Prevention of adult height and bone mineral deficits in delayed male puberty with short stature

Sir,

In a recent paper[1] we demonstrated that the pubertal surge of testosterone secretion coincided with a height *and* mineralisation spurt in normal boys. Subsequently, the height velocity slowed down while bone mineralisation continued at a high rate, at least until age 20 years. In the absence of a sizeable increase in serum testosterone, as in delayed puberty with short stature, the spurts did not appear.[1] At the same time Martin *et al.*[2] consolidated scanty observations indicating that testosterone, endogenous as well as exogenous, stimulated growth hormone secretion in this condition. They also commented upon the final height reached by such boys, stating that this was usually less than expected. Thus, these boys appeared to have lost linear growth potential in an irreversible manner. As the growth hormone stimulating action of testosterone appears to cease after adolescence,[3] Martin *et al.*[2] suggested a more aggressive approach to the treatment of male delayed puberty associated with short stature.

In the above letter we present a unifying concept strongly suggesting that growth hormone governs bone growth as well as the delivery of calcium and phosphate for optimal mineralisation. We also suggest that testosterone, in part at least, exerts its actions on bone growth and mineralisation through its stimulatory action on growth hormone secretion. The clinical and experimental evidence for this concept will be presented (S Krabbe, I Transbøl, C Christiansen, in preparation). We certainly agree with Martin *et al.*[2] who suggested that to achieve normal adult height in this 'syndrome', or extreme variant of normality, treatment has to be initiated earlier than usual, probably as early as age 13 or 14 years. Although we have no data on the bone mineral content of adult males who have undergone delayed puberty, we suggest that earlier treatment may also serve to prevent subnormality respecting the ultimate storage of bone mineral. Although probably unimportant during the first decades of adult life such a deficit may reach clinical significance during senescence.

References

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2 Martin L G, Grossman M S, Connor T B, Levitsky L L, Clark J W, Camitta F D. Effect of androgen on growth hormone secretion and growth in boys with short stature. *Acta Endocrinol* 1979; **91**: 201–12.

3 Merimee T J, Fineberg S E. Studies of the sex based variation of human growth hormone secretion. *J Clin Endocrinol Metab* 1971; **33**: 896–902.

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Congenital absence of the sternum

Sir,

In response to the request made by Dr Haque[1] I report a further case.

A baby girl weighing 3·84 kg was born on 13 September 1970 to an apparently healthy primigravida aged 23 years. Labour was induced at 42 weeks' gestation and there was some hydramnios. The baby was normal apart from complete absence of the manubrium sterni and upper part of the body of the sternum. Aortic pulsation could be seen through the skin and herniation of the lung occurred when the baby cried. There were no respiratory problems, she fed normally, and her developmental progress was entirely normal. She was followed up until age 4 years during which time her growth was normal. Follow-up ceased because her family moved to another area. Her mother gave birth to a normal boy 2 years later. The mother's weight and blood count were normal and there was no suggestion that she had any nutritional deficiency. I do not know the aetiology in this case.

Reference

1 Haque K N. Letter: Congenital absence of the sternum. *Arch Dis Child* 1979; **54**: 905–6.

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The place of noninvasive methods in the diagnosis of acute scrotum in newborns

Sir,

I read with interest the paper by Hitch *et al.*[1] The dilemma that faces one when dealing with patients in this category[2] has been reduced by routine use of the cord and scrotal

Doppler flowmeter examination.[3] The possibility of using a noninvasive procedure to differentiate easily and accurately between ischaemic and nonischaemic testicular lesions is welcomed, and this method should soon become indispensible in cases of acute scrotum; it would certainly help to avoid unnecessary urgent operations in newborn infants and children.

References

1 Hitch D C, Shandling B, Lilly J R. Recognition of bilateral neonatal testicular torsion. *Arch Dis Child* 1980; **55:** 153–4.

2 Iuchtman M, Jacob E T, Wagner I, Berant M. Testicular torsion in the newborn. *Int Surg* 1976; **61:** 100–1.

3 Iuchtman M, Zoireff L, Assa J. Doppler flowmeter in differential diagnosis of acute scrotum in children. *J Urol* 1979; **121:** 221–2.

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Dr Hitch and co-workers comment:

Neonatal testicular torsion mandates early recognition and surgery if hormonal secretion, let alone spermatogenesis, is to follow. We believe that neonatal testicular torsion is recognisable and with the advent of testicular scanning[1] it can easily be verified. Our preliminary experience with Doppler flow studies has been less encouraging. Further experience with these techniques is needed.

Reference

1 Hitch D L, Gilday D L, Shandling B, Savage J P. A new approach to the diagnosis of testicular torsion. *J Pediatr Surg* 1976; **11:** 537–41.

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Continuous sodium valproate or phenobarbitone in the prevention of 'simple' febrile convulsions

Sir,

Ngwane and Bower concluded their recent paper[1] with the recommendation that any child below age 18 months who presents with febrile convulsions should be treated with either sodium valproate or phenobarbitone, but analysis of their paper gives only meagre evidence in support of this proposition. A recurrence rate of 1 in 18 for sodium valproate compared with 7 in 21 for the 'untreated' group, when the necessary correction for small numbers is applied, gives $\chi^2 = 3{\cdot}1$ which is not significant (admittedly, Fisher's exact test gives $P = 0{\cdot}03$ which is significant). No statistical test is given for the difference between a recurrence rate of 4 in 21 for the phenobarbitone group and 7 in 21 for the untreated group; in fact, with the correction for small numbers, $\chi^2 = 0{\cdot}49$ which is not significant (and the exact test gives $P = 0{\cdot}16$ which is also not significant). Moreover, an incidence of 7 recurrences out of 21 cases in the untreated group might be thought atypically high. Five recurrences out of 20 would be more in keeping with common experience, in which case the difference in favour of the phenobarbitone group for practical purposes vanishes completely.

Do these results really suggest that in preventing recurrence of simple febrile convulsions, either treatment (sodium valproate or phenobarbitone) is better than none? Perhaps this is so for sodium valproate, but for phenobarbitone a more scientific conclusion would surely be 'not proved'?

Reference

1 Ngwane E, Bower B. Continuous sodium valproate or phenobarbitone in the prevention of 'simple' febrile convulsions. Comparison by a double-blind trial. *Arch Dis Child* 1980; **55:** 171–4.

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Dr Ngwane and Dr Bower comment:

When we applied a strict definition of 'simple' febrile convulsions in an attempt to obtain a uniform population, thereby reducing the numbers from 265 to 64, we realised that the small numbers would invite statistical argument. Nevertheless, we maintain that our recommendation that any child below age 18 months who presents with a febrile convulsion should be treated with either phenobarbitone or sodium valproate is validated by our results. We showed that the results for all treated subjects were statistically superior than for the untreated group ($P<0{\cdot}05$). The treatment was either phenobarbitone or sodium valproate, administered in a double-blind manner, and the results were similar in the two treatment groups. We agree that the difference between the results in the phenobarbitone and untreated groups is not significant, nor did we claim the contrary. There is strong evidence from previous work (discussed in our article) that phenobarbitone is more effective than nothing, and our trial was designed to discover if sodium valproate was as effective as phenobarbitone. Dr Giles surely has his