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February 23, 1944

Mr. Ivan Sabourin
Aldred Building
Montreal, P. Q.
Canada

Dear Mr. Sabourin:

While the matter is still fresh in my mind I am writing you at length on my impression of the situation on Asbestosis in Quebec in so far as it involves relationships between the producers, the medical profession and the Compensation Commission. One conference with Mr. Sharp, Dr. Vidal and Mr. Dick gave me a much clearer picture than I had gotten previously. Before doing so I offer a few comments on autopsy material.

I am enclosing my report on the lung tissues of Philemon Tardif. Of necessity the conclusions are not too satisfying as they are based only upon examination of two blocks of tissue, approximately 2 x 4 centimeters in diameter without even an indication of the parts of the lung from which they were taken. However, they like the material from the two previous cases, do demonstrate that the Thetford operations produce limited amounts of indisputable Asbestosis. In the presence of advanced tuberculosis this is obscured and not likely to be identified except on microscopic examination of pulmonary tissue. X-ray during life will not show it.

Today I have also examined in gross the sagital section through the entire right lung of Majorique Perron which we collected yesterday from the toxicological laboratory in Montreal. This is a much more satisfactory specimen with which to work. It likewise shows a combination of widespread chronic tuberculosis upon a background which will probably prove on microscopic examination to be minimal Asbestosis. Sections will be prepared as soon as possible for microscopic examination. In order to make my report complete in this case, I would appreciate it if you would send me a brief summary of the occupational and clinical history including: How long employed, character and date of first symptons, date of quitting work, whether Koch's bacillus was found in sputum and date of death. I neglected to copy this information from your record.

This autopsy material on fatal cases and the X-rays that are now reaching the Commission are bound to create in their minds the impression that tuberculosis is an all too common complication of Asbestosis. If I were in their position with my experience limited to evidence of this kind I would find it difficult to resist the conclusion that there is causal relationship between even slight amounts of

asbestosis and tuberculosis of the lungs. I have told you repeatedly as I told Mr. Sharp yesterday that I did not agree with this conclusion but I doubt whether he or Dr. Vidal will be persuaded when they see a succession of cases like those which are now passing before them. They must be made to realize that these cases are the exceptions rather than the rule and that there is a great majority of employees in the asbestos industry who are free from tuberculosis.

I would therefore urge that immediate steps be taken t survey every employee in the asbestos industry in Quebec so that the necessary statistics can be assembled. Dr. Stevenson's X-ray films alre taken on the men at Asbestos will provide you with material for a preliminary report. Coupled with this there might be an analysis of mortal data contrasting the total death rate from tuberculosis in Thetford and Asbestos with that in other industrial communities of the Province. It would be still more convincing if you could show the separate rates of males and females unless the number of the latter employed in the asbestos plants constitutes an appreciable proportion of the female population. Finally the age distribution of deaths from tuberculosis in those employed in the asbestos plants as contrasted with that in other occupations would help in determining whether the occupational factor wa contributory.

My own observations elsewhere and your intimation that the rate for tuberculosis was higher in St. Hyacinthe than in Thetford and / or Asbestos make me confident that the companies would acquire information of greatest service to themselves by such a survey and analysis. If asbestosis specifically is predisposing to tuberculosis in the same sense as silicosis does the effect should be just as apparent in North Carolina and Pennsylvania as it seems to be in Quebec or England But the films of employed asbestos workers in the States show a very low incidence of associated tuberculosis. It is my firm belief that in Quebec and England, factors outside the occupation are responsible.

As I sensed the situation in Montreal yesterday the asbestos industry will have to produce evidence to prove this point and start on it immediately or the industrial commission will become increasingly convinced that our position is biased and not founded upon truth.

Furthermore the industry would undoubtedly profit by undertaking a general program of community control of tuberculosis. By instituting case finding machinery not only among employees but among the members of their families, the open cases of tuberculosis would be discovered and the sources of spread of the infection could be removed.

One of the iron mining companies whom we serve has such a program with a public health nurse, a program for tuberculin testing of school children and follow up of the contacts of every known case of open tuberculosis. The program has paid dividends by practically eliminating tuberculosis with resultant lowering of compensation costs and the good will of the employees is unmeasurable. The asbestos industry of Quebec has a serious problem because its labor force is drawn from a population in which the tuberculosis rate is high but their situation is no worse than that which our other clients attacked successfully in an iron mining community.

The second point which is becoming increasingly apparent to me is that the asbestosis of the Quebec mines and mills is quite generally less severe and extensive than the asbestosis of the fabricating plants of the States. Comparison of the X-ray findings in the two groups reveals very few cases with typical patterns among Thetford or Asbestos mill men. Some asbestosis is unquestionably produced for we have microscopic evidence of it in the four autopsy cases now available but it is not advanced enough to replace much of the functional parts of the lungs and thus cause the X-ray shadows that are generally recognized as characteristic of the disease. (Most of us disagree with Dr. Pendergrass when he says in Lanza's book that there is no characteristic pattern of asbestosis). If my observations are correct this relatively slight amount of disease should not cause disability for only the framework of the lungs is involved leaving most of the parts that carry on the functions of respiration intact.

But here again the Canadian industry is on the defense against its medical profession and their representatives on the advisory board to the industrial commission. The doctors have seen only their own local manifestations of asbestosis without opportunity to compare it with the really severe manifestations that are produced elsewhere. They find the abnormal shadows in the X-ray films of exposed workmen, which they are convinced must cause symptoms and disability. They find asbestosis bodies in the lungs of autopsied cases associated with minimal tissue reactions or overwhelming manifestations of concurrent pulmonary infections and now they are sure of their ground.

In order to defend itself the industry in Quebec must have more facts which will require time to collect, analyze and publicize. These findings will have to be contrasted with those in the fabricating plants either in Asbestos or elsewhere. In the city of Asbestos the spinning and weaving mill has been in operation for only four years which is the minimum time to produce much disease in those exposed, but in another year or two one may expect to find evidence of it here unless I am much mistaken.

Mr. Ivan Sabourin (4) February 23, 1944

As I have told you in conversation, our experimental observations lead me to believe that the true hazard in asbestos plants is proportional to the amount of small, individual, uncrushed fibres in the industrial atmosphere. I suspect that the reason why one sees little or no typical asbestosis in the mines and mills is because there is probably too little such fibre in the air of these plants. Conversely the reason for the "typical" disease in the fabricating plants is the fact that the processes there open the bundles of asbestos fibre and tear them into lengths short enough to be inhaled in dangerous quantities.

The mills and mines are dusty but the dust is largely serpentine with relatively few fibres large enough to do harm to the lungs. This dust enters the lungs where it produces reaction around blood vessels and other framework structures that are just like any other inert dust. We see the shadows of such inert reaction manifested in X-ray as an exaggeration of the normal shadows of blood vessels. This condition is not asbestosis nor necessarily preasbestotic any more than the identical changes in men working in hard rock mines are necessarily presilicotic. In free silica industries regardless of exposure we do not diagnose silicosis from X-rays until we find the characteristic pattern of modulation; in the asbestos industry we should do likewise and refrain from a diagnosis of asbestosis until the X-ray reveals the characteristic pattern of haziness or fine stippled appearance spreading throughout the lower thirds or more of the lung fields. The situation with asbestosis is even more difficult than with silicosis for today the requisite condition of the dust exposure is not appreciated by the medical profession.

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Therefore I strongly urge that surveys be made to compare the relative quantity and nature of the dusts in the mills and the fabricating plants in order to present proof that in the first instance the percentage of thin fibre of inhalable dimensions is insignificant in comparison with the quantity created in spinning and weaving. It is going to be hard to persuade physicians of the importance of these factors but similar factors govern the reaction to quartz and the development of silicosis. These physical limitations of atmospheric dust often determine the outcome in contested cases involving a diagnosis of silicosis and I forsee that they will have equal weight in asbestosis. A competent industrial hygienist should collect and analyze enough of the atmospheric samples in weaving and spinning mills and in the mines and processing plants to establish this basic difference so that it will stand beyond controversy. We have the experimental background; we now must demonstrate its industrial application. When correlated with the results upon exposed workmen, I feel certain that our position will be secure.

To summarize: I would make the following recommendations to your clients, the Canadian Asbestos Producers in order that they may defend themselves against a growing conviction among the local physicians that employment in this Quebec industry is highly dangerous.

1. Correct an erroneous impression of the Industrial Commission that asbestosis is frequently associated with pulmonary tuberculosis.

   a) Statistical data based on X-ray of all employed workmen to indicate the incidence of pulmonary tuberculosis

   b) Comparative mortality statistics on tuberculosis in Asbestos and Thetford and in other communities of Quebec indicating the frequency by sex and age.

2. Consider the possibility of inaugurating a program of tuberculosis case finding and control in the general populations of Asbestos and Thetford in order to eliminate sources of new infection among employees.

3. Make a detailed comparative study of the nature of the dust in the fabricating plants and the asbestos mines and mills.

I trust that you will agree with these recommendations and be willing to pass them along to your clients.

May I take this opportunity to thank you for your delightful hospitality while I was in Montreal and thank you for making my visit profitable and pleasant.

Sincerely yours,

Leroy U. Gardner, M. D.
Director

LUG:RH

COPY

February 28, 1944.

Mr. Ivan Sabourin
Aldred Building
Montreal, P.Q.,
Canada

Dear Mr. Sabourin:

I have your letter of February 22 asking for comments on the relationship between asbestosis and susceptibility to tuberculosis. This question cannot be answered decisively from a study of post mortem material alone. Medical problems of this nature are usually solved on the basis of probabilities and it is this matter of probabilities on which we need further evidence in the case of tuberculosis associated with asbestosis.

Merewether in his "Memorandum on Asbestosis" Tubercle November and December, 1933 and January, 1934) admits that "the data on the subject are meager." He reports that of 374 workers while at work, only 4 showed signs of active tuberculosis and that the British Silicosis and Asbestos Medical Board in its first periodic examination of workers in this industry found only one case of asbestosis and tuberculosis in 1512 examinations. On the other hand the English post mortem material at that time revealed 35.7% of the cases complicated by tuberculosis. (Subsequently he appears to have found more cases if Dr. Riddell is correctly informed). In the publication cited Merewether concludes:

> "The evidence therefore, points to the hypothesis that at present the added risk to asbestos workers from pulmonary tuberculosis is limited and definitely less than in workers exposed to free silica; virulent massive infections will as in the general population, cause death; lesser ones will result in more widespread fibrosis than would appear in cases amongst the general population and slight infections, in common with many other affections, are likely to precipitate death in cases of advanced asbestosis where they wouldn't do so in the general population." The underscoring is mine but I agree with these views.

When Dr. Merewether was in this country last year he visited me for several days and we spent most of our time discussing these problems. He still accepts our experimental work which proves that there is no specific susceptibility to tuberculosis induced by asbestosis and he, like ourselves, is anxious to accumulate comparative statistics from various parts of the world to establish the general clinical experience.

As I indicated in my last letter the Quebec experience is not yet analyzed; there is only an impression that most of the asbestos workers die of tuberculosis without taking into account the excessive prevalence of such infection among the population of the Province. This impression needs verification by careful analysis of morbidity and mortality statistics collected from the employees of the asbestos industry, from their families not exposed to asbestos dust and from other communities in the Province of Quebec. Such data should then be compared with similar statistics for asbestos workers in other countries. If there is a specific occupational predisposition to tuberculosis it will become apparent on such analysus just as it has for silicosis. Wherever silicosis is being created, the high mortality from tuberculosis cannot be hidden.

I do not believe that there is much tuberculosis among asbestos workers in the United States and suspect that the excess mortality from this cause in England is probably due to the low living standards of industrial workers. Possibly selection of cases coming to autopsy has influenced the picture. American studies have thus far failed to demonstrate enough tuberculosis among asbestos workers to cause comment. The U. S. Public Health Service Study of Asbestosis in the Asbestos Textile Industry in North Carolina, (Bulletin 241), issued in 1938, hardly mentioned tuberculosis, dismissing the subject with the statement on page 51 that "there were only 2 cases of active, pulmonary tuberculosis in the group" (541 employees). Shull, (Radiology 27, No. 3, 279-292: 1936) examined 71 of 100 cases discharged from the plants where the Public Health Service made its study 15 months later and found 8 cases complicated by tuberculosis of slight to moderate extent but only 2 were active. These two subsequently died of this cause and in 2 others the infection later became active. Shull states in his conclusions that "asbestosis does not predispose to tuberculosis." Presumably he infers, although he does not say so, that the 4 cases of active disease among 541 workmen represented accumulated disabilities when the examination program was inaugurated. Donnelly (J. Ind. Hyg. and Tox. 18, No. 4, 222-228:1936) also examined 151 discharged employees including the 100 reported by Shull. He gives more details on the cases of tuberculosis; One of them had worked in the asbestos industry for 5 months and the others 15 months. The remaining 2 had worked 6 and 10 years respectively and their infections might have been aggravated by exposure to asbestos dust. He points out, however, that an incidence of 2 cases among 151 industrial employees is no higher than in any other industrial group. McPheeters, (J. Ind. Hyg. and Tox., 18, 229-239: 1936) likewise examined and reported on these cases and he is in entire agreement with the other reporters. In his conclusions he states, "No activating or aggravating effect of asbestos dust or asbestosis or tuberculosis was observed." Finally I believe I told you that I myself, had just returned from North Carolina where I had reviewed chest films of some 100 employees of the local asbestos industry and saw only one that showed any indication of adult type pulmonary tuberculosis. This was completely

healed. I commented on the fact to the members of the Medical Board of the State Industrial Commission with whom I was conferring and the Chairman, Dr. Vestal, told me that tuberculosis was a rare finding among asbestos workers although common enough in other industrial workers in their state. It would therefore, appear that in North Carolina asbestosis does not favor or predispose to tuberculosis.

I have just now completed a review of 125 films for an asbestos fabricating plant in another state. In this plant the amount of severity of the asbestosis on the first examination in 1930 was well marked, approximately 40% of the group showing definite X-ray evidence of the disease. Nevertheless, the total adult type tuberculosis rate was only 5%; four of these cases occurred in persons with asbestosis and 2 of them were considered probably active.

A second examination was made upon 67 of the original group after an interval of 5 years during which they remained at work. Now the incidence of asbestos had increased to 62% but the tuberculosis rate had fallen to 2.9%. A third examination was done on 57 of the original group who still remained in employment after a further interval of 3 to 4 years; in this group the incidence of asbestosis was over 80% but that fo tuberculosis only 3.5%. In spite of excessive amounts of severe asbestosis the tuberculosis rate remained within the limits usually found in American industrial populations.

The behavior of the tuberculosis in these people was not unusual. For example one man, a spinner for 13 years, was 31 years old when first examined. He had at that time an early non-specific dust reaction and evidence of old tuberculosis in the top of his lungs. In two succeeding examinations his dust reaction steadily increased to reach first stage asbestosis 9 years after he was first seen. His tuberculosis however, remained apparently uninfluenced. Most of the others behaved in the same manner. These people had already been exposed to asbestos dust for periods of 3 to 18 or more years when they were first examined and the three subsequent examinations during the following 9 years give us a good opportunity to follow what might have developed. No new case of tuberculosis appeared. If it had not by that time the chances were not very great that they would later.

After they left the asbestos industry we do not know what happened to most of them but on a few there are insurance records that indicate the cause of death. Two died of tuberculosis. One had had his infection when he was examined in 1930 at the age of 50. At that time he had an early asbestosis after weaving for 10 years and evidence of associated chronic tuberculosis. He was advised to leave which he did and died of his infection about 3 years later. The second man aged 32 had been a weaver for 12 years and had first stage asbestosis but no evidence of tuberculosis at the time. He left his job after the

first examination for reasons not stated. Three years later he was reported to have died of tuberculosis in a sanitorium. Such cases could occur anywhere.

It is possible that if we had follow-ups on more of these cases after they left the asbestos industry we would find more persons who subsequently contracted tuberculosis and died of it. However, the evidence now available indicates no such tendency. Animal experiments, contrary to those upon silicosis, disclose no theoretical reasons for suspecting that asbestosis creates any special susceptibility to the tubercle bacillus of Koch.

I am becoming more and more convinced that that the high tuberculosis rates reported in English autopsy statistics are due to causes other than occupation. I believe that the same must hold true in Quebec where the amount of severity of the asbestos dust reaction is very much less than it is in the fabricating plants in the United States.

I believe I have covered the other points raised in your letter and will therefore not prolong this discussion further.

Very truly yours,

Leroy U. Gardner, M. D.
Director

LUG:RH

cc to Mr. Vandiver Brown