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Reply

EDITOR,—Dr Dowling is right to emphasise that the eradication rate we obtained in our *H pylori* positive, NSAID associated peptic ulcers is fairly low (56%), and more convincing conclusions could have been made by using a more effective eradicating regimen (that is, omeprazole based triple therapy) providing a cure rate of at least 85–90%. Unfortunately, when this clinical study was originally planned (spring 1993), dual therapy (omeprazole with amoxycillin) was considered one of the most effective eradicating regimens available; however, it should be emphasised that the *H pylori* cure rate obtained with omeprazole/amoxycillin in this series is comparable with that reported previously in NSAID unrelated peptic ulcers using the same combination of drugs.[1 2] We also agree on the fact that our results should be considered preliminary because of the limited number of NSAID associated ulcers treated, and they need to be confirmed in further clinical trials involving larger series of patients on long term NSAID treatment with gastric or duodenal, or both, ulcer disease.

However, two important points emerge from our study. First, unlike NSAID unrelated peptic ulcers, eradication of *H pylori* is not associated with the "cure" of NSAID associated ulcers which tend to recur rapidly after initial healing if the patient continues to receive the NSAID but stops taking antiulcer medication. This necessarily implies that the risk of recurrence induced by the presence of *H pylori* infection, if any, is small and it is only additional to the main risk of receiving long term NSAID treatment. Second, *H pylori* status does not seem to have an important role in the healing of these lesions. This is not only confirmed by the observation that the rate of healing is not increased by eradication of *H pylori*, but also from the fact that *H pylori* negative ulcers respond to omeprazole just as well as *H pylori* positive ones. The major determinant of healing response to the antisecretory compound in these patients seems to be the concomitant intake of the NSAID during the healing phase, which delays the healing process irrespective of the type of drug used.

Recently, Hawkey *et al*,[3] in a very large study involving 541 patients with NSAID related gastroduodenal ulcers or erosions, have shown that infection with *H pylori* does not hamper the healing efficacy of omeprazole 20–40 mg daily; on the contrary, it tends to be associated with higher healing rates, perhaps as a result of the increased antisecretory effects of proton pump inhibitors in this setting.[4]

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Endoscopic papillectomy

EDITOR,—I read the recent article on endoscopic papillectomy by Dr Farrell *et al* (*Gut* 1996; **39:** 36–8) with considerable interest because of a simple experiment done in 1964, which indicated that papillectomy might be hazardous.[1] The closed duodenal loop model of haemorrhagic necrotic pancreatitis closely resembles severe human pancreatitis and is caused by the reflux of duodenal contents through the papilla of Vater[2]; it was suggested that pancreatitis could be produced by much smaller intraduodenal pressures provided that the papilla of Vater was damaged of incompetent.

The duodenal papilla in humans and in dogs is lined by mucosal folds or 'valvules', which serve to prevent regurgitation of duodenal contents.[3 4] The isolated dog duodenum was filled with coloured saline maintained at a pressure of 30 mm Hg without any fluid escaping from the cut ends of the pancreatic ducts or common bile duct. A simple mucosal papillectomy was performed at the main pancreatic duct, the duodenotomy incision closed, and the intraduodenal pressure raised again to 30 mm Hg. Within two minutes coloured saline oozed from the main pancreatic duct but not from the separate lesser pancreatic duct and associated common bile duct. When the common bile duct papilla was also excised saline escaped from the common bile duct.[1] The histological picture of the papilla, before and after excision (photographed), plus the findings above suggest that: (1) the mucosal portion of the papilla of Vater serves a useful purpose in preventing duodenal reflux; (2) papillectomy may increase the risk of developing pancreatitis, depending on the nature of the underlying pathology for which papillectomy was performed.

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Reply

EDITOR,—We are grateful for Dr McCutcheon's comments and while it is possible that endoscopic papillectomy may increase the risk of developing pancreatitis the fact remains that none of our 10 patients have suffered pancreatitis or ascending cholangitis after papillectomy (25 months follow up to date). In reply, we feel two points should be made. Firstly, the closed duodenal loop model described is not comparable with our patients' situation as there was no distal obstruction and hence no cause for reflux of duodenal contents through the damaged papilla of Vater. Secondly, while the common or external sphincter are excised at papillectomy both internal sphincters (biliary and pancreatic) are preserved hence serving as a natural barrier to reflux of duodenal contents. Finally, we would only recommend endoscopic papillectomy as a means of aiding cannulation in limited circumstances such as a large obstructive ampullary tumour, or an exophytic ampulla with an ectopic orifice or low bile duct stones.

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BOOK REVIEWS

Textbook of Gastroenterology. Volumes 1 and 2. Yamada T, Alpers D H, Owyang C, Powell D W, Silverstein F E. (Pp 3456; illustrated; £176.00.) Philadelphia: Lippincott-Raven. 1995. ISBN 0-397-51492-1.

A review of the second edition of this book published in 1995 was only requested in late 1996 (accompanied by an invoice marked "rush!") which explains the interval between publication and this review.

I was brought up on *Gray's Anatomy* to achieve adult height around the dining table. A wide selection of two volume books on gastroenterology could now do this job admirably.

This remarkable book has more than 200 contributors, mostly North American with a smattering of other contributors from seven different countries. Its approach is so different that comparison with other textbooks of gastroenterology is inappropriate. No other book could find the reviewer at the end of volume one, some 67 chapters and 1500 pages later, still not yet through the pylorus. This is not an obsession with the oesophagus and stomach, but extensive consideration of basic mechanisms relevant to clinical problems (26 chapters) and, more importantly, an extended section of similar length concerned with approaches to the symptomatic patient. This section is particularly relevant for the younger postgraduate whose patients in real life present with a constellation of symptoms rather than a specific diagnosis. This focused approach is both a strength and a weakness as