

LETTERS TO THE EDITOR

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DMSAs after UTI—scan more children, not less

EDITOR,—Deshpande and Verrier Jones have recently concluded that it is not worth undertaking dimercaptosuccinic acid (DMSA) scans in children over 1 year of age who present with a "simple" urinary tract infection (UTI).¹ Their argument has three strands. First, they interpret their data as indicating a very low chance of children over 1 year having a renal scar, especially if the UTI is diagnosed by a general practitioner at home rather than in hospital. Second, they argue that subjecting children to DMSA scanning is financially and emotionally very costly. Third, they feel there is little value in identifying renal scars. We disagree with all three points.

WHAT IS THE CHANCE OF FINDING A SCAR IN A CHILD AFTER A "SIMPLE" UTI? Deshpande and Verrier Jones argue that the prevalence of scars is so low in older children

that it is not justified to perform DMSA scans after 1 year. Yet their own data show that the prevalence in their 124 patients is very similar under and over 1 year (7/52 (13%) *v* 7/72 (10%); $p=0.52$, χ^2). This confirms our study of 2842 children who had DMSA scans after a first recognised UTI.² The prevalence of scars was similar at every age from infancy to 16 years.

Deshpande and Verrier Jones advise using the site of diagnosis of the UTI as well as age to select which children should have a DMSA scan. They seem to be assuming that children diagnosed in hospital are likely to have had a more severe illness (and greater scarring risk) than children diagnosed at home by their general practitioners. Their argument has two flaws. One is that there are many local factors that may influence where the diagnosis is made, but which do not relate to the severity of illness. These will vary but will include the organisation, quality and ease of availability of primary and secondary health care services as well as geographical and social factors. Clearly, it cannot be assumed that their deceptively simple surrogate marker for illness severity will reliably predict scarring risk outside their own centre. A further problem is that their small numbers (54 diagnosed at home, 18 in hospital) give poor predictive power for this association. We also made a crude assessment of illness severity in our study of scar prevalence in children after a UTI,² noting if they had fever, anorexia, malaise, or required hospital admission (but not who made the diagnosis). Younger patients were much more likely to have a severe illness by any of these criteria (table 1), yet their prevalence of scarring was no greater. We also investigated whether these illness severity markers distinguished between the 92 children who had scars and 232 of the unscarred children who were scanned on the same day. Though scarred children were symptomatic slightly more often, the differences were small, so these criteria would not provide a clinically useful screening tool, either before and after the fourth birthday (table 2).

WHAT IS THE COST OF A DMSA?

Deshpande and Verrier Jones' description of DMSA scans bears almost no relation to our experience of them. We use local anaesthetic cream and distraction techniques routinely during venepuncture and find this combination extremely successful. We have not found it necessary to sedate children, nor do we

recognise psychological trauma occurring in the children or their parents. A typical comment from one of our families is how interesting their day had been! The effective radiation dose of a DMSA is up to 0.7 mSv.³ This is equivalent to about 4 months extra background radiation in the UK, or 6 weeks in Sweden.

THE VALUE OF IDENTIFYING RENAL SCARS

Deshpande and Verrier Jones seem concerned that "the emphasis on imaging tests" has overshadowed the importance that needs to be given to "the diagnosis and treatment of infection". Whilst we agree that there is a need for an emphasis on accurate diagnosis and treatment, especially in the very young, we see no conflict between providing a service that delivers prompt diagnosis and treatment, and applying a systematic imaging protocol.

Though the primary aim of imaging children's urinary tracts after a UTI is to identify risk factors that will allow us to prevent renal scarring, there is undoubtedly value in diagnosing scars that have already occurred. Reflux nephropathy is the commonest cause of hypertension in children.⁴ Children that have their blood pressure monitored because of known renal scarring can receive early treatment. By contrast, children that present unexpectedly with severe hypertension following unrecognised or uninvestigated UTIs may have a high morbidity, and a significant mortality. Similarly, children identified as having extensive renal scarring can have treatments gradually introduced if their renal function begins to decline, rather than presenting with the complications of severe renal impairment such as rickets, poor growth, tiredness and anaemia, or even sudden death from hyperkalaemia.

In summary, whilst we acknowledge the importance of knowing the cost of every intervention and test, we are concerned that their value must also be fully appreciated. Investigations that inform families about their child's condition, and allow monitoring to direct early treatment and prevent unpleasant or permanent sequelae are inherently valuable. Since a DMSA scan performed after a childhood UTI has a similar chance of identifying scarring at any age, we currently advocate undertaking one in every child after their first recognised UTI.

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Table 1 The frequency of clinical findings in children investigated for a UTI, by age group. Children aged 8 to 16 years are grouped together because of small numbers

Clinical variables	Frequency (%) according to age in years									
	<1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	>8	
Age	<1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	>8	
Febrile	50	48	37	28	18	36	22	17	32	
Vomiting, anorexia, or malaise	73	57	40	37	23	29	26	21	30	
Admitted to hospital	59	35	26	12	8	3	4	12	23	

Table 2 The frequency of clinical findings in children investigated for a UTI, grouped according to whether they had scars. The statistical difference between the frequencies in the scarred and non-scarred children are assessed separately for children under and over 4 years using the χ^2 test.

Clinical variables	Under 4 years old			Over 4 years old		
	% scarred (n=38)	p value for χ^2	% non-scarred (n=102)	% scarred (n=54)	p value for χ^2	% non-scarred (n=130)
Febrile	47.4	0.57	37.3	37.0	0.03	20.8
Vomiting, anorexia, or malaise	68.4	0.04	45.1	33.3	0.21	23.1
Admitted to hospital	39.5	0.28	27.5	13.2	0.75	10.2

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Age specific aetiology of anaphylaxis

Routine hospital data analysis suggests the incidence of anaphylaxis is rapidly increasing in England.^{1,2} Although an acute life threatening disorder, anaphylaxis is often managed sub-optimally,³ one of the major difficulties being prompt recognition of the disorder.⁴ An appreciation of how aetiology varies with age may aid clinicians in arriving at a quick and accurate diagnosis.

Using the Hospital Episode Statistics database, we studied 2323 emergency NHS admissions over the four year period from 1 April 1991 to 31 March 1995, with a primary diagnosis of anaphylaxis (*International Classification of Diseases* (ICD) 9: 995.0; 999.4). Three cases were excluded because of invalid age codes; 17% of admissions occurred in children aged under 16 (n = 385). Overall, aetiology was recorded for 52% (n = 1207) of admissions, the most commonly recorded triggers being drugs (61%), food (16%), and venom (11%).

Studying age specific aetiology (table 1) reveals that food related anaphylaxis becomes relatively less frequent with increasing age (p < 0.001) whereas the proportion of drug triggered admissions increases with age (p < 0.001). No venom related admissions were noted in infants, but in all other age groups the proportion of venom triggered admissions remained stable.

Differences in age specific patterns of admission may result from variations in susceptibility, exposure, or both. Alternatively, these patterns may reflect recording biases, which may operate differentially. Care also needs to be taken in interpreting these data because aetiology was not recorded for almost half of the anaphylaxis admissions studied. Despite these reservations, in view of the unprecedented number of cases available for study, our findings are likely to provide the most reliable picture of variations in anaphylaxis aetiology with age. Further progress will be dependent on achieving more comprehensive recording of trigger agents, particularly in children, and the development of a more extensive set of ICD codes for anaphylaxis that allows recording of triggers such as nuts and latex.¹

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BOOK REVIEW

Poverty and child health. N Spencer. (Pp352, 2nd ed.) Radcliffe Medical Press. ISBN 1-85775-477-8.

I noted contrasting newspaper headlines in the week I read this book (February 2001): "5m Britons living on the breadline" (referring to the Breadline Europe survey in September 1999), and "Parents are to blame for child poverty", a Peterborough City Council Leader commenting on the finding of the Poverty in Peterborough 2000 report. These summarise the breadth of attitudes represented in the country, and doubtless too in paediatricians, over the causes of child poverty. How much is it really parents' fault that children go to school dirty, with holes in their shoes, and without any breakfast, or indeed do not go to school at all? The blame culture is common, particularly among the conservative media, and those who live in poverty are well aware of this. Low income parents know that they carry the responsibility for parenting their children adequately. The evidence is that mothers will give up treats, trips, and an adequate diet for themselves in order to feed and clothe their children.

The Breadline Europe survey mentioned above measured poverty on the yardstick of the public's assessment of the absolute essentials of life: households lacking at least six of these were categorised as being "a lot" below the level of income needed to avoid absolute poverty. Nine per cent of the population came into this category. And the current European Union figures put the UK at the bottom of the league with a massive one in three children living in poverty. As UNICEF put it, "the UK emerges as a serious contender for

the title of worst place in Europe to be a child".

So, what is new in Spencer's second edition? In reviewing the first edition in this journal in May 1997 I wrote of the limited space given to health services approaches to tackle health inequalities. There is considerably more on this area in the new edition. Figures on poverty are updated, and there is a new section on measuring child health, though this is rather inadequate on the "assets" (health) as opposed to the "deficits" (disease). Perhaps the main change since the first edition is that there is a Labour government which has made a commitment to ending child poverty by 2020, ten years too long, in the view of the Child Poverty Action Group. In the meantime, how can we as paediatricians reduce the effects of poverty on children's health?

In Spencer's eyes, the most important means are political, through backing policies of redistribution: this would require a higher level of taxation for the well off. Spencer shows that Britain is bottom of the list for income redistribution in Europe. Sweden is at the top, with those on welfare achieving 83% of national average economic well being compared to 48% in UK.

Secondly, paediatricians should be aware of and support specific social policies aimed at families such as maternity allowance, additional benefits for lone parents, and child benefit.

Thirdly, there are specific health sector interventions of known effectiveness that paediatricians and their Royal College might take forward in collaboration with others. The basic principles of these are equity, empowerment and participation, intersectoral working, information and data monitoring, accessibility, flexibility, and advocacy. Examples of evaluated programmes are the accident reduction programme in Harlem, New York, which uses innovative community development methods, and the community mothers scheme in Dublin, which trains local volunteers in a home visiting programme.

The involvement of paediatricians in such schemes is limited at present, but could be considerable. Resolution for 2002: find out what is happening locally in measures to tackle poverty in child health, and contribute! Spencer's book will be essential advance reading.

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Table 1 Emergency anaphylaxis admissions by age and aetiological trigger.

	Children < 16	All ages	Infants <1	Preschool 1 to 5	Junior 6 to 10	Adolescent 11 to 15	Elderly > 55	χ^2 with 1 df	p value
Food	60 (41)*	190 (16)*	16 (62)	25 (48)	8 (32)	11 (26)	12 (4)	121.0	<0.001
Meat/fish	1 (1)	19 (2)	1 (4)	0 (0)	0 (0)	0 (0)	4 (1)	0.028	0.86
Berries/seeds/mushrooms/plants	9 (6)	25 (2)	1 (4)	6 (12)	1 (4)	1 (2)	3 (1)	16.4	<0.001
Food other	43 (30)	111 (9)	12 (46)	15 (29)	6 (24)	10 (24)	3 (1)	104.0	<0.001
Food unspecified	7 (5)	35 (3)	2 (8)	4 (8)	1 (4)	0 (0)	2 (1)	9.5	0.002
Drug	49 (34)	738 (61)	7 (27)	15 (29)	9 (36)	18 (43)	227 (75)	69.0	<0.001
Antibiotics: penicillin	4 (3)	158 (13)	1 (4)	1 (2)	0 (0)	2 (5)	64 (21)	25.0	<0.001
Antibiotics: other	2 (1)	70 (6)	0 (0)	0 (0)	1 (4)	1 (2)	27 (9)	9.9	0.002
Analgesics: anti-rheumatics	0 (0)	55 (5)	0 (0)	0 (0)	0 (0)	0 (0)	18 (6)	7.1	0.008
Analgesics: other	0 (0)	57 (5)	0 (0)	0 (0)	0 (0)	0 (0)	12 (4)	34.0	0.07
Vaccines	17 (12)	54 (4)	3 (12)	2 (4)	4 (16)	8 (19)	10 (3)	8.4	0.004
Drug other	26 (18)	344 (29)	3 (12)	12 (23)	4 (16)	7 (17)	96 (32)	8.0	0.005
Insect venom	15 (10)	136 (11)	0 (0)	7 (13)	3 (12)	5 (12)	35 (12)	0.53	0.47
Other	21 (14)	143 (12)	3 (12)	5 (10)	5 (20)	8 (19)	30 (10)	0.62	0.43
Total admissions with aetiology recorded	145	1207	26	52	25	42	304		
Total admissions (% aetiology not recorded)	385 (62)	2320 (48)	69 (62)	145 (64)	73 (66)	98 (57)	499 (39)	45.0	<0.001

*Table shows count (percentage) unless otherwise stated