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Correspondence

Neuropathic bladder and spinal dysraphism

Sir,

We support Borzyskowski and Neville[1] in their plea for early recognition of occult forms of spinal dysraphism. Although experience suggests that it is rare to find lessening of neurological impairment in such patients after surgery, further deterioration may be prevented.

The correct use of the term spinal dysraphism is important; in a recent postgraduate examination a question on spinal dysraphism elicited replies which totally ignored all open forms such as myelocele. In 1886 von Recklinghausen wrote about the araphic theory in the causation of open and closed spinal defects.[2] The term dysraphism is derived from that paper, and it was used again by Lichtenstein.[3] Other authors, such as Gryspeerdt,[4] have dealt with lesions including tethered cord and diastematomyelia under the general heading of occult spinal dysraphism or, more correctly, crypto-dysraphism (G Crawford, 1981, personal communication). There is growing confusion by the omission of the word *occult*. In their first paragraph Borzyskowski and Neville[1] mention spina bifida as distinct from dysraphism and then cite a reference to occult spinal dysraphism.[5] We wonder whether a paper by James and Lassman entitled 'Spinal dysraphism'[6] although clearly dealing with occult forms only, might have increased the misunderstanding.

Unfused neural arches, commonly described as spina bifida occulta, are found in about 5% of normal adults and in some children with delayed fusion;[7] this radiological finding must be related to other bony lesions—such as a widened interpedicular space, bone spur, or enlarged intervertebral foramen—before being considered significant. However, the presence of neurological abnormalities merits further investigation in the presence or absence of spina bifida occulta.

References

1 Borzyskowski M, Neville B G R. Neuropathic bladder and spinal dysraphism. *Arch Dis Child* 1981; **56:** 176–80.

2 von Recklinghausen F. Untersuchungen über die spina bifida. *Arch Pathol Anat* 1886; **105:** 243–51.

3 Lichtenstein B W. Spinal dysraphism. Spina bifida and myelodysplasia. *Arch Neurol Psychiatr* 1940; **44:** 792–8.

4 Gryspeerdt G L. Myelographic assessment of occult forms of spinal dysraphism. *Acta Radiol [Diagn] (Stockh)* 1963; **1:** 702–17.

5 Anderson F M. Occult spinal dysraphism: a series of 73 cases. *Pediatrics* 1975; **55:** 826–35.

6 James C C M, Lassman L P. Spinal dysraphism. An orthopaedic syndrome in children accompanying occult forms. *Arch Dis Child* 1960; **35:** 315–27.

7 Lorber J, Levick K. Spina bifida cystica. Incidence of spina bifida occulta in parents and in controls. *Arch Dis Child* 1967; **42:** 171–3.

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Dr Neville and Dr Borzyskowski comment:

We accept that the term spinal dysraphism should be used as the term for all midline fusion defects of the spinal cord. However, it has become conventional to separate open spina bifida from the closed fusion defects with significant neurological lesions. The letter from Dr Levick and Mr Sharrard points out that this view is appearing in examination answers. Several authors have used this terminology for closed lesions with significant neurological defect, and occult spina bifida or occult spinal dysraphism may be confused with the minor degree of non-fusion of L5 or S1 neural arches. The first sentence of our paper should have read 'The causes of a neuropathic bladder in childhood are numerous, and include open spina bifida (which forms the largest group), closed spinal dysraphism, spinal cord tumour, trauma, and myelitis.' We welcome the writers' support for the need for early recognition of such patients in whom the external manifestations are often quite minor.

Nursing sick children

Sir,

Your Annotation[1] was both timely and encouraging. For years we have been trying to gain recognition of the importance of meeting the needs of children by utilising existing trained staff and by maintaining or re-establishing training facilities for sick children's nurses. Despite the highly commended Court report,[2] little progress has been made in implementing its recommendations. This is because there is resistance to change, particularly on the part of those who have the power to initiate it but who have failed to understand the relevance of such change in the light of growing emphasis in child care.

Paediatric nurses in Scotland have some support (but it is not known for how long) from the Scottish Home and Health Department and the General Nursing Council for Scotland, to the extent that some semblance of a training scheme for nurses specialising in the care of sick children has been retained. However, we feel that the time has come for our case to be represented far more strongly at EEC level because the pattern of nurse training is greatly influenced by the EEC Committee. As a nurse teacher I know that the existing programme for student nurse

training in this field does not fully meet all the needs of children from birth to adolescence, and that we must be prepared to extend our knowledge and expertise so that the RSCN is more readily accepted in all fields of child care. We need the support of paediatricians and our colleagues. However, if paediatricians are willing to work with nurses who do not have the necessary qualifications, and if our nurse administrators are prepared to accept such nurses, then our position will not only be substantially weakened, but it will also have a detrimental effect on the services available for sick children.

References

1 Chantler C. Nursing sick children. *Arch Dis Child* 1981; **56:** 241–2.

2 Court S D M, chairman. *Fit for the future.* Report of the Committee on Child Health Services. London: HMSO, 1976.

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Survival of children with chronic renal failure

Sir,

Last year we reported 10 years' experience with regular haemodialysis and renal transplantation in the treatment of children with end stage renal disease (ESRD).[1] We should now like to update this report with the results from 1979 and 1980.

By 31 December 1980, 98 children, aged under 15 years, had been accepted for treatment of ESRD. Eighty-eight of these children have received 111 renal allografts (88 first grafts, 19 second grafts, and 4 third grafts; 50 live related donor grafts and 61 cadaver grafts) and 65 of them currently have a functioning graft, 10 are on dialysis, and 13 have died. Of the 10 children not transplanted, 4 were maintained on haemodialysis, 2 children (aged 10 months and 2½ years) were on hospital peritoneal dialysis awaiting cadaver grafts, 1 with the haemolytic uraemic syndrome had recovered renal function after 12 months on home haemodialysis, and 3 had died.

Actuarial patient survival in all 98 children accepted for treatment of ESRD was 81% at 5 years and 78% at 10 years, while the 5-year survival rate for the 66 children treated in the six years since 1 January 1975 was 90%; at 31 December 1978, the 5-year survival rate for all children treated was 76% and that for the 57 children treated during the preceding six years was 83%.

Actuarial graft survival for first grafts performed since 1 January 1975 is shown in the Figure. Live donor graft survival was 86% at 3 years and 76% at 5 years, compared with 71% at both 3 and 5 years for the six years ending in December 1978. First cadaver graft survival has also improved from 47% to 65% at both 3 and 5 years.

The trend towards improved results[1] has therefore continued, despite accepting younger children with more complex problems on to the programme. Recipients of

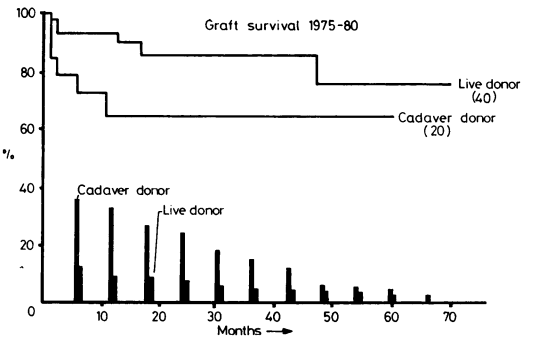

Figure *Actuarial survival of first live donor and cadaver donor grafts in children 1975–80. Number of grafts at risk at each analysis point are shown.*

cadaver grafts are now transfused on at least five occasions before transplantation and this may have contributed to the improved results.[2]

A grave problem that has become worse during the last 6 months is the poor supply of cadaver kidneys for transplantation and we especially need kidneys from children in order to treat infants and the younger children. We would ask our colleagues to consider contacting their local transplant unit if suitable kidneys become available.

References

1 Chantler C, Carter J E, Bewick M, *et al.* 10 years' experience with regular haemodialysis and renal transplantation. *Arch Dis Child* 1980; **55:** 435–45.

2 Opelz G, Graver B, Terasaki P I. Induction of high kidney graft survival rate by multiple transfusion. *Lancet* 1981; **i:** 1223–5.

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Bed occupancy not an index of efficiency

Sir,

Dr Forrester should be congratulated on his excellent Short Report[1] which clearly demonstrates what paediatricians have known for a long time. In our hospital group (and presumably in many others) bed occupancy is counted at midnight; this means that if a child is in with a febrile convulsion for 2 days and 1 night, according to the administrators his bed is occupied for only one day, making the statistics even more confusing.