The main criticism of Love's paper arises from the nature of the material available to the authors. Ideally, to answer the question in which they were interested, an 11 year follow up study of a well defined population of miners and ex-miners would be necessary. This was not available, so they made the best use they could of the three cross-sectional studies of working miners. However, the brute fact remains that only 1677 of the original 6191 were re-examined. After we have allowed for reasonable exclusions the re-examination rate is 28·7%. The chance of this population being representative of the original population is small and the possibility of inbuilt bias large.

Two other points can be made. The authors themselves only claim that all their variables, including "dust exposure," explained 6% of the variance and their results do not seem compatible with the Medical Research Council's careful epidemiological studies summarised at the Royal Society of Medicine meeting.[2] I think that we shall have to wait some time for the final answer.

A. L. COCHRANE
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[1] Love RG, Miller BG. Longitudinal study of lung function in coalminers. *Thorax* 1983;**37**:193–7.
[2] Cochrane AL. An epidemiologist's view of the relationship between simple pneumoconiosis and morbidity and mortality. *Proc R Soc Med* 1976;**69**:12–4.

SIR,—Unfortunately a number of unwarranted inferences are likely to be drawn from Dr Anthony Seaton's editorial "Coal and the lung" (April 1983, p241). It is clear that coal mining is associated with a reduction in ventilatory capacity and that the latter is independent of the presence of coalworkers' pneumoconiosis.[1 2] Dr Seaton quotes a paper that states that in the past the highest dust exposure had roughly the same effect on FEV_1 as cigarette smoking.[3] Such a comparison is misleading. Unfortunately the National Coal Board questionnaire records current smoking habits and not pack years. To compare the effects of maximal dust exposure to those of average cigarette consumption is inappropriate, and it would be more relevant to compare the rate of decline in the FEV_1 in the most dust exposed to that of the heaviest smokers. Moreover, airways obstruction develops in only 13–15% of cigarette smokers.[4] Thus the paper by Love and Miller[3] compares the effects of cigarette smoking, which induces significant obstruction in a minority, with that of dust, which induces minor obstruction in the vast majority of those with prolonged exposure.[2 5] This is a specious and misleading comparison.

Similarly, Love and Miller deliberately selected older subjects with a long history of dust exposure.[3] Since it is known that cigarette smoking reduces life expectancy by about 11 years, and also leads to premature disability,[6] then the loss to the study from death and disability will have been significant. In contrast, there is no evidence to suggest that dust is similarly lethal, and indeed in one prospective study of coalminers, in which cigarette smoking had a significant effect on the standardised mortality ratio, no discernible effect was apparent from increasing years spent underground.[7] The population studied by Love and Miller was therefore composed of a survivor group resistant to the effects of smoking.

WKC MORGAN
Chest Diseases Unit
University Hospital
London, Ontario
Canada

[1] Rogan JM, Attfield MD, Jacobsen M, Rae S, Walter DD, Walton WH. Role of dust in the working environment in development of chronic bronchitis in British coal miners. *Br J Ind Med* 1973;**30**:217–26.
[2] Hankinson JL, Reger RB, Morgan WKC. Maximal expiratory flows in coal miners. *Am Rev Respir Dis* 1977;**116**:175–80.
[3] Love RG, Miller BG. Longitudinal study of lung function in coal miners. *Thorax* 1982;**37**:193–7.
[4] Fletcher CM, Peto R. The natural history of chronic airflow obstruction. *Br Med J* 1977;i:1645–8.
[5] Kibelstis JA, Morgan EJ, Reger R, Lapp NL, Seaton A, Morgan WKC. Prevalence of bronchitis and airway obstruction in American bituminous coal miners. *Am Rev Respir Dis* 1973;**108**:886–93.
[6] Doll R, Peto R. Mortality in relation to smoking: twenty years' observation in British male doctors. *Br Med J* 1976;ii:1520–7.
[7] Ortmeyer CE, Costello J, Morgan WKC, Sweiker D, Peterson M. The mortality of Appalachian coal miners: 1963–1971. *Arch Environ Health* 1974;**29**:67–72.

***These letters were sent to Dr Seaton, who replies below.

SIR,—Dr Kennedy has often made the point that he believes nitrogen dioxide to be an important cause of emphysema in coalminers, though readers of his paper on the subject[1] will not, I think, be wholly convinced by the case he made. Our recent work has shown a relationship between measured exposure to respirable coalmine dust in life and the risk of having centriacinar emphysema post mortem in men who also showed a fibrotic reaction to dust. In addition, it has been possible to show a similar relationship between risk of emphysema and lung coal content. In both cases the risk was reduced with higher levels of quartz and other silicates in the dust. This work has recently been submitted for publication. We do not have figures for exposure to oxides of nitrogen in this study, but we have recently investigated the relationship between exposure to these oxides, respiratory symptoms, and FEV_1 in coalminers.[3] We find that current levels of the gases, as Dr Kennedy would wish, are low and we are unable to demonstrate any relationship between exposure to oxides of nitrogen and respiratory symptoms or FEV_1.

I do not dispute that in the 1960s there were times when dangerously high concentrations of nitrogen dioxide may have followed shotfiring, and vigilance is clearly necessary to ensure that this does not recur. Whether the present low levels of nitrogen dioxide in coalmines play any part in the causation of "emphysema" must remain an open question. If they do, the effect on FEV_1 from our recent studies seems to be so small as to be undetectable—in any case it is less than the effects of cigarette smoking and coal dust exposure.[2]

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Professor Cochrane, curiously, has taken issue with only one of the five references that I provided in support of the idea that there is a relationship between coal dust exposure and risk of emphysema and disabling airways obstruction. The paper by Love and Miller deals with one aspect of this—namely, the relationship between fall in FEV_1 over 11 years and measured dust exposure. Cochrane's criticisms were of course anticipated and discussed in that paper. Though the sample was indeed small, the authors pointed out that the bias introduced by migration from mining of more disabled men would be likely to make the effect they found if anything an underestimate. More recent work, shortly to be submitted for publication, in which we have included ex-miners in our studies has increased our confidence in the validity of these findings.

Professor Cochrane's point about the high residual variability in our lung function changes is a good one. This degree of unexplained variation is a general problem in longitudinal studies of lung function and was also noted by Fletcher and colleagues.[3] Is it not remarkable that, despite this "noise," a significant effect of coal dust exposure has nevertheless been found in our studies?

Professor Cochrane's last point ignores the fact that our studies have made use of careful longitudinal measurements of individual miners' exposure to coal dust whereas the MRC studies used radiological category of pneumoconiosis as a surrogate for such exposure. We also find no gradient in FEV_1 in relation to pneumoconiosis category when we correct for dust exposure and our results are therefore compatible with those obtained by the MRC. Ours, however, go further; most miners even with high dust exposure do not develop pneumoconiosis, and even in those with category O radiographs it has been shown that higher dust exposures are associated with lower levels of FEV_1.[4] This effect is detectable in both smokers and non-smokers. The MRC studies were careful, but they did not include the exposure measurements essential for answering the question "Does coal dust exposure increase a man's risk of airways obstruction?" I feel that Professor Cochrane should now concede that his careful studies have been superseded.

The points that Dr Morgan makes are fully discussed in the paper by Love and Miller that he cites. The point I was trying to get across was that exposure to respirable coalmine dust has a measurable effect on FEV_1. I hope that our current research will amplify this point in that we are studying men who have left the coalmining industry and taking full account of lifetime smoking habits.

A SEATON
Institute of Occupational Medicine
Edinburgh

[1] Kennedy MCS. Nitrous fumes and coal miners with emphysema. *Ann Occup Hyg* 1972;**15**:285–300.
[2] Robertson A, Dodgson J, Collings P, Seaton A. Exposure to oxides of nitrogen: respiratory symptoms and lung function in British coalminers. *Br J Industr Med* (in press).
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[4] Rogan JM, Attfield MD, Jacobsen M, Rae S, Walker DD, Walton WH. Role of dust in the working environment in development of chronic bronchitis in British coal miners. In: Walton WH, ed. *Inhaled particles III.* Old Woking, Surrey: Unwin, 1971: 883–94.

Late snaring of Lillehei-Kaster prosthesis by a fragment of left atrial monitoring catheter

SIR,—For proper postoperative management of patients undergoing open heart surgery continuous measurement of the left atrial pressure is routine in many cardiac centres. The use of a left atrial monitoring catheter is, however, not without risks and its early complications have been described.[1,2] We report a late complication resulting in snaring of a mitral Lillehei-Kaster prosthesis 13 months after its insertion.

A 51 year old woman who had an uneventful implantation of a Lillehei-Kaster mitral prosthesis on 29 January 1982 was readmitted with sudden haemodynamic deterioration on 23 February. She was in low cardiac output state with pulmonary congestion, raised jugular venous pressure, and absent prosthetic clicks. The electrocardiogram showed acute right ventricular strain and echocardiography confirmed the absence of movement of the prosthesis. She was operated on as an emergency to relieve the obstructed prosthesis. It was anticipated that the prosthesis would be occluded by clot, but in fact there was a fragment of left atrial pressure monitoring catheter wrapped around the strut of the prosthesis in such a way as to immobilise it completely. This was removed, and after being washed the prosthesis moved normally. It was well healed into the mitral annulus and was therefore not replaced. The left atrium was closed over a new left atrial pressure monitoring line and the cardiopulmonary bypass was discontinued. The patient made an uneventful postoperative recovery.

We have measured the left atrial pressure routinely in our unit for 10 years. We place the E–Z catheter (Deseret Company, Sandy, Utah, USA) in the left atrium soon after the initiation of cardiopulmonary bypass. To prevent displacement during the operation, the catheter is sutured to a superficial bite of pericardium. Once the patient is stable haemodynamically, the catheter is removed by a sharp tug that breaks its anchorage to the pericardium. It is routinely checked for its integrity.

Poppet jamming of a Lillehei-Kaster prosthesis due to impaction of a left atrial monitoring line during mitral valve replacement and the occurrence of recurrent bacteraemia due to an abandoned fragment of a left atrial catheter have been described.[1,3] In this case, obviously the catheter had snapped at its point of fixation and the retained fragment caused the immobilisation of the disc. The incident calls for a careful inspection of the catheter after it is withdrawn, preferably by one of the members of the surgical team actually involved in the procedure, and emphasises the fact that jamming of the disc prostheses could be due to factors other than thrombosis or entrapped sutures.

The technique of removal of surgically implanted monitoring catheters deserves mention. These are made of viscous elastic material which will stretch if slowly drawn on, thus becoming weaker and more liable to break. Sharp traction, however, will free the catheter from its retaining pericardial stitch by breaking the stitch or avulsing a pinch of pericardial lining while the catheter is in its unstretched,

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