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The Management of Patients Who Have "Failed" Antireflux Surgery

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Gastroenterologists may be called upon to manage patients who have had antireflux surgery that failed. The available literature on this topic comprises predominantly reports on retrospective, observational studies written by surgeons who often have focused on how technical deficiencies in performing the operation led to the failure. Such reports are of limited value to the gastroenterologist seeking guidance on patient management. Furthermore, comparisons among the reports are confounded by the lack of a standardized definition for failed antireflux surgery. This report critically reviews the available literature, and suggests a practical approach to the management of patients who have symptoms that were not completely relieved, that reappeared later, or that were caused by antireflux surgery.

Medical therapy for gastroesophageal reflux disease (GERD) focuses on suppressing gastric acid secretion, but does little to correct the anatomical and physiological abnormalities that predispose to gastroesophageal reflux (1). In contrast, antireflux surgery attempts to correct the reflux diathesis by returning the gastroesophageal junction to its normal anatomical location within the abdomen, and by creating a barrier to reflux through fundoplication and through repair of the diaphragmatic crurae that surround the distal esophagus (2). Both medical and surgical therapies can be highly successful in healing the symptoms and signs of GERD, but both occasionally fail. This report deals with the management of patients with failed antireflux surgery.

A MEDLINE search of reports published in English since 1966 using the search strategy "(fundoplication or antireflux) and (failed or failure or unsuccessful)" yielded 235 articles. A review of these articles and their references revealed 44 reports dealing primarily with the evaluation and/or management of failed antireflux surgery (excluding reports that focused exclusively on postoperative dysphagia) (3–47). There were no reports of randomized, controlled therapeutic trials. The available literature comprised predominantly reports on retrospective, observational studies. Furthermore, most of the reports are written by surgeons who focused primarily on how technical deficiencies in the performance of the operation may have led to failure. Such reports are of limited value to the gastroenterologist who must treat patients who have failed fundoplications.

One fundamental problem that confounds comparisons among these reports is the lack of a standardized definition for failed antireflux surgery. Fundoplications can "fail" in any number of ways. For example, the surgery can fail to control the primary GERD manifestation for which the operation was prescribed. Most commonly that manifestation is heartburn, but fundoplication also is used to control extraesophageal GERD manifestations like asthma and laryngitis, or atypical GERD symptoms like noncardiac chest pain. Few would argue that the operation is a failure if the primary symptom is unchanged or worse after surgery. But what if the symptom is still present, only improved to the point that it is more tolerable or more readily controlled with medication? Is that a failure or an incomplete success?

Another frequently used definition of failure includes the appearance of new symptoms such as dysphagia, bloating, or diarrhea after surgery. If the operation has eliminated the primary complaint, one could argue that the new symptom should be deemed a complication rather than a failure. Timing is also an issue in defining surgical failures. Symptoms can occur early or late after fundoplication. Should the operation be considered a failure if the patient had days, weeks, months, or years of relief before symptoms appeared or recurred?

Although these semantic issues may confound comparisons among reports, their subtleties are likely to be lost on the patient who has disabling symptoms following a fundoplication. Such a patient probably will not be comforted by reassurance that his or her symptoms do not meet the criteria for a surgical failure. This report does not advocate any specific, arbitrary definition for failed antireflux surgery. Rather, the report suggests a practical approach to the management of patients who have symptoms that were not completely relieved, that reappeared later, or that were caused by antireflux surgery.

SYMPTOMS INCOMPLETELY RELIEVED OR REAPPEARING LATER AFTER ANTIREFLUX SURGERY

There are two major categories for symptoms that either were incompletely relieved or reappeared later after antireflux

surgery: (1) Symptoms that are due to gastroesophageal reflux that was not eliminated by the operation, and (2) symptoms that are not due to gastroesophageal reflux. The distinction between these categories often can be made on the basis of a careful history and endoscopic examination. In some cases, a barium contrast study (upper gastrointestinal [UGI] series), esophageal pH monitoring study, and esophageal manometry also may be needed.

History

It is important to ascertain specifically what is the symptom that has not been relieved by fundoplication, especially for patients who complain of persistent heartburn. Patients often do not understand the meaning of the term "heartburn," and they may use it to describe a variety of symptoms that are related only indirectly or not at all to gastroesophageal reflux (48). GERD frequently is associated with functional symptoms that may not be due to the reflux of gastric contents (49). In the VA cooperative study of medical and surgical therapies for GERD, for example, approximately 50% of the medically treated patients complained of abdominal distention and fullness during a 1-yr follow-up period (50). Such complaints are more likely to be epiphenomena unrelated to GERD, or manifestations of the poorly defined gut motility disorder that predisposes to GERD, rather than symptoms caused directly by gastric juice refluxing into the esophagus. Nevertheless, some patients refer to these functional symptoms as "heartburn."

Patients who are most likely to respond well to antireflux surgery are those who have GERD symptoms that respond well to medical antireflux therapy, and who have pathological acid reflux demonstrated by pH monitoring (51). However, symptoms that were not due to gastroesophageal reflux in the first place are unlikely to be relieved by antireflux surgery. For the patient who complains of postoperative heartburn, therefore, the physician should ask for a specific description of the sensation, and should ask whether the sensation differs from that experienced preoperatively. Such a careful history will help to distinguish an operation that failed for technical reasons from a failure of preoperative diagnosis.

Endoscopy

For patients with symptoms after fundoplication, endoscopy can answer several important questions: (1) Is there reflux esophagitis? The presence of reflux esophagitis after antireflux surgery provides objective evidence that the operation has not controlled GERD (2). Is there another lesion that can explain the symptoms? The finding of a gastric or duodenal ulcer, for example, might explain new symptoms of abdominal pain. (3) Does the fundoplication appear to be anatomically correct? In this regard, the endoscopist should attempt to ascertain the location, orientation, and span of the fundoplication, and the presence of paraesophageal herniation.

The two most commonly used fundoplication procedures (Nissen and Toupet) create characteristic folds in the proximal stomach that usually can be seen best with the endoscope in the retroflexed position (Figs. 1 and 2) (34, 52). The folds of the fundoplication should be located just below the diaphragm. If the folds are seen above the diaphragm, it is an indication that the fundoplication has herniated into the

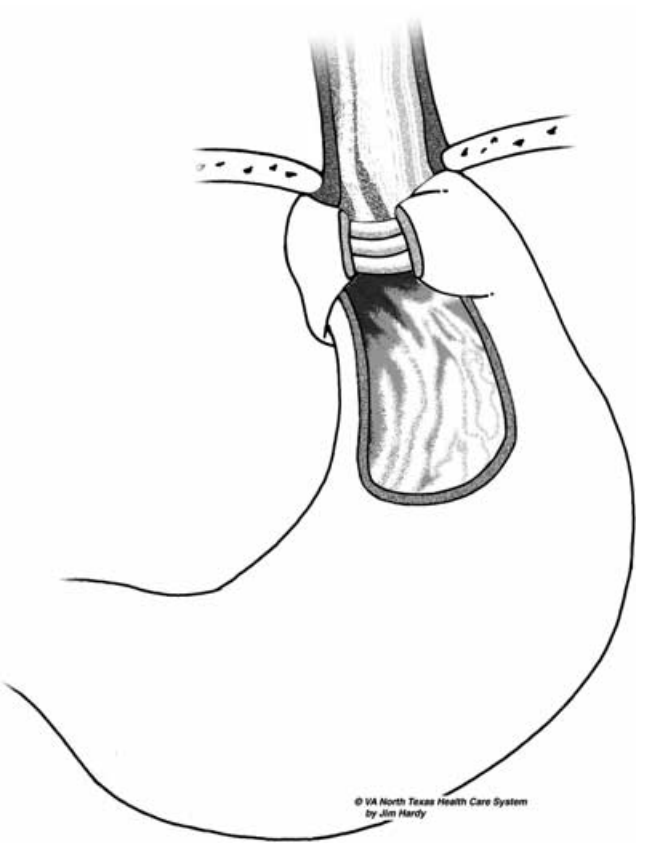

Figure 1. An anatomically correct fundoplication. Note that the folds caused by the fundoplication are located just below and parallel to the diaphragm. The folds should measure approximately 1–2 cm in span.

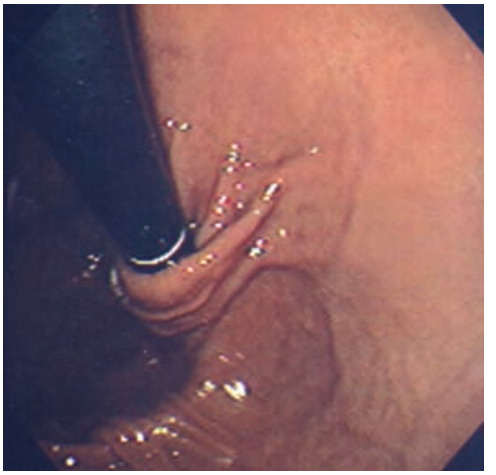

Figure 2. Endoscopic photograph of an anatomically correct Nissen fundoplication, retroflexed view. The fundoplication folds are located below the diaphragm, and the folds run parallel to the white distance line on the endoscope. (Photograph provided by Dr. David Johnson.)

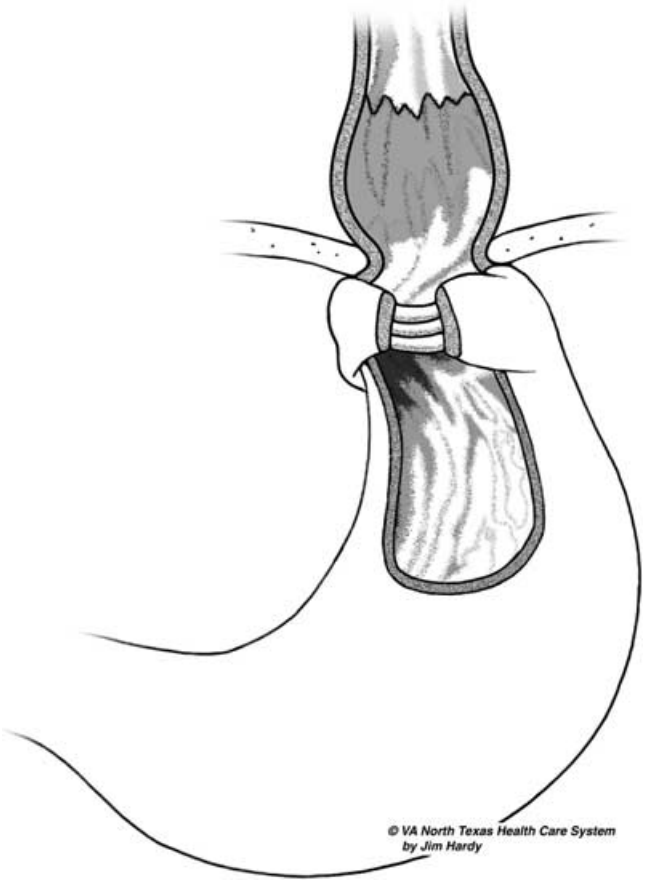

Figure 3. A slipped fundoplication. There is a pouch of stomach proximal to the fundoplication folds. A slipped fundoplication can occur in two ways: (1) The fundoplication is fashioned in the correct location, but a portion of the stomach later herniates ("slips") through the fundoplication; or (2) the surgeon mistakes the proximal stomach for the distal esophagus, and inadvertently fashions the fundoplication around the stomach.

chest, which usually results from disruption of the crural repair (28). If there is a pouch of stomach proximal to the folds of the fundoplication, the condition is called a "slipped" fundoplication (*e.g.*, a "slipped Nissen") (Fig. 3). A slipped fundoplication can occur in two ways: (1) The fundoplication is fashioned in the correct location, but a portion of the stomach later herniates ("slips") through the fundoplication; or (2) the surgeon mistakes the proximal stomach for the distal esophagus, and inadvertently fashions the fundoplication around the stomach. Although the latter situation represents an initial surgical error rather than a later slippage (herniation), the condition is called a slipped fundoplication despite the misnomer. Finally, the absence of fundoplication folds suggests total disruption of the antireflux procedure (the "missin' Nissen"). Any of these abnormalities can render the antireflux surgery ineffective.

The folds of a properly constructed fundoplication should be oriented parallel to the diaphragm. An oblique orientation of the folds suggests twisting of the fundoplication, or improper construction of the wrap using the body rather than

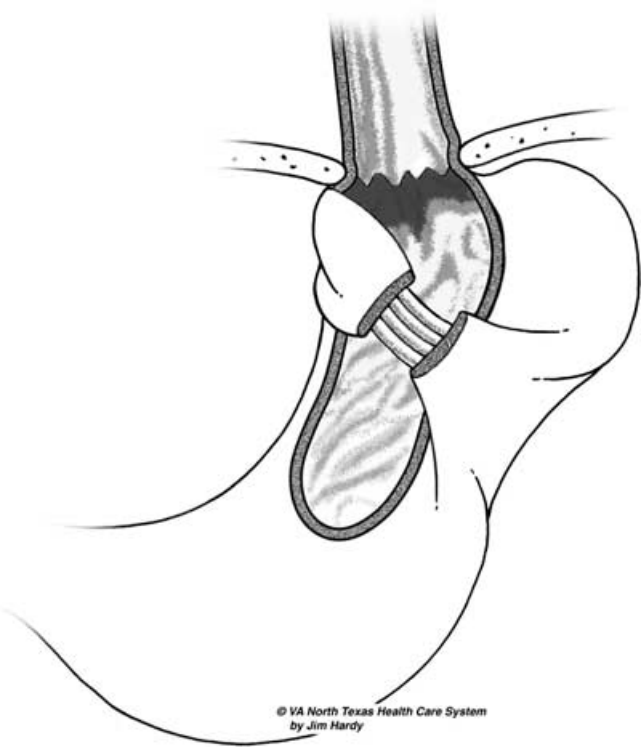

Figure 4. Obliquely oriented fundoplication folds. This can be caused by herniation of the wrap or by improper construction of the wrap using the body rather than the fundus of the stomach.

the fundus of the stomach (Figs. 4 and 5) (34). Either of these conditions can cause postoperative gastroesophageal reflux, dysphagia, or both. The folds should measure approximately 1 to 2 cm in span. A wider span indicates a too-generous fundoplication that can cause dysphagia. A paraesophageal

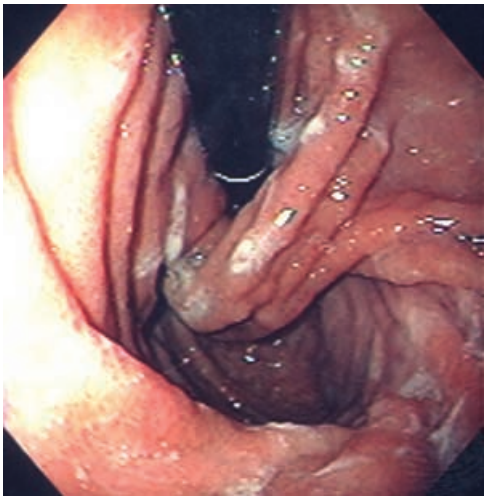

Figure 5. Endoscopic photograph of a slipped Nissen fundoplication, retroflexed view. The fundoplication folds are oriented obliquely to the white distance line on the endoscope, and there is a pouch of stomach proximal to the folds. (Photograph provided by Dr. David Johnson.)

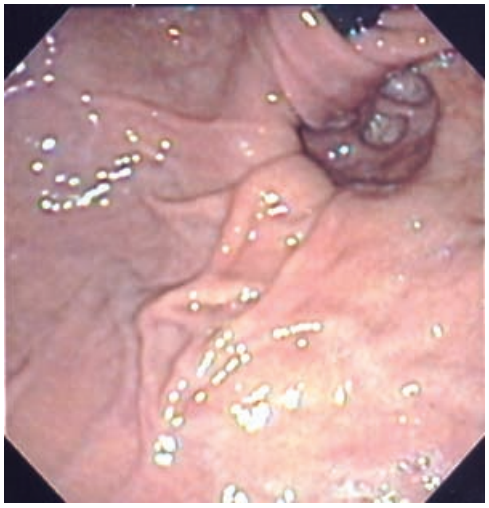

Figure 6. Endoscopic photograph of a paraesophageal hernia, retroflexed view. The herniated pouch of stomach is located next to the fundoplication folds. (Photograph provided by Dr. David Johnson.)

hernia also can cause dysphagia by pressing on the distal esophagus (Fig. 6). The herniated portion of the stomach in these cases usually originates from the fundoplication itself, and may result from attempts to construct a "floppy" wrap (28).

UGI Series

Endoscopic examination clearly is more sensitive than barium contrast radiography for identifying mucosal lesions of the esophagus like reflux esophagitis (53). However, paraesophageal hernias and anatomical relationships among organs may be better appreciated by UGI series than by endoscopy. If the endoscopist suspects paraesophageal herniation, or has any question about the anatomy of the repair, an UGI series often can help to define the anatomy. For patients with postfundoplication dysphagia, the radiologist also can provide an assessment of the efficacy of esophageal emptying.

Esophageal pH Monitoring

Ambulatory monitoring of esophageal pH can document the pattern, frequency, and duration of acid reflux after fundoplication, and can establish the association between episodes of acid reflux and symptoms (54). An esophageal pH monitoring study can help to establish that postoperative symptoms are due to acid reflux. The demonstration of normal esophageal acid exposure and lack of symptom correlation with reflux episodes strongly suggest that the postoperative complaints are not caused by persistent acid reflux. Alternatively, the demonstration of abnormal acid reflux and the correlation of symptoms with reflux episodes establish that the operation has failed at its primary goal.

Esophageal Manometry

Esophageal manometry can be useful to document whether there is an esophageal motility abnormality that may be contributing to the postfundoplication symptoms. Esophageal manometry is most likely to be helpful for the evaluation of postoperative dysphagia (see below).

Symptoms due to Persistent Gastroesophageal Reflux after Fundoplication

If the above procedures indicate that the postfundoplication symptoms are due to persistent gastroesophageal reflux, then the two major therapeutic options are medical antireflux therapy or surgical revision of the fundoplication. Perhaps endoscopic antireflux procedures may be used for this indication in the future, but presently there is little published information on endoscopic therapy for patients with failed antireflux surgery. Proton pump inhibitors (PPIs) are highly effective antisecretory agents (1) and, in theory, postfundoplication symptoms that are due to acid reflux should respond well to PPI therapy. Few data are available specifically on the efficacy of PPIs in patients with failed antireflux surgery, however, and it is difficult to provide a precise estimate on how often PPI therapy will be successful in this setting. Nevertheless, if symptoms appear to be due to persistent acid reflux, then a trial of PPI therapy is warranted. If the symptoms are well controlled by PPI therapy, reoperation may not be necessary.

A number of reports have dealt specifically with the results of reoperation for patients with failed antireflux surgery (4, 6, 8, 9, 11, 12, 14–16, 19–25, 27–31, 33, 36–43). The primary reason for reoperation in these series was persistent gastroesophageal reflux (manifested by heartburn and/or regurgitation) in approximately 50% of patients, dysphagia in approximately 30%, and the combination of persistent reflux and dysphagia in approximately 20% patients. Although none of these reports can be considered definitive, there is a consensus among the authors on several issues regarding reoperation for failed antireflux surgery: (1) Reoperation is more challenging technically than the primary repair. (2) The success rate for symptom relief after reoperation (70–85%) is somewhat lower than that reported for the primary repair. (3) The success rate appears to decrease with subsequent reoperations. In one series, for example, good-to-excellent results were found in 85% of patients who had one reoperation, 66% of patients who had two reoperations, and only 42% of patients who had three or more reoperations (8). (4) The mortality rate is higher than that for the primary repair. For the first antireflux surgery, the mortality rate is approximately 0.1–0.3% (55–57). For reoperation, the mortality rate appears to be approximately 1% (34).

A number of surgical authorities advocate reoperation for patients who have symptomatic gastroesophageal reflux that clearly is due to a correctable, technical failure of antireflux surgery. However, there is no compelling reason to choose reoperation over continued medical therapy if that therapy is controlling the symptoms and signs of GERD. There are no randomized trials showing that one form of therapy is

superior to another in this setting, and there are substantial hazards associated with reoperation.

For patients who are interested in another antireflux operation, it is important to clarify specifically what are the goals that they are trying to achieve with this treatment. For example, is the patient hoping to get relief from troublesome symptoms that are not responding to medical therapy? If that is the case, then before recommending another procedure the physician should ascertain that the unresponsive symptoms are in fact due to reflux, and are likely to respond to fundoplication. Patients who have atypical symptoms that persist despite adequate PPI therapy may have functional disorders or other problems unlikely to respond to procedures designed to prevent reflux. Some patients request surgery because they think it will prevent esophageal cancer (58). Such patients should be counseled regarding their true risk for this cancer, and should be informed that a cancer-preventive role has not been established for any GERD treatment. Patients who request reoperation because they find it inconvenient to take antisecretory medications should be informed of recent data suggesting that many patients continue to take these medications after antireflux surgery (58, 59).

SYMPTOMS CAUSED BY ANTIREFLUX SURGERY

Dysphagia

Approximately 50% of patients experience dysphagia immediately after fundoplication, presumably as a consequence of the edema and inflammation caused by the surgery (60, 61). These patients are treated with dietary modifications and reassurance, and the dysphagia usually resolves spontaneously within 2–3 months. However, some 3% to 24% of patients experience dysphagia that persists beyond 3 months, or that is so severe as to require treatment beyond dietary modifications (60, 61).

Table 1 lists the causes of persistent or severe postfundoplication dysphagia. Patients with peptic esophageal strictures may have dysphagia that does not improve after fundoplication, especially if the operation is not successful in controlling reflux esophagitis (26). Indeed, even in the absence of peptic stricture, severe reflux esophagitis can cause dysphagia (62). Endoscopy can establish whether there is an esophageal stricture or inflammation that might be contributing to postfundoplication dysphagia. If there is reflux esophagitis, a trial of PPI therapy is warranted. If the dysphagia resolves with PPI therapy, no additional treatment may be required. If there is a peptic stricture, dilation therapy should be performed.

Table 1. Causes of Persistent Post-Fundoplication Dysphagia

GERD-related
Peptic esophageal stricture
Recurrent reflux esophagitis
Mechanical obstruction resulting from fundoplication
Fundoplication too long or too tight
Slipped fundoplication
Paraesophageal hernia
Esophageal motility disorder
Ineffective esophageal motility
Achalasia missed preoperatively
Achalasia developed postoperatively

If the endoscopy or barium swallow suggests that the fundoplication has resulted in a mechanical obstruction of the esophagus, a trial of dilation therapy usually is warranted (60, 61). Mechanical obstruction of the esophagus can result if the fundoplication is too long or too tight, if the diaphragmatic crural repair constricts the esophagus, or if there is an inflammatory reaction to mesh or other foreign materials that are occasionally used in the repair. With such mechanical obstructions, dilation can relieve the dysphagia in one-half to two-thirds of cases. Among patients for whom dilation therapy is successful, approximately 75% require only one session of dilation therapy. Most of the published experience involves esophageal dilation with mercury-filled rubber or polyvinyl bougies, with patients dilated to a mean diameter of approximately 18 mm (54 French gauge). The available reports of small series describe no complications from esophageal bougienage for postfundoplication dysphagia, even when the dilations were performed within the first week after surgery (60, 61).

For patients who have dysphagia due to a slipped fundoplication or paraesophageal herniation, a number of authorities advise that dilation therapy usually will fail and that reoperation will be necessary in most cases (28). Indeed, the reported success rate for dilation therapy in this setting is less than 30% (60). However, in the absence of randomized therapeutic trials comparing dilation and reoperation, the relative efficacy of the procedures in relieving dysphagia is not clear. The failure rate for nonoperative therapy may be exaggerated due to a number of biases that affect the outcome of observational studies. For example, if physicians start dilation therapy with the bias that it will not work, then they will be predisposed to abandon the treatment early in favor of operative therapy (self-fulfilling prophecy).

Manometric studies on patients with severe GERD often exhibit evidence of ineffective esophageal motility in which some wet swallows elicit low-amplitude peristaltic waves, low-amplitude simultaneous contractions, failed peristaltic sequences, or no contractions in the esophagus (63, 64). For such patients, antireflux surgeons traditionally have advocated the performance of a "loose wrap" such as a Toupet fundoplication to prevent the postoperative dysphagia that might result from a "tight wrap" such as a conventional Nissen fundoplication. Although a recent randomized trial found that such preoperative motility abnormalities had no effect on the frequency of postoperative dysphagia, irrespective of the type of fundoplication performed (65), ineffective esophageal motility undoubtedly can contribute to postfundoplication dysphagia. Nevertheless, the finding of ineffective esophageal motility on manometric examination has no clear therapeutic implications for the patient with postfundoplication dysphagia. Prokinetic agents such as

metoclopramide have not been shown to be helpful in this setting, and a trial of dilation therapy usually is warranted with the rationale that even a minimal mechanical obstruction might contribute to dysphagia in patients with abnormal esophageal motility. One group has suggested that esophageal resection and reconstruction by colonic or jejunal interposition is preferable to another antireflux operation for patients who have postfundoplication dysphagia associated with severe ineffective esophageal motility (26). The evidence to support such an aggressive and potentially hazardous treatment is very limited, however.

Achalasia is an important diagnosis to consider in patients with postfundoplication dysphagia, especially if no fixed mechanical obstruction is apparent by endoscopy or barium swallow, and the response to conventional dilation therapy with bougies or through-the-scope balloons is poor. Manometrically, classical achalasia is characterized by incomplete relaxation of the lower esophageal sphincter (LES) with swallowing (*i.e.*, swallowing causes LES pressure to fall to a nadir value that remains >8 mmHg above gastric pressure), and aperistalsis in the body of the esophagus (64). There are also a number of atypical disorders of LES relaxation (achalasia variants) in which swallowing causes incomplete relaxation of the LES, but some peristalsis is preserved. These atypical disorders of LES relaxation can behave like achalasia clinically, and in their response to treatments aimed at reducing residual LES pressure.

Approximately one-third of patients with achalasia have heartburn that can be indistinguishable from that of GERD (66). Such patients may be misdiagnosed as having GERD, and may be referred for fundoplication when they have a poor response to medical antireflux therapy. Fundoplications performed on patients with achalasia mistakenly diagnosed as GERD can result in severe dysphagia. Such a mistake is especially likely if esophageal manometry is not performed before the fundoplication (67).

Both the atypical and classical forms of achalasia have been reported to develop after, and presumably as a consequence of, antireflux surgery. In one study of 103 patients who had esophageal manometry before and after laparoscopic Nissen fundoplication, the mean residual pressure after LES relaxation was found to increase from 1.2 mmHg preoperatively to 10.8 mmHg after surgery (68). Pursnani *et al*. described similar results on residual LES pressure in 20 patients who had pre- and postoperative esophageal motility studies (69). Thus, antireflux surgery can create the functional equivalent of an atypical disorder of LES relaxation. In another report of 250 patients who had laparoscopic Nissen fundoplications and preoperative esophageal motility studies showing no achalasia, seven patients (3%) developed achalasia postoperatively (defined as complete disappearance of esophageal peristalsis on postoperative manometry or fluoroscopy with or without incomplete LES relaxation) (70). Unlike patients with dysphagia due to the mechanical effects of fundoplication (whose symptoms appear early after surgery), the patients with postfundoplication achalasia in this series did not develop severe dysphagia until a mean of 135 days postoperatively. In contrast, Ellingson *et al*. described nine patients who developed manometrically confirmed, classical achalasia after fundoplication, all of whom experienced dysphagia in the immediate postoperative period (71). These reports all suggest that fundoplication can cause classical or atypical achalasia, but the mechanism is not known. It is not clear whether the incomplete relaxation of the LES results from injury to the innervation of the sphincter or from mechanical effects of the operation.

Although no therapy for achalasia can reliably restore esophageal peristalsis, dysphagia often responds well to treatments that reduce residual LES pressure through pharmacological or mechanical means (*e.g.*, botulinum toxin injection, pneumatic dilation, Heller myotomy) (72). In the aforementioned series of seven patients with achalasia that developed after Nissen fundoplication, three improved when they were treated with botulinum toxin injection, and one improved after Heller myotomy (70). In contrast, neither of the two patients who were treated with another antireflux procedure had improvement in their dysphagia. In a series of 14 patients who had pneumatic dilation using 30 mm to 40 mm diameter balloons for postfundoplication dysphagia that did not respond to bougienage, 9 patients (64%) had a good response and there were no serious complications (73). The nadir LES relaxation pressure correlated with success for pneumatic dilation in this study. All six patients whose predilation nadir LES pressure was ≥ 10 mmHg had a good response to the procedure. Pneumatic dilation was successful in five of nine patients (56%) with postfundoplication achalasia in another series, but one patient had a balloon-induced perforation that required surgical repair (71). These and other studies suggest that pneumatic dilation can be effective in patients who have postfundoplication dysphagia with manometric features of incomplete LES relaxation (74). The perforation rate does not appear to differ substantially from that for patients with achalasia who have not had antireflux surgery, but experience with pneumatic dilation in the postfundoplication setting is limited.

Gas-Bloat Syndrome

The gas-bloat syndrome comprises an ill-defined and variable constellation of symptoms that have been assumed to result from the inability to vent gas from the stomach into the esophagus after fundoplication (13). In addition to the sensation of bloating, which is itself poorly defined, other features that variably have been considered part of the gas-bloat syndrome include abdominal distention, early satiety, nausea, upper abdominal pain, flatulence, inability to belch, and inability to vomit. In a number of reports that have dealt with gas-bloat syndrome, furthermore, the authors have not even provided a specific definition for the disorder. The cause of the syndrome is not clear, but proposed mechanisms include: (1) inability of the surgically altered GEJ to relax in response to gastric distention (13); (2) aerophagia, a frequent habit of patients with severe GERD, that continues in the postoperative period

when the air cannot easily be vented (75); (3) impairment of meal-induced receptive relaxation and accomodation of the stomach with accelerated gastric emptying (76); and (4) surgical injury to the vagus nerves, which delays gastric emptying and interferes with the transient LES relaxation that is part of the normal belch reflex (77–79).

The reported frequency of gas-bloat syndrome has ranged widely, probably due to differences in the definition of the disorder as well as to differences among patients and surgical techniques. In one recent study of 84 patients who responded to telephone questionnaires about their antireflux operations, 16 (19%) described the new onset of bloating after surgery (80). In the VA trial of medical and surgical therapies for GERD, both the medical and surgical patients were questioned for symptoms of gas-bloat syndrome at their quarterly clinic visits (50). During the first year of the study, 81% of the surgical patients had at least one symptom of the gas-bloat syndrome. However, 60% of the medically treated patients also had at least one of those symptoms. The symptoms generally were mild in both treatment groups, and their presence did not preclude satisfaction with therapy. In the large majority of cases in published series, furthermore, it appears that the symptoms of gas-bloat syndrome resolve spontaneously within six months (13).

Authorities recommend that patients with mild gas-bloat syndrome should be treated with: (1) dietary modifications to avoid gas-producing foods, (2) gas-reducing agents such as simethicone, (3) prokinetic agents such as metoclopramide, (4) advice to avoid aerophagia, and (5) reassurance that the symptoms usually resolve spontaneously within months (13). There is no convincing evidence that any of these measures are effective in relieving the symptoms of gas-bloat syndrome, however. The Hill antireflux procedure, in which the surgeon anchors the lower esophageal sphincter within the abdomen and creates a flap-valve mechanism to prevent reflux, appears to have a very low incidence of gas-bloat syndrome. Some patients with severe gas-bloat have been treated successfully by reoperation in which the original repair was taken down, and a Hill posterior gastropexy was performed (81). Such invasive therapy rarely should be needed, and should be initiated only after a reasonable trial of conservative measures has failed, and only after small bowel obstruction and delayed gastric emptying have been excluded (see below).

For patients with debilitating gas-bloat after antireflux surgery, it is important for the clinician to differentiate gas-bloat syndrome from a mechanical small bowel obstruction (*e.g.*, one caused by adhesions), and from delayed gastric emptying due either to peptic ulceration that causes gastric outlet obstruction or to vagal injury that impairs gastric motility. Antireflux surgery in adults uncommonly results in adhesions that cause small bowel obstruction, although children appear to be susceptible to this phenomenon (82). Radiographic tests such as computed tomography or enteroclysis may occasionally be necessary to exclude a mechanical small bowel obstruction in a patient with severe gas-bloat syndrome. Endoscopy can identify peptic ulceration causing mechanical obstruction of the gastric outlet. It may not be so easy to exclude delayed gastric emptying due to vagal injury in patients with severe gas-bloat syndrome, however.

Antireflux surgery usually accelerates the gastric emptying of both liquids and solids (75, 83). Vagotomy, in contrast, can delay gastric emptying of solids by interfering with antral motility and pyloric relaxation (78, 84). Therefore, vagal injury during fundoplication might cause delayed gastric emptying with symptoms similar to those of gas-bloat syndrome. Radionuclide tests that can document delayed gastric emptying are widely available (85), and the demonstration of delayed gastric emptying by these tests in a patient with severe symptoms of gas-bloat suggests that vagal injury could be contributing to the problem. This finding might have therapeutic implications, because it suggests a role for treatments aimed at relaxing the pylorus such as pyloroplasty or botulinum toxin injection. A number of functional tests are available to document the integrity of the gastric vagal innervation (*e.g.*, the Congo red test, sham-feeding with gastric acid analysis, sham-feeding with measurement of plasma levels of pancreatic polypeptide) (86, 87). However, these tests are not performed widely, and their clinical utility in identifying patients who would benefit from procedures such as pyloroplasty and botulinum toxin injection has not been established.

For symptomatic patients with delayed gastric emptying documented by radionuclide tests, treatment can begin with the prescription of frequent, small-volume meals that are low in fat and fiber. Prokinetic agents such as metoclopramide and erythromycin have been recommended, although there is little evidence that these medications are useful in patients with delayed gastric emptying after fundoplication. The management of patients who have troubling symptoms despite these measures is disputed. Nutrition can be supported using jejunostomy tubes or parenteral nutrition, but these are unattractive, long-term therapeutic options. The insertion of percutaneous endoscopic gastrostomy (PEG) tubes for "venting" has been tried, but there is no clear documentation for the success of this procedure (13, 88). Furthermore, PEG tube insertion can be complicated by leakage and infection, and the procedure causes adhesions between the stomach and abdominal wall that might complicate subsequent surgical attempts to revise the fundoplication. Simple revision of the fundoplication alone is not likely to improve gastric emptying that has been compromised by vagal injury. A gastric drainage procedure such as pyloroplasty or gastroenterostomy might be indicated if the symptoms clearly are due to antropyloric dysfunction caused by vagal injury. However, no preoperative test has been shown to predict reliably which patients will benefit from such invasive therapy. In this situation, the clinician can consider a trial of botulinum toxin injection into the pyloric sphincter muscle, even though only very limited observational data support this practice.

In one report of three patients with severe gastroparesis, one of whom had symptoms develop after Nissen

fundoplication, the injection of 200 U of botulinum toxin into the pylorus resulted in symptomatic improvement in all three with no complications (89). In another report of 10 patients with severe gastroparesis, one of whom had symptoms develop after Toupet fundoplication, the injection of 80–100 U of botulinum toxin into the pylorus resulted in significant improvements in symptoms and gastric emptying without complications (90). Clearly, further study is needed to determine the precise efficacy and safety of this procedure. However, this therapy appears to be reasonably safe, the rationale for its use is plausible, and limited data suggest that some patients will benefit. For patients with documented, severe gastroparesis after fundoplication for whom conservative measures have failed, therefore, a trial of botulinum toxin injection seems warranted before considering more invasive therapies.

Diarrhea

Diarrhea is a frequent complication of antireflux surgery. In a study of 84 patients who responded to a telephone questionnaire about their antireflux operations, 15 (18%) described the new onset of diarrhea after surgery (91). The diarrhea developed within 6 wk of the operation in 10 of those 15 patients. In four cases, the diarrhea was associated with fecal incontinence. Diagnostic testing for the cause of diarrhea was not performed routinely, but no etiology other than fundoplication was found in the seven patients who had such testing. Within a 2-yr follow-up period, only 2 of the 15 patients (13%) had complete resolution of their diarrhea. Other reports have described even higher rates of postfundoplication diarrhea (up to 33%), but those studies did not describe whether diarrhea was present before the operation (91, 92). The cause of postfundoplication diarrhea is not known. Suggested mechanisms include (1) accelerated gastric emptying, (2) vagal injury, and (3) postoperative dietary modifications. There is no specific treatment for patients with postfundoplication diarrhea, and their management is empirical.

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REFERENCES

1. DeVault KR, Castell DO, and The Practice Parameters Committee of the American College of Gastroenterology. Updated guidelines for the diagnosis and treatment of gastroesophageal reflux disease. Am J Gastroenterol 1999;94:1434–42.
2. Hinder RA, Libbey JS, Gorecki P, et al. Antireflux surgery: Indications, preoperative evaluation, and outcome. Gastroenterol Clin North Am 1999;28:987–1005.
3. Polk HC Jr. Fundoplication for reflux esophagitis: Misadventures with the operation of choice. Ann Surg 1976;183:645–52.
4. Leonardi HK, Crozier RE, Ellis FH Jr. Reoperation for complications of the Nissen fundoplication. J Thorac Cardiovasc Surg 1981;81:50–6.
5. Leonardi HK, Ellis FH Jr. Complications of the Nissen fundoplication. Surg Clin North Am 1983;63:1155–65.
6. Hatton PD, Selinkoff PM, Harford FJ Jr. Surgical management of the failed Nissen fundoplication. Am J Surg 1984;148:760–3.
7. Hatfield M, Shapir J. The radiologic manifestations of failed antireflux operations. AJR 1985;144:1209–14.
8. Little AG, Ferguson MK, Skinner DB. Reoperation for failed antireflux operations. J Thorac Cardiovasc Surg 1986;91:511–7.
9. Stirling MC, Orringer MB. Surgical treatment after the failed antireflux operation. J Thorac Cardiovasc Surg 1986;92:667–72.
10. Low DE, Mercer CD, James EC, et al. Post Nissen syndrome. Surg Gynecol Obstet 1988;167:1–5.
11. Siewert JR, Isolauri J, Feussner H. Reoperation following failed fundoplication. World J Surg 1989;13:791–6.
12. Martin CJ, Crookes PF. Reoperation for failed antireflux surgery. Aust N Z J Surg 1990;60:773–8.
13. Low DE. Management of the problem patient after antireflux surgery. Gastroenterol Clin North Am 1994;23:371–89.
14. Rieger NA, Jamieson GG, Britten-Jones R, et al. Reoperation after failed antireflux surgery. Br J Surg 1994;81:1159–61.
15. Siewert JR, Stein HJ, Feussner H. Reoperations after failed antireflux procedures. Ann Chir Gynaecol 1995;84:122–8.
16. DePaula AL, Hashiba K, Bafutto M, et al. Laparoscopic reoperations after failed and complicated antireflux operations. Surg Endosc 1995;9:681–6.
17. Stein HJ, Feussner H, Siewert JR. Failure of antireflux surgery: Causes and management strategies. Am J Surg 1996;171:36–40.
18. Dallemagne B, Weerts JM, Jehaes C, et al. Causes of failures of laparoscopic antireflux operations. Surg Endosc 1996;10:305–10.
19. Ellis FH Jr, Gibb SP, Heatley GJ. Reoperation after failed antireflux surgery. Review of 101 cases. Eur J Cardiothorac Surg 1996;10:225–31.
20. Alexander HC, Hendler RS. Laparoscopic reoperation on failed antireflux procedures: Report of two patients. Surg Laparosc Endosc 1996;6:147–9.
21. Lim JK, Moisidis E, Munro WS, et al. Re-operation for failed anti-reflux surgery. Aust N Z J Surg 1996;66:731–3.
22. O'Reilly MJ, Mullins S, Reddick EJ. Laparoscopic management of failed antireflux surgery. Surg Laparosc Endosc 1997;7:90–3.
23. Deschamps C, Trastek VF, Allen MS, et al. Long-term results after reoperation for failed antireflux procedures. J Thorac Cardiovasc Surg 1997;113:545–50.
24. Frantzides CT, Carlson MA. Laparoscopic redo Nissen fundoplication. J Laparoendosc Adv Surg Tech A 1997;7:235–9.
25. Bonavina L, Chella B, Segalin A, et al. Surgical therapy in patients with failed antireflux repairs. Hepatogastroenterology 1998;45:1344–7.
26. Gadenstatter M, Hagen JA, DeMeester TR, et al. Esophagectomy for unsuccessful antireflux operations. J Thorac Cardiovasc Surg 1998;115:296–300.
27. Floch NR, Hinder RA, Klingler PJ, et al. Is laparoscopic reoperation for failed antireflux surgery feasible? Arch Surg 1999;134:733–7.
28. Horgan S, Pohl D, Bogetti D, et al. Failed antireflux surgery. What have we learned from reoperations? Arch Surg 1999;134:809–17.

29. Hunter JG, Smith CD, Branum GD, et al. Laparoscopic fundoplication failures: Patterns of failure and response to fundoplication revision. Ann Surg 1999;230:595–606.
30. Pointer R, Bammer T, Then P, et al. Laparoscopic refundoplications after failed antireflux surgery. Am J Surg 1999;178:541–4.
31. Curet MJ, Josloff RK, Schoeb O, et al. Laparoscopic reoperation for failed antireflux procedures. Arch Surg 1999;134:559–63.
32. Soper NJ, Dunnegan D. Anatomic fundoplication failure after laparoscopic antireflux surgery. Ann Surg 1999;229:669–76.
33. Szwerc MF, Wiechmann RJ, Maley RH, et al. Reoperative laparoscopic antireflux surgery. Surgery 1999;126:723–8.
34. Waring JP. Management of postfundoplication complications. Semin Gastrointest Dis 1999;10:121–9.
35. Voitk A, Joffe J, Alvarez C, et al. Factors contributing to laparoscopic failure during the learning curve for laparoscopic Nissen fundoplication in a community hospital. J Laparoendosc Adv Surg Tech A 1999;9:243–8.
36. Bais JE, Horbach JMLM, Masclee AAM, et al. Surgical treatment for recurrent gastro-oesophageal reflux disease after failed antireflux surgery. Br J Surg 2000;87:243–9.
37. Awad ZT, Anderson PI, Sato K, et al. Laparoscopic reoperative antireflux surgery. Surg Endosc 2001;15:1401–7.
38. Neuhauser B, Hinder RA. Laparoscopic reoperation for failed antireflux surgery. Semin Laparosc Surg 2001;8:281–6.
39. Serafini FM, Bloomston M, Zervos E, et al. Laparoscopic revision of failed antireflux operations. J Surg Res 2001;95:13–8.
40. Granderath FA, Kamolz T, Schweiger UM, et al. Is laparoscopic refundoplication feasible in patients with failed primary open antireflux surgery? Surg Endosc 2002;16:381–5.
41. Legare JF, Henteleff HJ, Casson AG. Results of Collis gastroplasty and selective fundoplication, using a left thoracoabdominal approach, for failed antireflux surgery. Eur J Cardiothorac Surg 2002;21:534–40.
42. Johnsson E, Lundell L. Repeat antireflux surgery: Effectiveness of a Toupet partial posterior fundoplication. Eur J Surg 2002;168:441–5.
43. Granderath FA, Kamolz T, Schweiger UM, et al. Long-term follow-up after laparoscopic refundoplication for failed antireflux surgery. Quality of life, symptomatic outcome, and patient satisfaction. J Gastroinest Surg 2002;6:812–8.
44. Braghetto I, Csendes A, Burdiles P, et al. Results of surgical treatment for recurrent postoperative gastroesophageal reflux. Dis Esophagus 2002;15:315–22.
45. Heniford BT, Matthews BD, Kercher KW, et al. Surgical experience in fifty-five consecutive reoperative fundoplications. Am Surg 2002;68:949–54.
46. Khajanchee YS, O'Rourke RW, Lockhart B, et al. Postoperative symptoms and failure after antireflux surgery. Arch Surg 2002;137:1008–13.
47. Kamolz T, Granderath FA, Bammer T, et al. Failed antireflux surgery: surgical outcome of laparoscopic refundoplication in the elderly. Hepatogastroenterology 2002;49:865–8.
48. Spechler SJ, Jain SK, Tendler DA, et al. Racial differences in the frequency of symptoms and complications of gastro-oesophageal reflux disease. Aliment Pharmacol Ther 2002;16:1795–800.
49. Pimentel M, Rossi F, Chow EJ, et al. Increased prevalence of irritable bowel syndrome in patients with gastroesophageal reflux. J Clin Gastroenterol 2002;34:221–4.
50. Spechler SJ. Comparison of medical and surgical therapy for complicated gastroesophageal reflux disease in veterans. Department of Veterans Affairs Gastroesophageal Reflux Disease Study Group. N Engl J Med 1992;326:786–92.
51. Hinder RA. Surgical therapy for GERD: Selection of procedures, short- and long-term results. J Clin Gastroenterol 2000;30(3 Suppl):S48–50.
52. Johnson DA, Younes Z, Hogan WJ. Endoscopic assessment of hiatal hernia repair. Gastrointest Endosc 2000;52:650–9.
53. Ott DJ. Radiographic techniques and efficacy in evaluating esophageal dysphagia. Dysphagia 1990;5:192–203.
54. Adhami T, Richter JE. Twenty-four hour pH monitoring in the assessment of esophageal function. Semin Thorac Cardiovasc Surg 2001;13:241–54.
55. Perdikis G, Hinder RA, Lund RJ, et al. Laparoscopic Nissen fundoplication: where do we stand? Surg Laparosc Endosc 1997;7:17–21.
56. Rantanen TK, Salo JA, Sipponen JT. Fatal and life-threatening complications in antireflux surgery: Analysis of 5502 operations. Br J Surg 1999;86:1573–7.
57. Carlson MA, Frantzides CT. Complications and results of primary minimally invasive antireflux procedures: A review of 10,735 reported cases. J Am Coll Surg 2001;193:428–39.
58. Vakil N, Shaw M, Kirby R. Clinical effectiveness of laparoscopic fundoplication in a U.S. community. Am J Med 2003;114:1–5.
59. Spechler SJ, Lee E, Ahnen D, et al. Long-term outcome of medical and surgical treatments for gastroesophageal reflux disease. Follow-up of a randomized controlled trial. JAMA 2001;285:2331–8.
60. Wo JM, Trus TL, Richardson WS, et al. Evaluation and management of postfundoplication dysphagia. Am J Gastroenterol 1996;91:2318–22.
61. Malhi-Chowla N, Gorecki P, Bammer T, et al. Dilation after fundoplication: Timing, frequency, indications, and outcome. Gastrointest Endosc 2002;55:219–23.
62. Triadafilopoulos G. Nonobstructive dysphagia in reflux esophagitis. Am J Gastroenterol 1989;84:614–8.
63. Kahrilas PJ, Dodds WJ, Hogan WJ, et al. Esophageal peristaltic dysfunction in peptic esophagitis. Gastroenterology 1986;91:897–904.
64. Spechler SJ, Castell DO. Classification of oesophageal motility abnormalities. Gut 2001;49:145–51.
65. Fibbe C, Layer P, Keller J, et al. Esophageal motility in reflux disease before and after fundoplication: A prospective, randomized, clinical and manometric study. Gastroenterology 2001;121:5–14.
66. Spechler SJ, Souza RF, Rosenberg SJ, et al. Heartburn in patients with achalasia. Gut 1995;37:305–8.
67. Castell DO. Esophageal manometry prior to antireflux surgery: Required, preferred, or even needed? Gastroenterology 2001;121:214–6.
68. Mathew G, Watson DI, Myers JC, et al. Oesophageal motility before and after laparoscopic Nissen fundoplication. Br J Surg 1997;84:1465–9.
69. Pursnani KG, Sataloff DM, Zayas F, et al. Evaluation of the antireflux mechanism following laparoscopic fundoplication. Br J Surg 1997;84:1157–61.
70. Stylopoulos N, Bunker CJ, Rattner DW. Development of achalasia secondary to laparoscopic Nissen fundoplication. J Gastrointest Surg 2002;6:368–78.
71. Ellingson TL, Kozarek RA, Gelfand MD, et al. Iatrogenic achalasia. A case series. J Clin Gastroenterol 1995;20:96–9.
72. Spechler SJ. AGA technical review on treatment of patients with dysphagia caused by benign disorders of the distal esophagus. Gastroenterology 1999;117:233–54.

73. Hui JM, Hunt DR, de Carle DJ, et al. Esophageal pneumatic dilation for postfundoplication dysphagia: Safety, efficacy, and predictors of outcome. Am J Gastroenterol 2002;97:2986–91.
74. Gaudric M, Sabate JM, Artru P, et al. Results of pneumatic dilatation in patients with dysphagia after antireflux surgery. Br J Surg 1999;86:1088–91.
75. Kamolz T, Bammer T, Granderath FA, et al. Comorbidity of aerophagia in GERD patients: Outcome of laparoscopic antireflux surgery. Scand J Gastroenterol 2002;37:138–43.
76. Lindeboom MYA, Vu MK, Ringers J, et al. Function of the proximal stomach after partial versus complete laparoscopic fundoplication. Am J Gastroenterol 2003;98:284–90.
77. Vansant JH, Baker JW Jr. Complications of vagotomy in the treatment of hiatal hernia. Ann Surg 1976;183:629–35.
78. Ireland AC, Holloway RH, Toouli J, et al. Mechanisms underlying the antireflux action of fundoplication. Gut 1993;34:303–8.
79. Fich A, Neri M, Camilleri M, et al. Stasis syndromes following gastric surgery: Clinical and motility features of 60 symptomatic patients. J Clin Gastroenterol 1990;12:505–12.
80. Klaus A, Hinder RA, DeVault KR, et al. Bowel dysfunction after laparoscopic antireflux surgery: Incidence, severity, and clinical course. Am J Med 2003;114:6–9.
81. Hocking MP, Maher JW, Woodward ER. Definitive surgical therapy for incapacitating "gas-bloat" syndrome. Am Surg 1982;48:131–3.
82. Wilkins BM, Spitz L. Adhesion obstruction following Nissen fundoplication in children. Br J Surg 1987;74:777–9.
83. Maddern GJ, Jamieson GG. Fundoplication enhances gastric emptying. Ann Surg 1985;201:296–9.
84. Chang TM, Chen TH, Tsou SS, et al. Differences in gastric emptying between highly selective vagotomy and posterior truncal vagotomy combined with anterior seromyotomy. J Gastrointest Surg 1999;3:533–6.
85. Parkman HP, Miller MA, Fisher RS. Role of nuclear medicine in evaluating patients with suspected gastrointestinal motility disorders. Semin Nucl Med 1995;25:289–305.
86. Thirlby RC, Patterson DJ, Kozarek RA. Prospective comparison of Congo red and sham feeding testing to determine vagal innervation of the stomach. Am J Surg 1992;163:533–6.
87. Balaji NS, Crookes PF, Banki F, et al. A safe and noninvasive test for vagal integrity revisited. Arch Surg 2002;137:954–8.
88. Moulis H, Vender RJ. Percutaneous endoscopic gastrostomy for treatment of gas-bloat syndrome. Gastrointest Endosc 1993;39:581–3.
89. Lacy BE, Zayat EN, Crowell MD, et al. Botulinum toxin for the treatment of gastroparesis: A preliminary report. Am J Gastroenterol 2002;97:1548–52.
90. Miller LS, Szych GA, Kantor SB, et al. Treatment of idiopathic gastroparesis with injection of botulinum toxin into the pyloric sphincter muscle. Am J Gastroenterol 2002;97:1653–60.
91. Swanstrom L, Wayne R. Spectrum of gastrointestinal symptoms after laparoscopic fundoplication. Am J Surg 1994;167:538–41.
92. Kozarek RA, Low DE, Raltz SL. Complications associated with laparoscopic anti-reflux surgery: One multispecialty clinic's experience. Gastrointest Endosc 1997;46:527–31.