

- 4 Guslandi M, Passaretti S, Sorghi M, Ballarin E. The mucus-bicarbonate barrier in erosive prepyloric changes. *Gastroenterology* 1986; **90**: 1445.
- 5 Guslandi M, Masci E, Ballarin E, Imbimbo BP, Daniotti S. Luminal bicarbonate outflow in chronic antral erosions is suppressed by pirenzepine. *Hepato-Gastroenterol.* (in press).
- 6 Scarpignato C. Pirenzepine and mucosal protection: an overview. In: Cheli R, Molinari F eds. *Pirenzepine-knowledge and new trends*. Verona: Cortina International. 1986; 75–88.

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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References

- 1 Guslandi M, Ballarin E. Luminal leakage of HCO₃ in chronic antral erosions. *Hepato-Gastroenterol* 1986; **33**: 221–2.
- 2 Nesland AA, Berstad A. Erosive prepyloric changes in persons with and without dyspepsia. *Scand J Gastroenterol* 1985; **20**: 222–8.
- 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.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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References

- 1 Rowe PH, Mason RC, Jourdan MH. Effect of cimetidine and omeprazole on aspirin and taurocholate induced gastric mucosal damage. [Correspondence] *Gut* 1987; **27**: 5892.
- 2 Rowe PH, Lange R, Marrone G, Matthews J, Silen W. In vitro protection of amphibian gastric mucosa by nutrient HCO₃ against aspirin injury. *Gastroenterology* 1985; **89**: 767–78.
- 3 Rowe PH, Starlinger MJ, Kasdon E, Marrone G, Silen W. Effects of simulated systemic administration of aspirin, salicylate and indomethacin on amphibian gastric mucosa. *Gastroenterology* 1986; **90**: 559–69.
- 4 Lacy ER, Ito S. Microscopic analysis of ethanol damage to rat gastric mucosa after treatment with a prostaglandin. *Gastroenterology* 1982; **83**: 619–25.
- 5 Robert A, Nezamis JE, Lancaster C, Hanchar AJ. Cytoprotection by prostaglandins in rats: prevention of gastric necrosis produced by alcohol, HCl, NaOH, hypertonic NaCl and thermal injury. *Gastroenterology* 1979; **77**: 433–43.

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