Correspondence

Central lymph node changes and progressive massive fibrosis in coalworkers

SIR,—We were very interested in the "central hypothesis" for the aetiology of progressive massive fibrosis in coalworkers proposed by Dr RME Seal and colleagues (July 1986;**41**:531–7). The Joint Coal Board medical division has maintained a continuous series of postmortem studies on lung tissue of coalworkers since 1949 and data on 1070 cases have been analysed to date. All but a few of these lungs have been studied in the fixed inflated state.[1 2] In this series a total of 72 cases were found to be of progressive massive fibrosis (defined as at least one dust lesion over 2 cm in diameter). The hilar lymph node histopathological findings from these 72 cases have been classified on sections of the hilar region according to the scheme proposed by Dr Seal and others without knowledge of the severity of progressive massive fibrosis in the lung parenchyma. Parenchymal progressive massive fibrosis was graded on sagittal sections through both lungs, cut to include the upper and lower lobes. The grading system was similar to the International Labour Office radiographic grading system—namely: *category A*—a lesion having a greatest diameter exceeding 2 cm and up to and including 5 cm, or several lesions, each greater than 2 cm, the sum of whose greatest diameters does not exceed 5 cm; *category B*—one or more lesions larger or more numerous than those in category A, whose combined area does not exceed the equivalent of one third of one lung surface; *category C*—one or more lesions whose combined area exceeds the equivalent of one third of one lung surface. Lymph node histopathology grade was compared with parenchymal progressive massive fibrosis grade (table 1). We also compared the presence or absence of progressive massive fibrosis in each lung when the pathological appearances of the lymph nodes differed between right and left sides (table 2).

Table 1 *Central lymph node histopathology grades and progressive massive fibrosis (PMF) grades in lungs from 72 coalworkers*

Progressive massive fibrosis grade	*Central node histopathology grade*				
	0	*1*	*2*	*3*	*4*
A	0	9	10	10	3
B	0	7	12	18	1
C	0	2	0	0	0

χ^2_1 (categories B + C pooled, categories 0–2, and 3–4 pooled) = 1·32, p > 0·2; NS.

Table 2 *Side of maximum gland pathology grade where right and left sides differed in relation to side showing progressive massive fibrosis (PMF)*

Side showing PMF	*Right*	*Left*
Right	2	1
Left	1	1
Both	4	5

It can be seen from tables 1 and 2 that there is no significant association between the extent of lymph node disease and the extent of progressive massive fibrosis. If the "central" hypothesis of leakage of dust material from a ruptured hilar lymph node into a vessel or bronchus held true, then one would expect such an association to be present—that is, the more "leak back" of dust material the larger the parenchymal progressive massive fibrosis lesion. Our findings are thus against such a "central" hypothesis.

Of the four cases where erosion into a vessel or bronchus was observed, three had the lowest degree of progressive massive fibrosis. Two had erosion into artery only, one into bronchus only, and one into bronchus and artery. The degree of progressive massive fibrosis was, however, significantly correlated with coal dust exposure as estimated by years worked at the coal face ($r = 0{\cdot}37$, $p < 0{\cdot}001$).[1] No cases of progressive massive fibrosis have come to necropsy since 1972, emphasising the association between progressive massive fibrosis and the much higher dust exposures which existed in the past. In New South Wales extensive dust control measures have been in operation since 1949 and shot firing ceased as a mining technique around 1960. New South Wales coal is nearly all bituminous in rank with very little anthracite, and this may partly explain the differences between the aetiology of progressive massive fibrosis in South Wales and New South Wales.

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1 Leigh J, Outhred KG, McKenzie HI, Wiles AN. Multiple regression analysis of quantified aetiological, clinical and postmortem pathological variables related to respiratory disease in coal workers. *Ann Occup Hyg* 1982;**26**:383–400.
2 Leigh J, Outhred KG, McKenzie HI, Glick M, Wiles AN. Quantified pathology of emphysema, pneumoconiosis and chronic bronchitis in coal workers. *Br J Ind Med* 1983;**40**:258–63.

SIR,—We were interested in the data presented by Drs Leigh and Wiles. We do not, however, consider that these are necessarily evidence against the hypothesis put forward in our paper. We found an association between the *presence* of progressive massive fibrosis, or secondary dust foci, and the extent of central lymph node dust related changes. Drs Leigh and Wiles confined their analysis to lungs with progressive massive fibrosis (of at least 2 cm in diameter), so losing the most important part of the analysis. It is not really surprising that they failed to find an association between *size* of progressive massive fibrosis lesions and extent of central lymph node changes; we believe that central changes are important in initiating progressive massive fibrosis within the associated area of lung parenchyma but the subsequent size of these lesions may depend on several other variables.

We are also concerned about the lymph node sections that the authors had available for study from a series going back to 1949 and not collected specifically to display the lymph nodes. It is crucial to look for changes in and around the appropriate node or nodes when progressive massive fibrosis or secondary foci are confined to one area of the lung (as we illustrated in our paper), and this does not appear to have been considered in the Australian study. In our study we were aware that histological assessments were "underestimating" changes in and around lymph nodes because of the difficulty of obtaining sufficient sections (for example, to include a branch of the pulmonary artery), and it is likely that this was also a problem in the study of Drs Leigh and Wiles.

We agree that dust exposure (however estimated) is an important factor associated with progressive massive fibrosis and this has been demonstrated repeatedly. Yet dust exposure is not the only factor concerned and some men seem more "susceptible" than others. Our paper suggested a mechanism for the production of progressive massive fibrosis in individual men. We feel that careful prospective studies are now required to examine this hypothesis further.

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Book notices

Manual of Cardiac Arrhythmias. EK Chung. (Pp 307; $30.) New York: Yorke Medical Books, 1986.

The *Manual of Cardiac Arrhythmias* describes disturbances in cardiac rhythm from innocuous atrial ectopics to serious digoxin associated arrhythmias for which treatment is mandatory and may be life saving. A brief summary of the anatomy and electrophysiology of the specialised conducting system is followed by a chapter on each of the common arrhythmias according to site of origin. More importantly, the clinical significance of each arrhythmia is explained. The author succeeds in explaining clearly the concepts behind tachyarrhythmias such as re-entry, circus movement, and automaticity; and there are two well written chapters on digoxin and its toxic effects. Less common arrhythmias such as coronary sinus rhythm and the ECG findings in cardiac transplant patients are described, although, unfortunately, no example of an ECG from a transplant patient is presented. The tone is didactic, authoritative, and repetitive only in so much as is needed to make each chapter stand on its own. Whatever weaknesses there are are those of omission rather than commission—for example, the section on the differential diagnosis of broad complex tachyarrhythmias could have emphasised the frequency and relative importance of 12 lead ECG findings in arriving at a correct diagnosis. Details of antiarrhythmic drug treatment reflect the narrower choice of agents available in the United States of America. Amiodarone and mexilitene are considered to be still under investigation and flecainide, probably the most effective of all drugs in ventricular tachycardia, is not mentioned. Despite this the general advice about the treatment of arrhythmias is firmly based on general principles and can be adapted to local conditions. This is an excellent textbook which will be read avidly by physicians, cardiologists, medical students and others concerned with cardiac care, including coronary care nurses and technicians.—MG and RH

Manual of Exercise ECG Testing. EK Chung. ($30.) New York: Yorke Medical Books, 1986.

This book is a comprehensive work based mainly on the author's long personal experience. It covers most clinical and practical aspects of exercise ECG testing. It has been formatted and edited to facilitate its use as a concise and simple guide to the subject and makes no attempt to enter into complexities of exercise physiology. The text is well illustrated with ECG tracings and tables. Although much of the material is repeated it provides easy reading. The only irritating feature is the excessive use of numbered points in the text which makes the book read rather like a shopping list. The book covers most practical aspects of exercise testing and the chapters on its use after myocardial infarction (chapter 8) and the interpretation of arm exercise ECG tests (chapter 10) are especially well written and useful. Much of the practical detail is valuable since this book has gathered together a great deal of information about exercise protocols etc, which are difficult to find elsewhere The book is aimed at the general physician with an interest in cardiology and the cardiologist either trained or in training. It will be of considerable use to physiological measurement technicians, as a practical guide, and is of at least theoretical interest to nurses concerned with cardiac care.—MG and RH

Cardiac Arrhythmias—Self Learning. EK Chung. (Pp 220; $30.) New York: Yorke Medical Books, 1986.

This self learning volume contains 200 examples of cardiac arrhythmias with questions after each ECG and a brief discussion on the reverse of the page. It is sufficiently comprehensive to serve as an excellent source of revision. It may be taken either alone or with the *Manual of Cardiac Arrhythmias*. The overall clarity and authority of both books commend them as a quick source of reference in any coronary or intensive care unit and as an introduction to the salient aspects of common cardiac arrhythmias for medical students, coronary care nurses, and junior hospital doctors with or without a special interest in cardiology and even cardiac surgeons and anaesthetists.—MG and RH

Notice

Symposium on Surgical Stapling

The first British Symposium on Surgical Stapling will be held on 22 and 23 October 1987 in Manchester. The chairman will be Professor WD George. Further details from Conference Co-ordinates, Confer House, 69 Kingston Road, New Malden, Surrey KT3 3PB.