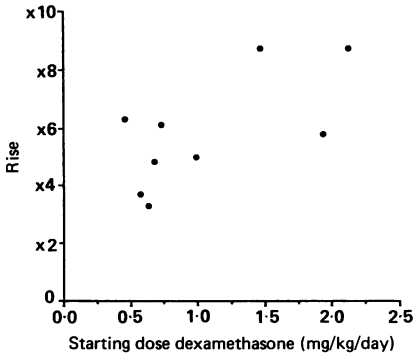

Figure 1 Weight change during first week.

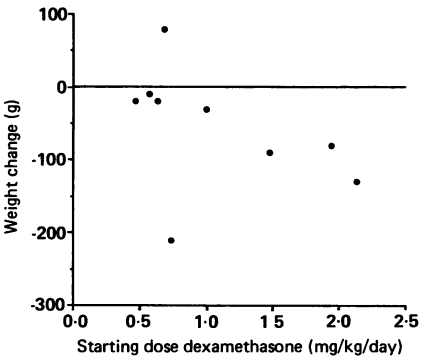

Figure 2 Rise in blood urea concentrations.

the week before treatment (before treatment mean 0·87 mmol/l and on treatment mean 4·73 mmol/l). This catabolic state appeared to be a dose related effect (see figs 1 and 2).

The importance of adequate nutrition in premature infants is well accepted and the likelihood of inducing a catabolic state should be borne in mind when considering dexamethasone treatment. Future studies of growth and nutrition in premature infants should take into account the use of dexamethasone, and further studies of the role of dexamethasone in bronchopulmonary dysplasia should include the effectiveness of lower dose treatment.

P D MACDONALD
P GALEA
L G ALROOMI
Glasgow Royal Maternity Hospital, Rottenrow, Glasgow G4 0NA

1 Mammel MC, Green TP, Johnson DE, *et al.* Controlled trial of dexamethasone therapy in infants with bronchopulmonary dysplasia. *Lancet* 1983;i:1356–8.
2 Avery GB, Fletcher AB, Kaplan M, *et al.* Controlled trial of dexamethasone in respirator dependent infants with bronchopulmonary dysplasia. *Pediatrics* 1985;75:106–11.

Which routine test for kidney function?

SIR,—In a recent article Parkin, Smith, and Brocklebank studied some simple tests for assessing glomerular filtration rate in children with known renal disease.[1] One of the tests studied performed better than the others. It was a height:creatinine index, where a value below 2·1 indicated a low glomerular filtration rate with a sensitivity of 91%.

The calculations for the predictive values of the different tests, however, have also been calculated from the 80 samples in their study.

Table 1

	Test result	Plasma clearance of ^{51}Cr edetic acid		Total
		<80 ml/min	*>80 ml/min*	
Height:creatinine <2·1	+	20	100	120
	–	2	378	380
		22	478	500

Table 2

	Test result	Plasma clearance of ^{51}Cr edetic acid		Total
		<80 ml/min	*>80 ml/min*	
Height:creatinine <1·5	+	15	10	25
	–	7	468	475
		22	478	500

Twenty two of these were from patients with a low glomerular filtration rate. The prevalence of low glomerular filtration rate in this group is 28%. The prevalence of the disease, for which one is looking in the tested population, influences the predictive values for the test quite a lot. The only thing the reader of the article knows about these children is that they all have a renal disease, and that the physician caring for them has decided to measure their glomerular filtration rate by ^{51}Cr edetic acid clearance. A prevalence of low glomerular filtration rate of 28% in this group means that the process of selecting these 72 children, who were tested, from all the others with known renal disease has been quite efficient. The authors now propose that this unknown procedure is to be replaced by using height: creatinine <2·1 as the selecting instrument for suspicion of low glomerular filtration rate. Let us see what happens. The prevalence of reduced glomerular filtration rate is low in the population. Let us presume that the general practitioners with an interest in paediatrics, who are the non-nephrologists the authors recommend the test for, will have to think about the renal function in at least 500 cases of renal disease before they will find 22 cases with low glomerular filtration rate. Using the sensitivity and specificity given in the article we can calculate the following table (see above: table 1).

From this table we can get the predictive value of a normal test to be 99%, but the predictive value of an abnormal test is only 17%.

The authors don't recommend the height: creatinine index <1·5 because its sensitivity is too low. What will happen if we apply it to the 500 children we are talking about with a renal disease and unknown renal function? (see above: table 2).

The predictive value of a normal test is still very good, 98%, and the predictive value of a pathological test is much better, 60%. But this test leaves one third of the group we are looking for undetected. So even if we only have to make 25 clearance tests to find 15 of the 22 with a low glomerular filtration rate compared with 120 clearance investigations to find 20 of them, the five we lose by this method can be quite important.

But both these calculations are based on the assumption that the risk for a low glomerular filtration rate is evenly distributed in the 500 individuals, and that the height:creatinine index will work in the same way in all cases. I don't think that is true. These children all had a known renal disease, and the risk of having a low glomerular filtration rate must be different between diagnosis. If, by using this information, one can reduce the group of 500 children to a more manageable lot of say 100, and then by applying the height:creatinine index as the authors suggest and with the value <2·1, the *whole* procedure will work quite well. I think that is what the authors have done, but they must not keep the hidden part of the screening they have performed hidden from the reader.

S BLOMSTRAND
Department of Paediatrics, Lasarettet, Box 850, S–501 15 Borås, Sweden

1 Parkin A, Smith HC, Brocklebank JT. Which routine test for kidney function? *Arch Dis Child* 1989;**64**:1261–3.

Drs Parkin and Brocklebank comment:
The purpose of the study was to help paediatricians to identify children with reduced glomerular filtration rate and then to decide when to refer those children to a specialist paediatric nephrology centre. We accept the analysis of Dr Blomstrand but the context of our investigation was not screening for reduced glomerular filtration rate in the general population. Given that the children have established renal disease, the decision as to which diagnostic test to use must be made. To this end we have identified normal ranges of β_2 microglobulin and plasma creatinine concentrations in children who have measured normal glomerular filtration rate and given some guidance about which is the most appropriate test to use.

We would agree that this is not an unscreened population and that the results in the general population might be different.

Neonatal purpura fulminans and transient protein C deficiency

SIR,—We present a unique case of an otherwise well neonate presenting with purpura fulminans due to transient protein C deficiency.

A 4310 g girl was born to unrelated parents after an uncomplicated pregnancy and delivery. There was no relevant family history. Vitamin K_1 was injected at birth and a routine examination showed a healthy infant. From 7 hours of age there was rapid development of areas of 'bruising' affecting the trunk and limbs typical of purpura fulminans. Her vital signs were normal. At 18 hours of age she had a right sided clonic convulsion. Oliguria was present with the serum creatinine concentration rising to 114 μmol/l. Ultrasound of the heart and brain and a renal isotope scan were normal. The full blood count, prothrombin time, and activated partial thromboplastin time were normal. A septic screen was clear. The clinical picture was consistent with

homozygous protein C deficiency. However, a functional protein C (Acticlot, American Diagnostica) concentration of 5% (reference range day 0, 8·5–60%[1]) made homozygous deficiency unlikely. Protein S and antithrombin III concentrations were normal.

Within 24 hours of the administration of 10 ml/kg of fresh frozen plasma there were visible improvements in the skin lesions and cerebral and renal function. By 7 days the infant had completely recovered. Fresh frozen plasma was given daily and then second daily until the 23rd day by which time the protein C concentration had risen to 35%. By 6 months the protein C concentration was within the adult range (55–186%).

Neurodevelopmental assessment at 9 months of age showed, a normal infant. The parental protein C concentrations were normal.

Protein C concentrations are known to be low in healthy neonates.[1] Therefore previous authors have advised caution in evaluating infants with apparent protein C deficiency.[2] The finding that purpura fulminans, previously only associated with homozygous deficiency, may also occur in transient protein C deficiency, further highlights the care that must be taken in evaluating infants with low protein C concentrations.

C MINUTILLO
P J PEMBERTON
M L N WILLOUGHBY
P J PRICE
Princess Margaret Hospital for Children, GPO Box D184 Perth, WA, Australia 6001

L R MARSHALL
Royal Perth Hospital

1 Bennhagen R, Holmberg L. Protein C activity and antigen in premature and fullterm newborn infants. *Acta Paediatr Scand* 1989;**78**: 34–9.
2 Manco-Johnson MJ, Marlar RA, Jacobson LJ, Hays T, Warady BA. Severe protein C deficiency in newborn infants. *J Pediatr* 1988;**113**: 359–63.

BOOK REVIEWS

Foetus into Man. By J M Tanner. (Pp 280; £6 paperback.) Castlemead Publications, 1989. ISBN 0–948555–24–6.

This is the second edition of an excellent book describing the journey of physical growth in terms that Professor Tanner hopes 'the biologically unsophisticated reader will understand and the biologically sophisticated approve'. They certainly will!

Human growth is described from conception through embryogenesis, fetal life, the transition at birth, infancy, and childhood to maturity at adolescence. The main outline and chapter contents closely follow those of the first edition but they have been all brought up to date, and some extensively rewritten. There is a valuable bibliography at the end of the book that contains for each chapter some key up to date references and suggestions for further reading for those interested to pursue in greater depth the aspects covered.

Topics covered in 12 chapters include the features of the growth curve; the complex mosaic of differential growth and compositional changes of the body tissues and organs; and factors that regulate and organise growth. Influences on prenatal growth and subsequent size at birth; chromosomal and endocrine control of sexual dimorphism throughout childhood; and a masterly description of puberty with its endocrine regulation lucidly explained are also included. Other topics are the outcome of interactions between genetic make up and the environment, including a particularly well updated account of the effects of nutrition on growth and auxological characteristics of various races, and standards of normal growth with useful charts and figures for practical use in the clinic. Especially welcome is a section on what must be one of the most remarkable aspects of the growth process, 'catch up' growth. The book is rounded off with a succinct account of some common disorders of growth.

The text is written in Professor Tanner's well known inimitable and personal style that has little difficulty in attracting and maintaining the reader's attention. But the ease of this style should not be allowed to conceal the tremendous amount of factual information that is contained. The principal objective of the book is to describe human growth to such diverse audiences that might include teachers, practitioners, students, paramedical workers, and parents and I find it difficult to imagine how any of these groups will not derive benefit and enjoyment from the book. There is something in it for everyone, from the concerned parent of a short boy to the paediatrician dealing in the clinic setting with a difficult growth problem.

I will continue to recommend this book to my students as compulsory reading in their child health course and for those preparing for postgraduate diplomas. Were the many professional workers both in hospital and the community who are at some time involved in problems concerned with the physical growth of children to have this book on their shelves for immediate reference far fewer misconceptions and misunderstandings would arise. Unnecessary clinic referrals would decrease. A lot of parental anxiety would be removed, and the current eagerness for manipulating the growth curve of normal children would be less enthusiastically applied.

D P DAVIES
Professor of child health

Food Intolerance in Infancy. Edited by Robert N Hamburger. (Pp 317; $47·50 hardback). Raven Press, 1989. ISBN 0–88167–545–8.

I suspect that many British paediatricians have a sneaking mistrust of allergists. There is also a reluctance to ascribe every childhood symptom to food allergy. Although the diagnosis of cows' milk allergy has achieved widespread acceptance, there is an impression that food allergy has a certain social cachet and goes with other trappings of affluence. Although one would expect bona fide food allergy to be more common among the socially deprived, in practice it seems to be less prevalent among those who cannot afford expensive exclusion diets. The editor of this multiauthor book recognises this scepticism about food allergy in the introduction. Throughout the book there is a well balanced approach avoiding statements about food allergy which are not supported by scientific data.

The book is essentially the edited proceedings of an international symposium on food intolerance and food allergy. The book consists of five sections and the first section deals with the immunological and chemical basis of food allergy. This is followed by sections on food allergy in general and the manifestations of cows' milk allergy in particular, which I found especially interesting. The chapters on the effects of cows' milk exclusion diets on sleep disturbance and colic were very thought provoking. However, I am still not sure that I will immediately advocate such a diet as the first line of treatment for children who are referred to me with behaviour problems. Dr Eastham's chapter provides a timely reminder of the possible disadvantages as well as potential benefits of soya protein formulas.

I found the section on the Carnation hypoallergenic formula the least interesting and least relevant part of the book. It was the only section of this otherwise excellent symposium to include the name of the sponsors and seemed to be the least objective scientifically. I remain to be convinced that the hypoallergenic formula is superior to protein hydrolysate formulas that are currently available. As Dr Hamburger himself pointed out 'just how hypoallergenic is the new formula?'

Overall I warmly welcome this book which will be of interest to general paediatricians, paediatric gastroenterologists, and immunologists. It should certainly find a place in every paediatric library and will also be of interest to some general practitioners and health visitors.

A W BOON
Consultant paediatrician

The Pediatric Spine III: Cysts, Tumors, and Infections. Edited by Antony Raimondi, Maurice Choux, Concezio Di Rocco. (Pp 215; DM 228 hardback.) Springer Verlag, 1989. ISBN 3–540–96804–0.

This small volume, the fourth in the ongoing series of publications entitled *Principles of Pediatric Neurosurgery*, concerns itself with certain aspects of the paediatric spine. The purpose of the series is to present the reader with an updated comprehensive view of selected topics in paediatric neurosurgery. The volumes are appearing at a greater frequency than one per year and rapid advances in a particular field, or a sense of previous neglect, dictate the choice of subject of each book. While there is a strong transatlantic presence among the contributors, the three principal editors are European and each volume is multiauthor. The series is written mainly for paediatric neurosurgeons and those training in the field, but both the current volume and the previous issues contain much of interest to the paediatric neurologist, oncologist, radiotherapist and neuroradiologist, as well as the general paediatrician and paediatric surgeon.

In the present issue, intraspinal tumours occupy at least 50% of the text, and the discussion is detailed and very practically orientated in terms of neuroradiological protocol, surgical approach, and radiotherapeutic considerations. Reproduction of imaging techniques (myelography, computed tomography, and magnetic resonance imaging) is first class