ALTERATIONS IN LARYNGEAL MUCOSA AFTER EXPOSURE TO ASBESTOS

Sir,—I am surprised that there has been no response in your postbag to the paper by Kambić et al (1989; 46:177–23). Lest silence be taken as universal agreement that the results justify the use of the term "laryngeal asbestosis," let me protest. The authors compared workers in asbestos cement plants with a control group living in a mountain settlement with extremely favourable climatic conditions. The high incidence of chronic laryngitis in the workers was attributed to asbestos and no attention whatsoever was given to the effects of cement dust. It is obvious that in order to study the effects of asbestos, the control group should be cement workers not using asbestos.

D DAVIES 3 The Sandholes, Farnsfield, Newark NG22 8HQ

Authors' reply:

Our suggestion for the use of the term laryngeal asbestosis has been justified in several recent publications.1 It is established that as well as asbestos and cement, many other known and unknown harmful cofactors must be considered in the development of chronic laryngitis.3 The question is, to what extent particular factors participate in the aetiopathogenesis, but that was not the purpose of our work.

Those who read the article carefully will realise that the occurrence of chronic laryngitis correlates with the degree of workplace pollution with asbestos fibres (see table 6 in the original paper); this is considered a convincing proof for the aetiology of laryngeal lesions among the workers studied.

Scanning electron microscopy on biopsy specimens from 10 workers who needed surgical treatment (stripping of the vocal cords), showed that three had asbestos fibres on the epithelium.

After they changed their work so that they were no longer directly exposed to asbestos dust, and after they ceased alcohol abuse and smoking, the laryngeal mucosa was found to be within normal limits. It is our strong belief that this fact at least partially elucidates the aetiology of the aberrations discussed.


Asbestos related abnormalities among United States merchant marine seamen

Sir,—An important piece of data not provided in the paper by Selikoff and colleagues (1990;47:292–301) is a breakdown of their series according to the radiological ILO classification of parenchymal abnormalities corresponding to pneumoconiosis (0/1, 1/0, 1/1, etc). To a lesser extent a listing of the pleural abnormalities consistent with pneumoconiosis would also be worthwhile. Although radiographic reproductions for publication are somewhat limited in their ability to demonstrate the smaller irregular nodules of pneumoconiosis, especially in the lower profusion category, at least one or two examples in the 1/0, 1/1 range would have been helpful to the reader in order that a definite opinion of the results of this study may be formed.

It has been my observation, as has been reported by others,2 that even among experienced "B" readers, the 0/1, 1/0, and 1/1 categories of profusion for small opacities are difficult areas on which to agree. This becomes even more problematic when en face basal pleural plaques are present.

Currently we are attempting further to define this group of patients with en face pleural plaques and questionable small opacities by the use of high resolution computer tomography (HRCT), as has been suggested by Gamsu.4 It would be of interest to know if there are any patients in the group of Selikoff et al that may have been in this category of en face pleural plaques complicating the interpretation of small opacities.

PETER J BARRETT 10 Martin's Lane, Hingham, MA 02043, USA

Authors' reply:

We read Barrett's comments on our recent paper reporting radiological abnormalities in merchant marine seamen with interest. Parenchymal abnormalities consistent with effects of exposure to asbestos were present in less than 17% of the entire group; in more than half the cases (9%), these were the only radiological abnormalities. Table 1 gives the distribution of the ILO score for parenchymal small opacities; in most cases profusion was in category 1 (1/0, 1/1, 1/2).

We agree with the comments on pleural fibrosis en face and the difficulties encountered in interpreting parenchymal small opacities when such pleural changes are present. We compared the distribution pattern of the ILO score for small opacities in the subgroups without pleural fibrosis face on, circumscribed pleural fibrosis face on, and diffuse pleural fibrosis face on. The proportions of profusion score category 1 (1/0, 1/1, 1/2) and category 2 (2/1 and higher) were higher in the presence of pleural fibrosis face on, especially diffuse pleural fibrosis (table 2).

These findings could be interpreted as indicating that the presence of pleural fibrosis face on makes a positive parenchymal score more likely. Another possibility, suggested by many population studies, is that there exists a genuine significant association between parenchymal and pleural

<table>
<thead>
<tr>
<th>Parenchymal small opacities</th>
<th>ILO score profusion</th>
<th>No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0/0</td>
<td>2258 (67-9)</td>
<td></td>
</tr>
<tr>
<td>0/1</td>
<td>510 (15-3)</td>
<td></td>
</tr>
<tr>
<td>1/0</td>
<td>390 (9-3)</td>
<td></td>
</tr>
<tr>
<td>1/1</td>
<td>190 (5-7)</td>
<td></td>
</tr>
<tr>
<td>1/2</td>
<td>27 (0-8)</td>
<td></td>
</tr>
<tr>
<td>2/1 and higher</td>
<td>30 (0-9)</td>
<td></td>
</tr>
</tbody>
</table>