PEPTIC ULCER SURGERY - AN OBITUARY (PART TWO)

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The demise of gastrectomy

The debate in the immediate post-war decade was less about which surgical operation to carry out on peptic ulcer and more about the extent of the gastric resection. By the early 1950s the American Gastroenterological Association Committee on Peptic Ulcer had documented the mortality and recurrence rates for 2,441 patients having gastrectomy for duodenal ulcer and followed up for a mean of 2.5 years. The operative mortality averaged around 2% and recurrence rate about the same. Despite these impressive results, doubts grew about the operation. Gordon-Taylor[36] in the UK as early as 1929, and later Inglefinger[37] in the US had alerted the surgical community to the possibility of anaemia and weight loss in the long-term but both remained enthusiastic gastrectomists.

The post-war years also saw the rise of the formal 'follow-up clinic' which for the first time was able to achieve a comprehensive review of patients for several years after the operation with little loss to follow-up. Subtotal resection - as advocated for example by Visick - could reduce recurrence to negligible levels, but only at the expense of frequent 'dumping', 'small stomach' syndrome, and more severe nutritional and metabolic sequelae.

Anaemia after gastric resection was first described by Moynihan (Figure 9) who suggested that the cause was lack of acid; Faber was the first to suggest that there was in addition a macrocytic component. Fischer[38] found that 7% (of 766) patients were anaemic 22-30 years after resection but in none of these was the anaemia megaloblastic. Even with the prolonged follow-up possible in the studies published in the 1980s, the incidence of post-gastrectomy anaemia varied from 5% to 40%.

Vitamin B_{12} deficiency varied from 10% to 50% in the published literature. Some large series, however, have shown no vitamin B_{12} deficiency on long-term follow-up. Metabolic bone disease following gastrectomy was documented by Everson in 1952.[39] Because of the need, until recently, to establish the diagnosis by bone biopsy, studies have almost certainly included senile osteoporosis as many of the post-gastrectomy patients studied had reached the age group in which this is common. The incidence has consequently varied in the reported literature from 0.4% to 43%.

Weight loss after Polya gastrectomy was documented by Muir,[40] and by Wells and Welbourne[41] who stressed the importance of its relationship to pre-operative weight. Wallensteen[42] reviewed 17 published studies and found the incidence of significant weight loss after gastrectomy to vary between 10% and 75%. Ivy and Raunch both found that post-operative weight loss was related to the extent of the gastric resection. McLean and Johnston suggested that the main cause of post-gastrectomy weight loss was reduced intake as a result of reduced gastric capacity, the now generally accepted theory.

As more and more reports were published showing the

FIGURE 9
Berkeley Moynihan - later Lord Moynihan of Leeds (1865-1936). Along with his mentor Mayo-Robson he was influential in establishing gastro-enterostomy in the treatment of duodenal ulcer.

long-term nutritional and metabolic sequelae which could follow gastric resection, so vagotomy and drainage increasingly found favour. Stammers and Alexander-Williams, in an authoritative monograph[43] published in the early 1960s, recorded the results in their series of 75% gastrectomy with a Billroth II anastomosis. Despite a low operative mortality of 1.4% and a recurrent ulcer rate of only 2%, they concluded that the operation should be abandoned because of long-term metabolic sequelae.

In what was to be the last major controlled randomised trial comparing subtotal gastrectomy against vagotomy + gastro-enterostomy and against vagotomy + antrectomy, the Leeds / York trial[44] showed an advantage for gastrectomy in terms of Visick grading (a simple scoring system to assess the symptomatic result after peptic ulcer surgery), recurrent ulceration, and no difference in operative mortality or 'post-gastrectomy syndromes'. Yet despite this conclusion, the authors were unable to recommend gastrectomy because long-term sequelae[45] compared unfavourably with vagotomy in studies like Cox's[45] and because the results of

gastrectomy deteriorated with time as reported in the Leeds' follow-up clinic.[46]

GASTRO-ENTEROSTOMY
Gastro-enterostomy (figure 11) was first performed in September 1881 by Wölfer (Figure 10), Billroth's first assistant, during the latter's temporary absence from Vienna.[47] According to Moynihan's later account,[9] Wölfler was about to close the abdomen when his assistant Nicolandoni suggested that a bypass be attempted. An ante-colic gastro-jejunal anastomosis was fashioned with a long anterior loop. The patient lived for four months. When Billroth himself later attempted the procedure the patient died after a few days from what sounds like a stomal obstruction. The result of this early disaster was that the procedure fell into disrepute in Billroth's clinic and was developed elsewhere.

Courvoisier, Professor of Surgery at Basle, introduced the first development. As a result of a series of cadaver studies, he concluded that taking the jejunum through the transverse mesocolon would be less likely to produce obstruction. In 1883, Courvoisier's pride in the success of this, the first retrocolic gastro-enterostomy, clouded his clinical judgement. He took the patient by carriage to a meeting of the Swiss Medical Association on the fifth post-operative day. Although she apparently survived this ordeal, she died five days later.[48] The first successful results from a series of posterior gastro-enterostomy were reported by von Hacker in 1885. According to the historical review

FIGURE 11
Gastro-enterostomy.

by Olch and Harkins,[49] gastro-enterostomy for peptic ulcer was probably first performed in 1894 by Doyen, whose light occlusion bowel clamps remain in general use today.

From its earliest days the procedure was complicated by the problem which Billroth had encountered at his first attempt at the operation - afferent loop obstruction. A variety of modifications were tried in attempts to overcome this. In the earliest days the mistaken concept that partially obstructing the efferent loop would produce equal resistance to gastric outflow via the stoma resulted in several unsuccessful variations.

A physiological rather than mechanistic approach was adopted by Rockwitz[50] who described the first iso-peristaltic anastomosis in 1887. Mayo confirmed the benefits of the short loop but considered that there was no advantage in the iso-peristaltic technique. The rationale for the operation when performed for duodenal ulcer was that the gastro-enterostomy would divert food from the duodenum and thereby 'rest' the duodenum. The retrocolic 'no-loop' operation rapidly gained in popularity and, with increasing experience, mortality rates fell.

The operation was championed by two hugely influential Leeds' surgeons, Mayo-Robson and Moynihan, whose advocacy of this as a safe, effective operation was responsible for its widespread adoption in Britain as the procedure of choice in the treatment of duodenal ulcer from the turn of the century - a position which did not alter until the 1930s. The influence of W.J. Mayo (Figure 12), who had learned the technique at Mikulicz clinic, ensured the predominance of gastro-enterostomy as the mainstay of duodenal ulcer treatment in the US until the 1930s.

The decline of gastro-enterostomy
The complication which was to bring about the demise of gastro-enterostomy, namely stomal ulceration, had been noted independently by Braun and Hahn as early as 1899. The problem had been reported in the early years of the century in the English language literature by authoritative figures like Mayo[51] (co-founder of the clinic that bears his name), and Wilkie.[52] It was generally accepted that the advantage of much lower mortality more than compensated for this complication.

The problem of jejunal ulceration was recognised increasingly throughout the 1920s, and prompted Pribram in 1923 to suggest that 'gastro-enterostomy is a disease and

FIGURE 10
Anton Wölfler (1850-1917) was assistant to Billroth in Vienna when he performed the first gastro-enterostomy. He later became Professor of Surgery in Prague.
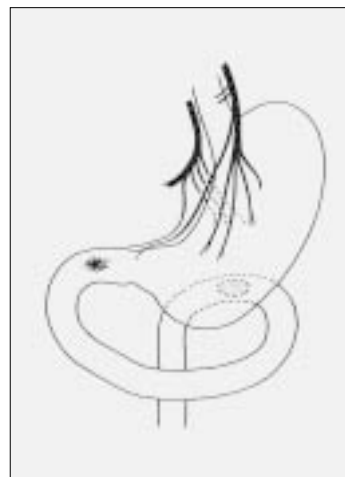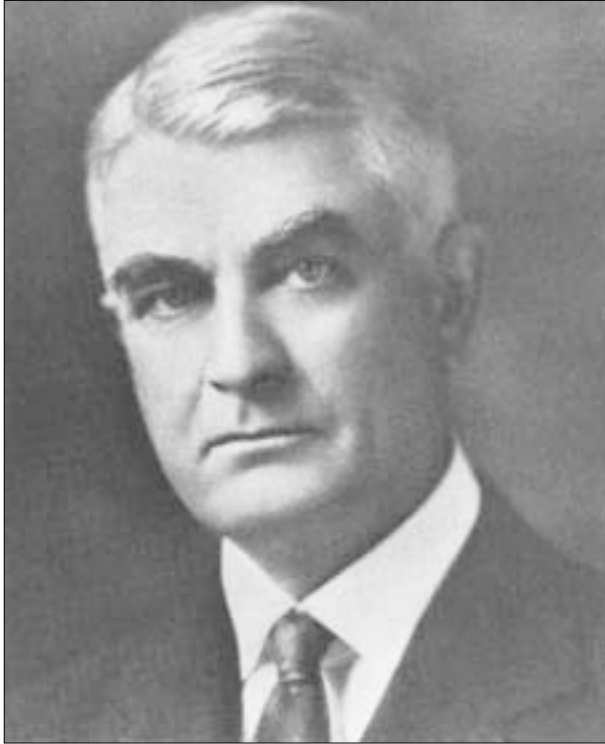
FIGURE 12
William J. Mayo (1861-1939) along with his father William W. and his brother Charles made the Mayo Clinic a leading world centre.

not an operation'. It was Lewisohn who in 1925 published the series which was to move American surgeons away from gastro-enterostomy as the standard treatment for duodenal ulcer.[33] Lewisohn had worked for two years as assistant to Czerney where he had learned not only surgical techniques but the value of careful audit. His report of a stomal ulceration rate of 25% proved a major influence on American surgeons. At the Mayo clinic the procedure declined year on year from a peak in 1936. By the late 1930s gastro-enterostomy had been supplanted in the US by gastrectomy as the favoured operation. Wangensteen in 1942 recorded its 'obituary as the standard operation for duodenal ulcer'.

Similarly in Britain gastro-enterostomy as sole treatment for duodenal ulcer was being performed less often in the middle years of the century, despite Moynihan's continued advocacy of the procedure into the 1930s. Sherren, in 1925, went so far as to say that gastro-enterostomy had done 'more than any other operation for the good of the human race'[53] - a view dismissed as hyperbole within a few years.

Heanage Ogilvie's opinion was to prove influential:[54] he calculated that the true incidence of recurrent ulcer after gastro-enterostomy was 20%. On the basis of this, and the low mortality in the published results of the time, he advocated gastrectomy which had become the favoured procedure in continental Europe. Ogilvie's views were enhanced by the esteem in which he was held in the UK and by his position of seniority in the British Army during the Second World War – in which he rose to the rank of major-general. In 1956 he scathingly condemned gastro-enterostomy by writing that: 'For the unskilled surgeon on the unfit patient it has much to recommend it.'

Wells[35] records that after 1945 gastro-enterostomy was rapidly supplanted in the UK by partial gastrectomy because

the long-term results were unsatisfactory in 25% of cases. The era of more complete and long-term gastric follow-up had dawned. Clark,[55] from Illingworth's unit, was able to report a follow-up rate of 77% at 20 years. At 10 years, 43% of patients had recurrent symptoms described as severe in 23%. Tanner reported a 50% recurrence rate on long-term review. Other procedures had replaced it in all but a handful of centres by the late 1950s.

Last outposts of gastro-enterostomy
The continuing use was recorded in two units (both in Edinburgh) into the 1960s, and in one of these into the 1970s. Farquharson[56] expressed misgivings about the widespread use of gastrectomy with its attendant sequelae: he recorded 5,170 cases followed up for 4-10 years with a recurrent ulcer rate of 3.5%. In the Western General Hospital, Edinburgh, the selective policy in which gastro-enterostomy was reserved for patients with low acid output (maximal acid output less than 50) was proposed by Bruce and colleagues.[57] This followed their observation that jejunal ulcer was more frequent in high acid secretors. In one of the last published accounts of the results of gastro-enterostomy for duodenal ulcer, Small and Smith[58] recorded 139 patients with pre-operative MAO of less than 30 mEq/hr who were followed up for 4-18 years. The recurrent ulcer rate was 2.2%. Results like this, however, were too late to save gastro-enterostomy as an accepted operation for duodenal ulcer.

THE SCIENTIFIC BASIS OF LOWERING GASTRIC SECRETION
The earliest scientific observations on the role of the vagus in gastric secretion were made by Sir Benjamin Brodie.[59] In a series of experiments published in 1814, he demonstrated that copious volumes of watery fluid were produced in the stomachs of dogs by an intravenous injection of arsenic but not in dogs whose vagal nerves were pre-sectioned. Beaumont,[60] among his observations of the gastric fistula of Alexis St. Martin, noted alterations in the volume of gastric secretion in times of emotion. Heidenheim developed an isolated denervated antral pouch in the dog which was to prove the experimental model for many of the subsequent studies of the controls of gastric secretion.

The contributions of Pavlov and his school to our understanding of secretory controls were immense. Jurgens[61] was able to demonstrate in Pavlov's laboratory that subdiaphragmatic vagal section in dogs could abolish acid from the fasting gastric juice. Pavlov reported a now famous series of experiments demonstrating a cephalic or psychic phase of gastric secretion mediated through the vagi. The side-effects of vagal section were also described about this time: Cannon[62] showed that in cats vagotomy delayed gastric emptying, while Ungar[63] found gastric dilatation and stasis in vagotomised dogs.

Thus, by the turn of the century, the physiological basis for vagal section as a means of reducing gastric acid secretion was well established. Measurement of gastric acid secretion had become common in clinical practice. In the absence of investigative tools, a syndrome called 'hypersecretion' was described. It was Moynihan[21] who clarified the true nature of this 'syndrome'. The features were those of duodenal ulcer. He wrote: 'Hypersecretion is not a disease in itself. Hypersecretion is duodenal ulcer.'

TRUNCAL VAGOTOMY

Jaboulay in 1899 was probably the first to perform vagotomy in man (according to McCrea),[64] in an attempt to treat the abdominal pains of tabes dorsalis. Exner[65] performed a subdiaphragmatic truncal vagotomy to try to control the vomiting in tabetic patients with 'gastric crisis'. In 1914 Exner and Schwarzmann[66] reported 20 cases of gastric crisis treated by vagotomy, with success in half. Exner also gave this first description in man of gastric stasis following the procedure, and advocated a gastrostomy to overcome this complication. By 1920 Bircher,[67] a Swiss surgeon, was able to report the results of vagotomy on 20 patients whose symptoms suggested a diagnosis of gastric ulcer. The symptoms were reportedly abolished but at operation the diagnosis of ulcer was confirmed in only one instance.

Latarjet's writings in 1922 had to wait some 40 years before being afforded the credit they deserved.[68] After cadaveric dissection, this Lyon surgeon described in detail his technique of vagotomy in which the anterior trunk was divided below its hepatic branch and the posterior trunk below its coeliac branch. Eight of his patients with gastric ulcer were cured by vagotomy alone and seven by vagotomy and gastro-enterostomy.

Hartzell[69] showed that vagal section in the dog resulted in a marked reduction in free and total acid secretion. McCrea[64] of Manchester was the first surgeon to suggest that the vagi contributed to chronicity and complications of ulcer and recommended vagotomy for these problems. Pieri,[70] an Italian surgeon, endorsed this view and reported successful results in eight patients with peptic ulcer. In 1926 Klein in New York[71] performed anterior truncal vagotomy as an adjuvant to gastrectomy for duodenal ulcer with the express objective of further lowering acid output. In 1938 the same group[72] was able to report 26 cases from the Mount Sinai Hospital, all of whom became achlorhydric after the procedure.

Failure of surgeons to consistently perform a complete vagotomy inevitably led to a high failure rate. Surgeons failed to appreciate the high origin of a branch to the fundus (later described by Grassi as the 'criminal nerve'),[73] and the presence of branches to the lesser curve, which pursue an independent pathway to the parietal cell mass.

Although Birchner in Germany and Pieri in Italy performed vagotomy in patients with peptic ulcer, it was the methodical experimental and clinical work of Dragstedt and his colleagues which firmly established the place of truncal vagotomy in clinical practice from the early 1950s. In 1943 he reported two patients[74] in whom transthoracic truncal vagotomy had been performed for chronic duodenal ulcer: the gastric acid secretion was lowered post-operatively and the ulcers healed. Further reports followed rapidly over the next few years. Dragstedt changed to an infra-diaphragmatic vagotomy and reported the same lowering of acid secretion and ulcer healing.

In addition to recording the benefits of vagotomy, Dragstedt and his colleagues also noted some problems.[75] Whilst acid secretion was reduced, it was not abolished. Furthermore, intravenous histamine still produced an acid secretory response. These observations and the resultant failure to control the disease in some patients provided evidence for those who regarded vagotomy as inadequate treatment. Dragstedt's group in continuing animal research had shown the antrum (and thus antrectomy) to be the

key to the abolition of ulcer disease in the dog. By 1951 he was able to report that when the entire canine stomach was isolated from the intestinal tract and its vagal supply interrupted, ulcers did not develop.

The second problem which Dragstedt described was that of gastric stasis as a result of the section of the vagal motor fibres to the antrum. Thus the need for a drainage procedure was recognised early.

Pyloroplasty

Pyloroplasty was first performed by Heineke in 1886. A longitudinal incision was made through all layers of the anterior wall of duodenum and antrum across the pylorus, and was sutured transversely to avoid narrowing the gastric outlet. Mikulicz[76] described an identical procedure two years later. The Heineke-Mikulicz pyloroplasty was to become the most widely performed drainage procedure. It was not used initially as a drainage procedure but rather for closure following excision of anterior ulcers, and then for infantile congenital hypertrophic stenosis. (In the latter condition the results were almost always fatal before pyloromyotomy was introduced by Ramstedt[77] of Munster in 1912 - although there are competing claims.)

The first pyloroplasty for duodenal ulcer was performed in 1896 at the Mayo Clinic and excision of the ulcer with closure as a pyloroplasty was reported from there in 1902. Judd[78] reported a series of such cases concluding that the procedure was successful in about 60%.

Weinberg and colleagues from California described a single layer pyloroplasty which they claimed was superior to the double-layered Heineke-Mikulicz.[79] In their report of 500 cases they claimed only 5% of patients suffered untoward side-effects which they believed was due to the elimination of retrograde movement of food seen with gastro-jejunal anastomoses.

Yet vagotomy and gastro-jejunostomy as advocated by Dragstedt increased in popularity in the UK throughout the 1950s and 1960s. When Dragstedt adopted the Weinberg pyloroplasty as his favoured drainage procedure, vagotomy and pyloroplasty became the most popular operation for duodenal ulcer in the English-speaking world in the 1960s and 1970s. Subsequent prospective randomised trials comparing these drainage procedures, however, showed no difference in results between them.[80]

Truncal vagotomy on trial

Given the enthusiasm with which truncal vagotomy had been adopted it was surprising that several controlled studies found problems. The Leeds-York trial[44] found that vagotomy and gastro-enterostomy was inferior to vagotomy and antrectomy, and also to subtotal gastrectomy in terms of Visick grading, recurrent ulcer and the incidence of diarrhoea. Furthermore vagotomy and drainage was not associated with a lower incidence of operative mortality or of post-gastrectomy symptoms. Other trials by Cox,[45] Price,[81] Howard[82] and Postlethwait[83] in the early 1970s failed to show a clear benefit for vagotomy. While the mortality rate tended to be higher following gastrectomy, the recurrent ulcer rate tended to be lower. Truncal vagotomy and drainage probably remained the preferred operation because of its lower mortality rate and because of concern about sequelae of gastrectomy. Iron deficiency and megaloblastic anaemia, steatorrhoea and 'dumping' began to be reported although none as severe as could occur

after gastrectomy. But the most severe post-vagotomy complication was diarrhoea which, in a small proportion of unfortunate individuals, could permanently blight their quality of life, with little hope of cure.

The controlled trials of the late 1970s and early 1980s showed a clear advantage for highly selective, over truncal, vagotomy, but doubts about the recurrence rate after the former, combined with its technical challenge, ensured a role into the 1990s for truncal vagotomy.

VAGOTOMY AND ANTRECTOMY

Vagotomy combined with antrectomy (Figure 13) represented an approach to duodenal ulcer disease which more than any other was based on a clear understanding of the physiology of gastric acid secretion. Arguably the procedure abolished both the cephalic and gastric phases of acid secretion without the need for extensive gastric resection with its untoward sequelae. It was undoubtedly the procedure which most successfully abolished the ulcer diathesis.

Klein[71] in 1929 published the first report of the combination describing anterior truncal vagotomy combined with Billroth I gastrectomy in eight patients with duodenal ulcer 'with hyperacidity'. The problem of hyperacidity in the aetiology of duodenal ulcer was appreciated by Winkelstein and Berg,[72] who in 1938 advocated vagotomy and partial gastrectomy and were able to demonstrate achlorhydria as a result of the procedure.

Dragstedt's group[84] showed that in dogs with a Pavlov pouch, gastric secretion was reduced after vagotomy, but subsequent antrectomy completely abolished secretion in the isolated stomach pouch.

The experimental work of Storer[85] in Dragstedt's laboratory demonstrated in the dog the superiority of vagotomy and antrectomy in preventing recurrent ulceration. In the Mann-Williamson preparation, vagotomy alone protected against ulceration in 55% of animals, antrectomy alone in 66% while vagotomy combined with antrectomy was effective in 83%. Kay[86] compared the acid response to histamine in man following those three procedures: vagotomy reduced maximal acid output by 60%, antrectomy by 70% and a combination of the two procedures by 95%. The first clinical reports of complete truncal vagotomy, as advocated by Dragstedt, combined with gastric resection, came from Farmer and Smithwick[87] from Boston, who performed a 50% gastrectomy, arguing that by removing the antrum in addition to performing truncal vagotomy they could in one operation reduce the two major stimuli to parietal cell acid production. They made the important observation that this procedure reduced acid output as much as radical gastrectomy, alone or combined with vagotomy. Edward and Herrington described a 40% gastrectomy a procedure which they christened 'antrectomy'.[88] Dragstedt's group[89] showed in a series of animal experiments an 86% reduction in acid output in both Pavlov and Heidenhain pouches and virtual complete protection against stomal ulcer in the Mann-Williamson preparation. The procedure was taken up with enthusiasm in the US with numerous reports of series with recurrent ulcer rates of less than 1%. In many centres in North America this remained the operation of choice for duodenal ulcer until the demise of surgical treatment.
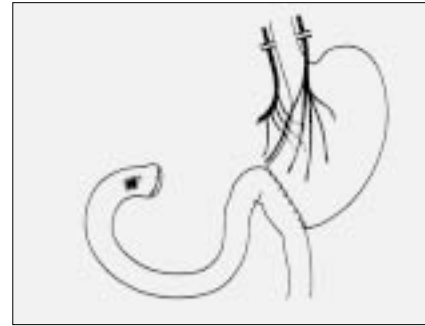

FIGURE 13
Vagotomy and antrectomy.

SELECTIVE VAGOTOMY

Latarjet's was the earliest report[68] on a technique of vagal section which sought to selectively divide vagal fibres to the stomach whilst preserving those to the rest of the intestinal tract. Although drainage procedures had overcome the problems of gastric stasis, they had brought their own sequelae. Dumping could be attributed to gastric incontinence, and bile vomiting to reflux into the stomach through the afferent loop or the incompetent pylorus. Diarrhoea was thought to result from parasympathetic denervation of the abdominal viscera, which in addition caused bloating, excessive borborygmi and abdominal discomfort.

Franksson of Stockholm devised a selective denervation without a drainage procedure but, in the light of his subsequent experience, came to recommend one. Independently, Jackson[90] and Moore[91] in the US described partial gastric vagotomy, which consisted of preservation of the coeliac division of the posterior vagus but division of the anterior vagal trunk. It was Griffith and Harkins who in 1957 published the first account of selective vagotomy.[92] They subsequently produced evidence, based on insulin tests of vagally-mediated acid output, that the procedure resulted in satisfactory denervation of the parietal cell mass. Because the motor innervation of the antrum was divided, a drainage procedure was still required.

Early results of selective vagotomy from Griffith[93] and from Burge[94] appeared promising both in terms of recurrent ulcer and post-vagotomy sequelae. As had happened so often in the saga of operations for duodenal ulcer, controlled trials and, more especially, long-term follow-up studies by Sawyers,[95] Mason[96] and others showed no advantage for the new procedure over truncal vagotomy and drainage. Longer follow-up[97] confirmed that the problems of dumping, vomiting and diarrhoea were no different following selective vagotomy and drainage. As it was more technically demanding and time-consuming, the operation enjoyed only a brief vogue.

HIGHLY SELECTIVE VAGOTOMY (PARIETAL CELL VAGOTOMY)

In 1953 Griffith, working in Harkins laboratory in Seattle, performed on dogs a procedure which he termed 'parietal vagotomy'. Antral motility was retained whilst complete denervation of the parietal cell mass was achieved as demonstrated by negative insulin tests. Griffith sought the opinion of Drs Dragstedt and Hollander about the new technique, but neither the pioneer of truncal vagotomy nor the developer of the insulin test approved. They rejected the concept of selective or parietal vagotomy

because they believed that any one vagal fibre to the stomach, if left intact, could re-innervate the entire stomach (the 'all-or-none' law). Griffith describes three parietal vagotomies carried out in 1956 but because of the disapproval of his seniors he was not allowed to repeat the procedure. He states that the three patients did 'quite well without either dumping or recurrence'.[98]

Burge recognised the advantages of avoiding a drainage procedure and published an account of selective vagotomy without drainage.[99] He believed that bilateral selective vagotomy would preserve the innervation to the prepyloric stomach. His failure to appreciate the anatomy of the nerve of Latarjet resulted in its division with consequent gastric stasis.

Holle and Hart in Munich in 1967, apparently unaware of Griffith's work, had shown in the experimental model that the vagal innervation of the antrum actually inhibited gastric secretion.[100] This led them on to develop a parietal cell vagotomy preserving the antral innervation in order to preserve this antral inhibition of acid secretion.

Griffith, meanwhile, had continued with dog experiments designed to produce evidence to show that the 'all-or-none' law did not apply. He aimed to further demonstrate that the vagal innervation of the stomach was segmental and that the hepato-biliary vagal fibres did not contribute to gastric innervation. These hypotheses were finally proved by Amdrup and Griffith in 1969.[101] On the basis of this knowledge, Amdrup (Figure 14) began to perform parietal cell vagotomy in man in 1969, the same year that Johnston in Leeds began to practise highly selective vagotomy. The initial experience with this operation was reported by Johnston and Wilkinson in 1970[102] and by Amdrup the same year.[103] The difference in nomenclature was readily explained. Amdrup mapped out the parietal cell mass with dye or pH measurement, while Johnston depended on anatomical landmarks to identify vagal fibres. Recurrent ulcer proved to be the main problem associated with this operation.

For a given recurrence rate to be meaningful, the length of follow-up of the series is crucial. Recurrent ulcer after highly selective vagotomy continued to present throughout the length of follow-up, so that those series with longer follow-up will have higher recurrence rates. Indeed in one of the series with the longest follow-up, only one-third of recurrences were manifest at five years.[103] In one of the last reviews of HSV from Edinburgh, there was 20% recurrence rate at 12 years, although the diathesis had been modified to the extent that in over 90% of patients symptoms were cured or greatly improved.[105]

Other variations on the selective vagotomy theme were described by Taylor in Edinburgh.[106] His lesser curve sero-myotomy reduced the time and the tedium of HSV. Hill[107] developed a further variant combining anterior HSV (latterly, sero-myotomy) with posterior truncal reducing the operating time still further.

THE DEMISE OF SURGICAL TREATMENT

With the knowledge that duodenal ulcer can be successfully treated in most patients with simple oral therapy to eradicate *H. pylori*, surgical interest in duodenal ulcer has all but disappeared. As duodenal ulcer incidence continues to decline by the year and peptic ulcer diathesis becomes less aggressive by the decade, these will surely represent the last surgical contribution to the management. It was ironic that by the time these ulcer operations, which were certainly the safest and perhaps most effective, had been developed, their raison d'être had gone.

A good obituary should only hint at the bad while emphasising the good. From the perspective of an era where ulcers are cured by a week's course of tablets, it is all too easy to look back in horror at the attempts of twentieth century surgeons to treat disease and brand these as barbaric. Yet the surgery of peptic ulcer provided one of the best examples to date of the application of anatomy, physiology and pathology to treat a common disease. For most of its recipients, ulcer surgery was helpful, for others it was life-saving, but for a small, important minority, ulcer operations blighted (and often shortened) the rest of their lives.

FIGURE 14

Erik Amdrup (right), Danish pioneer of highly selective or parietal cell vagotomy, pictured with the author (left).

REFERENCES

36 Gordon-Taylor G. The remote results of gastrectomy. *Brit J Surg* 1929; 16:641-645.
37 Inglefinger FJ. The late results of total and subtotal gastrectomy. *N Engl J Med* 1944; 231:321-327.
38 Fischer AB *et al.* Late mortality following Billroth II resection for duodenal ulcer. *Acta Chir Scand* 1985; 151:43.
39 Everson TC. Collective review: Nutrition following total gastrectomy. *Int Abst Surg* 1952; 95:209.
40 Muir A. Postgastrectomy syndromes. *Brit J Surg* 1949; 37:165-178.
41 Wells CA, Welbourne RB. Post gastrectomy syndromes. *Brit Med J* 1951; 1:546.
42 Wallensten S. Results of the surgical treatment of peptic ulcer by partial gastrectomy according to Billroth I and II methods: a clinical study based on 1256 operated cases. *Acta Chir Scand* 1954; (suppl 191).
43 Stammers FAR, Williams JA. *Partial gastrectomy*. Stammers FAR, Williams JA, editors. London; Butterworths; 1963.
44 Goligher JC, Pulvertaft CN, de Dombal FT *et al.* Five to eight year results of Leeds / York controlled trial of elective surgery for duodenal ulcer. *Brit Med J* 1968; 2:781-787.
45 Cox AG. Comparison of symptoms after vagotomy with gastrojejunostomy and partial gastrectomy. *Brit Med J* 1968; 1:288-290.
46 Pulvertaft CN. The late results of gastric resection. *Brit J Surg* 1964; 51:414-419.
47 Wolfler A. Gastro-Enterostomie. *Zentrabl Chir* 1881; 45:705-708.
48 Waugh JM, Hood RT. Gastric operations: A Historic Review, Part I. *Quart Rev Surg Gynec Obstet* 1953; 10:201-213.
49 Olch PD, Harkins HN. *A historical review of gastric surgery in Surgery of the Stomach and Duodenum*. Harkins HN, Nyhus LM editors. London: Churchill; 1962.
50 Rockwitz C. Die Gastroenterostomie au die Strassburger Chirurgischen Klinik. *Dtch Z Chir* 1887; 25:502-564.

51 Mayo WJ. Complications following gastro-enterostomy. *Ann Surg* 1902; 36:231-244.

52 Wilkie DPD. Gastro-jejunal and Jejunal Ulceration following gastroenterostomy. *Edin Med J* 1910; 5:316-327.

53 Sherren J. Gastro-jejunostomy. *Lancet* 1925; 2:1007-1016.

54 Ogilvie H. The place of surgery in the treatment of peptic ulcer. *Lancet* 1935; 1:641-642.

55 Clark DH. Posterior Gastro-enterostomy in peptic ulcer: long term results. *Brit Med J* 1951; 1:57-61.

56 Farquharson EL. Doubts and misgivings about the treatment of duodenal ulcer. *Lancet* 1956; 3:849-851.

57 Bruce J, Card WI, Marks IN *et al.* The rationale of selective surgery in the treatment of duodenal ulcer. *J Roy Coll Surg Edin* 1959; 4:85-104.

58 Small WP, Smith AN. Gastroenterostomy in a selective surgical policy for duodenal ulcer. In: *Surgery of the stomach and duodenum*. 3rd ed. Nyhus LM, Wastell C, editors. Boston: Little, Brown and Co; 1977.

59 Brodie BC. Experiments and observations on the influence of the nerves of the eighth pair on the secretions canal after splanchnic and vagus nerve section. *Science* 1906; 27:764.

60 Beaumont W. Experiments and observations on the influence of the nerves of the eighth pair on the secretions of the stomach. *Philos Trans* 1814; 104:102.

61 Pavlov IP (translation WH Thompson). The work of the digestive glands. London: Griffen; 1902

62 Cannon WB. On the motor activities of the alimentary canal after splanchnic and vagus nerve section. *Science* 1906; 27:764.

63 Ungar E, Bettman M, Rubaschow S. Die doppelseitige intrathorakale vagotomie. *Berl Klin Wechnscher* 1911; 48:939.

64 McCrea ED. Nerves of the stomach and their relation to Surgery. *Brit J Surg* 1926; 13:621-648.

65 Exner A. Tabische Krisen, Ulcus Ventriculi und Vagus. *Wien Klin Wechnschr* 1912; 25:1405-1406.

66 Exner A, Schwarzmann E. Gastriche Krisen und Vagotomie. *Mitt Grenzneb Med Chir* 1914; 28:15.

67 Bircher E. Die Resektion von Aesten der N. Vagus zur Behandlung gastrischner Affectionen. *Schweitz Med Wechenschr* 1920; 50:519-522.

68 Latarjet A. Resection des nerfs de l'estomac. Technique operatoire: Resultats cliniques. *Bull Acad Med Paris* 1922; 87:681-691.

69 Hartzell JB. The effect of section of the vagus nerves on gastric acidity. *Am J Physiol* 1929; 19:161.

70 Pieri G. La Resezione soprodiagraumatica e sottodia fraumatica. *Ann Ital Chir* 1927; 6:941-947.

71 Klein E. Left vagus section and partial gastrectomy for duodenal ulcer with hyperacidity. *Ann Surg* 1929; 90:65-69.

72 Winkelstein A, Berg AA. Vagotomy plus partial gastrectomy for duodenal ulcer. *Am J Dig Dis* 1938; 5:497-501.

73 Grassi G. Anatomy of the 'criminal branch' of the vagus and its surgical implications. In: *Surgery of the stomach and duodenum*. 3rd ed. Nyhus LM, Wastell C. Boston: Little, Brown & Co; 1977; 60.

74 Dragstedt LR, Owens FM. Supradiaphragmatic section of the vagus nerves in the treatment of duodenal ulcer. *Proc Soc Exp Biol Med* 1943; 53: 52-155.

75 Dragstedt LR, Woodward ER, Storer EH *et al.* Quantitative studies on the mechanism of gastric secretion in health and disease. *Ann Surg* 1950; 132:626-629.

76 Mikulicz J von. Zur Operativen Behandlung des stenosirenden Mageneschwueres. *Arch Klin Chir* 1888; 37:79-90.

77 Ramstedt C. Zur Operation der angeborenen Pylorusstenose. In: Versammlung deutscher Naturforscher und Arzte in Munster (Westfalen). *Zentrabl Chir* 1912; 39:1741-1742.

78 Judd ES, Hazeltine ME. The results of operations for excision of ulcers of the duodenum. *Ann Surg* 1930; 92:563.

79 Weinberg JA, Stempein SJ, Movius HJ *et al.* Vagotomy and pyloroplasty in the treatment of duodenal ulcer. *Am J Surg* 1956; 92:202-207.

80 Kennedy T, Johnston GW, Love AHG *et al.* Pyloroplasty versus gastrojejunostomy: Results of a double blind controlled trial. *Brit J Surg* 1973; 60:949-953.

81 Price WR, Grizzle JE, Postlethwait RW *et al.* Results of operation for duodenal ulcer. *Surg Gynec Obstet* 1970; 131:233-244.

82 Howard RJ, Murphy WR, Humphrey EW. A prospective randomised study of the elective treatment for duodenal ulcer: two-to-ten-year follow-up study. *Surgery* 1973; 73:256-260.

83 Postlethwait RW. Five year follow-up results of operations for duodenal ulcer. *Surg Gynec Obstet* 1973; 137:387-392.

84 Woodward ER, Lyon ES, Landor J *et al.* The Physiology of the gastric antrum. *Gastroenterology* 1954; 27:766.

85 Storer EH, Woodward ER, Dragstedt LR. The effect of vagotomy and antral resection on the Mann-Williamson ulcer. *Surgery* 1950; 27:526-530.

86 Kay AW. Gastro-intestinal surgery and human physiology. *J Roy Coll Surg Edin* 1962; 7:275-288.

87 Farmer DA, Howe C, Porrell WJ *et al.* The effect of various surgical procedures upon the acidity of the gastric contents of ulcer patients. *Ann Surg* 1951; 134:319.

88 Edward LW, Herrington JL. Vagotomy and gastro-enterostomy - vagotomy and conservative gastrectomy. A comparative study. *Ann Surg* 1953; 137:6.

89 Woodward ER, Bigelow RR, Dragstedt LR. The effect of resection of the antrum of the stomach on gastric secretion in Pavlov pouch dogs. *Am J Physiol* 1950; 162:99-109.

90 Jackson RG. Anatomic study of the vagus nerves with technic of transabdominal selective gastric vagus resection. *Arch Surg* 1948; 57:333-352.

91 Moore FD. Follow-up of vagotomy in duodenal ulcer. *Gastroenterology* 1948; 11:442-452.

92 Griffith CA, Harkins HN. Partial gastric vagotomy. An experimental study. *Gastroenterology* 1957; 32:96-101.

93 Griffith CA. Gastric vagotomy versus total abdominal vagotomy. *Arch Surg.* 1960; 81:781-788.

94 Burge HW. Vagal nerve section in chronic duodenal ulceration. *Ann R Coll Surg. Engl* 1960; 26:231-234.

95 Sawyers JL, Scott HW, Edwards WH *et al.* Comparative studies of the clinical effects of truncal and selective gastric vagotomy. *Am J Surg* 1968; 115:165-172.

96 Mason MC, Giles GR, Graham NG *et al.* An early assessment of selective and total vagotomy. *Brit J Surg* 1968; 55:677-680.

97 Sawyers JL, Herrington JL, Burney P. Proximal gastric vagotomy compared with vagotomy and antrectomy and selective gastric vagotomy and pyloroplasty. *Am J Surg* 1977; 186:510-517.

98 Griffith CA. Selective gastric vagotomy. In: *Surgery of the Stomach and Duodenum*. Nyhus LM, Wastell C editors. 3rd ed. Boston: Little, Brown & Co; 1977.

99 Burge HW, Hutchison JSF, Longland CJ *et al.* Selective nerve section in the prevention of post vagotomy diarrhoea. *Lancet* 1964; 1:577-579.

100 Holle F, Hart W. Nueu Wege der Chirurgie das Gastroduodenal ulcus. *Med Klin* 1967; 62:441.

101 Amdrup BM, Griffith CA. Selective vagotomy of the parietal cell mass. *Anns Surg* 1969; 170:207.

102 Johnston D, Wilkinson AR. Highly selective vagotomy without a drainage procedure in the treatment of duodenal ulcer. *Brit J Surg* 1970; 57:289-296.

103 Amdrup E, Jensen H-E. Selective vagotomy of the parietal cell mass preserving innervation of the undrained antrum. *Gastroenterology* 1970; 59:522-527.

104 Hoffman J, Olesen A, Schulze S. Prospective 14-18 year follow-up study after parietal cell vagotomy. *Brit J Surg* 1987; 74:1056-1059.

105 Macintyre IMC, Millar A, Smith AN *et al.* Highly selective vagotomy 5-15 years on. *Br J Surg* 1990; 77:65-69.

106 Taylor TV. Lesser curve seromyotomy - an operation for duodenal ulcer. *Brit J Surg* 1979; 66:733-737.

107 Hill GL, Barker MCJ. Anterior highly selective with posterior truncal vagotomy. *Brit J Surg* 1978; 65:702.