

Wood
Ellman

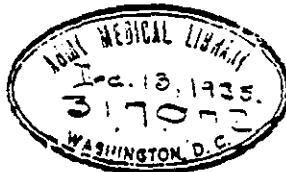
U50B

PLAINTIFF'S
EXHIBIT
SA-39

The BRITISH JOURNAL OF RADIOLOGY

FOUNDED 1896

New Series
VOLUME VII
JANUARY TO DECEMBER, 1934



PUBLISHED MONTHLY BY
THE BRITISH INSTITUTE OF RADIOLOGY
32 WELBECK STREET, LONDON, W.1

PNEUMONOCONIOSIS.

(A Discussion held at the Institute on January 19, 1934)

PART III—PULMONARY ASBESTOSIS

By PHILIP ELLMAN, M.D., M.R.C.P.

Physician to: The Tuberculosis and Chest Clinic, East Ham; The London Clinic and Institute of Physical Medicine, Pimlico; Battersea General Hospital; and Consultant, London County Council.

(Received February 21, 1934)

THE kind invitation of your President is my excuse for partaking in this discussion on "the pneumoconiosis." I have, during the course of my work, had an opportunity of seeing a number of cases of asbestos pneumoconiosis—pulmonary asbestosis—and it has, furthermore, been my good fortune to have had the help and close co-operation of my friend and teacher in chest radiology, Dr. Stanley Melville, in the radiological study of my cases.

The close study which has been devoted to silicosis, hitherto considered to result from exposure to silica dust, has established fairly clearly the clinical and pathological results which follow, together with the greatly increased predisposition to succumb to tuberculosis. Similar close study is to-day being given to the effects which follow upon the inhalation of another dust arising when asbestos is used in industry. The significance of the silicates, as exemplified by asbestos in the production of a pulmonary fibrosis, added to the recent work of Dr. W. R. Jones, of the Royal School of Mines, in connection with the significance of the silicates as exemplified by "serricite" in the causation of silicosis, is of outstanding importance. If the work of W. R. Jones is confirmed by others, as is not unlikely, our whole conception of the "pneumoconioses" will undergo marked revolutionary changes.

A full description of the mineral, asbestos, and of the industries in which it is employed, has been given by Merewether and Price.

The history of the development of our knowledge of pulmonary asbestosis as a clinical entity is, however, noteworthy.

Whilst the use of asbestos in industry can be traced to antiquity, it is only in more recent years that the harmful effects of prolonged exposure to asbestos dust has been realised. It is only during the last six or seven years that the subject of pulmonary asbestosis can be said to have received any real attention as a serious industrial lung disease. The first case, the now

famous "Montague Murray" case, was not suspected as an industrial pulmonary disease at the time. It occurred in a young man who came under the care of Dr. Montague Murray, at Charing Cross Hospital in 1899. He had worked chiefly in the card-room of an asbestos factory for some ten years, and was apparently exposed to asbestos dust for the greater part of that period. He died in 1900, and post-mortem findings showed an extensive degree of pulmonary fibrosis but no tuberculosis. Photomicrographs of the lung showed spicules of asbestos.

This case only came to light some six years later, *i.e.* in 1906, when Dr. Murray gave evidence before the Departmental Committee on Compensation for Industrial Diseases.

The second case was reported by Fahr, in 1914, to the Hamburg Medical Society, but it was not actually until 1927 that any real attention was focussed on this disease, when W. E. Cooke and Stuart MacDonald discovered the "curious golden-yellow bodies." In 1929 Stewart and Haddow found these highly characteristic "curious bodies" in the juice obtained by lung puncture, and in the sputum. They suggested that these bodies should be called "asbestosis bodies." In the same year, Gloyne demonstrated that the asbestosis body has a central core of asbestos fibre. A large series of cases of pulmonary asbestosis was reported by Burton Wood in 1929, with special reference to the radiological features. I have reported cases of pulmonary asbestosis out of a smaller series during 1930 and 1931.

Since 1927 tremendous strides have been made on the subject of pulmonary asbestosis, and a vast amount of literature has sprung up.

The chemistry and industrial uses of asbestos have been fully described by Merewether and Price. The clinical features have already been dealt with this evening.

Radiological Features

Radioscopic examination of the chest followed by a technically satisfactory radiograph affords, in my view, the most reliable single piece of evidence in establishing a diagnosis of the stage and extent of the disease in cases of pulmonary asbestosis. Radiography discloses the presence of a fine, diffuse fibrosis, which has been claimed to be characteristic of asbestosis. It must, however, be emphasised that other dusts capable of producing pulmonary fibrosis may possibly lead to a condition indistinguishable from that of asbestosis. Certainly Pancoast and Pendergrass doubt whether anyone viewing a number of radiograms, indicative of pneumoconiosis, sustained in several industries, could select those of asbestosis subjects. We may take it

PLATE XXVI

[Philip Ellman]



FIG. 5.

CASE 1. (6) Fine punctate mottling distributed in all zones, the apices remaining free (stage iv). A suspicious annular shadow in the left infraclavicular region is suspicious of a cavity.

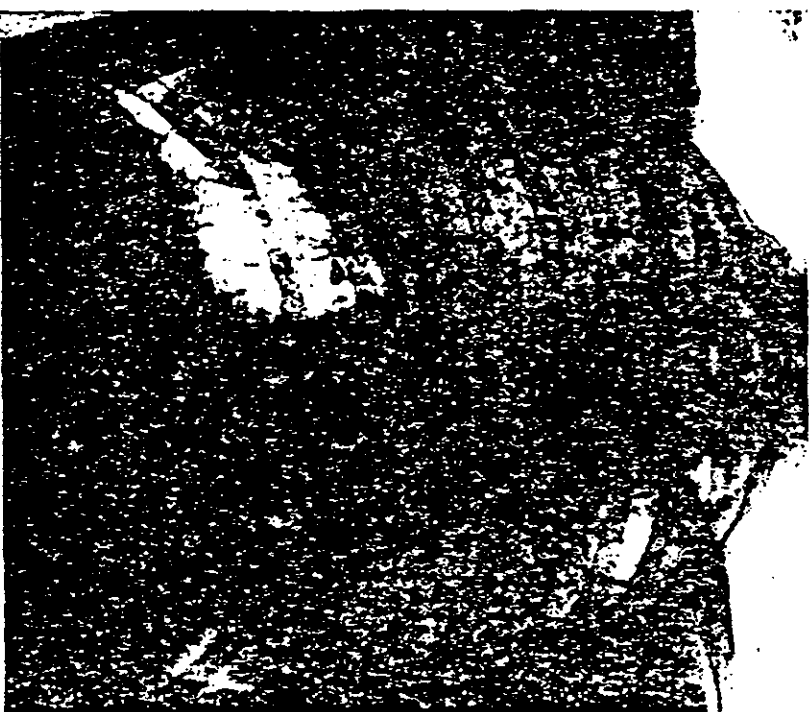


FIG. 6.

CASE 1. (6) The same patient some fourteen months later. There is very definite extension of the disease. Whilst the appearances are typical of asbestosis, the mottling in the upper zones of both lungs suggests the possibility of a superadded tuberculous infection. The annular shadow in the left infraclavicular region is now very well defined.



FIG. 8.

CASE 2. (1) Note the characteristic mottling with bilateral basal fibrosis. The right interlobar can be seen to be thickened and below its normal position, having been drawn down by the basal fibrosis. There is no evidence of tuberculous infiltration of the parenchyma of either lung.



FIG. 9.

CASE 2. (2) Same case nearly two years later. Note now the definite tuberculous infiltration involving the left upper and middle zones with excavation. This has complicated the original pure asbestosis. Extension of the fibrosis of the right lower zone can be seen.

that all such pneumoconioses, including asbestosis, are due to a *silicate* dust, that is, are examples of a "silicosis."

However, radiograms of pulmonary asbestosis are certainly very characteristic.

Dr. Stanley Melville has co-operated closely in the study of my cases from the radiological aspect.

Four stages of the disease have been detailed by me elsewhere.*

The diffuse mottling has been aptly described by Burton Wood as presenting a "ground glass" appearance.

Melville says the impression is that of a blurred smearing of the involved zones, as though a piece of india-rubber had been rubbed across the film.

The radiological findings confirm the clinical findings that the disease is characterised by a fine diffuse fibrosis, commencing at, and involving principally, the bases of the lungs. Of course, in the advanced cases all zones become involved.

Pathological Features

A few points about the pathological features may now be noted, with special reference to the characteristic asbestosis bodies.

Macroscopic Appearances

The visceral pleura is usually considerably thickened, while the pleural sacs may be partially or even completely obliterated by dense fibrous adhesions. The bases of the lungs are usually firmly adherent to the diaphragm.

Bronchiectasis is fairly common; bronchiectatic cavities sometimes occur. Specimens often show a honeycomb appearance of the lung. Usually the lungs are firm on palpation, tough, and airless; the areas of dense fibrosis show greyish-black mottling owing to carbon immobilisation. The upper lobes, by contrast, may be spongy, crepitant, and emphysematous; this compensatory emphysema may often be detected clinically and radiologically.

Microscopic Appearances

The interstitial tissue of the lungs is everywhere increased, so much so that in some cases the alveolar structure can no longer be detected; it is completely obliterated; but in other areas there is evidence of compensatory emphysema. Indeed, although fibrosis may appear to the naked eye to have escaped the apices, much fibrosis may be seen in them on lung section. In and around the densely fibrous areas, asbestosis bodies are found in profusion; they tend to occur in radially arranged clumps.

*These four stages are detailed in my paper in the *Journal of Industrial Hygiene*, July 1933, from which much relevant matter in this discussion, including illustrations, has been taken.

Carbon pigment is present in varying quantities. Advanced areas of fibrosis can be found, associated with dilated bronchioles and bronchiectatic change. In addition to the dense fibrosis, inflammatory catarrhal changes may be observed, with desquamation of alveolar and bronchiolar epithelium, and thickening of the alveolar and bronchiolar walls. Here asbestosis bodies tend to be fewer in number.

Foreign body giant cells, distinct from tuberculosis giant cells, may be present in the connective tissue: Gloyne, who has recorded these cells, and termed them "asbestosis giant cells," suggests that they present an attempt to destroy the asbestos fibres by phagocytosis. These cells are larger than tuberculosis giant cells; their cytoplasm has a stippled and pigmented appearance, in contrast with the structureless caseating appearance of tuberculosis giant cells.

Asbestosis Bodies

These highly characteristic golden-yellow "curious" bodies, termed "asbestosis bodies" in 1929 by Stewart and Haddow, are found in the sputum and fibrosed lungs of asbestos workers. They have been present in all autopsies upon cases of pulmonary asbestosis. They vary in size and shape; but, characteristically, they have bulbous enlargements at the extremities, with a regularly or irregularly segmented body, resembling dumb-bells; when fractured they are club-shaped. The appearance of fully formed bodies has been aptly compared by Gloyne to beads on a necklace; the beads vary in size, and represent the irregularly segmented body. These bodies have been found by various observers to vary in length from 20 to over 200 microns. An asbestos fibre forms the central core of each body, and can frequently be detected. The golden-yellow material covering each fibre contains an iron substance, which gives a prussian blue reaction; probably it is a silicate, which protects the fibre from further change. When stained with ammonium sulphide, the central core of an asbestos fibre stains lightly against a well stained body.

Asbestosis bodies are probably asbestos fibres which have become coated with colloidal golden-yellow material—probably iron silicate—derived from body tissues, oxyhaemoglobin being the chief source. In addition to being found in the sputum and lung juice, they have been found in the faeces (Gloyne) and even in the spleen (H. L. Stewart Bucher and Coleman), which raises the question as to how they gained access to this organ. Presumably they travelled either by embolic spread in the blood stream, or by phagocytic cells. The bodies have also been found in the upper abdominal lymph glands (M. J. Stewart, personal communication).

The presence of asbestosis bodies in the faeces is of practical value where no sputum is available, since, as in cases of phthisis, the sputum may have been swallowed rather than coughed up.

The bodies are best seen with an oil immersion lens, and show up clearly, without staining, as golden-yellow structures. They can, however, be stained by hæmotoxylin, by potassium ferrocyanide, and hydrochloric acid, giving the prussian blue reaction, and by ammonium sulphide.

Asbestosis Bodies in Clumps

The bodies may be found in the lung tissue either singly or in clumps, and may occur similarly in the sputum, although they more usually are

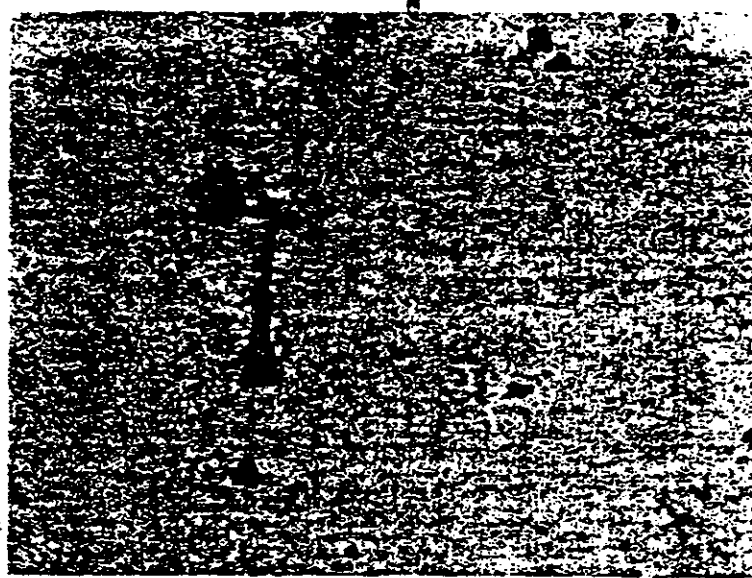


FIG. 1.—Pseudo asbestosis bodies.
(By courtesy of Prof. S. Lyle Cummins)

found in the sputum singly or in groups of two or three. The bodies tend to be scanty if there is little bronchial secretion, and, in my experience, given a pulmonary fibrosis, the profusion of bodies in the sputum appears to show a tendency to vary directly with the extent of the disease. When the bodies are more abundant in the sputum, they may occur in radially arranged clumps; such an occurrence indicates disintegration of lung tissue by either simple suppurative broncho-pneumonia, or secondary tuberculous infection. This occurrence of bodies in clumps in the sputum is comparable to the presence of elastic tissue in phthisis (M. J. Stewart).

What have been called "pseudo-asbestos bodies" have recently been recorded as found in the lungs of a coal-miner working in the mines of Alabama, who had never worked in asbestos, and of a Lancashire coal-miner. Apparently other dusts may simulate asbestos fibres, and lead to similar reactions with the lung tissues.

A recent observation by Kettle is interesting and noteworthy in this connection:

"Crystalline silica heavily coated with iron (silica, 59.9 per cent.; ferric oxide, 40.1 per cent.) and injected into the tissues appeared to be as inert as pure ferric oxide itself. It would appear that the silica is prevented from going into solution by its very adequate coating of insoluble ferric oxide.

"This observation has an important bearing on pulmonary asbestosis.

"The familiar asbestosis bodies appear to consist of asbestos spicules, which have become coated with iron. They are insoluble and remain for many years apparently quiescent in the pulmonary tissue and alveoli. They are not formed, however, immediately the asbestos enters the body, but only after an interval of some weeks, and presumably damage to the lung occurs as the result of solution from the surface of the spicule before it is rendered inert by the coating of iron."

Kettle records the fact that asbestosis bodies injected into the subcutaneous tissues of mice are as inert and harmless as the iron coated crystals of silica. This protective action of the colloid substance is a point of considerable interest.

Asbestosis Bodies in Sputum

The presence of asbestosis bodies in sputum indicates merely exposure to asbestos dust; it cannot alone justify a diagnosis of pulmonary asbestosis. A diagnosis of a diseased condition of the lungs depends upon the cumulative findings of: (a) exposure to asbestos dust; (b) clinical evidence of pulmonary fibrosis; (c) radiological evidence of fine, diffuse, pulmonary fibrosis; and (d) asbestosis bodies in sputum, faeces, or lung tissue.

It is astonishing how asbestosis bodies persist in the sputum for years, even despite short periods of exposure. In one case a patient was exposed to asbestos dust for one year, yet the bodies were present in the sputum five years later. Apparently, once the bodies are formed, unless they are carried away or excreted in the sputum, they remain in the lungs more or less permanently without being dissolved.

Pulmonary Asbestosis in a Dog

The following account of asbestosis found in a dog is noteworthy, since there is a significant contrast to the findings in the human subject.

A rough-haired terrier dog, ten years old, was kept for ratting purposes in an asbestos factory, where he spent the greater part of his life, and was exposed to asbestos dust during most of the time. He developed a cough, and increasing dyspnoea for two years, during the last six months of which he became thin and emaciated. The dyspnoea became so distressing that the dog was lethalled in its own interests. I was fortunate enough to secure the thoracic organs,



FIG. 2.
Macroscopic appearance of dog's lung in this case.

trachea, heart, and lungs, which were kindly examined for me by Dr. N. Schuster. The following is a summary of her findings already reported.

The macroscopic appearances showed that, whilst there was no sign of acute pleurisy, there was a general opacity of the visceral pleura, with patches of localised pleural thickening, and bands of adhesions between the lobes. The

lungs had lost their usual spongy consistency; on section there was evidence of diffuse fibrosis under the pleura. The bronchioles were dilated; carbon pigment was to be seen scattered throughout the lung. Evidence of any acute inflammatory process was completely absent.

Microscopic examination revealed much the same conditions as in the human subject. Chronic interstitial fibrosis of a fine diffuse nature with dilatation of the bronchioles was present. Fibres of asbestos were seen in large numbers, both in the alveoli and interstitial tissue of all parts of the lung. The fibres corresponded exactly, under the microscope, with the appearance of pure asbestos after it had been crushed. *But no asbestosis bodies were found.*

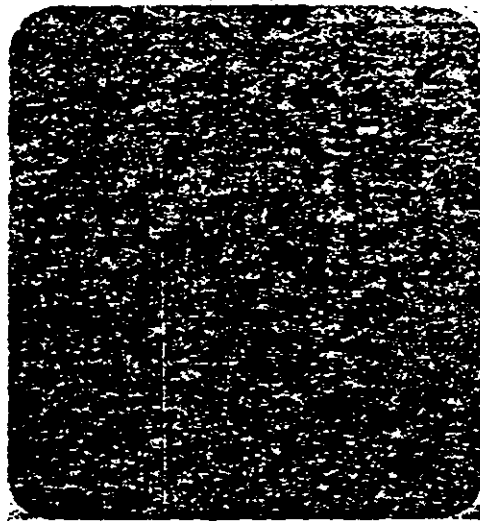


FIG. 3.—Foreign body giant cell. Note coarse stippled segmented appearance. The presence of asbestos fibres, represented by fine lines, can just be seen.

(Microphotograph by Dr. W. Susman),

this important negative fact was confirmed by Professor M. J. Stewart and Dr. Gloyne. Professor Stewart informs me that he has recently made an examination of another dog, which stayed from 7 p.m. to 7 a.m. in an asbestos factory for over nine years, and at death was 13½ years old. Here also he was unable to find any asbestosis bodies, and only a few asbestos fibres were present. Apparently, this second dog did not have the same opportunity as the first of inhaling the asbestos dust, since it was only at the factory with the night watchman.

This absence of asbestosis bodies was also observed by Stewart in three

rats from an asbestos factory, although fibres were present in the lungs. Gloyne, however, after careful search, has found one small body, together with numerous fibres, in the lungs of a grey rat caught on an asbestos factory premises.

Although the lung changes in the case of this dog were closely allied to those of asbestosis in the human lung, two noteworthy features were absent—*asbestosis bodies*, and evidence of acute inflammation. A possible relationship between the formation of *asbestosis bodies* and an acute inflammatory exudate might suggest itself, had not *asbestosis bodies* been found experimentally in guinea pigs in the absence of any appreciable inflammation; apparently they are produced during normal metabolism.

The Tuberculosis risk in Asbestosis

Tuberculosis is generally recognised as the most serious risk to which silicotic patients are exposed; but the question of tuberculosis complicating asbestosis has remained rather an open one. Until recently, the opinion was held that for tuberculosis to complicate asbestosis was comparatively rare, and this was regarded as a distinctive feature in contra-distinction to silicosis.

Recently, however, Professor Kettle, working experimentally with animals, finds that silica and asbestos dusts belong to a group of active substances which produce lesion when introduced into the subcutaneous tissues; they both produce serious pulmonary fibrosis, and both assist the growth of tubercle bacilli. With greater experience of this disease, clinical evidence would seem to support this experimental work, as there is little doubt that an increasing number of cases of superadded tuberculosis are being seen in subjects who originally had an uncomplicated pulmonary asbestosis. Such cases may not be met with in an examination of workers at work with no symptoms; they tend rather to be seen among those who have left work and are under treatment. Hence Merewether, in 1930, found only four active cases of tuberculosis in an examination of three hundred and seventy-four asbestos workers actually at work, and formed the impression that there was no outstanding susceptibility to tuberculosis among these workers. On the other hand, Wood and Gloyne, in 1931, were able to trace twelve cases of tuberculosis, ten of which were active, in a series of fifty-seven cases of asbestosis; and among thirty-five deaths from asbestosis referred to in the Report of the Chief Inspector of Factories for 1931, tuberculosis was a complicating or terminal factor in eleven cases. Among twenty of my own definite cases of asbestosis, the majority of whom were not at work, seven had tuberculosis, of whom three are now alive.

A typical lesion of mild activity, with an early stage of pulmonary

asbestosis, was present in one of my cases who had been exposed to asbestos dust for ten years; but the work, which consisted in coating lead pipes, did not entail exposure to high dust concentrations. Tubercle bacilli and asbestosis bodies have been found in sputum. This case has been watched for three years, during which the tuberculosis has remained stationary. In this connection Wood and Gloyne have pointed out that obsolescent tuberculosis may remain quiescent in spite of exposure to asbestos dust.

Another case—one of pure pulmonary asbestosis—under my care, has, during the last few months, developed pulmonary tuberculosis, which I can say quite definitely, from clinical, radiological, and sputum controls, has been implanted upon the asbestosis with decided aggravation of all symptoms. This case has since come to autopsy. Among my series, serial skiagrams raise the suspicion of superadded tuberculous infiltration in two cases. I am further informed by Professor Stewart, that at autopsy, when no microscopic evidence of lung tuberculosis has been observed, tuberculosis may be found histologically.

My own limited experience tends to show that tuberculosis, as a complication of asbestosis, is by no means uncommon; the risk may or may not be equal to that in silicosis; one cannot be sure that it may not even prove to be perhaps even greater; but that there is a risk, the evidence quoted and my own observations, appear to establish beyond doubt. Other things apart, recognition of the existence of this risk is an important factor when considering what preventive measures should be taken to control this occupational disease. The existence of this risk establishes that no person with tuberculosis in any form should be allowed to enter the industry, nor should anyone in whom tuberculosis at a later stage is detected be permitted to continue in the industry. The risk here is to the other workers, as well as to themselves.

Finally, let me conclude with a description of two typical cases.

Case 1. A female, aged 35. This woman worked in an asbestos factory from 1916 to 1921, had been quite healthy previous to working there, and had never had chest trouble. In 1919, cough and dyspnoea developed and became worse, but she continued at work until 1921, when she was compelled to give it up. In 1921 she was notified as a case of pulmonary tuberculosis; her weight was then 119 lbs.; it is now 99½. She has twice been in a sanatorium; although the sputum has been examined in numerous occasions, tubercle bacilli have not been found.

Clinical examination. I first saw her in May 1930, when there was present cough, dyspnoea, and occasional sweats. She was thin and emaciated, and her face was cyanosed. The chest expansion was poor, and the apices were retracted. Expiratory signs were prolonged in the upper zones, but diminished in the

lower zones; dry, crackling, scattered crepitations were heard throughout both lungs, but especially over the left. The heart was drawn over to the left side. The sputum, carefully examined in June 1930, was found to contain asbestosis bodies, which have been found repeatedly since, both singly and, on one occasion, in clumps.

X-ray examination in November 1930 gave evidence of basal pleurisy, with heart and mediastinum displaced to the left. Both lungs showed punctate mottling generally distributed. The appearances were characteristic of those found in uncomplicated pulmonary asbestosis. In January 1932 there was evidence of bilateral pleurisy, with the heart, mediastinum, and trachea



FIG. 4.

CASE 1. Asbestos bodies in clumps in the sputum.

displaced to the left. The appearances were typical of asbestosis, but the mottling in the upper zones suggested superadded infection, with apparently a small cavity in the upper zone of the left lung, although the annular shadow suggestive of this can just be detected in the first skiagram.

Diagnosis. This patient exhibits an advanced degree of asbestosis. Her serial skiagrams have given important information regarding the progress of the case, with a suspicion of superadded tuberculous infection, though no bacilli have yet been found.*

*Quite recently tubercle bacilli have been found in the sputum, definitely proving the superadded tuberculous infiltration.

Case 2. Female, aged 34. She was engaged in an asbestos factory from 1911 to 1917. She was first seen in September 1937 for a slight cough, which had persisted for some years. The cough had troubled her seriously during the previous nine months, when she had recommenced work at the factory, after being away for some eighteen years. There had been dyspnoea with the cough, which had become progressively worse, and was marked on exertion. During the next four months there was not much loss of weight; the cough persisted, with some muco-purulent expectoration, half an ounce in twenty-four hours; it was never blood-stained. There were severe night sweats, and the appetite was poor. Lassitude was present. Asbestos corns were present, especially on the hand and elbow.



FIG. 7.

CASE 2. Asbestos bodies occurring in clumps in the sputum.

Clinical examination. Physical signs in the chest were mainly at the base, and typical of those of a bilateral pulmonary fibrosis, with pleural thickening at the right base.

X-ray examination disclosed some scoliosis, with dorsal convexity to the left; the heart tended to lie in the hollow of the curve. Movement of the right side of the diaphragm was restricted. On the right side there was evidence of pleural thickening at the base, with lowering and thickening of the interlobar septum. The basal linear striation was exaggerated, and there was some

ill-defined mottling of all zones. On the left side some fibrosis was detected at the base.

The tenacious muco-purulent sputum was found to contain numerous and mixed organisms, mainly bacillus friedlander, and pneumococcus. No tubercle bacilli were found. Asbestos fibres and asbestosis bodies were present.

All symptoms had become much aggravated by July 1932, and the weight had dropped from 113 lb. to 96 lb. The pulse was rapid, and there was evening pyrexia. Sputum increased in amount, and tubercle bacilli were found in October 1932, after frequent examinations. Asbestosis bodies of great variety were present, with *definite bodies in clumps*; the condition suggested a rapid, complicating tuberculous process.

X-ray examination now showed restricted movement of the right side of the diaphragm, and increased fibrosis at the right base. On the left side there was infiltration of the upper and middle zones, with excavation in the upper zone, with pleural involvement, and basal fibrosis.

Diagnosis. The case was originally one of pure pulmonary asbestosis, complicated later by tuberculosis. Here the tuberculosis was implanted upon an asbestosis. The clumping of the asbestosis bodies indicated disintegration of lung tissue.

Prognosis

An interesting feature of this disease is the length of time which may elapse between exposure to the dust and a fatal termination, and the fact that this period is only one-half of that in silicosis. Apparently the dust gains access

COMPARISON BETWEEN DATA DERIVED FROM FATAL CASES OF SILICOSIS AND ASBESTOSIS. (CHIEF INSPECTOR OF FACTORIES AND WORKSHOPS, 1931)

Disease	Number of Deaths	Average age at death	Duration of Employment in Years		
			Shortest	Longest	Average
Silicosis	89	54.1	2.8	57.0	42.5
Silicosis with tuberculosis ..	116	51.7	2.0	57.0	32.5
Asbestosis	24	40.6	4.4	28.0	15.1
Asbestosis with tuberculosis ..	11	45.7	2.6	18.0	13.5

to the lungs and produces pulmonary fibrosis as the result either of actual mechanical trauma, or of a toxic effect comparable to that exerted by silica in cases of silicosis; asbestosis bodies appear to lie inert in the tissues. The dust particles, once they have gained access, continue to injure the lungs, and the disease is a progressive one, which, if sufficient dust is present, ends fatally,

the end being determined by some intercurrent complication, such as acute broncho-pneumonia or phthisis.

Symptoms of pulmonary asbestosis as a general rule first appear some five to fifteen years after the first exposure to dust, the time depending largely upon the nature and concentration of the dust; in one of my cases, symptoms developed within one year after exposure commenced; but in other cases, exposed to a minimum of dust, no symptoms have developed within ten years of the first exposure.

In an established case of asbestosis with symptoms, the dyspnoea is usually out of proportion to the clinical findings as regards fibrosis, while in a case of silicosis, one has the impression that dyspnoea, even though the clinical findings of fibrosis are advanced, becomes manifest only on exertion.

Again, a case of simple silicosis often looks well, declares that he is well, and is even offended when any reflection is cast upon the soundness of his lungs. In contrast to this, a case of asbestosis is cyanotic, emaciated, anxious, and often obviously going downhill. Such, then, is the clinical picture of the state of *sufferers* from asbestosis. Only by an extension of meaning could the word "sufferer" be applied to anyone with simple silicosis.

Summary

Clinical, radiological, and pathological findings of the occupational disease, asbestosis, have been presented. This disease must be grouped with silicosis as a very serious pneumoconiosis. The average length of employment in fatal cases is only one-half that of silicosis.

A diagnosis, which is most likely to be made during the winter months, when acute respiratory complications occur, must depend upon a combination of factors: (a) opportunities for inhaling asbestos dust; (b) the occurrence of asbestosis bodies in the sputum; (c) clinical and radiological findings of fine diffuse, pulmonary fibrosis *commencing at the bases*; the characteristic radiological findings are probably the most important reliable single piece of evidence in early diagnosis.

The clinical features of the disease indicate that the onset of symptoms usually occur after some five to ten years of exposure to the dust; the degree of dyspnoea and emaciation, the complexion, the absence of hæmoptysis, and the very scanty expectoration, are all characteristic features.

Inhalation of asbestos dust must be expected sooner or later to produce pulmonary fibrosis, depending upon (a) length of exposure, and (b) nature and concentration of the dust.

The histological features of asbestosis are essentially (a) diffuse chronic

interstitial fibrosis of the lungs, with areas of acute catarrhal changes, and (b) the presence of characteristic asbestosis bodies, appearing either singly or in clumps.

The presence of isolated asbestosis bodies in the sputum is indicative merely of previous exposure to asbestos dust, and does not necessarily have any clinical significance; if the bodies occur radially arranged in clumps, they suggest disintegration of pulmonary tissues.

Pulmonary asbestosis, once established, is a progressive disease with a bad prognosis; its treatment can only be symptomatic.

The tuberculosis risk in asbestosis must be reckoned with, even though time has yet to indicate whether it is less, equal to, or perhaps even greater than that in silicosis.

REFERENCES

- Annual Report, Chief Inspect. of Factories 1931, London, H.M. Stationery Office, 1931, 112.
 COOKE, W. E. and MACDONALD, S., *B.M.J.*, 1927, 2, 1024.
 ELLMAN PHILIP, *Proc. Roy. Soc. Med.*, 1930-31, 24, 526, 541, 699, "Chest Disease in General Practice, 1932, p. 116, "*Journ. Indust. Hygiene*, 1933, xv, 165.
 GLOYNE, S. R., *Tubercle*, 1930, 11, 151; *ibid.*, 1930, 12, 158; *Lancet*, 1932, 1, 1351.
 JONES, W. R., *Journal of Hygiene*, 1933, xxxiii, 307.
 KETTLE, E. H., *Journ. Path. and Bact.*, 1932, 35, 395.
 MEREWETHER, E. R. A., and PRICE, H.M. Stationery Office, 1930.
 MEREWETHER, E. R. A., *Journ. Indust. Hyg.*, 1930, xii, 253.
 PANCOAST, H. K., and PENDERGRASS, E. P., *Am. Journ. Röntgen.*, 1931, 26, 556.
 SCHUSTER, N. H., *Journ. Path. and Bact.*, 1931, 34, 751.
 STEWART, M. J., and HADDOW, A. C., *Journ. Path. and Bact.*, 1929, 32, 172.
 STEWART, M. J., TATTERSALL, N., and HADDOW, A. C., *Journ. Path. and Bact.*, 1932, 35, 737.
 STEWART, H. L., BUCHER, C. J., and COLEMAN, E. H., *Arch. Path.*, 1931, 12, 916.
 TYLECOTE, F. E., and DUNN, W. J. S., *Lancet*, 1931, 2, 632.
 WOOD, W. B., *Tubercle*, 1929, x, 353.
 WOOD, W. B., and GLOYNE, S. R., *Lancet*, 1931, 2, 954.

INVITATION TO VISIT MESSRS. PHILIPS' FACTORY AT EINDHOVEN, HOLLAND.

A very cordial invitation to British members of the Fourth International Congress of Radiology to visit Eindhoven has been extended by Dr. Bouwers, of Messrs. N. V. Philips, Gloeilampenfabrieken. It is suggested that a limited number travel as a party from London to Eindhoven, thereafter carrying out the following programme. After visiting the laboratories and factory, the party would leave Eindhoven on the afternoon of Friday, July 20, and proceed by bus to Cologne, where the night would be spent. On Saturday, Frankfurt would be reached by bus along the Rhine. After visiting the Hoffelder Clinic on Sunday, the buses would go to Freiburg or Baden-Baden in the Black Forest; next day (Monday) the journey to Zurich would be completed. One or two buses (each taking 35 people) would be provided, but members would pay their own hotel and meal expenses.

Since only a limited number can join this party, it is essential that application to join be sent to the General Secretary, British Institute of Radiology, by May 31.