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Review

A relic or still relevant: the narrowing role for vagotomy in the treatment of peptic ulcer disease

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Abstract

BACKGROUND: Given the rise of medical treatment for peptic ulcer disease (PUD), surgical treatment is necessary only in select cases and emergencies. The authors assess the current relevance of surgical vagotomy to treat PUD and its complications.

DATA SOURCES: Although historically significant, selective and highly selective vagotomy is very technically challenging, and highly selective vagotomy has a relatively narrow indication and high recurrence rates. Vagotomy and gastrectomy is associated with significant side effects. Two types of vagotomy remain relevant, within a narrow scope. Truncal vagotomy and pyloroplasty is safe and efficacious through a laparoscopic approach in certain emergent cases. Vagotomy and Roux-en-Y gastrojejunostomy can be used to treat severe PUD refractory to medical management.

CONCLUSIONS: The role of vagotomy in the management of PUD has a rich history but predated pharmacologic control of acid and understanding of the role of *Helicobacter pylori* in the disease. Thus, the current role of vagotomy is significantly limited. Specifically, the emergent use of truncal vagotomy is warranted for patients who are either resistant or allergic to proton pump inhibitors.

The landscape for the treatment and management of peptic ulcer disease (PUD) has drastically changed over the past 50 years. Treatments started as primarily dietary and evolved through a period of surgical predominance to the current treatment landscape that relies mostly on medical management. In this report, we review the increasing evidence supporting a diminished, yet necessary, role for surgery in the management of PUD. Specifically, the role of vagotomy is examined and reevaluated in the surgical management of complicated PUD.

History of Vagotomy

The role of surgery in the treatment of PUD dates back to Andre Latarjet, who in 1922 reported the first human vagotomy in a study of 24 patients.[1] However, given the high rates of delayed gastric emptying, <100 operations were performed between 1922 and 1940. Instead, there was growing focus on the role of gastric resection during this time.[2,3] However, Lester Dragstedt revived the role of vagotomy in the management of PUD and published his findings from >200 thoracic vagotomies in 1947. Dragstedt's work was greatly influenced by growing scientific data that supported the idea that vagal denervation could

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favorably reduce acid secretion and thereby improve the clinical course of duodenal ulcers.[4,5] The following decades saw the rise of vagotomy with various modifications. In 1953, an evaluation of 200 patients undergoing vagotomy (and 40% with gastrectomy) showed excellent results in 93.4% of patients.[6] Dragstedt et al[7] also went on to publish a critical evaluation of 100 patients undergoing truncal vagotomy and pyloroplasty. Thus, from the rejuvenation of vagotomy in 1947 by Dragstedt, vagotomy in combination with either pyloroplasty or antrectomy, started to become the gold standard for the treatment of PUD and its most common complications, including bleeding and obstruction.

However, by the 1970s and 1980s, H_2 receptor antagonists for gastric acid suppression and proton pump inhibitors (PPIs) were introduced, forever changing the treatment of the disease. In a landmark report, the results of a double-blind controlled trial of cimetidine showed ulcer healing in 66% of patients within 6 weeks.[8] Concurrently, Drs Barry Marshall and Robin Warren completely altered the conventional wisdom regarding the etiology of PUD by proving that *Helicobacter pylori* was found in the stomachs of 75% to 85% of patients with PUD.[9–11] Their discovery earned them the Nobel Prize in medicine in 2005 and ushered in a paradigm shift in both the understanding and treatment of PUD. In the United States, *H pylori* infection and the use of nonsteroidal anti-inflammatory drugs are the predominant causes of PUD, accounting for 48% and 24% of cases, respectively. Specific to *H pylori*, the pathophysiology of PUD is now firmly based in the fact that *H pylori* infection and concurrent inflammation cause increased levels of gastrin release and decreased gastric mucous and duodenal mucosal bicarbonate.[12] The decline of protective factors and increased secretion of gastric acid is instrumental in the ability of *H pylori* infection to cause PUD.[13]

Given the discovery of the crucial role of *H pylori* in the etiology of PUD, the decrease in the incidence of *H pylori* infection secondary to improved sanitation, and the exponential rise of PPIs in the management of the same, the continued role and importance, if any, of surgical vagotomy in the treatment of PUD is brought into question.[14,15] Numerous studies have firmly established the positive effect of vagal stimulation on gastric acid secretion compared with the antisecretory effects of PPIs.[16–18] In the remainder of this article, we review the current roles of various types of vagotomies and explore current patterns of practice for the treatment and management of the most common presentations of PUD, including bleeding, obstruction, and perforation.

Types of Vagotomy for Peptic Ulcer Surgery

Selective vagotomy

The first truncal vagotomy dates back to Latarjet in 1921, and the procedure gained popularity through the work of Dragstedt in 1943. However, given the lack of a drainage procedure, one third of patients developed delayed gastric emptying after truncal vagotomies. Thus, Dragstedt advocated the addition of a drainage procedure, and the next 30 years also saw a progressive refinement of the vagotomy to increase selectivity.[5]

Selective vagotomy, defined as the division of vagal fibers to the stomach with preservation of the hepatic and celiac branches, dates back to 1922 and was first described by Wertheimer[19] and Latarjet.[1] In 1947, Jackson and Franksson, independently, introduced selective posterior vagotomy and total anterior vagotomy. This was followed, in 1948, by 2 significant advances. First, Moore was responsible for a selective vagotomy that included total denervation of the stomach with preservation of the hepatic and celiac supplies of the vagus, resulting in fewer postvagotomy side effects. Franksson also performed a selective anterior and posterior vagotomy with preservation of the pyloric ramus without drainage. Work on refinement of the selective vagotomy technique continued, and in 1963, a study presented results from 52 patients who underwent selective gastric vagotomy. It confirmed that selective vagotomy, by preserving enervation of the gallbladder, pancreas, and major parts of the intestine, was associated with less diarrhea and dumping syndrome compared with truncal vagotomy alone. It was also considered to be at least as effective as truncal vagotomy in establishing full vagal denervation.[20] A 1969 study confirmed that selective vagotomy is capable of reducing the incidence of dumping syndrome but is unable to control duodenal ulcer diathesis given the high secretory potential of the innervated antrum of the stomach.[21]

Although it is of historical significance, there are very few practicing physicians who use selective vagotomy. This operation did not gain wide clinical acceptance because it was much more challenging than truncal vagotomy, and it was unclear that the scientific advantages of the operation translated to actual clinical benefit. Selective vagotomy appears to have no role in the current management of PUD.

Highly selective vagotomy (HSV)

Given the interest in combating delayed gastric emptying and reducing the need for drainage procedures, continued emphasis was placed on increasing the selectivity of the vagotomy operation. HSV was first introduced by Griffith and Harkins in 1957. A landmark report from 1970, "Highly Selective Vagotomy Without a Drainage Procedure in the Treatment of Duodenal Ulcer" by Johnston and Wilkinson,[22] provided significant evidence to support the continued role of HSV in the treatment of PUD. Technically they described selective denervation of the parietal cell mass by dividing the nerve branches of the anterior and posterior vagus as they enter the stomach. Because these nerves are comingled with their blood supply, this was usually accomplished by close devascularization of the lesser curvature of the stomach from just above the gastroesophageal junction to the "crow's foot" on the antrum. They defined the clinical indication for HSV as chronic PUD and treated 25 patients