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?

ELLEN F HEINEMAN

Department of Health and Human Services,
Occupational Studies Section,
Environmental Epidemiology Branch,
6130 Executive Blvd,
Rockville, MD 20892, USA

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-