^{99m}Tc dimercaptosuccinic acid (DMSA) scan in urinary tract infection

Sir,

Dr Smellie *et al*, in their recent paper on ^{99m}Tc dimercaptosuccinic (DMSA) scan in patients with established renal scarring,[1] quoted a paper from this hospital.[2] They failed to quote the principal findings of this paper, however, namely that a DMSA scan is the most accurate method of detecting renal scars in children 0–5 years old. In our paper we compared intravenous urograms, ultrasound, and a DMSA scan carried out over a six month period in 63 children known to have vesicoureteric reflux. A total of 60 children were found to have scarring on DMSA scans, 50 on ultrasound, and only 36 on intravenous urograms. The severity of the scarring was assessed and a highly significant difference was obtained for coupled comparisons of the three modalities—that is, between DMSA scanning and ultrasound, ultrasound and intravenous urography, and DMSA scanning and intravenous urography. None of the changes identified on a DMSA scan in this group was reversible indicating that the appearances were not the result of recent acute infection.

The data were also assessed according to the age of the children, and the discriminating power between the techniques became less noticeable with increasing age.

In our hands ^{99m}Tc DMSA scanning is neither expensive nor time consuming (about £35 per examination). Reactions to DMSA are exceptional but there is still a definite morbidity and mortality associated with contrast media, even the non-ionic form. In our department scanning is carried out 1·5 hours after injection and takes 15–20 minutes. We never perform DMSA and diethylenetriamine pentacetic acid (DTPA) scans simultaneously.

In this hospital the DMSA scan is used as the first line investigation of the renal tract in urinary tract infection. Intravenous urography is carried out only for assessment of possible structural abnormality, particularly before surgery. Reliance on intravenous urography and ultrasound, especially in those under 5 years old, will result in inadequate detection, treatment, and follow up of renal scarring.

References

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[2] Monsour M, Azmy AF, MacKenzie JR. Renal scarring secondary to vesico-ureteric reflux. Critical assessment and new grading. *Br J Urol* 1987;**60:**320–4.

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The teenage coeliac: follow up study of 102 patients

Sir,

We read with interest and pleasure the paper by Kumar and colleagues on the teenage coeliac.[1] We were surprised, however, that height distribution of their teenagers with coeliac disease was nearly normal despite the fact that nearly half of them (45/102) confessed not to adhere to the gluten free diet and most (36/44) of the biopsy specimens showed jejunal mucosa abnormality.

In our longitudinal study we followed up 43 children with coeliac disease who, after initial diagnosis at the mean age of 13 months (range 4–31 months) and a gluten free diet for one to three years (mean 15 months), abandoned this treatment and returned to a normal gluten containing diet.[2] We followed patients up with a yearly clinical and anthropometric assessment and a duodenal biopsy. There was a gradual decrease in the number of the assessed patients because they either returned to the gluten free diet or were lost to follow up. All measurements were interpolated by Tanner's method to obtain data at regular yearly intervals. Such interpolated data were transformed into standard deviation scores (SDS) according to Tanner's formula SDS=(xe–Xm)/SDm where xe is the measurement at the relevant age and Xm and SDm are normal mean and normal standard deviation at the age in question, respectively (see table). Means of standard deviation scores were compared with the normal means by the Student's *t* test for unpaired groups. We found that at each of the yearly age intervals the persistent histological relapse was paralleled with significant height deficit.

Similar linear growth retardation was evidenced by Colaco *et al* in their cross sectional study of Irish adolescents with coeliac disease aged between 17·8 and 18·5 years—therefore after their pubertal spurt.[3]

The 102 coeliac children reported by Kumar *et al* are a 'mixed society'; more than half (58/102) did not have a biopsy specimen taken at the time of the study. Of the 44 who did have a biopsy, eight had normal mucosa, 19 showed moderate and 17 gross abnormality. Some of these children were at their pubertal spurt, some were older. It is

Table *Mean and SD of height expressed as standard scores (SDS) at yearly age intervals in children with 'neglected' coeliac disease and persistent histological relapse*

Age at assessment (years)	*Mean height SDS (SD)*	*p Value*	*No of patients*
3	−0·65 (0·88)	<0·001	35
4	−1·10 (0·90)	<0·001	29
5	−0·76 (0·90)	<0·001	24
6	−1·08 (0·84)	<0·001	19
7	−0·86 (0·97)	<0·01	16
8	−1·06 (0·81)	<0·001	15
9	−0·85 (0·67)	<0·01	10