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PULMONARY ASBESTOSIS II

Including the Report of a Pure Case[1]

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For a number of years one of us (W. A. S.) has observed, in the chest clinic at the Roper Hospital (Charleston) and at clinics held in certain sections of Charleston County, cases of respiratory disease which possessed characteristics not ordinarily encountered. The symptoms presented by these patients were cough of varying severity, usually slight but occasionally severe dyspnoea, expectoration and, in some, loss of weight. There were few or no constitutional symptoms. The physical signs consisted in most instances of poor expansion and fine râles at both lung bases. When roentgenograms were obtained they have been interpreted as being "negative" in some cases, as showing moderate degrees of granular mottling in others, and in a few rather extensive fibrosis. Sputum examinations failed to show tubercle bacilli. The occupational history revealed that at one time or another, for periods varying from months to years, these people had worked in an asbestos factory. Several such patients were diagnosed and treated as having pulmonary tuberculosis.

The significance of the relationship of the occupation and the pulmonary disability was realized when in the fall of 1927 an adult male, about 40 years of age, who had worked for 17 years in an asbestos plant, was seen in consultation with a local physician. This man exhibited the characteristic picture of the terminal stage of respiratory failure. He was emaciated, cyanotic and dyspnoeic. He had a severe cough, productive of a large amount of mucopurulent sputum in which no tubercle bacilli were found. The fingers were moderately clubbed; and the nails curved and cyanotic. There was a moderate daily rise of temperature. The expansion was diminished, resonance impaired over the lower lobes, and there were numerous coarse and moderately coarse râles over the entire chest. The roentgenogram showed fairly distinct granular and linear

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opacities at the lower portion of both lung fields, the apices were emphysematous, and there was well-defined "shagginess" about the heart. This man was seen on only a few occasions before he died. No necropsy was obtained. The diagnosis of pneumonoconiosis, possibly due to the inhalation of asbestos dust, seemed justified, and the case was reported as such to the Medical Society of South Carolina.

These experiences led to the conviction that prolonged exposure to asbestos dust presented a very definite health hazard, but until recently the matter had not been subject to proof. That the same impression had prevailed among physicians practising in or near asbestos factories is indicated by the statement of Cooke (1) that "Medical men in areas where asbestos is manufactured have long suspected the dust to be the cause of chronic bronchitis and fibrosis"; and of Sir Thomas Oliver (2), who points out that he has visited asbestos factories in America and has seen cases of pulmonary asbestosis at Armley, Leeds. He does not specifically state that he has seen cases of this disease in this country. Simson (3) in the introduction to his article on *Pulmonary Asbestosis in South Africa* says, "It has been known for some time that workers exposed to the dusty atmosphere arising from some processes involved in the preparation of asbestos materials suffer from pulmonary disability." From these expressions it would seem that the baneful effects of asbestos dust were long recognized, but until Cooke's (1) report in 1924, in which he describes the unusual morbid anatomy of the lungs, there was no conclusive evidence of relationship of this dust to pulmonary pathological changes.

As American medicine contains only meagre reference to this subject, it would seem timely to review briefly the progress that has been made in the study of this disease, analyze the cases reported, and at the same time put on record a complete case of pure pulmonary asbestosis, the first, so far as we have been able to ascertain, that has come to necropsy in this country.

The first readily available record of this condition is the case reported by Cooke (1) in 1924. That of a woman aged 33 years, who commenced work in an asbestos factory at 13 years of age and was almost continuously employed until two years before her death in 1924. Her symptoms were cough, dyspnoea, expectoration and hæmoptysis, all of which gradually increased in severity, being followed later by sweats and fever. Signs were first those of "fibrosis of the lungs" and two years prior to death the signs of cavitation appeared. The

necropsy revealed the peculiar type of pulmonary fibrosis in which granular dark brown pigment was found. There was also fibrocaseous tuberculosis.

In 1926 Pancoast and Pendergrass (4), together with Miller and Landis, examined 17 asbestos workers in the United States, two of whom showed "first stage changes and the other fifteen definite second stage appearance." This was apparently only a roentgen study and the classification was based from the point of view of silicosis. They rather discounted Cooke's findings, believing his fibrosis as likely due to tuberculosis alone.

In 1927 Cooke (5) published a more detailed discussion and elaborated on the subject of asbestosis. He cited the patient observed by Dr. H. Montague Murray at Charing Cross Hospital and reported in the Charing Cross Gazette in 1900. This was a man of 38 years of age who was admitted to the Hospital in 1899 and died in 1900. It is stated that the man informed Dr. Murray that he was the sole survivor of ten men who started work with him in the carding room of an asbestos plant ten years previously. The necropsy showed fibrosis with what Dr. Murray thought were "spicules of asbestos" in the lung sections.

Stuart McDonald (6), to whom was referred a specimen of Cooke's first case for histological study and to whom we are indebted for an excellent description of microscopic appearances of the lung tissue in this disease, refers to a case of Dr. Grieve's, sections from the lungs of which presented "appearances practically identical," death, however, resulting from bronchopneumonia.

Sir Thomas Oliver (2) reports in the same journal that he examined with Dr. Grieve of Armley, Leeds, two women suffering with pulmonary asbestosis. One was 48 years of age and had worked in an asbestos factory for thirty years. She gave up work the year before on account of shortness of breath and cough. She was much emaciated, and expansion of the chest was one inch. The physical signs present were flattening of percussion note at the bases. The breath-sounds were exaggerated at the top of both lungs and diminished at the bases. "Small dry friction sounds" were heard at the right base. "Moist tinkling sounds suggestive of cavity" were heard on the left, and the apex of the heart was displaced upward and outward.

The other patient was 30 years of age and had worked in an asbestos factory 18 years. She developed cough and asthma four years previously. She emained away from work for three months and then worked for three years. She had dragging pains in the chest, shortness of breath, and cough. The chief signs were moist râles in both axillae and small friction with crepitation heard at both bases. No tubercle bacilli were found in the sputum of either.

In 1928 Simson (3) reported four cases coming to autopsy in whose lung substance were found the "golden yellow bodies" now believed to be pathognomonic of asbestosis. The first subject was an adult native South African who had worked in asbestos for one year, and nine weeks before death he developed acute miliary tuberculosis. The second was also a male native who

had worked two years in a mill. He had well-marked lung fibrosis in which the "asbestosis bodies" were readily found. This case apparently died of uncomplicated pulmonary asbestosis. In two other asbestos workers dying of lobar pneumonia Simson also found these characteristic bodies.

In a communication to the editor of the *British Medical Journal*, September 5, 1928, M. J. Stewart (7), in describing his method of "immediate diagnosis of pulmonary asbestosis at autopsy" by squeezing the juice from a small piece of fibrotic lung on a slide to find the "brown bodies" of this disease, reports four cases. One was a woman 34 years of age who had been employed in asbestos work for 16 years. Both lungs were extensively fibrosed, and very anthracotic, but there was no evidence of tubercle, syphilis or silicosis. In the other three cases Stewart gives no details but states that the "asbestosis bodies" were found by Dr. F. L. Taylor in the lungs of these workers after death.

Stewart and Haddow (8), in directing attention to their method of exploratory lung puncture to obtain secretion for examination for "asbestosis bodies," report a case successfully diagnosed by this procedure. They also report the finding of these bodies in the sputum of this and one other patient. It appears that they were the first to suggest the value of sputum examination in the diagnosis of this condition.

Seiler (9), of Glasgow, reported in 1928 a case of pneumonoconiosis due to inhalation of asbestos dust. This was a man of 40 who had been associated with the asbestos industry for 22 years. He had cough, breathlessness, loss of weight, and lassitude for a period of several months. His physical signs and roentgenograms were characteristic of fibrosis of the lung. The patient was still living.

W. Burton Wood (10), of London, in an article on pulmonary asbestosis, which he illustrates with roentgenograms, bases the diagnosis of the 15 cases reported on the occupational history and the clinical and roentgenological manifestations. No record of the finding of asbestosis bodies appears.

In a subsequent article Wood in collaboration with Page (11) reports in detail the clinical and pathological findings on one of these patients who had died. This was a woman, aged 34, of nine years' service in an asbestos factory. She suffered with dyspnoea, loss of weight, and palpitation. She was emaciated and pale, her skin having a violet tinge. The physical signs showed flattening of the left side of the chest, impaired resonance over both bases, and "crackling crepitations of fibroid type" were heard over the whole of the left and the base of the right lung. The roentgenogram showed granular mottling throughout both lungs. The heart was slightly displaced to the left and its left border was obscured by heavy shadows in the lower lung field. At necropsy was found the typical fibrosis with amorphous dark brown pigment and numerous "golden yellow bodies." These bodies were also found in the expressed lung juice. Bronchopneumonia was apparently the terminal event. Later (12) these authors report a similar case, necropsy showing an associated tuberculosis.

Wood and Gloyne (13) recently reported having seen 37 cases, 15 of which were previously reported. In four the diagnosis was doubtful. There were four upon whom postmortem examinations were made, two of which were reported by Wood and Page, the other two being previously unreported cases.

Merewether (14) has recently reported upon a comprehensive group study of asbestos workers for the detection of pulmonary disability. Of 775 workers engaged in the more dusty processes of manufacture 374 were examined. Ninety-five, or 25.4 per cent, of these showed pulmonary fibrosis attributable to asbestos dust, and 21, or 5.8 per cent, were classified as showing a prefibrotic condition. The tabulation of those examined, by years employed in the industry, showed a marked increase in percentage of occurrence of fibrosis, varying from nothing in the first four years to more than 80 per cent affected in 20 years or more of exposure. The diagnosis of these cases was based on the occupational history, and the clinical and roentgen findings. The sputum was apparently not examined for asbestosis bodies.

Lynch and Smith (15) recently reported two necropsies on asbestos workers, one dying of gunshot wounds and one of lobar pneumonia, in both of which the lungs presented deposits of yellowish-brown pigment and asbestosis bodies. Included in this article was a report of four other cases, two of which had associated pulmonary tuberculosis, one syphilis, and the fourth pulmonary fibrosis with progressive cardiac failure.

The finding of asbestosis bodies in the sputum confirmed the diagnosis in three of these. In the fourth it was anticipated that because of the length of exposure and advanced pulmonary disease the exhibition of these bodies would be a simple procedure, while, as a matter of fact, with copious sputum and numerous examinations, none was found. Lung puncture was not attempted.

A Complete Case of Pure Pulmonary Asbestosis

This patient was a white male, 46 years of age when first coming under observation at the chest clinic at Roper Hospital in November, 1925. His physician had diagnosed pulmonary tuberculosis. This opinion was confirmed and he was sent to Pinehaven Sanatorium for treatment. No tuberculosis was known in his family and his history of past illnesses is unimportant.

Occupational History: He commenced work in a local asbestos plant in 1911 or 1912 as a carder, working steadily until 1919, losing two weeks during that year, and four weeks in 1920. He was out in 1921 and worked only three and one half months in 1922. He was out again in 1923, but worked from June, 1924, to May, 1925, making a total of approximately 11½ years.

Present Illness: In 1914 or 1915 he began to cough and to bring up a

small amount of sputum. In 1918 or 1919 he developed a pain in the lower right chest, which was aggravated by movement, but not affected by the cough or by deep breathing. Shortly after the pain commenced he began to lose weight in spite of having a good appetite. At about the same time he noticed that his breath was getting short. The pain and expectoration persisted, he tired more easily, and breathlessness became more pronounced. In August his left lower chest began to pain him and in October he expectorated a small amount of blood. He had no fever or night sweats and did not feel weak. The chest pains, loss of weight (23 pounds), and shortness of breath caused him to seek medical advice.

Physical Examination: November, 1925: A very lean man, 65 inches in height, weighing 97½ pounds, color somewhat dusky, lips slightly cyanotic. The finger-nails were slightly cyanotic and curved to some extent, but not clubbed. The neck veins were prominent. The chest wall was much emaciated, badly shaped, and of the "cobbler type." Expansion was very poor and unequal, being less on the left. Resonance was impaired anteriorly and posteriorly throughout the left side, and there was bronchial breathing to the second rib and fourth dorsal spine, with moderately coarse and coarse râles practically over the entire lung. On the right side there were moderately coarse and coarse râles to the fourth rib and eighth dorsal spine.

The roentgen report by Dr. A. R. Fatt is as follows:

Examination of the chest with the fluoroscope and film shows considerable amount of mottling in both lungs. Some in right upper but a great deal in left upper, sufficient to completely block the first interspace. In the second left interspace there is some breaking down with cavity formation about 2 cm. in diameter. (See figure 1.)

The examinations of other systems were negative. Sputum tests were negative for tubercle bacilli.

This patient remained in the Sanatorium from November 9, 1925, to January 15, 1926, when he became tired of the restrictions and deserted. During this period his temperature stayed within normal limits, except for one week in December when there was a daily rise to 100°. On the rest regimen he seemed to improve, his cough and dyspnoea lessened, and he gained 20 pounds.

He returned to work in the asbestos plant for several months in 1926, later obtaining employment in one of the city parks as a gardener. In

June, 1927, he was treated at Roper Hospital for acute prostatitis. He returned to the chest clinic at Roper Hospital in August, 1929, on the suggestion of the tuberculosis nurse. Although his cough, expectoration and chest pains were still present he had little complaint except for dyspnoea; the latter had grown progressively worse and was much aggra-

FIG. 1. CASE 10392. ROENTGENOGRAM, NOVEMBER, 1925

vated by exertion. He had considerable difficulty in doing any work at all. Except for appearing somewhat more "dusky," the physical examination showed little change except that the râles were more widely distributed. A roentgenogram made at this time was interpreted by Dr. R. B. Tait as follows:

There is scattered density throughout the chest. Hilar markings are very dense, and there are diaphragmatic adhesions on both sides. There is probably cavity formation in the middle left, although this is not positive, due to the peculiar appearance of the whole chest. (See figure 2.)

FIG. 2. CASE 10392. ROENTGENOGRAM, AUGUST 27, 1929.

He was not seen again until April 5, 1930, when he returned to the clinic seeking hospitalization. His dyspnoea, cough and expectoration had increased, and for the past two months his feet and legs had become swollen. He was weak, emaciated, cyanotic, and the lower extremities

were oedematous. Resonance was impaired over both lungs and there were numerous râles of all sizes over the entire chest. He was admitted to Roper Hospital, where he died of congestive heart failure about three weeks later.

His sputum was negative for tubercle bacilli and for asbestosis bodies on four examinations. The blood Wassermann was negative. The

FIG. 3. Case 1092. ROENTGENOGRAM, APRIL 10, 1930.

haemoglobin was 85 per cent, white cells 7,975, lymphocytes 17 per cent, transitionals 1.5 per cent, polynuclears 75.5 per cent, eosinophiles 3.5 per cent, basophiles 2.5 per cent. The urine showed a trace of albumen and coarsely granular casts on two examinations.

Dr. R. B. Tait's roentgen report of April 10, 1930, is as follows:

There is a large amount of fibrosis scattered throughout both sides with evidence of a cavity in the middle portion of the left, and a larger cavity in the middle portion of the right. Both of the apices appear to be evenly consolidated which suggests a small amount of fluid causing a partial collapse of the lungs. Heart and mediastinum are very much displaced to the left." See figure 3.)

Autopsy

No. 10302: The body had been embalmed prior to the autopsy. There was marked subcutaneous oedema, especially of the feet and legs to above the knees, and some of the hands and forearms. The toes and fingers were stubby, but there was no cyanosis, perhaps interfered with by the embalming. The sternum was flattened above and definitely depressed over the epigastrium.

The pericardium and heart were definitely displaced to the left, the apex well outside the nipple line at about the fifth interspace. The heart weighed 345 gm. empty, was definitely broadened but not lengthened, the right heart furnishing the breadth, the left not enlarged. The right cavities were well enlarged and open, the left ventricle closed. The right myocardium measured 8 to 10 mm. in thickness well up from the apex toward the pulmonary orifice. Microscopically the muscle fibres were not of uniform size, and there was generally a distinct enlargement of those of the right. They were of indistinct outlines and striations, and those of the right ventricle were frequently vacuolated. There was an increase of stroma, and the veins, particularly of the right, were definitely distended with blood.

The aorta was the seat of a minor atheroma, and the whole vascular system, outside the heart, was in good condition.

Both pleural sacs were completely obliterated by old adhesions, usually very dense, especially over the whole right lung and the upper and lower left. The diaphragm was up to about the fourth rib on the left but was low on the right. The left lung was retracted well over to the outer wall and high, occupying about one half to two thirds of the usual space. The pleura generally was thick and cartilage like, especially over the apex and base. The lung was coarse, leathery, nodular and lumpy over the upper half and the base, the middle being more spongy and air bearing. The vessels and bronchi were very prominent and the framework coarse, variously there were prominent emphysematous bullae in the parenchyma especially in the apex where there was a group or chain of honey-

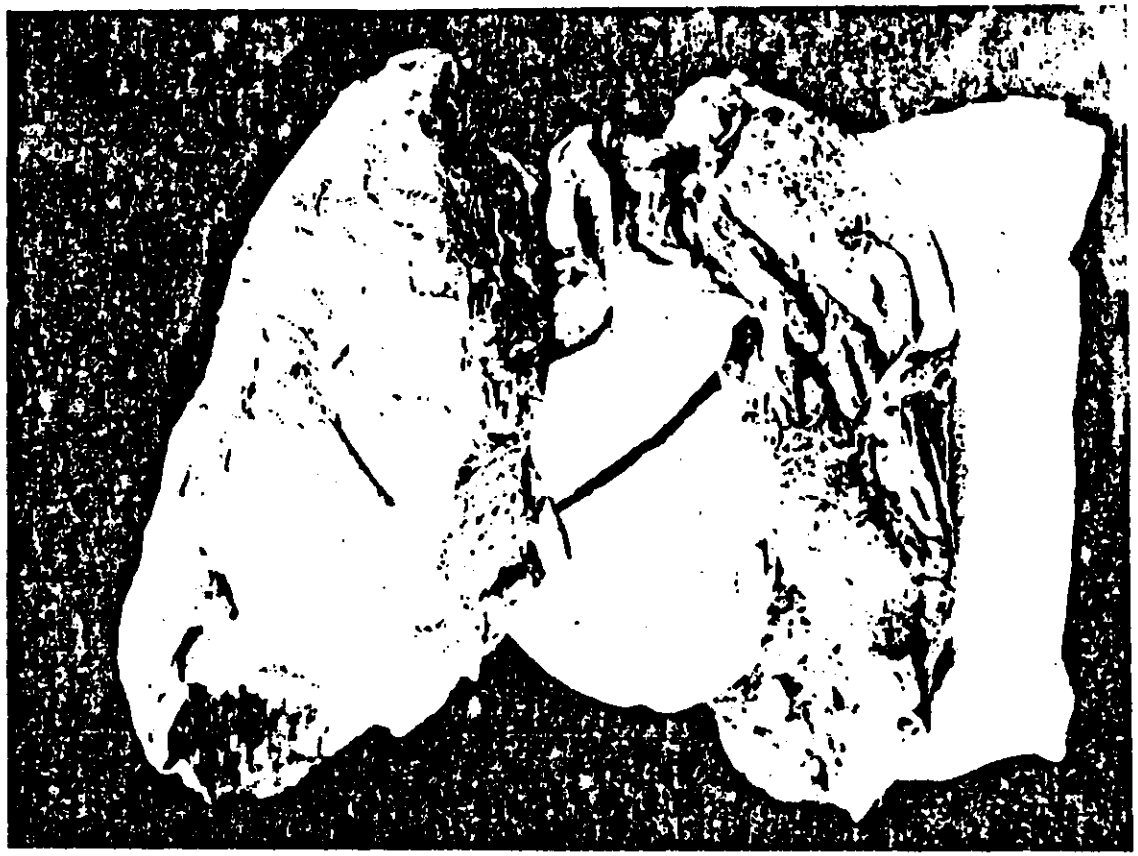

Fig. 1.—Lung sliced with Extreme Fibrosis of Lungs and Hypertrophy of Right Heart. (X 1/3.)

combed sacs. The interlobar pleura was obliterated. The bronchi appeared congested. The hilum lymph nodes were inconspicuous, did not appear enlarged, and were smoky black. There was no evidence of tuberculosis.

The right lung was large, filling the whole right chest and encroaching some toward the left. (See figure 4.) Its whole pleura was very thick and cartilage-like. The interlobar pleura was sealed outwardly, but was open, a clean membrane presenting, between the adjacent lobes. The lung was very much like its fellow generally, the upper lobe densely fibrous and lumpy, vessels and bronchi prominent, and a mass of em-

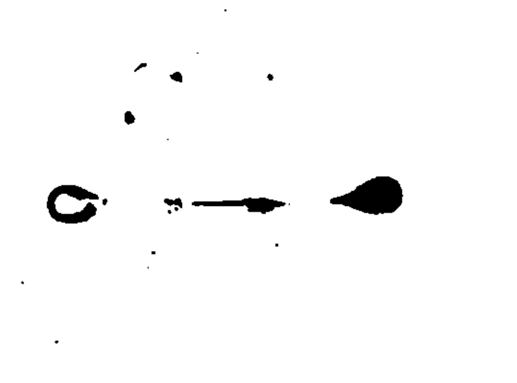

FIG. 5. GIANT CELL PHAGOCYTE CONTAINING AN ASBESTOSIS BODY. MONONUCLEAR PHAGOCYTES WITH GRANULAR PIGMENT. FRESH UNSTAINED [illegible] OF LUNG SUBSTANCE. CASE 10602. PHOTOGRAPH [illegible]

physematous bullae in the apex. The middle lobe was not so fibrous, nodular or lumpy, and had prominent bronchi and air bullae. Both lungs, especially the right, bore much frothy fluid, which, expressed and examined, revealed numerous asbestosis bodies, free, in sheaves or clumps, and ingested by giant cells. (See figure 5.) The two ends were often engulfed by different giant cells. There were also a few dust cells, with fine black granular pigment, and masses of a yellowish amorphous substance, of the color of the asbestosis bodies, in cellular debris.

Microscopically there was an extreme grade of hyalinizing fibrosis of the lungs, universally but irregularly distributed. The pleura was thick

and fibrous, and there was marked interlobular fibrosis. Scattered here and there were irregularly rounded areas of hyaline fibrous tissue, in somewhat laminated form, within which were masses of greenish-black granular substance. (See figure 6.) Here were also areas of liquefaction and calcification in the centre of these hyaline nodules. A large part of the alveoli were obliterated or virtually so. Some lobules remained open, the sacs having thick fibrous walls. In these open alveoli the epithelium was sometimes cuboidal and there were fairly numerous

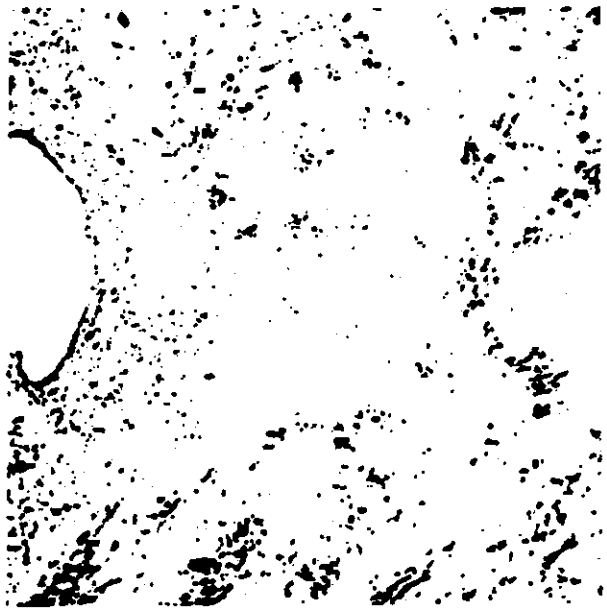

FIG. 6.—LATE STAGE WITH EXTENSIVE FIBROSIS AND BRONCHIECTASIS. Case 10892. PHOTOMICROGRAPH. × 87·5

large round phagocytes, some with a group of nuclei; some mononuclear. These macrophages contained black or greenish-black or brownish granular pigment and an occasional asbestosis body. Where the lung was less fibrous there were young connective-tissue cells and lymphocytic accumulations. The bronchioles were dilated and their walls thick and fibrous. In some areas the lobules of alveoli showed marked emphysema, large empty sacs with thin walls. Asbestosis bodies in typical forms, with a variety of architectural figures, yellowish brown, clubbed, dumb-bell and rod forms, were to be found widespread, singly or in groups

within giant cells in the alveoli of less fibrous areas, singly in the alveolar walls and interlobular tissues showing the younger fibrosis. (See figure 7.) Associated with them was much granular substance of the same color, as if from disintegrated asbestosis bodies. Beside these pigments there was much ordinary black anthracotic material around the vessels of the interlobular tissues. The interlobar pleura was especially thick.

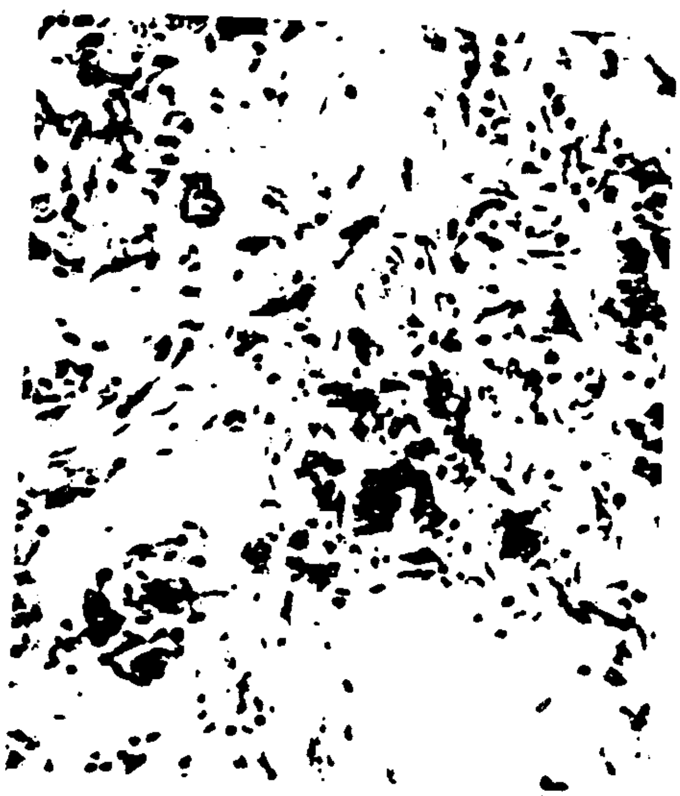

FIG. 7. PROGRESSIVE FIBROSIS WITH ASBESTOSIS BODIES AND GRANULAR PIGMENT IN ALVEOLI AND FIBROUS TISSUE. CASE 10302. PHOTOMICROGRAPH. ×280

The large bronchi were practically normal. The peribronchial lymph nodes were the seat of marked fibrosis, oedema, atrophy of follicles, and accumulation of masses of black and yellowish-brown granular pigment, of the same order as that in the lung.

The liver was grossly and microscopically in a state of extreme chronic passive congestion, which state, in lesser degree, was conspicuous in spleen, kidneys and other viscera.

FIG. 8.—ASBESTOS BODY AS SEEN IN SPUTUM AFTER DIGESTING WITH 10 PER CENT SODIUM HYDROXIDE. FROM ANOTHER CASE. PHOTOMICROGRAPH ×950

COMMENTS ON THE CASE

This man entered the employ of a local asbestos plant shortly after it had commenced operation. The factory buildings were renovated old structures which had been erected for other purposes. It has been ascertained that ventilation was extremely poor and no provision had been made for removal of dust at this time. No one suspected the dangerous properties of the dust. It seems reasonable to assume that the man was exposed for many years to air heavily laden with finely powdered asbestos. When this man first came under observation five years before his death his lungs were already irreparably damaged and it was not anticipated that he would survive many months. It was during the last years of his life that the diagnosis of fibroid tuberculosis, which had been made in spite of the absence of toxic symptoms and absence of tubercle bacilli, was questioned. This previous diagnosis was largely due to upper-lobe involvement and to a misinterpretation of the roentgen films.

The irregular dense opacities surrounding areas of translucency, due to emphysema, suggested cavitation, even when reread in the face of knowledge to the contrary. No cavities were found postmortem. There was a small pneumothorax on the right side on a level with the superior fissure, which extended downward, separating the upper and middle lobes. This shows up very well in roentgenograms of August, 1929. (See figure 2.) The other cavities suspected in the roentgenograms were undoubtedly groups of very large emphysematous bullae.

It is remarkable that no asbestosis bodies were encountered in the sputum, as this was mucopurulent and copious in the terminal stages of the patient's illness. These bodies were found in abundance in the expressed lung juice and in the alveoli in lung sections. On account of the extreme fibrosis and the fixed condition of the lungs it is likely that there was very little expulsion of material from alveoli into the bronchial tract. Perhaps in such late cases sputum examination for asbestosis bodies may not be as valuable as in earlier stages.

This is undoubtedly a pure case of long-standing asbestosis, with consequent extreme hyaline fibrosis of the lungs, and consequent obliteration of a large part of air bearing tissue, emphysema, bronchiectasis, increased pulmonary resistance to the circulation, hypertrophy of the right heart with eventual degeneration, fibrosis and congestion of the heart, progressive heart failure, with generalized passive congestion of the viscera.

and death from slow cardiac failure, the natural end-result of uncomplicated disease of the lungs of this extent and character. It is interesting to note in the lung changes the common laminated hyaline fibrous nodule, which has been described heretofore as a characteristic lesion of the fibrosis of pulmonary silicosis.

SUMMARY

In a survey of all available literature on the subject up to the present time we have collected 172 cases of pulmonary asbestosis. There are references to this subject in one or two abstracts, notably those of Bridge (16) and of Sir Thomas Oliver (17), but specific cases are not enumerated. In four cases belonging to Wood's series the diagnosis was doubtful, and in the majority of others the diagnosis was based entirely on clinical and roentgen findings. There were 27 in which the diagnosis was confirmed by the finding of the asbestosis bodies in the sputum, in the lung juice by puncture, or by necropsy. Necropsy has been made on 18 cases. In three of these the disease was complicated by pulmonary tuberculosis, three by lobar pneumonia, three by bronchopneumonia, and one was a traumatic death. In 4 the authors failed to give a complete report, stating only that the necropsy confirmed the diagnosis. Including the very first case recorded, that of Murray in the Charing Cross Gazette of 1900, which apparently received little attention until resurrected by Cooke, there are now 4 records of necropsy on uncomplicated pulmonary asbestosis. Except those reported by ourselves, and those by Pancoast and Pendergrass, and four others by Simson from South Africa, these cases have all developed in the British Isles.

Since this article was submitted for publication the following report has been encountered: Mills, R. G., Pulmonary Asbestosis: Report of a Case, *Minnesota Medicine*, 1930, xiii, 495. This was apparently a pure case in which death occurred some seventeen years after exposure to asbestos dust in South America.

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