Progression of irregular opacities in asbestos miners

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ABSTRACT All white and mixed race men who were employed in South African asbestos mines and mills between 30 November 1970 and 30 November 1975 were studied. The men who had two radiographs available, the first taken some time between the above two dates and the latest available radiograph which had to be at least two years after the first one numbered 1454: 793 continued exposure after the first radiograph and 661 did not. The films were read by a panel of three readers. Data available included age, years of exposure to asbestos and other mining, intensity of exposure to asbestos and other dust, and smoking habit. Progression was expressed as the difference between the average readings of radiograph 2—radiograph 1 in minor categories per year of irregular opacities. Changes in pleural abnormality were also measured. No differences of progression in the profusion or change in size of the irregular opacities were found between the two groups or in the number of zones affected. "New attacks" appeared equally frequently between the two groups. No difference in the change in extent of any type of pleural change was seen. It appears that once a dose of asbestos sufficient to initiate the disease has been retained it is inexorably progressive.

The effect of removal from exposure to asbestos dust on progression of radiological change has been studied by several authors working on relatively small groups of exposed men who have usually been exposed to chrysotile or chrysotile with a small admixture of amphibole.¹⁻⁹ In one study the exposure was purely to Western Australian crocidolite.¹⁰

The present study was performed on men whose predominant exposure was to South African amphibole (crocidolite and amosite) asbestos. The objectives were:

- (1) to determine if the progression of irregular opacities was slowed down or stopped or reversed after exposure to asbestos ceased, particularly in the case of low profusion of irregular opacities; and
- (2) to determine whether irregular opacities appeared de novo after exposure ceased and if so how commonly.

Methods

STUDY SUBJECTS

All 2185 white and mixed race men employed by asbestos mines and mills on 30 November 1970 or who had entered employment (often by re-employment) up to 30 November 1975 were studied in a radiological cross sectional study.¹¹ The men from the cross

sectional study were chosen for this study if they had had a follow up radiograph taken at least two years after the radiograph used for the cross sectional study. The maximum follow up time was up to mid-1989. Workers with missing exposure data or no follow up radiograph were excluded. The group remaining for the progression study amounted to 1454 men. Of these, 793 had exposure after the first radiograph and 661 did not. Eighty five men had exposure to chrysotile only.

RADIOLOGICAL READINGS

A panel of three experienced radiograph readers read the films using the full 1980 ILO classification of Radiographs of the Pneumoconioses, the standard films and the side by side reading method. The films were marked to indicate the chronological order, the film used in the original cross sectional study (marked 1) and the latest available film (marked 2), but the dates of the films were covered so that the readers were blinded as to the duration of the period between the two films. The readers had no other information except that they knew the men had been exposed to asbestos. Nearly all the films were 35 × 43 cm. In some cases only 100 mm films were available for the dates required and then these were read. Only posteroanterior views were read.

The profusion of irregular opacities was read in minor categories and recorded so that each minor

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category was assigned a value between 0 and 10—that is, 0/0 became 0, 0/1 became 1, and, finally, 3/4 became 10 (0—was not used). The distribution of the opacities was graded by a score of one to six according to the number of zones affected. The size of the irregular opacities was graded with a numerical score from 0 to 3 as follows $ss = 1$, $st/ts = 2$, $ut/tu/tt = 3$. The score for pleural change was expressed as an area, calculated as extent times width where width was measurable. Width in terms of a, b, c was transformed into 1, 2, 3. For en face plaques, width was assumed to be one and the area therefore was the same as the extent. All pleural changes in right and left hemithoraces were then summed. No distinction was made between plaques and diffuse pleural thickening.

MINING EXPOSURES

Mining exposures to asbestos, gold, and other mining (for copper, tin, or antimony, for example) were coded in terms of years of exposure and also in terms of fibre-years in the case of asbestos. Years of exposure were calculated from shifts worked (280 shifts = 1 year). Fibre-years were computed by summing over all the occupations the man had worked in asbestos mines and the years spent in a particular occupation weighted by the estimated fibre level for that occupation at that particular mine at the relevant time. Fibre levels were available since 1945 for individual asbestos mines at different calendar periods and for different occupations. The groups are characterised by relatively short service, on average under conditions of moderately intense fibre exposure. A threshold limit value of 2 fibres/cc was imposed in 1984 only shortly before the termination of this project. It should be noted that the fibre exposures have been continuously computed for the subjects in this study since 1970 and updated every six months. In the earlier period if the total exposure of the man who left the mine amounted to less than 500 fibre-shifts the exposure was recorded as zero. Fibre-years worked before November 1970 were computed using information given by the man himself as to the mine he had worked at and the job he had held for different calendar periods. Asbestos, gold, and other mining exposures were calculated for two periods. The first period was from the start of mining exposure to the date of the first radiograph and the second was the period between the two radiographs.

SMOKING HABIT

Smoking habits were established at the time of first radiograph and categorised as never smoker (18.5%), current smoker 1–10 cigarettes/day (21.9%), ex-smoker > 2 years (3.4%), current smoker > 11 cigarettes/day (54.3%), unknown (1.9%). Cigars and pipe tobacco were converted to cigarettes as follows; small cigar 2, large cigar 5, ounce of tobacco 28. The subjects

with the unknown smoking values were excluded from the analysis in which smoking was considered.

STATISTICAL ANALYSIS

Three progression variables were calculated for the profusion of irregular opacities over the three readers:

- (a) average progression rate/y (APRIO)

$$\text{APRIO} = 1/3 \sum_{i=1}^3 (\text{reading } 2_i - \text{reading } 1_i) / \text{time (y)}$$

- (b) average progression (APIO)

$$\text{APIO} = 1/3 \sum_{i=1}^3 (\text{reading } 2_i - \text{reading } 1_i)$$

- (c) progression (yes/no) by one or more major category (PIO).

The average major category or profusion was calculated by averaging the profusion score over the three readers and then the categories were defined as follows: category 0/0 was assigned to average profusion score of < 0.5, 0/1 to score 0.5–< 1.5, 1/0–1/2 to score 1.5–< 4.5, 2/1–2/3 to score 4.5–< 7.5, and > 2/3 to score > 7.5. Then PIO was assigned the value 1 if the average category of profusion changed by one or more, major categories and 0 otherwise.

The relation between the variables (a) and (b) and exposure to asbestos was estimated by means of a stepwise multivariate linear regression analysis (SAS package, PROC STEPWISE and PROC REG). The potential confounding effects of smoking (SMOKE), gold mining and other mining exposure, age at first radiograph (AGE), the average initial reading for irregular opacities (AIO1), years of follow up (YFUP), and year of birth were considered in the analysis. The standardised means were calculated with analysis of covariance (SAS package, PROC GLM). The logistic regression model (PROC LOGIST) was applied for the analysis of variable (c)—that is, progression yes/no, using the same potential confounding variables as for the linear regression analysis. The central limit theorem which states that whenever “n” is moderately large then the mean is approximately normally distributed regardless of the distribution of the underlying variable was applied when comparing the two groups.

Results

COMPARISON OF THE EXPOSED AND UNEXPOSED MINERS

Table 1 describes the study population. There were 793 men who had been exposed after the first radiograph and 661 who had not. The two groups differed significantly in their history of exposure to asbestos mining up to the time of the first radiograph, the years

Table 1 *Comparison between miners exposed and miners not exposed to asbestos mining after the first radiological examination for exposure characteristics*

	<i>Exposure after first radiograph</i>				
	<i>Yes (n = 793)</i>		<i>No (n = 661)</i>		
<i>Characteristic</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>p</i>
Year of birth	37.7	10.7	37.6	10.8	0.82
Age at 1st radiograph	36.4	10.9	35.2	10.9	0.04
Year of 1st radiograph	74.1	1.2	72.8	1.5	0.0001
Year of 2nd radiograph	82.1	3.4	81.5	3.7	0.002
Year of follow up	8.0	3.4	8.7	3.8	0.0001
Mining exposure to 1st radiograph:					
Asbestos (y)	6.7	6.5	4.2	5.5	0.0001
Asbestos (f/y)	38.4	73.1	23.2	61.4	0.0001
Gold (y)	2.0	4.8	3.1	5.7	0.0001
Other (y)	1.1	3.2	1.3	3.1	0.40
Between 1st and 2nd radiograph:					
Asbestos (y)	4.5	3.3			
Asbestos (f/y)	6.2	9.8			
Gold (y)	0.3	1.1	1.0	2.4	0.0001
Other (y)	0.6	1.6	1.8	3.3	0.0001
Smoking category:					
	<i>No</i>	<i>%</i>	<i>No</i>	<i>%</i>	
Never smoked	148	18.7	121	18.3	
1-10 cig/day	176	22.2	143	21.6	
Ex-smoker	29	3.7	20	3.0	
≥11 cig/day	424	53.5	365	55.2	
Unknown	16	2.0	12	1.8	

of exposure were 6.7 (SD, 6.5) and 4.2 (SD, 5.5) and fibre-years were 38.4 (SD 73.1) and 23.2 (SD 61.4) for those exposed and not exposed after first radiograph, respectively. Several other characteristics showed statistically significant differences between the two groups (see table 1), but the differences were not considered important for this study. Smoking categories were virtually identical in the two groups. The mean years of follow up were similar for the exposed and the unexposed groups, 8.0 (SD, 3.4) and 8.7 (SD, 3.8) years respectively. The maximum follow up was 16.5 years.

COMPARISON OF THE RADIOGRAPH READERS

Table 2 compares the three readers for the number of cases in whom they considered progression of IO had taken place and for the degree of progression, expressed as major categories. Reader 2 read progression more frequently and to a greater extent than the other two readers, and reader 1 read apparent regression more commonly. The differences between the readers were considered to be acceptable. Table 3 shows the proportions of agreement on whether progression was present or not among the three readers. Agreement was considered satisfactory, the proportion was over 0.7 in all cases. This reasonable agreement, however, was in some measure due to agreement that progression was absent and there were somewhat larger disagreements when readers thought there was progression; when reader 1 thought there was progres-

sion (n 429) reader 2 agreed in 282 (66.7%) and reader 3 agreed in 277 of 436 (63.5%). When reader 2 thought there was progression reader 3 agreed only in 53.4%.

AVERAGE PROGRESSION RATE/YEAR OF IRREGULAR OPACITIES

Table 4 gives the factors found to be significantly related to the average progression rate of irregular opacities a year (APRIO). The 28 subjects with missing smoking data were excluded from this analysis. The results are presented for all the subjects and then separately for those exposed after the first radiograph and for those not so exposed. Age at first radiograph, average profusion of irregular opacities at first radiograph, smoking category, and years of exposure to asbestos to first radiograph were found to be significant predictors for both groups. The estimated regression coefficients did not statistically differ for the two groups as no significant interaction between any of them and the group (0, 1) variable was observed. It should be noted, however, that for the group not exposed after the first radiograph the average profusion at first radiograph was the most important predictor. Asbestos exposure after the first radiograph was a significant factor for those who continued exposure only in terms of fibre-years. Years of exposure in gold mines was not significantly related to progression rate and the exposure to other mining was of marginal significance in both the exposed and unexposed group.

AVERAGE PROGRESSION OF PROFUSION

The variables found to be significantly related to the average progression of profusion (APIO) were AGE, years of asbestos mining exposure to first radiograph (YAEXP1), YFUP, AI01, and SMOKE. The presence of asbestos exposure after the first radiograph was not found to be statistically significant. Table 5 shows the effect of years of follow up on APIO. The means for APIO were standardised for the effect of the other variables. The miners with 12 or more years of follow up had the highest value for progression. Even though the miners with the longest follow up were on average

Table 2 *Progression by minor categories for three readers*

<i>Progression (No of categories)</i>							
<i>Reader</i>		<i><0*</i>	<i>0</i>	<i>1</i>	<i>2</i>	<i>>3</i>	<i>Total</i>
1	No	43	972	180	137	122	1454
	%	3.0	66.9	12.4	9.4	8.4	100.1
2	No	22	859	239	138	169	1427†
	%	1.5	60.2	16.8	9.7	11.8	100.0
3	No	13	988	211	133	102	1447†
	%	0.9	68.3	14.6	9.2	7.1	100.1

* <0 indicates apparent regression.

† Reader 2 rejected 27 films as unreadable, reader 3 rejected seven.

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Table 3 Agreement on progression between the three readers

	Progressed	Yes	No	Total
<i>Reader 2</i>				
Reader 1	Yes	282	147	429
	No	264	734	998
	Total	546	881	1427*
Proportion in agreement = 1016/1427 = 0.71				
<i>Reader 3</i>				
Reader 1	Yes	277	159	436
	No	169	842	1011
	Total	446	1001	1447*
Proportion in agreement = 1119/1447 = 0.77				
<i>Reader 3</i>				
Reader 2	Yes	290	253	543
	No	148	729	877
	Total	438	982	1420*
Proportion in agreement = 1019/1420 = 0.72				

*Number of radiographs rejected as unreadable were 27 for reader 2 and seven for reader 3.

younger, the highest progressors among them were men who were over 35 (of 202 men with more than 12 years follow up in 90 aged under 30, irregular opacity change was 0.31, whereas in 65 aged 35–44 it was 1.78). The table confirms that the two groups did not differ in their pattern of progression of irregular opacities with respect to AGE, YFUP, and AIO1. The APRIO is also given for comparison.

PROGRESSION BY MAJOR CATEGORY OF PROFUSION IN IRREGULAR OPACITIES

Table 6 presents the mean radiological readings for irregular opacities in the second radiograph according to the readings in the first one for those who were and those who were not exposed after the first radiograph separately. A considerable amount of progression is evident in both groups, 80/648 men whose initial

reading was 0/0 (12.3%) in the exposed group and 58/549 (10.6%) in the unexposed group progressed to a reading of 1/0 or more—that is, new “attacks.” The difference was not significant. Of those read 0/1 in the first film, 53/88 (60%) progressed in those with exposure after first radiograph and 46/69 (67%) had so progressed in those without further exposure. The table does not indicate that the subjects exposed after the first radiograph progressed more than those not so exposed. The variables found to be significantly related to the presence of progression (PIO) were AGE, YAEXPI, YFUP, SMOKE, and AIO1 and again the two groups were not found to differ. In addition we also examined the factors related to progression (a) from category 0/0 to category 0/1, (b) from category 0/0 to >0/1, and (c) from category >0/1 to any higher category. For group (a) only AGE and YFUP were found to be significant. For groups (b) and (c) all the four variables mentioned above were statistically significant, but a pattern emerged in the strength of the association. For group (c) AGE and SMOKE were higher predictors than for group (b) and for group (b) YFUP was a stronger predictor.

Discussion

This is a relatively large radiological progression study on asbestos miners and millers with relatively few years of exposure and accumulated fibre-years (table 1); most of the subjects were exposed to amphibole. The reading of the films was by the side by side method to the full 1980 ILO classification. This choice was supported by most of those who have studied the problem of reading radiological progression in pneumoconioses and the relative merits of side by side and independent randomised reading.^{12–14} The readers

Table 4 Factors related to the progression rate/year of irregular opacities (IO), averaged over the three readers (APRIO)

Variable	β	SE	R ²	p
(a) For all 1425 subjects				
Age at 1st radiograph	0.003	0.0004	0.12	0.0001
AVIO profusion at 1st radiograph	0.050	0.007	0.04	0.0001
Smoking category	0.015	0.003	0.01	0.0001
Asbestos exposure to 1st radiograph (y)	0.004	0.001	0.01	0.0001
(b) For 777 subjects exposed to asbestos after 1st radiograph				
Age at 1st radiograph	0.003	0.001	0.09	0.0001
Av 10 profusion at 1st radiograph	0.035	0.009	0.02	0.001
Smoking category	0.017	0.005	0.01	0.001
Asbestos exposure to 1st radiograph (y)	0.003	0.001	0.01	0.002
Asbestos exposure after radiograph (f/y)	0.016	0.005	0.01	0.003
Other exposure (y)	–0.003	0.002	0.003	0.08
(b) For 648 subjects not exposed to asbestos after 1st radiograph				
Av 10 at 1st radiograph	0.068	0.01	0.18	0.0001
Age at 1st radiograph	0.003	0.001	0.06	0.0001
Asbestos exposure to 1st radiograph (y)	0.004	0.001	0.01	0.0001
Smoking category	0.013	0.004	0.01	0.004
Other exposure to 1st radiograph (y)	0.004	0.002	0.005	0.04

In five cases two readers considered a film unreadable. These were omitted from the analyses. Those with missing smoking data were also omitted from these analyses.

Table 5 *Change in profusion of irregular opacities (IO) adjusted for other effects*

Exposed						Change in IO from 2nd radiograph	
Years of follow up	No	Age at 1st radiograph	Exposure to 1st radiograph (y)	Exposure from 1st-2nd radiograph (f/y)	AVIO at 1st radiograph	APIO	APRIO
<3	86	36.1	4.9	3.6	0.22	0.36	0.14
3-5	110	36.1	6.4	4.7	0.29	0.46	0.11
5-7	114	36.1	7.2	5.5	0.30	0.60	0.10
7-9	72	39.5	8.1	4.6	0.26	0.97	0.11
9-12	308	36.9	7.3	7.9	0.28	0.77	0.07
≥12	65	31.9	3.9	7.6	0.12	0.96	0.09
	755						
Not exposed						Change in IO from 1st-2nd radiograph	
Years of follow up	No	Age at 1st radiograph	Exposure to 1st radiograph (y)	Exposure from 1st-2nd radiograph (f/y)	AVIO at 1st radiograph	APIO	APRIO
<3	66	36.7	4.2	0.0	0.23	0.29	0.12
3-5	82	34.0	3.4	0.0	0.13	0.48	0.11
5-7	73	37.2	5.5	0.0	0.39	0.54	0.10
7-9	71	37.4	5.7	0.0	0.45	0.77	0.10
9-12	208	35.5	3.9	0.0	0.25	0.78	0.08
≥12	137	32.9	3.6	0.0	0.15	1.00	0.08
	637						

AVIO = Average profusion of irregular opacities, APIO = average progression, APRIo = average progression rate/y.

Table 6 *Distribution of the mean radiological readings for irregular opacities (IO) on second radiograph according to the initial reading and according to the presence of asbestos exposure after the first radiograph*

	Second reading						
First reading	0/0	0/1	1/0-1/2	2/1-2/3	3/2-3/4	Total	%
(a) Subjects exposed to asbestos after 1st radiograph							
0/0	421	147	75	4	1	648	84.1
0/1	3	32	48	4	1	88	11.4
1/0-1/2	0	0	21	11	0	32	4.2
2/1-2/3	0	0	1	2	0	3	0.4
Total	424	179	145	21	2	771	100.0
%	55.0	23.2	18.8	2.7	0.3	100.0	100.0
(b) Subjects not exposed to asbestos after 1st radiograph							
0/0	377	114	50	8	0	549	84.6
0/1	0	23	40	4	2	69	10.6
1/0-1/2	0	0	18	8	4	30	4.6
2/1-2/3	0	0	0	0	1	1	0.2
Total	377	137	108	20	7	649	100.0
%	58.1	21.1	16.6	3.1	1.1	100.0	

were blinded as to the time lapse between two radiographs which varied from two to 16.5 years, knowledge of which might have influenced their readings.

Agreement among readers as to progression was probably as good as might be expected (tables 2 and 3) bearing in mind that progression was measured in minor categories and that few cases attained a profusion of category 2/1 or higher (table 4). Observer variation both for profusion and progression may be expected to be at its greatest in the lower categories. The progression in profusion of irregular opacities was analysed as average progression rate/year (APRIO),

average progression in profusion (APIO), and as progression by one or more average major category of profusion (PIO). The factors found to be related to all three variables were age at first radiograph, years of exposure to asbestos to first radiograph, average profusion of irregular opacities at first radiograph, tobacco smoking, and asbestos fibre-years after the second radiograph for the miners who continued exposure. No interaction effects were found to be statistically significant.

Age at first radiograph was found to be the strongest predictor of progression rate in the whole cohort and in the exposed group. The age effect was

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found to be much the same for the two groups ($b = 0.003$, $SE = 0.001$, table 4). The significant effect for age may be a true influence of age on progression (possibly due to immunological and endocrine changes with age). Nevertheless, in part at least one must consider the possibility that it is a reflection of non-specific radiological change with age (due in particular to smoking). The interaction between AGE and SMOKE was more significant than the individual effect on its own but when AGE and SMOKE were included in the model it ceased to be significant, nevertheless indicating the association between age and smoking.

There is no consensus, however, on the effect of age on progression. Souranta *et al* in a small study with a follow up of only three to four years found progression to be greater in younger men.⁵ Liddell *et al*, however, found age at first radiograph to be a leading variable for the progression rate of irregular opacities.⁶ Cookson *et al* in their study found age to have a weak effect¹⁰ as did Viallat *et al*.⁹ Becklake *et al* found no effect of age.²

Smoking habit was clearly related to progression rate in this study in the total group and in the subgroups, and the effect was found for all three readers when analysed individually.

Viallat *et al*⁹ and Souranta *et al*⁸ found that non-progressors smoked significantly less than progressors. Jones *et al*,⁵ Becklake *et al*,² and Rubino *et al*⁷ failed to find an effect of smoking whereas Liddell *et al* actually found that smoking was protective.⁶ Our group has previously shown that smoking habit has an important effect on the reading of irregular opacities whereas smoking was not found to be associated with asbestosis at necropsy.¹⁵

Average reading of irregular opacity profusion on first radiograph was strongly related to progression rate in all groups, and for the group unexposed after the first radiograph the average profusion was found to be the most important predictor of progression rate, even more important than age. This result is consistent with the notion that asbestosis is an inexorably progressive disease in most cases once it has started. Liddell *et al* found in their study that irregular opacities on the first film was a major predictor of rate of progression of irregular opacities.⁶ Souranta *et al* also found that with increasing category of irregular opacities progression increased from their 1976–7 reading to 1979–81 (in 85 patients).⁸

The years of exposure to asbestos to first radiograph was found to be a significant predictor of rate of progression for the combined and the individual groups. Fibre-years surprisingly was not significant once the years of exposure were entered into the regression model, except for the period between first and second radiographs in those who continued exposure. The reason why years of exposure is a far

stronger factor than fibre-years may be because estimations of fibre concentrations in the earlier years (before the introduction of the membrane filter in the early 1970s) measured by the konimeter and thermal precipitator were not accurate enough. Cumulative asbestos exposure (fibre-years) was found to be significantly related to rate of progression in some studies^{5,9,10} but another study found little evidence of a relation.⁶

Years of follow up were significantly related to progression in profusion of irregular opacities (table 5) and to the presence of progression by one or more major categories of profusion. Table 5 also shows that the two groups did not differ in the progression pattern with respect to the years of follow up, age, and exposure to first radiograph.

It should be emphasised that the maximum follow up period after first radiograph was only 16.5 years but there were over 608 miners with more than 10 years of follow up. Becklake *et al* notes the importance of "lag time"¹² and Cookson *et al* state that many of their subjects would not have been considered as progressing at 20 years of follow up whereas they had progressed at 30 years.¹⁰ In our study 65 men with further exposure and 137 without who had more than 12 years of follow up had the same degree of change between the two radiographs and the same rate of progression. They were also virtually of the same average age and exposure up to the first radiograph and profusion reading on first radiograph (table 5).

The distribution of the mean radiological readings for irregular opacities on the second radiograph for the various initial readings (table 6) for the two groups separately show two features of interest. New attacks (readings of 1/0 or higher on the second radiograph when the initial reading was 0/0) were fairly common—that is, 80/648 (12.3%) for those with exposure after first radiograph and 58/549 (10.6%) for those without such exposure. The difference is of no significance. A reading of 0/1 had a serious prognosis in this study: 53/88 (60%) progressed to a reading 1/0 or greater in the exposed group and 46/69 (66%) in the unexposed group.

The average change in the size of the irregular opacities with time was identical in the two groups. Two out of three readers found increases in 22.8% of the exposed and 22.4% of the unexposed. For increase in the number of zones affected the findings were 25.5% and 26.8% respectively. Costophrenic angle obliteration had appeared in 3.9% and 4.4%, respectively, in the two groups. Pleural calcification had appeared or progressed in 2% of both groups. The calculated area of pleural change (see methods) had progressed in 13.8% and 14.8%, respectively.

This study has found absolutely no differences in the evolution of radiological change between the group who continued exposure after the first radiograph and

those who did not despite the fewer years of exposure and fewer fibre-years in the latter group up to the date of first radiograph. The same conclusion was reported from a study of workers exposed to asbestos (non-miners)³ over a similar period of follow up as in this study.

There are some possible weaknesses in the study. A relatively short period of follow up means that the group who continued exposure had had only a mean of 4.5 years of exposure and 6.2 fibre-years after the first radiograph and that the time may have been too short for the additional exposure to take effect and the dose too low.

Cookson *et al* have shown how important duration of follow up is as progression in their study continued throughout 33 years and progression from category 0 to category 1 continued to happen throughout that period.¹⁰ Although their group was exposed to higher doses over a shorter period (median duration 37 months, median cumulative exposure 91 fibre-years), it is highly probable that new attacks and progression will continue to occur in the two groups in this study. Possibly with longer follow up the men who ceased exposure may be shown to have benefited.

To our knowledge, there are no published data to allow a comparison of radiological progression with time between chrysotile and amphibole exposed men. In the study of Liddell *et al* over a follow up period of 20 years in chrysotile miners the study group was selected in a different manner to this study.⁶ Nevertheless, his finding of new attacks of 12.5% to 16.2% is comparable with 12.3% and 10.6% in our exposed and unexposed groups, respectively. Unfortunately progression rate was calculated in different ways in the two studies and are not comparable.

Our results indicate that once a dose of asbestos sufficient to initiate the disease has been retained in the lungs the disease is inexorably progressive. The dose needed to initiate is no doubt influenced by many individual factors, genetic factors, differences in the rate of clearance of dust (which may, for instance, be affected by age and bronchial anatomy).

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