Does growth hormone treatment improve final height attainment of children with intrauterine growth retardation?

SIR,—The potential indications for the therapeutic use of growth hormone have widened with the availability of more abundant supplies of growth hormone. One of the new potential indications such as Turner’s syndrome, intrauterine growth retardation (IUGR), etc., the growth-promoting benefits of growth hormone have usually been assessed in the short term by comparing the changes in height velocity SD score during the first treatment year and the pretreatment year, and, in some instances, by quantitating the psychological benefits gained by improved growth during the middle childhood years. In the long term the benefits of growth hormone can be assessed by comparing final height in a treated group with that either in an untreated group or with the original pretreatment height prediction of the treated group.

In their recent article on the use of growth hormone in children with IUGR, Stanhope et al present three year growth data and suggest that the results are rather disappointing.1 This conclusion is based on the fact that the height for bone age score did not change significantly over the three years of the study. With knowledge of the natural history of IUGR, Stanhope et al do, however, still believe that their growth hormone treated children may attain a greater final height than would have been the case if they had remained untreated. In their discussion Stanhope et al state that ‘only the continuation of clinical trials such as this until final height will answer the question’. Others might feel that the current results are sufficiently disappointing for the study to be concluded now. Certainly if the design of the study remains in its present form then, as the authors acknowledge, there is a real possibility that growth hormone treatment will reduce the duration of puberty and compromise final height prognosis. Thus, I would suggest that, if the study is to continue, one of their groups of IUGR children, who received growth hormone during prepubertal life, should not receive growth hormone during puberty. Furthermore, their results emphasise that individual children with IUGR seen by paediatricians around the UK should not be offered growth hormone on an ad hoc basis.

Finally, if the authors believe that possible growth hormone treatment induced gain in height for IUGR children can only be interpreted when the children attain final height, is much lower by publishing the three year growth data?

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Kawasaki disease

SIR,—I read with interest the annotation on Kawasaki disease by Levin et al.1 I would like to draw attention to the fact that this disease, like any other vasculitis, can have a predominantly neurological presentation, with an encephalopathic/encephalitic illness. I have seen three such patients who all presented with high fever, diminished consciousness, a minimal increase in lymphocytes in the cerebrospinal fluid with normal protein and glucose, and a rash. In one child, a 7 year old, the most striking physical sign was bilateral papilloedema.


Inherited metabolic diseases in the sudden infant death syndrome

SIR,—Holton and his coworkers set out to investigate among other causes the prevalence of medium chain acyl CoA dehydrogenase deficiency (MCAD) in cases of sudden infant death syndrome (SIDS).1 They assert that the claim ‘MCAD deficiency could cause 3% of cases of SIDS’ is not supported by their findings.

Unfortunately their work, while exhaustive in its scope, demonstrates the difficulty in looking for rare events in small samples. Using binomial probability theorem

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P(r)=\frac{\alpha}{r!(\alpha-r)!}p^r(1-p)^{\alpha-r},\]

the chance of finding 0 cases in a sample of 70 with a true population prevalence of 3% is 0.118. What the authors can say from their work is that with a 0.05 confidence, the true prevalence of MCAD in cases of SIDS is less than 4.2% and using a 0.01 confidence they can state the prevalence is less than 2.4%. Support for their claim of a low prevalence rate of MCAD in SIDS is found in a recent report of carrier rate of the K 329E mutation for MCAD in population screening.2 These estimate the carrier rate to be between 1/40

Late intravenous gammaglobulin treatment in Kawasaki syndrome

SIR,—We wish to comment on the interesting article on intravenous gammaglobulin treatment for Kawasaki disease in which the Author mentioned it seems reasonable to treat if the disease is still active.1 In a recent study published by Marasini et al suggest a favourable effect of gammaglobulin in established coronary abnormalities, at least in the mid-term follow up.2

They studied two groups of children with a similar image of coronary abnormalities. The first group (nine children) was treated with aspirin (100 mg/kg/day) until the fever disappeared, followed by aspirin (5 mg/kg/day) and dipiridamole (5 mg/kg/day). The second group (seven children) received intravenous gammaglobulins (400 mg/kg/day) for five days after the 10th day from the onset of the disease, when coronary artery abnormalities had already developed.

In the first group four patients had a complete resolution of their abnormalities. Five had persistent abnormalities including three with myocardial infarction (one died from his myocardial infarction). In the second group, all coronary abnormalities had resolved during follow up.

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