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Merewether

Merewether

H.K. Medical Inspector of Factories
Empire House
159, Gt. Charles St.,
Birmingham, 3

4th Dec., 1934

Dear Dr. McPheeters:

As promised I have thought over the points raised in your letter of the 15th October and have endeavoured to discuss them in the following pages. In view of the official post I hold, I must, however, state that the opinions expressed are only my own personal ones and not necessarily in accordance with the opinions of others competent to judge, nor assented to by the Home Office.

Before one can discuss profitably questions relating to the establishment of any compensation scheme it is essential for us to agree on one or two fundamental points: the first of these is:-
"With what aim is compensation to be paid?", i.e.

- (a) Merely a payment to a worker or his next of kin as some recompense for having had the misfortune to contract an occupational disease - in this case asbestosis, - or
- (b) As an effort to recompense a worker and those strictly dependent on him for actual financial loss directly occasioned by the effects of the disease.

In the first case the solution is easy since as soon as the disease is diagnosed definitely a payment can be made to the worker or to his estate and the matter is finished.

In the second case the problems raised are much more complicated, but payment of compensation on this principle is more just in its application and probably less expensive. In other words, in the first case payment is made irrespective of the presence or not of any disability due to the disease, and in the second case the presence of disability, or of death from the disease, is essential and the extent of the disability material in assessing the amount of compensation to be paid.

Now it is necessary to decide (1) what is disability and (2) whether any particular worker has the disease and is in fact disabled by it.

- (1) Disability is a function of effort or work - if a man is disabled he cannot work so well - and wages are a function of work done: therefore disability is, shortly, "inability to earn ordinary wages." Obviously if we apply the term in this sense, although an affected worker's

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physical condition may remain exactly the same, his disability will vary with the type of work to which he is put. This, as you will agree, is material in the case of asbestosis since one man with developed asbestosis can continue to earn his ordinary wages (in the absence of some intercurrent infection) if employed in one process (e.g. narrow band weaving) whereas he could not if employed in some other process (e.g. transporting sacks of asbestos, shovelling and mixing).

If, therefore, method (b) above is adopted as the basis of compensation, it follows that the amount of compensation to be paid does not depend only on the presence and extent of the disease, but also on the type of work of which he is now capable of doing, whether such work can be found for him, and the proportion the wages he can earn at the new kind of work bears to that which he earned at the old work.

Apart from such questions as adjustment of compensation in relation to number of dependants, a further subsidiary question has to be considered, that of the procedure where it is dangerous for the workman, although not totally disabled to continue work in the processes.

- (2) Whether any particular worker has the disease and is in fact disabled by it.

As just mentioned, the other factor in the assessment of "disablement" from asbestosis of a worker, is the presence and extent of the disease, and this is where, as Pancoast has so lucidly explained, we all come up against a considerable fog.

You will agree that it is easy enough, after consideration of the clinical and radiological features and the industrial history, to come to a correct decision in the majority of cases of advanced asbestosis, but is quite another matter to say in a particular case "This man has not got asbestosis and never will have if he has no more exposure to the dust." Not only, to my mind, are there no definitely recognisable stages of asbestosis, but the earliest definitely recognisable signs of asbestosis are of ominous import.

Let us put it this way:- From the practical point of view one can consider a series of asbestos workers, like those you have examined, and divide

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them up with fair ease into:-

1. Cases of advanced asbestosis with "disability" in the sense we have just considered.
2. Cases of developed asbestosis
(a) with "disability"
(b) without "disability"
3. Cases of diagnosable asbestosis with no appreciable "disability"
4. The remainder.

(I am leaving out for the moment any cases complicated with tuberculosis.)

It is Group 4 which is so very difficult to sort out. From the practical point of view of group compensation, it is not really important to do so, but if it is necessary for any reason to make a diagnosis and a prognosis in each individual case, then one does realise the complexity of the business.

Let us consider what types one will find in this Group 4, firstly from the clinical and radiological point of view,

- (a) The apparently perfectly normal: fine physical specimens with no abnormal clinical signs in the chest and no suspicious radiological features at all.
- (b) The same, but with an extremely slight general impairment of percussion note to very light percussion, and possibly a few scattered sticky pleural like crepitations at bases and mid-zones: no suspicious radiological features.
- (c) The same as (b), but with very slight "haziness" at the bases or in the mid-zones (in men), or in the mid-zones in women.

These three classes may be considered together:- Have they asbestosis? Will they ever have it if they have no further exposure to dust?

You will agree that there is no definitely diagnosable degree of asbestosis present, yet (c) represents the earliest recognisable changes. What then will happen to these people even with no further exposure? The reply is that it is impossible to answer this question without definite knowledge of the nature of the work on which they have been engaged, its dustiness and the length of exposure.

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exposure.

In other words "Have these workers trapped in their lungs the 'fibrosis-producing amount' of dust?" and if this question is answered in the affirmative, "Has a 'maturation' period sufficiently long to permit of the development of a diagnosable degree of asbestosis, elapsed? These two points are discussed slightly in sections VI, VIII, and X of my Memorandum.

Now it is possible, with precise knowledge of the process on which engaged, its dustiness, and the length of exposure, to consider individually the workers in classes (a), (b), and (c) and make a very accurate forecast of what will happen to them sooner or later. Some will never develop a disabling degree of asbestosis, some will be dead from it in three or four years.

The importance of this method of attack in these difficult cases is that with its aid one can separate these workers, all apparently healthy, all capable of passing the strictest life insurance examination into

- (1) Those which will develop definite asbestosis within a comparatively short space of time and should be re-examined at intervals of six months.
- (2) Those which one need not concern oneself about for a very varying and indefinite period.

In parenthesis one may note here another advantage. It is the above Group I of cases of rapidly developing asbestosis due to short exposure to dense concentrations of dust which are liable to discredit an organized system of examination by State experts for compensation purposes. For example such a case may be examined by the experts, and clinically and radiologically nothing definite is found; during the next year the worker develops tubercle, further examination discloses a radiological picture which is conclusive only as regards the tubercle; the worker dies within the next 12 months or so and post-mortem not only much tuberculosis is revealed but also extensive asbestosis. The question then raised is "Why did not the expert discover the asbestosis?", the explanation being, of course, that at the time of the first examination, although the worker had in his lungs, "a fibrosis-producing amount of dust", the fibrosis had not had time to mature; it did go on maturing steadily, however, until death, but at the time of the second examination the tuberculosis obscured and prevented recognition of what would have been radiologically a definitely diagnosable degree of asbestosis in the absence of tuberculosis.

Obviously therefore the history of exposure and the concentration of dust to which exposed is always important, and in some cases supremely important, if mistakes are to be avoided. Also whether there is or has been exposure to other dusts, the nature of them, and amount of exposure is also important, owing to the resulting modification of the radiographic picture. Incidentally I have seen one or two definite cases of combined silicosis and asbestosis,

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the two types of fibrosis in one case which came to autopsy occurring typically and independently.

- To revert to classes (b) and (c) (page 3), the significance of the very slight general impairment of note (less in degree than that one seen in definite asbestosis) and the sticky crepitations is of interest. I think that in workers who have only a short history of exposure and in whom there has been no time for the maturation of any appreciable degree of fibrosis, these signs indicate very recent or present exposure to dense concentrations of dust; I suppose they actually reflect mechanical blocking up of bronchioles with dust. Similar crepitations appear from time to time in cases of developed or advanced asbestosis due to other causes as well. I only mention these points because, when present in such cases as we are considering, they are suggestive of exposure to much dust.

(d) Workers with pre-existing pulmonary disease or sequelae:-

these, of course, may have had very varying lengths and degrees of exposure. With short exposure, the principles outlined above are helpful; with longer exposures, although the clinical signs of asbestosis, such as they are, may be totally or partially obscured, the radiological appearances, are however, as a rule, sufficiently evident somewhere in the film, especially if taken in conjunction with the history of exposure.

In this connection I must say how impressed I am with the "inevitability" of asbestosis: I have yet to come across convincing evidence that of two men working side by side at the same dusty process for the same length of time, one will be taken and the other left. Per contra, in silicosis, as you will agree, this does appear to occur. At the risk of wearying you I would repeat that with a history of a certain length of exposure to a certain concentration of dust one can say without fear of error that a particular worker must have an easily recognisable degree of asbestosis if we could take out his lungs and examine them.

(e) With concurrent pulmonary disease apart from tuberculosis.

Here, of course, until the acute or subacute condition clear up, nothing much can be done other than to consider from the industrial history, amount of exposure to asbestos, etc., whether it is possible for the worker to have developed a recognisable degree of asbestosis. Large hearts with associated congestive phenomena and pregnancy may produce misleading appearances in the radiographic appearances.

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(f) With Tuberculosis

(1) With pre-existing fibroid type:

Apart from the radiographic appearances at the bases, and of the other lung if unaffected or less affected, the slight impairment of note at the bases, as compared with the normal, together with the history of amount of exposure to asbestos are helpful.

(2) With concurrent acute or subacute infection.

Admittedly, you will agree, the diagnosis of the asbestosis element is often extremely difficult.

One type has already been mentioned on page 4. Another type is where the tuberculosis supervenes on a definite degree of asbestosis. The tubercular infection appears to have a pronounced effect on the radiographic appearances of asbestosis remote from the main site of the tubercular lesion, and diagnosis may be very difficult if the worker is seen then for the first time. However, as a rule, clinical examination of the chest does disclose some evidence of asbestosis in the remainder of the lungs. As I have mentioned elsewhere, it is the uniformity of the slightly abnormal physical signs found which are so suggestive of the diffuse fibrosis of asbestosis.

The Interaction of Asbestosis and Tuberculosis.

This aspect is being most actively discussed at the moment. Some points are discussed in Section IX of my Memorandum. To attempt to examine the question critically is beyond the limits of a letter, but it may be worth mentioning a few items which strike me, as the question is so important from the compensation point of view. These are:-

- (1) The number of verified deaths from asbestosis or asbestosis with tuberculosis in which full particulars available is still far too small to make any dogmatic assertions.
- (2) Many writers have assumed that necessarily, the relationship of asbestosis and tuberculosis should be the same as that which is accepted to exist between silicosis and tuberculosis: there is no reason why this must be so, since the diseases differ in several fundamental ways.
- (3) The figures extant relating to silicosis are compiled almost entirely from male deaths: a very large female element is employed in the asbestos trade: no account has been taken of this or of the proneness of adolescent females to tubercle.

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- (4) No effort has been made by writers to separate out from the deaths which have occurred from asbestosis with tuberculosis, those deaths which inevitably would have occurred from tubercle, and which so far as one can judge have not been accelerated by the presence of asbestosis.
- (5) At the present moment, - including all cases in which full particulars are available, - of the total deaths in this country from asbestosis and asbestosis with tuberculosis, 39 per cent had tuberculosis, and of the total deaths in this country from silicosis or silicosis with tuberculosis, 56 per cent had tuberculosis.
- (6) Therefore, emphasizing that the figures for asbestos and asbestosis with tuberculosis deaths are far too small to draw any conclusions, all one can say is that so far there is no evidence that the risk of tuberculosis supervening is so pronounced in asbestosis as in silicosis. If in addition one takes into account the factors mentioned in (4) above and in my Memorandum, the difference as to tubercle risk in the two diseases becomes more pronounced.

Still it is certainly no use arguing this point until we have sufficient statistics. The position is still, I think, exactly as given on page 21 of my paper. That there is a relationship between asbestosis and tuberculosis must be taken into account in any scheme of compensation, and knowledge is too slender at present to do otherwise than to say that compensation should be paid where death takes place from pulmonary tuberculosis and a recognisable degree of asbestosis is also found present, and that workers with tuberculosis should not be permitted to work in processes involving a risk of asbestosis.

Now with regard to your specific questions, you will have observed from the foregoing discussion and from the tenour of Dr. Pancoast's answers that both he and I hold the view that it would be impossible in the present state of knowledge to evolve a scheme of compensation for asbestosis based on the identification of a series of stages of the disease. All one can do is to decide whether workers:

- (1) Have a recognisable degree of the disease.
- (2) Have or have not pulmonary tuberculosis in addition.
- (3) Are or are not "distilled".
 - (a) Totally.
 - (b) Partially.

(Question 1. The minimal asbestosis lesion.

(Question 5. Is asbestosis a progressive disease, etc?

Since I hold the views I do concerning the "fibrosis producing amount" of asbestos dust (page 13 of my Memorandum), the answer to

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Question 5, in my view, is "Yes", and, moreover, when the earliest recognisable radiographic appearances (which can only be identified with a reasonable degree of certainty by comparison of the particular case in all its aspects with other cases - of which all the details, including radiograms and results of post-mortem examination are known, - in one's "library" of cases) appear, that amount of fibrosis is a present risk to the worker, as there is more fibrosis present than is reflected in the radiographic appearances and sufficient to militate against his recovery from, say, an attack of pneumonia. How far that "minimal" amount of fibrosis will "progress" depends on the amount of new fibrous tissue which will be laid down, which in turn depends on,

- (a) the amount of asbestos dust trapped in the lungs;
- (b) whether the "maturation" period has elapsed since last exposure to asbestos.

Of course, the maturation of the fibrous tissue involves also the production in severe cases, of local emphysematous bullae, which may rupture causing partial pneumothorax, and of local retractions and contractions and strangulation resulting in bronchiectasis and non-tubercular cavitation.

At the moment therefore I think one can say that the "minimal" asbestosis lesion is more of academic interest rather than of medico-legal importance, and really can only be thought of (in the living worker) as the opinion of a particular expert (with considerable experience of the disease and with a large "library" of cases and films for comparison purposes), on a particular case. A post-mortem standard of a "minimal" asbestotic lesion could, I suppose, be laid down.

Q.2.

Supposing a man, otherwise healthy, was put before one and he had this "minimal asbestotic lesion", and really minimal, i.e. the earliest degree reasonably recognisable by an unbiased expert as above, then I think the prognosis of the case is bad, although, very exceptionally, no inevitably so. One would have to consider the amount and extent of exposure and how far maturation had progressed; - and on these, apart from complications and the question of what further exposure to dust there will be, depends whether the man will live for years or die in a comparatively short space of time.

The "minimal lesion", therefore as assessed in terms of clinical signs and radiographic appearances, is, if expressed in terms of probable length of life, a variable standard.

Questions 3 and 4/

These have already been commented on. There are no definite stages of asbestosis: roughly one could grade them into early, developed, and advanced, and those with tubercle, but such a grading would neither necessarily be a measure of the "disability" nor of the prognosis.

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Question 6

This I have endeavoured to answer on page 7.

I think that now I have come to the end of the points you have raised in your letter. Porgive me if I have appeared dogmatic, that is only because I have been trying to give you my own feelings on the subject.

I am afraid this is a very long letter, but I have endeavoured to answer also other subsidiary questions which you would have probably wished to discuss if we had been face to face instead of conducting the matter by correspondence.

I don't suppose for one moment that you will agree with all I have said on this very difficult subject: I should like to know any points with which you are at variance with me and also any other points which you would like to discuss with me.

One last word, - Compensation, however generous, is at the best but a cruel recompense for Prevention, and this strikes me very forcibly in connection with asbestosis and silicosis. These are two very deadly diseases, are they not, and the outcome is so inevitable, granted sufficient exposure to the dusts. For these reasons and since the provision of compensation without enacting stringent measures directed against the suppression of dust in the causative industrial processes is liable to throw unexpected strains on the financial structure of the compensation scheme, it is very desirable to attack the problem from both aspects at the same time.

It has given me pleasure and profit to think over the points you have raised.

With kind regards,

Yours sincerely

M.R.A. Herzowether

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FACTORY DEPARTMENT—HOME OFFICE

Telephone: Central 6776



In reply, please address as below
H.M. MEDICAL INSPECTOR OF FACTORIES,
EMPIRE HOUSE,

GT. CHARLES STREET,
BIRMINGHAM, 3.

6th. November, 1935.

Personal

Leroy U. Gardner Esq., M.D.,
Director,

The Saranac Laboratory for the study of
Tuberculosis of,
The Edward L. Trudeau Foundation,
Post Office Box 661,
7, Church Street,
Saranac Lake,
NEW YORK, U.S.A.



Dear Dr. Gardner,

In continuation of my letter of the 12th.

August, I have now with the courtesy of Captain
J. G. Ross, Manager of the Asbestos Corporation Ltd, *of Canada,*
and Mr. W. A. Janitch, representative of the
Corporation in this country, obtained some accurate
information as to "King's Floats". This I enclose
and you will see that only about 10% of it is non-
fibrous material. The percentage of fibre is higher
than I expected. You will see, however, that in test
4 after washing ^{the} material ^{which} had passed through a 200
mesh sieve, only 8.65% of non-fibrous material was
obtained.

Of course one might argue that this method
of analysis is not sufficiently delicate to settle

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2.

LEROY U. GARDNER ESQ., M.D.

the question you raised in your letter of 25/7/35, but
it seems to me that if with this test only 8.65% of
the material which passed through the 200 mesh sieve
was non-fibrous any samples of ^{Kings} floate you used for your
experimental work would contain quite a high enough
percentage of fibre provided it was well mixed. I have
the small boxes of samples referred to in the test, if
you would like to further examine them yourself. Mr.
Janitch very kindly said that I could make what use I
liked of the facts in this test so I am enabled to send
it on to you.

I am not sure whether I mentioned it to you
before or not but I am being led to a conclusion of the
immense amount of importance of the fibrous nature of
asbestos dust; this because I have been making a little
enquiry into the effect of French Chalk which is non-
fibrous, and I have come to the conclusion that it
certainly does not have the effects of asbestos on the
lungs. Judging by the results of my examinations of
workers, etc, and it seems to me that the reason for
this is the different physical characters of French
Chalk and chrysotile asbestos, although they are
practically identical chemically.

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3.

LEROY D. GARDNER ESQ., M.D.

In this connection, the Canadian asbestos interests are inclined to think that Canadian chrysotile is less harmful than other varieties of asbestos on account of the fact that they feel certain that Asbestosis would have made itself so very much more evident amongst their workers at Thetford and elsewhere in Canada than it has done. This suggestion I have only heard in the last few days, but coming to my notice as it does after a considerable period of cogitating on the French Chalk problem, there may be something of truth in the suggestion, although, of course, nobody has yet produced any concrete evidence on the matter. It would, however, fit in with my theory as to the importance of the fibrous character of asbestos dust and of the non-fibrous character of French Chalk as an explanation of the different effects of these two dusts when inhaled into the lungs. Canadian chrysotile as compared with chrysotile from other sources certainly is more talcy or alippery to the feel.

I enclose you two extracts from the Annual Report of the Chief Inspector of Factories for the years 1933 and 1934. On pages 63 - 65 of the 1933

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4.

LEROY U. GARDNER ESQ., M.P.

report and page 15 of the 1934 report you will find a
very short synopsis of some of the results of this
enquiry of mine.

With kindest regards,

Yours very sincerely,

S. A. A. Mervin