LUNG FUNCTIONS AND CHEST RADIOGRAPHS IN SHIPYARD WORKERS EXPOSED TO ASBESTOS

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Lung functions and diffusing capacity for carbon monoxide were examined in 209 shipyard workers occupationally exposed to asbestos, among whom there was an equal number of smokers and non-smokers. The workers were classified into groups according to the lung fibrosis profusion and the length and width of the pleural plaques in accordance with the 1980 ILO International Classifications of Pneumoconioses. With regard to the profusion of the parenchyma, workers classified in category 2 (subcategories 2/1 and 2/2) had significantly lower FVC and FEV₁ values than the workers in categories 0 (P<0.01) and 1 (P<0.05). With an increase in the category of profusion there was a trend towards a fall in mean MEF₂₅, MEF₅₀ and MEF₇₅ values, but it was not significant. Diffusing capacity for carbon monoxide was identical for all categories. There were no significant differences between the workers without pleural plaques and those with visible plaques in respect to lung function values. With an increase in the length and width of the plaques FVC, FEV₁, MEF₂₅ and MEF₇₅ values tended to become lower. The results demonstrate that the changes detected on the radiographs of the lungs and pleura are the early indicators of possible asbestos disease in shipyard workers.

Key terms: asbestos exposure, occupational exposure, parenchymal fibrosis, pleural plaque, ventilatory lung function

Asbestosis of the lung parenchyma or pleura occurs as a reaction of the body to inhaled, accumulated and deposited asbestos particles. A sufficiently sensitive, non-invasive method for detecting the initial changes due to asbestosis has not yet been devised. A reduction in certain lung functions has been reported for persons with a history of long-term asbestos exposure but without radiographically visible asbestosis of the lung parenchyma and/or pleura. The functions most frequently involved are vital capacity (1-4), forced expiratory volume in the first second (5-6), diffusing capacity for carbon monoxide (7-8) and more recently, small airways flow limitation (9-12). Some authors claim that only when fibrosis of the lung parenchyma and/or pleura is clearly visible is there a fall in the above parameters (13-26).
This investigation aims at establishing the value of certain lung functions as an early indicator of changes in ventilatory function and/or diffusion function of the lungs in shipyard workers who have been occasionally exposed to low concentrations of asbestos.

**SUBJECTS AND METHODS**

The subjects in the investigation were 350 male workers from three shipyards in Croatia, aged 48.8±12.2 years, range 26-65 years, who were employed as pipefitters, carpenters, joiners, electricians and ship insulation workers. Their workplaces involved occasional exposure to asbestos of an average duration of 22.8±10.1 years. Asbestosis was suspected in all of them on the basis of occupational history and/or chest radiograph taken in a local hospital for lung diseases.

Respiratory data were recorded by means of the questionnaire of the Committee for Chronic Bronchitis of the British Medical Research Council (27). Data on other diseases, including inherited, were also taken. Each worker was examined by an occupational health specialist and if necessary by other specialists as well. Special attention was paid to detecting changes in the locomotor system and thorax deformities that could affect the ventilatory function of the lungs. The workers were classified into non-smokers, light, moderate, and heavy smokers according to the number of cigarettes smoked, following the recommendations of Brinkman and Coates (28). Fifty-one workers were excluded from the investigation because of confirmed acute respiratory, systemic and other diseases that could have produced a direct or an indirect effect on respiratory function.

For a total of 299 subjects posteroanterior and oblique chest radiographs were taken once again and analysed in accordance with the International Classification of Pneumonconioses (29). Analysis was done by comparison with a set of films of the International Labour Organization and was carried out independently by two occupational health specialists and a radiologist. The result on which at least two experts had agreed was taken to be final. Changes in the lung parenchyma were classified with regard to the form and size of the shadow, the extent of profusion and the number of lung segments where the shadows were visible. Visible thickenings of the pleura were also classified by shape, length and width.

Lung function tests were performed on a VICATIST 5 (MINHARDT) apparatus, The Netherlands. Diffusing capacity for carbon monoxide (DLCO) was measured on a Transfer test apparatus, P.K. Morgan Ltd., U.S.A., by the single breath method after Quanjer (30). Before the test body mass and height of each worker were measured and age was recorded. After having received detailed instructions on how to perform the test the workers completed three quick, forced expirations in a standing position (with a nose peg). The best value was recorded. Forced vital capacity (FVC) was measured as a static, and forced expiratory volume in the first second (FEV1) as a dynamic lung function test. Readings of the maximum expiratory flow were taken at 25% FVC (MEF25), 50% FVC (MEF50) and 75% FVC (MEF75). The Tiffeneau index (FEV1/FVC:100) was calculated for each worker. Impairment of respiratory function in relation to predicted values was calculated from the difference between the measured and predicted normal values. The values according to Quanjer (30) were taken as normal. A reduction in FVC, FEV1, Tiffeneau index and DLCO values of 20% and higher than predicted was considered to be clinically significant. The MEF25, MEF50 and MEF75 values 40% lower than expected were taken to be pathological. For statistical analysis Student's t-test and Mann-Whitney test were used.
RESULTS

Table 1 gives the chest X-ray changes in relation to the workers’ age, length of exposure and time from the beginning of asbestos exposure. The plaque characteristics are presented in Table 2. All plaques were bilateral. No worker had diffuse pleural thickening, pleural effusion, mesothelioma of the pleura or lung cancer. The degrees of profusion of lung fibrosis can be seen in Table 3.

Table 1 Characteristics of the examined groups of asbestos-exposed shipyard workers

<table>
<thead>
<tr>
<th>Chest X-ray</th>
<th>n</th>
<th>%</th>
<th>Age* (yrs)</th>
<th>Duration of* exposure (yrs)</th>
<th>Time elapsed* from beginning of exposure (yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without visible changes</td>
<td>101</td>
<td>33.8</td>
<td>47.1±5.1</td>
<td>17.3±11.1</td>
<td>22.4±10.8</td>
</tr>
<tr>
<td>Pleural plaques only</td>
<td>68</td>
<td>22.7</td>
<td>45.1±5.2</td>
<td>71.5±14.1</td>
<td>26.6±17.2</td>
</tr>
<tr>
<td>Parenchymal fibrosis only**</td>
<td>42</td>
<td>14.0</td>
<td>48.1±9.2</td>
<td>22.7±11.7</td>
<td>29.3±9.6</td>
</tr>
<tr>
<td>Parenchymal fibrosis and pleural plaques*</td>
<td>88</td>
<td>29.4</td>
<td>52.1±13.1</td>
<td>30.1±9.3</td>
<td>34.8±11.1</td>
</tr>
<tr>
<td>Pleura – total</td>
<td>156</td>
<td>52.2</td>
<td>49.0±11.1</td>
<td>26.2±10.1</td>
<td>31.2±3.6</td>
</tr>
<tr>
<td>Parenchymal fibrosis total**</td>
<td>130</td>
<td>43.5</td>
<td>50.8±13.3</td>
<td>77.6±11.3</td>
<td>33.0±13.2</td>
</tr>
<tr>
<td>Total no. of subjects</td>
<td>299</td>
<td>100</td>
<td>48.8±12.2</td>
<td>22.8±10.1</td>
<td>28.0±14.2</td>
</tr>
</tbody>
</table>

* Mean value ± standard deviation
** Parenchymal fibrosis Category 1 and higher

Table 2 Workers classified according to the length and width of plaques (n=156)

<table>
<thead>
<tr>
<th>Width of plaque</th>
<th>Length of plaque</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Width</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>a</td>
<td>119</td>
<td>23</td>
</tr>
<tr>
<td>b</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>TOTAL (%)</td>
<td>125 (80.1)</td>
<td>30 (19.2)</td>
</tr>
</tbody>
</table>

Table 3 Workers classified according to subcategory and category of lung parenchymal profusion (n=299)

<table>
<thead>
<tr>
<th>Subcategory of profusion (ILO)</th>
<th>No. of subjects</th>
<th>%</th>
<th>Category of profusion (ILO)</th>
<th>No. of subjects</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>0/0</td>
<td>53</td>
<td>17.2</td>
<td>0</td>
<td>169</td>
<td>56.2</td>
</tr>
<tr>
<td>0/1</td>
<td>116</td>
<td>38.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/0</td>
<td>86</td>
<td>28.8</td>
<td>1</td>
<td>118</td>
<td>39.8</td>
</tr>
<tr>
<td>1/1</td>
<td>29</td>
<td>9.7</td>
<td>2</td>
<td>12</td>
<td>4.0</td>
</tr>
</tbody>
</table>
Table 4 Lung function* according to category of parenchymal fibrosis

<table>
<thead>
<tr>
<th>Category of profusion (ILO)</th>
<th>No. of subjects</th>
<th>FVC</th>
<th>FEV₁</th>
<th>% FEV₁/FVC</th>
<th>MEF₂₅</th>
<th>MEF₅₀</th>
<th>MEF₇₅</th>
<th>DLCO</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>169</td>
<td>85.6±14.6⁺</td>
<td>84.5±16.6⁺</td>
<td>90.2</td>
<td>76.0±14.2</td>
<td>82.2±31.1</td>
<td>85.3±25.2</td>
<td>90.2±16.8</td>
</tr>
<tr>
<td>1</td>
<td>113</td>
<td>86.2±14.8⁺</td>
<td>82.0±17.7⁺</td>
<td>89.2</td>
<td>71.8±32.5</td>
<td>78.9±31.6</td>
<td>82.1±26.4</td>
<td>93.6±14.9</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>74.1±21.6⁺</td>
<td>73.5±18.2⁺</td>
<td>83.1</td>
<td>64.8±28.3</td>
<td>76.5±6.0</td>
<td>80.3±14.2</td>
<td>98.7±3.0</td>
</tr>
</tbody>
</table>

Lung function parameters are expressed in percentages of predicted values according to Quanjer, 1983 (30)
* : P<0.05
** : P<0.01
* Mean values ± standard deviation

Table 5 Lung function* in workers with fibrosis of the parenchyma category 0 classified according to the width of pleural plaques

<table>
<thead>
<tr>
<th>Pleural plaques</th>
<th>n (%)</th>
<th>FVC</th>
<th>FEV₁</th>
<th>% FEV₁/FVC</th>
<th>MEF₂₅</th>
<th>MEF₅₀</th>
<th>MEF₇₅</th>
<th>DLCC</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>101 (59.7)</td>
<td>90.9±21.2</td>
<td>86.0±7.2</td>
<td>94.6±17.6</td>
<td>75.3±28.2</td>
<td>81.8±22.1</td>
<td>83.2±17.5</td>
<td>90.1±16.2</td>
</tr>
<tr>
<td>A</td>
<td>67 (35.6)</td>
<td>87.5±18.4</td>
<td>86.5±4.4</td>
<td>93.4±11.2</td>
<td>71.2±13.2</td>
<td>82.3±17.3</td>
<td>80.1±11.2</td>
<td>91.2±11.0</td>
</tr>
<tr>
<td>C</td>
<td>1 (0.5)</td>
<td>84.1±0</td>
<td>82.1±0</td>
<td>97.3±0</td>
<td>72.0±0</td>
<td>78.2±0</td>
<td>73.2±0</td>
<td>92.0±0</td>
</tr>
</tbody>
</table>

* Mean value ± standard deviation
** % of subjects from the total number with parenchymal fibrosis category 0
The numbers of smokers and non-smokers were equal, as were those of light, moderate and heavy smokers.

The lung function values and diffusing capacity for carbon monoxide did not significantly differ between categories 0 and 1, nor was their reduction significant clinically. Significantly reduced FVC and FEV\(_1\) values were found in workers classified as category 2 in relation to categories 0 (P<0.01) and 1 (P<0.05). The reduction was also significant clinically. The MEF\(_25\), MEF\(_50\) and MEF\(_75\) showed a tendency to diminish with a higher category of parenchymal fibrosis, although without statistical significance between the categories. Reduced lung function values were of no clinical significance in any of the groups. Diffusing capacity for carbon monoxide did not vary between the investigated categories for parenchymal fibrosis neither was there a trend towards a reduction (Table 4).

In workers with parenchymal fibrosis category 0 and visible pleural plaques the FVC, FEV\(_1\), MEF\(_25\), and MEF\(_75\) values were usually lower than in those without plaques. A slight trend towards a reduction in FVC and FEV\(_1\) with an increase in the length and/or width of the pleural plaque when the parenchymal fibrosis was of the same degree can be seen in Tables 5 and 6.

### Table 6 Lung function in workers with parenchymal fibrosis category 0 classified according to the length of the pleural plaques

<table>
<thead>
<tr>
<th>Pleural plaques n (%)**</th>
<th>FVC</th>
<th>FEV(_1)</th>
<th>FEV(_1)/FVC</th>
<th>MEF(_25)</th>
<th>MEF(_50)</th>
<th>MEF(_75)</th>
<th>DL(_{CO})</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 101 (59.7)</td>
<td>84.3±21.2</td>
<td>94.6±17.6</td>
<td>75.3±28.2</td>
<td>81.8±22.1</td>
<td>83.4±17.6</td>
<td>90.1±16.2</td>
<td></td>
</tr>
<tr>
<td>1 35 (42.5)</td>
<td>89.8±17.4</td>
<td>93.6±11.2</td>
<td>68.2±17.2</td>
<td>78.3±19.2</td>
<td>81.3±17.4</td>
<td>91.3±57.0</td>
<td></td>
</tr>
<tr>
<td>2 13 (7.7)</td>
<td>87.1±12.2</td>
<td>92.0±10.2</td>
<td>97.2±11.2</td>
<td>77.2±11.2</td>
<td>78.3±14.5</td>
<td>91.6±13.3</td>
<td></td>
</tr>
</tbody>
</table>

* Mean value ± standard deviation
** % of subjects from the total number with parenchymal fibrosis category 0

### DISCUSSION

The results of this work are only partly in agreement with the results of other authors regarding the effect of asbestos on lung function. We have not established a correlation between a fall in the lung functions and the length of exposure to asbestos, although such a correlation has been reported by other authors (4, 7, 11–14, 16, 17, 31, 32).

A statistically significant fall was seen in both FVC and FEV\(_1\) only in those workers whose grade of fibrosis was classified as category 2 (subcategories 2/1 and 2/2) as opposed to those from categories 0 and 1 (subcategories 0/0–1/2). The fall in FVC and FEV\(_1\) only in cases of clearly visible fibrosis is a sign of disease progression and cannot be a test for early detection of asbestosis or a parameter depending on the degree of exposure (4, 14, 31–34).

Our subjects showed a trend towards a reduced flow through the small airways following increase in the category of parenchymal fibrosis, although there were no significant differences between the examined groups. Begin and co-workers (3, 8) claim that the reduced flow through the small airways unaccompanied by visible X-ray changes is a
result of asbestos-induced alveolitis that is later histopathologically confirmed. This may account for the controversial values of these parameters reported in the literature.

The value of diffusion capacity for carbon monoxide as a sensitive test for the diagnosis of asbestosis is also controversial. Harber and Smithman consider this test to be sensitive but non-specific (16). Other authors are of the same opinion (7, 8, 35, 37). Becklake and co-workers (4) on the other hand, report a reduction in this parameter only in subjects exposed to asbestos with parenchymal fibrosis, subcategory 2/1 and higher. The values of diffusion capacity for carbon monoxide did not differ among the groups of our workers, nor did the length of exposure to asbestos have any effect on them. The question is whether the asbestos-induced inflammation helps diffusion in certain lung segments. This could be the reason why this test tends to yield variable results with different authors.

For subjects with visible pleural plaques a significant lowering of FVC and FEV1 has been reported by several authors (9-12, 25, 34, 38, 39). According to Lumley those subjects have significantly reduced diffusing capacity for carbon monoxide (40). Other authors (4, 6, 12, 13) have failed to find a significant variation in the given parameters even in subjects with visible pleural plaques and simultaneous parenchymal fibrosis. Our results show only a slight trend towards a reduction of FVC, FEV1, and MEF25 with an increase in the length and width of the pleural plaque; a statistically significant reduction of FVC and FEV1 depended only on the degree of lung fibrosis progression.

This work leads to the conclusion that in the early stages of asbestosis of the lung parenchyma and pleura, there are no parameters at rest sufficiently sensitive to detect impaired ventilatory lung function caused by asbestos. Restrictive impaired ventilation was present only in advanced asbestosis of the parenchyma (category 2). In our experience, although not sufficiently sensitive, the radiograph of the lung and pleura is the earliest non-invasive indicator of possible asbestos disease in a population exposed to small concentrations of asbestos such as shipyard workers.

REFERENCES


Sažetak

PLUĆNE FUNKCIJE I RENDGENOGRAM PLUĆA I PLEURE U BRODOGRADILJENIH RADNIKA IZLOŽENIH AZBESTU

Plućne funkcije i difuzijski kapacitet za ugljik monoksid istraživani su u 299 brodogradiljnih radnika profesionalno izloženih azbestu. Među ispitanicima bio je podjednak broj pušača i nepušača.

Dobiveni rezultati analizirani su u skupinama ispitanika svinđanih prema prozresi plućnog parenhima fibrozom te duljinu i širinu pleuralnih plakova. U odnosu na prozreset parenhima, ispitanici svinđani u kategoriju 2, supkategorije 2/1 i 2/2, imali su statistički značajnu stručenost FVK i FEV1 u odnosu na pleuranog svrstane u kategoriju 0 (P<0,01) i kategoriju 1 (P<0,05). Uočen je trend pada prosječnih vrijednosti MEF25, MEF50 i MEF75 s porastom kategorije prozresi ali bez statističko značajnosti. Vrijednosti difuzijskog kapaciteta za ugljik monoksid bile su podjednake u svim kategorijama. Nije bilo statistički značajne razlike u ispitanicih bez pleuralnih plakova u odnosu prema ispitanicima s vidljivim plakovima. Uočen je trend pada FVK, FEV1, MEF25 i MEF75 s pocecanjem i širine i duljine plaka. Rezultati pokazuju da je u odnosu na istraživanje plućne funkcije rendgenogram pluća i pleure raniji znak moguće azbestne bolesti u brodogradiljnih radnika.

Institut za medicinsku istraživanja i medicinu rada Sveučilišta u Zagrebu, Zagreb, Hrvatska

Ključne riječi: izloženost azbestu, fibroza parenhima, pleuralni plak, profesionalna izloženost, ventilacijska plućna funkcija