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49. **Carl Johan Jacobson,** Copenhagen.

A case of acute Pulmonary Asbestosis.

Case Record:

C. H., mechanic, 25 years old, admitted for bronchial asthma to this department on January 18, 1935.

Apart from scarlet fever in childhood and herniotomy in adolescence (for traumatic hernia), the patient gave a past history of good health. In particular, no disposition to pulmonary tuberculosis, no hay fever, no previous attacks of asthma.

During the last year the patient has worked in a shop at electrowelding of rails and boilers. The welding is done with asbestos-covered electrodes, and it gives rise to a great deal of smoking with production of asbestos dust which is hurled out in the air. On welding inside the boilers, he says, there is formed a great deal of such dust. He thinks, that such electro-welding of boilers from the inside is an exceptional specialty of the factory in which he works; this is not the case, however, according to the chief physician of the factory inspection. The illness, on account of which he now enters the hospital, began about 6 weeks ago with violent spells of coughing and copious expectorate. The coughing spells were followed by marked shortness of breath. He gradually became so dyspnoeic that he had to sit up in bed all night for the past 10 days, and the cough would set in at once as soon as he tried to lie down. He would also get very dyspnoeic on the slightest exertion. He quit work 8 days before admission, but it did not seem as if rest at home made his attacks of asthma less severe. He says there has been no palpitation of the heart or other functional disturbances. He has never worked with lead.

On admission, the patient was found to be in a fair state of nutrition. The respiratory excursions of the chest were small; the heart was lung-covered; posteriorly, the 12th rib marked the inferior border of the lungs. The percussion sound was resonant. Whistling and sonorous ronchi were heard everywhere, but there were no areas of dullness or other signs of infiltration. The heart sounds were clear, and the action regular. The physical examination revealed no other particular abnormalities. The temperature was normal on admission, and kept normal throughout his stay in the hospital. During the first days he produced a good deal of expectorate (about 50 c.c., daily), but this subsided rapidly. The most striking feature was his marked dyspnoea.

Laboratory tests: Wassermann negative. Sedimentation test (1 hour): 2 mm. Pirquet negative. Vital capacity of the chest, estimated on the day after admission, 2100 c.c. (60% of the normal for his weight and sitting height).

X-ray examination showed conspicuous details of lung structure, but no definite signs of beginning silicosis in the form of infiltration, small areas of density or similar abnormalities. X-ray diagnosis: Bronchitis? Emphysema (slight degree).

No tubercle bacilli were found in the expectorate; and the blood picture was perfectly normal.

Under treatment with nicordamin, potassium iodide, magnyl, and rest in bed, the patient improved rapidly, and got up after 6 days. After other 6 days

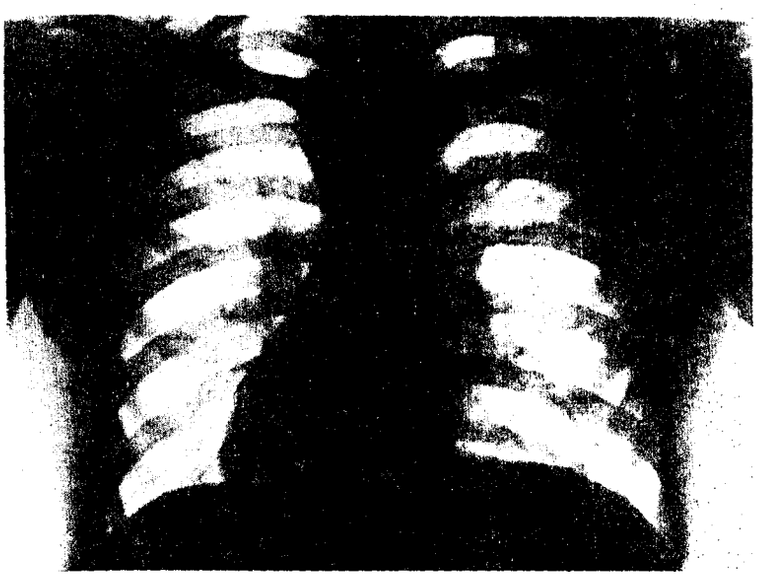

Fig. 1. Roentgenogram of the chest on admission to hospital.

he was given incandescent-light treatment and respiratory exercises, with good result. After 20 days in the hospital, he was discharged, as feeling well, for continued ambulant treatment with 12 incandescent-light baths and 12 respiratory exercises.

When admitted, he had been rather exhausted, but he improved rapidly, gaining 5 kg. in weight in 12 days. Clinical diagnosis: Bronchial asthma, Bronchitis, Emphysema, and Acute pulmonary silicosis (Asbestosis).

Comments. To make an unquestionably sure diagnosis of asbestosis, the presence of "asbestos bodies" must be demonstrated. I tried, therefore, to find such bodies in the expectorate (altogether 200 c.c.). But before I mention this examination in detail, it may be appropriate, I think, to give a brief review of the history of asbestos and pulmonary asbestosis.

Asbestos was known even in ancient times, and the Romans mined it from the Alps and the Ural Mountains. It has been mentioned by HERODOTUS, PLINY and MARCO POLO; the last author reports that it was used by the Tartars.

Although its valuable fire-resisting properties have been known for thousands of years, asbestos has not been employed for industrial purposes to any particular extent before the last part of the past century. In the asbestos factories the various processes of manufacture generated a very considerable amount of dust, and in the working classes the manufacture of asbestos has long been considered unsafe work.

It is rather strange, therefore, that prior to 1924 there had been reported only 1 case of disease due to inhalation of asbestos dust. This was a fatal case of pulmonary asbestosis in a 33-years old man who died in 1900 in the Charing Cross Hospital, London. The case was described by Murray who made the diagnosis of pulmonary fibrosis. The diagnosis was confirmed on autopsy, and microphotos of lung sections showed "spicules of asbestos". This was the first and, down to 1924, the only record of death due to asbestos. In 1924, Cooke described a fatal case of pulmonary asbestosis in which the post-mortem examination revealed the presence of peculiar, so-called asbestos bodies in the lungs. These bodies were elongated and measured from 20 to 70 μ in length; many were clubbed at the ends or swollen on the middle; they were brownish-yellow in colour and gave a positive Prussian blue reaction on account of their iron content.

Subsequently these bodies have been described by several authors (STROEBE, STEWART), and their nature has been investigated very thoroughly by BEGER.

Asbestos is a magnesium silicate, of which there are numerous varieties, most of them containing some iron. It has now been found that the organism is able to transform and in part destroy this silicate. BEGER has demonstrated what COOKE had already found it reasonable to assume: that carbon dioxide and the moist heat of the organism can hydrolyse the silicate. The course of this process is probably as follows:

After the asbestos dust has entered the lung tissue, it is acted upon by the acid body fluids, and the metallic atom is liberated from the crystallic lattice. The particles are now surrounded by

protein which envelops them like a coat of gel, and the silicate deteriorates while the iron remains. Asbestos bodies are excreted now and then with the expectorate. The organism has thus a chance of ridding itself of the absorbed asbestos.

As mentioned, asbestos bodies were demonstrated first in the lungs in patients who died of pulmonary asbestosis. Stewart has demonstrated them in the blood from punctures of the lung and in sputum, but only in patients who had been suffering from asbestosis through a number of years. He states, that it is difficult to find these bodies in the expectorate, and that they are to be looked for during an exacerbation of the bronchitis of the respective patient. As it did not seem advisable to perform a puncture of the lung in such a recent case as the one here reported, I collected 200 c.c. of sputum from our patient. Stewart had destroyed the sputum with antiformin, but I employed Burkes's method for demonstration of minerals in silicotic expectorate.

I evaporated the 200 c.c. of sputum till it formed a very thick butter-like mass, from which I made smears on slides; a part of it was treated with hydrochloric acid. By means of the polarization microscope and by incineration of the mass, it was possible to demonstrate several large refractive aggregates and smaller markedly refractive particles. But by comparison of these bodies with asbestos fibres that had been treated with hydrochloric acid in the same manner as the expectorate, nothing definite could be derived.

Mr. R. Bøgvad, M. S., who has been kind enough to carry out the mineralogical examination arrived at the following conclusion: None of the scarce filliform particles have the slightest resemblance to the asbestos bodies mentioned in the literature; most likely, these particles have nothing to do with asbestos or with asbestos bodies.

One of the electrodes which the patient used in his work has been examined. In the packing was found asbestos (actino lite) which is more resistant to acids than is the chrysotile asbestos found in asbestotic lungs by other investigators (Beger, Cooke).

The clinical picture of pulmonary asbestosis is described by Oliver as resembling in the early stage any other form of silicosis and differing but slightly from pulmonary tuberculosis. He emphasizes in particular the marked shortness of breath on slight exertion, deficient respiratory capacity and physical debility; as

differentiating signs he points out the absence of fever, a degree of dyspnoea in excess of the physical signs, a greater amount of general disability, and comparative absence of night sweat.

LÖRZ holds that pulmonary asbestosis can be classified by itself as a special characteristic occupational disease. It is associated with coughing, expectoration and shortness of breath, in particular on exertion and in foggy weather.

The patient's physician has told me—on May 8, 1935—that the patient is now feeling fairly well, the expectoration is minimal and he has regained his capacity for work to a fair degree.

Reexamination of the patient here in the hospital on May 13, 1935, shows the vital capacity of the chest to be 4000 c.c. (*i.e.*, 111% of the normal capacity for his weight and sitting height).

X-ray examination of the chest shows a mild degree of bronchitis and emphysema.

Auscultation of the lungs: There are still signs of emphysema, but no signs of bronchitis.

The diagnosis of pulmonary asbestosis is made on the anamnestic data, the sudden onset of dyspnoea, the finding of emphysema, bronchitis and asthma, and the prominent lung structures in the X-ray picture.

But the diagnosis cannot be said to be established with certainty unless the presence of asbestos is demonstrated in the expectorate. Yet, theoretically there can hardly be any doubt that the greater part of the asbestos will be deposited in the connective tissue, so that it is not very likely to be found in the expectorate in fairly mild or recent cases.

There is, besides, the possibility that this might perhaps be a case of poisoning through a production of noxious substances from the electrodes or from the welded material. The first possibility comprises the production of nitro-compounds which are markedly irritating to the respiratory tract. The welded material may also give off some noxious substances, especially if it is oiled—*e.g.*, acrolein.

The rapid improvement of the symptoms in this patient, I think, is chiefly attributable to his stay in the hospital by keeping him away from his dangerous job; in addition, his lungs were given a chance and time to break down and eliminate the asbestos.

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strated indisputably also in experimental studies (Berger), differentiates asbestosis from the other forms of silicosis. If Jones' view is correct, that ordinary silicosis is due in part to silicates, especially sericite—and many things suggest it is correct—it is an obvious conclusion that the silicates break down too, even though more

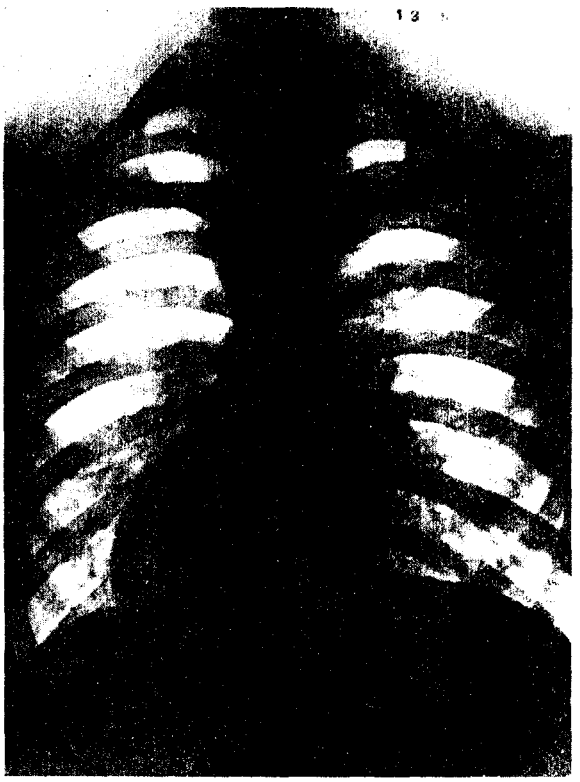

Fig. 2. Roentgenogram of the chest, nearly 5 months after Fig. 1.

slowly. In his work on silicosis, Böhme (1934) mentions the hydrolysis of asbestos and concludes: "Für Quarz und Silicate ist wahrscheinlich ein ähnlicher, wenn auch sehr langsamer Auflösungsvorgang in der Lungen anzunehmen".

Asbestosis is therefore to be designated as a special form of silicosis (or chalicosis), to which the individual resistance presumably is fairly great, and in which there is a marked tendency to spontaneous recovery, at any rate in the initial and early stages.

In recent years, investigation into occupational diseases and their

diagnosis have gained headway rapidly in this country, and I thought therefore it might be of interest to present before this congress an early case of pulmonary asbestosis. For early clinical demonstration of this condition, before the patient shows definite roentgenographic lesions and demonstrable asbestos bodies in the sputum, will give him the best chance of recovery. In analogy with tuberculosis and the demonstrable presence of tubercle bacilli in the sputum—once asbestos bodies are demonstrated in the sputum, the diagnosis of pulmonary asbestosis is established, but the prognosis is often to be regarded as less favourable.

I wish to thank my chief, Dr. N. R. CHRISTOFFERSEN, for inviting me to present this paper. I am also obliged to Mr. R. BÖGVAD for his kind assistance in the mineralogical part of this study.

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