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 Pneumocystis carinii pneumonia occurred much less frequently than Pneumocystis carinii pneumonia in homosexual men in London over a four year period, 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 more frequent 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 and 26 had bacterial infection (19 had pneumonia and seven had bronchitis), a ratio of 2:1:1. Between these two periods admission policy remained unchanged.

We agree with Dr Crowley and colleagues that observed differences in incidence may be ascribed to 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 illnesses.1

The view that prophylaxis against Pneumocystis carinii pneumonia is "unnecessary" greater incidence of other respiratory infections is endorsed by Chien et al,1 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 no 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 different infections now accounted for 27% (and 44% had non-respiratory infections).

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 rheuma-

toid 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 unit six weeks later with acute dyspnoea and bilateral lung cracks. 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 (SaO2) fell to 80% despite increasing concentrations of oxygen. His creatinine kinase, cardiac enzymes 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 methyldopa one on three successive days. Within 24 hours of his starting on this his SaO2 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 had been permanently discontinued and his pulmonary function is slowly improving with a reducing dose of oral prednisolone.

Pneumonitis due to methotrexate can cause 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

1 Jeffrey AA, Miller RF. Bacterial pneumonia in homosexual patients positive for HIV antibody. Thorax 1991;46:771P.
3 Peck AD, Grant AD, Poole NM, Miller RF. Changes in pattern of respiratory disease in HIV positive patients in a referral centre in the United Kingdom between 1986-7 and 1990-1. Thorax (in press).