Correspondence

Update on lung disease in coal miners

SIR—In his editorial (1987;44:145-8) Soutar fortifies some of his arguments by quoting two papers that describe certain investigations carried out by my colleagues and me when I was associated with the National Institute of Occupational Safety and Health. In doing so, he either quotes out of context or misinterprets several of our findings.

Firstly, in his remarks concerning coalworkers' pneumoconiosis, he states that "irregular opacities are related to dust exposure ... and to an impairment of lung function." He supports this comment by quoting, among other references, a paper by Amandus et al.1 In reality, Amandus and his coworkers found that the two main factors associated with the presence of irregular opacities in coal miners were cigarette smoking and increasing age. A surrogate measure of exposure to dust—namely, years worked underground—was also associated to a far lesser degree. Moreover, in non-smoking coal miners irregular opacities were not associated with impaired lung function. In virtually every investigation of the frequency and presence of irregular opacities, whether in coal miners or other dust exposed populations, there has been an association between prevalence of irregular opacities and cigarette smoking. It is abundantly apparent that irregular opacities are found in many occupations other than coalmining, including workers exposed to kaolin,2 man made mineral fibres,3 and silica.4 In addition, they have been noted to occur in non-dust exposed groups including women and here again, cigarette smoking played a pre-eminent part.5 6 Certainly, one cannot attribute the presence of irregular opacities in non-smoking granite shed or man made mineral fibre workers to emphysema and fibrosis. No one has yet suggested that exposure to silica in the absence of conglomerate silicosis leads to emphysema.7 Surely coalworkers are susceptible to the same influences and agents that lead to the presence of irregular opacities in other dust and non-dust exposed workers. The power and validity of the association between coal dust and irregular opacities are relatively weak and multiple regression analysis becomes less reliable when there is collinearity between such factors as age, cigarette smoking, and exposure to dust. The association between them becomes even more tenuous when the interobserver variation in regard to the reading of irregular opacities is taken into consideration.8 Although the paper quoted to indicate the problem with wide interobserver variation originates from the United States, I have similar unpublished data from Britain.

Secondly, Soutar postulates that there is "an element of restrictive lung defect in coalminers" since, in the dust exposed groups, both the FEV1 and FVC are reduced. Contrary to what he states, obstruction is often associated with a decrease in both the FEV1 and FVC, but by contrast, the residual volume (RV) is always increased and the total lung capacity (TLC) is often increased.9 10 In true restrictive impairment, all lung volumes are decreased. An increased RV is an early sign of obstructive impairment and the increase occurs at the expense of the FVC.9-11 Later, the TLC also increases but to a lesser extent; when it does so it partially and temporarily masks the effects of the increase in the RV. In the absence of a knowledge of all lung volumes the mere presence of a reduction in the FEV1 and FVC cannot be taken as an indication that restrictive impairment is present. If Soutar rereads our paper he will note that one of the points we made was that the RV was appreciably increased in smokers whether they had bronchitis or not.12 There was, in addition, a slight but significantly increased RV in non-smokers who had industrial bronchitis or what he terms occupational bronchitis (fig 4). Secondly, he will note that we did not suggest that the airflow limitation occurs in the upper airways, although we believe that most of the obstruction is located in central airways. Indeed our exact words were “flows at low lung volumes were unchanged unless expressed either at absolute lung volumes or as a percentage of TLC, this being a reflection of an increase in RV.” Finally, although he maintains that some coalminers showed severe airflow obstruction due to dust exposure, he concedes that most are smokers and yet attributes the airflow obstruction mainly to dust exposure. This statement is difficult to reconcile with the postmortem study carried out by Fernie et al at the institute in which they were unable to find evidence of pulmonary hypertension or cor pulmonale in the absence of a history of cigarette smoking or PMF.13

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References

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Dr Soutar replies:
Morgan takes issue with some of my necessarily brief remarks on small irregular opacities in the chest radiographs of coalminers, and the type of lung functional impairment related to exposure to coalmine dust.

It is not disputed that irregular opacities in coalminers are related to age and may be related to smoking. I quoted evidence that irregular opacities are related also to a surrogate index of dust exposure (years worked underground)."1 2 and to direct measurements of cumulative exposures to dust.3 Despite his doubts, Morgan associates himself with the paper by Amandus et al.,1 in which the authors state "smoking, age, and years underground were independent factors which each contributed significantly to the prevalence of irregular lesions."

Inter-reader differences in the interpretation of the appearances of the chest radiographs are well recognised,3 4 but agreement between readers may still be sufficient under some circumstances to enable the demonstration of relations between certain radiographic appearances and other variables. Amandus et al (Morgan among the authors) studied inter-reader differences in recognising irregular opacities, and recommended that "epidemiological studies that depend on the differentiation of these types of opacity should involve a majority opinion from several interpreters rather than relying on one reader only. In addition the rigid training of readers periodically by the same standards should help to keep this variation within acceptable limits."5 Observing these precautions Dick et al confirmed a positive relation between irregular opacities and lifetime cumulative dust exposure which is unlikely to be due to chance.6

Referring to the functional implications of irregular opacities, Morgan correctly states that Amandus et al showed impairment of function related to irregular opacities in smokers but not in non-smokers.1 In addition to the other evidence I quoted of functional impairment related to irregular opacities,1 5 6 a new study confirms impairments of function related to irregular opacities in coalminers.7 Most (86%) of the coalminers were smokers or ex-smokers, but results from 104 non-smokers included in that study were consistent with an opacity related impairment in these men also.

Secondly, (Morgan's notation), of course restrictive functional defects cannot be identified with complete confidence on the basis of spirometric measurements alone, though some patterns of abnormality can be suggestive. Our epidemiological studies have shown that dust exposure is related on average to a parallel reduction of FEV1 than FVC, a pattern contrasting with the effects of smoking, which is related to a much greater reduction of FEV1 than FVC, and corresponding reduction of the FEV1/FVC ratio.8 The former pattern is also shown by the group of non-smoking miners with bronchitis (assumed to be "industrial bronchitis") described by Hankinson et al.9 Table 1 in their paper shows that FEV1, FVC, and FEV1/FVC ratio in these men were on average 0.171, 0.181, and 0.3% respectively lower than in an age and height matched group of non-smoking miners without bronchitis. By contrast, the FEV1, FVC, and FEV1/FVC ratio in a group of smoking miners with bronchitis were 0.431, 0.231, and 5.5% respectively lower than in the non-smoking miners without bronchitis. Trying to understand these differences in conventional terms, I suggested in relation to dust induced defects "an element of a restrictive lung defect as well as airflow obstruction. Morgan for his part suggested on the basis of these and additional measurements "that dust induced bronchitis is primarily affecting the larger or upper airways, and is not associated with concomitant destruction of lung parenchyma" (page 178, 3rd para, lines 14–17), while later in the article suggesting additional changes in small airways.9

To resolve these differences, further characterisation of the functional effects of dust exposure is needed, and this will be available shortly. This new information may enable the disabling pathological lesions eventually to be identified by helping to direct the pathologist's attention to the relevant parts of the lung.

Finally Morgan refers to my mention of a small group of coalminers who showed a severe inverse relation between dust exposure and FEV1. Our reasons...
for attributing the decrement in FEV₁ to exposure to dust are explained clearly in the source paper; and just as we are not yet clear on the functionally important pathological changes related to dust, we also have little basis on which to judge the relevance of Morgan's point on pulmonary hypertension or cor pulmonale. Incidentally, the study by Fernie et al included only eight non-smoking miners without progressive massive fibrosis, too small a group to provide substantial support for Morgan's argument.

References

Incidence of cancer among vinylchloride and polyvinylchloride workers: further evidence for an association with malignant melanoma

SIR—Recently Heldaas et al presented the results of a cancer morbidity study in vinylchloride exposed workers (1987;44:278–80). They are suggesting a relation between exposure to VC and the incidence of malignant melanomas. This suggestion is based on visual comparison of observed/expected cases in subgroups, stratified on levels of exposure or on years from first exposure (resp tables 1 and 2).

A statistically significant trend between duration or level of exposure and specific cancer morbidity may be tested according to Breslow et al. This test on significant trend was applied on the observed/expected cases of melanoma in relation to level of exposure (table 1) or in relation to years from first exposure (table 2). The probability, that the distribution of observed/expected cases of melanoma in the subgroups of table 1 and 2 was due to chance, was respectively 65% and 22%.

The only significant finding is the increase in the number of cases of melanoma in the total group of workers. Such an increase, however, was also found in cohort studies of workers with widely differing occupations. Moreover, in all other cohort studies of workers exposed to vinylchloride no increase of mortality from melanoma was found. Therefore, the present epidemiologic evidence does not support a causative relation between the incidence of melanoma and exposure to occupational vinylchloride.

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References

Drs Heldaas, Laugård and Andersen reply:

Ten Berge seems to put a great deal of emphasis on the statistical inference of the results in our study. We are less concerned about the statistics; in our view the emphasis should be put more on the design of the study. We think that the quality of epidemiological studies in occupational medicine should be judged primarily by the characterisation of the level and duration of exposure to the potentially harmful agent, and to what extent selection has been avoided, and whether confounding has been dealt with in a proper way. Occupational diseases with presumed long latency periods have also to be dealt with in a manner which takes care of this phenomenon.

A small sample size is a common weakness in the design phase of small studies. Ten Berge, however, seems to interpret this problem of small sample size and failure to obtain "statistical significance" as being evidence for non-causality. It is not obvious from ten Berge's letter how he has tested statistical significance.