INVESTIGATION INTO THE HAZARDOUS USE OF ASBESTOS. NORTHERN IRELAND 1960 - 76

by

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INTRODUCTION

THE opportunity for medical research arises from the fortuitous coincidence of the right patients, the right investigators, time and nowadays money. The last often seems the main barrier to successful research. But perhaps because the financial demands of the work I am going to describe were not great at first, money did not hold it up.

During the 1950’s Dr. Elliott McCaughey† collected in Belfast some 15 cases of what appeared to be primary pleural tumours and described them as a pathological entity “Primary Diffuse Mesothelioma of the Pleura”. His findings in the form of a thesis and subsequently published (McCaughey, 1958) were not universally accepted. Primary localised pleural tumours had been described in the past (Stout, A. P., 1952) but both clinically and pathologically these were not the same as those described by McCaughey. Isolated cases had been described elsewhere, but the Belfast cases had been collected over a number of years and derived from a population of only 1½ million. It was considered a rare tumour, but not so rare in Belfast as elsewhere.

The curious discovery of 16 cases within one year in a Kimberley sanatorium forced Wagner and his colleagues (Wagner, Sleggs and Marchand, 1960), to look for an environmental cause. Asbestos bodies were seen in the lungs and further investigation revealed that these patients had been exposed to blue asbestos from the mines, tips and dirt roads along the crocidolite bearing hills of the N.W. Cape Province.

When he came to the British Isles, Wagner visited the only person who had reported a large series of diffuse mesotheliomas to discuss the histological features and to look for asbestos bodies in the underlying lung. The findings with respect to the peculiar cellular pattern of the tumour and the presence of asbestos bodies in the lung proved to be the same in the Belfast patients as in the South Africans.

By this time Professor Wade†† and the author had admitted to their care as acute medical emergencies two patients with diffuse pleural malignancy. One was diagnosed as mesothelioma after three surgical biopsies and the second was diagnosed only at autopsy. They were impressed by two things, the peculiar

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clinical pattern of the illness and the fact that two cases of so ‘rare’ a disease should present to the same medical unit within a year.

Professor Pemberton, the Professor of Social and Preventive Medicine, was at the time carrying out a study of byssinosis in the linen mills. But Professor Wade and the author had had experience of research into environmental disease and were therefore able to undertake the work which was obviously needed. They carried this out with the active help and encouragement of Professor Pemberton and the Medical Inspector of Factories (Dr. Swain) and in collaboration with Dr. Elliot McCaughey.

PRELIMINARY STUDIES

There were three questions to be answered:
(1) Were the Belfast tumors related to asbestos exposure?
(2) Where was the asbestos exposure occurring?
(3) What was the extent of the hazard created by this exposure?

Question 1: Relation of tumours to asbestos exposure

Before embarking on an expensive long-term study we needed an answer to the first question. By December 1965 occupational histories had been obtained from 42 patients identified with mesothelioma of the pleura either from them or their relatives. Occupational histories were also obtained from age and sex matched controls (Elmes and Wade, 1965). These histories were obtained by trained social workers (Mrs. Dudgeon and Mrs. Simpson) and the results showed a statistically significant association between occupational exposure to asbestos and mesothelioma. Three-quarters of the mesothelioma patients had been so exposed whereas only one-quarter of the controls had been exposed (Table 1).

| Patients with Exposure | Heavy | Light | None | Total |
|------------------------|-------|-------|------|-------|
| Mesothelioma           | 8     | 24    | 10   | 42    |
| Matched controls       | 2     | 7     | 33   | 42    |
| Asbestos bodies        | 2     | 12    | 6    | 20    |
| No asbestos bodies     | 1     | 4     | 15   | 20    |

Combining the light and heavy groups the differences are significant: Between mesothelioma and controls $P > 0.001$; between asbestos bodies and none $0.02 > P > 0.01$.

In a series of related studies certain other facts emerged. Firstly the presence of asbestos bodies in the lungs were usually but not always attributable to a history of occupational exposure (see Table 1). Secondly 21 (88%) our of 24 patients with mesothelioma had asbestos bodies in the lung whereas they were present in
only 6 (25%) of matched controls (Table 2). Thirdly that asbestos bodies were present in the lungs of about a quarter of the elderly males dying in Belfast at that time. There was no relationship between the finding of asbestos bodies and

| Patients                        | Number examined | Per cent with bodies | Significance of difference |
|---------------------------------|-----------------|----------------------|---------------------------|
| With mesothelioma               | [...]           | [...]                |                           |
| Matched controls                | 24              | 88                   | \( X^2 = 19.05 \)        |
| With carcinoma of bronchus      | [...]           | [...]                |                           |
| Most recent 50 of these         | 100             | 20                   |                           |
| Matched controls                | 50              | 18                   | \( X^2 = 0.94 \)         |
| Without cancer of lung          | [...]           | [...]                |                           |
| Aged 50 to 59                   | 100             | 14                   | \( X^2 = 5.18 \)         |
| Aged 60 to 69                   | 100             | 27                   | \( 0.05 > P > 0.02 \)    |

the presence of lung cancer. The work indicated that there was no special risk of lung cancer but perhaps a quarter of the male population were at risk of developing mesotheliomas. With development of more precise methods of measuring the asbestos content of the lung, it now seems that both these initial conclusions were misleading. But these initial studies justified further work on question (1) as well as the need to answer questions (2) and (3).

**Question 2: Where was the asbestos exposure occurring?**

When the mesothelioma cases were plotted on a map there was no indication that they lived in any particular area as there had been in the South African cases. This supported the concept that the exposure was occupational. When 62 cases were available, analysis showed that 45 had worked in the shipyard and 17 had never worked in the shipyard (see Tables 3 and 4). Ten of the shipyard workers and two of the others worked with insulating material. The relatively large number of plumbers and boilermakers was thought to be due to the exposure to asbestos which they described vividly. The general impression was that for many years all workers inside a ship were exposed from time to time to the dust produced during the application or removal of insulation. The men responsible for the insulation work were employed by one or other of a group of contractors, most of whom were financially dependent upon the asbestos industry in England. Because these employers acknowledged the hazards of asbestos in England they realised the importance of establishing safer working conditions in Northern Ireland, where (unlike England, Wales and Scotland) asbestos regulations had not existed. So that it was with the active help of the employers, the Medical Inspectors of Factories (Drs: Swain, Paisley and Hood), the insulation
Table 3
Mesotheliomas in Shipyard Workers
45 of a group of 62 cases

| INSULATORS:— |          |          |          |          |      |
|--------------|----------|----------|----------|----------|------|
| Full-time    | ...      | ...      | ...      | ...      | 5)   |
| Transient    | ...      | ...      | ...      | ...      | 5)   |

| OTHER OCCUPATIONS (Helpers and Labourers included):— |          |          |          |          |      |
|-----------------------------------------------------|----------|----------|----------|----------|------|
| Plumbers, Pipefitters and Boiler Makers             | ...      | ...      | ...      | ...      | 17   |
| Engine Fitters                                     | ...      | ...      | ...      | ...      | 4    |
| Platers, Riveters and Welders                      | ...      | ...      | ...      | ...      | 4    |
| Electricians                                        | ...      | ...      | ...      | ...      | 8    |
| Other                                               | ...      | ...      | ...      | ...      | 2    |

Table 4
Mesotheliomas in Non-Shipyard Workers
17 of a group of 62 cases

| INSULATORS:— |          |          |          |      |
|--------------|----------|----------|----------|------|
| Heating Engineers and Boilermen                     | ...      | ...      | 2       |
| Builders’ Labourers and Joiners                      | ...      | ...      | 6       |
| Dockers                                              | ...      | ...      | 1       |

| NO KNOWN EXPOSURE:— |          |          |          |      |
|---------------------|----------|----------|----------|------|
| Linen Workers (Women) | ...      | ...      | 2       |
| Sawyers and Timber Workers                             | ...      | ...      | 2       |
| Baker                                                          | ...      | ...      | 1       |
| Postman                                                        | ...      | ...      | 1       |

contractors and the trades unions that we were able to track down the sources of asbestos exposure and identify the most exposed population.

There was a small factory filling asbestos cloth quilts with fibrous asbestos employing up to 20 women at a time which had opened during the war to supply the shipyard. No other long-term factory exposure was important at that time. The follow-up of these women has not been completed. The factory has since been closed and reopened but no longer handles asbestos material.

The only other groups continuously exposed to asbestos dust were the insulation workers and the men responsible for cutting and fitting marinite partitioning in the ships. These latter were shipyard employees and it has not been possible to study them in detail. The insulation workers all belonged to one branch of the Transport and General Workers Union. Although this working group had belonged to this union branch for many years none had survived to the retiring age to draw on their own pensions fund. Mrs. Simpson, who became responsible
for contacting all these men and bringing them for follow-up, found that they were aware of the risks of their trade and had modified their way of life accordingly. Up until the hazards of asbestos received wide publicity it was not uncommon for several generations within one family to follow this trade, and as we subsequently discovered it was not uncommon for several of the men within one family to die prematurely leaving the women dependent on the remaining men. As a group they earned relatively high wages and spent the money on enjoying their shortened lives.

**Question 3: The extent of the hazard created by exposure**

Helped by the Pneumoconiosis Unit of the Medical Research Council (Dr. J. C. Gilson, Dr. J. C. Wagner and Dr. P. D. Oldham) and the London School of Hygiene (Dr. M. Newhouse and Dr. Thomson) formal research studies were set up to determine the health hazards for the insulation workers.

(i) **Living Workers.** Dr. J. Langlands and Dr. W. Wallace, both then working in the Department of Therapeutics, carried out two studies. One (Wallace and Langlands, 1971) was a comparison of the clinical, physiological and radiological findings in 50 randomly selected insulation workers, with 50 employees of the Belfast Corporation matched for age, height and smoking habits. The elaborate measurements and tests carried out on this group were part of an international attempt to discover the best method of detecting and measuring the extent of asbestos induced disease. Although this comparison revealed that the insulation workers showed some physiological evidence of asbestosis, these tests were no more sensitive than good chest x-rays. Nine out of the 50 men showed definite radiological evidence of asbestosis and 11 others showed changes probably due to asbestos. These workers were more likely to have a productive cough, basal rales and clubbing of the fingers and on physiological testing they were more likely to show a restrictive lung lesion with some impairment of gas transfer. They showed no more evidence of airways obstruction than the controls.

The second study (Langlands, Wallace and Simpson, 1971) was the application of these tests to all the men then working as insulators (lagger) and achieved 93 per cent co-operation which indicates the high level of effort put into the work and the good co-operation from the employers and men. Evidence of asbestosis and pleural lesions increased with the age and duration of exposure. Radiological evidence of parenchymal lung damage, basal rales, finger clubbing, a restrictive lung lesion and impairment of gas transfer appeared to be the signs of serious lung disease. Evidence of this type of damage increased from 13 per cent of men with only 10 years exposure to 85 per cent of those with more than 30 years. Pleural changes alone on x-rays were not usually associated with evidence of serious lung damage, but did not appear until many years after first exposure. High levels of cigarette smoking were associated with increased physiological evidence of airways and parenchymal lung disease in workers with normal and abnormal radiographs but there was no evidence that cigarette smoking increased the incidence or severity of radiological change.
Although these studies revealed ample evidence of occupational lung disease they did not reveal why the men were not surviving to the retiring age. The 80 per cent of the men over 60 who had radiological evidence of asbestosis did not often have a life threatening degree of impairment of lung function.

(ii) Dead Workers. The reason became apparent when the third study was analysed (Elmes and Simpson, 1971). In order to get the full picture of the hazard it was necessary to find out how many men had had to give up work and why. 168 men who were working in 1940 were identified and as far as is known this represented the entire population of insulation workers in Northern Ireland. By 1966 five were untraced, but 98 had died when age adjusted predictions based on mortality amongst Northern Ireland males predicted 37 deaths. Most of the unpredicted deaths were due to cancer of the respiratory tract, especially primary lung cancer. The two diseases then thought to be caused by asbestos exposure were less important. Mesothelioma caused only a third as many deaths as lung cancer. Asbestosis appeared to contribute to between 9 and 21 of the 61 unpredicted deaths. This study of mortality has been extended for another 9 years and one must suppose that the full extent of the hazard to this group of men has now been revealed (Elmes and Simpson, 1977). Unlike most prospective morbidity and mortality studies this group of men showed no initial advantage compared with the general population but the excessive mortality did not become statistically significant until the third five year follow-up period (1950-55, Figure 1). This

Fig. 1. SURVIVAL OF INSULATION WORKERS
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excess of deaths continued until 1965. Those still surviving (who were youngest amongst the original workers in 1940), now have nearly the same annual mor-

tality rate as predicted (Figure 2). Nevertheless these hardy survivors still show a relative excess of deaths due to cancer (Figure 3). During the last 10 years there

![Bar graph showing observed and expected deaths from 1940 to 1970.](image)

**Fig. 2. DEATHS FROM ALL CAUSES**
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have been no deaths due to asbestosis nor have there been any excess due to gastro-intestinal cancer. There were eight deaths due to gastro-intestinal cancer between 1950 and 1959 when only 0.2 were expected. Mesotheliomas have accounted for about one-third of the unexpected deaths due to cancer since 1950 and bronchial cancer the rest. Even before 1965 the amount of asbestos dust to which these men were exposed had started to fall because of the replacement of dry powder material by preformed slabs. Since 1965 there has been a substitution of glass and
rock wool material for asbestos and limpet spraying has almost completely dis-
appeared. Although there have been changes in the pattern of deaths amongst
insulation workers there is no evidence from this series that changes in work
environment have had any effect on the total death rate yet.

MESOTHELIOMAS

The spur which has led to these epidemiological studies was the high incidence
of pleural mesothelioma. Although this tumour was not the main cause of un-
predicted deaths in insulation workers, its frequency in other workers with less
occupational exposure to asbestos is alarming because apparently trivial exposure
may lead to a tumour 40 years later. With the help of the physicians, surgeons
and pathologists in Northern Ireland it has been possible to investigate large
numbers of patients with this disease during the last 15 years. The Northern
Ireland cases were used as the test group for a much larger study of proven cases
dying in the United Kingdom between 1960 and 1970 (Elmes and Simpson, 1976).
The clinical picture which emerged is of a tumour which most frequently arises
in the parietal pleura but can arise in the peritoneum. It occurs at a wide age
range from about 30 to over 80 years and in this country the majority (perhaps
95 per cent) have had some occupational exposure. The remainder may constitute
the natural incidence of this disease which would occur if no asbestos was used.
In Northern Ireland one such case appears to occur every 3-5 years. Most of the
12 to 15 cases we see here every year have had exposure to asbestos in the
shipyard.

Mesotheliomas usually produce rather vague symptoms at first, a dull ache or
 heaviness in one side of the chest, breathlessness of gradual onset or a dry cough.
These symptoms are common enough in the elderly and few patients are referred
to the hospital until they have had the symptoms for 4-5 months. At this stage
the clinical picture may be typical but it is difficult to prove the diagnosis.
Although other signs of asbestos disease (basal rales, pleural calcification, club-
bing of the fingers and radiological or physiological evidence of asbestosis) are
uncommon, a history of exposure is very important. In about half the cases there
is a serous effusion, 30 per cent have a bloody effusion and the remainder have a
solid lesion. Only in the serous efusions is it possible to make a diagnosis by
cytology. Even open biopsy may be unsuccessful in all three types unless a
pleurectomy is attempted. The prognosis from the time of referral to death
averages about a year but ranges from a few weeks to five or more years. In
retrospect it is possible to say that the tumours with a predominantly epithelial
cell pattern tend to survive for nearly 18 months, whereas those with a mainly
spindle celled sarcoma pattern survive for only 8 months.

During the latter part of the illness pain due to infiltration of the chest wall
and spinal column and breathlessness due to the accumulation of fluid and the
immobilisation of one lung dominate the clinical picture. The pain responds
poorly to radiotherapy, root section and cytotoxic drugs. It is better relieved

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by aspirin than by powerful analgesics, whose addictive properties lead to problems in patients with a relatively long survival.

Death is usually associated with compression of vital structures in the mediastinum, the oesophagus or major blood vessels. It may also result from spread of tumour to the opposite pleura or peritoneum. Pulmonary embolus is often a terminal event. Distant metastases are found at autopsy in nearly half the patients but seldom contribute to the clinical illness.

This detailed clinical picture which has been built up is the result of careful observations by many observers. Dr. C. F. Stanford has for instance been investigating the contribution of damage to the sympathetic chain by tumour to the subjective and objective changes observed in the skin of these patients.

CONTINUING RESEARCH

There are several important unanswered questions. The simple solution to the problem would seem to be the prohibition of the use of asbestos in any form. However, it is an essential component in certain fire preventing situations where it probably saves more lives than it kills. It is also a very cheap and efficient reinforcement for sheet cement materials and for brake linings. Although substitutes have been devised they are very much more expensive and we have no guarantee that they will be appreciably safer than asbestos. A large programme of research is going on elsewhere to settle these points.

Meanwhile we must make the continued use of asbestos as safe as possible. Continuous monitoring of exposed workers will determine whether the new measures have been effective. This applies to asbestos factory workers and insulators and especially to maintenance and demolition workers where old insulation has to be removed. Dr. Jean Langlands is doing this work in Northern Ireland and she has the unenviable job, with Mrs. Simpson's help, of determining whether the abnormalities she is detecting in workers are due to old exposure (before the regulations) or to new.

This sort of surveillance is effective as far as asbestosis is concerned. In so far as the elimination of asbestosis nearly eliminates the carcinoma risk, it will also prevent the second fatal illness produced by asbestos. The mean interval between first exposure and death in this province is 42 years and exposure need only last a few months. From the introduction of the current regulations until the incidence of mesothelioma falls to half its peak level (which has not yet been reached), will be about 40 years or the year 2010. This is dependent on crocidolite being the main cause of occupational mesothelioma. More work is needed and will depend in part on the sophisticated electron probe analysis of the lungs of patients dying of mesothelioma. Considerable amounts of material have been collected here for this purpose with the painstaking help of many pathologists and my assistants (Dr. D. P. Bell, Miss Wheeler, Miss Stevenson and Marie Gordon).
CONCLUSION

At a critical time for the prevention of increasing illness and death from asbestos exposure this province provided the material and the people for important research. The work carried out here was done synchronously with work in other parts of the British Isles, in the USA and South Africa and also on the continent of Europe. This work was co-ordinated at a series of international meetings and helped by funds from the World Health Organisation and various funds for research in cancer. The work in Northern Ireland was a significant contribution to the whole in spite of the relatively small scale on which it could be carried out. Its main contribution was to stimulate others to carry out large scale studies to confirm our findings. The main drawback of the Northern Ireland work was that there was no information on either total dose levels or the contributions of the different types of asbestos. The main advantage of the work here was the willingness of people to pool their information and to collaborate. I am sorry I cannot make out a list of all their names because there were many hundreds and they came from many different walks of life.

REFERENCES

ELMES, P. C. and SIMPSON, MARION, J. C., (1971). Insulation workers in Belfast. 3. Mortality 1940-66. British Journal of Industrial Medicine, 28, 226.

ELMES, P. C. and SIMPSON, M. J. C. (1976). Clinical aspects of mesothelioma. Quarterly Journal of Medicine, 45, 427.

ELMES, P. C. and SIMPSON, M. J. C., (1977). Belfast insulation workers. A further study of mortality due to asbestos exposure (1940-75). British Journal of Industrial Medicine. In press.

ELMES, P. C. and Wade, O. L., (1965). Relationship between exposure to asbestos and pleural malignancy in Belfast. Annals New York Academy Science, 132, 549.

LANGLANDS, J. H. M., WALLACE, W. F. M., SIMPSON, M. J. C., (1971). Insulation workers in Belfast 2. Mortality in men still at work. British Journal of Industrial Medicine, 28, 317.

MCCAUGHEY, W. T. E. (1958). Primary tumours of the pleura. Journal of Pathology and Bacteriology, 76, 517.

STOUT, A. P. (1952). Tumours of the pleura. Harlem Hospital Bulletin, 5, 54.

WAGNER, J. C., SLEEGGS, C. A. and MARCHAND, P., (1960). Diffuse pleural mesothelioma and asbestos exposure in North West Cape Province. British Journal of Industrial Medicine, 17, 260.

WALLACE, W. F. M. and LANGLANDS, J. H. M. (1971). Insulation workers in Belfast. 1. Comparison of a random sample with a control population. British Journal of Industrial Medicine, 28, 211.