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 multisection 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 radio-

therapists 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

be reduced; at least three of the patients of Kumar *et al* probably fell into this category. We agree that gluten seems to harm the teenager less than in the young child and that a very few teenagers and young adults may seem to tolerate gluten for long periods. We also know that some patients don't realise that they are not completely well until they have experienced a considerable improvement in well being on a strict gluten free diet. Recent evidence suggests that complying with a gluten free diet reduces the risk of malignancy.³

In their final paragraph the authors are ambivalent about advocacy of a gluten free diet, although at one time they did advocate one. We would be gratified to learn that they accept that non-compliance with a gluten free diet would be harmful for most of their patients.

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J COLACO, B EGAN-MITCHELL, F M STEVENS,
P F FOTTRELL, C F MCCARTHY, and
B M McNICHOLL
University Department of Medicine,
Regional Hospital, Galway, Ireland

Drs Kumar, Milla, and Professor Walker-Smith comment:

Thankyou for inviting us to reply to the letter of Colaco *et al*. None of us disagrees that a strict gluten free diet is mandatory in children and, indeed, this is the practice we all follow. There is also no doubt that shortness of stature can be due to untreated coeliac disease and, in our paper, three of the patients with their heights in the <3rd centile group in fact presented with this complication.

The point of our paper was to show that, despite being repeatedly advised to adhere to a strict gluten free diet, many teenagers did not follow this advice. Our clinics also have a dietician in attendance and the 'teenage' coeliac clinic has now been running for over 16 years with all patients being carefully monitored. The morphometric measurements of the jejunal mucosa, however, showed that many patients had abnormal mucosa despite stating that they were on a strict diet. It is well known that patients often say what they feel their doctor would like to hear!

To answer specific points: we reported measurements in only 70 of 102 patients as the remaining had been measured with less accurate techniques. The criticism put forward would also apply to their own paper where only 45 of 52 patients were reported—a far smaller number than in our paper. One explanation for the difference between our papers could be that although all our patients had reached puberty, perhaps some of the patients of Colaco *et al* had not as this point was not mentioned in their paper. Finally, if Colaco *et al* 'find it difficult to accept' that our patients

were well 'despite gluten ingestion' we can only refer them to the references quoted in our paper or, indeed, to any clinician who sees patients with coeliac disease on a regular basis.

IgG antibodies to *Aspergillus fumigatus* in cystic fibrosis

Sir,

We read with interest the report by Forsyth *et al* showing a significant negative correlation between the serum titre of IgG antibody to *Aspergillus fumigatus* and the Shwachman score and several indices of pulmonary function in cystic fibrosis.¹ The authors speculate that high antibody titre might lead to more severe disease through an increase in the amount of inflammation in the lung, though they acknowledge that conversely it might merely reflect colonisation of the damaged lung in those with more severe disease. Unfortunately they do not give details about *Pseudomonas aeruginosa* colonisation and specific *P aeruginosa* IgG serum antibodies in their patients.

We have recently done a similar study in 36 patients with cystic fibrosis aged 1-19 years. IgG ELISA to *A fumigatus* metabolic and somatic antigens was performed and the serum titre was obtained by comparing the results with reference serum. We found, as Forsyth *et al*, a significant negative correlation of the titre with forced expiratory volume in one second ($r\ 0.61$, $p<0.01$), forced vital capacity ($r\ 0.59$, $p<0.01$), peak flow ($r\ 0.58$, $p<0.01$), Shwachman score ($r\ 0.55$, $p<0.01$), forced expiratory flow measured between 25% and 75% of expired vital capacity ($r\ 0.54$, $p<0.01$), and a positive correlation with age ($r\ 0.42$, $p<0.01$). The closest association (figure) was, however, with the number of serum precipitins to *P aeruginosa* (determination kindly done by Dr Niels Højby, Statens Seruminstitut, Copenhagen, Denmark) ($r\ 0.79$, $p<0.001$). Raised serum precipitins to *P aeruginosa* have previously been associated with advanced lung disease and chronic *P aeruginosa* bronchial infection in cystic fibrosis.² Any independent effect of

Figure Correlation of serum titre of IgG antibodies to *Aspergillus fumigatus* and number of serum precipitins to *Pseudomonas aeruginosa*. $y=24.328x+246.085$, $r=0.9$.