

PLAINTIFF'S
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Asbestosis — A Diagnostic Enigma

A Personal View

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Although the carcinogenic properties of asbestos are presently attracting a good deal of attention, asbestosis is still the earliest lung disease resulting from exposure to it and its incidence in an exposed population is the most useful indicator of the degree of dust control exercised over a period of time.

The diagnosis of asbestosis depends upon: (1) An adequate occupational exposure history. (2) Physical signs of pulmonary fibrosis. (3) Progressive radiological changes. (4) Confirmatory measurements of altered lung function.

Asbestosis is a clinical entity and is readily diagnosed when all the above-mentioned criteria are met. Problems in diagnosis are encountered when one or more of the diagnostic criteria listed above cannot be substantiated. At the present time every effort is made to diagnose the disease in its early stages in the hope that removal from further exposure will prevent the direct and indirect complications.

Regulations to control the asbestos industry in the United Kingdom were made in 1931.¹ The evidence upon which they were based was gathered in 1929 by Merewether and Price, the former a medical inspector of factories (who later became senior medical inspector) and the latter an engineering inspector of factories.² Merewether selected for his studies the textile branch of the industry (a branch manufacturing industrial textiles for insulation, friction and packing material applications), and some preliminary processes in other branches. Each individual's previous industrial history, subsequent to leaving school, was noted in detail. Merewether considered this to be essential in order to exclude people whose previous work may have been in any of the numerous processes involving exposure to free silica and other dusts.

Merewether's investigations led him to state: "To sum up, therefore it appears probable that concentration of dust and length of exposure as factors in the production of fibrosis are interdependent within certain limits. While it seems necessary for

the production of generalized fibrosis of the lungs that a definite minimal quantity of dust must be inhaled, the lower the concentration of dust in the air breathed, the longer the lapse of time before the fibrosis is fully developed, and within a certain limit, the higher the concentration of dust, the sooner the fibrosis becomes fully developed and the more intense the involvement of the lung tissue."

He went on to hypothesize that, in the light of the above reasoning and the evidence which pointed to it, the application of dust control measures would cause "firstly, a great increase in the length of time before workers develop a disabling fibrosis, and secondly, the almost total disappearance of the disease, as the measures for the suppression of dust are perfected."

The improvements made in the British manufacturing industries after the 1931 Asbestos Industry Regulations took effect may be said to have occurred slowly and progressively from 1931 onwards and although much was achieved before World War II, the major advances in dust control probably took place in the 1950's.³

During the years of World War II regulations were relaxed and conditions in factories deteriorated. One of the problems in quantifying dust exposure data is how to take account of the effect of historic events on working hours and working conditions. There is no way at present of analysing the effects of depressions, booms and other socio-economic phenomena when attempting to measure cumulative dust exposure over any given period of time.

Because of the limitations of the 1931 regulations, and because the regulations applied to manufacture but not usage of products, the hoped for reduction in cases of asbestosis in the U.K. did not occur at a national level. The Senior Medical Inspector's Advisory Panel on Asbestos, in its report to the minister of labor in 1967⁴ gave the following reasons for the increasing incidence of asbestosis: (a) The clinical criteria for diagnosis have changed since the 1920's (b) Laggards, the total of whom was not known, tended to be excluded from the process to which the 1931 regulations applied. (c) There had been an increase in overall consumption of asbestos and a rising population of exposed workers. (d) Medical supervision of workers in the asbestos industry (originally recommended by Merewether) had not been extended to workers in new processes as these were introduced.

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Comparison Between Merewether's Findings in 1929 and Prevalence of Asbestosis in 1975.*						
Lewinsohn 1975				Merewether 1929		
No. of Years Employed	No. Exposed	No. of Cases Certified	Group Incidence %	No. Examined	No. of Cases Diagnosed	Group Incidence %
0 - 9	737	1	0.125	230	36	15.5
10 - 14	88	0	—	84	27	32.1
15 - 19	66	3	4.5	28	15	53.5
20 & Over	82	4†	4.9	21	17	80.9
Totals	1033	8	0.77	363	95	26.2

*Only cases of asbestosis in receipt of Industrial Injuries Benefit are included in the 1975 figures
†3 cases with more than 20 years exposure were first exposed before 1933 (Table VI)

The Advisory Panel did record, however, that although they were inclined to accept the growing use of asbestos as the most probable explanation of the increase in cases, "conversely there is no evidence pointing to a decrease in the attack rate in the industry as a whole although there is such evidence in certain important asbestos using factories."

In 1969, new regulations known as the Asbestos Regulations 1969 were made in the U.K. and they took effect in May 1970.¹² These regulations recognized two things, among many others, namely, the importance of their application to both manufacture and usage of asbestos products and the acceptance of a level of dustiness below which there was no hazard to health. The latter principle was acknowledged by the publication of "Hygiene Standards for Airborne Asbestos Dust Concentrations for Use with Asbestos Regulations 1969" in Technical Data Note 13 (Rev.).¹³

Technical Data Note 13 states that where the dust concentration is less than 2 fibres/ml, HM Factory Inspectorate will not seek to enforce the substantive provisions of the Regulations. This figure is derived from the simple assumption that in order to accumulate an exposure limit of 100 fibres/ml during a working lifetime, the dust level should not exceed an annual average of 2 fibres/ml $\times$ 50 years $\times$ 2 f/ml = 100 f. years/ml., 50 years $\times$ 4 f/ml = 100 f. years/ml. etc.

'Adequate' Occupational Exposure History

In the determination of an adequate occupational exposure history it is necessary to ascertain the type of asbestos fibre in use. There are four main varieties in commercial use, namely:

Chrysotile — white — 95% world production.

Crocidolite — blue -)

Amosite — brownish -) 5% of world production.

Anthophyllite — brownish -)

It is accepted that all varieties of asbestos can give rise to asbestosis. Asbestosis might be complicated by the development of carcinoma of the lung, and the risk of this complication is multiplied many times by cigarette smoking.

Before discussing the clinical features of asbestosis, one further difficulty in obtaining an adequate occupational exposure history requires reference. Merewether attempted to quantify the dustiness in the textile branch of the industry but was only able to produce a rough guide. The respirability of asbestos dust, because it is composed of fibres and not of spherical or uniform particles, depends upon its aerodynamic behavior which is related to fibre diameter and falling speed. It was not until suitable instruments became available in the late 1940's and early 1950's that routine environmental monitoring could be introduced into industry. The British Occupational Hygiene Society made use of dust measure-

ments and clinical data from a Rochdale asbestos textile factory in its first attempt to set "Hygiene Standards for Chrysotile Asbestos Dust" in 1968. Prior to this the only standard recorded was in the U.S.A. and was 5,000,000 asbestos particles per cubic foot as measured by means of the midjet impinger. The B.O.H.S. Standard recommended a cumulative dust exposure limit of 100 fibre years per cubic centimeter. This would allow a 1% risk of developing basal rates, which were considered as the earliest physical signs due to the effects of asbestos exposure.

In the medical surveillance of asbestos workers in Britain we are now concerned with two populations for follow-up, namely, those people who have worked in the industry when dust measurements were not made and when variable dust control measures were taken and those who have entered since May 1970 when new improved conditions were demanded by new regulations and when environmental monitoring could be added to the data for epidemiological evaluation. HM Chief Inspector of Factories in his Annual Report, 1974,¹ states, "139 new cases of asbestosis were recorded by DHSS during the year. These continued to reflect conditions in past years when the long-term effects of asbestos dust on the Health of Workers was not fully appreciated. The latent period for this disease is such that annual figures cannot yet be expected to reflect improved conditions following the introduction of new legislation in 1970."

What constitutes an adequate occupational history? There is insufficient evidence available from industry to enable this question to be completely resolved, but such evidence as there is allows certain standards to be set in the hope of reducing the risk and providing a base-line for further study. (B.O.H.S. 1968).

Physical Signs of Pulmonary Fibrosis

Where exposure is known, the presence of asbestos bodies and fibers in sputum is of little importance, since their presence simply confirms exposure and their absence does not indicate freedom from disease. The presence of basal rales (crepitations or crackles) and finger clubbing have long been accepted as important clinical findings in conjunction with a history of asbestos exposure. The British Occupational Hygiene Society's Committee on Hygiene Standards, in their publication 'Hygiene Standards for Chrysotile Asbestos Dust' considered basal rales as the "key symptom." The crackles are characteristically of high pitch and occur in end-inspiration, persisting after coughing and most prominent in the dependent areas of the lungs.

It is important to establish that the crackles are persistent and not due to other diseases resulting in pulmonary fibrosis.

Until it is possible to record lung sounds and preserve graphic records, the presence or absence of these fine crackles remains a

subjective sign which will be elicited in different ways by different observers according to the quality of their stethoscopes, the degree of presbycusis from which they suffer and the ability of the patient to breathe in a controlled manner.

Progressive Radiological Changes

X-ray technology and classifications of pneumoconioses have changed considerably during the past 46 years since Merewether surveyed workers in the asbestos textile industry. The radiological criteria which he used to diagnose asbestosis are not defined in his report.

Radiological changes considered significant in the diagnosis of asbestosis by the BOHS Committee were increased general opacity of the lower lobes, blurring of the cardiac outline, pleural thickening and adhesions. Isolated areas of calcification, unconnected with the above changes, were not considered as necessarily or probably asbestotic in this series.

It appears likely that Merewether and the BOHS Committee were concerned with relatively far-advanced disease.

The BOHS Committee recognized the onset of the disease to be gradual and hence difficult to define. All the features of the disease may occur to varying degree and, indeed, the severity of the alteration of the separate features may well be related to the type of past dust exposure. Thus the particular set of criteria used to decide whether asbestosis is present or absent will vary.

Various systems of classification have been used to attempt to standardize the description of radiological opacities. The International Labor Office's 1959 Classification⁸ was, until recently, the most widely used but had certain difficulties with regard to asbestosis. The classification was recently modified by a UICC working group and extended to include irregular opacities such as occur in asbestosis as well as other abnormalities.⁹ The ILO/UC International Classification of Radiographs of Pneumoconiosis, 1971,⁴ is designed to describe "persistent radiological opacities in the lung fields provoked by mineral dust" and to allow them to be categorized according to size and shape and to indicate their profusion or extent in the lung fields. A set of standard films illustrating all categories is issued by the ILO.

Confirmatory Measurements of Altered Lung Function

Lung function tests have three main uses, namely: (a) To establish base-line values in order to assess lung function, periodically using each worker as his/her own control. (b) To confirm the clinical and radiological diagnosis. (c) To assist in the assessment of disability in established disease.

It is not proposed to discuss (a) and (c) but merely to describe briefly the value of lung function tests in confirming the diagnosis. According to Badier et al.¹¹ in asbestos workers vital capacity reduction precedes category 2 or 3 radiological changes by 10 to 15 years; the latter changes do not occur until 20 years of exposure. After 30 years of exposure, the incidence of functional and radiographic abnormalities is approximately the same.

The tests most readily used according to Becklake et al.¹⁰ are those of ventilatory capacity, which include measurement of the FEV₁, FVC and FEV/FVC¹⁰.

Lung volumes and gas transfer should be measured in all suspected cases to obtain confirmatory evidence of pulmonary fibrosis.

General Remarks

An attempt has been made to indicate that asbestosis is not an

easy condition to diagnose. It requires intimate knowledge of the industry and the use of its products. The view is shared by Elmes who, in relation to a particular case which he diagnosed as asbestosis of nonoccupational origin, has subsequently published an account of the autopsy findings indicating that his diagnosis was incorrect. The diagnosis established at autopsy in this case was chronic active fibrocaseous tuberculosis.¹¹

Asbestosis is not a clear-cut entity except in the advanced stages of disease and very often a mistaken diagnosis can be made which, if communicated to the individual concerned before all diagnostic avenues have been explored and the disease confirmed, can lead to psychological stress and breed ill-will in a community. It is sound practice in the United Kingdom to discuss all suspected cases with the Members of the Pneumoconiosis Medical Panel and to accept their judgment with regard to diagnosis. There may be a few people who have some, but not all, diagnostic criteria, who are not considered to be suffering from asbestosis. Many of these cases are elderly and on the verge of retirement. They are symptom-free, their earning capacity is not affected and they are unable to find alternative work if they have to change jobs.

Furthermore, the dust conditions which probably produced the slight changes in their x-rays should no longer exist and they should now be employed in conditions where the Asbestos Regulations, 1969 ensure their safety. Unless they develop symptoms, or clinical findings indicate definite changes, there does not appear to be any need to take further action. The first indication of deterioration must elicit a rapid response leading to immediate further investigation, diagnosis and appropriate compensation.

In the interpretation of lung function test results it should be borne in mind that there is a wide range of "normality" for most available measurements made and it is thus important to establish for each individual worker a "normal" base-line value prior to exposure and to observe deviation from the base-line in order to assess deterioration of lung function during his working lifetime. The measurement of FEV₁ and FVC, is a simple procedure and the test equipment available is robust, transportable and dependable. The use of lung function tests in the future in the surveillance of asbestos workers is obvious and should form an essential part of any preventative medical programme.

Physiological tests alone cannot prove the diagnosis of asbestosis, but merely the abnormal pattern of lung function which characterises diffuse pulmonary fibrosis from any cause. In combination with occupational history, physical signs and radiological changes they give confirmatory evidence of the presence of the disease and assist in assessing its severity. They are essential in the investigation of suspected asbestosis.

A number of points arise as a result of the increasingly widespread use of the ILO/UC Classification of radiographs. The commonest changes recorded by most observers are in category 1 and relate to irregular small opacities. It would appear that the presence of irregular small opacities in smokers can significantly affect the interpretation of x-rays and that in the older age group these changes can also occur.¹⁴

The significance of category 1 (irregular small opacities in asbestos workers working in low dust concentrations), unless demonstrated to be related to cumulative dust exposure when reviewed on a serial basis, cannot yet be determined. There is no clear-cut dividing line between early change and disease. The diagnosis of asbestosis should not be made purely on the basis of a slight alteration in radiological appearance and in the absence of serial review radiographs, a history of asbestos exposure (including

some knowledge of dust levels and fibre type) and confirmatory clinical findings of pulmonary fibrosis.

Many workers who have been exposed to asbestos never develop any x-ray changes and only some develop asbestosis. It is not morally justified to suggest to a symptom-free, otherwise healthy individual that he or she is suffering from an incurable dust disease with a concomitant cancer risk, if there is not definite proof. If the disease is "definitely suspected" then the individual has a right to know, to cease exposure and to seek compensation. It is not yet known whether the improved methods of examination available permit the detection of the disease at a stage where further progression can be prevented if exposure ceases. The disease is now almost certainly diagnosable at an earlier stage than 46 years ago in Merewether's time. The disease will probably progress in the more advanced cases even when exposure to asbestos dust has ceased.

The British worker has changed his job habits and it is unusual to find him working in one firm or one job for as long as 20 years. Asbestosis is a preventable disease — it now remains to prove whether lung cancer and mesothelioma are preventable by the same means. With regard to lung cancer the eradication of the cigarette habit will undoubtedly also lead to the eradication of this complication of asbestosis.¹¹

In a large asbestos textile factory in Rochdale the prevalence of asbestosis has been reduced in 1975 to 0.77% among the current labour force compared with Merewether's finding of 26.2% in 1929. (Table). This reduction has been achieved by conscientious effort on the part of management to eliminate the disease. In recent years there has been a growing awareness among workers in industry of their role in the prevention of occupational disease and when enlightened management takes advantage of this new willingness to cooperate, then useful practical measures follow. Confrontation is not the way to better health. Elimination of occupational disease depends upon joint consultation and sensible application of the most reliable and practicable preventive measures, implementing Legge's aphorism that unless and until the employer has done everything, and everything means a great deal, the workman, no matter how willing he may be to do so, cannot protect himself.

The world cannot do without asbestos at the present time as it

forms an essential component in many sophisticated engineering production and everyday devices. Without asbestos, more lives would undoubtedly be lost from the hazards of fire and most kinds of energy conservation, transportation and industry would be unable to function effectively.

References

1. The Asbestos Industry Regulations, (S.R. & O 1931 No. 1140), 1931.
2. Merewether ERA and Price CW: Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry. Part 1. Occurrence of Pulmonary Fibrosis and other Pulmonary Affections in Asbestos Workers. H.M.S.O., London, 1910.
3. British Occupational Hygiene Society: Committee on Hygiene Standards: Hygiene standards for chrysotile asbestos dust. *Ann Occup Hyg* 11:47, 1968.
4. Problems arising from the use of Asbestos. Memorandum of the Senior Medical Inspector's Advisory Panel London: H.M.S.O., 1967.
5. Department of Employment 1974. Annual Report 1974. H.M. Chief Inspector of Factories: London. H.M.S.O. (Cmd) 6122.
6. International labour office meeting of experts on the international classification of radiographs of the pneumoconioses. *Occup Safety Hlth* 9:2, 1959.
7. UICC/Cincinnati classification of the radiographic appearances of pneumoconioses. A co-operative study by the UICC committee. *Chest* 58:57, 1970.
8. International Labour Office. International Classification of Radiographs of Pneumoconioses. ILO U/C Classification 1971: 1972.
9. Bader ME, Bader RA, Teirstein AS et al: Pulmonary function and radiographic changes in 598 workers with varying duration of exposure to asbestos. *Mt. Sinai J Med* 37:492, 1970.
10. Becklake MR, Fournier-Massey G, McDonald IC et al: Lung function in relation to chest radiographic changes in Quebec asbestos workers. *Bull Physiother Res* 6:637-659, 1970.
11. Elmes PC: Incorrect diagnosis of asbestosis. *Postgrad Med J* 50:250-251, 1974.
12. The Asbestos Regulations. London: H.M.S.O. (1969 No. 690).
13. Department of Employment. Hygiene Standards for Airborne Asbestos Dust Concentration for Use with Asbestos Regulations. Technical Data Note 13 (Rev.), 1969.
14. Amandus HE, Lapp NL, Jacobsen G, and Reger RB: Significance of irregular small opacities in radiographs of coalminers in the U.S.A. *Brit J Industr Med* 13:13, 1976.
15. Berry G, Newhouse ML and Turok M: Combined effect of asbestos exposure and smoking on mortality from lung cancer in factory workers. *Lancet* 2:476-479, 1972.

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