THE DISEASES OF OCCUPATIONS

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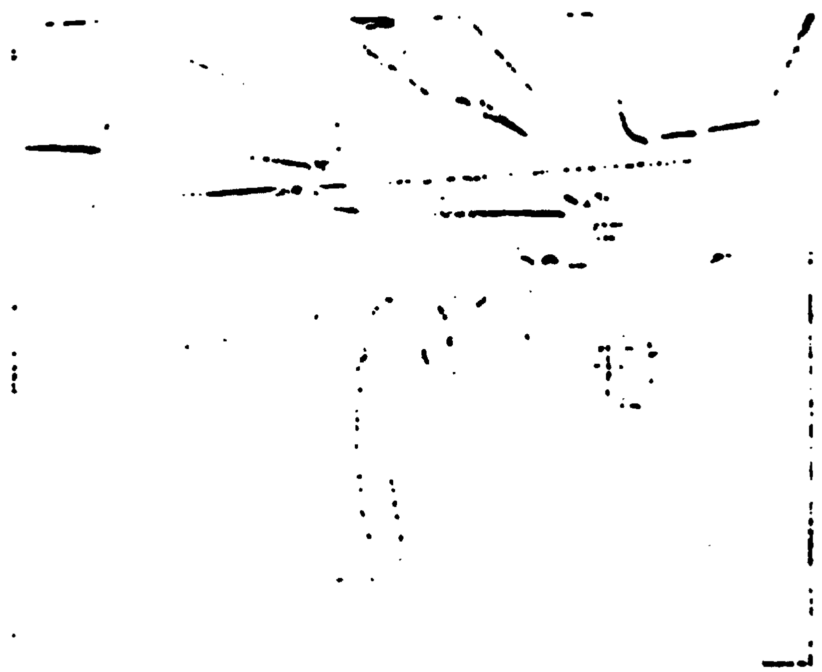

[illegible] Patients with Silicosis at Timmins, Ontario, breathing Aluminium Powder from an Experimental Treatment Chamber
(By courtesy of Dr. H. W. Jacob)

(a) Asbestos

[illegible]stos is the name given to a series of minerals composed of silicates [illegible]gnesium and iron. It is sometimes referred to as earth flax, stone flax [illegible]ntain cork. It is silky and fibrous in form, and this fibrous structure [illegible] many of the characteristics of *asbestosis*, the disease caused by [illegible]ation of the dust. Asbestosis is a more serious disease even than sili[illegible]. In addition to this asbestos workers face the risk of cancer of the [illegible] of mesothelioma of the pleura and peritoneum both of which are [illegible] fatal diseases.

History of Asbestos

On a small scale, asbestos was made into cloth at least 2,000 years ago. [illegible]dotus (450 B.C.) relates how the Romans mined it in the Italian Alps [illegible] Ural Mountains, using it for enshrouding corpses before cremation to [illegible] of easy collection of the ashes for burial. They named it *amianthus*, [illegible] without miasma, undefiled or incorruptible. Pliny (A.D. 50) refers [illegible] to difficulties in the weaving of asbestos, but also to the use of [illegible]ators to avoid the inhalation of dust. Strabo (30 B.C.) and Plutarch [illegible] refer to the wicks of the lamps of the vestal virgins as *asbesta*, the [illegible]enchable, inextinguishable or inconsumable. It is stated that Charle[illegible] possessed a tablecloth made of it, which was cleansed by passage through fire. However, although the occurrence of asbestos is widespr. throughout the world, it is only since about 1890 that it has been used industrial purposes. In 1880 the annual world production was only : tons, a figure which had risen to 200,000 tons by 1920 and nearly 3,500,c tons by 1967.

Sources and Varieties

Canada produces 65 per cent of the world's asbestos. The remain comes from Italy, Cyprus, Finland, Russia, South Africa, Australia, N. Zealand, the United States of America and Peru. The term asbestos i collective name applied in commerce to a variety of silicate minerals whi differ from each other in chemical composition and physical properti but resemble one another in their finely fibrous nature and flexibilit. Their value depends on the facility with which they can be split in long and short flexible fibres, their resistance to acids and their insulatin properties with respect to heat and electricity. Most of the asbestos commerce is fibrous serpentine, especially chrysotile, $3MgO.2SiO_2.2H_2$(This has the longest and strongest fibres and can be spun. Asbestos derived also from the hornblende (amphibole) group of minerals, of whic the most important are crocidolite and amosite. These consist of fibres to short for spinning, but more stable chemically and more resistant to aci and heat than chrysotile. The hornblende varieties contain less magnesiu: and usually more aluminium and iron, crocidolite being mainly a silica of iron, $NaFe(SiO_3)_2.FeSiO_3$. It is also known as *blue asbestos*, an allusion t its prevalent colour, and as *Cape blue*, because formerly it was mined onl in Cape Province, South Africa. Amosite, $(MgFe)O.SiO_2$, which occurs i workable quantities only in the Union of South Africa, is named from th initial letters of Asbestos Mine of South Africa, the title of the mine wher 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 automobile, shipbuilding, electrical, building, chemical and other trade with a variety of useful products. These include a wide range of insulation products for pipe lagging, as well as flat and corrugated cement sheets for building work, and also roofing shingles, cements, tiles and fireproof paints. Wool-asbestos fibre is even used in the filter pads of certain types of respirator. From modest beginnings in a small disused Austrian paper mill in 1893 the asbestos-cement industry has constantly expanded so that by 1967 it was estimated that over 100,000 people were employed in it. In 1912 an Italian engineer built the first machine for making seamless asbestos-cement pipes. In 50 years the demand for such pipes for use in domestic installations, water mains and sewerage has greatly expanded in at least 40 countries. In brake linings and clutch facings the asbestos is [illegible] wire threads. Asbestos is made into acid-resisting [illegible] for chemical pans, valve packings, gaskets, jointing [illegible] steam pipes, and is used for coating the bulkheads [illegible]. Long-fibred asbestos, which can be spun and [illegible] used for fire-fighting suits, firemen's helmets, sheets [illegible] curtains, boiler mattresses and for the coverings of [illegible] acts as a flux. Domestic uses include plastic floor [illegible] boards, oven cloths, table and cooking mats. In [illegible] being made with asbestos-asphalt for road surfacing. The [illegible] in many of these industries is highly mobile and distributed over a large number of small units. In addition there is a vast number of "do it yourself" enthusiasts who may be exposed intermittently to highly concentrated asbestos dust.

Mining

In South Africa asbestos is obtained [illegible] open cast and deep mines. Drilling and explosives are used [illegible] rock is hard and mechanical excavation when it is softer. The [illegible] milled to remove the host rock and the fibrous asbestos is partially teazed out before it is packed into bags. Where labour is cheap some of the separation of rock from fibre is done by hand with a cobbing hammer (fig. 399) usually by men but sometimes by women often accompanied by their babies. Until recently large amounts of asbestos fibre escaped into the air from the mines and mills so that not only the miners and millers but the whole population of the mining towns and the surrounding countryside have been exposed. From the air a cloud of asbestos dust can be seen over the small mining townships. It was customary to transport asbestos from the mines to the factories in jute bags. Sometimes a boy who did this using a donkey wagon would sleep on the asbestos bags during part of the journey. This exposure of a whole series of transport workers could be eliminated by using sealed impervious bags.

FIG. 399.—Cobbing hammer used in asbestos mining in Canada
(By courtesy of Canadian Johns-Manville Co., Ltd., Asbestos, (Que.))

Work in Factories

For the manufacture of asbestos textiles 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. The crushing process is dusty. The material then passes by vacuum suction into a closed-in machine which delivers flocculent, soft, fibrous material, free from grit. 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 fibres are laid parallel. From the end of this machine the material is received as a sliver or rope. Stripping the carding machines (fig. 400) and cleaning the cylinders with a wire brush was a very dusty occupation before modern types of exhaust ventilation were applied locally. 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 mill-boards, 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

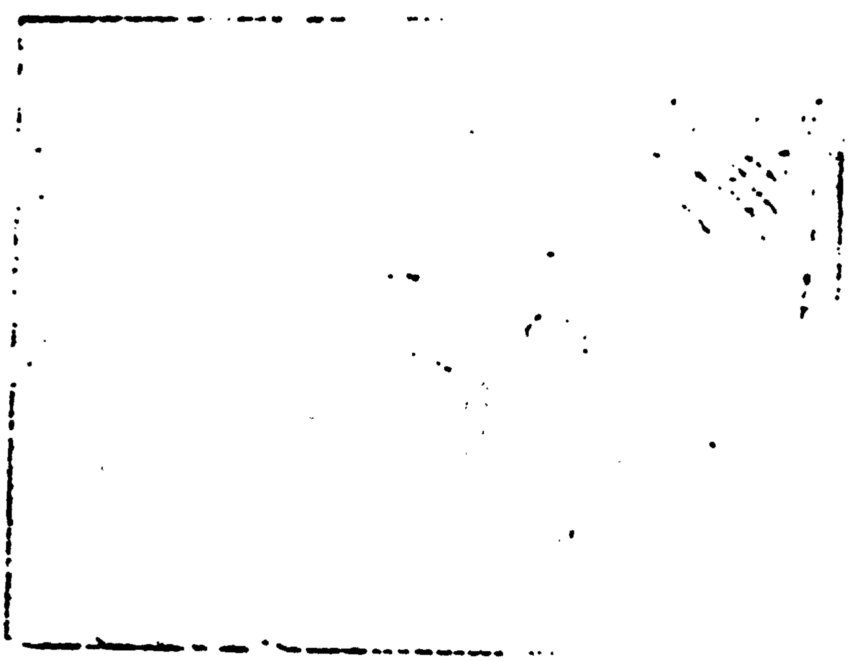

FIG. 400.—Stripping an Asbestos-carding Machine before a modern type of Exhaust Ventilation was applied locally
(By courtesy of British Belting and Asbestos, Ltd.)

[illegible] asbestos by hand, scoop or shovel, levelled by beating [illegible] sewing.

Use of Asbestos Goods

[illegible] products of asbestos factories are used in the manufacture of workshops, houses, power units, ships, cars and domestic stoves, by a variety of skilled tradesmen with their helpers. These men sometimes use powdered material which they mix with water and apply on the spot whilst the mixture is wet, or they use prefabricated blocks or mattresses which require cutting, sawing or shaping to make them fit. The materials are usually rough-finished when they leave the factory and release some dust when handled by storemen and transport workers. Hot water pipes of copper and other metals are insulated by covering them with asbestos. Steam pipes are similarly treated with asbestos, wire and cement. The job is known as pipe-lagging and it is carried out in shipbuilding, in factory construction and in private houses. At one time the asbestos was applied wet but now prefabricated materials shaped to the job are used dry, the workman sawing them up to the required lengths as he goes on with his work. It follows that the new method produces dust whereas the old method did not. Building workers, interior decorators and many others also use asbestos materials. Electricians, plumbers, fitters and painters may work in confined spaces and may create dust cutting or drilling these materials in the course of their work. Most asbestos materials are coated with paint or sandwiched between other substances so that they do not present a continuing risk until the building or installation is demolished. But asbestos roofing and brake linings unhappily cause some general atmospheric pollution, and certain uses such as the lining of ducts for air conditioning with soft asbestos, and the use of unsealed ceiling tiles cause continuous pollution of the air of office and factory buildings where people work all day.

Asbestos in Fire Protection

In 1903 the Paris Métro, as a result of a fire in which 83 people died, changed from cast iron brake blocks to asbestos-resin blocks. The fire had been started by metallic dust, causing a short circuit. Asbestos-resin brake blocks have been used by the London Underground for this and other reasons ever since. In 1932 when the *Queen Mary* was being built, mock cabins were constructed and ignited to test different materials, and it was then that asbestos-based board established its supremacy in fire protection. Today asbestos insulation board is increasingly used for compartment walls where large numbers of people congregate, for example in offices, stores, schools, hospitals and multi-storey flats. Asbestos-cement sheets, widely employed for side-cladding and roofing are weather-proof and will resist the surface spread of flame. Asbestos textiles are used for furnace fire curtains and other types of fire shield, of which the theatre fire safety curtain is one. Wherever workers are exposed to great heat or to the risk of hot metal splashes the use of protective clothing is desirable. For this purpose aluminized asbestos textile materials have been developed. In a major fire steel girders prove surprisingly vulnerable and their distortion may cause the collapse of floors. Their resistance to fire can be enhanced by treating them with asbestos spray. This forms a homogeneous coating and has the advantage of adding but little weight (Brown, 1967).

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 asbestos bodies, a diagnosis was made of typical fibroid phthisis. Difficulties of certification delayed further action and cases of asbestosis continued to occur. Special 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 were then made in some German factories, especially in Dresden (Krüger and others, 1931) and Berlin (Gerbis and Ucko, 1932). In Great Britain the report by Merewether and Price led to safety measures being adopted in factories and to the workers being kept under medical surveillance. But although improvements in dust control were effective in most of the asbestos textile factories new cases are still being diagnosed by the Panels of the Ministry of Social Security. Furthermore we now know that many people with asbestosis or mesothelioma had contact with asbestos only outside industry either through living near a mine or a factory or through dusty clothes being brought home from work. The story of mesothelioma of the pleura and peritoneum is related on p. 1019.

Thirty Cases in One London Factory

Between 1930 and 1934 thirty-seven workers in asbestos all from one factory 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, disintegrator, 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 laryngitis was obtained in three cases and of conjunctivitis in two. Warts on the fingers had been present at one time in thirteen cases and had subsequently disappeared. It is amosite, a heavier and thicker fibre than chrysotile, which may, in some operations, penetrate the skin and form a *corn* or *wart*. Histologically the lesion shows extreme keratosis (Alden and Howell, 1944).

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 dulness 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. The tubercle bacillus was found in the sputum of five of the above thirty cases, and four of these died.

Asbestos Bodies in the Sputum

Thirteen cases showed asbestos 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. 401). They can be stained by hæmatoxylin. The demonstration of asbestos bodies in the sputum has not proved to be a useful diagnostic technique

FIG. 401.—Asbestos bodies in the sputum magnified 1,000 diameters

because so many patients in industrial communities have some asbestos in their lungs which are otherwise normal. Therefore the presence of asbestos bodies in the sputum indicates that the patient has been exposed to asbestos dust at some time in the past only, and as a solitary positive finding is certainly not an indication of asbestosis. In fact, one London Hospital patient with sputum due to broncho-pneumonia apparently released asbestos bodies more readily at the height of the illness, the numbers in the sputum diminishing as the inflammatory condition resolved.

Asbestos Bodies in the Lungs at Necropsy

In a case of asbestosis necropsy may show large numbers of asbestos bodies in the lung sections both in alveolar spaces and within the thickened alveolar walls. They are readily demonstrated in smears of fluid scraped from the cut surface of the lung. In asbestosis the presence of asbestos bodies may be demonstrated in the hilar lymph nodes also. It is important to emphasize that asbestos bodies are seen more often in the absence of asbestosis. In many of these cases the number of asbestos bodies is small;

as a result they are less readily demonstrated, but by examining thick unstained sections of lung their identification is facilitated. These sections are thirty microns in thickness, being approximately six times as thick as standard sections; in addition to visualizing a greater volume of lung parenchyma there is less likelihood of asbestos bodies being knocked out of the sections when the paraffin blocks are cut. At The London Hospital it has been possible to identify asbestos bodies in the lungs in 30 to 40 per cent of routine adult necropsies. Lungs of such cases may contain only one or two asbestos bodies per section; generally there is a negative occupational history, but many of these patients have lived and worked in the East End of London for many years. Since other structures may superficially resemble asbestos bodies it is valuable to identify them with greater certainty. The heat resistant property of asbestos is utilized when sections of lung are incinerated by heating to 600° C. which effectively removes the lung parenchyma. Surprisingly the characteristic shell of the asbestos body remains intact so that these structures are still readily recognizable. In fact, since the incineration removes the carbon deposits in the lung too, many more asbestos bodies are revealed in the post-incineration specimens (Smith, 1967).

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 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).

Pleural Plaques

Pleural plaques are frequently present in asbestosis. They may be nodular or smooth and are composed of very firm, white hyaline material which may be focally calcified. They have a characteristic distribution and are usually confined to the parietal pleura. The usual sites are the central tendons of the diaphragms and in parallel lines over the ribs of the postero-lateral chest wall, but plaques may be present also behind the sternum just lateral to the mid-line. Plaques, with the same distribution, are more commonly seen in patients without asbestosis. However, examination of thick, unstained lung sections reveals small numbers of asbestos bodies in over 80 per cent of these cases. On microscopy pleural plaques are composed of hyaline, acellular fibrous tissue arranged parallel to the pleural surface; they often contain variable sized foci of calcification which when extensive may be visible on chest X-ray. Films such as these may be misinterpreted unless the possible presence of calcified plaques is borne in mind (Hourihane, Lessof and Richardson, 1966).

Respiratory Function Tests

These tests are of little value in the detection of early asbestosis in the individual case unless serial readings over previous years are available. The pattern of functional impairment includes low total lung volume, low vital capacity without evidence of airways obstruction, low pulmonary compliance and low diffusing capacity. The extent of the diffusion defect is the best estimate of disability. Williams and Hugh Jones (1960) reported abnormalities in diffusing capacity for carbon monoxide, before X-ray changes had occurred in factory workers. However other workers have found this test less sensitive than X-rays of the chest in the early detection of asbestosis.

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. Since about 1930, as conditions in the factories improved, and tuberculosis declined, the onset of asbestosis became delayed so that now it requires an average of 17 years' exposure to develop. 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 broncho-pneumonia.

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 effects 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.

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. Later, with the decline of tuberculosis, these percentage figures both for asbestosis and silicosis have become less.

Association with Carcinoma of the Lung

The first recorded case of carcinoma of the lung associated with asbestos was described by Lynch and Smith (1935). By 1955, according to Doll, a total of 61 cases had been reported. We have already seen (p. 818) that Doll (1955) reached the conclusion that asbestos workers faced a risk of cancer of the lung ten times as great as that in the general population; and that McCullough (1959) collected particulars of a series of 365 patients with asbestosis among whom 18 per cent had cancer of the lung. But the carcinogenic effects of asbestos are evidently not confined to the lungs, for Keal (1960) found a high incidence of peritoneal carcinoma in women with asbestosis. He showed that during the period 1948–58, twenty-three women were admitted to The London Hospital with asbestosis; fifteen of these had subsequently died, four with carcinoma of the lung, nine with carcinomatosis peritonei; of the latter, one was certainly ovarian in origin, and this was possibly the primary site in four others, but in the remaining two no primary site could be found.

Mesothelioma of the Pleura and Peritoneum

The idea of an association between mesothelioma and exposure to asbestos came from Cape Province, South Africa and was suggested by Wagner, Sleggs and Marchand in 1960. They found their cases in the asbestos hills west of Kimberley where communities tend to cluster round the mine workings. Schools have been built close to mounds of asbestos on which children played. There was asbestos dust in the air, in houses and in the streets. Within three years they collected [illegible] patients with mesothelioma, nearly all of whom had evidence of exposure to crocidolite often non-occupational. By contrast, there are no reported cases from the vicinity of amosite workings. They recorded 31 cases of diffuse pleural mesothelioma. Histological proof of asbestosis was present in eight of these cases. Prior to this work only occasional cases of this condition had been associated with asbestosis. In 1946 Wyers published the first case of mesothelioma of the pleura to be diagnosed in an asbestos worker in Great Britain. Up to February 1965 the total number of cases diagnosed in the British Isles was 150 (Gilson, 1966). In contrast to the average latent interval of twenty years between the first exposure to asbestos and the development of carcinoma of the lung, the interval between the first exposure and the onset of symptoms from mesothelioma may vary from between 25 and 50 years with an average of 33 years. While some patients give a story of continuous exposure to asbestos over many years, a significant proportion give a history of only transient exposure sometimes amounting to a few weeks only, many years before. Such patients may show little or no evidence of fibrosis of the lungs but do show large or small quantities of asbestos bodies in the lung parenchyma. Some of these latter cases show identical amounts of asbestos in the lung to that observed in many patients living in the East End of London. This raises the question of whether minor degrees of community contamination by asbestos are capable of giving rise to mesothelioma.

Exposure in Factory, Shipyard and Community

Hourihane (1964) examined tissues and records of 83 cases of mesothelioma from the files of the pathology department of the London Hospital. Fifty-six of these were pleural, and 27 peritoneal tumours. Occupational and residential histories were obtained from the relatives of 76 of these cases by Newhouse and Thompson (1965). Forty out of 76 gave a history of occupational or domestic exposure to asbestos. Only 9 of 76 control patients suffering from other diseases gave such a history. Among those with no evidence of occupational or domestic exposure, one-third lived within half a mile of a large asbestos factory. Seventeen cases of mesothelioma were investigated in the Merseyside area. Exposure to asbestos was confirmed in 14. Eleven patients gave clear histories of handling asbestos as laggers, boiler makers and ship repairers. Two female patients had been sorters of used sacking (Owen, 1964). Mesothelioma occurs with equal frequency in the two sexes, which is in contrast to the striking male preponderance in carcinoma of the lung. The condition is invariably fatal in months rather than years.

Symptoms and Signs of Mesothelioma

The principal symptom is dyspnoea, usually rapidly increasing, and it may be associated with pain in the chest, cough and haemoptysis. The signs are those of pleural effusion usually with complete collapse of the lung. The fluid removed is straw-coloured and sometimes bloodstained. Cytological examination shows malignant cells. Metastases are rare but they may occur as generalized lymphadenopathy, small nodules in the subcutaneous tissues over neck and chest or nodules on the tongue.

X-rays of the chest show massive thickening of the pleura and sometimes a tumour which quickly increases. The fluid of course is seen and sometimes a loculated effusion is present. Thickening of the oblique fissure may be seen. Ultimately the pleural cavity becomes obliterated. A progressively enlarging mediastinal tumour is common. Thoracoscopy usually shows tumour nodules on the thickened pleura with the lung collapsed and immobile. Thoracoscopic pleural biopsy should be used as a method of diagnosis.

Morbid Anatomy of Mesothelioma

Mesothelioma is an uncommon primary tumour of the serosal surfaces and may occur in the pleura, peritoneum or pericardium. The pleural and peritoneal tumours, either alone or together, are the most frequent, although all or any combination of the three may occur. Pleural mesotheliomas are massive white tumours which tend to surround the lung spreading to involve both the fissures and main pleural space and to cause collapse. Peritoneal tumours usually involve all surfaces of the abdominal cavity. Usually the distribution of tumour is uneven, with a main mass perhaps involving the greater omentum and with discrete or confluent nodules elsewhere. Persistently recurring effusion or ascites may occur. Lymph node metastases are common but discrete metastases within parenchymatous organs are not seen. The liver may be closely invested by tumour with distortion and even erosion of its capsule, but all the tumour seen is in continuity with that in the peritoneal cavity. Mesotheliomas tend to show a mixed histological picture consisting of epithelial patterns in some areas and spindle-cell fibromatous tumour in others.

Preventive Measures

In asbestos mining wet rock drilling is imperative. Hand cobbing should be replaced by crushing in enclosed machines. Asbestos must not be dumped carelessly about the mine workings and towns. Transport must be carried out by the use of strong impervious bags. In the factories of Great Britain legislation has been effective in controlling the severity of the disease but cases do still occur. 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. 403). 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 holders (Wyers, 1949). Education of the worker [illegible] at a high standard of good housekeeping (fig. 402). Workers [illegible] asbestos dust such as those in shipbuilding, ship-breaking [illegible] lagging and those handling asbestos as stevedores and [illegible] workers should wear efficient dust masks at such times [illegible]

DO NOT USE AIR-HOSE
TO CLEAN YOUR CLOTHES

FIG. 402.—Notice in use in an asbestos factory in Canada.
(By courtesy of Canadian Johns-Manville Co., Ltd., Asbestos, Que.)

work produces dust. Since mesothelioma seems to be associated in some cases with very small exposures to asbestos a system of control by licence of the import and use of what is probably the most dangerous form of asbestos, crocidolite or blue asbestos, has been advocated. In 1967 British Rail announced that asbestos was no longer used as insulation in railway carriages because of possible danger to men in the workshops where panelling was removed, and glass fibre was used instead. The British

FIG. 403.—Portable Vacuum Apparatus for cleaning Asbestos Carding Machine. Note the Revolving Brush fitted with a Cover and connected to a Vacuum Cleaner
(Report on Conference concerning Methods for Suppressing Dust in Asbestos Textile Factories, 1931)

Asbestos Industry has proposed that all asbestos fibres shipped to Great Britain shall be packed in dustproof bags, and that all blue asbestos shall in addition be shipped on pallets which can be handled mechanically.

Tipping of Asbestos Waste

In July 1966 at Buxworth, Derbyshire, a local enquiry by the Ministry of Housing and Local Government revealed that a firm using asbestos in the manufacture of brake linings and clutch facings had dumped its waste in disused quarries for about 40 years. At first there were no houses in the immediate vicinity of the quarry used, tipping was done from open lorries and the dust was not bagged. In July 1964 space in the first quarry had become exhausted and tipping was transferred to another quarry only 60 yards from the nearest house. By July 1966 contractors were tipping 230 tons monthly and the conditions under which this was done were totally unsatisfactory. Asbestos dust formed 25 per cent of the material dumped and although this was bagged, sometimes a bag would fall from a lorry and remain on the ground split open and uncovered. The manufacturing firm had disclaimed responsibility for the waste once it had left their gates. Although public health inspectors had visited the site regularly the nuisance persisted. From what we now know of the etiology of mesothelioma, it is clear that uncontrolled tipping of asbestos waste such as happened in the village of Buxworth should be sternly repressed.

Draft Asbestos Regulations, 1968

These long-awaited draft regulations, to supersede those of 1931, have been issued and circulated to interested parties for criticism. They will now apply to all factories, building operations, civil engineering work, shipbuilding and ship-repairing; and particularly to lagging and delagging, which are known to be dangerous operations not previously covered. Any process in which asbestos dust is liable to be given off to a dangerous extent will be carried out either under an exhaust draught which will effectively prevent the escape of asbestos dust into the air of the workplace or in some equally safe way. If this is not possible, workers must be given protective clothing and approved respiratory-protective equipment. Any person undertaking a process in which crocidolite is used will have to notify the district inspector of factories. But the Factories Act cannot protect private users. We know that inhalation of dust from such activities as sawing or sanding material containing asbestos presents the hazard of malignant mesothelioma outside as well as inside industrial situations. It seems not unreasonable, therefore, to insist that materials containing asbestos should be clearly marked to show this, so that adequate precautions can be taken by the users. For instance, amateur handymen sawing materials containing asbestos could easily wear a lightweight dust mask, and either work out of doors or in a well ventilated room. Meanwhile many doctors have asked whether the properties of asbestos are as unique as the salesmanship of the asbestos companies appears to indicate. There are many other mineral fibres such as glass wool, rock wool or slag wool, which have useful inherent fire-proofing properties but not the fibrogenic or carcinogenic effects on the lungs. Is it certain that the properties of asbestos justify the risks of asbestosis, carcinoma of the lung and mesothelioma which are entailed by its use?

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

A. P., 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. Dyspnoea, 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 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 un-

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steady 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 D4 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 asbestos 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 asbestos 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 asbestos 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 occur 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 rocks. Talc mining is carried out mainly in the United States of America and Manchuria. The largest deposit in Europe is on the northern slope of the Pyrenees.

Uses

Talc is the first mineral man encounters upon entering the world, for it is the main constituent of talcum powder and of toilet powders in general. It is soft and has a greasy feel, and on that account the massive impure substance has been called *steatite* or *soapstone*. This is the *French chalk* used by tailors for marking cloth. Perhaps 90 per cent of all the talc produced is marketed in the form of an impalpable powder. Nearly half the total output is used in the manufacture of paint and distemper. In ceramics, talc is used for the manufacture of tiles, electrical porcelain, table-ware and refractories. Nearly 25 per cent of talc production goes into the manufacture of paper in which the mineral serves for filling and glazing better than kaolin. It is widely used as a dusting agent for rubber tyres, rubber gloves, furs and for packing parachutes to prevent rotting from moisture. Workmen who handle it for such purposes often call it *pounce*.

Pneumoconiosis in Talc Mining and Milling

In New York State, disabling pneumoconiosis occurred in 14·5 per cent of the workers in the talc mining and milling industry. The dust contained fine, straight, needle-like fibres. The talc fibrosis was associated with an increased susceptibility to tuberculosis (Siegal and others, 1943). Marked radiological changes were found in nine out of thirty-four men employed in a talc mill in Norway for periods lasting from seven to twenty-four years; there was less disablement than would have been expected from the X-ray appearances (Bruusgaard and Skjelbred-Knudsen, 1949).

A Case proved by Necropsy

The first case of this type proved by necropsy to occur in Great Britain was published by McLaughlin and others in 1949. The man was fifty-one years of age and had worked in a rubber-tyre factory since he left school at fourteen years of age. The only industrial dust to which he was exposed was that of talc. For two years he had complained of increasing dyspnoea, which was partly due to heart failure arising from rheumatic carditis with aortic incompetence. The X-ray film showed nodular shadows distributed over both lung fields. At necropsy, throughout the lungs there were grey nodules which had coalesced in some places, especially in the lower lobes. Microscopically the nodules showed whorling, but this had not the typical appearance of silicosis. Within the nodules were fibre-like structures from 10 to 90 microns in length, arranged singly and in clumps; the non-fibrous particles apparently had been eliminated. Some of the fibre-like structures were similar to asbestos bodies, and the appearances suggested that talc pneumoconiosis and asbestosis are similar diseases and that the former may be caused only by the fibrous varieties of talc. Asbestos is more actively fibrogenic than talc, and this might be due to the higher proportion of fibres in asbestos.

(c) Sericite

Sericite is of geological rather than commercial importance. It is a variety of the mica known as muscovite, $K_2O.3Al_2O_3.6SiO_2.2H_2O$. It occurs as minute shreds or in very thin veins and is a secondary mineral formed by hydrothermal alteration of silicates such as the felspars. It forms acicular crystals and has been suggested as a cause of pulmonary disease by Mavrogordato (1926), Badham (1927) and Jones (1933). Since it occurs in many rocks in association with quartz it is not surprising that it has been found in the lungs of miners with silicosis. It is a residual material only and has not by itself been found to cause progressive fibrosis of the lungs, possibly because the fibrous particles are too small to have the effect caused by asbestos.

(d) Sillimanite

Until about 1920 the minerals of the sillimanite group were almost unknown outside the geological world, but now they are in great demand for making high-grade refractories. They are all aluminium silicates capable of withstanding high temperatures. Andalusite comes from California and the Transvaal; kyanite and sillimanite are quarried in India. Porcelain made from them is characterised by its high melting-point, low coefficient of expansion, resistance to shock and low electrical conductivity, properties which render the porcelain eminently suitable for sparking-plugs, laboratory ware, thermo-couple tubing and special refractory bricks used in electric and forging furnaces and cement kilns.

Sillimanite Pneumoconiosis

Sillimanite presents a fibrous crystalline form in the raw state and when calcined, but, unlike asbestos, the fibrous form is lost when it is crushed. When fused in a furnace at 1,545° C., sillimanite is converted partly or wholly into mullite. Gärtner (1947) suggested that this fact may be related to the histological changes, with the formation of small irregular nodules, found in a portion of lung of a furnace worker, and reported by Gärtner and van Marwyck (1947). In the examination of sixteen workers exposed to dust of calcined sillimanite, Henry and Middleton (1936) found radiographic changes but no disability, and no signs or symptoms which could be related to the dust exposure.

(e) Glass Wool

Fibrous glass, *fibre glass* or *glass wool*, is a manufactured silicate used in industry for sound and heat insulation, air filtration, as a filter in resin-bonded laminates, and as an inert base for textile manufacture or packing processes. Inhalation of fibre glass dust does not cause pneumoconiosis. Schepers (1959) showed in three animal species that the dust, when inhaled, behaves as a biologically inert substance. In dust produced by the machining of plastic materials containing a glass wool filler the working atmosphere may contain particles of glass wool from about 5 to 20 μ in length. These particles may be inhaled by the workmen but despite their physical similarity to asbestos particles there is no evidence that fibrosis of the lungs results or that *fibre glass bodies* similar to *asbestos bodies* are produced (Gardner, 1942). The spicules of fibre glass, or glass wool may, however, cause irritation of the skin, conjunctivae and upper respiratory tract. Implantation of splinters of glass wool in the region of the nail fold of the finger may produce paronychia sometimes with secondary malformation of the nail (McKenna, Smith and Maclean, 1958). Away from the nail fold fibres of spun glass wool may produce warts on the fingers. The particles will penetrate normal clothing but woollen underclothing may provide some protection, as may barrier creams for the exposed skin. The skin irritation disappears following a bath and a change of clothing. Glass wool does not itself cause dermatitis. However, oils used as lubricants in the manipulation of the fibre may cause oil folliculitis. Likewise in the plastics industry an epoxy resin may be added to the resins used as binders for the glass fibre filling material. Should dermatitis then occur it is caused not by the glass-fibre but by the epoxy resin.

Non-fibrous Silicates

In the absence of quartz and of fibrous structure, silicate minerals appear to produce radiographic changes accompanied by symptoms which are less severe than might be expected from the radiographic signs. The amount of change depends on the concentration of dust and the period of exposure.

(a) Mica

Micas are complex aluminium silicates combined with either potassium, magnesium or iron. The more common are muscovite, $K_2O.3Al_2O_3.6SiO_2.2H_2O$, biotite, $(KH_2)_2(MgFe)_2(AlFe)_2(SiO_4)_3$, and phlogopite, $(KH)_3Mg_3Al(SiO_4)_3$.

Sources

Muscovite, or white mica, is transparent and colourless in thin sheets, and is distinguished by its highly perfect cleavage. It is not decomposed by sulphuric acid. The name is derived from Muscovy in Russia, where the mineral was in early use as window glass. In commercial deposits the crystals vary from a few inches to a foot or so in diameter and up to two inches in thickness. They are referred to as *books* of mica because they can be cleaved into sheets almost as easily as separating the pages of a book. India supplies 70 per cent of the world total of sheet mica. Large quantities of muscovite are mined in the United States of America, but only a small percentage is of sheet-quality, the rest being used as ground mica. Phlogopite, or amber mica, is mined in Canada and Madagascar. It is difficult to differentiate from muscovite, except that it is decomposed by sulphuric acid. Biotite varies in colour, being either brown, black or dark green, and even in thin sheets, in contrast to muscovite, it has a smoky colour and is spotted with impurities. For this reason it is rarely used in sheet form but is employed as ground mica.

Uses

Owing to its transparency and heat resistance, muscovite is used for the chimneys of oil-lamps, for peep-holes in stove doors, ovens, furnaces and optical lanterns, and is then often termed isinglass stone, after isinglass, which is a semi-transparent form of gelatin. No other mineral has better cleavage, flexibility or elasticity; it is possible to roll a sheet of muscovite a thousandth of an inch in thickness into a cylinder a quarter of an inch in diameter, and its elasticity would enable the sheet to flatten out again quite