the only person who has ever read any of my own publications. The authors say that Stevens and Nelson[2] found that formula milk reduced the incidence of iron deficiency anaemia whereas the study that was designed to look at the effect of iron in formula milk provided no evidence at all to justify this statement. There was no evidence that formula milk was responsible for the low incidence of iron deficiency anaemia in the children who were studied and no evidence that iron in formula milk was an important source of dietary iron for these infants.

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1 Morley R, Abbott R, Fairweather-Tait S, MacFadyen U, Stephenson T, Lucas A. Iron fortified follow on formula from 9 to 18 months improves iron status but not development or growth: a randomised trial. *Arch Dis Child* 1999;**81**:247–52.
2 Stevens D, Nelson A. The effect of iron in formula milk after 6 months of age. *Arch Dis Child* 1995;**73**:216–20.

Dr Morley and colleagues comment:

We apologise for misquoting Stevens' paper; this was an editing error when we amalgamated two papers. The reference for the statement "Iron fortification of milk formula . . . has been shown to reduce the incidence of iron deficiency anaemia" should have been: Moffatt ME, Longstaffe S, Besant J, Dureski C. Prevention of iron deficiency and psychomotor decline in high risk infants through use of iron fortified formula: a randomised trial. *J Pediatr* 1994;**125**:527–34.

The iron content of the three milks was also misquoted and should have been: cows' milk 0.5 mg/litre; iron fortified formula 12 mg/litre; unfortified formula 0.9 mg/litre. This correction strengthens rather than weakens our conclusions.

Estimating the genetic potential in stature

EDITOR,—Midparental height is an important measure in estimating a child's target height—the genetic potential in stature. Height reference values that allow for parental height are more appropriate for growth evaluation in paediatric clinics. We read with interest the recent paper by Wright and Cheetham on the strengths and limitations of parental heights as a predictor of attained height.[1] The authors concluded that midparental height was a useful indicator of the expected height for children when their parents were of average stature but misleading when used to assess short children. We have recently reported the same findings based in 2402 Swedish children.[2] We observed that the regression coefficient between midparental height and a child's final height was approximately 0.6 in standard deviation scores (it was 0.5 for children 8 years of age in the paper by Wright and Cheetham).

We believe that the linear function of midparental height could be used to estimate a child's target height, rather than midparental or corrected midparental height, which Wright and Cheetham implicitly used to represent a child's genetic target height. The meaning of midparental height is different for children with short, average, and tall parents. The parents' heights not only reflect the parents' genotype in stature, but also mirrors the extrinsic influences the parents experienced during their own growth span. This provides a biologically meaningful explanation of the so called "regression to the mean phenomenon". For instance, the intrinsic genetic potential in stature of short parents is usually much greater than their measured heights; consequently, the following generation is usually taller due to a better manifestation of the intrinsic growth potential.[2]

We agree that short children attending paediatric clinics are usually shorter than their target height, whatever method is used for estimation. The height of parents is important for clinical evaluation of short children. A short child with tall parents is certainly more likely to have a pathological cause than a short child of short parents. It is not appropriate to consider midparental height itself as a simple measure of target height. Clearly, midparental height is not misleading for any child if its linear function is used for estimating a child's target height—the genetic potential in stature.

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1 Wright CM, Cheetham TD. The strengths and limitations of parental heights as a predictor of attained height. *Arch Dis Child* 1999;**81**:257–60.
2 Luo ZC, Albertsson-Wikland K, Karlberg J. Target height as predicted by parental heights in a population-based study. *Pediatr Res* 1998; **44**:563–71.

IGFBP-3 as a predictor of growth hormone deficiency

EDITOR,—We read with interest the paper by Mitchell and colleagues[1] and wish to add our own observations on this subject. In 1996 the Regional Endocrine Laboratory started to provide a service for the measurement of insulin-like growth factor binding protein (IGFBP-3) following early reports that this was a good marker of growth hormone secretion. We then undertook a retrospective audit of the measurement of serum insulin-like growth factor (IGF-1) and IGFBP-3 as predictive markers of growth hormone deficiency (GHD) in children undergoing growth hormone stimulation tests (glucagon and insulin tolerance tests). Between October 1996 and January 1998, 93 children had simultaneous measurements of IGF-1 and 78 children had measurements of IGFBP-3. We defined GHD as a peak growth hormone level of < 20 mU/litre and complete GHD as a peak < 10 mU/litre in response to a stimulation test.

The results for IGF-1 and IGFBP-3 were compared to reference ranges for age available in the laboratory and classified as low or normal. The reference range for IGF-1 was constructed by the laboratory using their own assay and that for IGFBP-3 being supplied by the manufacturers of the kit (Nichols Institute, San Juan Capistrano, California, USA). We calculated their sensitivity and specificity as predictors of GHD using the two different cut off levels and the likelihood ratio—that is, the likelihood that the result would be seen in someone with as opposed to someone without GHD (table 1).

Eight children had both a low IGF-1 and IGFBP-3, which produced a sensitivity of 22.2% and specificity of 90.4%, with a likelihood ratio of 2.3 in predicting GHD. Therefore the combination of a low IGF-1 and low IGFBP-3 would be highly suggestive of GHD, but a significant number of children with GHD will have normal values for either of these two markers.

Table 1 Sensitivity and specificty of IGF-1 and IGFBP-3 in predicting growth hormone (GH) deficiency

	Peak GH < 10 mU/l	*Peak GH < 20 mU/l*
IGF-1		
Sensitivity	37.5%	29.5%
Specificity	79.7%	79.6%
Likelihood ratio	1.85	1.5
IGFBP-3		
Sensitivity	31.5%	27.8%
Specificity	76.3%	76.2%
Likelihood ratio	1.33	1.2

Thus it can be seen that a single measurement of IGFBP-3 performed no better than IGF-1 as a marker of growth hormone secretion despite previous claims. Neither marker had a high likelihood ratio and would therefore not be good as a single predictive test. Although we realise that some of the normal IGFBP-3 results could have resulted from the presence of IGFBP-3 protease activity interfering with the assay in children with radiation induced GHD this is not likely to alter our findings significantly.

Thus we agree with Mitchell *et al* and other authors[2] that IGFBP-3 measurements are not good predictive markers of growth hormone secretion and do not replace the need for careful clinical evaluation and growth hormone stimulation tests in short, slowly growing children.

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1 Mitchell H, Dattani MT, Nanduri V, Hindmarsh PC, Preece MA, Brook CGD. Failure of IGF-1 and IGFBP-3 to diagnose growth hormone insufficiency. *Arch Dis Child* 1999;**80**:443–7.
2 Tillmann V, Buckler JM, Kibirige MS, *et al*. Biochemical tests in the diagnosis of childhood growth hormone deficiency. *J Clin Endocrinol Metab* 1997;**82**:531–5.

Raised serum transaminases: not always liver disease

EDITOR, Too often, the pursuit of detailed investigation supersedes clinical suspicion and decision making. A 3 year old boy was referred to our service for investigation of chronic liver disease. The patient was reported to be a well child, whose development was "within normal limits"; a 2 cm hepatomegaly was found during an admission for a chest infection. Subsequent investigations revealed normal serum bilirubin, γ glutamyl transpeptidase, alkaline phosphatase, and albumin. The only abnormality was a persistently raised alanine aminotransferase (507 IU/litre) and it was this that prompted referral to a liver centre.

Retrospectively it became apparent that the boy had some motor delay, having first walked at the age of 2 years. On clinical examination he was mildly hypotonic and demonstrated a positive Gower's sign. In view of this and the isolated increase in alanine aminotransferase, serum creatinine kinase measurement was requested to determine whether the origin of the transaminase was in fact muscle. The serum creatinine kinase was severely raised at 22 000 μmol/litre and the