LETTERS TO THE EDITOR

Helicobacter pylori and peptic ulcer recurrence

SIR,—I suppose that according to Moss and Calam's criteria (*Gut* 1992; **33**: 289–92) I should be called 'perverse,' yet I find it difficult to totally accept the concept that eradication of *Helicobacter pylori* is pivotal in preventing duodenal ulcer recurrence. In particular, the apparent parallelism between the lower relapse rate of ulcers treated with colloidal bismuth subcitrate and the antibacterial activity of the drug does need to be accepted with caution.

First of all colloidal bismuth subcitrate alone does not eradicate *H pylori* but only suppresses temporarily the microorganism and within one month only 10 per cent of subjects remain free of *H pylori*.[1] The reduction in the rate of ulcer recurrences, if related to the disappearance of *H pylori*, should be limited to early relapses occurring during the first month after colloidal bismuth subcitrate withdrawal. On the contrary, follow up studies carried out over 12 months show that colloidal bismuth subscitrate healed ulcers remain in remission to a greater extent than those healed with H2 blockers throughout the observation period.[2] Furthermore, if more than 70% of relapsing ulcers are *H pylori* positive, on the other hand 75% of Helicobacter-positive duodenal ulcers do not relapse.[3] Thus other factors must explain the longer remission of colloidal bismuth subcitrate healed ulcers.

A decrease in the rate of duodenal ulcer recurrence after treatment is discontinued has been seen also in patients healed with sucralfate, an anti-ulcer agent devoid of any effect on *H pylori*. Although evidence is conflicting, an analysis of pooled data from the literature shows that duodenal ulcers healed with sucralfate stay healed significantly longer than those initially treated with cimetidine or ranitidine.[4] Therefore, as already suggested by us,[5] and by others[6 7] it would seem that treating ulcers with drugs strengthening mucosal defences (irrespective of which gastroprotective drug is used and regardless of *H pylori*) results in longer lasting healing than after treatment with gastric acid inhibitors.

M GUSLANDI
Gastroenterology Unit,
S Raffaele Hospital,
Via Olgettina 60 20132, Milano, Italy

1 Rauws EAJ, Tytgat GNJ. Cure of duodenal ulcer associated with eradication of Helicobacter pylori. *Lancet* 1990; **335**: 1233–5.
2 Miller JP, Faragher EB. The potential impact of Campylobacter pylori on the treatment of duodenal ulcer disease. *Scand J Gastroenterol* 1989; **24** (suppl 160): 39–45.
3 Bianchi Porro G, Lazzaroni M. Campylobacter pylori and peptic ulcer therapy. *Gastroenterol Clin Biol* 1989; **13B**: 107–11.
4 Lam SK. Implications of sucralfate-induced ulcer healing and relapse. *Am J Med* 1989; **86** (suppl 6A): 122–6.
5 Guslandi M. Importance of defensive factors in the prevention of peptic ulcer recurrence. *Acta Gastroenterol Belg* 1983; **46**: 411–8.
6 McLean AJ, Harrison PM, Ionnides-Demos PM, Byrne L, McCarthy P, Dudley JF. The choice of ulcer-healing agent influences duodenal ulcer relapse rate and long-term clinical outcome. *Aust NZ J Med* 1985; **15**: 367–74.
7 Miller JP, Faragher EP. Relapse of duodenal ulcer: does it matter which drug is used in the initial treatment? *BMJ* 1986; **293**: 1117–8.

Reply

SIR,—We agree that the remission prolonging effect of DeNol does not in itself demonstrate the role of *Helicobacter pylori* in duodenal ulcer disease. The role of *H pylori* is shown, however, by the vast difference in relapse rates between patients who have and those who have not achieved eradication on triple therapy. Relapse rates are very much lower if the organism is eradicated.

J CALAM
S MOSS
Royal Postgraduate School of Medicine,
Hammersmith Hospital,
Du Cane Road,
London W12 0NV

Helicobacter pylori and peptic ulcer recurrence

SIR,—In the leading article by Moss and Calam (*Gut* 1992; **33**: 289–92) the point is made that colonisation of the duodenum only occurs in the presence of gastric metaplasia. Duodenal ulceration is invariably accompanied by gastric metaplasia, its presence suggesting a loss of normal mucosal defence. If this is so, any treatment that can both heal duodenal ulceration and restore normal duodenal mucosa should thereby restore normal mucosal resistance and also eliminate *Helicobacter pylori* colonisation.

We reported a small study[1 2] comparing duodenal ulcer relapse and the histological and ultrastructural changes in duodenal mucosa following healing and after one year's maintenance treatment with either sucralfate or cimetidine. At the end of one year we found a significant increase in the number of biopsy specimens showing absent or minimal gastric metaplasia in the patients on sucralfate (eight of 11) compared with those on cimetidine (five of 14). It was noted, in addition, that in the absence of gastric metaplasia no *H pylori* organisms were seen on either light or electron microscopy, and organisms were very rarely seen when there was minimal gastric metaplasia.[3] The subsequent relapse rate during the two years after cessation of treatment reflected these changes, being only two of 11 in the sucralfate group compared with nine of 13 in the cimetidine group, one patient being lost to follow-up.

These findings suggest that an alternative approach to the elimination of *H pylori* from the duodenum in the management of duodenal ulcer would be treatment aimed at the restoration of normal duodenal mucosa as well as ulcer healing. Sucralfate, which has no direct effect on *H pylori* organisms, was found to achieve this in a number of cases. It is possible that other treatments which enhance mucosal defence may be found to have similar results.

F I TOVEY
Y C YIU
E M HUSBAND
L BAKER
A P JAYARAJ
Department of Surgery,
University College and Middlesex School of Medicine,
The Rayne Institute,
University Street,
London WC1 6AU

1 Tovey FI, Husband EM, Yiu Chu Yiu, Baker L, McPhail G, Lewin MR, *et al.* Comparison of relapse rate after healing of duodenal ulceration and after one year's maintenance with cimetidine or sucralfate: a light and electron microscopy study. *Gut* 1989; **30**: 586–93.
2 Tovey FI, Husband EM, Yiu Chu Yiu, Baker L, McPhail G, Jayaraj AP, *et al.* Differences in mucosal appearances and in relapse rate in duodenal ulceration treated with sucralfate or cimetidine. *Am J Med* 1989; **86** (suppl 6A): 141–4.
3 Tovey FI, Baker L, Husband EM, McPhail G, Yiu Chu Yiu, Jayaraj AP. Therapy in *Helicobacter pylori* infection. *Lancet* 1990; ii: 1381.

Estimation of total hepatic blood flow by duplex ultrasound

SIR,—Carlisle *et al* suggested that the measurement of liver blood flow using duplex ultrasound might provide valuable haemodynamic information in a wide variety of liver diseases, such as cirrhosis and hepatic tumours (*Gut* 1992; **33**: 92–7).

Using duplex ultrasound and duplex/colour Doppler sonography, we have measured portal venous and total liver blood flows in 40 control subjects, 115 patients with colorectal cancer, 25 with gastric carcinoma, and 15 with breast carcinoma over the last two years. Fifty eight of these patients had overt liver metastases. While Carlisle *et al* measured blood flow in the hepatic artery proper, we measured the hepatic arterial blood flow component in the common hepatic artery segment, because blood flow measurement was more consistent in the latter. In the hepatic artery proper, measurement of the cross sectional area involved a far higher percentage error because of the small diameter of the vessel. Accurate visualisation and correction for the angle was also more of a problem as the hepatic artery proper often spirals around the portal vein in close proximity to the common bile duct. These factors would severely affect the accuracy of the measurements in a number of patients. Indeed, Carlisle *et al* could only include 10 of 40 subjects in their study for flow measurements. We managed to obtain measurements in almost all of our patients. Our estimation of total liver blood flow was made on the assumption that the gastric and gastroduodenal arterial components were not significant under a 12 hours fasting condition. Also, we were more interested in evaluating the diagnostic potential of duplex/colour Doppler sonography rather than absolute flow measurements in individual vessels.

We showed that in patients with overt liver metastases there was (a) a significant increase in hepatic arterial blood flow compared with controls, (b) a significant reduction in portal venous blood flow, (c) no significant change in total liver blood flow. The increase in hepatic arterial blood flow was reflected in an increase in both the time averaged velocity of blood and the cross sectional area of the artery while the reduction in portal venous blood flow was mainly associated with decreased cross sectional area.

We defined the Doppler perfusion index, as the ratio of hepatic arterial to total liver blood flow and showed that there was clear separation of Doppler perfusion index values in colorectal cancer patients with overt liver metastases and control subjects.[1 2] Similar findings also applied to metastases from breast, gastric, oesophageal, and carcinoid cancer.[3] We concluded that measurements of Doppler perfusion index using duplex/colour Doppler sonography may be of value in the detection of small and 'occult' hepatic metastases. In our study, some of the patients who had an apparently curative resection of the primary colorectal cancer had abnormally raised Doppler perfusion index values. This is interesting because Leveson in 1985, using dynamic scintigraphy to measure liver blood flow indirectly, had a similar group of patients with abnormal hepatic haemodynamics who went on to develop overt hepatic