In the present case no involvement of the medulla was found. Extensive fibrous meningitis resembling gummatous basilar meningitis has also been described by Askanazy[11] Rosenblath,[13] Henneberg,[10] and others. As far as is known, the cysts are very little toxic, although it appears that pyrexia occurred at the onset of infection or early in the course of a case of disseminated cysticercosis described by Major R. Priest[15] in 1926.

The case we record proved fatal after nearly six years of symptoms; but the parasites, especially when localized in the eye or brain, may live for long periods, very rare cases being noted up to twenty years (Kuchenmeister, Roth-Ivanoff, Pfeifer, and others).

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A CASE OF PNEUMOCONIOSIS.

RESULT OF THE INHALATION OF ASBESTOS DUST.

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(*With Special Plate.*)

THE asbestos industry has been in existence for over two thousand years, and it is remarkable that the possibility of damage to the lungs, arising from the inhalation of dust during its manufacturing process, has not been investigated until within comparatively recent years. Very few cases of pneumoconiosis due to asbestos dust have been recorded in medical literature, and for this reason the notes on the following case may be of some interest.

The first recorded case was that reported by the late Dr. Montague Murray of Charing Cross Hospital in 1900. No other case was reported, and the inference that asbestos dust might be the cause of an extensive pulmonary fibrosis was lost sight of for some time. In 1924[1] and 1927[2] Dr. W. E. Cooke published his exhaustive reports on another case, and described in detail the pathological changes and his "curious bodies," which are apparently a unique feature of the microscopical picture. This stimulated other investigations, and Simson[3] has reported similar findings in workers employed in the crushing of asbestos rock, as apart from the manufacturing processes. In most of the cases hitherto reported the extensive fibrosis has been associated either with pulmonary tuberculosis or an unresolved pneumonia, and there has always been the slight doubt as to whether the inhalation of asbestos dust was the primary cause of the fibrosis.

The patient, a man aged 40 years, was sent for an opinion of his chest condition to the tuberculosis dispensary. The question of tuberculosis was raised as a consequence of his complaint of cough and breathlessness, combined with loss of weight and lassitude, extending over a period of several months.

Previous History.—There was no history of tuberculosis in the family. No history could be elicited of any illness in early life which might have led to a pulmonary fibrosis, and, apart from the symptoms which he considered to be associated with his occupation as an asbestos worker, he has had no other illnesses.

Occupation.—When a boy he had worked for a very short period in a glass works. Thereafter, for a period of twenty-two years, he has been associated with the asbestos industry. During this period he has been employed in the various processes of asbestos work: for four and a half years he worked at a carding machine, which is considered the dustiest part of the work; for seven and a half years at the weaving of asbestos cloth, and for the remainder of the period at the manufacture of the millboard. The various processes differ considerably in their degree of dustiness, and therefore in the amount of asbestos dust inhaled by the various types of workers. As, however, a number of different processes, evolving a varying amount of dust, may be carried out in the same room, the dust produced in one process may affect workers at another process. It will be noted that this man must have worked in the asbestos industry for a period during which less importance was paid to the prevention of dust than at the present time.

History of Present Illness.—The patient has had a cough from within a few months of starting work at asbestos, but he states that this was "from the throat," and appeared to have been purely the result of dust irritation. Within recent years the cough has increased in severity, and there has been a little sputum first thing in the morning. The first symptom to cause anxiety was breathlessness, which developed five years ago, and this has become accentuated since then. His general condition has remained satisfactory, and his particular symptoms were not sufficiently severe to interfere with his employment until a few months ago. At this time he complained of lassitude, loss of weight, frequent night sweatings, and an occasional slight aching of the left shoulder. On no occasion has there been haemoptysis.

Notes on Examination.—He is a spare-built man; weight 7 st. 11 lb.; temperature, pulse, and respirations within normal limits. No obvious dyspnoea noted on examination. Cyanosis of lips and cheeks, but not a marked feature. Well-marked finger clubbing noticed by the patient for at least two years. Sputum scanty, and of a mucoid nature. Tubercle bacillus absent on frequent examination.

Examination of Chest.—Inspection showed expansion to be poor generally (one inch by measurement); there was hypertrophy of the extraordinary muscles of respiration; the supraclavicular fossae were markedly hollowed; the heart apex beat was in the fifth intercostal space within the nipple line. The percussion note was flattened throughout; there was relative dullness over the right upper lobe, both in front and behind. On auscultation the respiratory murmur was diminished generally, and of a harsh quality over the right upper lobe anteriorly and posteriorly; expiration was prolonged all over the chest; rhonchi were general, with fine pleural friction at both bases.

Other Systems.—*Examination of other systems was negative.*

X-Ray Examination.—The radiogram of the chest shows a fine mottling of a silicotic type throughout both lungs, more marked on the right side and at both bases, with indication of a definite fibrosis, especially on the right side. (See special plate.)

Progress.—The patient was admitted to hospital for observation, and has now been an in-patient for six months. During this period his general condition has improved considerably, and he has gained over 1 st. in weight. The cyanosis which had been noted on first examination has to a great extent disappeared, but there is still some evidence on the cheeks and lips. The cough, which is now manifestly less troublesome, is still present, and there is a scanty mucoid sputum. Physical examination of the chest reveals no new feature apart from the absence of bronchitic sounds. The temperature and pulse remained within normal limits during the period of observation and treatment in hospital.

Conclusion.

There seems little doubt that this is a definite case of pulmonary fibrosis, the result of the inhalation of asbestos dust. The recent development of symptoms of toxaemia, associated with long-standing pulmonary symptoms, was at first suggestive of a chronic pulmonary tuberculosis with an acute exacerbation. However, the physical signs, the persistent absence of the tubercle bacillus in the sputum, and the maintenance of temperature and pulse within normal limits demanded further investigation. The typical radiographic features (signs more marked in the basal portions of the lungs) and his long period of employment with asbestos point definitely in the direction of a fibrosis of occupational origin, especially as there is neither a history of other illnesses in childhood or adolescence to support a more common cause of pulmonary fibrosis, nor does this correlation of history, symptoms, and physical signs occur in these latter cases. Toxaemic symptoms in the absence of a tuberculous affection are of special interest; whether they are the result of the absorption of some constituent of the asbestos rock or not is a point which requires further investigation.

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