THE MEDICAL JOURNAL OF AUSTRALIA, 1983(September 3); 2(5): 221-224.

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AL JOURNAL OF AUSTRALIA September 3, 1983 v.2, no 5, pp.221-224

nore antibiotic agents, but for more appropriate e we already have. To this end, since this paper tted, the Board of Management of the Royal Hospital has approved an educational campaign, umer advertising techniques, to further improve antibiotic agents in hospital practice (see pages

gements

nowledge the contribution of the following people: Professor John hairman of the working parties responsible for the first two editions of elines, without whom the concept would not have become a reality; Antibiotic Guidelines subcommittee, who have ensured that each ooklet is up-to-date and authoritative; Dr Rod Taylor and Dr John ical-year medical students, conducted our first antibiotic audit; and Mr Director of Pharmacy, and his team of ward pharmacists and pharmacy t whom the subsequent audits would not have been possible.

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10 AVR 1984

l Articles

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pensable asbestos-related disease in New South es

PLAINTIFF'S EXHIBIT
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Robert Barnes

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

(Med J Aust 1983; 2: 221-224)

ARD of pulmonary fibrosis in asbestos workers in in was demonstrated by Merewether and Price in 1949, Merewether reported a more ominous between cancer of the lung and asbestos ex- x years later, Doll reported that lung cancer was of death of 54% of workers who were suffering tosis.[3]

, Wagner described the association of crocidolite amphiboles) with mesothelioma.[4] The evidence 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

TABLE 1: Type of work performed and causes of death

...se	Number of deaths									
	Asbestos products	Lagger and sprayer	Power house	Boilermaker	Carpenter	Welder	Miner	Wharf labourer	Other	Total
...osis	13	10	1	3	—	—	1	—	1	29
...hogenic ...inoma	34	8	11	—	—	—	—	8	11	67
...helioma	22	30	3	6	9	6	2	4	20	101
	68	48	15	9	9	6	3	9	32	197

NHMRC has advised the prohibition of any mining, ...ation and use of crocidolite in Australia,[1] but it is, ...rely, still present in lagging which covers old boilers ...am pipes, and in many older buildings in which it was ...s a spray coat for fireproofing.

...) other possible causes of malignant mesothelioma are ...e, a fibrous zeolite, which is present in the local rocks ...al used for building walls in Anatolia, Turkey,[2] and ...ite, which is used as a whitewash in another area of ...ountry.[3] Both the reports cited describe an almost ...ic form of malignant mesothelioma in Turkish ...s in these areas.

...ods and results

...s a retrospective study of 197 workers who have been ...nized by the Workers' Compensation (Dust Diseases) ... of New South Wales as having died from asbestosis or ...bestos-associated disease during the years between ...ary, 1968 (when asbestosis became recognized by the ... in N.S.W. as compensable), and December 31, 1983. ...ese 197 deaths, 29 have been from asbestosis, 67 from ...hogenic carcinoma, and 101 from malignant ...helioma of the pleura or peritoneum.

... types of work performed by these workers are shown ...ble 1; the majority of the workers were employed in the ...tos-products industry. Asbestos laggers and sprayers ... the next most commonly affected, followed by ...rhouse workers, boilermakers, welders and carpenters. ...aths from asbestosis, where a fairly long and heavy ex...e would be expected, occurred mostly in workers in the ...tos products and manufacturing industry. Deaths from ...thelioma and, to a lesser extent, bronchogenic car...ma (in which the exposure, although expected to be ...y, may not necessarily be over a lengthy period) were ...ered over a much broader range of industries. Some of ...xposures were for relatively short periods, but were pro...y quite heavy. For example, during World War II, ...tos was wrapped around welding rods to make them ... more slowly. Examination of such rods, which were ...d amongst old stock at Vickers-Cockatoo Dockyards, ...ed that they were wrapped in crocidolite thread. One of ... rods was then used experimentally (with all due precau...), and the air levels of asbestos fibre were many times ...ter than the threshold-limit value (TLV) for chrysotile ...g/m³ of air) and over 100 times greater than the TLV for ...idolite (0.1mg/m³ of air).

...ical signs and symptoms

... most common initial symptom in workers with malignant mesothelioma, 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	21	33

ancy, and the mean exposure periods are shown in

gh the lengths of industrial exposure to asbestos similar if only the averages of the three groups are d, the range is much greater and the minimal ex- times much less in the case of malignant iomas. The shortest exposure to asbestos was in one who developed a malignant mesothelioma 26 years dling hessian bags of crocidolite for one month. rkers, such as those employed in powerhouses and ms, had environmental exposures while working ers applying and removing asbestos. Another only remembered exposure was using asbestos a laboratory for a few months 30 years previously. erage survival times from diagnosis for the three s are shown in Table 3. Both bronchogenic car- nd malignant mesothelioma would be expected to sed reasonably promptly, whereas a worker with may have fairly advanced disease before the condi- ognized. Therefore, the survival from the develop- st[illegible]s to death could be expected to be longer verage of 7.3 years shown in Table 3.

TABLE 3: Average survival times

	Average age at diagnosis (years)	Average age at death (years)	Life expectancy (years)
	55	63	7.3
ic carcinoma	58.2	60	1.8
a	59.3	60	0.7

Figure, the percentage survival after the diagnosis elioma is shown as a histogram. It is interesting to two persons with mesothelioma are still alive after ger periods than indicated here and, therefore, are ded in this series. One worker, in whom diagnosis by open lung biopsy in 1972, is reasonably well, in constant low-grade chest pain; the second whom diagnosis was made by transbronchial lung 1978, is still working and feels quite well. The first d a pleurodesis which seems to have controlled the f pleural effusions, but the second has received no motherapy or radiotherapy.*

E 4: Smoking and incidence of asbestosis and malignant disease

	Incidence		
	Asbestosis	Bronchogenic carcinoma	Mesothelioma
	45%	71%	50%
>10 <20	23%	8.5%	4.5%
	14%	3%	3.5%
	[illegible]	[illegible]	[illegible]

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.

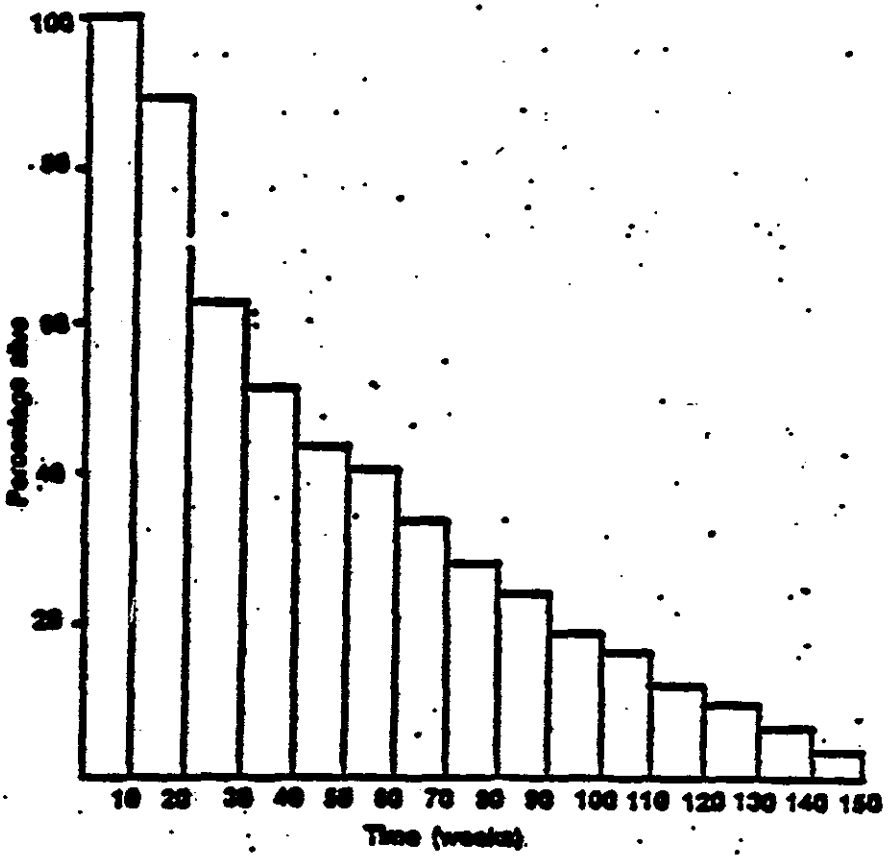

FIGURE: Survival periods of workers with malignant mesothelioma.

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.[12]

The amphibole fibres of asbestos (those of crocidolite rather than of amosite) are more strongly implicated than

e fibres leaches out,[4] and the fibres eventually ate and are absorbed.

on microscopy has enabled the identification of lung sections. This has shown that the bulk of dust fibres in the lungs vary in length from 5 μm to nd that the chrysotile fibres are the shortest, those ite are the longest and thickest, and those of te are intermediate. Studies have also shown that er fibres (that is, those of the amphiboles, amosite idolite) are likely to undergo the process of coating. presence of large numbers of coated fibres reflects an to amphibole asbestos, while uncoated fibres are ly those of chrysotile.

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

9 deaths from asbestosis do not include the deaths of workers who have been recognized as suffering d who were compensated for, pulmonary asbestosis. rker with asbestosis who dies has his or her clinical X-ray films, results of lung function tests, death cer- and results of the post-mortem examination (if possi- ewed by a panel of three specialists in occupational ases. For example, death from cor pulmonale as a f extensive lung fibrosis would be accepted as due to is, but death from a coronary occlusion, left-sided ilure, or cancer of any organ, excluding the lungs or , would be classified as death not due to asbestosis. of a further 43 workers who suffered from compen- bestosis were certified as "death due to a cause other bestosis".

pting these criteria, the number of deaths from lioma in this series (101 deaths) is almost double that onchogenic carcinoma (67 deaths), and more than nes that from asbestosis (29 deaths). The greater in- of malignant mesothelioma, compared with that of genic carcinoma, in New South Wales is inconsis- h all overseas reports, and is difficult to explain. Of the reporting of malignant mesothelioma has very mproved since the setting-up of the mesothelioma ance programme about a year ago, and the appoint- f a field officer to investigate each reported case. In e of bronchogenic carcinoma, the association with g is so well recognized, both by the medical profession the general public, that the relationship to asbestos re in the workplace might, perhaps, be overlooked in

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.

Acknowledgements

I am grateful to the Chairman of the Workers' Compensation (Dust Diseases) Board for permission to publish this article, and to Miss A. Leishow for preparation of the manuscript, tables, and graphs.

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