Biphasic lung diffusing capacity: detection of early asbestos induced changes in lung function

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Abstract

Asbestos related changes in the single breath carbon monoxide diffusing capacity (D_LCO) were longitudinally analysed in 14 subjects exposed predominantly to chrysotile asbestos in an asbestos cement factory. These subjects were examined annually over the past nine years; their lung function was initially characterised with increased D_LCO as the sole functional abnormality and they had normal chest radiographs. The radiological examination included a chest *x* ray film and, in the past two years, high resolution computed tomography (HRCT). A biphasic D_LCO change was found: an initial increase followed by a relative decrease. The increase in D_LCO was mainly caused by an increase in the membrane component (Dm). Indomethacin treatment applied after the sixth annual follow up significantly reduced D_LCO and Dm. The decrease in D_LCO correlated well with the parenchymal abnormalities found on HRCT, whereas the chest *x* ray film profusion score for small opacities (ILO classification) was unchanged. In conclusion, the data suggested that, as well as the absolute values of pulmonary function tests, the measurement of progression of functional parameters is essential in the assessment of pleural and parenchymal disease of the lung related to exposure to asbestos. High resolution computed tomography is suggested as the radiological method of choice in subjects with an isolated decrease in D_LCO. Exposure to asbestos can be associated not only with a reduction in D_LCO, but also with a temporary increase in D_LCO caused by a subclinical inflammatory reaction.

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The diagnosis of asbestosis is based on a combination of clinical and radiological findings and measured changes in lung function.[1] Clinical symptoms, physical findings, and functional abnormalities (reductions in forced vital and diffusing capacities) were usually evident only in the advanced stages of disease and appeared too insensitive for early detection of asbestosis.[2,3] Recent studies have shown, however, that functional abnormalities may precede roentgenographic changes in the asbestos exposed subjects.[4-6] The radiological diagnosis of asbestosis has been recently improved by HRCT, which has increased sensitivity over the standard *x* ray film or conventional CT for the detection of early pleural and parenchymal changes.[7]

In the present study we performed a nine year follow up and analysed lung function and radiological data in a group of asbestos cement workers, with particular emphasis on single breath carbon monoxide diffusing capacity (D_LCO) and HRCT.

Subjects and methods

The study population consisted of asbestos cement workers. A continuous survey was started in 1979 with the purpose of investigating asbestos associated health hazards in exposed subjects. The study population was composed of all exposed subjects regardless of whether they had any symptoms. All subjects had complete medical and occupational histories taken and underwent physical examination, resting pulmonary functional tests, and chest radiography. Pulmonary function test results at the end of the sixth follow up indicated that 14 workers of the total number of 215 (7%) had a greater than

Table 1 Pulmonary function and radiological data in 14 asbestos exposed subjects at the start of nine year follow up period

No	Expo (y)	Functional data						Chest radiographs (ILO profusion score)
		D_LCO (%)	D_LCO/VA (%)	Dm (%)	Vc (%)	FVC (%)	FEV_1(%)	
1	23	91	95	114	94	93	98	0/0
2	5	88	84	86	113	84	92	0/0
3	9	90	93	106	107	96	90	0/0
4	4	93	96	96	90	87	84	0/0
5	20	103	96	99	86	93	93	0/0
6	17	87	94	118	94	88	90	0/0
7	14	94	98	106	89	98	94	0/0
8	7	96	101	94	85	93	102	0/0
9	2	90	94	98	92	102	90	0/0
10	19	93	91	97	103	94	98	0/0
11	15	86	89	103	86	95	103	0/0
12	7	87	96	98	94	102	92	0/0
13	10	96	97	89	90	94	94	0/0
14	12	100	99	110	83	91	93	0/0
Mean (SD)	12 (7)	92 (5)	95 (4)	101 (9)	93 (9)	94 (5)	94 (5)	

Expo = Duration of asbestos exposure at the start of the study.

120% increase in predicted D_LCO. These 14 workers had normal pulmonary function (including D_LCO) and chest radiographs, and were asymptomatic at the start of the study (table 1). They had been involved in manufacture of asbestos cement products, where chrysotile asbestos has been predominantly used. Their average age was 42 (SD 8) years with a mean asbestos exposure of 21 (SD 6) years at the end of the nine year follow up period. They formed the study population and comprised 11 non-smokers and three smokers.

Pulmonary function testing included measurements of the forced vital capacity (FVC), and the forced expiratory volume in one second (FEV_1). The best of three readings was chosen on the basis of the largest absolute sum of FVC and FEV_1. The values obtained were expressed as percentages of the predicted values.[8] Measurement of D_LCO was with the single breath technique in an upright seated position (Morgan MK-4, England).[9] The same equipment was used for all nine follow ups in D_LCO measurements. For single breath (10 second breath hold) D_LCO measurements, an inspired concentration of 0·3% CO, 9% He, 21% O_2, and balance nitrogen was used. The arithmetic mean of two acceptable tests for each subject was used and D_LCO values were corrected for haemoglobin concentration.[10] Estimations of the membrane diffusing component (Dm) and the pulmonary capillary blood volume (Vc) were done according to the methods of Roughton and Forster.[11] Measurements of D_LCO were made at two alveolar oxygen tensions, namely, during inhalation of room air and after breathing 100% O_2 for 20 minutes. Bates' equation ($1/\Theta = 0{\cdot}0057\ PAO_2 + 0{\cdot}75$) and a λ value of 2·5 were used for estimation of D_LCO.[12] Reference values for D_LCO, D_LCO/VA, Dm, and Vc were taken from Cotes.[13] All 14 subjects were without any clinical abnormalities known to affect D_LCO volume (anaemia, asthma).

After the sixth annual follow up all 14 subjects were offered 125 mg of indomethacin per day for seven days and they consumed the drug regularly. The transfer factor and its components (Dm and Vc) were measured the day after completion of the treatment.

Radiological examination included postero-anterior and lateral chest radiographs, which were scored independently by two thoracic radiologists trained in the ILO international classification for pneumoconioses.[14] The HRCT was performed in all subjects with a Siemens SOMATOM scanner. Scans were made at five levels through the lower thorax in both prone and supine positions for a total of 10 images.[15] Scans were acquired at full expiration by using 2 mm thickness, seven second scan acquisition time, 720 projections, 125 kV, 780 mAs, and a strong edge enhancement algorithm. All HRCT scans were photographed at three window settings—namely for the pleura and mediastinum, for the lungs, and for the lung and pleura. The HRCT scoring was done according to Aberle and coworkers[7] by two radiologists and the probability for asbestosis was scored on a five point scale (from 1 = normal, to 5 = abnormal and high probability for asbestosis).

Statistical analysis was performed with the paired Student's t test and $p < 0{\cdot}05$ was taken as the level of significance. For comparison of the various functional and imaging tests Pearson's correlation was used.

Results

At the start of the study, all subjects had normal lung function and chest radiographs (table 1). The onset of exposure to asbestos ranged individually from two to 23 years. At the sixth follow up, their lung function

Table 2 Pulmonary function and radiological data in 14 asbestos exposed subjects at sixth follow up

No	Expo (y)	Functional data						Chest radiographs (ILO profusion score)
		D_LCO (%)	D_LCO/VA (%)	Dm (%)	Vc (%)	FVC (%)	FEV_1(%)	
1	28	133	140	147	102	90	96	0/0
2	10	148	146	143	124	76	93	0/0
3	14	153	173	164	116	95	88	0/0
4	9	152	163	161	104	85	86	0/0
5	25	162	174	150	129	93	93	0/0
6	22	157	161	121	94	92	96	0/0
7	19	131	148	173	108	97	88	0/0
8	12	140	108	128	92	90	94	0/0
9	7	150	146	133	94	98	93	0/0
10	24	141	158	146	114	93	95	0/0
11	19	144	152	138	88	94	106	0/0
12	12	136	143	148	132	98	94	0/0
13	15	152	135	152	104	94	90	0/0
14	17	128	136	142	86	92	89	0/0
Mean (SD)	17 (6)	144 (10)	149 (16)	146 (14)	107 (14)	92 (6)	93 (5)	

Table 3 Pulmonary function and radiological data in 14 asbestos exposed subjects at the end of nine year follow up period

No	Expo (y)	Functional data						Radiological data	
		D_LCO (%)	D_LCO/VA (%)	Dm (%)	Vc (%)	FVC (%)	FEV_1(%)	ILO profusion score	HRCT PARE
1	32	96	103	108	87	89	95	0/0	2
2	14	119	126	126	117	71	88	0/1	2
3	18	108	113	106	109	101	92	0/0	1
4	13	88	108	93	85	75	83	0/0	3
5	29	113	125	126	114	92	90	0/0	2
6	26	82	85	91	85	87	94	0/1	2
7	23	85	88	90	94	96	95	0/0	2
8	16	104	114	113	101	92	96	0/0	1
9	11	92	97	107	97	96	94	0/0	2
10	28	112	121	132	120	91	95	0/0	1
11	23	101	112	108	90	94	101	0/0	2
12	16	118	128	127	122	100	93	0/0	1
13	19	111	115	125	93	91	95	0/0	2
14	21	97	104	109	85	88	91	0/1	2
Mean (SD)	21 (6)	102 (12)	110 (13)	111 (14)	98 (14)	90 (8)	93 (5)		

HRCT PARE = HRCT score for probability for parenchymal fibrosis (1 (low probability) indicates no parenchymal disease; 2 (intermediate probability) indicates parenchymal disease with possible relation to asbestosis; and 3 (high probability) indicates parenchymal asbestosis).

was characterised by the following findings compared with data collected during the first follow up: increased D_LCO ($p < 0.0001$), D_LCO/VA ($p < 0.0005$), Dm ($p < 0.0005$), and Vc ($p < 0.01$) (table 2). Chest radiographs were normal (table 2). The last follow up showed that all subjects had D_LCO, D_LCO/VA, Dm, and Vc in the normal range ($\pm 20\%$ of the predicted values), whereas two subjects had FVC below 80% (table 3); D_LCO ($p < 0.05$) and D_LCO/VA ($p < 0.01$) were still increased, however, when compared with the findings in the first follow up. Pleural thickening was found in four subjects (three focal and one diffuse) on the chest *x* ray film. Pleural thickening was seen on HRCT in six subjects, including the four subjects with pleural thickening on chest *x* ray film. All subjects had an ILO profusion score for small opacities (an index for parenchymal abnormalities) below 1/0 (table 3). The

Table 4 Correlation between various functional and imaging tests in 14 asbestos exposed subjects at the end of nine year follow up

Correlation*	r	p Value
D_LCO (% predicted) *v* HRCT score	0·650	0·012
D_LCO/VA (%) *v* HRCT score	0·545	0·042
Dm (% predicted) *v* HRCT score	0·586	0·029
Vc (% predicted) *v* HRCT score	0·689	0·008
FVC (% predicted) *v* HRCT score	0·574	0·030
FEV_1 (% predicted) *v* HRCT score	0·359	NS
ILO profusion *v* HRCT score	0·389	NS
ILO profusion *v* D_LCO (% predicted)	0·112	NS
ILO profusion *v* D_LCO/VA (%)	0·199	NS
ILO profusion *v* Dm (% predicted)	0·109	NS
ILO profusion *v* Vc (% predicted)	0·167	NS
ILO profusion *v* FVC (% predicted)	0·530	0·049
ILO profusion *v* FEV_1 (% predicted)	0·258	NS
D_LCO (% predicted) *v* Dm (% predicted)	0·927	0·00012
D_LCO (% predicted) *v* Vc (% predicted)	0·817	0·00025

*Pearson's correlation; NS = not significant.

HRCT analysis showed nine subjects, four subjects, and one subject to have intermediate, low, and high probabilities for developing asbestosis respectively (table 3).

Table 4 shows the statistical correlations between the various functional and imaging tests using Pearson's correlation. The HRCT had a higher correlation with functional tests and was possibly more sensitive than conventional chest radiography, although the small sample size (n = 14) makes this conclusion tentative.

Figure 1 shows the individual D_LCO changes in exposed subjects during the nine years of follow up. A biphasic change in D_LCO was noted—namely, an initial increase followed by a relative decrease. The timing of the increase in D_LCO in relation to the start of exposure to asbestos was individual. Because VA was unchanged, D_LCO/VA changed in a similar manner to D_LCO (fig 2). Figures 3 and 4 show the individual Dm and Vc changes. The initial D_LCO increase was mainly due to an increase in Dm, but not in Vc.

Figures 5 and 6 show the effect of indomethacin treatment for seven days on D_LCO, Dm, and Vc in 14 subjects after the sixth annual follow up. When compared with the corresponding pretreatment findings, mean D_LCO and Dm showed a significant reduction after treatment. On the other hand, indomethacin treatment had no effect on Vc.

Discussion

Lung function tests were suggested to be more sensitive than chest radiographs in detection of early asbestosis.[16] An increase in static elastic recoil pressure was reported in asbestos exposed subjects, but this test is non-specific and unsuitable for routine measurements.[17] Measurement of D_LCO is another sensitive functional test for the diagnosis of asbestosis. Picado and coworkers[16] found only 10 patients with decreased D_LCO in a group of 42 patients with diagnosed asbestosis. The same authors suggested that D_LCO changes do not regress, so that progressive reduction in D_LCO may be a reliable finding for the diagnosis of asbestosis.[16] We have shown previously that functional abnormalities may precede radiological changes in asbestos exposed subjects; the FVC and D_LCO decrements were seen with ILO profusion score below 1/1,[5] and a biphasic mid-expiratory change in flow rate (the initial

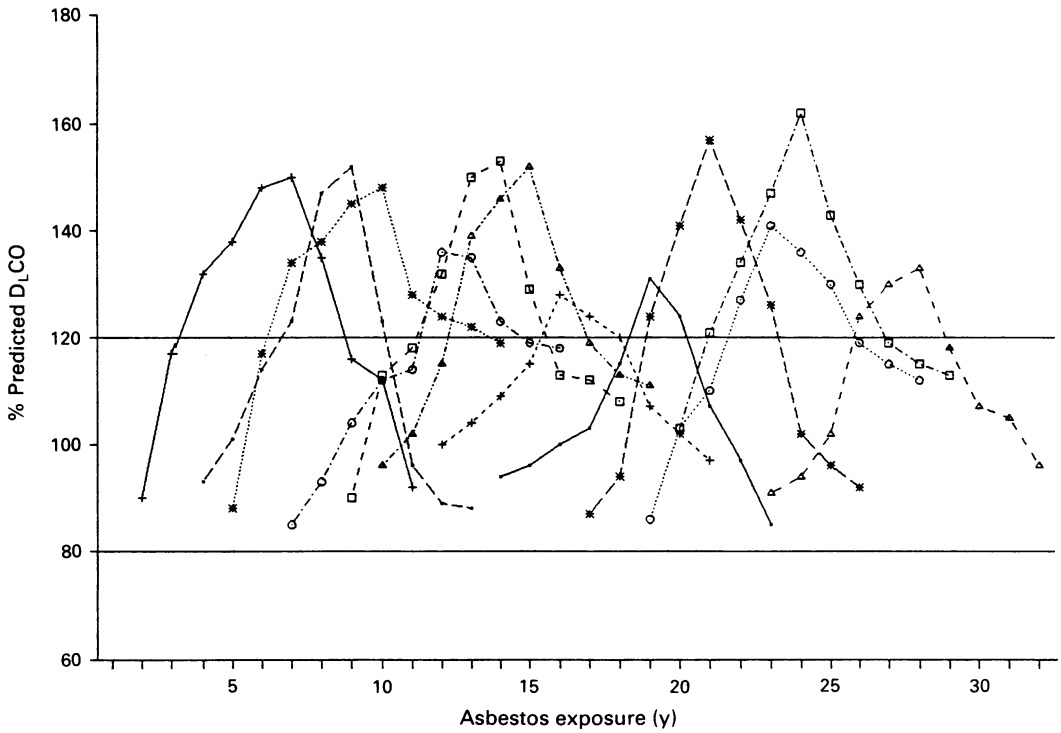

Figure 1 The time course of the individual D_LCO changes in 14 asbestos exoposed workers during nine years of follow up. The values are expressed as per cent of the predicted normal values. Horizontal lines represent limits of the normal range.

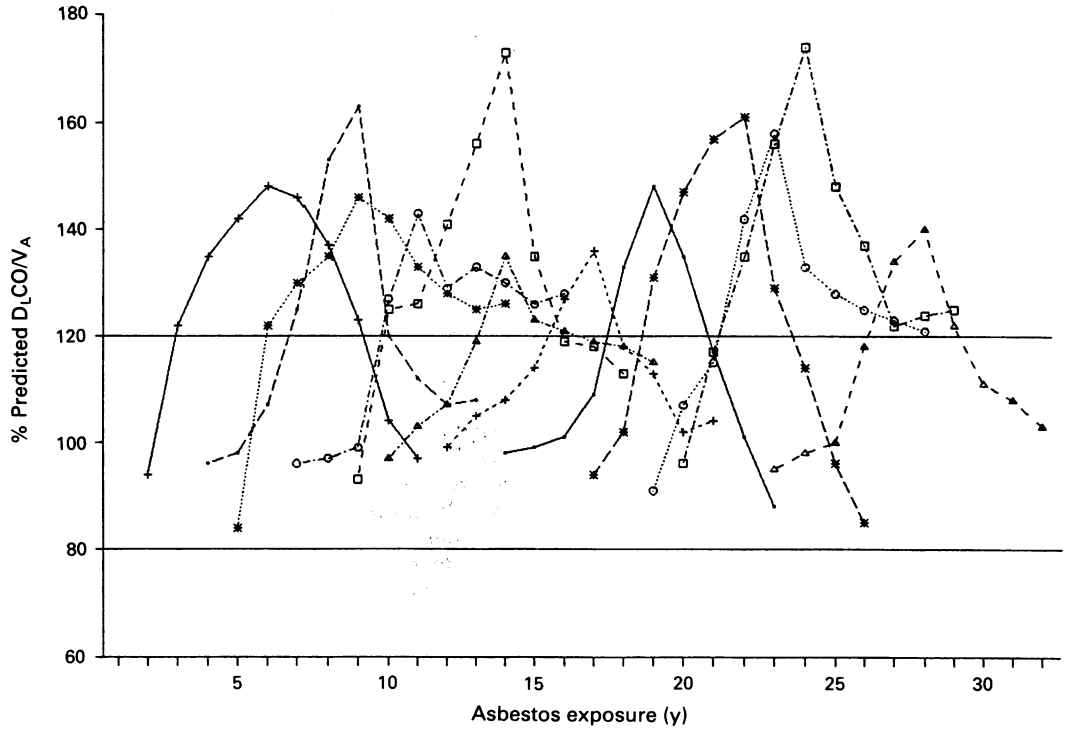

Figure 2 The time course of the individual D_LCO/VA changes in 14 asbestos exposed workers during nine years of follow up. Horizontal lines represent limits of the normal range.

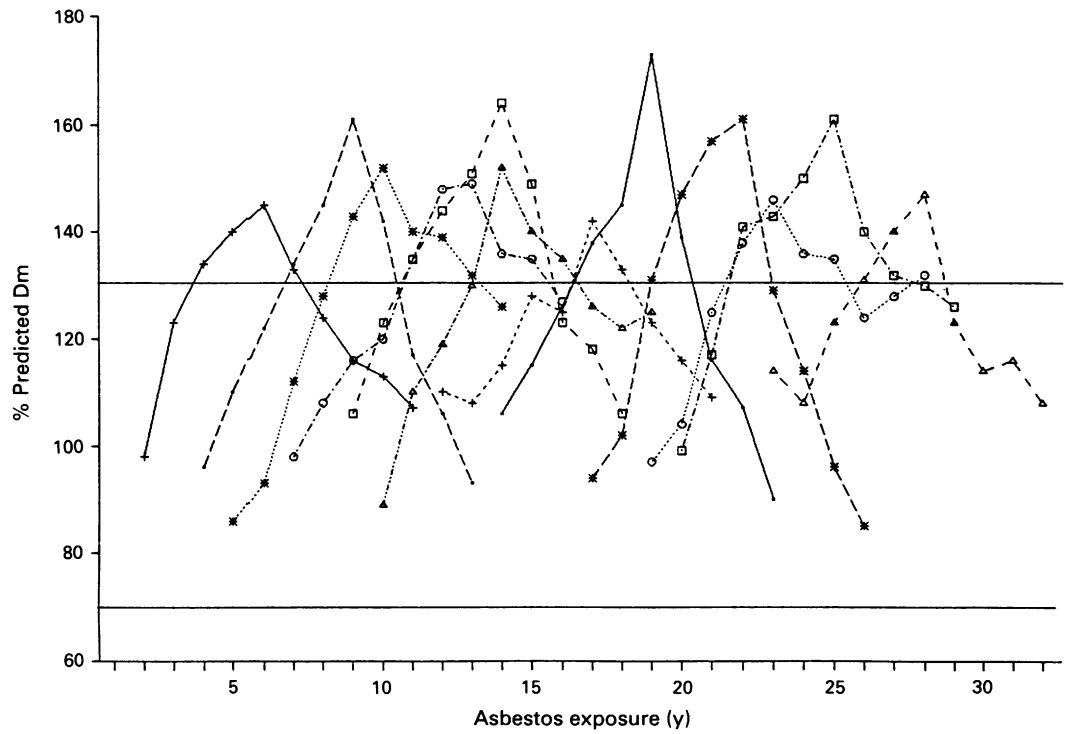

Figure 3 The time course of the individual Dm changes in 14 asbestos exposed workers during nine years of follow up. Horizontal lines represent limits of the normal range.

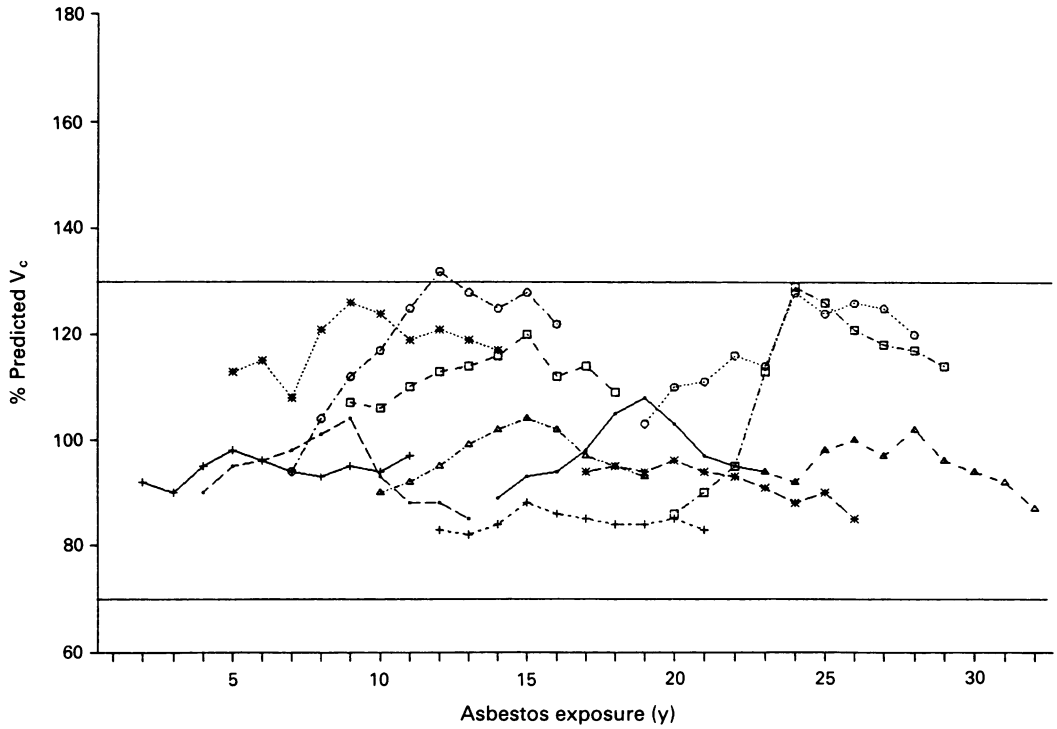

Figure 4 The time course of the individual Vc changes in 14 asbestos exposed workers during nine years of follow up. Horizontal lines represent limits of the normal range.

increase followed by a decrease) in some non-smoking workers was the earliest functional sign indicative of the future development of parenchymal asbestosis.[6] In most subjects, mid-expiratory flow rate and D_LCO decrements were correlated with abnormal HRCT suggestive of asbestosis, whereas chest radiographs were unchanged.[6]

The present study showed that in some subjects functional abnormalities precede radiological (chest *x* ray film) findings. A biphasic change in D_LCO was found—namely, an initial increase, as the earliest functional sign of exposure to asbestos, followed by a relative decrease. This functional response (D_LCO increase) was timed individually in relation to the start of exposure to asbestos (fig 1). After radiological abnormalities were found (as detected with HRCT

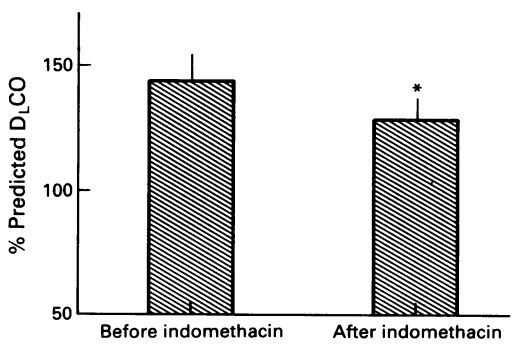

*Figure 5 The effect of indomethacin (125 mg once a day for seven days) on D_LCO after the sixth annual follow up (n = 14). Values are mean with SD. *p < 0·01 compared with pretreatment mean values.*

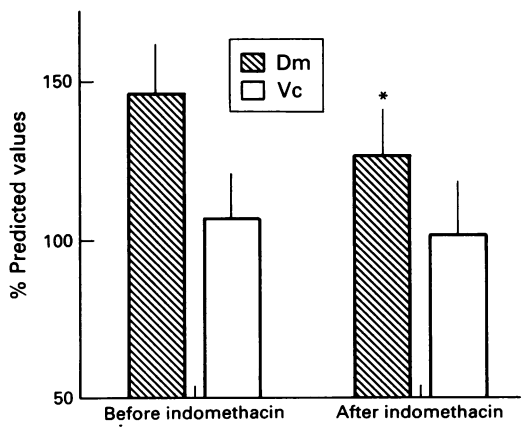

*Figure 6 The effect of indomethacin (125 mg once a day for seven days) on Dm and Vc after the sixth annual follow up (n = 14). Values are mean with SD. *p < 0·01 compared with pretreatment mean values.*

but not on the chest x ray films), a relative decrease in D_LCO was seen, but the absolute values were still in the normal range of the predicted values. Despite this fact, these subjects had a substantial decrease in D_LCO relative to the values observed in the previous few years of the follow up.

Another problem in the assessment of tests of pulmonary function is the fairly wide range of predicted values ($\pm 20\%$). Theoretically, a subject could start with an FVC or D_LCO of 120% of predicted values, and despite a large reduction (up to 40%), still be described as "functionally normal". This emphasises the need for establishing dynamic measurements of lung function as the main determinant in the diagnosis and prognosis of pulmonary disease.

The early phase of asbestosis can be characterised by multifocal inflammatory responses and it has been named "chronic interstitial pneumonia".[18] Pulmonary uptake of gallium was found to be increased in many subjects without roentgenographic changes associated with asbestosis, but who had been exposed either to chrysotile[19] or crocidolite.[20] This suggested the presence of subclinical pulmonary inflammation in these subjects. In a recent report, Garcia and coworkers[21] showed that asbestos fibres can increase the release of prostaglandin PGI_2 from the endothelial cell monolayer in a dose-dependent manner in vitro. As an active pulmonary vasodilator,[22] PGI_2 may increase the pulmonary blood volume. The process of inflammation is associated with local vasodilatation and increased blood volume. As inhalation of asbestos fibres leads to chronic multifocal inflammatory response, it is possible that D_LCO was initially increased in our subjects due to the increase in the pulmonary capillary blood volume. The membrane diffusion (Dm) and the pulmonary capillary blood volume (Vc) changed in a manner similar to D_LCO in our subjects (table 2), but most of the increase in D_LCO was due to an increase in Dm. It has been shown that dynamic exercise induces similar changes in Dm and Vc.[23] Possible mechanisms for these effects during exercise are the opening of capillaries of smaller diameter or thinner walls, and stretching of the capillaries to produce flattening of their cross section, and thinning of their walls.[23] The possible explanation of the finding that the increase in D_LCO in our subjects was predominantly caused by an increase in Dm could be the enhanced solubility of CO in an inflamed environment, rather then a decrease in the thickness of the alveolocapillary membrane.

To test the hypothesis that an asbestos induced increase in D_LCO was caused by increased local prostaglandin production, we investigated the effect of seven day indomethacin treatment in our subjects after the sixth annual follow up (figs 5 and 6). Indomethacin, an inhibitor of prostaglandin synthetase (cyclooxygenase), caused significant reductions in D_LCO and Dm in comparison with pretreatment values. Post-treatment D_LCO and Dm values were, however, still above those predicted. The possible explanation for this discrepancy could be asbestos induced increased concentrations of other local vasodilators known to be released in inflammatory reactions (for example, histamine, bradykinin).

Recently, HRCT has been shown to be more sensitive than chest radiography in detecting pleural and parenchymal pulmonary abnormalities.[7 24] Staples *et al*[24] reported that in 169 asbestos exposed subjects with no evidence of asbestosis on chest radiographs (ILO grade $< 1/0$), 34% had abnormalities on HRC consistent with asbestosis. Our data, although limited by the small sample size ($n = 14$), also support the argument that HRCT has a higher correlation with functional tests and that it may be more sensitive than conventional chest radiography in asbestos exposed subjects. We found pleural thickening on HRCT in six out of 14 subjects, although this was mild in five of them. In all six subjects, parenchymal abnormalities were also present on HRCT, thus preventing the selective investigation of the effects of isolated pleural thickening on pulmonary function. Staples *et al*[24] reported that pulmonary function in subjects with pleural plaques on HRCT was not significantly different from those without pleural plaques when parenchymal analysis by HRCT was normal.

In conclusion, our data show that dynamic functional findings precede radiological abnormalities on the chest x ray film in some subjects exposed to asbestos. The radiological method of choice in the early detection of asbestosis, indicated by reduction in D_LCO is considered to be HRCT, as suggested by other investigators. Our results suggest that exposure to asbestos can lead to a biphasic change in D_LCO (initial increase followed by relative decrease) and that D_LCO might be an early and sensitive functional indicator of future interstitial asbestosis. The initial increase in D_LCO was probably due to a subclinical inflamatory response. Prostaglandins seem to be one of the inflammatory mediators of an increase in D_LCO as indicated by a partial return of D_LCO to normal after treatment with indomethacin.

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Accepted 19 August 1991

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