Gut, 1965, **6**, 113

Effect of different foods on the acidity of the gastric contents in patients with duodenal ulcer

Part I A comparison between two 'therapeutic' diets and freely-chosen meals[1]

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EDITORIAL SYNOPSIS This study shows that the gastric acidity in 12 duodenal ulcer patients was no greater when they took a completely 'free-choice' diet than when they took a typical 'therapeutic' diet as conventionally advised for peptic ulcer.

The acidity of the gastric contents in patients with duodenal ulcer tends to be higher than normal. This is true after a gruel test meal (Barford, 1928) and in patients taking a light diet (Ronald, 1939; James and Pickering, 1949). One aim of most dietary regimes for peptic ulcer is to reduce the acidity of the gastric contents. In this study, the acidity of the gastric contents has been measured in patients with duodenal ulcer while they took two 'therapeutic' diets and the results are compared with those obtained when the same patients took freely-chosen 'normal' meals.

METHOD OF STUDY

SELECTION OF PATIENTS Each of the 12 patients studied suffered from a radiologically-proven duodenal ulcer, with a typical history and without evidence of delayed gastric emptying. The experimental nature of the test, and the reason for it, were carefully explained to each patient, and the experiment was only performed with his willing cooperation.

CONDITIONS OF THE EXPERIMENT The patients were all admitted to hospital for medical or surgical treatment of the ulcer. During the tests they were allowed up and were encouraged to lead as normal a life as possible in the ward. Smoking was not prohibited. Antisecretory drugs were discontinued. No regular antacids were given but patients were allowed antacid tablets if pain occurred. Only two patients required any tablets, one taking eight tablets over three days and the other two tablets. A hypnotic was given at night and sedatives were continued if already prescribed.

DIETS STUDIED The two 'therapeutic' diets, in use at a London teaching hospital at the time of study, were constituted as shown below:

The gastric II diet was generally used soon after the patient's admission to hospital for medical treatment of the ulcer and the convalescent diet was advised at the end of the period of in-patient treatment.

For the 'freely-chosen' diet, the patients were offered a menu giving as wide a choice as possible. They were encouraged to choose 'normal' food and no restrictions on the type or amount of food taken were imposed. The main course taken at each meal is set out in Table I; vegetables, dessert, bread, pastries and beverages were taken in addition. No patient complained that his symptoms were aggravated by this diet.

Each test period lasted 24 hours from 8 a.m. on one day to 7 a.m. on the next. The order of the test periods was

[1]This work was included in a thesis submitted by one of the authors (J.E.L.-J.) to the University of Cambridge for the degree of M.D.

	Gastric II Diet	*Convalescent Gastric Diet*
Breakfast	Weak tea; 1 egg; 2 slices thin toast	Cereal; egg, fish, or grilled bacon; toast
Lunch	1½ tablespoons flaked fish, minced chicken, tripe or sweetbread; creamed potato and sieved vegetables; cereal, custard, or jelly	Fish, tripe, sweetbread, chicken, lamb, minced beef or mutton; mashed potato and vegetable puree; cereal, custard, jelly, or sponge
Tea	Weak tea; 3 slices thin bread and butter	Weak tea; toast or bread and butter
Supper	Soup and toast; cereal or custard	Soup; fish, egg, or cheese dish; pudding
	10 a.m. } 5 oz. milk 2 p.m. } drink if 8 p.m. } desired	

TABLE I

MAIN COURSE TAKEN BY PATIENTS WHEN GIVEN A FREE CHOICE

Patient	*Breakfast*	*Lunch*	*Supper*
1	Fish (haddock)	Lamb chop	Eggs and chips
2	Poached egg	Roast beef	Beef roll
3	Fried bacon	Steak and kidney	?
4	Bacon and egg	Chicken	Lamb chop
5	Bacon and egg	Fish (plaice)	Beef salad
6	Bacon and egg	Chicken	Chicken puffs
7	Bacon and egg	Heart	Minced meat
8	Bacon and egg	Roast beef	Roast lamb
9	Bacon and egg	Steak	Roast lamb
10	Bacon and sausage	Meat casserole	Fish
11	Bacon and egg	Fish and chips	Cheese on toast
12	Bacon and egg	Roast beef	Ham salad

varied from patient to patient according to a Latin-square pattern.

INTUBATION AND ASPIRATION OF SAMPLES A plastic, radio-opaque, Ryle's tube, 3 mm. in external diameter (10 Fr gauge), and having four aspiration holes (approx. 1 mm in diameter) placed between 5 and 8.5 cm. from the tip, was used.[1] It was passed through the nose and the tip was guided fluoroscopically into the likely position of the gastric antrum, using the left leaf of the diaphragm and the vertebral bodies as landmarks.

Samples of 5 to 10 ml. of gastric contents were withdrawn hourly on the hour, and before meals were begun. They were stored in a refrigerator until the acidity was measured.

MEASUREMENT OF ACIDITY The *p*H of the unfiltered samples was determined to the nearest 0·05 *p*H unit at room temperature with the sealed glass electrode, standardized at *p*H 4 and *p*H 9. The presence of visible food or bile in each sample was recorded.

ANALYSIS OF RESULTS The 'acidity' of a fluid is determined by the hydrogen ions in it. The acidity of a buffered solution cannot be accurately measured by removing hydrogen ions, so altering the equilibrium, as occurs during titration with a base. An electrometric method has to be used for determining acidity. A strong acid, such as hydrochloric acid, is believed to be completely ionised at all concentrations. Its solution properties, however, including acidity, depend not on the concentration of ions present but on their 'activity', a concept which takes account of deviations from ideal behaviour due to electrostatic interactions between the ions. Though concentrations of hydrogen ion can be calculated for a strong acid by correcting electrometric determinations by published 'activity coefficients', the results may be unphysiological as it is likely that biological properties depend rather on the solution properties and thus on the 'activity' of the ion.

The glass electrode neither measures concentrations of hydrogen ion nor does it measure precisely activities of the ion. These limitations do not limit the usefulness of the method which gives a 'practical scale of acidity' (MacInnes, 1948). For these reasons neither concentrations nor activities of hydrogen ion are calculated in this paper but the results are expressed as *p*H units.

The *p*H scale, being logarithmic, is convenient for expressing ratios but not so convenient for expressing arithmetic differences. Thus *p*Hl bears the same ten-fold ratio to *p*H2 in terms of hydrogen ion activity as does *p*H2 to *p*H3. The arithmetic differences in hydrogen ion activity, however, between these values are very different as they also bear this ten-fold ratio to one another, being 90 mEq./l. in the former and 9 mEq./l. in the latter. When comparisons have to be made between groups of samples of different *p*H, three methods of analysis can be used. 1 The samples obtained in one of the test periods can be grouped into different *p*H ranges and the distribution within these ranges compared with that found in other test periods. For setting the limits of each range a logarithmic, *e.g.*, *p*H 1·1-1·5, 1·6-2·0, etc., or an arithmetic, *e.g.*, *p*H 1·0-1·1, 1·1-1·22, 1·22-1·4, 1·4-1·7, >1·7, scale can be used, each range in the latter corresponding to an approximate increment of 20 mEq./l. in hydrogen ion activity. 2 A mean value can be obtained by dividing the sum of a number of *p*H values by the number of samples. Such a mean, while convenient for certain statistical analyses (Mitchell, Hunt, and Grossman, 1962) does not represent the mean acidity because it takes no account of the logarithmic nature of the *p*H scale. The true mean acidity can be derived only from the reciprocal of the anti-logarithm of the *p*H values, the value obtained may then be converted back to the *p*H scale as the 'mean *p*H'. It is true that such a value gives weight to the most acid samples but this is inevitable when a mean is obtained from values with such a wide range, the highest value being perhaps ten thousand times greater than the lowest value. 3 The results may also be assessed by considering the diets in pairs and subtracting the values for samples at corresponding times in each patient. The mean difference for all the patients at a particular time may be calculated, or, from the mean difference for each patient over the day, an overall mean difference may be obtained. The significance of these differences may be assessed by Student's t test. As Mitchell *et al.* (1962) point out, each individual difference between a pair of values represents a division and the anti-logarithm of the difference is the ratio of the acidity of one sample to that of the other. However, the anti-logarithm of a mean difference obtained for several pairs of samples does not represent a ratio between the mean acidities of the samples because of the arithmetic manipulation of logarithms involved in the calculation.

RESULTS

The *p*H values of all the samples obtained[2] have been analysed as follows:

DISTRIBUTION OF SAMPLES IN DIFFERENT *p*H RANGES The results from all the patients for each of the test periods have been aggregated. Since a diet is likely to exert most effect during waking hours, the test

[1] J. C. Franklin and Co. Simplastic tube.

[2] Full details of the results for each patient have been published elsewhere (Lennard-Jones, 1964) or may be obtained from the authors.

TABLE II

DISTRIBUTION OF SAMPLES IN DIFFERENT *p*H RANGES USING A LOGARITHMIC SERIES OF *p*H RANGES

Diet	*p*H *Range*						*Total*	*Period*
	⩽1·5	1·6-2·0	2·1-2·5	2·6-3·0	3·1-3·5	>3·5		
Gastric II	138	65	26	20	9	23	281	24 hours
Convalescent	136	68	29	19	13	15	280	(8 a.m.-7a.m.)
Free-choice	116	79	26	9	16	28	274	
Gastric II	67	50	20	16	8	16	177	Day-time
Convalescent	65	49	23	17	11	11	176	(8 a.m.-10 p.m.)
Free-choice	49	58	21	7	14	23	172	
Gastric II	71	15	6	4	1	7	104	Night-time
Convalescent	71	19	6	2	2	4	104	(11 p.m.-7 a.m.)
Free-choice	67	21	5	2	2	5	102	

periods have been divided to show results obtained from 8 a.m. to 10 p.m. and from 11 p.m. to 7 a.m. In Table II the results are shown using a logarithmic series of *p*H ranges and in Table III the results obtained during waking hours are shown using an approximate arithmetic series of *p*H ranges.

TABLE III

DISTRIBUTION OF SAMPLES IN DIFFERENT *p*H RANGES DURING WAKING HOURS (8 a.m. TO 11 p.m.) USING AN APPROXIMATE ARITHMETIC SERIES OF *p*H RANGES

Diet	*p*H *Range*					*Total*
	⩽1·1	1·11-1·2	1·21-1·4	1·41-1·7	>1·7	
Gastric II	2	11	37	53	74	177
Convalescent	2	13	29	45	87	176
Free-choice	2	4	27	50	89	172

Both during the whole 24-hour period and during waking hours there were fewer samples of high acidity (*p*H ⩽ 1·5) and more samples of low acidity (*p*H > 3·5) when the free-choice diet was taken than when the 'therapeutic' diets were taken. A similar trend is shown in Table III.

MEAN ACIDITY The mean *p*H, determined from the arithmetic equivalents and converted back to the logarithmic scale, at each hour of the day for the three test periods is shown in Figure 1. The results for each test period are similar, with a fall in acidity after each meal followed by a rise to a maximum just before the next meal. Invididual differences at different times are such as might have occurred by chance.

DIFFERENCE BETWEEN CORRESPONDING *p*H VALUES The results for the three diets have been compared hour by hour in each patient, taking the diets in pairs. The *p*H of the sample for one diet at a particular time has been subtracted from the *p*H of the sample for the other diet of the pair at the same time. Having determined the mean difference over the whole day, during waking hours or at night, for each

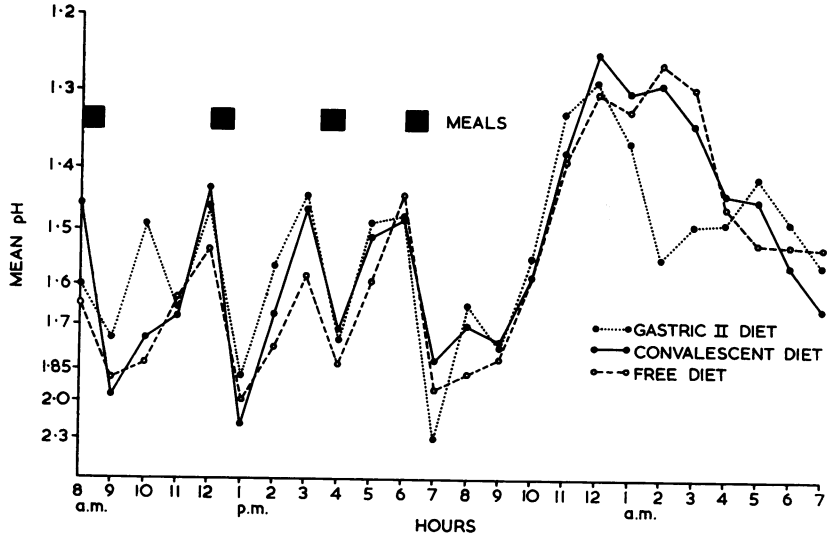

FIG. 1. *Mean acidity at different times of the day.*

patient, the mean differences for all the patients taken together has been calculated. The mean differences were small and not significant ($P > 0.05$). The largest mean difference, $+0.21 \pm 0.15$ *p*H units, was found between the 'free-choice' and the 'convalescent' diets confirming the trend towards lower acidity with the free-choice diet noted previously.

DISCUSSION

Therapeutic diets for peptic ulcer are hallowed by tradition and the concepts upon which they are based have deep roots in medical history. Celsus, in the first century A.D., was concerned that food should soothe and not irritate the stomach, and wrote, 'if the stomach is infested with an ulcer... Light and glutinous food must be used, but not to satiety. Everything acrid and acid is to be avoided'. Paulus Aeginata wrote in similar vein towards the end of the sixth century. The Arab School, around the tenth century and represented by Avicenna, emphasized mainly rest for the stomach, 'the very fact of taking nourishment is a tax on the digestive faculties, therefore the withdrawal of a certain amount of food means a corresponding alleviation for them.' William Hunter (1784), consulted about a boy wasting away with persistent abdominal pain and vomiting, reasoned that it was important 'to avoid offending a very weak stomach, either with the quantity or quality of what is taken down' and recommended frequent small quantities of milk. His patient recovered. During the last century, milk became the generally accepted mainstay of treatment for peptic ulcer. Sippy (1915), knowing the buffering effect of milk upon the acidity of the gastric contents, advocated repeated milk drinks as part of a programme designed to neutralize completely the gastric acid during waking hours. Hurst and Stewart (1929), whose modified Sippy diets have been widely used in England, inherited all these principles; 'The diet should produce as little secretion of acid as possible ... should render it innocuous by combining with it... should also contain nothing which can irritate the ulcerated mucous membrane', and they recommended milk, cream, and purées to achieve these aims. Many gastroenterologists today base their dietary advice on similar concepts; for example, Bockus (1963) aims to ensure a 'reduction in both the motor and secretory activity of the stomach' and considers that the 'ideal food for the fulfilment of the criteria for the dietary management of ulcer is milk'.

Dietary restriction, by its monotony, difficulty, and social handicap, is an imposition upon a patient, fully justified if it achieves its ends but unjustified if it is ineffective. The results of the present study do not support the hypothesis that the two diets studied reduce the acidity of the gastric contents as compared to a freely-chosen diet. Only one factor, acidity, has been studied and other factors, particularly those which influence gastric motility and emptying, such as the consistency and composition of the food, may be therapeutically important but have not been investigated. This study has also been limited to two diets, representative of one type, and it is possible that other types of diet may be effective in reducing acidity.

Several previous authors have shown that the acidity of the gastric contents tends to be lower when patients take food than when they take repeated drinks of a milk-cream mixture (Nicol, 1939; Kirsner and Palmer, 1940; Bingle and Lennard-Jones, 1960). These findings must be interpreted cautiously because there is a transitory fall in acidity after each drink which is not detected if gastric samples are withdrawn one hour later (Levy, 1950; Bingle and Lennard-Jones, 1960). Palmer (1933) showed that the Sippy regime does not always eliminate 'free acid' from the stomach. Wosika and Emery (1935) elaborated this work and demonstrated that the acidity was lower when patients took six feeds daily, with hourly milk and cream, than when they took milk-cream alone at the beginning of treatment, or three feeds daily later in treatment. Nicol (1939), in an excellent study, found similar results in that the mean acidity of gastric samples was greater when patients took an 'hourly' diet (5 oz. milk + 1 oz. cream, milk pudding, or vegetable purée) than when they took a 'two-hourly' diet (same regime + three main meals). He was not able to show any difference in mean acidity between the two-hourly diet and a 'light' diet (four meals + 5 oz. milk at night). He attributed the relatively lower acidity while patients took the two-hourly and light diets to their greater bulk and protein contents. The only study comparable to the present investigation is that of Lawrence (1952) who compared the mean gastric acidity of two groups of patients with peptic ulcer (site unspecified), one group taking a Lenhartz regime and the other a normal diet, all taking olive oil, antacids, and atropine. Little difference was found between the two groups but the validity of this finding is questionable as the comparison was not made in the same patients.

Three controlled clinical trials of diet in the treatment of duodenal ulcer have been performed. In one (Lawrence, 1952), performed among inpatients with peptic ulcer at an unspecified site, no difference in healing rate or duration of pain was found between patients taking a Lenhartz or a normal hospital diet. In the others, performed

among out-patients with duodenal ulcer, no difference was found over six months (Truelove, 1960), or one year (Doll, Friedlander, and Pygott, 1956), in the rate of healing or number of relapses between patients recommended a 'gastric diet' and others not recommended a dietary regime.

The usefulness of many therapeutic diets recommended for duodenal ulcer is questionable. It could be, however, that some dietary factor is important in aetiology or treatment. In subsequent papers, observations upon the effect on the acidity of the gastric contents of altering the frequency, composition, and consistency of meals will be reported.

SUMMARY

The acidity of the gastric contents in 12 patients with duodenal ulcer has been measured, using a sampling technique, while they took on three days two different 'therapeutic' diets and freely-chosen 'normal' meals. No difference was demonstrable between the diets, though there was a trend towards slightly lower acidity during waking hours with the 'free-choice' diet.

We thank the physicians under whose care these patients were admitted, particularly Dr. F. Avery-Jones, Dr. T. D. Kellock, and Dr. E. N. Rowlands, for permission to study their patients and for their encouragement. The study depended on the help so willingly given by the dieticians at University College Hospital and the Central Middlesex Hospital, notably Miss G. M. Cave and Miss J. Fletcher. We are very grateful to Miss P. B. Wilcox for her help with the *p*H measurements and to Mrs. I. M. Prentice for the diagram.

REFERENCES

Aeginata Paulus. (1844). *The Seven Books*, translated by F. Adams, vol. I, Book III, Chap. 37. Sydenham Society London.

Avicenna (1930). A Treatise on *the Canon of Medicine of Avicenna;* with a translation by O. C. Gruner. Luzac, London.

Barford, L. J. (1928). A statistical enquiry into the etiology, symptoms, signs and results of treatment in 166 cases of gastric and duodenal ulcer. *Guy's Hosp. Rep.*, **78**, 127-157.

Bingle, J. P., and Lennard-Jones, J. E. (1960). Some factors in the assessment of gastric anti-secretory drugs by a sampling technique. *Gut*, **1**, 337-344.

Bockus, H. L. (1963). *Gastroenterology*, 2nd ed., vol. I. Saunders, Philadelphia and London.

Celsus (1756). *Of Medicine.* Translated by J. Greive, Book IV, Chap. V. Wilson and Durham, London.

Doll, R., Friedlander, P., and Pygott, F. (1956). Dietetic treatment of peptic ulcer. *Lancet*, **1**, 5-9.

Hunter, W. (1784). The successful cure of a severe disorder of the stomach by milk taken in small quantities at once. *Med. Obs. Soc. Phys. Lond.*, **6**, 310-318.

Hurst, A. F., and Stewart, M. J. (1929). *Gastric and Duodenal Ulcer.* Oxford University Press, London.

James, A. H., and Pickering, G. W. (1949). The role of gastric acidity in the pathogenesis of peptic ulcer. *Clin. Sci.*, **8**, 181-210.

Kirsner, J. B., and Palmer, W. L. (1940). The effect of various antacids on the hydrogen-ion concentration of the gastric contents. *Amer. J. dig. Dis.*, **7**, 85-93.

Lawrence, J. S. (1952). Dietetic and other methods in the treatment of peptic ulcer. *Lancet*, **1**, 482-485.

Lennard-Jones, J. E. (1964). Food and gastric acidity in duodenal ulcer. M.D. Thesis Univ. of Cambridge.

Levy, J. S. (1950). Comparative buffering capacity of intact and predigested protein following hourly feedings. *Gastroenterology*, **16**, 370-376.

MacInnes, D. A. (1948). Criticism of a definition of pH. *Science*, **108**, 693.

Mitchell, R. D., Hunt, J. N., and Grossman, M. I. (1962). Inhibition of basal and postprandial gastric secretion by poldine and atropine in patients with peptic ulcer. *Gastroenterology*, **43**, 400-406.

Nicol, B. M. (1939). Control of gastric acidity in peptic ulcer. *Lancet* **2**, 881-884.

Palmer, W. L. (1933). Fundamental difficulties in the treatment of peptic ulcer. *J. Amer. med. Ass.*, **101**, 1604-1608.

Ronald, J. (1939). The value of therapeutic measures in duodenal ulceration. *Brit. med. J.*, **2**, 1033-1034.

Sippy, B. W. (1915). Gastric and duodenal ulcer: medical cure by an efficient removal of gastric juice corrosion. *J. Amer. med. Ass.*, **64**, 1625-1630.

Truelove, S. C. (1960). Stilboestrol, phenobarbitone, and diet in chronic duodenal ulcer. *Brit. med. J.*, **2**, 559-566.

Wosika, P. H., and Emery, E. S., Jr. (1935). The effectiveness of the Sippy regimen in neutralizing the gastric juice of patients if the amount of alkali is not varied. *Ann. intern. Med.*, **9**, 1070-1077.