

Third wave of asbestos-related disease from secondary use of asbestos

A case report from industry

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Abstract An occupational health survey conducted in a workshop in which asbestos cement was used showed initial atmospheric asbestos levels ranging from 1,9 to 27,5 fibres per millilitre of air. Radiological changes suggestive of asbestos-related pleural disease were found in 2 workers (2,5%), while 3 (3,8%) had borderline features of asbestosis. The survey confirmed that uncontrolled and hazardous use of asbestos continues in industry despite public awareness of its dangers and the Asbestos Regulations of 1987.

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Yorld-wide, the historical pattern of the emergence of asbestos-related disease has been described as appearing in a series of waves.1 The first wave occurred during the mining and milling of asbestos in the early part of the century. This was followed by the intensified use of the substance during the 1930s, particularly in the shipbuilding, construction and manufacturing industries. The devastating effects of this second wave are still being observed today. More recently a third wave, resulting from the use of asbestos articles, e.g. sheeting, cement and friction products, has been described.

Since little is known about the extent of exposure to the third wave in South Africa, the results of an occupational health survey conducted in a workshop which used asbestos cement are reported. The survey was undertaken: (i) to assess the current asbestos hazard by measurement of asbestos fibre levels; and (ii) to identify workers with asbestos-related respiratory disease.

Subjects and methods

The workplace

The workplace, which employed 80 workers with current or previous occupational exposure to asbestos, was a subsidiary of a large local company. In one of the workshops asbestos cement sheets were cut into component parts for the production of an insulating device designed to prevent electrical shorts along an extensive power distribution and switching system. The sheets were cut by a manually operated machine, sanded into

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shape by hand and then assembled. While an extractor system was installed at both the cutting and sanding sections, it was only partially effective in the former and non-operational in the latter. There was poor enclosure of the workshop with free movement of air into adjacent areas. There was no vacuuming or wetting system to control asbestos fibres emitted into the atmosphere. Similarly, no personal dedusting mechanism was installed. Instead, a compressed air facility was used. The company had previously measured asbestos fibre levels, which were reportedly within normal limits.

Asbestos fibre measurements

In 1992 asbestos-in-air measurements were done by the National Centre for Occupational Health (NCOH) Occupational Hygiene Department using the standard method for the collection and counting of fibres.2 Asbestos levels were measured at an initial visit and 5 weeks later after a workplace clean-up. Five samples, 4 personal and 1 static, were taken initially. At the repeat visit 8 samples, 4 personal and 4 static, were taken. Analysis of the asbestos samples to determine the type of asbestos was performed using a scanning electron microscope (SEM).

Evaluation of workers

Full-size postero-anterior chest radiographs were taken on all 80 workers who currently or previously worked in the asbestos workshop. The radiographs were taken by an experienced radiographer using the NCOH mobile X-ray machine. A questionnaire was administered by two trained interviewers to elicit information such as age, race, sex, smoking history and length of asbestos exposure. Two experienced readers, blind to exposure details, independently read the chest radiographs into the 1980 International Labour Office (ILO) International Classification of Radiographs of Pneumoconioses. Where there was consensus between the two, the reading was accepted. Where there was disagreement, a third reader was called upon and the median opinion was accepted.

Results

Table I shows asbestos fibre levels in the workshop at the time of the initial NCOH inspection and after the subsequent clean-up. Initial measurements ranged from 1,9 fibres per millilitre of air to 27,5 fibres/ml. Repeat measurements dropped to a maximum of 1,7 fibres/ml. On both occasions the highest measurements were recorded in the section where asbestos sheets were sanded. Although a substantial drop in the fibre levels was observed after the clean-up, none of the personal sample measurements was below the action level for asbestos in industry of 0,5 fibres/ml.3 Fibres examined under the SEM had the characteristics of chrysotile.

TABLE I.

Asbestos-in-air measurements at workshop before and after workplace clean-up

Sample type	Job description	Asbestos levels (fibres/ml)
Before clean-up	4-14-12-1-1	
Static	N/A	2,2
Personal	Sanding asbestos sheets	27,5
Personal	Sweeping workshop	5,7
Personal	Assembling asbestos sheets	1,9
Personal	Drilling asbestos sheets	8,6
After clean-up		
Personal	Drilling asbestos sheets	0.6
Personal	Sanding asbestos sheets	1,7
Personal	Sweeping workshop floor	1,4
Personal	Sanding asbestos sheets	0,5
Static	N/A	0,6
Static	N/A	0,2
Static	N/A	0,2
Static	N/A	0,01

N/A = not applicable; static sample = sampler situated in workplace environment; personal sample = sampler attached to individual worker.

Of the 80 workers examined radiologically, 48 (60%) had more than 5 years' exposure to asbestos; of these, 32 (40%) had more than 10 years' exposure. The median exposure was 7 years and the interquartile range was 3 - 12 years. Fifty (62,5%) were current smokers and 4 were ex-smokers.

Table II presents radiological findings. Sixty-five (81,3%) of the 80 radiographs were normal and 12 (15%) were abnormal, while the remaining 3 (3,8%) could not be read owing to poor quality. Seven of the 12 abnormal radiographs displayed lesions that were not related to asbestos exposure, including fractured ribs, active tuberculosis and healed tuberculosis. Two workers (2,5%), with 4 and 11 years' asbestos exposure respectively had asbestos-related pleural disease but no parenchymal disease. This was confirmed on a high-resolution computed tomographic scan in both cases. The remaining 3 (3,8%) had evidence of equivocal parenchymal disease suggestive of asbestosis — all 3 had a significant smoking history and their respective asbestos exposure times were 1, 6 and 14 years.

TABLE II.

Radiological findings in 80 workers employed in a workshop using asbestos cement

Radiological finding	No.	%
Normal	65	81,3
Abnormal	12	15
Asbestos-related pleural disease	2	2,5
Parenchymal lesions 0/1	3	3,8
Other (non-occupational lung disease)	7	8,8
Inconclusive owing to poor quality	3	3,8

Discussion

The Asbestos Regulations of 1987³ set the exposure limit for asbestos at 1 fibre/ml. In addition, the regulations describe the action level for asbestos as the level at or above which certain prescribed preventive measures must be taken, and this is set at 0,5 fibres/ml. The factory inspectorate under the Department of Manpower is responsible for ensuring implementation of the regulations.

The occupational hygiene results of this survey demonstrate that despite the existence of these regulations the asbestos hazard in this workplace has gone unidentified and is consequently uncontrolled. An important consideration is that of quality control — the

workplace had previously relied solely on internal readings without objective assessment of the method employed and results obtained. Not one of the initial NCOH measurements was below the exposure limit for asbestos, the lowest level of 1,9 being close to double. The highest measurement, over 27 times the exposure limit, was recorded in the sanding area where the extraction system was broken. During the past 10 years the NCOH has performed asbestos-in-air measurements at 7 other secondary asbestos users. Of these, 5 had at least one measurement above 1 fibre/ml, while the highest levels for these workplaces ranged from 1,7 to 9 fibres/ml (NCOH Reports: 11/82, 8/91, 9/86, 26/88, 4/88, 10/91, 16/90). One possible explanation for the poor control of asbestos is the lenient penalties for contravening the regulations: contravenors may be fined no more than R1 000 and R5 per day for a continuous offence, or imprisonment for not more than 6 months and 1 day for each additional day of continued offence, amounting to no more than 90 days.3

While the repeat measurements showed a major improvement, 5 were still above the action level -2 of which were above the exposure limit for asbestos.

The only asbestos-related disease diagnosed from the radiographic survey was benign pleural disease in 2 workers (2,5%). This is considerably less than the prevalence of pleural disease reported by Irwig *et al.*⁴ among South Africans employed in amosite and crocidolite mines in the 1970s. The overall prevalence in the latter study was 7,6%; however, this varied with years of exposure. Workers with less than 1 year of exposure had a prevalence of 2,5%, while prevalences of 5,7% and 11% were found in workers exposed for 3 - 7 and 7 - 15 years respectively.

According to the ILO Classification, asbestosis is defined as profusion of parenchymal lesions of 1/0 or more. Asbestosis was not diagnosed in any of the 80 workers radiographed. However, 3 radiographs (3,8%) were considered to have profusion of 0/1. While the diagnosis of asbestosis is doubtful in the subject with only 1 year of asbestos exposure, the 0/1 category may be significant in the remaining 2 subjects. In a study by Sluis-Cremer and Hnizdo,⁵ in which radiographs of 1 454 asbestos miners and millers were reviewed, it was found that a 0/1 reading had a serious prognosis — 60% of subjects progressed to a reading of 1/0 or greater on a later radiograph.

The low prevalence of disease may be explained by the fact that more than half of the workers radiographed had less than 10 years of asbestos exposure, and only 6 were first exposed more than 20 years ago. Although high rates of radiological disease have been reported in short-exposure amosite workers, the diagnosis was made at least 20 years after first exposure. Another possible explanation for the low prevalence of disease is that sick workers may have left the workforce, with the healthy survivors remaining behind (a healthy-worker effect.

The high incidence of cigarette smoking (62,5%) is particularly worrying in view of the combined ill-effects of asbestos exposure and smoking. Only 4 workers with a smoking history had stopped smoking. Hammond *et al.*⁸ showed that among asbestos workers, ex-cigarette smokers have substantially lower death rates than persistent smokers.

In conclusion, the survey suggests that: (i) despite the 1987 Asbestos Regulations, asbestos levels in sectors of industry remain uncontrolled; (ii) the third wave of asbestos disease described internationally can be expected locally — even though the extent of its effect is not yet known; (iii) South African practitioners can expect to diagnose asbestos-related lung disease in the future and need to be familiar with procedures for compensating sufferers; (iv) smoking cessation programmes in asbestos-using workplaces deserve greater attention.

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