THE AETIOLOGY OF BENIGN LESSER CURVE GASTRIC ULCER: VAGOTOMY AND PYLOROPLASTY IN ITS TREATMENT

Hunterian Lecture delivered at the Royal College of Surgeons of England
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by

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"AETIOLOGY", WROTE PAVLOV more than 60 years ago, "is the weakest branch of medicine"; yet only when the cause of a disease is known can we sensibly seek a cure. He went on: "We must regret that pathology has not yet taken its proper place as an experimental science—as pathological physiology."

Forty years later Lester Dragstedt, fulfilling Pavlov's dream, gave us the science of physio-pathology as applied to gastric disease, and from it, for the first time, an understanding of the aetiology of every form of peptic ulceration. How these two men would have enjoyed each other's company!

To foresee the future and to understand the present we must turn always to the past. Let us look, then, for a moment at "the past" of my subject to-day.

History

Kelling, in 1918, had already learnt that a low gastric resection, leaving in position a high juxta-cardiac benign gastric ulcer, cured the lesion (Fig. 1*a*). In 1923 Madlener confirmed his findings.

Since that time this procedure, the so-called Kelling-Madlener operation, has been widely used on the Continent and, in more recent years, both in America and in this country.

Before he died, Finsterer, giving the late results of this method, stressed not only the low mortality and morbidity of this distal gastric resection, but said, "The lasting results are as good with the Kelling-Madlener method as with resection taking the ulcer."

Surely we may with reason conclude that the cause of such benign gastric ulcers must lie in that part of the stomach excised by this low gastrectomy; it must lie either in the antrum or in the pyloric canal, or in both.

Madlener went further. He claimed that lesser curve gastric ulcers were cured if as little as 5 cm. of the prepyloric stomach was excised, together with the pylorus (Fig. 1*b*). Some years later, in 1934, Smoler reported that nothing more than excision of the pylorus was necessary (Fig. 1*c*).

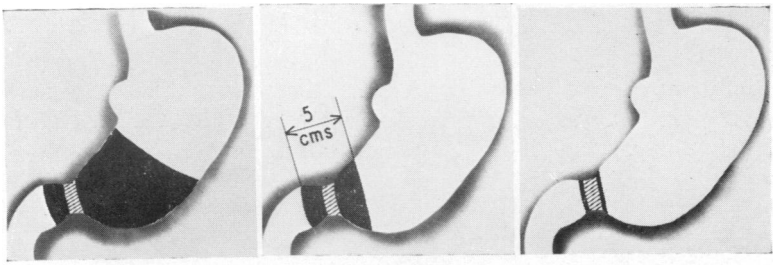
If this is true, cannot we too go further, and with reason claim that the cause of benign gastric ulcer lies at the pylorus?

I hope to show in this lecture that such simple reasoned deduction leads to the truth.

Gastric retention and gastric ulcer

For many years radiologists and clinicians have associated benign lesser curve gastric ulcer and gastric retention. Carman, in 1917, recorded two patients with duodenal ulceration and benign lesser curve ulcer. In both the stomach was one-third full of barium at six hours. In 1931, Emery and Munroe reported gastric retention in 50 per cent of cases of gastric ulcer, and Bull (1935) in 25 per cent. More recently Dragstedt (1959) has put this figure at 80 per cent.

The cause of this retention has been the subject of much speculation. Stone and Ruggles (1932) thought it was due to pylorospasm with pyloric muscle hypertrophy, while Bull (1935) believed it to be caused by inhibition of gastric peristalsis. Feldman (1946) postulated pylorospasm and Golden



(*a*) (*b*) (*c*)

Fig. 1. (*a*) Gastrectomy below (Kelling, 1918; Madlener, 1923). (*b*) The 5 cm. excision (Madlener, 1923). (*c*) Pylorus only (Smoler, 1934).

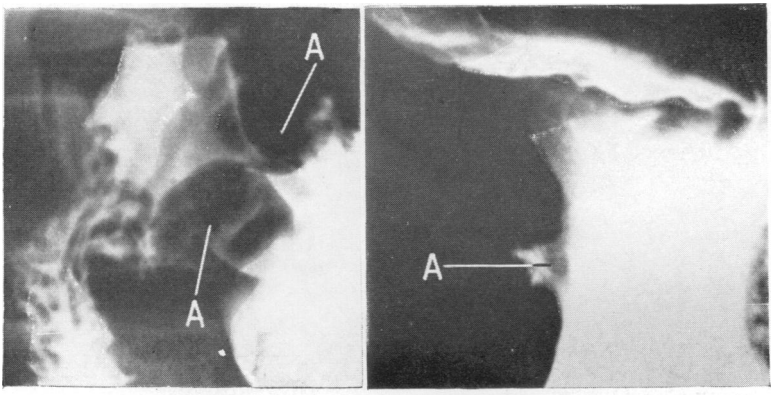
(1937) pyloric hypertrophy. Dragstedt (1959) thought that both the gastric retention and the low gastric acid levels found in patients with benign lesser curve gastric ulcer were explained by a state of underactivity of the vagus nerves with diminished motor and secretory function. Shanks *et al.* (1938) quoted Hurst as referring to gastric ulcer with normal pyloric function, with pyloric achalasia, with pylorospasm, with organic pyloric obstruction, and with failure of the pylorus to open.

We see from this extensive literature how many minds have been focussed on the pylorus in benign gastric ulcer.

For many years surgeons have recognized that *duodenal* ulceration with *duodenal* stenosis caused gastric retention in association with lesser curve gastric ulcer. In these circumstances the gastric ulcer has been regarded as secondary to the retention. These are often treated by

vagotomy and pyloroplasty in the belief that the pyloroplasty, by dividing the stenosed duodenum and by overcoming the gastric retention, would cure the gastric ulcer, while the added vagotomy would cure the duodenal ulcer. This is now an accepted procedure.

When vagotomy was first used by Dragstedt and others in the 1940s, gastric retention was often profound and prolonged. Typical chronic benign lesser curve gastric ulcers sometimes formed and were considered to be secondary to the retention. After total abdominal vagotomy without gastric drainage, not only is the tone lost in the denervated stomach, but there is also pyloric dysfunction, either from pylorospasm or from a



(*a*) (*b*)

Fig. 2. (*a*) A = the Finney hump. (*b*) A = lesser curve gastric ulcer.

failure to relax. This change at the pylorus may be demonstrated during operation by internal digital examination of the pyloric ring before and after nerve section.

Here again we find our thoughts turning to the pylorus in patients with benign ulcer and gastric retention, for this complication does not occur if a good pyloroplasty is made.

If, during vagotomy and pyloroplasty, the pyloroplasty stoma is made too small, then gastric retention may follow and a chronic lesser curve gastric ulcer develop. Such ulcers are cured when the retentioned stomach is drained by gastrojejunostomy.

Figure 2 (*a*) shows such a badly made pyloroplasty. The Finney procedure has been used on the superior as well as the inferior aspect of the pylorus. Two " Finney humps " project into the lumen of the pyloroplasty, which is narrowed. As a result there is gastric retention (Fig. 3*a*) and a lesser curve gastric ulcer (Fig. 2*b*). This ulcer was cured by gastrojejunostomy.

Usually, however, benign lesser curve gastric ulcer is not due to stenosis of the duodenum, neither does it follow vagotomy. It is thought of as a *primary* disease of obscure origin, to be treated surgically by gastric resection. We must question the primary nature of the lesion, and, too, the need for resection.

Perhaps the most astute observation was made by Huber and Huntington in 1948. These two radiologists from New York City wrote: " Gastric retention due to organic obstruction at or near the pylorus often precedes, and may have a causal relationship to the subsequent development of gastric ulcer of the lesser curvature."

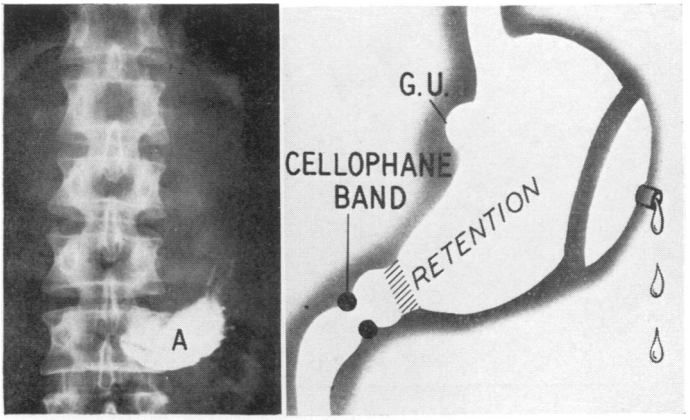


Fig. 3. (*a*) A = gastric retention. (*b*) Gastric retention (de la Rosa *et al.*, 1964).

Recently de la Rosa *et al.* (1964) have shown that gastric retention produced in dogs by " wrapping cellophane tape around the duodenum just distal to the pylorus " was sometimes followed by " chronic progressive gastric ulcer " (Fig. 3*b*). This finding had earlier been reported by Rigler *et al.* (1955). In 1901 Van Yzeren produced chronic gastric ulcers in rabbits after vagotomy, and these he found were cured by gastrojejunostomy. Linares *et al.* (1964) reproduced this work and found that gastroenterostomy or pyloroplasty protected against gastric ulceration.

Vagotomy and pyloroplasty done for duodenal ulceration, in the presence of a wide pyloroplasty may, from transient gastric retention following vagotomy, cause a transient gastric ulcer, which will heal as gastric tone returns with the passing weeks (Burge, 1964).

Gastric retention does not in itself cause gastric ulcer. Many patients with pyloric channel disease and gastric retention never develop a lesser curve gastric ulcer. Lesser curve ulcers form, apparently, only in people

who are genetically liable. Probably blood groups and blood group substances, as well, probably, as other unknown factors, play a part.

Retention in any organ usually spells distal organic obstruction. This reasoning led me to make an internal digital examination of the pyloric ring through a small prepyloric gastrotomy incision in patients with benign lesser curve gastric ulcer. A mucosal stenotic lesion of the ring was not infrequently found. I was led to study the history of the pyloric channel syndrome and to search for this condition in my own practice. Later I was to find that others, especially Rhind (1959), had found this lesion before me. His is an important paper.

The pyloric channel syndrome

Many papers* have been written on this subject since Butsch gave us his in 1935.

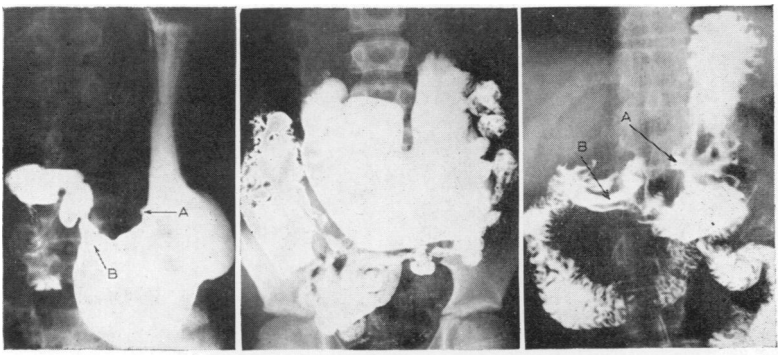


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Fig. 4. (*a*) A = lesser curve gastric ulcer; B = tapering antrum. (*b*) Gastric retention. (*c*) A = gastric ulcer; B = antral gastritis.

The clinical picture

All writers seem to agree that the clinical picture is typical of peptic ulceration in the distal stomach, with intermittent attacks of pain about one hour after food. Distressing vomiting is a common feature, due, apparently, to pylorospasm.

X-ray appearances

These have long been described by radiologists. They are:

1. Tapering of the pre-pyloric stomach (Fig. 4*a*).
2. Gastric retention (Fig. 4*b*).
3. An abnormal pre-pyloric gastric mucous membrane, commonly referred to radiologically as " antral gastritis " (Fig. 4*c*).

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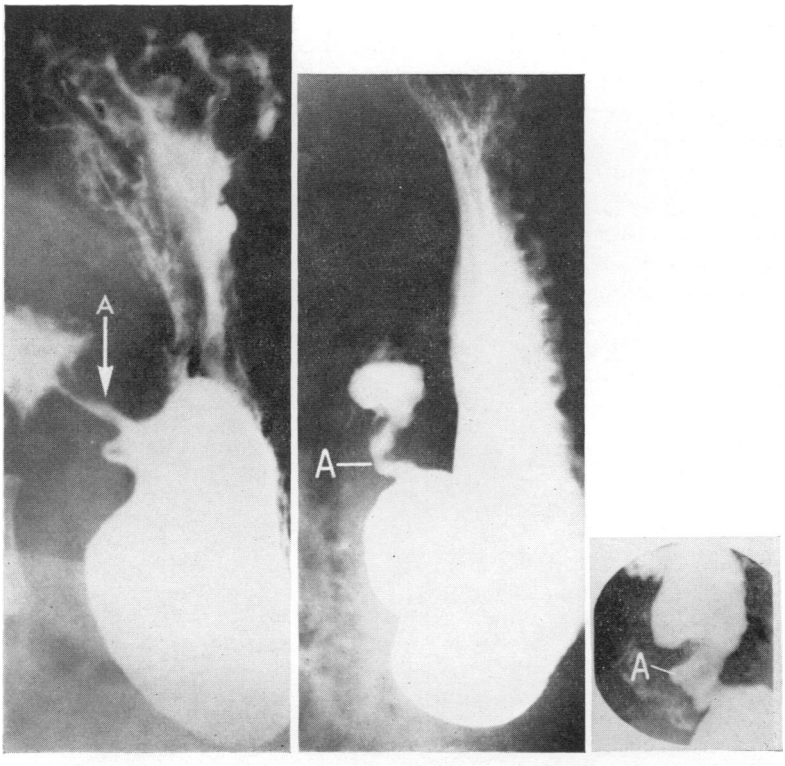

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Fig. 5. (*a*) A = apparent elongation and narrowing of the pyloric canal from spasm. (*b*) A = pyloric and pre-pyloric deformity. (*c*) A = pyloric deformity.

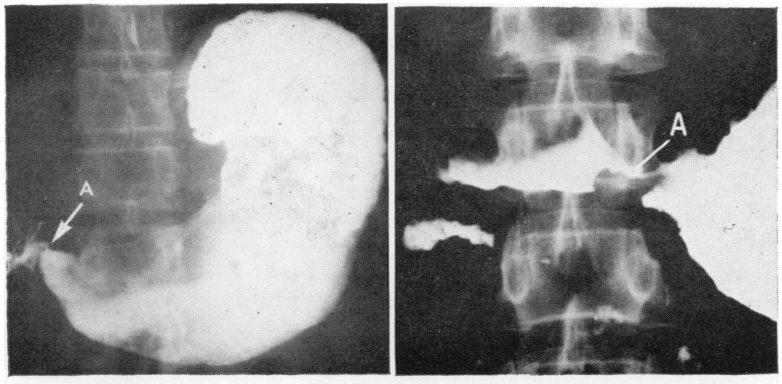

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Fig. 6. (*a*) A = narrow pyloric canal. (*b*) A = pyloric ulcer.

4. Elongation of the pyloric canal (Fig. 5*a*).
5. Persistent narrowing of the canal (Fig. 6*a*).
6. An ulcer crater on the canal (Fig. 6*b*).
7. Various deformities of the pyloric canal, including, often, the duodenal base (Fig. 5*b* and *c*).

The pathology

This disease has always been regarded primarily as a mucosal one, involving the pre-pyloric stomach and the pylorus, and perhaps the mucous membrane of the duodenum beyond. There is evidence of pre-pyloric gastritis histologically, there is muscle hypertrophy, both pyloric and pre-pyloric. This hypertrophy has commonly been held to be secondary to the underlying mucosal disease. Not only is there secondary pre-pyloric and pyloric spasm, but later in the disease fibrosis causes organic narrowing of the pyloric ring. Rossle (1935) believed that the muscle hypertrophy, both pyloric and pre-pyloric, was caused by continued spasm. He called it an " activity hypertrophy ".

Boas (1898), too, thought that the hypertrophy was a direct result of the mucosal disease, and used the term " stenosing gastritis ". Konjetzny (1936) believed the hypertrophy secondary to what he called " gastro-duodenitis ".

Usually no ulcer crater is found in the mucous membrane, but sometimes one is present. We are reminded here of peptic oesophagitis with its primary mucosal pathology and muscle hypertrophy. In this condition also there is seldom an ulcer crater.

The mucosal stenotic lesion at the pylorus

In early cases the pyloric lesion is, apparently, nothing more than pylorospasm during the attacks. Later, with fibrosis and hypertrophy, there is a definite histological picture. Golden (1937) noticed that the hypertrophy was often unilateral, and this has been so in the only two cases in which I have been able to obtain a complete section of the pylorus. In both these patients there was associated benign lesser curve gastric ulcer.

When a finger is introduced internally into the pylorus through a pre-pyloric gastrotomy incision (Fig. 7*a*), mucosal stenosis is often found. This appears to be due to sub-mucosal fibrosis. The muscle lumen, as Rhind showed, is not narrow, and the pylorus, on external examination, may be in every way normal. In some cases there is a suspicion of thickening while in others there is obvious change typical of what surgeons have for so long called idiopathic adult hypertrophic pyloric stenosis. Any attempt to estimate the size of the mucosal opening by taking the pylorus between finger and thumb may be misleading, for the muscle lumen may be felt and not the mucosal.

In 1932 Stone and Ruggles described a case in which " the tip of the little finger would not enter the pyloric canal ". Rhind (1959) dissected the mucous membrane from inside the muscle (Fig. 8*a* and *b*); he found

that the mucous membrane, when dissected out, was stenosed at the pylorus, whereas the surrounding muscle coat was in no way narrowed. Section of the muscle tube showed hypertrophy " to about twice the normal thickness ". He reported that looking back to early X-ray

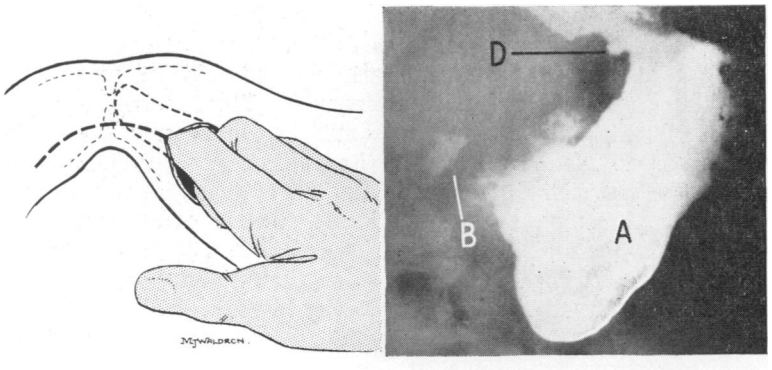

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Fig. 7. (*a*) Pyloric channel disease. (*b*) A = retentioned stomach; B = pylorus not filled; D = lesser curve gastric ulcer.

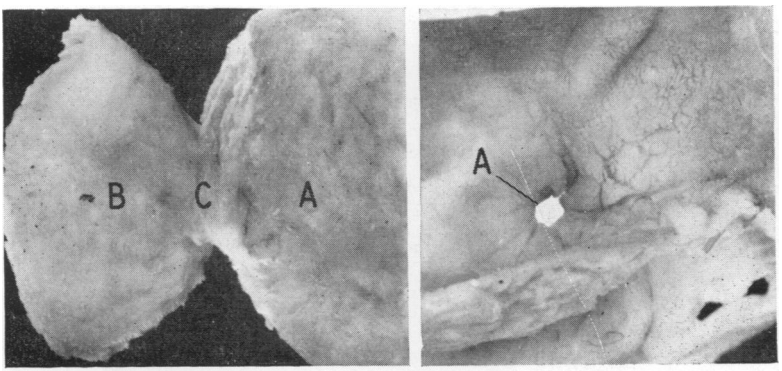

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Fig. 8. (*a*) A = stomach; B = duodenum; C = stenosed pylorus. (*b*) A = stenosed pyloric opening.

films in his case " it was immediately obvious that the mucosal stenosis seen in the specimen gave a perfect explanation of the abnormal appearance of the pyloric region ".

Figure 7 (*b*) is an X-ray of a patient with a chronic lesser curve gastric ulcer. The stomach is large and there is gastric retention. The pyloric

area does not easily fill. All the features of pyloric channel disease and its associated gastric ulcer are present. Figure 9 (*a*) shows the pylorus seen from the duodenal side in the gastrectomy specimen. The opening is small and rigid. Figure 9 (*b*) is a section across the pylorus showing the

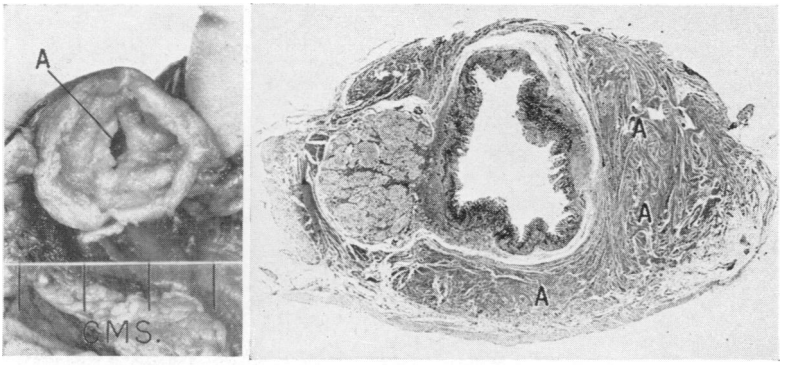

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Fig. 9. (*a*) A = small rigid pyloric opening. (*b*) A = unilateral muscle hypertrophy.

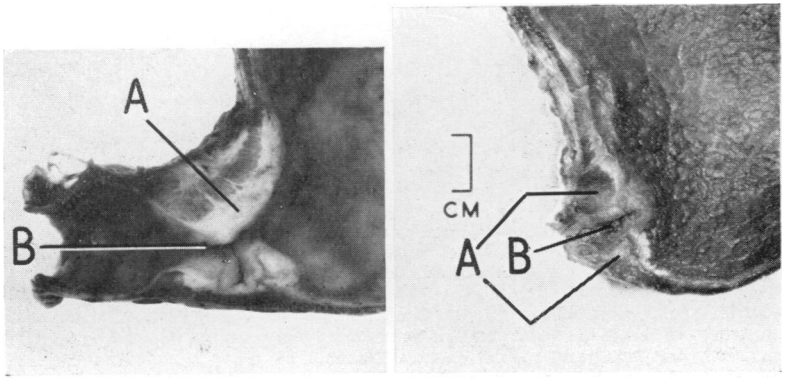

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Fig. 10. (*a*) A = asymmetrical hypertrophy and fibrosis; B = narrowed pyloric canal. (*b*) A = pyloric muscle hypertrophy; B = elongated stenosed pyloric canal.

asymmetrical hypertrophy at the pylorus. Figure 10 (*a*) and (*b*) are excellent examples of pyloric channel disease.

This is, I believe, the usual cause of gastric ulcer—this little lesion found long ago and then forgotten. Here Kelling, Madlener, Smoler, de la Rosa, Rigler, and Dragstedt all fall into line. Here the past meets the present to foretell the future. The surgical treatment of benign lesser curve gastric ulcer has been changed.

Gastric acid levels

In our work at the West London Hospital we have found a picture of hyposecretion of gastric acid in pyloric channel disease, and this has been the finding of other workers. Commonly there is no free hydrochloric acid in the basal juice and there is a low response to histamine.

Associated pyloric channel disease and lesser curve gastric ulcer

Foulk *et al.* (1957) wrote: " Seven of our 42 pyloric channel ulcers were associated with a second active benign gastric ulcer situated above the channel." These cases the authors excluded from their analysis.

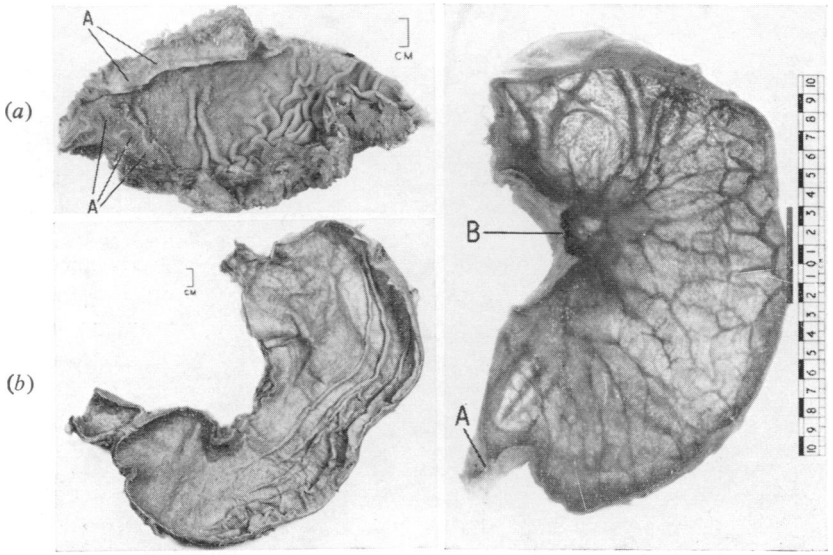

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Fig. 11. (*a*) A = pyloric and pre-pyloric hypertrophy. (*b*) Normal stomach to illustrate normal pyloric and pre-pyloric muscle. (*c*) A = pyloric and pre-pyloric muscle hypertrophy and pyloric canal stenosis; B = chronic lesser curve gastric ulcer in a retentioned stomach.

Our own studies have shown, we believe, the presence of duodenal or pyloric channel disease in almost every one of our cases of benign lesser curve gastric ulcer. Not uncommonly duodenal disease is present in the *base* of the cap, where it can easily involve and block the pyloric canal. It is difficult then to distinguish cases of pyloric channel disease from cases of duodenal ulceration; indeed, the two diseases are commonly associated. Figure 11 (*a*) is taken from a specimen in the museum of the Royal College of Surgeons of England. It shows the severe hypertrophy of pyloric channel disease, yet the record states that the duodenal ulcer which was present is not included in the specimen.

A study of museum specimens

Because of this association I have, with the help of the pathologists concerned, examined almost every specimen in the museums of the London teaching hospitals in which the pylorus is intact and a benign lesser curve ulcer is present. There is hardly one which does not clearly show evidence of pyloric channel disease or of duodenal ulceration. Since these two conditions are often seen without lesser curve ulcer, we must conclude that the gastric ulcer is secondary to the more distal lesion.

Figure 11 (*b*) shows the appearance of a normal pylorus and prepyloric muscle, and Figures 11 (*c*) to 14 (*c*) are some of the specimens taken from the museums.

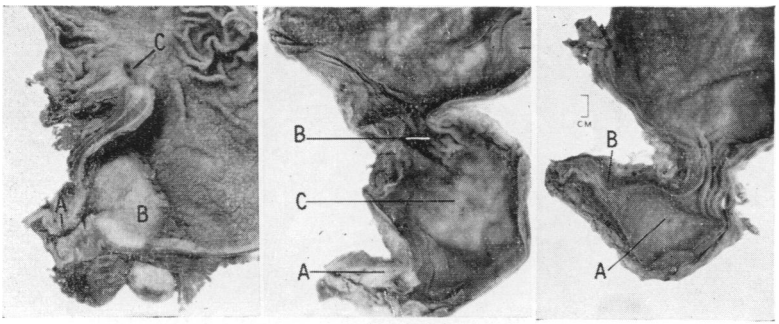

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Fig. 12. (*a*) A = benign pyloric channel disease with severe pyloric canal stenosis; B = pre-pyloric carcinoma; C = chronic lesser curve gastric ulcer. (*b*) A = pyloric channel disease; B = hour-glass deformity; C = distended, obstructed lower loculus. (*c*) A = distended lower loculus; B = pyloric hypertrophy.

The role of acid in gastric ulcer

It is generally agreed that gastric ulcer cannot occur in stomachs unable to secrete free hydrochloric acid. The disease does not occur in patients with pernicious anaemia. Ricketts *et al.* (1949) claimed that the role of hydrochloric acid in benign lesser curve ulcer was proved because these ulcers healed if achlorhydria developed and was maintained for a period of five months or longer. They stated that the ulcer does not recur during a period of achlorhydria.

In Dragstedt's paper of 1951 we read: " The effect of vagotomy on gastric ulcers is interesting in view of the fact that a hypersecretion of gastric juice is not present in these patients. Almost without exception the secretion in the empty stomach, and in response to the ingestion of food, is within the normal range or depressed. Nevertheless, 16 out of 26 patients with gastric ulcer (62 per cent) secured a good result from

vagotomy. While gastric secretion was not increased in these patients, nevertheless *complete vagotomy produced a significant reduction in secretion*, and it seems likely that this accounts for the beneficial results secured."

Gastrin and gastric ulcer

Dragstedt (1959) believes that 80 per cent of gastric ulcers are caused by an augmented hormonal phase of gastric acid secretion due to retention. He believes they are caused by gastrin. The evidence is strong.

1. The relationship between gastric ulcer and gastric retention is well established. Gastric retention augments the hormonal phase of gastric acid secretion by increasing gastrin release.

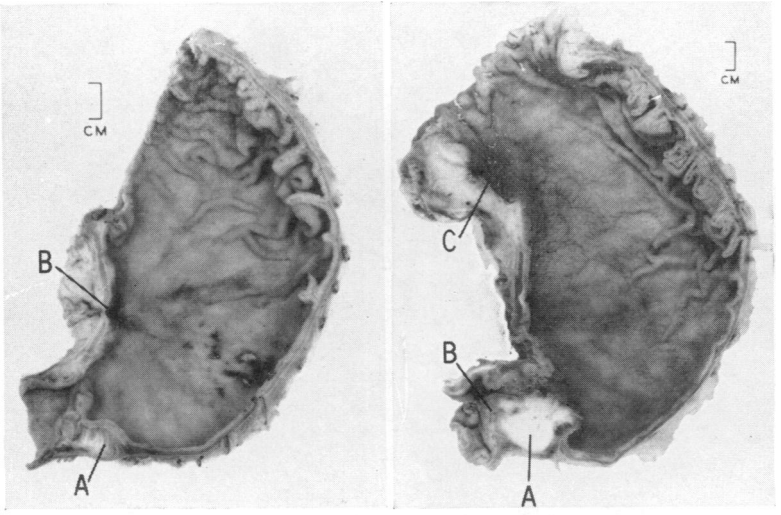

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Fig. 13. (*a*) A = asymmetrical pyloric and pre-pyloric hypertrophy; B = lesser curve gastric ulcer. (*b*) A = pyloric carcinoma; B = benign pyloric channel disease; C = lesser curve gastric ulcer.

2. It is claimed that the active principle of the pancreatic tumour in the Zollinger-Ellison syndrome is identical with gastrin. This substance gives rise not only to very high and persistent gastric levels but also to the most serious forms of duodenal, anastomotic, and gastric ulceration.

3. Pavlov's " round ulcer ". Pavlov recorded that a round ulcer developed in the Pavlov pouch of one of his dogs. This ulcer bled violently and then perforated. At the same time there was " a continuous and increasing hypersecretion " of acid. He was fascinated by this observation and commented that this increase of acid was not due to the " psychic and centrally excited flow " but to the " much weaker and

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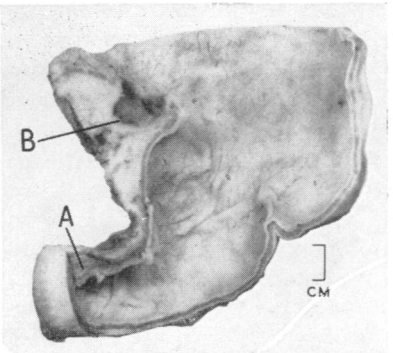

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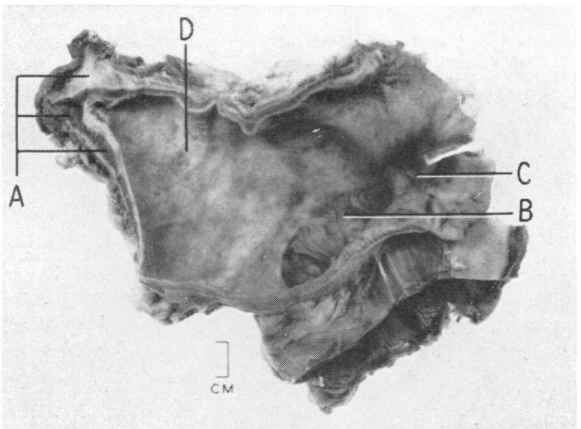

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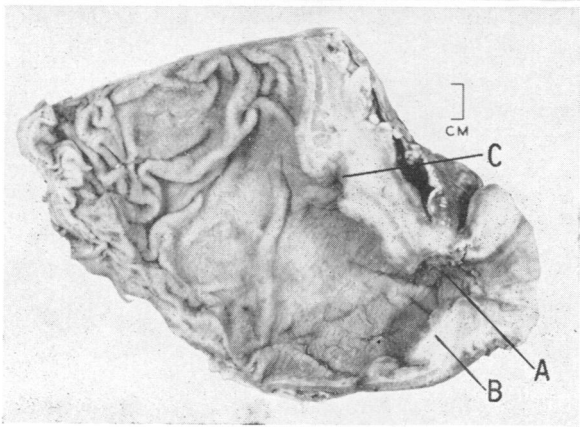

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Fig. 14. (*a*) A = unilateral hypertrophy; B = gastric ulcer. (*b*) A = stenosing pyloric channel disease; B = gastro-jejunostomy; C = benign gastric ulcer; D = retentioned antrum. (*c*) A = pyloric ring ulcer; B = long-standing pyloric fibrosis and hypertrophy; C = lesser curve gastric ulcer.

chemically excited secretion ". It seems that his round ulcer was associated with an augmented hormonal phase of acid secretion.

4. Gregory and Tracy (1964) have extracted from the antral mucosa of the hog's stomach two almost identical peptides. These they have named G1 and G2. These substances have a curious action in that they stimulate acid secretion in conscious dogs with denervated fundic pouches, but, if larger doses are given, the acid output is small and the juice is rich in pepsin. Gillespie and Grossman (1963) believe that gastrin is its own inhibitor. It has been suggested (*Lancet*, 1965) that this finding casts " some doubt on the ideas of Dragstedt *et al.* (1964) about the mechanism of stasis in the formation of gastric ulcers ". However, if gastrin and the active principle of the Zollinger-Ellison tumour are identical, then this objection to Dragstedt's hypothesis is difficult to understand, for in the Zollinger-Ellison syndrome the gastric acid levels are usually persistently high and peptic ulceration is relentless, severe, and serious. Gastrin has now been synthetized. The brilliant work of Gregory and his team has put us on the threshold of great things.

Vagotomy and pyloroplasty in the treatment of lesser curve gastric ulcer

If pyloric or duodenal stenosis, fibrotic and permanent, or spastic and transient, is the cause of lesser curve gastric ulcer, then nothing more than pyloroplasty, by draining the antrum, should cure it. There is little doubt that it does. Strauss (1924) performed excision of the lesser curve ulcer together with pyloroplasty in 21 patients. There were no recurrences eight years later. Walton (1934) used wedge resection and gastrojejunostomy, and reported a recurrence rate of 1.8 per cent.

These are the findings which we would expect with regard to the lesser curve gastric ulcer itself. Indeed, we have already seen that benign lesser curve gastric ulcer caused by gastric retention following vagotomy and a bad pyloroplasty may be cured by gastrojejunostomy.

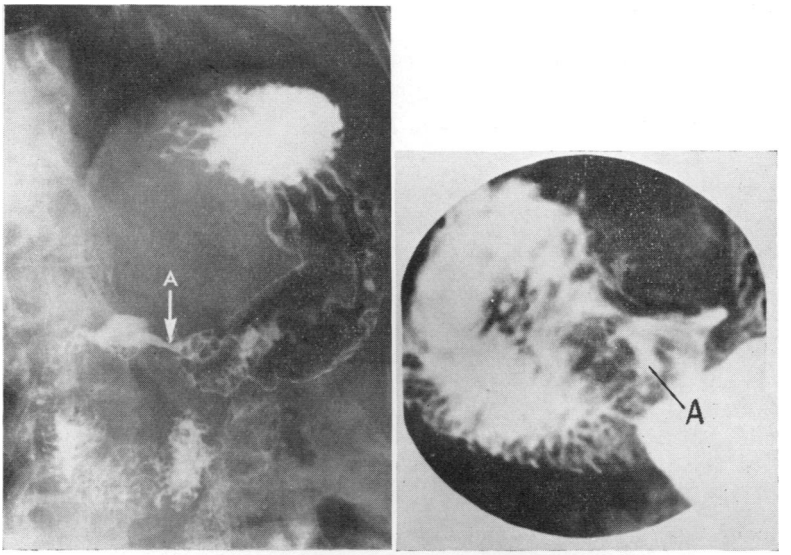
Our problem, then, is not whether gastric resection or vagotomy and pyloroplasty should be used in the treatment of benign lesser curve gastric ulcer; it is to choose between vagotomy and pyloroplasty and pyloroplasty without vagotomy. Perhaps one day a surgeon may again have the courage to perform pyloroplasty without vagotomy in a series of cases and follow them for an adequate time. There are, I believe, good reasons for adding vagotomy in every case.

Duodenal ulceration and pyloric channel disease, in spite of the differing gastric acid pictures, not infrequently co-exist; sometimes the pylorus is blocked by duodenal disease in the base of the cap, up against and involving the pyloric ring. Pyloroplasty alone would therefore sometimes leave basal duodenal disease untreated. Both duodenal ulceration and pyloric channel disease are cured by vagotomy and pyloroplasty. I cannot see that we dare use pyloroplasty alone. The following case illustrates this opinion:

Case. A Billroth I resection was done in a patient with recurrent lesser curve gastric ulcer. *There was no free acid in the resting juice and the response to histamine was low.* Following this resection there was no gastric retention, yet an ulcer formed on the stoma. Looking back at the pre-operative X-ray films there were obvious signs of pyloric channel disease (Fig. 15*a*). Vagotomy cured this anastomotic ulcer in spite of the low acid picture seen before the first operation.

In massive haemorrhage and in perforation

When vagotomy and pyloroplasty became established as a correct and proper procedure for duodenal ulceration, its application to both massive haemorrhage and to perforation naturally followed. In duodenal perforation, certainly if the disease is long established, it is not only an accepted operation but, I believe, the one of choice.



(*a*) (*b*)

Fig. 15. (*a*) A = abnormal pyloric canal. (*b*) A = basal duodenal ulcer crater.

In massive haemorrhage from duodenal ulcer the only criticism of vagotomy and pyloroplasty is that bleeding may continue or recur in the early post-operative days before the operation has had time to cure the lesion. Nevertheless, vagotomy and pyloroplasty, with under-running of the ulcer, is widely used.

Almost exactly the same situation is now present in gastric ulceration. I cannot here speak from experience. I believe that suture of the perforation together with vagotomy and simple drainage is the correct treatment. It will be necessary to establish the innocent nature of the ulcer by excision or by biopsy.

So too with massive haemorrhage. We must reconsider the surgical treatment. Bilateral selective vagotomy and pyloroplasty, with underrunning of the ulcer crater, seems attractive and in every way parallel to the position in duodenal ulceration.

There is, however, an important difference. Even when a good pyloroplasty with a wide stoma is made, there is often, temporarily, some degree of antral retention caused by gastric denervation. This retention may, at least in theory, so augment the hormonal phase of gastric acid secretion in these early post-operative days as to make worse the condition of the ulcer, leading, perhaps, to recurrent haemorrhage.

Probably, therefore, if vagotomy and drainage is used for massive haemorrhage from gastric ulceration, gastrojejunostomy rather than pyloroplasty should be chosen as the drainage operation. In this way post-operative atonic antral retention might be avoided.

Alternatively, the gastric antrum should be excised by hemigastrectomy, with or without vagotomy, for antrectomy alone—if our theories are true—must be sufficient to cure gastric ulceration. If this is done, a high-lying ulcer may be left in position (the Kelling-Madlener operation). A mid-gastric or low ulcer would be excised.

Only experience will set the pattern of the surgical treatment in these cases of massive haemorrhage, when we come in these present times, and in the future, to apply our newly gained knowledge to old problems.

The results

Let us look now at the results of our work at the West London Hospital when vagotomy and pyloroplasty is used for uncomplicated lesser curve gastric ulcer.

PATIENTS FOLLOWED UP

O ..... Symptom free.
H ..... Healed
UH ..... Unhealed
AH ..... Almost healed
} Barium meals.
UT ..... Untraced.
R ..... Gastric retention.

	Months				*Years*					*History*
	1	2	3	6	1	1½	2	2½	3	*in years*
1.	O	O	H	O	O	O	O	O	O	10
2.	O	AH	O	O	O	H	O	O	H	6
3.	H	O	O	O	O	O	O	O	H	7
4.	O	H	O	O	O	O	O	O	H	2
5.	O	O	UT	UT	UT	UT	UT	UT	UT	10
6.	O	O	O	O	H	O	O	H	O	5
7.	O	O	H	O	O	H	O	O		2
8.	H	O	O	H	O	H	O	H		10
9.	H	O	O	O	H	O	O	H		7
10.	O	O	O	O	O	O	O	H		10
11.	O	O	O	O	H	O	O	H		6
12.	O	O	O	H	O	O	O	H		2
13.	O	H	O	O	O	O	O	O		5
14.	O	O	O	O	O	O	O	H		4
15.	O	O	O	H	O	O	O	H		16
16.	H	UT	UT	UT	UT	UT	UT	UT		7
17.	H	H	O	O	O	H	O	H		3
18.	H	H	O	O	O	H	O	H		6
19.	O	AH	H	O	O	H	O	H		10
20.	O	O	O	H	O	O	O	H		3
21.	O	O	H	O	O	O	H			2
22.	O	H	O	O	O	O	H			2
23.	O	O	O	O	O	O	H			13
24.	O	H	O	O	O	O	H			3
25.	O	O	O	UT	UT	UT	UT			4
26.	O	H	O	O	O	O	H			16
27.	O	O	H	O	O	O	H			4
28.	O	O	H	O	O	UT	UT			3
29.	O	O	O	H	O	O	H			2
30.	O	O	H	O	O	O	UT			6
31.	O	O	H	O	O	O	H			15
32.	O	H	O	H	O	O	H			2
33.	O	H	O	O	O	O	H			2
34.	O	H	O	O	O	H				2
35.	O	O	O	H	O	H				1
36.	H	O	O	O	H	O				7
37.	O	H	O	O	O	H				5
38.	H	O	O	O	O	H				10
39.	UH	AH	O	H	O	H				4
40.	O	O	UH	H	O	H				4
41.	H	O	O	O	O	UH	R			4
42.	O	O	H	O	H					3
43.	O	H	O	O	H					6
44.	O	O	O	O	H					1
45.	O	O	H	O	H					6
46.	O	O	O	O	H					4
47.	H	O	H	O	H					12
48.	O	O	O	H	H					2
49.	H	O	O	O	H					7
50.	UT	UT	UT	UT	UT					22
51.	H	O	O	H	UT					2
52.	O	O	UH	O	H					6
53.	H	O	O	O	H					30
54.	O	O	O	O	H					5
55.	O	O	O	O	H					1
56.	O	H	O	H						8
57.	O	O	O	H						20
58.	O	H	O	H						6
59.	O	O	O	H						1
60.	O	UH	O	AH	R					2
61.	O	O	O	H						4
62.	O	O	O	H						50
63.	O	H	O							7
64.	O	O	H							6
65.	UH	UH	H							4
66.	O	H	O							3/12
67.	O	H	O							15
68.	O	O	H							5
69.	H	O	H							8
70.	O	O								3/52
71.	O	O								10
72.	O									

The problem of malignancy

When the subject of vagotomy and pyloroplasty in the treatment of benign lesser curve gastric ulcer has been discussed in surgical meetings, objection has not been raised on the grounds that it would fail to cure the lesion. Criticism of the procedure has always been that malignancy may be overlooked.

Sometimes primary gastric cancer masquerades as a simple ulcer. Sometimes malignant change takes place in a longstanding benign lesion.

Even at gastrotomy the surgeon may not be sure of the nature of the ulcer. Frozen section may fail to reveal a definite diagnosis. If the doubtful lesion is simple, nothing more than vagotomy and pyloroplasty is needed to cure it. If it is malignant, and operable, then nothing less than a radical gastric resection should be done. I believe there is only one proper way of handling this problem. The doubtful ulcer must either be removed by wedge excision, or its edge must be biopsied in one or more quadrant. If a frozen section leaves no doubt about the diagnosis, then the way is clear. If this technique is not used, or if the report from it is doubtful, then vagotomy and pyloroplasty is performed. If the ulcer is low on the lesser curve, biopsies are taken through the pyloroplasty incision. If it is high, a short gastrotomy incision is made at a higher level opposite the ulcer. Biopsy of a high lesser curve lesion is easily taken in this way.

Only, then, if the paraffin section proves malignancy, is a second operation undertaken, and a properly planned radical cancer procedure performed. *Never* must this be done for an ulcer thought to be malignant but proved finally to be benign. Especially is this so if the ulcer is high in the juxta-cardiac area.

DISCUSSION

Gastric ulcer—one cause or two?

When gastric retention can be demonstrated on X-ray examination, the aetiology of benign lesser curve gastric ulcer seems clear and vagotomy and pyloroplasty an eminently reasonable treatment. What, then, of those cases—and there are some—in which, at the time the barium examination is made, there is no evidence of gastric retention and no evidence of pyloric or duodenal disease?

The use of a food/barium mixture in place of the usual barium will apparently demonstrate retention in a greater number of patients. Oberhelman (1959), reporting the technique, wrote: " To date the majority of patients with benign gastric ulcer, without mechanical obstruction at the pylorus, have shown significantly lengthy emptying times ranging from 8 to 15 hours after ingestion of the test meal."

There is, however, I believe, a more important reason for the absence of X-ray evidence of gastric retention in patients with a lesser curve gastric

ulcer *at the time the examination is made.* Pyloric channel disease is, like duodenal ulceration, an episodic disease. In both, marked narrowing of the lumen may not give X-ray evidence of retention until a fresh attack, from oedema and from spasm, occludes more severely the already narrow site. We have seen that pyloric channel disease is an episodic lesion characterized by pain and vomiting. When there is vomiting there is also retention.

The pylorus is a very special site, for a small ulcer occurring on an otherwise normal and fully wide pylorus may cause retention and vomiting from severe pylorospasm. The following case is an important one, for it illustrates the apparent paradox of a gastric ulcer caused by, yet without, retention.

Case. This patient with dyspepsia vomited his food for five days; presumably he had gastric retention and an augmented hormonal phase of gastric acid secretion. On admission to hospital three weeks later with haematemesis an immediate barium meal showed a lesser curve gastric ulcer. The base of the duodenal cap was irregular (Fig. 15*c*). There was no gastric retention. Operation was later undertaken for continued haemorrhage. The pylorus and duodenum appeared normal. A finger introduced through a prepyloric gastrotomy incision showed no evidence of mucosal narrowing. The pyloric canal was fully wide. A lesser curve gastric ulcer, from which the patient was bleeding, was found and oversewn. When the pyloroplasty incision was completed, a small inactive looking ulcer was present on the pyloric ring. The patient remains well two years later.

Here we have, then, an example of a patient with no organic block and no retention *at the time of X-ray examination.* Had this tiny ulcer been healed at the time of operation, this patient would have been classified as a case with no evidence whatever of pyloro-duodenal disease.

It is this kind of patient which makes me think that there is only one cause of benign lesser curve gastric ulcer, and that gastric retention from a pyloric or duodenal lesion.

I like to think, with Dragstedt, that the circulation in this case was flooded with gastrin during the phase of retention three weeks before admission. This gastrin caused the ulcer. As the pyloric lesion healed the retention disappeared. Three weeks later the lesser curve ulcer was still present. This is not surprising. A wound may be slow in healing, although the injury causing was instantaneous. The injury in this case I believe was gastrin.

Is there a cause of lesser curve ulcer other than retention? Gastrojejunostomy proximal to a stenosed pylorus or duodenum may certainly cause lesser curve ulcer from antral retention. It is possible to imagine that gastrojejunostomy without pyloric or duodenal stenosis, and therefore without antral retention, might cause a lesser curve ulcer. The gastrin " tap " in the antrum is turned off by acid from the body and fundus entering the antrum and changing the pH of its mucosa. In a patient with a gastrojejunostomy and without pyloro-duodenal obstruction, in theory at least, the gastric acid could fail to enter the antrum to turn off the gastrin tap and, in effect, augment gastrin release. I have, I think,

seen two patients in which this hypothesis seemed a possibility. In both the gastric ulcer was cured by vagotomy and pyloroplasty after taking down the gastrojejunostomy.

Are there any other causes of benign lesser curve gastric ulcer of which we are as yet unaware?

Lesser curve gastric ulcer—one operation or two?

If there are two or more different causes of benign lesser curve gastric ulcer, we would not expect one operation to cure them all. Rather might we expect that we would have to differentiate one type of ulcer from another, and in some way choose the operation for each patient. We are reminded at once that some surgeons believe that duodenal ulceration has more than one cause, and that the operation used in any one case must be chosen, and that this choice depends on gastric acid studies. Fortunately, vagotomy and pyloroplasty seems to cure all patients with duodenal ulceration, and the so-called combined operation of vagotomy and antrectomy appears to be based on error.

So, too, with vagotomy and pyloroplasty for benign lesser curve ulcer. This operation seems to cure them all. Probably, then, there is only one cause; but if there is more than one we need not worry. We are surgeons and we aim to cure; others, rightly, will seek the reason.

CONCLUSION

In conclusion may I say that I believe lesser curve gastric ulcers are caused by gastric retention, persistent or transient, from pyloric or duodenal disease. Whatever the truth may be, it does seem that vagotomy and pyloroplasty cures them all, and gastric resection, in my opinion, no longer has a place.

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