

CORRESPONDENCE

Coalmining, emphysema, and compensation

Sir,—In his editorial, Seaton (1990; 47:433-5) makes a plea for awarding compensation to coal miners with simple coal workers' pneumoconiosis (CWP) who have a reduced forced expiratory volume in one second (FEV_1). He justifies his recommendation by attributing the reduction in the FEV_1 to emphysema, which he claims has been induced by coal dust. In doing so he states that the Industrial Injuries Advisory Council (IIAC) did not appreciate the strength or logic of his previous submission; a statement that would seem to imply that the members of the IIAC are perhaps a trifle obtuse.

We share Seaton's view that coal miners have an excess of emphysema over and above the general population. Indeed, some of our earlier studies carried out by the United States Public Health Service, and in which he participated, made this evident. In this connection, the Appalachian Laboratory for Occupational Respiratory Disease (ALFORD) showed that simple CWP in non-smokers was associated with an increase in residual volume (RV) and total lung capacity (TLC) and in some instances a slight to moderate loss of elastic recoil.¹⁻³ Hankinson and colleagues who were working at ALFORD also showed that simple CWP was associated with an increase in air space size.⁴ Although some of the increase in RV occurring in non-smoking coal miners with simple CWP can be attributed to industrial bronchitis, this cannot explain the increase in TLC.^{1,2,5} The increment in TLC must be a consequence of the fact that there is decreased elastic recoil of the lungs. We also accept that cumulative exposure to respirable dust is associated with a greater prevalence of both pneumoconiosis and fibrosis and that the severity of the latter is related to the category of the simple CWP. Similarly we accept the concept that the higher the category of simple CWP and fibrosis, the more extensive is the emphysema.⁶ Seaton's next inference that the emphysema which is present causes obstruction is, however, much more difficult to accept. Were this the

case, the FEV_1 should decrease as the category of simple CWP increases in much the same manner as the RV and TLC increase as the category of CWP worsens. That it does not is all too obvious from other studies.^{2,7,8} By contrast, in cigarette smoke induced emphysema the extent of the emphysema is clearly related to FEV_1 , RV, and TLC. Similarly we ask why is it that, despite the increased air space size shown in subjects with simple CWP, there is no associated fall in the FEV_1 , and why is the mean FEV_1 of subjects with category 0 the same as those with simple CWP?^{2,7} Surely if there is more emphysema in those with simple CWP, and the emphysema is associated with airways obstruction, the FEV_1 should decrease with increasing category of simple CWP. Seaton fortifies his argument by alluding to 95 non-smoking miners with CWP, who showed an inverse relation between FEV_1 and the extent of centracinar emphysema present in them. We can show him similar findings, however, and a slightly reduced FEV_1 in a similarly selected and comparably dust exposed group of non-smoking coal miners without simple CWP who do not have any significant loss of elastic recoil or other evidence of emphysema, but who have bronchitis.

It is also pertinent to note that a rather similar decline in the FEV_1 and the FEF_{25-75} is present in gold miners who are exposed to silica.⁹ Here again cumulative dust exposure is associated with a greater prevalence of silicosis and a lower than normal FEV_1 and FEF_{25-75} , but as the Institute of Occupational Medicine and others have pointed out, the one thing silica is not associated with is emphysema.^{6,10} This also needs to be borne in mind in view of Seaton's endorsement of the hypothesis, "wherever there is fibrosis, there is also emphysema." Certainly this is not true of simple silicosis, healed miliary tuberculosis, and the various fungal diseases seen in North America. Moreover, one wonders whether the overdistension and airways obstruction that are seen in advanced tuberculosis and sarcoidosis have anything in common with focal dust emphysema of coal miners.

We support the concept expressed in the Pearson Report that equal disability should receive equal compensation, no matter whether the cause is occupationally related, natural disease, or another person's negligence.

What is necessary is a fair and uniformly administered system, not a system that, on somewhat tenuous evidence, rewards a smoking coal miner with airways obstruction while ignoring a smoking slate worker with the same problem.

In trying to convince others, Seaton seems to have convinced himself. We would remind him he also failed to convince the late Archie Cochrane, whose many contributions Seaton generously recognised. We hope the IIAC will give due weight to the old adage, namely, "Never to swallow anything whole unless you happen to be a python, otherwise you might choke on it."

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miners: *A clinico-pathological study of emphysema, airways obstruction and massive fibrosis*. Edinburgh: Institute of Occupational Medicine, 1989. (Report No TM/89/05.)

Author's reply

I am pleased to note that my old friends Gee and Morgan accept that coalminers have an excess of emphysema over the general population, although I would remind them that this was not measured and related to measured dust exposure until the work of Ruckley and colleagues.¹ It was, however, the early work in Morgan's laboratory which first excited my personal interest in this confusing subject. Gee and Morgan misquote Ruckley *et al* when they say that they showed a relation between extent (or category) of pneumoconiosis and emphysema. What was reported in that paper and confirmed in recent work was a relation between prevalence of pathological centriacinar emphysema in the lungs of a large group of coalminers and their *lifetime dust exposure*. This has now been confirmed in non-smoking miners.² It is essential to my argument that this central fact is understood.

This being apparently the case, there should be no difficulty in appreciating that if also decrement in FEV₁ is related to lifetime dust exposure and, from several unconnected studies, there is a general inverse relation between extent of centriacinar emphysema and FEV₁, then it is plausible to suggest that decline in FEV₁ may be due to emphysema caused (at least in part) by exposure to dust. One purpose of my article was to give the evidence for this and to quote some supportive work on mechanisms.

I am disappointed that Gee and Morgan are unimpressed with my hypothesis that injury to the lung commonly causes fibrosis and emphysema simultaneously. Certainly, many conditions present to the clinician as primarily one of these, but pathologically there is usually a minor component of the other. In silicosis of the accelerated type or silicotic progressive massive fibrosis, of which I have recently seen a surprising number of cases, emphysema is often a major component. Incidentally, the Institute of Occupational Medicine has never suggested that exposure to silica does not cause emphysema, rather that quartz in coal dust appears to reduce the strength of the relation

between exposure to dust and prevalence of emphysema. This is consistent with the hypothesis that quartz is less liable to cause emphysema and more liable to cause fibrosis than coal. But the general hypothesis I have proposed is one that should be tested experimentally by those interested in the mechanisms of lung disease who, at present, seem to be divided into those studying fibrosis and those studying emphysema.

My other main point related to the award of Industrial Injuries Benefit in the British context, a system of no fault compensation very different from making a claim of injury due to an employer's negligence. In this British system, it is necessary for an employed person to show he has a prescribed disease, and my argument was concerned with a fair system for coalminers in the light of current knowledge. It was not concerned with people with disabilities due to other causes, matters open for the Industrial Injuries Advisory Council to review as and when they feel the evidence is worth considering. My suggestion is relatively simple, and recognises (as do Gee and Morgan) that coalminers are at increased risk of emphysema. I have expressed the opinion that in the presence of radiological evidence of exposure to coalmine dust (pneumoconiosis), a reduced FEV₁ should be attributed to that man's dust exposure and benefit paid accordingly. This, like every other system of dividing a continuum into two, is not absolutely fair, but is the fairest way I can think of, taking account of the scientific evidence and the law and regulations as they stand.

In civil litigation such matters have to be solved by courts on the balance of probabilities, and I can see that some may argue that all airflow obstruction in miners is due to dust as others would argue that it is all due to smoking. The truth, as ever, lies somewhere between and in the case of coalminers I believe we have sufficient evidence to have a stab at finding a just settlement.

Finally, Gee and Morgan might perhaps agree that before swallowing anything, they should look at it carefully. Some things are best swallowed whole. Try crunching your antibiotics—they often taste quite bitter.

1 Ruckley VA, Gauld SJ, Chapman JS, *et al*. Emphysema and dust exposure in a group of coalminers. *Am Rev Respir Dis* 1984;129:528–32.

2 Ruckley VA, Fernie JM, Campbell SJ, Cowie HA. *Causes of disability in coalminers: a clinico-pathological study of emphysema, airways obstruction and massive fibrosis*. Edinburgh: Institute of Occupational Medicine, 1989. (Report No TM/89/05.)

Sedentary work in middle life and fracture of the proximal femur

Sir,—I read the paper of Cooper, Wickham, and Coggon (1990;47:69–70) with great interest. A future study might expand the exposure assessment by using a full occupational history to consider the role of prior occupational activity levels at multiple points in time. The authors did not comment on the lack of a linear dose response trend with increasing proportion of the day spent sitting or on the suggestion, in results for women excluding housewives and for men, that risk could be J shaped. Although the observed increased risk for weight bearing jobs compared with intermediate jobs could be a chance finding, its appearance in both sexes is intriguing. Would the authors be willing to speculate on reasons for a J shaped curve and whether they would expect such findings in a subsequent study?

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Authors' reply

We agree with Heineman that the non-linear risk gradient found in our study of occupational activity and hip fracture is interesting. We were circumspect in our interpretation of it as we considered that the confidence intervals around many of the odds ratios, particularly in men, were wide. Also, a previous case-control study of hip fracture, in which an activity score had been constituted at age 50 years from occupational and leisure activities, suggested progressively increasing fracture risk with declining activity level.¹

A potential biological explanation for a J-shaped relation is the non-linear interrelation of physical activity, muscle strength, and bone mineral density. It has been shown that the threshold level at which osteoblastic activity can be induced is relatively low,² and it might be that above this level, the protective effects of exercise are less obvious. Alter-



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