Gut

Gut publishes original papers and reviews concerned with practice and clinical research in gastroenterology. The field includes the basic science, molecular biology, physiology and diseases of the alimentary tract, the liver and pancreas including epidemiological, medical, surgical, radiological or histopathological aspects. Case reports will only be accepted if of exceptional merit. Letters related to articles published in *Gut* or with topics of general professional interest are welcomed. Authors are encouraged to include the names and addresses of four experts whom the authors consider suitable to peer review their work.

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REFERENCES These follow the Vancouver system – that is, references numbered consecutively in the text and listed numerically with titles abbreviated in the style of *Index Medicus, Standard journal article* – (list all authors when six or less; when seven or more, list first three and add *et al*): James A, Joyce B, Harvey T. Effect of longterm cimetidine. *Gut* 1979; **20**: 123–4. **NB: Accurate punctuation is essential.**

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LETTERS TO THE EDITOR

Chronic lymphocytic gastritis

SIR,—We read with interest the article by Crampton *et al* on 'Chronic lymphocytic gastritis and protein losing gastropathy.'[1] We are currently reviewing the clinical data of a large series of patients with the diagnosis of chronic lymphocytic gastritis (CLG), collected over the past six years.

We would like to add some comments from our own experience of the association between CLG and protein losing enteropathies. We collected clinical and laboratory data from 160 patients. Eighteen had lower limb oedema, which in 14 cases was of cardiac or renal origin or was related to prolonged venous stasis. However, in the other four patients, who had hypoproteinaemia as did Crampton's cases, no cause outside of the gastrointestinal tract was found to account for the protein loss. Ninety seven of the 160 patients of this series had complete biochemical investigations. Interestingly, 18 (18%) had abnormal serum protein concentrations, lower than the lower limit of normal by at least 10%. This may be related to the common observation that the enlarged gastric folds, characteristic of the varioliform features of the disease, are covered by a thick coat of mucus and crossed by large serpiginous erosions.[2]

We published a series of 44 patients with CLG in 1986[3] where weight loss and anorexia, also described by Crampton *et al* in their patients, were considered to be common symptoms. Indeed, 66% of patients presented with weight loss and 33% with anorexia. These symptoms were sufficiently severe in some cases to mimic clinically the presentation of gastric cancer and particularly, because of the enlarged rugal folds, gastric lymphoma.

Possible confusion with Menetrier's disease must be borne in mind. In fact, some of our patients carried the clinical and radiological diagnosis of Menetrier's disease. Furthermore, in one patient who came to surgery suffering from protein loss and peripheral oedema there was a complex pathological picture, with features of both CLG and Menetrier's disease, almost suggesting a relation between the two conditions. Leaving aside the debate concerning the infantile form of Menetrier's disease, it must be said that it shares clinical features with CLG, and like CLG can resolve spontaneously over a few months.[4]

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1 Crampton JR, Hunter JO, Neale G, Wight DGD. Chronic lymphocytic gastritis and protein losing gastropathy. *Gut Festschrift* 1989; **30**: 71–4.
2 Haot J, Hamichi L, Wallez L, Mainguet P. Lymphocytic gastritis: a newly described entity. A retrospective endoscopic and histological study. *Gut* 1988; **29**: 1258–4.
3 Haot J, Jouret-Mourin A, Delos M, *et al*. Anatomoclinical study of a series of chronic gastritis characterized by intra-epithelial lymphocytic infiltration. *Acta Endoscopica* 1986; **16**: 69-74.
4 Delos M, Jouret-Mourin A, Wallez L, Willette M, Mainguet P, Haot J. Histological follow-up of a series of gastritis characterized by intra-epithelial lymphocytic infiltration. *Acta Endoscopica* 1986; **16**: 185–7.

Goblet cell carcinoid of the appendix

SIR,—The article by Park *et al* on 'Goblet cell carcinoid of the appendix' (*Gut* 1990; **31**: 322–4) is interesting, but the results presented do not justify their recommendation that all such patients should be treated by a right hemicolectomy. Four of the patients are alive and free of recurrent disease after appendicectomy. One of the patients treated by right hemicolectomy is dead. In only one patient was residual tumour found in the caecum and appendiceal stump and his follow up is only two months. Only one patient has presented late after appendicectomy with recurrent disease. This was 15 years after the original surgery and we are not told how long after his right hemicolectomy his current follow up is. It seems more sensible to recommend hemicolectomy for patients in whom the tumour is close to the base of the appendix, but the case for routine right hemicolectomy has not been substantiated in this paper.

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Reply

SIR,—We would agree that many patients with goblet cell carcinoid of the appendix appear to do well after simple appendicectomy. This tumour, however, has a more sinister prognosis than is generally accepted for carcinoid tumours. In our study the five year actuarial survival was 60% and in a review of published reports 16% of all cases had recurrence.[1] As stressed in our original article it is not possible to predict the behaviour on histological criteria, including position of the tumour. Of the four patients in our study with recurrent disease, two had proximal and two had distally situated tumours. Until better prognostic factors are identified, we think that a right hemicolectomy offers the best chance of preventing recurrence in otherwise healthy patients.

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1 Edmonds P, Merino MJ, Livolsi VA, Duray PH. Adenocarcinoids (mucinous carcinoid) of the appendix. *Gastroenterology* 1984; **86**: 302–9.

One minute urease test

SIR,—After setting up the one minute urease test in our laboratory we came across some interesting findings. In patients who had undergone treatment for *Helicobacter* with bismuth the biopsy specimen was positive on histological examination but negative on the urease test. In our study the sensitivity of the one minute urease test fell from 75% to 43%. No change was seen in the placebo group. Although the organisms are still present, they lose their ability to produce urea after treatment. The urease test should not be used once treatment has begun.

If the treatment regimen has to be monitored, it is best to send the specimen for histological examination.

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Opposition to 'Gut'

SIR,—Apropos of the editor's note on Harold C Edwards, the first editor of *Gut* (*Gut* 1989; **30**: 1659), in which Edwards's recollection of 'the vigorous discussion concerned with choosing a title and the opposition to "Gut" which was pretty strong' is cited, the readers of this journal may be interested and perhaps amused to know that when a group of young clinical investigators in gastroenterology met in New Haven in the early 'sixties to organise the Eastern Gut Club a vigorous discussion concerned with choosing a title arose, and the opposition to 'Gut' was also pretty strong. The opposition to that word by some finicky members of the east coast establishment collapsed, however, when it was pointed out that the recently published journal of the British Society of Gastroenterology was called *Gut*.

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