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much for more antibiotic agents, but for more appropriate use of those we already have. To this end, since this paper was submitted, the Board of Management of the Royal Melbourne Hospital has approved an educational campaign, using consumer advertising techniques, to further improve the use of antibiotic agents in hospital practice (see pages 246-247).

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Original Articles

Compensable asbestos-related disease in New South Wales

Robert Barnes

ABSTRACT: Deaths in New South Wales from asbestosis or asbestos-related disease are analysed and reasons for the unusual distribution of the deaths are discussed.

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THE HAZARD of pulmonary fibrosis in asbestos workers in Great Britain was demonstrated by Merewether and Price in 1930.[1] In 1949, Merewether reported a more ominous association between cancer of the lung and asbestos exposure.[2] Six years later, Doll reported that lung cancer was the cause of death of 54% of workers who were suffering from asbestosis.[3]

In 1960, Wagner described the association of crocidolite (one of the amphiboles) with mesothelioma.[4] The evidence from South Africa has been consistent with the theory that malignant mesotheliomas have been confined to the crocidolite mines and crocidolite-containing asbestos products in that country. No convincing evidence has yet been produced to show that white asbestos (or chrysotile) can cause malignant mesotheliomas, and anthophyllite, which has been mined and used in Finland in manufacturing processes with relatively high environmental dust levels,[5] has not been reported to be associated with malignant mesothelioma.

Amosite (or brown asbestos), which is also an amphibole, was almost certainly the cause of mesotheliomas reported in 1981 in factory workers who were using a mixture of chrysotile and amosite in asbestos board manufacture.[6]

In Australia, the chrysotile form of asbestos has been mainly used in asbestos-products manufacture, but crocidolite (or blue asbestos) has been preferred for such work as boiler and steam pipe lagging, limpet spraying for fire protection, and in certain processes in which acid resistance is required. Crocidolite was either imported from South Africa or obtained from Australia's only blue asbestos mine at Wittenoom, in Western Australia.

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TABLE 1: Type of work performed and causes of death

Disease	Number of deaths									
	Asbestos products	Lagger and sprayer	Power house	Boilermaker	Carpenter	Welder	Miner	Wharf labourer	Other	Total
Asbestosis	13	10	1	3	—	—	1	—	1	29
Bronchogenic carcinoma	34	6	11	—	—	—	—	5	11	67
Mesothelioma	22	30	3	6	9	5	2	4	20	101
Total	69	46	15	9	9	5	3	9	32	197

The NHMRC has advised the prohibition of any mining, importation and use of crocidolite in Australia,[1] but it is, unfortunately, still present in lagging which covers old boilers and steam pipes, and in many older buildings in which it was used as a spray coat for fireproofing.

Two other possible causes of malignant mesothelioma are erionite, a fibrous zeolite, which is present in the local rocks and soil used for building walls in Anatolia, Turkey,[2] and tremolite, which is used as a whitewash in another area of that country.[3] Both the reports cited describe an almost epidemic form of malignant mesothelioma in Turkish villages in these areas.

Methods and results

This is a retrospective study of 197 workers who have been recognized by the Workers' Compensation (Dust Diseases) Board of New South Wales as having died from asbestosis or an asbestos-associated disease during the years between February, 1968 (when asbestosis became recognized by the Board in N.S.W. as compensable) and December 31, 1983. Of these 197 deaths, 29 have been from asbestosis, 67 from bronchogenic carcinoma, and 101 from malignant mesothelioma of the pleura or peritoneum.

The types of work performed by these workers are shown in Table 1; the majority of the workers were employed in the asbestos-products industry. Asbestos laggers and sprayers were the next most commonly affected, followed by powerhouse workers, boilermakers, welders and carpenters.

Deaths from asbestosis, where a fairly long and heavy exposure would be expected, occurred mostly in workers in the asbestos products and manufacturing industry. Deaths from mesothelioma and, to a lesser extent, bronchogenic carcinoma (in which the exposure, although expected to be heavy, may not necessarily be over a lengthy period) were scattered over a much broader range of industries. Some of the exposures were for relatively short periods, but were probably quite heavy. For example, during World War II, asbestos was wrapped around welding rods to make them burn more slowly. Examination of such rods, which were found amongst old stock at Vickers-Cockatoo Dockyards, showed that they were wrapped in crocidolite thread. One of these rods was then used experimentally (with all due precautions), and the air levels of asbestos fibre were many times greater than the threshold-limit value (TLV) for chrysotile (2mg/m³ of air) and over 100 times greater than the TLV for crocidolite (0.1mg/m³ of air).

Clinical signs and symptoms

Breathlessness was the most common presenting symptom in workers with asbestosis (80%), whereas pain (56%) was the most common initial symptom in workers with malignant mesotheliomas, and cough (62%) in those with bronchogenic carcinoma.

The most frequent findings on clinical examination were basal crepitations in persons with asbestosis, and pleural effusions in those with a malignant tumour.

The diagnosis of asbestosis was made after consideration of several factors, including the evidence of diffuse interstitial fibrosis on a chest X-ray film, fine inspiratory crepitations at the lung bases on auscultation, changes in the FEV/FVC ratio of a restrictive nature, and changes in the results of other pulmonary function tests, such as gas transfer factor (DLCO) and lung compliance (tested by means of a body plethysmograph).

If there was sufficient doubt in the diagnosis (for example, a suggestive chest X-ray film, but doubtful occupational exposure), the Dust Diseases Board delayed recognition pending further evidence, such as a high asbestos fibre count in material obtained by a transbronchial resection. This is a relatively minor procedure, in contrast to the more invasive process of an open lung biopsy.

The recognition for compensation purposes of asbestos-induced bronchogenic carcinoma is also bound by certain rules. There must be evidence in the lung fields (other than the presence of a tumour) of asbestos-induced fibrosis, or the presence of a greater quantity of asbestos fibres in lung tissue (obtained by transbronchial or trephine biopsy) than the accepted level for an urban dweller.

The presence of pleural thickening or pleural plaques alone is not sufficient evidence for a diagnosis of asbestosis, but would be accepted as evidence of asbestos exposure in the recognition of asbestos-induced bronchogenic carcinoma.

Survival and latent period

The average ages at death, the latent period from the first (known) asbestos exposure to the development of the disease

TABLE 2: Average age at death, latent period, and mean length of asbestos exposure

Disease	Age at death (years)	Exposure (years)	Latent period (years)
Asbestosis	63 (46-81)	19 (2-35)	31 (17-59)
Bronchogenic carcinoma	60 (43-80)	21 (6-50)	30 (7-51)
Mesothelioma	60 (41-80)	21 (0.5-52)	33 (11-55)

or malignancy, and the mean exposure periods are shown in Table 2.

Although the lengths of industrial exposure to asbestos dust are similar if only the averages of the three groups are considered, the range is much greater and the minimal exposure time much less in the case of malignant mesothelioma. The shortest exposure to asbestos was in one worker who developed a malignant mesothelioma 26 years after handling hessian bags of crocidolite for one month. Other workers, such as those employed in powerhouses and boiler-rooms, had environmental exposures while working near laggers applying and removing asbestos. Another worker's only remembered exposure was using asbestos gloves in a laboratory for a few months 30 years previously.

The average survival times from diagnosis for the three conditions are shown in Table 3. Both bronchogenic carcinoma and malignant mesothelioma would be expected to be diagnosed reasonably promptly, whereas a worker with asbestosis may have fairly advanced disease before the condition is recognized. Therefore, the survival from the development of asbestosis to death could be expected to be longer than the average of 7.3 years shown in Table 3.

TABLE 3: Average survival times

Disease	Average age at diagnosis (years)	Average age at death (years)	Life expectancy (years)
Asbestosis	55	63	7.3
Bronchogenic carcinoma	58.2	60	1.8
Mesothelioma	59.3	60	0.7

In the Figure, the percentage survival after the diagnosis of mesothelioma is shown as a histogram. It is interesting to note that two persons with mesothelioma are still alive after much longer periods than indicated here and, therefore, are not included in this series. One worker, in whom diagnosis was made by open lung biopsy in 1972, is reasonably well, although in constant low-grade chest pain; the second worker, in whom diagnosis was made by transbronchial lung biopsy in 1978, is still working and feels quite well. The first worker had a pleurodesis which seems to have controlled the build-up of pleural effusions, but the second has received no active chemotherapy or radiotherapy.

TABLE 4: Smoking and incidence of asbestosis and malignant disease

Smoking habit (packs/year)	Incidence		
	Asbestosis	Bronchogenic carcinoma	Mesothelioma
Heavy (>20)	45%	71%	50%
Moderate (>10 <20)	23%	8.5%	4.5%
Light (<10)	14%	3%	3.5%
Nil	18%	17.5%	43%

Role of smoking

Other studies have demonstrated that smoking has no bearing on the incidence of malignant mesothelioma, but greatly increases the likelihood of bronchogenic carcinoma.[9,11] It seems reasonable to hypothesize that smokers would inhale more asbestos dust than non-smokers when exposed to similarly poor environmental conditions.

In Table 4, the possible correlation between smoking and the development of disease is demonstrated.

As can be seen, 43% of mesothelioma sufferers were non-smokers, compared with 18% of those with asbestosis and 17.5% of those with bronchogenic carcinoma. This is significant ($P < 0.02$) and strengthens the argument for banning smoking wherever asbestos materials are made or handled, in an endeavour to reduce both the incidence of bronchogenic carcinoma and (possibly) the incidence of asbestosis.

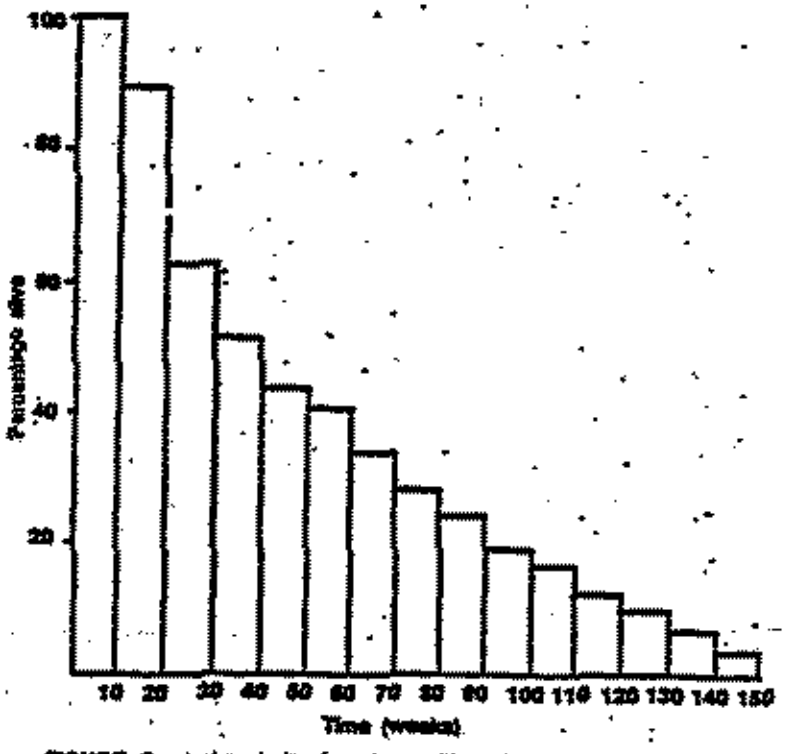

FIGURE: Survival periods of workers with malignant mesothelioma.

Discussion

Asbestosis has been recognized as a hazard of asbestos exposure since Merewether's report in 1930.[1] New risks of such exposure, such as cancer of the lung, pleura and peritoneum and, possibly, of the larnyx, bowel and bladder, have been reported, and legislation controlling the threshold limit values of asbestos dust should reduce the incidence of these diseases in the future. Unfortunately, bad industrial practices in the past have left a legacy of illness and death which can be expected to continue for another three decades or longer. This is particularly true in the case of malignant mesothelioma, in which the average latent period is 43 years.[9]

The amphibole fibres of asbestos (those of crocidolite rather than of amosite) are more strongly implicated than chrysotile fibres, and there is compelling evidence to suggest that these fibres have the greatest carcinogenic effect on cells whose diameter is less than 0.25 μm and whose length is greater than 9 μm.[12] Another reason for this difference in effects is that the uncoated amphiboles are thought to remain in the tissue unchanged for life, whereas the magnesium in

chrysotile fibres leaches out,[14] and the fibres eventually *disintegrate and* are absorbed.

Electron microscopy has enabled the identification of fibres in lung sections. This has shown that the bulk of asbestos-dust fibres in the lungs vary in length from 5 μm to 25 μm, and that the chrysotile fibres are the shortest, those of amosite are the longest and thickest, and those of crocidolite are intermediate. Studies have also shown that the longer fibres (that is, those of the amphiboles, amosite and crocidolite) are likely to undergo the process of coating, and the presence of large numbers of coated fibres reflects an exposure to amphibole asbestos, while uncoated fibres are most likely those of chrysotile.

The cases of mesothelioma in this series have all been verified by the Australian Mesothelioma Surveillance Programme, and, since early in 1981, an attempt has been made to obtain lung tissue for asbestos-fibre counts in all cases of mesothelioma and bronchogenic carcinoma. In fact, unless there is other evidence of asbestosis (diffuse interstitial fibrosis, basal crepitations, and so on), no award is given to a person with bronchogenic carcinoma if the fibre count is not greater than the levels in the lungs of normal urban dwellers (that is, a fibre count not greater than 350 000 fibres per gram of dry lung issue). Twelve workers in this series received compensation on the basis of greater than normal asbestos fibre counts in lung tissue.

The 29 deaths from asbestosis do not include the deaths of all those workers who have been recognized as suffering from, and who were compensated for, pulmonary asbestosis. Each worker with asbestosis who dies has his or her clinical records, X-ray films, results of lung function tests, death certificate and results of the post-mortem examination (if possible) reviewed by a panel of three specialists in occupational chest diseases. For example, death from cor pulmonale as a result of extensive lung fibrosis would be accepted as due to asbestosis, but death from a coronary occlusion, left-sided heart failure, or cancer of any organ, excluding the lungs or bronchi, would be classified as death not due to asbestosis. Deaths of a further 43 workers who suffered from compensable asbestosis were certified as "death due to a cause other than asbestosis".

Accepting these criteria, the number of deaths from mesothelioma in this series (101 deaths) is almost double that from bronchogenic carcinoma (67 deaths), and more than three times that from asbestosis (29 deaths). The greater incidence of malignant mesothelioma, compared with that of bronchogenic carcinoma, in New South Wales is inconsistent with all overseas reports, and is difficult to explain. Of course, the reporting of malignant mesothelioma has very much improved since the setting-up of the mesothelioma surveillance programme about a year ago, and the appointment of a field officer to investigate each reported case. In the case of bronchogenic carcinoma, the association with smoking is so well recognized, both by the medical profession and by the general public, that the relationship to asbestos exposure in the workplace might, perhaps, be overlooked in many patients.

The true incidence of mesothelioma is difficult to estimate for many reasons. Many cases which were diagnosed on clinical grounds were not confirmed by histological methods, or by a post-mortem examination after the death of the sufferer. Death certificates are notoriously unreliable and tend to underestimate the true incidence. The rate for Glasgow for the years 1970-1980 was 16.6/1 000 000 population per year.[16] This relatively high rate probably reflects the prevalence of shipbuilding in the area, as other rates for the United Kingdom in earlier reports were as low as 2.57/1 000 000.[17]

The rates for Australia will no doubt be more accurately assessed with the setting up of the Mesothelioma Register, and with the drive by the various State thoracic societies to be on the lookout for, and to notify, each case.

We can expect the number of cases of asbestosis and bronchogenic carcinoma (from asbestos exposure, but possibly not from smoking) to decrease after the introduction of the Asbestos Regulations in 1978, which control the permissible dust levels in industry. Unfortunately, with the average latent period for the development of mesothelioma being 42 years, many asbestos-induced mesotheliomas can be expected to continue to occur for at least another three to four decades.

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