paid. The cost savings from non-transplanted livers were equally impressive even at the discounted price of £30,000 per transplant. Are we to believe that these spare livers would not be used for some equally deserving cases thus resulting in no net saving to the health service? As a paediatrician I remain unconvinced by the arguments advanced that a national screening programme at two weeks after delivery will solve this clinical dilemma.

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1 Mowat AP, Davidson LL, Dick MC. Earlier identification of biliary atresia and hepatobiliary disease: selective screening in the third week of life. *Arch Dis Child* 1995; **72:** 90–2.

Professor Mowat and Dr Dick comment:
We are please to have Professor Matthew's support in trying to achieve surgical treatment for all infants with biliary atresia by 60 days of age. Because we share some of the concerns he expresses, we do not advocate screening for biliary atresia but selective screening or more correctly case finding by detecting conjugated hyperbilirubinaemia in jaundiced infants to detect all forms of hepatobiliary disease. Most will have other hepatobiliary disorders for which early and specific treatment is desirable. By screening at the same time as the infant is being assessed by community health care professionals much of the cost and logistic difficulties will be minimised.

King's Healthcare Trust is undoubtedly in the real world. Next year the cost for a direct bilirubin will increase to £4.00 including all overheads! Since submitting our paper an infant aged 18 days with biliary atresia was 'overlooked' by a member of our junior staff. The total serum bilirubin concentration was 72 μmol/l. We cannot stress too strongly the infant with biliary atresia in the first weeks of life appears well. The only constant abnormal clinical feature is jaundice which may be very mild and urine which is persistently yellow and never colourless. In the last two years 25 infants and children in UK died while on waiting lists for liver transplantation. If any of these were alive because a selective screening made transplantation unnecessary for one child with biliary atresia, would any paediatrician object?

Because the optimum time for screening is controversial, community staff in our district are testing for conjugated hyperbilirubinaemia in jaundiced infants of different ethnic backgrounds. This study funded by the Children's Liver Disease Foundation will clarify logistical difficulties and the prevalence of benign jaundice in the third and fourth week after birth.

Double blind placebo controlled trial of pizotifen syrup in the treatment of abdominal migraine

EDITOR,—Now and then the concept 'abdominal migraine' appears in the literature as if it were a fact. I have always been reluctant to accept it as a special entity. The only thing that distinguishes it from recurrent abdominal pain in Apley's definition is the exclusion of the milder cases.[1 2] The demonstration of a special visual evoked response pattern in children with migraine and abdominal migraine is of course interesting.[3] But it is necessary to do this test in an *unselected* group of children with recurrent abdominal pain, to see if it delimits a special group among these children, or if it is a common phenomenon in children with recurrent abdominal pain. Even if it should delimit a special group it might just be a question of severity.

I am not able to refute the existence of abdominal migraine. But until now nothing except severity seems to justify the concept. Migraine in a close family member is a prerequisite for the diagnosis abdominal migraine.[2] But not even this criterion seems to be of any help, as accumulation of several kinds of presumed psychosomatic symptoms including headache is very common in children with recurrent abdominal pain and in their families.[4] I would still prefer the expression recurrent abdominal pain for all bellyachers, at least until we know more about aetiology and pathogenesis.

These reflections should be seen as a comment on the paper of Symon and Russell showing effect of pizotifen in children with abdominal migraine.[5] It is of course important to show that pizotifen does work. But the paper gives rise to two important questions. How does pizotifen work on all children with recurrent abdominal pain? And does the effect of pizotifen in a group of children with severe pain justify the migraine diagnosis?

Aetiology of recurrent abdominal pain is not known with certainty, but it is likely that psychosomatic mechanisms are operative. In the complex pathogenesis different peptides and motility may be important factors.[6] It is in this context that the effect of pizotifen should be considered.

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1 Apley J. *The child with abdominal pains.* 2nd Ed. Oxford: Blackwell, 1975.
2 Symon DNK, Russell G. Abdominal migraine: a childhood syndrome defined. *Cephalalgia* 1986; **6:** 223–8.
3 Mortimer MJ, Good PA, Marsters JB, Addy DP. Visual evoked responses in children with migraine: a diagnostic test. *Lancet* 1990; i: 75–7.
4 Christensen MF, Holm E, Sahlholdt I. Recidiverende mavesmerter hos danske skolebørn. *Ugeskr Laeger* 1984; **146:** 2690–5.
5 Symon DNK, Russell G. Double blind placebo controlled trial of pizotifen syrup in the treatment of abdominal migraine. *Arch Dis Child* 1995; **72:** 48–50.
6 Lindberg T. Recurrent abdominal pain in childhood. *Acta Paediatr* 1994; **83:** 775–6.

Dr Symon and Dr Russell comment:
Recurrent abdominal pain is a symptom and not a diagnosis. We find no difficulty in accepting that children with recurrent headaches may be suffering from a wide variety of different diseases, including migraine, tension headaches, and even cerebral tumours. Similarly recurrent abdominal pain may be the final symptom of a wide variety of disease processes. In our practice the commonest cause of recurrent abdominal pain is constipation. The concept that all recurrent abdominal pain is psychosomatic in origin has been discredited by the absence of any statistically significant differences between children with recurrent abdominal pain and pain free children with regard to various psychological variables thought to be associated with psychogenicity.[1]

The children whom we treated in our trial were not 'bellyachers' but were suffering from recurrent severe disabling symptoms. Unlike bellyachers their symptoms came in discrete attacks with complete normality between episodes. We accept that the term 'abdominal migraine' is not universally accepted and the arguments for this were fully rehearsed in a recent clinical controversies article.[2] Perhaps there would be fewer objections if the syndrome had a different eponymous name such as Buchanan's syndrome,[3] as some people wish to reserve the term migraine solely for headaches on the basis of its presumed etymological derivation from hemicrania.

We would not expect pizotifen to be of benefit in all children with recurrent abdominal pain and logically we feel that it is unlikely that pizotifen would be of value in recurrent abdominal pain other than abdominal migraine. We are not aware of any trials of the use of pizotifen in recurrent abdominal pain other than our own trial in abdominal migraine.

To lump together all children with recurrent abdominal pain as having psychosomatic pathology is to do grave disservice to those patients who come to us seeking relief of their symptoms.

1 McGrath PJ, Goodman JT, Firestone P, Shipman R, Peters S. Recurrent abdominal pain: a psychogenic disorder? *Arch Dis Child* 1983; **58:** 888–90.
2 Symon DNK. Is there a place for 'abdominal migraine' as a separate entity in the IHS classification? Yes! *Cephalalgia* 1992; **12:** 346–8.
3 Buchanan JA. The abdominal crises of migraine. *J Nerv Ment Dis* 1921; **54:** 406–12.

Medicalisation of the normal variant – treatment of the short, sexually immature adolescent boy

EDITOR,—I enjoyed Christopher Kelnar's annotation but as a non-endocrinologist am unhappy about his advice for delayed puberty in the absence of disease that 'boys over 14 years of age ... who have impaired self image and social withdrawal not responding to reassurance' should be considered for treatment which 'should not be denied when appropriate'.[1]

There are two issues. Firstly the widespread use of potent endocrine agents for a self limiting condition. Can we really be sure that there will be no long term adverse effects during the lifetime of the individuals concerned or, indeed, of their progeny? 'Patients need to know whether they want to take the risks and doctors need to be accountable', states Brendon Nelson, the president of the Australian Medical Association, in considering the unexpected long term consequences of another endocrine intervention, Creutzfeldt-Jakob Disease.[2] The prospect of permanent gross dwarfism probably, even in retrospect, justified the, at the time unpredictable and thus unquantifiable, long term risk. Does the transient and common phenomenon of delayed puberty? We must surely include permanence as well as severity and incidence in any therapeutic cost benefit analysis.

Secondly, and more importantly, we need to be careful, as paediatricians, not to narrow the range of accepted normality and to medicalise normal variation. A teenager with delayed puberty may have impaired self image and social withdrawal at the age of 15. Where is the evidence that short term manipulation of the situation with drugs is of long term benefit to the psychological health of the future man, quite apart from its implications

for those at the extremes of the *normal* range for fat/thin, short/tall, clever/stupid, clumsy/agile, white/black individuals?

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.

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1 Kelnar CJH. Treatment of the short, sexually immature adolescent boy. *Arch Dis Child* 1994; **71:** 285–7.
2 Zinn C. Victims of Creutzfeldt-Jakob disease prompt code of conduct. *BMJ* 1994; **309:** 1321.

Dr Kelnar 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.[1]

Selective and appropriate hormone treatment is not designed to 'narrow the range of normality' (nor will it do so) but to relieve distress.[2] 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 Boyds' 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.

1 Brown DC, Butler GE, Kelnar CJH, Wu FCW. A double blind, placebo controlled study of the effects of low dose testosterone undecanoate on the growth of small for age, prepubertal boys. *Arch Dis Child* 1995; **73:** 131–5.
2 Kelnar CJH. Pride and prejudice – stature in perspective. *Acta Paediatr Scand (Suppl)* 1990; **370:** 5–15.

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 multidermatome 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 is used to treat male pattern baldness.[1] It has been tried, unsuccessfully, to modify acute hair loss during chemotherapy[2]; 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.

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1 Savin RC, Alton AV. Minoxidil: update on its clinical role. *Dermatol Clin* 1993; **11:** 55–64.
2 Granai CO, Frederickson H, Gajewski W, Goodman A, Goldstein A, Baden H. The use of minoxidil to attempt to prevent alopecia during chemotherapy for gynaecological malignancies. *Eur J Gynaecol Oncol* 1991; **12:** 129–32.

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.[1] The clinical presentation in both these cases was very similar to our original report in 1994[2] and to the cases of fibrotic strictures in cystic fibrosis which have been described subsequently.[3] 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. If both these investigations are normal, then there is little to be gained by proceeding to contrast studies.

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1 Green MR, Southern KW, Wolfe SP, Littlewood JM, Najmaldin AS, Wyatt JI. Colonic strictures in cystic fibrosis [Letter]. *Arch Dis Child* 1995; **72:** 191.
2 Smyth RL, van-Velzen D, Smyth AR, Lloyd DA, Heaf DP. Strictures of ascending colon in cystic fibrosis and high-strength pancreatic enzymes. *Lancet* 1994; **343:** 85–6.
3 Taylor CJ. Colonic strictures in cystic fibrosis. *Lancet* 1994; **343:** 615–6.

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 Carty'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 a colonic 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.[1] 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 use. 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 in the care of these children to reject this approach and concentrate on working with schools and parents to support these