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 biodisposability of cimetidine.¹

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- 1 Cugler R, Brand M, Somogyi A. Impaired cimetidine absorption due to antacids and metoclopramide. *Eur J Clin Pharmacol* 1981; 20: 225-8.

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 seric 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