in group c (6) ranitidine, 150 mg/bid. χ^2 with Yates' correction and Wilcoxon's test for pair differences were used for statistical analysis.

Our results show there was a reduction in the percentage of patients with gastritis grade 4 and 3 put together, by the end of six weeks. The difference was substantial but not significant, in patients treated with CBS (before treatment: 50%, after treatment: 17%). The small number of patients in group c does not guarantee any relevant suggestion for this group. No differences were found in gastritis grading in any one of the studied group, before and after treatment.

There was a substantial but insignificant reduction in the percentage of HLO positive, grade 3 and 2 patients by the end of six weeks treatment with CBS or sucralfate; 25% of CBS and 15% of sucralfate treated became HLO test negative. On the contrary, all the patients treated with ranitidine became HLO positive, grade 2 and 3, by the end of six weeks.

No significant differences were found in HLO test grading in any group of patients before and after treatment.

None of the drugs studied significantly improved gastritis, although CBS reduced substantially the percentage of patients with gastritis grade 4 and 3. Others have found CBS to significantly improve *Helicobacter* associated gastritis in patients with non-ulcer dyspepsia¹ as well as in a mixed population of patients suffering from duodenal ulcer, gastric ulcer and non ulcer dyspepsia, by the end of four weeks treatment.² This discrepancy could be caused by the rather small number of patients in our study. Another interesting finding was that two patients on sucralfate therapy became *Helicobacter pylori* negative, by the end of six weeks treatment. Others³ have found no influence of sucralfate on *Helicobacter pylori*. Our finding could be due to sampling error or perhaps the concomitant use of some other drugs that is, amoxicillin or metronidazole by these two patients.

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Lectin binding sites in the jejunal mucosa of patients with gluten sensitive enteropathy

SIR, — We have read with interest the article by Vecchi *et al* (*Gut* 1989; 30: 804-10) concerning the histochemical distribution of glycoconjugates investigated by lectins in the jejunal mucosa of patients with gluten sensitive enteropathy and subtotal villous atrophy. Four patients with coeliac disease and 10 patients

with dermatitis herpetiformis were studied; in addition 10 jejunal biopsies taken from patients with irritable bowel syndrome, showing normal morphology were used as controls. Although our experience has been reported in a previous study¹ this was not cited by Vecchi *et al*. In our study we have investigated the lectin binding sites in duodenojejunal mucosae from 119 coeliac children (67 with subtotal villous atrophy and 52 with partial villous atrophy after gluten free diet for 12 months) (age range 1-12 years); moreover, 16 biopsies obtained from infants with short stature and normal intestinal mucosa and nine specimens obtained from infants suffering from postenteritis syndrome were also utilised as normal and pathological controls respectively.

It would be of interest to know the age of patients studied in the paper by Vecchi *et al*; in addition no information about the blood groups of patients is available in this study.

Comparisons can be made on the different results obtained by Vecchi *et al* with ours. Our findings show WGA and DBA in normal mucosae are different from those of Vecchi *et al*, in which a constant positivity at the tip of villi and in goblet cells was observed; the latter findings were similar to our pathological control mucosae. Moreover, Vecchi *et al* considered 10 normal patients suffering from chronic diarrhoea with a final diagnosis of irritable bowel disease; these patients are indeed comparable with our pathological controls. Therefore we contend that only patients without gastrointestinal symptoms and histologically normal intestinal mucosa must be chosen as normal controls, especially in studies assessing lectin binding sites in pathological tissues.

In pathological specimens, there is striking disagreement about the distribution of some lectins; in particular WGA was evident in our coeliac mucosae and pathological controls, whereas no reactivity was encountered in normal control mucosae. On the contrary, Vecchi *et al* reported no differences in the WGA pattern in pathological and 'normal control' mucosae. As regards DBA distribution, Vecchi *et al* observed an evident decrease of goblet cells reactivity in mucosae with subtotal villous atrophy in comparison with controls and they suggested an incomplete maturation of goblet cells; on the contrary we have documented the appearance of goblet cells reactivity in coeliac patients and pathological control mucosae. The DBA reactivity is considered to be a specific marker of human colonic mature goblet cells,^{2,3} as also outlined by Vecchi *et al*; it is noteworthy that colonic mature goblet cells contain acidic sulphated mucosubstances, which are absent in normal duodenojejunal mucosa. In coeliac mucosae, however, we have previously shown with high iron diamine - alcian blue pH 2.5 (HID-AB method), the presence of weak and strong acidic sulphomucins.⁴ This fact may be related to the DBA reactivity found in our coeliac mucosae, suggesting the appearance of new specific lectin binding sites. Additional evidence on the appearance of sulphated glycoproteins was the finding of the PNA expression in our coeliac mucosae in comparison with normal and pathological control mucosae. The latter datum is another divergent point with negative findings by Vecchi *et al* in their control and pathological mucosae.

Finally, Vecchi *et al* suggest that further studies about the lectin binding pattern in the jejunum of patients with GSE on a gluten free diet would be useful. In the aforementioned paper, we have extensively studied the lectin binding pattern in 52 treated coeliac patients on

a gluten free diet for at least 12 months; in particular, data concerning WGA, DBA and PNA obtained in these patients were similar to those found in untreated coeliac children. The hypothesis of a primary defect of the glycoconjugates metabolism in coeliac disease remains to be investigated in more extensive studies.

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Reply

SIR, — We appreciate the comments of Barresi and colleagues, who point out some very important matters of discussion on the use of lectin histochemistry. We feel, however, that the two studies cannot be considered fully comparable because of clinical and methodological differences.

First, while Barresi *et al* report the results obtained in a pediatric population,¹ our series consisted of adult patients only.² Although it is not known if age may induce changes in the lectin binding pattern in human subjects, this seems to be the case in animals.^{3,4} Indeed, the lectin binding pattern observed by Barresi *et al* differs not only from what we have observed but also from what has been observed by other authors.^{5,7}

In fact, as also mentioned by our colleagues, DBA has been shown to react with at least some of normal jejunal specimens studied by other authors.⁵ Furthermore, WGA reactivity of both epithelial and goblet cells is a common finding in normal jejunal mucosa, as described by other authors^{6,7} and is one of the most consistent findings in our experience.^{2,8} Staining with PNA has been reported to occur even in normal specimens by other authors⁹ but no evidence of staining was observed by us in either normal or pathological jejunal mucosa.^{2,8}

If these differences are indeed due to the fact that the only normal controls studied so far are those reported by Barresi *et al*,¹ as the authors suggest, is also not known. Their statement is based on the absence of any gastrointestinal symptom in their children of short stature. Short stature in children and chronic diarrhoea in adults, however, are both suggestive of coeliac disease. Thus, we believe that the presence of normal jejunal mucosa architecture, together with the negativity of other functional and serological evaluations (which were all performed in our patients) should be considered as sufficient evidence to rule out the disease in our patients with diarrhoea as well as in their children with short stature.

Lectin histochemistry is a useful and interesting tool for the study of tissue glycoconjugates. One should keep in mind, however, the fact that the use of different fixatives and of different tissue processing techniques may affect the presence and availability for staining

of the carbohydrate side chains. Thus, we feel that data obtained using different methods should be compared very cautiously. In our hands, the use of formalin fixed tissues has given the most reproducible results.

As far as the persistence of changes in lectin binding pattern after gluten free diet, Barresi *et al* provide very interesting and exciting results. We agree that further studies on this matter are still needed, as the patients studied by Barresi after 12 months of gluten free diet still showed partial villous atrophy. The persistence of changes observed by Barresi *et al* in these patients might in fact reflect the incompleteness of recovery rather than the expression of a primary defect.

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Gastroenterologists and nutritional support

SIR,—At the inaugural committee meeting of the Small Bowel/Nutrition Section of the British Society of Gastroenterology in September 1988 it was agreed that a survey should be undertaken to assess the degree of influence and/or participation of BSG members on the practice of clinical nutrition support in the United Kingdom. A brief questionnaire was circulated to 1500 BSG members in December 1988. All replies until June 1989 were collected and analysed; 168 completed questionnaires were returned—a response rate of 11.2%. The breakdown of occupation of respondents is listed in the Table.

Ninety per cent of respondents were responsible for decision making for enteral nutrition of their own patients, but only 45% were responsible for decision making concerning total parenteral nutrition for their own patients. Eighty nine per cent of respondents were also responsible for enteral nutrition support of other clinicians' patients, dropping to only 29% for total parenteral nutrition. Some form of nutrition team or advisory group was available to 39% of respondents, and 7% were in charge

TABLE Completed questionnaires

Respondents (n)	Occupation/status
68	Consultant physicians
36	Consultant surgeons
25	Consultant gastroenterologists
9	Senior registrars (medicine/gastroenterology)
7	Consultant paediatric gastroenterologists
4	Professors of medicine
4	Senior lecturers/readers (surgery)
4	Consultant paediatrician
3	Professors of surgery
3	Senior lecturers (medicine)
2	Senior registrars (surgery)
1	Professor of child health
1	Professor of paediatric gastroenterology
1	Senior lecturer (child health)

of those teams. Six per cent of respondents had access to a team which solely advised on total parenteral nutrition. Three respondents said that nutrition teams were in the process of being 'set-up'. If a nutrition support team was not available (61%), over half respondents stated that 'no-one specific' was responsible for providing nutritional support or that there was 'variable' responsibility. Thirty per cent stated that nutrition support advice was given by the patients' own clinical team, 6.5% had advice provided by dietitians, 5% by consultant anaesthetists, and 3% by pharmacists.

In summary, most of the respondents admitted to some involvement with nutrition support—predominantly enteral. A minority of BSG members were involved with decisions for total parenteral nutrition, even with their own patients. Less than half of the BSG membership had access to nutrition teams. The poor response rate for the questionnaire of 11.2% can be compared with a response rate of 73.4% for a much more detailed and complex questionnaire sent to district dietitians.¹

The results of this small survey suggest that members of the BSG play a very minor role in decision making for nutritional support. This is perhaps surprising considering the frequency with which gastroenterological patients (both medical and surgical) require nutritional support. It is to be hoped that the formation of the Small Bowel/Nutrition Section of the BSG will stimulate greater interest and participation of gastroenterologists in the practice of nutritional support as so many gastrointestinal diseases are associated with nutritional problems.

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Randomised, double blind comparison of omeprazole and cimetidine in the treatment of symptomatic gastric ulcer

SIR,—We have read with interest the recent article by Bates *et al* (*Gut* 1989; 30: 1323-8) but we wish to comment on the presentation of the results. Indeed in several Figures of this article (Figs 3, 4, and 5) the per cent of patients is given without the absolute values being stated in either the text or on the figures. We feel that by so doing valuable information might be 'lost', such as, how many patients on cimetidine took no antacids, etc.

It is also stated that more patients on cimetidine than on omeprazole took antacids and it

might be interesting to know in what amount and at what time antacids were taken, as this could interfere with bioavailability of cimetidine.¹

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Reply

SIR,—Dr Plas raises some interesting points relating to our recent paper. In the text, we stated that diary card data were available for 80-83% of randomised patients, and Figures 4 and 5 relate to this subset of the 105 patients randomised to omeprazole and 92 to cimetidine. Over the first 14 days of the trial, significantly fewer patients receiving omeprazole took antacids compared with those receiving ranitidine ($p=0.001$). At day 14 seven of 74 patients receiving omeprazole and 27/70 receiving cimetidine also took antacids. From days 2-14, the total antacid consumption in the omeprazole group was 597 tablets and in the cimetidine group 885 tablets ($p=0.01$). Patients recorded their daily antacid consumption but not the time at which they took these tablets. A single tablet of the antacid used in our study has a neutralising capacity of 13 mmol HCl, compared with 156 mmol HCl used in the study quoted by Dr Plas; moreover, in our study, the antacid did not contain aluminium. In normal practice, patients take antacids prn and we feel that our study was a fair reflection of this, and therefore of the relative efficacies of omeprazole and cimetidine.

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CREST syndrome: nodular regenerative hyperplasia of the liver and primary biliary cirrhosis an overlap syndrome?

SIR,—We have read with interest the paper by Mc Mahon *et al* about an association between nodular regenerative hyperplasia of the liver, CREST syndrome and primary biliary cirrhosis.¹ We should like to make the following comments. First, the authors state that their report is the third recorded case of association between CREST syndrome and nodular regenerative hyperplasia of the liver; in our opinion they have failed to quote two other previous reports concerning this association.^{2,3} Moreover, the paper by Hautfova *et al*⁴ was actually the first report of an association between CREST syndrome, nodular regenerative hyperplasia of the liver and primary biliary cirrhosis. Second, we consider that the diagnosis of primary biliary cirrhosis in the report of McMahon *et al* is based on insufficient data to establish the usual diagnosis criteria of primary biliary cirrhosis. The authors argued that increased serum IgM concentration, cholestasis, and decrease in bromosulphthalein clearance which were observed in their patient, favoured the diagnosis of primary biliary cirrhosis. Increase in serum IgM concentration, however, is found in a variety of systemic diseases association with nodular regenerative hyperplasia of the liver^{4,5} and was also observed in our patient (personal communication).² Raised alkaline phosphatase activity is noted in 67% cases of nodular regenerative hyperplasia of the liver.⁶ A decrease in bromosulphthalein clearance is of no value in the presence of cholestasis and has been reported in patients with nodular