LETTERS TO THE EDITOR

Screening for growth: towards 2000

EDITOR,—Jefferson and Forster devote 15 column inches to the virtues of growth monitoring but offer just four lines of 'results'.1 However, they introduce a protocol that refers any child with a growth velocity below the 25th or above the 75th centile to 'generate appropriate referrals and not swamp the system'. We would have thought that by definition 50% of children would grow outside these centile limits.

The issues raised by growth monitoring are not simple, however much we may wish them to be so. 'Children cannot be relied upon to grow along the centile lines on the standard growth charts—they deviate from them both up and down.'

This can be illustrated by examination of a commonly quoted reason for monitoring height in prepupal schoolchildren—the identification of girls with Turner's syndrome. The figure shows the growth curves for these girls superimposed on the 1990 normal centile chart. Note that between the ages of 5 and 9 the curve for the taller girls (those who would not be identified by a single measurement) crosses less than one centile channel (0-67 SDs on these charts). At least one normal girl in every 50 will cross one complete centile channel over this time span (calculated by Cole from French and English longitudinal data2 3). Turner's syndrome has been reported to occur in one girl in every 2000, and probably at least half of these are identified at birth or in the preschool years. There is therefore perhaps one undetected case of Turner's syndrome in every 4000 girls at 5 years of age and it follows that at least 80 normal girls must be referred for every case of Turner's syndrome discovered.

Dr Jefferson comments:

Dr Cole and Professor Hall have 'impeccably' measured our column inches accurately; would that the profession measured children metrically, regularly, and as diligently.

The use of height velocity in most height monitoring programmes is over two year periods which lessens the effect of measurement inaccuracy and year to year variation. The probability of two successive yearly velocities in a normal child falling below the 25th centile is around 0·25×0·25 (0·0625) units. That is, only 6·25% of healthy children will grow that slowly over a whole two year period.

It is the experience of paediatricians measuring children regularly that normal children do not deviate from their centile lines and sequential measurements are highly correlated as is shown by the original 'Tanner data.'

The illustrated Turner normal data superimposes 'old' Turner's syndrome data on the new 1990 normal charts and may not take account of secular trend in the Turner's syndrome group but it does show that at least 50% of girls with Turner's syndrome are not identifiable by height or height velocity alone before the age of 5 years. Therefore continual monitoring (as outlined in our letter) is necessary to detect these individuals and, just as importantly, others with non-endocrine, chronic disease/deprivation not identifiable by other means.

We and many of our colleagues would strongly support a similar kind of systematic study to look at the benefits of growth monitoring before the measurements at 7 and 9 years are deleted from the child health surveillance protocol.

1 Brook CGD, Hindmarsh PC, Healy MJR. A better way to detect growth failure. BMJ 1986; 293: 1186.

Transitent gluten intolerance

EDITOR,—In a recent paper Meuli et al described genetic differences in HLA-DR phenotypes between children with coeliac disease and those who presented with transient gluten intolerance.1

The latter children had a history of small intestinal mucosa after gluten challenge.2

There were 16 of these children and they continued on a normal gluten containing diet for a period of five to 15 years with the small intestinal mucosa remaining normal. They were described as having transient gluten intolerance. It seems a pity to introduce yet another name for the syndrome called temporary in the classic paper of McNeill et al2 but in all ESPGAN reports has been referred to as transient gluten intolerance.3-5

Establishment of a firm diagnosis of transient gluten intolerance is central to this report.

These cases do not fulfil the very strict criteria for this syndrome as described by McNeill et al and cited by the authors.2 There was no initial proof of gluten intolerance by a rigorous challenge as McNeill et al advocated. However, they are consistent with the less strict practical criteria that I have published,6 which leave some doubt about a final diagnosis. Furthermore the authors do not allude to the observations of Polanczo and Larrauri who have pointed out the difficulty of ever certainly excluding the diagnosis of coeliac disease in such patients.7

They have described five children who took five to nine years to relapse after return to a normal gluten containing diet. Thus for reasons unknown, delay in relapse may take several years, after the reintroduction of gluten to the diet. Thus while it is possible, that the children described did have transient gluten intolerance, it is also possible some in time will prove to have coeliac disease. Although the evidence that there is a genetic difference in HLA phenotype between the two groups of children argues in favour of a distinction from coeliac disease. It is yet possible that the 16 children are a heterogenous group. Some indeed may have had coeliac disease for example those who had the DR3/DR7 phenotype and will eventually relapse.

The authors do not tell us whether all 16 children were under the age of 2 years at presentation. This is of some importance for the validity of the revised ESPGAN criteria for coeliac disease.8 If there were any children diagnosed as having transient gluten intolerance aged more than 2 years at the onset of symptoms, yet who have not relapsed after many years of gluten ingestion, this observation would challenge the validity of the revised criteria. Such a finding does not appear to have been published before.

J A WALKER-SMITH

University Department of Paediatric Gastroenterology, Royal Free Hospital, Pond Street, London NW3 2GQ


D M B HALL

Department of Paediatrics, Florence Nightingale Children's Hospital, Western Bank, Sheffield S10 2TH

REFERENCES


5 "The SD of the change in height SD score between two ages is given by [(SDr×r) + (SDt×t)] where r is the corre- lation between height SD score at the two ages. For ages 5 and 9 years this correlation is about 0·964,4 so the SD of the change in height SD score between 5 and 9 years is 0·35, and the 95% confidence interval is 2·20-0·56-0·70 units. That is, about one channel width (0·67 units on the 1990 charts) so 2-3% of children can be expected to fall (and an equal proportion to rise) by more than one channel width between 5 and 9 years.

6 Dr Cole and Professor Hall have 'impeccably' measured our column inches accurately; would that the profession measured children metrically, regularly, and as diligently.

7 The use of height velocity in most height monitoring programmes is over two year periods which lessens the effect of measurement inaccuracy and year to year variation. The probability of two successive yearly velocities in a normal child falling below the 25th centile is around 0·25×0·25 (0·0625) units. That is, only 6·25% of healthy children will grow that slowly over a whole two year period.

8 It is the experience of paediatricians measuring children regularly that normal children do not deviate from their centile lines and sequential measurements are highly corre- lated as is shown by the original 'Tanner data.'

9 The illustrated Turner normal data superimposes 'old' Turner's syndrome data on the new 1990 normal charts and may not take account of secular trend in the Turner's syndrome group but it does show that at least 50% of girls with Turner's syndrome are not identifiable by height or height velocity alone before the age of 5 years. Therefore continual monitoring (as outlined in our letter) is necessary to detect these individuals and, just as importantly, others with non-endocrine, chronic disease/deprivation not identifiable by other means.

10 We and many of our colleagues would strongly support a similar kind of systematic study to look at the benefits of growth monitoring before the measurements at 7 and 9 years are deleted from the child health surveillance protocol.

1 Brook CGD, Hindmarsh PC, Healy MJR. A better way to detect growth failure. BMJ 1986; 293: 1186.
Acyclovir in chickenpox

EDITOR,—Virological evidence for the reactivation of chickenpox contracted in infancy has recently been reported and is related to the immune status of the host. Secondary attacks of chickenpox and early reactivation as zoster have been reported after the treatment of normal children with chickenpox suggesting that the immune response may be impaired after acyclovir treatment. We report the case of severe primary varicella infection in an infant who should have been protected by passive maternal antibody. His mother had been treated with acyclovir for chickenpox before delivery. A 25 year old woman presented at 38 weeks' gestation with a vesicular rash. The diagnosis of chickenpox was confirmed by the detection of specific IgM antibodies to varicella zoster virus and she was treated with acyclovir 800 mg five times daily for seven days. Nine days after the development of the rash she delivered a healthy boy. Six days after delivery he developed a vesicular rash and fever, and varicella zoster virus was detected in vesicular fluid. He was successfully treated with a five day course of acyclovir (100 mg/kg per day).

This infant was born nine days after his mother developed chickenpox and, in accordance with current guidelines for the UK, he did not receive passive immune globulin. We postulate that the use of acyclovir to treat the mother's infection may have affected her immune response to the virus leading to reduced passive transfer of immunity to her fetus. When he was born he was at increased risk of varicella infection, which he subsequently developed. This case highlights concerns over the effect of acyclovir on the immune response to chickenpox and also suggests that the present guidelines for passive immunisation against varicella zoster virus may leave a proportion of infants born to mothers treated with acyclovir at unnecessary risk.

PETER J JENKS
JUDITH BREUER
Department of Virology, Royal Hospitals NHS Trust, 37 Ashfield Street, London E1 1BB

Infant length measurements

EDITOR,—Like Professor Frank Falkner I was interested to read Dr Doull's article on the reliability of infant length measurement,2 though a little disappointed to find no reference to the Neonatometer—an instrument for measuring crown-heel length in infancy designed and written up by Bob Holding (from Holtain Ltd) and myself 24 years ago1 in the Archives. This particular invention was given to the technique in the training of observers with the neonatometer, '95% of all observations of crown-heel length were likely to lie between plus and minus 3.4 mm of the true value'. These represented accuracy, length is important — not only for the more immediate assessment of growth status but also to help evaluate a problem of growth in an older child by looking back at earlier measurements.

D P DAVIES
Department of Child Health, University of Wales College of Medicine, Heath Park, Cardiff CF4 4XN


Transient gluten intolerance.

J A Walker-Smith

Arch Dis Child 1996 74: 183-184
doi: 10.1136/adc.74.2.183-b

Updated information and services can be found at:
http://adc.bmj.com/content/74/2/183.3.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/