LETTERS TO THE EDITOR

Changes in asthma prevalence

Prevalence of asthma symptoms and percentage fall in peak expiratory flow rate (PEFR) with exercise in 1973 and 1988

	1973 (n=817) %	1988 (n=965) %	1988–1973 / 1973 %
Asthma ever	5·5	12·0	118
Wheeze ever	17·0	22·3	31
Wheeze in the past 12 months	9·8 (n=812)	15·2 (n=960)	55
>45% fall in PEFR on exercise	0·4	1·3	238
45–36% fall in PEFR	0·5	1·0	111
35–26% fall in PEFR	1·1	1·7	60
≤25% fall in PEFR	98·0	95·9	−2

SIR,—There have been many studies of asthma prevalence, though the widely varying methods used makes comparisons over time difficult. Anderson reviewed prevalence surveys of wheezing illness in the United Kingdom and found the data did not support the assertion that there has been an increase in the proportion of the child population who experience wheezing illness.[1]

The repeat survey by Burr *et al* used identical methodology to their earlier survey and they also avoided the problem of changes in illness labelling.[2] Their study is very important to our understanding of trends in asthma mortality and morbidity. The authors of this excellent study might have placed more emphasis on the appreciable prevalence changes in the severe end of the asthma spectrum. When looked at in this way, the study provides support for a small increase in the prevalence of wheezing illness and more noticeable increases in asthma labelling and current wheeze (past 12 months). It also showed that the percentage increase in the proportion of the population with a fall in peak expiratory flow rate with exercise was greater the larger the fall in peak expiratory flow rate (table), suggesting asthma is becoming more severe. This disproportionate increase in the prevalence of severe asthma is consistent with surveys of hospital admissions in both New Zealand[3] and the United Kingdom,[4] which have found evidence for an increase in admission rates for severe asthma in children.

There is an urgent need to explain the increase in prevalence of severe asthma and this must include the possibility that current therapy has an adverse effect on morbidity.[5]

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Nutrition in cystic fibrosis

SIR,—In response to Dr David's excellent and comprehensive review of current management in cystic fibrosis,[1] we would like to report our own experience in dealing with nutritional aspects of the disease. This is important as malnutrition may affect pulmonary function,[2] and nutritional repletion not only improves linear growth but also can be shown to produce a reversal in the trend for deteriorating lung function[3] and aid immune competence.[4]

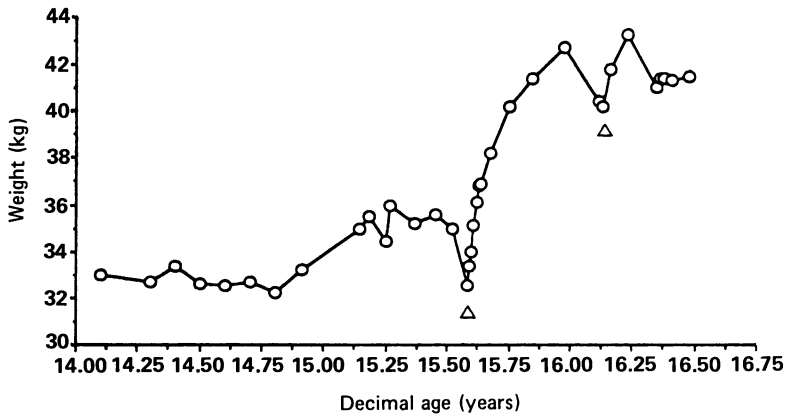
We have not found any advantage in the use of protein hydrolysates for infants requiring formula feeds. Although on paper such feeds composed of amino acids/peptides, glucose polymers, and medium chain triglyceride should be absorbed without added pancreatic enzyme supplements, in our experience diarrhoea is better controlled and weight gain enhanced with supplements. Casein hydrolysates that have a high carbohydrate content and relatively low energy density (2·814 kJ/ml or 0·67 kcal/ml), offer little benefit over cheaper standard baby milks of similar or higher energy density that are readily supplemented with glucose polymers or carbohydrate/fat mixtures to achieve energy densities of 3·36–4·2 kJ/ml (0·8–1·0 kcal/ml).

We have also found overnight enteral feeds useful in maintaining or improving nutrition. Many centres are, however, reluctant to employ nasogastric feeding because of patient resistance, concerns that tubes will be coughed up, and problems with enzyme delivery. Our experience is that polyurethane tubes are well tolerated providing medical and nursing staff and parents adopt a positive attitude. Percutaneous gastrostomy may be more acceptable for patients requiring long term nutritional support but gastrostomy is not without complications. Specialised feeding formulas are usually adopted to avoid giving enzyme supplements. Alternatively, many centres wake the child once or twice through the night to administer extra enzymes, yet satisfactory weight gain can be achieved using whole protein feeds by giving enzymes at the start and finish of a continuous overnight feed as the following case report illustrates.

Case report

A girl of 14 years had gained weight poorly for more than four years, falling from the 10th to well below the 3rd centile despite encouragement to take a high energy, high protein diet, nutritional supplements (Fresubin, Fresenius), and the use of a microsphere pancreatic enzyme preparation. She had chronic lung disease (forced expiratory volume in one second <30% predicted) and *Pseudomonas aeruginosa* colonisation. After admission with an increased productive cough and weight loss a fine bore 'Silk' nasogastric tube (Silk and Corsafe 6 French gauge 56 cm; E Merck Ltd) and Kangaroo 330 enteral feeding pump (Sherwood Medical) were used to deliver an overnight (10 hour) feed (Fortison Energy Plus; Cow and Gate) which, after four days, provided 65% of her recommended daily intake for energy and 86% of recommended daily intake for protein (6370 kJ (1500 kcal) and 50 g protein). Pancreatic enzymes (Pancrease, Cilag) were given at the start and end of the feed period only in a dose equivalent to that used with main meals. Although the patient had a chronic cough the tube, which was left in situ throughout the feed period, was well tolerated. Diarrhoea did not occur despite the use of a 'non-elemental' feed and weight gain was rapid (see figure). After a two week course of intravenous antibiotics the patient was discharged to continue overnight feeds at home. An increase of 10·2 kg was achieved over five months.

Continuous overnight nasogastric feeding without frequent enzyme supplementation appears a simple solution to nutritional



Effect of supplementary nasogastric feeds on weight gain. Feed period is indicated by arrows.

deficiency in patients with cystic fibrosis, which can easily be performed in the home.

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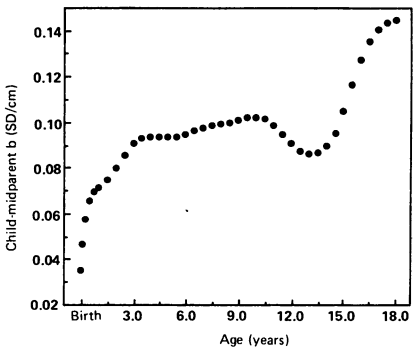
Parent-specific evaluation of growth

SIR,—We have read with interest the recent paper by Dr Sorva and colleagues, concerning reference data for stature of children conditional upon the statures of parents and that of the child at 1 year of age.[1] We are troubled by the use of data of unknown validity and reliability as the basis of reference data. Common protocols were not used for measuring children, it is unknown whether recumbent length or erect stature was measured at the youngest ages, and all parental and some of the child statures were self reported rather than measured. Each of these deficiencies will, to an unknown degree, add random error and perhaps bias. We and others have documented appreciable bias as well as increased random error when using reported statures.[2] More practically, the added random error will attenuate correlations and regression coefficients that are the bases of the equations estimating standard deviation scores for children. While the authors acknowledge their less than perfect data, there remains concern over the validity of the findings.

The final equations for children (their table 2) are to be applied irrespective of the age of the child. In our investigations on the Fels Longitudinal Study, we found that the pattern of parent-child relationships for stature changed so greatly across age that it was necessary to consider age.[3] This can be appreciated from the figure, which presents the regression coefficients (b) of standard deviations in length (birth to 2·5 years) and stature (3·0 to 18 years) of boys, relative to the mid-parent stature (average of maternal and paternal statures). Clearly, there is systematic and non-linear age covariance in the midparent-child statural relationship scaled according to standard deviation scores in child stature. Unfortunately, Dr Sorva and colleagues do not provide any conventional summary or diagnostic statistics allowing evaluation of the adequacy of fits for the equations in their table 2 across the entire age range; those relating to final stature in their table 3 are insufficient for this purpose.



Regression coefficients (b) of standard deviations in length and stature relative to midparent stature.

We have provided previously a method of adjusting recumbent length or stature of children (birth to 18 years) according to parental statures.[3] All measurements of children and parents were taken under standardised research conditions. The parent-specific adjustments were specifically normed relative to the internationally recommended reference data from the US National Center for Health Statistics, and US national estimates of parental statures. Exact estimation equations, computer programs,[3] and simplified tables for clinical ready reference[4] have been presented. We agree with Dr Sorva that a valid method of parent-specific evaluation of stature in children provides an important clinical tool to separate the normal genetic contribution of parental stature to stature of children from other factors that affect stature such as malnutrition and disease.

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The taste of milk

SIR,—One in four bottle fed babies change their milk formula in the first weeks of life, often in response to non-specific symptoms.[1–3] This tendency is common in Portugal too. Often, the substituted milk has a similar composition to the original, but is a different brand. In this situation, mothers often express satisfaction with the switch to the new milk.

I cannot help reflecting that three out of four bottle fed babies accept, without reluctance, the very same type of food with no variation in composition (or presumably taste) six times daily during early infancy. This monotony, which is contrary to the most basic rule of dietary habits, might just prove too much for one in four babies—at least we should give the matter some further thought.

I presume that the evolution of the taste learning process has developed through breast feeding. It is well known that the composition of maternal milk is subject to important variations, which are part influenced by the maternal diet. Are there similar variations in the taste of maternal milk, thereby making mealtimes somewhat more exciting for babies? Might such variations in taste, during early infancy, facilitate weaning and the natural appetite for a varied diet in toddlers? Conversely, is there any evidence that bottle feeding is associated with a narrow 'taste span' in toddlers?

For those babies who are not breast fed, perhaps there is nothing wrong with encouraging various brands of milk according to the whim of babies (and their mothers).

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