Alkaline areas in gastric mucosa after gastric surgery

W. M. Capper, T. J. Butler, and K. G. Buckler

From the Department of Surgery, Southmead and Frenchay Hospitals, Bristol

EDITORIAL SYNOPSIS  These studies demonstrate that duodenal reflux into the stomach after gastric surgery causes an adjacent area of mucosal alkalinity likely to be due to an atrophic gastritis.

The variations in size of the alkaline area of the gastric mucosa in disorders such as gastric ulcer, duodenal ulcer, and combined gastric and duodenal ulcer have been reported previously (Capper, Laidlaw, Butler, and Richards, 1962; Capper, Butler, Buckler, and Hallett, 1966). The areas were defined at operation using a pH monitoring system, and were confirmed histologically afterwards. Recently, opportunities have occurred permitting the application of the same technique to the study of gastric mucosa following previous gastric operations.

MATERIAL AND METHODS

The following groups of patients were investigated:—
(a) Two patients after Billroth I operation had recurrent ulcers requiring vagotomy and refashioning of the stoma.
(b) Four patients after Polya operations required conversion of the anastomosis to a Billroth I for steatorrhoea.
(c) Two patients after gastrojejunostomy developed further ulceration and vagotomy or vagotomy and antrectomy was necessary.
(d) One patient after hemigastrectomy with a Roux-en-Y anastomosis had a long afferent loop and conversion to a gastro-duodenostomy was done.

In each case the gastric remnant was opened at operation and the pH of the mucosa was recorded during continuous histamine stimulation (Capper et al., 1962). The monitoring procedure was started at the anastomosis and carried on towards the fundus.

RESULTS

The findings are illustrated in Figure 1. Despite previous excision of the existing alkaline zone ('antrum'), there was a new alkaline area in each case adjacent to the stoma. The peristomal area following gastrojejunostomy is particularly worthy of note (Fig. 1c). An important exception to these findings was seen in the patient with the Roux-en-Y anastomosis. The gastric mucosa was found to be acid-secreting right down to the line of the anastomosis with the jejunum. This was the only case in the series where there was no reflux of duodenal contents into the gastric remnant.

DISCUSSION

The per-operative study of the pH of gastric mucosa during histamine stimulation depends on the presence or absence of oxyntic cells in the mucosa under test. The appearance of a new alkaline zone, where the mucosa was previously acid-secreting, indicates that the oxyntic cells must have disappeared. This change only occurs, however, when duodenal contents have free access to the gastric remnant.

In his classical experiments on dogs, Lawson (1964) has shown that atrophic gastritis with disappearance of oxyntic cells in fundic mucosa follows a Billroth I and Polya gastrectomy (Figs. 2a and b). When duodenal contents were diverted to flow over the gastric mucosa (Fig. 2c), peristomal gastritis and similar changes in the antrum ensued; this change extended beyond the previous antrofundic line to involve adjacent fundic mucosa. Before each procedure was carried out the normality of the various mucosal areas was established histologically. When the duodenal contents were diverted away from the stomach by a Roux-en-Y anastomosis (Fig. 2d), the gastric mucosa remained normal in all respects. These studies indicate the importance of duodenal contents in the causation of mucosal changes in the stomach. Further, Lawson established that the change was not due to bile or pancreatic juice alone but to the combination of the two.

Our observations in man, using a different method of assay, confirm Lawson's findings in dogs. We assessed the disappearance of oxyntic cells in vivo by estimating the altered pH of the mucosa, whereas Lawson recorded histological changes, which consisted of atrophic gastritis, superficial gastritis, the replacement of oxyntic cells by mucus-secreting cells, and a tenfold increase in mitoses. It appears, therefore, that a major cause of atrophic gastritis in man and dogs is duodenal reflux.

At this juncture, it is worth recording a note of caution with reference to the histological examination of secondary gastrectomy specimens. Because of the difficulty sometimes experienced in the
Alkaline areas in gastric mucosa after gastric surgery

FIG. 1a, b, c, and d. Illustration of the findings of pH monitoring of gastric mucosa following various gastric operations. The black area indicates the extent of the new alkaline zone. The arrow shows the direction of flow of duodenal contents.

FIG. 2a, b, c, and d. Illustration of the procedure and findings in dogs (Lawson, 1965). The black area indicates the zone of gastritis.
differentiation of true pyloric mucosa and altered fundic mucosa, the changes reported here occurring around the anastomosis should be recognized otherwise the retained antral mucosa may be falsely interpreted. If there is doubt about the nature of alkaline-secreting mucosa from the stomach, whether it is true antral mucosa or altered fundic mucosa, then gastrin assay is the only certain method of identification.

SUMMARY
The experimental findings of Lawson (1964), following gastric surgery in dogs showing histological changes of atrophic gastritis with disappearance of oxyntic cells, are confirmed in man by pH monitoring of gastric mucosa when re-operation has been necessary. They strongly suggest that reflux of duodenal contents over gastric mucosa is the cause of the change and the variations encountered in the alkaline areas. The fact that the alkaline areas in man increase proximally from the pylorus or extend from the anastomosis is best explained by the occurrence of duodenal reflux.

Variation of the degree and extent of duodenal reflux with the resulting loss of oxyntic cells may be a factor in the variations of acid output. Caution is expressed with reference to the interpretation of the histology of alkaline-secreting gastric mucosa, and gastrin assay may be needed to determine its true nature.

We are particularly indebted to Mr. H. H. Lawson for details of his experimental procedures and findings.

REFERENCES