Correspondence

Duodenal ulceration and postoperative recurrence

SIR,—We have read with interest the leading article by Mr R M Kirk (*Gut* 1988; **29:** 1625–7). He draws attention to reports of the presence of parietal cells in the duodenum and suggests that these may enable 'susceptible mucosa to come into contact with freshly secreted acid that has not yet been partially diluted, adsorbed, absorbed, and neutralised'. During the past six years we have had a particular interest in gastric metaplasia in association with duodenal ulceration and have devised scoring systems for recording light and electron microscopical changes.[1–3] During this time several hundred duodenal biopsies have been examined and we have not yet seen any oxyntic cells in the presence of gastric metaplasia. Typically the mucosal surface is flat, without any gastric pits and is covered by epithelial cells secreting PAS staining mucus.

This stresses the importance of understanding the exact meaning of any references to the presence of gastric mucosa in the duodenum. It is important to distinguish between gastric metaplasia and the presence of heterotopic gastric mucosa. The former often involves widespread areas of mucosa covered with mucus secreting surface epithelial cells staining with PAS accompanied by varying degrees of inflammatory cell infiltration and frequently the presence of *Campylobacter pylori*. The latter involves scattered small islands of parietal cells, usually in association with Brunner's glands lying superficial to the muscularis mucosae, sometimes with occasional chief cells or small clusters of surface epithelial cells. Both are reported as occurring more frequently in association with duodenal ulceration.

Gastric metaplasia is thought to be either a defence mechanism or a manifestation of mucosal damage in response to hyperacidity, whereas the presence of heterotopic mucosa may be developmental and a possible course of localised hyperacidity contributing to ulcer formation.

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Reply

SIR,—Heterotopia is usually defined as anomalous differentiation of tissue and is considered to be a primary affair, while metaplasia is considered to be an alteration of tissue after it has differentiated normally. The distinction is somewhat artificial. When the stomach first develops it is lined with columnar epithelium which later differentiates into the various cells found in the adult stomach,[1] yet one would not call this metaplasia. In the adult, gastrin, or other as yet unidentified tropic factors may switch progenitor cells of the antrum to produce parietal cells, or of the fundus to produce functionally competent intestinal cells. The sweeping cell changes that occur during embryonic development are not necessarily once and for all time.[2]

Biopsy specimens from the duodenal cap may not be truly duodenal, as the duodenogastric junction is often indistinct and does not necessarily correspond with the muscular ring. Gastric cells in the anatomical duodenal bulb may be the distal end of the antrum.[3]

Gastric metaplasia is often stated to be either a defence mechanism or a manifestation of mucosal damage while heterotopia is thought to be a developmental anomaly. Both are, however, reported to be more frequent in the presence of duodenal ulceration. In both conditions, therefore, the mucosa is subject to the same acid attack. *Campylobacter pylori* as a possible factor in duodenal ulceration would be expected to colonise the gastric mucosal cells associated with islands of 'heterotopia' as they do in 'metaplasia', so that the presence of the organisms is not pathognomonic of metaplasia.

In duodenal ulcer patients the antrum is small owing to distal extension of the gastric fundus. It is likely that the discovery of parietal cells in the antrum and various types of gastric cells in the duodenal bulb,[3,4] represents this distal drive rather than being a response to damage.[5]

The parietal cells identified in the duodenum in gastrectomy specimens were sited in the ducts and may be missed in biopsy specimens taken through fibreoptic endoscopes.

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Balloon dilatation of benign oesophageal strictures

SIR,—We read with interest the recent paper in your journal by Cox *et al* (*Gut* 1988; **29:** 1741–7). The authors report that both methods are equally safe, but believe that bougie dilatation is better in reducing the incidence of redilatation, and recommend that balloon dilatation should be reserved for very select cases. Both methods used, involve radiology, and the patient staying in hospital overnight.

We are currently involved in the prospective evaluation of transendoscopic balloon dilatation as an outpatient procedure, for benign peptic strictures. We use a Rigiflex TTS transendoscopic balloon catheter (supplied by Keymed, Essex). Strictures are dilated under direct vision and do not require *x*-ray control. Over the past 18 months, we have performed 51 dilatations on 41 patients. The mean age group was 77·6 years. All procedures have been performed on an outpatient basis. No complications have been encountered. We have found the procedure an effective method of relieving symptoms, with only 10 patients requiring redilatation. We find this a safe and cost effective way of managing a common problem in an elderly population.

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Benign oesophageal stricture in Barrett's oesophagus

SIR,—I enjoyed the paper by Atkinson and Robertson[1] which showed that patients with benign oesophageal stricture associated with Barrett's oesophagus do well with conservative management. Their study showed that only 11 of 23 patients (48%) required more than one dilatation during follow up. In a much smaller series of such patients included in a paper by Barbezat and myself, seven patients with Barrett's oesophagus and benign oesophageal stricture were followed up and only three (42%) needed more than an initial dilatation.[2] I was interested that Atkinson and Robertson[1] found that 41% of their 56 patients with Barrett's oesophagus had a benign oesophageal stricture. In contrast, Barbezat and I found only 10 patients with benign oesophageal stricture (19%) in a series of 52 patients with Barrett's oesophagus from Dunedin, New Zealand. In other series listed in Atkinson and Robertson's paper, benign oesophageal stricture was reported in 31% to 81% of patients with Barrett's oesophagus. I think the discrepancy between our figures and the others may be explained by the selection of cases in the series. Many of the reported series with much higher figures for the prevalence of benign oesophageal stricture are surgical series; surgeons are likely to see patients with troublesome symptoms and/or complications of Barrett's oesophagus. Patients with Barrett's oesophagus but few or no symptoms and no complications, are not going to be referred for surgery. This type of bias can be seen in a previous series from Dunedin, which included all patients with Barrett's oesophagus referred to the thoracic surgical unit between 1952 and 1973. All 45 patients in the series had evidence of benign oesophageal stricture on barium swallow and 44 of the 45 patients complained of dysphagia.[3] Similar bias may be present in series such as those of Atkinson and Robertson,[1] which report cases from centres with a high reputation in the management of oesophageal disease and who are likely to acquire patients with symptomatic or complicated Barrett's oesophagus. Barbezat and I found 52 patients with Barrett's oesophagus from among all patients endoscoped in a medical gastroenterology unit which provided all endoscopic services for the Dunedin area in a period from January 1981 to December 1986. Ten per cent of patients endoscoped with evidence of gastro-oesophageal reflux had Barrett's oesophagus; these figures are similar to those from other centres.[4,5] A quarter of our patients with Barrett's oesophagus did not have symptoms related to the oesophagus. I submit that our series contained a broader and perhaps more representative spectrum of Barrett's oesophagus and in such series the numbers of patients with complications such as benign oesophageal stricture will be less. The latest figures from our own unit at Dudley Road Hospital support this view; 35 cases of Barrett's oesophagus have been diagnosed from among all patients undergoing endoscopy over the last two years and only four patients had benign oesophageal stricture. Barrett's oesophagus is common and endoscopists are becoming much more aware of it, therefore it is likely that many cases diagnosed in the future will be at an earlier stage and without complications.

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