Adrenocortical activity in children

I read with interest the report on adrenocortical activity in children receiving inhaled corticosteroids by Lipworth and colleagues1 but am perplexed by the reliance to the clinical practice. Although it is possible to demonstrate biochemical evidence of suppression of the hypothalamic-pituitary-adrenal axis with sensitive tests such as the measurement of overnight urinary cortisol excretion, this is likely to have little clinical significance. There is to date no evidence that inhaled corticosteroids taken at recommended doses have caused clinically significant adrenal suppression.

The major side effect of inhaled corticosteroids in childhood is growth suppression.1,2 In a group of children aged 7–9 years who received beclomethasone dipropionate at a dose of 400 µg/day there was no significant effect on overnight urinary cortisol. Despite this, there was clinically relevant growth suppression in those children who received beclomethasone dipropionate (difference of 1 cm in growth over seven months of study).3 I therefore feel that the authors’ conclusions are premature as no conclusions can be drawn from the safety profile of either budesonide or fluticasone propionate can be derived from this study.

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Fenoterol and asthma deaths

After reading the correspondence on this vexatious topic published in the August issue of Thorax I am more deeply puzzled than ever by the acrimony displayed by the Wellington and Auckland medical schools over the role played by fenoterol in the 1977/82 “epidemic” of asthma deaths in New Zealand. I visited that country in 1982 at the invitation of its Asthma Foundation which hoped I might be able to shed some light on the cause of the “epidemic” that had given rise to serious public concern. I suspected that a therapeutic innovation, available without prescription, analogous to that of Medihaler Iso Forte® in the UK, had crossed the lips of any of the many respiratory physicians I met during my visit to New Zealand in 1982. I was clearly given to understand that the only β2 agonists used in the treatment of asthma at that time were salbutamol and terbutaline. Had I been informed of the widespread prescription of fenoterol inhalers during the 1977/82 “epidemic” I would certainly have questioned its safety.

As I pointed out in a letter to the New Zealand Medical Journal published on 11 April 1990, one metered dose (then 200 µg) of fenoterol was equivalent to four metered doses (400 µg) of salbutamol. If a fenoterol inhaler was four times more dangerous than salbutamol inhaler, it is possible that use of a fenoterol inhaler in patients with severe acute asthma must have carried a similar risk to that of overuse of other β2 agonists delivered by Nebuliser and overuse of Medihaler Iso Forte® in the 1970s which must all have contributed to “epidemics” of asthma deaths.

I would urge my colleagues in New Zealand to accept that view, which may seem simplistic but carries more conviction with practical clinicians than the sterile semantics that have occupied so much space in medical journals during the past few years.

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5 Grant IWJ. Asthma in New Zealand. BMJ 1983;286:718.

Home environment and asthma

Butland and colleagues found that increased use of non-feather pillows was the only domestic indoor exposure that appeared to explain a 20% increase in the population prevalence odds of wheeze in children of 0–4 years. Croydon, London between 1978 and 1991.1 This extends their previous epidemiological studies on synthetic material-filled pillows as a risk factor for severe asthma in adolescence. In the study they hypothesised that material-filled pillows may release volatile organic compounds, possibly influencing the airway response to inhaled allergens.

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In their latest study no hypothesis for the association between wheezing and the use of non-feather pillows is put forward. Environmental exposures to allergens were not directly examined and, in particular, levels of the major house dust mite allergen Der p 1 in feather and non-feather pillows were not available. They discard the possibility that house dust mite allergens could be a reason for the increased prevalence of wheeze based on a study that showed no change in domestic reservoir levels of Der p 1 between 1979 and 1989. However, in that study Der p 1 levels were measured in dust samples from mattresses, bedroom floors, and living room carpets, not from pillows.

We have recently demonstrated and confirmed in a follow up study that synthetic pillows contain a mean of eight times more total Der p 1 allergen than feather pillows. Given that people spend many hours in close contact with pillows, it could be argued that exposure to higher levels of Der p 1 in non-feather pillows may explain the findings of Butland and colleagues relating the increased use of non-feather pillows with an increase in the prevalence of wheeze. Recent evidence shows clear correlations between exposure to house dust mite allergens in the bed and the severity of various parameters used in the measurement of asthma severity.

We agree with Butland and colleagues that further studies are required to explain the link between increased wheeze and the use of non-feather pillows — not only toxicological and epidemiological studies, as suggested by the authors, but further investigations of allergen levels in different types of pillows to determine whether our New Zealand based findings are applicable in the UK and elsewhere.

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NOTICES

Sixth Bronchitis Symposium

The Sixth Bronchitis Symposium on the link between asthma and COPD will be held in Groningen, The Netherlands on 24–26 August 1998. For further information contact the Secretariat Department of Pulmonary Diseases, PO Box 30,001, 9700 BB Groningen, The Netherlands. Telephone: +31 50 361 2357 or 3532. Fax: +31 50 361 9320. E mail: e.pick@int.azg.nl or c.w.verver@int.azg.nl.

CORRECTION

Survival of patients with severe α1-antitrypsin deficiency

In a paper entitled “Survival of patients with severe α1-antitrypsin deficiency with special reference to non-index cases” by Seersholm, Kok-Jensen and Dirksen published in Thorax in 1994 it has been pointed out by Dr James K Stoller of the Cleveland Clinic, Cleveland, Ohio, USA that the labels on the key of figure 1 on page 696 were transposed. A correct version of the figure is shown below.
