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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. They¹ and Nesland and Berstad² 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, Sata³ 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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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.¹ The importance of histology has been reported in the gastric mucosal injury by aspirin^{2,3} and ethanol.⁴ In the latter study, the theory that prostaglandins achieved complete cytoprotection of the gastric mucosa against injury by absolute ethanol⁵ 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

examinations are likely to produce multiple biopsies and this has immediate implications for the histopathology laboratory. The handling and processing of these small biopsies requires particular care and, furthermore, they often pose considerable diagnostic problems requiring the examination of multiple levels and the application of histochemical techniques.

The Pathology Section of the British Society for Gastroenterology is currently investigating the effect of endoscopic procedures on the workload of histopathology laboratories. When this survey is completed we will be able to more precisely estimate the implications of increasing any endoscopic facility. In the meantime, however, it is important that our clinical colleagues, whose efforts to improve endoscopic facilities we warmly applaud, should not lose sight of the implications this has on the histopathology laboratory both in terms of workload and in the need for specialist expertise in interpretation.

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Ginger and carbon dioxide as thromboxane synthetase inhibitors: potential utility in treating peptic ulceration

SIR.—In a recent report, the thromboxane synthetase inhibitor dazmegrel was found to afford cytoprotection in an animal model of gastric mucosal damage, and the therapeutic potential of thromboxane synthetase inhibition in cytoprotection was stressed.¹ Thromboxane A₂ has been implicated in the pathophysiology of peptic ulceration.² Interestingly, cimetidine acts as a thromboxane antagonist.³

I would like to suggest the conjoint use of two potent thromboxane synthetase inhibitors: ginger^{4,5} and carbon dioxide.⁶

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thromboxane A₂ synthesis in man. *Thrombosis Res* 1984; **35**: 105–9.

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Disodium cromoglycate and prednisolone enemas in the treatment of ulcerative colitis

SIR.—We have read with interest the study by Grace *et al* (*Gut*, 1987; **28**: 88–92) on the treatment of ulcerative colitis with disodium cromoglycate (DSGC) or prednisolone enemas. It may be of interest that in 1977 we reported to the 21st National Congress of the Italian Society of Gastroenterology (SIGE), similar data, although on a limited number of patients.

In our study, the action of DSCG administered in doses of 200 mg/100 ml by enema twice a day for two months was compared double-blind with 21-prednisolone phosphate 20 mg/100 ml twice daily also given by enema (enemas were packed in identical bags). Twelve patients with ulcerative colitis were included in the trial, all on a longterm treatment with sulphasalazine 2 g/daily who presented a moderate relapse and continued to take their maintenance dose during the trial. Treatment evaluation was based on symptoms, appearance at sigmoidoscopy and rectal biopsy (with count of eosinophils and mast cells), with patients being assessed at presentation and at the end of an eight week period of treatment.

Results showed a favourable therapeutic action of both drugs with a slight preference for DSCG (normalisation of rectal mucosa in six patients v five in the prednisolone group). From the histological point of view, the results were difficult to interpret: eosinophil count frequently very high in both groups, did not seem to be significantly influenced by the two drugs. Symptoms disappeared in all patients at the end of the treatment period.

These preliminary data support the hypothesis that allergic processes may play a definite, although minor role in the pathogenesis of ulcerative proctitis, and show that DSCG when given as enema exerts a similar therapeutic effect on mild, distal colitis as steroid enemas, thus supporting the recent conclusions of Grace *et al*.

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