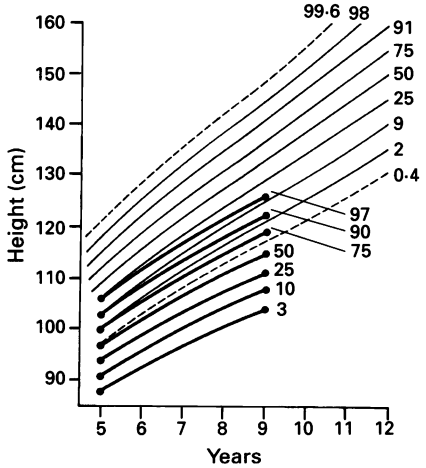
LETTERS TO THE EDITOR

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] We would like to know how a protocol that refers any child with a growth velocity below the 25th or above the 75th centile can '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 prepubertal schoolchildren – the identification of girls with Turner's syndrome. The figure shows the growth curves for these girls[2] superimposed on the 1990 nine 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 data[3 4*]). 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.



Curves for girls with Turner's syndrome superimposed on the 1990 nine centile height chart.

Growth monitoring, like many other aspects of child health surveillance, involves a search for needles in haystacks. That does not necessarily mean that it should not be done but, as with so many other areas of medical care, common sense choices do not always work out so well in practice. The downside of ineffective monitoring and screening is not only economic – more important are the worry generated, the waste of scarce professional expertise and, not least, the waste of parents' time. What we need from our endocrinology colleagues is not emotive pleas but a multicentre systematic study of the contribution made by growth monitoring to the earlier detection of growth disorders.

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1 Jefferson IG, Forster L. Screening for growth: towards 2000. *Arch Dis Child* 1995; **73:** 87.
2 Lyon AJ, Preece MA, Grant DB. Growth curve for girls with Turner's syndrome. *Arch Dis Child* 1985; **60:** 932–5.
3 Cole TJ. Growth charts for both cross-sectional and longitudinal data. *Stat Med* 1994; **13:** 2477–92.
4 Bailey BJR. Monitoring the heights of pre-pubertal children. *Ann Hum Biol* 1994; **21:** 1–11.

*The SD of the change in height SD score between two ages is given by $\sqrt{2}(1-r)$, where r is the correlation between height SD score at the two ages. For ages 5 and 9 years this correlation is about 0·94,[3 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 $\pm 2\times 0{\cdot}35 = 0{\cdot}70$ units. This 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.

Dr Jefferson comments:

Dr Cole and Professor Hall have 'imperially' 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 only around $0{\cdot}25\times 0{\cdot}25$ (0·0625), that is, only 6·25% of healthy children will grow that slowly over a whole two year period.[1]

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 large multicentre 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 Brooke CGD, Hindmarsh PC, Healy MJR. A better way to detect growth failure. *BMJ* 1986; **293:** 1186.

Transient 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 previously had a clinical presentation consistent with coeliac disease accompanied by hyperplastic villous atrophy followed by recovery on a gluten-free diet. The latter children had a normal small intestinal mucosa after gluten challenge.[1] 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 transitory gluten intolerance. It seems a pity to introduce yet another name for the syndrome called temporary in the classic paper of McNeish *et al*[2] 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 paper.

These cases do not fulfil the very strict criteria for this syndrome as described by McNeish *et al* and cited by the authors.[2] There was no initial proof of gluten intolerance by early gluten challenge in these children, as McNeish *et al* advocated. However, they are consistent with the less strict practical criteria that I have published,[6 7] which leave some doubt about a final diagnosis. Furthermore the authors do not allude to the observations of Polanco and Larrauri who have pointed out the difficulty of ever certainly excluding the diagnosis of coeliac disease in such patients.[8] 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 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.[5] 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.

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1 Meuli R, Pichler WJ, Gaze H, Lentze MJ. Genetic difference in HLA-DR phenotypes between coeliac disease and transitory gluten intolerance. *Arch Dis Child* 1995; **72:** 29–32.
2 McNeish AS, Rolles CJ, Arthur LJH. Criteria for the diagnosis of temporary gluten intolerance. *Arch Dis Child* 1976; **51:** 275–8.
3 Meeuwisse GW. Diagnostic criteria in coeliac disease. *Acta Paediatr Scand* 1970; **59:** 461–3.
4 McNeish AS, Harms K, Rey J, Shmerling DH, Visakorpi J, Walker-Smith JA. Re-evaluation of