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UC-3835
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ASBESTOS INTERNATIONAL ASSOCIATION
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68 GLOUCESTER PLACE, LONDON W1H 3HL

# MEMORANDUM

TO: Medical Advisory Panel & Dr. H.C. Lewinsohn
Dr. W.J. Smither
Dr. I. Tretner

AIA/20/1/16/HAS

FROM: Director-General

28 October 1981

Criteria for the Diagnosis of Asbestosis and Considerations in the Attribution of Lung Cancer and Mesothelioma to Asbestos Exposure

As you will recall the above "Criteria" document has been agreed by the MAP and has been distributed to Member Associations.

Following this, the Executive Committee supported the suggestion that the final version should be published in a reputable journal under your names and subject to peer review.

Prior to such publication it was considered courteous to circulate the paper to the ScAP so that they would have an opportunity to comment if they wished.

Somewhat to our surprise (and gratification) considerable interest was shown by the ScAP and comments were received from nine members.

Their comments and suggestions were not too many nor too radical and it seemed best, in order to save time, that Dr. Murray, Dr. Browne and myself should go through the comments and amend the text where we considered it was necessary. This, with the Executive Committee's agreement, we have now done and the amended document is forwarded herewith.

It is proposed that we discuss the paper at the next MAP meeting on 1/2 December 1981 so that it can then be published.

I do not anticipate that there will be much change to this paper at the coming MAP meeting and it will consequently be published very much in this form and content. Unless I hear from you to the contrary I will assume that you are agreeable to having your name appear at the footnote on page one.

Sir Neville Stack

P.S. You will notice that the paper has been given an AIA Memorandum number. This is for administrative convenience.

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# CRITERIA FOR THE DIAGNOSIS OF ASBESTOSIS AND CONSIDERATIONS IN THE ATTRIBUTION OF LUNG CANCER AND MESOTHELIOMA TO ASBESTOS EXPOSURE

Prepared by the Medical Advisory Panel*
to the Asbestos International Association

## SECTION 1 ASBESTOSIS

### DEFINITION

1.1. 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.

### CRITERIA FOR DIAGNOSIS

1.2. **History**

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

1.3. **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.

1.3.1. **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.

* S.F. McCullagh (Australia), Chairman; G. Aresini (Italy); K. Browne (UK); B. Korsgaard (Denmark); J. Lepoutre (Belgium); M. Lesage (Canada); H.C. Lewinsohn (USA); F. Mansour (Lebanon); M.C. Mills (UK); W.R. Paul (USA); C. Raffaelli (France); W.J. Smither (UK); I. Tretner (Germany); R.B.K. Tucker (South Africa); R. Murray (Convenor).

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1.3.2. **Abnormal Breathlessness.** This, occurring initally 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.

1.3.3. **Clubbing.** This is a sign seen in many lung and other diseases including asbestosis where it may be of late occurrence. It develops slowly but the rapid development of finger clubbing, particularly if painful, in a person with asbestosis suggests lung cancer.

1.3.4. **Dry Cough and Ill-Defined Chest Pain or Discomfort.** 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.

1.4. **Radiological Aspects**

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

1.4.1. 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.

1.4.2. The recording of pulmonary changes on the radiograph should follow the system used in the ILO Classification*. This system describes irregular or linear opacities as follows:-

i) Small (s) - width up to about 1.5 mm.
ii) Medium (t) - width over 1.5 mm and under 3 mm.
iii) Large (u) - width over 3 mm and up to 10 mm.

The changes may be of such profusion as to obscure the normally sharp margins of the bronchovascular structures. Small (p), medium (q), or rarely, large (r) rounded opacities may also be present. In advanced disease the sharpness of the cardiac and diaphragmatic borders are sometimes obscured.

1.4.3. Pleural changes are commonly present and are often the first radiological indicator of exposure to asbestos. Diffuse or localised pleural thickening or circumscribed pleural plaques, with or without calcification, may occur and

* The ILO International Classification of Radiographs of Pneumoconiosis (1980) is designed to ensure international comparisons of radiological data. Sets of films are obtainable from ILO Publications, International Labour Office, CH1211, Geneva 22, Switzerland.



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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.

1.4.4. 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 is helpful.

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

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

1.4.7. Since pleural changes may occur in the absence of parnchymal fibrosis, the use of the term "pleural asbestosis" is undesirable.

1.4.8. 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.

1.5. **Other Investigations**

1.5.1. **Asbestos (Ferruginous) Bodies.** When a productive cough is present asbestos fibres or ferruginous bodies in the sputum are evidence of exposure to asbestos but are not diagnostic of asbestosis.

1.5.2. **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.

1.6. **Lung Function Abnormalities**

The characteristic abnormalities are those of restrictive lung disease.



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1.6.1. 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.

1.6.2. While a restrictive pattern is consistent with the diagnosis of asbestosis, it is not specific to this disease. Asbestosis may develop with little or no detectable restrictive defect in the early stages. A predominantly obstructive picture is uncommon in the absence of a smoking history.

1.6.3. More sophisticated measurements in the assessment of restrictive lung disease (e.g. the measurement of transfer factor and lung compliance) are diagnostically helpful, but are not essential to the routine medical surveillance of a group of asbestos-exposed workers.

### 1.7 Differential Diagnosis

When there are clinical, radiological, or lung function abnormalities in asbestos workers, the exclusion of simulative disease is necessary for the correct management of the individual. This is increasingly important as, with improving occupational hygiene standards, asbestosis becomes less common.

### 1.8 Comment

The art of diagnosis, always a matter of weighing probabilities and looking at the total evidence, demands expert judgement 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.

## SECTION 2 MESOTHELIOMA

2.1 Diffuse malignant mesothelioma is a rare primary tumour in the general population, occurring in the pleura or peritoneum. When attributable to asbestos the latent period between asbestos exposure and onset of the disease is usually 20 to 40 years or longer.



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2.2 It must be emphasised that the definitive diagnosis of mesothelioma is not easy. In asbestos workers it may be associated with asbestosis though the level of fibrosis can be minimal. In some cases there is no evidence of fibrosis even 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 ascities with or without abdominal pain. Metasteses, although rare, can affect other organs, but local spread is the usual mode of progression. Hitherto no treatment has been successful, the outcome being invariably fatal.

2.3 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 ot 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.

2.4 While cases of mesothelioma may arise in the general population with no obvious cause, the majority of mesothelioma cases are related to asbestos exposure. The proportion of cases attributable to asbestos exposure varies; it is greatest in the more highly industrialised communities, especially those with a large shipbuilding or ship repairing industry. In industrial populations generally, most mesothelioma cases may be ascribed to asbestos although even in these communities no relationship with asbestos can be established in a proportion varying from 10% to over 30%. In 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.

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

2.6 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 mesothelioma 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.

2.7 There is epidemiological and pathological evidence of an exposure - response relationship between inhaled asbestos and tumour formation. Recent work does not support the ofen stated idea that any slight, casual or brief asbestos exposure may lead to mesothelioma.



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2.8 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 mesothelioma will only be determined in the future.

## SECTION 3 LUNG CANCER

3.1 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.

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

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

3.4 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.

## SECTION 4 CANCER OF OTHER SITES

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

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