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]

Twenty two percent of these children with CDH were still not walking at the age of 18 months. Thus the commonly taught axiom that CDH does not delay the onset of walking is incorrect. Indeed, in a later study that I made in 1962, into the way in which the diagnosis came to be made in 244 cases of CDH (average age 19 months), 'late walking' was the fourth most common form of presentation (11%), after 'abnormal gait' (53%), 'short leg' (29%), and 'associated deformity' (21%).[4] No correction was made for gestational age but then CDH is uncommon among preterm infants.[4 5] In conclusion, while the late diagnosis of CDH is becoming less common, the failure of a term infant to walk at 18 months remains a useful marker for this condition.

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1 Johnson A, Goddard O, Ashurst H. Is late walking a marker of morbidity? *Arch Dis Child* 1990;65:486–8.
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5 Dunn PM, Evans RE, Thearle MJ, Griffiths HED, Witherow PJ. Congenital dislocation of the hip: early and late diagnosis and management compared. *Arch Dis Child* 1985;60: 407–14.

Drs Johnson, Goddard, and Ashurst comment: While acknowledging Dr Dunn's point that some degree of delay in achieving motor milestones is to be expected among very preterm infants, the high rate of impairment among the late walkers in this group means that 'immaturity' should not be too readily accepted as the sole reason for late walking. As so often in clinical practice, a balance needs to be achieved between causing unnecessary anxiety to parents and yet remaining aware of the risk of associated impairment in all late walkers, particularly those who were born very preterm.

There was just one infant among the late walkers who had associated congenital dislocation of the hip. This had been detected, however, before the age of 18 months.

Lichen sclerosis

SIR,—As a paediatrician who has encountered eight new cases of lichen sclerosis in young children over a period of 18 months, I would like to add my observations to those of Drs Priestley and Bleehan and Dr Harrington.[1 2]

The earliest of these cases was a girl aged 6 years, who presented with dysuria and bleeding after a visit to a male neighbour—a regular babysitter. The girl gave a clear account of repeated sexual abuse involving frictional interlabial trauma. The neighbour admitted to these offences as well as to those on other children has since served a prison sentence. The findings were of gross lichen sclerosis with pallor, friability, and haemorrhage, with the hymen showing partial disruption at one site.

Of the other seven cases seen, a further two were associated with clear histories of sexual abuse. In another child, extensive hymenal damage was associated with behavioural and family factors, and abuse remains a worrying possibility.

Lichen sclerosis has therefore been noted by me in three children where sexual abuse was described, as well as in children presented by protective agencies, because of this and other behavioural symptomatology.

The suggestion that the presence of lichen sclerosis in someway excludes sexual abuse must be unacceptable. Indeed, if I were to use the statistical reasoning of Dr Berth Jones *et al* in terms of my own limited experience,[3] the presence of these changes would be linked to a sexual abuse diathesis in such significant numbers (50%), as to turn this argument on its head with equal frailness.

If lichen sclerosis is the outcome of chronic trauma in some children, then it must follow that every case requires careful study. Perhaps we should also be considering the possibility that these changes may relate to a local immune response to the recurrent presence of substances like semen, or contraceptive lubricant, both in adults and children.

My personal approach is to accept lichen sclerosis in children as a spontaneous unexplained phenomenon. I would not accept hymenal disruption, sexualised behaviour, or a child's disclosure as part of this phenomenon. The presence of lichen sclerosis in these circumstances can neither be considered 'spontaneous' nor 'unexplained'.

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1 Priestley BL, Bleehan SS. Lichen sclerosis and sexual abuse. *Arch Dis Child* 1990;65:335.
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Surveillance for anaemia: risk factors in pattern of milk intake

SIR,—We were interested to read Mills' study from east London, of the prevalence of anaemia in early infancy,[1] and fully agree with his conclusion that iron status is closely related to the type of milk consumed.

Prompted by a high reported incidence of iron deficiency in China, and concern in Hong Kong that babies were having a poor iron intake during weaning, in the mid 1980s we investigated the iron status of a sample of full term healthy Hong Kong Chinese babies. These babies were being followed up to monitor their growth and nutritional state and most were formula fed. Of our initial cohort of 174 babies only 8, 4, and 2% were breast fed at 2, 4, and 6 months respectively.[2]

Mean (SD) daily iron intakes at 6, 12 and 18 months were 8(5), 9(3), and 8(3) mg respectively, comfortably within the WHO dietary allowance of 5–10 mg. The mean (SD) haemoglobin concentration in 123 babies, at 18 months was 124 (8·9) g/l. Eleven had a haemoglobin concentration <110 g/l. Of these, seven had β thalassaemia trait, two showed a satisfactory response to iron treatment (2 mg/kg/day) for three months, one had an unchanged haemoglobin, and one defaulted further follow up. Three of 112 babies with haemoglobin of >110 g/l had a mean corpuscular volume <70 fl. Mean (SD) serum ferritin concentration at 18 months in 128 infants was 155·9 (24·8) μg/l, with only one having a concentration <7 μg/l.

We attributed this very low (~2% excluding β thalassaemia trait) incidence of iron deficiency to infant feeding practices, which provide: (i) an adequate iron intake during the weaning period from fortified milk formulas, iron fortified cereals, meat, and fish and (ii) a negligible consumption of pasteurised cows' milk with its low iron content and risks of causing occult intestinal blood loss.

These practices contrast with those described in Mills' study,[1] where, with an incidence of anaemia of 22%, continued breast feeding, with the early giving of pasteurised milk, were risk factors for anaemia. But the value of prolonged formula feeding, with iron fortified milks that comply with international standards, as one means of lessening the incidence of iron deficiency in early childhood, was evident from both our investigations.

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1 Mills AF. Surveillance for anaemia: risk factors in patterns of milk intake. *Arch Dis Child* 1990; 65:428–31.
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Collecting 24 hour urine samples from children

SIR,—Collection of urine over several hours for metabolic investigations in infants and toddlers is notoriously awkward: disposable napkins frequently produce contaminated samples, and modified cots are only practicable for small infants.[1 2] The following modified technique imposes few restraints on the patient, and is therefore metabolically accurate, and may be used for accurate collections of up to 72 hours.[3]

A bag is constructed with a narrow tube inlet and a larger outlet. The Hollister 24 hour paediatric urine collection bag is particularly appropriate, having a reinforced outlet provided. The inside pocket of the bag is removed and the bag resealed using a heated bar sealer. A nasogastric tube of approximately 8F guage is inserted 2 cm through a small hole in the top corner of the bag, and sealed in place using spray on plastic adhesive (for example, Dow Corning) and plastic adhesive tape.

The air diaphragm pump (for example, the Charles Austen series), collection vessels, and tubing are attached to provide an air flow of some 3 l per minute, with positive pressure directed into the top of the collection bag, and