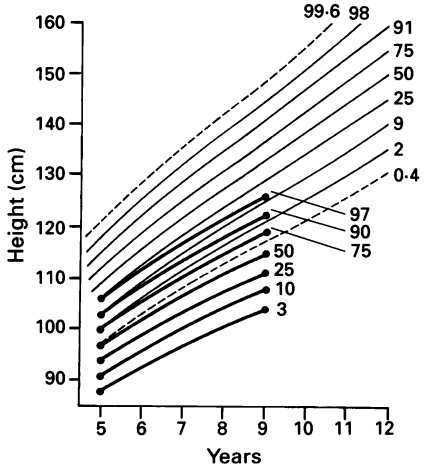
LETTERS TO THE EDITOR

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.
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*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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diagnostic criteria for coeliac disease. *Arch Dis Child* 1979; **54:** 783–6.
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7 Walker-Smith JA. Transient gluten intolerance. Does it exist? *Neth J Med* 1987; **93:** 1356–62.
8 Polanco I, Larrauri J. Does transient gluten intolerance exist? Kumar PJ, Walker-Smith JA, eds. *Coeliac disease: 100 years.* Leeds: University Printing Service, 1989: 226–31.

Toledo type brachyolmia

EDITOR,—I read with interest the paper by Grain *et al* dealing with the first UK case of a type of brachyolmia (short trunk) which is associated with both peripheral corneal punctate opacities only seen by slit lamp and a qualitative abnormality of glycosaminoglycans (chondroitin sulphate).[1]

These data confirm our previous findings in four siblings with this autosomal recessive condition.[2 3] We agree with the authors' statement that these cases represent a distinct type of spondylar dysplasia. Natural history, physical examination, and ophthalmological, radiographic, and biochemical findings in the case reported by Grain *et al* coincide with those of our cases, except for two points. First, some of our cases had irregular chondrocostal ossification. Second, advanced bone age was not present in our cases and this may explain why final adult height in our male cases (3–10th centile) was not as short as the one predicted for the case reported by Grain *et al* (3rd centile).

As stated by the authors, this disease may be a currently unrecognised cause of short stature. We have suggested for the diagnosis of this brachyolmia, type I, that a slit lamp examination as well as detailed glycosaminoglycan studies should be performed as routine procedures.[4] The latter test is currently available only in some laboratories, but it is of crucial importance for the diagnosis of brachyolmia type I. As autosomal recessive and autosomal dominant patterns of inheritance are involved in the four types of brachyolmia,[1] the distinction among them will allow an adequate clinical management of the patients and will give further support for adequate genetic counselling.

Present efforts in this type of brachyolmia should be directed to DNA studies and among the candidate genes one should include those involved in glycosaminoglycan metabolism. Sequencing and cloning of the gene for brachyolmia would allow a more precise diagnosis and genetic counselling for this condition.

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1 Grain L, Duke O, Thompson G, Davies EG. Toledo type brachyolmia. *Arch Dis Child* 1994; **71:** 448–9.
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Acyclovir in chickenpox

EDITOR,—Virological evidence for the reactivation of chickenpox contracted in infancy has recently been documented and is related to the immune status of the host.[1] 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.[2] 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 five times daily).

This infant was born nine days after his mother developed chickenpox and, in accordance with current guidelines for the UK, he did not receive zoster immune globulin.[3] 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.

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1 Terada K, Kawano S, Hiraga Y, Morita T. Reactivation of chickenpox contracted in infancy. *Arch Dis Child* 1995; **73:** 162–3.
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Expulsion of ventriculoperitoneal shunt tubing

EDITOR,—A baby girl of 18 months was admitted to our paediatric ward in September 1982 with a two week history of irritability, vomiting, and refusal to sleep. She had a fever of 38·5°C and a lumbar puncture showed cerebrospinal fluid protein of 3·65 g/l, 750 polymorphonuclear leucocytes$\times 10^6$/l of cerebrospinal fluid, and Gram positive cocci on staining.

A diagnosis of pneumococcal meningitis was made and the child was treated with triple chemotherapy: penicillin, sulphadimidine, and chloramphenicol as was routine in 1982. She remained critically unwell and developed a third and sixth nerve palsy on the left side. Computed tomography of the head showed marked dilatation of the lateral and third ventricles and she was referred to the neurosurgeons who subsequently inserted a ventriculoperitoneal shunt. The child recovered from her meningitis but remained globally retarded in her development with regular seizures and unable to speak.

At the age of 14 years, she represented with apparent, recurrent abdominal pain. Physical examination was unhelpful. There seemed to be no area of local tenderness or guarding. She continued to eat well and her bowels moved normally. Urine culture and analysis was negative and a plain abdominal radiograph failed to reveal any abnormality. The apparent abdominal pain that the child was suffering persisted intermittently for several weeks and she was reviewed and examined on several occasions. No clinical evidence of organic disease was elicited. After approximately eight weeks of intermittent symptoms the child passed a plastic tube in her stool which was clearly the distal portion of the ventriculoperitoneal shunt.

I am unaware of any reports of this particular complication of ventriculoperitoneal shunting.

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Infant length measurements

EDITOR,—Like Professor Frank Falkner[1] 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 ago[3] in the *Archives*. This paper showed that, provided careful attention 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 accurate and reliable measurements. The constant pressure pad fitted to the number counter, allowing it to automatically lock, added particular precision. We also showed that mothers were well able to hold the head.

But a good reliable instrument is one thing, it is quite another to convince people of the value of length measurement in infants. Along with weight and head circumference, 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.

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1 Falkner F. Infant length measurements. *Arch Dis Child* 1995; **73:** 379.
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