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CONTENTS:

PULMONARY TUBERCULOSIS FROM A PUBLIC HEALTH STANDPOINT (Page 8)
R. H. Sundberg, M.D., F.A.C.P., San Diego, California

DEMONSTRATION OF CANCER CELLS IN PLEURAL FLUIDS (Page 10)
Alfred Goldman, M.D., F.A.C.P., St. Louis, Missouri

A NOTE ON THE EARLY DIAGNOSIS OF PULMONARY TUBERCULOSIS (Page 13)
Jaharlal Ghosh, M.D., M.R.C.P., M.R.C., Calcutta, India

THE MODERN TREATMENT OF PULMONARY TUBERCULOSIS (Page 14)
John B. Andosca, M.D., Boston, Massachusetts

TUBERCULOSIS IN ATHLETES (Page 16)
C. B. Young, M.D. and E. G. Faber, M.D., Tyler, Texas

EDITORIALS, ORGANIZATION NEWS, SANATORIA

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OUR BEST WISHES FOR THE NEW YEAR

Studies in Asbestosis

MOSES J. STONE, M.D.
Boston, Massachusetts

The diagnosis of asbestosis, its clinical course, the effect it produces on organs other than the lungs, as well as its various complications and sequelae, are very much in the controversial stage. Only within the last decade has the subject of asbestosis really received the attention of the medical world. The literature on this subject is still rather vague and only recently has the hazardous nature of exposure to asbestos dust been recognized and studied. To be sure, asbestos is not a new mineral[1]. The mineral mentioned in the writings of Herodotus and the second Pliny, was known to the Romans who mined it from the Alps and the Urals. In 1900, Montague Murray recorded in the *Charing Cross Hospital Gazette*, a case of disease due to inhalation of asbestos dust.

Hoffman was the first American to focus attention on the magnitude of the asbestosis problem in the United States. In the same year, Lynch and Smith reported their findings on asbestosis bodies found in the alveoli and bronchioles and in the fibrous and necrotic areas. Gloyne and others have described the asbestosis body as a core of asbestosis fiber surrounded by iron containing deposits. The bodies are slender, elongated, segmented structures with bulbous ends and stain blue when treated with potassium ferrocyanide. The essential features of the pathological changes of asbestosis appear to be pleuritis, marked diffuse fibrosis with contraction of the lungs, and the presence of the asbestos fibers and the asbestosis bodies. Thickening of the visceral pleura with varying degrees of pleural and pericardial adhesions is often found. Obliterating layers of fibrous tissue may surround the bronchioles and pulmonary end arteries. Lynch and Gloyne note that the presence of asbestos fibers in the mouth and nose are an indication of exposure, but not necessarily of disease. Essentially the presence of asbestosis bodies in the sputum indicates exposure to asbestos dust, but it does not absolutely denote disease.

This report is based on a study of 180 patients examined by the author in collaboration with the late Dr. John B. Hawes, II. The great majority of patients had been employed in a factory manufacturing asbestos brake linings with an average exposure time of five to fifteen years.

Classification

Of the 180 patients, 32 were diagnosed as negative, although these individuals claimed disability. X-ray films as well as physical signs failed to show any abnormalities that could be attributed to asbestosis. Seventy-eight patients were classed in Stage I, when there was definite limitation of chest expansion (less than 2 inches), in addition to roentgenological evidence of increased lung markings. Unlike silicosis, the very early fibrotic changes are very indefinite and only after considerable experience, together with careful standardized x-ray technique and proper history, can a diagnosis of early asbestosis be made. In Stage II, there were fifty-four patients who had definite symptoms and definite x-ray evidence of pulmonary fibrosis. Included in Stage III, were sixteen patients who had definite symptoms as well as roentgenological evidence of marked pulmonary involvement. The average length of exposure of the Stage I patient was eight years; Stage II, 10 years; Stage III, eleven years. While there is slight evidence that the degree of fibrosis is apt to increase with the length of exposure, there are other factors such as intercurrent pulmonary infections that play a part in the development of fibrotic changes in the lung structures.

Dyspnoea was the outstanding symptom in all patients. Dyspnoea on slight exertion, tightness of the chest, cough, general fatigue were among the other symptoms noted. In Stage III the patients also complained of inability to sustain any effort, cough, expectoration, tightness of the chest, anorexia and loss of weight.

1 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 nickel and iron and aluminum.

physical findings were quite meager and were not in proportion to the symptoms offered by patients. Many cases showed dullness at the bases with hyperresonance at the apices associated with diminution of chest expansion. Ascultation disclosed prolongation of the expiratory phase with some high pitched dry rales. Associated bronchitis or bronchiectasis may be present.

X-ray examination constitutes the most important single procedure in the diagnosis of asbestosis. The shadows are much finer, lighter and more granular than the nodular fibroid type of infiltration found in silicosis. In the early stages a relatively slight increase in density in the lung bases is seen. With progression of the disease and thickening of the pleura, a fine lace-like fibrosis is found. The typical granular or "ground glass" appearance is then noted, especially in the mid-lung and basilar areas. The bronchovascular markings are increased and peribronchial and pleural thickenings are noted. Many cases show limitation of motion and elevation of the diaphragm. With associated disease the parenchymatous changes become more marked. In the series of cases studied, bronchiectasis, bronchitis and bronchopneumonia were found to be more frequently associated with asbestosis than was tuberculosis. In addition, tuberculosis did not impress the author as being a serious complication of asbestosis. Only one individual in the total series subsequently developed active pulmonary tuberculosis.

In 150 patients the cardiac aspects of asbestosis were studied. Ninety patients (60 per cent) showed prominent pulmonary vessels due either to enlargement or increased peribronchial fibrosis. Asbestosis was associated with an increased transverse diameter in 33 per cent of the cases and 13 per cent had marked right-sided hypertrophy. The cardiac outline is obscured and blurred by the superimposed increased fibrous tissue. The thickening of the pleura, the marked emphysema and the pulmonary fibrosis may cause rotation or lateral displacement of the heart. Roentgenoscopic examination is essential to determine enlargement of the heart shadow and alterations in the cardiac silhouette. The x-ray appearance of asbestosis may be greatly altered by the congestion due to associated heart disease. It is also evident that the dyspnoea noted early in this disease is of cardiac or circulatory, rather than of pulmonary origin.

A small group of patients were reexamined three years following the first examination. X-ray, electrocardiographic, blood studies and vital capacity examinations were done. The vital capacity was found to be 50-70 per cent below the normal, calculated on the basis of height and weight. The impression gained from the study was that in cases of advanced fibrosis the disease may progress even after exposure ceases. The heart was not affected unless pulmonary fibrosis was marked. In the light of the blood studies, fibrosis is the result of irritation due to asbestos fibers and not the result of infection. Since the first examination, 18 patients have died. Most of the patients still pursue gainful occupation, but are unable to perform duties that demand much physical exertion.

The impressions gained from the above study are that asbestosis is a debilitating disease caused by exposure to asbestos dust, tuberculosis is an infrequent complication and pulmonary infections, other than pulmonary tuberculosis, are fairly frequent complications. In advanced asbestosis, the right ventricle may dilate with subsequent pulmonary congestion. Like silicosis, asbestosis is caused by exposure to asbestos dust and should be classified as an industrial hazard. Hence such patients should be entitled to compensation under the industrial accident laws.

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