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

Sir,

In a recent paper 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. At the same time Martin et al. consolidated scanty observations indicating that testosterone, endogenous as well as exogenous, simulated 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, Martin et al. 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 Transbol, C Christiansen, in preparation). We certainly agree with Martin et al. 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.

Congenital absence of the sternum

Sir,

In response to the request made by Dr Haque 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


M E R STONEMAN
Department of Paediatrics, West Kent General Hospital, Maidstone, Kent

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

Sir,

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

References

Congenital absence of the sternum.

M E Stoneman

*Arch Dis Child* 1980 55: 657
doi: 10.1136/adc.55.8.657-a