LETTERS TO THE EDITOR

Atopy, bronchial responsiveness, and symptoms in wheezy 3 year olds

EDITOR,—There is now increasing evidence that most children with recurrent wheeze in the first year of life do not have atopic asthma and that ‘wheezy bronchitis’ is a distinct entity with a different natural history. Although it is now clear that infants are able to respond to bronchodilators, it is well known that β agonists are rarely effective in this age group during wheezing episodes. Despite this, it is still widely believed that bronchospasm is the main cause of wheeze in these children. The recent paper by Nicola Wilson et al suggests that non-atopic infants who wheeze have greater bronchial responsiveness than atopic infants.1 In contrast, a recent study from Perth could find no evidence of increased bronchial responsiveness in recurrently wheezy infants when compared with matched controls.2 The group from Perth suggest that their results support the notion that diminished lung function present from birth predisposes infants to lower respiratory tract symptoms.

An alternative explanation may be that excessive mucus production with or without mucosal oedema is predominantly responsible for the symptoms experienced by infants with wheezy bronchitis. This would explain why antihistamines often appear more effective than β agonists. It would also explain why inhaled steroids are frequently of benefit in patients with recurrent or persistent respiratory symptoms as steroids inhibit both the production and release of mucus. Coarse ‘ruttly’ upper airway noises suggestive of mucus secretions are extremely common in young infants and many recurrently wheezy children have ‘ruttles’ between exacerbations. ‘Noisy breathing’ is reported as being always present in 11% of children and they frequently have symptoms from birth. It may well be that infants with wheezy bronchitis have an increased capacity to secrete mucus in response to stimuli such as virus infections, either because of a tendency from birth or as a response to environmental events such as ventilation in the neonatal period, acute bronchiolitis, or ‘passive smoking’. Increased baseline production of mucus might explain the increased airways resistance seen in these infants.1 Anticholinergics will inhibit mucus production from submucosal glands but will have no effect on goblet cell secretion and therefore cannot be expected to relieve totally symptoms in those with prominent goblet cell hyperplasia. These are more likely to be the ones with increased capacity to secrete mucus due to stimuli such as passive smoking and those with ‘postbronchiolitic symptoms’. It is possible that treatment of these infants, if any is required, would improve if we consider using drugs which inhibit mucus production or newer mucolytics.


Dr Wilson comments: I was interested in Dr Everard’s suggestion that the syndrome of ‘wheezy bronchitis’ might be explained by increased mucus production rather than bronchospasm. He may well have a point. However, contrary to his implication, we did not propose that the increased bronchial responsiveness found in the non-atopic children in our study was responsible for their virus induced wheeze. We concluded that the episodic wheeze associated with colds in the first three years of life, in this hospital based population, was a characteristic of asthma and non-atopics, apart from the age of onset. Often, early asthma precipitated by viral infections can only be distinguished from wheezy bronchitis retrospectively.

It is therefore very doubtful whether the syndrome of wheezy bronchitis can altogether be explained on the basis of mucus production.


Dr Brett and coauthors comment: We read with interest the comments of W H Nikolazik and M H Schöni. Their comment that after one year in the trial there was no difference between FEV_1, FEF_25–75, FEV_1/FVC and FEV_1/FRC in treated and control groups is quite correct, but the data in table 2 show that on entry to the trial both these values were worse in the treated group. Both in the observation group, hence during the trial period both these parameters improved in the treated group and deteriorated or remained stationary in the observation group. Other parameters (for example serum IgE titre, white cell count, % neutrophils) showed a similar pattern.

We are unclear about the exact meaning of the comment on longitudinal changes: we give absolute values of parameters on entry and after one year, from which changes can be calculated. The statistics and conclusions are not affected by the method of presentation, although we thought our choice made the results easier to follow. We are also unsure what is meant by the suggestion that we should ‘reveal all the essential data not yet mentioned’—the parameters given in tables 1 and 2 (as means with the range) were chosen as being the most meaningful in monitoring the progress of pulmonary infection from the more than 20 which we routinely monitor. We would agree that p<0.10 is not generally considered significant and had no reason otherwise when we included it, for completeness, in table 2.

Audit of screening for congenital hypothyroidism

EDITOR,—We read with interest the audit of a screening programme for congenital hypothyroidism.1 From the data available the birth prevalence for congenital hypothyroidism in the period of study was calculated to be 1:3770. The authors state ‘This level of prevalence is similar to 1:3980 found from 36 centres in 12 European countries and the 1:4200 reported from the USA, which suggest that it is unlikely that a significant number of cases, other than the known false negatives, were missed’. There are a number of problems with this assumption:

(1) In the audit 62 cases were detected out of 228 289 livebirths over a period of seven years, giving a birth prevalence of 1:3682 and not 1:3770 as stated in the article.

(2) If the 95% confidence intervals for the data presented were to be worked out it would give the possible range of birth prevalence as 1:2872 to 1:4803 (using the Poisson distribution). This means, that for 228 289 livebirths, the region could have had between 48 and 79 cases of congenital hypothyroidism. Taking the upper limit would imply that the programme could have missed up to 19 cases—that is, false negatives that this audit picked up and 17 others that were not.

(3) An examination of the data from European countries mentioned in the statement above showed that 14 European countries (37 centres) had a collective birth prevalence of 1:3598 but there was intercountry variation from 1:2860 in the Netherlands to 1:5770 in Austria.7 There is nothing to suggest that the


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M Everard

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