LETTERS TO THE EDITOR

Growth hormone deficiency in children with chromosomal abnormalities

SIR,—I read with some surprise the letter by Schwartz and Duck on the treatment with growth hormone of two children with chromosome deletions associated with multiple malformations, microcephaly, and mental handicap.[1] Though I too am intrigued that a cause of poor growth in some children with chromosome disorders might be mediated through a lack of growth hormone (although I cannot understand why they used growth hormone in their second case, as there was a very adequate maximum growth hormone response to insulin provoked hypoglycaemia (21·4 mIU/l)), this has surely to be tempered with a sense of proportion. Is it really appropriate clinical practice to use growth hormone in children with such major multiple physical and neurodevelopmental handicaps?

This example highlights for me what is becoming an increasingly worrying dilemma concerning growth hormone treatment. If, to children with true growth hormone deficiency, we add girls with Turner's syndrome, children who fail to grow as a consequence of intracranial irradiation for treatment of malignant disorders, children with other chronic diseases such as chronic renal failure, and now children with chromosome disorders and dysmorphisms, the queue for growth hormone treatment, an extremely costly item, becomes a long one. This is even without 'normal' short children whose treatment with growth hormone is surely an option we should now very seriously question.

Against a background of increasing financial constraints within health authorities the euphoria following the ready availability of biosynthetic preparations of growth hormone in 1985 will need to be better disciplined. Because growth in the short term can be accelerated after administration of growth hormone, and because there might also result an increase in predicted height of a few centimetres, does not mean it has to be used; this is an assumption that seems to be insidiously becoming part of paediatric practice.

More needs to be learned, beyond simple anecdotal experience, of possible detrimental effects to psychological wellbeing of shortness, which is often the major criterion for considering treatment. We also need to look in more detail at psychological outcomes of treatment. The problem of shortness is often as much a family concern as it is one belonging to the child, and parents' attitudes have to be better understood, as herein so often lies the source of the problem. At the same time we need to do as much as we can to help some children and their parents understand and cope with one of the apparent unfairnesses of life, as short stature is now so often viewed in our society, and not raise the false spectre of 'a pill for every ill'. The reality that sometimes some degree of a shorter than perceived ideal stature might be unavoidable may simply have to be accepted.

In the case of growth hormone treatment, it is difficult to avoid the conclusion that eventually there will need to emerge priorities in its prescription, which whether we like it or not, are likely to be determined in the end as much by monetary considerations as clinical need.

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1 Schwarz HP, Duck SC. Growth hormone deficiency in children with chromosome abnormalities. *Arch Dis Child* 1990;65:334.

Early identification of hearing loss: screening and surveillance methods

SIR,—Scanlon and Bamford have recently emphasised the poor sensitivity of the distraction hearing test as a screening procedure for the detection of childhood deafness.[1] A retrospective study conducted by us in Warrington revealed similar limitations of the distraction test. Of 41 children with severe, sensorineural deafness only 12 cases were detected at a mean age of 11 months as a result of failing the distraction test. Sixteen babies passed the test but were later found to be a deaf at the considerably later age of 41 months. This indicates that if screening procedures in the case of these latter children were unsatisfactory, a false negative group could have been inadvertently created where misplaced parental confidence contributed to delayed diagnosis. Most cases in our series were diagnosed as a result of referral after parental concern.

Even if the sensitivity of the distraction test was 100%, 8 months of age is far too late to make the initial diagnosis of nerve deafness as, by the time appropriate referrals are subsequently made after failing the test, several months may elapse before hearing aids are fitted.

The test itself is technically difficult to perform and the subtleties of result interpretation are often underestimated. It is difficult to maintain standards when large numbers of children need to be screened.

The conditions under which the test is performed are often unsatisfactory and sound proofing of all clinic premises would be prohibitively expensive.

Is it ethical to continue with a screening procedure knowing that at best it has poor sensitivity and that at worst it can actually be harmful by contributing to a delay in diagnosis? Alternative methods of diagnosis are currently being evaluated in West Berkshire. We await their results with great interest.

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1 Scanlon PE, Bamford JM. Early identification of hearing loss:screening and surveillance methods. *Arch Dis Child* 1990;65:479–85.

Purchasers, providers, and community paediatricians

SIR,—Dr Appleyard's analysis of the problems awaiting children's services raises a further question.[1]

The process of contracting is described by the Department of Health as a dialogue in which 'all participants should be clearly identified as being either purchasers or providers'.[2] Dr Appleyard argues that logically this implies that consultants working in provider units will not be allowed to give advice to the purchasers of their services. This represents a considerable challenge to community paediatricians, who have a dual role.

The community paediatrician is a provider of services, for example, for children with special needs. But equally, he or she has important purchaser functions, in terms of local management, and information gathering. For example, immunisation and preschool surveillance are increasingly devolved to general practitioners, while the community paediatrician retains responsibility for the system centrally.

Information is power in the new health service, and it is central to the work of community paediatricians to be able to advocate the health needs of their child populations.

Division of this role would destroy the contribution of the community paediatrician to an integrated child health service. Unfortunately this may be an inevitable consequence of the NHS Review, which aims to provide services according to consumer choice rather than population needs.

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1 Appleyard WJ. Children's services in the new NHS—a struggle for survival? *Arch Dis Child* 1990;65:635–7.
2 Nichol DK. *Contracts for health services: operating contracts.* London: HMSO, 1990. (Department of Health EL(90) MB/24, 27.2.90.)

Is late walking a marker of congenital displacement of the hip?

SIR,—Johnson and colleagues record that 10% of 4275 infants born in 1984–5 and studied in Oxford health region had not walked by 18 months of age.[1] They point out that this figure is considerably in excess of that of 3–5% cited by studies in the 1950s[2] and 1960s.[3] The average gestational age of their late walkers at birth was 36·2 weeks. Not surprisingly, 46% of infants born before 28 weeks fell into this group, for no correction was made for preterm delivery. I find it hard to accept their argument that it is appropriate to ignore the 'lost' prenatal months, even though very preterm infants may have an associated impairment. Apart from the interpretation of the medical assessment, judging a preterm infant against a developmental scale designed for term infants is likely to create anxiety among parents whose child is slow to attain his or her milestones.

In the Oxford study five (1·2%) of the late walkers had orthopaedic problems. It would be of interest to know whether any of these children had congenital dislocation of the hip (CDH). Of course this diagnosis is now usually made long before an infant walks. This was not so 30 years ago, however, when I made a small study of 65 children born in Birmingham in whom the diagnosis was made after walking had commenced. The mean age of starting to walk in this group was 16·5 months (range: 11–28 months) as compared with 13·7 months for the whole population.[2 3]