ASBESTOS—A NEW HAZARD

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SYNOPSIS

In recent years there has been an increase in the incidence of a rare malignant tumour—mesothelioma—affecting the lining of the lung or abdominal organs. The evidence points to a relationship between the occurrence of this tumour and exposure to asbestos, and in some cases the exposure need only be slight both in intensity and in duration.

This means that not only the manufacturing, but also the user industries may be at risk, and new legislation will shortly be introduced with the aim of eliminating the hazard. This must ensure that the worker is fully protected but care must be taken not to introduce too fussy restrictions which would hamper industry without adding to safety.

Introduction

New legislation will shortly be introduced describing the measures to be taken to avoid exposure to asbestos in occupations not covered by the present Asbestos Regulations. The purpose of this paper is to explain the background to this.

It has been known since early in the century that people exposed to fairly high concentrations of asbestos over a long period of time are liable to develop a particular kind of dust disease of the lung called asbestosis. The main symptom is breathlessness, which is usually progressive, and death may occur after a few years. The Asbestos Regulations of 1931, which became effective in 1933, were designed to prevent this disease, and with the active and willing co-operation of the firms concerned, have largely succeeded in their purpose.

Later it was noticed that asbestos workers had an increased liability to cancer of the lung. Doll[1] found that, in the textile industry, in men exposed for 20 or more years, the risk of cancer of the lung was 10 times the normal.

Both these hazards—asbestosis and cancer of the lung—require considerable and prolonged exposure and so are confined to asbestos miners and workers in asbestos factories and textile mills; and they are now firmly under control. It is not with these that we are concerned here. It has been found that a third hazard exists. This is called mesothelioma, a malignant growth of the pleura (the lining membrane of the lung) or of the peritoneum (the lining membrane of the abdominal organs). But whereas to produce asbestosis about seven years of considerable exposure is normally required—and even longer in cancer of the lung due to asbestos—mesothelioma may occur, not only in heavily exposed workers, but in some cases where there has been a less severe and a shorter exposure.

Thus mesothelioma has been recorded in people who are only intermittently exposed at work, *e.g.* laggers, and in people who work in the vicinity of those who are intermittently exposed. It has also been recorded in the families of men who have been exposed, the presumption being that they may inhale the fibres from contaminated clothing; it has also occurred in people living near to, but not actually working in, an asbestos factory.

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The Proposed Regulations

Assuming that there is a causal relationship between slight asbestos exposure and mesothelioma, the implications are important. It means that many firms who never considered themselves to have an asbestos risk, in fact have one—not only to their laggers and occasional laggers, but also to those working in their near vicinity. The Ministry of Labour, advised by the Senior Medical Inspector of Factories (himself advised by a panel of experts) is revising the Asbestos Regulations of 1931 so as to cover not only the manufacturing industry, but also the user industries.

These new regulations may be difficult to apply and enforce because the interpretation of what constitutes " a process in which asbestos is processed or used, or in which it is applied to or removed from an article " may vary, and in marginal cases could depend on the attitude of management or of the Local Inspector of Factories. The big manufacturing firms require no guidance—three of them have established the Asbestos Research Council to sponsor research and to perfect working conditions. Indeed, they have recently issued a Recommended Code of Practice for handling asbestos products used in thermal insulation. But many small user firms who have no medical officers still have no idea that there is a possible hazard, and to them the new regulations, without more detailed explanation, might appear to be unnecessarily stringent.

The Relationship Between Light Exposure to Asbestos and Mesothelioma

There is a great deal of evidence of a relationship between light asbestos exposure and mesothelioma, some of it more reliable than others. It is often difficult to prove a medical fact conclusively, and one has to take a balanced view, based on the occupational and residential histories; the epidemiological figures; the clinical findings; the various pathological investigations and X-rays; and finally, in the last resort, the post-mortem findings. In the case of mesothelioma there are many difficulties. It normally takes about 40 years after the initial exposure for the disease to develop and this can make the occupational history a difficult one to establish as an ill man may find it hard to think back with accuracy all that length of time and the relatives may often be unreliable historians. The residential history, too, for the same reason, may be unreliable—a short period of residence

near an asbestos factory may not be recollected or even known. Although this may be regarded as giving an *under*-estimate of the number of cases giving an occupational or residential history of exposure it can also be interpreted the other way—it may be obvious that the interrogator is interested in asbestos exposure and the patient might think it is to his advantage to give himself the benefit of the doubt and say " yes ". He may also do so because he is very ill and tired.

Another difficulty is that there are many forms of asbestos commonly used in industry and they vary in their apparent carcinogenicity, crocidolite being the most, and amosite the least, associated with mesothelioma. In fact, there is no reliable evidence yet that amosite alone is carcinogenic.

The oil content of the fibres can also complicate the picture. Some mineral oils occur naturally in asbestos; some, particularly jute oil, contaminate asbestos in processing, storage, and transport. Moreover, untreated crocidolite and amosite (but not chrysotile) contain polycyclic aromatic hydrocarbons including the proven lung carcinogen 3 : 4 benzpyrene.

However, in spite of these valid points, much evidence remains although it must be subjected to close scrutiny. The incidence of mesothelioma is increasing—one hospital in London, and one in Belfast, are each admitting one new case a month.[2] When one considers that up to August 1966 there were only 500 authenticated cases in the literature this appears to be significant. But it can be argued that earlier in the century, with chest surgery, radiology, and pathology less advanced than it is today, many cases diagnosed as cancer of the lung or as T.B. were, in fact, mesotheliomas and so the increase is not as great as it may appear to be.

Eighty per cent of all cases have been shown to have had contact with asbestos, and sometimes the exposure has been short and insufficient to produce asbestosis.[3] Consecutive post-mortems of 500 people dying in the U.S.A. from any cause showed that 30% of the men and 20% of the women had evidence of exposure to asbestos. Six per cent of the men showed evidence of *heavy* exposure.

Animal experiments show that inoculation of any type of asbestos fibre into the peritoneum of rats will cause mesothelioma. The value of this evidence is small because injection of silica and other substances, none of which have been connected with mesothelioma in man will similarly cause the tumour to develop in rats.

Wagner, Sleggs, and Marchand[4] described 33 cases of pleural mesothelioma in the mining areas of South Africa, all but one exposed to asbestos. The type of asbestos was crocidolite in each case. Some of these cases had only minor exposure—in childhood—and were therefore residential and not occupational.

Kiviluoto[5] found a high incidence of pleural plaques in people living near an asbestos (anthophyllite) mine in Finland. Pleural plaques are thickenings of the lining membrane of the lungs, sometimes with calcification. They are so closely associated with exposure to asbestos that their presence can be taken as evidence of asbestos exposure.[6] They can be detected in life by X-rays and are easily demonstrated post-mortem.

Keal[7] followed up the cases of 23 women admitted to the London Hospital with asbestosis between the years 1948–1958. Fifteen had died, nine of them from peritoneal cancer.

Enticknap and Smither[8] mention primary peritoneal tumours reported in N. America in workers exposed to chrysotile. They also describe a pathological specimen of the lung of a South African, aged 44, who died of mesothelioma, who was not occupationally exposed, but who lived near asbestos dumps in his youth.

Newhouse and Thompson[9] did a retropsective survey of 83 cases of mesothelioma treated at the London Hospital going back to 1917. In 76 of the cases full occupational and residential histories were obtained. They compared them with the same number of in-patients from the same hospital (matched by sex and, as far as possible, by age) suffering from other diseases. Forty (52·6%) gave a history of occupational or domestic exposure, *i.e.* living in the same house as an asbestos worker, compared with nine (11·8%) in the other group. Of those who were not exposed occupationally or domestically, 30·6% of the mesothelioma patients, compared with 7·6% of the non-mesothelioma group lived within half a mile of an asbestos factory.

Most important, out of the 31 patients with occupational exposures, only ten were in jobs to which the Asbestos Regulations of 1931 applied. The remaining 21 were considered to be suffering from occupational mesothelioma, but they were not protected. There were nine " domestic " cases—seven women and two men. With the women, the usual history was of the wife who washed her husband's overalls. In one case, a relative said that the husband, a docker, used to come home " white with asbestos " every evening for three or four years, and his wife would brush him down. The two men were exposed when, as boys of eight or nine, their sisters used to come home from working in an asbestos factory with dust on their clothes. One of these girls died of asbestosis. The brother who died of pleural mesothelioma had no other exposure to asbestos.

In the *British Medical Journal*[10] it is stated: " *one woman who had calcified plaques on X-ray had been exposed 25 years before, when she held asbestos sheets intermittently over a period of six weeks, while her husband sawed them to make rabbit hutches.*"

Lancet[11] report on the death of an asbestos worker. The post-mortem in this case showed multiple mesotheliomas " typical of the late effects of asbestos ". During the deceased's last illness, asbestos particles were found in the sputum. His only known exposure to asbestos was for six months, 30 years before, on a grinding machine, when he was 16 years old. The pathologist, in evidence at the inquest, was satisfied that an exposure for six months, 30 years before, could have been the cause. The coroner directed that the papers be sent to the pneumoconiosis panel, in the hope of obtaining industrial death benefit for the widow.

Bohlig[12] found that although asbestosis is now a rare occupational disease in Germany the incidence of mesothelioma has risen markedly since 1955.

This briefly is the outline of the case. There seems to be no doubt that *prolonged* exposure to asbestos can cause mesothelioma and indeed it has recently been scheduled as an Industrial Disease; but this need occasion no undue concern now regarding initial exposures, as the firms and places where this could occur have long since taken stringent precautions. The problem is light and intermittent exposure, because this involves places of work not covered by the 1931 Regulations. The Ministry of Labour, in framing the new regulations, has decided to take no chances. Naturally, it has had authoritative advice, because the Advisory Panel on Asbestos, which reported to H.M. Senior Medical Inspector on existing medical knowledge on the health hazards of asbestos is composed of experts from the medical profession and the Medical and Chemical Branches of H.M. Factory Inspectorate.

As a further example of informed medical opinion, the following letter, dated 15.7.65, was sent by a distinguished London chest surgeon to a man's employers:

" *Dear Sirs,*

I have recently operated on one of your employees who had a cancer of his lung. When we came to examine the removed tissue, it was found that it had very probably been caused by breathing in asbestos dust. This is a known and serious cause of lung cancer. The employee in question had never worked with asbestos at any

time in his life, but I understand you periodically have asbestos sheathing removed from water-pipes running along the ceilings of some of your work rooms. While this is being done, the work people are present, and are therefore exposed to the resultant dust. Exposure, even for short periods, may have serious and lasting results, and I feel that these circumstances should therefore be brought urgently to your attention."

We must be careful not to get the matter out of proportion. Mesothelioma is still a comparatively rare disease, and there are as yet only about 500 authenticated cases reported. Moreover, it is known that some forms of asbestos are more likely carcinogens than others, *e.g.* crocidolite is the most likely to be dangerous, and the other forms far less likely, particularly amosite. Under the present Regulations there are two main exclusions:

(1). Processes which do not give rise to dust, *e.g.* where the asbestos is wet, or treated with grease, and

(2). Where the degree of exposure is sufficiently slight —no more than eight hours a week.

It has been suggested that these exclusions continue in the new Regulations, except that the eight hours a week be lowered still further. If, in addition, a form other than crocidolite is used, and an agreed low and apparently safe Maximum Allowable Concentration were introduced, this suggestion would appear to be reasonable. The National Institute of Health in the U.S.A. recommends an MAC of five million particles per cubic foot of air or 176 particles/cm^3. This should be lowered much further, and maybe allowed certain limits of variation according to the size of the particle.

The problem is a difficult one. The safety of the workman must be paramount, but in protecting him one must not stifle industry with restrictions which do not add to his safety. It is certain that many user firms will come within the scope of the new Regulations, which envisage segregation of other workers, exhaust ventilation, wet weaving, vacuum cleaning, protective clothing, *etc.* Equally, other user firms which satisfy the Inspector of Factories on all of a number of laid down safety measures should be allowed exemption. It seems certain that public opinion will also have to be more informed and disquiet allayed because more cases of mesothelioma are likely to occur as the latent period of development expires in people exposed before the danger was realised. Now that the hazard is recognised, with care and good management in dealing with it can be limited as effectively as asbestosis and cancer of the lung.

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DISCUSSION

Dr. H. C. LEWINSOHN said that he had little to add to the substance of Gadian's paper, but wished to draw attention more carefully to the follow-up of the study by Doll[1] in the textile industry and to show how effective the precautionary measures taken by the industry had been in reducing the risk of cancer of the lung, so that it was now no greater than expected in the population. He hoped that if this could be done with regard to asbestosis and cancer of the lung, the problem of mesothelioma would present no greater difficulty in its solution.

Table I showed the mortality in workers in scheduled areas in an asbestos textile factory. The Asbestos Industry Regulations of 1931 took effect in 1933 and therefore 1 January, 1933 was the date at which exposure was first looked at. The first group had had more than 10 years of exposure to asbestos at that time in January, 1933. The second group had had between five and ten years of exposure at that time, the next, less than five years, and 104 men had had no exposure at all—in other words, recent entrants to the industry. Some of the fourth group would be included in the fifth; they overlapped. Two cohorts therefore emerge consisting of men with more than 20 years exposure on 30 June, 1964 and more than 10 years exposure on 30 June, 1964. Looking at the causes of death from all cancers in these various groups and comparing them with the expected number of deaths in the population, it would be noticed that of the 57 who had had more than ten years exposure in 1933, the observed deaths from carcinoma were 19 as opposed to the expected 3·56(±4). Taking cancer of the lung and pleura specifically, 15 of the 19 were due to these causes as opposed to 1·6(±2) which would have been expected. There was about 14 times greater risk of developing cancer of the lung if a man commenced his working career ten years

TABLE I.—*Mortality in Workers in "Scheduled Areas" in an Asbestos Textile Factory*

Number in Group	Exposure at 1 Jan. 1933	Exposure at 30 June 1964	All Neoplasms Observed	All Neoplasms Expected	Cancer of Lung and Pleura Observed	Cancer of Lung and Pleura Expected
57	> 10 years	> 20 years	19	3·56	15	1·06
45	5–10 years		8	2·70	5	1·01
16	> 5 years		1	0·66	1	0·27
104	None		1	1·73	1	0·78
489	None	> 10 years	11	14·25	6	5·78
190*	None		2	2·16	1	0·15

* All female

before the Regulations became effective (1 January, 1933). If 15 were subtracted from the 19, the remaining four neoplasms were what one would expect in the population anyway. Table I illustrates that since 1933 the incidence of cancer of the lung and pleura in the exposed population studied has reached a level compatible with that expected in the general population. The same applies to the figures for all neoplasms. Gadian had said that this particular risk to the industry appeared to have been brought under control by the measures which had been taken and which were originated by the Regulations. These figures illustrate this quite clearly with regard to the workers in scheduled areas in this asbestos textile factory.

Mesothelioma of the pleura and also of the periteneum was not associated with the handling of asbestos until Wagner, Sleggs and Marchand[2] reported their findings in the crocidolite mining area of the North West Cape Province of South Africa. It was now generally accepted that this form of asbestos was an important cause of mesothelioma although the fact that in many cases of this condition no exposure whatever to asbestos of any kind could be ascertained leads one to wonder if crocidolite was the only cause of mesothelioma. The concept of dose relationship was challenged by the minimal exposure theory of which the symposium had already heard. The evidence in the medical literature other than that presented by Wagner *et al.* was by no means conclusive but was very suggestive. One should point out that *often although exposure of short duration might be on record* in an individual case, this did not necessarily imply the only exposure in that case, as some may have taken place which was either not recalled, recorded, or known of. Furthermore, exposure might be intermittent, but on the other hand, as in the case of laggers removing old lagging, it could be very heavy if work was done in a confined unventilated space.

The cases which were often quoted as having had minor exposure in childhood in the crocidolite mining area in fact had substantial exposure, as the roads were often made of asbestos slag, lorries trundled their loads through the villages spilling asbestos as they went, and the children played on the asbestos mine dumps.

On experimental evidence with regard to the role of the different types of asbestos in producing asbestosis, asbestosis with carcinoma, and mesothelioma, Wagner[3] has shown that the amphiboles (crocidolite and amosite) cause fibrosis more rapidly than chrysotile. As far as malignancy is concerned there appears from the South African evidence to be a definite association between exposure to crocidolite dust and the development of mesothelioma. With regard to carcinoma there is no definite indication that any type of asbestos is any more involved than any other. Preliminary results would indicate that chrysotile is more rapidly eliminated from the lungs and may even be soluble and absorbed and eventually excreted. Crocidolite and amosite remain in the lungs for much longer than chrysotile and seem to resist the tissue's attempts to get rid of them.

The main evidence suggesting that chrysotile is implicated in the development of the mesotheliomas comes from the United States of America. Firstly, Selikoff's studies and secondly, those in Pennsylvania by Mancuso, O'Donnell and Lieben. The Selikoff studies were criticised by the experts as they felt there was no proof of a definite chrysotile exposure, and some doubt on the fact that crocidolite may have been imported into the States earlier and in larger amounts than Selikoff stated. Information from Mancuso and O'Donnell is that there is no evidence that material other than chrysotile was used in Pennsylvania. In this country it is not possible to determine the exact nature of exposure in most cases and a mixed exposure is the almost invariable rule.

A further consideration to take into account when assessing the risks attached to the handling of any one particular type of asbestos is that chrysotile represents 95% of world production and is associated with a minimal and in fact doubtful incidence of mesothelioma whereas crocidolite comprises something between 3 and 3·5% of world production and is associated with a relatively large number of cases.

Gadian mentioned pleural plaques as an index of exposure to asbestos, and, in particular, mentioned the findings of Kiviluoto in Finland where anthophyllite was mined. As yet no cases of mesothelioma had been diagnosed from this area. In support of the environmental exposure theory, Gadian mentioned the domestic cases described by Dr. Muriel Newhouse in her 1965 publication. Dr. Lewinsohn pointed out that although nine cases of mesothelioma were found where the only exposure known was contact with a relative working in an asbestos factory, only two of the presumed " carriers " actually were found to have asbestosis. One would have expected in the 1930's that had these " carriers " been so contaminated as to expose their contacts, they themselves should have succumbed from the effects of the hazard. The case of the young man who was exposed to asbestos for six months, 30 years previously, when he had worked on a grinding machine, was probably an example of a heavy exposure. It was unlikely that 30 years ago, grinding would have been done under exhaust ventilation and using damping methods.

Gadian had stated the facts clearly, fairly, and rationally. The industry in which Dr. Lewinsohn was employed was aware of these facts and was doing all in its power to investigate alternatives for the known hazardous forms of asbestos, and was actively engaged in research and in encouraging research into the health hazards encountered in the handling of all forms of asbestos. As an example of this research, work done by the Physics Research Department of Turner Brothers Asbestos Co. Ltd on disintegration or wear of brake linings has shown that no free fibres can be recovered after braking tests and that a dehydrated magnesium silicate known as " Forsterite " appears to be the source of dusts. No information is available as to whether Forsterite can produce lung disease. Other work on this subject has shown that resinated particles can be recovered but no free fibres. Even if Forsterite is a health risk the dilutional effect of the atmosphere would, of course, minimise it. Mesothelioma should not be associated with asbestos brake linings as crocidolite is not used for this purpose.

Mr. D. L. WILLIAMS said that it had been made perfectly clear that exposure to the dust from asbestos need only be for a short period of time for it to have dangerous or serious effects. What risk did Gadian think there was from working permanently in a building which had asbestos sheeting.

Mr. E. W. F. WHALLEY asked whether Gadian thought that some people were more susceptible to the effects of asbestos than others.

Mr. B. Y. WALKER said that a television programme that dealt with the subject left the impression that it was only blue asbestos that was a cause of worry.

Dr. Gadian had not said that: would he therefore comment?

Mr. N. B. SIBLEY asked if there was any evidence that other silicate fibres such as mineral wool had the same hazard.

Mr. M. KNEALE said that many of the most useful products used were in fact asbestos-cement, not asbestos as such. He asked if there was anything to be said about that.

Mr. D. G. FURZEY asked what Gadian had to say about the fine asbestos dust arising from the disintegration of brake linings from the cars on the roads today.

Mr. C. BAILEY asked if Gadian could enlarge on the Government's proposed new Regulations. Intermittent exposure had been mentioned, but were the effects cumulative? If so, then he was concerned with such cases as the quoted example of the woman who developed calcified plaques, after working with her husband sawing up asbestos sheets for six weeks. If one assumed she was exposed for as much as 50 hours a week for this period, then on a comparative basis the present 8 hour permissible limit would allow considerable damage to occur after 9 months. On the same basis it would appear that even if this permissible limit is reduced by a factor of four in the new regulations, it would still seem inadequate.

Mr. W. P. HOWARD said that the actual imports of blue crocidolite only represented between 3 and $3\frac{1}{2}\%$ of total imports of asbestos fibre, and that the total percentage of chrysotile fibre in terms of commercial production represented 85%. In order to keep the whole subject in perspective, these figures should be remembered since the main risk of mesothelioma seemed to be from blue crocidolite.

Mr. A. V. BAILEY asked for interim advice on the direction in which specifications for lagging work could be improved in the immediate future.

Mr. K. OSBELDISTON said that he did not agree that laggers were intermittently exposed. He would have thought that laggers were regularly exposed to asbestos dust and certainly, in his experience, more laggers contracted asbestosis than other trades. He asked for Gadian's comments.

Hazards associated with asbestos were a subject which his company had studied in great detail. The risk now was that, as distinct from people working with the material who might contract asbestosis or mesothelioma, there were people working in the vicinity who might be exposed for a short time. He thought that this was where some tightening of the Regulations was required. It was covered to some extent by the new recommended Code of Practice produced by the Asbestosis Research Council but he thought that this was a particular aspect that should be emphasised.

Dr. GADIAN in reply, referred to the asbestos building and said he thought there was very little risk. The risk was when the sheeting was cut. The normal asbestos sheeting was in fact sufficiently protected from forming asbestos dust to prevent hazard. It was in maintenance and removal that some risk might be present.

It was definitely so that some people were more susceptible than others. There were people who had been exposed to the maximum over many years and had not developed either mesothelioma or, in some cases, asbestosis, whereas others had had the slighter exposure, and had. It was considered that there might be personal idiosyncracies, some sort of immune reaction on the part of some people. That was not peculiar to the asbestos hazard. It took place, for example, in the bladder cancer hazard where some men, before the danger was known, virtually wallowed in the bladder carcinogen and did not develop tumours, and others for whom precautions were taken did develop them.

He did not think it was the case that risk was only from blue asbestos, but it was accepted that blue asbestos unquestionably was by far the most carcinogenic.

With regard to other silicates, the hazard with asbestos was due to the fact of it being a fibrous silicate; non-fibrous silicate was harmless and he did not think that any other silicate had in fact been connected with mesothelioma.

With regard to the use of asbestos cement, the problem was the same as with asbestos sheeting. It was when the thing was broken up and asbestos dust might get about that any hazard would arise.

With regard to disintegration of brake linings, they presented a problem where people who were not occupationally, residentially, or in any way normally exposed to asbestos, were exposed to tiny amounts in the atmosphere from brake linings. That did not affect the issue any more than the fact that there were all sorts of dangerous carcinogenic fumes from chimneys and trains, *etc.* All that happened was that there was a slight inherent liability from living in the present age but one was liable to add to it oneself by smoking or exposing oneself to asbestos.

With regard to the new Regulations, the main line would be wetting, enclosure, separation, segregation, ventilation; these were the main lines; and definition at some point of what constituted the process involved.

On dust concentration, the figure given was purely empirical and was taken from a comparative silica figure.

Dr. A. WOLFF referred to the concern expressed by a previous speaker about the hazard to laggers. He thought that this might not be a serious problem as lagging was usually carried out in the wet state and, to the best of his knowledge, most of the lagging material was now based on mineral wool, and not on asbestos.

Dr. GADIAN said that the first thing would be, if one were starting a new man on lagging, one would preferably start a man over the age of 40. That was the accepted practice in many industries. A man should be informed if there was a true hazard. One hoped that there would not really be one but when there was a hazard, he should be properly protected. The methods used would naturally be the damping down of dust, ventilation, segregation, and protective clothing. He would have a respirator, a suit, gloves, boots, *etc.*, and vacuum cleaning should be provided. The amount of vacuum cleaning depended on how much and how often it took place. One would not get involved in highly expensive devices when the exposure was very slight. One had to be realistic about it.

Mr. OSBELDISTON asked Gadian whether more laggers suffered from asbestosis than other members of the population. Would he agree that laggers were exposed continuously rather than intermittently?

Dr. GADIAN said that it varied. Some were exposed continuously, some were not. Some were exposed intermittently because they were doing other jobs and came to lagging when the situation arose. He had no figures on the question whether more laggers suffered from mesothelioma than from asbestosis but obviously there must be fewer because mesothelioma was a very rare disease still.

He did not think that there should be a hazard from asbestos clothing in hot atmospheres. The hazard was only from an asbestos fibre being loose in the atmosphere and inhaled.

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