Asthma trends

Causes of wheeze and asthma may differ

EDITOR,—Two papers report the outcome of childhood asthma in Tasmania[1] and Melbourne[2] in subjects now in their 30s. We reported a 25 year follow up of schoolchildren in Aberdeen[3,4] and think that our findings influence the interpretation of these Australian papers.

In the 1964 random community survey that provided the baseline for our study, subjects were classified as having asthma, "wheeze in the presence of respiratory infection" (wheezy bronchitis), or no respiratory symptoms (comparison subjects).[5] Review after 25 years of subjects from each group showed that 61% of those who had had asthma in childhood continued to wheeze in adult life, compared with 30% of those who had had wheezy bronchitis; 11% of the comparison subjects had developed wheeze since the original study.

Of the subjects who had not had symptoms in childhood who were reviewed by Mark A Jenkins and colleagues, 10·6% had developed symptoms by the age of 29-32,[1] a similar percentage to that in our study. Of those who had had symptoms in childhood, 25·6% continued to experience symptoms as adults, a much smaller percentage than we had found. The reason for the difference from our results may lie in the ages at the time of the original studies: the Tasmanian children were identified at age 7, while ours were selected at 10 to 15, when a number of wheezy children would have already grown out of their symptoms. Another explanation may lie in the definition of symptoms in adults: Jenkins and colleagues defined them as the "occurrence of an asthma attack within the previous 12 months," which is a more stringent definition than that used in our study (wheeze in the past 12 months) or the study by Helmut Oswald and colleagues (wheeze in the past three years).[2] The Tasmanian survey of 1968 failed, however, to discriminate between children with asthma and those with "wheeze only in the presence of respiratory infection" (wheezy bronchitis), which we believe is the most likely reason for the apparent lower prevalence of symptoms in the adults in their study.

Oswald and colleagues report that 36% of those with wheezy bronchitis in childhood continued to wheeze as adults. This result was similar to ours, although the percentage they reported for asthmatic children with symptoms persisting at age 30 was higher (78%), probably because the Melbourne sample was "enhanced" at age 10 by the addition of 79 children who suffered from more severe asthma.

Our finding that the natural course of wheeze in the presence of infection differed from that of asthma led us to hypothesise that the pathogenesis of these two conditions may also differ. We believe that the results presented from the two Australian studies are compatible with our hypothesis.

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Different nebulisers may explain Australian findings

EDITOR,—Jennifer K Peat and colleagues report that the prevalance of airway responsiveness increased 1·4-fold to twofold over 10 years in children in two separate towns in New South Wales.[1] We believe that an important confounding factor that could account for at least some of the observed increase was the change in the nebulisers used to test airway responsiveness in the two surveys in the study: DeVilbiss glass hand held 40 nebulisers were used in 1982 and DeVilbiss 45 plastic hand held nebulisers in 1992. The same group reported no increase in the prevalence of airway responsiveness in an adult population over nine years when there had been no change in the challenge protocol.[2]

The output of a nebuliser is usually measured by weight loss per activation, but this has been shown significantly to overestimate the output of a variety of jet nebulisers because of concomitant evaporative water loss.[3] Using a fluoride tracer technique, we have shown that this is also true for both types of DeVilbiss hand held nebulisers. Furthermore, although weight loss was similar for both types of nebuliser, the true drug output of the DeVilbiss 45 nebuliser was almost twice that of the DeVilbiss 40 nebuliser.[4]

Another important performance characteristic of nebulisers that may differ between these two types is the droplet size of the aerosol generated. Certainly, less than half of the output of the DeVilbiss 40 nebuliser has been shown to be of respirable size—that is, <6 μm mass median diameter[5]—and this proportion will probably be higher for the more efficient, newer DeVilbiss 45 nebulisers.

It would be interesting to compare the prevalence of positive results of histamine challenge tests in 1982 with the proportion of children who had a 20% drop in forced expiratory volume in one second either before or at the dose step of 1·95 μmol histamine in 1992—that is, the maximum "real" dose that we believe was delivered in the first study.

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Optimal treatment includes inhaled steroids

EDITOR,—Peter D Phelan makes some alarming assertions in his editorial on the epidemiology of asthma in children.[1] Firstly, he claims that there is no evidence to support the use of inhaled corticosteroids long term. Recently, however, Agertoft and Pederson reported significant reductions in annual admissions to hospital and improvements in the forced expiratory volume in one second in over 200 asthmatic children who used inhaled budesonide for three to six years.[2]

Secondly, optimal long term control of the disease requires early treatment with inhaled steroids.[3] Forced expiratory volume in one second is significantly higher in children who receive budesonide within two years of the onset of asthma.

Thirdly, many studies have raised the question of suppression of growth. This concern is not new and has resulted in guidelines on the use of spacer devices for high doses of inhaled steroids to reduce systemic absorption. Although in some studies there seems to be some constitutional delay, long term studies have shown no significant changes in growth velocity or weight gain.[4,5]

Having documented tests of adrenal function in more than 60 asthmatic children over the past seven years, we have found no biochemical evidence of adrenal suppression at doses of 400 μg of inhaled corticosteroids and only one impaired result of a short tetracosactrin test at doses >1200 μg. We therefore hope that adequate control of asthma will continue to be seen as