THE

DISEASES OF OCCUPATION

By

Donald Hunter

London

English University Press

1955.

Description of disease or injury.	Nature of occupation.
38. Tuberculosis (*cont.*)	*Any occupation involving:* (*b*) in attendance upon a person or persons suffering from tuberculosis, where the need for such attendance arises by reason of physical or mental infirmity; (*c*) as a research worker engaged in research in connexion with tuberculosis; (*d*) as a laboratory worker, pathologist or post-mortem worker where the occupation involves working with material which is a source of tuberculous infection, or in an occupation ancillary to such employment.

First Schedule, Part 2

Pneumoconiosis. Fibrosis of the lungs due to silica dust, asbestos dust or other dust, and including the condition of the lungs known as dust reticulation.

1. Any occupation involving—
   (*a*) the mining, quarrying or working of silica rock or the working of dried quartzose sand or any dry deposit or dry residue of silica or any dry admixture containing such materials (including any occupation in which any of the aforesaid operations are carried out incidentally to the mining or quarrying of other minerals or to the manufacture of articles containing crushed or ground silica rock);
   (*b*) the handling of any of the materials specified in the foregoing subparagraph in or incidental to any of the operations mentioned therein, or substantial exposure to the dust arising from such operations.

2. Any occupation involving the breaking, crushing or grinding of flint or the working or handling of broken, crushed or ground flint or materials containing such flint, or substantial exposure to the dust arising from any of such operations.

3. Any occupation involving sand blasting by means of compressed air with the use of quartzose sand or crushed silica rock or flint, or substantial exposure to the dust arising from such sand blasting.

4. Any occupation involving—
   (*a*) the freeing of steel castings from adherent siliceous substances;
   (*b*) the blasting of metal castings to free them from adherent sil

ceous substance by means of any abrasive, by a blast of compressed air, by steam or by a wheel;

(c) the moulding of iron castings with the use of siliceous materials as a facing powder or parting powder;

(d) substantial exposure to the dust arising from any of the foregoing operations.

5. Any occupation in or incidental to the manufacture of china or earthenware (including sanitary earthenware, electrical earthenware and earthenware tiles), and any occupation involving substantial exposure to the dust arising therefrom.

6. Any occupation involving the grinding of mineral graphite, or substantial exposure to the dust arising from such grinding.

7. Any occupation involving the dressing of granite or any igneous rock by masons or the crushing of such materials, or substantial exposure to the dust arising from such operations.

8. Any occupation involving the use, or preparation for use, of a grindstone, or substantial exposure to the dust arising therefrom.

9. Any occupation involving—

(a) the working or handling of asbestos or any admixture of asbestos;

(b) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos;

(c) the cleaning of any machinery or plant used in any of the foregoing operations and of any chambers, fixtures and appliances for the collection of asbestos dust;

(d) substantial exposure to the dust arising from any of the foregoing operations.

10. Any occupation involving—

(a) work underground in any coal, tin, slate or hæmatite iron-ore mine;

(b) the working or handling above ground at any coal or tin mine of any minerals extracted therefrom, or any operation incidental thereto;

(c) the trimming of coal in any ship, barge or lighter, or in any dock or harbour or at any wharf or quay;

(d) the sawing, splitting or dressing of slate, or any operation incidental thereto.

Byssinosis. Employment for a period or periods amounting in the aggregate to not less than twenty years, in any occupation in any room where any process up to and including the carding process is performed in factories in which the spinning or manipulation of raw or waste cotton is carried on.

Health and Welfare of the Miner

In contrast with the early years of the nineteenth century, when it was far from being admitted that the State had either a right or a duty to inter-

FIG. 338.—Patients with Silicosis at Timmins, Ontario, breathing Aluminium Powder from an Experimental Treatment Chamber
(By courtesy of Dr. H. W. Jacob)

ASBESTOS AND OTHER FIBROUS SILICATES

The question as to whether silicates are toxic when inhaled has been much debated since Jones (1933) put forward the view that sericite, a potassium aluminium silicate, is the cause of much industrial silicosis. His view has received but little support. Certain silicates are noxious, and the chief of these is the group of fibrous mineral silicates known as asbestos. To a less extent talc produces a fibrous reaction in the lungs; sillimanite and glass wool seem to be harmless.

(*a*) Asbestos

Asbestos is the name given to a series of minerals composed of silicates of magnesium and iron. It is sometimes referred to as earth flax, stone flax or mountain cork. It is silky and fibrous in form, and this fibrous structure determines many of the characteristics of *asbestosis*, the disease caused by inhalation of the dust. Asbestosis is a more serious disease even than silicosis, but it is far less prevalent (Stewart, 1947).

History of Asbestos

On a small scale, asbestos was made into cloth at least 2,000 years ago. Herodotus (450 B.C.) relates how the Romans mined it in the Italian Alps and Ural Mountains, using it for enshrouding corpses before cremation to

permit of easy collection of the ashes for burial. They named it *amianthus*, meaning without miasma, undefiled or incorruptible. Pliny (A.D. 50) refers not only to difficulties in the weaving of asbestos, but also to the use of respirators to avoid the inhalation of dust. Strabo (30 B.C.) and Plutarch (A.D. 70) refer to the wicks of the lamps of the vestal virgins as *asberta*, the unquenchable, inextinguishable or inconsumable. It is stated that Charlemagne possessed a tablecloth made of it, which was cleansed by passage through fire. However, although the occurrence of asbestos is widespread throughout the world, it is only since about 1890 that it has been used for industrial purposes. In 1880 the annual world production was only 500 tons, a figure which had risen to 1,300,000 tons by 1951.

Sources and Varieties

Canada produces 65 per cent of the world's asbestos. The remainder comes from Italy, Cyprus, Finland, Russia, South Africa, Australia, New Zealand, the United States of America and Peru. The term asbestos is a collective name applied in commerce to a variety of silicate minerals which differ from each other in chemical composition and physical properties, but resemble one another in their finely fibrous nature and flexibility. Their value depends on the facility with which they can be split into long and short flexible fibres, their resistance to acids and their insulating properties with respect to heat and electricity. Most of the asbestos in commerce is fibrous serpentine, especially chrysotile, $3MgO.2SiO_2.2H_2O$. This has the longest and strongest fibres and can be spun. Asbestos is derived also from the hornblende (amphibole) group of minerals, of which the most important are crocidolite and amosite. These consist of fibres too short for spinning, but more stable chemically and more resistant to acids and heat than chrysotile. The hornblende varieties contain less magnesium and usually more aluminium and iron, crocidolite being mainly a silicate of iron, $NaFe(SiO_3)_2FeSiO_3$. It is also known as *blue asbestos*, in allusion to its prevalent colour, and as *Cape blue*, because formerly it was mined only in Cape Province, South Africa. Amosite, $(MgFe)O.SiO_2$, which occurs in workable quantities only in the Union of South Africa, is named from the initial letters of Asbestos Mine of South Africa, the title of the mine where it was first worked. Other varieties are actinolite, $CaO.3(MgFe)O.4SiO_2$, and tremolite, $CaO.3MgO.4SiO_2$.

Uses

Capable of being spun into yarn and woven into cloth or of mixing with cement, rubber, resin, plastics or graphite, it provides the engineering, electrical, building, chemical and other trades with a variety of useful products. These include a wide range of insulation products, as well as flat and corrugated cement sheets for building work, and also roofing shingles, cements, tiles and fireproof paints. In brake linings and clutch facings the asbestos is usually reinforced with wire threads. Asbestos is made into acid-resisting filtering cloth, linings for chemical pans, valve packings,

gaskets, jointings and lagging material for steam pipes, and is used for coating the bulkheads of ships and marine piers. Long-fibred asbestos, which can be spun and woven into textiles, is used for fire-fighting suits, firemen's helmets, sheets and ropes, theatre safety curtains, boiler mattresses and for the coverings of welding rods where it acts as a flux.

Mining, Crushing and Disintegrating

Asbestos is mined both in open pits and by underground methods. After blasting, the longest asbestos fibres are separated from the mother rock by cobbers. These men are exposed to much dust because they do

FIG. 389.—Stripping an Asbestos-carding Machine before a modern type of Exhaust Ventilation was applied locally
(By courtesy of British Belting and Asbestos, Ltd.)

their work by hand with a cobbing hammer. Raw asbestos in crude and fibre form is imported into Great Britain and stored in sacks in open sheds. The sacks are carried to a rotary mill and opened, two or three at a time, by hand. This process is dusty. The material then passes by vacuum suction into a closed-in machine which delivers flocculent, soft, fibrous material, free from grit.

Carding and Card Cleaning

For the manufacture of yarn and cloth, cotton is often incorporated with the fibre before carding. The carding machine consists of large, fast-running cylinders, covered with innumerable minute wire points to ensure

that the
is receiv
and clear

Spinning

Spinn
weaving
rusting ce
boards, a
cement ar
secure the
becoming
out upon
room. Sul
polishing,
entirely by
asbestos b
sewing.

Historical

The da
light with
tosis know
Murray in
years and w
room, the o
He died in
revealed ex
found to co
phthisis. Di
asbestosis co
Cooke (192
seemed to e
pulmonary fi
Merewether
have been m
and others,

Thirty Case

Between
Out-patient D
and twelve w
asbestosis. Se
asbestosis was
in more than
cupations in

that the fibres are laid parallel. From the end of this machine the material is received as a sliver or rope. Stripping the carding machines (fig. 389) and cleaning the cylinders with a wire brush is a very dusty occupation.

Spinning and Weaving

Spinning, doubling and weaving follow the process of carding. Dry weaving is usually practised, for wet weaving has the disadvantage of rusting certain fittings on the machinery. For non-textiles, such as millboards, a wet process is employed. Fibred asbestos is mixed with clay, cement and water, and run through a series of sedimentation tanks to secure the finest of the pulp. This passes along the machine as an emulsion, becoming drier and more doughy on its way, until it is eventually rolled out upon a cylinder. From this it is cut, removed and dried in the drying-room. Subsequent operations, such as sawing, grinding, turning and polishing, give rise to much dust. Insulating mattresses are made almost entirely by hand from lengths of asbestos cloth, and are filled with fibred asbestos by hand, scoop or shovel, levelled by beating and finished by sewing.

Historical Summary

The dangers of asbestos remained for long unsuspected. They came to light with a somewhat disconcerting suddenness. The first case of asbestosis known in Great Britain was observed in 1900 and described by Murray in 1907. The man had worked on a carding machine for fourteen years and was the last survivor of ten men who had worked in the card room, the others all having died about the age of thirty with lung disease. He died in Charing Cross Hospital at the age of thirty-four, and necropsy revealed extensive diffuse pulmonary fibrosis. Although the lungs were found to contain asbestosis bodies, a diagnosis was made of typical fibroid phthisis. Difficulties of certification delayed further action and cases of asbestosis continued to occur. Real attention was drawn to the disease by Cooke (1924). But it was the case published by Seiler in 1928 which seemed to establish an unequivocal relationship between asbestos and pulmonary fibrosis which precipitated the inquiry leading to the Report of Merewether and Price (1930). Reports of a heavy incidence of asbestosis have been made in some German factories, especially in Dresden (Krüger and others, 1931) and Berlin (Gerbis and Ucko, 1932).

Thirty Cases in One Factory

Between 1930 and 1934 thirty-seven workers in asbestos attended the Out-patient Department of the London Hospital; twenty-five were women and twelve were men. Of these, thirty were judged to be suffering from asbestosis. Seven of these died; and in four necropsy showed that the asbestosis was complicated by pulmonary tuberculosis. Many had worked in more than one department of the same factory, but the principal occupations in order of frequency were as follows: spinner, weaver, dis-

integrator, mixer, mattress maker, card cleaner, storekeeper, slab maker, presser and machinist. The average length of exposure before the onset of symptoms was seven years. Of the patients who died, one was a weaver who had been exposed for eighteen months and another a storekeeper who had been at work for nineteen years. In many cases there was a long interval between removal from exposure and the onset of symptoms: the longest such interval was eight years.

Symptoms

The presenting symptom was dyspnœa. This was early, conspicuous and always out of proportion to the signs found in the chest. It was nearly always associated with non-productive cough, as many as eighteen cases having little or no sputum. Hæmoptysis never occurred in the absence of pulmonary tuberculosis: it was found in three cases. In sixteen cases pain occurred between the shoulders, under the scapula or behind the sternum; it was never more than slight. In two cases a diagnosis of acute pleurisy had been made during the period of employment in asbestos, but in no case had a pleural effusion occurred. A history of warts was obtained in thirteen cases, of laryngitis in three and of conjunctivitis in two.

Physical Signs

The patients were usually well nourished. Clubbing of the fingers was present in six cases. Cyanosis was occasionally noticed, but was only slight. Examination of the chest showed limitation of the respiratory excursion on both sides. The percussion note was never seriously impaired, but slight dullness was sometimes found at the bases. The breath sounds were vesicular, and auscultation sometimes revealed scattered, medium, crepitant râles, often with a metallic tone, and situated especially at the bases. Added sounds were absent on repeated examination in ten cases. For obvious reasons the condition is not unilateral, and therefore displacement of the heart and other mediastinal structures was not found.

Asbestosis Bodies in the Sputum

Thirteen cases showed asbestosis bodies in the sputum. These are golden-yellow structures of elongated bead-like form, often with bulbous ends (Cooke and Hill, 1927). The appearance of fully formed bodies has been compared to beads on a necklace. The beads vary in size and make up an irregularly segmented body. These bodies have been found to vary in length from 20 to over 200 microns. An asbestos fibre forms the central core in each body (Gloyne, 1929). They are best seen with an oil immersion lens, and show up clearly without staining as golden-yellow structures (fig. 390). They can be stained by hæmatoxylin. The tubercle bacillus was found in the sputum of five of the above thirty cases, and four of these died.

Radiographic Appearances

The fibrosis in asbestosis is much more diffuse than that due to silica. In silicosis it occurs in the form of numerous more or less isolated foci, but

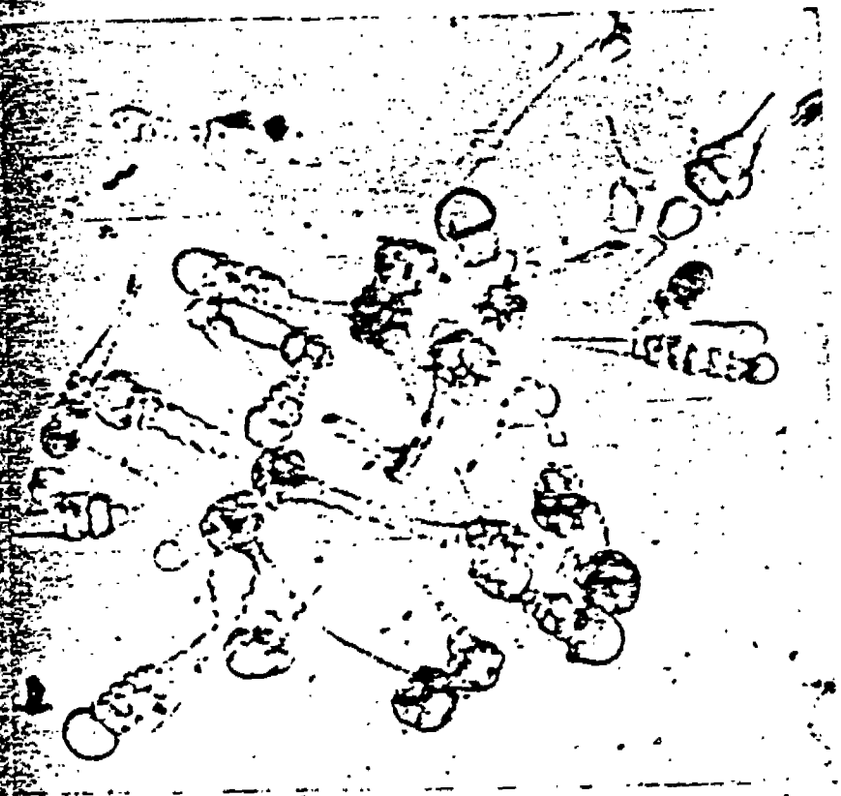

FIG. 390.—Asbestosis Bodies in the Sputum magnified 1,000 diameters

in asbestosis as a fine network throughout the lungs. In radiographs it is so fine as to resemble ground glass or fine cobweb (plate XVI). Homogeneous opacities of this nature are seen at both bases, and may later become more obvious as a fine punctate stippling. The inter-lobar pleura is usually thickened, and there is a shaggy outline to the heart shadow. Ultimately the fine punctate mottling can be observed to have spread beyond the limits of the lower zone to the middle and even the upper zone, but the apices usually remain free. This fine, diffuse, punctate mottling stands out in definite contrast to the nodulation seen in silicosis. In fact, in advanced asbestosis the degree of shadowing present would have little significance if seen in silicosis (Timmermans, 1931).

Prognosis

Broadly speaking, the length of time which may elapse between exposure to the dust and a fatal termination is only one-half of that in silicosis. In other words, in asbestosis the fibrosis develops more rapidly. With continued exposure to high concentrations of dust, the fibrosis may be

fully developed in seven years, and may cause death after about twelve years' exposure as compared with thirty-five years for all cases of silicosis. The shortest length of exposure to asbestos dust which has ultimately caused death is eighteen months. It must be remembered that the existence of a clinically recognizable degree of asbestos fibrosis in any individual is an additional adverse factor in the prognosis of any acute infection of the lungs such as pneumonia or bronchopneumonia.

Pathology of Asbestosis

At necropsy pleural adhesions are extensive and dense, and there are often thick subpleural plaques of fibrous tissue. Large areas of the lung are tough owing to the presence of fibrous tissue; emphysema is extensive but usually localized to the lower and apical parts of the lungs. There are two views regarding the mode of development of asbestosis. According to the one, the action of the dust is chemical; the other view supposes that the asbestos fibres act mechanically. Experiments carried out at the Saranac laboratories do not support the chemical theory, since aluminium hydroxide which neutralizes the toxic action of quartz does not prevent asbestosis. Moreover, asbestos fibres shorter than 20 microns were found to be relatively innocuous, whereas if the action were chemical an increase in potency would have been expected with reduction in particle size below 5 microns, as in the case of silica. A comparison of the effect of asbestos fibres 15 microns long with those 2·5 microns long (King and others, 1946) showed that rabbits receiving the long fibres developed a nodular reticulin formation comparable with experimental silicotic nodules; animals receiving the short fibres developed a diffuse interstitial reticulin formation.

Effect of the Size of the Fibre

The localization of the asbestos lesions appeared to depend on the size of the fibres; with long fibres, too big for removal by phagocytes, the reaction resulted in the formation of connective tissue within the alveoli; with shorter fibres capable of being taken into the alveolar walls, there was diffuse interstitial fibrosis. These workers suggest that if the fibres were shorter than 2·5 microns they would be completely removed from the alveolar walls and fail to produce fibrosis. Johnstone (1948) concluded from clinical investigation that workers exposed to high concentrations of extremely fine asbestos dust did not suffer from asbestosis. The Saranac workers conclude that asbestosis is caused by mechanical irritation from the asbestos fibres during the movements of respiration, and that this peculiarity is related to the flexibility of the fibres not possessed by other foreign bodies. The view that movement of the lungs is a factor is supported by the absence of reaction to asbestos in organs other than the lung. Wyers (1949) points out that if the fibrous character is absent, as in the case of serpentine, the chemical equivalent of asbestos, the dust is inert.

Incidence of Associated Pulmonary Tuberculosis

Precise information as to the incidence of pulmonary tuberculosis amongst asbestos workers cannot be obtained, but the Factory Department figures for 1935 suggest a less close association between asbestosis and tuberculosis than is the case with silicosis and tuberculosis. Thus among 702 deaths from silicosis the disease was accompanied by tuberculosis in 56 per cent, whereas among eighty-two deaths from asbestosis only 36 per cent showed associated tuberculosis.

Association with Carcinoma of the Lung

In an analysis of 235 cases of asbestosis by the Factory Department (1947), cancer of the lung or pleura was found to occur in thirty-one (13·2 per cent, average age 52·1 years). These figures were contrasted with the much lower incidence of cancer of the lung in 6,884 cases of silicosis, of whom 1·32 per cent had carcinoma (average age 59·4 years). Usually the carcinoma appears many years after exposure to asbestos has ceased (see case 38, p. 882).

Preventive Treatment

In asbestos mining wet rock drilling is imperative. In Great Britain legislation has been effective in controlling the disease. In factories and textile mills the principle of locally applied exhaust ventilation must be enforced to prevent the escape of asbestos dust into the air of any room in which work is done. Hand cleaning of the cylinders of carding machines is prohibited while any other person is present, nor may it be done by hand strickles or other hand tools. Those cleaning cylinders, filling insulating mattresses and entering chambers containing loose asbestos should be provided with an efficient apparatus by means of which they can breathe air free from dust. Suppression of dust in the cleaning of carding machines is best ensured by the use of a revolving brush fitted with a cover and connected to a portable vacuum cleaner (fig. 391). By the use of closed-in machines, handling should be reduced to a minimum. In one factory, spinning frames have been totally enclosed in *perspex* with an ingenious device which permits easy access to bobbins (Wyers, 1949). Education of the worker must aim at a high standard of good housekeeping.

Case 37. Asbestos spinner—exposure to asbestos dust for five years—asbestosis.

A. R., a woman, aged 27, had worked from 1924 to 1929 in the spinning department of an asbestos works where the protection from dust was poor. In 1928 she suffered from a dry cough, and in 1930 from shortness of breath and occasional pain in the right side of the chest on breathing.

On examination: five warts on fingers, the largest 3 by 1 mm. No clubbing of fingers. Dyspnœa, but no cyanosis. Apex beat not displaced, and heart sounds normal. There was diminished movement of the base of the right lung, with slight dullness, diminished breath

FIG. 391.—Portable Vacuum Apparatus for cleaning Asbestos Carding Machine. Note the Revolving Brush fitted with a Cover and connected to a Vacuum Cleaner
(Report on Conferences concerning Methods for Suppressing Dust in Asbestos Textile Factories, 1931)

sounds and extensive creaking pleuritic sounds. A few crepitations at both bases. No sputum. X-ray showed the heart to be normally situated, and there were fine homogeneous opacities resembling ground glass at the bases of both lungs. In 1935 the shortness of breath was worse; there was a dry cough. On examination there were no warts on the fingers, and the physical signs in the chest were unchanged.

Case 38. Asbestos carder and weaver—exposure to asbestos dust for three years—latent interval of twenty-five years—death from compression paraplegia, secondary carcinoma of spine; primary carcinoma of lung; asbestosis.

M. C., a married woman, aged 43, L.H. Reg. No. 41149/1947.

1919–22: between the ages of fifteen and eighteen she worked as carder and weaver in an asbestos factory. It is not known whether she cleaned the carding machines with a steel handbrush, but this method was in use at the time. She gave up the work owing to shortness of breath.

25 years: she had frequent brief attacks of breathlessness sometimes with cough but was not disabled.

16 years: at the age of twenty-seven she married, and later had one miscarriage but no further pregnancy. She remained active, doing all the duties of a housewife.

Four months: sudden onset of aching pain in the back of the neck without any apparent precipitating factor. The pain radiated to both shoulders and to both sides of the chest, was continuous and severe enough to interfere with sleep. It remained for some time practically unchanged, but had gradually increased in severity during six weeks.

Three weeks: she felt unsteady on getting out of bed in the morning and since then she had become progressively weaker and more unsteady on her legs. There were occasional involuntary movements of the legs, usually flexion of the knees with plantar flexion of the ankles. Occasional paræsthesiæ of the legs. Hesitancy of micturition. Bowels open regularly.

On examination: temperature 99° F. Pulse 112. Respiration 22. A nervous distressed woman, in considerable pain. Furred moist tongue. No pallor. No enlarged lymph glands or œdema. Regular pulse. Apex beat 1 inch lateral to mid-clavicular line. Blood-pressure 120/90. Heart sounds normal. There was some impairment of resonance and diminution of breath sounds with coarse crepitations at the bases of both lungs. The abdomen, including digital examination of rectum and vagina, showed nothing abnormal. Nervous system: an anxious but co-operative lady. Cranial nerves normal. Upper limbs normal. Lower limbs—approximately symmetrical spastic paralysis with brisk reflexes, ankle clonus and extensor plantar responses. Abdominal reflexes absent, and some weakness of lower trunk musculature. Loss of sensation to pin-prick, postural sense and vibration up to the level of D6.

Investigations: catheter urine: trace of albumin; deposit; calcium oxalate crystals; culture sterile. Blood count: hæmoglobin 89 per cent, red blood cells 4,720,000 per c.mm., colour index 0·94, platelets 255,000 per c.mm., white blood cells and differential count normal. Cerebrospinal fluid: clear, colourless fluid, under normal pressure, rising unevenly on jugular compression to 300 mm., and falling similarly; no increase in cells, protein 500 mgm. per cent, Lange 111,211, Wassermann reaction negative. X-ray of chest: lung markings heavy throughout, excessive fine interlacing shadows at bases, no nodulation. X-ray of spine—the body of D_4 is smaller than its neighbours, especially in vertical height, and is mottled. The pedicles are not visible.

Progress: while in hospital the pain continued without much alteration. In the middle of the second week she suddenly developed severe pain in the left chest, became dyspnœic, collapsed, pale and cyanosed. Some relief was obtained from morphine and oxygen. This was followed by fever up to 101·8° F., with rapid respiration rate and two further attacks of chest pain. Electrocardiogram at this time showed absent Q and inverted T in lead 3 with right axis deviation, the rate being 180. She became steadily weaker and died 2½ weeks after admission.

Necropsy: Professor Dorothy Russell made the necropsy (L.H.P.M. 282/1947), of which the following is a summary.

Secondary carcinoma of thoracic vertebræ invading epidural space. Primary carcinoma of lung. Asbestosis.

Dark greyish-black reticular fibrosis throughout both lungs, especially lower lobes and right middle lobe, with conspicuous subpleural fibrosis (0·5 cm. deep). Area (3 × 3 × 2 cm.) of pale grey carcinoma beneath pleura of mesial aspect of left upper lobe. Clear yellow pleural effusion, left (3 oz.). A few fibrous pleural adhesions over posterior surface of left lung and mesial aspect of right upper lobe. Secondary carcinoma of hilar glands, most marked right, and one in superior mediastinum. Secondary carcinoma of greater part of bodies of 2nd to 4th thoracic vertebræ. Collar of growth encircling dura over 4th and 5th thoracic segments. Patchy softening of spinal cord at this level. Nodule (1 cm. diam.) of secondary carcinoma in mesial surface of left parietal cortex. Secondary carcinoma in supraclavicular glands. Swollen red pulp in spleen. A well-nourished, edentulous woman.

Microscopic. Lungs: conspicuous anastomosing bands of dense fibrosis containing numerous asbestosis bodies, often in clumps, and associated with foreign body giant-cells. Similar zone beneath pleura of lower lobes; less marked and interrupted over middle lobe. Congestion and emphysema of residual lung tissue; many macrophages and occasional asbestosis bodies in alveolar spaces. *Left upper lobe:* similar changes, together with ill-defined, infiltrating tubular, columnar-celled and polygonal trabecular carcinoma beneath mesial pleura, apparently arising from bronchus. *Gland from right hilum:* focus of secondary polygonal-celled carcinoma; anthracosis and a few collections of asbestosis bodies. *Right and left supraclavicular glands:* extensive secondary carcinoma. *Fifth thoracic segment with dura:* extensive secondary carcinoma of epidural tissues, not penetrating dura. Permeation of many veins. Engorgement of pial veins. Ill-defined wedges of ischæmic softening in both lateral columns of spinal cord extending into crossed pyramidal tracts, and a few smaller similar foci in anterior and posterior columns. *Thoracic vertebræ 2–3 with intervertebral disc:* extensive osteoclastic and osteoplastic secondary carcinoma of spongiosa and periosteum. Considerable necrosis of medullary tissue. *Left parietal cerebrum:* nodule of secondary carcinoma in superficial cortex. *Spleen:* slight myeloid reaction in pulp.

(*b*) Talc

Talc is a hydrated magnesium silicate, $3MgO.4SiO_2.H_2O$. The purest deposits occurs in association with dolomite and magnesian limestone, where the talc owes its origin to the hydration of the magnesium silicate minerals which were formed during the earlier metamorphism of the carbonate