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Obesity and GERD: Pathophysiology and Effect of Bariatric Surgery

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Abstract Epidemiologic, endoscopic, and pathophysiologic studies document the relationship between obesity and gastroesophageal reflux disease (GERD). Increased body mass index and accumulation of visceral fat are associated with a two- to threefold increased risk of developing reflux symptoms and esophageal lesions. Given this association, many studies were designed to evaluate the outcome of reflux symptoms following conventional and surgical treatment of obesity. Among bariatric procedures, gastric sleeve and banded gastroplasty were shown to have no effect or even worsen reflux symptoms in the postoperative setting. Gastric banding improves reflux symptoms and findings (endoscopic and pH-measured distal esophageal acid exposure) in many patients, but is associated with de novo reflux symptoms or lesions in a considerable proportion of patients. To date, Roux-en-Y gastric bypass is the most effective bariatric procedure that consistently leads to weight reduction and improvement of GERD symptoms in patients undergoing direct gastric bypass and among those converted from restrictive bariatric procedures to gastric bypass.

Keywords Gastroesophageal reflux disease (GERD) · Body mass index (BMI) · Esophageal reflux monitoring · Esophageal manometry · Upper gastrointestinal endoscopy · Proton pump inhibitors (PPI) · Gastric banding · Roux-en-Y gastric bypass · Sleeve gastrectomy

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Introduction

Gastroesophageal reflux disease (GERD) is the most common gastrointestinal diagnosis in the ambulatory care setting in the United States [1]. It is estimated that about 20% to 30% of US adults experience heartburn and/or acid regurgitation (hallmark symptoms of GERD) at least weekly [2]. The most efficient pharmacologic agents to treat GERD are proton pump inhibitors (PPIs); the annual cost of these medications totals $10 billion in the United States. Thus, exploring the mechanisms of GERD and evaluating alternative therapies has never ceased to be a major topic in research and clinical practice.

The increasing prevalence of overweight and obesity is a major concern regarding the health status of the adult and adolescent population. Results of the US National Health and Nutrition Examination Survey (NHANES) found in 2004 that about 66% of the adult US population is either overweight (body mass index [BMI] 25–30 kg/m^2) or obese (BMI>30 kg/m^2) [3]. The prevalence of obesity more than doubled between 1980 and 2009, as indicated by Centers for Disease Control and Prevention surveys. Even more concerning is the prevalence of extremely obese (BMI> 40 kg/m^2) individuals of 4.8% to 5.1% in the general population. Because many of these patients fail conventional pharmacologic and dietary therapies, bariatric surgery remains their only option for treating obesity.

The relationship between GERD and obesity was investigated recently. Epidemiologic studies report 2- to 2.5-fold increased risks of gastroesophageal symptoms in obese patients. Although these symptoms can be effectively

treated with PPIs, several studies were interested in investigating the effects of bariatric surgery on gastroesophageal reflux.

The present review presents studies on the epidemiology and pathophysiologic aspects of the relationship between obesity and reflux, and analyzes the results of studies aimed at investigating the effect of various bariatric procedures on gastroesophageal reflux disease. The review is based mainly on studies published in English and indexed in Medline between 2000 and 2011.

Obesity and GERD

Pathophysiologic mechanisms leading to GERD involve the presence of hiatal hernia, weak lower esophageal sphincter (LES), transient lower esophageal sphincter relaxation, altered gastroesophageal pressure gradient, and esophageal factors (eg, poor esophageal clearance and altered esophageal motility). Although alteration in frequency and characteristics of transient lower esophageal sphincter relaxation, leading to gastroesophageal reflux episodes, is currently considered to be the most important pathophysiologic mechanism of GERD, recent demographic developments of BMI in the United States prompted several authors to investigate more carefully the contribution of obesity to the pathogenesis of GERD. Pathophysiologic hypotheses on the effects of obesity in GERD include concerns that increased fat (in particular visceral fat) might alter the pressure dynamics at the gastroesophageal junction favoring reflux; altered eating habits of obese patients with large, high-caloric meals leading to delayed gastric emptying; and changes of LES resting pressure, fundic distention, and hormonal changes (eg, cholecystokinin, ghrelin) favoring the occurrence and perception of gastroesophageal reflux episodes.

Given the increased prevalence of both gastroesophageal reflux disease and obesity, many authors have investigated the relationship between these two conditions. In 2005, El-Serag et al. [4] performed a cross-sectional study to determine the prevalence and risk factors for GERD in volunteers (VA employees). Participants were asked to complete a GERD questionnaire, provide information on their height and weight, and invited for an upper gastrointestinal endoscopy with biopsies from the distal esophagus. Using these data, the authors analyzed the association between BMI, GERD symptoms, and endoscopic visible erosions using multiple logistic regression analyses. Of the 915 individuals who received the questionnaire, 512 (54%) returned the questionnaire, 453 had complete and interpretable responses including weight and height, and 196 (43% of respondents) underwent upper gastrointestinal endoscopy. With regard to symptoms, 118 of 453 (26%) participants reported weekly heartburn and/or regurgitation and 44 of 196 (22%) had erosive esophagitis. The prevalence of weekly GERD symptoms increased from 17.6% in individuals with BMI<25 kg/m^2, to 27.9% in those with BMI ranging from 25 to 30 kg/m^2, and 34.6% in those with BMI>30 kg/m^2 (P=0.001 for linear trend). A similar trend (P=0.01) was observed in the relationship between BMI and erosive esophagitis, with prevalence of 12.5% in patients with BMI<25 kg/m^2, 29.8% in those with BMI ranging from 25 to 30 kg/m^2, and 26.9% in those with BMI>30 kg/m^2. The multiple logistic regression analysis found a strong positive association between obesity and frequent GERD symptoms (OR 2.44; 95% CI, 1.27–4.67) and between BMI>25 kg/m^2 and erosive esophagitis (OR 2.75; 95% CI, 1.24–6.13). The authors summarized these findings as indicative that higher BMI increases the risk of GERD symptoms and erosive esophagitis, independent of demographic features and dietary intake.

In 2006, Jacobson et al. [5] reported on the association of BMI and GERD symptoms in women based on data from the Nurses' Health Study. The Nurses' Health Study (established in 1976) included 121,700 registered female nurses who completed questionnaires focusing on risks for cancer and cardiovascular diseases. In 2000, supplemental questions regarding GERD symptoms were sent to 12,192 participants without asthma and/or chronic obstructive pulmonary disease. A total of 10,545 (86%) returned the questionnaires, with 2306 (22%) participants reporting GERD symptoms at least once a week, 256 (11%) of whom with severe to very severe (ie, affecting/greatly affecting lifestyle) symptoms. The multivariable analysis found BMI to be an independently associated risk factor for frequent and severe GERD symptoms (BMI 30–35 kg/m^2: OR 2.03, 95% CI, 1.72–2.41 for frequent reflux symptoms and OR 2.56, 95% CI, 2.05–3.29 for severe reflux symptoms; BMI>35 kg/m^2: OR 1.96, 95% CI, 1.58–2.33 for frequent reflux symptoms and OR 2.35, 95% CI, 1.76–3.09 for severe reflux symptoms).

In addition to these epidemiologic studies linking obesity to GERD, pathophysiologic studies have examined the influence of increased body weight, waist circumference, and visceral adipose tissue to GERD. Pandolfino et al. [6] reviewed high-resolution manometry recordings in 285 patients, focusing on pressure changes across the esophagogastric junction during respiratory cycles, measuring intragastric and intraesophageal pressures and calculating the gastroesophageal pressure gradient. Factoring BMI and waist circumference (WC), the authors found a significant correlation of BMI and WC with intragastric pressure (inspiration, BMI [r=0.57], WC [r=0.62], P<.0001; expiration, BMI [r=0.58], WC [r=0.64], P<.0001); and gastroesophageal pressure gradient (inspiration, BMI [r=0.37], WC [r=0.43], P<.0001; expiration, BMI [r=0.24], WC [r=0.26], P<.0001). Multivariate analysis

adjusting for age, gender, and patient type did not alter the direction or magnitude of this relationship. In addition, obesity was associated with separation of the esophagogastric junction pressure components (BMI, r=0.17, P<.005; WC, r=0.21, P<.001) suggestive of temporal developing hiatal hernia. Based on these findings, the authors concluded that obese subjects are more likely to have esophagogastric junction disruption (leading to hiatal hernia), and augmenting the gastroesophageal pressure gradient provided a perfect scenario for reflux to occur.

El-Serag et al. [7] reported on the influence of obesity on distal esophageal acid exposure in 206 patients who underwent conventional 24-hour distal esophageal pH monitoring. In their study, patients were divided into three groups based on BMI (ie, normal-weight BMI<25 kg/m^2, overweight BMI 25.0–29.9 kg/m^2, and obese BMI> 30 kg/m^2). The authors noted significantly (P<0.05) higher distal esophageal acid exposure in the group of obese patients (percentage of time pH<4: 7.7%±9.4%) compared to normal-weight patients (percentage of time pH<4: 5.0%± 6.0). Similar observations were made for postprandial acid exposure and number of reflux episodes (total, upright, and recumbent). These parameters were numerically higher in the overweight group compared to the normal-weight group, but did not reach statistical significance. After controlling for age and gender, BMI>30 kg/m^2 was associated with an almost threefold increased risk of having abnormal distal esophageal acid exposure compared to patients with normal BMI (OR 2.91; 95% CI, 1.24–6.81; P=0.014). When adjusted for waist circumference by including it in the same model, the association between BMI>30 kg/m^2 and measures of esophageal acid exposure became attenuated for all, and not significant for some, thus indicating that waist circumference may mediate a large part of the effect of obesity on esophageal acid exposure. The authors interpreted these findings as indicative that obesity increases the risk of GERD by increasing distal esophageal acid exposure.

Crowell et al. [8] confirmed these results in a study evaluating the influence of BMI on distal esophageal acid in 157 patients undergoing wireless 48-hour capsule pH monitoring. Using the same cut-off values to categorize patients as normal weight (BMI 18–25 kg/m^2), overweight (BMI 25–30 kg/m^2), and obese (BMI>30 kg/m^2), the authors compared distal esophageal acid exposure measured by a wireless capsule positioned 6 cm above the endoscopically visible Z-line, or 5 cm above the manometrically located proximal border of the lower esophageal sphincter. The association between percentage of time pH<4 and BMI showed a significant linear trend for total, upright, and recumbent distal esophageal acid exposure. Compared to normal-weight patients, overweight patients had OR 1.47 (95% CI, 0.69–3.13) and obese patients had OR 5.01 (95% CI, 1.94–12.95) of having an abnormal total distal esophageal acid exposure (ie, percentage of time pH<4: > 5.3%). Furthermore, in obese and overweight patients, percentage of time pH<4 was higher during the second monitoring day compared to the first day. The authors interpreted these data as supportive for the role of obesity in the development of GERD.

In 2009, Schneider et al. [9] showed that obesity increases not only the distal esophageal acid exposure but also the number of gastroesophageal reflux episodes, as assessed by combined impedance-pH monitoring. These authors compared distal esophageal acid exposure and number of acid and non-acid reflux episodes detected by 24-hour impedance pH monitoring in 16 healthy volunteers (BMI<35 kg/m^2), 11 obese patients (BMI 35–39 kg/m^2), 23 super-obese patients (BMI 40–49 kg/m^2), and 17 super-super-obese patients (BMI>50 kg/m^2). They found higher DeMeester scores and number of impedance-detected acid reflux episodes in obese, super-obese, and super-super-obese patients compared to the healthy controls (BMI< 35 kg/m^2). On the other hand, no differences were found in distal esophageal acid exposure and number of reflux episodes between the subgroups of obese patients. The authors interpreted these findings as indicative for the contribution of obesity in the pathogenesis of GERD, and on the other hand, that the severity of GERD is not directly related to the stage of obesity.

Recent studies documented that not all fat is “bad fat” when it comes to gastroesophageal reflux disease. In a cross-sectional, case-control study, Lee et al. [10••] reported on the relationship between erosive esophagitis and visceral fat accumulation quantified by abdominal CT scan in 100 patients with erosive esophagitis Los Angeles classes A through D and 100 age- and gender-matched controls without esophagitis. Esophageal erosions were assessed by endoscopy and body fat distribution by detailed analysis of a 10-mm-thick slice at the level of the fourth lumbar vertebrae. Areas of adipose tissues were defined as areas with tomographic attenuation between −150 and −50 HU, and further defined as visceral fat (VF) and subcutaneous fat (SF) based on intra-abdominal versus extra-abdominal localization. The authors found that patients with erosive esophagitis had higher BMI (26.1 kg/m^2 vs 24.4 kg/m^2; P= 0.028), higher waist-to-hip ratio (0.92 vs 0.89; P=0.021), and almost significant areas of total abdominal fat (214.4 cm^2 vs 175.6 cm^2; P=0.054). Based on definitions of visceral and subcutaneous fat, the authors found that patients with erosive esophagitis had higher VF areas compared to controls (104.7 cm^2 vs 75.9 cm^2; P=0.014), but similar SF areas (109.7 cm^2 vs 98.7 cm^2). A multivariable analysis identified BMI>30 kg/m^2 (OR 2.12; 95% CI, 1.57–2.86), waist-to-hip ratio >0.9 (OR 2.11; 95% CI, 1.17–3.92), and visceral fat area >137.35 cm^2 (OR 3.23; 95% CI, 2.77–3.83) as the main anthropomorphic risk

factors for developing erosive esophagitis. Based on these findings, the authors concluded that further investigations should focus on the visceral fat as a major determinant of GERD.

The demographic, pathophysiologic, and endoscopy studies described above underscore the contribution of obesity—in particular, visceral accumulated fat—to the development of GERD symptoms and lesions. Thus, studies evaluating the effects of bariatric surgery on GERD were warranted.

Effect of Bariatric Surgery on GERD

The most frequently used bariatric procedures include gastric banding, sleeve gastrectomy, and gastric bypass. These procedures have been shown to be effective in weight reduction (their primary goal), but they also influence gastroesophageal reflux in various ways.

Gastric Banding

Gastric banding is the least invasive, restrictive bariatric procedure that does not open any segment of the gastrointestinal tract, and therefore has the lowest risk of leakage (Fig. 1). The procedure consists of implanting an adjustable band around the proximal stomach; a reservoir placed subcutaneously allows adjustment of the size of the band by the amount of fluid used to inflate the band. The filling of the band is adjusted according to symptoms and weight loss. Concerns that this procedure might worsen gastroesophageal reflux were based on the concept that fluid and nutrient retention in the pouch could reflux much more easily from the pouch, and the distension of the pouch might alter LES pressure dynamics.

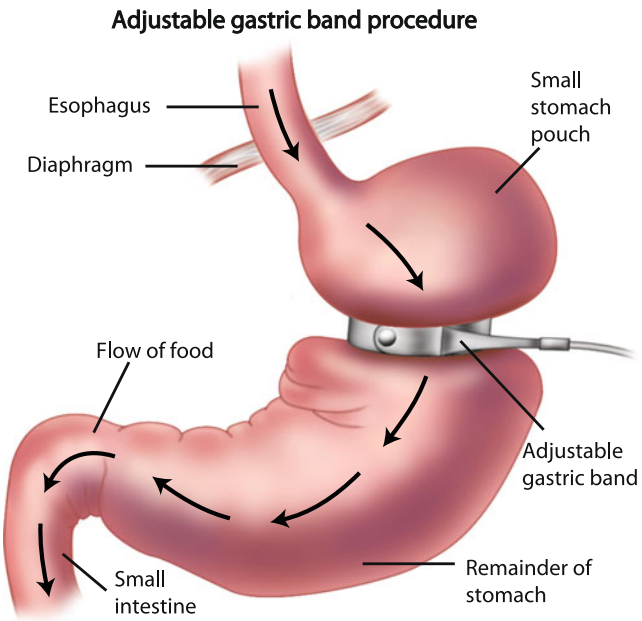

Fig. 1 Gastric banding procedure. The gastric band creates an obstruction to flow, allowing content to remain in the stomach section above the band and below the lower esophageal sphincter. This favors gastroesophageal reflux if the gastric pouch fills rapidly. On the other hand, the gastric band provides a second barrier preventing content from the gastric corpus and antrum to reflux back into the esophagus

In 1999, Dixon and O'Brien [11] were among the first to report on the effects of gastric banding on GERD symptoms in obese patients. In this cohort, 48 of 274 (16%) patients had reflux esophagitis requiring PPI therapy preoperatively. At 2-year follow-up after band placement, 36 (76%) patients reported complete resolution of reflux symptoms and seven (14%) reported marked improvement. No change or aggravation of reflux symptoms occurred in three (6%) and two (4%) patients, respectively. The authors concluded that placement of the band probably acts directly to reduce reflux. A more plausible explanation for the rapid improvement of reflux symptoms following surgery is the reduced size of ingested meals, and later, weight loss resulting from gastric banding.

In 2006, Tolonen et al. [12] presented a study designed to answer the question whether gastric banding reduced or increased gastroesophageal reflux. Thirty-one patients (5 male, 26 female, mean age 44±11 SD years) underwent symptom assessment, upper gastrointestinal endoscopy, and 24-hour pH and manometry recordings before and after gastric banding. After a median time of 19 months (range 7–32 months), symptomatic patients decreased from 48.4% preoperatively to 16.1% postoperatively (P=0.01), medication for GERD decreased from 35.5% to 12.9% (P=0.05), and the diagnosis of GERD on 24-hour pH recordings decreased from 77.4% to 37.5% (P=0.01). Total number of pH-detected reflux episodes decreased from 44.6±23.7 preoperatively to 22.9±17.1 postoperatively (P<0.001), distal esophageal acid exposure (percentage of time pH<4) decreased from 9.5%±6.2% to 3.5%±3.7% (P<0.001), and the DeMeester score decreased from 38.5±24.9 to 18.6±20.4 (P=0.03). Based on these data, the authors concluded that a correctly placed gastric band is an effective antireflux barrier.

In 2010, Rebecchi et al. [13] reported the incidence of GERD in 100 patients undergoing laparoscopic adjustable silicone gastric banding (LASGB) or laparoscopic vertical banded gastroplasty (LVBG). Patients were randomly assigned to either LASBG or LVBG. Follow-up consisted of administering the Gastroesophageal Reflux Disease Health-Related Quality-of-Life (GERD-HRQOL) scale at 3, 12, and 96 months, and performing esophageal manometry, 24-hour pH monitoring, and endoscopy at 12 and 96 months. At the 1-year follow-up, 13 (26%) LASGB patients and 11 (21.6%) LVBG patients developed GERD. In most cases,

GERD was attributed to pouch dilation or poor compliance and required either reoperation (10 LASGB patients and 3 LVBG patients) or endoscopic dilation of the neo-pylorus (4 LVBG patients). Eight years after bariatric surgery, 3 of 26 (11.5%) patients who underwent LASGB and 4 of 45 (9%) patients who underwent LVBG were actively treated with PPIs for GERD. Based on these data, the authors concluded that in the long term, gastric restrictive procedures do not increase the prevalence of GERD, and the increased incidence of GERD in the early follow-up is often from technical defects or poor patient compliance.

A recently published systematic review by de Jong et al. [14•] summarizes the findings of 20 studies including 3307 patients. This meta-analysis found a decrease in the prevalence of reflux symptoms from 32.9% (16–57) preoperatively to 7.7% (0–26.9) postoperatively, and a decrease in the use of antireflux medication from 27.5% (16–38.5) preoperatively to 9.5% (3.1–19.2) postoperatively. On the other hand, this meta-analysis found that 15% (6.1–20) of the patients developed new reflux symptoms following gastric banding. With regard to esophageal lesions, the prevalence of erosive esophagitis decreased postoperatively from 33.3% (19.4–61.6) to 27% (2.3–60.8), but newly developed esophagitis was observed in 22.9% (0–38.4) of patients. Physiologic measurements documented pathological gastroesophageal reflux in 55.8% (34.9–77.4) preoperatively and postoperatively in 29.4% (0–41.7) of the patients. The authors concluded that adjustable gastric banding has antireflux properties resulting in resolution or improvement of reflux symptoms, normalized pH monitoring results, and a decrease in esophagitis in the short term. However, a subset of patients reported worsening or new reflux symptoms and/or development of de novo esophagitis during long-term followup.

Sleeve Gastrectomy

Sleeve gastrectomy is becoming the most frequently used restrictive bariatric procedure (Fig. 2). The stapling of the stomach from the cardia to the pylorus, tailored on a large-size dilator, creates a tubular stomach with decreased reservoir function; moreover, removal of a large part of the gastric fundus leads to decreased levels of ghrelin [15].

In 2006, Himpens et al. [16] reported on the 1- and 3-year results of a prospective randomized study comparing laparoscopic gastric banding and laparoscopic sleeve gastrectomy. Of 80 candidates for bariatric surgery between January 1 and December 31, 2002, 40 were randomly assigned to undergo laparoscopic gastric banding and 40 to undergo laparoscopic sleeve gastrectomy. Laparoscopic sleeve gastrectomy was more effective than laparoscopic gastric banding in reducing weight at 1 year (median weight loss after sleeve, 26 kg [range 0–46 kg] vs banding, 14 kg [range −5–38 kg]) and at 3 years (median weight loss after sleeve, 29.5 kg [range 0–45] vs banding 17 kg [range 0–40]). However, GERD appeared de novo after 1 year in 8.8% of patients with gastric banding and in 21.8% of patients with sleeve gastrectomy (P=NS), and after 3 years in 20.5% of patients with gastric banding and in 3.1% of patients with sleeve gastrectomy (P=NS). Based on these findings, the authors concluded that gastric sleeve was more likely to promote GERD, whereas gastric banding led to an improvement of esophageal erosions.

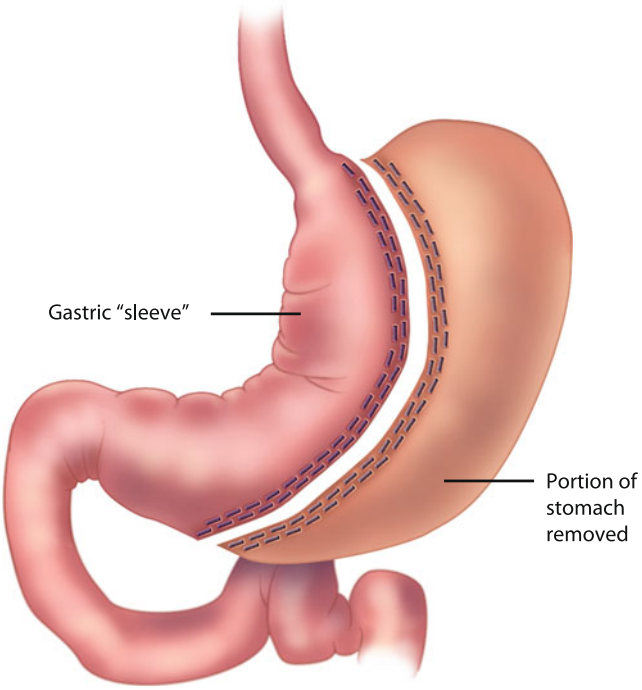

Fig. 2 Gastric sleeve procedure. Although technically easier to perform, especially by laparoscopy, leaving a larger fundus maintains the reflux-favoring mechanisms of transient lower esophageal sphincter relaxations. On the other hand, a small fundic and corpus volume is accompanied by poor distensibility, promoting upper gastrointestinal symptoms and reflux

In 2011, Miguel et al. [17] reported the results of a nonrandomized, prospective, controlled clinical study including 65 women (aged 20 to 60 years old, BMI 40 to 45 kg/m^2) who underwent gastric bypass (N=32) or sleeve gastrectomy (N=32). At baseline, 6 of 33 (18%) patients in the gastric sleeve group and 9 of 32 (28%) patients in the gastric bypass group had endoscopically visible esophageal erosions (P=NS). One year following the bariatric intervention, the percentage of patients with erosive esophagitis rose in the sleeve gastrectomy group to 14 of 31 (45%) and decreased in the gastric bypass group to 2 of 32 (6%). The prevalence of erosive esophagitis was significantly different in the post-sleeve versus post-bypass group (P<0.001). Based on these findings, the authors concluded that sleeve

gastrectomy worsens erosive esophagitis, whereas gastric bypass improves esophageal lesions.

In 2011, Lazoura et al. [18••] investigated the influence of the final shape of sleeve gastrectomy on GERD symptoms. The study included 85 consecutive patients who underwent laparoscopic sleeve gastrectomy [18••]. Symptoms of GERD (heartburn, regurgitation, and vomiting) were assessed preoperatively and at 1, 6, and 12 months postoperatively. The postoperative anatomy was assessed by gastrografin studies routinely performed in all patients on the third postoperative day. The shape of the remaining stomach was classified according to the system proposed by Werquin et al. [19]. The desired tubular pattern was achieved in 66% of patients, a superior pouch was present in 26% of patients, and an inferior pouch was present in 8% of patients. Symptom intensity for regurgitation and vomiting was higher in patients with tubular pattern and superior pouch compared to patients with inferior pouch at 1, 6, and 12 months postoperatively. Based on these findings, the authors concluded that the shape of the remaining stomach has an impact on GERD in patients after laparoscopic sleeve gastrectomy, arguing that an inferior pouch (ie, preservation of the antrum) might be beneficial regarding post-sleeve reflux symptoms.

In summary, currently available data indicate an increased prevalence in esophageal erosions and GERD symptoms in patients undergoing sleeve gastrectomy. Recent data argue that the shape of the remaining stomach plays an important role in the development of reflux, and revising or redesigning sleeve gastrectomy might help prevent patients from developing this complication following bariatric surgery.

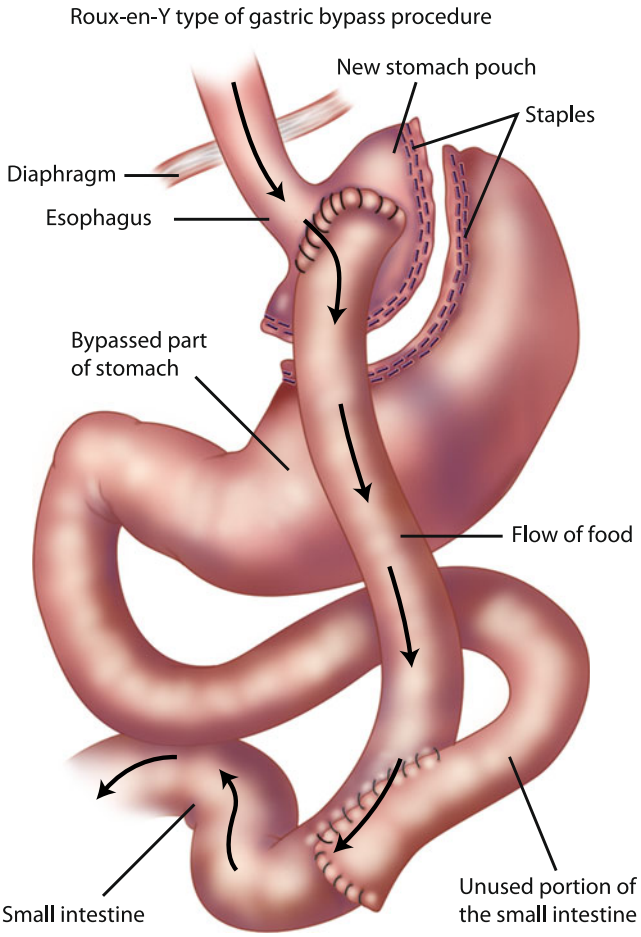

Fig. 3 Gastric bypass operation. The small gastric pouch attached to a jejunal loop provides an adequate antireflux mechanism, because gastric distension is diminished, and consequently transient lower esophageal sphincter relaxations. Problems can occur if the gastrojejunal anastomosis scars, creating an obstruction to flow out of the gastric pouch

Gastric Bypass

Gastric bypass with Roux-en-Y diversion is the most complex and best investigated bariatric intervention (Fig. 3). This procedure involves stapling the stomach to create a small gastric pouch, stapling the small intestine (typically jejunum), and then reestablishing continuity of the gastrointestinal tract by attaching the jejunal loop to the gastric pouch (gastrojejunal anastomosis) and anastomosing the diverted stomach, duodenum, and proximal jejunum back to the jejunum (jejunal-jejunal anastomosis). The procedure is highly effective for weight loss, as documented by initial studies in the mid 1970s [20].

In 2003, Foster et al. [21] reported on the outcome of gastrointestinal symptoms in patients undergoing laparoscopic Roux-en-Y gastric bypass. The authors evaluated 19 gastrointestinal and general well-being symptoms on a visual analogue scale (0–100 points) in 43 patients prior to gastric bypass and in 35 patients 6 months after gastric bypass. From this evaluation, the authors noted significant improvement in abdominal pain, 23.3±26.4 versus 8.6±13.5 (P=0.003); heartburn, 34.0±26.6 versus 8.0±14.0 (P<0.001); acid regurgitation, 28.1±24.0 versus 10.7±21.0 (P=0.001); gnawing in epigastrium, 19.3±22.7 versus 7.5±16.0 P=0.01); abdominal distention, 38.2±31.5 versus 11.1±19.2 (P<0.001); eructation, 27.7±24.4 versus 15.5±16.9 (P=0.01); increased flatus, 40.2±25.7 versus 25.2±25.3 (P=0.005); decreased stools, 5.4±16.8 versus 17.4±20.0 (P<0.001); increased stools, 23.9±26.7 versus 6.5±11.7 (P<0.001); loose stools, 29.7±26.5 versus 17.5±20.0 (P=0.03); urgent defecation, 34.3±26.5 versus 14.3±19.3 (P<0.001); difficulty falling asleep, 44.1±38.4 versus 27.5±32.9 (P=0.05); insomnia, 42.4±36.2 versus 21.6±30.5 (P=0.008); and rested on awakening, 65.1±33.8 versus 30.5±28.8 (P<0.001). Symptoms that did not improve following gastric bypass surgery included nausea/vomiting, borborygmi, hard stools, incomplete evacuation of stool, and dysphagia. Based on these findings, the authors concluded

that gastric bypass surgery improves many gastrointestinal symptoms, including GERD symptoms, without inducing dysphagia.

In 2004, Perry et al. [22] investigated the effects of Roux-en-Y gastric bypass for recalcitrant GERD in morbidly obese patients. The study included 57 patients with refractory GERD and BMI>35 kg/m^2 scheduled to undergo laparoscopic gastric bypass. Forty-eight (84%) of patients had either hiatal hernia or reflux esophagitis and two (3%) had Barrett esophagus. Preoperatively, 31 of 57 (54%) patients used high doses of PPI, or PPI twice daily, and 17 of 57 (30%) patients used high doses of H_2-blocker. At a mean follow-up of 18 months (range 3–30), patients lost on average 40 kg (range 16–70 kg) and all patients reported symptom improvement or no symptoms of GERD. Postoperatively, only 3 of 57 (5%) patients used PPI twice daily, and all 17 patients who used high doses of H_2-blocker were using a low dose of ranitidine (ranitidine, 150 mg daily). Based on these results, the authors concluded that laparoscopic Roux-en-Y gastric bypass should be considered for treating GERD in morbidly obese patients.

Conversion to gastric bypass is often used in patients with reflux symptoms following restrictive bariatric procedures (ie, gastric banding or gastric bypass). Westling et al. [23] reported on the outcome after converting patients with unsuccessful gastric restrictive surgery to gastric bypass. A total of 44 patients underwent conversion from adjustable gastric banding (N=26), vertical banded gastroplasty (N=13), and gastric banding (N=5). The main reasons for conversion from gastric band (both fixed and adjustable) were band erosions (N=12) and reflux esophagitis (N=11), whereas for vertical banded gastroplasty, the staple line disruption led to weight loss failure (N=12). In addition to more efficient weight loss, patients reported prompt resolution of reflux symptoms and vomiting. These results led the authors to conclude that conversion from restrictive bariatric procedures to Roux-en-Y gastric bypass is an effective treatment modality for failures of gastric band or vertical banded gastroplasty.

In 2010 Gagné et al. [24] reported on the results of laparoscopic revision of vertical banded gastroplasty to Roux-en-Y gastric bypass in 105 patients. In addition to a significant reduction of excess weight by 47%, conversion from vertical banded gastroplasty to gastric bypass improved dysphagia in all (100%) patients reporting this symptom prior to conversion, and 95% of patients reported marked improvement of reflux symptoms. Based on these findings, the authors concluded that revision of vertical band gastroplasty to gastric bypass is a feasible procedure with acceptable weight loss and reversal of weight-related comorbidities and symptoms.

Langer et al. [25] recently published a first report on conversion from sleeve gastrectomy to Roux-en-Y gastric bypass. Eight of 73 (11%) patients with sleeve gastrectomy underwent conversion to gastric bypass because of severe reflux (N=3) confirmed by pH monitoring or weight loss failure (N=5) about 3 years after laparoscopic sleeve gastrectomy. Conversion led to an important weight reduction (15±8 kg) in patients reoperated for weight loss failure, and improved reflux in all three patients who had reported severe reflux, as assessed at a median follow-up of 14 months. Patients with reflux symptoms after sleeve gastrectomy were able to discontinue acid-suppressive medication after conversion to bypass.

In summary, studies investigating gastroesophageal reflux in patients undergoing gastric bypass report significant reduction in prevalence of erosive esophagitis and reflux symptoms. Furthermore, small studies indicate that conversion of restrictive bariatric procedures to gastric bypass is successful in treating newly developed reflux symptoms and weight loss failure.

Conclusions

Epidemiologic, pathophysiologic, and endoscopic data underscore the contribution of obesity, in particular visceral fat accumulation, to the development of GERD. Bariatric surgery interventions influence GERD in obese patients, but their effect depends on the procedure used. As detailed in a systematic review by De Groot et al. [26••], vertical banded gastroplasty and sleeve gastrectomy had no influence or even increased GERD, whereas studies on the effect of laparoscopic adjustable gastric banding on GERD report conflicting results. Roux-en-Y gastric bypass had a positive effect on GERD, even though its effects were mainly evaluated by GERD questionnaires. In conclusion, from an esophageal perspective, the preferred bariatric procedure is the laparoscopic Roux-en-Y gastric bypass.

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References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
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1. Shaheen NJ, Hansen RA, Morgan DR, et al. The burden of gastrointestinal and liver diseases, 2006. Am J Gastroenterol. 2006;101:2128–38.

2. Locke III GR, Talley NJ, Fett SL, et al. Prevalence and clinical spectrum of gastroesophageal re- flux: a population-based study in Olmsted County, Minnesota. Gastroenterology. 1997;112:1448–56.
3. Ogden CL, Carroll MD, Curtin LR, et al. Prevalence of overweight and obesity in the United States, 1999–2004. JAMA. 2006;295:1549–55.
4. El-Serag HB, Graham DY, Satia JA, Rabeneck L. Obesity is an independent risk factor for GERD symptoms and erosive esophagitis. Am J Gastroenterol. 2005;100:1243–50.
5. Jacobson BC, Somers SC, Fuchs CS, et al. Body-mass index and symptoms of gastroesophageal reflux in women. N Engl J Med. 2006;354:2340–8.
6. Pandolfino JE, El-Serag HB, Zhang Q, et al. Obesity: a challenge to esophagogastric junction integrity. Gastroenterology. 2006;130:639–49.
7. El-Serag HB, Ergun GA, Pandolfino J, et al. Obesity increases oesophageal acid exposure. Gut. 2007;56:749–55.
8. Crowell MD, Bradley A, Hansel S, et al. Obesity is associated with increased 48-h esophageal acid exposure in patients with symptomatic gastroesophageal reflux. Am J Gastroenterol. 2009;104:553–9.
9. Schneider JM, Brücher BL, Küper M, et al. Multichannel intraluminal impedance measurement of gastroesophageal reflux in patients with different stages of morbid obesity. Obes Surg. 2009;19:1522–9.
10. •• Lee HL, Eun CS, Lee OY, et al. Association between erosive esophagitis and visceral fat accumulation quantified by abdominal CT scan. J Clin Gastroenterol. 2009; 43:240–3. *This article describes the first study analyzing separately the influence of BMI, waist-to-hip ratio, and intra-abdominal versus subcutaneous fat on GERD.*
11. Dixon JB, O'Brien PE. Gastroesophageal reflux in obesity: the effect of lap-band placement. Obes Surg. 1999;9:527–31.
12. Tolonen P, Victorzon M, Niemi R, Mäkelä J. Does gastric banding for morbid obesity reduce or increase gastroesophageal reflux? Obes Surg. 2006;16:1469–74.
13. Rebecchi F, Rocchietto S, Giaccone C, et al. Gastroesophageal reflux disease and esophageal motility in morbidly obese patients submitted to laparoscopic adjustable silicone gastric banding or laparoscopic vertical banded gastroplasty. Surg Endosc. 2010 Jul 30. [Epub ahead of print].
14. • de Jong JR, Besselink MG, van Ramshorst B, et al. Effects of adjustable gastric banding on gastroesophageal reflux and esophageal motility: a systematic review. Obes Rev. 2010; 11:297–305. *The authors describe a relevant meta-analysis (systematic review) on the effects of gastric banding on GERD and esophageal motility.*
15. Langer FB, Reza Hoda MA, Bohdjalian A, et al. Sleeve gastrectomy and gastric banding: effects on plasma ghrelin levels. Obes Surg. 2005;15:1024–9.
16. Himpens J, Dapri G, Cadière GB. A prospective randomized study between laparoscopic gastric banding and laparoscopic isolated sleeve gastrectomy: results after 1 and 3 years. Obes Surg. 2006;16:1450–6.
17. Miguel GP, Azevedo JL, de Souza PH, et al. Erosive esophagitis after bariatric surgery: banded vertical gastrectomy versus banded Roux-en-Y gastric bypass. Obes Surg. 2011;21:167–72.
18. •• Lazoura O, Zacharoulis D, Triantafyllidis G, et al. Symptoms of gastroesophageal reflux following laparoscopic sleeve gastrectomy are related to the final shape of the sleeve as depicted by radiology. Obes Surg. 2011; 21:295–9. *The article describes very important studies indicating that not all sleeve gastrectomies are the same with regard to the risk of developing GERD symptoms*.
19. Werquin C, Caudron J, Mezghani J, et al. Early imaging features after sleeve gastrectomy. J Radiol. 2008;89:1721–8.
20. Mason EE, Printen KJ, Hartford CE, Boyd WC. Optimizing results of gastric bypass. Ann Surg. 1975;182:405–14.
21. Foster A, Laws HL, Gonzalez QH, Clements RH. Gastrointestinal symptomatic outcome after laparoscopic Roux-en-Y gastric bypass. J Gastrointest Surg. 2003;7:750–3.
22. Perry Y, Courcoulas AP, Fernando HC, et al. Laparoscopic Roux-en-Y gastric bypass for recalcitrant gastroesophageal reflux disease in morbidly obese patients. JSLS. 2004;8:19–23.
23. Westling A, Ohrvall M, Gustavsson S. Roux-en-Y gastric bypass after previous unsuccessful gastric restrictive surgery. J Gastrointest Surg. 2002;6:206–11.
24. Gagné DJ, Dovec E, Urbandt JE. Laparoscopic revision of vertical banded gastroplasty to Roux-en-Y gastric bypass: outcomes of 105 patients. Surg Obes Relat Dis. 2010 Nov 5. [Epub ahead of print]
25. Langer FB, Bohdjalian A, Shakeri-Leidenmühler S, et al. Conversion from sleeve gastrectomy to Roux-en-Y gastric bypass–indications and outcome. Obes Surg. 2010;20:835–40.
26. •• De Groot NL, Burgerhart JS, Van De Meeberg PC, et al. Systematic review: the effects of conservative and surgical treatment for obesity on gastro-oesophageal reflux disease. Aliment Pharmacol Ther. 2009; 30:1091–102. *This article is a very important systematic review on the effects of dietary measures and surgical therapy for obesity on GERD*.