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ASBESTOSIS BODIES IN SPUTUM AND LUNG*

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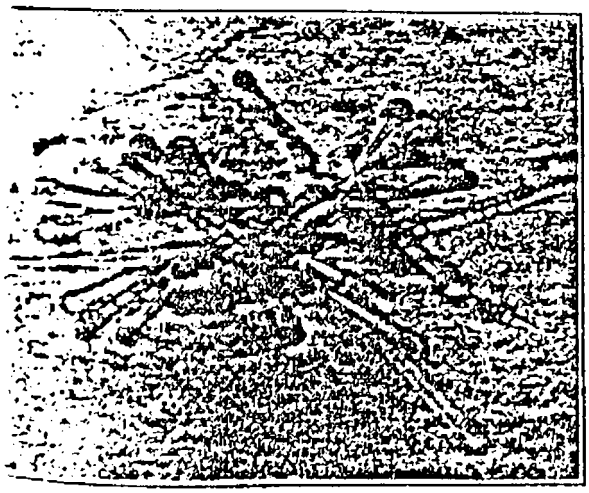
...estosis has, of comparatively recent time, assumed ...ition of prominent and perhaps peculiar inter... the field of pneumonoconiosis and occupa... disease. While the asbestos industry is ... than two thousand years old, it is only ...n the past few years that it has assumed ...minent place which it now occupies in ...dustrial arts. Until recently asbestosis ...een assumed to be essentially silicosis, and ...ree silica has been thought to constitute ...ngerous factor in pneumonoconiosis as ...ted with the relative harmlessness of ...r dusts.

...has come to attention within the last three ...ur years that there is a peculiar character... in the state of asbestosis, not found in sili... and it is our purpose in this report to ...d some data that have come to hand, since ...man medicine thus far has no report on ...peculiar condition and to the end that ...ular interest may be stimulated in its ... in the extensive asbestos industry. It is ...ted that a further report of our studies ...e made at a later time.

Cooke[1] recorded an autopsy on an ...ker who had been exposed for ..., the last five years intermittently, ...ing extensive fibrosis of the lungs and ...ar tuberculosis. At this time there was ... the lungs only black particles, from 3 to 393 ...ns in length, of various shapes.



...—Asbestosis bodies in sputum concentrate; reduced from a photo... ...ain a magnification of 705 diameters.

...1927, Cooke[2] again recorded this case and cited ...which a patient who had worked in an ... for ten years showed at autopsy pulmonary ...spicules of asbestos in the lungs. These two cases are apparently the only records of autopsies in cases of asbestosis up to that time.

In a companion article, McDonald[3] recorded a histologic study of the lung tissue in Cooke's case and stated that the appearances were practically the same as in another case the sections of which were in the possession of Dr. I. M. D. Grieve of Armlay, Leeds. McDonald found, in addition to well marked diffuse interstitial pneumonia, chronic bronchitis and emphysema, anthracosis and extensive tuberculosis, certain peculiar foreign bodies in the alveoli, bronchioles and

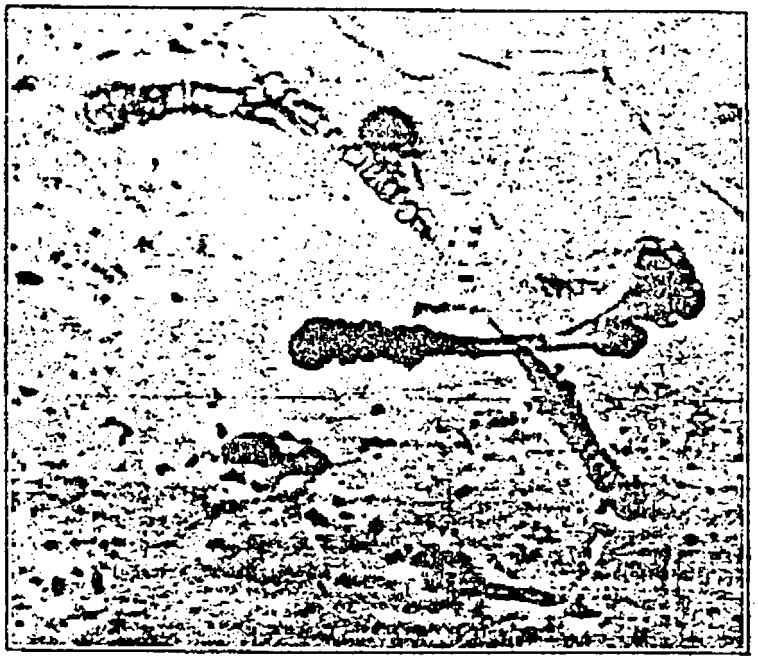


Fig. 2.—Asbestosis bodies in sputum; magnification about 950 diameters.

interstitial fibrotic areas. This is the first recorded observation of these objects, which have since been called "asbestosis bodies." There was considerable disagreement among zoologists, botanists and others as to their nature; McDonald advanced the hypothesis that they are portions of asbestos in process of alteration and absorption by hydrolysis, with the passing of the silica into a colloidal state and later into a gel.

In 1928, Simson[4] followed with a report of four autopsies on asbestos mill workers in South Africa, the first being done in 1926 without discovery of the asbestosis bodies at the time. One of these subjects worked for twelve months in the mill, developed tuberculosis and died; the second worked for two years in the same mill and had a prolonged pneumonia during this time. This patient had neither tuberculosis nor pneumonia at autopsy but fibrosis of the lungs. The length of exposure of the third and fourth patients is not given, but both died of lobar pneumonia and both showed very little fibrosis. In the lung tissues of all of the four were found "golden yellow" segmented structures with rounded ends.

The descriptions and illustrations by McDonald and Simson make it clear that they were dealing with the same object and that it is the same as we have found in the lungs and sputum of asbestos workers.

In October, 1929, there occurred in the service of one of us (K. M. L.) an autopsy on a patient dead of

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1. Cooke, W. E.: Fibrosis of Lungs Due to the Inhalation of Asbestos Dust, Brit. M. J. 2: 147 (July 26) 1924.
2. Cooke, W. E.: Pulmonary Asbestosis, Brit. M. J. 2: 1024 (Dec. 3) 1927.
3. McDonald, S.: Histology of Pulmonary Asbestosis, Brit. M. J. 2: 1025 (Dec. 3) 1927.
4. Simson, F. W.: Pulmonary Asbestosis in South Africa, Brit. M. J. 1: 685 (May 26) 1928.

a gunshot wound. This was a Negro man who, at the time of his death, was a worker in an asbestos mill, having done such work for a total of twenty-eight months during a period of about three years. At the autopsy there was nothing of significance beyond the gunshot wounds of the chest and abdomen. In microscopic examination of the tissues, however, the peculiar bodies under consideration here were found in the sec-

Fig. 3.—Asbestosis bodies in lung with phagocytes and black granular pigment within alveoli in pneumonia; reduced from a photomicrograph with a magnification of 475 diameters.

tions from both lungs, in alveoli, associated with fine black granular pigment and mononuclear cells, some being intimately associated with large mononuclear phagocytes, in the bronchioles, where there was also the black granular material and some mononuclears, in the alveolar walls, which were thickened, and within the connective tissues of the interlobular areas along the course of vessels and bronchi. Here there was a definite increase of fibrous tissue and much accumulation of black granular pigment.

At about the same time occurred another autopsy on a Negro man who was working in an asbestos mill at the time of development of lobar pneumonia from which he died. He had been in this occupation almost continuously for a period of four and one-half years. In this case, also, it was in the microscopic study of the tissues from the autopsy that the asbestosis was discovered, gross examination revealing only massive pneumonia of the whole right lung and upper left lobe.

Sections of all parts of the lungs from this case revealed the "asbestosis" bodies. There were great numbers of them in the alveoli, the walls of the alveoli, the deeper pleural tissues, the bronchi and the interlobular framework tissues, and they even occurred in thrombi of veins and in the peribronchial lymph nodes. Associated with them was much black granular pigment, in the alveoli, interstitial tissues and lymph nodes, and in these nodes was also a heavy deposit of yellow-brown granular substance of the same color as the asbestosis bodies. Mononuclear and polynuclear giant cell phagocytes were prominent in intimate connection with the asbestosis bodies in the alveoli, bronchi and veins. They contained much of the black granular material in the alveoli and bronchi, also. In the walls of the alveoli and bronchi and in the framework structures where these bodies lay was a cellular increase in fibrous tissue.

The peculiar bodies in the lungs of these two patients were first suspected of being mycelia and spores of a fungus, possibly *Aspergillus*. On investigation of the subjects and after we had learned of their occupation, the reports of McDonald and Simson were encountered and identification with the objects observed by them was established.

It then occurred to mind that these bodies should be found in the sputum of such patients, since they occurred in considerable numbers in the alveoli and bronchi, and that sputum examination might prove to be an important measure in the diagnosis of asbestosis. Consequently search was made for them in sputum of asbestos workers from the clientele of one of us (W. A. S.) Specimens of sputum were examined by direct wet smear preparation and by examining the concentrated sediment of sputum after digesting it in 10 per cent sodium hydroxide solution, kept warmed to about 80 C. until the mucus was well digested and the mixture watery.

The first sputum examined came from a man who by clinical and roentgen examination had active fibrocaseous tuberculosis and also a moderate grade of pneumonoconiosis. He had not worked in asbestos for approximately a year but had been previously so occupied for about ten years. The sputum contained great numbers of tubercle bacilli. Asbestosis bodies were readily found in direct wet slide preparations and in large numbers in the sodium hydroxide digest concentrate.

It was at this point that it came to attention that such sputum tests had already been made. In 1929 Stewart and Haddow,[5] and in 1930 Wood and Gloyne,[6] showed that they could be found by digesting the sputum with equal parts of concentrated antiformin and examining the centrifugated deposit. Subsequently we have found the asbestosis bodies in the sputum of two other asbestos workers and have failed to find them in one.

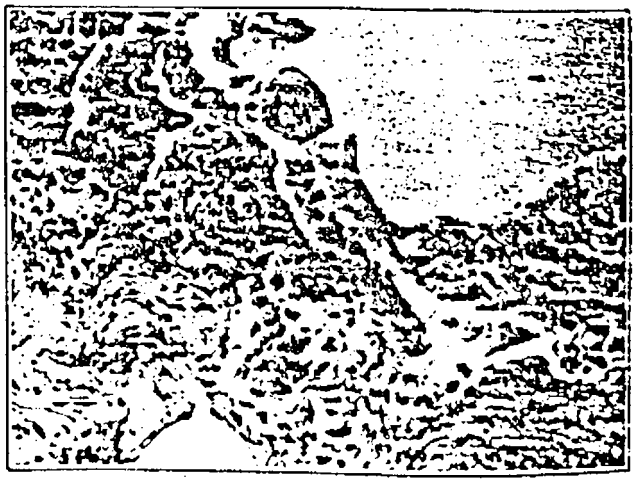

Fig. 4.—Asbestosis bodies with phagocytes and black granular pigment in fibrous lung; reduced from a photomicrograph with a magnification of 250 diameters.

The second patient was a Negro woman who had worked in an asbestos factory from 1918 to 1924 but not since. She had no evidence of pulmonary disease and was under treatment for syphilis. None of the asbestosis

5. Stewart, M. J., and Haddow, A. C.: Demonstration of the Previously Undescribed Bodies ("Asbestosis Bodies") in Material Obtained by Lung Puncture and in the Sputum, J. Path. & Bact. 32:172 (Jan.) 1929.

6. Wood, W. B., and Gloyne, S. R.: Pulmonary Asbestosis, Lancet 1:445 (March 1) 1930.

bodies were found in direct unconcentrated wet preparation. In the sodium hydroxide digest concentrate they were sparse and were not as long as those found in the case. The sputum was mucopurulent.

third patient had not worked in asbestos for ximately two years. He was employed in an asbestos factory for seven months in 1924 and for about two years from 1926 to 1928. He had active fibrocaseous tuberculosis of the lungs, with tubercle bacilli in the sputum but no condition distinguishable as pneumonoconiosis. The first sputum was mucoid in character and insufficient for concentration. No asbestosis bodies were found in direct wet unconcentrated preparations. In this specimen were large numbers of dust cells, with fine black particles and numerous fine needle-like crystals. The second sputum was mucopurulent. A few asbestosis bodies were found in the sodium hydroxide digest concentrate but none in the unconcentrated preparations. These sparse bodies were usually small, but some were long and thick.

The fourth sputum examination was done in the case of a white man who had worked in an asbestos factory for about fourteen years but not since 1926. He had advanced fibrosis of the lungs, thought to be a late stage of pneumonoconiosis, without any evidence of tuberculosis, and was in a state of advancing cardiac failure. Examination of sputum on three separate occasions by both direct and concentrated preparation failed to reveal any asbestosis bodies.

It was anticipated that this case, on account of the length of exposure and the extent of pulmonary disease, should have the best opportunity of exhibiting these bodies, but this expectation has not been realized. The patient had been exposed within about a year, and showed by far the greatest number of asbestosis bodies in the sputum, but the other two patients had not been more recently exposed, one had not worked in asbestos for about five years, and neither of these two gave any distinguishable evidence of pneumonoconiosis.

It remains a subject for further study as to the relation of the occurrence and time of duration of these bodies in the sputum to the extent of exposure to asbestos dust, and as to the state or stage of asbestosis or other associated conditions in relation to their expulsion in the sputum.

The asbestosis bodies are quite variable in size, color and form but generally are of a characteristic structure. In the sputum, from which they may be better studied alone, they have a central filament of a transparent, slightly greenish tinged needle-like crystal. This is taken to be an asbestos crystal. On this filament is deposited, in varying quantity, nodules, blobs and segments of a homogeneous refractive substance, varying from a shining light yellow or greenish-yellow to a deep mahogony brown, depending on the thickness of the deposit. In general this deposit occurs more on the extremities, usually in a spherical blob here, tapering toward the middle of the filament, which may be bare, giving the appearance of two clubs or baseball bats joined small end to small end. Again there may be a pearl of the deposit on the extremities and the shaft have a cylindric deposit of segments or disks of uniform or irregular size, giving the whole body the appearance of a dumb-bell. The clubbed form may break in the middle, leaving two indian-club forms. Again, the middle and extremities may be the seat of more or less symmetrical deposits of globules, piled up, to give a variety of architectural figures. The segments or disks of deposit along the crystal filament may be pressed closely together or they may be separated by an interval at which the needle-like central crystal may be seen. Sometimes the shaft is covered by a homogeneous non-segmented deposit.

In the lungs they are of the same forms, but their full length may not be seen and they usually appear shorter. The size of the body varies widely in length and thickness, with the length of the filament and the amount and uniformity of the deposit. No extensive measurements have been made in this study, but in the lungs they have been measured from 12 to 100 microns long and from 1 to 12 thick, the shaft being narrower than the ends. In sputum they have been measured from 1 to 12 microns thick and up to 140 microns long.

Asbestosis bodies do not take the ordinary tissue and sputum dyes and they have been found in their natural form in one of these cases in slide preparations of sputum stained by the ordinary carbolfuchsin method for tubercle bacilli.

They may be stained on the slide by the prussian blue reaction for iron, the heavier and darker brown bodies taking a deep indigo blue, this shading to a lighter color in proportion to the heaviness and darkness of the yellow-brown substance. This reaction indicates a considerable iron content to the deposit. This iron has been thought to signify that the asbestosis body comes from the iron content of asbestos. Since the material is evidently a deposit within the lung on an asbestos crystal, this does not appear to be a satisfactory explanation. It is suggested that the iron content, if not the whole substance of the deposit, is of tissue origin, probably from the blood.

Clinical Notes, Suggestions and New Instruments

THE EXECUTION OF ROBERT H. WHITE BY HYDROCYANIC ACID GAS

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Robert H. White was executed by hydrocyanic acid gas at the Nevada State Prison, June 2, 1930. A study of the heart and respiratory action was made at the time of the execution, a Bowles stethoscope being applied on the bare chest wall over the apex of the heart. The tube leading to the ear pieces on the outside of the prison wall was connected to the stethoscope while the prisoner was being strapped to the chair.

This was at 4:36 a. m. The heart action at that time was 108, strong and regular. The gas was started generating at 4:37½. At 4:38 the pulse rate was 120, regular and strong. A small inspiration was taken at 4:37¾, at which time the prisoner indicated that he smelled the gas. At 4:38 he took a very deep inspiration, turning his head toward the gas. He gave a spasmodic cough, his head fell forward, and he became unconscious. Following this first deep inspiration there was a complete stopping of the heart action for fifteen seconds. After that short period of time, or at 4:38¼, the heart again began to beat in an irregular manner, continuing thus for fifteen seconds, when it became regular and strong. There was no apparent loss of power in the heart action. After this time, for two minutes the heart became slower, beating 100 times a minute at 4:41½ and eighty times a minute at 4:44. At 4:46½ the heart beats were distinctly regular but becoming very weak. The last heart beat was noted at 4:47.

Respirations during this time after the first deep inspiration were convulsive and irregular.