CRITERIA FOR DETERMINING WORK RELATED HEAVY ASBESTOS EXPOSURE

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INTRODUCTION

The objective of this paper is to derive from case records, guidelines for whicb occupations and operations constitute heavy asbestos exposure.

Schedule 3 of the Workers' Compensation and Assistance Act, Western Australia (1981) contains a list of specified industrial diseases for which Workers' Compensation may be obtained. One of the diseases listed in Schedule 3 is lung cancer associated with "any process entailing heavy exposure to asbestos dust." The term "heavy exposure" is not defined.

METHOD

All claimants for Workers' Compensation for pneumoconioses attend at the Perth Chest Clinic. Perth Chest Clinic records of Workers' Compensation claimants diagnosed by the Pneumoconiosis Medical Panel as silico-asbestosis or asbestosis were examined for Wittenoom Australian Blue Asbestos (ABA) workers for the period 1 July 1987 to 31 December 1987 (15 cases) and the DOSHWA Asbestosis Register was examined for "non ABA" workers for the period 1 January 1979 to 31 December 1987 (36 cases). Mining and milling of blue asbestos was located in the town of Wittenoom, which is in the North-West of Western Australia, from 1943–1966. "Non-ABA" cases were further divided into Waterside Workers (9 cases), Asbestos-Cement Products Manufacturing Workers (9 cases), Railway Workers (5 cases) and "Other" (13 cases).

All the cases on the Perth Chest Clinic records who successfully claimed Workers' Compensation for mesothelioma from 1 January 1979 until 31 December 1987 were examined (101 cases). All the cases on the Perth Chest Clinic records who successfully claimed Workers' Compensation for lung cancer from 1985 (when lung cancer became a specified industrial disease under the Act) until 31 December 1987 were examined (12 cases). For each of the above three disease categories, the resulting data were classified according to occupation and duration of exposure.

RESULTS

Asbestosis and Silico-asbestosis

From 1 January 1979 until 31 December 1987 (9 years) the DOSHWA Asbestosis Register contains the names of 154 people. Of these, 118 (77%) had worked for ABA in Wittenoom.

As it is generally accepted that having worked mining or milling crocidolite asbestos at ABA for even relatively short periods constitutes a history of heavy exposure, it was decided to look at only a small number of ABA workers, namely those who had successfully claimed Workers' Compensation for asbestosis or silico-asbestosis between 1 July 1987 and 31 December 1987 (15 cases). Of these 15 cases, occupational histories were available for 14, and, of these, only 6 (40%) had worked exclusively in one type of operation, the remaining 8 having worked in multiple operations. These 14 workers had been engaged in a total of 21 different operations at ABA. The mean duration of working at ABA was 43 months (range 3–96 months).

Information was obtained from the Perth Chest Clinic records for the 36 "non-ABA" workers registered on the DOSHWA Asbestosis Register from 1979 until 1987 inclusive.

Of the 9 Waterside Workers registered, 78% were involved exclusively in the one operation of loading/unloading bags of asbestos. The mean duration of employment for waterside workers was 235 months (range 84–384 months). Of the 9 Asbestos-Cement Products Manufacturing Workers, 4 (44%) of these had worked in a single operation. The nine workers had worked in a total of 19 different operations. The mean duration of employment for this group of workers was 231 months (range 28–416 months). Of the 5 Railway Workers, 4 (80%) had worked in only one type of operation. The mean duration of working for the Railways was 223 months (range 72–504 months). The number of cases of asbestosis and silico-asbestosis amongst the different occupational groups described above, together with the mean durations of employment, standard deviations and ranges are summarized in Table I.

Mesothelioma

All the cases of mesothelioma in the Perth Chest Clinic records from 1 January 1979 until 31 December 1987 were examined. There was a total of 101 cases of mesothelioma during this period. Of these, 9 cases were excluded because there was insufficient information regarding either the person's occupation or the duration of employment in a particular occupation. DOSHWA's Mesothelioma Register contains the names of a number of people who lived in Wittenoom but who did not

work in the mine or mill at ABA. There were eight such cases identified up until November 1985. The last group under consideration contained 16 workers. This group covered a wide variety of different occupations including carpenters, truck drivers and insulation workers. The mean duration of employment in this group was 176 months (range 1–420 months). The numbers of cases of mesothelioma amongst the different occupational groups described above, together with the mean durations of employment, standard deviations and ranges are summarized in Table I.

Lung Cancer

Of the 12 lung cancer patients who successfully claimed Workers' Compensation under the Act between July 1985 and 31 December 1987, 6 worked at ABA, 2 were Waterside Workers, 2 worked in the Railways, one for an Asbestos-Cement Products Manufacturer and one was an insulation worker. For the overall group of 12 lung cancer patients, the mean period of employment was 127 months (range 10–372 months). Of these 12 patients, all except for one (who gave a history of having been a lifelong non-smoker) had a history of moderate to heavy cigarette smoking over several years. Of the 11 smokers, it was possible in 10 of them to estimate the total cigarette consumption over their lifetime in terms of pack-years where one pack-year represents a person's smoking a packet of 20 cigarettes per day for a year (=7300 cigarettes per year). The mean lifetime cigarette consumption was 37.7 pack years (range 18–56 pack years). The numbers of cases of lung cancer in the other occupational groups are listed in Table I along with the corresponding means, standard deviations and ranges.

Lung cancers attributable to asbestos exposure should, strictly-speaking, be called bronchial carcinomas, as should the vast majority of lung cancers that are caused by other known agents.[1] All the common histological forms can occur (squamous carcinoma, small or oat-cell carcinoma and 3 adenocarcinoma). Amongst the 12 cases of lung cancer considered in this paper, there were 4 adenocarcinomas, 4 small cell carcinomas, 2 squamous cell carcinomas, one case who had two separate tumours (one a squamos cell carcinoma and one a small cell carcinoma) and one case in whom the histopathological diagnosis was not recorded.

DISCUSSION

A widely held belief is that the increased risk of lung cancer due to exposure to asbestos occurs only where asbestosis is already present.[2] This theory contends that exposures to asbestos which are insufficient to cause asbestosis are also insufficient to cause lung cancer. The alternative point of view,

Table I

Numbers of Cases of Asbestosis/Silico-Asbestosis, Mesothelioma and Lung Cancer Amongst Different Occupational Groups with Mean Durations of Employment, Standard Deviations and Ranges (in Months)

	Asbestosis or Silico-asbestosis		Mesothelioma		Lung Cancer	
A.B.A.	N	14	N	61	N	6
	MEAN	43	MEAN	22.0	MEAN	39.3
	S.D.	25.1	S.D.	26.1	S.D.	46.8
	RANGE	3-96	RANGE	1-132	RANGE	10-133
RAILWAYS	N	5	N	5	N	2
	MEAN	223	MEAN	185	MEAN	94.5
	S.D.	180	S.D.	139	S.D.	36.1
	RANGE	72-504	RANGE	72-420	RANGE	69-120
ASBESTOS CEMENT PRODUCTS MANUFACTURING	N	9	NIL		N	1
	MEAN	231			MEAN	372
	S.D.	159			S.D.	N/A
	RANGE	28-416			RANGE	N/A
WATERSIDE WORKERS	N	9	N	8	N	2
	MEAN	235	MEAN	166	MEAN	264
	S.D.	111	S.D.	141	S.D.	102
	RANGE	84-384	RANGE	24-396	RANGE	192-336
OTHER	N	13	N	16	N	1
	MEAN	150	MEAN	176	MEAN	195
	S.D.	148	S.D.	137	S.D.	N/A
	RANGE	24-456	RANGE	1-420	RANGE	N/A
			N	90	N	12
			MEAN	71.1	MEAN	127
			S.D.	107	S.D.	125
			RANGE	1-420	RANGE	10-372

which is also widely held, is that there is no demonstrated threshold level of exposure to asbestos below which there is no increased risk of lung cancer.[2]

Chase et al (1985)[3] have proposed, as its final determination, a "risk apportioned to asbestos" that is represented by a number between zero and one. This number reflects the strength of the evidence in support of each individual lung cancer being related to asbestos exposure. In 1984, Mowe et al[4] analysed mineral fibre concentrations in lung tissue by scanning electron microscopy in 73 males with malignant mesothelioma and in 36 controls who died of cardiovascular or cerebrovascular diseases. Their investigation showed apparent differences in the median lung fibre concentrations between occupational groups with different levels of asbestos exposure as judged from their occupational histories. Mowe and Gylseth[5] investigated 141 cases of malignant mesothelioma registered by the Cancer Registry of Norway 1970–79. Sixty-five of the cases were classified into four groups according to criteria of estimated probability of occupational asbestos exposure. These were definite, probable, possible and unlikely or unknown exposures.

In a survey undertaken by the Mines Department in 1966,[6] measurements were made of the concentrations of airborne respirable fibres of crocidolite greater than 5 microns in length in various workplaces at ABA in Wittenoom. A 0 to 10 scale of estimated fibre levels for both before and after September 1957 (when a less dusty mill started operating) applicable to all 87 job categories at ABA was developed using the judgement of an ex-superintendent of operations at Wittenoom (who had a detailed knowledge of all jobs on the site throughout the production period). Unfortunately, as far as could be determined, there are no records of similar information concerning airborne fibre concentrations pertaining to non-ABA work situations which would have prevailed in Western Australia at the time that contemporary Workers' Compensation claimants would have been occupationally exposed to asbestos. The occupations of the six ABA workers who developed asbestosis and who worked in a single type of job category were compared with their scores on the above 0–10 point scale. Because of the small numbers involved, it is difficult to draw any conclusions from this comparison although it is interesting to note that two workers whose fibre concentrations were considered to be relatively low (2 points) still had sufficient exposure to cause asbestosis after only three years of employment in each case.

Any approach which attempts to uncover a relationship between different occupations and heaviness of asbestos exposure cannot take into account variations in how various processes are performed. In other words, such an approach is unable to make allowances for the fact that one type of operation can result in different degrees of exposure according to the ways in which the work processes are performed. It would be simple and convenient if, for cases of asbestos-induced occupational lung disease, one could look at the durations of employment of the workers in various occupations and operations and assume that there is an inverse relationship between duration of occupational exposure to asbestos and the "heaviness" of exposure to asbestos entailed. If such an assumption were valid, one could then easily classify different occupations/operations according to the "heaviness" of exposure (e.g., heavy, moderate, mild, negligible, etc.). Unfortunately, there are a number of reasons why such an assumption would be invalid as well as a number of other difficulties with this approach. These include:

1. By utilizing Perth Chest Clinic records, one immediately introduces a form of selection bias, namely that patients with asbestos induced occupational lung disease who have not claimed Workers' Compensation are immediately excluded from further consideration.

2. There is a strong possibility of recall bias with respect to patients' recollections of their occupational histories.

3. The problem of multiple exposures refers to people who have been exposed to asbestos either in different occupations or who have performed different operations involving asbestos exposure while working in the same occupation.

4. The problem of intermittent exposure applies to the majority of cases where the information necessary to calculate an accurate equivalent continuous exposure was lacking.

5. The main problems encountered in recorded occupational histories were incomplete descriptions of jobs and incomplete information regarding the durations of exposure to asbestos.

CONCLUSION

A solution to this problem may be found in studies which compare the amounts of asbestos fibre measured in lung tissue for equivalent durations of asbestos exposure in different occupations. Until such time as the results of these types of studies become available, it will be necessary for medical panels charged with the responsibility of making judgements regarding "heaviness" of asbestos exposure to exercise clinical judgement and assess each case on its merits. Heaviness of exposure to asbestos can be thought of as the product of intensity and duration. This formula is not necessarily valid however because it does not separate the effects of the two variables.[7] In the absence of any better alternative, it seems reasonable to make use of this formula, provided it is recognized that it may be an over-simplification of the true state of affairs. In determining the intensity of asbestos exposure in the absence of direct dust exposure measurements, one has to rely on an accurate description of the job to obtain an indirect indication of intensity. This can be partially corrohorated by a knowledge on claimants who did similar jobs such as the subjective degree of dustiness of the working environment. The other parameter in the above formula is that of duration. In this regard the most important point to recognize is the distinction between duration of employment and duration of exposure. Apart from intensity and duration of exposure, the type of asbestos to which the claimant was exposed should be taken into account.

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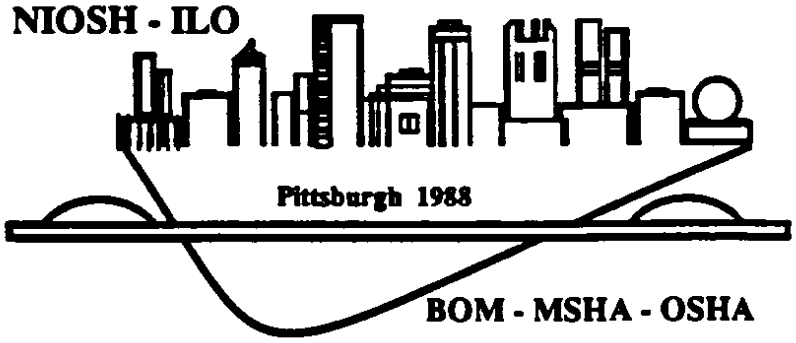

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