CORRESPONDENCE

Exercise capacity in coal workers’ pneumoconiosis: an analysis using causal modelling

Sir,—The paper by Cooper and Johnson (1990;47:52–7) is a supreme example of hysteron-proteron. Whereas I have little doubt that their results are for the most part correct, at least as far as their cohort is concerned, the inferences that they have drawn cannot be applied to coal miners as a whole.

Firstly, implicit in their analysis of the group is the assumption that disability claimants are representative of coal miners as a whole and this is certainly not the case. The authors’ efforts to exclude subjects with heart disease is a testament to this. No less than 169 of a total of 1064 subjects were excluded on account of heart disease, with the exclusions being based on a series of relatively insensitive criteria. A further 205 subjects were eliminated because of back or leg pain. Thus more than a third of their claimants had complaints that were not related to the lungs. This left those subjects whose symptoms appeared to be predominantly due to a reduction in ventilatory capacity, and even here the causes of a reduction of either or both of the forced expiratory volume in one second (FEV₁) and the forced vital capacity (FVC) are legion and are certainly not limited to coal dust or the other factors considered by the authors.

Secondly, the authors state that “years of mining” does not affect the FEV₁. This statement goes against all published evidence as far as underground miners are concerned.1–3 Thus the authors have no reference population for comparison, and without such it is impossible to know how the FEV₁ compares with a comparable cohort of non-miners. Similarly it has been shown repeatedly that coal miners as a group have a lower FEV₁ and more bronchitis than a suitable reference population of non-miners.4 Moreover, the reduction in the FEV₁ seems to appear in the first years after starting mining. There is also compelling evidence in coal miners that as dust exposure increases the ventilatory capacity declines further and at a rate above and beyond that due to ageing alone.5 That Cooper and Johnson could not demonstrate this is a reflection of the bias inherent in their selection of disability claimants.

Thirdly, whereas the statement that the FVC is affected by coal mining is undoubtedly true, the authors would have been wise to probe more deeply and review some of the older publications. In pure restrictive disease, the FVC and FEV₁ are reduced to roughly the same extent. Cooper and Johnson claim that years spent mining have affected the FVC but not the FEV₁. This observation would seem to require an explanation. There are only two instances where the FVC may be or appear to be selectively reduced in the absence of a comparable reduction in the FEV₁. The first occurs when the forced expiratory volume manoeuvre is terminated prematurely before completion of the exhalation; a common happening in severe obstruction where the forced expiratory volume manoeuvre may take 15 seconds or more to complete.6 The second occurs when the residual volume (RV) is increased without a comparable increase in total lung capacity (TLC) and is a frequent finding in coal miners whether they have coal workers’ pneumoconiosis (CWP) or not.7 It may result from either small airways obstruction as a consequence of the deposition of dust in the peripheral airways or from decreased elastic recoil resulting from focal emphysema.8 Both of these may be observed in working miners with few, or even no symptoms. Deposition of dust is unrelated to the radiographic category of CWP, whereas the decreased lung recoil is found in CWP and tends to be related to radiographic category.9 Moreover, to assume that the decrease in FVC seen in coal miners in the absence of progressive massive fibrosis or another disease is ever sufficiently severe to cause disability is unrealistic.

The authors go on to claim that the PaCO₂, but not the PaO₂, was related to decreased exercise tolerance. They do not state whether this observation occurred with a decreased or increased PaCO₂, but one assumes a decrease. Either way the inference must be in error, as there is an inverse relation between the PaO₂ and the PaCO₂. If a subject hyperventilates, the decline in the PaCO₂ will be matched by a roughly comparable rise in the PaO₂. Such a relation is inevitable assuming Dalton’s law is correct.

Penultimately, the authors refer to a paper of ours in which we investigated the role of blood gas determination in the assessment of disability in United States coal miners.10 The authors go on to say that our assertion that blood gas analysis was not helpful was not based on a multivariate study. Had they read our paper more closely, they would have seen that there was no need to do multivariate analysis as only two subjects met the disability criteria and both had heart disease and congestive failure. All the claimants had been referred to us for exercise testing and blood gas analysis by the Department of Labour and we did not arbitrarily exclude any from our analysis because of other diseases that explained their hypoxaemia. The authors also seem surprised that the PaO₂ does not correlate with exercise, and that subjects with a low PaO₂ often show the same exercise tolerance as those who have normal values. This is hardly a new observation.

Finally, those of us who have been working in this field for many years are fully aware that simple CWP, categories 2 and 3, is associated with certain minor pulmonary impairments. Indeed, similar minor impairments such as an increased closing volume may occur in those who have no radiographic evidence of CWP, but who have been exposed to dust for some years. To equate such impairments with disability—that is, an inability to carry out a specific task—is no more justifiable than it is to presume that a person not exposed to dust who happens to be a cigarette smoker and who has a borderline FEV₁, of 79% of the predicted or an abnormal closing volume is disabled.

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References

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Sir,—The article by Cooper and Johnson (1990;47:52–7) raises some epidemiological issues. Their enthusiasm for multivariate statistical methods leads them to underestimate their use for many years in respiratory epidemiological studies.

There are important conceptual differences between variable types in their relation to each other and to various outcomes. Multivariate methods are generally used to control or adjust for confounding or to display effect modification by extraneous variables affecting the relation between exposure and outcome variables.

For an extraneous variable to be a confounder it must not be an intermediate step in the causal pathway between exposure and outcome. By entering exposures such as smoking or experience of mining and intermediate variables such as forced expiratory volume in one second [FEV1] and forced vital capacity [FVC] into the same regression analysis of exercise minutes, the authors have erred epidemiologically. This may explain why the contributions of smoking and age were not significant and why this finding was “contrary to common wisdom.” It is also not clear from the text whether they forced age into the regression model or not, bearing in mind that mining experience is likely to be strongly colinear with age.

Causal is a difficult word to use in epidemiology. It would perhaps have been better for the authors to have taken an a priori decision about which variables to examine in relation to which outcomes. The hypothesis thus stated, the remaining task is then to choose the most appropriate statistical method that does not violate basic epidemiological (or Statistical) requirements.

Two other epidemiological points relate to the issues of misclassification and external validity. The authors generalise rather hastily from their findings. Because they find no relation between mining experience (years underground) and FEV1, they make the rather radical recommendation that FEV1 be dropped as a criterion for determination of black lung disability if their findings are substantiated by others. Yet studies on both United States and British populations of coal miners have shown effects of dust exposure on FEV1. Mining experience is a relatively poor proxy indicator of exposure to dust. Also, it is not clear whether or not the authors excluded spirometric “test failures” as required by the ATS (1979) criteria they employed. A misclassified indicator of true exposure to dust together with the exclusion of “test failures,” which biases associations with FEV1 towards the null, could suppress an association between exposure to dust and FEV1.

It appears to us to be somewhat artificial to characterise respiratory disability in coal miners as purely restrictive. There is evidence that non-specific exposure to dust in coal mines (and elsewhere) may cause obstruction (decreased FEV1) rather than restriction (decreased FVC) (and elsewhere). Both restriction and obstruction could afflict the same worker and both could contribute to respiratory disability as measured by exercise capacity.

We should like to end with a question. Were cases of progressive massive fibrosis excluded from the study group? This is not clear from the text and could influence FVC findings.


Authors’ reply: Morgan raises several questions.

Firstly, he is concerned that we excluded subjects from our study who had heart or musculoskeletal problems. As the purpose of the study was to investigate relations among coal mining, pulmonary function, and tolerance to exercise, it would not have made sense to include subjects with known coronary heart disease, or those who stopped the treadmill test because of their back hurt. We fail to see how these exclusions introduce bias.

He also suggests that our sample of 690 coal miners may have had a reduction of their ventilatory capacity for reasons other than coal mining or cigarette smoking. We attempted to exclude subjects with heart disease. The prevalence of other conditions that reduce ventilatory capacity is very low in our region so the probability that unlikely diseases were present in sufficient numbers to bias the results significantly in the sample of coal miners is quite low.

Morgan, and Myers and Bachmann, take issue with our finding that “years of mining” did not predict FEV1 in subjects without complicated pneumoconiosis. Contrary to Morgan’s statement that this “goes against all published evidence,” there are others who support this belief. In a contemporary textbook of chest medicine, Becklake states that “most evidence supports the view that simple coal workers’ pneumoconiosis (CWP) is a condition not only without symptoms but also without demonstrable effect on lung function.” If, as Becklake states, simple CWP has no demonstrable effect on lung function, failure to find an association between years of mining and FEV1 is not so surprising. Myers and Bachmann cite two studies. In one, Attfield’s results are the same as ours; years of mining underground had no significant association with FEV1 (see his table 4, model 1). Attfield did find an association between FEV1 and “years at the (coal mine) face.” We note that with 1100 miners studied, the best fitting model in Attfield’s analysis explained only 12% of the variance of FEV1, and when the special “mine effects” term...