LETTERS TO THE EDITOR

HIV related bronchiectasis

In their review of new developments in pulmonary diseases affecting HIV infected individuals (March 1995;50:294–302) Mitchell and Miller do not discuss bronchiectasis. They mention indolent bronchopulmonary Pseudomonas aeruginosa infection comparable to that seen in cystic fibrosis, but do not comment on bronchiectasis which is now well described in adults and children with HIV infection.

Bronchiectasis in a series of HIV infected adults was first reported from Oxford in 1992, and the association has subsequently been confirmed in reports from the USA. The incidence of bronchiectasis in the HIV infected population remains to be established; it is frequently undiagnosed because of a low index of suspicion and because chest radiographs may be normal or non-specific. High resolution computed tomographic (CT) scanning is the investigation of choice. The aetiology is likely to be multifactorial, but recurrent bronchopulmonary infection is probably one of the most important contributing factors. Some cases have been seen following Pneumocystis carinii infection alone, while other cases have been related to various endobronchial lesions. Most cases of bronchiectasis in HIV infected adults are seen following recurrent episodes of pyogenic infection with common pathogens such as Strep-tococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis, and Staphylococcus au-reus. In paediatric practice bronchiectasis has emerged as a complication of lymphocytic interstitial pneumonitis. A report of the isolation of Burkholderia (formerly Pseudomonas) cepacia from one case of bronchiectasis in an adult with AIDS gives cause for concern. This organism has a predilection for the lungs of patients with cystic fibrosis and has caused several epidemics associated with cystic fibrosis. The potential exists for B cepacia to pose a similar threat to HIV patients with chronic lung disease.

At Boston City Hospital in the past year we have diagnosed bronchiectasis in three patients, aged 7, 10, and 16 years, out of 60 children with AIDS. All were boys and had congenitally acquired HIV infection. Each had experienced many previous episodes of pneumonia, and the two younger patients had previously experienced lymphocytic interstitial pneumonitis, based on chest radiographic findings or histopathological examination. Symptoms of chronic productive cough, exercise limitation, and persistent basilar pulmonary infiltrates suggested bronchiectasis, which in each case was confirmed by a chest CT scan. Sputum culture yielded S pneumoniae, H influenzae, nontypable, beta-lactamase negative, and normal flora respectively.

We conclude that bronchiectasis has become a significant problem in HIV infected patients, and has the potential to cause substantial morbidity.

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HIV infection is a major cause of morbidity. This is a summary of a recent review by Drs McFarlane and Lipworth (February 1995;50:134–8) that in vivo salbutamol shows characteristics of a β2 adrenoceptor partial agonist in a state of high adrenergic tone (exercise) is based upon the observation of similar effects of salbutamol and the non-selective β adrenoceptor antagonist, propranolol, in augmenting exercise-induced hyperkalaemia. The authors state that the mechanism of this augmentation of exercise-induced hyperkalaemia is by β2 adrenoceptor antagonism since propranolol and the selective β2 adrenoceptor antagonist ICI 118 551 would show similar effects in this respect. It is the latter assumption that we would like to challenge. We think that it is more likely that the effect of salbutamol on the exercise-induced increase in potassium is due to some other yet unknown mechanism.

Concerning the effects of ICI 118 551 on exercise-induced hyperkalaemia, the authors refer to a paper on observations of the effects on plasma potassium levels in six healthy volunteers during a game of squash. However, in that paper ICI 118 551 did not influence the exercise-induced increase in plasma potassium levels, as was also concluded by the authors of that paper. The difference in the mean plasma potassium level at the end of the exercise period between the placebo and active treatment groups can be explained by the difference in baseline potassium levels before exercise. Moreover, in another paper a direct comparison between propranolol and ICI 118 551 of the effects on plasma potassium levels during exercise was made under standardised conditions – that is, during incremental ergometer exercise. In that experiment only plasma potassium levels after pretreatment with propranolol were different from placebo, but plasma potassium levels after ICI 118 551 were identical to those after placebo treatment.

On the basis of these two studies, it is very unlikely that blockade of β2 adrenoceptors is involved in the phenomenon of exercise-induced hyperkalaemia. Either non-specific β blocker or a non-β adrenoceptor-mediated mechanism shared by several drugs of this class is involved. We think that the latter notion makes the conclusion of the paper by Grove et al concerning the β2 adrenoceptor partial agonist activity of salbutamol at least subject to doubt. In the light of this, the implicit warning for the possible negative effects of salbutamol in the setting of acute asthma is unsubstantiated, particularly since this drug has been shown for many years to be of great clinical value in this potentially life threatening situation.

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β2 agonist/antagonist activity of salbutamol

The main conclusion of the paper by Drs Grove, McFarlane and Lipworth (February 1995;50:134–8) that in vivo salbutamol shows characteristics of a β2 adrenoceptor partial antagonist in a state of high adrenergic tone (exercise) is based upon the observation of similar effects of salbutamol and the non-selective β adrenoceptor antagonist, propranolol, in augmenting exercise-induced hyperkalaemia. The authors state that the mechanism of this augmentation of exercise-induced hyperkalaemia is by β2 adrenoceptor antagonism since propranolol and the selective β2 adrenoceptor antagonist ICI 118 551 would show similar effects in this respect. It is the latter assumption that we would like to challenge. We think that it is more likely that the effect of salbutamol on the exercise-induced increase in potassium is due to some other yet unknown mechanism.

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