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Dear Bob:

The brevity and simplicity of your questionnaire is appreciated but after several attempts I don't believe I can respond to your general inquiry by using it. You will forgive me therefore if I do it in letter form as follows.

Item 1a. As best I can learn, H. W. Johns of New York was the first in the U.S.A. to use asbestos in the manufacture of roofing, in 1858. The asbestos was obtained from deposits in nearby Staten Island. This was a rather small operation but grew rapidly to include textiles, insulations and packings. This expansion was undoubtedly accelerated by the development of asbestos deposits in Quebec that were started in the late 1870's.

Item 1b. It is almost certain that the earliest published observations of asbestos related disease were those from England and I don't know what to give you as a date for the U.S.A. I am aware that by the late 1920's and early 30's animal experimentation was being conducted at Saranac Lake, New York. I believe this stemmed predominantly from the interest of the Metropolitan Life Insurance Company.

Item 1c and d. I do not have any information on these questions. Mr. Fenner of Johns-Manville is going to try to develop information specifically for Johns-Manville and if he is able to do so I will forward this to you at a later date.

Item 1e. There are no data in the U.S.A. with respect to this question. These abnormalities have not been subject to report to any Federal or state agency in the past or present. Workmen's compensation cases might give some information along these lines but would, I believe, be so unsatisfactory as to not merit an effort in that direction.

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Item 1f. No system of notification either statutory or voluntary, exists in the U.S. insofar as I know.

Item 1g. Because of the magnitude I am loathe to embark on an answer to this series of requests. For mining and milling specifically I believe the data by McDonald and associates and the data from Finland are the only ones deserving the mantle of "epidemiological study". Neither of these are related to populations in the U.S.A. Adhering to U.S.A. data, I believe that the studies of Enterline and of Mancuso re mixed exposures would be the most representative. Enterline and also Weill have published some information with respect to asbestos-cement sheets and pipe. I don't know of any information on the manufacture of insulation but of course, Selikoff's extensive studies on insulation workers is available. I don't know of any epidemiological studies on friction materials. You can see that I have not mentioned all of the authors that appear in the literature and I am not certain that I have even mentioned the principle ones from the U.S.A. To explore this problem in a satisfactory way is just more of a task than I have time for at the moment and I suspect you know about as much about it as I do anyway. Perhaps you might want to make some use of the accumulated information developed by the ICEH under Dr. Pelnar. I am sure you would also find very extensive information in Dr. Selikoff's laboratory.

Item 1h. Confining the answers to information in the U.S.A. I am aware only of the work that has been done by Anderson and by Selikoff. Perhaps others have contributed in this regard.

Item 1i. Insofar as I know, all of the commonly used varieties of asbestos have been implicated with respect to pulmonary fibrosis and bronchogenic cancer. With respect to mesothelioma, chrysotile, amosite and crocidolite have been implicated while thus far tremolite and anthophyllite have not been. Insofar as I know, all varieties have been associated with pleural plaque development. Other diseases such as laryngeal and gastrointestinal tumors have not been sufficiently reported on to permit any kind of comment about kinds of fibers that might be associated with these disease abnormalities.

For all of my responses to Item 2, I am speaking only from my personal point of view.

With respect to item 2A. a 1&2, I must say that I don't believe that either macroscopic or microscopic appearances or examinati[illegible] are utilized for associating death with asbestos exposure. Th[illegible] association it seems to me is more related to severity of the fibrosis and whether or not causes totally unrelated to asbesto[illegible]

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exposure exist and more reasonably account for death. One also runs into the very difficult question of whether or not a bronchogenic cancer or gastrointestinal cancer or mesothelioma in the specific individual can be attributed to asbestos exposure. In this regard, one usually looks for a history of rather heavy exposure and some evidence of lung tissue reaction before making that kind of association.

Item 2Ab. It seems to me that the evidence favors the concept that mesothelioma associated with asbestos exposure might occur in the absence of recognizable fibrosis as diagnosed during life and possibly even by autopsy although this latter point has not been thoroughly examined. Because of a high incidence of bronchogenic cancer having nothing to do with asbestos exposure it is very difficult in my judgment to say that an individual who has bronchogenic cancer and who has been exposed to asbestos has necessarily a relation between the two. Where dose response data is available (thus shown by McDonald, et al), it appears clearly demonstrated that an excess of bronchogenic cancer occurs only after there has been a rather heavy cumulative exposure. I recognize that this is a relative term. At the moment I believe that when definite clinical evidence of pulmonary fibrosis ascribed to asbestos exposure coexist with bronchogenic cancer it is reasonable to make an association between the bronchogenic cancer and exposure to asbestos. Up to levels of fibrosis less than this I remain uncertain as to whether or not one can ascribe coexisting bronchogenic cancer to asbestos exposure in this specific individual. The McDonald's have also shown that an excess of gastrointestinal cancer appears only in those who are most heavily exposed and I believe the comments just made with respect to bronchogenic cancer would hold there also.

Item 2Ac. I believe this is answered more suitably by an experienced pathologist.

Item 2Ba 1, 2 and 3. The clinical diagnoses of asbestosis seems to be based on criteria that vary somewhat from country to country. In the U.S.A. I believe the diagnoses is most commonly made on the basis of radiological changes plus a reliable history of substantial exposure to respirable asbestos fibers plust the ability to exclude other causes of radiological changes that mimick those caused by asbestos. In this regard, a series of films over a span of several years showing persistence or perhaps progression of the change with no periods of regression is most helpful. Little dependence on basal crepitations is

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manifested in this country and I don't know of anybody who utilizes dyspnea as clinical evidence for the diagnosis of asbestosis. I might say that basal rales when present and determined to not be caused by congestive failure or chronic bronchitis are looked upon by some as helpful provided there is evidence in the radiograph. Dr. Murphy in the U.S. has underway a rather extensive study using recorded lung sounds in an effort to see whether or not abnormal lung sounds can be used in helping to establish diagnosis of asbestosis either with or without the presence of radiologic abnormalities.

Item 2Ca. I don't believe that any lung function changes can be regarded as pathognomonic of asbestos related disease. Every one of the measurements you list can be abnormal singly or in combination with each other as a result of diseases other than asbestosis. It can be said, however, that items 1, 2, 3 and 4 are commonly abnormal in individuals who have pulmonary asbestosis (fibrosis of the lung).

Item 2Da. The ILO/UICC system is not commonly used by most radiologists who on a day by day routine look at x-ray films of those persons who have been exposed to asbestos. On the other hand, when epidemiological studies are being carried out, I believe that the system is rather universally used in the U.S.A.

Item 2Db. For the most part in the U.S.A., a narrative x-ray reading is used for those persons who have been exposed to asbestos. This is somewhat in contrast to the custom in compensation proceedings for coal workers where the UICC system is rather universally used. I believe that in some of the larger employee populations being observed for control purposes the use of the UICC system is beginning to be utilized.

Item 2Ea & b. I believe I have indicated earlier, for both control and compensation purposes in the U.S.A., there is general use of a combination of (a) single 14 x 17 P.A. chest film, (b) FVC and FEV_1, (c) an estimation of cumulative exposure and (d) clinical information to exclude other causes of whatever manifestations might be present.

Item 2Fa, b & c. I don't quite understand this question. As accurate as possible an exposure history should always be obtained as a part of the mechanics of arriving at a diagnosis with respect to asbestos related disease. Along those lines one must explore the occupational, para-occupational and neighborhood exposures.

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Dr. George W. Wright to Dr. Robert Murray on the History and Diagnosis of Asbestos Disease (1978) Published by AsbestosClaims.Law
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As I am sure you are more than well aware, the variability between physicians and agencies with respect to the way in which they look at many of the questions you ask is considerable. I have tried to give you my own personal views in the matter. There is still some lack of agreement on the criteria for diagnosis of asbestos-related disease between those individuals responsible for carrying out this procedure. There also are considerable chances for difference of opinion when one looks at the question of the degree of certainty of the diagnosis. The criteria for certainty as opposed to suspect might be different.

It is regretable that we don't have better records with respect to the frequency of disease and the circumstances under which it occurred here in the U.S.A. Most of the compensation procedures are based on regulations put forth by the individual States and these have varied considerably across the Nation. Recordkeeping throughout the industry in the past has been rather poor but I think has improved markedly in the past few years, especially in the industries having larger numbers of employees. Unfortunately, the data is still rather meager and would pose almost unsurmountable obstacles to anyone who would attempt to collate the information that does exist. For this reason those who have wanted to examine one or another aspects of the overall relationship between asbestos and disease have had to start from scratch so to speak, and develop their data as best they can from what is available from past and contemporary records.

If you come to the U.S.A. sometime in the future, we would welcome seeing you here in Denver.

Cordially yours,

George W. Wright, M.D.

/jh
cc H. C. Lewinsohn

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