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LETTERS TO THE EDITOR

Mycobacterial infection in patients infected with the human immunodeficiency virus

Dr M Helbert and colleagues (January 1990;45:45-8) reported a 6% incidence of Mycobacterium tuberculosis in a case series of 207 patients with AIDS and 34 patients with the AIDS related complex. At Central Middlesex Hospital we have been concerned in the care of 12 patients with AIDS, three of whom had M tuberculosis. Two developed tuberculosis a few months before the diagnosis of AIDS; one was an immigrant from sub-Saharan Africa and the other an immigrant from the Indian subcontinent. The third patient, who was white, developed tuberculosis as a late complication of AIDS. Of the remaining nine patients, two were sub-Saharan African immigrants (one of whom developed an infection with Mycobacterium avium-intracellulare), one was West Indian, and the other six were white.

Experience in the United States has shown that M tuberculosis in patients with AIDS is much more common in groups which have a higher prevalence of previous tuberculous infection.12 A recent prospective study of intravenous drug misusers provided strong support for the idea that tuberculosis in these patients is a consequence of reactivation of latent tuberculous infection rather than a primary infection.2 We believe that the high prevalence of M tuberculosis in our patients was due to the high proportion of ethnic minorities (42%) in whom there is an increased prevalence of tuberculosis. In England and Wales during 1983 the notification rates of tuberculosis for the Indian subcontinent and West Indian ethnic population were estimated to be 24 times and four times, respectively, that of the white population.3 In sub-Saharan Africa the risk of infection with tuberculosis is also substantially greater than that of England and Wales.4

Dr Herbert and colleagues in their discussion considered only intravenous drug misuse as a possible influence on the prevalence of tuberculosis within their population. No mention was made of the ethnic background of their patients. We believe that this is important in the assessment of M tuberculosis in HIV infected patients. Of the 23 HIV positive patients attending Central Middlesex Hospital, nine are from sub-Saharan Africa or the Indian subcontinent and three are intravenous drug misusers, reflecting the demography of the local population.5 It has recently been suggested that in the United Kingdom people infected with HIV who have not had BCG vaccination should be tuberculin tested and offered prophylactic treatment if they are positive.6 False negative tuberculin reactions are recognised, however, in HIV positive patients⁷ and in symptomatic patients must be combined with a chest radiograph and appropriate bacteriological specimens. The demography of our population suggests that tuberculosis is likely to be a continuing problem. We intend to implement routine tuberculin testing in our patients who have not had BCG vaccination and to offer

antituberculous treatment or prophylaxis as necessary.

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It is now generally accepted that the greater incidence of tuberculosis in patients with AIDS than in the general population is due to breakdown of foci of tuberculous infection acquired before infection with the human immunodeficiency virus (HIV). The incidence of tuberculosis in a group of patients with AIDS will therefore depend mainly on the extent to which the group was infected with tuberculosis before acquiring HIV infection. On this basis further useful epidemiological information might be available from the series presented so successfully from the clinical standpoint by Dr M Helbert and others (January 1990;45:45-8). If the series includes any non-white patients it would be helpful to see an analysis of the incidence of tuberculosis by ethnic group. An analysis of the incidence of tuberculosis by broad age groups in the white ethnic group might also be informative, as those over age 50 years in 1983-8, when this series was collected, were found to be at least 40% tuberculin positive when aged 13 years or more in 1949-50, while those under 30 years of age in 1983-8 were less than 2% tuberculin positive when aged 13 years in the early 1970s.2 Similar information from other series would also be most helpful in assessing the likely impact of the developing AIDS epidemic on tuberculosis in Britain. Separate analysis by age of other mycobacterial infections in patients with AIDS might help to reveal differences in pattern from disease due to Mycobacterium tuberculosis.

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AUTHORS' REPLY Of the 15 patients with tuberculosis and HIV infection, one was of Indian origin (age 43), one was of Afro-Caribbean descent but brought up in Britain (age 21) and one was a white man who had lived in central Africa (age 52). The rest of the

patients were white and had lived in Europe all their lives. None of these patients was an intravenous drug user.

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Assessment of reversibility of airway obstruction in patients with chronic obstructive airways disease

We were interested to read the article by Nisar and colleagues (March 1990;45:190-4) describing reversibility in response to salbutamol and oral prednisolone in patients with chronic airflow obstruction. Their data suggested that a response to 5 mg of nebulised salbutamol, defined as an increase in FEV, of at least 15% and 200 ml, could be used as a means of identifying patients likely to respond to treatment with corticosteroids. Such a screening test would be extremely helpful in reducing the time spent performing steroid trials to determine optimal treatment for this group of patients. Our own results, recently published,1 when analysed according to the criteria used by the Liverpool group do not show the same conclusions, and we are concerned lest chest physicians withhold potentially beneficial treatment in disabled patients with chronic airflow obstruction.

Our reanalysis shows that, of 13 patients who responded to oral prednisolone by increasing their FEV, by at least 15% and 200 ml, five (38%) showed no response to 10 mg nebulised salbutamol. When we retain our own criteria for response (a 20% improvement in FEV₁, or forced vital capacity (FVC) or peak expiratory flow (PEF), over baseline), 19 of 39 responders to prednisolone did not show an improvement in FEV, of 15% and 200 ml after 10 mg salbutamol. Hence in our patients the use of response to nebulised salbutamol as a screening test caused us to miss up to half of the patients who showed benefit. The increase in response rate when FVC and PEF are included in the response criteria also suggests that with the use of FEV₁ as the sole physiological measure of response many patients responding to treatment would be missed. Indeed, in our paper only 29 of the 65 responses to treatment (oral prednisolone or inhaled beclomethasone) were in FEV

We would urge physicians to offer a trial of steroid treatment to all patients with chronic airflow obstruction, and to assess response using FVC and domiciliary PEF monitoring in addition to FEV, measurements and subjective measures; otherwise a significant number of patients will be denied the benefits of treatment.

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 Weir DC, Gove RI, Robertson AS, Burge PS. Corticosteroid trials in non-asthmatic chronic airflow obstruction: a comparison of oral prednisolone and inhaled beclomethasone dipropionate. *Thorax* 1990;45:112-7).

AUTHORS' REPLY We thank Drs Weir and Burge for their interest in our paper and for raising some important issues. It was helpful