*Thorax* 1991;46:33–38



# Abnormal lung function associated with asbestos disease of the pleura, the lung, and both: a comparative analysis

Kaye H Kilburn, Raphael H Warshaw

**Abstract**

**The impairment of lung function associated with different types of asbestos related disease was examined in 1298 men. The 310 men with circumscribed pleural lesions (plaques) or diffuse pleural thickening without asbestosis were compared with 596 men with asbestosis only and with 322 men with pleural abnormalities and asbestosis, as classified from chest radiographs by ILO pneumoconiosis criteria. Spirometric indices and total lung capacity (TLC; determined by planimetry) were measured and expressed as percentages of predicted values. Non-smoking men with pleural disease only had reduced values of mid and terminal expiratory flows (80·6 and 69·9% predicted) and a reduced $FEV_1$ (89% predicted) with a forced vital capacity (FVC) of 94% predicted. TLC was 104% predicted. Thus they had airways obstruction without restriction. Non-smoking men with pulmonary asbestosis (ILO profusion of opacities mostly 1/0 and 1/1) had pulmonary function similar to that of men with pleural disease. $FEV_1$ and FVC and flow rates at other lung volumes were lower in smokers with asbestosis (after adjustment for duration of smoking) than in the non-smokers with asbestosis. Airflow limitation was worse in the men with both pleural abnormalities and pulmonary asbestosis with lower values for mid expiratory flow, $FEV_1$ and FVC (but not TLC) than those with either abnormality alone, in both non-smokers and current smokers. Men with diffuse pleural thickening that included the costophrenic angles had more airways obstruction and air trapping and lower FVC values than those with circumscribed pleural disease.**

**University of Southern California School of Medicine, Environmental Sciences Laboratory, 2025 Zonal Avenue, Los Angeles, California 90033**
K H Kilburn

**Worker's Disease Detection Services, San Dimas, California 91773, USA**
R H Warshaw

Reprint requests to:
Dr Kilburn

Accepted 2 October 1990

Whether pleural plaques or diffuse pleural thickening (or a combination of the two) due to asbestosis are associated with impairment of lung function remains controversial.[1] The issues include whether impairment is present, the nature and degree of such impairment, and whether those with asbestos related pleural disease only have or will develop pulmonary asbestosis. Because asbestos fibres must pass through the lungs to reach the pleural space it is conceivable that individuals with pleural plaques or thickening have some pulmonary fibrosis that would be detectable histologically but not by chest radiography. Such subradiographic fibrosis, which could be detected by microscopic examination of histological sections of lung, could impair function.[2]

We therefore compared the pulmonary function of asbestos exposed workers with pleural abnormalities only, workers with pulmonary abnormalities only, and workers with both pleural and pulmonary abnormalities as determined radiographically. In addition, we compared the effects of different types of pleural lesions on pulmonary function. Pleural abnormalities were divided into (1) pleural calcifications only; (2) circumscribed lesions (pleural plaques) only; (3) diffuse pleural thickening; and (4) diffuse pleural thickening with obliterated costophrenic angles. Data on subjects in overlapping categories were not analysed. Findings are based on 1298 subjects selected from 4572 male boilermakers and pipefitters who were studied by one medical team using a standard protocol.

## Methods

The 1298 men were studied at 20 sites across the United States from Los Angeles, California, in the west to Mobile, Alabama, and Jacksonville, Florida, in the south and to Minneapolis, Minnesota, in the north. Most men were active or retired members of either the International Brotherhood of Boilermakers, Blacksmiths, Welders, Forgers, and Iron Ship Makers or the United Association of Plumbers and Pipefitters. Others were from metal or chemical trades. The study was approved by the Human Subjects Institutional Review Board of the University of Southern California. Informed consent was obtained from each worker. All men were white and had been first exposed to asbestos at least 15 years previously, and all had been exposed for at least five years. They included 259 non-smokers, 969 current smokers, and 70 ex-smokers, who were studied by questionnaires, physical examination of the chest,

radiography, and spirometry. The questionnaire was administered by trained interviewers. It included occupational history; asbestos exposure (proximity and duration); and medical, pulmonary, and cardiovascular history (with criteria for defining chronic bronchitis and asthma, based on standard questions from DLD-78 in the epidemiology standardisation project.[3] The diagnosis of chronic bronchitis was made when phlegm had been produced most days of the week for at least three months a year in two successive years. The diagnosis of asthma required the presence of episodic wheezing that was relieved spontaneously or with medication, with "normal breathing" between attacks. The chest was examined for size, shape, deformity, and quality of breath sounds (normal, decreased, or absent; rales; wheezing); the extremities were examined for cyanosis, clubbing, and oedema.

Spirometry was performed with rolling seal spirometers (Ohio 822), with the subjects standing and wearing a nose clip, and followed the ATS Snowbird recommendations.[4] Care was taken to ensure full or at least 10 seconds' expiration. Spirometric recordings were digitised from the tracing with the best effort and measured for volumes and flows, and the data were corrected to body temperature. Percentage of predicted value was calculated for each measurement by using regression equations derived from 188 Michigan men in the model group, which thereby adjusted the values for height, age, and years of cigarette smoking.[5] Volumes and flows were expressed as means for each group and as mean percentages of predicted values. Lung area was measured by planimetry of posterior-anterior and lateral radiographs and used to calculate total lung capacity (TLC) from the regression equation developed by Harris *et al.*[6]

Posteroanterior and lateral chest radiographs were obtained on standard 14 × 17 inch films with a Picker portable *x* ray machine (KV 120–130 and suitable grid); they were read in the presence of each worker for profusion of irregular opacities—that is, for pulmonary asbestosis and for type and extent of pleural asbestos signs on the basis of the 1980 ILO criteria for pneumoconiosis.[7] Films were interpreted by a single reader. Consistency of reading was high as judged by interval repeat readings of 100 films during the study, which were within 5% of the original readings. Workers with unilateral pleural abnormalities who had a history of trauma or infection matching their lesions were not considered to have asbestos pleural disease. Where radiographs were unsatisfactory or of borderline readability repeat films were taken until quality was ILO class 1 or 2. Men with pleural asbestos disease only were further classified into four exclusive groups: those with calcified pleural plaques only, pleural plaques only, diffuse pleural thickening, and diffuse pleural thickening with blunted or obliterated costophrenic angles. Diffuse pleural thickening was defined as uniform thickening on both lateral pleura not affecting the costophrenic angles. The same pleural classification was applied to men who had pulmonary asbestosis in addition.

All data were recorded in a machine readable (mark sense) format and machine read into a microcomputer (Atari 1040ST). Data were analysed after transfer to a Compaq computer by using Stata statistical programs including linear regression, a correlation matrix, and Student's *t* test (two tailed). Pulmonary function is presented in tables 1 and 2 as group mean % predicted values derived from regression equations in which they were compared with the values from 188 men in a stratified random population sample of Michigan whose pulmonary function has been modelled.[5] Thus each man's measurements were expressed as percentages of the values predicted from the data on the model population after adjustment for height, age, and years of cigarette smoking. To make population comparisons we used the pulmonary function values from all 370 Michigan men from a random population sample. To do this we added to the 188 normal men used to model pulmonary function 182 men who had one or more of the following: sputum production, dyspnoea, wheezing, angina pectoris, previously diagnosed lung disease, coronary heart disease, hypertension, rales, clubbing, cyanosis. Thus the population for comparison with the groups of men exposed to asbestos included all the men studied, not just those selected for absence of symptoms or of cardiorespiratory diseases. The significance of differences between group means of men with asbestos pleural signs and of those with asbestosis and the 370 Michigan men were determined with Student's *t* test. A p value below 0·05 defined significance.

## Results

Details and lung function values of the 259 men who had never smoked cigarettes are shown according to three categories of asbestos disease in table 1 and those of the 969 current cigarette smokers in table 2. For these comparisons we excluded 686 ex-cigarette smokers and others in ethnic groups for which there were no agreed predictive equations for pulmonary function measurements. When analysing the types of pleural disease we included 23 ex-smokers in table 3 and 47 ex-smokers in table 4.

In the 259 men who had never smoked cigarettes those with both pleural signs and pulmonary asbestosis (group 3) were slightly older and had had a correspondingly longer exposure to asbestos than those with pleural signs only (group 1) or those with asbestosis (group 2). Chronic bronchitis and asthma were more prevalent in these non-smokers than in the reference population of 370 men. Expiratory flows, including $FEV_1$, were lower in all three non-smoking groups with asbestos disease than the predicted values based on age, sex, and height derived from the reference group.[5] FVC was also decreased whereas TLC was normal. Although pulmonary function

*Table 1 Comparison of non-smokers with pulmonary impairment associated with pleural plaques and diffuse thickening, pulmonary asbestosis, and both pleural signs and pulmonary asbestosis (mean (SD) values)*

| | *Pleural signs only (n = 108)* | *Pulmonary asbestosis only (n = 98)* | *Pleural and pulmonary (n = 53)* |
|---|---|---|---|
| Age (y) | 61·9 (9·9) | 61·4 (10·0) | 67·0 (8·5) |
| Height (cm) | 173·2 (7·8) | 173·3 (7·3) | 172·7 (7·6) |
| Weight (kg) | 85·0 (16·7) | 87·9 (14·2) | 90·2 (18·1) |
| Asbestos exposure (y) | 32·4 (11·9) | 30·3 (9·7) | 33·7 (10·1) |
| Chronic bronchitis (%) | 30·6 | 20·4 | 32·1 |
| Asthma history (%) | 13·0 | 10·2 | 11·3 |
| Lung function | | | |
| Absolute values | | | |
| FVC (l) | 4·00 (0·79) | 4·01 (0·88) | 3·65 (0·83) |
| $FEV_1$ (l) | 3·01 (0·67) | 3·07 (0·74) | 2·74 (0·71) |
| $FEV_1$/FVC | 75·2 (7·7) | 76·4 (7·3) | 75·2 (9·1) |
| $FEF_{25-75}$ (l/s) | 2·46 (1·18) | 2·60 (1·16) | 2·24 (1·21) |
| $FEF_{75-85}$ (l/s) | 0·50 (0·33) | 0·55 (0·35) | 0·46 (0·32) |
| TLC (l) | 7·20 (1·00) | 7·21 (1·17) | 7·04 (1·05) |
| RV (l) | 3·20 (0·87) | 3·20 (0·89) | 3·39 (0·92) |
| RV/TLC | 44·4 (9·6) | 44·4 (9·8) | 48·2 (10·1) |
| % of predicted values | | | |
| FVC | 94·1 (15·0) | 93·6 (16·0) | 88·7 (17·0) |
| $FEV_1$ | 88·8 (15·3) | 90·0 (17·7) | 84·2 (17·2) |
| $FEF_{25-75}$ | 80·6 (33·8) | 85·3 (35·9) | 76·6 (36·7) |
| $FEF_{75-85}$ | 69·6 (37·9) | 78·5 (50·3) | 72·6 (49·4) |
| TLC | 104·1 (13·6) | 103·7 (13·5) | 101·6 (14·7) |

FVC—forced vital capacity; $FEV_1$—forced expiratory volume in one second; $FEF_{25-75}$, $FEF_{75-85}$—forced expiratory flow at 25–75% and at 75–85% of vital capacity; TLC—total lung capacity; RV—residual volume.

values were lower in the 53 men with pleural disease and asbestosis than in the other groups, the small differences were not significant. Mean body weight was significantly less in non-smoking men with pleural calcification only than in other groups.

Among the current smokers the 202 men with pleural disease were only slightly older than those with asbestosis (62 versus 58 years); 57 (28%) had chronic bronchitis and 33 (16·5%) had asthma. They had had more than 30 years of smoking cigarettes and 33 years of exposure to asbestos. Mid ($FEF_{25-75}$) and terminal expiratory flows, $FEV_1$, and FVC were lower ($p < 0·0001$) than those of the reference group. The 498 men with asbestosis only had reductions in lung function similar to those of men with pleural disease only. The 269 men with pleural disease and asbestosis were oldest (64 years) and more had asthma (26%). They had the most abnormal pulmonary function measurements (FVC 86%, $FEV_1$ 80%, $FEF_{25-75}$ 61%, TLC 112%, and RV/TLC 53% of predicted values). All values except TLC were below those of the reference population ($p < 0·0001$),[5] and FVC, $FEV_1$ and $FEF_{25-75}$ were below the values seen in the groups with pleural disease and with asbestosis only ($p < 0·001$). The increases in RV/TLC were associated with a decrease in $FEV_1$/FVC. When there was radiographic evidence of pleural signs only or asbestosis only, the reduction in pulmonary function was very similar. Men with both pleural disease and asbestosis had greater functional impairment than those with either disease alone.

*Table 2 Pulmonary impairment in currently smoking men associated with pleural asbestos disease compared with pulmonary asbestosis alone and with both pleural signs and pulmonary asbestosis (mean (SD) values)*

| | *Pleural signs only (n = 202)* | *Pulmonary asbestosis only (n = 498)* | *Pleural and pulmonary (n = 269)* |
|---|---|---|---|
| Age (y) | 61·6 (8·3) | 58·4 (9·8) | 64·0 (9·2) |
| Height (cm) | 173·8 (7·1) | 174·4 (6·2) | 173·5 (6·3) |
| Weight (kg) | 83·7 (14·6) | 86·0 (16·2) | 84·4 (14·8) |
| Asbestos exposure (y) | 33·0 (9·2) | 31·2 (10·0) | 33·8 (9·7) |
| Duration of smoking (y) | 32·2 (14·7) | 33·5 (13·0) | 36·9 (13·7) |
| Cigarettes/day | 26·2 (14·2) | 28·8 (13·8) | 27·4 (14·3) |
| Chronic bronchitis (%) | 28·2 | 36·9 | 37·2 |
| Asthma history (%) | 16·8 | 18·5 | 25·6 |
| Lung function | | | |
| Absolute values | | | |
| FVC (l) | 3·94 (0·82) | 4·08 (0·87) | 3·72 (0·85) |
| $FEV_1$ (l) | 2·76 (0·82) | 2·80 (0·81) | 2·47 (0·79) |
| $FEV_1$/FVC | 69·1 (12·0) | 68·1 (12·4) | 67·4 (12·6) |
| $FEF_{25-75}$ (l/s) | 1·98 (1·18) | 1·92 (1·20) | 1·56 (1·05) |
| $FEF_{75-85}$ (l/s) | 0·28 (0·25) | 0·39 (0·30) | 0·33 (0·25) |
| TLC (l) | 7·85 (1·14) | 7·88 (1·10) | 7·92 (1·14) |
| RV (l) | 3·91 (1·15) | 3·80 (1·11) | 4·20 (0·82) |
| RV/TLC | 49·3 (10·4) | 47·8 (10·7) | 52·6 (10·2) |
| % of predicted values | | | |
| FVC | 90·1 (16·1) | 90·5 (16·5) | 86·0 (16·4)* |
| $FEV_1$ | 86·5 (21·6) | 85·0 (20·6) | 79·9 (21·3)* |
| $FEF_{25-75}$ | 73·8 (37·6) | 69·8 (40·0) | 60·8 (35·9)* |
| $FEF_{75-85}$ | 73·9 (39·9) | 71·6 (47·5) | 70·9 (42·9) |
| TLC | 110·8 (13·9) | 110·7 (14·6) | 112·1 (15·1) |

*Significantly different from pleural only and from pulmonary only. Abbreviations as in table 1.

To determine whether circumscribed or diffuse pleural disease had different effects on pulmonary function, we combined smoking groups (data on 23 ex-smokers and 202 current smokers were adjusted for duration of smoking) and divided the 333 men with pleural disease into four categories: 45 had calcified plaques only, 98 had circumscribed plaques only, 129 had diffuse pleural thickening only, and 61 had diffuse pleural thickening together with blunting or obliteration of costophrenic angles (table 3). Data on men whose abnormalities overlapped these categories and those with diaphragmatic plaques only were not analysed further. Although there appeared to be a gradient of abnormality of lung function—from pleural calcification, which impaired function least, to plaques and then to diffuse thickening—the differences between the subgroups were not statistically significant. Men in all groups had flows significantly below those of the reference group. The 61 men who combined diffuse pleural thickening and blunting of one or both costophrenic angles, however, had significantly lower FVC, FEV and $FEF_{25-75}$ values than the other groups. $FEV_1$/FVC fell in parallel with these reductions and RV/TLC increased as TLC did not decrease. Mean body weight did not vary significantly between the groups, though men with calcification weighed less than those with diffuse pleural thickening.

When we compared the same four categories of pleural disease in the 369 men who also had asbestosis (irregular opacities with a profusion of 1/0 or more), we found no gradient of impairment from pleural calcification to plaques to diffuse pleural thickening (table 4). As in the "pleural only" comparisons, however, those whose diffuse thickening caused blunting of the costophrenic angles had more impairment than the other three groups and this difference was significant. The 84 men with asbestosis and diffuse pleural thickening with blunted or obliterated costophrenic angles had significantly lower flow rates and FVC

*Table 3 Pulmonary function impairment associated with four types of pleural signs: calcified plaques only, plaques only, diffuse pleural disease only, and diffuse pleural disease with blunt costophrenic angles in workers without irregular opacities of pulmonary asbestosis (mean (SD) values)*

| Group: | A<br>Calcification only (n = 45) | B<br>Plaques only (n = 98) | C<br>Diffuse thickening only (n = 129) | D<br>Diffuse thickening with blunt costophrenic angles (n = 61) |
|---|---|---|---|---|
| Age (y) | 64·8 (7·5) | 63·3 (8·6) | 60·5 (8·9) | 61·3 (9·0) |
| Height (cm) | 171·2 (6·0) | 173·5 (6·3) | 174·0 (7·0) | 175·4 (7·0) |
| Weight (kg) | 71·0 (14·2) | 81·2 (11·3) | 90·5 (15·4) | 88·1 (17·7) |
| Asbestos exposure (y) | 34·6 (8·5) | 32·5 (10·0) | 32·1 (10·9) | 32·2 (9·2) |
| Duration of smoking (y) | 23·1 (18·1) | 18·7 (17·5) | 23·1 (18·0) | 28·7 (14·8) |
| Cigarettes/day | 24·8 (14·7) | 26·6 (12·9) | 27·0 (16·8) | 25·5 (11·2) |
| Chronic bronchitis (%) | 33·3 | 27·6 | 28·7 | 38·8 |
| Asthma history (%) | 20·0 | 10·2 | 14·0 | 16·4 |
| Lung function | | | | |
| Absolute values | | | | |
| FVC (l) | 4·02 (0·70) | 4·07 (0·67) | 3·90 (0·79) | 3·77 (0·92) |
| $FEV_1$ (l) | 2·80 (0·76) | 2·94 (0·67) | 2·83 (0·72) | 2·61 (0·82) |
| $FEV_1$/FVC | 68·8 (13·5) | 71·9 (9·4) | 72·4 (10·0) | 68·4 (12·4) |
| $FEF_{25-75}$ (l/s) | 2·09 (1·24) | 2·20 (1·20) | 2·12 (1·11) | 1·82 (1·08) |
| $FEF_{75-85}$ (l/s) | 0·44 (0·28) | 0·43 (0·29) | 0·40 (0·27) | 0·37 (0·26) |
| TLC (l) | 7·67 (0·98) | 7·68 (1·02) | 7·48 (1·24) | 7·79 (0·92) |
| RV (l) | 3·64 (0·85) | 3·61 (0·72) | 3·58 (0·82) | 4·02 (0·84) |
| RV/TLC | 47·1 (8·5) | 46·4 (9·4) | 47·3 (9·8) | 51·4 (10·7) |
| % of predicted values | | | | |
| FVC | 98·6 (15·3) | 94·5 (11·6) | 88·8 (13·9) | 84·8 (17·7)* |
| $FEV_1$ | 92·2 (22·5) | 90·5 (16·2) | 86·2 (17·7) | 79·4 (20·8)* |
| $FEF_{25-75}$ | 79·7 (45·8) | 78·7 (37·9) | 75·9 (35·0) | 66·3 (34·6)* |
| $FEF_{75-85}$ | 85·5 (45·3) | 75·0 (43·6) | 68·5 (35·0) | 68·2 (41·5) |
| TLC | 108·7 (11·3) | 109·4 (12·8) | 102·9 (13·7) | 106·6 (12·7) |

*p < 0·0001 in the comparison with groups A, B, and C. Abbreviations as in table 1.

than the other groups with both asbestosis and pleural disease. Further subdivision of pleural disease into the ILO categories according to the extent of pleural plaques and the extent of diffuse pleural thickening—that is, ILO categories 1, 2, and 3—showed no significant correlations with RV/TLC in regression analysis. Thus the most sensitive indicator of airways obstruction could not distinguish differences within the category of pleural disease.

## Discussion

The expiratory flow limitation and air trapping seen in workers with pleural disease was interpreted as airways obstruction. This airways obstruction was the same as that seen in men

*Table 4 Pulmonary impairment associated with four types of pleural signs: calcified plaques only, plaques only, and diffuse pleural disease without and with obliterated costophrenic angles in 369 men with pulmonary asbestosis according to radiographs (mean (SD) values)*

| Group: | A<br>Calcification only (n = 42) | B<br>Plaques only (n = 95) | C<br>Diffuse thickening only (n = 148) | D<br>Diffuse thickening with blunt costophrenic angles (n = 84) |
|---|---|---|---|---|
| Age (y) | 68·7 (8·7) | 65·0 (9·0) | 63·5 (8·7) | 66·2 (9·2) |
| Height (cm) | 172·8 (5·3) | 173·9 (6·4) | 173·2 (6·6) | 172·5 (6·6) |
| Weight (kg) | 77·6 (18·3) | 82·9 (15·6) | 89·4 (14·6) | 86·0 (14·4) |
| Asbestos exposure (y) | 35·3 (7·8) | 33·8 (9·8) | 34·7 (19·2) | 33·4 (11·1) |
| Duration of smoking (y) | 32·4 (20·5) | 29·2 (18·0) | 29·3 (17·4) | 31·8 (16·4) |
| Cigarettes/day | 20·4 (11·0) | 24·7 (12·9) | 28·4 (15·6) | 28·5 (15·1) |
| Chronic bronchitis (%) | 33·3 | 33·7 | 31·8 | 44·0 |
| Asthma history (%) | 27·8 | 16·8 | 25·0 | 22·6 |
| Lung function | | | | |
| Absolute values | | | | |
| FVC (l) | 3·56 (0·74) | 3·90 (0·78) | 3·76 (0·77) | 3·30 (0·78) |
| $FEV_1$ (l) | 2·35 (0·68) | 2·65 (0·69) | 2·65 (0·68) | 2·17 (0·72) |
| $FEF_{25-75}$ (l/s) | 1·47 (0·38) | 1·80 (1·09) | 1·84 (1·09) | 1·30 (0·79) |
| $FEF_{75-85}$ (l/s) | 0·31 (0·20) | 0·36 (0·25) | 0·38 (0·28) | 0·31 (0·22) |
| TLC (l) | 7·66 (1·43) | 7·80 (1·24) | 7·47 (1·06) | 75·1 (0·96) |
| $FEV_1$/FVC | 65·9 (13·4) | 68·4 (12·1) | 70·4 (10·3) | 65·4 (13·1) |
| RV/TLC | 52·7 (9·7) | 49·6 (8·6) | 49·3 (9·8) | 55·9 (9·8)* |
| % of predicted values | | | | |
| FVC | 86·7 (16·7) | 90·6 (15·3) | 87·9 (16·1) | 79·0 (16·3)† |
| $FEV_1$ | 80·1 (19·6) | 85·2 (18·8) | 85·0 (19·0) | 71·8 (19·6)† |
| $FEF_{25-75}$ | 60·3 (31·4) | 68·8 (37·8) | 68·9 (35·5) | 51·3 (28·4)† |
| $FEF_{75-85}$ | 74·6 (45·2) | 74·5 (49·1) | 74·4 (45·0) | 69·5 (49·0) |
| TLC | 106·6 (18·8) | 106·7 (15·5) | 103·2 (13·3) | 104·5 (13·1) |

*p < 0·05 in the comparison with groups B and C; †p < 0·0001 in the comparison with groups A, B, and C. Abbreviations as in table 1.

with asbestosis only. When signs of both pleural and pulmonary asbestos disease were present airflow limitation was significantly worse than when either was present alone. Among those with pleural disease there was a progressive increase in airways obstruction through the different types of disease, the rank order being calcified plaques, plaques, diffuse pleural thickening, diffuse thickening with blunted costophrenic angles. The only statistically significant reduction, however, was seen when costophrenic angle blunting accompanied diffuse thickening, regardless of whether asbestosis was present or not. Such blunting appears to indicate an additional disorder that interferes with emptying of the lung. Although diaphragmatic movement may be compromised, fibrotic strands would appear to extend deeply into the lower lobes. Such strands have been seen on extended scale computed tomograms.[8 9]

This study extends previous reports of pulmonary functional impairment associated with pleural disease and does so with a moderately large number of non-smoking men. It is the first large study to show that functional impairment from pleural asbestos disease approximates in severity that due to asbestosis in non-smokers in a population of men exposed to asbestos studied cross sectionally. In fact, the data suggest that airways obstruction occurs in men who have been exposed to asbestos whether their radiographic abnormalities are pleural or pulmonary, but that combined pleural and pulmonary asbestosis produced more obstruction. Thus there is airways obstruction with either pleural disease only or pulmonary disease only and additional airways obstruction with a reduced vital capacity and increased residual volume when pleural and pulmonary forms of asbestosis are combined. Furthermore, when diffuse pleural thickening causes obliteration of the costophrenic angles, there is still more impairment in both the absence and presence of radiographic signs of asbestosis. Total lung capacity was not decreased as radiographic abnormalities increased. In currently smoking subjects with pulmonary asbestosis, pleural disease further reduced function.

The differences in body weight between men with different kinds of pleural disease is unexplained. Although the explanation may simply be easier recognition of calcification in thin than in fat people, and a greater likelihood of detecting diffuse thickening in the latter, other possibilities, including metabolic reasons, should be considered.

Understanding of the physiological impairment underlying asbestos related pleural disease has evolved since 1968. Reductions in FVC and maximum voluntary ventilation were associated with pleural calcification in British dockyard workers accepted as having asbestos disease by the pneumoconiosis panel.[10] Forty men with pleural calcification, 46 with plaques, 48 with diffuse thickening, and 11 with pulmonary fibrosis showed progressive impairment in function by comparison with 41 "normal" men.[11] Although Zitting[12] found a decreased $FEV_1$ and FVC in 130 Finnish workers with combined pleural abnormalities and pulmonary asbestosis (ILO profusion grades 0/1, 1/0 and 1/1), they did not determine whether pleural disease alone impaired function. In a German study pleural disease alone reduced $FEV_1$ but to a lesser extent than did pulmonary asbestosis.[13] Swedish construction workers with pleural densities had significantly reduced $FEV_1$ and flows at different lung volumes ($MEF_{75}$, $MEF_{50}$, $MEF_{25}$), closing volume, FVC, and transfer factor for carbon monoxide (TLCO). They also had more chronic bronchitis than did asbestos exposed men and unexposed controls.[14]

Pleural plaques in men who had never smoked and in smokers 20–25 years after asbestos exposure was associated with a reduction in $FEV_1$ and FVC and a 40% reduction in compliance in Uppsala[15] and diffuse pleural disease was associated with a reduced $FEV_1$, TLC and TLCO in 37 British dockyard workers.[16] The greatest functional impairment occurred in those with obliteration of costophrenic angles and extensive pleural thickening, as in the present study. In 129 sheet metal workers with bilateral pleural changes and 89 with unilateral pleural plaques only, FVC was reduced but flows were not,[17] which suggests to us that vital capacity manoeuvres may have been incomplete owing to early termination of effort. A reduction in FVC was also more common in 87 non-smoking men with pleural plaques than in 115 non-smoking controls from shipyards in Gothenberg, Sweden.[18] $FEV_1$ was below 80% of predicted in 11% of 200 Norwegian men under 70 years of age who had pleural plaques.[19]

Small airways obstruction, increased air trapping, and decreased FVC were found in 89 railway carriage repair workers with pleural plaques[20 21] and decreased $FEV_1$ and FVC (but no reduction in TLCO) in 377 railway yard workers with pleural plaques.[22] Cotes and King[23] reported reduced expiratory flows and FVC with diffuse pleural thickening, and a further reduction with blunting of the costophrenic angles, in 122 men. No additional impairment was found in men with pulmonary asbestosis, perhaps because of the low profusions of opacities. Pulmonary asbestosis developed over 10 years of observation in 10% of 201 British shipyard workers with pleural abnormalities.[24] The prevalence of plaques increased from 24% to 32% over four years in 75 Norwegian construction workers, in association with a fall in $FEV_1$ and FVC in about 6%.[25]

These studies support the idea of a worsening of pleural disease with time and the appearance of asbestosis in men with pleural plaques. When combined with the results of the present study, they suggest that in the presence of any radiographic sign of asbestos disease there is functional impairment. $FEV_1$ and FVC were reduced when circumscribed or diffuse pleural thickening became apparent and were further reduced when costophrenic angles were blunted. The degree of impairment from circumscribed or diffuse pleural thickening was

similar to that from pulmonary asbestosis (irregular opacities) alone. Obliteration of costophrenic angles was associated with additional functional impairment in men with pleural disease only and in those with combined pleural disease and asbestosis. Extended scale computed tomography has shown fibrosis extending from pleura into lung in some patients with diffuse thickening and blunted costophrenic angles.[8 9] Some of these patients have a history of pleural effusion, which may be one mechanism for these changes.[8]

The inference from this study and earlier data is that when pleural thickening is visible on a radiograph terminal and respiratory bronchioles are also diseased,[26] though the changes are not sufficiently radiodense to be seen on the radiograph. Local zones of restricted lung movement due to circumscribed or diffuse pleural scarring would not obstruct airways. Air trapping may conceivably occur, however, when there is symphysis of parietal to visceral pleura, as when costophrenic angles are obliterated. Plaques or diffuse pleural thickening did not reduce or "restrict" total lung capacity, not did they produce a different pattern of impairment from the orderly continuum of obstruction in small airways proceeding to air trapping and a reduced vital capacity seen in pulmonary asbestosis.[27] We think therefore, that pleural asbestos disease signifies the presence of pulmonary asbestosis that is beneath the threshold for detection by routine chest radiography. The probable lesions are cellular infiltrates and fibrosis around small bronchioles,[26] limiting flow in these airways as measured by spirometry.

In conclusion, men exposed to asbestos who have pleural disease have impaired pulmonary function. Those with calcified pleural plaques only have the least reduction in function but have more pulmonary impairment than men with asbestos exposure alone, and calcified plaques therefore are more than a marker of exposure.


1 American Thoracic Society. The diagnosis of nonmalignant diseases related to asbestos. ATS statement. *Am Rev Respir Dis* 1986;**134**:363–8.
2 Kilburn KH, Warshaw RH. Pulmonary functional impairment associated with pleural asbestos disease: circumscribed and diffuse thickening. *Chest* 1990;**98**:965–72.
3 Ferris BG Jr. Epidemiology standardisation project. *Am Rev Respir Dis* 1978;**118**:7–54.
4 American Thoracic Society. Snowbird workshop on standardisation of spirometry. ATS statement. *Am Rev Respir Dis* 1979;**119**:831–8.
5 Miller A, Thornton JC, Warshaw R, Bernstein J, Selikoff J, Teirstein AS. Mean and instantaneous expiratory flows, FVC, and $FEV_1$: prediction equations from a probability sample of Michigan, a large industrial state. *Bull Eur Physiopathol Respir* 1986;**22**:589–97.
6 Harris TR, Pratt PC, Kilburn KH. Total lung capacity measured by roentgenograms. *Am J Med* 1971;**50**:756–63.
7 International Labour Office. *U/C International classification of radiographs of pneumoconiosis*. Geneva: International Labour Office, 1980. (Occupational safety and health series.)
8 Wright PH, Hanson A, Kreel L, Capel LH. Respiratory function changes after asbestos pleurisy. *Thorax* 1980; **35**:31–6.
9 Aberle DR, Gamsu G, Ray CS, Feuerstein IM. Asbestos-related pleural and parenchymal fibrosis: detection with high resolution CT. *Radiology* 1988;**166**:729–34.
10 Leathart GL. Pulmonary function tests in asbestos workers. *Trans Soc Occup Med* 1968;**18**:49–55.
11 Lumley KPS. Physiological changes in asbestos pleural disease. In: Walton WH, ed *Inhaled particles IV*. New York: Pergamon, 1977:781–7.
12 Zitting A, Huuskonen MS, Alanko K, Mattsson T. Radiographic and physiological findings in patients with asbestosis. *Scand J Work Environ Health* 1978;**4**:275–83.
13 Arzt GH, Pirtkien R, Rosenthal H. Review of lung function data in 195 patients with asbestosis of the lung. *Int Arch Occup Environ Health* 1980;**45**:63–79.
14 Hedenstierna G, Alexandersson R, Kolmodin-Hedman B, Szamosi A, Tollqvist J. Pleural plaques and lung function in construction workers exposed to asbestos. *Eur J Respir Dis* 1981;**62**:111–22.
15 Fridriksson HV, Hedenstrom H, Hillerdal G, Malmberg P. Increased lung stiffness in persons with pleural plaques. *Eur J Respir Dis* 1981;**62**:412–24.
16 McGavin CR, Sheers G. Diffuse pleural thickening in asbestos workers: disability and lung function abnormalities. *Thorax* 1984;**39**:604–7.
17 Baker EL, Dagg T, Greene RE. Respiratory illness in the construction trades: 1. The significance of asbestos-associated pleural disease among sheet metal workers. *J Occup Med* 1985;**27**:483–9.
18 Jarvholm B, Sanden A. Pleural plaques and respiratory function. *Am J Ind Med* 1986;**10**:419–26.
19 Hilt B, Lien JT, Lund-Larsen PG. Lung function and respiratory symptoms in subjects with asbestos-related disorders: a cross-sectional study. *Am J Ind Med* 1987; **11**:517–28.
20 Hjortsberg U, Orback P, Arborelius M, Ranstam J, Welinder H. Railroad workers with pleural plaques: I. Spirometric and nitrogen washout investigation on smoking and non-smoking asbestos exposed workers. *Am J Ind Med* 1988;**14**:635–41.
21 Hjortsberg U, Orback P, Arborelius M, Ranstam J, Welinder H. Railroad workers with pleural plaques: II. Small airway dysfunction among asbestos exposed workers. *Am J Ind Med* 1988;**14**:643–7.
22 Oliver LC, Eisen EA, Greene R, Sprince NL. Asbestos-related pleural plaques and lung function. *Am J Ind Med* 1988;**14**:649–56.
23 Cotes JE, King B. Relationship of lung function to radiographic reading (ILO) in patients with asbestos related lung disease. *Thorax* 1988;**43**:777–83.
24 McMillan GHG, Rossiter CE. Development of radiological and clinical evidence of parenchymal fibrosis in men with non-malignant asbestos-related pleural lesions. *Br J Ind Med* 1982;**39**:54–9.
25 Ohlson CG, Bodin L, Rydman T, Hogstedt C. Ventilatory decrements in former asbestos cement workers: a four year follow up. *Br J Ind Med* 1985;**42**:612–6.
26 Wright JL, Churg A. Morphology of small-airway lesions in patients with asbestos exposure. *Hum Pathol* 1984;**15**: 68–74.
27 Kilburn KH, Warshaw RH. Effects of asbestos exposure, the presence and severity of asbestosis and of cigarette smoking on air ways obstruction, lung volume and air trapping. *Am Rev Respir Dis* (in press).