

Duodenal pH values in normal controls and in patients with duodenal ulcer¹

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Recent studies by Wormsley and Mahony (1967) and by Banks, Dyck, Dreiling, and Janowitz (1967) have shown that the ratio between the secretory capacity of pancreatic bicarbonate and gastric acid is lower in duodenal ulcer patients than in normal subjects. It has therefore been suggested (Wormsley and Mahony, 1967; Rovelstad, 1956) that duodenal neutralization is insufficient in this disease.

Some studies have supported this theory by demonstrating higher duodenal pH values in normal controls than in subjects with duodenal ulcer (Kearney, Lomfort, and Osterberg, 1941; Atkinson and Henley, 1955; Archambault, Rovelstad, and Carlson, 1967). Other studies, however, have not been able to show such a difference (Rhodes and Prestwich, 1966) and this question can still be considered open. The present study investigates this problem further by measuring fasting and post-prandial duodenal pH values in normal and ulcer subjects. By selecting the cases so that the two groups did not differ with respect to gastric secretory capacity, a possible difference due to the gastric hypersecretion usually associated with duodenal ulcer could be eliminated.

MATERIAL AND METHODS

The study comprised two groups of adult men, nine with duodenal ulcer and eight without ulcer disease. The two groups were identical with respect to gastric secretory capacity as determined by the augmented histamine test: ulcer group 35.3 m-equiv/hr \pm 8.1, control group 34.8 m-equiv/hr \pm 5.8.

All subjects in the ulcer group had a typical history and a definite niche was demonstrated in the bulb during a barium meal examination. This x-ray examination was also performed in the control group where it showed a normal stomach and duodenum in all cases.

The pH electrode used (GK 282c, Radiometer, Copenhagen) was specially designed for measurements in the gastrointestinal tract (Rune, 1968).

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The electrode was connected to a pH meter 27 (Radiometer), and the pH was continually recorded on a Beckman potentiometer recorder. The pH meter was also connected to an 'average-pH analog computer' (Medi-tronic, Copenhagen), showing the mean pH in each period and the percentage of time the pH had been below 3 and above 5. Before each study the zero point and sensitivity of the electrode was adjusted in two buffers, with a pH of 7.4 and 2.0 respectively. This procedure was repeated after each study. In a few cases significant changes in zero point or span had developed during the measurements *in situ*; these cases were excluded and the electrodes discharged. The pH measurements were all performed 6 to 8 cm distal to the pylorus, *ie*, in the proximal part of the descending duodenum.

To secure a constant and reproducible position the patient was kept lying on the x-ray table during the investigation so that frequent fluoroscopic controls could be made. In addition potential difference measurements were used to monitor the position of the electrode (Archambault *et al*, 1967; Andersson and Grossman, 1965). This method is based upon studies demonstrating that the potential of the luminal surface of the gastric mucosa is negative to that of the duodenal mucosa. This potential difference was measured continuously by means of two polyvinyl tubes (internal diameter 0.91 mm) filled with saturated KCl and glued to the pH electrode in such a way that one ended 6 cm and the other 9 cm proximal to the bulb of the glass electrode. These tubes were closed distally with porous plugs, through which the liquid junction was established. The flow of KCl through the plugs was approximately 10 μ l/hr. Proximally the tubes ended in two calomel half-cells, connected to a second pH meter, serving as a millivoltmeter.

After an overnight fast the pH electrode was passed through a nostril into the stomach and advanced into the duodenum under fluoroscopic control. When the distal part of the descending duodenum was reached measuring the potential difference was started. In this position a potential difference of less than 10 mV was usually found. The electrode was then slowly withdrawn until a sharp rise in potential difference was observed. The magnitude of this increment was approximately 30 mV. During the rest of the study this position was controlled with short intervals by means of the potential

TABLE I
FASTING AND POSTPRANDIAL pH MEASURED 6 TO 8 CM DISTALLY TO THE PYLORUS

Period ¹	Mean pH			Percentage of Time with pH below 3			Percentage of Time with pH above 5		
	A	B	C	A	B	C	A	B	C
Normal subjects (n = 8)	6.3 (±0.9)	5.3 (±0.9)	5.2 (±1.1)	6 (±11)	4 (±13)	9 (±22)	87 (±20)	67 (±34)	64 (±37)
Patients with duodenal ulcer (n = 9)	6.3 (±1.1)	5.4 (±0.9)	5.4 (±1.0)	3 (±12)	2 (±4)	2 (±4)	88 (±22)	65 (±29)	65 (±31)

¹A: Forty min before the meal

B: First 40 min after the meal

C: Forty to 100 min after the meal

difference and by fluoroscopic examination. The position of the electrode was corrected if necessary. After the meal the potential difference often diminished; in these cases the position of the electrode was controlled by fluoroscopy alone.

The patients were studied for one to two hours in the fasting state. Then the meal, consisting of 170 g of fried minced meat was given, and the measurements were continued for another two hours. For each 20-min period the mean pH and the percentage of time the pH was below 3 and above 5 was registered.

The maximal gastric secretory capacity was determined by the augmented histamine test.

RESULTS

No significant difference was found between the duodenal pH in the two groups, either in the fasting state, or after the meal. Table I shows the results summarized into three periods: 40 min before the meal, the first 40 min after the meal, and the following 60 minutes. The mean pH fell from 6.3 in the fasting period to values around 5.4 after the meal. No systematic difference was found between the first and the second postprandial period. It can also be seen that the pH fell below 3 for short periods only, while fluctuations between pH 3 and 5 occurred in approximately 30% of the time after the meal. The frequency of the fluctuations was the same in the two groups, but within the groups they were rather widely distributed, as demonstrated by the standard deviations.

DISCUSSION

The aim of the study was to see if the duodenal neutralization was insufficient in a group of patients with duodenal ulcer. The results show that the pH 6 to 8 cm distal to the pylorus was relatively stable around 5 to 6 in the fasting state as well as after a meal. From these pH values, together with the values for the duodenal carbon-dioxide tension found in dogs (Rune and Henriksen, 1968) and man (Rune, 1968, unpublished), the bicarbonate concentration in the duodenal contents can be calcu-

lated. This leads to concentrations between 2 and 20 m-equiv per litre, demonstrating a surplus of base in the duodenum. Furthermore no difference was found in the pH between the ulcer group and the control group. Therefore it seems a reasonable conclusion that the duodenal secretion of bicarbonate is sufficient in patients with duodenal ulcer. This, however, does not exclude the possibility of a decreased neutralization in the duodenal bulb, due to insufficient regurgitation and mixing of the pancreatic juice.

SUMMARY

Eight normal subjects and nine patients with duodenal ulcer were studied. The gastric secretory capacity was the same in the two groups. The pH electrode was placed 6 to 8 cm distal to the pylorus, as judged from a combination of frequent fluoroscopic controls and continuous registration of the potential difference across the pylorus. In the fasting state the mean pH was 6.3 (SD 1.0) in both groups. After the meal, consisting of minced meat, it fell to values around 5.3 (SD 1.0); no significant difference was found between the two groups. It is therefore concluded that duodenal ulcer is not related to an insufficient capacity for neutralization in the duodenum.

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