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 subcitrate 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.

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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.

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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.

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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

metastases.⁴ Follow up studies are currently in progress.

We have also assessed the haemodynamic changes in patients with cirrhosis.⁵ As we expected, these patients also had abnormally raised Doppler perfusion index values but total liver blood flow was significantly reduced. The mechanisms underlying the changes in hepatic perfusion appear to be different in benign and malignant liver diseases. There is experimental evidence that the reduction in portal venous blood flow in subjects with liver metastases is because of increased splanchnic vascular resistance, whereas in cirrhosis, there is an increase in resistance to flow at the sinusoidal level of the liver. We have shown that the congestive index, defined as the ratio of the portal vein cross sectional area to the time averaged velocity of blood in the portal vein, was significantly raised in patients with cirrhosis compared with control subjects and patients with liver metastases. We concluded that duplex/colour Doppler sonography measurement of congestive index can discriminate between the perfusion changes because of the presence of hepatic metastases and those caused by cirrhosis, thereby increasing the diagnostic power of the technique.

The use of duplex/colour Doppler sonography rather than conventional duplex ultrasound would allow almost all patients with benign and malignant liver diseases to be assessed. It simplifies the examination with the ease of accurate identification of vessels of interest, and showing anatomical variations, and the duration of scanning is significantly reduced. This technique is, however, operator dependent and we suggest that a dedicated ultrasonologist is essential for meticulous standardised examinations to be performed, otherwise conflicting conclusions about duplex/colour Doppler sonography flowmetry in the abdomen will result. Further studies are required to explore the full diagnostic potential of this technique.

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Reply

SIR,—We wish to comment on the letter of Dr Leen *et al* to say that we are delighted that the estimation of the ratio of hepatic arterial flow to total hepatic blood flow using Doppler ultrasound does provide a valuable haemodynamic indicator in both malignant and cirrhotic disease, as we had hoped from our work.

We have noted that Dr Leen *et al* have applied a colour flow Doppler ultrasound system to their measurements and have chosen to study the common hepatic artery. Unfortunately, neither of these options was available to us since we did not have access to a colour flow system for this study. In addition, the original aim of our work was the study of the hepatic arterial buffer response in humans under non-invasive physiological conditions. We had previously established in a pilot study¹ that measurements of proper hepatic arterial flow were possible and the published work was undertaken as a precursor to studying proper hepatic artery and portal venous changes after feeding. The study of common hepatic artery flow was thus inappropriate since the gastric and gastroduodenal components would make a contribution to increase common hepatic artery flow when only hepatic arterial flow was required.

We had hoped to increase the population to whom the technique could be applied by showing that proper hepatic artery flow was a constant proportion of common hepatic artery flow. We showed, however, that the ratio of flow in the two vessels varies from subject to subject and seems to be dependent on the cross sectional areas of the vessels.

We are pleased that the measurements in the common hepatic artery of Leen *et al* do provide consistent results and thus extends the population to whom this technique is applicable. We are slightly concerned, however, that the gastric and gastroduodenal components are considered insignificant even in patients with gastric and oesophageal cancer!

We agree entirely with the concluding comments of Dr Leen and colleagues and hope that the full potential of flow measurements in the hepatic artery and portal vein using colour Doppler sonography will be exploited in both research and clinical applications in the future.

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BOOK REVIEWS

Current gastroenterology. Vol 11. Edited by G Gitnick. (Pp 386; illustrated; £69.95.) St Louis, MO: Mosby Year Book, 1991.

The problem with this book is that it is not about current gastroenterology. The authors did as they were told and reviewed 'last year's papers' in their subject but the year in question was mid-1988 to mid-1989. The book came out in late 1991. Such a delay reduces its value greatly. One reason for the delay may be the book's luxurious format – it is a hardback complete with photographs of x rays, CT scans

etc. But I suspect the main reason is that the authors had too much to cope with and could not meet their deadlines. Two of them had well over 300 papers to review. The ten chapters are, in the main, well written and they cover most of the important areas of gastroenterology, with little overlap. The chapter on nutrition is an oddity; it is a didactic review, has a relatively small reference list, and includes much non-gastrointestinal material.

If you like this kind of thing you will find it done just as well and much more currently in *Current opinion in gastroenterology*. I do question, however, the cost-benefit ratio of this approach and the value of the product. It is artificial to review a topic a year at a time and difficult to produce a readable, let alone, well argued article. I myself find articles of this kind hard to read and so tend not to read many of them. I suspect a market survey would show I am not alone. For the authors it is a hard slog producing a book like this; is it really worth all those ruined weekends?

K W HEATON

Ambulatory esophageal pH monitoring: practical approach and clinical applications. By Joel E Richter. (Pp 229; illustrated; £47.) New York: Igaku-Shoin Medical Publishers, 1991.

In his preface, Dr Richter notes that a practical 'how to do it' book on ambulatory oesophageal pH monitoring has not been available hitherto. This book aims to be a practical guide to the technique and to review its applications and limitations. Eighteen American authors have covered the subject comprehensively in seventeen chapters, beginning with an excellent little historical review and concluding with a section on alkaline reflux. Brief case reports, included in several chapters, provide succinct illustrations of why and how pH monitoring can (and sometimes cannot) be used to resolve a clinical problem.

This is undoubtedly a useful book for individuals who are introducing the technique to their own practice. They will soon, however, encounter the problems faced by everyone with experience of pH monitoring and the text is a little disappointing in its treatment of some of these difficult areas. The interpretation of oesophageal pH results obtained in patients with non-cardiac (or, more usually, normal coronary arteriogram) chest pain and in asthmatics could perhaps have been addressed more directly to aid the clinician who must make a clinical decision on the basis of the oesophageal findings.

My first consultation of the index – seeking guidance on equipment disinfection – referred me to the wrong page: the information provided in the section to which the index should refer is inadequate for a book claiming to be a practical guide. Other topics seem adequately and correctly indexed. Overall, the book scores 7 out of 10. There is nothing similar on the market and only by obtaining numerous journals can the reader obtain the information that is gathered together here in a sensible sequence. Like the technique itself, the book does not provide all the answers, but it is probably the best there is just now.

R C HEADING

Ulcer disease. Investigation and basis for therapy. Edited by Edward A Swabb and