

Correspondence

Mortality in cases of asbestosis diagnosed by a pneumoconiosis medical panel

SIR,—The statement by Dr II Coutts and colleagues that mortality in men with asbestosis is unrelated to profusion of small opacities (February 1987;42:111–6) merits further discussion. There should not have been any association in men who died of non-pulmonary conditions. For smokers who died of lung cancer some association might have been expected; it was possibly weak because even mild diffuse fibrosis constitutes a material additional risk.¹ For men dying of respiratory insufficiency there would have been a relationship if in most instances death was due to the parenchymal effects of asbestos. That this was not the case suggests the pleural effects were possibly of greater prognostic significance.

The authors refer only to parenchymal effects and to the whole group. It would be of interest to know for the subgroup of patients with respiratory failure if the mortality was related to the combined parenchymal and pleural abnormalities or to the latter alone. Pleural disease can seriously impair lung function.^{2,3}

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- 1 Kipen HM, Lilis R, Suzuki Y, Valciukas JA, Selikoff IJ. Pulmonary fibrosis in asbestos insulation workers with lung cancer: a radiological and histopathological evaluation. *Br J Ind Med* 1987;44:96–100.
- 2 Wright PH, Hanson A, Kreef L, Capel LH. Respiratory function changes after asbestos pleurisy. *Thorax* 1980;35:31–6.
- 3 Cotes JE, Fulton HS, King B, Hutchinson JEM, Woolley V. Relationship of lung function to x-ray reading (ILO) in patients with asbestos-related lung disease [abstract]. *Thorax* 1987; 42:207.

SIR,—In their paper discussing the mortality of patients with asbestosis diagnosed by a pneumoconiosis medical panel Dr II Coutts and others conclude that an increasing profusion of small opacities on the plain chest radiograph of applicants for compensation does not predict death. This finding is in contrast to the results of a study of West Australian claimants for compensation for asbestosis,¹ in which it was shown that the relative risk of death increased by a factor of 1.26 for each minor ILO category ($p = 0.0001$). Similar results were obtained by Liddell and McDonald.² The negative findings of Coutts *et al* may be a result of small numbers of subjects in the higher profusion groups, but it is difficult to tell from their data if this was the case. It is unfortunate that they do not cite this earlier study (which was published in 1985), as the information contained in it might alter the interpretation of their data: the finding that someone with a chest radiograph resembling a snowstorm fares no worse than an individual whose radiograph is almost clear is unexpected to say the least.

In their discussion of the relative frequency of different histological subtypes the authors make the qualified claim of an increase in the proportion of adenocarcinomas in their subjects, but as there are no control subjects this assumption may not be appropriate. The controlled studies that have

been carried out suggest that adenocarcinoma is not increased in patients exposed to asbestos,³ or even that there is an excess of squamous cell carcinomas.⁴

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- 1 Cookson WOC, Musk AW, Glancy JJ, de Klerk NH, Yin R, Mele R, Carr NG, Armstrong BK, Hobbs MST. Compensation, radiographic changes, and survival in applicants for asbestosis compensation. *Br J Ind Med* 1985;42:461–8.
- 2 Liddell FDK and McDonald JC. Radiological findings as predictors of mortality in Quebec asbestos workers. *Br J Ind Med* 1980;37:257–67.
- 3 Kannerstein M, Churg J. Pathology of carcinoma of the lung associated with asbestos exposure. *Cancer* 1972;30:14–21.
- 4 Baker JE, Reutens DC, Graham DF, *et al*. Morphology of bronchogenic carcinoma in workers formerly exposed to crocidolite at Wittenoom Gorge in Western Australia. *Int J Cancer* 1986;37:547–50.

SIR,—Both your correspondents comment on the lack of any relationship between profusion and mortality. Dr Cotes makes the reasonable point that one might have expected to see an increasing mortality from respiratory disease with increasing profusion of small opacities. No such trend was apparent when our mortality data were summarised for the first five years but such a pattern does begin to emerge at 10 years, although this trend is not statistically significant.

Dr Cookson and colleagues found that the relative risk of death increased by a factor of 1.26 for each minor ILO category. The figure was 1.5 for death from pneumoconiosis but, interestingly, profusion score did not predict death from lung cancer in their series. The proportion of lung cancer deaths in our series was much higher than in theirs (39% v 24%). If profusion score does not predict mortality from lung cancer, this could account for the difference between the two series. The claim that Liddell and MacDonald made observations similar to those of Dr Cookson and colleagues is incorrect. Those authors showed that small opacities of profusion greater than 0/1 were associated with increased mortality, but no attempt was made to examine the effects of increasing profusion thereafter on mortality.

It is true that controlled studies of asbestos exposed patients do not show an increase in the risk of adenocarcinoma but that is not the question which we tried to address. We wanted to know whether there was any difference in the frequency of histological types in cases of established pulmonary fibrosis. There are certainly difficulties in establishing an appropriate control group for this purpose. These difficulties are discussed in the paper and we would stand by our qualified claim that there is a relative increase in the frequency of adenocarcinoma.

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