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; 46:717–23). Let 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.

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Authors’ reply:
Our suggestion for the use of the term laryngeal asbestosis has been justified in several recent publications.13 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. 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.

1 Podolskaia EV. Precancerous conditions of the larynx in workers exposed to dust and their prevention. Vestn Otorinolaryngol 1989;2:67–9.
2 Parsons SM. Asbestos and cancer of the larynx. Is there a relationship? Laryngoscope 1990;100:25–61.
3 Kambić V, Radež Z, Preželj J, Žargi M. The role of testosterone in laryngeal carcinogenesis. Am J Otolaryngol 1984;5:344–9.

Asbestos related abnormalities among United States merchant marine seamen

Sir,—An important piece of data not provided in the paper by Selikoff and colleagues (1999;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, 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 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.

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1 International Labour Office. International classification of radiographs of pneumoconiosis. Geneva: ILO, 1980. (Series No 2.)
2 Parker DL, et al. Public health implications of the variability in the interpretation of “B” readings for pleural changes. J Occup Med 1989;31:775–80.
3 Ducatman AM, et al. “B” readers and asbestos medical surveillance. J Occup Med 1988;30:644–7.
4 Gamsu G. HRCT in the diagnosis of asbestos-related pleural parenchymal disease. Am J Ind Med 1989;16:115–7.

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 (9.9%), 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 fibrrosis face on, and diffuse pleural fibrrosis 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 fibrrosis face on, especially diffuse pleural fibrrosis (table 2).

These findings could be interpreted as indicating that the presence of pleural fibrrosis 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 fibrrosis.

Table 1 Parenchymal changes (small opacities) on chest x ray films of 3324 merchant marine seamen

| Parenchymal small opacities (ILO score profusion) | No (%) |
|--------------------------------------------------|--------|
| 0/0                                              | 2258 (67·9) |
| 0/1                                              | 510 (15·3)  |
| 1/0                                              | 309 (9·0)   |
| 1/1                                              | 190 (5·7)   |
| 1/2                                              | 27 (0·8)    |
| 2/1 and higher                                   | 30 (0·9)    |
fibrosis—that is, the likelihood of a positive parenchymal score is higher in those with pleural fibrosis.

The use of high resolution computer tomography (HRCT) will definitely contribute to clarification of these problems. It is of interest that HRCT generally detects more extensive pleural fibrosis than seen on the standard chest x ray film; it also detects interstitial pulmonary fibrosis in some cases in which no definite parenchymal abnormalities can be identified on the standard chest radiograph.

Asbestos: a chronology of its origins and health effects

Sirs—I read Murray’s recent article (1990;47:361–5) concerning the health effects of asbestos with interest. I agree that rational public health policy in this area must be based on the best available scientific evidence. None the less, I strongly disagree with Murray’s assessment of the use of asbestos in developing countries and his interpretation of the historical evidence concerning scientific knowledge of the carcinogenic potential of asbestos fibres.

Murray states that during the second world war period of asbestos use in shipbuilding “there was no knowledge of lung cancer or mesothelioma and work practices were poor as they were in many industries. It is improper for the apostles of hindsight to suggest that sufficient evidence existed about asbestos as to have been able to anticipate its effects.” As a consultant on the landmark asbestos property damage case Corporation of Mercer University v National Gypsum Co, I had an opportunity to review internal asbestos industry documents concerning industry research on the health effects of asbestos. As outlined in Broder’s book Outrageous misconduct: The asbestos industry on trial, it is clear that the asbestos industry actively suppressed the release of industry sponsored research showing that inhalation of asbestos fibres constituted a serious health risk. In the light of information obtained through legal discovery proceedings in hundreds of asbestos lawsuits it is undeniable that leaders of the asbestos industry conspired to misinform both the public and the scientific community about the dangers of asbestos. Unfortunately, thousands of innocent people paid with their lives for this misguided policy.

I must also disagree with Murray’s enthusiasm for the use of asbestos pipes in developing countries. Third World countries often become “dumping grounds” for toxic materials produced in industrialised nations. With markets for asbestos closed in many western countries, the asbestos industry must cultivate markets elsewhere. What better place than in nations that often lack adequate environmental and occupational regulations? Asbestos water pipes are often manufactured on site. Without proper regulation of such activities, we may, in several decades, witness the same dramatic increase in asbestos related deaths in the Third World as in the United States.

Such short sighted policies are akin to the production of tobacco and development of cigarette industries in Third World countries. Such countries now account for about 60% of world tobacco production with vigorous cigarette industries now established in China, Brazil, and Malawi. Agencies such as the World Bank have actively encouraged and supported the creation of cigarette industries in the third world (often with low interest loans) since it is an extremely profitable commodity and provides quick cash for economic development. Just as with asbestos, without considering the long term effects of such policies, the ultimate cost in terms of human mortality and morbidity cannot be appreciated.

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Authors’ reply:

I have read Huncharek’s comments on my paper with tolerance and understanding. He is entitled to his views as I am to mine, but, like many people, he is also an apostle of hindsight. I made the point that, during the war, when the ultimate priority was winning it, there was no knowledge of lung cancer or mesothelioma attributable to asbestos. If he has evidence that such knowledge existed, let him publish it.

I carry no torches for the asbestos industry. When I visited the United States asbestos industry in 1954 I was not pleased with what I saw and it was not as good as in the United Kingdom. I was told that there was no epidemiological evidence of lung cancer because necropsies were uncommon and in some states, illegal. Much has been made of the suppression of the Saranac Lake data, but it was not as reprehensible as is made out by Castleman and others. In any case, what was the United States government doing? They attended the ILO meetings of experts during the 1930s where asbestos was discussed, even though they were not members of the ILO at that time.

I do not know how many developing countries Huncharek has visited and if he has seen, as I have, the manufacture of asbestos cement materials, including pipes. I have recently seen two model asbestos cement factories, one in Thailand and the other in Nigeria. In the latter case, no diseases attributable to asbestos have arisen in twenty years. Maybe the ones who would have contracted the diseases died of gut infections when they were children!

I was fascinated by the description of the manufacture of asbestos cement pipes on site. Sheets and pipes need sophisticated modern equipment, some undoubtedly more modern than others; you can’t make them in “bush” factories. Bricks you can, but not pipes.

As well as his hindsight, he also indulges in prophecy. Not being the seventh son of a seventh son I cannot compete, but I can tell him that the hysteria in the United States about the removal of asbestos will result in more