FORUM

Coal mining, emphysema, and compensation revisited

Francis Bacon was aware that enthusiasts are seldom objective when he wrote. "Quod enim mavult homo verum esse, id potius credit" (*Novum Organon, Aphorism XLIX*). This might be loosely translated as "For what a man wishes were so, he more readily believes." In 1990 an editorial was published entitled "coal mining, emphysema, and compensation".¹ The editorial made a plea for compensation of coal miners who develop emphysema. The argument was that emphysema occurs more often in coal miners, and its presence is associated with respiratory impairment. Although conceding that emphysema could be induced by either cigarette smoking or coal dust, it was suggested that cumulative exposure to dust was an important factor in the induction of respiratory impairment in coal miners. Because in most instances information as to the magnitude of exposure to dust is unavailable, in trying to assess the effects of coal mine dust it was suggested that it was necessary to use a surrogate measure of exposure. In this regard it was indicated that dust induced emphysema is only demonstrable in the presence of pathological evidence of coal workers' pneumoconiosis, and that it is therefore reasonable to provide compensation for coal miners who have a reduced ventilatory capacity in association with early radiological changes of coal workers' pneumoconiosis (category 1/1). Seaton, the writer of the editorial, indicated that the argument had not been accepted by the Industrial Injuries Advisory Council (IIAC) in 1988, and it was furthermore suggested that the IIAC at that time had been unable to appreciate the extent or logic of the evidence. Despite the fact that more recently the IIAC has changed its position and recommended that chronic bronchitis and emphysema should be prescribed, there are many who still find the argument unconvincing and lacking in logic.

Seaton, in his editorial, relied mainly on three publications to support his thesis that coal mining may

lead to disabling impairment in the absence of progressive massive fibrosis and cigarette smoking.²⁻⁴ It is well established that the category of simple coal workers' pneumoconiosis is linearly related to the coal content of the lungs.^{5,6} In this context Ruckley *et al* have shown that the extent of emphysema found at postmortem increases with the amount of coal present in the lung and also with lifetime dust exposure.⁴ If this is so, and there seems little doubt that it is, then increasing category of simple coal workers' pneumoconiosis and its associated emphysema should be associated with a significant decrease in the forced expiratory volume in one second (FEV₁). Thus it should follow that the more dust present in the lungs, the more emphysema, and the worse the lung function. This is not the case, there being no difference in lung function in those with and without simple coal workers' pneumoconiosis.^{7,8}

Further evidence in support of the hypothesis is said to come from an examination of the lungs of a group of 95 non-smoking miners, which showed a clear inverse relation between the extent of the emphysema and the FEV₁.¹ The evidence for this statement is said to be contained in a technical report,⁴ which was not available to us at the time Gee and I questioned the validity of certain conclusions in Seaton's editorial.⁹ The report has since become available to us, but nowhere in the report is there any evidence to support the claim that there is an inverse relation between emphysema and the FEV₁ in non-smoking miners. Of the lungs obtained from the 95 non-smoking miners only 40 (42%) showed the presence of emphysema, centriacinar, panacinar or both. In this connection, the study showed that panacinar emphysema was entirely unrelated to dust exposure.⁴ Of the 40 subjects whose lungs showed emphysema, only 33 (35%) had centriacinar emphysema, of whom 23 also had progressive massive fibrosis, an acknowledged cause of centriacinar emphysema. This left only 10 subjects whose lungs showed centriacinar emphysema in the absence of progressive massive fibrosis. Of these 10, eight had fibrotic nodules—simple coal workers' pneumoconiosis—whereas the other two did not. The authors concluded that because

there were only two non-smoking subjects who had emphysema in the absence of coal workers' pneumoconiosis, no conclusions could be drawn as to the effect of dust in the induction of emphysema in those men who neither smoked nor had coal workers' pneumoconiosis.⁴ Moreover, emphysema was recorded as present when the disease affected as little as 1/30th of the lung, which the authors state is not of import, and would not usually be considered to have any functional significance. Elsewhere it has been shown that symptoms and impairment occur only when at least 20% of the lung is involved by emphysema.¹⁰

Only 24 of the 95 non-smoking miners included in the study had pulmonary function tests carried out during life, and no less than 10 of the 24 had progressive massive fibrosis.⁴ Of the remaining 14 non-smoking subjects, two had emphysema, whereas the other 12 did not. Only one of the two subjects with emphysema had simple coal workers' pneumoconiosis. In short, there were only two non-smoking miners with emphysema who had their FEV₁ measured, and they did not differ from each other. From these data it is impossible to recognise in non-smokers the obvious inverse relation between the FEV₁ and emphysema. The authors of the report concluded that "non-smokers with the highest expected lifetime exposures at British collieries would have a lower risk of developing centriacinar emphysema than a smoker with minimal dust exposure"; moreover, they were also unable to detect an exposure response between dust exposure and the extent of any emphysema found at postmortem.⁴

In their report the IIAC cite three papers that in their opinion were important in helping them reach their decision to recommend that chronic bronchitis and emphysema should become prescribed conditions in coal miners. Two of these papers had been published before the 1988/1989 IIAC statement; clearly the committee were slow to realise their merit. One was a study of 32 "lifelong" non-smoking Belgian coal miners who were compared with 34 steel workers of the same age.¹¹ The subjects were selected by the clerical staff. No details of how the subjects were selected were given, but they

were certainly not randomly chosen and it seemed that they all had symptoms. Although said to be lifelong non-smokers, it is evident that five were ex-smokers who had stopped at least eight years before the study. The contradictions occurring in different parts of the paper throw doubt on the validity of the study. The group of steelworkers had a mean resting PaO_2 of 96.6 mm, which is above the normal range, and is seldom found except in subjects below the age of 30 and who are also hyper-ventilating.

The second study that is quoted in support of the IIAC decision is that of 3380 United Kingdom coal miners in whom the effect of cumulative dust exposure on the FEV_1 in smoking and non-smoking miners was examined.² This study was started in 1953 but the rudimentary questionnaire on smoking habits was not introduced until 1958–59. Many of the men included in the first round of the study in 1953 had been working for 20 to 25 years. Dust measurements before 1953 were few and inaccurate.¹² The measurements of ventilatory capacity involved a direct readout with no permanent record. Moreover, of the 4122 subjects studied by Marine *et al.*, 4119 were said to have had satisfactory questionnaires and lung function tests. Nobody else has ever been able to obtain such compliance and indeed, Kaufmann and her colleagues, whom Marine *et al.* cite in support of their views, had a spirometric failure rate of 28.7%.¹³

The third study that is quoted in support is that of Attfield and Hodous.¹⁴ This was a study of 7139 United States coal miners. An attempt was made to show a relation between the FEV_1 and cumulative exposure to dust. The measurements of dust exposure used by them, however, had been shown by the United States government to be unreliable and had been rejected by the Bureau of Mines.^{15,16} Also, a group of anthracite miners were included in this study in whom no dust measurements had been made.¹⁷ Moreover, this is the only study that has shown that the effects of dust and cigarette smoking on the FEV_1 are the same—that is, a 5 ml annual decrement. Were this so, a man who had worked 40 years would lose only 200 ml, which is just about the variability of the FEV_1 when repeated on several

occasions over a period of 30 minutes or so.

It is perhaps coincidental that the IIAC recommendation to prescribe emphysema and chronic bronchitis in coal miners was sent to the Secretary for Social Security in November 1992 shortly after the Government had recommended closing all but a few of the remaining coal mines in Britain; a decision that was subsequently partially retracted and obviously much regretted by some of those who made it. Clearly the closure of most of the remaining mines will cause severe hardship to the miners and their families and as such they were and are entitled to recompense. If out of contrition for the hardship and suffering caused by closing the remaining coal mines the Government and its appointed Committees choose to pay compensation for the loss of mine jobs, then let them do so directly without distorting medical science to justify their decision.

Bertrand Russell must have been in sympathy with Francis Bacon's sentiments when in *Mysticism and hope* he wrote, "ethical considerations can only legitimately appear when the truth has been ascertained; they can and should appear as determining our feeling towards truth, and our manner of ordering our lives in view of the truth, but not as themselves dictating what the truth is to be." The rejection of truth to further one's concept of ethics or social justice inevitably leads to the ridiculous and the unconscionable.

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Seaton replies

I shall not restate the detail given in my original article, but invite interested readers to return to it. The epidemiological association between coalmine dust exposure, decrement in FEV_1 , presence of centriacinar emphysema, and mortality from chronic respiratory disease are strong and are supported by theoretical and experimental evidence. Morgan's article demonstrates either a careless reading of the literature or an unwillingness to accept this evidence. To consider some of his errors:

(1) He is incorrect when he says that Ruckley *et al* have shown that the extent of emphysema at post-mortem increases with lung coal content and with exposure to dust. They have shown that the risk of having centriacinar emphysema increases with increasing coal content and with exposure to dust.

(2) Lung function has often been shown to be worse (in epidemiological studies) in those with higher dust exposures than in those with lower. If in those with pneumoconiosis, dust exposure is corrected for, an *additional* effect of pneumoconiosis is not detectable. I have never claimed that simple pneumoconiosis impairs ventilatory function but simply suggest it as a reasonable index of heavy exposure to dust. The two papers that Morgan cites in support of his statement that lung function does not differ in those with and without simple coal worker's pneumoconiosis were both incorrectly referenced in his manuscript. Perhaps that is why he misrepresents them. Cochrane and Higgins (Morgan's reference 7), like other Medical Research Council scientists of that era, were puzzled by the fact that older miners with no pneumoconiosis tended to have lower ventilatory capacity than their contemporaries with category 1 disease or more. They speculated that perhaps "mining does in some ways accelerate, to a marked extent in some individuals, the normal decline in ventilatory capacity which occurs with age." Review of this study suggests that it had insufficient power to answer the question that Morgan asks of it. Morgan himself (his reference 8) showed a rise in residual volume in relation to coal workers' pneumoconiosis, but dismissed rather uncritically trends in FEV₁ without mentioning the likelihood of

a healthy worker effect among his (probably) small numbers of category 2 and 3 men. I quote two of his conclusions in that paper—"the mean FEV₁ and FVC of miners in certain geographic regions are substantially lower than they are in others. These differences cannot be accounted for by different smoking habits. It also seems probable that the chemical and physical composition of the dust might influence the prevalence of airways obstruction." I agree!

(3) Morgan is incorrect in claiming that I said that there is a clear inverse relation between *extent* of emphysema and FEV₁ in *non-smoking miners*. The relation between extent of emphysema and FEV₁ was apparent in the postmortem population of the two studies I quoted in all subjects combined. The numbers of non-smokers were too small for analysis in this respect. As I did say, however, a relation between dust exposure and the *risk* of having pathological centriacinar emphysema was demonstrated in non-smokers in the second report by Ruckley *et al* (Morgan's reference 4); I sent this report to Morgan in 1989 and am sorry to hear that he did not receive it.

(4) I note that Morgan believes that progressive massive fibrosis is an acknowledged *cause* of centriacinar emphysema. How on earth can that be? Surely the association between centriacinar emphysema, dust exposure, and progressive massive fibrosis, which Ruckley and colleagues demonstrated, can only be explained on the basis of independent effects of dust, as there is no anatomical relation between the two pathological lesions.

(5) Morgan again attempts to denigrate the careful epidemiological methods of the Coal Board's research. The measurements of dust

exposure, radiological categorisation, and ventilatory capacity have been well validated, and it is difficult to imagine better studies of such magnitude (50 000 miners over 25 years with prospective measurement of dust levels) being funded in the future.

Whether coalminers should receive Industrial Injuries Benefits is, of course, a matter for Government to decide. I am personally in no doubt that exposure to coalmine dust alone can, in occasional cases, cause clinically disabling emphysema. One such patient came to my clinic last month—a 70 year old lifetime non-smoker, heavily exposed to dust—who gave up mining at the age of 50 because of chronic cough and sputum and who is now disabled by radiological and physiological emphysema. He has no useful reversibility and is unresponsive to steroids, nor has he been exposed to birds; α -antitrypsin levels are normal. More commonly, the addition of dust exposure to smoking means that men who otherwise would not have reached a level of FEV₁ that causes disability do so.

Morgan says that there are many who find my arguments unconvincing and lacking in logic. So far I have only heard from him, but I accept there may be others. If there are, it would be interesting to hear their case for explaining away the relations that have now been shown in Britain, the United States, Australia, and Germany. Morgan calls Francis Bacon and Bertrand Russell as his witnesses. I prefer to call William Osler whom I have read on the subject and who made his observations on emphysema and coalmining long before cigarette smoking was an established practice.