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CONGRESS OF THE UNITED STATES

HOUSE OF REPRESENTATIVES

COMMITTEE ON EDUCATION AND LABOR

SUBCOMMITTEE ON LABOR STANDARDS

HEARING ON THE EXISTENCE OF ASBESTOS-RELATED DISEASES IN AMERICAN INDUSTRY AND THE MANNER AND ADEQUACY WITH WHICH THE VICTIMS OF THESE OCCUPATIONAL DISEASES ARE PRESENTLY COMPENSATED.

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RAYBESTOS MANHATTAN INCORPORATED

TRUMBULL, CONNECTICUT

MAY 1, 1979

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My name is Milton C. Lewinsohn. I am Corporate Medical Director for Raybestos-Manhattan, Inc., which is headquartered in Trumbull, Connecticut. I have been engaged in this capacity since October, 1976, prior to which I was Chief Medical Officer to TBA Industrial Products Company Limited in Rochdale, Lancashire, England, and Medical Adviser to the Turner and Newall group of companies. I was employed as a Pneumoconiosis Medical Officer in the Ministry of Pensions and National Insurance (now the Department of Health and Social Security) from 1963 to 1966, which provided me with further special experience and training in the field of occupational lung disease and the assessment of disability resulting therefrom. The curriculum vitae which is attached hereto as Appendix I lists my academic achievements, affiliations and publications.

I should like to present this statement in three sections, namely:

A. Asbestos and health - the matter in perspective,
B. The diagnosis of asbestos-related disease,
C. Assessment of Disability and Bill H.R.2760.

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A. Asbestos and Health - The Matter in Perspective

1. Asbestos is a collective mineralogical term including all asbestiform varieties of various minerals and refers to the industrial product obtained by mining and further processing. (1)
2. Biological effects attributable to asbestos exposure were described in the world medical literature as long ago as 1924 and in the United Kingdom legislation to regulate the asbestos industry was promulgated in 1931. (2,3)
3. The first condition which was found to be caused by exposure to asbestos was disabling fibrosis of the lungs which came to be known as asbestosis. (4) Post-mortem examination of lungs from asbestosis victims revealed the presence of unusual beaded, rod-like particles which were called asbestosis bodies because it was thought they only occurred in asbestosis cases. It was later shown that these bodies could be found in the lungs and sputum of persons exposed to asbestos but not necessarily suffering from asbestosis and so they became known as asbestos bodies, rather than asbestosis bodies. They may also be found in the lungs of the general population, because asbestos is naturally present in the ambient air. (5)
4. All varieties of asbestos are capable of causing asbestosis. The lungs of an asbestosis sufferer lose their elasticity,

become stiff (fibrous) and shrink in size, causing breathlessness, at first only on effort, but later, if the disease progresses, breathlessness at rest may occur as well. Sometimes the fingernails and toenails become rounded in both planes, i.e., from side to side and front to back, and raised from the nail-bed so that the angle between the nail and cuticle gradually disappears. The ends of the fingers become swollen and the digits assume a "drum-stick" appearance or clubbed appearance. Clubbing of the fingers is not always due to asbestos exposure, nor does it occur in every case of asbestosis.

5. The breathlessness associated with asbestosis occurs only when the disease is reasonably far advanced and lung function tests can then measure the severity of its effects. Some authors believe that changes in lung function precede changes in the chest X-Ray of asbestos exposed persons. Others find the opposite. Skilled X-Ray readers have learned to look for these radiological appearances and classify them according to severity, but there is still disagreement among experts as to the correlation between X-Ray appearances observed and the stage of the disease process. It is possible to see appearances on the chest X-Ray which do not necessarily represent disease as they

can occur normally in a proportion of unexposed persons, and which do not alter over a long period of observation. Correlations between lung function tests and X-Ray changes indicate that either might appear at an early stage of disease. Lung function measurement is very valuable in confirming the degree of impairment produced by the pulmonary fibrosis. (6-12)

6. When a physician listens to the chest of an asbestosis patient, he should hear typical crackling noises at the lower extremities of the lung when the patient is standing or seated. These noises are called crepitations or rales and may be present in an asbestos worker in the absence of X-Ray changes or other symptoms or signs. Many authorities on the subject regard these creps as the earliest positive evidence of the development of asbestosis in an exposed person. (13) The present British Occupational Hygiene Society Standard for Chrysotile Asbestos, which has been adopted in many countries and set at 2 fibers per cubic centimeter, was based on a study relating asbestos fiber count data to the development of crepitations as the earliest recognizable biological effect due to asbestos exposure. (14) This data is currently under review.

7. Asbestosis is dose-related and reduction of dust or reduction of duration of exposure or both, should therefore reduce

the incidence of this disease. There is convincing evidence from the world literature that improvements in dust control have significantly reduced the incidence of this disease in industries where they have been applied. (15, 16) The concern which has emerged since the late 1960's is the increased incidence of disease in workers not employed in such controlled industries, but mainly engaged in the insulation, building construction and ship building and repair industries, where asbestos was incorporated and handled in a dusty product. (17) The insulation industry is the example par excellence with the construction industry running a close second. (18) Many insulation workers (and others working in their vicinity) in shipyards and naval dock yards, are now being diagnosed as having asbestosis or other asbestos-related conditions. (19, 20) Asbestos use in insulation products is minimal nowadays and it has been replaced by other less hazardous materials. In some workers the X-Rays show evidence of thickening of the tissue membrane lining the lungs and chest cavity (know as the pleura). When they become infiltrated with calcium, they patchy areas of thickened calcified pleura may show up on X-Ray and are then called calcified pleural plaques. These changes are usually asymptomatic and not incapacitating. (12)

8. In the 1930's, in England and the U.S.A., a possible association was first noticed between asbestosis and the development of lung cancer. (21) Subsequent epidemiologic research confirmed this association in 1955 (22) and further work since then discovered the significant multiplicative effect of asbestos exposure and tobacco smoke on lung cancer in asbestos workers who smoke. (23) There are two schools of thought with regard to the way in which cancer in asbestos workers occurs, one school postulating that only those cases associated with asbestosis should be considered as being of occupational origin, while the other school of thought regards asbestos as a carcinogen capable of producing lung cancer and cancer of the gastro-intestinal tract even in the absence of asbestosis. (24) The evidence that asbestos causes gastro-intestinal cancer is not as positive as the evidence for lung cancer but, nevertheless, it is now accepted by government authorities drafting new legislation and standards and it is accepted for workers compensation in Ontario after 20 years of occupational exposure to asbestos.

9. There is evidence in the literature that cancer incidence declines in parallel with a decline in asbestosis incidence when sound dust control engineering methods are introduced. (25) The lapsed period between first exposure and development

of lung cancer is usually in excess of 20 years and often takes longer, therefore, the effects of engineering controls in industry have not yet become fully apparent. In one of the best documented studies to date, there is still a slight excess incidence of lung cancer in a group of workers in an English asbestos textile factory who first started employment after 1950. (26) This excess is probably due to the fact that in certain areas of the plant, dust levels remained high and have only come under control in the mid-1970's.

10. In the late 1950's and early 1960's, at about the time when the asbestos industry felt it was beginning to control the asbestosis and cancer hazards, another condition was found to be associated with asbestos exposure. This disease is known as diffuse malignant mesothelioma of the pleura (or peritoneum - the membrane lining the abdominal cavity and its contents). (27) It is a cancerous tumor which gradually spreads over the surface of the lung, into the fissures between the lobes of the lung and causes death by constricting the lung leading to asphyxiation. It is painful in its final stages and this, combined with progressive breathlessness, leads to a most uncomfortable death with much suffering. Unlike other cancers this tumor does not spread readily to other parts of the body but it has been

known to do so. Many authorities warn against surgical intervention as the tumor tends to spread rapidly through surgical scars and add to the patient's suffering.

11. The discovery of the association between mesothelioma and asbestos was a turning point in the approach to the control of this mineral fiber. The condition was first associated with asbestos in persons exposed to blue asbestos (crodidolite) from the North West Cape Province in South Africa. (27) Most of the cases occurred in persons whose contact with the fiber had not been occupational. They had lived near the mine, or had been carried on their (African) mother's back while she hand-cobbed asbestos bearing rock or they lived in the town where school playing fields, paths, and roads were made from asbestos mine tailings. This discovery introduced the concept of neighborhood exposure into the asbestos saga. In 1964 it was shown that similar neighborhood cases had occurred near an asbestos factory in London, but that relatives of workers exposed by contact with asbestos contaminated work clothes had also died of this malignant tumor. (28) There have been other reports from different parts of the world confirming this association between mesothelioma and asbestos. (29-31) These neighborhood exposures are thought to have occurred at a time when dust collection was not

installed in exhaust systems and the level of dust were many times greater than are now found in the air near asbestos factories. There is at present no evidence to suggest that the minute levels of asbestos in our environment present a risk to the general public. (32)

12. There is evidence which suggests that a number of factors are important in mesothelioma causation. Firstly, only fibers which are less than 3 micrometers in diameter are capable of reaching the depths of the lung and penetrating to the pleural surface. Secondly, the body can deal with and probably "inactivate" fibers less than 10-20 micrometers in length, but it cannot remove fibers of greater length. Thirdly, chrysotile fibers tend to be long, thin and curly and are intercepted by the branching of the airways into the lung thus being unable to reach the pleural surface; crocidolite, particularly from the North West Cape, is needle like, shorter than chrysotile on the whole and able to align itself in the air stream which carries it down into the depths of the lung. Crocidolite from the North East Transvaal in South Africa has more fibers greater than 3 micrometers than other varieties and does not appear to be associated with mesothelioma as yet. (33) Only one study has been reported in the world literature suggesting amosite as a cause of mesothelioma whereas in the amosite

mining area in South Africa no proven association between this asbestos variety and mesothelioma has as yet been found. (7) It is difficult to state with precision the role of chrysotile in mesothelioma causation because in all industrial situations exposures have been mixed. In the Quebec mining areas, the largest producers of chrysotile in the world, the majority of mesothelioma deaths could be connected with mixed exposures or known exposure to crocidolite. (32-34) The fourth factor, therefore, is that crocidolite appears to be the most likely fiber to cause mesothelioma, amosite less likely, chrysotile still less likely and to-date no known cases have occurred from exposure to anthophyllite asbestos. Cigarette smoking does not appear to be a factor in the causation of mesothelioma.

13. Asbestos does not appear to be the only cause of mesothelioma as in all published work to-date between 10-30% of cases studied have not been associated with asbestos exposure. In 1978, the suspicion that other materials could be partly responsible for mesothelioma cases without known exposure to asbestos, was apparently verified by the finding in Turkey that asbestos related diseases, mainly calcified pleural plaques, chronic fibrosing pleuritis and malignant pleural mesothelioma are endemic in some villages without asbestos deposits. (35, 36) In one village, where 11 mesothelioma

deaths occurred in 1974, there are no deposits of asbestos in the area, nor has there been any processing of such material brought in from elsewhere. Research has shown many fibers in the respirable size range in rock samples, samples from streets and fields of the village, but not from control villages 4 and 7 km further up the valley. These fibers were known to be erionite type zeolite. Thus, it would seem that fiber-shape and size is important in the eitology of malignant mesotheliома and fibrous minerals from sources other than asbestos mining or processing may be implicated in the epidemiology of this disease. (37, 38)

14. The crucial questions are whether asbestos can be used safely in manufacturing and whether, because of the neighborhood and domestic cases of mesotheliома which have occurred, presumably to levels of exposure less than in occupational situations, asbestos might pose a significant public health risk to the community. Sufficient time has not yet elapsed to verify the efficiency of modern dust control methods and work practices but the preliminary indications are that the answer will be in the affirmative with regard to the first question. The second question must be answered in a similar vein but as yet there is no evidence, indeed no means of collecting evidence, to indicate that the level of asbestos in the ambient air and general environment poses a significant health threat.

BIBLIOGRAPHY - SECTION A

1. Campbell, W. J., Blake R. L., Brown L. L., Cather E. E., and Sjoberg J. J.: Selected silicate minerals and their asbestiform varieties: mineralogical definitions and identification - characterization.
U. S. Dept. of the Interior, Bureau of Mines Information Circular 8751, 1977.

2. Cooke, W. E.: Pulmonary Asbestosis. Brit. med. J. 2: 1024 (1927).

3. Asbestos Industry Regulations, 1931. Statutory Rules and Orders No. 1140. (H.M.S.O.).

4. Cooke, W. E.: Fibrosis of the lungs due to inhalation of asbestos Brit. med. J. 2: 147 (1924).

5. Thomson, J. G.: Asbestos and the Urban Dweller
Ann. N. Y. Acad. Sc. 132 (Art 1.): 196 (1965).

6. Hunt, R: Routine Lung Function Studies on 830 Employees in an Asbestos Processing Factory. Ann. N. Y. Acad. Sc. 132 (Art 1.). 406 (1965).

7. Selikoff, I. J.; Lee, Douglas H. K.: Asbestos and Disease. (pp. 157)
Academic Press, Inc. 1978.

8. Bader, Mortimer E.; Bader, Richard A., Tierstein, A. S., Selikoff,

I. J.: Pulmonary Function in Asbestoses: Serial Tests in A Long-Term Prospective Study. Ann. N. Y. Acad. Se. 132 (Art 1.) 391. (1965)

9. Murphy, RLN., Gaensler, E. A., Ferris, B. G., Fitzgerald, M., Morrisey, W: Diagnosis of Asbestoses. Am. J. Med. 65: 498, 1978.

10. Becklake, M.: Lung Function. In: Biological effect of asbestos. International Agency for Research on Cancer, Lyon 1973 pp. 31-39.

11. Zitting, A., Huuskonen, M. S., Alanko, K., Mattsson, T.: Radiographic and physiological findings in patients with asbestosis. Scand. j. work environ. and health 4: 275-283, 1978.

12. Leathart, G. L.: Pulmonary Function Tests in Asbestos Workers. Trans. Soc. Occup. Med. 18: 49. 1969

13. Harries, P. G.: Clinical signs. In: Biological Effects of asbestos. International Agency for Research on Cancer, Lyon, 1973. pp. 19-24.

14. British Occupational Hygiene Society: Hygiene standards for chrysotile asbestos dust. Ann. Occup. Hyg. 11: 47-69. 1968

15. Asbestosis in Relation to type of fibre, dose, occupation and duration of exposure. (Chairman - M. Navratil; Rapporteur - H. Weill)

In: Biological Effects of Asbestos. International Agency for Research on Cancer, Lyon, 1973 pp. 155-165.

16. Becklake, M. R.: Asbestos-related diseases of the lung and other organs: Their epidemiology and implications for clinical practice. Am. Rev. Respir. Dis. 114: 187-227, 1976.

17. Selikoff, I. J., Hammond, E. C. and Churg, J.: Mortality experiences of asbestos insulation workers. Proceedings International Conference on Pneumoconioses, Johannesburg. H. A. Shapiro (ed.) Cape Town, Oxford University Press, pp. 180-186, 1970

18. Selikoff, I. J., Churg, J., and Hammond, E. C.: The occurrence of asbestosis among insulation workers in the United States. Ann. N. Y. Acad. Sci. 132, 139.

19. Harries, P. G.: Asbestos hazards in naval dockyards. Annals of Occupational Hygiene 11: 134.

20. Selikoff, I. J. and Hammond, E. Cuyler.,: Asbestos-associated disease in United States shipyards. CA-A Cancer Journal for Clinicians, 28: 87, 1978

21. Lynch, K. M. and Smith, A.: Pulmonary Asbestosis III: Carcinoma of Lung in Asbesto-Silicosis. Am. J. Cancer, 24: 56, 1935

22. Doll, R.: Mortatily from lung cancer in asbestos workers. Brit. J. industr. Med. 12: 81, 1955

23. Selikoff, I. J., Hammond, E. C., and Seidman, H.: Cancer risk of insulation workers in the United States. In: Biological effects of asbestos. International Agency for Research on Cancer, Lyon, pp. 31-39, 1973.

24. P. C. Elmes: Health Parameters other than mesothelioma. Department of Mines. Asbestos Symposium. Johannesburg, 1977 M. W. Glen (editor) pp. 9-16.

25. Knox, J. F., Holmes, S., Doll, R., and Hill, I. D.: Mortatily from lung cancer and other causes among workers in an asbestos textile factory Brit. J. industr. Med. 25: 293, 1968.

26. Peto, J., Doll, R., Howard, S., Kinlen, L. J., Lewinsohn, H. C.: A mortality study among workers in an English asbestos factory Brit. J. industr. Med. 34: 169, 1977.

27. Wagner, J. C.: Some pathological aspects of asbestosis in the Union of South Africa. Proceedings of the Pneumoconiosis Conference 1959, Johannesburg: Little, Brown & Co., Boston, 1960.

28. Newhouse, M. L., Thompson, H.: Epidemiology of Mesothelial Tumors in the London area. Ann. N. Y. Acad. Sci. 132 (Art 1.) 579, 1965.

29. Elmes, P. C.: The epidemiology and clinical features of asbestosis and related diseases. Postgrad. Med. J. 42: 623, 1966.

30. Rubino, G. F., et al: Epidemiology of pleural mesothelioma in Canada. Brit. J. industr. Med. 29: 436, 1972.

31. Lieben, J. & Pistawka, H: Mesothelioma and Asbestos Exposure. Arch. Environ. Health, 14: 559 1967.

32. Report of the Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer. In: Biological Effects of Asbestos. IARC Scientific Publications No. 8; Lyon, 1973 pp. 341-346.

33. Timbrell, V.: Physical Factors as Aetiological Mechanisms. In: Biological Effects of Asbestos. IARC Scientific Publications No. 8, Lyon, 1973 pp. 295-303.

34. Mc Donald, J. C. and Mc Donald A. D.: Epidemiology of Mesothelioma from Estimated Incidence.
Preventive Medicine, 6: 426-446, 1977.

35. Baris, Y. I., Arvinli, M, Sahin, A. A.: Environmental mesothelioma in Turkey (in press) Abstract only available)

36. Baris Y. I., Sahin, A. A., Ozesmi, N., Kerse, I., Ozen, E.,

Kolacan, B., Altimers, M., Goktepeli, A.: An outbreak of pleural mesothelioma and chronic fibrosing pleurisy in the village of Karain - Urgup in Anatolia. Therax, 33: 181-192, 1978.

37. Stanton, Mearl F: Some etiologic considerations of fiber carcinogenesis. In: Biological Effects of Asbestos, IARC Scientific Publications No. 8., Lyon, pp. 289-294.

38. Maroudas, N. G., O'Neill, C. H. and Stanton, M. F.: Fibroblast anchorage in carcinogenesis by fibers. Lancet, 1: 807-809, 1973.

B. The Diagnosis of Asbestos-Related Diseases

1. The diagnosis of asbestos-related diseases is not a simple matter because it depends upon the standards adopted by the examining physicians. (1) The Asbestosis Working Group of the Sub-Committee on Environmental Health of the Canadian Department of National Health and Welfare recommend that "uniform medical criteria for diagnosis should be established."

"The art of diagnosis, always a matter of weighing probabilities and of looking at the total evidence, is seldom subjected to greater challenge than in the lesser stages of asbestosis." (2)

2. Asbestosis as a disease-entity does not differ from all other forms of fibrotic lung disease. The diagnosis of asbestosis is always open to doubt in the living subject, and even the diagnosis of a histological specimen obtained by biopsy or at post mortem can be difficult. This is because asbestosis is a form of diffuse pulmonary fibrosis. This condition is not uncommon in all walks of life. (3) Asbestos bodies are found in the sputum of up to 25 per cent of all adults, whatever their occupation. (4)

3. An adequate definition of the term "asbestosis" is therefore required before a diagnosis can be made labelling an individual as suffering from it for the rest of his or her lifetime. The definition which most adequately fits the condition is as follows:

Asbestosis is a type of diffuse interstitial fibrosis of the lung which does not have a specific histological pattern to

distinguish it from interstitial fibrosis resulting from other causes, but which occurs in association with a history of exposure to the commonly used varieties of asbestiform minerals and may be characterized by the presence of asbestos bodies and asbestos fibers in the lungs greatly in excess of those seen in the general population.

4. Asbestos fibers also cause fibrosis of the pleura, and this may occur together with asbestosis of the lungs. Pleural fibrosis affects the parietal (lining of the chest wall) and visceral (lining of the lung itself) surfaces differently. Plaques of collagenous connective tissue develop on the parietal surface of the pleura along the lower ribs in the lateral and posterior surfaces and can also involve the diaphragm and sometimes the pericardium. Calcium may be deposited in these plaques after many years and they then become visible on chest radiographs. They are sometimes discovered during chest surgery and are often found at post-mortem, even when not seen on x-rays during life. This is because they frequently occur in areas not seen on the routine posterior-anterior chest radiograph. Special radiographic techniques may be required to detect them. (5)

The clinical effects of pleural plaques alone are minimal. (6) Plaques seldom produce symptoms and the only detectable effect has been a small reduction in lung function in studies of large groups of people. (7) When pleural plaques are large they may cause some measurable restriction in lung volume.

Calcified pleural plaques have to be distinguished from old haemorrhagic pleural effusions, and infection such as tuberculosis or empyema. Such calcified lesions are usually unilateral whereas pleural plaques seen as a result of exposure to asbestos are usually bilateral.

The visceral pleura may react acutely to asbestos and this will result in a form of pleurisy involving the bases and costophrenic angles with effusion (fluid). It may be unilateral at first and then affect the other side or may occur first on one side and then the other. It is important to be aware of this condition as it may not be recognised unless an exposure history is obtained for asbestos. It is important to differentiate it from malignant mesothelioma. (8-10)

Thickening of the visceral pleura occurs in asbestosis but is usually only detected on microscopy, although in a few cases of asbestosis it may develop over the lung bas.. showing up on a chest radiograph as a veiling which obscures the normal lung markings. In many films with advanced asbestosis it is difficult to determine how much of the altered pattern is due to lung disease and how much to fibrosis of the visceral pleura.

5. The diagnosis of asbestosis depends on:

(a) History of past exposure to asbestos dust: History taking requires scrupulous care and this means that it is as important to extract from the individual as accurate details as are possible on the nature of past occupations and exposure to asbestos dust as it is

to note details of past chest illnesses and injuries. The history is vital, but it must be properly taken and critically appraised, always being aware that memory of details of past occupations by the individual or relatives can often be imperfect and lead, if not recognised, at least to false statistics or embarrassment.

(b) Clinical signs and symptoms: Breathlessness on exertion is the commonest first symptom. Cough is not an early feature, but may occur later and be distressing. Sputum production appears to be associated with cigarette smoking. Some persons complain of aches and transient sharp chest pains, but they are rarely severe. Persistent pain should arouse suspicion of malignant disease or mesothelioma. The most important sign detected on physical examination is the finding of persistent high-pitched fine crackling sounds (crepitations, rales), best heard at the end of a maximum inspiration and persisting after coughing. These crackles are initially present in the lower axilla and extend later to a wider area. These sounds may be recorded electronically but the equipment is costly and cumbersome and reliance is mainly on the auscultatory technique of the physicians. Other physical signs, such as clubbing of the fingers are of little diagnostic value as they are not invariably present or necessarily related to asbestosis.

(c) Chest radiographic appearances: An internationally accepted system of recording radiographic appearances, applicable to the pneumoconioses, including asbestosis, became available in 1971.

It is known as the ILO U/C 1971 International Classification of Radiographs of the pneumoconioses and is obtainable from the International Labor Office, Geneva, Switzerland. The classification has recently been revised and a new version should become available this year. The lack of an adequate standardized classification prior to 1971 makes it imperative to re-read and report on all chest radiographs taken prior to this time as significant changes may have been overlooked or incorrectly reported.

The ILO U/C classification does not attempt to interpret the cause of the changes it describes and it cannot be used diagnostically without additional information such as occupational or exposure history, symptoms, signs and results of special laboratory tests performed. It's main use is as an epidemiological tool and it is extremely useful in assessing the incidence of pneumoconioses in study populations and this may be correlated with dust conditions enabling an evaluation of the effects of control measures which have been introduced. The standard films should always be compared with the films being read if errors in assessment of the degree of change are to be avoided.

The use of radiography in the medical surveillance of asbestos workers is accepted as invaluable, but a single radiograph, unless compared with earlier films, may be of little value in diagnosing asbestosis. In order to confirm the radiographic diagnosis it is essential to review all the available chest radiographs, preferably from the date of first exposure to the present time, taking into

consideration the additional information itemised in the preceding paragraph. It should always be remembered that the radiograph provides the most important diagnostic information, but the changes are not specific to asbestosis.

(d) Lung function:

Surveillance of asbestos workers is designed to detect the earliest effects of exposure as soon as possible so that advice may be given to the individual and the employer regarding job continuation or removal to alternative employment. It was believed at one time that alterations in certain lung function tests gave such an indication of the early effects of exposure. It should be recognised that the changes in lung function which can be measured are non-specific for asbestosis, but restrictive lung disease, if present, is important for diagnosis. Since asbestosis is usually a slowly progressive lesion and there may be many years without demonstrable changes occurring in radiographs, symptoms or lung function, it is necessary to establish a physiological base-line for evidence of deterioration in lung function.

Many factors other than asbestos dust exposure affect lung function results including other types of pulmonary fibrosis other forms of lung disease, cigarette smoking, equipment and techniques of measurement. An isolated abnormal result in an individual is not of specific diagnostic value and should be repeated and considered in relation to all the available additional information relating to the individual.

Established asbestosis causes a loss of diffusing capacity,

age-adjusted cancer death rates in general are leveling off and in some cases dropping off. Lung cancer is the number one site of cancer deaths among men. The death rate of men has increased more than 25 times in 45 years. The death rate is going up steadily for women. "Lung cancer is largely preventable, since 80% of it is caused by cigarette smoking," according to the American Cancer Society.

In view of this high incidence of lung cancer in the general population, especially in the cigarette smoking portion of the population, it is difficult to be certain in an asbestos worker who smokes cigarettes and also has none of the diagnostic criteria of asbestosis, and who developes a bronchial cancer, that it is in any way occupationally related. Lung cancer is such a common disease in males between the ages of 35-64 that cases will occur in asbestos workers even if the dust exposures have been at a level too low to increase the risk appreciably. The high incidence due to cigarette smoking alone will lead to cases occurring in asbestos workers without asbestosis. For the large series of cases studied in the USA and Canada by Selikoff, no evidence is presented showing that the excess cases (i.e. the number of cases observed over and above those expected) always have asbestosis. According to various authors, lung cancer is rare in non-smoking asbestos workers and the effects of cigarette smoking and asbestos dust are multiplicative. In the United Kingdom, acceptance of lung cancer in an asbestos worker as due to occupational exposure usually depends on the finding of evidence of asbestosis.

vital capacity, ventilatory capacity (restrictive) and compliance. Pleural plaques by themselves do not. Lung function tests may be normal even in the presence of other features of asbestosis or vice versa, may be abnormal in some exposed workers who have no symptoms or physical signs. Lung function tests would be of value if they were specific fo. the diagnosis of asbestosis, especially where the diagnosis is questionable, but there is some doubt about their ability to do this.

6. To summarize, therefore, it may be stated that, on current evidence, no single parameter gives a more sensitive indication than any of the others available in all situations. The chest x-ray, when classified on the ILO U/C 1971 scale by at least three experienced readers, is probably the most sensitive and specific single index (11-12). In diagnosing asbestosis several indices when combined and studied in relationship to each other, are preferable to a single index. Asbestosis is not easy to diagnose and all suspected cases should be referred to a competent and experienced group of physicians with special knowledge of occupational lung disease. This is one condition where a consensus opinion is invaluable, especially when the question of compensation arises.

7. The diagnosis of lung cancer is not difficult in comparison to asbestosis, but the decision as to whether it is due to occupational causes is not so straightforward. Asbestosis is a specific disease caused by the dust, whereas bronchical cancer is not, and in the individual case cannot be separable from that due to cigarette smoking alone. In the United States of America, except for cancer of the lung,

8. For the purpose of administration of a program to compensate asbestos-related diseases it is essential that a suitable definition of occupational lung cancer be devised. It is suggested that the following will suffice:

Lung cancer irrespective of histological type, occurring in an asbestos worker more than 20 years after first exposure with documented evidence of asbestosis diagnosed during life or discovered post-mortem, will be regarded as being of occupational origin. In the absence of evidence of asbestosis a history of continuous uninterrupted exposure to asbestos dust for 20 years or longer will also be regarded as confirmatory evidence of hazardous occupational exposure likely to be an etiological factor in the causation of disease. This definition applies regardless of the fact that work may have ceased many years prior to the diagnosis of this lung cancer.

9. Mesothelioma of the pleura and peritoneum, a very rare form of cancer, has in the last 20 years been shown to be highly related in older age groups to past asbestos dust exposure. Despite it being an almost specific occupational disease of asbestos workers (in older age groups), the diagnosis can easily be missed, especially in the peritoneal cases. This means that a most meticulous occupational history must be taken in cases of pleural or peritoneal effusion of uncertain origin. The diagnostic significance of the interval between first exposure and the occurrence of a mesothelioma is more useful than in the case of lung cancers. Mesothelioma very rarely develop in less than 20 years after first exposure, and may be delayed to over 50 years. Thus,

a mesothelioma appearing within the much shorter period of 20 years requires special review to ensure:

(a) the diagnoses is correct

(b) the occupational and residential histories are adequate, including inquiries about possible exposure at work or in childhood, either through family contact or from playing on dumps of asbestos fiber, or being near an asbestos factory.

It is possible that when mesotheliomas are being intensively looked for and recorded, that chance alone will result in a few non-occupationally related cases being discovered in those with possible exposure to asbestos, because such exposure is a common occurrence in nearly all occupations. If a mesothelioma appears 2 or 3 years after the first possible exposure to asbestos, it is possible that it is caused by something other than asbestos. Whereas, in the case of asbestosis and bronchial cancer the type of fiber to which this person was exposed is of no diagnostic significance, mesotheliomas are more likely to be related to exposure to amphiboles and crocidolite in particular. As has been mentioned in Section A of this statement, mesotheliomas can be found endemically in certain areas of Turkey, indicating that other minerals besides asbestos may be factors in its causation.

10. The association of asbestos with other types of cancer depends mainly on evidence from epidemiological studies and is much less defined than in the case of lung cancer and mesothelioma. It would appear that further clinical investigations are needed to determine whether gastro-intestinal cancer or cancer of the larynx in asbestos workers have special features which could allow them to be distinguished from similar tumors occurring in the general population. In the absence of asbestosis or other features indicating the effects of asbestos exposure it is difficult to attribute other types of cancer to asbestos in an individual case, particularly if a history of cigarette smoking is also obtained.

In the Canadian Province of Ontario the Workers Compensation Board follow certain guidelines for the adjudication of claims for cancer of the gastro-intestinal tract in asbestos workers. The essence of these guidelines is that exposure to asbestos must be occupational before a causal relationship can be established. Before awarding compensation the Board has to establish "there is a clear and adequate history of occupational exposure to asbestos dust, and while such occupational exposure cannot be quantitatively described, it should be of a continuous and repetitive nature and should represent or be a manifestation of the major component of the occupational activity." There has to be a minimal interval of 20 years between first exposure to asbestos and the diagnosis of gastro-intestinal cancer.

BIBLIOGRAPHY-SECTION B

1. Lewinsohn, H.C.: Asbestosis - A Diagnostic Enigma. A Personal View Occ. Med. 19: 607, 1977

2. Selikoff, I. J., Lee, D.H.K.: Asbestos and Disease. Academic Press, Inc., New York, San Francisco, London, 1978.

3. Talbott, J.A. and Calkins, E.: J. Am. Med. Ass. 185, 911, 1964.

4. Mourihane, D.O'B., Lessof, L., Richardson, P.C.: Lancet, 1, 1069, 1966.

5. Prager, L. (editor): Asbestos-Related Disease pp. 46-74. Grune & Stratton, New York, San Francisco, London, 1978.

6. Leathart, GL: Pulmonary Function tests in asbestos workers Trans. Soc. Occup. Med. 18: 49-55, 1968.

7. Rochleau MR, Fournier-Massey CC, McDonald, JC. et al: Lung function in relation to chest radiographic changes in Quebec asbestos workers. Bull. Physiopathol. Respir. (Nancy) 6: 637, 1970.

8. Gaensler EA, Kaplan AJ: Asbestos pleural effusion. Ann. Intern. Med. 74: 1, 178-191, 1971.

9. Chahinian, P., Hirsch A, Bignon J. et al: Les pleurésies asbestosiques non tumorales. Rev. Fr. Mal. Respir. 1: 9-39, 1973.

10. Rissenstein, MB: Pleural effusions in asbestosis. N. Engl. J. Med. 290: 1025, 1974.

11. Sheers G., Rossiter, C.E., Gilson, J.C., Mackenzie, FAF: UK Naval Dockyards asbestosis study: radiological methods in the surveillance of workers exposed to asbestos. Brit. J. industr. Med. 35: 195, 1978.

12. Weill, H., Jones R., (ed): The chest roentgenogram as an epidemiologic tool. Report of Workshop. Arch. environm. Hlth. 30: 435, 1975.

C. Assessment of Disability and Bill H. R. 2760

1. As a result of my experience as a Pneumoconiosis Medical Officer in the United Kingdom, I have come to abide by certain definitions of loss of faculty, disability and disablement which are as follows:

Loss of faculty means the total or partial loss of the normal use of organs or parts of the body, or the distruction or impairment of physical or mental functions, (e.g. the power to grip, to walk, to remember, to digest, to hear or to ventilate the lungs).

Disability denotes the specific loss of capacity for any of the ordinary activities of life imposed by the loss of faculty (thus the loss or impairment of the power to grip may involve the inability to hold a tool or operate a machine or play baseball).

Disablement is used to denote the total of the disabilities, that is, the aggregate loss of health, strength and power to enjoy life, and it is on the assessment of this disablement that the level of compensation will be determined.

Thus, exposure to asbestos results in asbestosis which may lead to loss of faculty (e.g. breathlessness on effort) and this imposes various disabilities for work or play (e.g. inability to walk distances or run) which together make up the disablement resulting from the dust exposure.

2. Bill H. R. 2740, Definitions, Sec. 102 (f), (g), (h) and (i):
In the assessment of disability a judgment is required based upon earning capacity which may be advantageous to some individuals and disadvantageous to others. Because the definition of disability is based on a decline in the level of earnings estimated from a person's highest annual income in the previous ten years, an individual with a severe disablement may possibly be compensated at a lower level than another person with only mild disablement because of difference in earning power. It is my personal opinion that a more equitable solution to the problem would be to base the assessment on the degree of disablement using the measured impairment of lung function detected by means of specified tests of lung function using a percentage scale for calculating the award, with a minimum payment of 10%.

Asbestosis may be a progressive disease and, therefore, compensated persons should be re-examined at least every two years and their degree of disablement reassessed, with increases in their level of compensation being awarded if necessary.

The presence of malignant disease would automatically qualify the person for an award of 100%. Where other related diseases develop, such as tuberculosis, bronchiectasis or even bronchitis in some cases, they should be assessed as aggravating factors and the degree of disablement calculated to take account of them. If, for example, tuberculosis is treated and improved, the assessment can be reduced accordingly.

It is obvious that a great deal of expertise is required to make the necessary judgmental decisions as well as confirm the diagnosis. I will deal with this later.

3. Bill H. R. 2740

I should like to draw attention to certain aspects of this Bill and state my personal opinion regarding these items.

(a) Page 1. A. Bill: The inclusion of the phrase "and/or the inhalation of asbestos coupled with the inhalation of asbestos coupled with the inhalation of cigarette tobacco smoke" opens the door to all cigarette smokers who can prove any exposure whatever to asbestos, irrespective of fiber type or airborne concentrations of respirable fibers.

(b) Page 2. Title 1. General Provisions (3): My comments above also apply to this paragraph. Lung cancer in an asbestos worker should only be accepted as an occupational disease if there is evidence of significant exposure. It is possible to argue with regard to the definition of significant exposure but it cannot be disputed that the pulmonary fibrosis (asbestosis) is more likely proof of cause and effect in a smoker than the absence of any of the other stigmata of asbestos exposure.

(2) This paragraph, if included in the final Bill, would be a precedent setting step in workers compensation legislation and would allow non-occupational lung disease to be classified under compensatable illness.

To summarise this section, I would say that the cause is just, but certain aspects are overstated and will liberalise the dispensation of compensation to include possibly non-occupational illnesses. With regard to tobacco-smoking the question to ask is whether industry or the state should compensate persons for the life-style they choose and for their life-habits such as cigarette smoking. Should not such individuals who multiply the risks involved, be asked to contribute towards the costs of the insurance which this Bill proposes to provide them with?

(c) Page 4. Definitions Sec. 102 (b): The term "affected person", if it includes members of an asbestos workers household sets a dangerous precedent. Such a person may develop an illness similar to an asbestos-related disease but with significantly less exposure and causally unrelated. This notwithstanding, such an "affected person" would receive compensation for a naturally occurring disease indistinguishable from asbestos-related disease. "Affected person" is thus too broadly defined and should only include the reference to occupational exposure.

(d) Page 10. (k) Class III: I believe that the insertion of this clause is unworkable and unjustified unless it is socially accepted that such a penalty should be imposed upon the manufacturer or purveyor of a consumer commodity, irrespective of the wishes of the majority of the community who utilise the commodity. Should vintners, brewers

or distillers be held responsible and pay for deaths and injuries incurred in road accidents involving drunken drivers?

(e) Pages 10-12. Title II: Claims for Benefits Occurring on or Before December 31, 1979. Sec. 201 (b): There are no internationally defined standards for diagnosis of asbestosis and for the determination of disability resulting from asbestos-related disease.

The criteria outlined in this section need to be clearly laid down and expanded in the guidelines to be published at a later date.

Pages 11-12, lines 24 onwards: "The Secretary shall accept the interpretation by a physician who is Board certified or Board eligible in the fields of preventive medicine/ occupational health, pulmonary medicine or radiology of a chest roentgenogram which is of acceptable quality.... if such roentgenogram has been taken by a radiologist or qualified radiologic technologist or technician.." This is unsatisfactory because the diagnosis of asbestosis and mesothelioma are often clear-cut or easy to make. It would be best if panels of experts could be established to examine claimants or death claims, similar to the British Pneumoconiosis Medical Panels. (see attached) The radiological features in the early stages of asbestosis are not clearly defined or definable as they are in other

forms of pneumoconiosis such as silicosis or coal-workers p eumoconiosis, therefore, reliance has to be placed on the assessment of the person as a whole, using all the diagnostic parameters demanded in this section. Although a physician may be Board Certified or Board eligible in the fields mentioned, such a physician may have no special sknowldge of occupational lung disease in general or asbestos-related diseases in particular. Occupational lung diseases are rarely seen by the "generalist" in any special field and are usually referred to physicians or surge ns with a known interest in the subject. There are very few physicians truly qualified to diagnose the asbestos related dise ses and unless a group of such persons is appointed for the purpose of the administration of this act, many unwarranted claims will be incorrectly decided and, perhaps, some justified claims may even be dismissed.

I would recommend that the Secretary be empowered to appoint physicians competent to decide on the medical facts presented to them and to apportion the percentage of disablement assessed. Only reports on radiographs prepared by a panel of three NIOSH "B" readers should be accepted in evidence and strict criteria should be adopted for interpretation of radiographic findings based upon internationally accepted standards. Each and every claim should be decided by the physicians who should reach agreement before recommending compensation or rejecting the claim. If it is not possible for them to

agree, the claim should be referred to a referee appointed by the Secretary whose decision will be final.

This concludes my statement. Thank you for inviting me to attend these hearings. I trust that my contribution will be of value to this Subcommittee in its further deliberations.

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