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important to note that the inhalation of CO gas tends to raise the basic pressure and so to interfere with the circulation to the tissues. This interference was evidenced by the estimation of the basic blood-pressure when the ration relapsed to 1·8 to 1, instead of the normal 3 to 1. Tuberculosis, by reducing the area of lung tissues available for oxygenation purposes, diminishes the amount of oxygen carried by the blood and as a result interferes with the oxidation of the penultimate products of digestion, giving rise to toxins which by themselves tend to raise the basic pressure. For this reason, whether the basic blood-pressure be raised as a result of tuberculosis or by the inhalation of CO gas, or both together, the fact that it was so raised was an important factor in connection with the prognosis of such a case.

REFERENCE.

[1] STEPHENS, G. ARBOUR. "Heart and Spleen in Health and Disease," 1932.

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# A MEMORANDUM ON ASBESTOSIS.

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## I.—INTRODUCTION.

THIS industrial disease of the lungs due to the inhalation of asbestos dust, although only recognised and accepted recently as an entity of serious import, has been, during the past few years, the object of much careful investigation in various countries. In Great Britain this has culminated in legislative action to control the disease and to provide for the compensation of affected workers.

It is timely, therefore, to pause to review the position in the light of all the knowledge obtained concerning the disease, and to appraise the value of the preventive measures adopted, considering critically not only whether they are adequate for the purpose, but also whether they are necessary. No matter what the particular occupational disease may be, such a survey of the position is necessary from time to time in the interests both of employees and employers for the reason that, although, ethically, the provision of measures for the prevention and compensation of an occupational disease is unassailable, it is none the less true that the imposition by legislative sanction of conditions of employment on an industry casts a definite financial burden not only on the industry as a whole, but also on individual employers and employees. Moreover, since methods of manufacture are essentially fluid, and changes in processes frequently result in a modification of the associated risk from occupational disease, clearly the compensation law and the scope of the preventive regulations applied to the industry must be modified also from time to time in conformity with the altered risk.

In the case of asbestosis, evidence already available is amply sufficient to demonstrate conclusively the necessity of particularising it as a com-

pensatable industrial disease and of enacting regulations for the industry designed to control the incidence of the disease and ultimately to abolish it.

Asbestosis is a new disease which illustrates only too clearly that the march of civilisation does not solely confer benefits on the world, but also harasses man with new problems and perplexities and with new diseases, or by the dissemination of diseases long thought circumscribed.

## II.—The Nature of Asbestos and the Growth of the Asbestos Industry.

Asbestos was known to the ancients as a substance, believed to be of vegetable origin, with unique properties: it could be spun and woven to form a non-inflammable cloth. For this reason and because of other attributes it was an early scientific curiosity and had an extremely limited vogue amongst the patrician element for some purposes, as, for instance, funeral wrappings and lamp wicks.

No serious attempt to exploit commercially the fire-resisting qualities of asbestos was made until the latter part of the nineteenth century, when, in 1876 in Italy and in 1878 in Canada, quarrying of the mineral was commenced. Some years elapsed, however, before difficulties in manufacture were overcome and advantage taken of the obvious potentialities of the mineral. Since then the increase in production has been remarkable. In 1880 the world production of asbestos was little over 500 short tons: it reached to 35,000 short tons in 1900, to over 200,000 in 1920, and to over 430,000 in 1929. From 1910 to 1929 the world production of asbestos increased over fourfold. The imports of asbestos (all grades) into the United Kingdom rose from 18,591 tons in 1922 to 33,520 tons in 1927. The figures for 1927 refer to Great Britain and Northern Ireland only. Of these quantities, 8,844 tons and 3,794 tons respectively, were re-exported. Thus the consumption of asbestos in this country trebled within five years.

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 flexibility. Their value depends on the facility with which they can be split up into long and flexible fibres for spinning and weaving, on their resistance to heat and acids, and on their insulating properties with respect to heat and electricity. Varieties of asbestos possess these characteristics in differing degree. Other properties of these minerals also find uses in industry.

Practically speaking, 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: constantly new uses for asbestos are being 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 materials in great variety. The phenomenal expansion of the motor car industry in the past twenty years has caused a corresponding increase in the demand for asbestos fabric brake linings, asbestos composition clutch rings, and latterly of moulded asbestos brake linings. Many other examples will come to mind.

The number of workers employed in the quarrying of asbestos and in the manufacture of asbestos-containing materials and articles is considerable, but the industry is very small as compared with, for example, the coal mining industry and, fortunately, only a small proportion is exposed to risk from the inhalation of asbestos-containing dust. In 1929, in Canada, which is responsible for 70 per cent. of the world's production of asbestos, 3,194 were employed in asbestos mines and mills, and a further 351 on the manufacturing side [1]. In this country it is estimated that the number of workers so exposed in manufacturing processes lies between 2,000 and 3,000. It is, therefore, nearly comparable, in point of number of workers exposed to appreciable risk from the inhalation of dust, to a silicosis-producing industry such as the refractories industry.

## III.—The History of Asbestosis and its Recognition as a Specific Industrial Disease.

Asbestosis may be defined as a specific occupational disease of the lungs caused by the inhalation of asbestos dust and characterised by progressive replacement of the essential active functioning tissue of the lung by inactive non-functioning fibrous or scar tissue.

It is essentially a pneumonokoniosis, a fibrosis of the lungs caused by the inhalation of dust, and therefore is in the same category as silicosis, which disease it resembles in some respects while differing considerably in others.

The first recorded case of asbestosis was that of a patient who died in 1900 in the Charing Cross Hospital, London. All that is known of this case is contained in the evidence given by Dr. Montague Murray before the Departmental Committee on Compensation for Industrial Diseases in 1906 [2]. The patient, aged 34 at death, had worked with asbestos for some fourteen years. Post-mortem examination revealed extensive diffuse pulmonary fibrosis with no evidence of pulmonary tuberculosis. This man stated that of the 10 men working in the cardroom when he went into it, he was the only survivor, all the others having died at ages round about 30.

This Committee was unable to obtain any evidence of additional cases, but in the same year (1906) Marchand and Riesal noted the presence of unusual bodies in the lungs of an asbestos worker [3]; in the same year also in the French official *Bulletin de l'Inspection du Travail* [4] the high mortality in an asbestos textile factory is reported upon and considered as probably due to a pneumonokoniosis from the asbestos dust.

In 1910, following a death certified as "acute pulmonary phthisis in an

asbestos worker" and an unverified report that seven other deaths from phthisis had occurred in the same factory [5], a survey of the industry by the British Factory Department did not disclose any evidence of the existence of a serious health hazard in the industry, nor were the replies to inquiries made to the Canadian Government suggestive of the existence of such a condition amongst the workers in the quarries and mills. At the request of the British Home Office, however, Professor J. M. Beattie in 1912 conducted some experiments on animals and concluded that the inhalation of asbestos dust would cause a mild degree of fibrosis.

Although definite proof was not forthcoming at that time, there were reasonable grounds for suspicion that the inhalation of much asbestos dust was to some extent harmful, and so from then onwards the British Factory Department pressed for the installation of exhaust ventilation in the more dusty processes.

In 1914, Fahr demonstrated to the Medical Society of Hamburg [3] the lungs of an asbestos worker with pneumonokoniosis and mentioned the occurrence of a large number of crystals in the lungs, but did not describe them more closely.

In 1917, during the Great War, the British Factory Department considered the matter again, but no further evidence incriminating asbestos had come to light either at home or abroad, and therefore no further action could be taken.

It was not until 1924 that anything further of moment occurred; in that year appeared a note by W. E. Cooke [6] concerning the death of an asbestos worker in which examination revealed not only extensive lesions of pulmonary tuberculosis, but also evidence of pulmonary fibrosis of a diffuse nature, which he attributed to the effects of asbestos dust. This case was fully described by Cooke and Stuart McDonald in 1927 [7, 8]. In the same year Cooke and Hill [23] and McDonald [8] gave the first detailed description of what are now known as the asbestosis bodies. The latter confidently asserted that they originated from the asbestos dust inhaled in the lungs and gave definite reasons for this opinion.

While from the practical point of view the ætiological relationship in this case between the inhalation of asbestos dust and fibrosis of the lungs would have been strengthened by the absence of a tuberculous infection, these two papers were, however, of the greatest importance and focussed the attention of investigators on the subject.

A few weeks later H. E. Seiler [9, 10] discovered an asbestos worker with signs of a diffuse pulmonary fibrosis with no evidence of a tuberculous infection; further investigation revealed no presumptive cause for the fibrosis other than the inhalation of asbestos dust.

This case, at that time the third of which the Factory Department had knowledge, was, however, the first in which the four essential conditions necessary to establish a relationship between the inhalation of asbestos dust and the development of pulmonary fibrosis could be demonstrated. These conditions are :—

(1) Work involving exposure to asbestos dust.

(2) The existence, demonstrable clinically and radiologically, of a definite pulmonary fibrosis.

(3) The absence of previous or present infections known to cause pulmonary fibrosis, e.g., tuberculosis, influenza, or pneumonia.

(4) The absence of previous or present work involving exposure to other dusts, which might cause pulmonary fibrosis.

When, therefore, the investigation of Seiler's case showed that other industrial and infective causes of pulmonary fibrosis could be excluded definitely, the necessity of deciding whether the supervention of this disease in an asbestos worker was an exceptional occurrence, or evidence of a grave health risk in the industry, was apparent, and a comprehensive inquiry in Great Britain was commenced in February, 1928. This investigation involved the clinical examination of 363 workers, or approximately 16·5 per cent. of the estimated population at risk from pure or nearly pure asbestos dust; 133 were examined radiologically and great care was taken to exclude all workers whose lungs might have been affected by previous work in other dusty occupations. The influence of work in different processes was assessed and the relative dustiness of them estimated. The results of this inquiry were published in 1930 [11, 12] and except where indicated, the conclusions and data given in this memorandum are derived from it and from the results of the first cycle of the statutory periodic medical examinations in the asbestos industry. Between the beginning of 1928 and 1930, and since, a number of other papers dealing with the clinical, pathological and radiological sides of the subject have been published both here and abroad. Many of these are referred to and are noted in the bibliography appended. Unfortunately no other general survey of the industry has yet appeared in print.

## IV.—Influences which Delayed the Recognition of Asbestosis as a Specific Industrial Disease.

It is of some importance to consider why, if asbestosis is a serious risk, this industry has only recently excited attention by reason of its raw material becoming suspect as a cause of industrial disease.

Several factors have contributed, but the main one appears to be that in the past it was not practically possible to obtain proof of the injuriousness of asbestos dust, considering the limitations imposed by the state of the industry, and the point reached by research work into the relationship between dust inhalation and diseases of the lungs.

In the closing years of the nineteenth century, the position with regard to dust diseases of the lung was chaotic; while it was realised, as had been known for centuries, that work in certain dusty occupations was dangerous, since so many workers died of lung disease, the nature of the disease, the precise causes and their mode of action were the subject of acute controversy. It was not so long before (in 1866) that Virchow, the great pathologist, at last admitted the possibility both that dust might be inhaled and might cause discolouration of the lung [13].

The literature was overloaded with many special names invented, some on very slender evidence, to describe the supposed effects of particular dusts. Some of these terms for non-existent conditions still crop up to-day. From 1900 onwards the attention of Governments and private investigators became more and more focussed on what is one of the gravest problems of

the age, that of the prevention and control of pulmonary disease amongst workers in what are now known as the silicosis-producing industries. In 1902 and 1903 the Milner Commission on Miners' Phthisis in South Africa pursued its investigations, to be followed by another Commission on the same subject from 1907-10, and by a third in 1911-12. The achievements of these Commissions and the succeeding labours of the Miners' Phthisis Prevention Committee, and the Miners' Phthisis Medical Bureau are classical. In 1914 the British Royal Commission on Metalliferous Mines and Quarries, in its report, stated the position then with extreme clarity as follows: "We do not know whether other dusts besides those containing free crystalline silica induce a pathological condition in the lungs, though the experiments of Professor Beattie on animals suggest that this may occur" [14].

It appears, therefore, that this focussing of energy on the all-important problem of silicosis, and the production of proof that the inhalation of free silica was responsible for the production of fibrosis of the lungs amongst workers in so many industries, tended to obscure the possibility that dusts other than those containing free silica might be injurious. In fact, it seems that the Royal Commission's reservation quoted above slipped from memory and the opinion grew that, from a practical point of view, if a dust contained free silica, it was harmful, if not it was negligible.

The second factor was the size of the industry. As mentioned above, only little more than 2,000 workers in Great Britain are exposed to-day to an appreciable risk from asbestos dust, and these are distributed in various parts of the country. Much less than this number have been employed in the more dusty, and hence more risky, processes for five years or more.

As noted above, the industry has expanded at a remarkable rate, but even so it seems probable that not more than between 35,000 and 40,000 workers in the world are exposed to-day to an appreciable risk from the inhalation of asbestos dust.

There are, of course, no precise data on which to assess this number accurately, but this estimate, which is based on the available figures for Great Britain and Canada, and the statistics of the world-production of asbestos, may not be far from the truth.

It is useless to look in the mortality statistics of various countries for any pointers that combined silica, far less asbestos, is dangerous. It was this very fact—the distinction as reflected in the vital statistics between industries where there is exposure to free silica and those in which the exposure is to combined silica—which at the same time incriminated free silica and apparently absolved combined silica. That this was the position is quite evident on reading the evidence submitted to the British Royal Commission on Metalliferous Mines and Quarries in the year just prior to the Great War. Incidentally, the fact that the vital statistics do not reveal an increased mortality from pulmonary disease amongst workers exposed to combined silica suggests strongly now that only some silicate minerals are dangerous when inhaled

Another important factor which prevented earlier recognition of the disease was that twenty years ago radiography of the lungs was in its infancy, and only just previously had that invaluable aid to diagnosis been

applied seriously in the investigation of silicosis [15]. Moreover, the radiographic picture of developed asbestosis is distinct from that of typical silicosis and when compared with the latter is, in the absence of definite knowledge, likely to be dismissed as of little import.

It is easy to understand, therefore, the influence of these and other factors in delaying the recognition of the disease; as is so often the case, much may be surmised with accuracy, but proof may have to await advances in collateral lines of research.

## V.—The Main Features of the Disease—Diagnosis—The Significance of the Asbestosis Bodies.

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 deposition of asbestos in the lungs [30]. 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 essential tissues of the lungs.

In common with other essential organs of the body the lungs have a large reserve of tissue for use in emergencies and to permit of a diminution in functional capacity due to advancing age or disease. For this reason and because fibrosis of the lungs is essentially a local disease, it is only when the fibrosis progresses to the extent of obliterating this reserve, that 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 infection 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.

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 bronchopneumonia, but may be a lobar pneumonia, bronchitis, influenza, or, less often, a subacute tubercular infection. With the exception of a mild degree of bronchitis which is more often mechanical in origin and not infective, being due to irritation of the retained dust, there is no evidence that the existence of developed asbestosis predisposes to the onset of such acute infections; but if, however, an acute infection does supervene, the presence of the asbestosis seriously impairs the chance of recovery. Details of cases in which the terminal

infection was tubercular could be given which demonstrate this added risk unequivocably: in these cases the onset of the tuberculosis was indubitably recent in origin and undoubtedly precipitated the fatal termination, but on post-mortem examination the extent of the tubercular lesions was clearly insufficient to have caused death under ordinary circumstances.

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

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.

*Clinical Signs.*—The most important single clinical sign is that of diffuse bilateral impairment of the percussion note: this is slight in degree and best elicited by very light and rapid percussion of the back of the chest from apex to base on each side, and is associated with a slight sense of resistance. The other physical signs do not differ materially from those presented by silicosis and need not be detailed here.

The symptoms exhibited also, as might be expected, closely resemble silicosis. For a considerable period the disease causes almost no inconvenience to the worker, and for a long time the symptoms may pass almost unnoticed. Between 50 and 60 per cent. of cases of asbestosis complain of 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 pneumonokoniosis 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 zones of the lungs; rarely, the fibrosis is most marked in the upper portions of the lungs. These factors modify the physical signs present, as also does the pre-existent state of the chest, and of the lungs upon which the fibrosis is implanted. The signs, however, are not so tenuous, as has been suggested, that it is impossible to make a diagnosis with fair certainty on physical examination alone. Radiographic examination of the chest is, however, invaluable and should never be neglected. A high level of technique is required and this 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 partially; in the latter case the radiographic picture is not only inconclusive but most misleading.

The cause of this is not far to seek and lies in the essential difference between the two types of fibrosis. While typical silicosis consists of dense

discrete and fairly large nodules of fibrous tissue embedded, as it were, in more or less normal and even slightly "ballooned" lung tissue, typical asbestosis consists of a network of fibrous tissue permeating the lung, the air cells in the immediate vicinity tending less to "balloon" than to collapse.

Minor variations in technique, therefore, will have in the case of the silicotic fibrosis comparatively little effect in reducing the contrast between the nodules and the sound lung on the resulting film, and the permissible margin is fairly large; not so, however, in the case of asbestosis, since the fibrous tissue lesion is far more diffuse and the areas of relatively sound lung tissue, with which contrast must be obtained if the fibrosis is to show on the film, are small and interspersed in the fibrous tissue network; hence, in the case of asbestosis, much care is necessary to take advantage of the small contrast available and to produce a film of real value in diagnosis and prognosis. The ascertainment and standardising of the optimum technique for this purpose is a complex problem which is occupying the attention of the Industrial Pulmonary Diseases Committee of the British Medical Research Council.

This fundamental difference between the two types of fibrosis referred to above is of great importance, explaining, as it does, a number of the features of the disease.

The diffuse dispersion, as it were, of the fibrotic lesions in the lungs in asbestosis as compared with the discrete localisation of the lesions in silicosis is reflected in both the physical signs and the radiographic picture of the disease—the diffuse slight general impairment of the percussion note, and the diffuse ground glass appearance [16] or veiling, as it were, together with the fine pinhead mottling of the radiographic film, being striking. The tendency to uniformity and the unobtrusive nature of the symptoms, physical signs and radiographic appearances are at the same time the most outstanding and the most deceptive features of the disease.

The radiographic appearances of the developed or advanced stages of the disease are distinctive, although they are not specific. While, as is the case with silicosis, certain radiographic appearances may be looked upon as typical of the disease, frequently modifications of and departure from the typical picture occur.

No one who has the opportunity of examining a large number of radiograms of the lungs of workers in different dusty occupations can fail to appreciate the infinite gradations which appear and how it is possible to duplicate a film from a worker in one industry with a film from a worker in another totally distinct industry.

Badham [17] was the first to describe the fine type of fibrosis which can be caused by an inorganic dust containing no free silica, and to draw attention to its serious nature.

An inquiry in the United States [18] showed that in cases of silicosis in granite cutters the radiographic features were atypical.

Pancoast and Pendergrass [19], in their comprehensive review of the whole subject, have given it as their view that the appearances of asbestosis in the radiographic film are not specific for that dust, and there can be no

doubt but that their opinion, founded on a very wide experience of radiography of workers in dusty trades, is the correct one.

In developed cases of pneumonokoniosis, however, the fairly large discrete mottling of the "snow-storm" of silicosis and the fine pin-head mottled ground glass appearance of asbestosis, do, it seems, represent the two typical extremes and reflect the two types of lesion found in the lungs, the discrete nodular silicotic type and the fine network of the asbestosis type. The infinite gradations that one sees in films of workers exposed to different inorganic dusts, often in mixture, therefore, it appears reasonable to suggest, represent the effects of the components of the dust encountered, according to their relative preponderance, on the lungs, and the radiograms accordingly approximate more to one or other end of the scale. In general, therefore, both the pathological and the radiographical appearances do appear to reflect more or less proportionately the actions of the fibrosis-producing silica and silicate. Radiograms of certain granite workers, of certain pottery workers, and of some mixers of boiler coverings, present examples of some of these gradations and illustrate this point.

In workers exposed to the insulating material known as "magnesia" (85 per cent. magnesium carbonate and 15 per cent. asbestos approximately), with signs of pulmonary fibrosis, one finds a type of radiogram which is totally distinct from either typical silicosis or typical asbestosis, and from appearances intermediate between the two. It is impossible to say what the underlying lung lesion may or may not be, as no autopsy findings are available. Haynes [20, 21], however, states that magnesia is a dangerous dust, so the radiographic picture may truthfully reflect a modification of the asbestos fibrosis effected by the introduction of a preponderance of a more quickly soluble dust.

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. Keating [22], of the Silicosis and Asbestosis Medical Board, has pointed out that pregnancy may produce a mottling suggestive of asbestosis. Presumably this is due to pressure on the diaphragm as well as to engorgement of the breast tissue. The film was one of an asbestos worker with less than two years' exposure, too short a time to produce radiographical appearances of developed asbestosis, in the absence of an additional period elapsing since the last exposure to permit of the development of the fibrosis. On first examination the film was very suggestive of developed asbestosis, but on close examination in the spaces between the fine mottling the lung field lit up well, which would not have been the case if the picture represented developed asbestosis.

In the light of present knowledge, therefore, one can say that asbestosis cannot be diagnosed with absolute certainty on either physical examination or radiological examination alone: with the aid of both, the pneumonokoniosis can be diagnosed with certainty if present in some degree, although not necessarily to an extent sufficient to cause either symptoms or any disablement. But it is not possible to say from physical and radiographical examination that an asbestos worker has not, trapped in the lungs, an

amount of dust sufficient to cause the development of serious fibrosis after a lapse of time.

The greatest possible aid to the diagnosis of asbestosis is to have available for comparison a series of films of asbestos workers, together with their whole history and the post-mortem findings if death has occurred.

The questions of degree of disablement and prognosis are discussed later.

*The Asbestosis Bodies and their Significance.*

In addition to spicules of asbestos, which may be very numerous, and other particles identified by Cooke [7] as derived from the raw material, certain foreign bodies are found in the lungs of asbestos workers.

These bodies were first seen apparently by Marchand and Riesal in 1906, and later by Fahr in 1914 [3], but Cooke and Hill [23] and McDonald [8] gave the first detailed description of them. They are microscopical in size, of a yellowish brown colour and of various shapes, but commonly with an elongated beaded appearance and with one or both ends bulbous. Gloyne [24], who gives the most comprehensive description of them and their attributes, illustrates 44 various forms.

They give both the Prussian blue and ammonium sulphide reactions for iron, but do not stain with ordinary aniline dyes, but may be made to take on colour by methods described by Gloyne which enable their structure to be studied more precisely and aid in photomicrography. The same observer has opened up a new field in this connection by the use of infra-red methods of photomicroscopy [25]. They are found in sections of the lungs, sometimes in enormous numbers, in direct films from the lung juice at the time of the post-mortem examination [26], in the juice obtained by lung puncture during life (Stewart and Haddow [27], first suggested by S. A. Henry), occasionally in direct films of the sputum, but frequently, although usually in small numbers, after digestion with antiformin and centrifuging (Stewart and Haddow [28]). They have been detected in the fæces (Gloyne [29]).

Dewirtz [48] and Gloyne [24] have both independently failed to find them in sections of the asbestos corns, which are common amongst asbestos workers and are caused by the irritation of the retained portions of spicules of asbestos which have penetrated the skin and broken off.

In experimentally dusted animals they occur in the lungs of guinea-pigs after an exposure of approximately seventy days; in the rabbit they have not been discovered after exposures as long as three-hundred-and-thirty days, and in the white rat they are very rare (Gardner and Cummings [30]). Gloyne [24] found one in a wild rat caught on an asbestos factory premises. Schuster [31] in a dog, long resident in an asbestos factory, could find no trace of the asbestosis bodies, although the lungs showed typical asbestosis.

Asbestosis bodies are not found in asbestos dust, but McDonald came to the definite conclusion that they were derived from asbestos fibres modified by residence in the lungs. The asbestos fibre forming the central core in the asbestosis body was identified by Gloyne [32]. The typical bodies have not been found in any other disease, but forms superficially resembling them have been found in a coal miner (Tylecote and Shaw Dunn [33] and Cooke [45]). How far, therefore, is the presence of the bodies in the sputum an aid to diagnosis of the disease?

Since they are found in the lungs following very short exposure to asbestos dust—Simson [34] has recorded the presence of the bodies in a human lung from a case with a history of only two months' exposure—the fact of their presence in the sputum can only be said to be indicative of exposure to asbestos dust. Where, however, the bodies are found in clumps, instead of individually, in the sputum, identical with the clumps seen in sections of the fibrosed lungs, their presence points to disintegration of lung tissue, and it is suggested (Stewart, Tattersall and Haddow [35]) that in such cases they have a far greater significance and that it is reasonable to conclude that there is a definite underlying asbestosis.

Since they may be found in the lungs, at any rate up to fourteen years after the last exposure to asbestos dust, their presence does not give any clue as to whether the exposure has been recent or long past.

## REFERENCES.

**No attempt has been made to compile a complete bibliography, but the following are particularly valuable in connection with the points discussed in the foregoing memorandum.**


[1] **Ross, J. G. "Chrysotile Asbestos in Canada." Canadian Department of Mines Publication. Ottawa: F. A. Acland, 1931. (The most comprehensive and valuable monograph on the subject.)**

[2] **Departmental Committee on Compensation for Industrial Diseases. Minutes of Evidence, Appendices and Index, 1907. Cd. 3496, p. 127; Report, 1907, Cd. 3495, p. 14.**

[3] KRUGER, ELIZABETH, ROSTOSKI and SAUPE. *Arch. f. Gewerbepath. u. Gewerbehyg.*, 1931, **2**, 558.

[4] AUKIBAULT. *Bulletin de l'Inspection du Travail*, 1906, p. 126. Paris: Imprimerie Nationale.

[5] Annual Report of the Chief Inspector of Factories and Workshops for 1910, p. 188. London: H.M. Stationery Office, 1911.

[6] COOKE, W. E. *Brit. Med. Journ.*, 1924, ii, 147.

[7] *Idem. Ibid.*, 1927, ii, 1024.

[8] McDONALD, S. *Ibid.*, ii, 1025.

[9] SEILER, H. E. *Ibid.*, 1928, ii, 982.

[10] SEILER, H. E., and GILMOUR, M. D. *Ibid.*, 1931, i, 1112.

[11] MERREWETHER, E. R. A. *Journ. Ind. Hyg.*, 1930, **12**, 198 and 239.

[12] MERREWETHER, E. R. A., and PRICE, C. W. Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry. London: H.M. Stationery Office, 1930.

[13] WILLIS, H. S. "Pneumonoconiosis and Tuberculosis," *Medicine*, 1930, **9**, 413. (A valuable monograph with many references.)

[14] Royal Commission on Metalliferous Mines and Quarries. Second Report, 1914. Cd. 7476, p. 146.

[15] IRVINE, L. G. A Review of the History of Silicosis on the Witwatersrand Goldfields. Silicosis. Records of the International Conference held at Johannesburg, 1930, p. 188. Geneva: International Labour Office, 1930.

[16] WOOD, W. B. *Tubercle*, 1929, **10**, 353.

[17] BADHAM, C. Notes on a Fine Type of Fibrous Pneumonokoniosis produced by Silicates and other Minerals. Studies in Indust. Hyg., No. 13, Rep. Dir.-Genl. Pub. Health, New South Wales, for 1927, Section L-E, Indust. Hyg., p. 103. Sydney, 1929.

[18] Russell, A. E., Britten, R. H., Thompson, L. R., and Bloomfield, J. J. The Health of Workers in Dusty Trades. II. Exposure to Siliceous Dust (Granite Industry), *U.S. Pub. Health Bull.*, No. 187, 1929, p. 87.

[19] Pancoast, H. K., and Pendergrass, E. P. *Amer. Journ. Roentgenol. and Rad. Therapy*, 1931, **26**, 591.

[20] Haynes, F. Annual Report Safety in Mines Research Board for 1928, p. 71. London: H.M. Stationery Office, 1929.

[21] *Idem.* Annual Report Safety in Mines Research Board for 1929, p. 52. London: H.M. Stationery Office, 1930.

[22] Keating, N. Personal Communication.

[23] Cooke, W. E., and Hill, C. F. *Journ. Roy. Micros. Soc.*, 1927, **47** (Series III), 232.

[24] Gloyne, S. R. *Lancet*, 1932, i, 1351.

[25] *Idem. Tubercle*, 1933, **14**, 208.

[26] Stewart, M. J. *Brit. Med. Journ.*, 1928, ii, 509.

[27] Haddow, A. C. *Ibid.*, 1929, **2**, 581.

[28] Stewart, M. J., and Haddow, A. C. *Journ. Path. and Bact.*, 1929, **32**, 172.

[29] Gloyne, S. R. *Tubercle*, 1931, **12**, 158.

[30] Gardner, L. U., and Cummings, D. E. *Journ. Indust. Hyg.*, 1931, **13**, 65 and 97.

[31] Schuster, N. *Journ. Path. and Bact.*, 1931, **24**, 751.

[32] Gloyne, S. R. *Tubercle*, 1929, **10**, 404.

[33] Tylecote, F. E., and Dunn, J. S. *Lancet*, 1931, ii, 632.

[34] Simson, F. W. Ann. Rep. S. African Institute for Med. Research, 1929, p. 64.

[35] Stewart, M. J., Tattersall, N., and Haddow, A. C. *Journ. Path. and Bact.*, 1932, **35**, 737.

[36] Wood, W. B., and Gloyne, S. R. *Lancet*, 1931, ii, 954.

[37] Report on Conference between Employers and Inspectors concerning Methods for Suppressing Dust in Asbestos Textile Factories. London: H.M. Stationery Office, 1931.

[38] Statutory Rules and Orders, 1931, No. 344. London: H.M. Stationery Office.

[39] Statutory Rules and Orders, 1931, No. 341. London: H.M. Stationery Office.

[40] Lovisetto, D. Pulmonary Asbestosis. Records International Conf. on Silicosis, Johannesburg, 1931, p. 506. Geneva: International Labour Office, 1931.

[41] Mussa, G. "Clinical and Radiological Notes on Pneumoconiosis due to Asbestos." *Ibid.*, p. 493.

[42] Loriga, G. "Pneumoconiosis in Italy." *Ibid.*, p. 493.

[43] Simson, F. W. *Brit. Med. Journ.*, 1928, i, 885.

[44] Statutory Rules and Orders, 1931, No. 1140. London: H.M. Stationery Office.

[45] Cooke, W. E. *Brit. Med. Journ.*, 1932, i, 656.

[46] "Asbestos, its Sources, Extraction, Preparation, Manufacture and Uses in Industry and Engineering." Berlin: Becker and Haag, 1928.

[47] *Asbestos* (a monthly trade journal published in Philadelphia, Pa.).

[48] Dewirtz, A. P. *Arch. f. Dermat. u. Syph.*, 1930, clxi, 1.

*(To be continued.)*

than for the thoracoplasty. If we take a broad-minded view of the presence of bacilli, and allow an open tuberculous case to restart work, it is always as a result of mature reflection. We take the patient's personal circumstances into consideration, and see that all necessary precautions are observed.

The long cure that we deem absolutely necessary after the operation, and which is mostly dependent on pathological-anatomical considerations, is a sufficient proof that we consider plombage as simply a process of collapse, which, in a relatively limited number of patients, can be applied with advantage.

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## A MEMORANDUM ON ASBESTOSIS.

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*(Continued from p. 81.)*

### VI.—Onset and Maturation of the Disease.

Within certain high and low limits, the concentration of 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. These limits will be discussed later as they have an important bearing on the scope of preventive measures.

Asbestos has a low specific gravity, is comparatively brittle, and the fibres may be split longitudinally, apparently to an indefinite degree, since there is no ultimate fibre comparable to a vegetable fibre such as cotton. Asbestos fibres have been measured down to a diameter of 0·00075 mm., but it can be demonstrated that even the finest filament measured is itself composed of fine threads (Ross [1]).

Any manipulation of asbestos, therefore, produces dust, which is easily projected into the air. Different varieties and grades of asbestos also differ materially in dustiness.

Exposure to asbestos 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. Thus, amongst 89 asbestos workers with under five years' employment examined in 1928 and 1929, no case of diffuse fibrosis clearly due to asbestos was discovered, and amongst 749 such workers examined recently by the Silicosis and Asbestosis Medical Board, only four cases of asbestosis were found. Amongst those working who have been employed between five and ten years the incidence rate is appreciable, and after ten years' employment a steep rise in the incidence rate occurs, and of those employed for twenty years or longer more than one-fourth were found by the Board to be affected. The incidence rates for the periods of employment 0-4 years, 5-9 years, 10-19 years, and 20 years and over, found from the

examination of 1,512 workers by the Board, are proportionate to the figures 1, 5·6, 30·4, 53·2.

While these figures show clearly the increasing risk with continued employment, it would be wrong to assume that the opposite inference which the figures appear to support—that so long as the period of exposure does not exceed five years the risk of contracting asbestosis is almost negligible—is correct.

Unfortunately, that inference is wholly untenable. The fact is 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. It has been shown that experimentally in guinea-pigs fibrosis due to inhaled asbestos commences after about five hundred days' exposure, and the inhaled dust trapped in the respiratory bronchioles remained localised there for at least two-and-a-third years (the length of the experiments up to the time of the report [30]).

Of the fatal cases in Great Britain of which full particulars are available, in two, examination over fourteen years after the last exposure to asbestos revealed numerous asbestosis bodies. Since these bodies are merely altered asbestos fibres and still contain an internal core of asbestos, and since unaltered asbestos fibres also can be found in the lungs years after the last exposure, it is evident that asbestos dust trapped in the lungs remains and continues to exert its fibrosis-producing powers for at least many years.

Examination of the particulars concerning cases of asbestosis in conjunction with the relative dustiness of the processes at which they worked leads to the conclusion that, while increasing concentrations of dust in the air reduce the period before developed asbestosis occurs, after a certain high concentration of dust in the air is reached a further increase in concentration does not further reduce the period of "maturation," as it may be called, of the fibrosis. Similar considerations also show that below a certain concentration (which will be referred to later), development of a disabling degree of asbestosis will not occur within the space of an average working lifetime.

It follows, therefore, and this is important from the practical point of view of prevention, that a certain minimum "fibrosis-producing amount," as it were, of asbestos dust must be trapped in the lungs in order to produce a potentially disabling or serious amount of fibrosis, and also that a certain "maturation" period must elapse before that amount of fibrosis is developed.

Confirmation that this hypothesis is, from the practical point of view, an accurate statement of the position, can be adduced from particulars of cases alive to-day, and from particulars of deaths which are now available. Certainly this minimum dose of dust can be trapped in the lungs under ordinary industrial conditions in under five years. Two examples will illustrate this:—

*Mattress worker*, employed four years and five months, died eight years after leaving this work at the age of 31. Four advanced asbestosis with no tuberculosis (verified by post-mortem examination).

*Miner*, employed two years and seven months, died twelve years later, at the age of

48, from a moderate degree of asbestosis, together with a superimposed tuberculosis of twelve to eighteen months' duration (verified by post-mortem examination).

Particulars of cases seem to show that with 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—which include not only the trapping period of the fibrosis-producing dose of dust, but also the maturation period of the fibrosis, which periods, of course, overlap.

The existence of this, which may be referred to as the fibrosis-producing period, i.e., the period which must elapse from the date of commencing work before a serious degree of asbestosis can be produced, accounts for the fact that it is not until the second five years of employment is reached that appreciable numbers of cases of asbestosis are discovered.

This period of seven years is, of course, the minimum, and few cases mature in this minimum period; in successive years, however, depending on the dustiness of the process in which employed, more cases mature. In the more dusty processes the fibrosis-producing period, as defined above, is commonly eleven years.

At this point, i.e., when fibrosis of serious import has matured—the worker is unduly short of breath on any extra exertion, has a tinge of cyanosis of the lips, and a little dry cough, mostly in the mornings. He is disinclined to climb stairs or walk up hills and he may even have changed from an upper flat or room to a lower one to avoid climbing the stairs. He still, however, remains at work and usually is not anxious about the state of his health; many refuse to admit any deterioration in health at this stage.

## VII.—Course of the Disease, Disablement Produced, and Prognosis.

What then is the outlook for those who have reached this stage of developed asbestosis?

This question, of crucial significance to the individual, the industry, and the legislature may be considered conveniently under three headings:—

### (1) *Does Asbestosis Shorten Life?*

The answer to this question is emphatically "Yes." Reference to the following tables will show that this is so.

Table I gives particulars of forty-two fatal cases of asbestosis or asbestosis with tuberculosis of which precise information is available; all except two were verified by post-mortem examination.

In Table II comparison is made between these 42 cases and 281 consecutive fatal cases of silicosis or silicosis with tuberculosis of which full particulars are available.

In Table III the cases of silicosis and of silicosis with tuberculosis are distributed in the industries concerned.

It will be observed that:—

(1) The average duration of employment sufficient to cause a fatal degree of asbestosis was only 15·2 years as compared with 40·1 years for

TABLE I

| Disease | Number of deaths | Age at death | | | Duration of employment in asbestos industry | | | Duration of exposure to asbestos dust in years | | | Period elapsing between last exposure to asbestos dust and death in years | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Longest | Shortest | Average | Longest | Shortest | Average | Longest | Shortest | Average | Longest | Shortest | Average |
| Asbestosis | 27 | 63 | 26 | 40·8 | 27 | 4·4 | 15·2 | 27 | 4·4 | 13·3 | 14·3 | 2 days | 2·96 |
| Asbestosis with tuberculosis | 15 | 67 | 22 | 41·6 | 24 | 2·6 | 11·7 | 24 | 2·6 | 11·3 | 12·2 * | 0·3 * | 4·6 * |

* Particulars only known for 12 cases.

TABLE II.

| Disease | Number of deaths | Average age at death | Duration of employment in years | | |
|---|---|---|---|---|---|
| | | | Longest | Shortest | Average |
| Silicosis .. .. .. | 114 | 54·1 | 57·0 | 2·8 | 40·1 |
| Silicosis with tuberculosis | 167 | 52·0 | 67·0 | 2·0 | 32·0 |
| Asbestosis .. .. .. | 27 | 40·8 | 27·0 | 4·4 | 15·2 |
| Asbestosis with tuberculosis | 15 | 41·6 | 24·0 | 2·6 | 11·7 |

TABLE III.

| Industry | Number of deaths | Average age at death | Duration of employment in years | | |
|---|---|---|---|---|---|
| | | | Longest | Shortest | Average |
| Pottery— | | | | | |
| Silicosis .. .. .. | 72 | 55·6 | 57·0 | 10·0 | 39·8 |
| Silicosis with tuberculosis | 75 | 55·2 | 67·0 | 13·0 | 37·9 |
| Sandstone— | | | | | |
| Silicosis .. .. .. | 21 | 57·3 | 57·0 | 20·0 | 40·4 |
| Silicosis with tuberculosis | 39 | 52·3 | 53·0 | 16·0 | 34·8 |
| Grinding of metals— | | | | | |
| Silicosis .. .. .. | 4 | 47·3 | 45·0 | 18·0 | 30·8 |
| Silicosis with tuberculosis | 26 | 51·1 | 48·0 | 2·8 | 30·2 |
| Sandblasting— | | | | | |
| Silicosis .. .. .. | 7 | 40·7 | 16·0 | 4·5 | 10·3 |
| Silicosis with tuberculosis | 16 | 44·2 | 20·0 | 2·5 | 8·3 |
| Manufacture of scouring powders— | | | | | |
| Silicosis .. .. .. | 3 | 37·0 | 11·0 | 5·25 | 7·8 |
| Silicosis with tuberculosis | 2 | 33·5 | 10·75 | 2·0 | 6·4 |
| Miscellaneous— | | | | | |
| Silicosis .. .. .. | 7 | 55·8 | 45·0 | 2·8 | 20·4 |
| Silicosis with tuberculosis | 9 | 50·9 | 34·0 | 11·0 | 21·0 |

all cases of silicosis. The actual average length of exposure to asbestos dust was, however, still less, and not more than 13·3 years.

(2) The shortest length of exposure to asbestos dust which ultimately caused death from advanced fibrosis of the lungs was 4·4 years.

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

(4) Although the figures in the asbestosis group are very small, it will be observed that in 35·7 per cent. the disease was accompanied by tuberculosis, while in the silicosis group 59·4 per cent. were accompanied by tuberculosis, suggesting, so far as the figures go, a lesser incidence of tuberculosis in asbestos workers compared with workers in the silicosis-producing industries.

Further confirmation is shown by the after-history of the 95 cases of asbestosis and five cases of asbestosis with tuberculosis found in the original inquiry in 1928. Of these 100 cases, although a number have migrated from the industry and have been lost sight of, 17 are known to have died, 10 from asbestosis, six from asbestosis with tuberculosis, and one from carcinoma of the pancreas, in whom also was found on post-mortem examination a considerable degree of asbestosis. Of the remainder, 13 are partially or wholly disabled on account of the disease and have been given certificates to that effect by the Silicosis and Asbestosis Medical Board. The toll of the disease is evident even in these incomplete figures.

### (2) *The Extent of the Disablement produced by Asbestosis.*

The amount of disablement produced by the development of asbestosis is surprisingly slight for a number of years. This is partly due to the character of the disease, and partly due 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, until the condition is advanced, although increasing inconvenience from shortness of breath on exertion 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.

### (3) *The Outlook for the Individual Affected Worker.*

The ultimate prognosis in those with developed asbestosis or in those who have incarcerated in their lungs a fibrosis-producing amount of asbestos dust, even though no appreciable fibrosis or only a slight amount can be detected, is bad. Death from the disease is inevitable sooner or later, if life is not cut short by accident or by some unconnected disease.

The immediate prognosis in any individual case is uncertain. With the presence of a moderate degree of asbestosis, many other factors, such as the worker's level of resistance to infection, his general health, the ease or arduousness of the daily round, and the state of his circulation, become important. These are only approximately calculable in each individual, and

the course of the disease and the immediate outlook for the patient are dependent far more on these extraneous considerations than on the precise degree of fibrosis present in the lungs.

It is remarkable to what extent the lungs can be affected and yet life in a fair degree of comfort remain. The reserve is, however, so slender 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 the fatal outcome. It is apparent, therefore, that while the onset of an acute respiratory infection is most ominous, the development of any disease which directly or indirectly throws an added strain on the heart or lungs is of serious import.

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

## VIII.—The Fibrosis-producing Amount of Asbestos Dust, and the Influence of Work in Different Processes.

As shown previously, if a certain, and as yet unknown, amount of asbestos dust is trapped in the lungs, death due to the development of asbestosis is an inevitable sequel, provided the worker is not carried off from some unassociated disease or from accident during the maturation period of the fibrosis.

It is important, therefore, 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 standpoint, can be inhaled with impunity.

Table IV.—Comparison between the Incidence Rates of Definite Fibrosis (Asbestosis) amongst Workers employed in Group (iii) (Less Dusty Processes) and the Corresponding Incidence Rates amongst Workers employed in Groups (i), (ii), (iv) and (v) taken together (More Dusty Processes).

| — | Incidence rates of asbestosis per cent. | | | |
|---|---|---|---|---|
| | 0-4 years employed | 5—9 years employed | 10—19 years employed | 20 and over years employed |
| Group (iii). (Number examined 500) | 0 | 2·4 | 8·3 | 5·0 |
| Groups (i), (ii), (iv) and (v) taken together. (Number examined 845) | 0·7 | 2·1 | 19·0 | 40·6 |

If only the slightest exposure to the dust results ultimately in death, then the scope of the necessary preventive measures is summed up in one word—prohibition—for, practically speaking, it is impossible to prevent such exposure. Fortunately, however, there is evidence that an appreciable amount of dust must be incarcerated in the lungs to produce a serious

degree of fibrosis. Inquiry into the relative dustiness of various processes and the relative incidence rates of asbestosis amongst workers in those processes provides data which go far to confirm this point of view.

The processes concerned may be divided into six groups in which similar processes are grouped together as follows: (i) Crushing, opening, disintegrating and mixing; (ii) carding; (iii) spinning, twisting, doubling, plaiting, &c.; (iv) insulating mattress making; (v) weaving and associated processes; (vi) miscellaneous.

The groups of processes differ in their average levels of dust production, that of group (iii) being easily the lowest in the absence of any dust suppression devices. Certain operations cause intense clouds of dust, although sometimes only momentarily. With high concentrations of dust, estimations of particle count are liable to gross errors of underestimation, but nevertheless such counts show that some operations may be at least twelve times as dusty as braiding, and probably much more so.

Furthermore, comparison between the incidence rates of asbestosis amongst workers in group (iii), those working in the less dusty processes and termed "spinners" for short, and the incidence rates amongst workers in the more dusty processes, groups (i), (ii), (iv) and (v) taken together, shows (Table IV) how much more serious is the risk in the more dusty processes. Moreover, the distinction is still more marked than is indicated in the table. The majority of spinners have either worked for a period in more dusty processes or have been or are exposed to dust generated in neighbouring more dusty processes. Also those employed at ring spinning, which is definitely a dusty process, and more so than flyer spinning, braiding and plaiting, are included in group (iii). The evil effects of exposure to high concentrations of asbestos dust for even a year or two has been already commented on. These factors have operated to increase the incidence of asbestosis amongst spinners, and in consequence the true incidence rates for this group are even less than those given in Table IV, and particularly so in the earlier years of employment. It seems clear, therefore, that amongst workers in the less dusty group of processes, the incidence of asbestosis is very much less than amongst workers in the more dusty processes. Examination of the average length of employment of cases of asbestosis, of the radiograms, and of the particulars of the recorded fatal cases, also indicates that not only are "spinners" less likely to contract asbestosis, but when they do, it takes longer to develop; in other words, in "spinners" the maturation period of the fibrosis is longer than in workers in the more dusty processes.

For these reasons and from examination of the varying conditions of exposure to dust amongst "spinners" in individual factories, it appeared reasonable to infer that the exposure of workers in this group to dust, as a whole, was not greatly in excess of the maximum safe limit.

One of the conclusions, therefore, of the original investigation was "that in order to prevent the full development of the disease amongst asbestos workers within the space of an average working life-time, it is necessary to reduce the concentration of dust in the air of the workrooms to a figure below that pertaining to spinning at the time over which these cases were exposed."

This deduction was, later, accepted, in the light of the evidence

available, and with the reservation that it was subject to alteration in conformity with further medical experience, as a safe basis on which it would be possible to work out appropriate dust suppression methods. For this purpose, therefore, the conditions arising from flyer spinning carried on without exhaust under good general conditions was considered as the safe criterion and was termed the "dust datum." The application of this dust datum is considered below.

## IX.—The Risk from Tuberculosis.

As mentioned previously, the risk to life associated with asbestos is a complex one. The fibrosis of the lungs itself, when it reaches an extreme degree, kills by mechanical embarrassment of the pulmonary circulation, causing back pressure and consequent heart failure. The presence of fibrosis in much less amount, while apparently not predisposing to the onset of acute respiratory infections, such as bronchopneumonia and pneumonia, does most adversely affect the chances of recovery from these diseases. Thirdly, there is the unknown influence of asbestosis on the course and outcome of totally unrelated diseases which cause added strain on the circulatory and respiratory systems.

In a category by itself comes pulmonary tuberculosis. The importance of determining whether or not the presence in the lungs of asbestos dust, or of asbestos dust and of fibrosis due to it, predisposes to the development of pulmonary tuberculosis cannot be overstressed. It does not follow, of course, that because pulmonary tuberculosis is so definitely an added risk in silicosis that similar considerations apply to asbestosis. The evidence, unfortunately, is at the moment insufficient to settle the question.

Gardner and Cummings [30] have shown that, experimentally, in guinea-pigs, primary tuberculosis infection is influenced only to a limited degree by inhaled asbestos and that the tendency to healing by fibrosis is marked. In this respect they emphasise the contrast between the effect of asbestos and of free silica in the form of quartz dust, with which the tendency was overwhelmingly towards the production of generalised chronic tuberculosis of the lungs and viscera.

These same workers found that, in another group of experiments where the tuberculous infection was implanted on to an existing asbestos fibrosis, 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.

Available data on this point relating to asbestos workers are very meagre, as might be expected, having regard to the comparatively small population at risk. Burton Wood and Gloyne [36] state that in their series of 57 cases of pulmonary asbestosis there were 10 cases of active tuberculosis. Nearly all these 57 cases were employed at the same factory where there were some 350 workers. It is probable, as they state, that the cases with tuberculosis are more prone to seek advice and that their

proportion with tuberculosis represents a maximum. Even so, the figures they produce emphasise the urgent need of full inquiry into this problem. These observers also make the important statement that obsolescent tuberculosis may remain quiescent in spite of exposure to asbestos dust.

Equally too, examination of workers whilst at work cannot indicate the true incidence of pulmonary tuberculosis, but such data are also suggestive. Of 374 workers examined while at work only four showed signs of active tuberculosis. The Silicosis and Asbestosis Medical Board, in the first cycle of periodic examinations of workers in the industry, found only one case of the mixed condition (asbestosis with tuberculosis) in 1,512 examinations.

Generally on post-mortem examination of the lungs of asbestos workers one finds that the cases showing active tuberculosis fall into three groups: (1) Those with evidence of rapid tuberculosis and very slight or no signs of asbestosis; (2) those with much fibrosis due to both diseases; (3) those with advanced asbestosis and a slight amount of active tuberculosis.

Of those in group (i) a number are definitely of the adolescent type, and the incidence rate of pulmonary tuberculosis in young women, in whom the majority of these cases have occurred, has increased in recent years. Unless, therefore, it can be shown that the presence of asbestos dust in the lungs favours the growth of the tubercle bacillus, this group of cases has no bearing on the question of an added risk of pulmonary tuberculosis in asbestos workers, since they would have occurred in the absence of exposure to asbestos.

From the experimental work of Gardner and Cummings, quoted above, it appears not unreasonable to deduce that, since the coincident inhalation of asbestos dust did not affect the ultimate healing of the lesions produced by a tuberculous infection of low virulence, whereas the inhalation of quartz not only prevented healing, but induced a generalised spread of the tuberculous infection, therefore the coincident inhalation of asbestos dust and tubercle bacilli of greater virulence would result in an infection, the outcome of which would be precisely the same, so far as risk to life is concerned, as if there had been no coincident exposure to asbestos dust.

If this hypothesis is correct, and the experimental work referred to does more than suggest it, the important practical point emerges that we can dismiss, as being unconnected with asbestos dust, the whole of the deaths in group (i) and a few of the deaths in group (ii). This is of major importance in connection with the employment of young persons in the industry.

The remaining cases in group (ii) include those cases of fibroid tuberculosis who become exposed to asbestos dust. In these the effect of the asbestos dust is to cause more fibrosis than would occur with either agent acting independently, but whether in the long run its influence is favourable or unfavourable would appear to depend on factors peculiar to each affected person. The number of these cases, however, obviously must be negligible. In this group, however, must be placed the apparently small number of cases in which, in spite of the presence of extensive asbestosis, a virulent, rapidly caseating tuberculosis does occur. These occur in middle life and would appear to depend more on the virulence of the particular strain of tubercle bacilli than on the presence of asbestos dust.

In the third group of cases where there is found on post-mortem examination much asbestosis and comparatively little active tuberculosis, the tuberculosis is the terminal and the immediate cause of death.

In these cases there is an advanced degree of asbestosis which, in itself, must cause death in the not too distant future; the tuberculous lesions, however, although of limited extent and evidently insufficient to cause death in a normal person, do in these cases cause death by obliterating the small remaining reserve of sound lung tissue over and above that just sufficient to maintain life. In these cases it is not the presence of asbestos dust in the lungs but the results of it, in the form of gross fibrosis, which have contributed to the fatal outcome. Certainly any other infection of the lungs or any other illness whatsover which demanded a reserve of sound lung tissue in excess of that actually available would have had the like effect. Death in these cases, therefore, is not the result of any particular adjuvant effect of asbestos dust on a tubercular infection.

To sum up on this question of added risk to asbestos workers from pulmonary tuberculosis, while the data, as yet, are far too fragmentary to be dogmatic on this subject, the burden of evidence points to the assumption that whatever added risk there may be it is less than that associated with silicosis.

The inhalation of asbestos dust does not appear to affect old inactive pulmonary tuberculosis, nor does it appear to modify the end-result of coincident tubercular infection.

While the presence of asbestos dust at any rate temporarily induces an exacerbation of a tubercular infection, it also conduces to the development of more fibrosis than would have occurred in its absence, which is a localising and reparative process. On the other hand, this effect results in permanent loss of function in a greater area of lung tissue with probably ultimately unfavourable results in individual cases.

A tubercular infection may, precisely as with other acute pulmonary infections, precipitate death in cases of advanced asbestosis, where, had the lungs been normal, recovery would have taken place.

The factors of prime importance, which are, of course, not local but general in their application, are massiveness of dose and virulence of the tubercular infection.

The evidence, therefore, points to the hypothesis that at present the added risk to asbestos workers from pulmonary tuberculosis is limited and definitely less than in workers exposed to free silica; virulent massive infections will, as in the general population, cause death; lesser ones will result in more widespread fibrosis than would appear in cases amongst the general population, and slight infections, in common with many other affections, are likely to precipitate death in cases of advanced asbestosis where they would not do so in the general population.

This appears to be the position at the moment; whether this risk will alter as the effects of measures for dust suppression in the industry become apparent, as is apparently happening in the silicosis-producing industries, cannot be foretold as yet.

*(To be continued).*

(3) It is essentially necessary to control pneumothorax cases with serial skiagrams.

(4) The hypothesis is advanced that sanecrysin through stimulation of the R.E.S. produces an immunologic (or immunobiologic) response in the body.

(5) Sanocrysin in conjunction with pneumothorax represents a worthy advance in the treatment of pulmonary tuberculosis.

In conclusion, I wish to acknowledge my indebtedness to Dr. H. C. Calvey for his very valuable help in taking the skiagrams; to Mr. Hall for aid in the preparation of this article; and to Dr. H. G. Trayer, Medical Superintendent, for permission to publish hospital material.

REFERENCES.

[1] PETERS, A. *Amer. Rev. Tub.*, 1928, **17**, 348.
[2] GILLIES, S. *Med. Journ. Austral.*, 1930, **ii**, 119.
[3] FINDLAY, G. M. "Recent Advances in Chemotherapy," 1930, p. 473.
[4] KRITSCHEWSKI, I. L. *Zentralbl. f. Bakt.*, 1927, **5**, 104.
[5] JUNGEBLUTT, C. W. *Zeitschr. f. Hyg. u. Infect.*, 1927, **107**, 357.
[6] FELDT, A., and SCHOTT, A. *Ibid.*, 1927, **107**, 453.
[7] JIMINEZ DE ASUA, F., and KUHN, M. J. *Compt. Rend. de la Soc. Biol.*, 1928, **99**, 1414.
[8] KOLMER, J. H., and SCHAMBERG, J. F. *Amer. Journ. Syph.*, 1933, **17**, 187.
[9] SCHRÖDER, G. *Zeitschr. f. Laryngol.*, 1932, **23**, 273-279.
[10] HUGHES, T. A., and SHRIVASTAVA, D. L. *Brit. Med. Journ.*, 1930, **ii**, 248.
[11] HOUGHTON, L. E. *Tubercle*, 1932, **13**, 393.
[12] SABIN, F. R. *Ibid.*, 1932, **13**, 206.
[13] CLARKE, B. R. *Brit. Med. Journ.*, 1929, **ii**, 576.

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O.K ✓

## A MEMORANDUM ON ASBESTOSIS.

BY E. R. A. MEREWETHER, M.D.

(*Continued from p.* 118.)

### X.—PREVENTIVE MEASURES.

As noted above (pp. 69), the risk from asbestosis in the asbestos industry is no less grave than the most serious risks from silicosis in the silicosis-producing industries.

While mortality rates for this disease amongst asbestos workers are not obtainable, all the evidence, and it is not inconsiderable, goes to show that in the absence of all preventive measures the appraisement of the risk to workers in the most dusty processes, which was made by the first recorded case and quoted previously (pp. 72) is substantially correct: of those employed in such processes continually from leaving school few will survive ten years' exposure and none will reach the age of 30.

Fortunately, in Great Britain, for more than twenty years, some measure of dust suppression, though an inadequate one, has been applied in the industry. This has come about by virtue of Section 74 of the Factory and Workshops Act, 1901, persuasion by H.M. Inspectors and co-operation of important firms in the industry in consequence of the vague belief, which could not be supported by evidence, that the dust was harmful. In some processes such as carding the amount of dust suppression has been considerable for quite this period.

On the production of proof of the existence of the specific disease, and of its incidence and serious nature, the gravity of the position and the necessity of enforcing a high standard of ventilation and of dust suppression was realised.

The usual steps under Section 79 of the Factory and Workshops Act of 1901 were then taken, the Secretary of State certifying that the manipulation of asbestos and the manufacture or repair of articles composed wholly or partly of asbestos and processes incidental thereto are dangerous, and a code of Regulations designed to abolish the risk was drawn up. These Regulations were termed the Asbestos Industry Regulations, 1931 [44] and by the direction of the Secretary of State came into force, with the exception of a few, on March 1, 1932. The excepted Regulations came into force six months and twelve months later, it being impossible to carry out the extensive alterations and additions to premises and ventilating machinery required under them by the date specified for the main body of the code. The complete code of regulations for the dust-producing processes of the industry has been in force therefore since March 1, 1933.

The carrying out of these regulations casts no light burden on the industry, nor could they have been applied in their entirety and with full effect within three years from the date of the certificate of the Secretary of State without the whole-hearted co-operation of the trade.

Problems of ventilating engineering of the utmost difficulty had to be faced, 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.

That this was the case was realised from the outset, and immediate steps were taken to arrange a conference of asbestos textile manufacturers and representatives of the Home Office to consider these practical difficulties and the best methods which could be adopted generally for suppressing dust in manufacturing and other processes.

The Conference agreed unanimously that everything practically possible should be done to suppress dust, and the manufacturers promised their full assistance to secure this result. It was then decided to set up a joint committee to consider the problem in detail and to collect all information available concerning dust suppressing methods.

The high public spirit shown by the leading British manufacturers in the industry, particularly in their readiness to pool their experience in regard to the difficult problems to be solved, enabled this Committee to do invaluable work.

Further experimental work was done and new methods tried out in various factories and by the expenditure of much time and labour, the

Committee were enabled to report in January, 1931, within six months of its constitution.

Its report is set out in a series of twenty-four agreements covering the main processes concerned and embodying concrete and detailed recommendations of a practical nature for dust suppression [37].

The Committee agreed on a practical standard of dust suppression based on the considerations previously discussed in this memorandum (Section VIII) and concluded, on the evidence available, that "For practical purposes, the conditions arising from flyer spinning 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."

The Agreements and Recommendations put forward in its report are aimed at the reduction of dust in other processes to this dust datum level. The Report of this Committee, together with certain suggestions of the General Council of the Trades Union Congress to whom the Report was communicated, were taken into account in drafting the Code of Regulations previously referred to which gave statutory effect to the Agreements arrived at by the Conferences.

These Regulations apply the following principles, as and where requisite, to achieve the agreed 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 material 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 (i.e., under the age of 18) in the most dusty processes.

Other preventive measures in force 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 (Section 12 of the Silicosis and Asbestosis (Medical Arrangements) Scheme, 1931) and, cases of fibrosis and of pulmonary tuberculosis are detected at the earliest possible moment. These are more conveniently discussed in the next section in connection with compensation for asbestosis.

## XI.—Compensation for Asbestosis.

In Great Britain by the Workmen's Compensation Act, 1925, as extended by the Workmen's Compensation (Silicosis and Asbestosis) Act, 1930, the Secretary of State was empowered to make a scheme of compensation for industries and processes involving exposure to asbestos dust, and also a general Scheme for co-ordinating the medical arrangements in connection with this Scheme and with all the schemes of compensation for silicosis in the silicosis-producing industries and processes.

Following this the Asbestos Industry (Asbestosis) Scheme, 1931 [38], providing for compensation for asbestosis and asbestosis accompanied by tuberculosis, was made and came into force on June 1, 1931. It applies to all workmen employed at any time on or after May 1, 1931, in any of a comprehensive schedule of specified processes. On the same date the Silicosis and Asbestosis (Medical Arrangements) Scheme, 1931 [39], came into force. This Scheme effectively simplified and co-ordinated the different medical arrangements already in force in connection with compensation for silicosis or silicosis accompanied by tuberculosis, and also applied the same general procedure in connection with compensation for asbestosis or asbestosis accompanied by tuberculosis.

Under this Scheme a whole-time Medical Board of specially qualified practitioners, supervised by a Chief Medical Officer, with the view of securing a uniform standard of efficiency, is charged with the duties of Periodical Medical Examination of workers employed in the specified industries or processes, of examination of workmen in connection with claims for disablement from these diseases, and of granting of the appropriate certificates in connection with disablement or death claimed to be due to these diseases. The Medical Board acts in different areas of the country by panels of not less than two members of the Board, and all certificates are granted with the authority of not less than two members, and are conclusive evidence of the matters therein certified in pursuance of the provisions of the Scheme. Furthermore, under Section 12 of the Scheme every new entrant must be examined, within two months of commencing employment, by a member of the Board or by other duly qualified medical practitioner specially appointed by the Secretary of State for the purpose. If he fails to satisfy the following requirements with respect to physique: (1) The chest must be at least of average development and the respiratory passages must be free from obstruction; (2) there must be no signs of disease of the lungs or heart; and (3) there must be no tuberculosis of any region; the case must be referred to the Board, who, if satisfied, must suspend the workmen from further employment in the industry or process, and shall certify accordingly.

The prescribed interval between the periodical examinations of asbestos workers is one year, and if a worker is found to be suffering from asbestosis or from tuberculosis to such a degree as to make it dangerous for him to continue work in the industry or process the Board must suspend the workman, and certify accordingly.

In the case of a death claim, the certificate of the Board can only be given after a post-mortem examination, except only in such cases where the workman at the time of his decease was in receipt of weekly payments

under the Compensation Scheme, and the Board is satisfied that a post-mortem examination is unnecessary.

The Schemes, therefore, in their application to asbestos workers, provide for the periodical medical examination of all workers exposed to any appreciable risk of developing asbestosis, for compensation for death and for partial or total disablement due to asbestosis or asbestosis accompanied by tuberculosis, for the suspension of workers suffering from asbestosis, asbestosis accompanied by tuberculosis, or tuberculosis to such a degree as to make it dangerous for them to continue work in the processes, and for the exclusion of new entrants who are unsuitable on medical grounds for employment in the industry.

It will be observed that, broadly speaking, these Schemes are effective in three directions:—

(1) They protect the worker and his dependants by providing expeditiously and economically for compensation if death or disablement is produced by asbestosis, by minimising any added risk from tuberculosis, and by preventing those who, on medical grounds, are especially liable to be adversely affected by work in the industry, from entering it.

(2) They protect the employer by raising the physical standard of entrants into the industry and by provision for expert scrutiny, on a uniform standard throughout the country, of claims for compensation.

(3) The data obtained from the records of the Board's examinations form the ultimate and only reliable criterion of the effectiveness of the preventive measures adopted in the industry.

## XII.—Asbestosis in Other Countries.

It appears that other European countries as well as Great Britain have experienced, prior to the Great War, isolated or vague warnings that the inhalation of asbestos dust might be dangerous, but that generally, owing to a variety of causes which have been discussed already, it was not recognised that these warnings were portents of a serious risk in the industry.

Thus, in the French official *Bulletin de l'Inspection du Travail* for 1906 [4], the year in which Dr. Montague Murray gave his evidence before the Departmental Committee on Compensation for Industrial Diseases, there appeared "Note sur l'Hygiène et la Sécurité des Ouvriers dans les Filatures et Tissages d'Amiante," by M. Auribault. He states that the atmosphere of the workrooms, "surchargée d'une multitude de particules siliceuses," is an eminently suitable milieu for the production of an occupational phthisis identical with those of potters, millstone makers, &c., and he gives "un exemple frappant" to corroborate this inference. He continues: "En 1890, une usine de filature et de tissage d'amiante s'établissait dans le voisinage de Condé-sur-Noireau (Calvados). Au cours de cinq premières années de marche, aucune ventilation artificielle n'assurait l'évacuation directe des poussières siliceuses produites par les divers métiers; cette inobservation totale des règles de l'hygiène occasiona de nombreux décès dans le personnel: une cinquantaine d'ouvriers et

d'ouvrières moururent dans l'intervalle précitée; le Directeur, précédemment propriétaire d'une filature de coton à Gonneville (Manche), avait recruté 17 ouvriers parmi son ancien personnel; 16 d'entre eux furent enlevés par la chalicose de 1890 à 1895."—How comparable is this to the statement of Montague Murray's case in 1900 that he was the last survivor out of ten employed in the cardroom.—M. Auribault continues that the workmen "justement effrayés," believed erroneously that lead poisoning derived from the asbestos "occasionnait la disparition rapide de leurs camarades." Following this, local exhaust ventilation, at first primitive in type but later improved, was applied to the cards, and the teasers (effilocheuses) were isolated, with the result that the mortality diminished considerably.

This is a valuable picture of the conditions which must have been common to asbestos textile factories in any country in the early days of the industry; moreover, he goes on to recommend ventilation of cards and enclosure of opening machines, and prohibition of employment of young persons under 18 unless there is "une captation rigoureuse des poussières."

The first record in Germany was a demonstration by Fahr to the Medical Society of Hamburg in 1914 of specimens and photomicrographs of a case of pneumonokoniosis in an asbestos worker. Fahr mentioned the occurrence of large numbers of crystals in the lungs but did not describe them more closely. He stated, however, that they had been seen by Marchand and Riesal in 1906, who had speculated on their origin.

In the last year or two a number of fatal cases have been reported from other countries, including South Africa, Germany, the United States of America and Italy.

German observers evidently regard the risk as serious, Kruger, Rostoski and Saupe [3] stating that of 52 workers whom they examined 30 showed definite lung changes. Their general conclusion was that moderately severe asbestosis takes some five years to develop. Of the workers they examined who had had ten years' or more exposure none was free from signs of the disease. From Italy [40], too, comes confirmation of the existence of severe pneumonokoniosis amongst workers, which can reach complete development after a period of seven to nine years, and can cause death after thirteen years' continuous exposure to much dust. Cases with a fatal issue are, however, Lovisetto continues, rare. Mussa [41] states that all workers who had worked for some time in the asbestos works showed lesions of pneumonokoniosis which could be recognised clinically and radiologically; he met with no case of association of pneumonokoniosis with tubercular lesions. Scarpa, however, prior to 1906, found 29 out of 30 asbestos workers to be suffering from tuberculosis [42].

Simson [43] in South Africa examined the lungs of two native asbestos mill workers, one of whom had been employed for twelve months and had died from a miliary tuberculosis, and the other, employed for two years, had apparently never recovered from an attack of lobar pneumonia a year before death. He comments, "the amount of the fibrosis in two of the human cases . . . was quite definite; and if due to the presence of asbestos dust, the initial rate of production was rapid when compared with present day non-infective silicosis on the Rand." The same observer, examining the lungs of a guinea-pig experimentally dusted by Mavrogordato for one hundred hours during a period of fifty-six days,

showed that this exposure ultimately resulted in the development of a slight generalised fibrosis.

Gardner and Cummings in the United States of America, whose detailed researches have been quoted already, showed that, in experimentally dusted guinea-pigs the fibrosis begins in those portions of the lungs where the dust is localised, and that this reaction can be detected in X-ray pictures after about two years' exposure to the concentration of dust used.

Evidence from all quarters, therefore, confirms the risk of pulmonary fibrosis attendant on the inhalation of asbestos dust. While, no doubt, the risk varies in different places and different works, depending on the local conditions and the amount of dust produced, the risk is general wherever more than minimal amounts of asbestos dust are produced in a manufacturing process. Of the risk associated with quarrying of the mineral this country has no experience, since there are no deposits of the mineral in Great Britain which are worked commercially. It is not unlikely that the risk of true asbestosis in quarrying the mineral may be less than in the comparable dusty manufacturing process owing to the low percentage of asbestos in the dust produced in the quarries and the open-air nature of the work, but the risk in the dusty milling and manufacturing processes is patent and serious.

## XIII.—The Outlook.

No discussion, however slight, of the characteristics of a specific occupational disease, of its effects on the workers and its reactions on employers and the State can be considered adequate in the absence of some examination of the position at present and of some appraisal of the prospect before the industry.

In asbestosis we have an industrial disease comparable in its general effects to silicosis, and equally menacing to the industrial host which harbours it. In Great Britain, fortunately, as compared with silicosis, the risk in point of number of workers exposed to the disease is a limited one, but it takes a grievous toll of those workers. When the inevitableness of the outcome of exposure for even a year or two to the high concentrations of asbestos dust commonly encountered in certain operations, and the shortening of life which results, is realised, the imperative and immediate need of adequate preventive measures is apparent.

The aim of these preventive measures is, of course, to make it impossible for new entrants into the industry to develop the disease, and the main agencies by which that goal can be attained are the triad which have served industrial hygiene so faithfully—localised exhaust ventilation, enclosure of dusty operations, and wet methods of manufacture.

A high standard is essential as is evidenced by the failure of past efforts, although those early ones were, however, partially effective. The practical engineering difficulties are great, but can be surmounted. In spite of these difficulties the industry is in a very favourable position, because experience in this country is showing that, in contradistinction to some of the silicosis-producing risks, the problems in the asbestos industry are finite ones and when tackled successfully do not leave any further subsidiary, but at present insoluble, problems.

Apart from the necessity of the application of effective methods of dust suppression to obvious dusty processes, such as opening and carding, and to others less obviously dusty, such as dry weaving and ring spinning, many incidental operations involving the handling of fiberised asbestos have to be eliminated and replaced by non-dusty enclosed mechanical methods.

In this connection a practical maxim of the greatest value is that every translation of fiberised asbestos in the factory produces dust which, if not controlled, is dangerous. It is this principle which underlies the dust suppression methods in use now in Great Britain.

Then, what of the outlook? If we accept that the dust datum level (p. 154) is for practical purposes a safe criterion of dust concentration—and although one cannot be dogmatic on this point as yet, nothing has arisen which casts doubt on its validity as a safe limit—and the dust produced in more dusty processes is suppressed generally to this level, and this standard is conscientiously maintained, then the outlook is bright.

Some years must elapse before the cases representing the accumulated risk disappear from the industry, but then it is believed that the minimal amounts of asbestos dust inhaled by workers under the new conditions will neither shorten life nor cause disablement. The position is strengthened by the evidence that any added risk from tuberculosis is less than in the silicosis-producing industries. Moreover, the added safeguard in this country of periodical medical examination at yearly intervals of almost all asbestos workers exposed to appreciable risk, with suspension from employment in such processes of all cases of open tuberculosis, diminishes this residual risk.

The influence of these three factors, the diminished power of asbestos as compared with free silica of aiding a tuberculous infection, suppression of dust in manufacturing processes to a level below that which will produce a disabling fibrosis within the space of an average lifetime and periodical medical examination of all workers exposed to potential risk, with exclusion of those workers with open tuberculosis, cannot fail to be effective in achieving the common goal. This goal is not the prevention of minimal and microscopical effects of asbestos dust, which must occur however slight the exposure, but the abolition of the risk from the development of diffuse fibrosis, and of any residual added risk from tuberculous infection.

The way is undoubtedly hard, and to attain the object the urge for incessant vigilance must arise in both the management and the workers *within* each factory.

In grappling with this problem, the asbestos industry, as compared with the silicosis-producing industries in tackling the problem of silicosis, starts with considerable tactical advantages because of the method of action of the dust, of the nature of the processes in which protection is required, and for other reasons. The problem of the control of asbestosis is definitely finite as compared with the control of silicosis, and it is this fact coupled with the evident desire of the leading manufacturers, as shown by the remarkable progress in dust suppression made in the past two years and their courage in dealing with the practical difficulties, which relieves the apprehension inevitably associated with a serious occupational disease, and cheers with the conviction of success.