

Laparoscopic sleeve gastrectomy and gastroesophageal reflux

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Abstract

Bariatric surgery is the only effective procedure that provides long-term sustained weight loss. Sleeve gastrectomy (SG) has emerged over the last few years to be an ideal bariatric procedure because it has several advantages compared to more complex bariatric procedures, including avoiding an intestinal bypass. However, several published follow-up studies report an increased rate of gastroesophageal reflux (GERD) after a SG. GERD is described as either *de novo* or as being caused by aggravation of preexisting symptoms. However, the literature on this topic is ambivalent despite the potentially increased rate of GERDs that may occur after this common bariatric procedure. This article reviews the mechanisms responsible for GERD in obese subjects as well as the results after a SG with respect to GERD. Future directions for clinical research are discussed along with the current surgical options for morbidly obese patients with GERD and undergoing bariatric surgery.

Key words: Gastroesophageal reflux; Sleeve gastrectomy; Morbid obesity; Bariatric surgery; Gastric bypass

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Core tip: Bariatric surgery is the only effective means to sustain weight loss. Sleeve gastrectomy (SG) has become popular because of its advantages over more complex bariatric procedures. However, an increased rate of gastroesophageal reflux (GERD) has been reported after SG that is either *de novo* or is caused by aggravation of preexisting symptoms. The literature is ambivalent about the implications for increased rates of GERD after SG. This article reviews the mechanisms of GERD in obese subjects, and the results from SG with respect to GERD. Future directions are discussed along

with current surgical options for obese patients with GERD and undergoing bariatric surgery.

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INTRODUCTION

Obesity is associated with significant morbidity and mortality linked to increased cardiovascular risk, osteoarthritis, diabetes, cancer, and gastroesophageal reflux disease (GERD). Must *et al*^[1] showed, in their cross-sectional study, a general pattern of increased prevalence and severity in overweight and obese subjects. This occurred consistently across all racial and ethnic groups and for all health conditions known to be related to obesity, such as cardiovascular disease, type-2 diabetes mellitus, hypertension, stroke, dyslipidemia, osteoarthritis, and some cancers, with the exception of high blood-cholesterol level.

There is strong published evidence that bariatric surgery is the only effective means to sustain long-term weight loss^[2-4]. This weight loss is also associated with the resolution of obesity-related comorbid conditions, which increase the risk of mortality associated with obesity^[2-4]. Three main procedures are used nowadays, including gastric banding, Roux-en-Y gastric bypass (LRYGBP), and the more recent sleeve gastrectomy (SG). The latter was introduced more than a decade ago as the first step in a biliopancreatic diversion with a duodenal switch and has, since then, been shown to be effective as a stand-alone bariatric procedure^[5,6]. SG has rapidly gained a large consensus worldwide in the bariatric community because of its several advantages, which include it being a simple and straightforward surgical technique without needing an intestinal bypass or causing any digestive anastomosis. This means that the whole digestive tract can be accessed without modifying its anatomy^[7].

Although the postoperative mortality and morbidity of SG has been reported to range between those of LRYGBP and gastric banding the results of long-term morbidity are less well known^[8]. Schauer *et al*^[9] recently demonstrated the superiority of the RYGB over a SG for the morbidly obese patients with remission type-2 diabetes at 3 years. A recent meta-analysis showed that a RYGBP was significantly more effective than a SG for the remission of obesity-related comorbid conditions, although no significant difference in weight loss was demonstrated^[10].

Obesity is associated with an increased risk of GERD, with up to 50% in morbidly obese patients suffering from this condition^[11]. It has also been

shown that obesity, itself is a risk factor for GERD through its mechanical alterations to the esogastric junction (EGJ), associated with transient relaxation of the lower esophageal sphincter (LES) and/or the presence of a hiatal hernia (HH), which may further exacerbate GERD. Indeed, the latter is considered to be the only independent predictor for GERD. HH impairs the EGJ flap, interfering with transmission of intragastric pressure (IGP) to the LES and modifying its closure. As a consequence, gastric content may reflux into the esophagus. In the obese patient, visceral fat, organomegaly, and elasticity of support core muscles and ligaments are important in generating an elevated IGP during inspiration and expiration, which is responsible, in turn, for increasing the gastroesophageal pressure-gradient during inspiration.

Although GERD can be particularly invalidating and may increase the risk of esophageal adenocarcinoma in the longer term^[12,13], the relationship between SG and GERD has not been fully elucidated. As there is no strong evidence regarding the influence of SG on GERD, we reviewed the current literature to determine whether SG could alleviate, cause, or exacerbate GERD.

GERD AND LAPAROSCOPIC SLEEVE GASTRECTOMY

GERD and obesity

GERD is a disorder of the upper gastrointestinal tract that is defined by heartburn and acid regurgitation, which develops when reflux of the stomach contents cause troublesome symptoms and/or complications, according to the Evidence-Based Consensus of the Montreal Definition and the Classification of Gastroesophageal Reflux Disease, issued in 2006^[14]. GERD impacts on the daily life of affected individuals, interfering with physical activity, impairing social functioning, disturbing sleep, and reducing productivity at work.

Different mechanisms are implicated for the occurrence of GERD and reflux esophagitis: *i.e.*, LES at a mediastinal position and/or with a short intra-abdominal length, a low resting LES pressure, transient relaxation of the LES, increased intra-abdominal or intragastric pressure, decreased esophageal clearance, increased acid sensitivity of the esophageal mucosa, and anatomic abnormalities of the EGJ junction, such as HH. Moreover, hormones and nutritional factors, such as fat or alcohol, can influence the resting pressure of the LES.

Obesity may promote GERD by increasing intra-abdominal pressure and the gastroesophageal pressure gradient, as well as inducing mechanical alterations to the EGJ. Pandolfino^[15] found a relationship between increasing BMI and the prevalence of GERD, with a high BMI being associated with an elevated risk of GERD. de Vries *et al*^[16] found

Table 1 Symptoms of gastroesophageal reflux disease

	GERD in obesity	De novo GERD after SG	Improvement of GERD after SG
Mechanism	Increasing BMI	Lack of gastric compliance	Reduced intra-abdominal pressure
	Increasing intragastric pressure	Increased intraluminal pressure	Reduced acid production
	Increasing gastroesophageal pressure gradient	Gastric fundus removal	Accelerated gastric emptying
	Hiatal hernia	LES pressure	Reduced gastric volume
		Final shape of the sleeve	
		Narrowing at the junction of the vertical and horizontal parts of the sleeve	
		Twisting of the sleeve	
		Dilation of the fundus	
		Persistence of hiatal hernia	

GERD: Gastroesophageal reflux disease; SG: Sleeve gastrectomy.

that increasing BMI was independently associated with increased intragastric pressure (IGP) during inspiration and expiration, which was responsible for an increase in the gastroesophageal-pressure gradient during inspiration. That study also showed that BMI, IGP, and the gastroesophageal-pressure gradient were strong independent predictors for HH, which was the only independent predictor of GERD. Visceral fat, organomegaly, and elasticity of the support core muscles and ligaments also play important roles in generating an elevated IGP. Indeed, not all patients with elevated IGP will develop a HH, and not every patient with a HH will develop GERD. Furthermore, there is no linear correlation with GERD severity.

If the anatomy of the EGJ flap valve is maintained without axial separation of the crura and LES, theoretically an elevation of IGP is transmitted to the intra-abdominal LES, and thus the EGJ remains closed. However, if the EGJ flap valve is obliterated, elevations in IGP may increase the volume of refluxate once the EGJ is forced open. Importantly, esophageal sensitivity varies from one individual to another and abnormal acid exposure is not always associated with GERD symptoms^[17]. This may be of major importance when explaining the variability of results reporting on SG and GERD (Table 1).

Mechanisms involved in de novo GERD after a LSG

Table 1 summarizes the mechanisms involved in *de*

novo GERD after a SG. Some cases are caused by the large compliant stomach being transformed into a long and narrow tube. This implies a lack of gastric compliance, with an increased intraluminal pressure that correlates inversely with the diameter of the gastric tube and is increased when the pylorus is closed. Other factors are related to dismantling of the anatomical antireflux mechanisms, including disruption to the Hiss angle and resection of the sling fibers in the distal part of the lower sphincter, which results in low esophageal-sphincter pressure. The final shape of the sleeve also plays a role as it may favor GERD and regurgitation when it is funnel-shaped. Technical mistakes include narrowing at the junction between the vertical and horizontal parts of the sleeve, twisting of the sleeve^[18], anatomical stenosis, and persistence of the gastric fundus and/or a HH that has not been diagnosed before surgery. The role of the gastric antrum has not been fully clarified but it is thought that extensive resection of the antrum may impair gastric emptying and favor GERD.

Mechanisms involved in improving GERD after a LSG

Four main principles seem to explain the improvement of GERD after a SG (Table 1), the decrease in intra-abdominal pressure due to weight loss, reduced acid production related to resection of the acid-producing gastric fundus, accelerated gastric emptying^[19,20], and reduced gastric volume. These all contribute to the diminution in gastric refluxate that putatively causes GERD symptoms.

PUBLISHED RESULTS ON THE INFLUENCE OF A SG ON GERD

Table 2 summarizes the data obtained from recent literature, which suggest a negative influence of SG on GERD. Thirteen studies included 5953 patients with a mean BMI of 42 ± 4 kg/m² (range: 37-55.5 kg/m²) and a mean follow up of 29 ± 22 mo (range: 3-72 mo). Of these studies, only one was a prospective randomized study^[21]; all the others were retrospective studies reporting on prospectively collected data^[22-32]. GERD assessment was based on clinical evaluations that included typical symptoms, such as heartburn, with a few studies using Montreal's criteria to define GERD.

Most studies explored the patients preoperatively using endoscopy; however, the use of esophageal manometry, an upper-gastro-intestinal contrast study, and 24-h pH/impedancemetry were inconsistently reported between the studies. All patients, except for those in the study by Arias *et al.*^[23], had preoperative GERD. The data showed a high percentage of persistent preoperative GERD, which occurred in up to 84% of cases in the retrospective review of DuPree *et al.*^[33], rather than a true worsening of symptoms.

Table 2 Negative impact of sleeve gastrectomy on gastroesophageal reflux disease

Ref.	Nature of the study	Patients, <i>n</i>	Pre-operative BMI (kg/m ²)	GERD evaluation	Follow-up (mo)	GERD (%) Preop	GERD (%) Postop
Himpens <i>et al</i> ^[21] , 2006	Prospective randomized: GB vs LSG	40	39	Clinical evaluation	36	-	<i>De Novo</i> At 1 yr: 21.8% At 3 yr: 3.1%
Arias <i>et al</i> ^[23] , 2009	Retrospective review	130	43.2	NA	36	0	<i>De novo</i> : 2.1%
Braghetto <i>et al</i> ^[25] , 2010	Retrospective review, and literature review	167	37 ± 4.4	Clinical score: EGD, EM	-	-	Increase
Braghetto <i>et al</i> ^[26] , 2010	Retrospective review	20	38.3	Clinical score: EM	-	-	Increase
Lakdawala <i>et al</i> ^[27] , 2010	Retrospective review	50	-	-	12	-	Increase
Himpens <i>et al</i> ^[22] , 2010	Retrospective review	30	39.9	NA	72	3.30%	23%
Carter <i>et al</i> ^[28] , 2011	Retrospective review	176	46.6	Clinical evaluation	24	34.60%	47.2%
							33.8% (of total) under medication
Howard <i>et al</i> ^[29] , 2011	Retrospective review	28	55.5	Clinical evaluation UGICS	8	7 (25%)	11 (39%) <i>De novo</i> : 18%
Soricelli <i>et al</i> ^[24] , 2013	Retrospective review: SG + HHR	378	44 ± 3.5	Clinical score: EGD, UGICS, EM. 24-h pH	18	60/378 (15.8%) SG: 19/281 (6.7%) SG+HHR: 41/97 (42%)	71/ 378 (18.7%) SG: 68 (24%) SG+HHR: 3/97 (3.1%)
Sieber <i>et al</i> ^[30] , 2014	Retrospective review	68	43 ± 8	Clinical evaluation: EGD, UGICS, EM	60	50%	Persistence : 44.1% <i>De novo</i> : 16%
Gorodner <i>et al</i> ^[31] , 2014	Retrospective review. Influence of LSG on GERD	14	40 ± 6	Demeester score: BM, EGD, EM. 24-h pH	14	4 (29%)	9 (64%)
Burgerhart <i>et al</i> ^[32] , 2014	Prospective study	20	47.6 ± 6.1	RDQ; EM. 24-h pH	3	14 (70%) Acid exposure: 4.1%	Persistence: 8 (57%) No change: 2 (14%) Worsening: 6 (43%) <i>De novo</i> : 10%
Dupree <i>et al</i> ^[33] , 2014	Retrospective review	4832	47 ± 9	Clinical evaluation	36	44.50%	Acid exposure: 12% Persistence: 84.1% <i>De novo</i> : 8.6%
Total: 13 studies							

LSG: Laparoscopic sleeve gastrectomy; GERD: Gastroesophageal reflux disease; BM: Barium meal; EGD: Esophogastroduodenoscopy; EM: Esophageal manometry; HHR: Hiatal hernia repair; UGICS: Upper gastrointestinal contrast study; NA: Not available; GB: Gastric banding; RDQ: Reflux-disease questionnaire.

In that study, proton-pump inhibitors (PPIs) were effective against the GERD symptoms.

Conversely, Sheppard *et al*^[34] investigated the incidence of GERD as defined by the use of PPIs after SG and RYGBP in a retrospective series of 387 morbidly obese patients. These authors found that GERD symptoms were significantly increased after SG compared to a RYGB. Interestingly, SG patients required more frequent PPI treatment, indicating the occurrence of *de novo* GERD in these patients. *De novo* GERD, which represents the negative impact of this procedure, was found in 2.1%-21% of cases described in Himpens *et al*^[22]'s study. Only one study, from Soricelli *et al*^[24], compared the outcomes of SG by differentiating patients with HH repair from those who had no HH repair during the SG procedure. The authors showed an overall higher postoperative percentage of GERD; nevertheless, they observed a significant decrease in GERD, from 42.1%-3.1% when HH repair was added to the sleeve procedure. These findings underlie the importance of HH and Hiss repair on postoperative GERD independently of the weight-

loss effect on GERD.

Table 3 summarizes the studies that reported a favorable impact of SG on GERD. Twelve studies included 1863 patients with a mean BMI of 51 ± 13 kg/m² (range: 36.5-65 kg/m²) and a mean follow-up of 20 ± 15 mo (range: 6-60 mo)^[19,20,35-44]. In these 12 studies, GERD assessment was always based on clinical evaluation without a direct systematic link to the Montreal criteria or the various clinical symptoms. Half of these studies did not include endoscopic assessment, two had data for 24-h pH/impedancemetry, two reported on esophageal manometry, and two had results from a barium meal as the preoperative assessment. All these patients had preoperative GERD.

Daes *et al*^[36], in their prospective evaluation of 382 patients, showed a 94% resolution of symptoms and emphasized the need for careful attention to surgical technique, such as avoiding relative narrowing at the junction between the vertical and horizontal parts of the stomach, and the importance of placing the anterior stomach wall and posterior stomach

Table 3 Positive impact of a sleeve gastrectomy on gastroesophageal reflux disease

Ref.	Nature of the study	Patients, n	Pre-operative BMI (kg/m ²)	GERD evaluation	Follow-up (mo)	GERD (%) Preop	GERD (%) Postop
Melissas <i>et al</i> ^[39] , 2008	Prospective study	14	49.5	CA	24	2 (14%)	1 (7%)
Nocca <i>et al</i> ^[40] , 2008	Multicenter prospective study	163	45.9	NA	24	10 (6.1%)	6 (3.6%)
Petersen <i>et al</i> ^[41] , 2012	Prospective study: 3 groups	37	50.5 and 47.5	CA; EM	NA	NA	LESP: 8.4 to 21.2 mmHg may protect against GERD
Chopra <i>et al</i> ^[42] , 2012	Retrospective review and analysis	185	49.0	CA; EGD	6	NA	Improvement: 46%
Daes <i>et al</i> ^[35] , 2012	Concurrent cohort study	134	39.0	CA; EGD	12	49.2%	<i>De novo</i> : 3.2% 1.50%
Rawlins <i>et al</i> ^[43] , 2013	Retrospective study	55	65.0	CA; NA	60	27%	27% 53% resolution 16% <i>de novo</i>
Santonicola <i>et al</i> ^[37] , 2013	Retrospective comparative LSG vs LSG + HHR	180 78 LSG 102 LSG + HHR	LSG: 36.5 LSG + HHR: 39.3	CA EGD If GERD: dc - BM	13-18	LSG: 39.2% LSG + HHR: 38.4%	LSG: 22.5%, <i>de novo</i> : 17.7% LSG+HHR: 43.3%, <i>de novo</i> : 22.9%
Sharma <i>et al</i> ^[19] , 2014	Prospective study	32	47.8	GERD SS EGD RS	12	CDS: 2.88 SS: 2.28 RS: 6.25% Esophagitis: 18.8%	CDS: 1.63 (<i>P</i> < 0.05) SS: 1.06 (<i>P</i> < 0.05) RS: 78.1% (<i>P</i> < 0.001) Esophagitis: 25%, reduction of severity
Rebecchi <i>et al</i> ^[20] , 2014	Prospective study A: PAE B: NAE	71	44.3	GSAS EGD BM EM 24-h pH	24	A: GSAS: 53.1 Demeester: 39.5 B: Demeester: 11.9	A: GSAS: 13.1 Demeester: 10.6 B: Demeester: 12 <i>de novo</i> : 5.4%
Pallati <i>et al</i> ^[38] , 2014	Prospective database	585	48.5	GERD-symptom grading based on medication use	6	All patients included	Score improvement 41% Worsening: 4.6% <i>de novo</i> : 9.2%
Del genio <i>et al</i> ^[44] , 2014	Prospective database Retrospective analysis	25	46.1	CA; HRiM, MII-pH	13	Patient excluded if preop. GERD	No <i>de novo</i> GERD
Daes <i>et al</i> ^[36] , 2014	Prospective evaluation	382	37.7	CA EGD	22	44.5%	2.6% 94% resolution of symptoms
Total: 12 studies							

NA: Not available; CA: Clinical assessment; UGICs: Upper gastrointestinal contrast series; LSG: Laparoscopic sleeve gastrectomy; HHR: Hiatal hernia repair; EGD: Esophagoduodenoscopy; GERD: Gastroesophageal reflux disease; dc-BM: Double contrast after a barium meal; CDQ: Carlsson Dent Score; GERD SS: GERD Symptom Score; RS: Radionuclide scintigraphy; GSAS: Gastroesophageal Reflux Disease Symptom Assessment Scale; EM: Esophageal manometry; PAE: Pathologic acid exposure; NAE: Normal acid exposure; HRiM: High-resolution impedance manometry; MII-pH: Combined 24-H pH-multichannel intraluminal impedance; LESP: Lower esophageal sphincter pressure.

wall in an equal and flat position when firing the stapler, in order to keep the sleeve from rolling and spiraling. Interestingly, in the study of Santonicola *et al*^[37], despite a decrease in GERD from 39.2% to 22.5%, there was no difference between pre- and postoperative symptoms after a SG that included or did not include an HH repair. These results emphasize the complex relationship between the mechanisms leading to the occurrence of GERD symptoms and the secondary improvements after weight loss and surgical repair, which increase factors such as the HH. Lastly, a prospective database from Pallati *et al*^[38], which included 585 patients, showed a 41% improvement in GERD symptoms, thus indicating that SG may also suit obese patients suffering from GERD.

MORBID OBESITY IN GERD CANDIDATES FOR BARIATRIC SURGERY

Role of a diagnostic work-up (HH, LES dysfunction) and intraoperative exploration

Resting LES pressure has been shown repeatedly to be normal in obese individuals. However, contradictory data exist on the gastroesophageal-pressure gradient. GERD depends on the pressure gradient between the stomach and esophagus. It has been shown that patients with reflux disease have more acid reflux during transient LES relaxation than normal subjects. Transient LES relaxation and HH are both involved in the mechanisms of GERD.

A preoperative diagnostic work-up is helpful when

Table 4 Laparoscopic sleeve gastrectomy and hiatal hernia repair

No change in GERD	Improvement of GERD
Santonicola <i>et al.</i> ^[37] , 2014	Cuenca-abente <i>et al.</i> (case report, no MeSH) 2006 Parikh <i>et al.</i> (case report, no MeSH) 2008 Korwar V <i>et al.</i> (case report, biological MeSH) 2009 Valera <i>et al.</i> (case report, MeSH) 2009 Merchant <i>et al.</i> (case report, biologic MeSH) 2009 Soricelli <i>et al.</i> ^[24,50] 2010 (mesh in 2 patients) and 2013 (no MeSH) Soliman ^[58] (no mesh, except 2 patients with large HH) 2012 Kotak <i>et al.</i> (case report, no MeSH) 2013 Gibson <i>et al.</i> ^[59] (no mesh) 2013 Daes <i>et al.</i> ^[55,36] (no MeSH) 2012 and 2013

GERD: Gastroesophageal reflux disease.

designing a surgical strategy; however, it has been well-demonstrated that the incidence of GERD symptoms in obese subjects does not correlate well with the severity of disease^[11]. Suter *et al.*^[45] found that 35.8% of morbidly obese patients had symptoms of GERD, of which 53% had HH and 31.4% had peptic esophagitis. Wilson *et al.*^[46] showed that there was an association between excess weight, HH, and reflux esophagitis, thus underlying the need for preoperative exploration. However, they also showed that the association between symptoms and disease was poor, with 51% of symptomatic patients not having esophagitis, and 23% of patients with esophagitis not having symptoms.

Furthermore, studying LES dysfunction may not accurately predict GERD symptoms^[47], and the incidence of HH is often underestimated by upper gastrointestinal endoscopy and/or a barium meal, which are the procedures currently used by most physicians as the preoperative work-up for morbidly obese patients who are candidates for bariatric surgery. For this reason, some authors suggest the need to assess hiatal crura peroperatively for the presence of a HH^[48,49]. Indeed, Soricelli *et al.*^[50] showed that “fingerprint” indentation of the diaphragm, just above esophageal emergence, is correlated with the presence of a crural defect. Interestingly, the same author reported that systematic repair of a HH diagnosed at the time of surgical exploration could effectively eliminate *de novo* GERD after SG^[24].

PROCEDURES USED IN MORBIDLY OBESE PATIENTS WITH GERD AND/OR HH AND UNDERGOING BARIATRIC SURGERY (RYGBP, LSG + HH FIXATION, WITH OR WITHOUT A MESH)

RYGB

A RYGB is considered the most effective bariatric

procedure for GERD symptoms as it limits acid production into the small gastric pouch and reduces esophageal reflux because of the Roux-en Y anatomy, which also retains the physical activity of the esophagus and gastric pouch within the abdomen^[51]. Several studies have confirmed that a RYGBP decreases exposure of acid to the esophagus^[52-54].

Kim *et al.*^[51] showed that conversion of a failed Nissen fundoplication to a RYGB resulted in excellent control of symptoms. Accordingly, Mion *et al.*^[54] and Madalosso *et al.*^[55] found similar results for a RYGP for GERD. Lastly, De Groot *et al.*^[56] compared RYGBP with restrictive procedures, such as gastric banding and vertical-banded gastroplasty, and found better control of symptoms associated with a RYGP. In this study, no data were available for SG as it is a relatively recent procedure.

The role of HH repair appears to be a main concern for some authors at the time of bariatric surgery. Although the need for systematic repair of the crura has only been partially studied for patients undergoing a RYGB^[57] this has been more widely investigated in SG surgery.

LSG + HH repair

Studies regarding the outcomes between laparoscopic sleeve gastrectomy (LSG) and HH repair are summarized in Table 4. Santonicola *et al.*^[37] showed no improvement of GERD symptoms after concomitant SG and HH repair. Moreover, after bariatric surgery, SG patients with a concomitant HH repair had a significantly higher frequency of heartburn than patients who underwent a LSG alone. In contrast, Soricelli *et al.*^[24] reported significant improvement of GERD symptoms after a SG with concomitant HH repair. They described repair of a posterior crura defect with two interrupted non-absorbable sutures, approximating to the right and left diaphragmatic pillars. HH repair was shown to be feasible and safe with no postoperative complications related to this procedure. The authors suggested that approaching the diaphragmatic crus from the left reduced damage to the anterior vascularization of the esogastric junction, which, if it was impaired, could be involved in the development of staple-line leaks after a SG. Furthermore, exposure of the hiatal area to the presence of a HH implies complete freeing of the posterior stomach wall and facilitates complete resection of the gastric fundus. This in turn is of great importance for the success of a SG in terms of weight loss but also avoids *de novo* GERD caused by acid secretion and regurgitation of the persistent gastric fundus content into the esophagus. In addition, the postoperative development of *de novo* reflux symptoms was significantly greater in patients who underwent a SG without an HH repair compared to those with an HH repair (22.9% vs 0%, $P = 0.01$). However, the follow-up for this study was short (12 mo) and midterm results (at the least) are needed before concluding on the role of HH repair.

Soliman^[58] reported favorable results in 20 patients who had concomitant SG and a posterior crural repair. Interestingly, two of the patients with a large HH (> 5 cm) had a polypropylene mesh repair. Thirteen patients reported resolution of GERD symptoms and five reported improvement leading to minimal doses of PPIs at a mean follow-up of 7 mo.

Gibson *et al.*^[59] analyzed the results of SG and HH repair in 500 patients. Interestingly, an anterior repair was performed in 265 patients and a posterior repair in 30 patients. The prevalence of GERD was reduced from 45% preoperatively to 6% ($n = 30$) postoperatively, and postoperative GERD was well controlled in all patients with PPI therapy.

Daes *et al.*^[35] 2012 found that concomitant SG and HH repair in 34 of 134 patients undergoing SG resulted in resolution of GERD symptoms in 94% of patients after a mean follow-up of 12 mo. The same author^[36] reported on simultaneous SG and HH repair in 142 patients out of 382 undergoing SG, and found that only 8 patients (5.6%) suffered with GERD postoperatively. Of the remaining 240 patients, who did not have a HH intra-operatively, only two developed GERD postoperatively. These data underline the importance of intraoperative exploration of the crural region to detect the presence of a HH, which is often missed at preoperative imaging and endoscopy.

The use of a mesh in HH repair has been described and advocated by authors in non-obese patient^[60,61]. Silecchia *et al.*^[62] studied the use of absorbable mesh fixed with a non-permanent device in 43 obese patients. Remission of GERD symptoms was observed in 90% of patients, and there were no mesh-related complications at a mean follow up of 17.4 mo; they also reported a 2.3% recurrence rate.

GERD COMPLICATIONS WITH A LSG

The issue of revisional bariatric surgery has been widely reported in the literature regarding GERD, weight-loss failure, or recurrence^[63,64]. Although a RYGBP is the procedure of choice when GERD complicates SG, some considerations should be given before directly converting from a SG to a RYGBP. Cheung *et al.*^[64] reported the results from revisional surgery after a SG (Re-SG and RYGBP) and found that both procedures were effective in achieving weight loss following a failed LSG. As weight loss may influence GERD symptoms, a Re-SG may also work as an effective tool to reduce GERD. Indeed, Silecchia *et al.*^[65] reported on the safety and efficacy of Re-SG (referred to by the authors as laparoscopic fundectomy) in cases where a residual fundus or neofundus is responsible for GERD symptoms. A Re-SG was done in 19 patients when a residual fundus or neofundus was found in patients with severe GERD symptoms. Of note is that cruroplasty was concomitantly done when a HH was found in this series. All patients had improved GERD symptoms and discontinued PPIs.

Noel *et al.*^[66] evaluated 36 patients (34 women and 2 men, mean age 41.3 years, with a BMI of 39.9) after a Re-SG to correct weight-loss failure and intractable severe GERD, related to pouch dilatation, occurring after a primary LSG. The mean interval of time from a primary SG to a ReSG was 34.5 mo (range: 9-67 mo). The ReSG was effective against GERD symptoms in the short term in this series.

HH is not only responsible for GERD but contributes to the incomplete removal of the gastric fundus, which is often missed at the time of a SG. The latter is responsible for acid secretion, which is then regurgitated back into the esophagus, especially if there are other factors such as a HH or an impaired LES, and if increased transient relaxation is present. Thus, Re-SG may be an option for patients with a persistent gastric fundus and or a HH responsible for GERD that is non-responsive to PPIs. However, this procedure should remain limited to patients in whom a relationship between GERD and a persistent gastric fundus is clear, and should be conducted by a specialized bariatric surgeon. If a HH is present, it should be fixed during the same procedure.

FUTURE DIRECTIONS

SG has become a very popular bariatric procedure in less than a decade because of its several advantages compared to more complicated procedures, including avoiding an intestinal bypass. However, SG as a surgical technique, although straightforward and less technically demanding, implies modification of some of the anatomical antireflux mechanisms. Furthermore, the presence of other factors, such as a HH or an impaired LES, may lead to the appearance of *de novo* GERD or aggravate a preexisting GERD. Although the extent of this problem is not known because the long-term results for SG beyond 10 years are not yet published, the potential consequences of an increased rate of GERD in the obese is alarming. Indeed, a body of literature shows that both obesity and GERD are responsible for the increased rate of adenocarcinoma of the cardia^[67]. Given the number of SGs done annually worldwide this problem should be carefully addressed.

Further randomized studies should address the problem of HH repair in candidates for a SG; the need to diagnose eventual dysfunction of the LES may contraindicate a SG, and thus the extent of a gastric antrum resection. When patients develop GERD after a SG resistant to PPI, a RYGBP remains the operation of choice, whereas some patients with residual fundus after a SG may be suitable candidates for a redo fundectomy or a Re-SG.

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