

Lung function impairment in relation to asbestos-induced pleural lesions with reference to the extent of the lesions and the initial parenchymal fibrosis

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Objectives The study focused on the effect of pleural lesions on the lung function of asbestos-exposed workers.

Methods A clinical check-up, chest radiography, high-resolution computed tomography (HRCT), and lung function testing were performed on 162 asbestos-exposed workers without any sign of parenchymal fibrosis on their chest radiographs. According to the HRCT scans, two subgroups were delineated, 97 subjects with pleural lesions and 65 referents without pleural lesions. Four categories of pleural lesions were specified according to the extent. Parenchymal changes, if identified on the HRCT scans, were recorded.

Results The radiographic sensitivity and specificity for pleural lesion detection when compared with that of HRCT were 64.9% and 98.5%, respectively. The HRCT scans showed parenchymal abnormalities in 46.3% of the participants, more frequently in those with pleural lesions (67.0% versus 15.4%, $P < 0.0001$). After the effect of parenchymal fibrosis was taken into account, pleural lesions were found to have a significant effect on the decrease in total lung capacity, forced vital capacity, and forced expiratory volume in 1 second, if being classified into category 2 or higher.

Conclusions Pleural lesions proved to have a negative effect on lung function, depending on their extent. The effect of the initial parenchymal fibrosis detectable in the HRCT scans only was also significant. A normal chest radiograph does not exclude the presence of pleural lesions or initial parenchymal fibrosis, with a possible negative effect on lung function.

Key terms asbestos exposure, asbestosis, pleural changes, pleural thickening, ventilation impairment.

In the Czech Republic, occupational exposure to asbestos used to be associated with the use of imported asbestos in the manufacture of friction and insulating materials, heat-resistant boards, textile products and cement products. At present, a single plant continues processing asbestos in limited quantities only. The other plants were either closed down or were converted to nonasbestos production. Up to 1995, compensation claims by occupationally exposed workers were considered justified if they presented with pulmonary asbestosis, pulmonary carcinoma associated with asbestosis, or

pleural or peritoneal mesothelioma. The compensation claims by occupationally exposed workers with pleural lesions started to be recognized in 1996. The amount of compensation is proportionate to the extent of the lung function impairment.

The aim of this study was to demonstrate possible changes in the lung function of asbestos-exposed workers with pleural lesions without concomitant signs of parenchymal fibrosis in the chest radiograph, more precisely, to determine how extensive pleural lesions should be to cause functional impairment and how

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initial parenchymal fibrosis still undetectable in a posteroanterior chest radiograph may be implicated in this impairment.

Participants and methods

Participants

At the beginning of 2000, three of the authors (JL, LR, BD) had 1199 former and current employees of asbestos-processing plants in their registers. Those with a confirmed history of occupational exposure to asbestos in their occupational records have been followed at regular intervals. Five hundred and ninety-one persons, mostly former asbestos-exposed workers, were followed in 2000. Chest radiographs showed normal findings for 312 of them (subgroup A), pleural changes alone for 180 of them (subgroup B), parenchymal changes alone [$\geq 0/1$ according to the International Labour Office (ILO)] for 53 of them (subgroup C), and both pleural and parenchymal changes for 46 of them (subgroup D). The following major criteria were used for their enrollment in the study: (i) documented occupational exposure to asbestos in their history, (ii) absence of parenchymal fibrosis on the posteroanterior chest radiograph (ILO $< 0/1$, ie, subgroups A and B). Anyone with a history of any disease likely to bias the chest radiograph and those with bronchial asthma were not enrolled. The participants were selected at random, separately from groups A and B. As many as 162 persons were investigated between 2000 and 2002 (98 from group A, 31.4%, and 64 from group B, 35.6%). Written informed consent was obtained from each person prior to the enrollment. The project was approved by the ethics commission according to the principles of the Declarations of Helsinki and Lisbon and World Medical Association Recommendations Guiding Physicians in Biomedical Research Involving Human Subjects. Each of the study subjects underwent all the examinations within 1 day. According to the HRCT scans, the participants were divided into two groups, (i) those with demonstrated pleural lesions (PL+) and (ii) those without lesions (PL-).

Assessment of occupational exposure to asbestos

The available results of the measurements of the particulate matter content in workplace air were used to estimate the asbestos exposure level. These measurements have been performed once a year since the 1950s. Until 1983, this parameter was exclusively the measured gravimetrically limit 2 mg/m³. Since 1979 the fraction of respirable particulate matter had also been measured on an irregular basis; its share in the total particulate matter content did not exceed 10–15%. Fiber counting

was started in 1984 (with a limit of 1 fiber/m³ and 0.6 fiber/m³ since 2000). The highest exposure levels were recorded up to 1975, the limits for the particulate matter being exceeded in most workplaces, four times the average in the most exposed places (ie, those manufacturing asbestos-textile products, heat-resistant asbestos boards, asbestos friction and insulating materials, and asbestos cement products). In extreme cases, the limit was even exceeded 15 to 500 times on a short-term basis. Medium exposure levels were typically recorded throughout the following period from 1976 to 1985, when the limits were exceeded two to three times the average in the most exposed workplaces while the mean levels either ranged within or slightly exceeded the norm in the other workplaces (asbestos cylinder head gaskets, asbestos paper products). The third period from 1986 to 1995 was characterized by a slump in the asbestos-processing industry. The limits were exceeded about two times in asbestos textile production and asbestos board production. Since 1996, asbestos-free production has prevailed, and the problematic plants have been closed down. The only plant still processing asbestos in the manufacture of asbestos boards meets the limits established for airborne particulate matter.

Case history and clinical examination

According to the occupational history data, the duration of exposure to asbestos and the latency period between the first occupational contact with asbestos and the date of examination were recorded. The asbestos exposure level was considered according to the occupational exposure duration. Dyspnea was assessed according to the criteria of the Medical Research Council (1), whereas chronic bronchitis was defined according to Dorland's Medical Dictionary (2). Reported angina pectoris and myocardial infarction were considered ischemic heart disease. Body weight and height were recorded as the body mass index (BMI). According to their smoking habits, the participants were categorized as nonsmokers (ie, those who had never smoked), ex-smokers (ie, those who had quit smoking at least 6 months prior to the examination), and current smokers (ie, the remaining persons).

Lung function tests

The lung function tests were performed in a sitting position with the use of a V-max Autobox 6200 DL body plethysmograph manufactured by Sensor-Medics (Yorba Linda, CA, USA). The total lung capacity (TLC), forced vital capacity (FVC), forced expiratory volume in 1 second (FEV₁), forced expiratory flow at 75% of the FVC (FEF_{75%}), forced expiratory flow at 25–75% of the FVC (FEF_{25–75%}), and diffusing capacity of

carbon monoxide (DLCO) were recorded and expressed in the percentage of the predicted values. The findings below either 80% (for TLC, FVC, FEV₁ and DLCO) or 70% (for FEF₇₅, FEF₂₅₋₇₅) of the predicted values were considered pathological. The used investigation protocol and predicted values comply with the requirements of the European Respiratory Society and the European Coal and Steel Community (3).

Chest radiographs

Standard posteroanterior chest radiographs (43×35 cm) were taken with a Siemens Multix UPH unit (Erlangen, Germany) at 60–65–70 kilovolts (exceptionally 73 kilovolts) according to the constitution of the patient, the computed milliamperes seconds values ranging from 2 to 15. They were evaluated independently by a radiologist and a team of three physicians experienced in the diagnosis of radiographic changes in asbestos-exposed persons on the basis of the standards of the 1980 ILO International Classification of Pneumoconioses (4). Pleural thickening (with or without obliteration of the costophrenic angle), craniocaudally exceeding one-fourth of the chest wall laterally was considered as large pleural lesions; less extensive pleural changes were classified as small pleural lesions.

Chest high-resolution computed tomography

The high-resolution computer tomography (HRCT) scans were taken with an Elscint Twin II CT unit (Haifa, Israel) at 140 kilovolts and 165 milliamperes seconds, with a matrix of 768×768 pixels. Sections 1-millimeter thick were obtained at 10-millimeter intervals at the end-inspiratory volume in the supine position, and then an additional three to four prone scans were taken (5, 6). The computed tomographic (CT) images were reconstructed with a high spatial frequency algorithm. A standardized window image of 1500 Hounsfield units (window width) and a level of –600 Hounsfield units (window center) was used for parenchymal visualization, and a window and a level of 2200 and –500 Hounsfield units, respectively, were used for the pleural evaluation.

Evaluation of the pleural lesions

The pleural lesions were divided into categories 0–4 according to their number, craniocaudal extent, width, and thickness (0 = not demonstrated, 1 = small, 2 = medium, 3 = large, 4 = very large). This categorization was based on the criteria of Lynche et al (7). If more than one plaque was found, the largest one was decisive for the categorization. These criteria are summarized in table 1.

Evaluation of parenchymal changes

Thickened intralobular and interlobular septal lines, subpleural curvilinear lines, parenchymal bands, ground-glass opacities, and honeycombing were recorded. The findings were classified as asbestosis or suspected asbestosis according to Algranti et al (8). An asbestosis diagnosis was made only when there were HRCT images compatible with definite signs of fibrosis (multiple bands or honeycombing) or in the presence of at least three of the described patterns of images, in a sequence of two or more scan sections, bilaterally and predominantly basal. The presence of one or more, less extensive abnormality patterns in the absence of definite signs of fibrosis did not lead to the diagnosis of asbestosis—in such cases the label “suspect” was used. The changes were considered indicative of asbestosis or suspected asbestosis only if identified in the independent portions of the lung. The changes seen only in the dependent areas of the lung were disregarded.

Statistical analysis

Simple summary characteristics (averages, standard deviations, proportions of various patient subgroups) were computed from the data available for the study group. Elementary analysis also included testing for differences between (i) group means (using two-sample

Table 1. Categorization of the pleural lesions found in the asbestos-exposed workers according to high-resolution computed tomography.

Craniocaudal extent (mm)	Pleural lesions			Category
	Number	Width (mm)	Thickness (mm)	
<80	1–3	≤30	1–2	1
<80	1–3	≤30	2–3	1
<80	1–3	≤30	>3	1
<80	1–3	31–50	1–2	1
<80	1–3	31–50	2–3	2
<80	1–3	31–50	>3	2
<80	1–3	>50	1–2	2
<80	1–3	>50	2–3	2
<80	1–3	>50	>3	3
<80	>3	≤30	1–2	1
<80	>3	≤30	2–3	2
<80	>3	≤30	>3	2
<80	>3	31–50	1–2	2
<80	>3	31–50	2–3	2
<80	>3	31–50	>3	3
<80	>3	>50	1–2	3
<80	>3	>50	2–3	3
<80	>3	>50	>3	3
≥80	≥1	≤50	.	3
≥80	≥1	>50	.	4

two-sided t-tests) and (ii) subgroup proportions (via a chi-square test in an appropriate contingency table).

A more-detailed analysis of the simultaneous effects of fibrosis and pleural lesions on the measured pulmonary functions (TLC, FVC, FEV₁, FEF₂₅₋₇₅, FEF₇₅, DLCO) was performed with a two-factor analysis of variance (ANOVA) with interaction. The strength of this approach lies in the fact that the effects of both factors (ie, fibrosis and pleural lesions) can be tested at once. Moreover, it is possible to test whether the effects of these factors are additive or interactional in nature. The test results are summarized as P-values in the tables.

If the interaction was not significant (hence, it was possible to separate the net effect of the pleural lesions), differences among the pleural lesion categories (0, 1, 2, 3, 4) were tested. Formally, we used orthogonal linear contrasts of the Helmert type [ie, the following comparisons: (i) 1 versus 0, (ii) 2 versus the average of 0 and 1, (iii) 3 versus the average of 0, 1 and 2, and (iv) 4 versus the average of 0, 1, 2 and 3].

In addition to the basic two-factor model, another larger ANOVA was used to test for simultaneous fibrosis and pleural lesion effects after adjustment for several selected potential confounders. The aim was to evaluate how stable the relationships between the pulmonary functions, fibrosis, and pleural lesions were with respect to selected potential confounders (smoking habits, chronic bronchitis, body mass index, ischemic heart disease).

Results

The characteristics of the two subgroups with or without pleural lesions on the HRCT scan are summarized in table 2.

The sensitivity and specificity of the conventional chest radiograph in detecting pleural lesions in the whole study group in comparison with that of the HRCT scan were 64.9% and 98.5%, respectively. The chest radiographs did not reveal 77% of the pleural lesions of category 1, 35% of category 2, 3.4% of category 3, and 11.8% of category 4. The conventional chest radiograph proved inadequate to detect initial parenchymal fibrosis. Although radiographically inapparent (ILO 0/0), parenchymal changes were detectable in the HRCT scans of 46.3% of the participants. Asbestosis and suspected asbestosis was found in 17 (10.5%) and 58 (35.8%) persons, respectively. Parenchymal abnormalities were significantly more frequent in the subjects with pleural lesions than in those without pleural lesions (67.0% versus 15.4%, $P < 0.0001$). No association between parenchymal changes and smoking habits was found ($P = 0.37$ for thickened interlobular septal lines, $P = 0.18$ for subpleural lines, $P = 0.14$ for parenchymal bands, $P = 0.35$ for ground glass opacities, $P = 0.94$ for honeycombing).

The mean values of the functional parameters studied (as the percentage of the predicted value) were either normal or only slightly lower than normal for both groups of participants; nevertheless, except for DLCO, they were all significantly lower for those with pleural lesions (table 3).

In light of the high proportion of parenchymal abnormalities on the HRCT scans, their possible effect on lung function could not be disregarded. An analysis of the possible concomitant effects of fibrosis and pleural lesions confirmed the assumption that even negligible parenchymal fibrosis may have a significant effect on lung function, as recorded for all the parameters studied. After adjustment for the effect of fibrosis, no effect was found for pleural lesions with respect to the following parameters: FEF₂₅₋₇₅, FEF₇₅, and DLCO. Nevertheless, the effect of pleural lesions was significant for TLC, FEV₁, and FVC even after adjustment for effects

Table 2. Characteristics of the 162 asbestos-exposed workers grouped according to the presence of pleural lesions in high-resolution computed tomography. (N = total number, MRC = Medical Research Council, BMI = body mass index)

Participants	Characteristic																			
	N	%	Male:fe- male ratio	Age (years)	Duration of occupational exposure to asbestos (years)		Latency period (years)		Dyspnea ≥2 on MRC scale (%)	Chronic bronchitis (%)	Non- smokers (%)	Ex- smokers (%)	Current- smokers (%)	Cigarette consump- tion (pack years)		BMI (kg/m ²)		Ische- mic heart disease (%)		
					Mean	SD	Mean	SD						Mean	SD	Mean	SD		Mean	SD
With pleural lesions	97	59.9	56:41	61.5	9.2	23.9	9.9	38.0	10.8	64.9	27.8	48.4	36.1	15.5	21.4	17.7	29.3	5.7	17.5	
Without pleu- ral lesions	65	40.1	32:33	53.3	10.1	18.8	10.9	28.4	11.0	35.4	20.0	47.7	23.1	27.2	19.8	14.5	28.7	5.6	6.2	
P-value	•		•	<0.001		0.002		<0.001		0.0002	0.26	0.92	•	•	0.68	0.52	0.04			

Table 3. Functional parameters of the percentages of the predicted values and the percentages of the pathological findings for the 162 asbestos-exposed workers of both subgroups (PL+ = subgroup with pleural lesions, PL- = subgroup without pleural lesions, TLC = total lung capacity, FVC = forced vital capacity, FEV₁ = forced expiratory volume in 1 second, FEV₇₅ = forced expiratory flow at 75% of FVC, FEV₂₅₋₇₅ = forced expiratory flow for 25–75% of the FVC, DLCO = diffusing capacity of carbon monoxide)

Function- al para- meter	Predicted value (%)					Pathological findings (%)		
	PL+		PL-		P-value	PL+	PL-	P-value
	Mean	SD	Mean	SD				
TLC	95.1	12.4	102.6	13.4	0.0003	10.3	1.5	0.030
FVC	96.7	16.9	106.3	14.6	0.0003	15.5	3.1	0.012
FEV ₁	92.3	17.2	101.7	15.1	0.0004	24.7	10.8	0.027
FEV ₂₅₋₇₅	70.7	24.5	82.6	27.1	0.004	50.5	32.3	0.022
FEV ₇₅	57.7	29.7	69.8	29.6	0.012	72.2	53.8	0.017
DLCO	87.6	16.7	90.5	14.8	0.26	33.0	24.6	0.23

Table 4. Effects of fibrosis and pleural lesions on the lung function of the 162 asbestos-exposed workers. (TLC = total lung capacity, FVC = forced vital capacity, FEV₁ = forced expiratory volume in 1 second, FEV_{75%} = forced expiratory flow at 75% of the FVC level, FEV_{25-75%} = forced expiratory flow at 25–75% of the FVC, DLCO = diffusing capacity of carbon monoxide)

Factor	P-value					
	TLC	FVC	FEV ₁	FEV ₂₅₋₇₅	FEV ₇₅	DLCO
Fibrosis (F) ^a	0.0124	<0.0001	<0.0001	<0.0001	0.0329	0.0354
Pleural lesions (PL) ^b	0.0005	0.0006	0.0030	0.0710	0.3778	0.2550
F×PL ^c	0.5371	0.0395	0.1026	0.6820	0.7310	0.9260

^a Corresponds to the test of the effect of parenchymal fibrosis upon a particular lung function parameter.

^b Corresponds to the test of the effect of pleural lesions on a particular lung function parameter after cleaning for the effect of fibrosis.

^c Corresponds to the fibrosis–pleural lesion interaction so that it tests the differences in the lung function averages for various levels of pleural lesions when the fibrosis status changes (from absent to present and vice versa).

Table 5. Effects of fibrosis and pleural lesions on the lung function of 162 asbestos-exposed workers after adjustment for selected confounders (smoking habits, chronic bronchitis, body mass index and ischemic heart disease) in a multifactorial analysis of variance. (F×PL = fibrosis and pleural lesion interaction, TLC = total lung capacity, FVC = forced vital capacity, FEV₁ = forced expiratory volume in 1 second, FEV_{75%} = forced expiratory flow at 75% of the FVC, FEV_{25-75%} = forced expiratory flow for 25–75% of the FVC, DLCO = diffusing capacity of carbon monoxide)

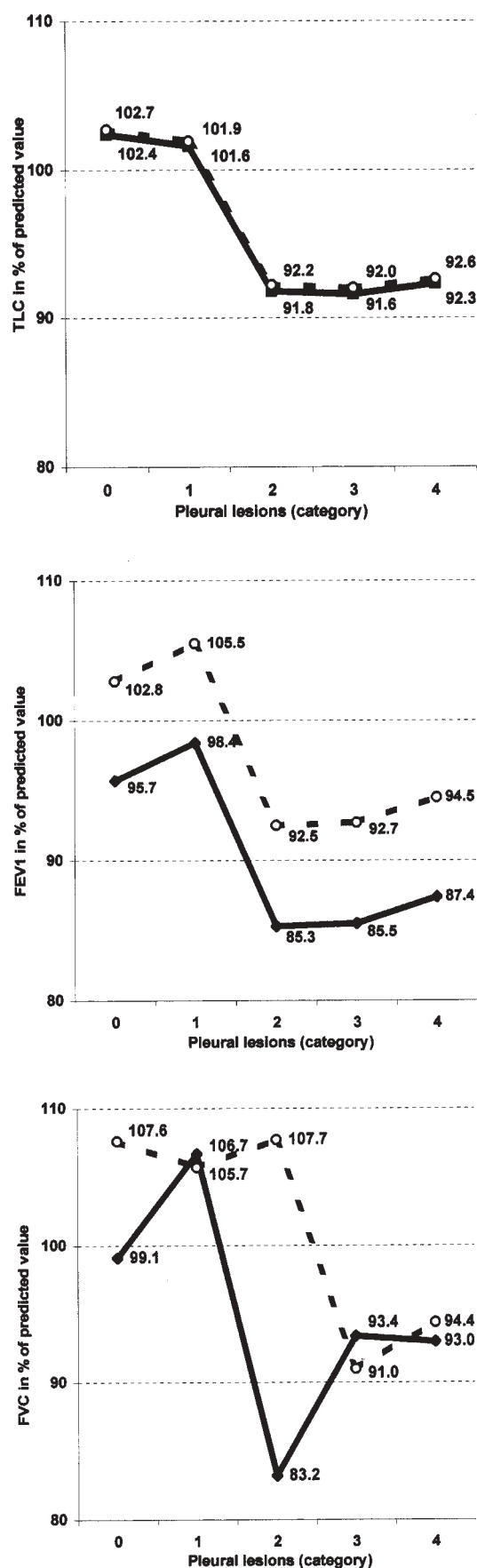
Factor	P-value					
	TLC	FVC	FEV ₁	FEV ₂₅₋₇₅	FEV ₇₅	DLCO
Smoking	0.2664	0.3579	0.0783	0.0725	0.0337	0.0583
Chronic bronchitis	0.1103	0.0440	0.0484	0.0820	0.1853	0.0174
Body mass index	0.0153	0.0287	0.0166	0.0318	0.1292	0.7075
Ischemic heart disease	0.1079	0.0750	0.0587	0.0219	0.8529	0.9260
Fibrosis	0.0612	0.0003	<0.0001	0.0001	0.0384	0.0598
Pleural lesions	0.0040	0.0019	0.0057	0.1473	0.3248	0.2959
F×PL	0.6400	0.0580	0.1498	0.6293	0.7617	0.8880

of fibrosis. The results of the analysis are given in table 4. These three parameters were analyzed in detail to determine how the extent of pleural lesions is implicated in lung function impairment.

Since pleural lesions and parenchymal fibrosis showed nonsignificant interaction for TLC and FEV₁ (see table 4), their effects could be clearly separated. A detailed analysis of the extent-dependent effect of pleural lesions (categories 0–4) showed the following: the TLC and FEV₁ values did not differ among the participants with pleural lesions in category 1 and those with pleural lesions in category 0 (1 versus 0, P=0.5142 and P=0.6566, respectively). A significant break point with a decrease in both the TLC and FEV₁ values was recorded for the participants with pleural lesions of category 2 (2 versus average 0 and 1, P=0.0006 and P=0.0014, respectively). Decreased values were also recorded for these parameters for higher categories of pleural lesions (3 versus average 0, 1 and 2: P=0.0128, P=0.0269, respectively; 4 versus average 0, 1, 2 and 3: P=0.1602, P=0.3381, respectively).

The situation was more complicated for FVC since the concomitant effects of fibrosis and pleural lesions were interactional in nature (see table 4). The participants with concomitant parenchymal fibrosis and pleural lesions showed a significant break point and a decrease in FVC with pleural lesions of category 2, the same being true for TLC and FEV₁. The participants with pleural lesions without concomitant parenchymal fibrosis had significantly decreased FVC values in the presence of pleural lesions of category 3. A diagrammatic representation of the mean TLC, FEV₁, and FVC values, depending on the extent of pleural lesions separately for the presence and absence of parenchymal fibrosis, is given in figure 1 (on page 393). Of the 66 cases of pleural lesions of categories 2–4, 15.2% remained undetected in the chest radiographs. Unrecognized pleural lesions of categories 2–4 accounted for 10.2% of all the negative chest radiographs.

In addition to the two-factor ANOVA, a larger ANOVA was used to test for the possible concomitant effects of fibrosis and pleural lesions after adjustment for selected potential confounders, namely, smoking habits, chronic bronchitis, body mass index, and ischemic heart disease. These factors may have biased the lung function results regardless of the effect of asbestos. The larger ANOVA model enabled testing of how stable the found relationships are. These relationships are summarized in table 5. A comparison of the results obtained prior to and after the adjustment for the confounders indicates that the patterns of significance and interaction between pleural lesions and fibrosis remained almost unchanged even after adjustment for the confounders, although the actual significance values (P-values) differed slightly.



Discussion

Pleural lesions are the most frequent radiographic finding among persons exposed to asbestos, and their incidence is significantly higher among occupationally exposed persons than among the general population (9). In the Czech Republic, the prevalence of pleural lesions has not been monitored. The only study (10) available to date was carried out in the 1970s; it documented a 16 times higher prevalence of pleural calcifications in chest radiographs of workers occupationally exposed to asbestos than for the general population over 40 years of age.

In our study, the diagnosis of pleural lesions was based on HRCT findings. Owing to its high sensitivity (11–13), HRCT has been generally considered the gold standard for assessing benign pleural changes. The pleural lesion categorization used by us can also be commonly used in practice for outpatients. It is based on a detailed description of pathology by a radiologist experienced in assessing asbestos-induced changes, the author's experience, and the diagnostic tools available, and literature data (7). We could not use the more accurate methods (eg, computer-derived volumetric measurements or a technique using cutout tracing paper) described in the literature (14–16) for categorizing pleural lesions in this study for technical reasons. Since the HRCT classification of pleural lesions has not yet been standardized, comparability of the results may pose problems.

Age, occupational asbestos exposure duration, and latency period were significantly higher for the participants with pleural lesions. Algranti et al (8) have shown that latency period is a significant predictor of pleural lesions (and parenchymal fibrosis), more significant than exposure duration. The effect of latency period on the incidence of pleural lesions was beyond the scope of our study, but a partial statistical analysis showed that the latency period was a significant factor influencing the lung function of the participants with pleural lesions when they were compared with those without pleural lesions.

Relationships between lung function and pleural lesions are primarily considered in light of the extent of the pleural lesions. The lung function parameters most frequently studied with reference to asbestos-induced pleural lesions are TLC, FVC, and FEV₁. In agreement with our conclusions, an effect of asbestos-induced

Figure 1. Means of total lung capacity (TLC), forced expiratory volume in 1 second (FEV₁), and forced vital capacity (FVC) as the percentage of the predicted value according to the extent of the pleural lesions, separately for the presence or absence of concomitant parenchymal fibrosis. (category of pleural lesions: 0=undemonstrated, 1=small, 2=medium-size, 3=large, 4=very large; solid line=fibrosis present; broken line=fibrosis absent)

pleural lesions on these parameters has been reported by many authors (8, 15–20). Nevertheless, other authors (11, 13, 21) have not found a poorer lung function in the presence of pleural lesions.

This discordance can be explained in several ways. One possible explanation is a shorter latency period. The participants in the study by Van Cleemput et al (11) had a similar exposure duration (25 years versus 25.6 years in our study) but were markedly younger (43 years versus 58 years in our study) and thus must have had a shorter latency period. Another possible explanation may be a smaller extent of pleural lesions. The participants in the study of Neri et al (21) showed signs of pleural lesions in their HRCT scans but not in their chest radiographs. The authors did not give the pleural lesion categorization in more detail. Neither did Soulat et al (13) find any relationship between pleural lesions and lung function. Nevertheless, attention was not paid to the extent of pleural lesions. It can be understood from the text that circumscribed plaques only without diffuse pleural thickening were observed. Possibly, pleural lesions of limited extent predominated, still not showing any effect on lung function. The study of Schwartz et al (17), describing a significant decrease in TLC, FVC, and FEV₁ in persons with diffuse pleural thickening only, is consistent with this assumption. Significant relationships in this regard were not found for persons with circumscribed plaques, even if they showed lower FEV₁ and FVC values than those without pleural lesions. Moreover, their study group was too small and therefore the statistical analysis may have been restricted in revealing statistically significant differences. Nevertheless, in a previous extensive study (22) based on chest radiographs of as many as 1211 exposed persons, Schwartz et al showed that even circumscribed plaques are independently significantly related to a decrease in FVC.

In contrast to Schwartz et al (17) and Kee et al (18), we did not find any significant effect of pleural lesions on DLCO. DLCO impairment was associated with parenchymal fibrosis. Nor did we find any significant effect of pleural lesions on FEF_{25–75} or FEF₇₅, as reported by Fischbein et al (20), Kilburn & Warshaw (23), and Hillerdal et al (24). Nevertheless, we showed that parenchymal fibrosis has a significant effect on these parameters. These authors assessed lung function with reference to pleural lesions diagnosed in chest radiographs. Therefore, it cannot be excluded that the decrease in FEF_{25–75} and FEF₇₅ may have been caused by initial parenchymal fibrosis still undetectable in a simple chest radiograph. On the other hand, the fact that no significant relationship was found between FEF_{25–75} or FEF₇₅ and pleural lesions could be attributed to the high residual variation of these parameters that, in turn, might have influenced the outcome of the statistical analysis.

Our study confirmed the hypothesis that pleural lesions have a significant effect on lung function, depending on their extent. Furthermore, only a significant effect of initial parenchymal fibrosis detectable in HRCT scans was found for lung function impairment. A normal chest radiograph does not exclude the presence of pleural lesions or initial parenchymal fibrosis, which may have already had a negative effect on lung function.

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