PULMONARY ASBESTOSIS.*

BY

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(With Special Plate.)

IN the BRITISH MEDICAL JOURNAL of July 26th, 1924 (p. 147), I published a short note on the woman who is the subject of this paper. The only similar case on record was that of a man admitted to the Charing Cross Hospital in 1899, where he died in 1900. Dr. E. L. Middleton of the Home Office kindly lent the notes of the case, and of the evidence given before the Departmental Committee on Industrial Diseases in 1906 by the late Dr. H. Montague Murray, under whose care the man had been. This patient, a man aged 33 years, had worked in the carding room of an asbestos factory for ten years prior to his admission to hospital. He informed Dr. Murray that he was the sole survivor of ten men who started work with him in the carding room; the others had died, presumably as the result of their occupation. A *post-mortem* examination was held and the diagnosis of pulmonary fibrosis was confirmed. Dr. Murray in his evidence refers to photomicrographs of lung sections which show "spicules of asbestos." These are the salient facts of the first and, down to 1924, the only record of a death due to asbestos.

That these two cases stand alone is very surprising. The asbestos industry is more than 2,000 years old, and we know that asbestos factories, up to quite recent years, have been devoid of any appliances for the prevention and extraction of dust. The remark of Dr. Murray's patient is suggestive, and medical men have long suspected asbestos dust to be the cause of lung conditions in workers in badly ventilated factories.

Asbestos is a physical paradox—a mineralogical vegetable, both fibrous and crystalline, elastic, and brittle; as capable of being carded, spun, and woven as wool, flax, or silk. A single strand can be spun to weigh less than an ounce to 100 yards, and a cloth manufactured which weighs less than 8 ounces to the square yard. It occurs in every country, but is never found in any two countries alike, nor, indeed, in any two parts of the same country.

Historical.

Asbestos is apparently indestructible, and its fire-resisting qualities were known to the ancients. The Romans mined it from the Italian Alps and the Ural Mountains. Herodotus (*circa* 450 B.C.) described a cremation cloth made from asbestos. Pliny (*circa* A.D. 50) mentions the difficulty in weaving it. Strabo (*circa* 30 B.C.) and Plutarch (*circa* A.D. 70) both speak of the wicks of the lamps of the Vestal Virgins being made from asbesta, so called because they maintained a perpetual flame without being consumed. Pausanius (*circa* A.D. 175) refers to a gold lamp made by Callimachus of Athens for Minerva, the wick of which was made of Carpasian linen, "the only linen which is not consumed by fire." Later (A.D. 1250) Marco Polo writes that he had seen Tartars using cloth that withstood fire which was made of a "Certaine Minerall of Earth found in a Mountayne."

Although its valuable properties have been known for thousands of years the modern adaptation of asbestos to the industrial arts dates from only a few years ago.

Composition.

Asbestos is one of the silicates, and its varieties are numerous. Wherever it occurs it is found associated with other minerals, more especially with chrome iron and magnetite. The composition of the well known Italian and Canadian fibres is as follows:

	Italian Fibre.	Canadian Chrysotile.
Silica	40.30	40.87
Magnesia	43.37	41.50
Ferrous oxide	0.87	2.81
Alumina	2.27	0.90
Water	13.72	13.55

The purest asbestos, having fibres of extraordinary length, occurs in Northern Italy. Asbestos may contain from 0.5 to 15 per cent. of iron oxide, but asbestos yarn is prepared from mineral as free as possible from iron. To get rid of the difficultly soluble iron, asbestos is soaked in orthophosphoric acid solution and washed in water before manufacture. The percentage of iron, then, is of recognized importance.

Manufacturing Process.

The process of manufacture resembles that of cotton. The crude mineral is subject to mechanical treatment in a grinding machine. The heavier rock is separated by gravity, and the remaining asbestos passed through carding, roving, and spinning machines, and from these to the weaving sheds.

During the carding process, and to a less extent in all the processes, a very considerable amount of dust is generated. In up-to-date factories all machines are fitted with extractor covers and the dust removed. In the first factory where the patient the subject of this paper was employed no method of dust removal was used, and the atmospheric conditions were occasionally so bad that workers in her particular room could not see each other.

Asbestos Fibre and Dust.

Microscopically asbestos fibre is seen to consist of two very different elements. The bulk of the fibre is translucent and glistening with here and there black opaque angular particles (Fig. 1). Minute black granules also are present. These black particles are actually part of the fibre but their appearance suggests a different chemical composition and different physical characters from the translucent portion. The dust generated during manufacture is seen to consist of these sharp angular particles and minute granules, suggesting, of course, that they are more brittle than the translucent part of the fibre. These particles are found in very small numbers in the finished article. Mr. T. H. Byrom, F.I.C., analysed several samples of dust, and found that the dusts containing the greater numbers of these black particles contained the largest amount of iron. The iron content of the finished article, raw material, and dust is as follows:

Finished article: Iron (as ferrous oxide)	0.1%
Crude raw material: Iron (as ferrous oxide)... ...	2.61%
Dust from carding room: Iron (as ferrous oxide) ...	18.4%

From these results it appears conclusive that the blackened brittle parts of the asbestos fibre are the iron-containing portions—the bugbear of the manufacturer, the cause of "dust," and a danger to the health of workers in the process of manufacture.

Clinical History of the Case.

The deceased, a woman aged 33 years, commenced work at the age of 13 years in an asbestos factory in which no provision was made for the extraction of dust. From an early age, soon after commencing work, she suffered from a cough, which did not interfere with her general health until 1917. She was then 30 years of age, and had been working thirteen years. From this time until 5 years later (1922) her attendances at work were intermittent owing to ill health. She missed occasional days and one or two periods of some weeks, until she finally ceased work in July, 1922.

Up to this she complained of cough, dyspnoea, expectoratio

DESCRIPTION OF PLATES.

FIG. 1.—Asbestos fibre, the bulk of which is translucent and in which are black angular iron-containing fragments. These constitute a large proportion of the dust generated in manufacture. (× 150.)

FIG. 2.—Large particles of asbestos in fibrotic area of lung. (× 150.)

FIG. 3.—Particle of asbestos 360 microns in length in necrotic area of lung. (× 150.)

FIGS. 4, 9, AND 12.—Curious bodies. (× 400.)

FIGS. 5, 6, 10, AND 11.—Curious bodies showing discoid arrangement and globular ends. Fig. 6 shows particles of these bodies and granular dust in a phagocytic cell. (× 1000.)

FIG. 7.—Fibrosed lung with clusters of the curious bodies lying free in alveoli.

FIG. 8.—Fibro-caseous area with giant cells.

Figs. 1 to 6 are reproduced by kind permission of the Editor of the *Journal of the Royal Microscopical Society.*

*This and the two following papers on this subject were read in the Section of Preventive Medicine at the Annual Meeting of the British Medical Association, Edinburgh, 1927.

and lassitude. The physical signs in her chest were those of fibrosis of the right lung. In July, 1922, signs of cavitation were noticed, the sputum became more profuse, with sweats and irregular temperature, and she died on March 14th, 1924.

An *x*-ray plate showed extensive fibrosis, more marked in the right lung, two calcareous glands at the root of the left lung, and two small calcareous particles in the base of the left lower lobe.

Macroscopical Appearances.

Right Lung.—The pleura is thickened over the entire surface of the lung, and shows the remains of dense adhesions to the chest wall and pericardium. The lung is firm and small. The glands at the root of the lung are large, and on section are black, show a thickened capsule, and some calcareous particles. On section, the lung is seen to be fibrosed and to a large extent airless, the lung tissue being replaced by fibrous tissue. Dense strands of fibrous tissue from the pleura intersect the lung. In the apex there is a large cavity, the size of a peeled tangerine orange. The middle and lower lobes show numerous small areas—varying in size from a hazel-nut to a pin's head—of caseation, some of which have proceeded to cavitation. The bronchi are dilated.

Left Lung.—The pleura is thickened and shows the remains of adhesions to the chest wall. The thickening and adhesions are not so marked as in the right lung. The lung is firmer than normal. At the root of the lung are two large calcareous masses, one the size of a large hazel-nut, the other about half that size—calcified tuberculous glands. The other glands are black and show periadenitis. In the left apex there is an area of old scar tissue about the size of a sixpenny piece, and a cavity the size of a walnut. Scattered throughout the lung are small areas of denser consistence than the rest of the lung, some of which show definite calcareous particles, others small areas of caseation. There is a considerable increase in the fibrous tissue.

Three outstanding features are presented by sections from this case. The first is the enormous amount of fine granular pigment in the peribronchial fibrous tissue, walls of alveoli, and in phagocytes scattered through the sections. The particles of this dust are similar in size and shape to the black granules seen in the asbestos fibre.

The second unusual feature is the presence of large solid angular particles (Fig. 2). These are situated in areas of fibrosis and in caseating areas. They vary in size from 3 to 360 microns in length. The particles are so large—masses of them are seen in certain areas—that they must have occluded small bronchi. Fibrosis of the alveoli supplied has taken place and later necrosis, as seen in Fig. 3.

We have never seen anything parallel to this in pneumonoconiosis due to other dusts, nor have we been able to find such occurrence in literature. On comparing these large particles with asbestos dust there is a striking resemblance in sizes, shapes, and colour. In fact, it is very easy to take each single particle found in the lung sections and immediately find its brother in a slide made from the dust.

We cannot think there is any reasonable doubt that the particles in the lungs are the heavy, brittle, iron-containing fragments of asbestos fibre. The more extensive involvement of the right lung is thus explained. The heavy particles would pass more easily down the more vertical right bronchus than the horizontal left bronchus.

HISTOLOGY OF PULMONARY ASBESTOSIS.

BY

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(*With Special Plate.*)

My remarks are confined to the histological appearances in the lungs in this condition, with special reference to certain foreign bodies of most unusual appearance which are present both in the alveoli and interstitial substance of the lungs. The observations are based almost entirely on material supplied from the case described by Dr. Cooke. The investigation has been conducted in the pathological department of the University of Durham College of Medicine.

I may state, however, that the appearances are practically identical with those observed in a second case of this condition, sections of which I have had an opportunity of examining through the courtesy of Dr. J. M. D. Grieve of Armley, Leeds.

Histology.

Numerous sections have been made from both lungs. The changes are more marked on the right side, but the appearances in the two lungs only differ in degree. They may be summarized as follows:

1. There is well marked diffuse interstitial pneumonia with chronic bronchitis and some emphysema.
2. There is well marked anthracosis.
3. There is an extensive tuberculous condition with chronic phthisis.
4. In the alveoli, bronchi, and bronchioles, and also in the interstitial fibrotic areas, are certain foreign bodies which will be described in detail later (Fig. 7).

As this communication deals specially with the nature of the foreign bodies, the general histological features will be dealt with very briefly.

The interstitial fibrosis is such as might be expected as a result of a combination of a pneumonoconiotic condition and a chronic tuberculous infection. The typical whorled formations seen in a more purely silicotic condition are not present. There is a marked endarteritis in the smaller branches of the pulmonary arteries; some are thrombosed and organized. Many of the smaller bronchi are obliterated; some have still caseous-looking centres. Some of the alveoli show the usual metaplasia of their lining cells into cuboidal form. The fibrosed and thickened walls of the bronchi in many places gradually merge into the areas of diffuse fibrous overgrowth. There are numerous foci of lymphocytic cells among the fibroblasts. Some of these seem obviously derived from lymphoid tissue in the bronchial walls. The interstitial fibrosis is progressive. The tuberculous condition is obvious histologically. Tubercle bacilli were not detected, but the histology is characteristic. The lesions are chronic in character, and there is no special indication of an acute exacerbation. There is well marked caseous bronchitis with lymphatic spread and numerous fibro-caseous deposits with giant cell systems (see Fig. 8). The bronchi, which are not specially the seat of tuberculous change, show catarrh with peribronchial thickening. There are numerous emphysematous areas. The alveoli show, in the majority of cases, some thickening of their walls, and contain many catarrhal cells, apparently derived from the lining cells; a similar catarrhal change is seen in the terminal bronchioles, some of which are dilated.

The Foreign Bodies.

The larger black and irregularly fragmented bodies which have been described by Dr. Cooke were not very obvious in the material I examined, but were clearly seen in some microscopical preparations of his which I had the opportunity of examining. I shall not refer to them specially, but confine my attention to certain highly characteristic and much smaller bodies which are abundant in all the sections examined. Some of these are free, but many are phagocytosed by the large mononuclear cells in the alveoli (Fig. 5). Some are easily included in comparatively small phagocytic cells, but the majority are larger, varying in size from 20 μ to 70 μ, or even more in the case of certain elongated forms. The smaller bodies are rounded and homogeneous, and all have a distinct yellowish-brown colour suggesting blood pigment. The longer forms have a highly characteristic appearance, strongly suggestive of some organic structure. Most have an annular appearance, which on closer examination can be resolved into a closely set series of rounded discoid bodies (Figs. 4, 5, 10, and 11). In some cases the globular forms are arranged along the more filamentous forms and occasionally are clustered at the ends of the rods, simulating sporangia of a hyphomycetes (Figs. 5, 6, 10, and 11). Some have club-like extremities at one or both ends of the filaments. Others, again, suggest the appearance of minute crustacean forms (Fig. 10), but closer examination does not support the idea of either vegetable or animal origin. These bodies do not stain with the ordinary aniline stains, but preserve their original yellow-brown colour. They are seen well in unstained sections. They give a characteristic prussian-blue reaction with potassium ferrocyanide and dilute hydrochloric acid. The reaction is not so obvious unless the solutions are slightly warmed. Where the bodies are too large to be phagocytosed by individual cells they tend to become surrounded by plasmodial masses. Many of the phagocytes contain much carbon pigment in addition. Though these bodies are mainly found in the alveoli and

(Illustrating the papers by DRS. W. E. COOKE *and* STUART MCDONALD.)

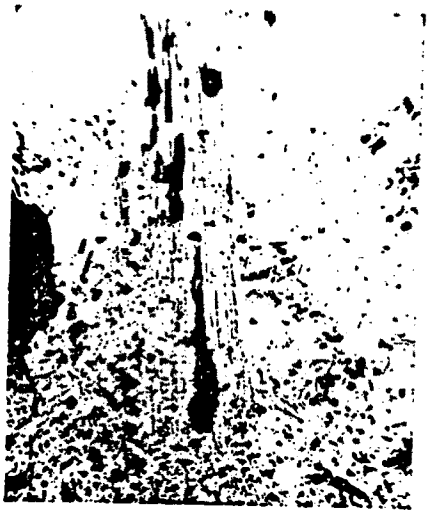
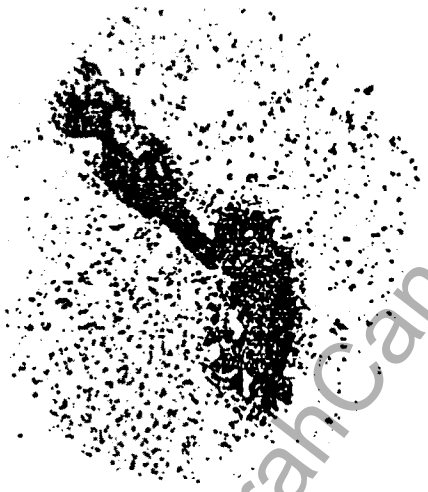


FIG. 1.

FIG. 2.



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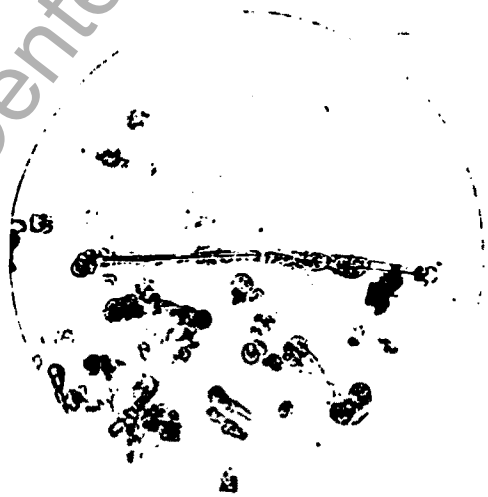

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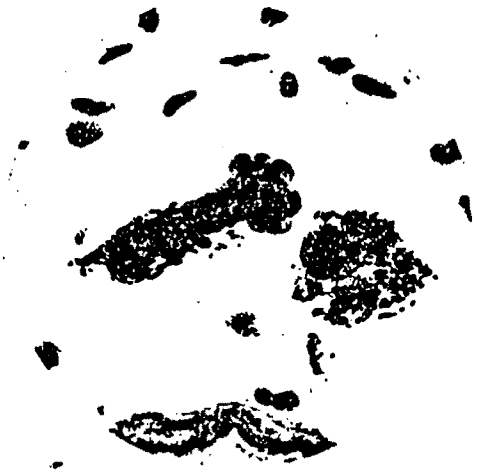
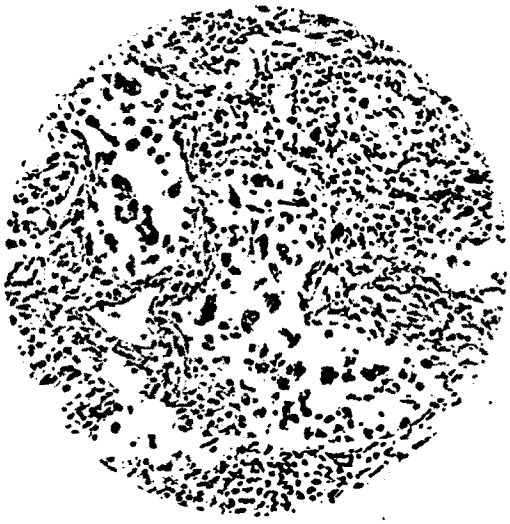


FIG. 6.

(Illustrating the papers by DRS. W. E. COOKE *and* STUART MCDONALD.*)*



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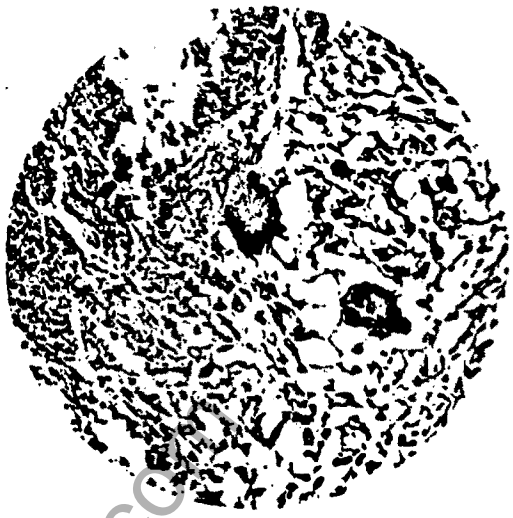

FIG. 8.

FIG. 9.

FIG. 10.

FIG. 11.

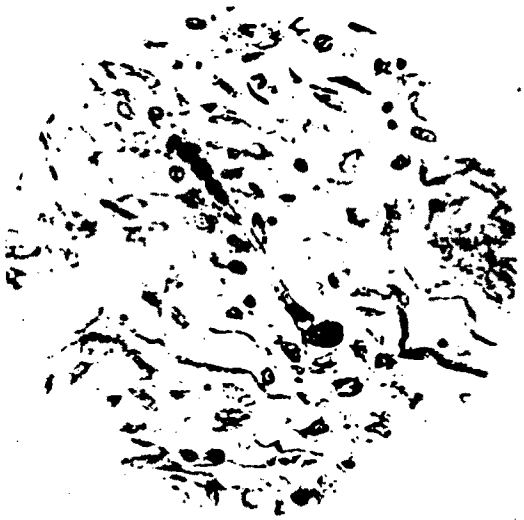

FIG. 12.

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