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

paramount as the deleterious effects of poorly controlled asthma are generally accepted to exceed the theoretical risks of treatment with inhaled steroids.

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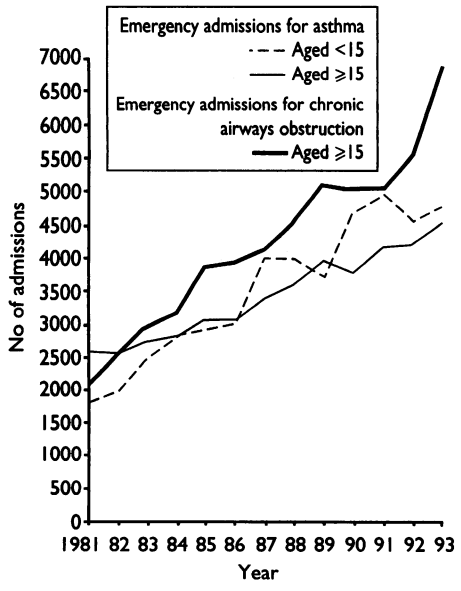
Emergency admissions increasing in Scotland

EDITOR,—S J Hyndman and colleagues have reviewed trends in admissions to hospital for acute asthma in East Anglia over the past 15 years and have identified a flattening of the rate of increase.[1] They have been hampered in their comparison with the general experience in England and Wales by changes in the system for gathering information.

Continuous data on admissions to hospital in Scotland are available from Scottish morbidity records collated centrally since the early 1960s. The data for admissions for asthma (strictly, discharges) have recently been reviewed, and the figure shows the trend in the number of admissions, for patients aged < 15 and ≥ 15, during 1981-93. The figure shows no evidence of any flattening in the rate of increase in emergency admissions for asthma in Scotland over this period.

Hyndman and colleagues review possible reasons for change in admission rates. Locally, there is no evidence either that patients with milder asthma are being admitted[2] or that the upward trend is due to a change in diagnostic preference from "chronic airways obstruction," for which admissions are also continuing to rise (figure). That readmission rates are stable in Scotland is suggested by the fact that the number of patients as a proportion of the number of admissions each year is stable at 71-75% for those aged < 5 and 78-81% for those aged ≥ 15.



Numbers of emergency admissions for asthma and for chronic airways obstruction (not elsewhere coded), Scotland, 1981-93.

The underlying causes of the observed flattening in the rate of increase in admissions to hospital in East Anglia are worth exploring further, not least to shed some light on whether this flattening indicates good practice in the care of patients with asthma or the opposite.

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Thunderstorm peak in Luton

EDITOR,—Virginia Murray and colleagues draw attention to the widespread impact of a thunderstorm on the night of 24 June on the number of patients with asthma attending accident and emergency departments across the south east of England.[1] My experience in general practice was similar. I was on call for the local deputising service at the time. The thunderstorm occurred in Luton between 2100 and 2200, and the first call for a visit to a patient with asthma occurred at 2120. In all, 23 calls out of 56 were to patients with asthma. Most of these patients complained of hayfever with difficulty in breathing, implying that they were not usually accustomed to wheeze.

Attendances at the accident and emergency department of Luton and Dunstable Hospital reflected the same pattern: 24 of 89 patients attended with asthma or acute shortness of breath. On the morning of 25 June five of the 10 patients who attended the surgery complained of asthma, which had started at the same time as the thunderstorm.

If my experience was repeated across the rest of the country there was a huge increase in cases of acute asthma over a short period; study of this would be well worth while.

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Somatostatin in gastroenterology

More studies needed

EDITOR,—We noted with concern the enthusiasm with which A Shulkes and J S Wilson endorse the use of somatostatin and its analogue octreotide in gastroenterology.[1] In particular, we have reservations concerning the reported role of octreotide in the management of enterocutaneous fistula since, despite a successful pilot study,[2] more studies seem warranted to clarify the timing and the cost-benefit of such treatment. Indeed, more recent studies of patients with such fistulas have failed to show any therapeutic advantage of octreotide.[3]

The work cited by Shulkes and Wilson was not, as they say, a controlled trial of octreotide but was a trial of a continuous intravenous infusion of native somatostatin.[4] The impracticality and financial implications of such treatment, which was associated with only a six day improvement in the time taken to healing, seem difficult to justify. In addition, Shulkes and Wilson fail to take into account that the article quoted lacks relevant clinical information, such as the timing of the infusion of somatostatin, the causes of non-closure of the fistulas, and the indications for stopping treatment and for reoperation. Furthermore, four patients were transferred from the group receiving placebo to the group receiving somatostatin after 15 days of total parenteral nutrition.

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Authors' reply

EDITOR,—We stated in our editorial that a "lack of randomised controlled trials with sufficient numbers" had "hampered clinical progress" with respect to accurate delineation of the role of somatostatin and its analogues in gastroenterology. The data from G L Carlson and colleagues illustrate the problem. In 1987 they concluded, after a blind crossover trial in 14 patients, that "octreotide significantly reduces enterocutaneous output and accelerates their spontaneous closure."[1] In 1989 they confirmed the success of the initial trial with a study of 27 patients, this time calling octreotide a "significant advance in the conservative treatment of . . . enterocutaneous fistulas."[2] In 1993, however, they seem to have resiled from their initial position after a trial in only 19 patients, published in a regional journal.[3]

We were aware of the apparently contradictory publications of Carlson and colleagues, but we found the multicentre trial of Torres *et al* to have been carefully conducted and to have contained the largest number of patients (20 in both the somatostatin and control groups),[4] and hence we cited it.

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