

within these adenomas of the duodenum. In an effort to determine the incidence of malignant degeneration, we have commenced a further prospective study, randomising patients to annual surveillance with biopsy, or aggressive endoscopic destruction, which includes endoscopic retrograde cholangiopancreatography in the evaluation. Patients found to have malignant degeneration are subjected to surgical excision. To further confuse the picture, patients with an apparently normal appearing ampulla on random biopsy, have been shown to have histologic adenomatous change, which in some patients extends up into the bile duct.

It is hoped, that with long term surveillance, and undergoing this randomisation, that we will obtain a better understanding as to the risk of cancer in the upper GI tract in patients with familial adenomatous polyposis.

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#### Symptoms of IBS and objective measurements of large bowel function

SIR.—While reading with interest the paper by Oettl and Heaton (*Gut* 1987; **28**: 146–9), we should like to take issue with some of the conclusions reached.

Their results are based on a highly selected group of patients, namely four of 30 patients with 'irritable bowel syndrome', and it is difficult to justify the claim that the results in these four should be valid for the whole group, given the undoubted heterogeneity of the condition. Further shortcomings are detailed by the authors themselves, such as the danger of making assumptions about motility based on what is essentially a study of whole gut transit time.

Finally, while agreeing that our understanding of the relationship between colonic function and symptoms in the irritable bowel syndrome is rudimentary, we should like to point out that work in this department has identified disordered colonic motility in approximately 30% of a group of patients with this condition.<sup>1</sup>

In conclusion, we feel that the study sample in this paper is too small and selected, to draw any firm conclusions.

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#### Erosive gastritis – does acid matter?

SIR.—The paper by Tatsuta *et al* on erosive gastritis<sup>1</sup> deserves comment. On the basis of the finding of 'large acid secreting areas' in their patients and because of the good therapeutic results obtained with pirenzepine, the authors suggest that the pathogenesis of this gastric disorder may involve high acid production. In agreement with Nesland and Berstad<sup>2</sup> we found, in a series of subjects with erosive gastritis of the antrum, that acid secretion was within the normal range and comparable to the secretory values observed in an age matched group of healthy controls.<sup>3</sup> On the other hand we reported in patients with chronic antral erosions an impairment of the gastric mucus-bicarbonate barrier, mucus secretion being qualitatively altered<sup>4</sup> with consequent luminal bicarbonate outflow through the eroded mucosa.<sup>5</sup>

The hypothesis that the pathogenesis of erosive gastritis is related to weakening of mucosal defences rather than to acid hypersecretion is consistent with our endoscopic findings after medical treatment. We observed complete disappearance of chronic antral erosions in 73% of cases after only four weeks of treatment with pirenzepine, compared with 47% healing in ranitidine treated patients ( $p=0.05$ ).<sup>5</sup> Furthermore pirenzepine was found to suppress  $\text{HCO}_3$  leakage into the gastric lumen even in subjects whose erosions were still unhealed,<sup>5</sup> which seems to suggest a functional recovery of the mucosal barrier preceding the anatomical repair. As the acid inhibiting activity of pirenzepine is much lower than that of ranitidine, the superior effect of the antimuscarinic agent in inducing endoscopic healing of erosive gastritis can be hardly explained on the basis of acid suppression. Thus, while I agree with Tatsuta *et al* on the good therapeutic activity of pirenzepine on erosive gastritis, it seems to me that this should be related to the property of the drug of strengthening the mucosal protective factors<sup>6</sup> which happen to be impaired in chronic gastric erosions.

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### Reply

SIR,—We thank Dr Guslandi for his comments about an impairment of the gastric mucus – bicarbonate barrier as a pathogenic factor in erosive gastritis. They<sup>1</sup> and Nesland and Berstad<sup>2</sup> found that acid secretion was within the normal range of healthy controls in patients with erosive gastritis of the antrum. We found that erosive gastritis was associated significantly more frequently with large acid secreting areas. We previously found a significant correlation between the extent of acid secreting areas and MAO. In fact, we found that gastric acid output in patients with erosive gastritis was high, and the same as in duodenal ulcer patients. Moreover, Sata<sup>3</sup> also reported acid hypersecretion in patients with erosive gastritis. Although I agree that pirenzepine has acid inhibiting activity and strengthening activity of the mucosal protective factors, it seems to me that acid hypersecretion has a more important role in pathogenesis of this disease.

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- 3 Sata H. Follow-up study of gastritis verrucosa. *Prog Dig Endosc* 1978; **12**: 27–31.

### Gastric cytoprotection by colloidal bismuth subcitrate (De-Nol) and sucralfate. Role of endogenous prostaglandins

SIR,—We read with interest the studies of Konturek SJ *et al* (*Gut* 1987; **28**: 201–5). There are however some important issues we would like to raise.

We continue to emphasise that the macroscopic assessment of gastric mucosal injury without any

histological corroboration is both misleading and incorrect.<sup>1</sup> The importance of histology has been reported in the gastric mucosal injury by aspirin<sup>2,3</sup> and ethanol.<sup>4</sup> In the latter study, the theory that prostaglandins achieved complete cytoprotection of the gastric mucosa against injury by absolute ethanol<sup>5</sup> was proved incorrect when microscopic studies of 'the cytoprotected uninjured gastric mucosa' revealed extensive surface mucosal injury. Do De-Nol and sucralfate prevent gastric surface cell injury? Without histology this important question is unanswered.

Another possible explanation of the data is that De-Nol and sucralfate induce a thick layer of mucus on the surface of the gastric mucosa – with the result that oral aspirin or ethanol does not reach the gastric mucosa. The measurement of serum salicylate and ethanol concentrations would solve this dilemma.

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### Future requirements for colonoscopy in Britain

SIR,—This report by the Endoscopy Section Committee of the British Society of Gastroenterology is indeed timely (*Gut*, 1987; **28**: 772–5). The diagnostic, therapeutic and surveillance indications for colonoscopy are clearly defined and we would not dispute but that the estimated requirements of about 160 colonoscopies per 100 000 population per year is a conservative one. Irrespective of the indication for colonoscopy, implicit in carrying out this procedure is the need to do biopsy; indeed most colonoscopic