for those at the extremes of the normal range for fat/thin, short/tall, clever/stupid, clumsy/age.

My own puberty was late. Not much fun at the time but the resultant temporary exclusion from full membership of the peer group has produced a useful long term lesson in coping with the natural ups and downs of life. Explanation, empathy, and reassurance are in my view better medicine in this area, as in many others, than use of medication.

R D H BOYD
Department of Child Health, St Mary's Hospital, Haslamag Road, Manchester M13 OJH


Dr Keinan comments
I am grateful to Professor Boyd for his comments on my annotation. I hope that I emphasised sufficiently that explanation and reassurance may be all that is required. A decision has to be made on clinical grounds as to whether that is the case – a situation frequently faced by paediatric endocrinologists and many general paediatricians.

I also discussed the poor quality of some previous studies and the need for more scientific information before definitive recommendations can be given. In that regard, studies are in progress in a number of centres and a further contribution from this department is soon to be published in this journal. Selective and appropriate hormone treatment is not designed to ‘narrow the range of normality’ (nor will it do so) but to relieve distress. The extent to which it achieves that must also be assessed scientifically and such studies are also in progress in this department and elsewhere. Not all boys presenting with short stature and pubertal delay are ‘future Professor Boys’ and some are likely to be significantly socially and psychologically disadvantaged at a time which is critically important for determining future work or career prospects. Potential physical consequences of delayed puberty also require proper prospective evaluation.

I believe, with Professor Boyd, that explanation, empathy, and reassurance are often enough. Where they are not, my view is that effective hormone treatments are now available and can reasonably be considered and prescribed on the basis of currently available scientific knowledge.


Minoxidil induced hair growth after leukaemia treatment?

EDITOR—Although hair loss is an invariable accompaniment of chemotherapy for acute lymphoblastic leukaemia (ALL), regrowth is usually prompt and complete. After unusually intensive and prolonged chemotherapy hair may not regrow properly. We report the successful treatment of one such case.

Case report
A 4 year old boy presented with common ALL. He was entered into the Medical Research Council (MRC) UKALL X trial, receiving 18 Gy as central nervous system prophylaxis.

After two years of treatment he was found to have central nervous system leukaemia and therefore was started on a relapse protocol (subsequently formulated as MRC UKALL R1). He tolerated this intensive regimen poorly and developed multiorgan shingles, so that after 16 weeks he was put on a maintenance regimen (vincristine, prednisolone, mercaptopurine, and methotrexate). He received a further 24 Gy of craniospinal irradiation.

After the later two year course of treatment the hair that regrew was only thin and wispy. It remained in this state for a period of 14 months. Minoxidil solution 2% was applied daily to the scalp. Over a period of nine months an almost normal head of hair was regained.

Abnormal hair growth was first noted as a side effect of the antihypertensive agent minoxidil. Topical minoxidil also stimulates hair growth and causes male pattern baldness. It has been tried, unsuccessfully, to modify acute hair loss during chemotherapy; we cannot find any examples of the use described here.

This patient’s hair did not improve for 14 months before the application of minoxidil, leading us to believe that minoxidil caused the hair regrowth. It would be of interest to hear of other’s experience in alleviating this distressing side effect.

MARK A VICKERS
CAROL J BARTON
Department of Pathology, Royal Berkshire Hospital, Reading, Berkshire RG1 3AN


Colonic strictures in cystic fibrosis?

EDITOR—We read with interest the letter by Green et al reporting two patients with cystic fibrosis who developed colonic strictures while receiving high strength pancreatic enzymes. The clinical presentation in both these cases was very similar to our original report in 1994 and to the cases of fibrotic strictures in cystic fibrosis which have been described subsequently. We disagree, however, with Green et al that bowel ultrasonography is unhelpful in the diagnosis of this condition, and indeed they provide no evidence to support this assertion. The typical findings on ultrasound in these strictures are of bowel wall thickening, with reduced peristalsis and free fluid associated with the lesions. Although the site and extent of the lesions can be most accurately defined by contrast studies, we would suggest that ultrasound is a more pleasant and less invasive initial procedure in the young child with abdominal pain.

Abdominal pain is a very common symptom in patients with cystic fibrosis, but because of the recent concern about fibrotic strictures, radiological investigations into the cause of such pain are now being performed early. Our practice is to perform a plain abdominal radiograph and ultrasound of the bowel so that these investigations are normal, then there is little to be gained by proceeding to contrast studies.

ROSALIND S MYTH
ELLIE CARTHY
DAVID HEAP
Respiratory Unit and Department of Radiology, Royal Liverpool Children’s Hospital, Alder Hey, Liverpool L12 2AP


Dr Green and coauthors comment:
We agree that bowel ultrasonography has a place in the diagnosis of colonic strictures, however, we feel that it is an observer dependent investigation. While it is a valuable screening procedure in Dr Green’s hands this may not be the case with less experienced interpretation. In the child with recurrent and troublesome abdominal pain it would be unfortunate to miss the occasional intussusception or in the case stricture by not proceeding to contrast studies. We would therefore be reluctant to suggest relying entirely on a normal plain abdominal film and ultrasound as routine practice in every centre.

Management of anaphylactic reactions to food?

EDITOR—Patel et al draw attention to the use of sew-on badges for children with potentially life threatening anaphylactic reactions. As a community paediatrician who has been responsible for the support of over 20 children with this problem over the last two years I must strongly disagree with their position. A detailed discussion with the parents of children in our area shows that they are keen that their children should not be labelled, either by badges or ‘minders’ in school. We must remember that these children are normal, but with a risk of serious reactions to foods. Support to schools must emphasise prevention (that is, exclusion of allergens from the environment) and management of the (unlikely) reaction. Labelling children may in fact reduce the focus of removing the allergen from the environment and thereby increase the risk to the child. Many food allergens are not obvious (for example, nut oils in foods) and we must not rely on badges to protect these children. The labelling approach is dangerous and may lead to the segregation of these children from their peers. It may also lead to bullying of these children, and encourage other children to offer them the ‘forbidden’ food. I would urge all paediatricians involved with children with food allergies to reject this approach and concentrate on working with schools and parents to support these