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.

DAVID G JAGELMAN

Cleveland Clinic Foundation,
Cleveland, Ohio, USA.

Symptoms of IBS and objective measurements of large bowel function

SIR,—While reading with interest the paper by Oettlé 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.[1]

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

G LAMONT, D EVANS, AND J REYNOLDS

Department of Surgery,
Queens' Medical Centre,
Nottingham.

Reference

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Erosive gastritis – does acid matter?

SIR,—The paper by Tatsuta *et al* on erosive gastritis[1] 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[2] 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.[3] 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[4] with consequent luminal bicarbonate outflow through the eroded mucosa.[3]

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 ($p=0{\cdot}05$).[5] Furthermore pirenzepine was found to suppress HCO_3 leakage into the gastric lumen even in subjects whose erosions were still unhealed,[5] 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[6] which happen to be impaired in chronic gastric erosions.

MARIO GUSLANDI

Institute of Internal Medicine,
University of Milan,
Italy.

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