

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<sub>1</sub> 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.<sup>3</sup> 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<sub>1</sub> 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<sub>1</sub>.<sup>4</sup> 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 I feel that Professor Cochrane should now concede that his careful studies have been superseded.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 coal-mine dust has a measurable effect on FEV<sub>1</sub>. 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.

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<sup>1</sup> Kennedy MCS. Nitrous fumes and coal miners with emphysema. *Ann Occup Hyg* 1972;15:285-300.

<sup>2</sup> 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).

<sup>3</sup> Fletcher C, Peto R, Tinker C, Speizer FE. *The natural history of chronic bronchitis and emphysema*. Oxford: Oxford University Press, 1976.

<sup>4</sup> 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.<sup>1,2</sup> 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.<sup>1,3</sup> 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,

strong state. It is not therefore satisfactory to use a gentler technique for its removal.

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- <sup>1</sup> Reyes LH, Ratzan KR, Rheinlander HF. *Serratia marcescens* bacteraemia originating from a catheter line in the left atrium after mitral valve replacement. *J Thorac Cardiovasc Surg* 1973;**65**:241-4.
- <sup>2</sup> Bricker DL, Dalton ML. Cardiac tamponade following dislodgement of a left atrial catheter following coronary artery bypass. *J Thorac Cardiovasc Surg* 1973;**66**:636-8.
- <sup>3</sup> Browdie SA, Agnew RF, Hamilton CS jun. Poppet-jamming during mitral valve replacement [letter]. *Ann Thorac Surg* 1978;**28**:591.

## Notices

### **Annual meeting of the European Academy of Allergology and Clinical Immunology**

The annual meeting of the European Academy of Allergology and Clinical Immunology 1984 will be held in Brussels on 16-19 May. The themes will be the immunological aspects of asthma, occupational allergy, and immunomodulation, with a postgraduate course on the treatment of allergic diseases. Further information from Professor R Pauwels, Department of Respiratory Diseases, De Pintelaan 185, B 9000 Ghent, Belgium.

### **Applied respiratory physiology**

A course in applied respiratory physiology will be held at the Royal Postgraduate Medical School, Hammersmith Hospital, from 26 to 30 March 1984. The topics will include mass spectrometry, body plethysmography, non-invasive monitoring, nocturnal hypoxaemia, control of breathing, regional lung function, airflow obstruction, diffusion and clinical pulmonary function testing. Visiting speakers will include Dr NR Anthonison (Canada). Application forms and further details are available from: School Office (SSC), Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS.

### **Ninth International Cystic Fibrosis Congress**

The medical/scientific programme of the Ninth International Cystic Fibrosis Congress will be held in Brighton from 12 to 15 June 1984. Papers on any aspect of cystic fibrosis may be submitted (closing date for abstracts 31 January 1984). The principal plenary sessions will include presentations on: prenatal diagnosis and carrier detection, pathophysiology (characteristics of the cystic fibrosis cell,

calcium metabolism, glycoproteins of cystic fibrosis mucus), the changing management of cystic fibrosis (aerosol antibiotics, the role of exercise, supplemental nutrition), and the basic defect. There will be an allied health professionals programme that will include parallel sessions on social aspects of cystic fibrosis, dietetics and nutrition, and physiotherapy. Further details are obtainable from the Congress Office, Cystic Fibrosis Research Trust, Alexandra House, 5 Blyth Road, Bromley, Kent BR1 3RS.

## Corrections

### **Bronchial gland dimensions in coalminers: influence of smoking and dust exposure**

Drs Douglas, Lamb and Ruckley in their paper (*Thorax* 1982;**37**:760-4) refer to work by Dr WKC Morgan and colleagues (*Br J Ind Med* 1978;**35**:285-91). It has become clear that Dr Morgan's work has been misquoted: Dr Morgan did not state that industrial bronchitis is confined to the upper airways. The authors regret this error.

### **Microbiological investigation of farmer's lung disease in Somerset 1963-83**

In the abstract by JP Anderson and FB Greatorex (September 1983, p 709), "acute" in the 6th line from the end should be "chronic"—that is, "In chronic farmer's lung disease. . . ."