management of spinal cord or cauda equina compression from recurrent primary or secondary malignancies was not discussed.

(6) The critical role of dexamethasone in emergency treatment was not mentioned.

(7) Chemotherapy now has a central role in the management of intraspinal malignancy,[5] yet receives no mention whatever. If a specific diagnosis can be made by biopsy (of the cord compressing lesion itself or of a separate site of tumour spread—for example, lymph node, bone marrow or, in the case of neuroblastoma, by a combination of biochemical and radiological criteria), laminectomy can be avoided. This is important because multisegment laminectomy can cause major morbidity. Besides the acute deterioration of neurological function, mentioned by Dr Cole, major cosmetic and functional consequences of laminectomy are also common. When radiotherapy and laminectomy are combined, these 'late effects' are particularly severe.[4]

(8) The role of 'lamina replacement' after laminectomy also deserves mention. The morbidity of the operation (especially in young children) will almost certainly be reduced by the general introduction of this technique.

J PRITCHARD
Department of Haematology and Oncology, Hospital for Sick Children, Great Ormond Street, London WC1N 3JH

J PUNT
Department of Neurosurgery, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH

Dr Cole comments:

The authors have made some valuable comments on the *treatment* of spinal cord tumours, but they have failed to appreciate the aims of the annotation. My remit in bringing these rare, frequently misdiagnosed tumours to the attention of the general paediatrician, was to emphasise clinical features and diagnosis rather than treatment. Annotations by definition are brief and thus have to be selective. Perhaps Dr Pritchard and Mr Punt will agree that it is more important that paediatricians be aware of the pitfalls in the diagnosis of these conditions, rather than the details of their highly specialised treatment. Management of intraspinal neoplasms is of course varied and complex, demanding the multidisciplinary skills of the paediatric neurosurgeon, neurologist, radiotherapist, neuroradiologist, and oncologist (preferably in a UK CCSG centre). The onus of suspecting the diagnosis, however, falls squarely on the shoulders of the general paediatrician.

A more careful reading of my text will confirm that nowhere have I stated that space occupying lesions below L1–L2 cause spinal cord compression or that the peak incidence of neuroblastoma is in the newborn period (points 1 and 3). Point 4 is perhaps a little unfair, not only have Dr Pritchard and Mr Punt misinterpreted what was said, they have also quoted out of context. The references provided were apposite to the original text. Many radiotherapists would, I think, concur that developments in their field have led to improved quality of survival (that is, outlook) for patients with spinal cord tumours.[6–8]

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The teenage coeliac

Sir,

We wish to comment on some defects in the study reported by Kumar *et al*[1] and to report our different findings.[2] The authors state that the height distribution of their patients was normal and unrelated to the strictness or otherwise of the gluten free diet, but we question this conclusion as they did not measure 32 of their 102 patients. They found that six of those measured (9%) remained below the third centile for height; if any of the 32 unmeasured had similar stature, the height distribution would clearly be below normal. Mean centile weights of those weighed were also below normal, so we find it difficult to accept that 'all patients were well and leading normal healthy lives' or that they 'remained well despite gluten ingestion' on the basis that they were regarded as asymptomatic, in view of the above findings and also that mucosal biopsies were taken only in 44 patients and 17 of these were grossly abnormal.

Our more comprehensive study showed a broad correlation between dietary compliance, height, weight, and mucosal state, serial height and weight being measured from mean age of diagnosis of 3·3 years to 10 years in 49 and 43 respectively of 52 patients. All patients regarded as complying well with the gluten free diet were of normal height and had normal or only slightly abnormal upper intestinal mucosa. Ten patients followed up from diagnosis at under 4 years to mean age 26 years, whose dietary compliance was poor from soon after diagnosis, were significantly shorter and lighter than normal and all had active enteropathy with appreciably lowered brush border enzymes. The mean heights and weights of those complying moderately were lower than those on good gluten free diets, but not significantly so.

We believe that if non-compliance with a gluten free diet starts in early childhood and persists, that adult stature will