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CONFIDENTIAL

CRITERIA FOR DIAGNOSIS OF ASBESTOSIS

The Medical Advisory Panel of the Asbestos International Association is producing a criteria document to assist industrial medical officers in diagnosing asbestos-related disease. The basic paper has now been finalised but publication will not take place until later in the year when all the references have been added and when the paper has been accepted by a reputable medical journal. The paper is not incompatible with that produced by Dr.Allardice a couple of years ago and indeed he contributed in the early stages towards the formulation of this paper.

When published the AIA paper will be a very important reference document for the asbestos industry and, indeed, for regulatory authorities in many countries. I am therefore sending you a copy of the final draft which can be used for internal purposes as required. However, as publication has not yet taken place I shall be grateful if you do not use it for external purposes or give any publicity to it.

H.D.S. HARDIE

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I. [redacted]

[redacted]

Asbestosis is a diffuse fibrosis of the parenchyma of the lung caused by exposure to respirable airborne asbestos fibres. The fibrosis is irreversible and in some persons progresses even after exposure has ceased.

[redacted]

1. History

There should be evidence of substantial occupational exposure (or substantial para-occupational exposure) to asbestos fibres.

2. Clinical Signs and Symptoms

Persistent basal inspiratory crepitations characteristic of interstitial pulmonary fibrosis may be heard but are not invariably present. Breathlessness and finger clubbing occur but are not specific signs in themselves.

2.1 Crepitations (crackles). These are fine basal inspiratory crepitations, usually bilateral which occur late in inspiration and persist after coughing or hyperpnoea. They are characteristically heard with each inspiration and on each occasion present very much the same pattern of sound. They are not essential to the diagnosis any more than they are pathognomonic of the disease. However, when not accounted for by another cause and when heard on at least two occasions a few months apart, given a history of exposure and radiological evidence of dust disease, they do provide valuable confirmation of the diagnosis. In the presence of equivocal radiological change, basal crepitations as here defined are a strong indication for the diagnosis of asbestosis.

2.2 Abnormal Breathlessness. This, occurring initially on exertion, is a symptom of asbestosis. There are, of course, many other causes of breathlessness and these must be excluded. In those under routine medical surveillance a provisional diagnosis would probably have been made before disabling dyspnoea occurs.

2.3 [illegible]. This is a rare event in lung and other diseases including asbestosis where it may be of little consequence. It develops slowly but the rapid growth rate of fibrous clubbing, particularly if painful, in a person with asbestosis suggests lung cancer.

2.4 [illegible]. These can occur in asbestosis, but infrequently, and then only late in the progression of the disease. They are not specific and of little diagnostic significance.

3. Radiological Aspects

Radiographic evidence of predominantly basal diffuse interstitial fibrosis is the characteristic change seen.

3.1 So that films are comparable they must be of good technical quality and of full size (approximately 350 mm x 430 mm and to include both costo-phrenic angles). Ideally inspiration should be such as to have brought the diaphragm below the fifth rib anteriorly and the tenth rib posteriorly.

3.2 The pulmonary changes as recorded on X-ray are those of small (width of up to about 1.5 mm (s^s)), medium (over 1.5 and under 3 mm (t^t)), or large (over 3 and up to 10 mm (u^u)), irregular or linear opacities. The changes may be of such profusion as to obscure the normally sharp margins of the bronchovascular structures. Small (p^p), medium (q^q), or rarely, large (r^r) rounded opacities may also be present. In advanced disease the sharpness of the cardiac and diaphragmatic borders are sometimes obscured.

3.3 Pleural changes are not a diagnostic criterion of asbestosis but are often present. Diffuse or localised pleural thickening or circumscribed pleural plaques, with or without calcification, may occur and need not be associated with parenchymal fibrosis. Transient pleural effusions have been reported. The pleural abnormalities can be sufficiently extensive to obscure the lung fields and may, in rare cases, lead to impairment of lung function.

3.4 In individual cases a narrative report by the reader of the radiograph as well as an interpretation of the relationship between the observed appearances and the occupational history may be helpful.

3.5 Where possible comparison of the most recent film with earlier films is highly desirable.

* These symbols make reference to the I.L.O. Classification of Radiographs of Pneumoconiosis (1980). This classification is purely descriptive and was specifically designed for epidemiological purposes and not for evaluating an individual.

3.6 A dose response relationship has been demonstrated between the extent of asbestos exposure and the radiological pleural changes. Radiological change correlates in general with an adverse effect on the long-term prognosis of an individual. However, in the absence of asbestosis, there is no adverse prognostic significance associated with pleural changes unless they are unusually severe.

3.7 Pleural changes do not constitute asbestosis and to speak of "pleural asbestosis" serves only to confuse.

3.8 Other causes of radiological changes must be carefully excluded prior to making the diagnosis of asbestosis. This is important in the early stages of the disease when the radiological changes are slight.

4. Other Investigations

4.1 Asbestos (Ferruginous) Bodies. When a pre-fibrotic sample is present asbestos fibres or ferruginous bodies in the sputum are evidence only of exposure to asbestos and are not diagnostic of asbestosis.

4.2 Biopsy is very seldom justified as a diagnostic procedure for asbestosis. The effects of exposure should already have been recognised on the grounds outlined in the foregoing criteria. Biopsy is justified if it is thought that an asbestos-exposed patient may be suffering from some other potentially treatable lung disease. Any surgeon about to conduct a thoracic operation on a person known to have been exposed to asbestos should be asked to obtain a specimen of lung tissue for histological examination.

5. Lung Function Abnormalities

The characteristic abnormalities are those of restrictive lung disease.

5.1 It is recommended that the F.V.C., F.E.V.$_1$ and F.E.V.$_1$/F.V.C. ratio be recorded routinely in standard fashion. A comparison of periodic lung function testing over a number of years is of much greater value than a single observation compared to the standard "normal" values. In assessing the results, allowance must be made for certain ethnic differences and the effects of smoking habits.

5.2 A restrictive pattern is consistent with the diagnosis of asbestosis, but not specific to this disease. A predominantly obstructive picture is uncommon in the absence of a smoking history.

5.3 More sophisticated pressure suits to detect restrictive lung disease are diagnostically helpful (e.g. the measurement of transfer factor and lung compliance) but are not essential to the routine medical surveillance of a group of asbestos-exposed people at this time.

[illegible]

When there are clinical, radiological, or lung function abnormalities in asbestos workers, the exclusion of simulative disease is important so that the correct management of the individual may be implemented. This facet is likely to be increasingly important as, with improving occupational hygiene standards, asbestosis becomes less common.

[illegible]

The art of diagnosis, always a matter of weighing probabilities and looking at the total evidence, demands expert judgment based on the interpretation of the above criteria. Each of them is on a scale of severity and it is possible to find high values in some, low values in others, or any permutation or combination of criteria in the individual cases. According to the circumstances the elements of the decision and action will vary.

II. [illegible]

1. Diffuse malignant mesothelioma is a rare primary tumour in the general population. Mesothelioma occurs in the pleura (lining of the lung) or the peritoneum (lining of the abdominal cavity). The latent period between asbestos exposure and onset of the disease is usually 20 to 40 years or longer.

2. It must be emphasised that the definitive diagnosis of mesothelioma is not easy. Mesothelioma in asbestos workers is usually associated with asbestosis which, however, may only be recognised at necropsy. Mesothelioma may occur after relatively brief but intense asbestos exposure. While pleural abnormalities are common in asbestos workers, there is no conclusive evidence that of themselves they predispose to mesothelioma. Early suspicion of the presence of pleural mesothelioma arises when pleural effusion or chest pain occurs in an asbestos worker. The tumour may completely encase the lung. Peritoneal mesothelioma may occupy a large amount of the abdominal cavity and produce ascites with or without abdominal pain. Metastases, although rare, can affect other organs, but local spread is the usual mode of progression. The outcome is invariably fatal.

3. Ideally the diagnosis will be based on careful necropsy in which particular attention is given to excluding the possibility

of a primary cancer at some distant site. Furthermore, the tumour tissue should be referred to one of the established mesothelioma reference panels so that histopathologists especially skilled in the diagnosis of this tumour may give their opinion. Such panels now exist in many countries.

4. Most cases of mesothelioma are related to asbestos exposure though the proportion of cases attributable to this cause varies. It is greatest in the most highly industrialised communities, especially those with a large shipbuilding or ship repairing industry. In this industrial population the majority of mesothelioma cases may be ascribed to asbestos exposure. In the other groups which have been adequately studied, the proportion of mesothelioma cases associated with asbestos exposure is lower but variable. Not all mesotheliomata are due to asbestos; for example, endemic mesothelioma in some areas of Turkey is believed to be due to a fibrous form of zeolite. Cases arise where no environmental causes can be determined.

5. Smoking habit plays no part in the genesis of mesotheliomas as far as can be determined at the present time.

6. This tumour is relatively uncommon even among those who have had substantial asbestos exposure. Many believe that the risk of developing mesothelioma is greatest with crocidolite, less with amosite, and apparently less with chrysotile, while anthophyllite appears never to have caused mesotheliomas in humans. This opinion, however, does not enjoy unanimous support and some authors hold that there is no justification for differentiating between the various kinds of asbestos and their biological effects.

7. There is epidemiological and pathological evidence of a dose response relationship between inhaled asbestos and tumour formation. Recent work does not support the often stated idea that any slight, casual or brief asbestos exposure may lead to mesotheliomas.

8. The long latency period between asbestos exposure and the development of the tumour makes elucidation of a dose response relationship difficult. Thus the effects of good dust control on the incidence of mesotheliomas will only be determined in the future.

III. LUNG CANCER

1. Cancer of the lung is the most common form of cancer in males in industrialised countries and the primary cause is cigarette

smoking. In asbestos workers who smoke, it is many times more common than in members of the general population who are not exposed to asbestos and who do not smoke.

2. Non-smoking asbestos workers under the conditions of past exposure appear to be at greater risk than non-workers in the general population. Even so, these non-smoking asbestos workers are at less risk of bronchogenic cancer than cigarette smokers who have not been exposed to asbestos in the general population. There is evidence from epidemiological studies that the lower levels of past exposure to asbestos do not pose a detectable excess risk for bronchial carcinoma development.

3. There are no specific pathological features by which an individual case of lung cancer can be attributed solely to asbestos exposure.

4. An adeno-carcinoma situated peripherally and particularly in a lower zone, is the type of tumour more likely to occur in a worker exposed to asbestos more than fifteen years previously. This is especially so in the presence of asbestosis.

IV. [illegible]

The evidence relating asbestos to cancer of other sites is equivocal and further data are awaited.

Document prepared by the Medical Advisory Panel to the Asbestos International Association.

1st April 1981