LETTERS TO THE EDITOR

Pulmonary complications of HIV disease

We read with interest the review by Drs DG Mitchell and RF Miller (May 1992;47:381-90). They refer to a study published in 1991 that reported that bacterial pneumonia occurred much less frequently than *Pneumocystis carinii* pneumonia in homosexual men in London over a four year period,[1] in contrast to a high relative frequency in an American population that included a large proportion of intravenous drug users.[2] Our findings suggest that bacterial pneumonias in HIV infected homosexuals in London may be occurring with a substantially greater relative frequency than previously reported.

We reviewed the case notes of all HIV seropositive patients admitted to our unit over six months (December 1991 to June 1992) in whom a diagnosis of chest infection was made. Sixty three patients were identified, representing 19% of admissions related to HIV infection. Forty one patients were diagnosed as having *Pneumocystis carinii* pneumonia; these had all undergone bronchoalveolar lavage and *Pneumocystis carinii* pneumonia was confirmed in 35 (32 homosexuals, three intravenous drug users). Twenty two patients were diagnosed as having bacterial pneumonia. For this survey we adopted the following definition of bacterial pneumonia: the presence of fever (>37·5°C) with new chest symptoms or signs and a new infiltrate on the chest radiograph and responsiveness to antimicrobials (not including anti-pneumocystis pneumonia cover). Fifteen patients fulfilled these criteria (12 homosexual, three intravenous drug users).

Our findings suggest that the ratio of cases of bacterial pneumonia to episodes of pneumocystis pneumonia requiring admission was around 1:2 overall and greater than 1:3 (12:32) in homosexuals, substantially higher than the 1:8 reported by Jeffrey and Miller.[1] The populations studied may not be directly comparable (for instance, our data only apply to patients admitted to hospital), but none the less the results support the possibility that the relative frequency of bacterial chest infections in patients with HIV disease may have increased. Factors responsible for this may include more effective prevention of *Pneumocystis carinii* pneumonia and an increase in the population of patients with lung damage related to previous chest infection.

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1 Jeffrey AA, Miller RF. Bacterial pneumonia in homosexual patients positive for HIV [abstract]. *Thorax* 1991;**46**;771P.
2 Magnenat JI, Nicod LP, Auckenthaler R, Junod AF. Mode of presentation and diagnosis of bacterial pneumonia in human immunodeficiency virus infected patients. *Am Rev Respir Dis* 1991;**144**:917-22.

AUTHORS' REPLY Dr Crowley and colleagues point out apparent differences between the incidence of bacterial pneumonia at the Middlesex Hospital, London (in an HIV positive population consisting of homosexual men,[1] and in a Swiss population in Geneva (57% of whom were injecting drug users).[2]

More recently we have reported a comparison of diagnoses in HIV positive men with respiratory problems admitted to a dedicated inpatient unit at the Middlesex Hospital in 1986–7 and 1990-1.[3] Of consecutive patients admitted with respiratory episodes in 1986–7 (33% of all admissions to the unit), 50 had *Pneumocystis carinii* pneumonia and five had bacterial infection (one had pneumonia and four had bronchitis, a diagnosis of pneumonia being made on the basis of criteria similar to those of Dr Crowley and colleagues), a ratio of 10:1; but in 1990–1, of 122 consecutive admissions for respiratory episodes (27% of all admissions), 59 had *Pneumocystis carinii* pneumonia and 26 had bacterial infection (19 had pneumonia and seven had bronchitis), a ratio of 2·2:1. Between these two periods admission policy remained unchanged.

We agree with Dr Crowley and colleagues that observed differences in incidence may be ascribed to use of antiretroviral drugs and use of primary and secondary prophylaxis against *Pneumocystis carinii* pneumonia, factors that also may contribute to prolonged survival from HIV disease. Many patients presenting with bacterial infection have advanced HIV disease and frequently have prior AIDS defining illness.[3]

The view that prophylaxis against *Pneumocystis carinii* pneumonia is "unmasking" a greater incidence of other respiratory infections is endorsed by Chien *et al*,[4] who have shown that before use of prophylaxis against *Pneumocystis carinii* pneumonia 68% of all HIV related admissions in Toronto were for infections. Of these patients, 48% had *Pneumocystis carinii* pneumonia, 20% had other respiratory infections (including bacterial pneumonia), and 32% had non-respiratory infections. After introduction of prophylaxis 67·5% of all admissions were for infections; only 29% of these patients, however, had *Pneumocystis carinii* pneumonia, whereas other respiratory infections now accounted for 27% (and 44% had non-respiratory infections).

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1 Jeffrey AA, Miller RF. Bacterial pneumonia in homosexual HIV positive patients [abstract]. *Thorax* 1991;**46**:771P.
2 Magnenat JI, Nicod LP, Auckenthaler R, Junod AF. Mode of presentation and diagnosis of bacterial pneumonia in human immunodeficiency virus infected patients. *Am Rev Respir Dis* 1991;**144**:917–22.
3 Pitkin AD, Grant AD, Foley NM, Miller RF. Changing patterns of respiratory disease in HIV positive patients in a referral centre in the United Kingdom between 1986–7 and 1990–1. *Thorax* (in press).
4 Chien S-M, Rawji M, Mintz S, Rachlis A, Chan CK. Changes in hospital admission pattern in patients with human immunodeficiency virus infection in the era of *P. carinii* pneumonia prophylaxis. *Chest* 1992;**102**:1035–9.

Acute pneumonitis associated with low dose methotrexate treatment for rheumatoid arthritis

We read with interest the paper by Dr MR Hargreaves and others (August 1992; 47:628-33). With the increasing use of low dose methotrexate in the treatment of patients with rheumatoid arthritis, drug induced pneumonitis is likely to become increasingly recognised. The correct diagnosis is not always easily made, however, and inappropriate management may be fatal.

We wish to report the case of a 65 year old man with a 20 year history of rheumatoid arthritis who started taking methotrexate 10 mg weekly in March 1992, at which time his pulmonary function and chest radiograph were normal. He was admitted to the coronary care unit six weeks later with acute dyspnoea and bilateral lung crackles. The chest radiograph showed bilateral upper zone shadowing and an electrocardiogram suggested left ventricular ischaemia. He was treated with intravenous diuretics and nitrates but failed to respond and became increasingly hypotensive, with consequent oliguria and acute renal failure. His oxygen saturation (Sao_2) fell to 80% despite increasing concentrations of inspired oxygen. Cardiac enzymes were normal, and dopamine was given through a central venous line—to no avail. Antibiotics were not administered.

The possibility of methotrexate pneumonitis causing hypoxia and consequent myocardial depression was considered and he was treated with 500 mg of intravenous methylprednisolone on three successive days. Within 24 hours of his starting on this his SaO_2 had risen to 95% and his blood pressure had become normal. Pulmonary function testing at this stage showed a pronounced restrictive defect. Renal function improved and he was started on prednisolone 60 mg daily. He was discharged one week later. At review a month after discharge his renal function and chest radiograph were almost normal. Methotrexate has been permanently discontinued and his pulmonary function is slowly improving with a reducing dose of oral prednisolone.

Pneumonitis due to methotrexate can clearly mimic left ventricular dysfunction and failure to suspect it may lead to a fatal delay. Immunosuppressive doses of steroids may be required in severe cases, though infection should first be excluded. Upper lobe abnormality, although unusual, may be especially likely to cause diagnostic confusion. Disproportionate hypoxia with a restrictive spirometric pattern may aid differentiation from primary myocardial disease. We hope that the accumulating data on methotrexate pneumonitis will alert physicians to the broad spectrum of its presentation.

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Tuberculosis contact tracing: are the British Thoracic Society guidelines still appropriate?

The suggestion made by Dr SF Hussain and colleagues (December 1992;47:984–5) of a review of tuberculosis contact tracing recommendations is timely. The number of

cases diagnosed as a result of the contact tracing described in this paper is small, however, and it would be a pity if the possibility of late tuberculosis were discounted on the grounds that no such cases were found in South Glamorgan. We recently reported on a child who was found to have a tuberculous pleural effusion six months after being a close contact of a patient with smear positive tuberculosis.[1] This child had been thought to be negative on being screened six weeks after the last contact. In children at least, longer follow up may be appropriate.

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1 Bush A, Warner JO. Tuberculosis in a contact. *Arch Dis Child* 1991;**66**:347-8.

BOOK NOTICES

Beta Blockers and Cardiac Arrhythmias. Prakash C Deedwania. (Pp 336; $115 US and Canada, $132 other countries.) New York: Dekker, 1992. ISBN 0-8247-8450-2.

This excellent book, the fifth in the "Fundamental and Clinical Cardiology" series, is directed towards cardiovascular physicians and research workers in this area. It is both detailed and comprehensive, but nevertheless remains perfectly readable. The opening chapters provide a clear background to the volume by explaining the ionic basis of cardiac action potentials and the electrophysiological effects of β adrenoceptor agonist and antagonist agents. The important concept of ventricular fibrillation threshold and its dependence on ischaemia and sympathetic activity is discussed, and forms a logical argument for the cardioprotective mechanism of β blockers. These ideas are expanded to form the rationale behind the use of β blockers for the treatment of arrhythmias in the setting of ischaemic heart disease and cardiac failure. Comparative clinical pharmacology and pharmacokinetics of β antagonists are then discussed with reference to the additional properties of β_1 selectivity and membrane stabilising and partial agonist activity. Most of the volume applies these fundamental concepts to the clinical setting and evaluates the role of β blockers in acute myocardial infarction and as primary antiarrhythmic agents. Interrelationships between catecholamines, myocardial ischaemia, and ventricular arrhythmias are emphasised, along with the beneficial effects of β blockade on risk of sudden death and the circadian rhythm of cardiovascular events. Conflicting effects of drugs on suppression of ventricular ectopy and the prevention of cardiac mortality are discussed with particular reference to the CAST study. The potential role of β blockers as primary antiarrhythmic agents is evaluated by discussing the clinical experience with conventional agents in this setting by comparison with the effect of class I agents and sotalol. The final chapter is a concise but comprehensive review of sotalol and its unique combination of β blocking and class III activity. Overall, the book is heavily clinically orientated, but throughout remains based on fundamental electrophysiological principles. It is an excellent overview of this area and is highly recommended.—NMW

Cardiopulmonary Physiology in Critical Care. Edited by Steven M Scharf. (Pp 472; £150.) New York: Dekker, 1992. ISBN 0-8247-8649-1.

This volume is one of a number comprising a series entitled "Fundamental and Clinical Cardiology". The stated aim of the editor was to bridge the gap between current concepts of the effects of disease on the cardiopulmonary system and the level of physiologic (sic) knowledge likely to be encountered in junior medical staff employed in what he terms the CCU (critical care unit). The 23 chapters (17 of which are contributed by the editor) are distributed between five sections, the logic for which is not immediately apparent. Thus part I, in dealing with mechanical concepts in cardiopulmonary physiology, includes a good deal of physics, much of which has limited clinical relevance. Part II includes detailed descriptions of different categories of pressure transducers, an electronic circuit diagram or two, and much complex mathematics relating to the principles of cardiac output measurement. The chapter dealing with regulation of peripheral blood flow covers the clinical implications of supply dependency of oxygen uptake, a hot potato among the critical care fraternity at present, in only a couple of pages and mentions little of the controversy surrounding this area, in which an understanding of basic physiology might influence clinical management in a major way. The pulmonary section was by far the best for my money, for the first time (in the chapter on respiratory muscle weakness) linking physiology to physical findings on examination and incorporating a well written contribution on gas exchange. Unfortunately for your reviewer, this represented an oasis of clinical relevance in an otherwise featureless sea of physiology, much of it explained in mathematical terms. In my experience, junior medical staff in the intensive care unit favour books that identify a clinical problem and indicate appropriate management options, each justified in physiological terms. By contrast, this book starts with the physiology and puts clinical medicine very firmly in second place.—TE

NOTICES

British Society for Allergy and Clinical Immunology and VI Charles Blackley Symposium

A joint meeting of the British Society for Allergy and Clinical Immunology (annual conference 1993) and the VI Charles Blackley symposium will be held from 2 to 4 August 1993 at the University of Nottingham. Details from BSACI conference secretariat, Congress House, 55 New Cavendish Street, London W1M 7RE (tel 071 486 0531; fax 071 935 7559).

Symposium on scoliosis

The ninth international Philip Zorab scoliosis symposium, on the theme of evaluating management (including cardiorespiratory aspects), will be held at Queen's College, Cambridge, from 15 to 17 September 1993. Details from the symposium secretariat, 42 Devonshire Road, Cambridge CB1 2BL (tel 0223 323437; fax 0223 460396).

CORRECTION

Portable liquid oxygen and exercise ability in severe respiratory disability

In the paper by R M Leach *et al* (October 1992;**47**:781–9) we regret an error on page 782, column 2, line 6 of the first full paragraph, which should read "endurance walk and a six minute walk were . . ."