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He found no evidence that gastric delay was an essential accompaniment of gastric ulceration. He also investigated the emptying time following the common gastric operations (Table II). Pyloroplasty alone did not shorten the emptying time. Crider and Thomas (1937) proved this by wedging open the pylorus in dogs. The addition of vagotomy to the standard operations (Table III) prolonged the emptying time often up to 10–12 hours. All these tests were done at more than one year after operation. *The slowest emptying post-operative stomachs are those in patients who have had vagotomy and pyloroplasty.* Table IV shows the degree to which vagotomy affects the emptying time. In 31 out of 35 cases of vagotomy and pyloroplasty the emptying time was over six hours; thus pyloroplasty, when added to vagotomy, is not a " drainage " procedure within the strict meaning of the term. *This operation which cures gastric ulcer prolongs the emptying time.* If stasis is a factor in the causation of gastric ulcer, it is unlikely that an operation which prolongs the emptying time could cause the ulcer to heal. A corresponding clinical observation was made by Salmon (1965). He found only 11 cases of classical gastric ulcer in a series of 104 patients with severe pyloric stenosis. This figure is much lower than one would expect if stasis is the primary aetiological factor.

Secretory pattern in gastric ulcer

I have been unable to find any evidence either in the literature or my own studies which suggests that hyperacidity occurs in lesser curve gastric ulcers. Indeed, the converse is true. Such observers as Pickering and James (1949), Watkinson (1951), Ball (1961), and Baron (1963) reported diminished acid secretion in this condition, often with a nocturnal phase when the gastric juice is alkaline. Welbourn and Burns (1965) have closely mimicked gastric retention by putting meat extract into the antrum. They failed to show any hypersecretion in the gastric ulcer stomach (Table V).

Size of the alkaline and acid areas of gastric mucosa

Our own studies (Capper *et al.*, 1966*b*) of gastric secretion have been rather more direct. We have determined the actual size of the acid and alkaline areas by monitoring the pH of the mucosa in more than 250 gastric operations and recorded the results on a standard chart (Figs. 1 and 2). The constant feature in gastric ulceration is a large alkaline area extending along the lesser curvature with a correspondingly reduced oxyntic cell mass. For hyperchlorhydria to occur a large oxyntic cell mass is essential. From an analysis of 190 cases we have shown that the mean alkaline area in a gastric ulcer is more than double that in a duodenal ulcer stomach. The reason for the low acid in a gastric ulcer is the small size of the oxyntic cell field. Up to the present time the evidence (Ferguson, 1950; Reed, 1962; Emas and Fyro, 1964) suggests that both the yield and the potency of the gastrin obtained from the antrum is much greater in D.U. than in G.U. Moreover, in the Zollinger-Ellison syn-

TABLE I

GASTRIC EMPTYING TIME IN PEPTIC ULCERATION
(K. Buckler)

	No. of cases	*Percentage of cases where stomach is completely empty at:* < 6 *hrs.*	> 6 *hrs.*
"Normals"	150	86%	14%
D.U.	183	68%	32%
G.U. (lesser curve) ..	58	62%	38%

TABLE II

POST-OPERATIVE GASTRIC EMPTYING TIME
(K. Buckler)

Operation	*No. of cases*	*Percentage of cases where stomach is completely empty at:* < 6 *hrs.*	> 6 *hrs.*
Billroth I	41	90%	10%
Polya	45	98%	2%
Gastro-enterostomy ..	9	90%	10%
Pyloroplasty alone ..	7	43%	57%

TABLE III

EFFECT OF VAGOTOMY ON GASTRIC EMPTYING TIME
(K. Buckler)

Operation: Vagotomy	*No. of cases*	*Emptying time:* < 6 *hrs.*	> 6 *hrs.*
+ Billroth I	27	26%	74%
+ Polya	22	54%	46%
+ Gastro-enterostomy ..	37	30%	70%
+ Pyloroplasty	35	11%	89%

TABLE IV

GASTRIC EMPTYING TIME WITH AND WITHOUT VAGOTOMY
(K. Buckler)

		Percentage empty at: < 6 *hrs.*	> 6 *hrs.*
Gastro-enterostomy	No Vagotomy	90%	10%
	+ Vagotomy	30%	70%
Pyloroplasty	No Vagotomy	43%	57%
	+ Vagotomy	11%	89%

TABLE V

GASTRIC SECRETION ON ANTRAL STIMULATION IN PEPTIC ULCERATION
(R. B. Welbourn and G. P. Burns)

Site of ulcer	*After meat extract HCl in mEq per hour ± standard deviation*
Duodenal	27.28 ± 14.9
Duodenal and gastric ..	26.9 ± 14.0
Gastric	5.24 ± 2.7

drome, where the gastrin-induced secretion is maximal, the ulcers are commonly in the duodenum or the jejunum and not on the lesser curvature.

In view of the gastric emptying time, the diminished secretory response and the reduced oxyntic cell field, the gastrin-hyperchlorhydria theory is untenable. There is no evidence of stasis nor of hypersecretion; indeed, the small oxyntic cell mass is incapable of producing excess acid.

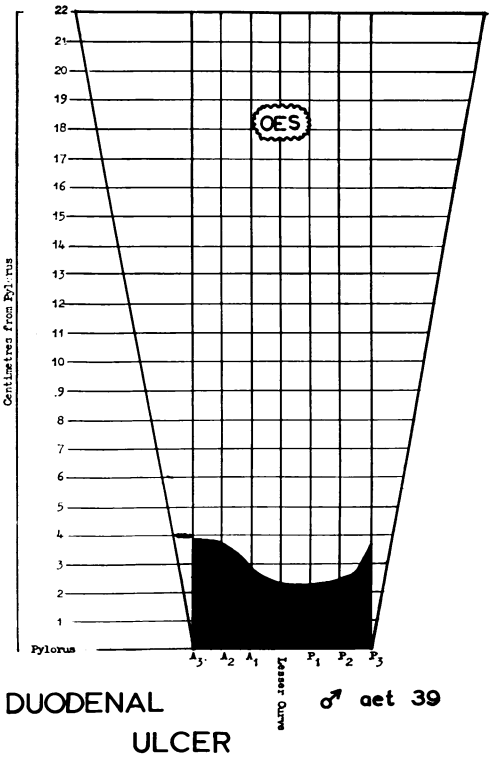


Fig. 1. Standard chart: gastric mucosal surface in sq. cm. recording pH changes for 3 cm. on either side of lesser curve. (Alkaline area black.)

Alkaline areas following surgery

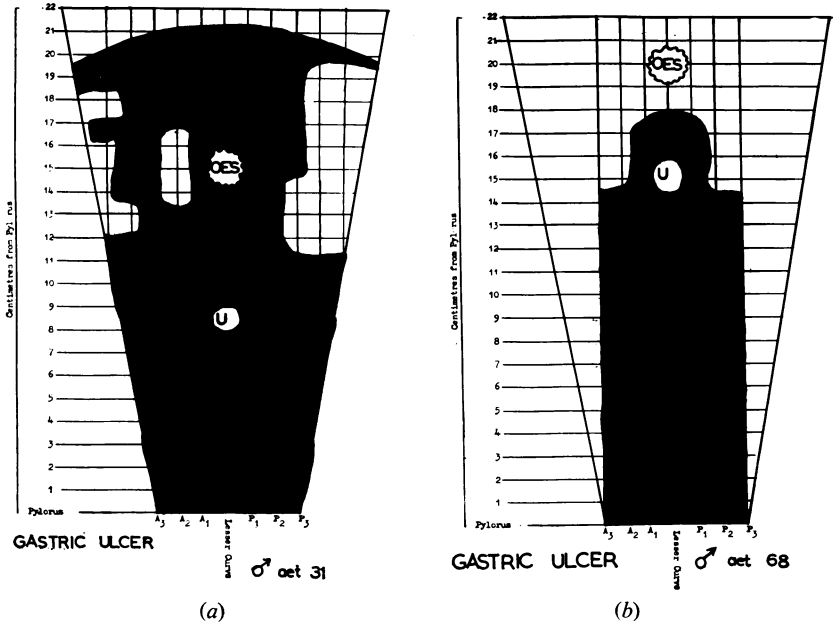
Consideration of these pH findings poses some critical questions, viz:

1. Why are the alkaline zones in gastric ulcer and duodenal ulcer so different, and, in particular, why are the alkaline areas in gastric ulcer so large?

2. What happens when the alkaline area is excised at gastrectomy? Does a new one form, or is the gastric remnant entirely without one?

The answer to these problems was elusive until we had opportunity

to monitor the changes in the gastric mucosa following various gastric operations. This opportunity arose when we had to re-operate on patients in whom the more severe complications, such as biliary vomiting, steatorrhoea, recurrent ulceration, and severe dumping, appeared following previous gastric surgery. It was found (Fig. 3) that alkaline areas appeared adjacent to the stoma in the Billroth I and Polya operations and also following gastro-enterostomy. Where a Roux-en-Y anastomosis had been carried out there was no new alkaline area (Capper *et al.*, 1966*a*). The gastric mucosa was acid right down to the junction with jejunal



(*a*) (*b*)

Fig. 2. (*a*) Chart of gastric ulcer. (*b*) Chart of gastric ulcer.

mucosa. Around the stoma, where duodenal regurgitation occurred, the oxyntic cells had disappeared. If no regurgitation occurred, the oxyntic cells remained. These findings are supported by the work of Lawson (1964). He kindly drew diagrams for us to record his findings (Fig. 4). He carried out gastric operations in dogs and found that, in the areas adjacent to the stoma, atrophic gastritis developed with the disappearance of the oxyntic cells. In addition, he found that the maximum change in the gastric mucosa was caused, not by bile or by pancreatic juice alone, but by both together. More recently we have been able to monitor a case where a gastric ulcer recurred following a Billroth I gastrectomy. The alkaline area extended to the site of the ulcer within 3 cm. of the cardia. We were able to show that there was a forceful regurgitation of duodenal juice well up into the stomach.

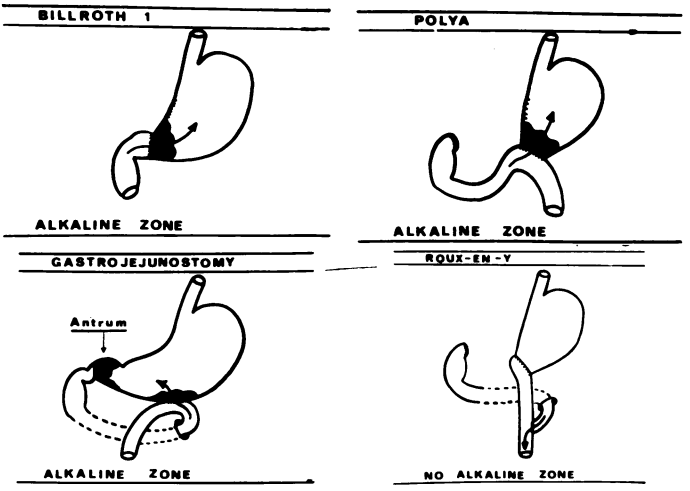


Fig. 3. Alkaline mucosal areas (black) following gastric operations. (pH monitoring.)

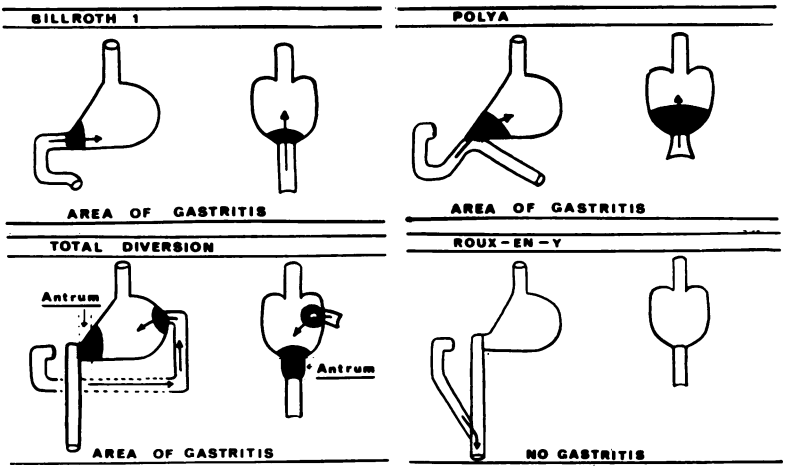


Fig. 4. Areas of gastritis recorded by Lawson (1964) after gastric operations (histological studies).

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denal cap. The effect was observed on an X-ray television screen. Not only did this test reveal the presence of pyloric competence or incompetence, but it also showed that there are marked variations of duodenal behaviour from time to time. Sometimes the duodenal activity, and especially the antiperistalsis, was rapid and forceful, and at other times it was slow and comparatively placid. It was variable in the same individual under different conditions of stress and rest. An almost constant finding, however, was that when the patient was supine, duodenal activity was greatly reduced and for a time completely absent.

The Pyloric Regurgitation Test (PRT) has been carried out in 113 cases. First of all, we examined 21 controls; these had no indigestion and were in hospital for hernia, migraine, carcinoma of colon (one case) or under out-

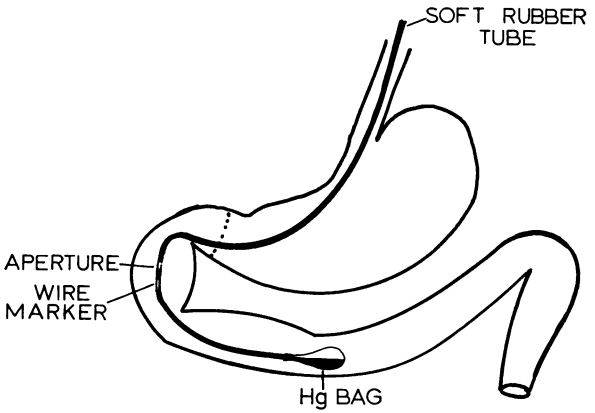

Fig. 5. Mercury-weighted tube for pyloric regurgitation test.

patient investigation for fibrositis. Pyloric regurgitation did not occur at all and we recorded duodenal peristalsis as being average in force and rate. In 41 cases with duodenal ulcer, the pylorus was completely competent in 18 but was incompetent in the remainder. In the early duodenal ulcers with classical symptoms of pain-food-relief, there was no regurgitation even when the duodenal cap was full. Peristalsis was normal or slow and, when the patient was supine, the rate of peristalsis became much reduced. We have examined a total of 29 cases with gastric ulceration. Twenty of these were active and had considerable pain after food; all of them showed pyloric incompetence and regurgitation. We have not seen an acutely painful gastric ulcer where forceful regurgitation was not present. In the remaining nine of the total there was no regurgitation. These had been under medical treatment; eight of them were pain free and the ulcer was presumably healing. The tendency in gastric ulcer was for duodenal peristalsis to be frequent and forceful and the pylorus incompetent, permitting the regurgitant flow to pass backwards into the stomach,

often directed between the gastric rugal folds and etching out the ulcer. In 22 non-peptic ulcer cases where there was gross incompetence and regurgitation, 11 had non-Addisonian achlorhydria, nine had oesophageal symptoms in the presence of a hiatus hernia, one had extensive atrophic gastritis without ulceration and one had a large ulcer cancer.

This test has revealed the presence of competence and incompetence of the pyloric valve and it has also shown considerable variations in duodenal behaviour especially in patients with gastric ulceration.

Bile in the gastric aspirate

Beaumont from his study of Alexis St. Martin (1833) stated: " Bile is never found in the gastric cavity in a state of health ". It appeared only, (1) under the influence of violent passion, (2) when the pyloric extremity was irritated by a tube or a thermometer pushed against it, or, (3) by pressure of the hand over the regions of the liver or duodenum. Sir William Osler (1902), in an address on William Beaumont, said, " It would be difficult to point to any observer who excelled him in devotion to truth and freedom from the trammels of theory or prejudice; where he lays down conclusions he does so with a degree of modesty and fairness of which few in his circumstances would have been capable ".

Beaumont's second statement may invalidate some of the observations of those who have reported the presence of bile in the gastric aspirate in cases of gastric ulcer. If the stomach tube is controlled radiologically, however, this criticism should not apply.

Watkinson (1951) found that bile staining was invariably present in cases of gastric ulcer where nocturnal neutralization occurred. Pickering and James (1949) showed that bile-stained gastric aspirate was more common in patients with gastric ulcer than in healthy people. Du Plessis (1965) studied fasting gastric aspirates for bile acid conjugates by paper chromatography and showed that there was an excessive amount of duodenal reflux in gastric ulcer although moderately high amounts occurred in a few of the duodenal ulcer cases. In none of the gastric ulcer cases was bile acid completely absent from the aspirate. Buckler (1965) in a very much larger series investigated both the night acid and the maximal histamine secretion for the presence of bile. Bile was completely absent in gastric ulcer cases in 3 per cent, in duodenal ulcers in 21 per cent, and in " normals " it was completely absent in 40 per cent. The bile concentration was recorded as being " high " in 10 per cent of duodenal ulcer and in 30 per cent of gastric ulcer cases. Any theory of the aetiology of gastric ulcer therefore must give due recognition to the increased amount of bile in the stomach in this condition.

Types of pyloric deformity

Pyloric incompetence, like mitral incompetence, is not uncommonly due to fibrosis of the valve. Burge (1964) has outlined very clearly the types

of pyloric deformity associated with gastric ulceration. They may be considered as:

1. *Deformity following duodenal ulceration*

Johnson (1957) has analysed this phenomenon very closely and has shown that the duodenal ulcer appears first, usually in Group " O " hypersecretors, and that the gastric ulcer when it occurs is often large and deep and tends to bleed profusely and undergo complications. In these hypersecretors there is often sufficient gastric acid to produce a deep and penetrating ulcer once the mucosa has been breached.

2. *The pyloric channel syndrome*

The deformity here is the result of submucosal fibrosis (Rhinds, 1959).

3. *Pyloric channel deformity due to carcinoma*

Malignant infiltration makes the pylorus rigid and incompetent. Not only may this result in speedy gastric emptying, but also in forceful regurgitation of duodenal contents into the stomach. In fact, some of the most forceful regurgitation we have seen has been through a malignant pylorus.

Many cases of gastric ulcer have no apparent pyloric deformity. It may be that increasing age, fatigue and asthenia (a not uncommon combination in gastric ulceration) are contributory factors in the production of incompetence in such cases.

Results of gastric surgery

In the last half-century many operations have been tried for gastric ulcer, but only a few have stood the test of time. The position has been reviewed recently by Tanner (1964) and by Farris and Smith (1963). In view of the observed importance of the pylorus, it would seem most satisfactory to divide the results of operations for gastric ulcer into two groups: (1) those that do not interfere with pyloric function, and (2) those that make the pylorus incompetent.

1. *Operations which do not interfere with pyloric function*

There are three main operations in this group:

(*a*) *Wedge excision* of the ulcer leads to a high incidence of recurrent ulcer at or near the original site. If it is accepted that a regurgitant stream from the duodenum is causative, the essential factor of pyloric deformity would persist after this operation and so an ulcer would tend to recur at the same site. When wedge excision of an ulcer is done at the same time as hiatus hernia repair, Butler (1965) reports that the ulcer recurs on the lesser curve at a lower site.

(*b*) *Sleeve resection* of the middle one-third of the stomach, together with the ulcer, is prone to relapse with a new ulcer on the lesser curve in roughly the same site and for the same reason as in wedge excision.

(*c*) *Gastro-enterostomy.* Tanner (1964) stated that this operation may or may not cure gastric ulcer. He said that most gastroscopists have seen quite a large persisting gastric ulcer on the lesser curve opposite a

gastro-enterostomy stoma. I have seen five such cases. Mangold (1958) found 12 patients who actually formed their gastric ulcer in the presence of a gastro-enterostomy which had been done previously for a duodenal ulcer. He added that there was no delay in emptying in these cases. Shipman (1965) recently reported a case where a gastric ulcer was treated by wedge excision, vagotomy and gastro-enterostomy. After six months another ulcer appeared at the site of the wedge excision. Gastro-enterostomy may well cure a prepyloric ulcer which, of course, is usually an " acid " ulcer with a hypersecreting stomach. The reason gastro-enterostomy does not necessarily cure a lesser curve gastric ulcer is that duodenal regurgitation is not necessarily abolished by this operation. Dragstedt *et al.* (1964) stated that to cure a gastric ulcer a gastro-enterostomy stoma must be close to the pylorus—an operation which presumably may affect pyloric function.

2. *Operations that make the pylorus incompetent*

Kelling (1918) and Madlener (1923) reported that a 5 cm. distal resection including the pylorus will result in the healing of a high benign gastric ulcer. Maurer (1951) strongly recommended such a procedure after his experience with 55 cases. Farris and Smith (1963) reported a high incidence of recurrence following wedge excision alone, but when pyloroplasty was added only one case out of 57 such operations had a recurrence. In addition, it may be said that pyloroplasty alone, a pylorectomy, an antrectomy, a Billroth I, and a Polya gastrectomy will usually cure a gastric ulcer. The only feature common to all these operations is abolition of the pyloric mechanism. Tanner (1964) has reported recurrences following Billroth I, and Holt and Lythgoe (1965) recurrences after pyloroplasty. These are rare, however, and in the two cases where we have found a recurrence following a Billroth I there was a narrowed stoma with powerful duodenal regurgitation as shown by the pyloric regurgitation test. I can find no record of a gastric ulcer recurring after a Polya gastrectomy. Presumably regurgitation, which occurs following this operation, is not the powerful stream such as may occasionally occur following the Billroth I procedure; in addition the pancreatic component is markedly reduced (Butler, 1961).

Post-vagotomy ulcer

Following vagotomy alone a number of surgeons reported that, although the duodenal ulcer healed, a gastric ulcer developed. Gastric stasis and antral stimulation were blamed for this. The probable mechanism, however, is that the duodenal ulcer heals as the result of the vagotomy, but this healing produces pyloric deformity which in turn leads to ideal conditions for the development of powerful duodenal regurgitation. On a similar basis the benefit of pyloroplasty when added to vagotomy is two-fold. It prevents (1) subsequent fibrotic pyloric stenosis and 24-hour gastric delay, and (2) pyloric deformity with ensuing forceful duodenal regurgitation.

CONCLUSIONS

The main conclusions to be drawn from the work may be summarized as follows:

1. There is no evidence that stasis is a factor in the causation of gastric ulcer.

2. There is no evidence that gastric hypersecretion accompanies gastric ulceration; in fact, there is positive evidence of a small parietal cell mass in this disease.

3. Active painful gastric ulcer is associated with pyloric dysfunction and abnormal duodenal behaviour.

4. The regurgitation of duodenal contents into the stomach causes atrophic gastritis, and the extent of this change is determined by the vigour and duration of the regurgitation.

5. These facts dispose of the stasis-gastrin hypersecretion theory; they indicate a change of emphasis from the stomach to the duodenum in gastric ulcer aetiology. Just as duodenal ulcer is the duodenal response to gastric secretion, so gastric ulcer is the gastric response to duodenal contents. The extent of duodenal regurgitation depends upon (1) the size of the pyloric aperture during the brief phase of duodenal systole, and (2) the vigour of duodenal activity. This concept is supported in particular by the results of gastric surgery. In addition it may be said that if the pylorus is completely incompetent following surgery, a gastric ulcer tends to heal.

DISCUSSION

The validity of these conclusions may be assessed by consideration of the following questions:

(a) Why does recumbency cause a gastric ulcer to heal?

When the patient is supine, duodenal peristalsis and antiperistalsis decrease in force and frequency, as is clearly shown on the pyloric regurgitation test. In this position, forceful duodenal regurgitation usually ceases.

(b) What happens when a gastric ulcer heals?

When a gastric ulcer heals the oxyntic carpet grows down again over the alkaline mucosa and covers the ulcer site. We have observed four such cases (Fig. 6). The mucosa covering the ulcer site had a pH of 4. This finding agrees with the observation of Watkinson (1951) that gastric acid increases when a gastric ulcer heals.

(c) How does pyloroplasty aid healing?

A wide incompetent pylorus does not permit a narrow forceful regurgitation. Barium studies after this procedure show a gentle ebb and flow of duodenal contents across the gastro-duodenal junction. Pyloroplasty decreases the pre-existing pressure gradients between stomach and duodenum.

(d) How can a regurgitant stream of duodenal contents pass around the lesser curve angle in a J-shaped stomach?

It must be remembered that the J-shaped stomach is a function of the amount and weight of barium in the stomach. It is apparent on P.R.T. that the regurgitant wave passes up chiefly along the magenstrasse and is guided by the avenue between adjacent rugae. It is obvious on the television screen that in some cases the stream is quite sharply curved or may be deflected as it enters the rugal avenue.

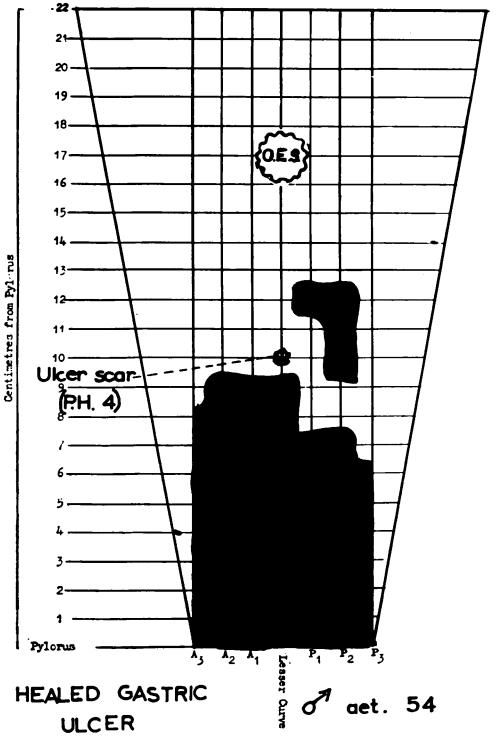

Fig. 6. Chart of healed gastric ulcer — ulcer site covered by acid mucosa.

As a final commentary, it may be worthwhile to contemplate the possible sequence of events in the natural history of gastric ulceration. It seems that the initial variation, possibly in response to psychological upset, is the appearance of abnormal duodenal activity manifest by both vigorous and frequent retro-peristaltic waves. When these occur in the presence of an incompetent pylorus, regurgitation of duodenal contents into the stomach appears. This regurgitation causes mucosal atrophy with disappearance of the thick layer of tenacious mucus covering and protecting the mucosa. The gastric ulcer subsequently appears in this zone of bare mucosa near its junction with the oxyntic cell mass. As soon as the

regurgitant alkaline wave recedes, acid juice will flow down again over this area, which is now ill-prepared to resist such an attack, and ulceration ensues. There must be some other factor which determines the actual location of the ulcer. The gastric peristaltic wave commences high up on the lesser curvature and it develops in magnitude as it descends. It is greatest in the middle third of the lesser curve—the commonest site for ulceration. It may be that the location of gastric ulceration is finally determined by the position of the descending peristaltic wall at the exact moment of duodenal regurgitation. An alternative view, one applicable to all sites of peptic ulceration, is that the junction of acid and non-acid secreting mucosa is subjected to waves of changing pH as secretions flow over it. The extremes of alkaline and acid pH levels occurring in the mucosal area under study in the stomach may well determine ulceration in functional zones. These suggestions are of course highly theoretical. Certainly anything which reduces the vigour of duodenal activity, such as bed rest, facilitates healing of the ulcer. Similarly, procedures which widen the pyloric aperture and thus alter the pattern of duodenal regurgitation also lead to resolution of the ulcer.

The association of duodenal regurgitation, atrophy of the mucosa, and gastric ulceration is further manifest by the fact that, as the healing process of gastric ulceration proceeds, the oxyntic cells regenerate and cover the former ulcer site. The position of the antro-fundic line depends on the degree of regurgitation and is not static.

This hypothesis really pushes the horizon of Truth further from us. Indeed, like the followers of King Canute, we are left wondering on the nature and ultimate control of the restless sea of duodenal activity!

ACKNOWLEDGEMENTS

This work was the outcome of the combined effort of a team and not an individual. My grateful thanks go to my friends and colleagues, Mr. T. J. Butler, Dr. G. Airth, Mr. K. G. Buckler, Mr. J. Kilby and finally to Mr. C. C. Hancock who, as Secretary of Southmead Hospital, supported the work in so many directions.

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B. R. KESBY, F.R.C.S.	Consultant Ophthalmologist, St. Margaret's Walsall, and Wolverhampton Groups of Hospitals.
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