Mastication and acid secretion

PUSHPA DUBEY
Ph.D.

S. NUNDY
M.Ch., F.R.C.S., F.R.C.P.

Department of Surgery, All India Institute of Medical Sciences, New Delhi-110029, India

Summary

The gastric acid response and the buffer capacity of the stomach were measured in 30 control subjects and 22 duodenal ulcer patients in response to a masticatory (solid) and a non-masticatory (homogenized) meal having the same chemical composition. The peak acid output values were equal after a masticatory and after a non-masticatory diet in control subjects (masticatory 18.1 ± 1.2 , non-masticatory 15.7 ± 1.3 mmol/hr) as well as in duodenal ulcer patients (masticatory 35.7 ± 1.5 , non-masticatory 33.7 ± 1.1 mmol/hr). The buffer capacity of the stomach contents 1 hr after the meal was significantly greater after a masticatory diet than a non-masticatory diet in both controls (14.6 ± 1.4 and 9.0 ± 1.9 mmol) and in duodenal ulcer patients (9.5 ± 1.5 and 7.5 ± 1.2 mmol). Duodenal ulcer patients had a significantly lower buffer capacity compared with controls. Masticatory diets may play a part in protecting individuals from developing duodenal ulcer.

KEY WORD: duodenal ulcer.

Introduction

Differences in the incidence and severity of duodenal ulcer disease in different parts of the world have been reported by various workers (Langman, 1973; Konstam, 1959). These striking geographical variations have been partly ascribed to the differences in the food habits in those places including the varying consistency of the diets (Tovey, 1979). For instance, it has been found that the incidence of duodenal ulcer is higher in South India where diets are sloppy and tend to be bolted down than in North India where diets are mainly solid and require mastication (Malhotra *et al.*, 1965). Even if solid and liquid diets are of similar chemical composition, physical differences might influence digestive functions.

The present study was designed to investigate the

effect of masticatory and non-masticatory homogenized diets having the same chemical composition and volume on the secretion of acid and buffer capacity of the stomach.

Material and methods

Patients. Fifty-two individuals were studied, 30 control subjects (mean age 36 ± 1.7 (s.e.m.) years) without any gastrointestinal disease and 22 patients (mean age 33 ± 1.1 years) with duodenal ulcer proved by barium meal or endoscopy.

Standard meal. This consisted of 30 g wheat chapatis, potato curry made with 50 g potatoes, 1 g oil (vegetable ghee), 1 g salt and 500 ml water.

Methods

In vitro studies

Effect of mastication alone on the pH of the diet. The patient was asked to chew the standard diet of chapatis and potatoes, and spit it out into a container in a total time of 20 min. The resulting mixture of the meal and saliva was homogenized and its pH measured using a Beckman's pH meter (Beckman Co., Philadelphia). The unmasticated meal alone was also homogenized separately and its pH measured.

In vivo studies

Augmented histamine test. After an overnight fast the patient was intubated with a Levin tube, the tip of which was positioned in an 'optimal' place in the stomach using the method of Hassan and Hobbs (1970). The resting juice was aspirated and discarded.

The stomach contents were then aspirated manually every 2 or 3 min and the aspirates divided into 15-min fractions. 'Basal aspiration' was continued for 1 hr in the middle of which period 10 mg of mepyramine maleate was injected intramuscularly. At the end of the basal period 0.04 mg/kg histamine acid phosphate was injected subcutaneously and gastric aspiration continued for a further hour.

Estimation of gastric acid secretion after meal. The overnight fasting patient was intubated with a 16 F Levin tube to the side of which was attached a small polyvinyl tube. This small tube had its distal opening 10 cm proximal to the tip of the Levin tube. The tip of the tube was positioned in an optimal place for aspiration using the water recovery method of Hassan and Hobsley (1970).

Basal acid output. This was estimated for 1 hr. The intragastric titration method (Fordtran and Walsh, 1973), was used for the estimation of acid secretory output. This involved maintaining the intragastric pH at 5.5 by aspirating aliquots every 2 min, measuring the pH and infusing, if necessary, 0.3 N sodium bicarbonate via the side tube.

Peak acid output. The masticatory diet of chapatis and potatoes was given to the patient who was instructed to chew it well and finish it in 20 min. On another day, the non-masticatory homogenized diet was swallowed. The rate of acid secretion was measured by intragastric titration for the next 4 hr.

Estimation of buffer capacity. The pH of the gastric juice alone was lowered to 2.5 by adding 0.1 N hydrochloric acid. It was then raised to 5.5 by infusing 0.3 N sodium bicarbonate into the stomach. The masticatory or non-masticatory meal was then consumed by the patient. One hour after the meal was taken, the intragastric pH was again lowered to 2.5 with hydrochloric acid and then raised to 5.5. The buffer capacity of the meal was calculated as the amount of alkali required to raise the pH of the gastric contents after the meal from 2.5 to 5.5 minus the amount of alkali required to raise the pH of the gastric juice from 2.5 to 5.5.

Calculations

Basal acid output. Sum of the last two 15-min values of acid in the basal period $\times 2$ (mmol/hr).

Peak acid output. Sum of the two highest acid output values after food or histamine $\times 2$ (mmol/hr).

Statistical analysis. The difference between groups were calculated using Student's paired *t*-test. Where appropriate results were subjected to correlation analysis.

Results

The effect of mastication on the pH of the diet

The pH of the masticated diet which was chewed and spat out was (mean $\pm$ s.e.) 6.5 ± 0.9 and that of the homogenized unmasticated diet was 6.3 ± 1.3 . This difference was not statistically significant ($P > 0.05$) indicating that saliva has little effect in altering the pH of the meal.

Mastication and acid secretion

The peak acid response to solid and liquid meals was significantly correlated both in control ($r = 0.86$, $P < 0.05$) and in patients with duodenal ulcer ($r = 0.72$, $P < 0.05$) and there were no significant differences between them (Fig. 1). The peak acid output after histamine in control subjects (16.3 ± 1.2 mmol/hr) was similar to that after solid (18.1 ± 1.2 mmol/hr) and homogenized (15.7 ± 1.3 mmol/hr) meals, but in patients with duodenal ulcer peak acid output after solid (35.7 ± 1.5 mmol/hr) or homogenized (33.7 ± 1.1 mmol/hr) meals were significantly higher than after histamine (29.1 ± 1.3 mmol/hr), ($P < 0.05$).

FIG. 1. Histograms representing the basal and peak acid output after histamine, masticatory and non-masticatory diets in controls and duodenal ulcer (D.U.) patients. (□) histamine; (▨) masticatory diet; (▩) non-masticatory diet. NS = non-significant.

Peak acid outputs after each of the stimuli were significantly higher ($P < 0.05$) in patients with duodenal ulcer than in controls.

Buffer capacity of the stomach

The buffer capacities after solid meals were significantly higher than after homogenized meals both in control subjects (14.6 ± 1.4 v. 9.0 ± 1.9 mmol) and in patients with duodenal ulcer (9.5 ± 1.5 v. 6.5 ± 1.2 mmol). With both types of meals, patients with duodenal ulcer had significantly lower ($P < 0.05$) buffer capacity than controls.

Discussion

This study shows that saliva alone has little effect on the pH of a standard diet, corroborating the findings of Baume (1968). Similarly, there is no difference in the gastric acid response to a masticatory or a non-masticatory diet. However, a masticatory diet has a buffer capacity after ingestion which is

significantly greater than that of a non-masticatory diet.

The reasons for this are matters for conjecture. The results are unlikely to be due to the protein binding and buffering capacity of the saliva, but possibly due to the different consistency of the two diets. The liquid diets may be emptied from the stomach more rapidly than the solid diets and therefore unable to bind acids (Hopkins, 1966).

A chewed diet mixed with saliva may also physically adsorb H^+ ions. Although the larger solid particles have much smaller binding surface areas relative to their weights than the finely dispersed particles, solid food stays in the stomach longer than one with a more liquid consistency.

The study also shows that the meal-stimulated acid secretory response in duodenal ulcer patients was significantly higher than the maximal acid response to histamine (MAO). This has also been demonstrated by Fordtran and Walsh (1973) but not by others (Rune, 1967; Malagelada *et al.*, 1976). The higher gastric acid response to a meal in our patients in comparison to histamine is probably not due to the use of a sub-maximal dose of histamine as it has already been demonstrated in our laboratory (unpublished data) that the optimal dose of histamine for maximal acid secretion in North Indian subjects is 0.04 mg/kg. The significantly higher meal mediated acid response in duodenal ulcer disease may be the result of several factors, such as increased parietal cell responsiveness to food, an increased vagal tone and an increased gastrin response (Fordtran and Walsh, 1973).

Patients with duodenal ulcer not only are acid hypersecretors but have a lower buffering capacity than control subjects. Our results on the gastric buffer capacity are similar to those of Fordtran and Walsh (1973) who have also shown a more rapid emptying of buffer after a meal in patients with duodenal ulcer. However, they are at variance with those of Jalan *et al.* (1979) who also used the same method but did not find any significant difference in the buffer capacity of patients with duodenal ulcer and control subjects. We cannot explain why this discrepancy has occurred. It should be emphasized however that Fordtran and Walsh's method mea-

sures only the emptying rate of buffer and not the emptying rate of the entire contents of the stomach. Our duodenal ulcer patients had therefore a higher acid secretion and lower buffer capacity than control subjects.

Both these mechanisms may be important in ulcer pathogenesis. The higher buffering capacity of the solid diets which require mastication may protect the gastric mucosa from ulceration and low buffer capacity of the non-masticatory diets reduces the efficiency of the buffer in preventing a rise in gastric acidity. This may account for the lower incidence of ulcer in those areas of the world where diets are mainly solids and require mastication.

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References

- BAUME, P.E. (1968) The source of gastric secretory inhibitor from human saliva. *Australian Journal of Medicine*, **17**, 42.
- FORDTRAN, J.S. & WALSH, J.H. (1973) Gastric acid secretion rate and buffer content of the stomach after eating. *Journal of Clinical Investigation*, **52**, 645.
- HASSAN, M.A. & HOBBSLEY, M. (1970) Positioning of subjects and of nasogastric tube during a gastric secretion study. *British Medical Journal*, **1**, 455.
- HOPKINS, A. (1966) The pattern of gastric emptying: a new view of old results. *Journal of Physiology (London)*, **182**, 144.
- JALAN, K.N., MAHALANABIS, D., MAITRA, T.K. & AGARWAL, S. (1979) Gastric acid secretion rate and buffer content of the stomach after a rice and a wheat based meal in normal subjects and patients with duodenal ulcer. *Gut*, **20**, 389.
- KONSTAM, P. (1959) Peptic ulcer in India. *Indian Journal of Medical Research*, **13**, 486.
- LANGMAN, M.J.S. (1973) Changing pattern in the epidemiology of peptic ulcer. *Clinics in Gastroenterology*, **2**, 4.
- MALAGELADA, J.R., LONGSTRETCH, G.F., SUMMERSKILL, W.H.J., GO, V.L.W. (1976) Measurement of gastric functions during digestion of ordinary solid meals in man. *Gastroenterology*, **70**, 203.
- MALHOTRA, S.L., SAIGAL, O.N. & MODY, G.D. (1965) Role of saliva in the aetiology of peptic ulcer. *British Medical Journal*, **1**, 1220.
- RUNE, S.J. (1967) Individual variation in secretory capacity of gastric acid to stimulation with solid food and with histamine. *Clinical Science*, **32**, 443.
- TOVEY, F.I. (1979) Peptic ulcer in India and Bangladesh. *Gut*, **20**, 329.

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