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PULMONARY ASBESTOSIS: ITS CLINICAL, RADIOLOGICAL, AND PATHOLOGICAL FEATURES, AND ASSOCIATED RISK OF TUBERCULOUS INFECTION*

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THE close study which has been devoted to silicosis, resulting 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, that arising when asbestos fibres are manipulated. A full description of the mineral asbestos, and of the industries in which it is employed, has been given by Merewether and Price (1). No further reference to this side of the question therefore is needed. This paper is restricted to presenting the clinical, radiological, and pathological findings in asbestosis, based upon an experience of cases seen during the last few years, some of which I have already reported (2, 3). While these findings are in close agreement with those of other observers they help to establish the existence of asbestosis as a disease with definite characteristics distinguishing it from silicosis.

*Received for publication March 17, 1933.

CLINICAL FEATURES

The slow development of a characteristic type of fibrosis distinguishes pulmonary asbestosis; it produces insidious lung changes; but the patient may be comparatively free from symptoms for several years, usually from 5 to 15; in some cases, before symptoms arise, years elapse after the worker has left the industry and his exposure to asbestos dust. Nevertheless, in my experience, one or two exceptions have occurred wherein symptoms have followed after exposure of 1 to 3 years or less. The onset of symptoms and

their severity depends to a large extent on the nature of the work, and on the amount of dust inhaled. Symptoms may even be absent despite the presence of clinical and radiological signs (4).

Dyspnoea.—Dyspnoea is one of the earliest symptoms, and at first occurs only on exertion. It is, however, progressive, and, ultimately, dyspnoea and even orthopnoea at rest may become established, which may be out of all proportion to the physical signs present.

Cough.—Cough sooner or later accompanies the dyspnoea; it is a harsh, non-productive cough, often associated with fleeting chest pains.

Expectoration.—Expectoration is often absent; in my cases it has varied a good deal. When present it is usually scanty in amount, and of a thick, tenacious, glairy, mucoid nature. Burton Wood has described it as resembling "semi-coagulated albumen." In a few cases, where there has been little bronchial secretion, almost clear fluid has been coughed up. During the winter months, when these patients are peculiarly liable to acute respiratory affections, the sputum may be more abundant and often more purulent, although pus cells have always been present. Haemoptysis has been strikingly absent, and even blood-stained expectoration has been the exception.

Anorexia.—Anorexia may occur early; it is usual in more advanced cases, but is by no means consistently present.

Cyanosis.—Cyanosis may be present, especially in more advanced cases; according to Merewether it is not necessarily dependent upon the degree of fibrosis. A dusky complexion is common to these patients, and Oliver speaks of a pronounced deadening of the skin, varying from mild bronzing to slight blueness.

Emaciation.—Emaciation has been rather a striking and distinctive feature in my cases, with a loss of weight out of all proportion to the physical signs. Slumber sweats have occurred in several cases.

Finger clubbing.—Clubbing was found in the more advanced cases, in which bronchiectasis was usually present.

Asbestos corns.—Corns form a characteristic skin lesion complained of by most patients. An asbestos fibre penetrates the skin, whereupon a hyperkeratosis forms round it; but no asbestosis bodies are found in these corns. They form on the hands and arms, and even on the skin of the legs, owing to dust fibres penetrating the stockings.

Seasonal incidence.—All symptoms become aggravated in the winter, when bronchial colds are experienced; hence during the winter months most of the cases are recognised. Patients may only discontinue their work because they are forced to do so on account of respiratory embarrassment on the least effort.

Whilst most of the symptoms present are similar to those of silicosis, emaciation and loss of weight out of all proportion to the physical signs, anorexia, the complexion and cyanosis are distinguishing features of asbestosis.

Clinical examination.—Examination of the chest shows limited expansion, even below 1 inch. The movement is especially limited at the bases, where, indeed, the fibrosis commences. The apices are frequently retracted. The

percussion note is impaired, especially at the bases, diminishing as the apex is approached.

Auscultation betrays diminution of breath sounds at the base, although harsh breath sounds with prolonged expiration may be heard over the upper zones, indicative of compensatory emphysema. The adventitious sounds are almost characteristic of the disease, comprising fine, dry, crackling râles; they are usually heard over the bases and in the axillary region. Pleural friction sounds, due to associated basal pleurisy, are not uncommon.

The physical signs in the average case are, in fact, those of basal fibrosis, spreading, in more advanced cases, upwards, although they are usually confined to the lower and middle zones. The apex is usually free; and the upper lobes are not infrequently emphysematous.

Case 1.—Female, aged 22. She worked in an asbestos factory for 4 years as an mattress maker. She had to sew asbestos cloth, and then stuff it with asbestos fibre. The stuffing had to be shaken in order to spread it out, thus producing dust; then the mattress required thumping to distribute the thickness of the packing, with the further production of dust.

She had always been healthy, and had no previous history of chest trouble, nor was there one of tuberculosis in her family.

Present illness.—When first seen, on January 2, 1931, she stated that her illness commenced 6 months previously with a dry, hacking cough which was associated with a loss of weight from 147 lbs. to 130 lbs. Two months ago she became conscious of shortness of breath on exertion, which became progressively worse. She complained of some pain at the base of the left lung, which was worse on inspiration. There were no sweats and no haemoptysis.

Clinical examination.—The patient was thin and pale, with cyanosed lips. There was evidence of corns on her hands. The chest expansion was limited, specially at the bases, and both apices were retracted. There was dullness at both bases, where breath sounds were diminished, and fine, dry, crackling crepitations were heard, chiefly over the left base.

X-ray examination revealed restricted diaphragmatic movements, especially on the left side. Linear striation at the bases was increased, with a faint homogeneous opacity, especially on the right side, where a fine punctate mottling could be observed.

A little glairy, tenacious, mucoid expectoration was obtained, in which were found a few asbestosis bodies and dust fibres, but no tubercle bacilli.

Diagnosis.—The case is one of an early degree of pulmonary asbestosis (Stage 1).

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. Modern radiological technic should be employed, with apparatus for radiography at a distance of 2 metres, and facilities for exposures of 1/10 second or less; in this way the quality of radiograms can be standardized, which is very necessary in following the development of a pneumoconiosis by means of serial radiograms taken over a period of time.

Radiography discloses the presence of a fine diffuse fibrosis, which has been claimed to be characteristic of asbestosis; but other allied dusts capable of producing pulmonary fibrosis may possibly lead to a condition indistinguishable from that of asbestosis. Certainly Pancoast and Pendergrass (5) doubt whether anyone viewing a number of radiograms, indicative of pneumoconioses, sustained in several in-

dustries could select those of asbestosis subjects; however radiograms of pulmonary asbestosis are certainly very characteristic.

Dr. Stanley Melville has coöperated closely in the study of my cases from the radiological aspect. Roughly four stages in order of progression of the disease have been observed. In the first stage, the movement of the diaphragm on one, and sometimes on both, sides, is restricted; the root shadows are unusually heavy; and the basal linear striation on one or both sides is increased. The right chest has not been observed to be specially involved more than the left; indeed the left base on a film is normally somewhat obscured by the heart shadow, and is not so visible. The trachea is centrally placed; the heart is usually normal in shape, size, and position; but the costo-phrenic and even the cardio-phrenic angle may be ill defined. Close examination of the film may detect some loss of translucency, and a fine homogeneous opacity or haze at one or both bases.

In the second stage the homogeneous opacity becomes more obvious, and a fine punctate stippling may be detected at one or both bases, while the basal linear striation is more obvious, with very definite restriction of diaphragmatic movements and sometimes blurring of the diaphragmatic angles. The inter-lobar pleura is usually thickened, and in my experience frequently drawn down to the base by the fibrosis.

In the third stage definite evidence of pleural involvement may be present, with or without mediastinal displacement, depending upon the inequality of fibrosis in the two lungs.

In the fourth and final stage, the fine punctate mottling can be observed to have spread beyond the limits of the lower zone to the middle and even upper zone, but the apices usually remain free. This fine diffuse punctate mottling stands out in definite contrast to the coarse nodular mottling seen in silicosis.

This diffuse mottling, so distinctive of asbestosis, has been described as presenting a "ground glass" appearance by Burton Wood (6), and by Merewether as the veiled appearance of the involved zone. Stanley Melville says the impression is that of a dirty, blurred smearing of the involved zones, as though a piece of india-rubber had been rubbed across the film.

The radiological features thus confirm the clinical findings that the disease is characterised by a fine diffuse fibrosis, commencing at, and involving principally, the bases of the lung.

When tuberculous infiltration complicates asbestosis, it can usually be detected and distinguished from the underlying fibrosis, just as in cases of silicosis.

Case 2.—A female, aged 55.—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 on 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 cya-

nosed. The chest expansion was poor, and the apices were retracted. Expiratory signs were prolonged in the upper zones, but sliminished 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 by Dr. Schuster 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 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. Here serial skiagrams have given important information regarding the progress of the case, with a suspicion of super-added tuberculous infection, though no bacilli have yet been found.

Case 8.—A female, aged 26. This girl worked as a teaser and carder for 6 years, from 1924 to 1930, when she was obliged to leave the factory because of giddiness, cough, and tightness of the chest, which had persisted for 4 years. These symptoms became progressively worse, and were associated with loss of weight.

The patient came under observation at the end of 1931, when she was thin and emaciated, and stated that she had lost 8 pounds in weight during the last 6 months. Her face was somewhat cyanosed.

Clinical Examination.—The chest expansion was poor, with definite dullness on both bases, especially the left, which decreased as the apices were approached, where no appreciable impairment of fulness was detected. Breath sounds were diminished at both bases, where fine, dry, crackling râles were heard, extending well up into the axillæ. The expiratory sounds were prolonged in the upper zone; and a few pleural friction sounds were heard at the left base.

X-ray examination in April, 1932, revealed restricted movement of both sides of the diaphragm, with evidence of pleural thickening at both bases. The right lung showed basal fibrosis, with characteristic punctate mottling of the lower zones. The left lung showed fibrosis and mottling of the lower and middle zones. The mottling was

FIG. 1.—Case 2. Asbestosis bodies in clumps in the sputum.

somewhat coarser than usual, and tended to conglomerate in the left middle zone, where there was a suggestion of tuberculous infiltration.

Expectoration was extremely scanty, but the trace present was tenacious and mucopurulent. Tubercle bacilli were persistently absent, but asbestosis bodies and fibres were present. At a subsequent X-ray examination in January, 1933, the mottling detected definitely indicated an increased spread of the fibrotic condition in both lungs during comparatively short period; the middle zones of both lungs were now completely involved; but there was no definite evidence of infiltration.

Diagnosis.—This case shows definite and

FIG. 2

FIG. 3

FIG. 2.—Case 2. Fine punctate mottling distributed in all zones, the apices remaining free (stage 4). A suspicious annular shadow in the left infraclavicular region is suggestive of a cavity.

FIG. 3.—Case 2. Same patient some 14 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. 4

Fig. 5

Fig. 4.—Case 3. Note the mottling in the right lower zone and left upper and middle zones.
Fig. 5.—Case 3. The same patient some 9 months later. Note the pronounced extension of the disease, especially on the right side, where it has spread into the middle zone and is beginning to invade the upper zone.

progressive spread of fibrosis in both lungs in less than 9 months.

Pathological Features*

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 immobilization. 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.

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, (10) 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 giant cells are larger than tuberculosis giant cells; their cytoplasm has a stippled and pigmented appearance, in contrast with the structureless cascating appearance of tuberculosis giant cells.

Case 4.—A female, aged 31: This woman worked in an asbestos factory, making mattresses, from 1919 to 1922. She had previously been healthy, and there was no tuberculosis in the family. After 2 years' work in the factory she began to feel languid, to lose her appetite, and to develop a cough. As the work did not seem to agree with her, she left the factory and went into domestic service; but she has never felt well. Every winter she has had a troublesome cough, with shortness of breath on exertion, and has been easily fatigued. Early in 1931 she consulted a doctor, who notified her as suffering from pulmonary tuberculosis.

Clinical condition.—When seen she was obviously ill, thin and wasted, very dyspnoeic, while her face was somewhat cyanosed. Sputum contained tubercle bacilli and asbestosis bodies, which were found in clumps on one occasion.

There were physical signs in the lungs of advanced phthisis, with cavitation in the left upper lobe, and, some months later, definite physical signs appeared in the right upper lobe. At the bases physical signs of a basal fibrosis, compatible with asbestosis, could be detected, viz.:—dullness,

* I am indebted to Professor M. J. Stewart for his valuable help in this section.

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She had always been healthy, and had no previous history of chest trouble, nor was there one of tuberculosis in her family.

Present illness.—When first seen, on January 2, 1931, she stated that her illness commenced 6 months previously with a dry, hacking cough which was associated with a loss of weight from 147 lbs. to 130 lbs. Two months ago she became conscious of shortness of breath on exertion, which became progressively worse. She complained of some pain at the base of the left lung, which was worse on inspiration. There were no sweats and no haemoptysis.

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A little glairy, tenacious, mucoid expectoration was obtained, in which were found a few asbestosis bodies and dust fibres, but no tubercle bacilli.

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Radiological Features

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In the second stage the homogeneous opacity becomes more obvious, and a fine punctate stippling may be detected at one or both bases, while the basal linear striation is more obvious, with very definite restriction of diaphragmatic movements and sometimes blurring of the diaphragmatic angles. The inter-lobar pleura is usually thickened, and in my experience frequently drawn down to the base by the fibrosis.

In the third stage definite evidence of pleural involvement may be present, with or without mediastinal displacement, depending upon the inequality of fibrosis in the two lungs.

In the fourth and final stage, the fine punctate mottling can be observed to have spread beyond the limits of the lower zone to the middle and even upper zone, but the apices usually remain free. This fine diffuse punctate mottling stands out in definite contrast to the coarse nodular mottling seen in silicosis.

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Clinical examination.—I first saw her in May, 1930, when there was present cough, dyspnœa, and occasional sweats. She was thin and emaciated, and her face was cya-

nosed. 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 by Dr. Schuster in June, 1930, was found to contain asbestosis bodies, which have been found repeatedly since both singly and, on one occasion, in clumps.

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Case 3.—A female, aged 26. This girl worked as a teaser and carder for 6 years, from 1924 to 1930, when she was obliged to leave the factory because of giddiness, cough, and tightness of the chest, which had persisted for 4 years. These symptoms became progressively worse, and were associated with loss of weight.

The patient came under observation at the end of 1931, when she was thin and emaciated, and stated that she had lost 8 pounds in weight during the last 6 months. Her face was somewhat cyanosed.

Clinical Examination.—The chest expansion was poor, with definite dullness on both bases, especially the left, which decreased as the apices were approached, where no appreciable impairment of percussion was detected. Breath sounds were diminished at both bases, where fine, dry, crackling rales were heard, extending well up into the axillæ. The expiratory sounds were prolonged in the upper zone; and a few pleural friction sounds were heard at the left base.

X-ray examination in April, 1932, revealed restricted movement of both sides of the diaphragm, with evidence of pleural thickening at both bases. The right lung showed basal fibrosis, with characteristic punctate mottling of the lower zones. The left lung showed fibrosis and mottling of the lower and middle zones. The mottling was somewhat coarser than usual, and tended to conglomerate in the left middle zone, where there was a suggestion of tuberculous infiltration.

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Expectoration was extremely scanty, but the trace present was tenacious and mucopurulent. Tubercle bacilli were persistently absent, but asbestosis bodies and fibres were present. At a subsequent X-ray examination in January, 1933, the mottling detected definitely indicated an increased spread of the fibrotic condition in both lungs during comparatively short period; the middle zones of both lungs were now completely involved; but there was no definite evidence of infiltration.

Diagnosis.—This case shows definite and

FIG. 2

FIG. 3

FIG. 2.—Case 2. Fine punctate mottling distributed in all zones, the apices remaining free (stage 4). A suspicious annular shadow in the left infraclavicular region is suggestive of a cavity.

FIG. 3.—Case 2. Same patient some 14 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. 4

Fig. 5

Fig. 4.—Case 3. Note the mottling in the right lower zone and left upper and middle zones.
Fig. 5.—Case 3. The same patient some 9 months later. Note the pronounced extension of the disease, especially on the right side, where it has spread into the middle zone and is beginning to invade the upper zone.

progressive spread of fibrosis in both lungs in less than 9 months.

Pathological Features*

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 immobilization. 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.

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, (10) 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 giant 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.

Case 4.—A female, aged 31. This woman worked in an asbestos factory, making mattresses, from 1919 to 1922. She had previously been healthy, and there was no tuberculosis in the family. After 2 years' work in the factory she began to feel languid, to lose her appetite, and to develop a cough. As the work did not seem to agree with her, she left the factory and went into domestic service; but she has never felt well. Every winter she has had a troublesome cough, with shortness of breath on exertion, and has been easily fatigued. Early in 1931 she consulted a doctor, who notified her as suffering from pulmonary tuberculosis.

Clinical condition.—When seen she was obviously ill, thin and wasted, very dyspnoeic, while her face was somewhat cyanosed. Sputum contained tubercle bacilli and asbestosis bodies, which were found in clumps on one occasion.

There were physical signs in the lungs of advanced phthisis, with cavitation in the left upper lobe, and, some months later, definite physical signs appeared in the right upper lobe. At the bases physical signs of a basal fibrosis, compatible with asbestosis, could be detected, viz.:—dullness,

* I am indebted to Professor M. J. Stewart for his valuable help in this section.

weak breath sounds, and dry, crackling râles, rather different from the râles in the upper zones. The signs were, however, overwhelmingly those of tuberculosis.

An X-ray examination in April, 1931, showed definitely restricted diaphragmatic movements on both sides. There was some dorsal scoliosis, the heart tending to be in the hollow of the concavity. The trachea was drawn to the left. The right lung was clear, whilst the left lung showed definite evidence of tuberculous infiltration of the upper and middle zones, with cavitation in the left upper zone. There was evidence

FIG. 6.—Case 4. Foreign body giant cell. Note asbestos body within the cell.

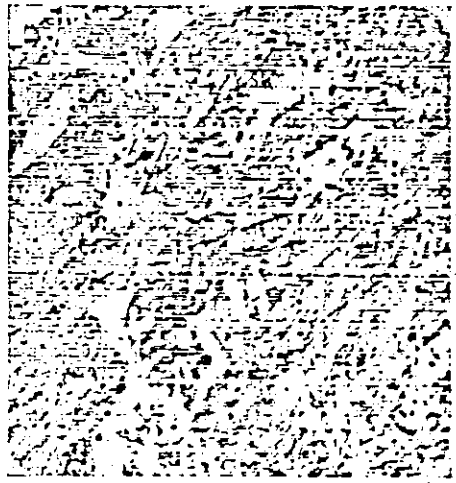

FIG. 7.—Case 4. Section showing diffuse asbestos fibrosis with asbestosis bodies in clumps, radially arranged. (Low power.).

of bilateral basal fibrosis, with pleural involvement of the left base.

The patient had institutional treatment in hospital and at a sanatorium for 6 months. In June, 1932, she discharged herself from the sanatorium and went home; she was then acutely ill, and had advanced physical signs of tuberculosis in both lungs. She died on August 30th, 1932.

Post mortem examination was made by Dr. S. R. Gloyne whose findings were as follows: the left pleural sac was found to be completely obliterated by adhesions, except at the extreme base, where there was a small free sac with greatly thickened pleura containing yellowish tubercles, and covered here and there with patches of plastic lymph. In one place in the middle of a sac was a strand of adhesions, which on separation showed lung immediately beneath the visceral pleural attachment, but there was no pneumothorax opening. The diaphragmatic pleura was thickened and completely adherent to the diaphragm.

The right pleura showed tough adhesions at the extreme apex. Over the middle of the upper lobe was a dense linear scar of old pleurisy with tags of plastic lymph attached to it. Here and there the visceral pleura showed white plaques of thickened pleura, especially over the upper lobe. There was one thickened nodule in the diaphragmatic pleura with adhesions to the diaphragm.

The left lung showed dense areas of asbestosis, almost confluent in the lower lobe. The upper lobe was completely involved in tuberculosis with several ragged cavities, and was practically destroyed by the cavities. The lower lobe showed a few caseous tubercles; in the upper part one of these was breaking down to form a small cavity.

The right lung also showed extensive areas of asbestosis. In the upper lobe there were numerous caseous deposits of tubercle from the size of a pea to that of a walnut. Middle and lower lobes also showed a few

minute discreet nodules of tubercle. No lesions in any other system.

Diagnosis.—Whilst the signs of tuberculosis tended to mask those of asbestosis, there was little doubt that this case was, in all probability, originally a case of pure pulmonary asbestosis complicated by an advanced degree of tuberculosis. Post mortem examination of the lungs showed advanced asbestosis in both lungs; clumped bodies of asbestosis had been observed in the alveoli and on one occasion in the sputum.

Asbestosis Bodies

Highly characteristic golden-yellow bodies 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 (10). 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 sulfide, 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, (11) they have been found in the fæces (12), and even in the spleen, (13) 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 fæces is as pointed out by Gloyne 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 haemotoxylin, by potassium ferrocyanide, and hydrochloric acid, giving prussian blue reaction, and by ammonium sulfide.

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 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, 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 (14). 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" (15) 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 (16) 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, fæces, 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 1 year; yet the bodies were present in the sputum 14 years later. In another case, exposed to the dust for only 10 weeks, the bodies were present in the sputum 5 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.

Case 5.—Female, aged 54. She was engaged in an asbestos factory from 1911 to 1917. She was first seen in September, 1930 for a slight cough, which had persisted for some years. The cough had troubled her seriously during the previous 9 months, when she had recommenced work at the factory, after being away for some eighteen years. There had been dyspnœa with the cough, which had become progressively worse, and was marked on exertion. During the next 4 months there was not much loss of weight; the cough persisted, with some muco-purulent expectoration, half an ounce in 24 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.

Clinical examination.—Physical signs in the chest were mainly at the base, and typi-

cal 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 inter-lobar 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 indicates disintegration of lung tissue.

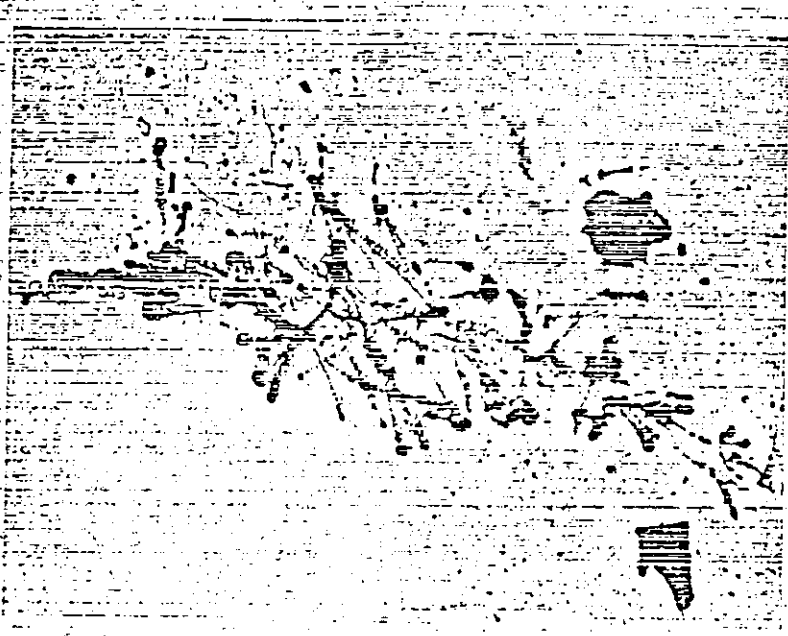

FIG. 8.—Case 5. Asbestosis bodies occurring in clumps in the sputum

Case 6.—A male, aged 45. This man worked as a card-room superintendent in an asbestos factory for 9 years, 1920 to 1929. His work brought him into contact with a high concentration of dust. He remained free from chest trouble until 1924, when he became conscious of mild dyspnoea on exertion, with an irritating, dry cough. The symptoms grew gradually worse, until in 1929 he was so breathless on the slightest exertion that he was compelled in October to cease work; he was then confined to bed

FIG. 9

FIG. 10

FIG. 9.—Case 5. Note the characteristic mottling with bilateral basal fibrosis. The right interlobar septum 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. 10.—Case 5. Same case nearly 2 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.

because of right-sided pleural effusion. He now suffered from progressive anorexia and palpitations; he was breathless even at rest; the face was cyanosed, with an earthy complexion; he was obviously wasted, and his weight had fallen from 203 lb. to 131 lb. Marked clubbing of the fingers was present.

Clinical examination.—The apices were contracted. The right side was practically immobile and the chest was flat. Movement of the left base was very restricted. There was dullness over the whole of the right chest and the left base. Breath sounds were diminished over the whole of the right chest, and some fine râles were heard at the base. The breath sounds over the left chest were coarse, and the expiration was definitely prolonged. Fine, characteristic, crackling râles were heard over the left base and in the axilla.

X-ray examination showed the right diaphragm almost completely obscured, while the movements of the left were very restricted. The trachea was slightly drawn over to the right. There was a dense opacity over the greater part of the right hemithorax, consistent with an encysted effusion. Fine punctate stippling was seen over the area not covered by the dense opacity at the right base. The left lung showed characteristic fine punctate mottling, involving the lower and middle zones.

Numerous asbestosis bodies, both singly and in clumps, were found in the sputum; but repeated examinations for tubercle bacilli were negative. Nevertheless, examination of pleural fluid showed it to be a clear tuberculous exudate.

This patient died on February 11, 1933. An autopsy by Dr. S. R. Gloyne showed evi-

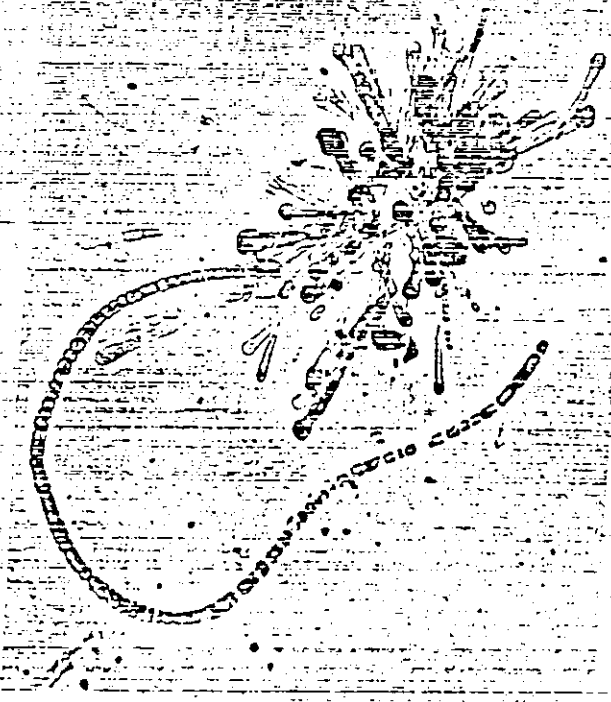

FIG. 11.—Case 6. Showing asbestosis bodies in clumps. The central asbestos fibre is well shown in the long curled body. (I am indebted to Prof. Matthew J. Stewart for the microphotographs of asbestosis bodies in clumps in Cases 5 and 6.)

dence of a right-sided tuberculous empyema with advanced bilateral pulmonary asbestosis. Tubercule bacilli were found in the pus, the fluid of which was clear and straw coloured when a paracentesis was done by me.

Diagnosis.—The case was clearly one of advanced pulmonary asbestosis, complicated by tuberculous pleurisy. Apparently there was some disintegration of lung tissue, since the asbestosis bodies were found in clumps.

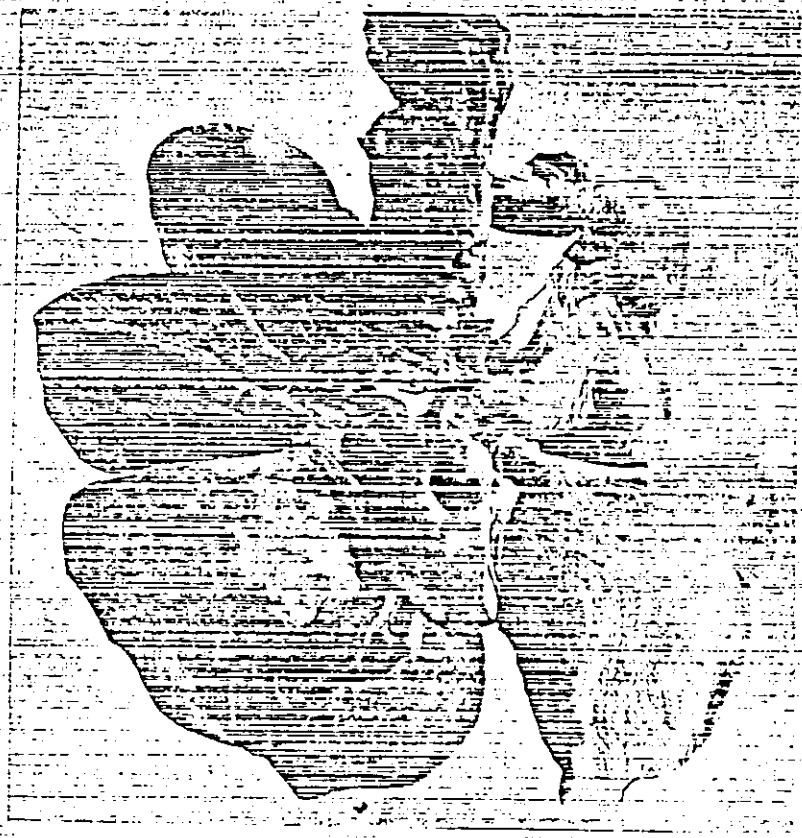

FIG. 12.—Case 7. Macroscopic appearance of dog's lung

Case 7.—A rough-haired terrier dog, 10 years old. This terrier 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 dyspnœa for 2 years, during the last 6 months of which he became thin and emaciated. The dyspnœa became so distressing that the dog was lethalled in its own interest. The thoracic organs, trachea, heart, and lungs (through the courtesy of Dr. Kerr who obtained them for me) were kindly examined by Dr. N. Schuster, whose findings have been reported elsewhere (8), but may be briefly summarized here.

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 localized 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 found 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; this important negative fact was confirmed by Professor M. J. Stewart and Dr. Gloyne. The former, indeed, informs me that he has recently made

FIG. 13.—Case 7. Section of dog's lung showing asbestos fibres. (Microphotograph by Dr. Schuster.)

an examination of another dog, which stayed from 7 p.m. to 7 a.m. in an asbestos factory for over 9 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 asbestos dust, since it was only at the factory with the night watchman.

This absence of asbestosis bodies was also observed by Stewart in 3 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 (9); apparently they are produced during normal metabolism.

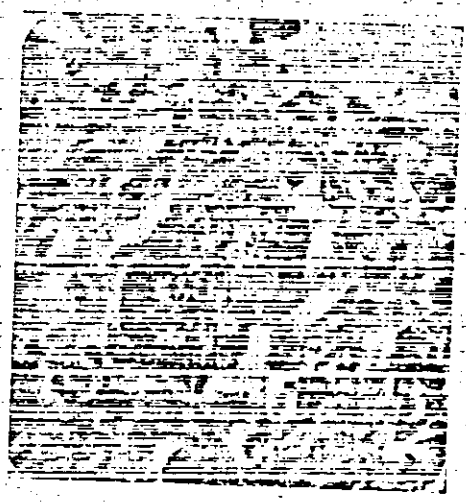

FIG. 14.—Case 7. Foreign body giant cell. Note coarse stippled pigmented appearance. The presence of asbestos fibres, represented by fine lines can just be seen. (Microphotograph by Dr. W. Susman, Pathological Department, Manchester University.)

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

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Recently, however, Professor Kettle, (16) working experimentally with animals, finds that silica and asbestos dusts belong to a group of active substances which produce lesions when introduced into the subcutaneous tissues; they both produce serious pulmonary fibrosis, and both assist the growth of tubercle bacilli. Clinical evidence would seem to support this experimental work, as there is little doubt that an increasing number of cases of tuberculosis are being seen in subjects exposed to asbestos dust. 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 attending clinics. Hence Merewether, (17) in 1930, found only 4 active cases of tuberculosis in an examination of 374 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, (18) in 1931, were able to trace 12 cases of tuberculosis, 10 of which were active, in a series of 57 cases of asbestosis; and among 35 deaths from asbestosis referred to in the Report of the Chief Inspector of Factories for 1931, (19) tuberculosis was a complicating or terminal factor in 11 cases. Among seventeen of my own definite cases of asbestosis, the majority of whom were not at work, six had tuberculosis, of which four were active.

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 10 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 3 years, during which the tuberculosis has remained stationary. In this connection Wood and Gloyne (18) have pointed out that obolescent tuberculosis may remain quiescent in spite of exposure to asbestos dust.

Another case, one of pure pulmonary asbestosis, (case 5) 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. Among my series, serial skiagrams raise the suspicion of superadded tuberculous infiltration in 2 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; 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 con-

tinue in the industry. The risk here is to the other workers, as well as to themselves.

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 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 5 to 15 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 1 year after exposure commenced; but in other cases, exposed to a minimum of dust, no symptoms have developed within 10 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 occurs after some 5 to 10 years of exposure to the dust; the degree of dyspnoea and emaciation, the complexion, the absence of haemoptysis, 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

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(a) length of exposure, and (b) nature and concentration of the dust.

The histological features of asbestosis are essentially (a) 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.

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