified as stage III. Two patients had evidence of old coronary disease. This was corroborated both by the X-ray as well as electrocardiographic examination. The electrocardiograms of the remaining patients were entirely normal. Sedimentation rates were normal with the exception of the two with coronary disease which showed an increased rate.

Our impression gained from the study of this small group is that in advanced fibrosis due to asbestosis the disease will progress even after exposure ceases. This, however, is not the case in the earlier stages. We also feel that the heart is not affected unless pulmonary fibrosis is marked. In the light of the blood studies, fibrosis is the result of irritation due to asbestos fibres, and is not the result of infection.

Since the entire group was first examined, 18 patients have died. The cause of death was given as follows:

Pulmonary tuberculosis	3 cases
Bronchopneumonia	5 cases
Lobar pneumonia	2 cases
Carcinoma	2 cases
Cardiac disease	2 cases
Cerebral shock	1 case
Accidental	2 cases
Brain tumor	1 case

Most of the patients are still able to pursue a gainful occupation although unable to perform duties that demand much physical exertion. Seventeen patients are semi-invalids, most of them complaining of cough and marked dyspnoea.

CONCLUSIONS

1. Asbestosis, like silicosis, constitutes an occupational hazard, arising from exposure to asbestos dust.

2. Pathological findings are those of pulmonary fibrosis and thickened pleura, most marked at both bases.

3. Dyspnoea is an early symptom and is the chief cause of early disability.

4. X-ray findings are not characteristic in early stages. In advanced asbestosis X-ray reveals lace-like interstitial fibrosis and thickened pleura at the bases giving the "ground-glass" appearance.

5. Bronchial and bronchopulmonary infections are common complications.

6. Tuberculosis is not a frequent concomitant of asbestosis.

7. Advanced asbestosis not infrequently leads to right ventricular hypertrophy and failure.

8. When exposure ceases, fibrosis does not progress unless the degree of involvement is already marked.

9. All health laws relating to dusty occupations should be rigidly enforced in all asbestos factories.

10. Workers developing pulmonary fibrosis in the course of exposure to asbestos dust should be entitled to compensation.

I am deeply indebted to Drs. George Levene, William D. Reid, Maurice A. Lesser of the Clinic for Cardiac Research of the Massachusetts Memorial Hospitals and to Dr. Lois C. Miller of the Department of Radiology for their coöperation and valuable assistance given me in preparation of this paper.

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