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SUBJECT INDEX TO ARTICLES. VOLUME XV

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AL HYGIENE

M.R.C.S., L.R.C.P., Lecturer in
ial Psychology, London School of
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Isaac Pitman & Sons, Ltd., 1933.
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THE JOURNAL OF INDUSTRIAL HYGIENE

VOLUME XV JULY, 1933 NUMBER 4

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

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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 some times 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. Staff 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 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 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 3.—A female, aged 20. 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 pounds in weight during the last 6 months. Her face was somewhat cyanosed.

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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 râles were heard, extending well up into the axillae. 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.

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

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, 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.

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 wit

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

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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.

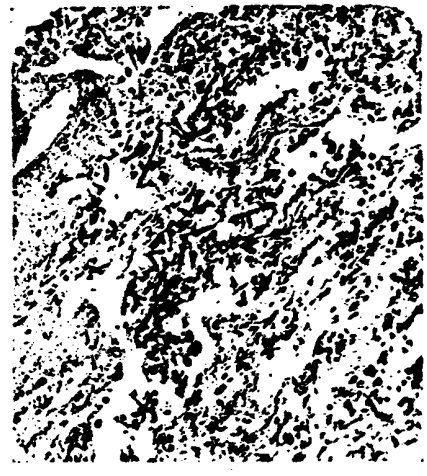

FIG. 7.—Case 4. Section showing diffuse asbestos fibrosis with asbestosis bodies in clumps, radially arranged. (Low power.)

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 feces (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 feces 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 bronchopneumonia, 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.
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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.

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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, feces, 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 34. 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 dyspnoea 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.

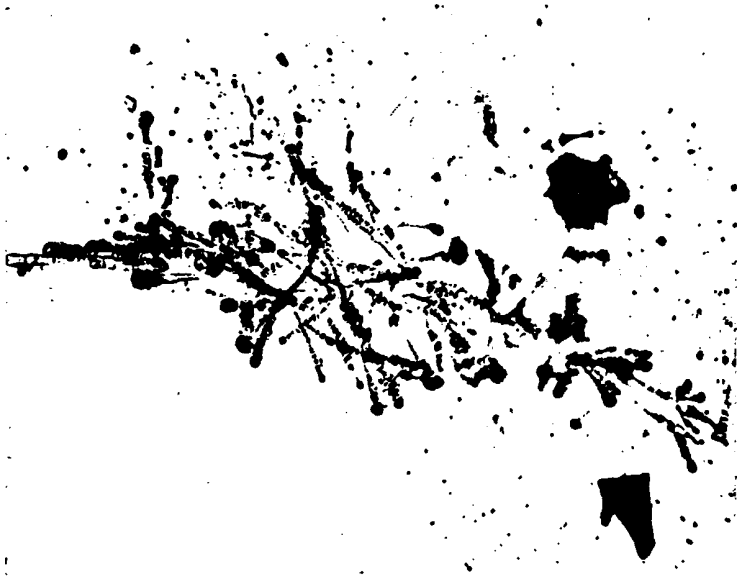

FIG. 8.—Case 5. Asbestosis bodies occurring in clumps in the sputum

Case 6.—A male, aged 43. 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 punctuate 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 evidence 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,

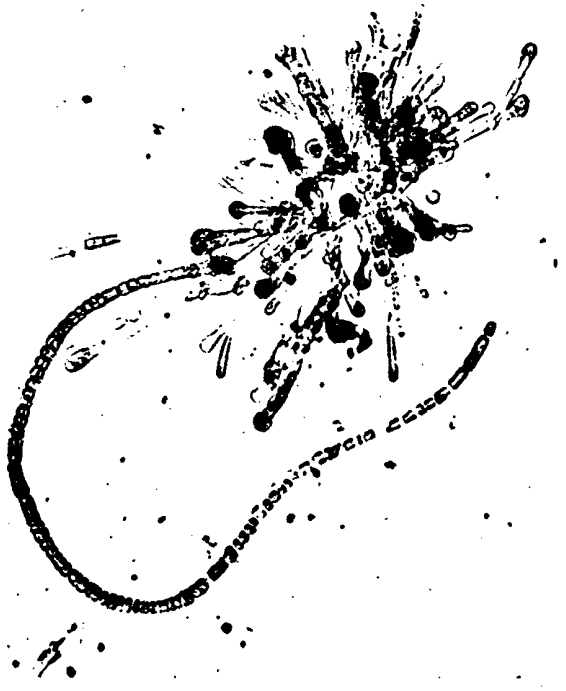

FIG. 11.—Case 6. Showing asbestosis bodies in clumps. The central asbestos fibre is well shown in the long curled body. (I am indebted to Prof. Matthew J. Stewart for the microphotographs of asbestosis bodies in clumps in Cases 5 and 6.)

FIG. 12.—Case 7. Macro

since the asbestosis bodies were found clumps.

Case 7.—*A rough-haired terrier dog, years old.* This terrier was kept for ratti purposes in an asbestos factory, where spent the greater part of his life, and exposed to asbestos dust during most of time. He developed a cough, and incre ing dyspnoea for 2 years, during the las months of which he became thin and em ated. The dyspnoea became so distress

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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 dyspnoea for 2 years, during the last 6 months of which he became thin and emaciated. The dyspnoea 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.

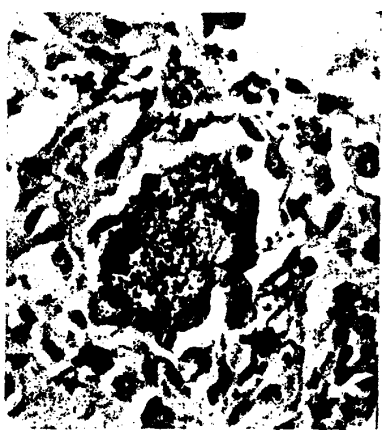

FIG. 14.—Case 7. Foreign body giant cell. Note coarse stippled pigmented appearance. The presence of asbestos fibres, represented by fine lines can just be seen. (Microphotograph by Dr. W. Susman, Pathological Department, Manchester University.)

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

feature in contra-distinction to silicosis.

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

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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

(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

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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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The relative toxicity of fluorine and arsenical insecticides was determined by feeding to white rats various accurately measured amounts of cyrolite, barium, fluosilicate, and lead arsenate, mixed with their food for 16 weeks. The arsenical compound was found to be several times as toxic as the fluorine compounds.—L. T. F.

DERATIZATION OF VESSELS BY MIXED OXIDES OF CARBON. *Sanneman. Bull. Office Internat. d'Hyg. Pub., 1932, vol. 24, pp. 641-644.*

SULFOKOSE IN SHIP FUMIGATION. *Hamel. Bull. Office. Internat. d'Hyg. Pub., 1932, vol. 24, pp. 645-646.*

DERATIZATION OF AUSTRALIAN COASTING VESSELS BY SULFUR. *G. A. Murray. Bull Office Internat. d'Hyg. Pub., 1932, vol. 24, pp. 647-651.*

GASEOUS FUMIGANTS FOR SHIPS. *C. Maselli. Bull Office Internat. d'Hyg. Pub., 1932, vol. 24, pp. 652-656.*

INJURIES DUE TO WORK WITH TOXIC WOODS, WITH PARTICULAR REFERENCE TO A NUMBER OF BRAZILIAN SPECIES. *F. W. Freise. Arch. f. Gewerbepath. u. Gewerbehyg., 1932, vol. 3, pp. 1-14.*

Symptoms and chemical substances associated with certain commercial woods are correlated.

DUST HAZARDS AND THEIR EFFECTS

THE DUST HAZARD IN PORCELAIN WORKS. *A. Hofbauer-Flatzeck. Abstr. as follows from Zentralbl. f. Gewerbehyg., 1932, vol. 19, pp. 105-111 in Bull. Hyg., 1932, vol. 7, p. 697.*

The author here reports the results of 1,339 X-ray examinations of porcelain workers of the Bavarian town of Selb. The results are tabulated according to the classes of work grouped with regard to the dust hazard, and the number of years a worker was employed in that class, and in cases where silicosis was found it is shown as slight, medium, or severe. The mortality from pulmonary tuberculosis among the general population of Selb was in 1930 about three times as high as in Bayern, and the number of new cases more than twice as many as in the neighbouring town of Stettin. The author considers that unless medical men understand more about the nature of the work in porcelain works, there is a tendency to regard cases of pure tuberculosis as silicosis, and overlook cases of silicosis which have occurred after a long latent period.

In grouping the occupations some difficulty was experienced, as the same word is used in different parts of Gemany to describe different kinds of work, in which the dust hazard may not be so great. Of 231 potters examined only 90 were free of silicosis, and there were 33 light cases, 39 medium, and 69 severe, while in 108 porcelain painters there was not a single case of silicosis. A number of other employments in porcelain works are given which are dust free. The medium and severe forms of silicosis were found only in workers who had been exposed to dust for over 11 years. In many cases the disease occurred many years after a comparatively short period of work with exposure to dust, and particulars are given of 3 cases in which severe silicosis occurred 40 years after work which exposed to dust. The periods of work were only from 4 to 9 years and there had been no exposure to dusty work after that. This long latent period requires further investigation.

SILICOSIS AND ASBESTOSIS. *Home Office, H. M. Stationery Office, 1932.*

"The object of this Memorandum is to set out briefly what the disease (silicosis) is, how and where it is produced, the means which can be taken to prevent it, the provision made for compensation, and the arrangements for the medical examination and certification of workmen."

"Within recent years it has been found that asbestos dust produces a fibrosis of the lungs (known as asbestosis) which resembles silicosis in many ways. A note on this disease is included."

Of particular interest is the following: "During the last three years there have been 80 cases (silicosis), including 30 deaths, amongst workmen employed in ganister mines and silica brick works; 179 cases including 25 deaths, in the getting and manipulation of sandstone at quarries and on p ises worked in conjunction therewith cases, including 87 deaths, in the po industry; 81 cases, including 32 deati the metal industries, including metal g ing and sandblasting; and 91 cases, incl 20 deaths, in coal mines."

It is a regrettable fact that while we i United States, have abundant eviden day of instances of both diseases we not even an approximate idea of our tistics thereon.

An excellent bibliography of 14 refer to British publications, mostly goverr tal, is appended.

OBSERVATIONS AND STUDIES ON SILICO DIATOMACEOUS SILICA. *R. T. Legg E. Rosencrantz. Am. Jour. Pub. H Oct., 1932, vol. 22, pp. 1055-1060.*

This paper summarizes the finding recommendations of an investigation silicosis hazard among workers enga mining diatomaceous earth in Santa B County, California. The deposit is posed of fossilized diatomes and n algae, and contains about 85 per cen silica. Workers are Mexican In selected by physical examination, ar authors state that these people hav highest tuberculosis rate in this co One hundred and eight were given examinations. The ages ranged from 56 years, and length of employment less than 1 to more than 5 years, 33 per having worked there 5 years. Pneu niosis of various degrees was present i per cent. of the cases, 60 men showing pneumoconiosis, 15 moderately adva and 6 in the advanced stage of the dis

The men were in excellent physica dition. The most striking physical found in 73 per cent. of the men, was cl fingers which two-thirds of the wo developed in the first year of employ

Dust concentrations were determi the U. S. Bureau of Mines, but unfortu are not included in this paper. The is a valuable contribution to the liter on the silicosis hazard from amo silica.—T. H.

MOLDING POWDER AS A CAUSE OF P NARY DUST DISEASE. *A. Abr*

lation of sandstone at quarries and on premises worked in conjunction therewith; 322 cases, including 87 deaths, in the pottery industry; 81 cases, including 32 deaths, in the metal industries, including metal grinding and sandblasting; and 91 cases, including 20 deaths, in coal mines."

It is a regrettable fact that while we in the United States, have abundant evidence today of instances of both diseases we have not even an approximate idea of our statistics thereon.

An excellent bibliography of 14 references to British publications, mostly governmental, is appended.

OBSERVATIONS AND STUDIES ON SILICOSIS BY DIATOMACEOUS SILICA. *R. T. Legge, and E. Rosencrantz. Am. Jour. Pub. Health, Oct., 1932, vol. 22, pp. 1055–1060.*

This paper summarizes the findings and recommendations of an investigation of the silicosis hazard among workers engaged in mining diatomaceous earth in Santa Barbara County, California. The deposit is composed of fossilized diatomes and marine algae, and contains about 85 per cent. free silica. Workers are Mexican Indians, selected by physical examination, and the authors state that these people have the highest tuberculosis rate in this country. One hundred and eight were given X-ray examinations. The ages ranged from 19 to 56 years, and length of employment from less than 1 to more than 5 years, 33 per cent. having worked there 5 years. Pneumoconiosis of various degrees was present in 68.5 per cent. of the cases, 60 men showing early pneumoconiosis, 15 moderately advanced, and 6 in the advanced stage of the disease.

The men were in excellent physical condition. The most striking physical sign, found in 73 per cent. of the men, was clubbed fingers which two-thirds of the workers developed in the first year of employment.

Dust concentrations were determined by the U. S. Bureau of Mines, but unfortunately are not included in this paper. The article is a valuable contribution to the literature on the silicosis hazard from amorphon silica.—T. H.

MOLDING POWDER AS A CAUSE OF PULMONARY DUST DISEASE. *A. Abraham. Abstr. as follows from Klin. Wochschr., 1932, vol. 11, pp. 1110–1113, in Chem. Abstr., 1932, vol. 26, p. 6035.*

Four case reports on injury from the manufacture of clay molding powder containing quartz, 31.17 per cent., feldspar, 50.4 per cent., and soluble silica, 1.79 per cent.; and lime molding powder containing quartz, 0.87 per cent., feldspar, 0.43 per cent., and soluble silica, 1.29 per cent.

CEMENT DUST INHALATION AND SPREAD OF TUBERCULOSIS IN GUINEA-PIG LUNGS. *J. Grober, and G. Riemerschmid. Abstr. as follows from Beitr. Klin. Tuberk., 1931, vol. 78, pp. 222–231, in Chem. Abstr., 1932, vol. 26, p. 6011.*

A series of guinea pigs was placed in the dustiest region of a cement establishment for 5 months after which it was found that the ash content of the lungs was greater than that of control animals while the calcium content of the lungs hardly differed from that of the cement-free animals. Twenty-four cement animals and an equal number of controls were injected intraperitoneally with human tubercle bacilli. The cement animals revealed the usual spread of tuberculosis but they rarely, in contrast to the non-cement animals, showed tuberculous changes in the kidneys or adrenals and still more rarely in the lungs, pleura, heart or bronchial glands. The pulmonary condition in the cement animals appeared to be less progressive than that of the controls. Whether this was due to a local deposition of calcium substances resulting in a chem. influence upon the lung tissue or a general change in the body as a whole requires explanation.

OBSERVATIONS ON THE PNEUMOCONIOSES. *E. H. Kettle. Abstr. as follows from Brit. Med. Jour., 1932, Aug. 13, pp. 281–283, in Bull. Hyg., 1932, vol. 7, p. 697.*

A thoughtful consideration is presented of present knowledge on dust diseases of the lungs. Most is known regarding the action of silica dust; still it is not clear whether the characteristic nodule originates in the localization of silica-containing phagocytes in pulmonary adenoid tissue, or as collagen deposited about silica-containing phagocytes

also presented. In connection with granite dust (25 to 40 per cent. quartz) the author points out that silicosis develops much more slowly than in an exposure to pure quartz dust and attributes this slower reaction to the possible neutralizing or inhibiting effect of the other constituents of granite. Attention is invited to the limitations of our present knowledge of many of the problems involved in pneumoconiosis and the need for more study of different types of dust both in the pure state and in measured combinations.—J. J. Bloomfield.

SILICOSIS OR PNEUMOCONIOSIS IN VERMONT GRANITE CUTTERS AND SLATE WORKERS. *Edward J. Rogers. New England Jour. Med., August 4, 1932, vol. 207, pp. 203–208.*

The author, who is the Medical Director of Vermont Sanatorium, has had excellent opportunity to observe pneumoconiosis and silicosis in Vermont during the twenty years of his service. He states that silicosis has been found by Jarvis, Russell, and others to be present in nearly all granite cutters. He quotes Russell that Italian and Swiss form the higher percentage of workers, the Scotch next, and Americans third.

The amount of silicosis found appears to be directly proportional to the concentration of dust to which the worker is exposed.

He comments on the absence of physical signs of disease in the chest and emphasizes the importance of dyspnea as an early symptom. Subsequent symptoms are disturbance of digestion, loss of weight, definite fatigue, and pleurisy. Occasionally the patient has a frank hemorrhage. There is also fever, which is not apt to be high.

The prognosis of tuberculosis in Silicosis is bad. Men break down with it several years after leaving their dusty work.

The author states that the amount of free silica in slate dust is probably greater than in granite dust. Finely powdered slate is, therefore, very dangerous to those exposed to inhaling it. However, the author is under the impression that the death rate from tuberculosis in the slate district is not as high as in the granite district. He points out that workers in marble dust appeared free of pneumoconiosis and tuberculosis.—W. I. Clark.

SEVERE SILICOSIS IN RELATION TO WORKMEN'S COMPENSATION LAWS. *E. Beintker. Schrift. a. d. Gesamtgeb. d. Gewerbehyg. No. 43, 1933, pp. 84.*

This monograph on the relation between the X-ray and clinical findings in silicosis is founded on the author's examination of 450 workmen who were exposed for several years to the inhalation of dust containing silica. Of these 338 were miners. Of 84 cases the author gives a résumé of the precedings, of the X-ray, and the clinical findings. As an objective test of the respiratory function he notes the time which the patient is able to hold his breath during which time his heart action is observed, and he watches toward the end of that time for symptoms of congestion such as acceleration of pulse, breathing rates, and swelling of the arm veins. During the entire examination breathing rates are observed. In all the cases he shows statistically that small chest expansion and small lung displacement are not necessarily typical for silicosis.

The author reports 63 cases in which he finds a discrepancy between the X-ray and the respiratory function excluding all cases of insufficiency from other etiology than silicosis. In 30 of these 63 cases he finds a severe decrease in function with only slight signs of fibrosis; in 33 cases the X-rays show a silicosis of second to third degree without marked change in the respiratory function. From this he reasons that it is not the fibrosis which produces the clinical signs of silicosis and he agrees with Reichman that emphysematous silicosis is not compensatory but a result of shrinking, traction, and resistance. This in itself perhaps causes a decrease of function so that in the most severe cases emphysematic parts and tentlike tractions of the lungs due to shrinking are almost detectable in the X-ray. He advances the theory that changes in the permeability of the walls of the alveoli result from mechanical irritation by dust particles. He attempts to explain the fact that particles of other dusts do not produce the same effect on the respiratory function; and believes that since phagocytosis of silica particles is slower than for other dusts, these latter remain in the alveoli a shorter time than does silica. His explanation is not altogether convincing since the effect of silica inhalation on the respirato reaction is not only quantitatively b qualitatively different from the effect other dust.

Beintker admits that his theory of t decrease in the permeability of the alve being the reason for the decrease in respi tory function is only a conjecture, as neith the X-ray nor the histological find ngs su port his theory.

The author ends with demanding that t decrease in respiratory function must considered more thoroughly than it is n by most German physicians who give exp opinion in cases of silicosis. He compar the finding of fibrosis in cases of silico to the finding of granulated erythrocytes lead poisoning, admitting the fibrosis o as an indicator that silicosis exists at but he denies that the degree of fibrosis be used as a test for the gravity of the c ease.—K. Stüber.

PNEUMOCONIOSIS. *J. R. Todhunter and B. Dixon. Lancet, November 19, 19 p. 1123.*

Attention is drawn to pneumoconiosis c to other dusts than silica. Five cases quoted in detail; in one the authors cla the condition to be indistinguishable fr silicosis; it occurred in a grease-mopper, a was associated with pulmonary tubercu sis. The second was somewhat similar, a occurred in a general laborer; the th affected an emery-wheel grinder; so did t fourth, which was associated with puln nary tuberculosis; the fifth and sixth w similar, there was fibrosis and pulmon tuberculosis after long exposure to em dust. The type of fibrosis, as seen p mortem, while somewhat similar to that silicosis, seems to have been less defi Men so affected experience an undue n tality from respiratory diseases, and grea care should be taken to safeguard them f the dust risk. There seems no good rea why such affected workers should not awarded compensation, just as in the of silicosis.—E. L. C.

PULMONARY ASBESTOSIS. *P. Ellman. L cet, 1933, February 4, pp. 252–53.*

The clinical, radiological, and pathol cal features of pulmonary asbestosis

effect of silica inhalation on the respiratory function is not only quantitatively but qualitatively different from the effect of other dust.

Beintker admits that his theory of the decrease in the permeability of the alveoli being the reason for the decrease in respiratory function is only a conjecture, as neither the X-ray nor the histological find ngs support his theory.

The author ends with demanding that the decrease in respiratory function must be considered more thoroughly than it is now by most German physicians who give expert opinion in cases of silicosis. He compares the finding of fibrosis in cases of silicosis to the finding of granulated erythrocytes in lead poisoning, admitting the fibrosis only as an indicator that silicosis exists at all, but he denies that the degree of fibrosis can be used as a test for the gravity of the disease.—K. Stüber.

PNEUMOCONIOSIS. *J. R. Todhunter and G. B. Dixon. Lancet, November 19, 1932, p. 1123.*

Attention is drawn to pneumoconiosis due to other dusts than silica. Five cases are quoted in detail; in one the authors claim the condition to be indistinguishable from silicosis; it occurred in a grease-mopper, and was associated with pulmonary tuberculosis. The second was somewhat similar, and occurred in a general laborer; the third affected an emery-wheel grinder; so did the fourth, which was associated with pulmonary tuberculosis; the fifth and sixth were similar, there was fibrosis and pulmonary tuberculosis after long exposure to emery dust. The type of fibrosis, as seen post-mortem, while somewhat similar to that of silicosis, seems to have been less definite. Men so affected experience an undue mortality from respiratory diseases, and greater care should be taken to safeguard them from the dust risk. There seems no good reason why such affected workers should not be awarded compensation, just as in the case of silicosis.—E. L. C.

PULMONARY ASBESTOSIS. *P. Ellman. Lancet, 1933, February 4, pp. 252–53.*

The clinical, radiological, and pathological features of pulmonary asbestosis are discussed from the author's personal experience. Dyspnoea is an early symptom, associated with a hard, non-productive cough. Anorexia was not unusual, and loss of weight out of proportion to the physical signs was a distinctive feature. Asbestos corns on the skin of the hands were often noted. The characteristic changes consisted of basal diffuse fibrosis, while the apices of the lungs were often hardly involved. The condition tends to interfere with the functions of the lungs, and to progress to a fatal issue more inevitably than does simple silicosis. On the other hand the tendency to succumb to inter-current tuberculosis is not so pronounced in asbestosis. Asbestos bodies are usually to be found in the sputum, and in the lungs post-mortem. When these bodies are found in clumps, they usually indicate the occurrence of disintegration of lung tissue. Apparently asbestos falls with silica into the group of active dusts which produce lesions in the tissues due to their toxic action after passing into solution. Such active dusts exert a definite influence on tuberculous infection, and assist the growth of the tubercle bacillus. In the author's series of 17 cases, 6 had tuberculosis. Serial skiagrams are of great value in following the progress of any case.—E. L. C.

SILICOSIS AND ALLIED DISEASES OF THE LUNGS. *M. J. Stewart. Lancet, 1933, February 4, pp. 253–254.*

A discussion was started by Professor Stewart at the Liverpool Medical Institution upon pneumoconioses. He distinguished between anthracosis, silicosis, and asbestosis, particularly indicating how the pathological characteristics of the two last named varied. He claimed that asbestosis was a more serious disease, proving fatal after 13 to 15 years employment, as compared with 32 or even 42 years in silicosis. Nevertheless, the tendency to tuberculosis was significantly less in asbestosis than in silicosis, a possible reason being that asbestosis was much more rapidly fatal, and so did not give tuberculosis a chance to develop. The tendency for pulmonary fibrosis in asbestos workers to be mainly at the base may be due to the action of gravity causing the long fine fibres to

pass downwards in the lung rather than in any other direction. An interesting discussion followed, to which Professor Dible and Professor Beattie contributed. One interesting point was the extent to which women rather than men succumb to asbestosis.—E. L. C.

INFRA-RED PHOTOMICROGRAPHS OF THE ASBESTOSIS LUNG. *S. R. Gloyne. Tubercle, 1933, vol. 14, no. 5, pp. 208–209.*

Interesting infra-red photomicrographs are published in contrast with illustrations taken on ordinary plates. The infra-red method without doubt shows up asbestos bodies in lung sections in far finer detail than does ordinary microphotography. The interest in this note lies in the possibilities it suggests of the use of infra-red photography in pathological work.—E. L. C.

OCCUPATION AND RESPIRATORY DISEASES. *A. E. Russell. South. Med. Jour., 1932, vol. 25, pp. 919–926.*

A summary of the knowledge on the relation between occupation and respiratory diseases is given. Some information is presented concerning the occurrence of silicosis in groups of granite workers. A steady rise in sickness and also in the prevalence of active cases of tuberculosis occurred in direct proportion to years of service in the industry and extent of dust exposure. These two groups of granite workers were exposed to an atmosphere containing 20 to 60 millions particles per cubic foot of air. The increase in morbidity and the prevalence of tuberculosis was not experienced by groups of workers who were exposed to less than 20 million particles per cubic foot of air. No such frequency of disability from tuberculosis occurred in other industries, two of which, anthracite coal mining and cement manufacturing, were dusty. The incidence of pneumonia in these last two industries was less than that of employees in general manufacturing. Workers in the granite and gold mining industries (both of which have silica dust hazards) had pneumonia in excess of those engaged in general manufacturing. It was shown that the dust hazard in the granite industry became excessive with the introduction of pneumatic tools and that while the death rate from tuberculosis in the general population has decreased in recent years, in certain dusty trades there has been an increase during the same period.—A. E. Russell.

PNEUMOCONIOSIS: A LIST OF REFERENCES. *Internat. Labour Office, Studies and Rep., Series F (Indust. Hyg.) No. 15, 1932, pp. 76.*

"This work is published in separate editions in English, French, and German—certain of the entries appear in French, the language in which the Bibliography was compiled. . . ."

References are given under: (1) Pneumoconiosis (93 refs.), (2) General pathology (268 refs.), (3) Experimental Research (106 refs.), (4) Clinic (172 refs.), and (5) Pathology (633 refs.).

Indices by subject, country, and author follow. It is difficult to see how anyone interested in industrial hygiene can afford not to have this book readily accessible. Authors and students will find it invaluable. (Available in America at the World Peace Foundation, 40 Mt. Vernon St., Boston for 50 cents.)

OCCUPATIONAL AFFECTIONS OF THE SKIN AND SPECIAL SENSES

COPPER DERMATITIS. *Erich Eisner. Abstr. from Dermat., Ztschr., Oct., 1932, vol. 64, p. 408, in Arch. Dermat. and Syph., Mar., 1933, vol. 27, p. 511.*

The author observed a patient in whom a lichenified eczema developed as a result of irritation from dust containing copper. Patch tests, as well as intra-cutaneous tests with copper sulphate solutions, were positive.

THE DIAGNOSIS OF OCCUPATIONAL DERMATITIS. *R. M. B. MacKenna. Jour. State Med., 1933, vol. 41, no. 1, pp. 5–20.*

This paper is based on a series of 100 consecutive cases of occupational dermatitis seen in private practice. Seventy-one per cent. were males. When distributed by age the peaks are found to be: one under age 25, and a second between ages 30 and 40. Cases which develop dermatitis before 25 possess to a greater degree than most a inherent susceptibility. Later in lif skilled workers may migrate to another fac tory, and meet with a new hazard, such, fo instance, as a French polisher who moved t a shop where the manager used turpentin substitute instead of turpentine, whic caused trouble; or again a painter who, a part of his work, was sent to paint a suga factory, and came in contact with dripping from sugar boilers and sugar dust; he de veloped sugar dermatitis. In the series c cases bakers and confectioners stood to with 24 per cent. of the cases, followed b French polishers with 17 per cent. and ce ment workers with 10 per cent. Exposur to oil claimed 8 per cent. Usually a perio of years is needed to sensitize to flour, bu 2 cases occurred after periods of 8 and 1 weeks respectively in different bakerie Toxic absorption from septic foci is seldo a factor in occupational dermatitis, whic usually commences as blebs on the finge and back of one hand, which spread rapidl to the other, and then to the forearms. Th palms of the hands are seldom affecte The rest of the body is not often attacke but if the thighs are affected the attack i usually a severe one, and if the body, oth than the shoulders and upper third of th chest, is concerned, the dermatitis is usuall of great severity and wide distribution; bu such cases are rare. Prevention lies i selecting the workers, selecting material and prophylactic measures. Much can b done in educating the workers with regar to the use of suitable cleansing material Often the physician needs to visit the worl shop to ascertain for himself the nature the hazard, which workers can seldom effi ciently describe. Differential diagnos may be difficult; it is mainly concerned wit deciding whether or not the condition caused by occupation. In some cases t lesions may be self-inflicted, but the malin gerer has seldom the skill to imitate derm titis lesions with success.—E. L. C.

THE NURSE IN INDUSTRY. *I. H. Charle Jour. State Med., 1933, vol. 41, no. pp. 47–52.*

The story is told how the Metropolita Life Insurance Company instituted i nursing service, which, far later, has spre

possess to a greater degree than most an inherent susceptibility. Later in life skilled workers may migrate to another factory, and meet with a new hazard, such, for instance, as a French polisher who moved to a shop where the manager used turpentine substitute instead of turpentine, which caused trouble; or again a painter who, as part of his work, was sent to paint a sugar factory, and came in contact with drippings from sugar boilers and sugar dust; he developed sugar dermatitis. In the series of cases bakers and confectioners stood top with 24 per cent. of the cases, followed by French polishers with 17 per cent. and cement workers with 10 per cent. Exposure to oil claimed 8 per cent. Usually a period of years is needed to sensitize to flour, but 2 cases occurred after periods of 8 and 12 weeks respectively in different bakeries. Toxic absorption from septic foci is seldom a factor in occupational dermatitis, which usually commences as blebs on the fingers and back of one hand, which spread rapidly to the other, and then to the forearms. The palms of the hands are seldom affected. The rest of the body is not often attacked, but if the thighs are affected the attack is usually a severe one, and if the body, other than the shoulders and upper third of the chest, is concerned, the dermatitis is usually of great severity and wide distribution; but such cases are rare. Prevention lies in selecting the workers, selecting materials, and prophylactic measures. Much can be done in educating the workers with regard to the use of suitable cleansing materials. Often the physician needs to visit the workshop to ascertain for himself the nature of the hazard, which workers can seldom efficiently describe. Differential diagnosis may be difficult; it is mainly concerned with deciding whether or not the condition is caused by occupation. In some cases the lesions may be self-inflicted, but the malingerer has seldom the skill to imitate dermatitis lesions with success.—E. L. C.

THE NURSE IN INDUSTRY. *I. H. Charley. Jour. State Med., 1933, vol. 41, no. 1, pp. 47–52.*

The story is told how the Metropolitan Life Insurance Company instituted its nursing service, which, far later, has spread to England. Groups of workers in factories, business establishments or offices, are insured *en bloc* for life, pensions, or sickness and accident. Cases of absence through sickness are notified to the local nursing association, perhaps the Queen's Institute for District Nursing, after a period of 2 days. On receipt of the call the nurse visits and offers service. Each visit is paid for by the Insurance Company at the rate of one shilling and fourpence. The nursing service definitely decreases absenteeism, which speeds up the productive capacity of industry. Stress is, however, laid on the need for co-ordinating all district nursing, so that at one and the same time a health visitor may not be inquiring about a pre-school child, and a school nurse, a district nurse, a tuberculosis nurse, together with the industrial service, be in attendance.—E. L. C.

DERMATITIS FOLLOWING THE WEARING OF DYED FABRICS. *E. M. Killick and J. T. Ingram. Lancet, January 14, 1933, pp. 77–80.*

The authors give full particulars concerning a case of dermatitis resulting from a garment which had been dyed black. It had been dyed solely with azo direct (substantive) dyes, which are normally used. On "patch" investigation the patient exhibited a specific idiosyncrasy to the dye, while normal subjects, and individuals suffering from other skin troubles, were negative. The authors claim that dermatitis arising from the use of paraphenylenediamine and allied chemicals is allergic in nature, rather than due to direct chemical irritation.—E. L. C.

ANOMALIES IN THE INTERPRETATION OF THE INDUSTRIAL DERMATOSES. *R. Prosser White. Jour. State Med., 1932, vol. 40, no. 10, pp. 559–572.*

An interesting but somewhat discursive discussion upon occupational skin troubles is presented. Workers may be made allergic to many industrial agents which can cause skin trouble; but every catarrhal inflammation of the skin is not a sensitized eruption. If a skin affection is called eczema, the worker cannot establish a claim for compensation, as he can if it is christened dermatitis. Once a worker is sensi-

The author states that radiologists more than any others need the salutary lessons of the deadhouse to correct their visionary interpretation of shadows, particularly of those radiating from the roots of the lungs. He states the unsatisfactory position today is due to lack of coöperation between physician, pathologist, chemist, and radiologist.

Diagnosis.—The history, knowledge of working conditions, and the presence of emphysema with dyspnoea altogether out of proportion to the amount of exercise, black morning sputum and minimal physical signs in the chest and the presence of fibrosis in the roentgen examination are the important points.

Prognosis.—If the diagnosis is made early and the patient removed from the mine and given an outdoor occupation, the disease appears to be not only arrested but considerable improvement in the lung condition and cardiac response takes place. Even the moderately advanced cases live many years in tolerable comfort. Infective conditions increase the gravity of the disease.

Prophylaxis.—Better ventilation, especially in dead-end places and cuttings, the universal adoption of wet drilling, and in special circumstances the use of respirators will materially lessen the incidence of anthracosis.

Treatment.—The essentials are removal of the patient from the mine dust and to guard against infection in an already damaged lung. Cough, dyspnoea, etc. are treated symptomatically. The author stresses the use of various drugs and diet and when there is cardiac failure the use of venesection.—E. P. Pendergrass.

POISONING BY MANGANESE ORES. *Salmon and Planque. Ann. d'Hyg., Pub. Indust. et Sociale, 1933, vol. 11, pp. 196–200.*

Some interesting cases are reported where men exposed to dust arising from mineral ores suffered from symptoms which presented the picture of manganese poisoning. Four men were affected, who presented symptoms of mental dullness, slow speech, pain in the muscles of the limbs, and difficulty in walking and the steppage gait. The picture is undoubtedly the same as was originally described in THIS JOURNAL (vol. 1, p. 183, and vol. 3, p. 231). Analysis of the ores showed them to contain 79 per cent. of manganese dioxide. The authors, however, somewhat unexpectedly attribute the toxic condition to the presence of 0.013 per cent. arsenic in the minerals. Such a small amount of arsenic present in dust would hardly be expected to exert any toxic influence.—E. L. C.

ON THE OCCURRENCE OF CLUMPS OF ASBESTOSIS BODIES IN THE SPUTUM OF ASBESTOS WORKERS. *M. J. Stewart, N. Tattersall, and A. C. Haddow. Abstr. as follows from Jour. Path. and Bacteriol., 1932, vol. 35, pp. 737–741, in Bull. Hyg., Jan., 1933, vol. 8, pp. 27–28.*

The authors describe two cases in which, in addition to single asbestosis bodies, a large clump of some 20 to 30 bodies, more or less radially arranged with their bulbous extremities outward, were found. The appearance of the clumps was similar to what is seen in histologic sections of the lung in cases of pulmonary asbestosis, where clumps of bodies occur either within the lung alveoli or embedded in fibrous tissue.

The authors suggest that the finding of a large clump or clumps of asbestosis bodies in the sputum is a clear indication of disintegration of lung tissue, either by a process of simple suppurative bronchopneumonia, or as a result of secondary tuberculosis infection. They believe that the fact that the sputum is rather roughly treated may account for the rarity of these clumps; on the other hand they have never found broken-up portions which might be expected to result from rough treatment. The radial arrangement of the bodies with the bulbous extremities at the periphery, indentical with that of the majority of the clumps in lung sections, shows, the authors consider, that such clumps are not artefacts but formations built up *in situ* within the lung alveolus from which they can only escape as a result of some secondary disintegrative process.

The authors conclude that since these clumps, which are only rarely encountered, point to disintegration of lung tissue their presence in sputum is of far greater significance than the finding of single bodies.—S. R. G.

THE DUST HAZARD IN THE COTTON INDUSTRY. *F. Koelsch. Abstr. as follows from Arch. f. Gewerbepath. u. Gewerbehyg., 1932, vol. 3, pp. 399–411, in Bull. Hyg., Jan., 1933, vol. 8, p. 27.*

The quantity of dust found in cotton mills may vary widely, as much depends on the kind and quality of the cotton, on the number of machines, and on the effectiveness of the exhaust ventilation. Chinese cotton gives off most dust. Among organic impurities in the dust are to be found the remains of seed pods and parts of fungi, and an analysis of the dust from American cotton shows about 50 per cent. of silica, and of the dust from Egyptian cotton about 29 per cent. Some of this was sand.

The author made some observations in a cotton mill where the rooms were spacious and well provided with exhaust ventilators and general ventilation, and where American long fiber cotton was used. Specimens of dust were taken at various places and counted with Owen's dust counter or the Zeiss konimeter. In a specimen taken near a carding machine there were 72 particles per cc. of air, of which about one-third were under 5μ. and a microphotograph of this specimen shows very little dust and particularly no fibers: this good result is believed to be due to the effective exhaust ventilation.

DUST, SMOKE, FUMES, AND VA
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CITY AIR POLLUTION AND ITS CONTROL. *H. B. Meller. Aerologist, Feb., and Mar., 1933, vol. 9.*

In two articles the author gives a good informative resumé of the air pollution problem for the lay reader, including control of combustion, physiologic and economic effects of pollution, and regulatory recommendations. Possible dangers to health are rather broadly covered with embodiment of intangible results difficult of proof.

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The long fiber cotton which comes mostly from America and Egypt scarcely reaches the lungs as the fibers are caught in the mucous membrane of the upper air passages. The short fiber cotton which comes from the East Indies and the small mineral particles are easily drawn into the lungs and there may give rise to disease.

The author refers to the latest international literature on the subject of these diseases and particularly to several English authors. Among the diseases mentioned are simple bronchitis, strippers' asthma, chronic bronchitis, silicotic dyspnea, weavers' cough, and tuberculosis.

The author investigated the health of 34 workers who had worked in the industry over 10 years. These were examined clinically and by X-rays and in 19 nothing abnormal was found; in 7 there was tuberculosis, but in none of these was there a family history of tuberculosis, and the remainder had bronchitis or difficulty of breathing. In no case were any changes found in the lungs resembling pneumoconiosis or pneumonomycosis.—A. J. C.

INDUSTRIAL DUSTS—PULMONARY TUBERCULOSIS AND PNEUMOCONIOSIS. *K. W. Jötten. Med. Lavoro, Feb. 1, 1933, vol. 24, pp. 48–50.*

Dusting experiments were made with a variety of industrial dusts using rabbits as experimental animals. The injurious effects of high silica dusts is emphasized.—L. T. F.

DUST, SMOKE, FUMES, AND VAPORS: THEIR DETERMINATION AND ELIMINATION

CITY AIR POLLUTION AND ITS CONTROL. *H. B. Meller. Aerologist, Feb., and Mar., 1933, vol. 9.*

In two articles the author gives a good informative resumé of the air pollution problem for the lay reader, including control of combustion, physiologic and economic effects of pollution, and regulatory recommendations. Possible dangers to health are rather broadly covered with embodiment of intangible results difficult of proof.

Practical scientific control has outstripped ordinance requirements which should be extended to control of combustion, dust, and sulfur acids, each of which is amenable to improvement. Raising requirements will stimulate betterment in design of automatic fuel feed devices; practical dust separation equipment is on the market; and even the status of pollution by sulfur acids, which presents just now the greatest technical difficulties, may be improved by regulations stipulating as low a sulfur content as feasible in the fuel sold, since it is possible to separate part of the sulfur in the coal as mined. Small apartments and homes are factors slighted in the consideration of control.—C. L. Pool.

(e) The hyaline nodules increase in size by extension at their periphery. Coalescence of adjacent nodules takes place and brings about involvement of further areas of the lung. (The conditions described under (c), (d), and (e) constitute the disease silicosis.)

Macroscopically the changes observed in silicosis are: (a) *In the early stage.* A variable number of palpable, pearly-white nodules up to 2 or 3 mm. in diameter on the pleural surface of the lung. On section, the cut surface of the lung is studded with pigmented foci, widely scattered, a moderate proportion of which are only palpable. The tracheo-bronchial lymph nodes may be slightly enlarged and deeply pigmented, and may exhibit foci of fibrous induration.

(b) *Later stages.* The fibrotic nodules are increased in number, size, and density, and coalescence of these may be found. The portions of the lung between the fibrotic nodules may be emphysematous. The tracheo-bronchial lymph nodes are smaller in size than those seen in the early stage and are fibrosed.

The presence of tuberculous infection usually modified the pathological appearance. Special attention was drawn to the three following types:

(a) In which the picture of silicosis above described may be little, if at all, modified, but in which only a biological test can demonstrate the presence of *B. tuberculosis.*

(b) In which the coexistence of silicosis and typical tuberculosis lesions is easily recognizable.

(c) In which the presence of tuberculosis is easily recognizable, but the existence of silicosis is more difficult to determine.

For compensation purposes, any stage of silicosis that is complicated with active tuberculosis should be considered as causing serious and permanent impairment and equivalent to third stage silicosis.

In order to claim compensation for silicosis, the claimant must have had the equivalent of 2 years' aggregate employment in an occupation or occupations involving exposure to a silica dust hazard in the State in which the claim is filed, unless the claimant was subjected to a prior physical examination authorized by the employer and at that time was not found to have silicosis. —E. P. Pendergrass.

SILICA IN THE ORGANISM AND SILICEOUS PARTICLES IN BLOOD. *L. Lematte and E. Kahane. Abstr. as follows from Compt. rend., 1933, vol. 196, pp. 575–578, in Chem. Abstr., May 20, 1933, vol. 27, p. 2479.*

A form of gelatinous silica from lung tissue, soluble in nitroperchloric acid, is called "silica of constitution." Compact mineral particles less attacked by the acid may come from respiratory dust. 0.3 to 0.7 mg. silicate per gram of dry fibrin comes from the blood; this amount is much less than the content reported by other authors. A deposit of silica particles of the second variety, too small to be weighed, regularly accompanies the other.

THE SILICIC ACID CONTENT OF LUNGS IN PNEUMOCONIOSIS. *Christian Hackmann. Abstr. as follows from Beitr. path. Anat., 1933, vol. 90, pp. 623–625, in Chem. Abstr., May 10, 1933, vol. 27, p. 2207.*

The amount of silica present in normal lungs is very small during childhood but tends to become greater with increasing age. In marked silicosis, injuries to the tissues are usually observed when the silica content exceeds 1 per cent. of the weight of the dried lung tissue, higher values being associated with greater degrees of injury.

REVIEW OF SILICOSIS. PART II. *Adelaide R. Smith. N. Y. State Dept. Labor, Industrial Bulletin, Apr., 1933, vol. 12, pp. 90–92.*

Factors in the development of silicosis are the type and percentage of silica or silicate in dust, the presence of other substances which may complicate the physiological reactions, the size of particles and degree of dustiness, and the length of exposure. Breathing of dust containing free silica in the cyrstalline or quartz form is most injurious, magnesium silicate is serious in producing "asbestosis," and other silicates cause milder disease. The usual damage from a high silica content appeared to be nullified where clay was present in the dust, but in other cases the presence of alkali was thought to increase the severity.

Only particles less than 10 microns in size are likely to enter the lungs, but most process dust encountered runs smaller. Much of the dust is invisible in the microscope, and even atmospheres rid of visible dust may cause silicosis. In jobs of moderate dustiness 20 to 50 million particles per cubic foot of air are found, but the many factors make it hard to say what exposure is safe. With granite dust of 35 per cent. free silica, 10 million per cubic foot are apparently safe.

Studies and figures abroad and here exemplify the ubiquity and seriousness of silicosis and concomitant tuberculosis, e.g. in mining and quarrying in Great Britain and the colonies, Germany and Italy; amongst grinders, sand blasters, foundry molders, and pottery workers in Germany; and amongst stone masons in Holland. The American figures for similar trades are astounding when first clearly comprehended, and pertinent summaries enlighten the effects on the lungs of spray coating, abrasives, cement, rock drilling, diatomaceous earth, slate milling, and talc mining occupations.—C. L. Pool.

COAL MINER'S LUNG: A PRELIMINARY ACCOUNT OF THE CHEMICAL ANALYSIS AND PATHOLOGY OF THE LUNGS OF COAL MINERS IN NEW SOUTH WALES. *C. Badham and H. Taylor. Med. Jour. Australia, April 29th, 1933.*

Details are given of the occupational history and post-mortem findings in 31 cases, of which 19 had been coal miners, 6 metalliferous miners, 1 a metal polisher, 1 an ore miller, 2 had no dust exposure, and 2 died from tuberculosis. The special methods used for determining the content in the lungs of silica and silicates, and of coally matter, are described. The opinion is expressed that different dusts set up different types of fibrosis in the lungs. The essential features of coal miner's lung are upper lobe consolidation, fibrotic nodules, non-tuberculous cavitation, and coal dust accumulation. This condition renders miners more vulnerable to respiratory diseases, such as bronchitis and pneumonia; in advanced cases cardiac disease with circulatory failure is aggravated. But it is doubtful whether tuberculosis can be aggravated or caused by a coal dust which has a high carbonaceous content. Compensation should be based upon disabiilty which may be absent in cases with well marked radiographic fibrosis; disability can only be determined by clinical methods.—E. L. C.

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INFRA-RED PHOTOMICROGRAPHS OF THE ASBESTOSIS LUNG. *S. R. Gloyne. Tubercle, Feb., 1933, vol. 15, p. 208.*

The article describes a preliminary attempt to employ infra-red photography in the study of inhaled particulate matter. "The photomicrographs reproduced (in the article) are taken on Ilford infra-red plates, using a 'pointolite' lamp for illumination and the infra-red filter. A 1/12 in. oil immersion lens was used in all cases." Focusing could not be carried out with the filter in place. "In each case a control photograph was first taken of identically the same field with an Ilford fine grain ordinary plate" and with this same focus a second exposure was then made on an infra-red plate. "The exposure of the latter was, however, 15 to 20 seconds longer."

A series of comparative photographs of asbestosis bodies indicates that in infra-red plates the detail is somewhat more pronounced, some of the bodies invisible in ordinary light are clearly discernible and the black pigment frequently seen in asbestosis does not register. However the author admits that these differences may be due to the fact that the infra-red filter alters the focus.—LEROY U. GARDNER.

THE EFFECT OF CEMENT DUST UPON WORKERS. *A. E. Russell. Abst. as follows from Am. Jour. Med. Sci., 1933, vol. 185, p. 330, in Can. Med. Ass. Jour., June, 1933, vol. 28, p. 687.*

The purpose of this investigation was primarily to measure in terms of physical condition and disabling sickness the effects upon the workers of exposure to known quantities of cement dust. The study was conducted throughout a period of nearly three years. The workers were examined twice during this period and where indicated special examinations and x-rays were made. If an employee remained away from work on account of illness the exact nature of the illness was ascertained.

In the mill where the investigation was conducted the air contained from 22 to 63 million particles per cubic foot of air. These particles were not larger than 10 microns. Chemical analysis showed that cement contains about 62 per cent. lime and 22 per cent silica. Other constituents

diarrhea and abdominal cramps in 1925; in 1926 he complained of mental confusion, incoördination, pain in the back, weakness of the legs and twitchings of the arms. Examined in April, 1927, he displayed pallor, a lead line on the gums, exaggerated equal reflexes and lead in both urine and feces. The blood pressure was 120 systolic and 80 diastolic, and never exceeded 130 systolic at any time during the illness. The hemoglobin in April, 1927, was 85 per cent., and there were no stipple cells.

Treatment during the first 4 days consisted of the administration of 4 cc. of syrup of hydriodic acid every 3 hours; during this phase of the treatment the output of lead in the excreta increased. Mobilization was attempted on the fifth day, calcium lactate 1 gm. and 1 quart of milk (each twice a day) being given. In a month the lead line had disappeared, and lead disappeared from the urine and feces. In Jan., 1928, the patient was sufficiently improved to permit de-leading. This was done by giving 1 gm. of ammonium chloride every hour and by requiring of the patient a calcium-poor diet. Included in the dietary were meat, liver, potato, rice, fruit and all milk-free foods; forbidden were milk, eggs, and green vegetables. Early in March, the patient reported that he had never felt better in his life; but on March 11, 1928, he experienced a series of tetany-like convulsions, with periods of pronounced cyanosis and marked apnea; each seizure began with twitching of the muscles of the right eye and spread to the entire body. Chvostek's and Trousseau's signs were elicited between attacks. Following the intravenous administration of 60 gm. of calcium chloride, the patient experienced relief. During the next 6 months he was well, but in the fall of 1928 he began to become worse. Headaches, confusion, and right hemianopia appeared. In Dec., 1928, a second series of convulsions occurred; they were similar in type and in response to treatment to the earlier seizures. The patient gained very slowly, his mental state remaining confused, and he died in Nov., 1929, of acute bronchopneumonia.

The case is almost certainly one of lead encephalopathy; the chemical components of the blood and urine rule out uremia, while the result of treatment makes the diagnosis of multiple sclerosis unlikely. That the convulsions were in the nature of tetany seems to be borne out by their appearance while the patient was on a low calcium diet, by spectacular improvement following treatment with calcium and by the existence of the Chvostek and Trousseau phenomena. The second spell occurred in the course of a diarrheal disease when the gastro-intestinal tract was unable to absorb injected calcium.

PREVENTION OF LEAD POISONING IN INDUSTRY. *G. H. Gehrmann. Am. Jour. Pub. Health, July, 1933, vol. 23, pp. 687–692.*

LEAD POISONING IN A GLASS BEAD COLORING WORKS. *F. W. Bickert. Abstr. from Zentralbl. f. Gewerbehyg., 1932, vol. 19, pp. 211–212, in Bull. Hyg., June, 1933, vol. 8, p. 400.*

Colored glass beads are at present prepared by introducing the color, mixed with white lead, inside the bead by an instrument resembling a pipette with a fine drawn-out point. Women who do this work at home warm the nozzle of the introducer between their lips after every two or three beads. An expert woman can do five gross of beads in one hour, and so brings to her mouth the color containing white lead 200 to 300 times an hour. Five women affected with lead poisoning were found. Although the teeth in all had been well cared for, all presented a distinct blue line, as well as small bluish patches on the mucous membrane inside the cheeks on a level with the lower teeth. Numerous basophilic punctate red blood corpuscles were found and there was hematoporphyrinuria. The women complained of pains in the limbs, especially in the calf muscles, and headache with loss of appetite. When the cause of the illness was recognized the use of white lead was stopped and titanium dioxide used in its place.

INVESTIGATION OF LEAD CONTENT IN THE TARTAR AND SALIVA OF WORKERS IN THE LEAD INDUSTRY. *J. Schmidt. Zentralbl. f. Gewerbehyg., May-June, 1933, N. S. vol. 10, pp. 101–104.*

Lead determinations were made on the tartar and saliva of a number of workers in the lead industry, a group of people suffering from lead poisoning, and on a rabbit and turtle which had been given lead poisoning artificially. A control lead determination was carried out as a check on the method of analysis. The results show that there is no relation between the symptoms of lead poisoning and the lead content in the tartar, and that the lead content of the tartar can therefore not be used as means of diagnosis. There is furthermore no relation between the "lead line" and lead content of the tartar. In no instance was any lead found in the saliva.

The author concludes that lead determination of the saliva or tartar as a method

DUST HAZARDS AND

THE MORBID ANATOMY AND HISTOLOGY OF ASBESTOSIS. *S. R. Gloyne. Tubercle, July, 1933, pp. 445–451.*

Asbestos dust differs greatly in structure and chemical composition. Occupational conditions in mines, quarries, and factories, so vary the exposure to dust that some workers experience massive, rapid doses, while others have only slow and gradual exposure to small doses. Operatives range from strong men to weakling girls, whose pulmonary tissue is not identical. Other diseases may confuse the pathological picture. Peculiar long needle-shaped particles characterise asbestos dust; the resulting fibrosis is diffuse, silicotic nodules being absent. The changing conditions lead to unusual forms of asbestosis. In the pleura the disease is marked by the obliterative nature of the process. The naked eye appearance of a lung is characterised by dense blue-black areas corresponding to the secondary lobules, surrounded by thick bands of interlobular connective tissue, generally with a reddened background of terminal bronchopneumonia in the less affected portions of the lung. Signs of disease in other organs are few. An excellent colored plate exhibits the naked eye appearance.—E. L. C.

THE MORBID ANATOMY AND HISTOLOGY OF ASBESTOSIS. *S. R. Gloyne. Tubercle, Aug., 1933, pp. 493–497.*

This article is a further contribution by

tartar and saliva of a number of workers in the lead industry, a group of people suffering from lead poisoning, and on a rabbit and turtle which had been given lead poisoning artificially. A control lead determination was carried out as a check on the method of analysis. The results show that there is no relation between the symptoms of lead poisoning and the lead content in the tartar, and that the lead content of the tartar can therefore not be used as means of diagnosis. There is furthermore no relation between the "lead line" and lead content of the tartar. In no instance was any lead found in the saliva.

The author concludes that lead determination of the saliva or tartar as a method of diagnosing lead poisoning is aimless.—A. D. B.

LEAD IN THE CEREBRO-SPINAL FLUID AFTER LEAD POISONING. *M. Kasahara and K. Arimichi. Abstr. as follows from Z. ges. exp. Med., 1932, vol. 81, pp. 696-697, in Physiol. Abstr., Aug., 1933, vol. 18, p. 370.*

Dogs and goats were used and the lead given *per os*. The cerebro-spinal fluid was obtained by suboccipital puncture and the lead estimated by Nakaseko's method. A goat of 15 kilo weight receiving 270 g. lead acetate in 10 days showed 0.95 g. lead in 10^6 cc. fluid. In dogs the fluid was collected from 5 poisoned animals and 0.28 g. of lead per 10^6 cc. of fluid found.

DUST HAZARDS AND THEIR EFFECTS

THE MORBID ANATOMY AND HISTOLOGY OF ASBESTOSIS. *S. R. Gloyne. Tubercle, July, 1933, pp. 445-451.*

Asbestos dust differs greatly in structure and chemical composition. Occupational conditions in mines, quarries, and factories, so vary the exposure to dust that some workers experience massive, rapid doses, while others have only slow and gradual exposure to small doses. Operatives range from strong men to weakling girls, whose pulmonary tissue is not identical. Other diseases may confuse the pathological picture. Peculiar long needle-shaped particles characterise asbestos dust; the resulting fibrosis is diffuse, silicotic nodules being absent. The changing conditions lead to unusual forms of asbestosis. In the pleura the disease is marked by the obliterative nature of the process. The naked eye appearance of a lung is characterised by dense blue-black areas corresponding to the secondary lobules, surrounded by thick bands of interlobular connective tissue, generally with a reddened background of terminal bronchopneumonia in the less affected portions of the lung. Signs of disease in other organs are few. An excellent colored plate exhibits the naked eye appearance.—E. L. C.

THE MORBID ANATOMY AND HISTOLOGY OF ASBESTOSIS. *S. R. Gloyne. Tubercle, Aug., 1933, pp. 493-497.*

This article is a further contribution by the above author and deals with the asbestos fibre and the tissue reaction it provokes. The asbestosis lung shows five kinds of particles: (a) carbon pigment common to all town-dwellers; (b) amorphous brown pigment, presumably blood; (c) sharp jagged particles, probably carbonaceous; (d) the asbestos fibre, and (e) the asbestos body. Asbestos fibres may also be found in the upper respiratory tract. The tissue reaction is three-fold: (a) thickening of the fibre by deposition of organic iron-containing material to form the asbestosis body; (b) cellular changes, of which the chief are accumulations of large phagocytic dust cells and asbestosis giant cells; and (c) the fibrosis common to all forms of pneumoconiosis. The asbestosis giant cell is not a cell in the true sense, as is the tuberculosis giant cell. It is a minute collection of phagocytes, fused together, and is an attempt on the part of the tissues to protect themselves against an irritant which cannot be lysed or absorbed. A characteristic immobilization, with long persistence, of phagocytes occurs, expecially of the asbestosis giant cell. The subject is to be continued.—E. L. C.

THE MORBID ANATOMY AND HISTOLOGY OF ASBESTOSIS. *S. R. Gloyne. Tubercle, Sept., 1933, pp. 550-558.*

This article is the last of a series by the same author dealing with asbestosis; it is

concerned with microscopic appearances. The characteristic of the asbestosis lung is the holding up of the dust fibres at the distal end of the respiratory bronchioles and in the alveolar ducts, with a cellular reaction of large mononuclear phagocytes and the occurrence of asbestosis giant cells. Later the lymphatics and adjacent air sacs are blocked with the dust and phagocytes. Finally, an increase of connective tissue follows round the bronchioles, alveolar sacs, capillaries, and venules, in the interlobular septa and beneath the pleura, completely obliterating all lung configuration. The characteristics of asbestosis are its diffuseness, its tendency to affect the lower lobes and set up pleural adhesions, the presence of asbestosis bodies, and concomitant tuberculosis and bronchopneumonia.—E. L. C.

RAPID DEVELOPMENT FROM LATENT TO SEVERE SILICOSIS. *Friedrich Scheidemandel. Ztschr. f. Tuberk., 1933, vol. 67, pp. 241–246.*

A case report is given of a man who had worked as a stone cutter for 29 years and who became ill in 1929, with pulmonary symptoms of cough, dyspnea, and weight loss which lead to roentgen examination. From this a diagnosis of bilateral tuberculosis was made. No evidence of pneumoconiosis was seen. Repeat films were made and not until July, 1931 was definite evidence of pneumoconiosis seen. The man died in April, 1932 and a post mortem examination was made in June, 1932 at the request of the Union to which the man belonged. Microscopic evidence of silicosis was found. It is concluded that there was so-called latent, unrecognized pneumoconiosis present at the first examination and that this progressed rapidly under the stimulation of the tuberculous infection.

The German law regarding the compensability of dust diseases is briefly discussed. The clinical signs of dyspnea and impaired circulation combined with unmistakable roentgen evidence of pneumoconiosis are necessary for this occupational disease to be considered worthy of compensation. It is only when tuberculosis is combined with pneumoconiosis that the former is compensable. The author believed the law is a bit too rigid in its terminology as there are cases where silicosis is present in sufficient degree to cause symptoms but not to result in definite roentgen changes.

Mention is made of the chemical effect of silica in the alkaline body fluids; also the experimental proof that silicosis favors the development and progress of a tuberculous infection. The necessity for autopsy examination for the conclusive diagnosis of silicosis in certain cases is noted.

Reproductions of four roentgenograms are given.—*E. P. Pendergrass.*

ETIOLOGY OF SILICOSIS. *A. J. Lanza. Jour. Am. Med. Assn., August 19, 1933, vol. 101, p. 583.*

This paper reviews briefly the known facts, discusses the recent Picher studies, enumerates some of the common causes of silicosis, and concludes with the following:

"Silicosis is a widely spread occupational disease and its peculiar relationship to tuberculosis, the difficulties of diagnosis, and the extreme difficulty associated with efforts for prevention and control, all combine to make it a disease of major importance. For this reason, I desire to emphasize most strongly the necessity for the precise recording of the patient's occupation, both by physicians and hospitals, especially in cases which present pulmonary symptoms. Not merely the patient's present but also his previous occupations should be ascertained.

Recent investigations and studies have revealed various types of pneumoconiosis which tend to confuse further both diagnosis and prognosis. This makes imperative an accurate marshalling of all the etiologic factors in the consideration of any case in which the inhalation of silica dust is concerned."

THE CLINICAL MANIFESTATIONS OF SILICOSIS. *R. R. Sayers. Jour. Am. Med. Assn., August 19, 1933, vol. 101, p. 580.*

As the author points out, this paper is essentially a review of the three papers recently published by him with Meriwether, Lanza, and Adams.—P. D.

SILICON DIOXIDE CONTENT OF LUNGS IN HEALTH AND DISEASE. *W. D. McNally. Jour. Am. Med. Assn., August, 1933, vol. 101, p. 584.*

This is a review with numerous references followed by tabulations of analytical data from the author's autopsy material. V his analytical procedure may be questi he shows that silicosis can be unfail proven by analyses of the lungs at aut He suggests 1.13 mg. of silica per gr dried lung as the normal. Specimens nitely silicotic contained varying am up to 26 mg. per gram of dried lung.—

MINERAL MATTER IN LUNGS. *W. E. C Brit. Med. Jour., Sep. 9, 1933, p. 49*

A method is given for preparing material so as to detect its mineral co The tissue is digested for 2 or 3 days trypsin; then the turbid fluid is centri and examined. Next the remaining and lung are reincubated for 24 hour acidified; then the fluid is centrifug before and examined. Finally the de in the centrifuge tubes are mixed strong sulfuric acid and potassium b mate and boiled; then the tube is c fuged and the deposit examined as b The first deposit contains carbonaceou other mineral particles; the second co in addition carbonaceous and "c matter; the final deposit contains insoluble silicates, diatoms, etc., free organic and "coaly" matter.—E. L. C

THE SILICA CONTENT OF LUNGS. *Sladden. Lancet, July 15, 1933, 123–125.*

Facts are presented gathered from p logical and chemical investigation lungs from colliers, stone-masons, po and others who were exposed to indu dusts. No effort is made to disti between silica and silicate in analyzin dried lung substance. The record divided into six groups according t silica content of the lung; this grou found to agree with the amounts of fi present in the lungs; the higher is th centage of silica, the greater is the an of fibrosis. Coal-dust alone, apart silica, was not found to cause serious sis. The conclusion is stated that the silica content exceeds 1.0 per ce the dried lung substance, fibrosis, c contributory towards death, is u present; quantities below this an while not negligible, are seldom asso with deaths attributable to pulmona

from the author's autopsy material. While his analytical procedure may be questioned, he shows that silicosis can be unfailingly proven by analyses of the lungs at autopsy. He suggests 1.13 mg. of silica per gram of dried lung as the normal. Specimens definitely silicotic contained varying amounts up to 26 mg. per gram of dried lung.—P. D.

MINERAL MATTER IN LUNGS. *W. E. Cooke. Brit. Med. Jour., Sep. 9, 1933, p. 492.*

A method is given for preparing lung material so as to detect its mineral content. The tissue is digested for 2 or 3 days with trypsin; then the turbid fluid is centrifuged and examined. Next the remaining fluid and lung are reincubated for 24 hours and acidified; then the fluid is centrifuged as before and examined. Finally the deposits in the centrifuge tubes are mixed with strong sulfuric acid and potassium bichromate and boiled; then the tube is centrifuged and the deposit examined as before. The first deposit contains carbonaceous and other mineral particles; the second contains in addition carbonaceous and "coaly" matter; the final deposit contains silica, insoluble silicates, diatoms, etc., free from organic and "coaly" matter.—E. L. C.

THE SILICA CONTENT OF LUNGS. *A. F. Sladden. Lancet, July 15, 1933, pp. 123–125.*

Facts are presented gathered from pathological and chemical investigation of 60 lungs from colliers, stone-masons, potters, and others who were exposed to industrial dusts. No effort is made to distinguish between silica and silicate in analyzing the dried lung substance. The records are divided into six groups according to the silica content of the lung; this grouping is found to agree with the amounts of fibrosis present in the lungs; the higher is the percentage of silica, the greater is the amount of fibrosis. Coal-dust alone, apart from silica, was not found to cause serious fibrosis. The conclusion is stated that when the silica content exceeds 1.0 per cent. of the dried lung substance, fibrosis, clearly contributory towards death, is usually present; quantities below this amount, while not negligible, are seldom associated with deaths attributable to pulmonary disease, except when tuberculosis has intervened; when the silica content exceeds 1.6 per cent., severe fibrosis, sufficient in itself to lead to death, is invariably present. Such severe fibrosis so damages the pulmonary drainage system that the accumulation of further dust inhaled is rapidly accelerated, thus greatly increasing the danger to life.—E. L. C.

SILICOTIC LUNGS: THE MINERALS THEY CONTAIN. *W. R. Jones. Jour. Hyg., Aug., 1933, pp. 307–329.*

This article is one of considerable interest. It is written by a geologist after examining the petrology of rocks, the dust of which has been blamed for causing silicosis, and the mineral residues of 29 silicotic lungs. The author gives reasons for concluding that pulmonary fibrosis, due to dust, is not in the main caused by quartz, or free silica, but by a mineral, sericite, a hydrated silicate of aluminium and potassium, also known as "secondary white mica." This mineral is present in minute fibres and scales in all rocks and materials incriminated as originating silicosis; it is also found in the residues and the lung tissue. No quartz-bearing rocks are known to start silicosis, except those containing in abundance fibrous aggregates of such silicate minerals as sericite and sillimanite; asbestos also is a fibrous silicate. Rocks containing a relatively small percentage of quartz, but much of these fibrous silicate minerals, have been recognized as causing silicosis. No opinion is expressed as to whether these minerals act merely as mechanical irritants, or induce chemical changes, in instigating the growth of fibrous tissue in the lungs. There are good petrological illustrations.—E. L. C.

SILICATES AND SILICATOSIS. *C. P. McCord. Indust. Med., July, 1933, vol. 2, pp. 4–12.*

"A discussion of the origin and effects of dusts containing little or no free silica." This is a review, with extensive quotations and citations from the literature.

THE EFFECTS OF COLLOIDAL SILICA ON EXPERIMENTAL TUBERCULOSIS IN GUINEA-PIGS. *S. L. Cummins and C. Weatherall. Jour. Hyg., Aug., 1933, pp. 295–306.*