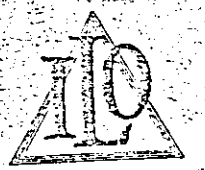
INTERNATIONAL LABOUR OFFICE

OCCUPATION AND HEALTH

Encyclopaedia of Hygiene, Pathology and Social Welfare,
Studied from the Point of View of Labour, Industry and Trades

SPECIAL SUPPLEMENT

INDUSTRIAL HEALTH IN WARTIME



MONTREAL

1944

25 cents. 1 shilling.

ASBESTOS

French: *Amiante.* — German: *Asbest.* — Italian: *Amianto.* — Spanish: *Amianto.*

Asbestos, although known to the ancients as a scientific curiosity since they were able to make a non-inflammable cloth from a substance believed to be of vegetable origin, had no commercial importance until the last quarter of the nineteenth century.

Then the unique fire-resisting and insulating properties of the mineral began to be exploited, and thereafter the expansion of the industry has been remarkable. The multifarious uses which are found for asbestos at the present day would have amazed the pioneers of the industry.

The fibrous minerals commercially known as asbestos are silicates, the silica being combined with metallic bases, mainly magnesium or iron and, to a less extent, calcium sodium or aluminium. The term is a collective name applied 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 the flexibility of the fibres.

For practical purposes, all that goes under the name asbestos in commerce is either fibrous serpentine, or a fibrous mineral of the hornblende group, of which the most important are crocidolite, amosite and tremolite. Serpentine asbestos or chrysotile is essentially a hydrated silicate of magnesium, containing little iron and almost no calcium. The hornblende varieties contain less magnesium and usually more calcium, aluminium and iron—crocidolite and amosite being mainly silicates of iron.

The *field of utility* of asbestos products has rapidly expanded and to-day is very large: new uses for asbestos are being constantly found. The mineral, the yarn, or the fabric composes, or is incorporated in, a vast number of articles, ranging from matches to filter pads, from paints to roofing tiles, from high pressure jointing to electrodes, and from brake-linings to insulating (electric, anti-noise) and fire-resisting materials in great variety.

As mentioned above, varieties and grades of asbestos differ widely in their chemical and physical qualities, and these considerations carry great weight in the choice of an appropriate grade of raw material for the purpose in view. Asbestos fibre varying in quality is of world-wide distribution, but over two-thirds of the world production of commercial fibre is derived from Canada, Rhodesia, South Africa, U.S.S.R. and Cyprus.

About four-fifths of the world's *production* of asbestos is unsuitable for spinning, and it is the discovery of industrial uses for these very short fibres and the dust-like waste which has been responsible for the phenomenal expansion of the industry as a whole.

Extraction

Hitherto asbestos has been got almost entirely by open-pit quarrying, but underground mining will have to be resorted to in increasing extent in the future in many properties.

After the mother rock containing the veins of asbestos fibre has been drilled and blasted, the loose long fibre together with adherent small pieces of rock is gathered up and sent to the cobbing shed, where, after drying, it is dressed (cobbed) by hand, freed from adherent rock, graded, screened, and then bagged ready for the market. This " crude " fibre still contains a proportion of rock, dust, and short fibre useless to the spinner, which is removed in the preparatory processes in the factory.

The broken material in the quarry after the removal of the crude fibre, where this is done, consists of rock containing the shorter fibre and still finer useful material, and barren rock. The former is sent to the mill for mechanical treatment, and the latter is dumped.

All grades of fibre other than those sold in the " crude " form, are produced mechanically in the mills. There the object is to extract as much fibre out of the rock as possible, while avoiding breaking up the fibre (and thus reducing its value) by unnecessary operations.

This is done by a series of crushing

operations, at the same time opening or fluffing out the fibre and screening out the useless sand. The fibre is collected by means of air suction at each stage, graded, cleaned and bagged.

The asbestos so produced is classified under an agreed defined system into "crude asbestos" and "milled asbestos". Crude asbestos consists of the hand-selected cross-vein material in its native or non-fibrous form. Milled asbestos consists of all grades produced by mechanical treatment of asbestos ore.

These two classes are further subdivided into nine standard groups. Crude asbestos is graded into two groups (1 and 2) and milled asbestos into seven groups (3 to 9), the determining feature being length of fibre. In the case of milled fibre further classification is made by means of the standard testing machine. This machine, of standard dimensions, comprises four boxes superimposed one on the other, the bottoms of the first three boxes from above downwards being wire screens of standard dimensions and progressively finer mesh: the lowest box retains the fine material which falls through the three screens. A weighed sample of asbestos (16 ounces) having been placed in the top box, the machine is mechanically agitated in a standardised method for exactly two minutes, and the asbestos remaining in each box is weighed. In this way the proportion of longer and shorter fibres in the sample is ascertained. The more fibre retained in the first box and the less fibre in the lowest, the higher the grade and the greater the value of the asbestos so tested.

In this way the seven groups of milled fibre are further subdivided into a number of grades. Thus in group No. 3 (spinning and textile fibres), there are seven standard grades ranging from the specification of 8—6—1—1 (minimum) for the top grade down to specification of 0—8—6—2 (minimum) for the lowest grade in this group when tested in the above way.

Asbestic or asbestine is a by-product from the mills in the nature of a residue containing a very low percentage of short fibre.

Manufacturing Processes

The asbestos, whether crude or milled, is now ready for dispatch to the factory. For textile purposes only the longer and better fibre comprised in the standard groups 1-3 (crude and spinning grades) are used.

Any of these alone, or blended with each other, or blended with cotton fibre, may be spun into yarn.

Asbestos suitable for this purpose has to be crushed, if in the crude state, and in all cases "opened" or "fiberised" before it is ready for carding. Separating (to remove iron) and sieving follow crushing, but precede opening.

The subsequent carding, doubling, spinning, and weaving processes proceed broadly as in the case of other textiles, but with essential modifications and restrictions caused by the different physical characters of the asbestos fibre.

Asbestos yarn is woven into cloth for insulation, mattress coverings, filtering material, fire curtains, fire-resisting clothing etc. Also, woven material is manufactured for belting for conveyors, brake linings and insulating tape.

Asbestos millboard and paper, and asbestos cement sheets, tiles, pipes etc. are made from short fibre mixed with other materials. Asbestos may be also made up with rubber for use as jointing, with bitumens for switchboard panelling, with synthetic resins, and with paints for a variety of purposes.

An important section of the industry is that engaged in the production of asbestos insulating materials. These include fiberised asbestos; "85 per cent. magnesia" (the remainder being asbestos fibre); mattresses made of asbestos cloth and filled with asbestos fibre, or "magnesia" or with other material; insulating compositions or plasters containing asbestos fibre and a variety of other materials, fiberised asbestos stiffened into thick sheets or moulded into shaped slabs or sections, and "air cell" insulating material built up from asbestos paper.

There are many other valuable uses of asbestos in manufacturing processes which need not be detailed here.

Risks to Health

All manipulation of asbestos fibre, by hand (as in sack filling or emptying, blending, sweeping and shovelling) or mechanically, produces dust which, if uncontrolled, is often in dangerous concentration. The inhalation of this dust over a period of time results in the development of a fibrosis of the lungs, a progressive replacement of the essential active func-

tioning tissue of the lungs by inactive and useless fibrosis or scar tissue. The disease produced, *asbestosis*, is therefore in the same category as silicosis, which it resembles in some respects while differing considerably in others. Since asbestos has been of commercial importance for scarcely fifty years, asbestosis is a modern disease. In contrast with silicosis we cannot trace the history of the baneful effects of the dust backwards over very many years.

The first recorded case of asbestosis was that of a patient of Dr. MONTAGUE MURRAY who died in 1900 in the Charing Cross Hospital, London. Post-mortem examination revealed extensive diffuse pulmonary fibrosis with no evidence of pulmonary tuberculosis. The records of this case together with the pathological specimens are still preserved. This man, aged 34 at death, had worked with asbestos for some fourteen years and stated that of the ten men working in the cardroom when he commenced, he was the only survivor, all the others having died at ages round about 30. Little more was heard for a number of years concerning the effects of asbestos dust on the lungs except isolated reports in England and France of high mortality among asbestos workers, and a note by MARCHAND and RIESAL of the presence of unusual bodies in the lungs of an asbestos worker.

This paucity of information was due to various causes. At that time the investigation and prevention of silicosis was occupying the attention both of Governments and of private investigators; the use of radiography in the diagnosis of diseases of the lungs was in its infancy; and the industry was small as regards the number of workers exposed to appreciable risk. Moreover, every new discovery emphasised the importance of silica in the free form as the predominant factor in the production of pneumonoconiosis.

Thus the real and accepted importance of free as opposed to combined silica in this respect tended to obscure the possibilities that some at least of the silicates might be equally harmful.

Although, for these reasons, enquiry in 1910-11 by the Factory Department of the British Home Office both in Great Britain and in Canada failed to produce evidence of a risk from the dust sufficient to necessitate scheduling processes in the industry as dangerous, the Department decided that suppression of the dust evolved in the more dusty processes was required. From that date, therefore, in Great Britain, active measures, although inadequate by present standards, were taken to suppress dust in the more dusty processes of the industry.

In 1924, however, the matter was raised again by the publication of a note by W. E. COOKE concerning the death of an asbestos worker, the result, in his opinion, of extensive pulmonary tuberculosis, together with a diffuse pulmonary fibrosis which he ascribed to asbestos dust. This case was fully described by COOKE and STUART MCDONALD in 1927. A few weeks later H. E. SEILER drew the attention of E. R. A. MEREWETHER to an asbestos worker in whom he had found signs of a diffuse pulmonary fibrosis with no evidence of tuberculosis, and further investigation established the absence of any infective or occupational cause other than asbestos dust.

Thereupon in February 1928 a comprehensive enquiry in Great Britain was undertaken and the results of this enquiry, which was completed in October 1928 and published in 1930, established the presence of a serious risk in the industry.

In the United States and Canada the Industrial Health Service of the Metropolitan Life Insurance Company carried out a similar survey between October 1929 and January 1931. LANZA, MACCONNELL and FEHNEL published the findings in 1935. These, together with the valuable study by FULTON, DOOLEY, MATTHEWS and HOUTZ, published in 1935, are the only general surveys of the industry.

In Italy, LOVISETTO published in 1930 the results of an enquiry, and G. MUSSA the results of clinical and radiological examinations.

Germany possesses valuable data relative to asbestosis based on clinical and laboratory research. It suffices to mention the works of BAADER, GERRIS (1931), BEGER (1933), BEINTKER (1934), ALWENS, KOPPENHÖFER (1935), etc.

ESSENTIAL FEATURES OF ASBESTOSIS

Asbestosis, the pulmonary fibrosis of asbestos workers, is insidious in its onset, irregular in its course, and variable in its mode of termination. It is helpful to visualise the disease as the slow growth of fibrous tissue (scar tissue) around the bronchioles or smaller air tubes of the lungs and between the air cells, wherever

the inhaled dust comes to rest. In contrast to silicosis the former is the important site of deposit of asbestos in the lungs, as was shown by GARDNER and CUMMINGS. While new fibrous tissue is being laid down like a spider's web, that deposited earlier gradually contracts. This fibrous tissue is not only useless as a substitute for the air cells, but with continued inhalation of the causative dust, by its invasion of new territory and consolidation of that already occupied, it gradually and literally strangles the breathing tissues of the lungs.

Asbestosis is, of course, essentially a local disease and therefore it is only when the fibrosis has obliterated that reserve of lung tissue normally present, and encroached upon the remainder which is essential for the normal functions of the individual, that symptoms appear. Then the appearance of undue shortness of breath on any extra effort draws the worker's attention to the fact that his health is not what it should be. The other symptoms of the disease, such as cough, are equally unassuming and are readily ascribed to some common and trivial cause.

From this point the progress of the disease is more rapid, since it is now encroaching on the remaining sound tissue of the lungs, already only just sufficient to maintain the worker in his ordinary daily activities. Ultimately, if no acute respiratory affection has precipitated a fatal termination, a stage is reached when the lungs can do little more than maintain life, and the shortness of breath becomes extreme.

To a great extent the outstanding features of the disease are the mechanical effects of this pervasive network of fibrous tissue induced by the retained asbestos dust. This prevents the proper aeration of the blood, becomes an increasing obstruction to the heart's action, and, in very advanced cases, the traction on the basal masses of fibrous tissue which have consolidated with the diaphragm, pleura and pericardium, still further mechanically embarrasses an already overburdened heart. LANZA and MCCONNELL have drawn attention to the radiological evidence of enlargement of the heart in asbestosis cases.

Usually the fatal issue is determined by the onset of some acute infection with which the remaining undamaged lung tissue is quite unable to cope: this is commonly a low grade broncho-pneumonia, but may be a lobar pneumonia, bronchitis, influenza, or less often, a subacute tubercular infection. There is no evidence that the existence of developed asbestosis predisposes to the onset of such acute infections, but if an acute infection does supervene, the presence of the asbestosis seriously impairs the chance of recovery.

Intercurrent attacks of dry pleurisy, which are partially responsible for the considerable thickening of the pleura which occurs, are common, but usually only cause slight and temporary disablement.

In the absence of intercurrent infections the fibrosis may progress to an extreme degree; bronchiectasis, non-tubercular cavitation, and spontaneous pneumothorax may occur. Ultimately the strain of maintaining the circulation through the partially strangled lungs becomes insupportable and general dropsy with an enlarged liver ushers in death from slow heart failure.

The most important single clinical sign is that of diffuse bilateral impairment of the percussion note; this is slight in degree and associated with a slight sense of resistance. It is best elicited by very light and rapid percussion of the back of the chest from apex to base on each side. This impairment of percussion note is more marked on the right side. The auscultatory signs are variable and depend on the extent and nature of the underlying changes in the lungs, on the extent of the fibrosis with its associated pleural thickening, the presence of intercurrent affections, bronchiectasis, tuberculosis, and on the degree of compensatory emphysema present. In the majority of cases the respiratory murmur is weakened, generally more on the right side, and often still more at the bases.

Asbestosis is a dry disease during most of its course, in the absence of intercurrent infections. This attribute, together with the diffuse distribution of the impairment of the percussion note, makes it a silent and unobtrusive disease, even more so than in the case of silicosis. The symptoms exhibited also closely resemble silicosis and may pass almost unnoticed by the subject for a considerable period, since so little inconvenience results. Between 50 and 60 per cent. of cases of asbestosis complain of slight cough and of undue shortness of breath on exertion.

and show a duskiness or slight blueness of the lips, which contrasts with the general pallor of the face not uncommonly seen.

Diagnosis

In common with other forms of pneumonoconiosis the diagnosis of the disease is fraught with difficulty; particularly is this the case in the early stages, in the late stages when associated with pulmonary tuberculosis, and in any stage if the disease is implanted on lungs already the subject of emphysema, or if some intercurrent infection has supervened.

The fibrosis, although diffuse and bilateral, may be most marked basally and on one side; less commonly the bases may be more or less emphysematous and the maximum fibrosis in the central zone of the lungs; rarely the fibrosis is most marked in the upper portion of the lungs. These factors modify the physical signs presented, as also does the pre-existent state of the chest, and of the lungs upon which the fibrosis is implanted.

Radiographic examination of the chest should never be omitted. A high level of technique is required which should be standardised. A technique which will produce an excellent film and demonstrate adequately silicotic lesions may fail to reveal the asbestos fibrosis entirely, or more often will reveal it only partially; in the latter case the radiographic picture is not only inconclusive, but most misleading.

The cause of this lies in the fundamental difference between the two types of fibrosis—the discrete nodular lesions of silicosis and the close network of asbestosis. This difference is reflected in the radiographic appearances of asbestosis, which are revealed typically as a general lack of translucency in the film together with a fine pin-head mottling. This is aptly referred to by Burton Wood as "the ground-glass appearance": associated with it is a shaggy appearance of the cardiac shadow. While, as is the case with silicosis, certain radiographic appearances may be looked upon as typical of the disease, frequently modifications of, and departures from, the typical picture occur. The radiographic appearances of the developed or advanced stages of the diseases are distinctive, although as Pancoast and Pendergrass maintain, they are not specific for that dust.

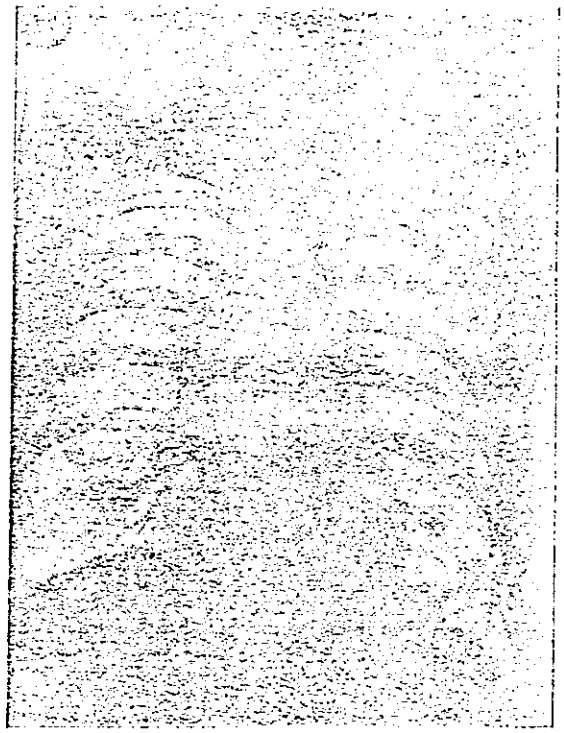


Fig. 1. — Developed asbestosis.

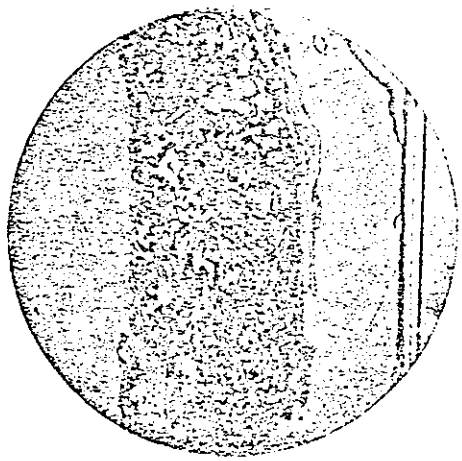


Fig. 1 *bis*. — Lung section (asbestosis).

Radiograms of asbestos workers are very puzzling—more so than in the case of silicosis—when it comes to assessing the degree of asbestosis present, particularly in the earlier stages and also in

women, owing to the shadows cast by the breast tissue. It is of the greatest value in the diagnosis of asbestosis to possess or to have access to a collection of radiograms of cases of asbestosis together with their clinical and industrial histories and the autopsy findings.

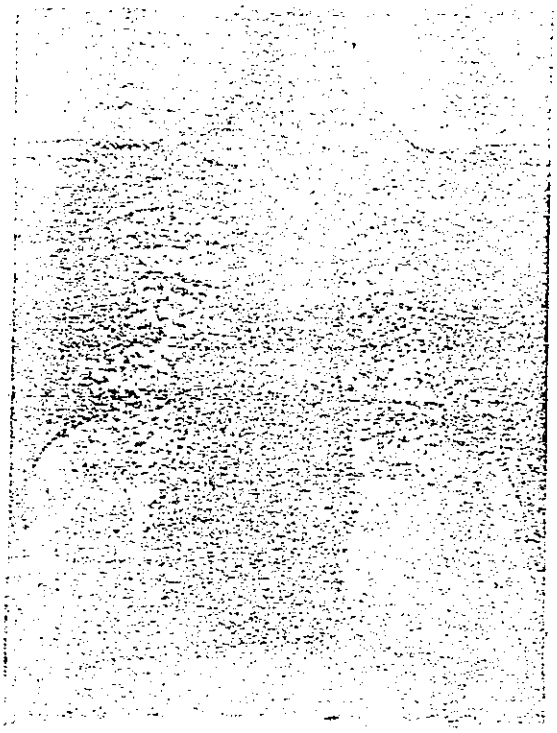


FIG. 2. — Silicosis.

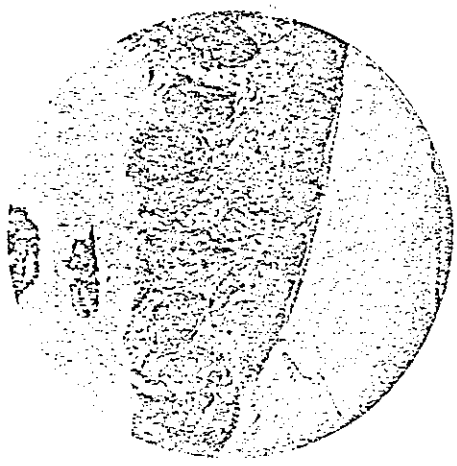


FIG. 2 *bis*. — Lung section (silicosis).

In the light of present knowledge, therefore, asbestosis cannot be diagnosed with absolute certainty on either physical examination or radiological examination alone; with the aid of both the pneumonoconiosis can be diagnosed with certainty if present in some degree, although not necessarily to an extent sufficient to cause either symptoms or any disablement. In very difficult cases where gross tuberculous lesions obscure the picture, then careful investigation of the subject's exposure to asbestos dust, particularly as to the dustiness of the process engaged in and the length of exposure, will enable a correct decision to be reached.

This latter point—careful study of the actual exposure to asbestos dust in each case—is a valuable aid in several ways, since, owing to the immobility of the asbestos fibres once they are occluded in the smaller bronchioles, it enables an estimate to be formed as to whether much or little asbestos dust is trapped in the lungs. Whether much or little dust is incarcerated in the lungs is of prime importance in coming to a correct conclusion as to the outcome in individual cases, particularly those in which the exposure has been to a dense concentration of dust for between one and two years. In such cases the radiograms will be negative since there has been no time for the fibrosis to develop, but if further radiograms are taken of these cases during the succeeding three or four years, the appearance and development of the fibrosis can be watched.

Pathological Features

As already mentioned, the first case of asbestosis in which pulmonary fibrosis was noted at autopsy occurred in 1900, but the first case in which a full microscopical examination of the lung was carried out did not occur until 1927. Records of this case were published by Cooke and Hill, and McDonald.

The pathological changes in the lungs may be considered under three headings:

(1) The alterations which take place in the asbestos fibre after it has reached the lungs;
(2) The reactions of the lung tissue; and
(3) Complications and sequelæ which follow.

The asbestos fibre, as found in the respiratory passages of the workers, is a highly refractile, fine, elastic rod, generally smooth but occasionally showing a sharp saw-like edge, and ends broken at varying angles. The fibres are of various lengths, the shortest being about the length of a tubercle bacillus, the longest extending across the whole field of the microscope. Short forms are the commoner and naturally are more likely to penetrate to the deeper recesses of the lung. The

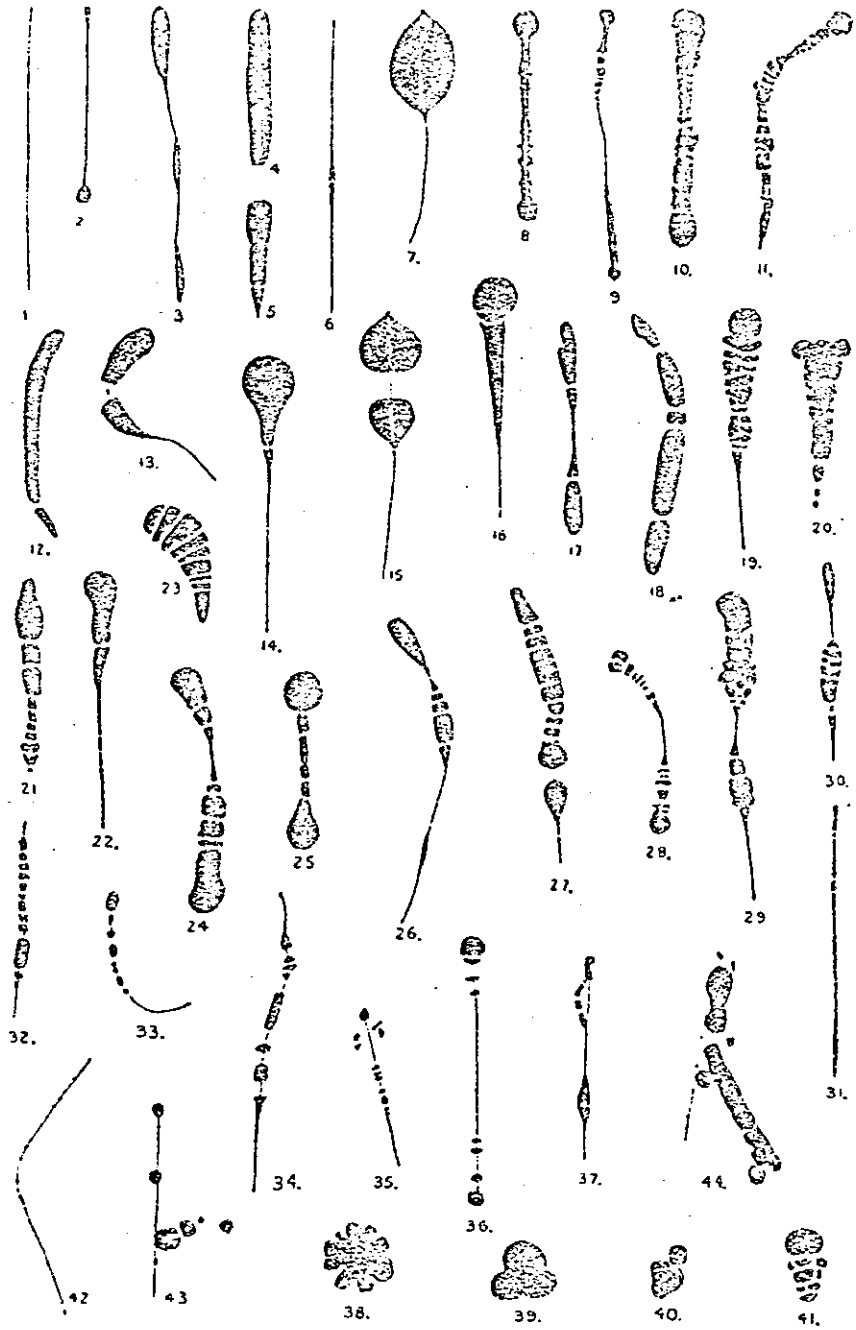


FIG. 3. — Asbestosis bodies (freehand drawings 1/12th obj.).

diameter of the fibres has not yet been determined, since they appear capable of splitting longitudinally into finer and finer constituent fibres, almost indefinitely. Small bundles or sheaves are quite common. In the lung these fibres become coated with a colloidal yellow pigment which in the course of time becomes crenated to form irregular segments, giving the appearance of minute crustaceans. These curiously shaped bodies were first noted by MARCHAND and RIESAL, in Germany, as early as 1906 and, later, by FAHR and FEIGEL in 1914, but the latter was unable to decide whether they were a crystalline product of haemoglobin or whether they were a direct result of dust inhalation; no further attention was called to them until 1927 when they were described by COOKE and HILL and MCDONALD, in Great Britain. The last-named suggested that they were almost certainly a product of the asbestos. GLOYNE finally showed the asbestos fibre lying in the centre of the body by dissolving the colloidal coating with concentrated sulphuric acid whilst under dark-ground illumination. These bodies have never been found in the original asbestos dust [1].

The tissue reaction to the asbestos fibre is dependent on at least two factors:

(1) The sharp, needle-like shape of the fibre which, for practical purposes, is indestructible, and

(2) The siliceous nature of the fibre.

The fibres are for the most part held up at the distal end of the respiratory bronchioles and in the alveolar ducts. Here a cellular reaction takes place consisting of the accumulation of large phagocytes and the production of a characteristic cell, known as the asbestosis giant cell, which, like other foreign body giant cells, is probably not a cell but a collection of partially degenerated phagocytes. As the disease advances the lymphatics in the neighbourhood become blocked. Lastly fibrous tissue is formed round the affected portions of the air passages, the blood capillaries and venules, the interlobular septa of the lung and in the pleura. The fibrosis thus produced is diffuse and readily distinguishable from the nodular fibrosis produced by free silica. Meanwhile the asbestos fibres become coated with a golden-yellow pigment which contains iron and is believed to be derived from blood proteins, to form the typical asbestosis bodies referred to above. It is not known how long all these changes take to bring about, but GARDNER and CUMMINGS have shown that in experimental animals small asbestosis bodies appear at the end of two months but fibrosis in the walls of the bronchioles was not noted until the end of 500 days. At the end of about two years the disease can be recognised

[1] BEGER (1933) has made a thorough histological and optical study of asbestos bodies. According to this author, the metallic element of the molecule of hydrated magnesium silicate is dissolved by the acid fluids of the body, leaving a silicic acid shell. The form of the needles and their power of double refraction remains intact. The asbestos body is subsequently formed by absorption of proteins by the needles of asbestos and subsequent coagulation in the form of gel. The silicic acid from the shell of the asbestos needle becomes dispersed in the proteid sheath of the asbestos body and becomes gradually disintegrated. There then occurs resorption by the body fluids of the proteid mass containing the silicic acid. Granulous bands of ferric oxide impart to the asbestos bodies their brownish colouring.

KOPPENHÖFER (1935) considers that the sheath of the asbestos bodies is not of a proteid nature and opposes BEGER's theory as to the formation and destruction of the asbestos bodies.

SUNDIUS and BYGDEN (1937), in a detailed study of asbestos bodies contained in the lungs of a worker who died of asbestosis, arrive at the following conclusions in regard to the mechanism of formation of the asbestos bodies. The organic substance of the latter is composed of proteid matter. The most important element—40 per cent.—of the *shell* of the asbestos body is iron oxide. The inorganic part of the shell does not come from the inhaled dust, but is of endogenous origin, coming from the body and principally from products derived from the blood pigment. In the light of this theory, the formation of asbestos bodies is not essentially connected with the outbreak of fibrosis. A causal relation between the two processes is perhaps doubtful in view of the fact that various authors have found these bodies in lungs of persons not suffering from asbestosis, and even in lungs free from fibrosis. On the other hand, it is well known that the formation of asbestosis fibrosis should be connected with the presence in the lung of asbestos needles and their action on the pulmonary tissue. On the basis of interstitial localisation of the fibrosis at the outset and its later extension to those parts of the lung most closely affected by respiratory movements, there may be accepted as plausible the theory of those authors who consider mechanical irritation and the injuries caused by the long rigid needles as a factor in the formation of fibrosis. The distribution of the fibrosis, moreover, seems to justify the hypothesis which accords a primary influence to respiratory movements on the migration of the large asbestos needles in the lungs.

LYNCH (1937) in an account of the mechanism of formation of "asbestos bodies" refers to the presence of similar bodies which he designates "silica bodies" found in silicosis with particular mention of their occurrence in a case of typical nodular fibrosis. He states further that similar formations have been noted in the lungs of coal workers (TURNCOTE and DUNN, 1936) as well as in the lungs of a considerable number of subjects who died of heart failure (particularly cases of rheumatism and arterio-sclerosis) without exposure to dust. The iron content and seeming common connection with pulmonary congestion and the local liberation of blood iron raises the question as to whether the iron of the asbestos body may not be so related at least in part, and whether congestion may at least favour production of all such bodies (asbestos bodies, silica bodies, and similar formations).

by the naked eye. It is probable that the disease takes longer than this to develop in man—at any rate to an extent sufficient to cause radiological and clinical signs.

When the lungs are examined by the naked eye after death they are seen to be large and densely fibrotic, and the pleura covering them thickened with plaques of old pleurisy. Often the lung is completely adherent to the chest wall, and in advanced cases to the diaphragm with the formation of a thick and extremely dense layer of fibrous tissue.

This anchoring of the lungs particularly to the diaphragm together with the general increase of fibrous tissue in the lungs which makes them firmer than normal is the probable explanation of a curious clinical sign sometimes seen. E. R. A. MEREWETHER noted that in some advanced cases the apices of the lungs may be easily seen rising in the sunken supra-clavicular areas with expiration, and descending with inspiration.

On cut surface the characteristic appearance is that of dense, blue-black polygonal areas of asbestos, cellular debris and pigment, corresponding to the secondary lobules of the lung and surrounded by thick bands of inter-lobular connective tissue. Generally there is a reddened background of terminal bronchopneumonia in the less affected portions of the lung. There are few naked eye signs of disease elsewhere.

Of the complications and sequelæ of pulmonary asbestosis four are outstanding:

(1) Purulent bronchitis;
(2) Bronchopneumonia;
(3) Pulmonary tuberculosis, and
(4) Emphysema, with occasional rupture of emphysematous bullæ causing spontaneous pneumothorax.

The purulent bronchitis may be of long standing. The bronchopneumonia is practically always a late event, and recovery very rare. The pulmonary tuberculosis is chiefly of the caseous type with little or no sign of repair and the emphysema compensatory to the fibrosis.

In addition to these four main complications may be noted two which are less common:

(1) Dilatation of the bronchial tubes resulting sometimes in what is known clinically as dry bronchiectasis, and
(2) Carcinoma.

The number of cases of the latter so far described is small and it is not yet established that there is any actiological connection between the two diseases.

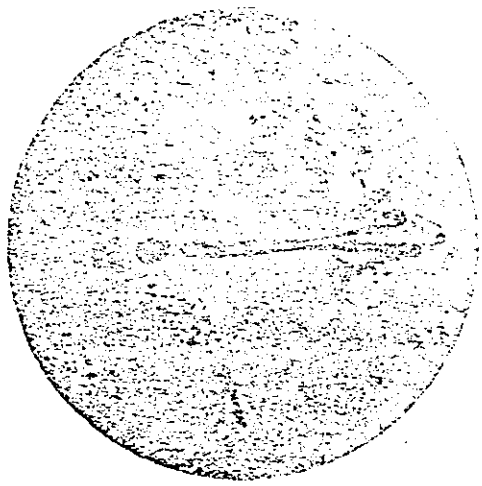


FIG. 4.

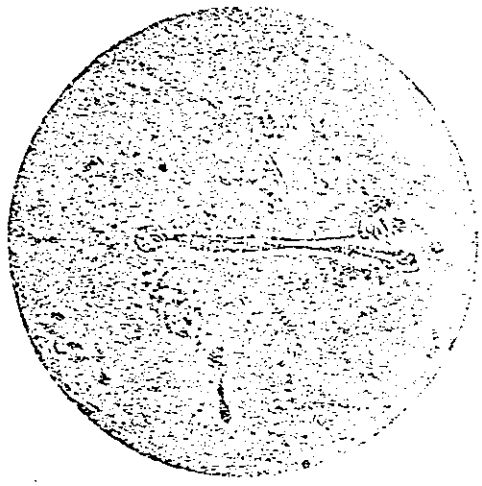


FIG. 5. — Infra-red photomicrographs of the asbestosis lung.

BRIDGE and HENRY have proposed that cancer, in order to be classified as of industrial origin must fulfil the following two conditions: (1) that the incidence-rate in the occupation under review should exceed that in the general population to a significant extent, and (2) that in the occupation concerned there should be sufficient association of a worker with a substance proved experimentally to

have carcinogenic properties. These two postulates cannot yet be regarded as having been fulfilled in the case of the

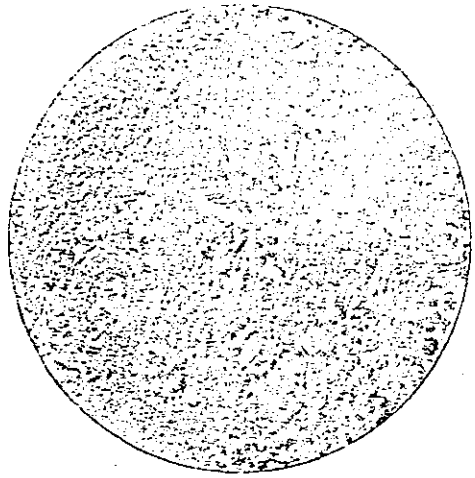


FIG. 6. — Lung section stained with haematoxylin, eosin, and van Gieson, showing fibrous tissue surrounding asbestos bodies.

FIG. 7. — Section of lung, unstained, showing asbestos bodies.

asbestos industry, but there is sufficient evidence to warrant careful observation in the future.

THE PROBLEM OF ASBESTOS DUST

Within certain high and low limits, the concentration of asbestos dust in the air of workrooms is the determining factor in the onset of the disease and also, within limits, concentration of dust and length of exposure determine the incidence rates in different processes in the industry.

Exposure to asbestos dust for less than five years can result in the development of a degree of asbestosis sufficient to cause death. Commonly, however, cases of definite asbestosis are not discovered on examination within five years of commencing work, although a few are found. Among those working who have been employed for between five and ten years in the absence of preventive measures the incidence rate is appreciable, and after ten years of such employment a steep rise in the incidence rate occurs. The incidence rates for the periods of employment 0-4 years, 5-9 years, 10-19 years, and 20 years and over, amongst 1,512 workers examined by the British Silicosis and Asbestosis Medical Board were proportionate to the figures 1, 5.6, 30.4, 53.2.

Although the incidence rate amongst those with less than five years exposure is so low, such exposure may be by no means negligible. The fact is, as suggested above, that work in a dense concentration of asbestos dust over a comparatively short period will lead inevitably to the development of a profound fibrosis, provided that the worker lives long enough for it to develop. As GARDNER and CUMMINGS have shown, the fibrosis takes time to appear, in fact there is a lag of some five hundred days before microscopical signs are demonstrable; it is much longer before the fibrosis matures and clinical and radiological signs are apparent. Correlation of the facts concerning particular cases of asbestosis with those concerning the relative dustiness of the processes at which they worked leads to the conclusion that this "period of maturation" of the fibrosis is not reduced below a certain minimum period, however high the concentration of dust in the air breathed may be.

Similar considerations also show that below a certain concentration, development of a disabling degree of asbestosis will not occur within the space of an average working lifetime.

It appears, therefore, that a certain minimum "fibrosis-producing amount", as it may be called, of asbestos dust must be trapped in the lungs in order to cause a disabling or serious amount of fibrosis, and also that a certain "maturation period" must elapse before that amount of fibrosis is developed.

Fatal cases of asbestosis have resulted from exposure as short as two years or

even a little less, although the fatal issue may be postponed for many years.

It is important to consider what is the amount of dust which will produce this result, or conversely, what is the amount of dust which, from the practical point of view can be inhaled with impunity.

Efforts are being made, notably by American investigators (W. B. Fulton and others), to establish in terms the concentration of dust in the air which can be permitted with safety [1]. In Great Britain the problem has been approached from a different angle, that of determining what processes are safe rather than an exact figure for concentration of dust. E. R. A. Merewether came to the conclusion "that in order to prevent the full development of the disease among asbestos workers within the space of an average working lifetime, it is necessary to reduce the concentration of dust in the air of workrooms to a figure below that pertaining to spinning at the time over which these cases were exposed".

Particulars of cases seem to show that with exposure to high concentrations of dust the minimum period of time which must elapse between the commencement of exposure and the production of a serious degree of asbestosis is approximately seven years—made up of the period of exposure during which the fibrosis-producing amount of dust is taken into the lungs and the maturation period during which the fibrosis develops—these periods, of course, overlap.

This period of approximately seven years, "the asbestosis production period", as it may be called, is the minimum, and few cases mature in this period; in successive years, however, depending on the dustiness of the process engaged in, more cases mature. In the more dusty processes, in the absence of adequate preventive measures, the asbestosis production period is commonly eleven years.

When asbestosis of serious extent has matured the worker is unduly short of breath on any extra exertion, has a little cyanosis of the lips, and also a little dry cough mostly in the mornings. He finds himself disinclined to climb stairs or walk up hills, but still remains at work and usually is not anxious about the state of his health.

The amount of disablement produced is surprisingly slight for a number of years. This is partly due to the character of the disease and partly to the nature of the work which, in the majority of processes in the industry in which there is a risk of asbestosis, does not involve much physical exertion. Those affected may, and often do, continue at work with occasional intermissions latterly, due to exacerbations of bronchitis etc., until the condition is far advanced, although increasing inconvenience from shortness of breath is experienced.

Usually these cases cease work a year or more before death, but sometimes a terminal bronchopneumonia, or other acute infection, commences while they are still at work, and there is no long period of invalidism.

It is remarkable to what extent the lungs can be affected by asbestosis and yet life in a fair degree of comfort remain. The reserve is, however, so slight that the addition of any burden to the system in the form of a disturbance of health which would only slightly inconvenience a normal person, may overcome the remaining resistance and precipitate a fatal outcome.

For these reasons and from consideration of the features present in the recorded fatal cases, the view must be accepted that the existence of even a moderate degree of asbestosis is a serious and ever present potential risk to life.

Since a worker with developed asbestosis may still remain at work and be little concerned as to the state of his health, the question may well be asked "Is asbestosis a serious disease?". To this question, unfortunately, the answer is emphatically "yes".

Asbestosis and Tuberculosis

Proof of this is now not difficult to find, as examination of data from the known fatal cases and comparison with similar data concerning fatalities from silicosis provide ample evidence. Fatalities from asbestosis and asbestosis with tuberculosis have now been reported from a number of countries. Of these the British figures are

[1] The Industrial Hygiene Division of the National Institute of Health (United States) has examined (1937) 543 persons in asbestos textile plants. An analysis of data shows that the maximum concentration of asbestos dust to which workers may be exposed without contracting asbestosis is in the neighbourhood of 5 million particles per cubic foot and it was determined by appropriate technical measures of control that the dust concentration could be reduced to less than 2.5 million particles per cubic foot.

the most complete, since the disease has been compensatable since 1 June 1931. The following figures are abstracted from the Annual Report of the Chief Inspector of Factories for 1935.

TABLE 1

Disease	Number of deaths	Average age at death	Duration of employment in years		
			Longest	Shortest	Average
Silicosis . . .	311	55.8	62.0	2.3	35.1
Silicosis with tuberculosis .	391	52.4	67.0	2.0	31.7
Asbestosis . .	52	41.9	27.0	1.5	12.4
Asbestosis with tuberculosis .	30	37.1	20.0	0.8	9.5

Table 1 gives particulars of 702 deaths from silicosis or silicosis with tuberculosis and of 82 from asbestosis or asbestosis

TABLE 2

Disease	Number of deaths	Average age at death	Duration of employment in years		
			Longest	Shortest	Average
Pottery:					
Silicosis . . .	159	58.0	62.0	2.8	39.2
Silicosis with tuberculosis .	164	54.1	67.0	5.0	36.4
Sandstone:					
Silicosis . . .	78	56.5	57.0	9.0	38.3
Silicosis with tuberculosis .	88	53.9	53.0	10.0	35.3
Grinding of metals:					
Silicosis . . .	26	56.1	56.0	18.0	34.3
Silicosis with tuberculosis .	73	52.1	52.0	2.8	31.5
Sandblasting:					
Silicosis . . .	15	44.5	20.0	4.0	10.7
Silicosis with tuberculosis .	36	44.5	20.0	2.0	8.4
Manufacture of scouring powders:					
Silicosis . . .	8	34.7	37.0	2.3	9.2
Silicosis with tuberculosis .	2	33.5	10.8	2.0	6.4
Miscellaneous:					
Silicosis . . .	25	50.3	45.0	6.0	23.5
Silicosis with tuberculosis .	28	49.9	30.0	9.0	25.6

with tuberculosis. In table 2 the cases of silicosis and of silicosis with tuberculosis are distributed according to the industries concerned.

It will be observed that:

(1) The average duration of employment in the asbestos industry sufficient to cause a fatal degree of asbestosis was 12.4 years as compared with 35.1 years for all cases of silicosis. The actual average length of exposure to asbestos dust was, however, still less.

(2) The shortest length of exposure to asbestos dust which ultimately caused death from fibrosis of the lungs was 1.5 years.

(3) Asbestosis is comparable with the most serious silicosis risks with respect to length of exposure which will cause a fatal degree of fibrosis.

(4) Although the numbers in the asbestosis group are small it will be noted that in 36.6 per cent. the disease was accompanied by tuberculosis, while in the silicosis group 55.7 per cent. were accompanied by tuberculosis, suggesting a less close association between asbestosis and tuberculosis than is the case with silicosis and tuberculosis.

Further confirmation is found in the after history of the 95 cases of asbestosis and the 5 cases of asbestosis with tuberculosis found by E. R. A. Merewether in his original enquiry in 1928. Of these 100 cases, although a number have migrated from the industry and have been lost sight of, 23 are known to have died, 12 from asbestosis, 9 from asbestosis with tuberculosis, and 2 from other conditions in both of whom a considerable degree of asbestosis was found on autopsy. Of the remainder a number are partially or wholly disabled on account of the disease.

As mentioned previously, the risk to life associated with asbestosis is a complex one. Primarily the fibrosis and the resulting mechanical embarrassment of the pulmonary circulation develop in step with each other. The supervention in an individual with asbestosis, therefore, of any disease which adds to this strain brings with it a greater risk to life than would be the case in a normal person. Amongst such diseases, infections of the respiratory tract, and especially bronchopneumonia and tuberculosis, hold first place.

The risk from tuberculosis requires special consideration because of its accepted importance in asbestosis as in silicosis and because of its infective nature.

The interaction of asbestosis and tuberculosis is of moment in determining both the real importance of asbestosis as an industrial risk and also the scope and probable effectiveness of projected preventive measures. There are a number of aspects to this relationship which have to be clearly defined and as yet the data are insufficient.

GARDNER and CUMMINGS, as a result of their experimental work on guinea-pigs, have established a point of the greatest importance, namely, that a primary tuberculous infection is influenced only to a limited degree by inhaled asbestos and that the tendency to healing by fibrosis is marked. They emphasised the contrast in this respect between the effect of asbestos and of free silica, with which the tendency was overwhelmingly towards the production of generalised chronic tuberculosis of the lungs and viscera.

These same workers found in another group of experiments where the tuberculous infection was implanted on to an existing asbestos fibrosis that the stimulating effect on the tuberculous infection was more marked than when the infection and inhalation of asbestos were instituted simultaneously, but the ultimate outcome had not yet been observed. They also stated that the combined action of asbestos dust and tubercle bacilli in the lung produced more fibrosis than did either agent acting independently.

In a subsequent paper, BURTON WOOD and GLOYNE reviewed a series of 100 cases of asbestosis which had been under their observation. In this group they found 30 cases with pulmonary tuberculosis, 21 being active and 9 obsolescent. They pointed out that the group with obsolescent tuberculosis emphasises the fact that obsolescent tuberculosis in the human is not necessarily reactivated by asbestosis.

In the series of deaths analysed above it was noted that asbestosis was associated with tuberculosis in a much lower percentage than was the case with silicosis.

J. RUSH SHULL in an analysis of 71 cases of asbestosis notes the presence of tuberculosis in 10, one of which showed a healed miliary tuberculosis.

Although it is not possible yet to answer categorically

(1) Does the inhalation of asbestos (*a*) antecedently, (*b*) coincidently, (*c*) subsequently to a tuberculous infection favour the development of the tuberculous process or not, and

(2) What is the effect of the presence of (*a*) a slight, (*b*) a moderate, (*c*) an advanced degree of asbestosis on (i) the implantation, (ii) the development of a tuberculous infection,

these observations suggest that whatever the added risk to asbestos workers from pulmonary tuberculosis may be (and there appears to be some), it is less than that associated with silicosis. It is worth recording that in two cases signs of both silicosis and asbestosis have been found at autopsy, in one of which there was also pulmonary tuberculosis.

PREVENTIVE MEASURES

The risk from asbestosis in the asbestos industry is no less grave than the most serious risks from silicosis in the silicosis producing industries. The preventive measures necessary, therefore, will be extensive and stringent. The essential is dust suppression in all processes to a safe level, which level may be determined by reference to a definite concentration of dust in the air of the workrooms or by reference to the amount of dust produced in a process which has been shown to be safe. There are advantages and disadvantages associated with each of these standards, but space does not permit of their discussion here. In Great Britain the second of these alternative methods was adopted. A Joint Committee of representatives of the Factory Department of the Home Office and of the Asbestos Textile Manufacturers agreed on a practical standard based on MEREWETHER's suggestion as to the relationship between the safe level of dust concentration and that evolved in spinning processes. This Committee concluded on the evidence then available that

"For practical purposes, the conditions arising from flyer spinning [1] carried on without exhaust under good general conditions may, it seems to the Committee, be taken as the "dust datum" . . . If, therefore, a particular process appears to give rise to dust in excess of that associated with such flyer spinning, the Committee regard the need for preventive measures as established."

[1] The dust production in ring spinning is above the safe level and the Regulations require the provision of local exhaust ventilation to the process.

In March 1932 a comprehensive Code of Regulations designed to suppress the dust produced in all processes to at least the level of that "arising from fiyer spinning carried on without exhaust under good general conditions" came into force.

These Regulations apply the following principles to achieve this standard: (1) Application of efficient localised exhaust ventilation at dust producing points. (2) Substitution of enclosed mechanical methods for hand conveyance and for dusty hand work generally. (3) Effective enclosure of dust-producing machines and plant. (4) Substitution of wet methods for dry. (5) Elimination of certain dust-producing appliances. (6) Effectual separation of processes to prevent unnecessary exposure to dust. (7) Use of sacks of close texture for internal work in the factory, and cleaning of them by machinery. (8) Efficient cleaning system. (9) Precautions to prevent dust from asbestos in storage chambers or bins entering the workrooms. (10) Regular examination and testing of ventilating plant; dust settling and filtering apparatus not to be allowed in workrooms. (11) Breathing apparatus of approved type to be provided for persons employed in certain operations.

The Regulations also prohibit the employment of young persons under the age of 18 in the most dusty processes.

In order to achieve the object of the Regulations problems of ventilating engineering of the utmost difficulty had to be solved, particularly on the textile side of the industry, where the application of local exhaust ventilation and other methods of dust suppression of a high standard to operations in which the necessity for it had never been envisaged before, was required.

Other preventive measures in force in Great Britain include the control of the disease by periodical medical examination of the workers, by which those unfitted by health reasons are prevented from entering the industry and cases of asbestosis and of pulmonary tuberculosis are detected at the earliest possible moment.

A practical maxim of the greatest value is that every translation of liberised asbestos in the factory produces dust which, if not controlled, is dangerous.

Compensation for Asbestosis

Great Britain was the first country to pass special legislation relative to compensation for asbestosis and asbestosis accompanied by tuberculosis for all workmen employed at any time on or after May 1931 in any process specified in a comprehensive schedule. In Germany the Order of 16 December 1935, and in France, the Order of 11 March 1937, grant compensation for *serious* asbestosis affecting workers coming within accident insurance legislation. In the United States the Law of 26 March 1935 passed in North Carolina provides for compensation of asbestosis in a certain number of industries[1].

The problem of compensation for asbestosis was considered by the Correspondence Committee on Industrial Hygiene of the International Labour Office which, at its last meeting in October 1935, decided to recommend to the Governing Body the following formula for inscription in the international schedule: "Asbestosis, with or without pulmonary tuberculosis, provided that asbestosis is an essential factor in causing the resultant incapacity or death" when occurring amongst workers engaged in "industries or processes recognised under national law or regulations as involving exposure to the risk of asbestosis". The Committee made the further recommendation that "it is advisable to recommend that those countries which so far are without adequate knowledge of the question should carry out in the near future the requisite enquiries and research for determining the extent of the occupational risk involved."

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[1] Among the States which accord compensation for asbestosis by blanket coverage, there are some in which the general coverage Law contains special provisions concerning asbestosis: Illinois (Law of 16 March 1936), Indiana (Law of 6 March 1929), Pennsylvania (Law of 1937).

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Figs. 1 *bis* and 2 *bis* are taken from lung sections stained by E. H. Sarsons, of Birmingham University, with his modification of Mallory's method.

Figs. 3 to 7 are taken from various publications by S. Roodhouse Gloyne.

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