Postoperative Problems 2011: Fundoplication and Obesity Surgery

W.O. Rohof, MD[a], R. Bisschops, MD, PhD[b], J. Tack, MD, PhD[c], G.E. Boeckxstaens, MD, PhD[c,*]

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The population of the Western world is abundantly exposed to food. Together with the introduction of fast food, this situation has contributed to an exponential increase in morbid obesity.[1,2] Similarly, the prevalence of gastroesophageal reflux disease (GERD) has increased significantly in the past decades, representing one of the most common gastrointestinal (GI) disorders in the Western world.[3] The fact that morbid obesity is a significant risk factor for GERD certainly contributes to this tendency.

Neurogastroenterologists are mainly confronted with GERD patients presenting at the outpatient clinic or in the endoscopy room, often referred by the primary care physician with symptoms resistant to proton pump inhibitors (PPIs). With the introduction of laparoscopic surgery, however, more patients, especially younger patients or patients unwilling to take lifelong PPIs, are treated surgically. Although laparoscopic antireflux surgery is very effective in controlling reflux, the neurogastroenterologist is increasingly confronted with postsurgery complications; a similar situation now exists for obese patients who have undergone bariatric surgery. Weight reduction and GERD are, for the most part, effectively treated by the relevant surgical techniques used, but patients with symptoms resulting from abnormal motility secondary to altered anatomy or stenosis as a consequence of these procedures are increasingly presenting at our motility unit. In the current review, we will focus on the

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[a] Department of Gastroenterology and Hepatology, Academic Medical Center, Amsterdam, the Netherlands
[b] Department of Gastroenterology, University Hospital of Leuven and Catholic University of Leuven, Leuven, Belgium
[c] Department of Gastroenterology, Translational Research Center for Gastrointestinal Disorders (TARGID), University Hospital of Leuven and Catholic University of Leuven, Herestraat 49, 3000 Leuven, Belgium
* Corresponding author.
E-mail address: Guy.Boeckxstaens@med.kuleuven.be

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postoperative complications of obesity surgery and fundoplication most commonly observed in the outpatient clinic.

OBESITY SURGERY

Obesity is a major medical problem that has seen such a dramatic increase in prevalence in the United States that it now exceeds 30% in both genders and in most age groups.[1,2] As obesity is a major risk factor for several serious medical conditions, such as arterial hypertension, cancer, diabetes, and cardiovascular diseases, this disorder should be rigorously treated.[4,5] Although different treatment modalities have been introduced, the best long-term results are currently obtained with obesity surgery.[4–6] For the most part, 1 of 3 different techniques is used to reduce food intake: laparoscopic adjustable gastric banding (LAGB), Roux-en-Y gastric bypass (RYGBP), and sleeve gastrectomy.

LAGB, introduced in 1993, is one of the most frequently performed surgical procedures to treat obesity worldwide, as it is a relatively simple to perform, minimal invasive and reversible. An inflatable device is positioned around the proximal stomach and then connected to a port reservoir, which is implanted 4 to 6 cm cranial to the xiphoid process and fixed to the periosteum of the sternum.[7,8] Approximately 4 weeks after surgery the band is filled with 2 to 4 ml of saline, thereby creating a small reservoir above the band, ensuring early satiation and reduced food intake. The volume is adjusted during follow-up so that, while solid food intolerance is avoided, food intake is reduced to approximately one-third to one-half of the volume ingested prior to surgery.[9] In the Roux-en-Y gastric bypass, the stomach is largely bypassed with only a small proximal gastric pouch remaining, while the stomach is drastically reduced in size by a sleeve gastrectomy.

In the early postsurgical period, complications such as leakage, infection, and bleeding can occur with all 3 procedures, but these are managed by the surgical team. Here, we will focus more on the long-term impact of these procedures on GI motility/physiology and the associated symptoms/complications, which will largely depend on the type of surgery to which the patient was exposed. Esophageal dysmotility disorders (dysphagia, esophageal dilatation, and heartburn [esophagitis])[10] are the main complications following LAGB, whereas dumping is the major complication of the 2 other techniques. These are the problems that the gastroenterologist is most likely to encounter.

Esophageal Dysmotility Disorders

In patients referred with dysphagia or heartburn following LAGB, displacement of the gastric band or an overinflated band should be excluded by imaging or endoscopy. A recent detailed analysis of a large series of patients (N = 167) treated with LAGB reported, indeed, that band deflation had to be carried out in approximately 30% (47 patients).[10] These patients presented with symptoms of solid food intolerance, nightly aspiration, and vomiting more than twice per week. On imaging, 7 patients had a hypercontractile esophagus, 34 had significant dilatation with anterior/posterior pouch slipping, and, most importantly, 6 patients presented with a major achalasia-like dilatation. The latter patients had to be reoperated on for band removal. Thus, radiographic evaluation of the position of the band and an estimation of transit through the esophagus are crucial. Data on esophageal motility assessed by esophageal manometry or impedance recordings, in this situation, are not available yet and are eagerly awaited.

The radiographic features described (ie, esophageal dilatation, hypercontractility [nutcracker-like esophagus], and, in the most advanced stage, an achalasia-like

picture) most likely result from the chronically increased resistance that has to be overcome by the esophagus and are very similar to the situation created by a too-tight fundoplication (see later). Initially, the esophagus will try to overcome the resistance, resulting in a hypercontractile state, which will gradually "exhaust" the esophagus with resultant dilatation and, ultimately, an achalasia-like picture. Interestingly, following deflation or removal of the gastric band, esophageal dilatation has completely reversed.[10]

After gastric bypass, dysphagia can develop due to a stricture at the stoma in up to 19% of patients. Usually, it is advised to perform balloon dilation only up to 15 mm in diameter, in order to preserve the restrictive function of the RYGBP. In most patients, resolution of symptoms can be obtained with endoscopic treatment.[11]

Dumping Syndrome

The dumping syndrome is a well-described complication of both gastric bypass and sleeve gastrectomy and has also been documented, albeit less frequently, following esophageal surgery. This condition results from too rapid passage of food from the stomach into the small bowel.[12] Under normal conditions gastric emptying is tightly controlled through intimate coordination of motility of the proximal stomach, acting as a reservoir, and the distal stomach, where mixing and grinding result in the reduction of the ingested food into particles small enough to leave the stomach. The accommodation-reflex of the proximal stomach is a vagovagally mediated motor pattern that inhibits the tone of the proximal stomach, thereby creating a reservoir to temporarily store food.[13] Impaired relaxation of the proximal stomach results in early satiation and has been identified as an important pathophysiological mechanism in functional dyspepsia.[14] The main task of the distal stomach, on the other hand, is to gradually brake down food particles to a diameter of 1 to 2 mm, the critical size for passage through the pylorus and thus exiting the stomach. Finally, the pylorus, by acting as a gatekeeper controlling the outflow of the stomach, also significantly contributes to gastric emptying.

After upper GI surgery, such as a sleeve or partial gastrectomy, vagotomy, or even esophageal surgery, the absence of the vagovagal accommodation reflex and/or anatomical reduction of the size of the proximal stomach leads to an impaired reservoir function of the stomach and accelerated gastric emptying.[15–17] In the case of either a Roux-en-Y gastric bypass surgery or a resection of the distal stomach (Billroth I and II), the grinding activity of the antrum and the sifting function of the pylorus are eliminated resulting in the rapid arrival of large food particles in the small intestine. The increased exposure of the duodenum to these large, less easily digested, particles is considered to be the main pathophysiologic mechanism responsible for the development of the dumping syndrome after esophageal and, more frequently, gastric surgery.[12]

Typically, dumping symptoms can be divided into early (within 30 minutes) and late (1 to 3 hours after meal ingestion). Early symptoms are divided into GI complaints, such as abdominal pain, diarrhea, borborygmi, bloating, and nausea and the more specific vasomotor complaints such as flushing, palpitation, perspiration, tachycardia, hypotension, and even syncope. Early symptoms are probably a result of a fluid shift to the hyperosmolar duodenal lumen, resulting in a decrease of circulating volume and, thereby, to postprandial tachycardia, hypotension and, eventually, although rarely, syncope.[12,18] Additionally, the carbohydrate overload leads to a rapid release of GI peptide hormones that alter GI motility and can, in itself, also lead to the observed hemodynamic effects.[18–20] Late symptoms occur up to 3 hours postprandially and include hypoglycemia, perspiration, hunger, fatigue, and syncope. The

excessive presentation of carbohydrates to the jejunum is thought to generate a rapid increase in insulin release via glucagon-like peptide (GLP-1), which eventually leads to hypoglycemia and related symptoms.[18,21]

The diagnosis of dumping syndrome is mainly based on clinical assessment and a modified oral glucose tolerance test. From 50 to 75 g of glucose solution is ingested after an overnight fast. Immediately before and up to 180 minutes after ingestion, blood glucose concentration, hematocrit, pulse rate, and blood pressure are recorded every 30 minutes. This provocative test is considered positive if late hypoglycemia (120 to 180 minutes) occurs or an early (30 minutes) rise in hematocrit (>3%) occurs. The best predictor of dumping, however, seems to be an increase in pulse rate (>10 bpm) after 30 minutes.[22] Assessment of accelerated gastric emptying can be helpful, but this test does not have good sensitivity and specificity.[18,22,23]

With the increased use of bariatric surgery, the incidence of dumping syndrome is rising; implying that clinicians should be familiar with the presentation and management of this syndrome. The first steps in the treatment of dumping syndrome are dietary measures. Patients are advised to eat more and smaller portions more frequently and to avoid drinking during the meal. Furthermore, the intake of fast-uptake carbohydrates should be limited. Most patients respond well to dietary measures, but for those who do not, acarbose is the next treatment step. Acarbose is an alpha-glycosidase hydrolase inhibitor that hinders the uptake of carbohydrates in the jejunum.[12,24] Small studies have indeed shown a reduction in hypoglycemia and an improvement in symptoms[24–27] (**Table 1**). However, because of its mechanism of action, acarbose is only helpful in patients with late dumping symptoms, as gastric emptying is unaffected. Furthermore, the frequent occurrence of side effects, such as bloating, flatulence, and diarrhea, hinder patient compliance. If patients fail to respond to acarbose, the next step is subcutaneous injection of somatostatin analogs, of which short-acting (octreotide) and long-acting (lanreotide, LAR) variants exist. Somatostatin analogs delay gastric emptying and small bowel transit and inhibit the release of GI hormones and insulin, and thus act on several pathophysiologic mechanisms involved in both the early and late phases of dumping. As a consequence, these agents have proven successful in the treatment of the postoperative dumping syndrome[23,28–34] (**Table 2**). As the long-acting variant has a confirmed effect on quality of life and is preferred by patients, monthly administration with LAR is indicated in patients with proven dietary-refractory dumping syndrome and impaired quality of life.[23,35] Known side effects, such as gallstone formation and steatorrhea, have to be considered in the decision on treatment.[23] If patients do not tolerate or do not respond to somatostatin analogues, surgery or continuous enteral feeding might be necessary but the results of these treatments are unpredictable.[12]

ANTIREFLUX SURGERY

Laparoscopic fundoplication is the recommended surgical therapy for GERD and is mainly used in patients who are unwilling to continue lifelong acid suppressive medication or who have experienced only partial therapeutic success. The first described, and most commonly used, is the Nissen fundoplication, in which a circumferential posterior wrap is made.[36] The Toupet fundoplication, on the other hand, creates a partial posterior wrap surrounding 270° of the esophagus.[37] Fundoplication has a high clinical success rate, even in patients with refractory symptoms to acid-suppressive medication, and in long-term follow-up.[38,39] In contrast to PPIs, both acid and weakly acid reflux episodes are reduced by fundoplication, most likely contributing to the excellent results reported.[40,41] However, postoperative symptoms

Table 1
Dumping syndrome studies

Study (year of publication)	No. of Patients	Treatment	Results
McLoughlin (1979)[24]	10	Acarbose 100 mg single administration before oral glucose tolerance test (OGTT)	Improvement of symptoms and attenuation of hypoglycemia during OGTT. Elevated plasma levels of gastric inhibitory polypeptide and insulin were reduced. No change in gastric emptying
Gerard (1983)[25]	24	Acarbose 100 mg single administration before OGTT	Attenuation of hypoglycemia during OGTT. Elevated plasma levels of insulin were reduced. Inhibition of sucrose-induced glucagon suppression
Lyons (1985)[26]	13	Acarbose 50 mg single administration before breakfast, continuation of therapy in 9 subjects	Attenuation of hypoglycemia. Elevated plasma levels of gastric inhibitory polypeptide, insulin, and enteroglucagon were reduced. Marked improvement in symptoms in some patients.
Hasegawa (1998)[27]	6	Acarbose 50–100 mg three times daily for a month	Attenuation of late dumping symptoms and glucose fluctuations. (uncontrolled)

Table 2
Controlled studies evaluating octreotide in dumping syndrome

Study (year of publication)	No. of Patients	Treatment	Results
Hopman (1988)[28]	12	Octreotide 50 μg vs placebo before OGTT	Improved dumping symptoms; suppression of postprandial rise in pulse rate; reduced peak insulin and increased nadir glycemia; delayed GI transit time
Primrose (1989)[29]	10	Octreotide 50 μg vs 100 μg vs placebo before OGTT	Reduced symptoms of early and late dumping; Attenuation of hypoglycemia; reduced pulse and systolic blood pressure changes
Tulassay (1989)[30]	8	Octreotide 50 μg vs placebo before OGTT	Improved dumping symptoms; no increase in insulin or gastric inhibitory polypeptide levels; suppression of rise in pulse and hematocrit
Geer (1990)[31]	10	Octreotide 100 μg vs placebo before a dumping provocative meal	Prevention of dumping symptoms, delayed gastric emptying and transit time, prevention of late hypoglycemia and of the rise in plasma levels of glucose, glucagon, pancreatic polypeptide, neurotensin and insulin.
Richards (1990)[32]	6	Octreotide 100 μg vs placebo before a dumping provocative meal	Prevention of dumping symptoms, fast induction of the migrating motor complex phase III in the duodenum
Gray (1991)[33]	9	Octreotide 100 μg vs placebo before a dumping provocative meal	Improvement of dumping symptoms, suppression of rise in hematocrit and pulse rate, inhibition of postprandial hypoglycemia
Hasler (1996)[34]	8	Octreotide 50 μg vs placebo before OGTT	Improvement of early and late dumping symptoms, suppression of rise in hematocrit and pulse rate, inhibition of postprandial hypoglycemia, no influence on gastric emptying rate
Arts (2009)[23]	30	Octreotide 50 μg vs placebo before OGTT	Improvement of early and late dumping symptoms, suppression of rise in hematocrit and pulse rate, inhibition of postprandial hypoglycemia.

such as dysphagia, bloating, flatulence, and the inability to belch are common and probably underreported.

Dysphagia

In a recent meta-analysis, the 2 most commonly used surgical antireflux techniques, total and partial fundoplication, were compared. Interestingly, partial fundoplication was associated with a significant reduction in the rates for postoperative dysphagia, dilatations for dysphagia, and surgical reinterventions compared to a total fundoplication, whereas the degree of symptom control, as well as rates of esophagitis and esophageal acid exposure, were similar for both treatments.[38] Therefore, the authors concluded that a partial fundoplication is the surgical antireflux procedure of choice for patients with GERD and, particularly in patients with abnormal preoperative manometry.

After fundoplication, early postoperative dysphagia occurs in up to 15% to 20% of patients, likely due to functional esophagogastric junction (EGJ) obstruction as a result of manipulation, local edema or hematoma.[38,42–45] Conservative measures such as supplemental feeding, dietary measures and reassurance are usually advocated, since symptoms resolve in most. More important is dysphagia, mostly for solids, persisting for longer than 2 to 3 months after surgery. Studies report that up to 10% of patients have persistent dysphagia that requires additional therapy.

The presence of preoperative esophageal motor dysfunction has been proposed to be predictive of the occurrence of postoperative dysphagia. Therefore, tailoring the degree of fundoplication according to status of preoperative esophageal motility has been advocated for a long time.[46–48] However, multiple studies have failed to demonstrate that preoperative manometry findings are a predictor of postoperative dysphagia or of the likelihood of reintervention for dysphagia after fundoplication.[43,48,49]

If patients present with persisting dysphagia, diagnostic evaluation is indicated.[42,49,50] A barium swallow and endoscopic evaluation are used to estimate the degree of stasis/stenosis and evaluate the intactness and position of the wrap. Anatomical abnormalities that can cause postoperative dysphagia are a too-tight wrap, displacement of the wrap (slipped Nissen), or hiatal fibrosis or stenosis; frequently no abnormalities are found.[51] Rarely, patients with achalasia misdiagnosed as GERD have been treated by a fundoplication.[52] Postoperative manometry should therefore be performed in patients in whom a preoperative examination is lacking. High-resolution esophageal manometry, on the other hand, is useful to demonstrate an elevated intrabolus pressure, indicative of EGJ obstruction.[53,54]

Interventions with proven treatment success are dilation and reoperation, indicated in 7% and 3% of patients after fundoplication, respectively.[49,55] Patients with anatomic abnormalities such as a slipped wrap should be considered for surgery early, but otherwise, dilations should be attempted first.[50] Dilations can be performed with bougies or through-the-scope balloons. Bougies and through-the-scope Rigiflex balloons range up to 20 mm in maximal diameter and are a safe and effective treatment option, particularly in patients without anatomic or manometric abnormalities.[50,56–58] Initial pneumatic dilation is performed with the minimal balloon volume of 30 mm, and has a success rate of 56% to 64%, although only studies of small sample size are available[56,57,59–61] (**Table 3**). If these endoscopic procedures are unsuccessful, surgical revision is indicated and is required in 3% to 6% of patients to resolve dysphagia.[55,62] It has to be emphasized though, that morbidity, treatment failure rates, and surgical complications are higher for redo surgery compared to the initial procedure, due to adhesions and the altered surgical anatomy. Nevertheless,

Table 3
Success rate of pneumatic dilation in postfundoplication dysphagia

Study (year of publication)	No. of Patients	Dilation Technique and Degree of Inflation (mm)	Success Rate	Serious Adverse Events
Gaudric (2001)[59]	16	Rigiflex balloon to 35 to 40 mm	9 of 16 (56%)	
Fumagalli (2007)[61]	8	Not specified	5 of 8 (63%)	
Hui (2002)[56]	14	Rigiflex balloon to 30 to 35 to 40 mm	9 of 14 (64%)	
Ellingson (1995)[60]	8	Rigiflex balloon to 30 to 35 to 40	5 of 8 (63%)	One perforation occurred, requiring surgery
Total	46		28 of 46 (61%)	

reoperation is still quite successful with success rates of 70% to 80%, most likely as these procedures are preferably performed in tertiary referral centers.[50,51,55]

Gas-Bloat Syndrome

The combination of bloating, the inability to belch, and flatulence are typical in the gas-bloat syndrome, observed in up to 20% of patients after fundoplication.[41,63] These symptoms are caused by the consequent reduction in the frequency of transient lower esophageal sphincter relaxations (TLESRs). This motor pattern is characterized by a prolonged absence of lower esophageal sphincter tone, accompanied by an inhibition of the crural diaphragm, and is widely recognized to be the major underlying pathophysiologic mechanism of GERD. However, as TLESRs are also the physiologic mechanism whereby one normally vents gas from the stomach, there is an increased risk for gas accumulation in the stomach following antireflux surgery.[40,64] Studies with pH-impedance before and after surgery have indeed demonstrated that fundoplication reduces the rate of gastric belches by 65%.[65] However, the frequency of supragastric belches is increased to a similar extent. Therefore, most patients probably do not notice the inability to belch or even report increased belching.[65]

Treatment is generally not necessary, and the number of affected patients declines with prolonged follow-up. Nevertheless, no evidence-based treatment is currently available for these patients, except for surgical revision. Partial fundoplication does lead to fewer symptoms of bloating and complaints of an inability to belch compared to complete fundoplication.[38]

Recurrent Reflux Symptoms

Long-term success rates for antireflux surgery vary between 85% and 90%.[62,66] However, studies have reported a high need for acid suppressive medication after a long period of follow-up: up to 62% in one study.[67] Risk factors for recurrent reflux symptoms after fundoplication are a high level of supine esophageal acid exposure and poor esophageal peristalsis before treatment.[68]

When a patient presents with recurrent reflux symptoms, it is important to objectively determine the relation between reflux episodes and symptoms. The gold standard is the ambulatory pH-impedance recording, as it detects pathologic acid

exposure and also provides an assessment of the correlation between symptoms and acid or nonacid reflux events.[69] If neither acid exposure nor a significant symptom association is detected, the diagnosis of functional heartburn is suspected, and empirical therapy with a low dose of tricyclic antidepressant as a pain modulator can be attempted.[69] When recurrent symptoms are due to reflux, acid-suppressive medication combined with baclofen should be considered. This compound has repeatedly been shown to reduce both acid and nonacid reflux, even in PPI-resistant patients.[70,71] Only in patients unresponsive to any of these measures should surgical reintervention be considered, but, as noted before, this will be more difficult than the initial procedure due to adhesions and the presence of an altered surgical anatomy.

Early Satiety and Dyspeptic Symptoms, Dumping Syndrome

As outlined earlier, the proximal stomach acts as a reservoir accommodating ingested food. During antireflux surgery, the fundus is wrapped around the esophagus to prevent reflux. The downside of this procedure, however, is that the reservoir function of the stomach is impaired. In addition, damage to the vagus nerve is a well known complication of antireflux surgery,[72] further contributing to impaired accommodation of the proximal stomach. Functional studies have confirmed the presence of impaired accommodation following antireflux surgery.[73] As this abnormality is associated with early satiety,[14] these findings most likely explain the development of dyspeptic symptoms, especially early satiety, following antireflux surgery. It should be emphasized that a considerable proportion of patients may already have complained of dyspeptic symptoms before surgery but, quite often, these symptoms have not been adequately elicited or assessed. Obviously, patient selection is of crucial importance to prevent the performance of surgery on patients with functional complaints. This is definitely a major clinical challenge, not only in daily practice but also for investigators studying clinical efficacy of new antireflux compounds.

Finally, although less seen frequently than after gastric surgery, dumping syndrome can develop following antireflux surgery, most likely due to the reduction in proximal stomach volume and injury to the vagus nerve.

SUMMARY

Although the surgical treatment of both GERD and obesity is very successful, these procedures have a significant impact on the physiology and function of the proximal GI tract. With the increasing prevalence of both GERD and obesity, more and more patients present at the motility outpatient clinic with symptoms related to surgical interventions for these medical problems. In this review, we describe the main complications following antireflux surgery: dysphagia, gas bloat syndrome, recurrent (persistent) GERD symptoms, and dyspeptic symptoms. The most common motility-related complications of obesity surgery are dumping syndrome and esophageal dysmotility.

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