

PLAINTIFF'S EXHIBIT

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SOME OBSERVATIONS ON ASBESTOSIS

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Abstract—Asbestosis, which is a fatal occupational disease is still occurring 30 yr after steps were taken to eliminate the disease. This is because of expansion of the industry, and especially of the insulating uses of asbestos. Nearly half the certified cases in the last two years occurred in insulators, whose work is not covered by the regulations introduced 30 yr ago.

The work of a lagger in a building is described and it is concluded that mixing of magnesia/asbestos plaster is the chief hazard.

It is suggested that efforts should be made to find, and use, a substitute for asbestos-fibre and that distribution of ready-mixed plaster should be considered. In the meantime the Asbestos Industry Regulations and the Silicosis and Asbestosis (Medical Arrangements) Scheme should be expanded to include the lagger and his work.

Until such steps are taken this essentially preventable disease will continue to cause unnecessary death and suffering.

INTRODUCTION

ASBESTOSIS is a fatal disease. It is due to a spreading fibrosis which not only restricts the expansion of the lungs but also interferes with uptake of oxygen into the blood. Eventually it causes death by slow suffocation. In a minority of patients a fatal cancer of the lung develops, and we seem to have been seeing this termination more frequently in recent years.

The disease was first reported by AURIBAUT, (1906) who ascribed to this cause the deaths of sixteen workers in an asbestos textile mill between 1890 and 1895 the first British case being that of MURRAY, (1907). By 1934 WOOD and GLOYNE were able to report their experience of 100 cases, mainly derived from the textile mills, in this country. Following the report of MEREWETHER and PRICE, (1930) legislation was introduced to protect the health of workers handling asbestos but this took a few years to become effective.

This disease takes, on average, 20 yr to become manifest but if the measures taken in 1931-35 were fully effective, there should by now, some 30 yr later, be no new cases occurring. In fact it has been my experience that the disease is occurring increasingly frequently and the object of this paper is to examine this possibility and to describe the changing pattern of incidence of the disease.

CERTIFICATIONS

Figure 1 taken from LEATHART, (1962) shows that certifications of asbestosis have not shown a satisfactory fall in recent years and that they may even be increasing. Like all certification figures they must be interpreted cautiously as there may well have been changes in diagnostic accuracy during these 30 yr; and there have been alterations of the law, especially the Pneumoconiosis and Byssinosis Benefit Scheme of 1952, which have brought more men into the compensation scheme.

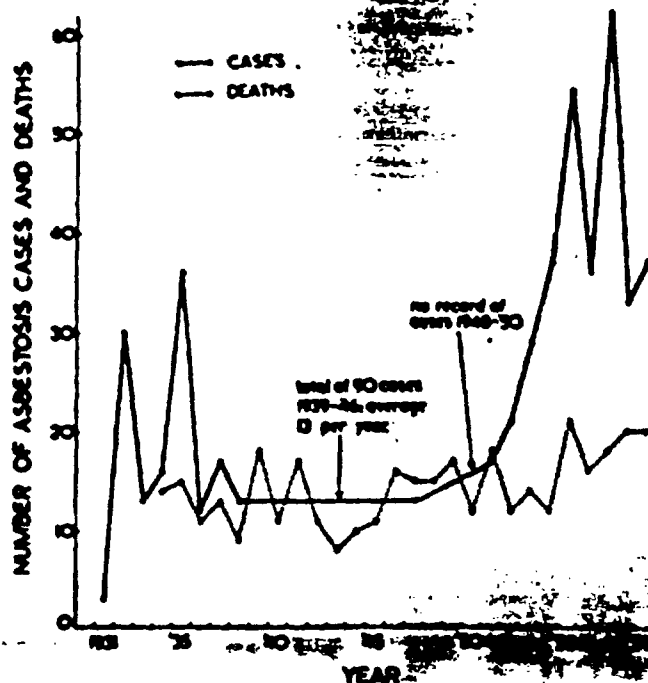


FIG. 1. Certifications of Asbestosis and Deaths, from 1931 to 1959. For the years 1952-59-42 cases certified under the Pneumoconiosis and Byssinosis Benefit Scheme have been added to the normal certifications (i.e., 7 per year).

Moreover the asbestos industry has expanded greatly during this period and there probably are more men exposed than there used to be.

THE ASBESTOS INDUSTRY

In 1935 the asbestos industry employed 10,000 workers; in 1958 there were 18,700 employees. Expansion of the industry is also reflected in increasing imports of asbestos, recorded in the annual reports of the Board of Trade, which are represented graphically in Fig. 2.

Most of the cases of asbestosis which are occurring nowadays started work in the industry before 1945. The further expansion of the industry since that date suggests that we may see many more cases in the future.

TABLE 1. ASBESTOS PRODUCTS IN THE UNITED KINGDOM (TONS)

	1930	1935	1958
1. Asbestos cement products	105,720	148,400	461,700
2. Packing, lagging, and jointing	2070	7263	42,900*
3. Brake and clutch linings	—	2243	10,000*
4. Other asbestos products (a)	22,274	22,187	—
5. Asbestos textiles (b)	—	—	16,424
6. Insulating materials (c)	—	—	36,000

(a) Mainly asbestos textiles

(b) Excluding those contained in 3

(c) Included under 2

* Approximate

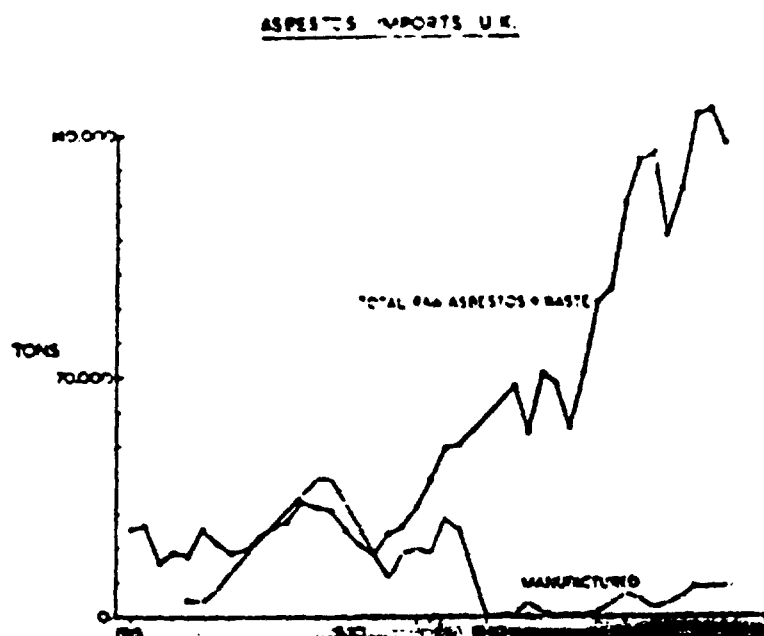


FIG. 2. Asbestos imports (tons) of the United Kingdom 1915-1957

The first cases of asbestosis were derived from asbestos textile mills, but analysis of the asbestos industry (based on the Board of Trade Censuses of Production) suggests that asbestos textiles have increased less than some other products (Table 1). It is unfortunate that information given by the censuses is not very detailed. However, it appears that the greatest actual increase has been in asbestos cement products, but the greatest percentage increase has been in insulating materials, which increased six-fold by weight, between 1935 and 1958.

CERTIFICATIONS IN DIFFERENT JOBS

Unhappily, growth of the insulating industry is also reflected in recent certifications of cases of asbestosis. The cases certified in 1960 and 1961 have been divided into groups derived from different occupations and are represented pictorially in Fig. 3*. It can be seen that 32 out of the 67 cases were employed on insulating or asbestos spraying.

The Asbestosis (Medical Arrangements) Scheme of 1931 is concerned with medical supervision of workers in textile mills and asbestos product factories and it is disturbing to find that in recent years the majority of cases of asbestosis have been occurring in the insulating industry which this Scheme does not cover. Moreover it is quite likely that regular medical supervision of insulators would reveal more cases than are at present coming to light through hospitals and chest clinics.

* I am indebted to Dr. J. WATKINS-PITCHFORD, of the Ministry of Pensions and National Insurance, for the certification figures.

The incidence of asbestosis among insulators is unknown. The total number of men employed is certainly less than the 18,700 in the asbestos products industry and probably is about 5000. In this part of the country rather fewer than 500 men are

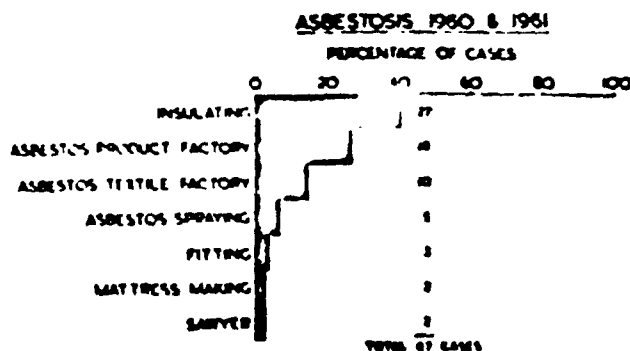


FIG. 3. Cases of asbestosis certified during 1960 and 1961 grouped according to occupation.

engaged on this work, but I have personally seen 18 cases of asbestosis amongst them. It therefore appears that asbestosis is relatively common in insulators.

In view of this it would seem justifiable to take a closer look at the insulating industry to see if steps can be taken to reduce the prevalence of asbestosis.

THE WORK OF AN INSULATOR

Insulators work in ships, power stations, factories and buildings, where they are required to lag boilers and pipes. The material most commonly used for this is a plaster consisting of 85 per cent magnesia bonded with 15 per cent of amosite asbestos. It is used in the form of pipe-sections, slabs, and powder. Valves and flanges are covered with asbestos mattresses (containing the same powder) and occasionally pure asbestos sections (bonded with sodium silicate) may be used. The lagger fastens slabs or preformed sections round the boiler or pipe with wire, then applies a coat of wet plaster by hand, and finishes off with a coat of waterproof cement. On board ship, where temperatures are higher, the pipe sections are finished off with a covering of asbestos cloth made from chrysotile asbestos.

The installation of a new heating system in a local hospital gave us the chance to watch the work in progress and to make a few measurements of dust concentration. The workers say that the mixing of powdered magnesia/asbestos plaster with water, and the stripping off of old lagging, are the dustiest processes. Mixing is done in a bucket, dust bin, or large box, depending on the amount required, and is illustrated in Fig. 4. The mixing process takes 5-15 min and is repeated every 2-4 hr. Usually the mixing is all done by one person, often by the apprentice who has just started work. Knowing that asbestosis is slow to develop one might suggest that it would be better if this dusty work were to be done by the elderly worker nearing retirement.

Often the work has to be done in confined spaces, such as between bulkheads in a ship, or in long ducts beneath the floor of a factory. Figure 5 shows a "lagger"

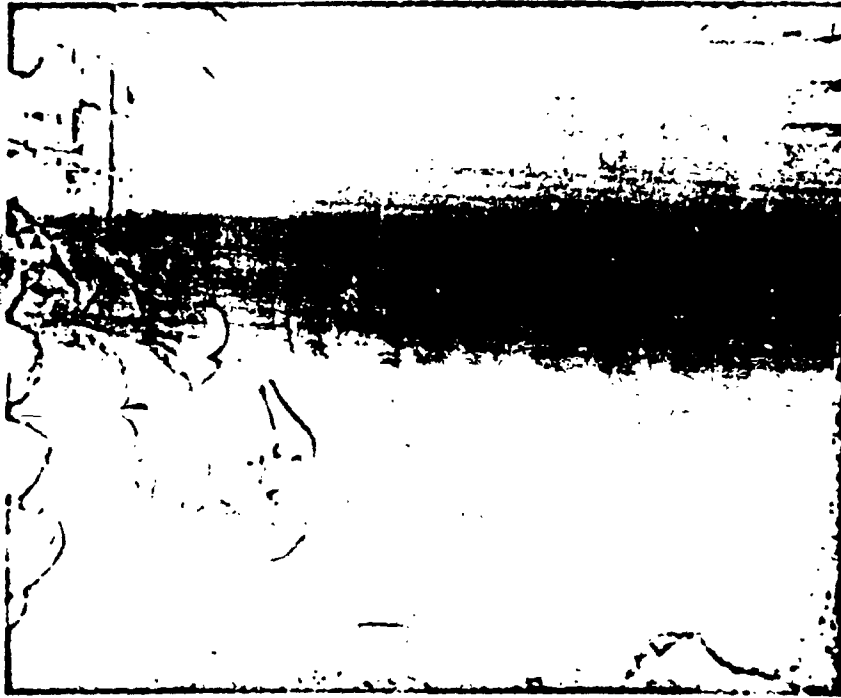


FIG. 4. Air sampling by thermal precipitator during the mixing of magnesia asbestos plaster with water.

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medical examination by a member of The Pneumoconiosis Medical Panel, and empowers the medical specialist to prohibit the employee from further work in contact with asbestos. In practice this scheme means examination at intervals not exceeding two years and the examination frequently, but not invariably, includes a chest radiograph. Men working in the insulating industry are not included within this scheme and, as things are at present arranged, the lagger is denied the benefits of environmental precautions at work and of periodic medical examination, but entitled to "compensation" when crippled by asbestosis. It would be unfair to the industry to omit to mention that some of the larger firms have a voluntary scheme for periodic medical examination which is entirely free to their employees. Such schemes are costly and tend to be ineffective because they are voluntary, and because the employees fear examination may lead to suspension from work. Now that so many of the certified cases of the disease are occurring in ladders it would appear that steps should be taken to make such examinations a legal obligation by broadening the Medical Arrangements Scheme*; and the preventive ideals of the Asbestos Industry Regulations should also be applied to lagging.

PREVENTION OF ASBESTOSIS IN LAGGERS

The principles upon which the prevention of dust disease depends, namely automation, total enclosure, exhaust ventilation, respirators, are well known (McLAUGHLIN, 1953) but few of them can be applied to the job of the lagger. Substitution of an innocuous material for asbestos would seem to offer the best solution, and, in fact, artificial rock wool and glass wool are being used in increasing amounts. Both are believed to be harmless to the lungs but our experience of them is not very extensive. Unless it is bonded with resin, glass wool is unpleasant to work with. The workers complain, not only of itching skin, but also of a "burning" sore throat and this suggests that bronchitis might be a late sequela.

Total enclosure is obviously impossible but the mixing of magnesia/asbestos plaster with water might be done centrally, and automatically. The objections to this are financial and based on the costs of distribution of ready-mixed plaster but since the plaster does not set (as cement does) and can be used days or weeks after mixing, delivery of a plaster slurry in bulk might be considered. Another possibility that might be considered is to make the plaster sticky before delivery, perhaps by the addition of a water soluble oil.

Exhaust ventilation is clearly impracticable but forced ventilation of confined spaces on board ship should lower the dust concentration. Personal protection of the worker, in the form of some sort of dust mask is theoretically feasible but it must be remembered that lagging, like all plastering, is heavy work for which the worker needs to breathe ten or fifteen-thousand litres of air in a shift. The dust filter must be capable of passing such a large volume of air without becoming clogged. Face-masks are often found to be too hot for comfort and this objection carries particular weight in the case of the lagger. His working conditions are hot and humid as the pipes on which he is putting wet plaster are usually hot at the time (in order to dry

* After presentation of this paper the author was informed that the T.U.C. and the Ministries concerned are at present engaged in discussions on how to achieve this.

cut the plaster) and we have recorded corrected effective temperatures as high as 89 °F (Table 2).

TABLE 2. RANGE OF CORRECTED EFFECTIVE TEMPERATURES EXPERIENCED DURING ASBESTOS LAGGING IN A HOSPITAL

Working area	Corrected effective temperature °F normal scale
Large boiler plant: 12 ft x 15 ft x 16 ft	65-89
Large duct 10 ft x 10 ft	67-79
Medium duct 7 ft x 6 ft	63-84
Small duct 5 ft x 5 ft	84

It therefore seems unlikely that the dust masks will be worn conscientiously and the provision of dust masks is probably not an effective preventive measure. More radical steps will have to be taken.

CONCLUSIONS

It is clear that asbestosis is occurring among workers in the insulating industry and it is felt that steps should be taken to deal with this problem. Closer supervision of the ladders' health, perhaps by extension of the Silicosis and Asbestosis (Medical Arrangements) Scheme, should help to focus the attention of employers on this hazard, and to define the extent of the problem. Prevention of the disease probably depends on substitution of an innocuous material for asbestos but something might be achieved by reducing the handling of dry asbestos plaster. The provision of respirators is less likely to be effective, because the hot and humid conditions in which a ladder works add materially to the discomforts of wearing a respirator.

The object of this communication is to reveal the hazards of the ladder's work to a wider audience in the hope that something further will be done, both by the Industry and by the Health Services, to prevent him developing a crippling and fatal disease.

Acknowledgements—I am grateful to Mrs. D. WEIGHTMAN for drawing Figs. 1, 2, 3 and 6, and to the Department of Photography in the University of Durham for Figs. 4 and 5. And I am especially indebted to DR. J. WATKINS-PITCHFORD for the help given by his Department and for the information used in the construction of Figs. 1 and 3.

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DISCUSSION

DR. F. P. GALL (Siebe Gorman, Chessington, Surrey): What about the possibility and advantages of wearing a dust mask during the mixing of the asbestos plaster which takes about 10 minutes of time?

DR. D. E. HICKIN (Occupational Hygiene Service, Slough): I would like to thank you for your interesting paper. During recent years our Dr. Challen looked into this problem and we also concluded that mixing and stripping were the hazardous processes though we did not have the opportunity to do dust counts. We did notice however, that even when mixing was done in the open air it is often done in the bottom of an old oil drum and this means that the man doing the mixing is bending down with his head in the dust cloud inside the drum. The point that I would like to make is that we were shown figures of dust counts of up to 300 particles/cm³. It is my own view that the M.A.C. of 175 applies to particulate dust and that a figure of 30 particles/cm³ would be more realistic for fibrous particles. This would make the ladders work more dangerous than Dr. Leathart has implied.

MR. S. G. LUXON (H.M. Factory Inspectorate): There is a legal requirement to take all steps to protect these people and I hope to enlighten you when I speak because this may help.

DR. J. S. MCINTOCK, N.C.B.: Normally when an industrial pulmonary disease is accompanied by a typical radiological appearance, this X-ray picture is taken as a major criterion in diagnosis and I think Dr. Leathart implied that this was the case in asbestosis. But I understand that asbestosis may now be diagnosed for compensation before any X-ray changes can be seen. Would Dr. Leathart care to comment? Is it possible that this may be one of the reasons for the recent rise in "certification" figures?

MR. F. B. CROSSLEY (I.M.I. (Kynoch) Ltd.): Would the particle size range 5-50 μ used in Dr. Leathart's experiments be within the respirable range?

PROF. R. E. LANE (Manchester University): Since it has been implied that mixing asbestos plaster with water is the cause of asbestosis it is very important to know how much time is spent on this process. Can I ask how long the mixing process takes and how frequently it is done during the shift?

DR. SMITHERS (Cape Asbestos Company): Dr. Leathart's excellent paper reminds me that we have had previous occasions on which to differ a little. Firstly I am not as gloomy as he on the question of incidence. The regulations promulgated in 1931 came into force in 1933. The high incidence of the later thirties reflected the working conditions of the pre-1933 years. There was, as he has shown, a drop in the number of new cases in the late forties and early fifties which reflected the improved conditions after 1933. The rise in incidence in the latter half of the fifties reflects the war-time working conditions when more people were employed on increased production at a time when black-out and security were more vital than ventilation.

A study of the number of years exposure before diagnosis in a series of cases gives a true picture of progress. In the series reported by the late Dr. Wyers, up to 1949 the average exposure of his cases was 10.5 years. In a series of cases from the same factory, fully investigated at Hammer-smith Hospital in 1959, the average exposure was 14.5 years. Thus in 10 years the improvement was already evident. (Table 1.)

On the question of incidence of malignancy our experience differs from Dr. Leathart's. The picture in this country is also very different from that seen in South Africa, where I recently saw some most interesting material. There one saw many more cases of pleural involvement and pleural malignancy. The South Africans find our incidence of what we may call parenchymatous malignancy quite astounding. Similarly the South African experience of peritoneal malignancy differs from ours. This will be the subject of a subsequent communication.

These variations in man's reactions to asbestos stimulated me to consider the differences between various types of asbestos. There are three main types of commercially important asbestos. These are chrysotile, crocidolite and amosite, the two latter are grouped as "amphibole" asbestos. They differ in geological formation, in chemical composition, in crystalline form, in fibre size and I suspect, in their effect on man. I have tabulated a few of their differences and the result is interesting.

TABLE 1. COMPARISON OF CHARACTERISTICS OF THREE FORMS OF ASBESTOS

Geological Formation	<i>Crocidolite</i>		<i>Amosite</i>		<i>Chrysotile</i>
	Metamorphosed Sedimentary Ironstones	Metamorphosed Sedimentary Ironstones	Metamorphosed Sedimentary Ironstones	Metamorphosed Sedimentary Ironstones	Magnesium rich igneous rocks
Chemical Formula	$\text{Na}_2\text{Fe}^{++}(\text{Fe}^{++}\text{Mg})\text{Si}_2\text{O}_7(\text{OH})_2$	$\text{Fe}^{++}\text{Mg}_2\text{Si}_2\text{O}_7(\text{OH})_2$	$\text{Mg}_3\text{Si}_2\text{O}_7(\text{OH})_2$		
Colour	Blue	Blue	Ash-Grey	White	White
Intrinsic Iron	40 per cent	40 per cent	40 per cent	NIL	NIL
Extrinsic Iron	Present	Present	Present	Present	Present
Oxidation	Slow at 25 °C, Rapid at 400 °C	Slow at 25 °C, Rapid at 400 °C	Negligible at 25 °C, slow above 400 °C	Negligible at 25 °C, slow above 400 °C	Non-Oxidizable
Mobility of Proton in (OH) Groups	High	Low	Low	Low	Not Known
Crystal Structure	Double Silicate Chains	Double Silicate Chains	Double Silicate Chains	Double Silicate Chains	Curved Silicate Sheet
Unit Cell Volume	900 (Å) ³	900 (Å) ³	905 (Å) ³	905 (Å) ³	720 (Å) ³
Specific Gravity	3.37	3.37	3.45	3.45	2.55
Fibre Diameter (Mean Minima)	0.15 μ	0.15 μ	0.5 μ	0.5 μ	0.03 μ
Tensile Strength	400,000 lb/in ²	400,000 lb/in ²	100,000 lb/in ²	100,000 lb/in ²	300,000 lb/in ²
3-4 Benzpyrene	Present + +	Present + +	Present +	Present +	Absent
Artificial Absorption of 3-4 Benzpyrene	Poor	Poor	Poor	Poor	Good
Elution of Hydrocarbons by Serum or Body Fluids	12 per cent in 3 days	12 per cent in 3 days	12 per cent in 3 days	12 per cent in 3 days	None Present
Reported Pleural Mesothelioma	90 + cases (Wagner, South Africa)	90 + cases (Wagner, South Africa)	None	None	2 (Cartier, Canada) 13 Possible (McCaughy, Ulster)

Consideration of the variations between types of asbestos may well lead to a better understanding of the nature of its effects on man. I feel it is no longer enough to report that a man has been exposed to asbestos. We must determine with which type or mixture of types of asbestos the man has worked.

PROF. R. C. BROWN: Can anybody in the audience tell us why asbestos is carcinogenic?

DR. G. L. LEATHART: First of all it was suggested that a dust mask might be worn by the men engaged on the mixing process. I think this is a perfectly practical suggestion and I see no reason why a mask should not be worn at this time. The time taken by the mixing process depends on the amount being mixed but on average it takes 5-15 min and is repeated at intervals of 2-4 hours.

I thank Dr. Hickish for his interest and with regard to the M.A.C. for asbestos dust I do not feel qualified to comment. I am quite prepared to accept his figure of 30 particles/cm³ as a more reasonable M.A.C. for fibrous particles, and I agree that this means that the dust hazard is greater than I had previously implied.

I am grateful to Mr. Luxon for his contribution on the part that can be played by the Factory Inspectorate.

I agree that in the past the diagnosis of asbestosis has depended mainly on the X-ray but that the diagnosis is now occasionally being made before radiological abnormalities are apparent. Diagnosis depends on the demonstration of persistent crepitations at the lung bases, and pulmonary function tests may also be some help. We do not know to what extent this may have influenced the certification figures.

I was asked whether the particle size range that we measured is outside the respirable range. I am sure that it is not. I agree that a spherical particle of 50 μ diameter would drop like a stone but the fibrous particles of asbestos are very light and float in the air much more readily. You will remember that fibres recovered from the human lung have measured up to 100 μ in length.

I am very grateful to Dr. Smither for coming here and making such an interesting contribution to the discussion. He said that he had crossed swords with me before but this is not strictly true. Our relationship has always been most friendly and I hope that it always will be so. Today, I am grateful to him for the information he has given us and for the interest he has shown in my paper.

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