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.

FRANK I TOVEY, E M HUSBAND, YIU CHU YIU, L BAKER, G MCPHAIL, AND A P JAYARAJ

Basingstoke District Hospital, Basingstoke, Hants and University College, London WC1

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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.

R M KIRK

Royal Free Hospital, Pond Street, London NW3 2QG

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