LETTERS TO THE EDITOR

Allergic alveolitis due to goose feathers in a duvet

It is not often that reports of rare conditions appear in textbooks before journals (November 1992;47:990–1). Allergic alveolitis due to feathers in cushions, however, was first described in 1975 in a patient in whom I had made the diagnosis some years before.[1] Although I have not seen a similar patient since, I thought it worthwhile writing in Crofton and Douglas's 4th edition: "One potential source of antigen that may be overlooked is feathers in cushions or pillows".[2] Incidentally, the patient I looked after had been ill, and a puzzle to her doctors, for about eight years. She had precipitating antibodies to bird serum but no exposure to birds and it was only on my second visit to her house that I tumbled to the possible cause, subsequently confirmed by challenge with the cushion feathers. She was cured by getting rid of the cushions.

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Effect of dietary sodium on the severity of bronchial asthma

Drs D Lieberman and D Heimer (May 1992;47:360–2) reported the effect of three levels of salt intake on peak expiratory flow in patients with mild asthma. They found that a low dietary salt intake had no effect, although a low salt intake has been associated with a low prevalence of asthma in some epidemiological studies.

Reversal may be more difficult than prevention, and it is important to note that the mean sodium excretion rate of 84 mmol/24 hours recorded with their "low" salt diet is not particularly low for a therapeutic diet. In hypertension, for example, there is evidence that a mean sodium excretion rate of 90 mmol/day is very close to the upper limit for a measurable therapeutic effect.[1]

The "low" salt diet for asthma still provided more sodium than potassium, giving a 24 hour urinary Na/K ratio of 1·12. In Australia the recommended dietary intake for potassium is 50–140 mmol/day, with an added recommendation that the Na/K ratio should not exceed 1·0.[2] Potassium has a natriuretic effect and this recommendation is intended for the whole population, on the evidence that it would help to prevent hypertension. It would be interesting to know if the electrolyte balance of a truly low salt diet has relevance to the treatment or prevention of asthma.

We have a consumer advisory group producing literature and a newsletter to help subscribers to achieve a sodium excretion rate below 50 mmol/24 hours.[3] Correspondence is invited to Salt Skip, GPO Box 1717, Hobart, Tasmania 7001, Australia.

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Notification of tuberculosis: how many cases are never reported?

The article by CD Sheldon *et al* (December 1992;47:1015–18) highlights the difficulties and inaccuracies encountered in studies on the incidence and nature of tuberculosis based on notification systems. At present even small changes in the practice of notification could profoundly affect our concept of tuberculosis as a declining or increasing health problem.[1] Sheldon *et al* recommend, amongst other things, notification of all positive cultures by microbiologists (as in Scotland).

England and Wales, as well as Scotland, have excellent reference facilities in the form of the Public Health Laboratory Service Mycobacterium Reference Unit and the Regional Tuberculosis Centres. These laboratories receive almost all cultured mycobacteria from the regions that they serve and are therefore able to carry out detailed studies on the changing trends in disease due to tubercle bacilli and other mycobacteria, and to monitor changes in the incidence of drug resistance.[2-4]

Although providing an unequivocal diagnosis, bacterial culture is only positive in a proportion of cases of tuberculosis. Thus, surveys based on bacteriological studies suffer from the same problem as those based on the notification system. If, however, the two systems could be combined by means of a computer link a much clearer picture of tuberculosis in Great Britain would emerge. Such dual reporting would enable weaknesses in the notification system to be delineated and remedied and would also facilitate an audit of the efficacy of procedures for obtaining clinical specimens and for the primary isolation of tubercle bacilli and other mycobacteria from such specimens.

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Attenuation of exercise induced asthma by local hyperthermia

I read with interest the paper by Dr S L Johnston and colleagues (August 1992;47: 592–7) indicating that local hyperthermia (inspired air temperature 42°C) significantly decreased the response to exercise challenge and had a trend towards protection against histamine challenge at one hour.

As the authors mentioned, one possibility might be the interfering effect of increased temperature on local inflammatory mediators. That view is supported by our previous results, which showed that body temperature modulates the bronchial responsiveness caused by platelet activating factor (PAF) in the rabbit.[1] Infused PAF, which at normal body temperature induced significant bronchial hyperresponsiveness, caused considerable hyporesponsiveness to inhaled histamine at a higher core body temperature (41·2°C). The change in body temperature did not have a significant effect on the ability of PAF to induce neutropenia, thrombocytopenia, or to alter levels of hematocrit (an index of microvascular leakage), or plasma corticosterone. The known effects of temperature on PAF receptor affinity, an increase in metabolic breakdown of PAF, or reduced secretion of secondary mediators, may be plausible basic mechanisms.

Interestingly, there has also been a clinical report in which a decrease in body temperature was found to induce airways obstruction.[2] An increase in body temperature is also known to ameliorate asthma symptoms[3] and, in the past, an artificial fever was once used to treat asthma.[4]

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BOOK NOTICES

Year Book of Pulmonary Disease. Roger C Bone and Thomas L Petty. (Pp 513; US$59.95.) Chicago: Mosby-Year Book, 1992. ISBN 0 8151 3884 9.

This is an interesting but curious book. The Mosby-Year Book Company publish 43 year books. This one is a collection of abstracts of papers published in 57 journals during 1990–1 selected by two eminent American respiratory physicians, Drs Bone and Petty. The 18 chapters of the book represent a conventional division between the standard topics of chest medicine and sensibly include sleep apnoea, critical care,

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