A FATAL CASE OF SILICOSIS

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(With Plates I—III, containing Figs. 1–6)

Investigations by the Inspectorate of Factories and Workshops have demonstrated a good many cases of silicosis among the workers in various industries in Denmark. In a comprehensive examination of porcelain workers, many were found to present marked silicotic changes in the lungs. One of these porcelain workers died on November 7th, 1933. As this case involves several interesting conditions, we think it is worth publishing.

It is the case of a porcelain turner, 63 years old, who had worked in the same factory for 46 years. His duties were the usual ones of a turner, mostly working with the wet or moist clay, but also cleaning, scraping and putting the finishing touches to the more dry, but not yet burned pieces of pottery—which work is done by means of rapidly rotating plates. This finishing produces a good deal of porcelain dust. In addition, some of the wet clay on the turner's hands and clothing will dry and be sent flying in the air by the motions of the turner. Further, minute particles of fine, moist clay will be thrown out in the air under the process of turning. There are thus many circumstances to explain why a very high percentage of silicosis[1,2] is found just among the turners, although the dust in the turnery is not so conspicuous as in many of the other shops in a porcelain factory.

This patient had always been in good health before. In 1932 he was confined to bed for about a week on account of "pneumonia," which is more likely to have been merely a fairly severe attack of bronchitis. A condition of dyspnoea had developed during the last two years before he died. His shortness of breath was aggravated gradually, and for the last year he was no longer able to ride his bicycle or walk the distance from his home to the factory, and he had to take the tram. The dyspnoea was most pronounced in the morning. Usually, when he woke up, he would have to wait a little to get his breath before he could dress and go to work. His family urged him to stay at home and report himself ill. He was a hardy fellow, however, and would not give up to his illness. His fellow-workers noticed that he was getting worse and worse, but he also refused to take their advice and report

[1] Gudjonsson (1933). [2] Hofbauer-Flatzech (1932).

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himself ill. On November 7th he was very short of breath in the morning and felt very poorly. His dyspnoea increased rather rapidly within about 15 min. He complained of pains in the chest and heart; then he became unconscious, with very marked dyspnoea and cyanosis. He was brought to the hospital, where he died a few hours later.

On July 20th, 1932, he had been examined roentgenographically together with other porcelain workers, with a view to the possible presence of silicosis. The X-ray plate of this patient (Pl. I, Fig. 1) showed: silicosis, in the third stage, with dense and partly confluent patches and motley areas with marked fibrosis; spontaneous pneumothorax on the right side, occupying the lower half of the right pleural cavity, where there are several conspicuous adhesions between the inferior surface of the lung and the diaphragm. The heart is displaced towards the left.

On admission to the hospital, the patient was still unconscious. The pulse was good, but the respiration very difficult. The lips were cyanotic. The skin was pale. He appeared on the whole to be moribund.

On account of the very poor condition of the patient, no further examination was made. He was given coramin every 15 min., after which the pulse improved somewhat. But he stayed unconscious. Attempts were made at lavage of the stomach, for the sake of any eventualities, but it had to be given up, as his condition was aggravated by this attempt. He kept going downhill rapidly, and died 2½ hours after admission, without gaining consciousness.

On autopsy, the findings were: pneumothorax sin. l. gr.; pleuritis dupl. fibr. magnu gradu; myofibrosis septi cordis; stasis hepat. et renum; silicosis pulm. duplex.

As to the condition of pneumothorax it should be mentioned that there were numerous adhesions between the lung and chest wall, that the lung felt very firm, and that it is probably on account of these two features that the lung had not collapsed to a greater extent. The adhesions, on both sides, were partly cord-like, partly flat and more extensive; there were cord-like adhesions between the inferior surface and the diaphragm. The lungs were markedly anthracotic, more than is found in many miners, their uneven surface reminding of terrazzo flooring. There were some very large emphysematous vesicles at the base of the lungs, the largest attaining the size of a fist.

The heart showed signs of beginning hypertrophy and scattered patches of myofibrosis, a few of these being located at or near the bundle of His-Tawara in the septum. (Microscopic examination of the septum, however, did not show any changes pronounced enough to constitute a cause of death.)

The autopsy revealed no other particular changes apart from stasis in the liver, kidneys and spleen. This is illustrated in Fig. 3, showing the posterior surface of the lungs, with the oesophagus laid open.

Fig. 4 shows very distinctly the uneven terrazzo-like surface of the lungs, with large areas of pleural adhesions covering parts of the right lung. Here

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will be noticed also a conglomerate of emphysematous vesicles, with the diaphragm suspended by the adhesions. The lungs were hard to the touch, inelastic, and filled with hard nodules, varying in size, the largest measuring about 5 cm. in diameter.

Cutting into the lung tissue produced a crackling sound, and when the knife was drawn over the cut surface it produced a harshly grating sound. A laboratory helper (not physician) who prepared the lungs, not knowing whence they came, exclaimed: "Why, the lungs are full of sand!"

Fig. 5 is a photomicrograph (×10) of a section from the left lung (van Gieson stain). It shows very plainly that the normal alveolar structures are largely replaced by more or less confluent dark areas, *i.e.* markedly anthracotic lobules separated by proliferating masses of connective tissue, lighter in colour.

Fig. 2 presents a roentgenogram of the lungs after removal from the chest. Here too the motley and large confluent densities are conspicuous features, besides large and small emphysematous vesicles. The lower parts are really quite transformed into conglomerates of vesicles.

It may be that Fig. 1 shows a suggestion of the marked emphysema of the right lung, as what appears to be evidence of pneumothorax is perhaps, at least in part, due to emphysema.

Microscopically, the firm nodular masses in the lungs (Fig. 6), small and large, and nearly one whole lobe, are found to consist of coarse fibrous connective tissue, with the fibres arranged in criss-crossing streaks, interspersed in several places with areas of markedly hyaline connective tissue. Between these streaks are accumulations of anthracotic pigment, embodied in large round cells with distinct nuclei, the cells being arranged in small groups of alveolar form (and, except for the colour of the pigment, they might recall a melanosarcoma with alveolar arrangement of the tumour cells). There are all sorts of transitions from these large round cells to smaller angular cells and elongated fibroblast-like cells, the most slender of which are found in the fibrous tissue.

Here and there are also areas of diffuse, but less intensely pigmented anthracosis, without intracellular arrangement of the pigment. There are, in addition, scattered in the connective tissue, a good many elongated slits which look as if they might contain pointed, spindle- or needle-shaped, bodies which do not take the stain. Here and there, in the marginal zone of the fibrous processes, are areas infiltrated with lymphocytes and fibroplastic processes with an abundance of spindle-shaped or polygonal connective tissue cells.

In one spot there is observed a "relatively small" polynuclear giant cell, well preserved, but without any cellular content of crystalloid nature.

The affection bears no resemblance to tuberculous changes, and *no tuberculous process whatever can be demonstrated in any of the sections*. The scanty "normal" lung structures show a slight thickening of the septa and some desquamation of the alveolar epithelium (chiefly cadaveric). Here and there are microscopic pigmented nodules of anthracotic cells in connective tissue.

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A small bronchus shows bronchiectatic dilatation with accumulation of desquamated epithelium; the wall of the bronchus is well preserved, quite thin, and engorged with congested capillaries. A larger, cartilage-containing, bronchus shows also bronchiectatic changes. Here the epithelium has disappeared and is replaced by granulation tissue, without anthracosis or any sign of tuberculosis.

Previously it was thought, although with some doubt, that SiO_2 might probably be the sole cause of silicosis—a view which is plainly evident from the conclusive definition of silicosis as given in the *Records of the International Conference at Johannesburg*, 1930: Silicosis is the pathological condition brought about in the lungs after persistent inhalation of SiO_2; and the more chemically pure the silicon, the more pronounced the condition of silicosis.

Recently, however, Jones (1933) has arrived at the opposite conclusion, finding that a compound silicate, sericite, is the true cause of silicosis in the cases examined by him.

This finding implies a possibility of an explanation of the peculiar fact, that silicosis is very frequent, for instance, among workers in the gold-mines of Transvaal, but rare in the Kolar gold-mines in India, although much quartz dust is produced in the working of the minerals in both places.

Similar peculiar differences in the frequency of silicosis are also known from Scotland, Wales and other districts.

In this connection it may be appropriate to give some mineralogical data concerning sericite:

SERICITE belongs mineralogically to the mica group; it is also called "secondary white mica." Sericite is a modification of muscovite, which belongs to the same group. The latter mineral is a hydrated potassium-aluminium-silicate, but with varying amounts of SiO_2 in the molecule. Muscovite crystallises in rather large platy crystals.

Sericite is finely crystallising muscovite, appearing often as fine pointed needles, measuring usually 5–10 microns, but often as small as 1–2 microns.

"Sericitisation" designates that transformation into sericite from other minerals (felspar, topaz, and others) which goes on extensively in nature under the influence of pneumatolytic and hydrothermal factors. Thus it is easy to understand that sericite may be present in many of the minerals and mineral mixtures employed in industry.

KAOLIN. Sericite and sericite needles are difficult to distinguish from other silicates which are chemically and mineralogically closely related—as, for example, the kaolinite group, the composition of which includes both aluminium, iron and chromium in the molecule.

Kaolinite is a hydrated aluminium-silicate which crystallises as finely scaly aggregates.

At the autopsy on the above-mentioned porcelain worker, parts of his lungs were employed to search for sericite by the method given by Jones (1933).

The technique was as follows:

The portion of the left lung used in this analysis weighed, in frozen fresh condition, 216 g.

To this lung tissue was added a total of 600 c.c. pure concentrated nitric acid (sp. gr. 1·40). The sliming of the tissue was completed in 11 days, where-

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after the black "slime" was boiled down and evaporated. There remained a black residue which, after calcination, was transformed into a cohesive mass, weighing 0·85 g.

The further examination of this mass was carried out, together with five histological preparations from the lung. Numerous sections were cut and examined, stained and unstained. In the course of these studies it was found that direct incineration of a piece of the lung gave better results as far as the mineralogical diagnosis is concerned.

Examination of the sections with the polarising microscope revealed minerals in considerable quantity, mostly quartz, and in addition—*in every section*—some needles, varying in size, up to 15 microns, besides countless needles about 1 micron in size.

These needles presented the following four characteristics: (1) Birefringent with (2) parallel extinction, (3) optically positive in longitudinal direction, (4) refractive index about 1·54.

Dense accumulations of minerals are observed in areas which contain many coal particles and in connective tissue.

Preparations obtained by the Jones method show numerous needles, measuring up to 5 microns, but consisting mostly of fragments of needles, probably broken in the somewhat rough handling of the material. These needles have the same four properties as given above.

A piece of lung that is incinerated yields needles which are larger and more distinct than those obtained by the Jones method.

For control material we have used:

(1) Apparently normal lungs from a woman who had never worked in any industrial plant, and had lived in the country throughout her last 28 years. No needles could be found in her lungs.

(2) Sections from tuberculous lungs. They showed some rod-like bodies, differing from the aforementioned and, probably, originating from the preparation.

(3) Sections from lungs of healthy guinea-pigs. These showed no needles identical with those described above.

Finally, kaolin powder was examined. It contained numerous needles which showed marked similarity to those demonstrated in the lungs of our case—in histological sections as well as in lung tissue treated after the Jones method and in incinerated lung.

Therefore we conclude that our fatal case was one of typical uncomplicated silicosis. The autopsy revealed no other condition which might have caused death. On the other hand, the changes in the lungs were so extensive and advanced that it is surprising that the man was able to go about with such lungs, as the lung tissue capable of respiratory function must have been reduced greatly in amount. The changes found in the heart are to be ascribed to the impaired circulation of the blood in the silicotic lungs—a condition involved in most cases of marked silicosis.

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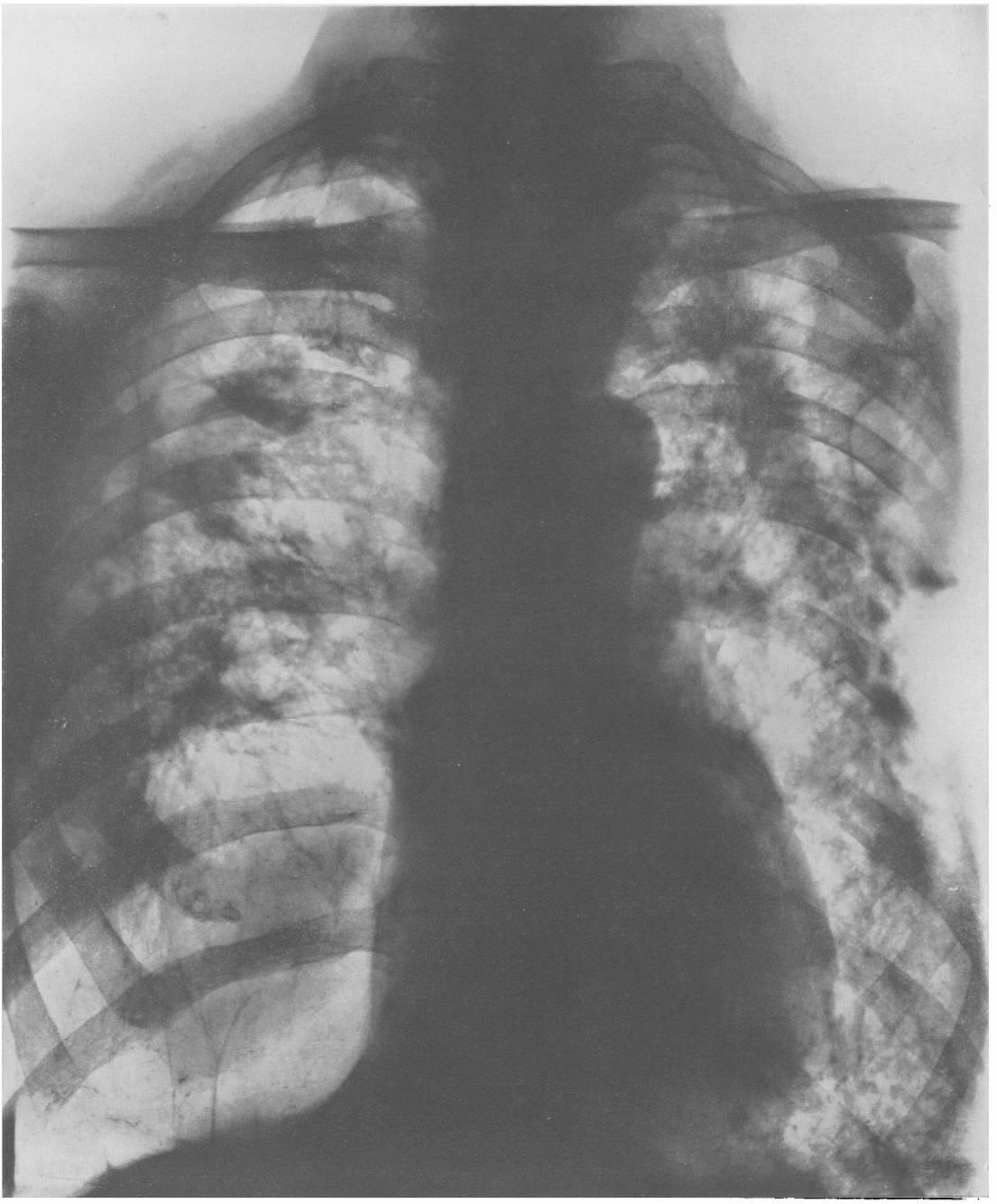

Fig. 1

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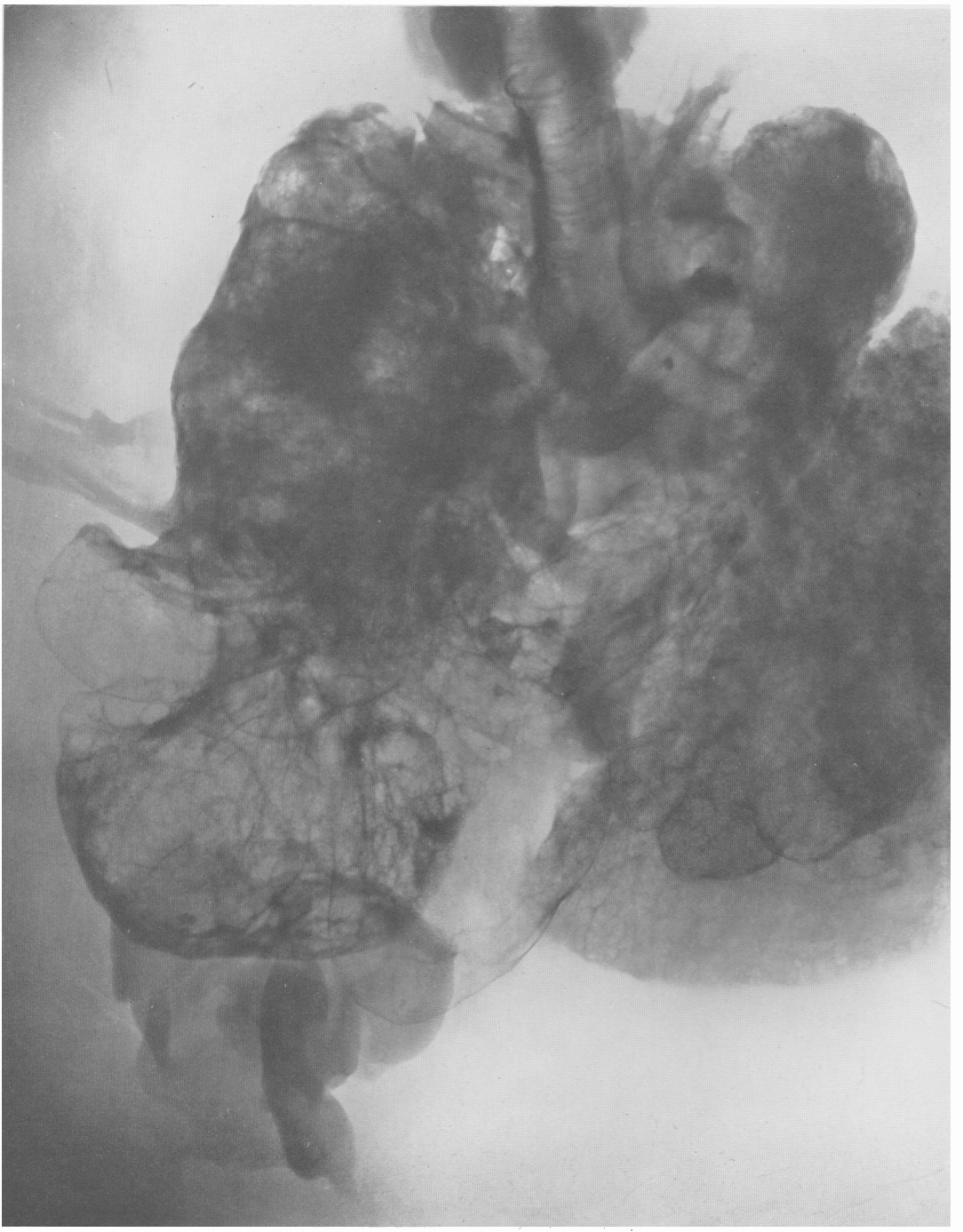

Fig. 2

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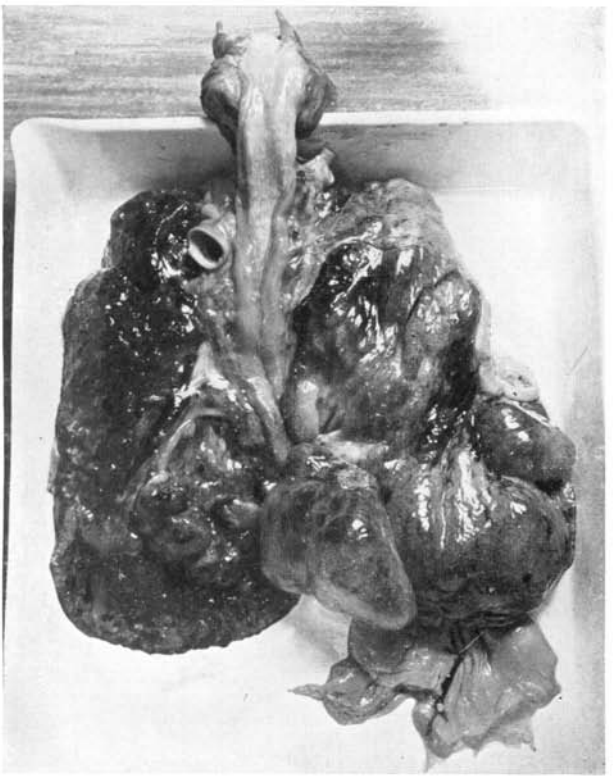

Fig. 3

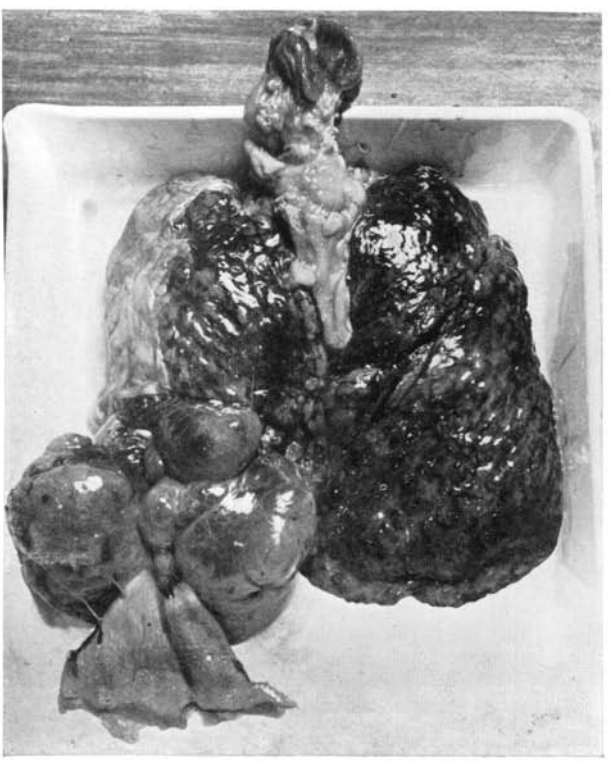

Fig. 4

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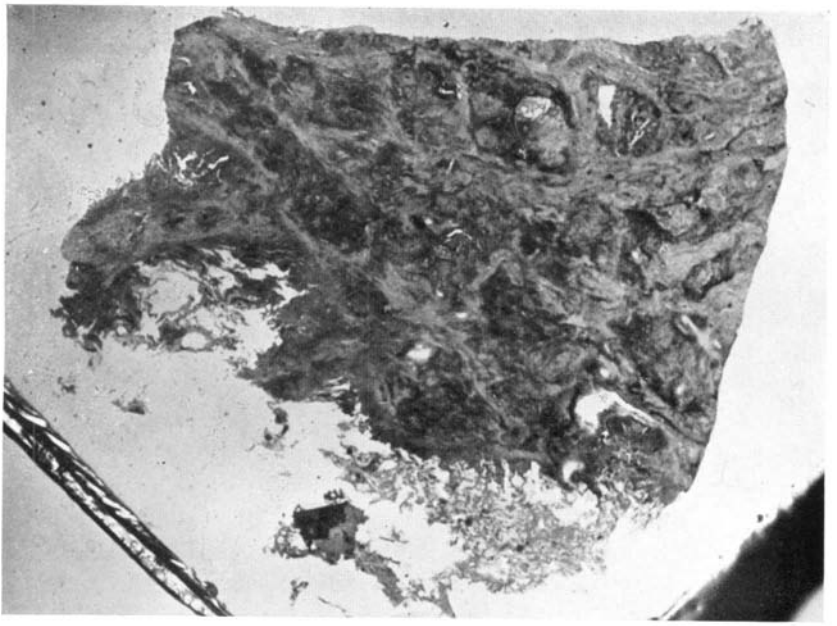

Fig. 5

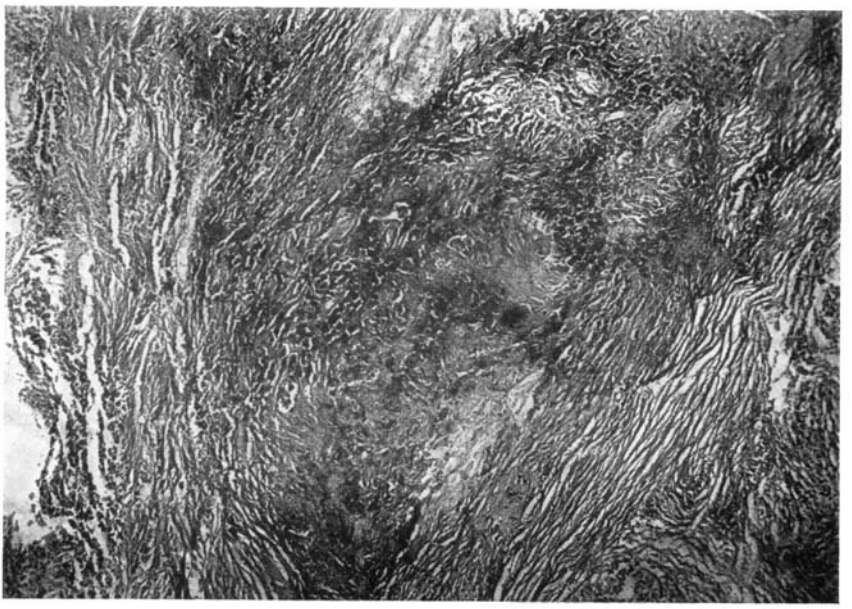

Fig. 6

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It is certain that the dust this man inhaled for years contained much silicic acid. In the factory where he worked, the dust on the shelves and stored chinaware had been previously analysed for silicic acid, and found to contain 50·6 per cent. thereof (Lind, 1933). Besides, as is well known, the crude materials used in manufacture of porcelain contain this substance in large quantities.

Yet, examination of the lungs showed that they were filled with needles of sericite or needles from kaolin, and we find it most probable that these needles constitute the main cause of the fibrosis in the lungs. Thus we are able to confirm the findings of Jones as far as fibrous minerals in silicotic lungs are concerned.

It seems to us that if Jones' theory is correct, all fibrous minerals are probably capable of the same effect as sericite; and, indeed, there are many minerals which occur in this form.

SUMMARY

1. A typical uncomplicated case of silicosis with fatal outcome in a porcelain turner is described.

2. Microscopic examination of lung tissue and of ashes therefrom showed numerous mineral needles resembling those found in kaolin from his factory.

3. The findings in this case are taken to support in part the theory advanced by W. R. Jones, that silicosis can be produced by fibrous minerals.

We are indebted to Mr Bögvad, M.S., for valuable aid in the mineralogical diagnosis.

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EXPLANATION OF PLATES I–III

PLATE I

Fig. 1. X-ray photograph taken of the patient on July 12th, 1932.

Fig. 2. X-ray photograph of the lungs removed at autopsy.

PLATE II

Fig. 3. Posterior view of the lungs with oesophagus laid open.

Fig. 4. Anterior view of the lungs.

PLATE III

Fig. 5. Photomicrograph of section of lung (× 10).

Fig. 6. Photomicrograph of a firm nodular mass in lung.

(For detailed explanation see text, pp. 167 and 168.)

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