Management of Complicated Peptic Ulcer Disease

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If anyone should consider removing half of my good stomach to cure a small ulcer in my duodenum, I would run faster than he.
C. E. Mayo, 1927

Our understanding of the pathophysiological features of peptic ulcer disease has rapidly evolved during the last century. Early understanding of gastric physiologic characteristics led to logical and time-tested surgical procedures aimed at acid reduction and a lowering of ulcer recurrence rates. A dramatic shift in treatment occurred with the recognition and growing knowledge of *Helicobacter pylori* (HP). Where once surgery dominated therapy for ulcer diathesis, medical therapy has now superseded. Just as gastric resection seemed radical to Dr Mayo, thus was medical treatment of peptic ulcer disease similarly approached with skepticism.

What was the balance of medical and surgical management of peptic ulcer disease as we entered the 21st century? Complications of peptic ulcer disease requiring operative intervention have remained important. However, the absolute number of procedures performed has significantly diminished in recent years.³ The reason for the decrease in surgical intervention, both emergent and elective, is multifactorial. Improvements in therapeutic endoscopy, the introduction of effective antacid therapy, and the recognition and treatment of HP infection have all greatly contributed to the successful nonoperative treatment of patients with peptic ulcer disease.² ³ With respect to complicated ulcer disease, treatment and eradication of HP infection have arguably led to a shift from treating patients operatively to treating them nonoperatively. This changing management scheme has occurred despite a relative paucity of data regarding the incidence and contribution of HP infection to the etiology of complicated peptic ulcers. Indeed, reports in the literature regarding the incidence of HP infection in a surgical cohort have been sparse. From a surgeon’s perspective, data relative to HP infection and the classic indications for surgery—perforation, bleeding, and gastric outlet obstruction (GOO)—have until recently been largely inferential based on treatment of those with uncomplicated peptic ulcers. Deviation from traditional surgical management might prove detrimental should other nonoperative modalities be used. The purpose of this discussion is to review the known pathophysiological features of peptic ulcer disease with a focus on those complications encountered by the surgeon.

**PATHOPHYSIOLOGICAL FEATURES**

**Acid Secretion**

Exceptional contributions by several physiologists and surgeons in the first half of the 20th century greatly added to our knowledge of gastric acid secretion.⁶ Ivan Pavlov, PhD, described the cephalic phase of acid secretion in the dog model and received the Nobel prize for his contributions. In France, Mathieu Jaboulay, MD, performed the first human vagotomy. André Latarjet, MD, detailed the lesser curve vagi, performed the first therapeutic vagotomy for treatment of an active peptic ulcer, and noted the deleterious effects of vagotomy on gastric emptying. William Bayliss, PhD, and Ernest Starling, PhD, elu-
acidified secretin and its contribution to the gastric phase of acid secretion. Lester Dragstedt, MD, PhD, reported on the importance of pyloroplasty as an adjunct to vagotomy to prevent gastric stasis. Further, he noted that the release of gastrin was due in part to an elevated gastric pH. He proposed that vagotomy combined with antrectomy was a superior operation for peptic ulcer disease owing to its eradication of the cephalic and gastric influences on acid secretion. In separate observations in the 1950s, Farmer and Smithwick at Boston University, Boston, Mass, and Edwards and Herrington at Vanderbilt University, Nashville, Tenn, reported the durability of vagotomy and antrectomy in the treatment of complicated peptic ulcer disease with long-term recurrence rates of less than 1%. Thus, the second half of the 20th century was noteworthy for several operations based on sound physiologic principles that resulted in effective surgical treatment of complicated peptic ulcer disease. Vagotomy and pyloroplasty, highly selective vagotomy, and vagotomy combined with antrectomy are most commonly used. While complications such as dumping, bile reflux, and gastric atony have been described, long-term morbidity has remained very low. These operations have proven to be safe and effective with very low ulcer recurrence rates.

HP Infection

Our knowledge of the etiology of peptic ulcer disease and surgery itself was soon to change by the description by Warren and Marshall in 1983 of a gram-negative bacteria noted in certain gastric biopsy specimens in association with antral inflammation. Originally named Campylobacter pylori, this organism was later renamed HP. Despite the growing body of literature (the majority of it nonsurgical), much is still unknown about this bacteria. Approximately one half of the world’s population and one third of American adults are thought to be infected, yet the majority are asymptomatic. The mode of transmission is not definitively known, but most likely transmission is from person to person either by the gastro-oral or fecal-oral routes. The decreasing incidence of HP infection in developed countries is thought to be related to improved sanitation and hygiene. Prevalence rates are higher in developing countries, and in these areas, infection is much more common in the young. Reinfection rates have not been defined. In the United States, seropositivity demonstrates a linear relationship with age, ranging from less than 20% at age 20 years to 50% at age 60 years.

The most dominant feature of HP is its ability to tolerate the stomach environment with its acidic pH, constant emptying, and rapidly exchanging epithelial layer. Several adaptive features allow this organism to survive. Its motility allows it to burrow through the mucin layer of the stomach to reach the gastric epithelial cells. Subsequently, adherins on the bacterial wall are able to attach to receptors on gastric mucosal cells. Receptors for HP (mainly Lewis B antigens) are present only in the gastric epithelium and not elsewhere in the gastrointestinal tract. The motility of the bacteria and its adherence mechanism allow it to colonize the stomach despite ongoing gastric motility.

Several metabolic features allow the organism to remain unharmed once colonization has ensued. Since it is microaerophilic, survival is possible even with the low oxygen levels observed in the gastric lumen. It is a facultative acidophih and thus its enzyme systems function well at a low pH. Helicobacter pylori generates a high catalase activity, which seems to be a protective mechanism against oxidative injury induced by neutrophil attack and hydrogen peroxide release. Finally, HP releases large quantities of urease, which hydrolyzes gastric intraluminal urea to ammonia and carbon oxide (the basis of the CLO [Camphylobacter-like organism] test). The ammonia effectively neutralizes local hydrochloric acid in the organism’s immediate environment—a process without which the bacteria cannot exist. Mutant HP that lack urease production fail to survive in the stomach.

Physiologically, HP results in a relative hypergastrinemia. This is thought to be on the basis of ammonia production, which locally raises pH exposed to G cells. In addition, somatostatin production—a negative feedback inhibitor of gastrin—is reduced. The impact of this hypergastrinemia on gastric acid secretion is less well characterized. In asymptomatic individuals with active infection, basal and peak acid outputs are normal. In symptomatic patients with an ulcer, basal and peak acid outputs may sometimes be elevated but may also be normal. Eradication of HP infection lowers acid output in those with elevated levels, though this may lag behind treatment by several months.

Helicobacter pylori causes additional dysregulations in gastric physiologic features. Mucosal inflammation results in gastric metaplasia within the duodenum. In conjunction with a high duodenal acid load, HP colonization is favored. This colonization results in further inflammation and additional gastric metaplasia with a resultant increase in HP density in the duodenal bulb. This cycle may eventually allow the development of frank ulceration. The inflammatory reaction caused by HP and the high duodenal acid production impairs bicarbonate secretion by the duodenal mucosa and diminishes neutralization of duodenal acid. In addition, glycine-conjugated bile acids that normally impair HP proliferation are precipitated in the presence of a high duodenal acid load and acidification of duodenal contents. Decreasing acid production and thereby bile acid precipitation by various acid-reducing medications is helpful in reversing this process but cannot in and of itself lead to complete regression or eradication of HP. Other environmental factors are involved. Stress and smoking increase gastric acid secretion and the duodenal acid load. Nonsteroidal anti-inflammatory drugs (NSAIDs) may injure duodenal mucosa and promote gastric metaplasia serving as a nidus for HP proliferation.

Various strains of HP exist, and virulence likewise differs. All strains of the bacteria cause mucosal inflammation but to a variable extent. Mechanisms whereby active ulceration occurs in infected individuals or not remains to be elucidated. In fact, peptic ulcer prevalence differs within and between countries despite a comparable prevalence of HP infection. One may hypothesize that either the virulence and possibly the density of the infecting organism is different and/or that other factors—environmental or genetic—are involved.
Nonsteroidal Anti-Inflammatory Drugs

Nonsteroidal anti-inflammatory drugs remain an important cause of peptic ulcers. They are widely prescribed and used particularly in high-risk-aging populations. Other risk factors for NSAID-induced ulceration are high-dose or multiple-NSAID use, comorbid illness, history of previous ulcer bleeding, cotherapy with corticosteroids or anticoagulation therapy, and probably concurrent HP infection.22,23 The pathophysiological features of toxic effects on the gastrointestinal tract relate to a suppression of gastric prostaglandins.24 The reduction in prostaglandins leads to a decrease in epithelial mucus, bicarbonate secretion, mucosal perfusion, epithelial proliferation, and ultimately the mucosal resistance to injury. The risk of life-threatening ulcer complications in long-term NSAID use ranges from 1% to 4%.25

*Helicobacter pylori* is present in approximately 50% of patients with NSAID-induced ulcer disease.26 It is the elderly population that is most at risk for HP infection as well as NSAID-induced complications. Whether active HP infection increases the risk of ulcer formation in those taking NSAIDs remains speculative. Two long-term longitudinal studies examining this issue have yielded conflicting results. Kim and Graham27 reported no significant increase in the incidence of gastroduodenal ulceration among long-term NSAID users with HP infection. However, patients with erosions detected at index endoscopy were excluded. In contrast, Taha et al28 found that patients with HP-positive ulcer erosions were more likely to progress to frank ulceration during NSAID treatment compared with HP-negative patients. Finally, Chan et al29 randomized patients with active HP infection to HP eradication vs no therapy prior to an 8-week course of NSAID treatment. Ulcers developed in 12 of 47 patients randomized to NSAID alone vs 1 of 45 receiving successful antimicrobial therapy. Taken together, these reports suggest that treatment of HP infection in those with presumed NSAID-induced ulceration seems reasonable. Whether routine HP infection assessment and treatment prior to embarking on an NSAID regimen is recommended remains unanswered at this time.

Several recommendations for treatment among those with NSAID-associated peptic ulceration can be made. All anti-inflammatory and antiplatelet drug treatments should be withdrawn. Any long-term anticoagulation should be temporarily reversed. Assessment and treatment of HP infection, if present, seems reasonable. If further anti-inflammatory drugs are to be used, consideration for cyclooxygenase inhibitor–specific therapy should be entertained.30 Data relative to any impact of NSAID use in the treatment algorithm for complicated ulcer disease will be discussed further.

**HP INFECTION AND UNCOMPLICATED PEPTIC ULCER DISEASE**

The impact of HP infection treatment on the reduction of uncomplicated ulcer recurrence has been well documented.31 In a report with prolonged follow-up evaluation, Van der Hulst et al32 studied 247 patients with histologically confirmed HP infection. One hundred eighty-six had documented eradication of active infection and ulcer healing (141 with duodenal and 45 with gastric ulcers), and further antacid therapy was ceased. All 186 patients completed surveillance endoscopy for 1 year without a single ulcer relapse. Ninety-six patients (64 with duodenal and 32 with gastric ulcers) had continued endoscopic follow-up for a median of 2.5 years (range, 0.5-9.8 years) without further ulceration and no evidence of HP reinfection. While the fate of those without HP eradication and the etiology of treatment failure are not reported, this study strongly suggests that elimination of HP infection prevents long-term ulcer recurrence without the need for prolonged antacid therapy. In a well-designed randomized controlled study, Graham et al33 reported on 109 patients with healed duodenal and gastric ulcers who had HP infection. Patients were randomized to HP infection treatment vs histamine antagonists alone. Treatment was discontinued after 16 weeks, and HP infection eradication was documented in those treated. Surveillance endoscopy was performed for a 2-year period. Ninety-five percent of those with duodenal ulcers not receiving HP infection treatment had ulcer recurrence vs 12% who received medical therapy. Corresponding rates for gastric ulcers were 74% and 13%, respectively. Other studies with prolonged follow-up periods corroborate the findings that eradication of HP infection markedly reduces recurrent gastroduodenal ulceration without the need for long-term antacid therapy.34-35 Can we expect results of HP infection eradication in those with uncomplicated ulcer disease to extrapolate to those with complicated ulcers encountered by the surgeon?

**SURGICAL COMPLICATIONS OF PEPTIC ULCER DISEASE: PATHOPHYSIOLOGICAL FEATURES AND MANAGEMENT**

**Perforation**

Acute perforations of the duodenum are estimated to occur in 2% to 10% of patients with ulcers. In the latter half of the 20th century, appropriate surgical management of perforated ulcers remained controversial. Many recommended simple patch closure in what is frequently an ill patient population, though others argued for a more formal antiulcer procedure.36-43 Recurrent ulcer disease was a concern unless prolonged antacid therapy was instituted or a definitive antiulcer procedure was performed. Long-term medical therapy had significant economic and compliance issues. Prolongation of surgery for a definitive procedure in the face of peritonitis was often felt to be ill advised and remained controversial. In the absence of long-term antacid therapy or a definitive antiulcer procedure, remedial surgical therapy for recurrent peptic ulcer was not uncommon.

Following simple patch closure, the clinical course of these patients remained difficult to predict. Many remained asymptomatic, but others required continued medical therapy to control ulcer symptoms or even surgical remediation. The natural history of perforated duodenal ulcer treated with suture application alone was well documented by Griffin and Organ.36 They followed the clinical course of 122 patients during a 25-year period.
Forty-six patients either died of unrelated causes or were lost to follow-up. Of the 76 followed up long-term, 5 died of recurrent ulcer-related complications, 5 had recurrent perforation, and 34 underwent a definitive antiulcer procedure because of recurrent symptoms. An additional 15 patients received prolonged medical therapy for persistent ulcer symptoms. In total, 48% of the original study population required further ulcer treatment. In the past, recurrent ulceration was thought to be on the basis of acid hypersecretion. Most recently, cocaine use and HP infection have been reported as important factors contributing to perforated duodenal ulcer.

The pathophysiological features of perforated duodenal ulcer in those abusing cocaine remains speculative. Most plausible is that perforation occurs because of a localized focal vasoconstriction or vascular thrombosis. Feliciano et al reported on the cases of 50 patients with cocaine-related perforations, which in their series represented almost 40% of all patients with juxtapyloric perforations in this inner city hospital. Omental patch closure alone was most often used. The authors suggest that this operation should suffice in the majority of patients but that a concurrent definitive antiulcer procedure should be considered in those with a history of gastroduodenal ulceration due to compliance issues. In the latter years of this study, the authors assessed their study population for HP infection by direct antral biopsy at the time of patch closure and found infection in 4 of 5 tested. It may be, as the authors hypothesize, that active HP infection predisposes crack cocaine addicts to gastroduodenal perforation.

Recent literature strongly implicates active HP infection as the cause of perforated duodenal ulcer. Reinbach et al noted an incidence of HP infection in 47% of 80 patients undergoing operation for perforated duodenal ulcer. Chu et al also reported an infectivity rate of 47% confirmed by postoperative endoscopy with biopsy at a mean of 6 years following surgery. Endoscopic evidence of an ongoing ulcer diathesis was found in 84.4% of those with active HP infection vs only 3.5% of those without, suggesting that if HP infection is not present, then recurrent ulcer disease following simple patch closure should be a rare event. Others have uniformly reported infectivity rates ranging from 70% to 92%.

Evidence suggests that medical treatment aimed at eradication of HP results in permanent resolution of a future ulcer diathesis without the need for long-term antacid therapy or surgical intervention. Ng et al performed a randomized controlled study examining this issue. Of 129 patients with duodenal ulcer perforation, 104 (81%) were infected by HP. The diagnosis was made via upper endoscopy and biopsy performed at the time of laparotomy. Postoperatively, patients were randomized to receive anti-HP therapy or a 4-week course of omeprazole alone after which treatment with all medications was discontinued. Patients completing the study protocol were followed up for 1 year with repeat endoscopy for ulcer surveillance and HP infection assessment by repeat biopsy. At 8 weeks following treatment, HP infection eradication rates were significantly higher in those treated for HP infection (84% vs 17%). At the 1-year endoscopic evaluation, 5% of the HP infection–treated group had evidence of recurrent ulceration vs 38% of those treated with antacids alone—a highly significant difference. It is important to emphasize that neither group received long-term acid-suppression therapy. Notably, the low ulcer recurrence rate in those treated for HP infection in this series is comparable with historical studies using immediate, definitive antiulcer procedures, suggesting that ulcer recurrence in the past may have been on the basis of persistent HP infection.

The etiology of perforated duodenal ulcers appears to be multifactorial but most often is associated with HP infection. Reports to date suggest that in most patients, simple patch closure alone with postoperative assessment for HP infection should suffice. Treatment and eradication of HP infection results in a very low risk for recurrent ulceration and allows withdrawal of long-term antacid therapy. Since a small percentage of patients will have other etiologies of ulcer disease (Zollinger–Ellison syndrome, Crohn’s disease), it is important to document HP infection and not treat empirically.

Bleeding

The incidence of gastroduodenal bleeding secondary to acid-peptic disease and hospital admissions for this complication have not significantly changed in the last 2 decades. Further, despite improvements in nonsurgical modalities such as proton pump inhibitors and therapeutic endoscopy, operation for peptic ulcer bleeding has remained constant; such operations are performed on 10% to 20% of all patients hospitalized for upper gastrointestinal tract hemorrhage. Evidence suggests that bleeding is more common as age increases. As such, mortality rates following ulcer bleeding have remained at approximately 10%, 61 The need for surgical intervention in this cohort remains important. In a large, prospective national survey by the American Society for Gastrointestinal Endoscopy (Oakbrook, Ill), 347 (15.6%) of 2225 patients with bleeding ulcers required surgical intervention. A further study at the University of California at Irvine noted that 19% of patients undergoing therapeutic endoscopy for ulcer bleeding required surgery. Importantly, surgery was necessary on an emergent or urgent basis in 70% and 100% of patients requiring operation in these respective series. Thus, surgery is most often necessary in the acute setting, typically within 48 hours of initial bleeding.

To what degree can therapeutic endoscopy obviate the need for surgical intervention and should it be considered more than once? Lau and attempted to answer this question in a randomized prospective study. During a 4-year period, approximately one third of 3500 patients admitted to the hospital with bleeding peptic ulcers received therapeutic endoscopy. Seventeen patients (1.5%) went directly to surgery following failure of primary endoscopic control. Bleeding recurred in 100 patients or 8.7% of the entire population. Median blood transfusion in this final group was 5 U, suggesting significant hemorrhage. Of the 48 patients randomized to repeat endoscopy, 27% percent underwent a failed endoscopy and required emergency surgical intervention with a postoperative complication rate of 46%. In those with a suc-
cessful second endoscopy, complications occurred in 14%. In contrast, 93% of patients going directly to surgery without repeat endoscopy achieved permanent hemostasis with a postoperative complication rate of 36%. On analysis, the authors noted that hypotension prior to the second endoscopy and an ulcer larger than 2 cm significantly correlated with endoscopic failure. Mortality in those undergoing repeat endoscopy was 10%, which was not significantly different from those undergoing surgical intervention for recurrent bleeding. Importantly, 4 of the 5 deaths in the repeat endoscopy arm occurred in those undergoing salvage surgical therapy for persistent bleeding. While a second therapeutic endoscopy may negate the need for surgery, fully one quarter of these patients will fail necessitating emergency surgery with a significant complication rate. Curiously, although the authors assessed for HP infection at the index endoscopy (via the CLO test), no results were reported.

With the recognition that HP infection significantly contributes to the etiology and persistence of uncomplicated ulcer disease, its role in the treatment algorithm for patients with significant gastrointestinal tract bleeding was questioned. Specifically, can therapy directed at HP infection eradication complement therapeutic endoscopy and reduce the number of patients requiring operation for hemorrhage? The question remains: Can early treatment for HP infection, if present, avert surgery in those with massive bleeding? Clearly, a rapid diagnosis of HP infection would be necessary. Techniques to diagnostically detect HP infection, such as serologic testing and histologic analysis, require several days for confirmation. Only 2 tests are available to rapidly assess for HP infection, the CLO (rapid urease) test and carbon 14 urea breath analysis. The breath analysis is not uniformly available in many institutions; further, data suggest that in those undergoing endoscopy for active bleeding, the CLO test lacks sensitivity with a substantial false-negative rate. Lee et al analyzed the diagnosis of HP infection in 55 patients with bleeding duodenal ulcers and compared results with 69 patients with uncomplicated ulcers. A variety of diagnostic methods to assess HP infection were used including the CLO test, serologic analysis, and microbiologic and histologic evaluation. The false-negative rate via the CLO test was significantly higher in those with bleeding ulcers vs those without (18.2% vs 1.4%; P<.05). These data suggest that in those undergoing endoscopy for active bleeding, the CLO test fails to detect HP infection with a substantial false-negative rate.

In 2 similarly designed and consecutively reported early studies, Rokkas et al and Jaspersen et al examined the impact of HP eradication in those patients with upper gastrointestinal tract bleeding secondary to peptic ulcer disease. The presence of HP infection in both studies was assessed by both the CLO test and histologic evaluation. Some 80 patients with bleeding ulcers and active HP infection were randomized to HP infection treatment vs antacids alone. Neither study reported what percentage of patients with bleeding ulcers had HP infection. Hemoglobin levels (9-10 g/dL) and transfusion requirements (2 U) at hospital admission were modest, suggesting this population would typically not be evaluated for surgical intervention. Only 5 of 51 patients in the study by Jaspersen et al required blood transfusion, and those with actively bleeding ulcers were excluded from analysis. It is unclear if any patient in the study by Rokkas et al underwent therapeutic endoscopy, and the number requiring transfusion was not reported. While HP infection elimination was effective in preventing recurrent ulceration in these studies, it is difficult to extrapolate these results to a population with massive bleeding, which is most commonly encountered by the surgeon.

It is becoming clear from several reports that the incidence of HP infection in a surgical cohort with significant bleeding is much lower, ranging from 39.1% to 55%, than in those with uncomplicated ulcers or minor degrees of bleeding. Data from the University of Tennessee, Memphis, suggest that in contrast to other populations, HP infection is even less common as age increases in those with massive bleeding. Further, this decreased infection in older populations could not be explained on the basis of excessive NSAID medication because use was equal in those with and without HP infection. In this series, only 2 of 39 patients ultimately undergoing operation for a bleeding ulcer had biopsies performed to assess for the presence of HP infection, most likely because of significant bleeding and the emergency nature of the procedure. In addition, one half of these patients required emergency operation before diagnosis of HP infection could have been made. In this study, a formal antiulcer procedure was performed in all patients with no patient having recurrent bleeding. Postoperative morbidity and mortality rates were 17.9% and 5.1%, respectively.

Thus, in the emergency setting with significant bleeding, rapid diagnosis of HP infection is frequently impossible. Many patients require emergency surgery, which does not allow any attempt at HP infection diagnosis. Surgery offers the lowest risk for rebleeding when compared with repeat therapeutic endoscopy. However, morbidity remains substantial in this cohort of patients with massive bleeding.

The incidence of HP infection in those patients with significant upper gastrointestinal tract bleeding secondary to peptic ulcer disease is significantly lower than in those with uncomplicated ulcers or minor degrees of hemorrhage. There are no data to substantiate a delay in surgical intervention to treat HP infection, and it would be anticipated that such a delay could be associated with an increase in morbidity and mortality in this population with significant transfusion requirements. There is no role for empirical treatment of HP infection in this cohort because the incidence of infection is substantially reduced. If HP infection is not present, the etiology of ulcer disease in this population is unknown but may be on the basis of acid hypersecretion. Thus, if surgery is to be performed, a definitive acid-reducing procedure should strongly be considered. In contrast to the arguments of others, performing a less radical operation in the face of massive bleeding with a known lower incidence of HP infection.
infection would leave up to 50% of this population at risk for rebleeding.

**Gastric Outlet Obstruction**

Benign GOO secondary to peptic ulcer disease remains prevalent and represents approximately 5% to 8% of ulcer-related complications. Obstruction necessitates operation in about 2000 patients per year in the United States. Pathophysiologically, pyloric channel stenosis leads to stasis, which raises the gastric pH resulting in pronounced gastric reflux and excess acid production. This is compounded by significant gastric distention that further increases gastric release. The combination exacerbates ongoing acid production resulting in a "vicious cycle." The incidence of HP infection in this cohort has, until recently, not been well defined and continues to suffer from small numbers for analysis. However, our understanding of its role in those with GOO is improving. Most recently, therapy for GOO primarily focused on 2 approaches, operative and nonoperative. Surgical intervention directed at a formal acid-reducing procedure has historically been the mainstay of therapy with repeatedly good results and low associated morbidity and mortality. Nonoperative management includes pneumatic dilation with or without treatment directed at HP infection. In many instances, pneumatic dilation is used primarily, and frequently repeatedly, before consideration for surgical referral.

Pyloric dilation for benign peptic stricture was first reported in 1982 and continues to be used, often as primary therapy. Its use, however, suffers from a lack of published data, especially with respect to long-term symptomatic improvement. Early studies focused primarily on the feasibility, safety, and short-term results of this technique. Studies with longer periods of follow-up have yielded conflicting results. With a median follow-up of 23 months, Perg et al noted sustained symptomatic improvement in 28 of 42 patients treated with pyloric dilation. Disario et al reported sustained relief in 24 of 30 patients, but 7 required repetitive dilation sessions. Kuwada and Alexander reported the longest follow-up to date with a median of 45 months. In their series, 16 of 19 patients initially treated successfully with pyloric dilation had a recurrence of symptoms at a median of 9 months following the index procedure. Similarly, Lau et al noted a recurrence of obstruction in 18 of 41 patients at a median of 39 months. These data would suggest that pyloric dilation may offer excellent initial symptomatic relief. However, one can expect an unacceptably high rate of recidivism in this population. Certainly, long-term antacid use is a necessary adjunct that would be costly over the lifetime of a young patient. The impact of antacid use on the prevention of recurrent GOO, however, remains questionable. In a study from the University of Tennessee, 3 patients underwent successful pyloric dilation followed by antacid therapy. All 3 patients eventually developed restenosis and underwent surgical management. Similar results were noted by Lam in a subsequent study. Morbidity in the form of perforation has been cited in 0% to 6% of patients undergoing this procedure, necessitating emergency operation with no reported mortality to date. One must also consider the surgical ramifications should pyloric dilation fail. Management of a difficult duodenal stump could be more problematic because of advanced fibrosis from 1 or more prior pneumatic dilations. In addition, failed pyloric dilation can lead to further weight loss and a high-risk patient for surgical intervention because of nutritional depletion.

Surprisingly, despite these recent reports on the use of pyloric dilation in those with benign GOO, the incidence of HP infection in this cohort was infrequently assessed. As such, its potential role in recurrent stricture formation in those with an initially successful pyloric dilation is unknown. Further, the impact on its eradication on the nonoperative management of benign peptic stricture can only be speculated. Only 19 of 42 patients in the series of pyloric dilation reported by Perg et al had concurrent biopsy performed for HP infection. Of these 19 patients, only 11 (57%) tested positive for HP infection. Five of these 11 patients were treated for HP infection following pyloric dilation, and all 5 remained asymptomatic at a median follow-up of 15 months. Another 5 of these 11 patients who tested positive for HP infection ultimately required surgical intervention. Unfortunately, it is unclear whether these patients were treated for HP infection or not. Gibson et al reported an HP infection incidence of only 33% in 24 patients undergoing surgery for GOO. In that series, 2 of 5 patients receiving pneumatic dilation preoperatively tested positive for HP infection though none received treatment. As previously noted, in the other 3, pyloric dilation failed, despite the concurrent use of antacid therapy. Very limited data suggest that treatment for active HP infection is an important adjunct to pyloric dilation if surgery is to be avoided. In a series of 19 patients reported by Lam, 9 (47%) had active HP infection. Six of these 9 patients received both antimicrobial therapy directed at HP infection and concurrent dilation. No recurrent obstruction was noted in these 6 patients at a median of 16 months.

Case reports have noted total resolution of symptomatic and endoscopic outlet obstruction with medical treatment directed at HP infection without concurrent pyloric dilation. However, no large series has confirmed this mode of therapy. In the series by Gibson et al, 3 patients received HP infection therapy alone without dilation, and this was unsuccessful in avoiding operation. Although medical treatment may eradicate an acute inflammatory process, its success in resolving a chronic fibrotic process noted by surgeons at the time of operative intervention would seem doubtful.

Surgery for benign GOO remains an important treatment modality either as initial therapy or following unsuccessful pyloric dilation. Options for treatment include highly selective vagotomy with some form of pyloroplasty, truncal vagotomy with gastroenterostomy, or truncal vagotomy with antrectomy. All have been reported with good results. While vagotomy and antrectomy may offer the lowest rate of ulcer recurrence, other modalities remain important, especially in those with significant duodenal scarring. Short- and long-term morbidity have been rare in reported series. Symp-
tomatic control following surgical intervention remains outstanding. In the series by Gibson et al,22 all but 1 of 16 patients followed up long-term after surgery reported Visick grade I or II symptoms (improved from Visick IV), and no patients required antacid medication.

Nonoperative management of benign GOO is hampered by a lack of long-term follow-up data. The incidence of HP infection in this population appears to be substantially less than in populations with uncomplicated ulcers and even less common than in those with perforated or massively bleeding ulcers. In patients with GOO and active HP infection, pyloric dilation combined with medical therapy directed at HP infection seems reasonable, but one must recognize that no substantial data as yet exist to substantiate this mode of therapy. In patients who test negative for HP infection, long-term success with pyloric dilation as reported in the literature remains poor, especially in the absence of permanent antacid use. Surgical therapy for this ulcer complication continues to offer definitive and durable symptomatic relief in this patient population.

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