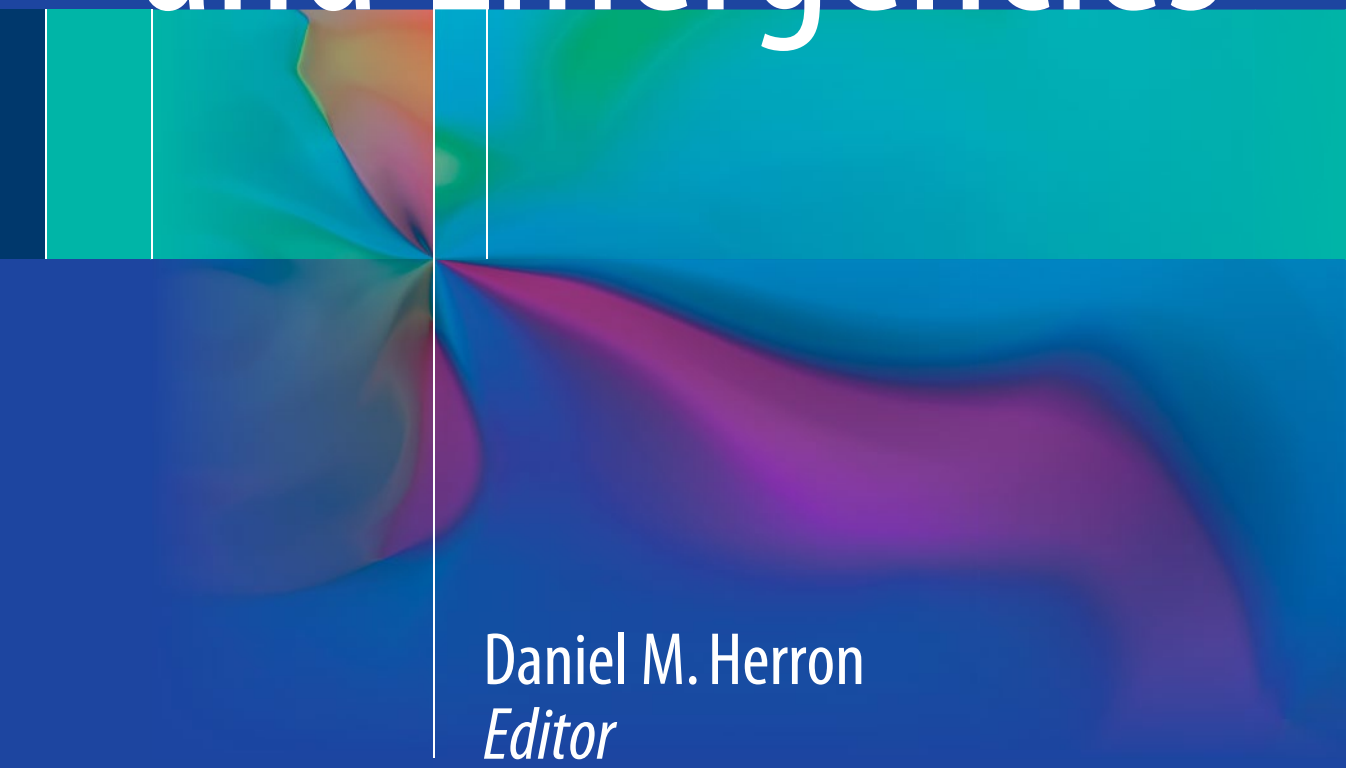
Bariatric Surgery Complications and Emergencies



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ISBN 978-3-319-27112-5 ISBN 978-3-319-27114-9 (eBook)
DOI 10.1007/978-3-319-27114-9

Library of Congress Control Number: 2016932398

Springer Cham Heidelberg New York Dordrecht London

Printed on acid-free paper

Springer International Publishing AG Switzerland is part of Springer Science+Business Media (www.springer.com)

Preface

The idea for this book germinated after a very brief session at the 2013 annual meeting of SAGES (the Society of American Gastrointestinal and Endoscopic Surgeons) in Baltimore, Maryland. The focus of the session was to briefly summarize bariatric surgery and its postoperative complications that may present to the emergency room. The target audience for the session was not necessarily the bariatric surgeon but rather the general surgeon who may be on call for that emergency room and who may be called to evaluate and manage the patient in the absence of a more specialized bariatric surgeon. The session was very brief, only 90 min long. Because the session included pediatric emergencies as well, only 45 min were available to summarize the entire spectrum of bariatric emergencies. However brief, the session focused our attention on the fact that there existed no summary or textbook, either for the general surgeon or the bariatric specialist, that focused primarily on bariatric complications and emergencies.

Given the continually increasing number of bariatric operations performed in the United States, we felt that the need for such a text was real. The American Society for Metabolic and Bariatric Surgery estimates that 158,000 bariatric operations were performed in 2011, 173,000 in 2012, and 179,000 in 2013. By 2014, the most recent year for which numbers are available, 193,000 bariatric operations were performed, 51.7 % of these being sleeve gastrectomy, 26.8 % Roux-en-Y gastric bypass, 9.5 % adjustable gastric band, 0.4 % biliopancreatic diversion-duodenal switch, and 11.5 % revisions[1]. While bariatric surgery has become remarkably safer since the laparoscopic revolution of the mid-1990s, bariatric patients still experience a broad spectrum of postoperative complications. Although some complications like wound infection are common to all general surgery patients, others such as erosion of an adjustable gastric band are unique to the bariatric patient. Still others, such as bowel obstruction, may be common to both general and bariatric surgery patients but may present very differently and require a very different evaluation and management plan in the bariatric population.

With 25 chapters written by internationally recognized experts in the field, this book represents our attempt to distill the issues of bariatric complications into a single volume. After a brief introduction and overview of the various bariatric procedures, subsequent chapters cover an extremely broad range of

[1] Estimate of Bariatric Surgery Numbers, 2011–2014. Published July 2015. https://asmbs.org/resources/estimate-of-bariatric-surgery-numbers accessed 30 August 2015

perioperative and postoperative issues, ranging from leaks, internal hernias, and bowel obstruction, to less commonly addressed topics such as post-bypass hypoglycemia and psychological complications after surgical weight loss. Additionally, several chapters focus specifically on steps that can be taken to avoid complications, both in the operating room and in the early postoperative phase.

I would like to take this opportunity to formally thank the many expert contributing authors who volunteered their time to this project. It is my hope, and the hope of contributing writers, that this book will be a valuable asset not only to bariatric surgeons and integrated health professionals but also to general surgeons and emergency medicine professionals who may be called upon to manage the bariatric patient who presents with a postoperative problem.

New York, NY Daniel M. Herron MD

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Introduction and Overview of Current and Emerging Operations

1

Daniel Shouhed and Gustavo Fernandez-Ranvier

1.1 Introduction

Obesity has become a worldwide epidemic associated with drastic deleterious effects on the health and mortality of patients. Approximately 112,000 deaths per year are related to obesity in the USA alone [1]. Furthermore, the combined direct and indirect costs of obesity were estimated to be $139 billion in 2009, roughly 5 % of the US national health expenditure [2]. Bariatric surgery has been established as the most effective and durable treatment for morbid obesity and shown to be superior to current medical therapies among large meta-analyses [3, 4]. With the advent of laparoscopy and significant reduction in perioperative morbidity and mortality, it is no surprise that the demand for bariatric surgery has exponentially grown, increasing from 8597 procedures in 1993 to greater than 200,000 surgeries in 2007 within the USA [5].

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1.2 Definitions and Surgical Indications

Morbid obesity is most commonly defined as having a body mass index (BMI) above 40 kg/m^2. Bariatric surgery has become established as the most effective and durable approach to treat morbid obesity and its associated comorbidities. According to the 1991 consensus guideline from the National Institutes of Health, candidates for surgical management of obesity in the USA include males or females with a BMI $\geq$ 40 kg/m^2 or a BMI $\geq$ 35 kg/m^2 with one or more significant obesity-related comorbidities such as type 2 diabetes mellitus, hypertension, hyperlipidemia, obstructive sleep apnea, or degenerative joint disease [6].

Surgical candidates must have previously attempted one or more nonsurgical weight-loss programs. They must understand that significant lifestyle changes including diet and exercise are mandatory and that postoperative follow-up and vitamin supplementation are a lifelong requirement. Patients must undergo a thorough psychological evaluation prior to surgery to rule out uncontrolled psychological illnesses or active alcohol or substance abuse. Finally, a surgical candidate should be medically optimized and able to tolerate general anesthesia and a major surgical procedure.

D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_1

1.3 Prevalence and Associated Health Problems

A report by the World Health Organization (WHO) indicated that at least 600 million adults around the world were obese in 2014 [7]. The rates of obesity have more than doubled in the last 25 years in some countries, including the USA, UK, and Australia [8]. In England, the prevalence of obesity among people aged 16 and over is 26 % for men and 24 % for women and the prevalence of morbid obesity is 2.5 % [9]. In the USA 6.6 % of adults are morbidly obese [10].

Obesity, particularly abdominal obesity, is associated with hyperinsulinemia often leading to the development of insulin resistance and subsequently type 2 diabetes mellitus (T2DM). In a 2012 study which estimated that 26 million Americans (8 %) satisfy the criteria for T2DM, more than 80 % of patients with diabetes were overweight and greater than 50 % were obese [11, 12]. Approximately 20 % of morbidly obese individuals have concomitant T2DM [4]. Insulin resistance and adipocyte cytokines may also lead to hypertension, dyslipidemia, vascular inflammation, and endothelial dysfunction, all of which promote the incidence of atherosclerotic cardiovascular disease [13, 14]. Patients who are afflicted with obesity and other metabolic derangements, such as hypertension and dyslipidemia, are at a twofold increased risk of developing cardiovascular disease and have a significantly increased risk of stroke, myocardial infarction, and death from such an event compared to unaffected individuals [15–17].

1.4 The Evolution of Bariatric Surgery

1.4.1 Jejunoileal and Jejunocolic Bypass

Surgical treatment for obesity emerged in the 1950s as physicians noted the weight loss effects of short-gut syndrome. It was noted that the majority of nutrient and fat absorption occurred in the small intestine; this observation served as the basis for the initial procedures targeted at weight loss for morbidly obese patients. Jejunocolic and then jejunoileal bypass were the first operations introduced and can be considered the archetype for the malabsorptive bariatric procedures. In these operations, now no longer performed, the proximal jejunum was connected to the distal ileum or colon, functionally "short-circuiting" the small intestine and resulting in a surgically induced short-gut syndrome. These operations functioned by limiting the intestinal surface area coming into contact with digested food, thereby decreasing caloric absorption.

The early results of these operations were promising as patients enjoyed significant weight loss; however, the benefits were soon outweighed by severe metabolic complications. Bacterial overgrowth of the bypassed loop of small intestine, termed "bypass enteritis," resulted in gas-bloat syndrome and foul-smelling flatus and stool. Absorption of bacterial toxins led to the development of polyarthralgia and hepatic failure, which became the leading cause of death related to these operations [18]. Alterations to the intestinal absorption of fatty acids, calcium, and oxalate led to an increased incidence of cholelithiasis, nephrolithiasis, and renal failure. Profuse diarrhea caused electrolyte imbalances and resulted in anal excoriation and hemorrhoids [19]. With these early operations, the mortality rate within the first 2 years of the operation was 4 % [20]. This alarmingly high mortality rate coupled with the severe morbidities described above resulted in the complete abandonment of jejunoileal and jejunocolic bypass.

1.5 Evolution of the Bypass

1.5.1 Loop Gastric Bypass, Roux-en-Y Gastric Bypass, and Laparoscopic Bypass

The first description of the gastric bypass was reported by Mason and Ito in 1967 based on the observation that patients who underwent partial gastrectomy with a Billroth II reconstruction

experienced significant long-term weight loss [21]. In their initial surgical technique the stomach was divided horizontally with a proximal gastric pouch of approximately 100–150 ml. Then, a loop of jejunum was brought cephalad through a retrocolic tunnel and anastomosed to the stomach pouch with a 12 mm diameter gastrojejunostomy.

In 1975, Mason and Printen modified the technique by reducing the gastric pouch volume to less than 50 ml to increase weight loss. Making the pouch smaller additionally served to reduce the gastric acid within it, thus decreasing the incidence of marginal ulceration and reflux disease [22]. A subsequent modification of the technique reported by Aldens in 1977 consisted of stapling of the stomach horizontally without gastric separation and a creation of an antecolic loop gastrojejunostomy [23]. Multiple complications were reported with the creation of the loop gastrojejunostomy, including dumping syndrome, bile reflux, and marginal ulcers among others [18]. A later report in 1977 by Griffen, described a modification to the Alden's technique with the creation of a retrocolic Roux-en-Y gastrojejunostomy [24]. This Roux-en-Y gastric bypass (RYGB) technique resulted in a lower rate of bile reflux and also decreased tension on the anastomosis (Fig. 1.1).

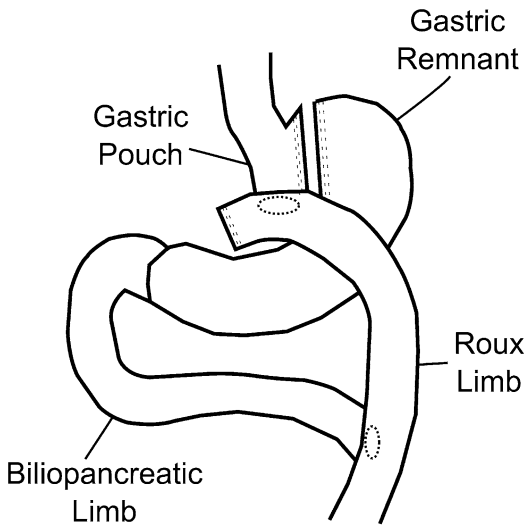

Fig. 1.1 Roux-en-Y gastric bypass (RYGB)

In 1994, Wittgrove and Clark performed the first laparoscopic Roux-en-Y gastric bypass (LRYGB). The proximal anastomosis was created using a circular stapler, with the introduction of the stapler anvil transorally using a peroral endoscopically placed wire similar to that used for percutaneous endoscopic gastrostomy (PEG) tube placement [25]. De la Torre and Scott described a variation of this technique with the introduction of the anvil of the stapler transabdominally [26]. In 1999, Higa reported LRYGB using a laparoscopic hand-sewn 2-layer gastrojejunostomy [27].

Long-term results from a number of different studies over the last three decades demonstrate that the excess weight loss with RYGB is 60–70 % at 5 years, 55–60 % at 10 years, and 50–62 % at 14 years [28, 29]. Although no longer the most commonly performed operation in many centers, the LRYGB is still considered by most bariatric surgeons to be the "gold standard" operation against which all other procedures are measured.

1.6 Gastroplasty

Many different types of gastroplasty have been used over the past three decades for weight loss purposes. However, these techniques have gradually fallen out of favor given their high rate of complications and the frequently inadequate weight loss obtained. The first gastroplasty procedure was performed by Mason and Printen in 1971 [30]. In their technique as originally described, they incompletely divided the stomach horizontally from the lesser curvature to the greater curvature, leaving a small conduit for the physiologic passage of food contents distally. This technique was ultimately unsuccessful in accomplishing adequate long-term weight loss and was abandoned [31]. In 1980, after several modifications of the technique, Mason described the vertical-banded gastroplasty or VBG [32]. In this technique, the stomach was stapled vertically but not divided using a TA-90 stapler after creating a through-and-through window across the anterior and posterior stomach walls. The remaining stomach conduit next to the lesser curvature

was then banded with a 1.5 cm wide polypropylene mesh collar creating a small 30 ml gastric pouch. A modification to this technique was also described by Laws, who used a silicon ring in place of the polypropylene mesh as a permanent, nonexpendable restriction of the pouch outlet [33]. Another modification of Mason's technique was described in 1990 by MacLean, in which the vertical staple line was created using a cutting stapler, thereby completely separating the stomach pouch from the greater curvature [34]. In 1994, Hess and Hess performed the first laparoscopic vertical-banded gastroplasty [35].

Because of significant food restriction and weight loss with the VBG technique, during the 80s and beginning of the 90s, it was used in many centers as the first line of treatment for morbid obesity. However, rates of weight regain were then noticed to be high due to patient's adaptation to high calorie food intake [34, 36, 37]. Complications of the VBG included gastric outlet obstruction secondary to stricture formation, perforation and leak, gastroesophageal reflux and staple line dehiscence with recanalization of the gastric lumen among the most common [38, 39]. For these reasons, the approach was ultimately abandoned.

1.7 Current Bariatric Procedures

1.7.1 Laparoscopic Gastric Bypass

The loop gastrojejunostomy, introduced by Mason and Ito in 1967, gradually evolved into the Roux-en-Y gastric bypass described by Griffen in 1977, with the advantage of reduced bile reflux and marginal ulcers and lowered anastomotic tension [40] (Fig. 1.1). The first series of LRYGB was reported in 1994 by Wittgrove and Clark, with an end-to-end gastrojejunostomy created using an endoscopically introduced anvil [25]. In the LRYGB, the uppermost part of the stomach is partitioned using a cutting surgical stapler to create a small gastric pouch, typically < 30 ml in size. A Roux limb, usually 100–150 cm long, is brought up to the stomach pouch and anastomosed using sutures, staples or a combination of the two. The Roux limb can be brought up in front of the colon (antecolic) or behind the colon (retrocolic). Superiorly, the Roux limb can travel in front of the bypassed stomach (antegastric) or behind it (retrogastric). Gastric juices from the bypassed stomach mix with bile from the liver and pancreatic secretions and pass through approximately 40–100 cm of jejunum referred to as the biliopancreatic limb, before joining the Roux limb to form the "common channel." Internal hernia spaces behind the Roux limb (Petersen defect) and at the distal anastomosis are closed to prevent future bowel entrapment; in retrocolic bypasses, the Roux limb is sutured to the retrocolic tunnel as well [41].

Multiple studies have confirmed that LRYGB is effective at achieving weight loss and resolving comorbidities, while maintaining an acceptably low rate of complications. A systematic review and meta-analysis from 2014 reported the average excess weight loss (EWL) after gastric bypass to be 64–73 % 2–3 years after surgery among 31 randomized controlled trials (RCTs) and 51–78 % among 8 observational studies [42]. Fewer studies were found reporting EWL at 5 years; the average EWL at 5 years among two observational studies was 58 %.

LRYGB is one of the most effective operations for achieving remission of T2DM. Since Pories et al. first described the effect of RYGB on the remission of obesity and diabetes mellitus [43], a large body of supporting literature has accumulated [3, 4, 44, 45]. In a meta-analysis of 621 studies, the average rate of diabetes resolution was 80.3 % among patients undergoing gastric bypass [3]. Four randomized controlled trials have reported that subjects who underwent RYGB were found to have a significantly higher rate of diabetes remission than patients who were only treated with medical therapy [46–49]. The same studies have also shown significant difference in secondary endpoints including hypertension, dyslipidemia, and proteinuria in patients undergoing surgery versus those being treated medically [48, 50]. RYGB has also shown to be effective in inducing diabetes remission in patients with a BMI $\leq$ 35 kg/m^2 [51].

Initially, gastric bypass was accompanied by a relatively high mortality rate. With refinements in technique through experience and proper patient selection, mortality has significantly decreased over time. A systematic review and meta-analysis from 2014 reported a perioperative mortality rate of 0.08 % for RCTs and 0.38 % for observational studies [1]. The mortality rate for greater than 30 days was reported to be 0.39 for RCTs and 0.72 for observational studies. The rate of complications after LRYGB was found to be 21 % and 12 % among RCTs and observational studies, respectively. Reoperation rates were approximately 2.6 % and 5.3 % among RCTs and observations studies.

Complications are typically classified as early or late based on their occurrence before or after the 30-day mark. The most common early complications include leakage, stenosis and bleeding. Most leaks occur at the gastrojejunostomy, though leaks can also infrequently be seen at the entero-enteral anastomosis and at the gastric pouch. Late complications include stricture formation at the gastrojejunal anastomosis, which may be the result of tension or ischemia at the anastomosis, subclinical leaks and/or exposure to excessive gastric acid [52]. Internal hernias, which may occur within the Petersen defect or at the enteroenterostomy site, can be catastrophic, and should be closed at the time of the initial operation [41]. Marginal ulceration, which may be a result of gastro-gastric fistula, is also seen infrequently and can typically be managed nonoperatively. The incidence of complications has been found to decrease with increasing experience of the surgical team over time [53].

1.7.2 Laparoscopic Sleeve Gastrectomy

The sleeve gastrectomy was first described in 1988 by Hess and contemporaneously by Marceau as a component of their biliopancreatic diversion with duodenal switch, or BPD-DS. This operation was a modification of the biliopancreatic diversion (BPD) operation first performed by Scopinaro in Italy [54, 55]. The BPD-DS involved creating a vertical gastric pouch approximately 100–150 ml in volume by resecting the greater curvature and preserving the antrum and pylorus (Fig. 1.2). The duodenum was divided at its first portion and the proximal aspect anastomosed to the ileum, creating the alimentary channel. This anatomy provided a significantly decreased rate of marginal ulceration, intestinal perforation, hypoproteinemia, hypocalcemia, and dumping syndrome, with maintenance of excellent weight loss, when compared to the original BPD [56].

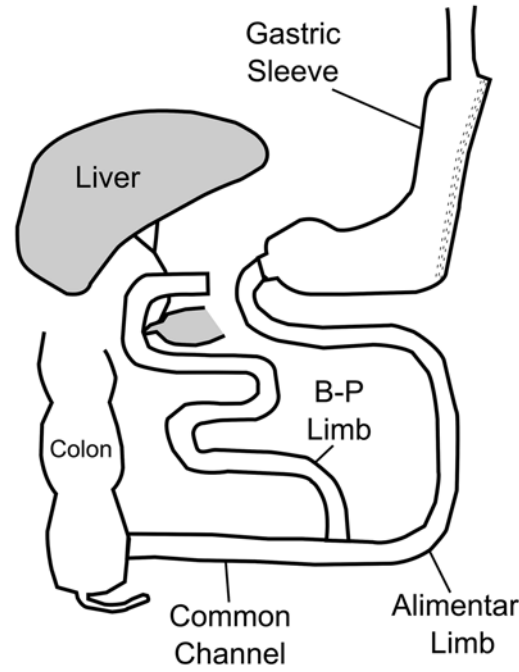

Fig. 1.2 Biliopancreatic diversion with duodenal switch (BPD-DS)

The first laparoscopic BPD-DS was performed by Gagner in New York in 1999 [57]. In an attempt several years later to decrease the morbidity and mortality of patients with a BMI over 60 undergoing BPD-DS, operations on these high-risk patients were performed in two stages: the technically simpler laparoscopic sleeve gastrectomy to allow for initial weight loss and comorbidity resolution, followed by completion of the BPD-DS anatomy approximately 6–12 months later [58–60]. Many of these staged patients achieved substantial weight loss with the sleeve gastrectomy alone, which ultimately led to

its recognition as a stand-alone primary weight loss operation [61, 62]. Over time, laparoscopic sleeve gastrectomy (LSG), also referred to as vertical sleeve gastrectomy has gained increased support from both surgeons and patients.

In the LSG, the greater curvature of the stomach is mobilized by dividing the gastrocolic omentum up to the angle of His superiorly and inferiorly to the antrum approximately 3–6 cm proximal to the pylorus [63]. The linear endoscopic stapler is serially applied to the stomach, beginning approximately 3–6 cm proximal to the pylorus and continuing upward. Staples designed for thicker tissue (e.g., black cartridge) are commonly used for the antral division, while reloads designed for thinner tissue (e.g., purple or blue) may result in better tissue compression and decreased bleeding. Staplers are fired adjacent to a calibrating bougie, typically 32–46 Fr size, which prevents excessive narrowing of the tubularized stomach (Fig. 1.3). Many surgeons feel that the use of buttressing material or oversewing of the staple line may potentially decrease the rate of postoperative bleeding or leakage, although the data are equivocal. This author's group has used both imbrication of part or all of the staple line with a running 2–0 polydioxanone suture and staple line buttressing with excellent results. The excised portion of the stomach is then removed through the largest port site, with or without wound protection. An intraoperative esophagogastroduodenoscopy may be performed to assess the patency of the sleeve, ensure intraluminal hemostasis, and rule out leakage from the staple line. Alternatively, a leak test may be performed with instillation of air or methylene blue dye through an orogastric tube.

An expert consensus statement was issued in 2011 to recommend best practice guidelines based on over 12,000 cases in an effort to reduce complications, improve efficacy, and move toward the adoption of standardized techniques and measures [64, 65]. Many recommendations were made. All panelists felt that use of a bougie was essential, while 87 % believed a 32 F–36 F to be the optimal size of the tubularized stomach. Most agreed that the closed height of the stapler should be at least 2.0 mm at the antrum and up to the incisura, while the closed height of the stapler beyond the incisura should be at least 1.5 mm. Additionally, it was felt to be important to fully mobilize the fundus before transection to prevent leaving behind too much stomach, particularly the fundus, which is relatively more distensible and may expand over time. Panelists also felt it was important to aggressively identify and repair any hiatal hernias. While the fundus should be fully mobilized, care should be taken to avoid stapling too near the gastroesophageal junction, as this may lead to narrowing of the esophagus or leaks at this point. Of note, many surgeons now feel that a bougie size of 40 Fr is preferred due to a potentially lower risk of leak [66].

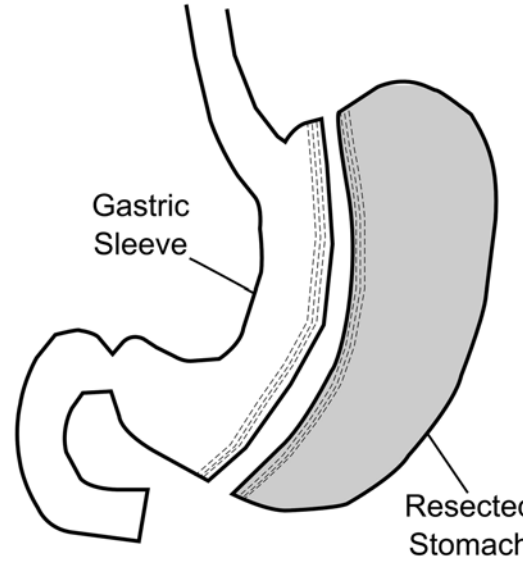

Fig. 1.3 Sleeve gastrectomy (SG)

EWL with sleeve gastrectomy ranges from 49 to 81 % [67]. The overall mean EWL 5 years or more after sleeve gastrectomy in a review of 16 studies was approximately 59 % [68]. Results from trials of LSG in patients with T2DM also show significant remission in the immediate postoperative phase. In observational cohorts, remission rates of T2DM are reported to range from 50 to 80 % at 12–18 months of follow-up [69–71]. The STAMPEDE trial compared outcomes between LSG and medical therapy, in addition to comparing RYGB to intensive medical therapy. At one-year follow-up, the rate of

T2DM remission after LSG was 37 % versus 12 % for the medically treated subjects. Although the rate of remission of T2DM at 3 years was greater among patients undergoing RYGB (38 %) versus LSG (24 %), patients undergoing LSG still demonstrated a significantly higher rate of remission ($p=0.01$) compared to patients who were treated with intensive medical therapy (5 %) [48, 50].

Based on the data of 12,799 laparoscopic sleeve gastrectomies from the International Sleeve Gastrectomy Expert Panel Consensus Statement of 2011, the average length of hospital stay after LSG was 2.5 ± 0.93 days. The conversion rate to open surgery was 1.05 % ± 1.85 %. The postoperative gastroesophageal reflux rate was 12.11 % ± 8.97 %. On average, patients experienced a 1.06 % leak rate and 0.35 % stricture rate [64]. The overall complication rate of LSG in large medical centers is < 15 % [72].

Despite a low overall mortality of 0.3 % with a leak-related mortality of only 0.1 % after LSG [66], the incidence of staple line leak and bleeding after LSG is perhaps the most concerning complication and potential target for technical improvement. Some controversy exists over the use of staple line reinforcement after sleeve gastrectomy [73]. A variety of surgical options including staple-line reinforcement, suture invagination, and biological sealant have been used to try and reduce the incidence of leak after sleeve gastrectomy. This topic will be discussed in greater depth in subsequent chapters.

1.7.3 Laparoscopic Adjustable Gastric Band Placement

This operation is often considered to be the least invasive bariatric procedure, although it does require the potentially permanent placement of a foreign body around the upper stomach. The first experiences in laparoscopic adjustable gastric banding (LAGB) were reported in 1993 [74–77]. Early approaches utilized the "perigastric" technique in which a retrogastric tunnel was created from the lesser curvature close to the gastric wall to the greater curvature about 2 cm below the cardia. Later approaches used the "pars flaccida" approach in which the pars flaccida was opened and a tunnel created behind the gastroesophageal junction above the level of the lesser sac. The adjustable silicone band could then be placed through this tunnel and secured anteriorly with a buckling device (Fig. 1.4).

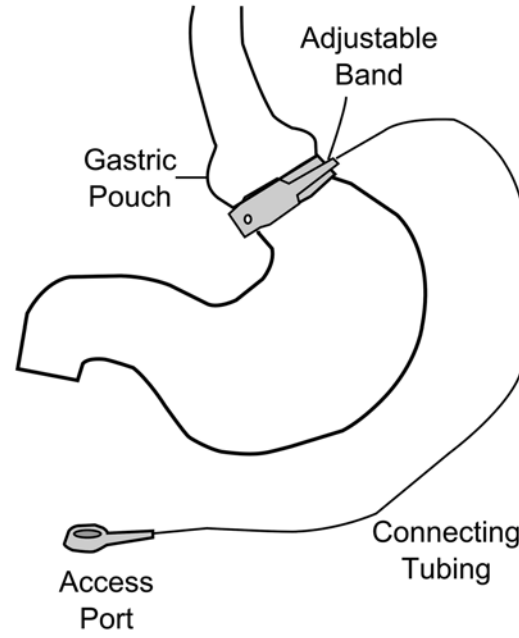

Fig. 1.4 Laparoscopic adjustable gastric band (LAGB)

For optimal results it is recommended that the pouch is sized to measure approximately 15 ml [78]. Additional recommendations include band imbrication anteriorly with two or more gastrogastric sutures to prevent band slippage [78]. The band is then connected to an access port which is implanted in the subcutaneous tissue in the abdominal wall, allowing percutaneous inflation of the gastric band with saline for regulation of the opening of the ring. It is recommended to wait 4–6 weeks prior to band inflation in order to allow adequate healing of the stomach imbrications to reduce the risk of slippage [78].

The average EWL in patients undergoing LAGB is approximately 46 % [79, 80]. Variable rates of diabetes resolution after LAGB have been reported in the literature. In a recent meta-analysis, approximately 57 % of patients demonstrated resolution of diabetes after LGB [3].

In a randomized controlled trial comparing patients with mild obesity (BMI 30–35 kg/m^2), significant differences in weight loss were observed at 2 years in patients who underwent LAGB (87.2 % EWL) when compared to a non-surgical group who was treated medically (21.8 % EWL) [81]. They also observed a decrease in the rate of metabolic syndrome from 38 to 3 % in patients undergoing surgery compared to 38 to 24 % in medically treated patients [81].

In another randomized controlled trial comparing patients who underwent LAGB to those treated medically for type 2 diabetes and weight loss, significant differences were observed after 2 years with remission of diabetes in 73 % of patients undergoing LGB and 27 % of those undergoing conventional medical therapy [82]. Surgically treated patients lost a mean of 62.5 % of excess body weight compared to 4.3 % in the conventional-therapy group. Seventy percent of surgical patients experienced remission of metabolic syndrome compared to 13 % of the medically treated patients. Remission of type 2 diabetes was related to weight loss and lower baseline glycated hemoglobin levels. The authors concluded that weight loss after 2 years of treatment with LGB resulted in significant resolution of type 2 diabetes and metabolic syndrome in the majority of the obese patients with BMI < 40 kg/m^2 when compared with medical treatment alone [82].

A randomized study from 2014 showed a more significant rate of diabetes remission among patients undergoing RYGB compared to those undergoing LAGB [83]. Rates of partial and complete remission of type 2 diabetes were 50 % and 17 %, respectively in the RYGB group and 27 % and 23 %, respectively, in the LAGB group ($p < 0.001$ and $p = 0.047$ between groups for partial and complete remission), with no remission in patients undergoing lifestyle and weight loss intervention [83].

Despite its low mortality and short-term morbidity, LAGB is associated with several late complications including band slippage, gastric erosion and gastric pouch dilatation [84]. Because of these issues, coupled with the reduced weight loss relative to gastric bypass and sleeve gastrectomy, and the "higher maintenance" required, the band has recently fallen out of favor and has been abandoned completely in many centers.

1.8 Biliopancreatic Diversion (BPD) and Biliopancreatic Diversion with Duodenal Switch (BPD-DS)

Scopinaro published his initial series of 18 patients undergoing biliopancreatic diversion in 1979 [85]. This procedure consists of a distal gastrectomy (antrectomy) leaving a proximal gastric pouch of about 200–400 ml volume [85]. The terminal ileum is divided 250 cm proximal to the ileocecal valve. The distal aspect of the divided ileum (alimentary limb) is brought up through a retrocolic tunnel and anastomosed to the remaining stomach. The proximal aspect of the divided ileum (biliopancreatic limb) is then anastomosed to the side of the distal ileum 50 cm proximal to the ileocecal valve, resulting in a common channel 50 cm in length. In a communication, Scopinaro reported an EWL of more than 70 % at 1 year and maintained for 20 years in the majority of the patients who underwent BPD [86].

In 1993, the BPD was modified by Marceau into the BPD-DS; he performed a vertical gastrectomy to create a gastric tube of approximately 200 ml volume based on the lesser curvature of the stomach rather than a horizontal gastrectomy as described by Scopinaro. With Marceau's technique, the pylorus was preserved, the duodenum was cross-stapled and then the enteric limb anastomosed to the proximal duodenum [87]. However, a high rate of failures and weight regain were observed after disruption of the staple line of the duodenum and subsequent recanalization of the normal gastric-duodenal transit. In 1998, Hess described a comparable BPD-DS but with a division of the duodenum, closure of the duodenal stump, and end-to-end anastomosis of the enteric limb to the proximal duodenum [54].

The BPD-DS operation is technically demanding, particularly when performed laparoscopically, and is associated with a higher degree of protein, nutritional, and vitamin deficiencies than any other bariatric currently used procedure. However,

it produces the greatest weight loss of any bariatric procedure and is the most likely to produce remission of diabetes. Studies suggest that mean EWL with BPD-DS at long-term follow-up ranges from 61 to 85 % [88–91]. In a systematic review, which included 48 studies for a total of 1565 patients comparing different bariatric surgical procedures, mean EWL at 2-year follow-up was 73 % with BPD-DS, 63 % with gastric bypass, 56 % with gastroplasty and 49 % with gastric banding. Diabetes resolution was greatest for patients undergoing BPD-DS (95.1 %), followed by RYGB (80.3 %), gastroplasty (79.7 %), and then LAGB (56.7 %). The proportion of patients with diabetes resolution or improvement was fairly constant at time points less than 2 years and 2 years or more [3].

In a prospective randomized controlled trial conducted among 60 patients by Mingrone et al., 95 % of subjects undergoing BPD achieved diabetes remission compared to 0 % in the medically treated group at 2-year follow-up. All patients had a history of at least 5 years of diabetes and glycated hemoglobin of 7.0 % or more. Remission was defined as a fasting glucose level of < 100 mg/dL and a glycated hemoglobin of < 6.5 % in the absence of pharmacologic therapy. There was also significantly greater improvement in total cholesterol levels, triglyceride levels and HDL levels among patients undergoing BDP versus medical therapy [49].

1.9 Emerging Bariatric Techniques

New bariatric devices and procedures intended to treat obesity are continually being developed. Endoscopic interventions such as transoral gastroplasty, the intragastric balloon, and the endoluminal gastrointestinal liner are a few devices that have gained recognition and demonstrated promising results [92]. Although these procedures are not as effective or sustainable at achieving weight loss as the surgical procedures that are being widely used, they have the potential to be less invasive, safer, and more cost-effective. These devices may hold potential for patients with early-stage obesity who do not yet qualify for traditional surgery; alternatively, they may serve as a bridge to traditional bariatric or non-bariatric operations for those who are too heavy to safely undergo surgery. Additionally such novel interventions may have potential as revisional procedures for failed bariatric surgical operations [93].

1.9.1 Intragastric Balloon

The intragastric balloon is one of the first endoscopic devices used for bariatric intervention. The balloon serves to reduce food consumption by occupying space in the stomach and inducing satiety. Since its inception in 1982, the intragastric balloon has undergone multiple transformations to minimize complications such as distal migration of the balloon leading to obstruction, ulceration and erosion, as well as nausea and vomiting that rarely require balloon removal. Earlier devices were designed as single balloons composed of silicone, which were inflated with approximately 400–700 ml of saline after being endoscopically deployed in the stomach. This type of balloon was removed from use in the USA due to problems with the complications noted above.

A newer model introduced by ReShape Medical (San Clemente, CA) and branded as the ReShape Duo is a dual-balloon device that is filled with 900 ml of saline and is designed to maximize space occupation in the stomach. This newer device potentially reduces the undesirable risk of migration, obstruction, and perforation conferred by the single balloon. If one balloon deflates in a dual-balloon device, the second balloon will maintain the device within the stomach, preventing migration and possible bowel obstruction while allowing the patient enough time to seek medical attention. The intragastric balloon is typically left in place for 6 months after which it is endoscopically deflated and extracted using a snare or basket [94].

Several investigators have evaluated the safety and efficacy of the intragastric balloon in the management of obesity. In the largest

reported study retrospectively analyzing the results of the intragastric balloon, the 6-month EWL was 33.9 % ± 18.7 % in 2515 patients. Patients with hypertension and diabetes achieved significant improvements in blood pressure and glycemic control. The authors reported five cases of gastric perforation (0.19 %), 2 of which were fatal [95]. Similar results were seen in a meta-analysis of 15 studies which demonstrated a 32 % EWL with a 0.1 % incidence of gastric perforation [96].

The intragastric balloon has also been used as a bridge for super-obese subjects with multiple medical comorbidities, allowing the achievement of short-term weight loss and reduction in comorbidities, potentially reducing the risk for subsequent traditional bariatric surgery [97]. The greatest pitfall of the device is the durability of weight loss. In a study looking at patients at approximately 5-year follow-up, only one-fourth of subjects sustained weight loss in the absence of any dietary or exercise regimen after balloon removal [98]. The Reshape balloon was approved by the FDA in July, 2015 for use in adult patients with a BMI of 30–40 kg/m^2. It remains to be seen whether the device will achieve significant clinical acceptance.

1.9.2 Duodenojejunal Bypass Sleeve

The endoscopic duodenal-jejunal bypass liner (EDJL), also known as the Endobarrier Gastrointestinal Liner (GI Dynamics Inc., Lexington, Mass), is a 60-cm impermeable fluoro-polymer liner, which is placed endoscopically and anchored at the duodenal bulb. Ingested food and gastric secretions pass through the interior of the liner while pancreatic enzymes and bile acids are diverted around the exterior of the liner. The EDJL is a temporary device, designed to be left in place for 6 months before endoscopic removal. The sleeve is intended to mimic the effects of gastric bypass surgery by delaying digestion and intervening with the body's metabolic functions, including alteration of incretin pathways [93].

In a multicenter, randomized clinical trial conducted in the Netherlands, 30 patients underwent EDJL and 11 were designated to adhere to a low-calorie diet alone. The mean percentage of EWL after 3 months was 19.0 % for patients who underwent a EDJL compared with 6.9 % for control patients ($P = .002$). All patients in the EDJL group had at least one adverse event, such as nausea, upper abdominal pain, pseudopolyp formation, or implant site inflammation. There were no serious adverse events, and all minor adverse events resolved either spontaneously of after temporary medication with no further sequelae [99]. Other studies have also demonstrated the efficacy of the EDJL in achieving significant weight loss; nonetheless, all series reported some degree of adverse events, a proportion of these classified as major adverse events, such as upper gastrointestinal bleeding, anchor migration, and stent obstruction [100].

A recent study explored the potential for EDJL in managing type 2 diabetes mellitus. At 1 year, patients who underwent the endoscopic procedure were found to have significantly lower requirements for insulin therapy than those who were treated with dietary interventions. Baseline glycated hemoglobin levels were 8.3 % for both groups and dropped to 7.0 % and 7.9 % in patients who underwent EDJL and dietary intervention, respectively ($p < 0.05$) [101]. The EDJL is approved for use in Europe to treat patients with type 2 diabetes mellitus and obesity for 12 months. However, the US pivotal trial of the device was terminated in July, 2015, due to higher than expected rates of hepatic abscess in clinical subjects. With the premature conclusion of this trial it appears unlikely that the device will ever see clinical use within the USA.

1.9.3 Vagal Nerve Blockade

In January of 2015, the FDA approved the vBloc device (Enteromedics, St. Paul, MN), a vagal nerve blocking device for obesity treatment. The device consists of two electrodes which are laparoscopically placed around the anterior and

posterior vagal nerve trunks at the esophagogastric junction, a subcutaneously placed neuroregulator and an external controller used to communicate with and provide power to the device. The electrodes are connected via leads to the neuroregulator, while the external controller communicates transcutaneously with the implanted neuroregulator through an external coil. The device is intermittently active for about half the day. The aim of reversible vagal blockade is to stop both ascending and descending neural traffic resulting in enhanced satiety, decrease in food intake, and weight loss [102].

The EMPOWER study was a randomized, double-blind, prospective controlled trial conducted in the USA and Australia [103]. When comparing the treated group with the control group at 12 months, there was no difference in overall weight loss measured as EWL (17 % ± 2 % vs. 16 % ± 2 %, p = NS). Similarly, the percentage of subjects attaining an EWL of ≥ 25 % was also not different between groups (22 % vs. 25 %, p = NS). However, treatment group participants who received at least 12 h of vagal block therapy a day achieved the level of weight loss anticipated in the design. Furthermore, a significant dose response of weight loss in relation to hours of device use for both groups coupled with the possibility that control patients may have received partial vagal blockade through low-energy safety or device checks confounded the interpretation of the trial's results.

The ReCharge trial, a subsequent randomized, double-blind, sham-controlled clinical trial involving 239 participants did show a difference in EWL between the two groups (24.4 % vs. 15.9 %, p = 0.002). Furthermore, a larger proportion of patients in the vagal nerve block group achieved 25 % or more EWL compared to the sham group (38 % vs. 23 %). The device, procedure or therapy-related serious adverse event rate in the vagal nerve block group was 3.7 %, which was significantly lower than the 15 % goal. The most common adverse events in the vagal nerve block group were heartburn or dyspepsia and abdominal pain attributed to therapy, all of which were reported as mild or moderate in severity [104].

References

1. Chang S-H, Stoll CRT, Song J, Varela JE, Eagon CJ, Colditz GA. The effectiveness and risks of bariatric surgery: an updated systematic review and meta-analysis, 2003–2012. JAMA Surg. 2014;149(3):275–87. doi:10.1001/jamasurg.2013.3654.
2. Terranova L, Busetto L, Vestri A, Zappa MA. Bariatric surgery: cost-effectiveness and budget impact. Obes Surg. 2012;22(4):646–53. doi:10.1007/s11695-012-0608-1.
3. Buchwald H, Estok R, Fahrbach K, et al. Weight and type 2 diabetes after bariatric surgery: systematic review and meta-analysis. Am J Med. 2009;122(3):248–256.e5. doi:10.1016/j.amjmed.2008.09.041.
4. Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. JAMA. 2004;292(14):1724–37. doi:10.1001/jama.292.14.1724.
5. Mechanick JI, Kushner RF, Sugerman HJ, et al. American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery medical guidelines for clinical practice for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric. Surg Obes Relat Dis. 2008;4(5 Suppl). doi:10.1016/j.soard.2008.08.009.
6. NIH Consensus Development Conference. NUTRITION for severe obesity. Nutrition. 1996;12(6):397–402.
7. World Health Organization. Obesity and overweight. Fact sheet N°311. 2015. http://www.who.int/mediacentre/factsheets/fs311/en/.
8. Lobstein T, Millstone E. Context for the PorGrow study: Europe's obesity crisis. Obes Rev. 2007;8 Suppl 2:7–16. doi:10.1111/j.1467-789X.2007.00354.x.
9. Lifestyles Statistics Team H and SCIC. Statistics on obesity, physical activity and diet. HSCIC Natl Stat. 2015;(March). http://www.hscic.gov.uk/catalogue/PUB16988/obes-phys-acti-diet-eng-2015.pdf.
10. Sturm R, Ph D, Economist S. Morbid obesity rates continue to rise rapidly in the US. Int J Obes. 2013;37(6):889–91. doi:10.1038/ijo.2012.159.Morbid.
11. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. The continuing epidemics of obesity and diabetes in the United States. JAMA. 2001;286(10):1195–200. doi:10.1001/jama.286.10.1195.
12. Leibson C, Wiliamson D, Melton III L, et al. Temporal trends in BMI among adults with diabetes. Diabetes Care. 2001;24(9):1584–9.
13. Lindsay RS, Howard BV. Cardiovascular risk associated with the metabolic syndrome. Curr Diab Rep. 2004;4(1):63–8. doi:10.1007/s11892-004-0013-9.
14. Koh KK, Han SH, Quon MJ. Inflammatory markers and the metabolic syndrome. J Am Coll Cardiol. 2005;46(11):1978–85. doi:10.1016/j.jacc.2005.06.082.
15. Alberti KGMM, Eckel RH, Grundy SM, et al. Harmonizing the metabolic syndrome: a joint interim statement of the international diabetes federation

task force on epidemiology and prevention; national heart, lung, and blood institute; American heart association; world heart federation; international atherosclerosis society; and international association for the study of obesity. Circulation. 2009;120(16):1640–5. doi:10.1161/CIRCULATIONAHA.109.192644.
16. Alberti KGMM, Zimmet P, Shaw J. Metabolic syndrome - a new world-wide definition. A consensus statement from the international diabetes federation. Diabet Med. 2006;23(5):469–80. ISI:000237012700003.
17. Olijhoek JK, van der Graaf Y, Banga J-D, Algra A, Rabelink TJ, Visseren FLJ. The metabolic syndrome is associated with advanced vascular damage in patients with coronary heart disease, stroke, peripheral arterial disease or abdominal aortic aneurysm. Eur Heart J. 2004;25(4):342–8. doi:10.1016/j.ehj.2003.12.007.
18. Moshiri M, Osman S, Robinson TJ, Khandelwal S, Bhargava P, Rohrmann CA. Evolution of bariatric surgery: a historical perspective. Am J Roentgenol. 2013;201(1):W40–8. doi:10.2214/AJR.12.10131.
19. Rucker RD, Horstmann J, Schneider PD, Varco RL, Buchwald H. Comparisons between jejunoileal and gastric bypass operations for morbid obesity. Surgery. 1982;92(2):241–9.
20. Scott HW, Dean RH, Shull HJ, Gluck FW. Metabolic complications of jejunoileal bypass operations for morbid obesity. Annu Rev Med. 1976;27:397–405.
21. Mason EE, Ito C. Gastric bypass in obesity. Surg Clin North Am. 1967;47(6):1345–51.
22. Mason EE, Printen KJ, Hartford CE, Boyd WC. Optimizing results of gastric bypass. Ann Surg. 1975;182(4):405–14.doi:10.1097/00000658-197510000-00006.
23. Alden JF. Gastric and jejunoileal bypass. A comparison in the treatment of morbid obesity. Arch Surg. 1977;112(7):799–806.
24. Griffen WO, Young VL, Stevenson CC. A prospective comparison of gastric and jejunoileal bypass procedures for morbid obesity. Ann Surg. 1977;186(4):500–9.
25. Wittgrove AC, Clark GW, Tremblay LJ. Laparoscopic gastric bypass, Roux-en-Y: preliminary report of five cases. Obes Surg. 1994;4:353–7. doi:10.1381/096089294765558331.
26. De la Torre RA, Scott JS. Laparoscopic Roux-en-Y gastric bypass: a totally intra-abdominal approach--technique and preliminary report. Obes Surg. 1999;9(5):492–8. doi:10.1381/096089299765552800.
27. Higa KD, Boone KB, Ho T, Davies OG. Laparoscopic Roux-en-Y gastric bypass for morbid obesity: technique and preliminary results of our first 400 patients. Arch Surg. 2000;135(9):1029–33. discussion 1033–1034.
28. Fobi MA, Lee H, Holness R, Cabinda D. Gastric bypass operation for obesity. World J Surg. 1998;22(9):925–35.
29. MacLean LD, Rhode BM, Nohr CW. Late outcome of isolated gastric bypass. Ann Surg. 2000;231(4):524–8. doi:10.1097/00000658-200004000-00011.
30. Printen KJ, Mason EE. Gastric surgery for relief of morbid obesity. Arch Surg. 1973;106(4):428–31.
31. Gomez CA. Gastroplasty in morbid obesity. Surg Clin North Am. 1979;59(6):1113–20.
32. Mason EE. Vertical banded gastroplasty for obesity. Arch Surg. 1982;117(5):701–6. http://www.ncbi.nlm.nih.gov/pubmed/7073493.
33. Laws HL, Piantadosi S. Superior gastric reduction procedure for morbid obesity: a prospective, randomized trial. Ann Surg. 1981;193(3):334–40. doi:10.1097/00000658-198103000-00014.
34. MacLean LD, Rhode BM, Forse RA. Late results of vertical banded gastroplasty for morbid and super obesity. Surgery. 1990;107(1):20–7.
35. Hess DW, Hess DS. Laparoscopic vertical banded gastroplasty with complete transection of the staple-line. Obes Surg. 1994;4(1):44–6. doi:10.1381/096089294765558890.
36. Balsiger BM, Poggio JL, Mai J, Kelly KA, Sarr MG. Ten and more years after vertical banded gastroplasty as primary operation for morbid obesity. J Gastrointest Surg. 2000;4(6):598–605.
37. Sugerman HJ, Starkey JV, Birkenhauer R. A randomized prospective trial of gastric bypass versus vertical banded gastroplasty for morbid obesity and their effects on sweets versus non-sweets eaters. Ann Surg.1987;205(6):613–24.doi:10.1097/00000658-198706000-00002.
38. Deitel M. Overview of operations for morbid obesity. World J Surg. 1998;22(9):913–8.
39. Miller K, Höller E, Hell E. Restrictive procedures in the treatment of morbid obesity -- vertical banded gastroplasty vs. adjustable gastric banding. Zentralbl Chir. 2002;127(12):1038–43. doi:10.1055/s-2002-36376.
40. Buchwald H, Buchwald JN. Evolution of operative procedures for the management of morbid obesity 1950–2000. Obes Surg. 2002;12:705–17. doi:10.1381/096089202321019747.
41. Comeau E, Gagner M, Inabnet WB, Herron DM, Quinn TM, Pomp A. Symptomatic internal hernias after laparoscopic bariatric surgery. Surg Endosc. 2005;19(1):34–9. doi:10.1007/s00464-003-8515-0.
42. Puzziferri N, Roshek TB, Mayo HG, Gallagher R, Belle SH, Livingston EH. Long-term follow-up after bariatric surgery. JAMA. 2014;312(9):934. doi:10.1001/jama.2014.10706.
43. Pories WJ, MacDonald KG, Morgan EJ, et al. Surgical treatment of obesity and its effect on diabetes: 10-y follow-up. Am J Clin Nutr. 1992;55(2):582S–5.
44. Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2011. Obes Surg. 2013;23(4):427–36. doi:10.1007/s11695-012-0864-0.
45. Kim S, Richards WO. Long-term follow-up of the metabolic profiles in obese patients with type 2 diabetes mellitus after Roux-en-Y gastric bypass. Ann Surg. 2010;251(6):1049–55. doi:10.1097/SLA.0b013e3181d9769b.
46. Ikramuddin S, Korner J, Lee W, et al. Roux-en-y gastric bypass vs intensive medical management for

the control of type 2 diabetes, hypertension, and hyperlipidemia: the diabetes surgery study randomized clinical trial. JAMA. 2013;309(21):2240–9. doi:10.1001/jama.2013.5835.
47. Liang Z, Wu Q, Chen B, Yu P, Zhao H, Ouyang X. Effect of laparoscopic Roux-en-Y gastric bypass surgery on type 2 diabetes mellitus with hypertension: a randomized controlled trial. Diabetes Res Clin Pract. 2013;101(1):50–6. doi:10.1016/j.diabres.2013.04.005.
48. Schauer PR, Bhatt DL, Kirwan JP, et al. Bariatric surgery versus intensive medical therapy for diabetes — 3-yearoutcomes.NEnglJMed.2014;370(21):2002–13. doi:10.1056/NEJMoa1401329.
49. Mingrone G, Panunzi S, De Gaetano A, et al. Bariatric surgery versus conventional medical therapy for type 2 diabetes. N Engl J Med. 2012;366(17):1577–85. doi:10.1056/NEJMoa1200111.
50. Schauer PR, Kashyap SR, Wolski K, et al. Bariatric surgery versus intensive medical therapy in obese patients with diabetes. N Engl J Med. 2012;366(17):1567–76. doi:10.1056/NEJMoa1200225.
51. Rao W-S, Shan C-X, Zhang W, Jiang D-Z, Qiu M. A meta-analysis of short-term outcomes of patients with type 2 diabetes mellitus and BMI ≤35 kg/m2 undergoing Roux-en-Y gastric bypass. World J Surg. 2015;39(1):223–30. doi:10.1007/s00268-014-2751-4.
52. Mathew A, Veliuona MA, Depalma FJ, Cooney RN. Gastrojejunal stricture after gastric bypass and efficacy of endoscopic intervention. Dig Dis Sci. 2009;54(9):1971–8. doi:10.1007/s10620-008-0581-7.
53. Birkmeyer JD, Finks JF, O'Reilly A, et al. Surgical skill and complication rates after bariatric surgery. N Engl J Med. 2013;369(15):1434–42. doi:10.1056/NEJMsa1300625.
54. Hess DS, Hess DW. Biliopancreatic diversion with a duodenal switch. Obes Surg. 1998;8(3):267–82. doi:10.1381/096089298765554476.
55. Marceau P, Biron S, St Georges R, Duclos M, Potvin M, Bourque RA. Biliopancreatic diversion with gastrectomy as surgical treatment of morbid obesity. Obes Surg. 1991;1(4):381–7.
56. Hess DS, Hess DW, Oakley RS. The biliopancreatic diversion with the duodenal switch: results beyond 10 years. Obes Surg. 2005;15(3):408–16. doi:10.1381/0960892053576695.
57. Ren CJ, Patterson E, Gagner M. Early results of laparoscopic biliopancreatic diversion with duodenal switch: a case series of 40 consecutive patients. Obes Surg. 2000;10(6):514–23. doi:10.1381/096089200321593715.
58. Regan JP, Inabnet WB, Gagner M, Pomp A. Early experience with two-stage laparoscopic Roux-en-Y gastric bypass as an alternative in the super-super obese patient. Obes Surg. 2003;13(6):861–4. doi:10.1381/096089203322618669.
59. Cottam D, Qureshi FG, Mattar SG, et al. Laparoscopic sleeve gastrectomy as an initial weight-loss procedure for high-risk patients with morbid obesity. Surg Endosc. 2006;20(6):859–63. doi:10.1007/s00464-005-0134-5.
60. Almogy G, Crookes PF, Anthone GJ. Longitudinal gastrectomy as a treatment for the high-risk super-obese patient. Obes Surg. 2004;14:492–7. doi:10.1381/096089204323013479.
61. Roa PE, Kaidar-Person O, Pinto D, Cho M, Szomstein S, Rosenthal RJ. Laparoscopic sleeve gastrectomy as treatment for morbid obesity: technique and short-term outcome. Obes Surg. 2006;16(10):1323–6. doi:10.1381/096089206778663869.
62. Tucker ON, Szomstein S, Rosenthal RJ. Indications for sleeve gastrectomy as a primary procedure for weight loss in the morbidly obese. J Gastrointest Surg. 2008;12(4):662–7. doi:10.1007/s11605-008-0480-4.
63. Gagner M, Deitel M, Erickson AL, Crosby RD. Survey on laparoscopic sleeve gastrectomy (LSG) at the fourth international consensus summit on sleeve gastrectomy. Obes Surg. 2013;23(12):2013–7. doi:10.1007/s11695-013-1040-x.
64. Rosenthal RJ, International Sleeve Gastrectomy Expert Panel, Diaz AA, Arvidsson D, Baker RS, Basso N, et al. International sleeve gastrectomy expert panel consensus statement: best practice guidelines based on experience of >12,000 cases. Surg Obes Relat Dis. 2012;8(1):8–19.
65. Deitel M, Gagner M, Erickson AL, Crosby RD. Third international summit: current status of sleeve gastrectomy. Surg Obes Relat Dis. 2011;7(6):749–59. doi:10.1016/j.soard.2011.07.017.
66. Parikh M, Issa R, McCrillis A, Saunders JK, Ude-Welcome A, Gagner M. Surgical strategies that may decrease leak after laparoscopic sleeve gastrectomy: a systematic review and meta-analysis of 9991 cases. Ann Surg. 2013;257(2):231–7. doi:10.1097/SLA.0b013e31826cc714.
67. Trastulli S, Desiderio J, Guarino S, et al. Laparoscopic sleeve gastrectomy compared with other bariatric surgical procedures: a systematic review of randomized trials. Surg Obes Relat Dis. 2013;9(5):816–29. doi:10.1016/j.soard.2013.05.007.
68. Diamantis T, Apostolou KG, Alexandrou A, Griniatsos J, Felekouras E, Tsigris C. Review of long-term weight loss results after laparoscopic sleeve gastrectomy. Surg Obes Relat Dis. 2014;10(1):177–83. doi:10.1016/j.soard.2013.11.007.
69. Leonetti F, Capoccia D, Coccia F, et al. Obesity, type 2 diabetes mellitus, and other comorbidities: a prospective cohort study of laparoscopic sleeve gastrectomy vs medical treatment. Arch Surg. 2012;147(8):694–700. doi:10.1001/archsurg.2012.222.
70. Lee W-J, Ser K-H, Chong K, et al. Laparoscopic sleeve gastrectomy for diabetes treatment in non-morbidly obese patients: efficacy and change of insulin secretion. Surgery. 2010;147(5):664–9. doi:10.1016/j.surg.2009.10.059.
71. Rosenthal R, Li X, Samuel S, Martinez P, Zheng C. Effect of sleeve gastrectomy on patients with diabetes mellitus. Surg Obes Relat Dis. 2009;5(4):429–34. doi:10.1016/j.soard.2008.11.006.
72. 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(11):1450–6. doi:10.1381/096089206778869933.
73. Gagner M, Buchwald JN. Comparison of laparoscopic sleeve gastrectomy leak rates in four staple-line reinforcement options: a systematic review. Surg Obes Relat Dis. 2014;10(4):1–11. doi:10.1016/j.soard.2014.01.016.
74. Broadbent R, Tracey M, Harrington P. Laparoscopic gastric banding: a preliminary report. Obes Surg. 1993;3(1):63–7. doi:10.1381/096089293765559791.
75. Catona A, Gossenberg M, La Manna A, Mussini G. Laparoscopic gastric banding: preliminary series. Obes Surg. 1993;3(2):207–9.
76. Forsell P, Hallberg D, Hellers G. Gastric banding for morbid obesity: initial experience with a new adjustable band. Obes Surg. 1993;3(4):369–74. doi:10.1381/096089293765559052.
77. Belachew M, Jacqet P, Lardinois F, Karler C. Vertical banded gastroplasty vs adjustable silicone gastric banding in the treatment of morbid obesity: a preliminary report. Obes Surg. 1993;3(3):275–8.
78. Belachew M, Legrand MJ, Vincent V. History of lapband: from dream to reality. Obes Surg. 2001;11:297–302. doi:10.1381/096089201321336638.
79. O'Brien PE, MacDonald L, Anderson M, Brennan L, Brown WA. Long-term outcomes after bariatric surgery: fifteen-year follow-up of adjustable gastric banding and a systematic review of the bariatric surgical literature. Ann Surg. 2013;257(1):87–94. doi:10.1097/SLA.0b013e31827b6c02.
80. Colquitt JL, Pickett K, Loveman E, Frampton GK. Surgery for weight loss in adults. Cochrane Database Syst Rev. 2014;8(8), CD003641. doi:10.1002/14651858.CD003641.pub4.
81. O'Brien PE, Dixon JB, Laurie C, et al. Treatment of mild to moderate obesity with laparoscopic adjustable gastric banding or an intensive medical program: a randomized trial. Ann Intern Med. 2006;144(9):625–33.
82. Dixon JB, O'Brien PE, Playfair J, et al. Adjustable gastric banding and conventional therapy for type 2 diabetes: a randomized controlled trial. JAMA. 2008;299(3):316–23. doi:10.1001/jama.299.3.316.
83. Courcoulas AP, Goodpaster BH, Eagleton JK, et al. Surgical vs medical treatments for type 2 diabetes mellitus: a randomized clinical trial. JAMA Surg. 2014. doi:10.1001/jamasurg.2014.467.
84. Egan RJ, Monkhouse SJW, Meredith HE, Bates SE, Morgan JDT, Norton SA. The reporting of gastric band slip and related complications; a review of the literature. Obes Surg. 2011;21(8):1280–8. doi:10.1007/s11695-010-0344-3.
85. Scopinaro N, Gianetta E, Civalleri D, Bonalumi U, Bachi V. Bilio-pancreatic bypass for obesity: II. Initial experience in man. Br J Surg. 1979;66(9):618–20. doi:10.1002/bjs.1800660906.
86. Scopinaro N. Comments to presidential address: gastric bypass and biliopancreatic diversion operations. Obes Surg. 2002;12(6):881–3. doi:10.1381/096089202320995763. author reply 884.
87. Marceau P, Biron S, Bourque RA, Potvin M, Hould FS, Simard S. Biliopancreatic diversion with a new type of gastrectomy. Obes Surg. 1993;3(1):29–35. doi:10.1381/096089293765559728.
88. Topart P, Becouarn G, Salle A. Five-year follow-up after biliopancreatic diversion with duodenal switch. Surg Obes Relat Dis. 2011;7(2):199–205. doi:10.1016/j.soard.2010.10.017.
89. Crea N, Pata G, Di Betta E, et al. Long-term results of biliopancreatic diversion with or without gastric preservation for morbid obesity. Obes Surg. 2011;21(2):139–45. doi:10.1007/s11695-010-0333-6.
90. Nelson DW, Blair KS, Martin MJ. ANalysis of obesity-related outcomes and bariatric failure rates with the duodenal switch vs gastric bypass for morbid obesity. Arch Surg. 2012;147(9):847–54. doi:10.1001/archsurg.2012.1654.
91. Baltasar A, Bou R, Bengochea M, et al. Duodenal switch: an effective therapy for morbid obesity - intermediate results. Obes Surg. 2001;11(1):54–8. doi:10.1381/096089201321454114.
92. Majumder S, Birk J. A review of the current status of endoluminal therapy as a primary approach to obesity management. Surg Endosc. 2013;27(7):2305–11. doi:10.1007/s00464-012-2765-7.
93. Štimac D, Majanović SK. Endoscopic approaches to obesity. Dig Dis. 2012;30(2):187–95. doi:10.1159/000336683.
94. Ponce J, Quebbemann BB, Patterson EJ. Prospective, randomized, multicenter study evaluating safety and efficacy of intragastric dual-balloon in obesity. Surg Obes Relat Dis. 2012;9(2):290–5. doi:10.1016/j.soard.2012.07.007.
95. Genco A, Bruni T, Doldi SB, et al. BioEnterics intragastric balloon: the Italian experience with 2,515 patients. Obes Surg. 2005;15(8):1161–4. doi:10.1381/0960892055002202.
96. Imaz I, Martínez-Cervell C, García-Álvarez EE, Sendra-Gutiérrez JM, González-Enríquez J. Safety and effectiveness of the intragastric balloon for obesity. A meta-analysis. Obes Surg. 2008;18(7):841–6. doi:10.1007/s11695-007-9331-8.
97. Spyropoulos C, Katsakoulis E, Mead N, Vagenas K, Kalfarentzos F. Intragastric balloon for high-risk super-obese patients: a prospective analysis of efficacy. Surg Obes Relat Dis. 2007;3(1):78–83. doi:10.1016/j.soard.2006.11.001.
98. Dastis SN, François E, Deviere J, et al. Intragastric balloon for weight loss: results in 100 individuals followed for at least 2.5 years. Endoscopy. 2009;41(7):575–80. doi:10.1055/s-0029-1214826.
99. Schouten R, Rijs CS, Bouvy ND, et al. A multicenter, randomized efficacy study of the EndoBarrier Gastrointestinal Liner for presurgical weight loss prior to bariatric surgery. Ann Surg. 2010;251(2):236–43. doi:10.1097/SLA.0b013e3181bdfbff.
100. Rodriguez-Grunert L, Galvao Neto MP, Alamo M, Ramos AC, Baez PB, Tarnoff M. First human experience with endoscopically delivered and retrieved duodenal-jejunal bypass sleeve. Surg Obes Relat Dis. 2008;4(1):55–9. doi:10.1016/j.soard.2007.07.012.

101. Koehestanie P, de Jonge C, Berends FJ, Janssen IM, Bouvy ND, Greve JWM. The effect of the endoscopic duodenal-jejunal bypass liner on obesity and type 2 diabetes mellitus, a multicenter randomized controlled trial. Ann Surg. 2014;260(6):984–92. doi:10.1097/SLA.0000000000000794.
102. Camilleri M, Toouli J, Herrera MF, et al. Intra-abdominal vagal blocking (VBLOC therapy): clinical results with a new implantable medical device. Surgery. 2008;143(6):723–31. doi:10.1016/j.surg.2008.03.015.
103. Sarr MG, Billington CJ, Brancatisano R, et al. The EMPOWER study: randomized, prospective, double-blind, multicenter trial of vagal blockade to induce weight loss in morbid obesity. Obes Surg.2012;22(11):1771–82.doi:10.1007/s11695-012-0751-8.
104. Ikramuddin S, Blackstone RP, Brancatisano A, et al. Effect of reversible intermittent intra-abdominal vagal nerve blockade on morbid obesity. JAMA. 2014;312(9):915. doi:10.1001/jama.2014.10540.

Anesthesia for the Bariatric Patient: Optimizing Safety and Managing Complications

2

Haobo Ma and Stephanie Jones

Patients undergoing bariatric surgery present special challenges to the anesthesiologist during perioperative management. Morbid obesity brings changes to patient anatomy and physiology, requiring meticulous preoperative assessment and planning to ensure safety intraoperatively and in the postoperative anesthesia care unit (PACU). Morbid obesity is associated with increased difficulty in airway management and ventilation. Careful airway assessment, experienced anesthesia personnel, and appropriate equipment are necessary for patient safety. A thorough understanding of pharmacology in morbidly obesity patients is also important. For example, an increased volume of distribution and amount of adipose tissue bring changes in drug pharmacokinetics, requiring adjusted dosing regimens of anesthetic induction agents, opioids, and muscle relaxants. Patients with morbid obesity also have increased respiratory complications during the postoperative period. Multimodal pain management can improve pain control as well as reduce respiratory complications. In this chapter, we discuss various aspects of anesthesia management with particular attention to increasing safety and reducing complications.

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2.1 Airway Management

Obese patients require special consideration in airway management. In the supine position, obese patients have decreased functional residual capacity resulting in lower tolerance of prolonged apnea [1]. Obesity, defined as a body mass index ≥ 30 kg/m^2, is an independent predictor of difficult mask ventilation [2]. Difficult intubation is also more common in obese than nonobese patients and oxygen desaturation during induction and intubation is more pronounced in obese patients [3]. A thorough history and airway evaluation to identify risks and predictors for difficult mask ventilation or tracheal intubation is important to facilitate planning for an appropriate airway management strategy.

Many medical and surgical conditions may affect airway management. Anesthesiologists need to carefully review the patient's past medical history to identify conditions such as previously difficult laryngoscopy or intubation, obstructive sleep apnea (OSA), oropharyngeal pathologies, or prior neck radiation. Morbidly obese patients frequently have a history of OSA, which is one of the predictors for difficult airway management. The STOP-BANG questionnaire (Snoring, Tiredness during daytime, Observed apnea, high

D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_2

blood Pressure, Body mass index >35 kg/m^2, Age, Neck circumference, and Gender) is a validated concise screening tool for OSA [4].

2.2 Mask Ventilation

Analysis of a large anesthesia record database identified predictors for difficult mask ventilation including body mass index (BMI) of 30 kg/m^2 or greater, beard, Mallampati class III or IV, age ≥57, limited jaw protrusion, snoring, and sleep apnea [2]. The presence of a beard is the only easily modifiable independent risk factor for difficult mask ventilation. Other predictors of difficult mask ventilation include neck radiation changes [5] and lack of teeth [6].

Placing the patient in sniffing position by extending the upper cervical spine and atlantooccipital joint while flexing the lower cervical spine can be utilized to assist mask ventilation. Placement of an oral airway and two-hand mask ventilation are other useful interventions. Reverse Trendelenburg position can shift weight caudad and facilitate diaphragmatic movement during mask ventilation. If ventilation via face mask is difficult, placement of a laryngeal mask airway may be attempted subsequently, if the patient is not yet ready for intubation or is unexpectedly difficult to intubate. When difficulty is encountered at any step of airway management, anesthesiologists should follow the American Society of Anesthesiologists (ASA) Difficult Airway Algorithm (Fig. 2.1).

2.3 Intubation

The ASA recommends components of a preoperative airway physical exam [7]. Table 2.1 displays some findings of the airway physical examination that may suggest the presence of a difficult intubation. BMI alone is not a predictor of difficult intubation [8]. A Mallampati score of III or IV and large neck circumference increase the potential for difficult intubation [9]. Limited jaw protrusion is also a predictor of difficult intubation [2]. Assessment of Mallampati classification when the patient's craniocervical junction is extended gives a better predictive value for difficult intubation in the morbidly obese population [10].

2.4 Optimizing Position and Preoxygenation

Obese patients have decreased functional residual capacity (FRC), reduced oxygen supply, and increased risk of developing hypoxemia when apneic [11, 12]. Preoxygenation in reverse Trendelenburg (head up) position extends the period prior to desaturation, allowing more time for intubation and airway control [13, 14]. Reverse Trendelenburg position also improves intraoperative oxygenation if appropriate from a surgical perspective [15]. A footboard will prevent the patient from sliding downward while in reverse Trendelenburg position. In addition, sniffing position can facilitate tracheal intubation [16].

Preoxygenation with 100 % O_2 either with 3-min tidal volume breathing or four vital capacity breaths provides equal and adequate arterial oxygenation for rapid sequence induction and intubation in the morbidly obese patient [17]. However, Gambee et al. [18] found apneic patients developed desaturation more rapidly after preoxygenation with four vital capacity breaths than did patients preoxygenated for 3 min. Thus tidal volume breathing for 3–5 min or eight vital capacity breathing over 60 s is recommended. The goal of preoxygenation is to achieve an end-tidal oxygenation concentration >90 %.

Application of continuous positive airway pressure (CPAP) of 10 cm H_2O during preoxygenation and positive end-expiratory pressure (PEEP) of 10 cm H_2O during mask ventilation after the induction of anesthesia can reduce atelectasis formation in morbidly obese patients and improve oxygenation [19]. This is a useful technique to prolong time to desaturation [20].

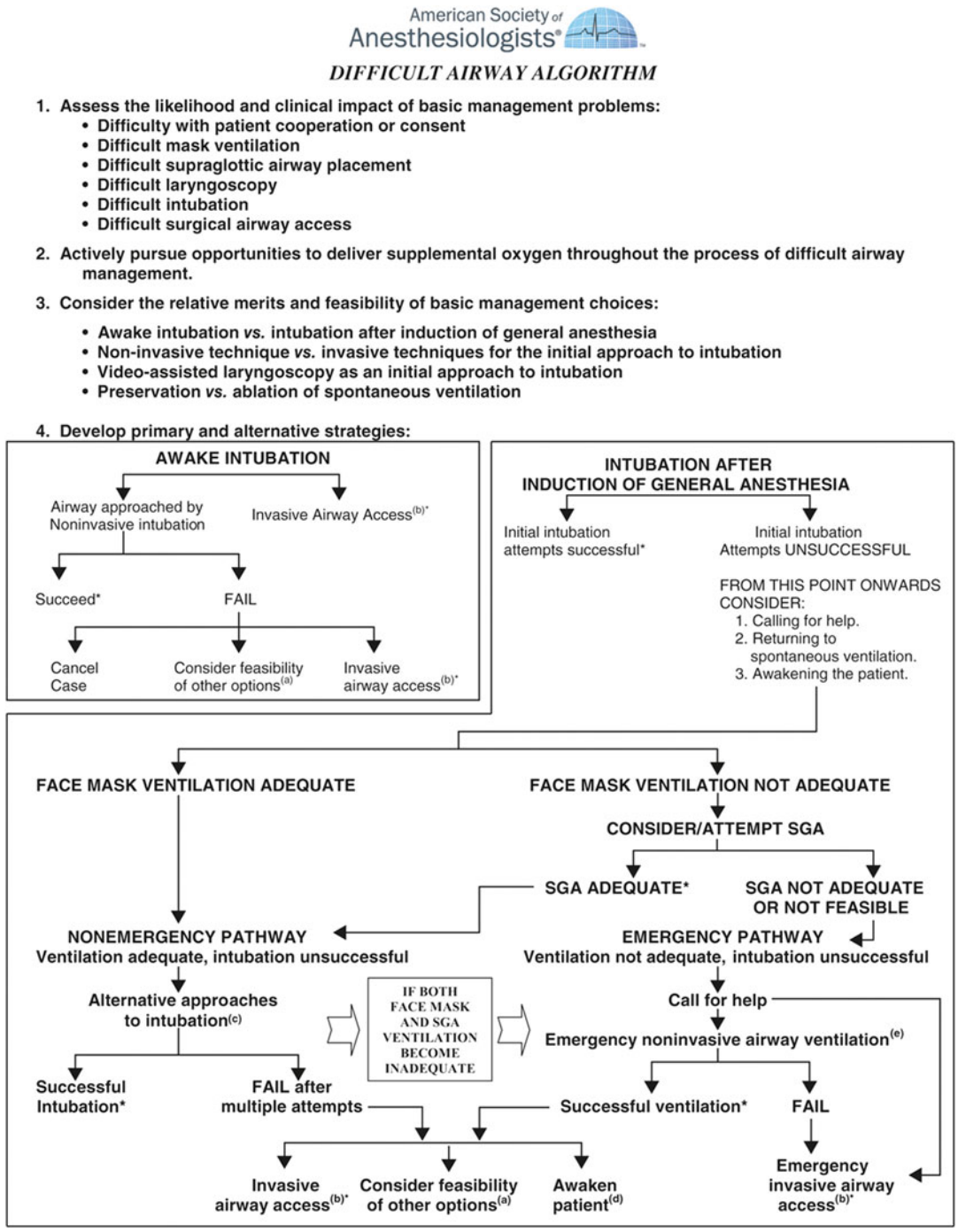

Fig. 2.1 American Society of Anesthesiologists Difficult Airway Algorithm [64] © Wolters Kluwer with permission

Table 2.1 Components of the preoperative airway physical examination [64]

Airway examination component	Nonreassuring findings
Length of upper incisors	Relatively long
Relationship of maxillary and mandibular incisors	Prominent "overbite" (maxillary incisors anterior to mandibular incisors)
Relationship of maxillary and mandibular incisors during voluntary protrusion of mandible	Patient cannot bring mandibular incisors anterior to maxillary incisors
Interincisor distance	Less than 3 cm
Visibility of uvula	Not visible when tongue is protruded with patient in sitting position (e.g., Mallampati class >2)
Shape of palate	Highly arched or very narrow
Compliance of mandibular space	Stiff, indurated, occupied by mass, or nonresilient
Thyromental distance	Less than three ordinary finger breadths
Length of neck	Short
Thickness of neck	Thick
Range of motion of head and neck	Patient cannot touch tip of chin to chest or cannot extend neck

2.5 Laryngeal Mask Airway

The laryngeal mask airway (LMA) is an important element of the difficult airway management algorithm [7] as a rescue option if face mask ventilation is not adequate. Even though most anesthesiologists feel the LMA is not appropriate to use as a definitive airway during bariatric surgery due to aspiration risk, the intubating LMA serves as an effective conduit for intubation. Frappier et al. used the intubating LMA in morbidly obese patients with a 96.3 % success rate [21]. Patients requiring rapid sequence induction were excluded from this study.

2.6 Choice of Laryngoscope

Choices for laryngoscopy include direct laryngoscopy with standard Macintosh or Miller blades, video laryngoscopes such as the Glidescope®, and flexible fiber-optic bronchoscopes. Selection among those devices is based on preoperative airway assessment. Video laryngoscopy improves intubation conditions in the morbidly obese patient [22]. If a non-video laryngoscope is chosen as the first line equipment, it is prudent to have a video laryngoscope readily available as backup.

Awake flexible fiber-optic laryngoscopy remains the gold standard for managing difficult airways. The patient with a history of failed intubation, upper airway abnormality, or with an expected difficult intubation from another cause may benefit from an awake fiber-optic intubation. Awake fiber-optic intubation requires an experienced operator. Proper topical anesthesia and careful sedation is key for success.

2.7 Extubation

A thorough evaluation of readiness for extubation must be performed at the conclusion of the anesthetic. There should be no ongoing indication to keep the patient intubated, such as hemodynamic instability. For patients who are difficult to intubate, proper timing of extubation, equipment availability, and the presence of skilled anesthesia providers are all vital for safe extubation. Before extubation, the following criteria should be met: spontaneous ventilation must be adequate, muscle relaxant fully reversed, airway reflexes fully recovered and the patient is following commands. If the intubation was difficult, the presence of a cuff leak should be documented prior to extubation to rule out glottic swelling due to airway trauma. To increase FRC before extubation, the patient should be placed in the reverse Trendelenburg position. For patients with OSA, extubation directly to noninvasive positive pressure ventilation may reduce airway obstruction and improve respiratory function [23].

2.8 Intraoperative Management

2.8.1 Intraoperative Monitoring

Standard intraoperative anesthesia monitoring requires continuous evaluation of oxygenation, ventilation, circulation, and temperature. Pulse

oximetry is used for the monitoring of oxygenation during the perioperative period and should be maintained as long as patients remain at increased risk for airway compromise [24]. Capnography is used to monitor ventilation intraoperatively. End-tidal carbon dioxide ($ETCO_2$) may not accurately reflect arterial carbon dioxide tension ($PaCO_2$) due to ventilation–perfusion mismatch in severely obese patients. Transcutaneous CO_2 ($TcCO_2$) monitoring may provide an alternative to end-tidal carbon dioxide monitoring, and has been shown to be more accurate than $ETCO_2$ at estimating $PaCO_2$ [25, 26].

Choosing a properly sized blood pressure cuff is important for accurate blood pressure measurement. The blood pressure cuff bladder width and length should be approximately 40 % and 80 % of the upper arm circumference, respectively. Blood pressure may be measured on the forearm if the upper arm is not anatomically amenable to blood pressure cuff placement. The decision to place an invasive monitor such as an arterial or central venous catheter should be guided by the clinical circumstances and comorbidities such as cerebral vascular disease, coronary artery disease, or pulmonary hypertension.

Obese patients are at increased risk for postoperative respiratory complications. Full reversal of neuromuscular blockade should be verified via both qualitative and quantitative measures. Instead of conventional qualitative train-of-four monitoring, intraoperative acceleromyography monitoring reduces the incidence of residual blockade and respiratory complications in the post anesthesia care unit [27].

Hypothermia has been associated with several perioperative complications, including wound infection, cardiac events, immune dysfunction, coagulopathy, and increased blood loss [28]. Body temperature should be measured during all bariatric operations. Forced air warming can be used to maintain normothermia intraoperatively.

2.8.2 Intraoperative Ventilation

Futier et al. demonstrated that using intraoperative low tidal volume lung protective ventilation in abdominal surgery reduced 7-day postoperative respiratory complications when compared with nonprotective ventilation [29]. The lung protective ventilation group used a tidal volume of 6–8 ml per kilogram of predicted body weight, PEEP of 6–8 cmH_2O, and recruitment maneuvers every 30 min. Chalhoub et al. used recruitment maneuvers following by PEEP in morbidly obese patients undergoing bariatric surgery and showed improvement in arterial oxygenation [30]. Erlandsson et al. used electric impedance tomography to optimize PEEP in morbidly obese patients. The PEEP level determined to prevent lung collapse and to improve gas exchange in morbidly obese patients in this study was around 15 cm H_2O. Based on current literature, low tidal volume based on predicted body weight, PEEP, and recruitment maneuvers are the three components to optimize intraoperative ventilation. Adjustments should be based on an assessment of ETCO2, oxygenation, hemodynamics, and patient volume status.

2.8.3 Intraoperative Positioning and Preventing Nerve Injury

Appropriate positioning of the morbidly obese patient is important for both the patient and the operating room staff. Attention should be paid to the weight limit of the operating table as the maximum allowable weight may vary dependent upon the orientation of the table. It may be necessary to use table extenders that attach to the siderails to accommodate the largest patients. Commercially available devices such as the HoverMatt® (HoverTech International, Bethlehem, PA) can be used to facilitate transfer of the obese patient between the operating room table and hospital bed. It is also critical to have adequate personnel to assist in moving and posi-

tioning morbidly obese patients. To minimize the risk of fall from the operating room table, straps and a footboard should be used for security. The use of a footboard is helpful to avoid downward sliding with reverse Trendelenburg position during induction, surgery (if indicated), and emergence. Given the higher risk of nerve injury in the obese population, all pressure points need to be adequately padded during surgery. Padding may include gel pads, foam, air-filled pads, or other padding materials.

2.9 Perioperative Drug Dosing

Identifying the appropriate dose of anesthetic medications can be challenging with obese patients. For the morbidly obese patient, dosing based on actual body weight overestimates requirements for fentanyl [31], cisatracurium [32], and rocuronium [33]. Different agents are dosed based on different body weight scalars such as total body weight (TBW), lean body weight (LBW), or ideal body weight (IBW), depending on their pharmacokinetic characteristics. Different phases of anesthesia, such as induction and maintenance, may require using different scalars in dosing calculations as well. For medications with limited pharmacokinetic data, anesthesiologists can begin dosing closer to the patient's estimated lean body mass (about 120 % of ideal body weight) and adjust as needed [34] (Tables 2.2 and 2.3).

Table 2.2 Body weight calculations

Body weight	Formula
Ideal body weight (IBW) [65]	Male: IBW = 50 kg + 2.3 kg/each inch above 5 feet Female: IBW = 50 kg + 2.3 kg/each inch above 5 feet
Lean body weight (LBW) [66]	Male: LBW = 0.33 × weight (kg) + 0.34 × height (cm) − 29.53 Female: LBW = 0.30 × weight (kg) + 0.42 × height (cm) − 43.30
Total body weight (TBW)	The patient actual body weight

Table 2.3 Weight-based dosing scalar recommendation for commonly used IV anesthetics [67]

Drug	Dosing scalar
Thiopental	Induction: LBM Maintenance: TBW
Propofol	Induction: LBM Maintenance: TBW
Fentanyl	LBM
Remifentanil	LBM
Succinylcholine	TBW
Vecuronium	IBW
Rocuronium	IBW
Cisatracurium	IBW

LBM lean body mass, *TBW* total body weight, *IBW* ideal body weight

2.10 Perioperative Pain Management

Because of respiratory depression and other side effects associated with opioids, multimodal pain management with minimized opioid use may be a better strategy in morbidly obese patients undergoing bariatric surgery [35]. A multimodal regimen works at different targets, from central to peripheral levels (Fig. 2.2). The use of multiple drugs, analgesic or adjuvant, in combination with opioids, achieves the best pain relief in obese patients during their postoperative course while minimizing side effects of opioids. Nonopioid analgesic options include NSAIDs, acetaminophen, alpha-2 agonists, NMDA receptor antagonists, magnesium, and neuropathic pain medications such as pregabalin or gabapentin. Feld et al. [36] used a nonopioid regimen including ketorolac, clonidine, lidocaine, ketamine, magnesium sulfate, and methylprednisolone. Nonopioid treated patients required less supplemental morphine PCA use and were also less sedated. However, more systematic studies are required before we can recommend specific protocols of multimodal pain management for bariatric surgery patients (see Fig. 2.2).

Local anesthetics via various delivery methods such as transversus abdominis plane (TAP) block or surgical site infusion can significantly reduce incisional pain. Spinal or epidural analgesia

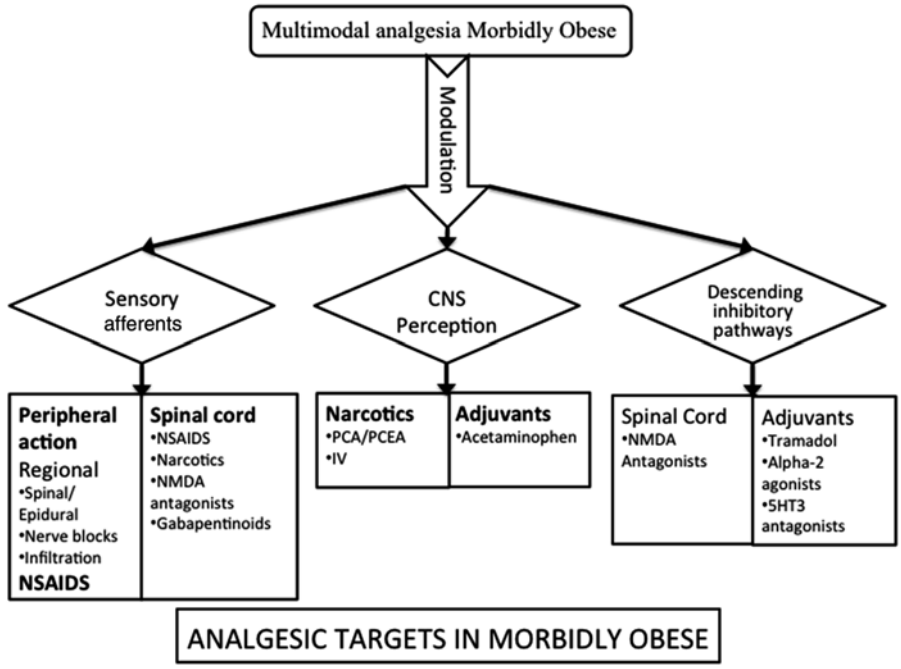

Fig. 2.2 Mutilmodal analgesic targets in morbidly obese patients [35]

provides excellent pain control for patients undergoing open bariatric surgery.

2.11 Nonopioid Analgesics

2.11.1 NSAIDs

Both selective and non-selective cyclooxygenase II (COX-II) inhibitors may be used in multimodal pain regimens. Govindarajan et al. [37] demonstrated a significant reduction in narcotic requirements with ketorolac during the first 24-h postoperatively in morbidly obese patients undergoing laparoscopic surgery. Ketorolac provides comparable postoperative pain relief to fentanyl while lowering the incidence of nausea and sedation [38]. Gastric perforation has been reported in the morbidly obese with prolonged use of non-selective COX inhibitors. Cox-II inhibitors have been advocated if long-term analgesics are necessary [39]. NSAIDs are most successfully used as a component of combination therapy rather than sole analgesics in morbidly obese individuals.

2.11.2 Acetaminophen

Acetaminophen is a centrally acting analgesic without sedative effect and with significant opioid sparing ability [40]. Obesity does not seem to alter acetaminophen pharmacokinetics. The dosage of acetaminophen should be based on ideal body weight. Acetaminophen doses at 6-h intervals can be used safely in the absence of other contraindications. A combination of acetaminophen and NSAIDs has been shown to be superior to either single therapy for managing mild to moderate postoperative pain.

2.11.3 Alpha-2 Agonists

Clonidine and dexmedetomidine are alpha-2 agonists with analgesic properties. Dexmedetomidine is more effective than clonidine in analgesia due to its increased selectivity for alpha-2A receptors. Dexmedetomidine can maintain airway tone and respiratory drive, making it is a good choice in morbidly obese patients. It lowers intraoperative

analgesic requirements and suppresses postoperative nausea vomiting (PONV) when run as an infusion at 0.2–0.8 mcg/kg/min [41]. Clonidine and dexmedetomidine can decrease postoperative rescue analgesic requirements by 25 % and 30 %, respectively, in morbidly obese patients [42].

2.11.4 Ketamine

Ketamine is a N-methyl-D-aspartate (NMDA) antagonist, which prevents glutamate action in pain transmission. It also reverses opioid induced hyperalgesia. Ketamine used as an analgesic adjuvant can reduce opioid use [36]. Hallucinatory side-effects can be avoided with a 0.5 mg/kg bolus dose followed by a continuous infusion of 2.0–2.5 mcg/kg/min for the first 48 h postoperatively [43].

2.11.5 Magnesium

Magnesium, by blocking NMDA receptors (at sites other than those blocked by ketamine) is known to reduce both intraoperative and postoperative analgesic requirements. Ryu et al. [44] added magnesium at a dose of 50 mg/kg at the time of induction and reported improved quality of postoperative analgesia in gynecological surgery cases.

2.11.6 Pregabalin and Gabapentin

Pregabalin and gabapentin inhibit calcium currents via high-voltage-activated channels, reducing neurotransmitter release and attenuating postsynaptic excitability. They are successfully used for chronic pain treatment. A large number of clinical trials indicate that pregabalin and gabapentin can be effective as postoperative analgesics. One oral dose of pregabalin 150 mg 2 h before laparoscopic sleeve gastrectomy reduced 24-h morphine use postoperatively [45]. One dose of gabapentin 600 mg 1 h before general anesthesia reduced opioid consumption in various surgeries [46]. Because of the sedative effect, pregabalin and gabapentin should be used cautiously in elderly patients. Both are eliminated solely by renal clearance and dosage must be adjusted in renally impaired patients.

2.11.7 Regional Blocks

The transversus abdominis plane (TAP) block is particularly effective in blocking T10 to L1 segments and is an attractive option for laparoscopic or open abdominal surgery. Bilateral TAP block in the nonobese patient has shown efficacy with midline incisions [47]. It can be used as a rescue option in situations of failed/difficult epidural analgesia in open bariatric surgery patients. Ultrasound guidance may reduce the challenge of performing TAP block in morbidly obese patients.

Infusion of local anesthetic at the surgical wound site is another convenient analgesia option for bariatric surgery. Both continuous flow devices and patient controlled pumps can be used. A bupivacaine pump has been found to reduce the use of opioids in morbidly obese undergoing laparoscopic bariatric surgery although its use is somewhat controversial [48].

Infusion of intraperitoneal 0.375 % bupivacaine for morbidly obese patients undergoing laparoscopic gastric banding showed significant pain score reduction postoperatively [49]. In a retrospective review, postoperative morphine use after Roux-en-Y bypass surgery was significantly lower in the bupivacaine intraperitoneal group than the control group [50].

2.11.8 Neuraxial Blocks

Neuraxial blocks such as spinal analgesia and epidural analgesia provide excellent pain control with less systemic side effects than intravenous opioids. They are often used in patients undergoing open bariatric surgery.

Michaloudis et al. [51] studied the use of continuous spinal analgesia for morbidly obese patients. Isobaric 0.5 % bupivacaine was used intraoperatively. Postoperatively, patients

received spinal analgesia as part of the pain regimen via a patient controlled intrathecal analgesia device (PCIA). The postoperative intrathecal solution contained bupivacaine 0.05 % and fentanyl 10 mcg/ml. The PCIA was discontinued on the fifth postoperative day. None of the subjects developed postdural puncture headache. The major complication was motor block.

Epidural analgesia use can reduce the use of postoperative opioids. Lowering opioid use is associated with less respiratory complications [52]. The morbidly obese patient may require a lower dose and volume of local anesthetic based on experiences in the obese obstetric population [53]. The increased depth from skin to epidural space adds challenge to epidural placement in morbidly obese patients. Ultrasound can provide visualization and measurement of depth of spinal structures (Fig. 2.3). The success of catheter placement in the obese population increases with ultrasound guidance [54] (see Fig. 2.3).

2.12 Postoperative Nausea and Vomiting (PONV)

Control of nausea and vomiting after bariatric surgery is an important consideration for patient comfort. A review in 2001 did not identify increased BMI as a risk factor for PONV [55]. However, a more recent study showed up to 42.7 % patients required antiemetic rescue medication after undergoing bariatric surgery even after triple PONV prophylaxis with dexamethasone, ondansetron, and scopolamine patch [56]. Ziemann-Gimmel et al. [57] used opioid-free total intravenous anesthesia (TIVA) for patients undergoing bariatric surgery and significantly reduced PONV incidence. The TIVA regimen included dexmedetomidine and propofol infusions during surgery and a ketamine bolus before incision. Postoperative multimodal pain management adding IV acetaminophen and IV ketorolac to an opioid regimen can further reduce the risk of PONV.

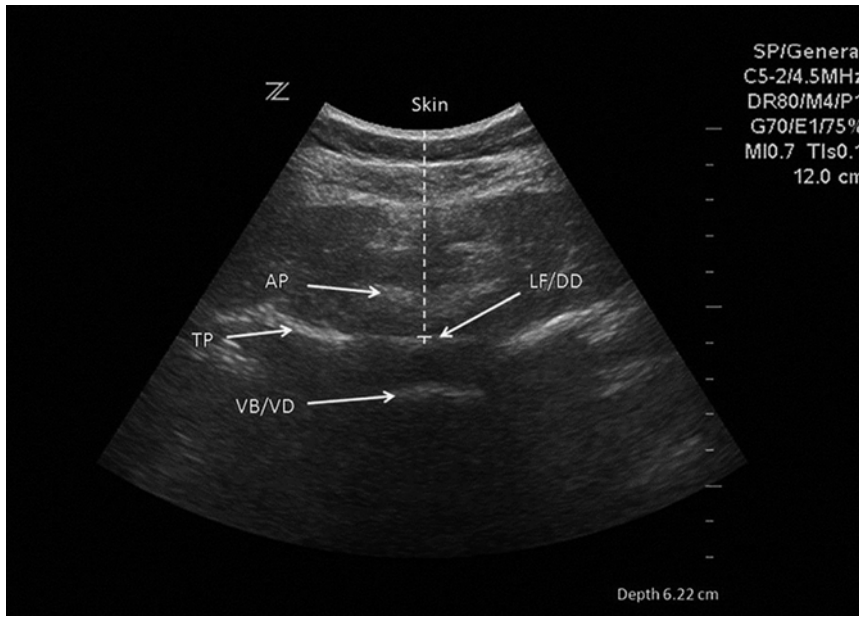

Fig. 2.3 Ultrasound imaging showing the ligamentum flavum-dura unit and the vertebral body/ventral dura. *LF* ligamentum flavum, *DD* dorsal dura, *VB* vertebral body, *VD* ventral dura, *AP* articular process, *TP* transverse process [68]

2.13 Respiratory Complications

Morbidly obese patients have a significantly higher risk of postoperative respiratory complications [58] such as hypoventilation and desaturation [59] due to atelectasis, sedation, and obstructive sleep apnea (OSA). OSA is a very common comorbidity in morbidly obese patients and is associated with increased risk of postoperative respiratory depression. Patients undergoing bariatric surgery require close monitoring in the post-anesthesia care unit (PACU). Even though the literature is insufficient to offer guidance regarding the appropriate duration of postoperative respiratory monitoring, continuous monitoring should be maintained as long as patients remain at increased risk. The proper postoperative respiratory management plan should include monitoring, avoiding supine position, supplemental oxygen, respiratory therapy, multimodal pain management, and use of continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) for OSA patients. Continuous postoperative monitoring with pulse oximetry is effective in detecting hypoxemic events and reducing the frequency of rescue events and intensive care unit transfers [60]. Supplemental oxygen should be administered continuously to those who are at increased perioperative risk from OSA until they are able to maintain their baseline oxygen saturation while breathing room air [24]. CPAP or noninvasive positive pressure ventilation (NIPPV) should be continuously administered postoperatively to patients who were using these modalities preoperatively, unless contraindicated by the surgical procedure. If frequent or severe airway obstruction or hypoxemia occurs during postoperative monitoring, initiation of nasal CPAP or NIPPV should be considered. Postoperative CPAP use after major abdominal surgery reduces atelectasis, pneumonia, and reintubation [61]. Neligan et al. [23] reported that the use of CPAP immediately after extubation improved lung function in morbidly obese patients with OSA undergoing laparoscopic bariatric surgery. Given that morbidly obese patients have a higher risk of tracheal reintubation, postoperative hand-off should include information regarding intubation and a plan if reintubation is needed.

2.14 Disposition from PACU

A physician should be responsible for the discharge of patients from the PACU per ASA standards. The consensus is that patients at increased perioperative risk from OSA should not be discharged to an unmonitored setting until they are no longer at risk of postoperative respiratory depression. To establish that patients are able to maintain adequate oxygen saturation levels while breathing room air, respiratory function should be determined by observing patients in an unstimulated environment, preferably while asleep. In other words, if the patient cannot maintain adequate oxygen saturation levels while breathing room air, the patient should be discharged to a monitored setting. Patients who are at high risk because of comorbidities, complications, or requiring ventilatory support may require ICU or intermediate level care unit admission [62]. Risk factors for ICU admission includes male gender, elderly patients, high BMI (>60 kg/m^2), pulmonary comorbidity, revision surgery, and reoperation [63].

2.15 Conclusion

A safe anesthetic for patients undergoing bariatric surgery requires meticulous preoperative, intraoperative, and postoperative care. Anesthesiologists need to fully understand the influence of obesity on patient anatomy, physiology, pharmacology, and pathophysiology. A thorough preoperative assessment will identify risk factors for airway management. Preoxygenation in reverse Trendelenburg position increases the safe period prior to desaturation. An anesthesiologist must always be prepared for the unanticipated difficult airway with appropriate equipment and sufficient help. In addition, vigilant intraoperative monitoring, optimal intraoperative ventilation, and avoiding pressure injury are important aspects of intraoperative management.

Multimodal pain management with both opioids and nonopioid modalities provides adequate pain control while minimizing side effects of opioids, such as respiratory depression and nausea and vomiting. Patients undergoing bariatric surgery carry a significant risk of developing respiratory complications, thus requiring close monitoring in the PACU and discharge to an appropriate postoperative setting.

References

1. El-Solh AA. Clinical approach to the critically ill, morbidly obese patient. Am J Respir Crit Care Med. 2004;169(5):557–61.
2. Kheterpal S, Han R, Tremper KK, Shanks A, Tait AR, O'Reilly M, et al. Incidence and predictors of difficult and impossible mask ventilation. Anesthesiology. 2006;105(5):885–91.
3. Juvin P, Lavaut E, Dupont H, Lefevre P, Demetriou M, Dumoulin JL, et al. Difficult tracheal intubation is more common in obese than in lean patients. Anesth Analg. 2003;97(2):595–600. table of contents.
4. Chung F, Yegneswaran B, Liao P, Chung SA, Vairavanathan S, Islam S, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. Anesthesiology. 2008;108(5):812–21.
5. Kheterpal S, Martin L, Shanks AM, Tremper KK. Prediction and outcomes of impossible mask ventilation: a review of 50,000 anesthetics. Anesthesiology. 2009;110(4):891–7.
6. Langeron O, Masso E, Huraux C, Guggiari M, Bianchi A, Coriat P, et al. Prediction of difficult mask ventilation. Anesthesiology. 2000;92(5):1229–36.
7. Enterlein G, Byhahn C. American Society of Anesthesiologists task F. [Practice guidelines for management of the difficult airway: update by the American Society of Anesthesiologists task force]. Anaesthesist. 2013;62(10):832–5.
8. Ezri T, Medalion B, Weisenberg M, Szmuk P, Warters RD, Charuzi I. Increased body mass index per se is not a predictor of difficult laryngoscopy. Can J Anaesth. 2003;50(2):179–83.
9. Brodsky JB, Lemmens HJ, Brock-Utne JG, Vierra M, Saidman LJ. Morbid obesity and tracheal intubation. Anesth Analg. 2002;94(3):732–6. table of contents.
10. Mashour GA, Kheterpal S, Vanaharam V, Shanks A, Wang LY, Sandberg WS, et al. The extended Mallampati score and a diagnosis of diabetes mellitus are predictors of difficult laryngoscopy in the morbidly obese. Anesth Analg. 2008;107(6):1919–23.
11. Berthoud MC, Peacock JE, Reilly CS. Effectiveness of preoxygenation in morbidly obese patients. Br J Anaesth. 1991;67(4):464–6.
12. Jense HG, Dubin SA, Silverstein PI, O'Leary-Escolas U. Effect of obesity on safe duration of apnea in anesthetized humans. Anesth Analg. 1991;72(1):89–93.
13. Dixon BJ, Dixon JB, Carden JR, Burn AJ, Schachter LM, Playfair JM, et al. Preoxygenation is more effective in the 25 degrees head-up position than in the supine position in severely obese patients: a randomized controlled study. Anesthesiology. 2005;102(6):1110–5. discussion 5A.
14. Boyce JR, Ness T, Castroman P, Gleysteen JJ. A preliminary study of the optimal anesthesia positioning for the morbidly obese patient. Obes Surg. 2003;13(1):4–9.
15. Perilli V, Sollazzi L, Bozza P, Modesti C, Chierichini A, Tacchino RM, et al. The effects of the reverse Trendelenburg position on respiratory mechanics and blood gases in morbidly obese patients during bariatric surgery. Anesth Analg. 2000;91(6):1520–5.
16. Adnet F, Borron SW, Dumas JL, Lapostolle F, Cupa M, Lapandry C. Study of the "sniffing position" by magnetic resonance imaging. Anesthesiology. 2001;94(1):83–6.
17. Goldberg ME, Norris MC, Larijani GE, Marr AT, Seltzer JL. Preoxygenation in the morbidly obese: a comparison of two techniques. Anesth Analg. 1989;68(4):520–2.
18. Gambee AM, Hertzka RE, Fisher DM. Preoxygenation techniques: comparison of three minutes and four breaths. Anesth Analg. 1987;66(5):468–70.
19. Coussa M, Proietti S, Schnyder P, Frascarolo P, Suter M, Spahn DR, et al. Prevention of atelectasis formation during the induction of general anesthesia in morbidly obese patients. Anesth Analg. 2004;98(5): 1491–5. table of contents.
20. Schumann R, Jones SB, Cooper B, Kelley SD, Bosch MV, Ortiz VE, et al. Update on best practice recommendations for anesthetic perioperative care and pain management in weight loss surgery, 2004–2007. Obesity. 2009;17(5):889–94.
21. Frappier J, Guenoun T, Journois D, Philippe H, Aka E, Cadi P, et al. Airway management using the intubating laryngeal mask airway for the morbidly obese patient. Anesth Analg. 2003;96(5):1510–5. table of contents.
22. Marrel J, Blanc C, Frascarolo P, Magnusson L. Videolaryngoscopy improves intubation condition in morbidly obese patients. Eur J Anaesthesiol. 2007;24(12):1045–9.
23. Neligan PJ, Malhotra G, Fraser M, Williams N, Greenblatt EP, Cereda M, et al. Continuous positive airway pressure via the Boussignac system immediately after extubation improves lung function in morbidly obese patients with obstructive sleep apnea undergoing laparoscopic bariatric surgery. Anesthesiology. 2009;110(4):878–84.
24. American Society of Anesthesiologists Task Force on Perioperative Management of patients with obstructive sleep apnea. Practice guidelines for the periopera-

tive management of patients with obstructive sleep apnea: an updated report by the American Society of Anesthesiologists Task Force on Perioperative Management of patients with obstructive sleep apnea. Anesthesiology. 2014;120(2):268–86.
25. Griffin J, Terry BE, Burton RK, Ray TL, Keller BP, Landrum AL, et al. Comparison of end-tidal and transcutaneous measures of carbon dioxide during general anaesthesia in severely obese adults. Br J Anaesth. 2003;91(4):498–501.
26. Xue Q, Wu X, Jin J, Yu B, Zheng M. Transcutaneous carbon dioxide monitoring accurately predicts arterial carbon dioxide partial pressure in patients undergoing prolonged laparoscopic surgery. Anesth Analg. 2010;111(2):417–20.
27. Murphy GS, Szokol JW, Avram MJ, Greenberg SB, Marymont JH, Vender JS, et al. Intraoperative acceleromyography monitoring reduces symptoms of muscle weakness and improves quality of recovery in the early postoperative period. Anesthesiology. 2011;115(5):946–54.
28. Hannenberg AA, Sessler DI. Improving perioperative temperature management. Anesth Analg. 2008;107(5): 1454–7.
29. Futier E, Constantin JM, Paugam-Burtz C, Pascal J, Eurin M, Neuschwander A, et al. A trial of intraoperative low-tidal-volume ventilation in abdominal surgery. N Engl J Med. 2013;369(5):428–37.
30. Chalhoub V, Yazigi A, Sleilaty G, Haddad F, Noun R, Madi-Jebara S, et al. Effect of vital capacity manoeuvres on arterial oxygenation in morbidly obese patients undergoing open bariatric surgery. Eur J Anaesthesiol. 2007;24(3):283–8.
31. Shibutani K, Inchiosa Jr MA, Sawada K, Bairamian M. Accuracy of pharmacokinetic models for predicting plasma fentanyl concentrations in lean and obese surgical patients: derivation of dosing weight ("pharmacokinetic mass"). Anesthesiology. 2004;101(3):603–13.
32. Leykin Y, Pellis T, Lucca M, Lomangino G, Marzano B, Gullo A. The effects of cisatracurium on morbidly obese women. Anesth Analg. 2004;99(4):1090–4. table of contents.
33. Leykin Y, Pellis T, Lucca M, Lomangino G, Marzano B, Gullo A. The pharmacodynamic effects of rocuronium when dosed according to real body weight or ideal body weight in morbidly obese patients. Anesth Analg. 2004;99(4):1086–9. table of contents.
34. Schumann R, Jones SB, Ortiz VE, Connor K, Pulai I, Ozawa ET, et al. Best practice recommendations for anesthetic perioperative care and pain management in weight loss surgery. Obes Res. 2005;13(2):254–66.
35. Alvarez A, Singh PM, Sinha AC. Postoperative analgesia in morbid obesity. Obes Surg. 2014;24(4): 652–9.
36. Feld JM, Laurito CE, Beckerman M, Vincent J, Hoffman WE. Non-opioid analgesia improves pain relief and decreases sedation after gastric bypass surgery. Can J Anaesth. 2003;50(4):336–41.
37. Govindarajan R, Ghosh B, Sathyamoorthy MK, Kodali NS, Raza A, Aronsohn J, et al. Efficacy of ketorolac in lieu of narcotics in the operative management of laparoscopic surgery for morbid obesity. Surg Obes Relat Dis. 2005;1(6):530–5. discussion 5–6.
38. Ding Y, White PF. Comparative effects of ketorolac, dezocine, and fentanyl as adjuvants during outpatient anesthesia. Anesth Analg. 1992;75(4):566–71.
39. Sasse KC, Ganser J, Kozar M, Watson RW, McGinley L, Lim D, et al. Seven cases of gastric perforation in Roux-en-Y gastric bypass patients: what lessons can we learn? Obes Surg. 2008;18(5):530–4.
40. Remy C, Marret E, Bonnet F. Effects of acetaminophen on morphine side-effects and consumption after major surgery: meta-analysis of randomized controlled trials. Br J Anaesth. 2005;94(4):505–13.
41. Tufanogullari B, White PF, Peixoto MP, Kianpour D, Lacour T, Griffin J, et al. Dexmedetomidine infusion during laparoscopic bariatric surgery: the effect on recovery outcome variables. Anesth Analg. 2008;106(6):1741–8.
42. Blaudszun G, Lysakowski C, Elia N, Tramer MR. Effect of perioperative systemic alpha2 agonists on postoperative morphine consumption and pain intensity: systematic review and meta-analysis of randomized controlled trials. Anesthesiology. 2012; 116(6):1312–22.
43. Schmid RL, Sandler AN, Katz J. Use and efficacy of low-dose ketamine in the management of acute postoperative pain: a review of current techniques and outcomes. Pain. 1999;82(2):111–25.
44. Ryu JH, Kang MH, Park KS, Do SH. Effects of magnesium sulphate on intraoperative anaesthetic requirements and postoperative analgesia in gynaecology patients receiving total intravenous anaesthesia. Br J Anaesth. 2008;100(3):397–403.
45. Cabrera Schulmeyer MC, de la Maza J, Ovalle C, Farias C, Vives I. Analgesic effects of a single preoperative dose of pregabalin after laparoscopic sleeve gastrectomy. Obes Surg. 2010;20(12):1678–81.
46. Chang CY, Challa CK, Shah J, Eloy JD. Gabapentin in acute postoperative pain management. Biomed Res Int. 2014;2014:631756.
47. Abdallah FW, Chan VW, Brull R. Transversus abdominis plane block: a systematic review. Reg Anesth Pain Med. 2012;37(2):193–209.
48. Cottam DR, Fisher B, Atkinson J, Link D, Volk P, Friesen C, et al. A randomized trial of bupivicaine pain pumps to eliminate the need for patient controlled analgesia pumps in primary laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2007;17(5):595–600.
49. Sherwinter DA, Ghaznavi AM, Spinner D, Savel RH, Macura JM, Adler H. Continuous infusion of intraperitoneal bupivacaine after laparoscopic surgery: a randomized controlled trial. Obes Surg. 2008;18(12):1581–6.
50. Cohen AR, Smith AN, Henriksen BS. Postoperative opioid requirements following roux-en-y gastric

bypass in patients receiving continuous bupivacaine through a pump system: a retrospective review. Hosp Pharm. 2013;48(6):479–83.
51. Michaloudis D, Fraidakis O, Petrou A, Farmakalidou H, Neonaki M, Christodoulakis M, et al. Continuous spinal anesthesia/analgesia for perioperative management of morbidly obese patients undergoing laparotomy for gastroplastic surgery. Obes Surg. 2000;10(3): 220–9.
52. Casati A, Putzu M. Anesthesia in the obese patient: pharmacokinetic considerations. J Clin Anesth. 2005;17(2):134–45.
53. Panni MK, Columb MO. Obese parturients have lower epidural local anaesthetic requirements for analgesia in labour. Br J Anaesth. 2006;96(1):106–10.
54. Carvalho JC. Ultrasound-facilitated epidurals and spinals in obstetrics. Anesthesiol Clin. 2008;26(1):145–58. vii-viii.
55. Kranke P, Apefel CC, Papenfuss T, Rauch S, Lobmann U, Rubsam B, et al. An increased body mass index is no risk factor for postoperative nausea and vomiting. A systematic review and results of original data. Acta Anaesthesiol Scand. 2001;45(2):160–6.
56. Ziemann-Gimmel P, Hensel P, Koppman J, Marema R. Multimodal analgesia reduces narcotic requirements and antiemetic rescue medication in laparoscopic Roux-en-Y gastric bypass surgery. Surg Obes Relat Dis. 2013;9(6):975–80.
57. Ziemann-Gimmel P, Goldfarb AA, Koppman J, Marema RT. Opioid-free total intravenous anaesthesia reduces postoperative nausea and vomiting in bariatric surgery beyond triple prophylaxis. Br J Anaesth. 2014;112(5):906–11.
58. Bamgbade OA, Rutter TW, Nafiu OO, Dorje P. Postoperative complications in obese and nonobese patients. World J Surg. 2007;31(3):556–60. discussion 61.
59. Ahmad S, Nagle A, McCarthy RJ, Fitzgerald PC, Sullivan JT, Prystowsky J. Postoperative hypoxemia in morbidly obese patients with and without obstructive sleep apnea undergoing laparoscopic bariatric surgery. Anesth Analg. 2008;107(1):138–43.
60. Taenzer AH, Pyke JB, McGrath SP, Blike GT. Impact of pulse oximetry surveillance on rescue events and intensive care unit transfers: a before-and-after concurrence study. Anesthesiology. 2010;112(2):282–7.
61. Ireland CJ, Chapman TM, Mathew SF, Herbison GP, Zacharias M. Continuous positive airway pressure (CPAP) during the postoperative period for prevention of postoperative morbidity and mortality following major abdominal surgery. Cochrane Database Syst Rev. 2014;8, CD008930.
62. Davidson JE, Callery C. Care of the obesity surgery patient requiring immediate-level care or intensive care. Obes Surg. 2001;11(1):93–7.
63. Helling TS, Willoughby TL, Maxfield DM, Ryan P. Determinants of the need for intensive care and prolonged mechanical ventilation in patients undergoing bariatric surgery. Obes Surg. 2004;14(8):1036–41.
64. Apfelbaum JL, Hagberg CA, Caplan RA, Blitt CD, Connis RT, Nickinovich DG, et al. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Anesthesiology. 2013;118(2):251–70.
65. Pai MP, Paloucek FP. The origin of the "ideal" body weight equations. Ann Pharmacother. 2000;34(9):1066–9.
66. Hume R. Prediction of lean body mass from height and weight. J Clin Pathol. 1966;19(4):389–91.
67. Ingrande J, Lemmens HJ. Dose adjustment of anaesthetics in the morbidly obese. Br J Anaesth. 2010;105 Suppl 1:i16–23.
68. Balki M, Lee Y, Halpern S, Carvalho JC. Ultrasound imaging of the lumbar spine in the transverse plane: the correlation between estimated and actual depth to the epidural space in obese parturients. Anesth Analg. 2009;108(6):1876–81.

3 Optimizing Perioperative Management: Perioperative Care and Protocols to Prevent and Detect Early Complications

Ambar Banerjee and Don Jay Selzer

Key Points

- Bariatric procedures are not without potential complications, though considered safe in the hands of an experienced surgeon.
- Preventive strategies, prompt diagnosis, and treatment of postoperative complications are important in reducing morbidity and mortality.
- Multidisciplinary approach is imperative in the preoperative period to identify and treat conditions that may result in adverse outcomes after surgery.
- Intraoperative considerations, including management of the airway, positioning of the patient, and drug pharmacokinetics, are important to reduce the risk of postoperative morbidity and mortality.
- Early complications, related to the commonly performed bariatric procedures, have been described in the chapter along with description of their presentation, work-up, prevention, and management.

3.1 Introduction

Since the National Institutes of Health (NIH) consensus conference over two decades ago, bariatric surgery has undergone remarkable progress and is now established as the only durable treatment for morbid obesity and its associated comorbidities [1]. Increasing experience and expertise in laparoscopic techniques have contributed to a significant decrease in morbidity and mortality associated with open bariatric procedures while achieving comparable weight loss results. Today, the commonly performed procedures include the adjustable gastric band (AGB), sleeve gastrectomy (SG), Roux-en-Y gastric bypass (RYGB), and biliopancreatic diversion with duodenal switch (DS) [2].

When performed at specialized centers, bariatric surgery is technically challenging but safe [3–5]. Perioperative complications vary based on the approach (open vs. laparoscopic) and complexity of the surgical procedure performed (adjustable gastric band vs. sleeve gastrectomy or gastric bypass or duodenal switch vs. revisional surgery) [6, 7]. As one may expect, incidence of an adverse intraoperative event is associated with an increased risk of major postoperative morbidity [8]. Although a detailed description of each complication and its management is outside the scope of this chapter, it provides an overview of programmatic efforts that prevent and/or help identify early perioperative complications of the four common bariatric surgical procedures performed in the USA.

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_3

3.2 Preoperative Considerations—Establish a Multidisciplinary Approach

The suitability of a candidate for bariatric surgery is determined through an intensive multidisciplinary evaluation process involving psychological, medical, and nutritional expertise [9]. This is as critical as the surgical arm of the process. It is instrumental for identification and possible intervention to arrest progression of conditions that may eventually lead to adverse outcomes in both the short and long term.

3.2.1 Psychosocial-Behavioral Evaluation

A psychosocial-behavioral evaluation is an essential component of preoperative preparation. It is now known that psychopathology may be related to bariatric surgery outcomes. The American Society for Bariatric and Metabolic Surgery (ASMBS) recommends the use of objective psychological assessment measures in conjunction with a clinical interview for the presurgical psychological screening (PPS). Components of the person-to-person interview are screening for psychiatric disorders, substance abuse or dependence (including alcohol), and assessing if the candidate has the capability to incorporate nutritional and behavioral changes before and after bariatric surgery [10]. The most widely used objective psychometric tests utilized for the purpose of PPS are Minnesota Multiphasic Personality Inventory – 2 Restructured Form (MMPI-2-RF) and the Beck Depression Inventory – II. The newer instrument, the MMPI-2-RF, has demonstrated good reliability, validity, and generalizability of scale scores across bariatric surgery candidate samples [11, 12].

A review of recent literature indicates that cognitive impairments present before surgery may predispose to poor weight loss following bariatric surgery. Some feel that a reduced cognitive ability inhibits the development and maintenance of appropriate eating behaviors. Although personality traits may not independently impact postsurgical weight loss among bariatric patients, they likely impact a patient's psychological and behavioral adjustment after surgery. Therefore, they may have an indirect effect on long-term weight loss [13].

The correlation between psychopathology and morbid obesity is complex and poorly understood. Patients with severe and chronic psychiatric disorders, such as adjustment disorders, personality disorders, or major depression, may have more difficulties accepting the behavioral changes imposed by surgery and thus be less likely to achieve successful weight loss [14, 15]. Ultimately, psychological distress secondary to obesity is highly likely to decrease with weight loss, which may contribute to better long-term weight maintenance and a better quality of life.

Compared to the general population, bariatric patients suffer from an increased risk of suicide and lifetime substance abuse. This is likely due to presurgical psychiatric and mood disorders or financial problems [16]. Suicidal ideation is considered to be a contraindication for surgery during preoperative work-up though the duration of such symptoms for exclusion may vary according to institutional guidelines. The authors' program excludes patients who have had suicidal attempts in the past 24 months from enlisting for bariatric surgery.

Chronic pain, another common issue arising in bariatric patients, may predispose the patient to a need for narcotic analgesics well in advance of considering surgery. In these patients, it is worthwhile to establish realistic expectations regarding the use of prescription drugs to manage chronic nonmalignant pain. These agreements may come in the form of contracts and may be regulated by local or state laws governing long-term narcotic prescription.

3.2.2 Preoperative Medical Evaluation and Clearance

3.2.2.1 Glycemic Control

Strict perioperative glycemic control with hemoglobin A_{1C} value of 6.5 to 7 % or less, a fasting blood glucose level of ≤110 mg/dl, and a 2-h postprandial blood glucose concentration of ≤140 mg/dl have been shown to reduce adverse

events and improve incidence of infections in the surgical population in general [10]. In patients with advanced microvascular or macrovascular complications, extensive comorbid conditions, and intractable diabetes, a more liberal preoperative hemoglobin A_{1C} goal of 7–8 % may be pursued to offer them the most effective opportunity for treatment of their diseases. A challenge may present when a preoperative patient is unable to achieve this level of control due to ongoing struggle with insulin resistance secondary to morbid obesity. A multidisciplinary approach is needed to make the best choice for the patient.

3.2.2.2 Cardiac Clearance

A preoperative cardiac evaluation is suggested for patients with ≥1 risk factors for coronary artery disease (CAD). Because obesity is an independent risk factor for CAD, the recommendation is to perform a cardiac evaluation in all obese patients seeking bariatric surgery. Risk for a perioperative cardiac event is based on functional capacity easily obtained in a thorough history and physical examination. The patient's ability to perform activities requiring at least four metabolic equivalents (e.g., climbing a flight of stairs or walking uphill) is considered a reliable predictor for a low-risk patient. Intermediate- or high-risk patients or those with a history of cardiac ailments should undergo a more in-depth preoperative evaluation that may require consultation with a cardiologist.

Patients with a recent percutaneous cardiac intervention present another challenge. To provide an adequate time for neo-intimal coverage and reduce the risk for thrombosis, the American College of Cardiology guidelines recommend dual-antiplatelet therapy (aspirin and usually a thienopyridine like clopidogrel) for ≥1 month in patients receiving bare metal stents and ≥12 months for those receiving drug-eluting stents [17]. Low-dose aspirin can be continued during the perioperative period when indicated. On the other hand, clopidogrel and many of the newer oral anticoagulants are known to be associated with a significant risk of bleeding and should be held in the perioperative period. The thienopyridine agents (e.g., clopidogrel and prasugrel) should be stopped 7–10 days prior to the surgery. Oral anticoagulants should be discontinued based on the half-lives of the various agents. Patients on warfarin should be advised to stop the medication 5–7 days prior to the procedure and may be bridged with fractionated/unfractionated heparin as opined by the cardiologist, based on their individual risk factors. Dabigatran (Pradaxa®), a direct thrombin inhibitor, should be discontinued at least 2 days or 3–5 days before surgery for patients with creatinine clearance of ≥50 ml/min and <50 ml/min, respectively. Newer generation direct factor Xa inhibitors (rivaroxaban and other xaban agents) may be discontinued 1–2 days before surgery due to their much shorter half-lives. Fewer data exists to guide one on when to restart these medications, but it is anticipated that the time should be when the surgeon determines minimal risk of postoperative bleeding. These patients should be advised to monitor their stools for melena after discharge to detect early gastrointestinal bleeding.

3.2.2.3 Obstructive Sleep Apnea

A prospective bariatric surgery patient should undergo a standardized screening for obstructive sleep apnea (OSA). The Berlin Questionnaire is commonly used to identify the risk (low to high) of sleep-disordered breathing. The questionnaire consists of three categories related to the risk of having sleep apnea. There are five questions about snoring (Category 1), four about daytime sleepiness (Category 2), one question about blood pressure (Category 3), and general questions about age, social background, gender, body height and weight, as well as neck circumference. If positive, a confirmatory polysomnography test should be obtained.

Patients with sleep disorders and intrinsic lung disease should be evaluated and managed by a physician specialist to help minimize perioperative complications. Perioperative exposure to sedation, anesthesia, and opioids increases pharyngeal collapse, decreases ventilatory response, and inhibits laryngeal respiratory modulated mechanoreceptors, leading to worsening of sleep apnea. Anesthetic medications and benzodiazepines also impair the arousal response, a protec-

tive defense mechanism against sleep apnea that helps in overcoming the airway obstruction. Furthermore, coexisting illnesses such as systemic hypertension, insulin resistance, coronary artery disease, and cardiac arrhythmias render the perioperative management of these patients even more complicated. A recent meta-analysis assessing the risk of sleep apnea on postoperative complications concluded that the incidence of oxygen desaturations, respiratory failure, cardiac events, and ICU transfers was higher in patients with OSA [18].

Patients with OSA who use continuous positive airway pressure (CPAP) preoperatively demonstrate a lower incidence of postoperative complications and shorter hospital length of stay when compared with those who were not on CPAP. Preoperative use of CPAP may decrease inflammation of the upper airway and tongue size, increase upper airway volume and stability, and improve pulmonary reserve through alveolar recruitment [19]. It has also been elicited that preoperative CPAP may help reduce nocturnal and pulmonary hypertension when consistently used over 3–6 months [20]. However, a consensus on the optimal duration of preoperative CPAP therapy has yet to be determined.

3.2.2.4 Estrogen Therapy

Any form of estrogen therapy (oral contraceptives in premenopausal women or hormone replacement therapy in postmenopausal women) should be discontinued at least 3 weeks prior to bariatric surgery to diminish the risks of perioperative venous thromboembolic phenomenon [10].

3.2.2.5 Tobacco

Smoking is a well-established independent risk factor for adverse surgical outcomes irrespective of the type of procedure. Increased BMI being a naturally constant risk factor in this population, modification of the other risk factors especially smoking is of utmost importance. Given the increased risk of poor wound healing, anastomotic ulcer, and overall impaired health, complete abstinence from smoking is recommended for at least 6 weeks prior to and after surgery [10]. A preoperative urine nicotine test is sometimes required by insurance and ensures compliance.

3.2.3 Optimize Preoperative Nutrition

3.2.3.1 Weight Loss

Preoperative weight loss may provide an opportunity to decrease perioperative complications [21]. Patients who present with a large left hepatic lobe and prolific mesenteric and omental fat often require higher intra-abdominal insufflation pressures of 18–20 mmHg for adequate operative field. These pneumoperitoneum pressures may further deteriorate the perioperative cardiopulmonary status by impairing venous return and increasing peak pulmonary airway pressures. Super morbid obesity (BMI >50) has generally been found to be associated with increased morbidity and higher risks of perioperative complications. In addition, preoperative weight loss may improve preoperative comorbid medical conditions (e.g., diabetes and hypertension), leading to a decrease in the risk of perioperative complications.

3.2.3.2 Malnutrition

Obese patients may present with mild-to-moderate malnutrition. Multiple publications in the literature have demonstrated obesity-related nutritional deficiencies, most commonly in vitamins B1, D, B6, and B12, folate, and trace minerals magnesium, iron, and zinc [22–24]. Supplementation of these essential micronutrients is important in the perioperative management of the bariatric patients. The deficiency of a particular micronutrient may also determine the type of procedure selected for the treatment of morbid obesity. Patients who are diagnosed with severe osteoporosis or iron-deficiency anemia are better served with non-malabsorptive bariatric procedures. A gastric bypass or a biliopancreatic diversion along with a higher risk of postoperative nutritional deficiencies may prove to be detrimental to the overall well-being of this subpopulation.

3.3 Intraoperative Considerations

3.3.1 Airway Management

Morbidly obese patients pose significant technical challenges for the surgical and the anesthesia teams alike. The obese patient's large tongue, redundant oropharyngeal tissue, atlanto-axial joint limitation due to cervical and thoracic fat pads, and presternal fat deposits inhibit movement of the laryngoscope and increase the difficulty of direct laryngoscopy (DL) [25]. Abnormal thyromental distance, neck range of motion, prognathism, higher Mallampati classification, and neck circumference are all predictors of a difficult airway and subsequent difficult intubation [26]. Head-elevated laryngoscopy position, where the patient's external auditory meatus is in a horizontal plane with the sternal notch to compensate for the fixed flexion due to cervical fat, may be used for optimal positioning [27]. In patients with unfavorable airways, an awake intubation may be appropriate. This is best performed with the help of a fiber-optic bronchoscope. Under extremely difficult circumstances, laryngeal mask airway may be used as an appropriate rescue device to ventilate the patient until a more definite airway is obtained. This can include a surgical airway as well under dire circumstances.

3.4 Drug Pharmacokinetics

Highly lipophilic substances, such as barbiturates and benzodiazepines, demonstrate altered pharmacokinetics in the morbidly obese with significant increases in the volume of distribution relative to normal-weight individuals. These drugs will likely require higher doses to reach the desired effect. They will also require a longer time to be eliminated from the patient's system. Due to more rapid and consistent recovery profile, desflurane with remifentanil has been suggested as one potion in this patient population. Complete muscular relaxation is crucial during laparoscopic bariatric procedures to facilitate ventilation and to maintain an adequate working space for visualization and safe manipulation of laparoscopic instruments [9, 28].

3.5 Respiratory Function

Morbid obesity is associated with the restrictive variety of pulmonary impairment, which leads to hypoxemia, hypoxia, and ventilation-perfusion mismatch, which are all aggravated in the supine position. A previous study had shown that most subjects demonstrated spirometry, lung volumes, and gas exchange measurements within the normal ranges [29]. Also, a definite relationship was not found between body size and spirometry. Hence, routine pulmonary function testing (PFT) may not be indicated in this population. However, if the patient presents with excessive daytime sleepiness, loud snoring, choking during sleep, resuscitative snorting, fatigue, hypersomnolence, impaired concentration and memory, a small oropharynx, and a thick neck, PFTs may be obtained to confirm the presence of obesity hypoventilation syndrome (OHS). It is defined by the presence of awake alveolar hypoventilation (arterial carbon dioxide tension [$PaCO_2$] >45 mmHg), which cannot be attributed to other pulmonary, endocrine, skeletal, or neuromuscular pathology in an obese individual. This diagnosis should prompt immediate initiation of noninvasive positive airway pressure therapy as untreated OHS can progress to acute, life-threatening cardiopulmonary compromise. In addition, untreated OHS is associated with a high mortality rate, a reduced quality of life, and numerous morbidities, including pulmonary hypertension, right heart failure, and angina.

Concomitant elevated intraperitoneal pressures during laparoscopic bariatric surgery may lead to further impairment and increased incidence of atelectasis [28, 30]. The anesthesia team should be wary of these difficulties and be prepared to take adequate safeguards during the course of the surgery and the immediate postoperative period.

3.5.1 Body Habitus

To reduce the technical difficulty in performing laparoscopic surgery in the morbidly obese, optimal positioning of the patient is of paramount importance. Particular attention should be paid to selecting appropriate instrumentation including an operating table rated to accommodate the patient's weight. It is necessary to provide padding of all pressure points to minimize the incidence of pressure ulcers and neural injuries as well as the possibility of rhabdomyolysis [31]. The patients should be appropriately secured to the operating table to aid in its safe maneuverability during the procedure. The use of an air mattress is also helpful for safe transfer from the operating table to the hospital bed, both for the morbidly obese patient and for the operating room staff.

3.6 Early Complications Common to All Bariatric Procedures

A morbidly obese patient undergoing a bariatric procedure is predisposed to certain early complications, irrespective of the type of surgery. These include infections, hemorrhage, deep venous thrombosis and pulmonary embolism, staple-line and/or anastomotic leaks, and acute nutritional deficiencies.

3.6.1 Infectious Processes

3.6.1.1 Surgical-Site Infections

Surgical-site infections (SSI) are the most common post-bariatric surgery infections. The Bariatric Outcomes Longitudinal Database (BOLD) study reported an overall incidence of SSI of 1.1 % [32]. These may be superficial or deep, surrounding organ spaces. SSI also increases the risk of incisional hernias. Skin commensals like *Staphylococcus aureus* and Streptococcus species, and Enterobacteriaceae and gram-negative bacteria, are the usual causative pathogens. Risk factors for SSI specific to the bariatric population include BMI greater than 50 kg/m^2, delayed antibiotic infusion, sleep apnea, bipolar disorder, and increased surgical time >180 min [33, 34].

Many SSI prevention measures are well described which include treating all existing infections prior to surgery, minimizing hair removal preoperatively (and if required removing hair immediately prior to surgery using clippers), achieving appropriate glucose control during the preoperative period, maintaining normothermia during the perioperative period, applying antiseptic skin preparation appropriately prior to surgery, ensuring proper hand/forearm antisepsis for surgical team members, maintaining a sterile field in the operating room, and providing appropriate antimicrobial prophylaxis. Expert surgical technique, including methods of wound closure, can also impact SSI risk. Most of these recommendations for the prevention of SSIs apply to bariatric surgery.

Current guidelines for surgical antibiotic prophylaxis recommend cefazolin before bariatric surgery. Due to the altered drug pharmacokinetics, the dose is adjusted based on the body weight of the patient. Additional intraoperative doses may be indicated for operative times exceeding 180 min or two times the half-life of the antibiotic. Clindamycin or vancomycin with an aminoglycoside may be administered in patients allergic to beta-lactams [35].

3.6.1.2 Pneumonia

Though respiratory tract infections are relatively rare after laparoscopic bariatric surgery, the risk of development of pneumonia may be increased in patients with prolonged intubation or hospitalization. The overall incidence of postoperative pneumonia is 0.4 % based on a retrospective review of the Bariatric Outcomes Longitudinal Database (BOLD) database comprising of 78,951 bariatric surgical patients [32]. History of smoking greater than 20 pack-years has been demonstrated to be associated with elevated risk of postoperative respiratory complications [36]. Early ambulation and incentive spirometry in the immediate postoperative period are important in decreasing the incidence of pulmonary complica-

tions. Optimal postoperative pain control is important for increased mobility and improved breathing. It can aid in decreasing the incidence of atelectasis and increasing recruitment of collapsed alveoli with improved tidal volumes. Regional anesthesia with the help of specialized continuous delivery systems (e.g., ON-Q pain relief system®) or with transversus abdominis plane block using long-acting local anesthetic (e.g., Exparel®) may achieve targeted pain relief in this patient population along with decrease in the dose of narcotic medications and their potential side effects. Noninvasive positive pressure therapy that was instituted in the preoperative period should be resumed in the immediate postoperative period to help in mitigating the restrictive pulmonary compromise associated with obesity and to help recruit the airways for adequate ventilation.

3.6.1.3 Other Infectious Complications

Catheter-associated urinary tract infections (CAUTI) and central line-associated bloodstream infections (CLABSI) may occur following bariatric surgery. Their incidence can be decreased by reducing the duration of use of these devices and by adhering to specific device care bundles aimed at preventing these infections. For example, many avoid the use of urinary catheters in bariatric surgical procedures altogether.

3.6.2 Venous Thromboembolism

Venous thromboembolism (VTE), including both deep vein thrombosis (DVT) and pulmonary embolism (PE), is a rare complication of bariatric surgery but a leading cause of postoperative mortality. The overall incidence of VTE was 0.42 % in 73,921 patients included in the BOLD database, where 73 % of these events occurred after discharge within 30 days after surgery [37]. The incidence of VTE was 0.29 % for laparoscopic procedures and 1.2 % for open procedures. Obesity predisposes patients to a hypercoagulable state due to a chronic inflammatory state, lower extremity venous stasis, sedentary lifestyle, and increased abdominal pressure. Additional commonly associated risk factors for fatal PE in this population included BMI >60 kg/m^2, truncal obesity, severe venous stasis disease, previous history of VTE, operative time greater than 3 h, and obesity-hypoventilation syndrome/sleep apnea syndrome [38].

The Michigan Bariatric Surgery Collaborative (MBSC), in a population-based study, determined the type of procedure to be the most important determinant for VTE. The probability of VTE was highest for patients undergoing the duodenal switch procedure, followed by open gastric bypass, laparoscopic gastric bypass, sleeve gastrectomy, and adjustable gastric band. They developed a preoperative risk calculator for identifying patients at especially high risk for VTE who would potentially benefit from more aggressive VTE prophylaxis, such as extended post-discharge thrombo-prophylaxis or postoperative therapeutic anticoagulation [39]. The same surgical group subsequently advocated for the use of low-molecular-weight heparin over unfractionated heparin in providing prophylaxis without an increase in bleeding complications [40].

The current guidelines of the ASMBS regarding VTE recommend using mechanical as well as chemical prophylaxis along with early ambulation [41]. Though there remains a lack of consensus regarding the initiation of thrombo-prophylaxis in the preoperative period due to the absence of strong evidence, multiple studies have recommended at least a single dose of heparin prior to surgery to reduce the risk of VTE. Perioperative chemoprophylaxis for patients, who are considered to be at high risk based on preoperative risk stratification, should be continued for 3–4 weeks after discharge from the hospital.

Immediate anticoagulation is indicated in patients with a high degree of clinical suspicion for VTE. If anticoagulation is contraindicated in the event of VTE, placement of a mechanical filter in the inferior vena cava may be considered to decrease the risk of further clot propagation and embolization. However, the MBSC had questioned the use of prophylactic IVC filters to prevent the occurrence of pulmonary embolism due

to the high risk of complications without any demonstrable benefits [42].

3.6.3 Hemorrhage

Postoperative hemorrhage is commonly multifactorial. Postoperative bleeding can be classified according to the location of the bleeding with respect to the stomach and intestine. The first location is extra-luminal. Although there is a potential for this bleeding to occur extra-peritoneal as well, most extra-luminal bleeding occurs within the peritoneal cavity. Most commonly, it occurs along the staple lines (e.g., gastroenterostomy, gastric sleeve/pouch or remnant, enteroenterostomy) or from inadequate control of short gastric, omental, or mesenteric vessels (Fig. 3.1). Trocar site bleeding and splenic and liver injuries from the retractor are less common.

The second location of postoperative bleeding is intraluminal. Again, it occurs most commonly along staple lines (e.g., gastroenterostomy, gastric staple line, or enteroenterostomy). Surgeons have employed the use of staple-line reinforcements and topical hemostatic agents/sealants for prophylaxis against such risk of bleeding. Dapri et al. showed the benefit of buttressing materials in reducing the incidence of postoperative bleeding without any statistically significant effect on the occurrence of leaks or operative time [43]. A systematic review of 30 articles, performed by Knapps et al., demonstrated a lack of statistical difference for staple-line leak, bleeding, and other major complications in laparoscopic sleeve gastrectomy with or without staple-line reinforcement [44]. Thrombin and other newer hemostatic-sealant agents (Tachosil®) have shown promise in reducing postoperative bleeding in some case series but major randomized controlled studies are lacking [45, 46]. Thus, the benefit of these agents in preventing episodes of hemorrhage is a matter of great debate in the surgical literature.

Another uncommon, but possible, source of bleeding is Mallory-Weiss tear resulting from severe vomiting or retching. Preoperative education regarding eating choices and a graduated process of dietary advancement should help to reduce this occurrence.

Although the clinical presentation of acute bleeding with a drop in hemoglobin/hematocrit, diaphoresis, tachycardia, hypotension, abdominal distention, hematemesis, and melena is instrumental

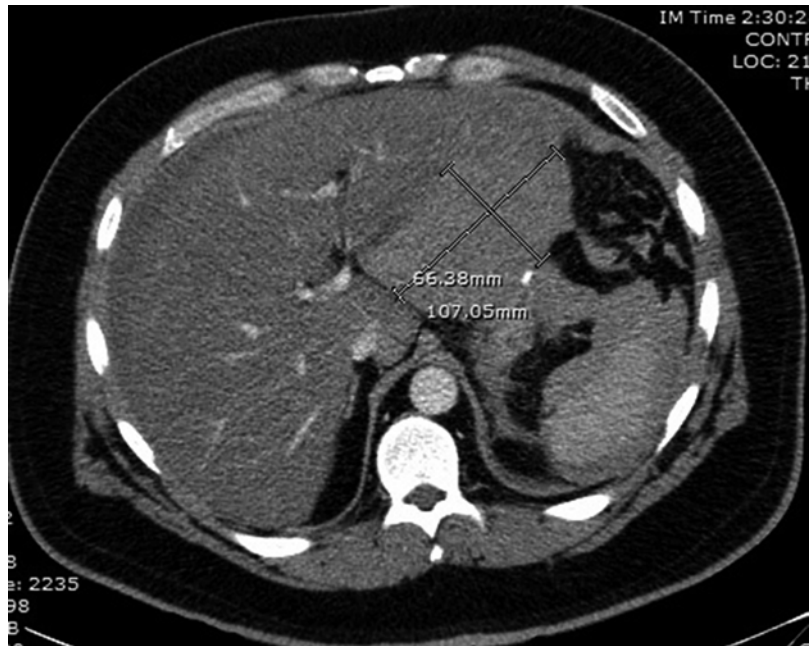

Fig. 3.1 Development of a hematoma anterior to the stomach following a laparoscopic sleeve gastrectomy

in making the diagnosis, identification and the corresponding control of the site of bleeding can be a challenge. Patients with early postoperative bleeding experience a significantly longer hospital stay (4.8 vs. 3.0 days, $p<0.0001$) and higher mortality rate (7.1 % vs. 0.9 %, $p<0.01$) compared with those without an early bleed [47].

The initial management of acute postoperative hemorrhage in this patient population does not differ from any other upper gastrointestinal bleed in a non-bariatric surgical patient. It includes adequate resuscitation, close monitoring of vital signs, serial blood counts, and discontinuation/reversal of anticoagulation. Blood product transfusion may be initiated when indicated. A majority of bleeding episodes will resolve without further surgical intervention. Patients with ongoing intraluminal bleeding along with high transfusion requirements will need further endoscopic exploration. It may reveal the site of bleeding on the inner aspect of the staple line, which can then be controlled by adrenaline injection, electrocoagulation, or endoclips. Hemodynamic instability and refractory bleeding, from the gastric remnant or other sites inaccessible to endoscopy or within the peritoneal cavity, can require surgical revisions. The operative goals are to evacuate the majority of the clots, attempt to identify, and control the site of hemorrhage if it is readily apparent or to oversew all staple lines if the patient is hemodynamically unstable and does not have an obvious bleeding source.

3.6.4 Nutritional Deficiencies

Anemia is the most common complication after a bariatric procedure and is estimated to occur in 20–49 % of patients. This may result from acute blood loss or secondary to deficiencies of iron, vitamin B_{12}, or folate. Bariatric patients may also present with deficiencies in magnesium, calcium, zinc, copper, vitamin D, thiamine, and vitamin A. However, a vast majority of these deficiencies do not manifest in the postoperative period as an early complication except thiamine, which can have severe neurological consequences [48].

Thiamine deficiency has been reported to occur in up to 29 % of patients. It can present within the first 6 weeks following the bariatric intervention. A high degree of suspicion and clinical alertness is required for its prompt diagnosis and adequate treatment should be instituted immediately. Vomiting after a bariatric procedure is the principal risk factor for development of thiamine deficiency in combination with poor food and supplement intake. Acute symptomatic thiamine deficiency may be precipitated by administration of intravenous glucose, which leads to interruption of the citric acid cycle and lactic acidosis. The deficiency may manifest as Wernicke's encephalopathy (ophthalmoplegia, nystagmus, ataxia, and mental status changes) or acute polyradiculoneuropathy (Guillain-Barre syndrome). If not recognized and treated, Wernicke's encephalopathy can progress to death or chronic neurological impairment known as Korsakoff's syndrome. The authors recommend prophylactic administration of intravenous thiamine 100 mg before starting intravenous fluids in patients at risk. Multivitamin supplementation should be carefully selected to include at least 3 mg of thiamine for prophylaxis. In patients demonstrating symptomatic deficiency, daily administration of 100–500 mg intravenous thiamine is recommended [49].

3.6.5 Anastomotic or Staple-Line Leak

Anastomotic or staple-line leak is one of the most dreaded complications of a bariatric procedure. It is considered as one of the strongest independent risk factors for postoperative mortality. The incidence of leaks, across different bariatric procedures, ranges from 1 to 5 %, depending on the series and the patient characteristics [50]. Revisional surgeries are associated with a higher rate of leaks and/or fistulas. It may present either early within the first 7 days after surgery or late, at a week or more after surgery. Early diagnosis of such a leak is critical to avoid progression of adverse outcomes of peritonitis, which include systemic inflammatory response, sepsis, multi-

organ failure, and lastly death. The clinical presentation of these patients is identical irrespective of the procedure. Findings like tachycardia, pyrexia, tachypnea, abdominal pain, oliguria, nausea, and vomiting and purulent drain output are the harbingers of a leak. It has been demonstrated that a persistent heart rate in excess of 120 beats per minute is a good indicator of an anastomotic/staple-line leak. Kolakowski et al. further reported in a study that the triad of tachycardia, tachypnea, and fever was 58 % sensitive and 99 % specific for detection of anastomotic leaks [51].

These findings can then be supported with either an upper gastrointestinal contrast study or a computerized tomography of the abdomen with water-soluble oral contrast (e.g., Isovue) to confirm the diagnosis. Sensitivity of upper GI contrast studies varies among reports between 22 and 75 % (Fig. 3.2). The interpretation of computerized tomography of the abdomen is user dependent and fails to demonstrate a high level of sensitivity in detecting early postoperative leaks in this patient population. When upper GI and CT are combined, up to one-third of patients will have both studies interpreted as normal despite the presence of a leak [52]. Lastly, surgical exploration is the most definitive assessment of the possibility of a leak with the highest sensitivity, specificity, and diagnostic accuracy. It should be implemented in the presence of negative imaging studies when there remains a high suspicion of a leak. Despite the invasiveness of re-exploration, it is a much safer intervention in view of the severe consequences of a delayed diagnosis of this complication.

Fig. 3.2 Upper gastrointestinal study showing extraluminal contrast from the proximal staple line of a gastric sleeve

Adjustable gastric band may occasionally present with a "leak," which is actually due to esophageal or gastric cardiac perforation due to blind tunneling in the retro-gastric fat during placement of the device. The most common location of leakage after a sleeve gastrectomy is at the proximal end of the staple line near the gastroesophageal junction followed by the site of intersection of consecutive staple lines. The gastrojejunostomy is considered as the high-risk anastomosis for a leak in a Roux-en-Y gastric bypass surgery.

Meticulous dissection, good surgical technique with gentle handling of tissues, and avoidance of tissue injury and ischemia are essential in preventing the occurrence and thereby avoiding the need to manage anastomotic/staple-line leaks. Most surgeons, including the authors, test the integrity of this anastomosis intraoperatively by insufflation of air via an orogastric tube, instillation of methylene blue through the same route, or flexible gastroscopy while keeping the anastomosis submerged in saline. Intraoperative gastroscopy is popular as it may also help in control of staple-line bleeds along with prompt evaluation of integrity of the anastomosis [53, 54]. Some authors advocate the use of fibrin sealant around the anastomosis to achieve better hemostasis around the suture line and prevent or decrease the incidence of anastomotic leaks [55].

When identified early in the postoperative course in a patient with unstable hemodynamics and/or florid sepsis, a return trip to the operating room is mandated for the management of leaks. A laparoscopic or open approach may be adopted based on the skill and expertise of the surgeon and associated patient factors. Control of the leak with possible repair, copious lavage of the peritoneal cavity, and placement of closed suction drains in

the area of the leak and other dependent spaces are the mainstays of its operative management. On occasions when surgical repair of these leaks are not possible (e.g., patients with delayed presentation and associated inflammatory reaction and friable tissue), lavage with wide drainage alone is considered safe. With a potential for prolonged limited oral intake after operative drainage, one should consider placement of a feeding gastrostomy in the gastric remnant of patients post-Roux-en-Y gastric bypass or a feeding jejunostomy in SG and DS patients. A naso-enteral feeding tube can also be considered for enteral nutrition to aid in the healing of the leak [56].

Placement of an intraluminal endoprosthesis may be considered to manage proximal leaks from gastric sleeves. These allow adequate enteral diversion to aid in optimal healing when left in situ over 4–6 weeks [57]. However, the risk of migration and erosion of enteric stents appear to overshadow the benefits. Occlusion of the leak by injection of fibrin glue also shows promise. Management of leaks in patients who are not amenable to placement of stents with endoclips and Over-the-Scope Clip (OTSC) (Ovesco Endoscopy, Tübingen, Germany) has also been reported and these may be employed based on the expertise of the surgeon or gastroenterologist [58, 59].

If the patient is hemodynamically stable and does not demonstrate any signs of sepsis, non-operative management with fluid resuscitation, intravenous antibiotics, and bowel rest may be considered along with placement of percutaneous drains in the intra-abdominal collections [56].

3.7 Acute Complications Specific to Adjustable Gastric Band

Laparoscopic adjustable gastric banding is a safe bariatric procedure. However, these patients can present with unique complications that must be recognized and managed appropriately to achieve good outcomes. The major early complications include band slippage, band erosion, stomal obstruction secondary to a food bolus, malposition of the band, port infection, and port or tubing malfunction.

3.7.1 Band Slippage

Slippage of an adjustable gastric band may be defined as a cephalad prolapse of the body of the stomach through the band or caudal movement of the band. Its reported incidence is between 1 and 20 % across studies over the years. The normal location of the band is at the angle of His. When slippage of the band occurs, complete stomal obstruction of the stomach can be precipitated secondary to proximal protrusion of a larger cross-sectional area of gastric body through the narrow diameter of the band. Placement of gastro-gastric tunnel stitches, usually two or more around the gastric band, is considered important for the prevention of band slippage. Many authors advocate an additional gastropexy stitch between the fundus of the stomach and the left crus of the diaphragm (Birmingham stitch) to prevent this complication [60]. Gastric band slip may be classified into five types. Type I prolapse involves upward migration of the anterior wall of the stomach through the band likely due to improper anterior fixation and disruption of the fixation sutures. Type II prolapse involves herniation of the posterior wall of the stomach through the band due to improper surgical technique. The pars flaccida technique for placement of the band is considered superior to the perigastric technique in minimizing the occurrence of this complication. Type III prolapse is defined as dilation of the proximal stomach pouch without any signs of obstruction or change in the angle of the band. It may be associated with dilation of the lower esophagus. It is caused by elevated distal pressure secondary to band over-inflation or due to overeating over a period of time. Type IV prolapse is an immediate postoperative event due to lower placement of the band on the stomach. Type V prolapse comprises necrosis of the herniated stomach wall as a result of progression of types I and II prolapse. Types I, II, IV, and V present as an acute complication and may mandate urgent/emergent surgical intervention for removal or repositioning of the band based on the presenting signs and symptoms. Type III is a chronic complication, which is managed non-operatively with band

deflation, food-portion control, and observation. Surgical treatment may be necessary if conservative management fails to reduce the size of the proximal stomach [61].

The initial presentation of a patient with band slippage includes persistent abdominal pain, dysphagia, vomiting, regurgitation, and food intolerance, which eventually may progress to gastric necrosis with perforation, upper gastrointestinal bleeding, and aspiration pneumonia. The radiological diagnosis is based on the orientation of the band on plain abdominal X-ray and an enlarged gastric pouch in an upper gastrointestinal contrast study or a computerized tomography of the abdomen with oral contrast (Fig. 3.3).

The first step in the management of this acute complication is complete band deflation by accessing the subcutaneous port with a Huber needle under strict aseptic precautions. Patients may be offered laparoscopic repositioning of the gastric band if conservative management fails to control symptoms. If reduction of substantial prolapse is not feasible or there is evidence of associated intra-abdominal infection, laparoscopic removal of the gastric band should be performed [62].

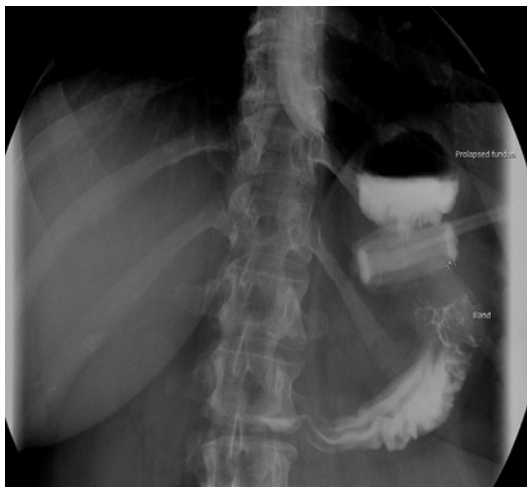

Fig. 3.3 Upper gastrointestinal study showing prolapse of the fundus of stomach secondary to slippage of an adjustable gastric band

3.7.2 Port-Site Infection

Early port-site infections are identified usually within the immediate postoperative period and present frequently as cellulitis. Use of perioperative antibiotics may help in reducing the incidence of this infection. If the infection involves an underlying abscess at the location of the port or failure of antibiotic treatment of the overlying cellulitis, the port should be removed. The proximal end of the tubing may be dropped within the peritoneal cavity for recovery at a later date following resolution of the infection. The possibility of band erosion leading to bacterial seeding of the tubing and access port should be considered and may be ruled out with upper endoscopy.

3.7.3 Port or Tubing Malfunction

Port or tubing malfunction may occur as a result of damage of the port septum or the tubing or as a result of inversion or dislodgement of the port. Damage to the port or to the tubing will result in slow leak of the injected fluid volume and manifest as a feeling of loss of restriction over a period of time after band inflation. This condition can be diagnosed by injecting contrast into the port under fluoroscopy, which will identify the site of leakage of contrast. Damage to the intra-abdominal tubing or the band can also be diagnosed during laparoscopy by injecting methylene blue dye intraoperatively in the port. Based on the site of leakage, surgical treatment may involve replacement of the port, tubing, or the band.

Port inversion may present with difficulty in accessing the port. This can be identified by abdominal radiography. Local exploration of the port site and fixation of the port to the underlying fascia will solve this problem. Surgeons have employed multiple methods of port fixation to avoid this problem including suturing to mesh and tacking the mesh to fascia. The recent addition of automatic fixation devices has greatly facilitated this process and has all but eliminated this problem.

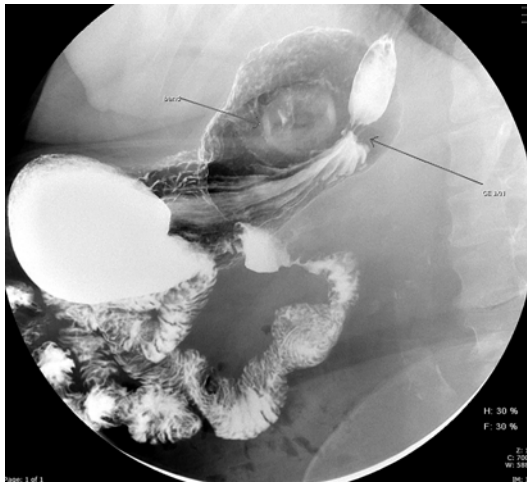

Fig. 3.4 Upper gastrointestinal study showing misplacement of an adjustable gastric band. The transit of contrast occurs outside the gastric band

3.7.4 Malposition of Gastric Band

Malposition or misplacement of gastric bands is a very rare but recognized early complication of gastric band surgery. It presents with symptoms of dysphagia and dyspepsia without any significant associated weight loss. This results due to erroneous placement of the band in the pericardial or retro-gastric fat pad. Misplacement of the band can be identified with a radiologic contrast study where contrast will be found to flow outside the circumference of the band [63] (Fig. 3.4). With diligent identification of anatomical landmarks, this issue can be avoided.

3.8 Acute Complications Specific to Sleeve Gastrectomy

Laparoscopic sleeve gastrectomy is a relatively new and effective surgical option for the management of morbid obesity. Initially conceived as a bridging operation in high-risk patients before biliopancreatic diversion or Roux-en-Y gastric bypass, it has established itself as an independent procedure with great potential. Early major complications include hemorrhage, staple-line leak, mid-gastric stricture, and porto-mesenteric venous thrombosis.

3.8.1 Mid-Gastric Stricture

This is a potential complication occurring in <1 % of patients after a laparoscopic sleeve gastrectomy. It can present acutely after surgery secondary to tissue edema or more commonly in a delayed fashion. It usually results from close proximity of the staple line at the level of the incisura angularis, resulting in an hourglass appearance of the stomach. Further inflammation and scarring at this site lead to formation of the stricture. These patients are at a higher risk for leaks at the proximal staple line due to the presence of narrow diameter and concomitant higher pressure at the level of the incisura. This can be avoided by placing a 34–40 Fr bougie and maintaining an appropriate distance away from the incisura while firing the first couple of stapler loads. The usual presenting symptoms are nausea, vomiting, dysphagia, and food intolerance. An upper gastrointestinal contrast study or endoscopy is diagnostic for this condition.

During its acute presentation, the treatment is usually non-operative with complete bowel rest and intravenous hydration. Symptoms due to mid-gastric stenosis may be caused by postoperative edema and resolve spontaneously in the absence of other pathologies like leak or abscesses. Endoscopic or radiological dilation may be indicated in the event of failure of expectant management. Multiple successive interventions may be required to treat the condition and ameliorate the symptoms [64]. The above may not be effective in patients with long segment stenosis when surgical therapy may be mandated in the form of seromyotomy or conversion to a Roux-en-Y gastric bypass [65].

3.8.2 Porto-Mesenteric Venous Thrombosis

Porto-mesenteric venous thrombosis (PMVT) is an infrequent complication of laparoscopic sleeve gastrectomy. A previous history of VTE is an important predictor for PMVT. It is hypothesized that the division of the short gastric ves-

sels during sleeve gastrectomy with change in blood flow, possible intimal damage of the splenic veins due to direct physical injury while operating in the lesser sac, and dehydration after discharge from the hospital may all contribute to formation of PMVT. The most common symptom on presentation is nonspecific abdominal pain. It may be associated with nausea, vomiting, diarrhea, and gastrointestinal bleeding. Physical examination may vary from low-grade fever, mild abdominal tenderness, and peritoneal signs to florid shock secondary to bowel ischemia. Contrast-enhanced CT scan of the abdomen is diagnostic for this condition. If the patients do not elicit signs of ischemic bowel, therapeutic anticoagulation with heparin is the recommended treatment, which may be subsequently transitioned to oral warfarin. Presence of bowel ischemia without necrosis or perforation may be treated with percutaneous thrombolytic therapy. Bowel necrosis and/or perforation will warrant exploration of the peritoneal cavity with resection of the affected bowel [66]. This raises the debate regarding the use of an antiplatelet agent or an anticoagulant in this patient population to prevent the development of this complication. Due to the relatively rare occurrence of PMVT, the increased risks of bleeding associated with prophylactic therapy should be carefully considered. The authors do not recommend this in their practice as good-quality studies advocating the same in the surgical literature are lacking.

3.9 Acute Complications Specific to Roux-en-Y Gastric Bypass

Gastric bypass has historically been considered as the gold standard procedure for the surgical management of morbid obesity. Until recently, the operation accounted for about 70 % of all bariatric surgeries performed worldwide. The most common early major complications associated with this procedure include anastomotic/staple-line leaks, postoperative hemorrhage, small bowel obstruction (SBO) due to variable etiology, and marginal ulceration.

3.9.1 Small Bowel Obstruction

SBO following a bariatric procedure is associated with considerable morbidity and mortality if not recognized and treated promptly. Obstruction can be classified into two groups based on the time of presentation after the primary surgery. Early SBO presents within the first 30 days of surgery while late SBO manifests beyond 30 days after surgery. The most common etiology of early SBO is an acute obstruction at the enteroenterostomy, attributed to technical problems with the Roux limb, while internal hernia and adhesive disease are responsible for majority of late SBO. Other causes of obstruction include incisional or port-site hernia, intussusception, anastomotic edema, angulation/kinking of the Roux limb, and hemobezoar. Laparoscopic bariatric procedures have interestingly higher incidence of SBO compared with open approach.

The symptoms related to early-onset SBO might be variable. Obstruction of the Roux limb presents with nausea, heartburn, vomiting, midepigastric pain, and upper abdominal fullness, which may be transiently relieved by emesis. A biliopancreatic limb obstruction, on the other hand, may be associated with nausea, abdominal fullness, tachycardia, hiccups, and shoulder and back pain. Gastric remnant dilation is a pathognomonic sign of biliopancreatic limb obstruction, which can subsequently progress to potential gastric necrosis and/or perforation. Common channel obstruction presents with features of both. Abnormal liver function tests and hyperamylasemia can result from obstruction of both the biliopancreatic limb and the common channel. The history and the physical examination of bariatric patients with early obstruction may often be vague. Hence, a high degree of suspicion and prompt and judicious surgical exploration is advocated to identify and treat these obstructions to prevent disruption of the new anastomosis or staple-line or intestinal necrosis, with subsequent perforation and peritonitis. CT scan with oral contrast is essential for the quick diagnosis of early SBO with the pertinent findings being dilated biliopancreatic limb or gastric remnant, location of small bowel loops in the left upper quadrant, and bowel wall thickening with proximal dilation.

3.9.1.1 Obstruction at Entero-Enterostomy

Technical errors contribute to kinking or obstruction at the enteroenterostomy. Postoperative edema, intraluminal hemorrhage with impaction of large clot at the anastomosis, and angulation of the Roux limb may all precipitate this complication. It leads to a closed-loop obstruction involving the biliopancreatic limb and the gastric remnant, which can be rapidly fatal if not recognized and decompressed [67]. Appropriate orientation of the Roux limb and placement of an anti-obstruction stitch (Brolin stitch) to prevent its kinking may help avoid this complication [68]. Stapled closure of the common enterotomy of these anastomoses can lead to increased incidence of obstruction at this site than hand-sewn closure. Intussusception of the jejunojejunostomy into the Roux limb has also been reported in 0.1–0.3 % patients. Surgical intervention should entail bowel resection and revision of anastomosis as it prevents recurrence [69].

3.9.1.2 Internal Hernia

Internal hernia is recognized as a frequent cause of SBO in the bariatric population undergoing laparoscopic surgery. It can occur at three possible locations after gastric bypass surgery: the Petersen defect between the Roux limb's mesentery and transverse mesocolon, the mesenteric defect of the jejunojejunostomy, and the transverse mesocolon defect which exists only in retrocolic bypasses [70]. The symptoms at presentation may suggest the site of obstruction as stated above. Physical examination and multimodality imaging should be implemented to make the diagnosis. The triad of CT scan findings that confirm the presence of an internal hernia includes whirling of the mesentery, location of the cecum and terminal ileum in the right upper quadrant of the abdomen, and the presence of majority of the small bowel loops on one side of the abdominal cavity. A significant number of patients may not have definite signs of an internal hernia due to frequent spontaneous reduction of the hernia. Hence, a very low threshold for surgical exploration is warranted in the presence of subtle signs of obstruction to prevent further morbidity and mortality. Laparoscopy is the procedure of choice where retrograde examination of small bowel should be performed from the ileocecal valve. The internal hernia, upon identification, should be gently reduced and the defect should be closed with non-absorbable suture. The authors recommend complete closure of all mesenteric defects with permanent suture during the primary bypass surgery to minimize occurrence of this complication [71].

3.9.1.3 Trocar-Site Hernia

A port-site/trocar-site hernia is an uncommon complication of laparoscopic surgery found in open fascial defects greater than 10 mm in diameter. Higher BMI is considered a significant risk factor for its development. Identification of such small incisional hernias can be exceedingly difficult in the bariatric population increasing its morbidity. A CT scan of abdomen and pelvis may often be necessary for diagnosis. Diagnostic laparoscopy is recommended for evaluation of the hernia contents and to rule out the presence of ischemic/necrotic bowel prior to repair of the hernia [72]. The authors advocate closure of all fascial defects larger than 1 cm to prevent the development of this complication. Though the omentum or the pre-peritoneal fat may herniate through open defects smaller than 1 cm, the risk of developing a clinically significant hernia involving a loop of small bowel is very low. Hence these small openings may not be closed at the end of the bariatric procedure.

3.9.2 Roux-en-O/Misconstruction

Another rare but devastating complication of laparoscopic gastric bypass includes the inadvertent anastomosis of the biliopancreatic limb to the proximal gastric pouch along with a misconstructed jejunojejunostomy. This so-called Roux-en-O reconstruction leads to a blind loop when patients present with complaints of abdominal pain, biliary emesis and esophagitis, and severe dehydration. Though the patients present with

this complication very early in the postoperative period, the diagnosis is usually delayed despite contrast studies and endoscopies. Hepatobiliary iminodiacetic acid (HIDA) scan is reported to help in the diagnosis by demonstrating reflux of radiotracer from the duodenum into the esophagus. Corrective surgery is necessary upon diagnosis of this problem. Making the Roux limb longer than the biliopancreatic limb may help to avoid this complication. The bowel may also be traced back to the ligament upon completion of the jejunojejunostomy to confirm the anatomy of the bypass [73].

3.9.3 Marginal Ulceration

A marginal ulcer corresponds to a peptic ulcer on the jejunal mucosa near the site of the gastrojejunostomy and has been reported to occur in 0.3–16 % of patients undergoing an RYGB [74]. The risk factors involved in its causation include operative technique, type of suture (absorbable vs. non-absorbable) used, patient age, history of previous gastric surgery, preoperative diabetes, coronary artery disease or peptic ulcer disease, and use of nonsteroid anti-inflammatory medications or tobacco [75]. In recognition of the risk of development of this complication, the patients should be started prophylactically on a proton pump inhibitor immediately after the surgery. However the duration of therapy continues to remain a source of much debate in literature. It is well known that a patient may still develop this complication despite being on prophylactic therapy even in the absence of common risk factors like smoking and alcohol abuse. Epigastric pain is the usual presenting symptom. It may be associated with nausea, vomiting, bleeding, or perforation. Upper GI endoscopy is often diagnostic and the ulcers are treated with proton pump inhibitors and sucralfate. Biopsy should be obtained while performing endoscopy to rule out the presence of *H. pylori* infection. Ulcers that present with intractability or perforation would require surgical revision of the anastomosis.

3.10 Acute Complications Specific to Biliopancreatic Diversion

Biliopancreatic diversion with duodenal switch (DS) yields very good and sustained weight loss where ingested food passes directly through a gastric sleeve into the ileum, bypassing the duodenum and jejunum. It is associated with an increased risk of complications. Most common early complications encountered in this patient population include bleeding, anastomotic and staple-line leaks, marginal ulcerations, SBO secondary to internal and incisional hernias, nutritional deficiencies, and anastomotic stenosis. The increased rate of complications is not surprising because DS is a longer and more complex procedure, requiring extended dissection, additional anastomoses, and a larger operating field because of the more distal enteroenterostomy compared with RYGB. Moreover, DS yields an additional number of potential leak sites, i.e., along the gastric sleeve and the divided duodenal bulb. This procedure is also notorious for its severe micro- and macronutrient deficiencies. However, these usually do not manifest in an acute setting.

3.11 Conclusion

Bariatric surgery is safe with a low rate of overall postoperative complications. However, these few complications have the potential to evolve into surgical emergencies with significant associated morbidity and mortality. Prevention, timely diagnosis, and prompt perioperative treatment can help avoid adverse outcomes.

References

1. NIH conference. Gastrointestinal surgery for severe obesity. Consensus development conference panel. Ann Intern Med. 1991;115(12):956–61.
2. Guidelines for laparoscopic and open surgical treatment of morbid obesity. American Society for Bariatric Surgery. Society of American Gastrointestinal Endoscopic Surgeons. Obes Surg. 2000;10(4):378–9.

3. Dumon KR, Murayama KM. Bariatric surgery outcomes. Surg Clin North Am. 2011;91(6):1313–38, x.
4. Thomas H, Agrawal S. Systematic review of obesity surgery mortality risk score—preoperative risk stratification in bariatric surgery. Obes Surg. 2012;22(7):1135–40.
5. Arterburn DE, Courcoulas AP. Bariatric surgery for obesity and metabolic conditions in adults. BMJ. 2014;349:g3961.
6. Lancaster RT, Hutter MM. Bands and bypasses: 30-day morbidity and mortality of bariatric surgical procedures as assessed by prospective, multi-center, risk-adjusted ACS-NSQIP data. Surg Endosc. 2008;22(12):2554–63.
7. Stenberg E, Szabo E, Agren G, Naslund E, Boman L, Bylund A, et al. Early complications after laparoscopic gastric bypass surgery: results from the Scandinavian Obesity Surgery Registry. Ann Surg. 2013.
8. Greenstein AJ, Wahed AS, Adeniji A, Courcoulas AP, Dakin G, Flum DR, et al. Prevalence of adverse intraoperative events during obesity surgery and their sequelae. J Am Coll Surg. 2012;215(2):271–7, e3.
9. Kuruba R, Koche LS, Murr MM. Preoperative assessment and perioperative care of patients undergoing bariatric surgery. Med Clin North Am. 2007;91(3):339–51, ix.
10. Mechanick JI, Youdim A, Jones DB, Garvey WT, Hurley DL, McMahon MM, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient—2013 update: cosponsored by American Association of Clinical Endocrinologists, the Obesity Society, and American Society for Metabolic & Bariatric Surgery. Endocr Pract. 2013;19(2):337–72.
11. Marek RJ, Ben-Porath YS, Ashton K, Heinberg LJ. Minnesota multiphasic personality inventory-2 restructured form (MMPI-2-RF) scale score differences in bariatric surgery candidates diagnosed with binge eating disorder versus BMI-matched controls. Int J Eat Disord. 2014;47(3):315–9.
12. Tarescavage AM, Wygant DB, Boutacoff LI, Ben-Porath YS. Reliability, validity, and utility of the Minnesota Multiphasic Personality Inventory-2-Restructured Form (MMPI-2-RF) in assessments of bariatric surgery candidates. Psychol Assess. 2013;25(4):1179–94.
13. Canetti L, Berry EM, Elizur Y. Psychosocial predictors of weight loss and psychological adjustment following bariatric surgery and a weight-loss program: the mediating role of emotional eating. Int J Eat Disord. 2009;42(2):109–17.
14. Wimmelmann CL, Dela F, Mortensen EL. Psychological predictors of weight loss after bariatric surgery: a review of the recent research. Obes Res Clin Pract. 2014;8(4):e299–313.
15. Kinzl JF, Schrattenecker M, Traweger C, Mattesich M, Fiala M, Biebl W. Psychosocial predictors of weight loss after bariatric surgery. Obes Surg. 2006;16(12):1609–14.
16. Peterhansel C, Petroff D, Klinitzke G, Kersting A, Wagner B. Risk of completed suicide after bariatric surgery: a systematic review. Obes Rev. 2013;14(5):369–82.
17. King 3rd SB, Smith Jr SC, Hirshfeld Jr JW, Jacobs AK, Morrison DA, Williams DO, et al. 2007 Focused Update of the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines: 2007 Writing Group to Review New Evidence and Update the ACC/AHA/SCAI 2005 Guideline Update for Percutaneous Coronary Intervention, Writing on Behalf of the 2005 Writing Committee. Circulation. 2008;117(2):261–95.
18. Hai F, Porhomayon J, Vermont L, Frydrych L, Jaoude P, El-Solh AA. Postoperative complications in patients with obstructive sleep apnea: a meta-analysis. J Clin Anesth. 2014;26(8):591–600.
19. Ryan CF, Lowe AA, Li D, Fleetham JA. Magnetic resonance imaging of the upper airway in obstructive sleep apnea before and after chronic nasal continuous positive airway pressure therapy. Am Rev Respir Dis. 1991;144(4):939–44.
20. Golbin JM, Somers VK, Caples SM. Obstructive sleep apnea, cardiovascular disease, and pulmonary hypertension. Proc Am Thorac Soc. 2008;5(2):200–6.
21. Van Nieuwenhove Y, Dambrauskas Z, Campillo-Soto A, van Dielen F, Wiezer R, Janssen I, et al. Preoperative very low-calorie diet and operative outcome after laparoscopic gastric bypass: a randomized multicenter study. Arch Surg. 2011;146(11):1300–5.
22. Isom KA, Andromalos L, Ariagno M, Hartman K, Mogensen KM, Stephanides K, et al. Nutrition and metabolic support recommendations for the bariatric patient. Nutr Clin Pract. 2014.
23. Gobato RC, Seixas Chaves DF, Chaim EA. Micronutrient and physiologic parameters before and 6 months after RYGB. Surg Obes Relat Dis. 2014.
24. Cole AJ, Beckman LM, Earthman CP. Vitamin D status following bariatric surgery: implications and recommendations. Nutr Clin Pract. 2014.
25. Juvin P, Lavaut E, Dupont H, Lefevre P, Demetriou M, Dumoulin JL, et al. Difficult tracheal intubation is more common in obese than in lean patients. Anesth Analg. 2003;97(2):595–600, table of contents.
26. Sheff SR, May MC, Carlisle SE, Kallies KJ, Mathiason MA, Kothari SN. Predictors of a difficult intubation in the bariatric patient: does preoperative body mass index matter? Surg Obes Relat Dis. 2013;9(3):344–9.
27. Levitan RM, Mechem CC, Ochroch EA, Shofer FS, Hollander JE. Head-elevated laryngoscopy position: improving laryngeal exposure during laryngoscopy by increasing head elevation. Ann Emerg Med. 2003;41(3):322–30.

28. Ogunnaike BO, Jones SB, Jones DB, Provost D, Whitten CW. Anesthetic considerations for bariatric surgery. Anesth Analg. 2002;95(6):1793–805.
29. Saliman JA, Benditt JO, Flum DR, Oelschlager BK, Dellinger EP, Goss CH. Pulmonary function in the morbidly obese. Surg Obes Relat Dis. 2008;4(5):632–9. discussion 9.
30. Pelosi P, Gregoretti C. Perioperative management of obese patients. Best Pract Res Clin Anaesthesiol. 2010;24(2):211–25.
31. Chakravartty S, Sarma DR, Patel AG. Rhabdomyolysis in bariatric surgery: a systematic review. Obes Surg. 2013;23(8):1333–40.
32. Nelson DW, Blair KS, Martin MJ. Analysis of obesity-related outcomes and bariatric failure rates with the duodenal switch vs gastric bypass for morbid obesity. Arch Surg. 2012;147(9):847–54.
33. Freeman JT, Anderson DJ, Hartwig MG, Sexton DJ. Surgical site infections following bariatric surgery in community hospitals: a weighty concern? Obes Surg. 2011;21(7):836–40.
34. Fischer MI, Dias C, Stein A, Meinhardt NG, Heineck I. Antibiotic prophylaxis in obese patients submitted to bariatric surgery. A systematic review. Acta Cir Bra. 2014;29(3):209–17.
35. Bratzler DW, Dellinger EP, Olsen KM, Perl TM, Auwaerter PG, Bolon MK, et al. Clinical practice guidelines for antimicrobial prophylaxis in surgery. Am J Health Syst Pharm. 2013;70(3):195–283.
36. Dossett LA, Dageforde LA, Swenson BR, Metzger R, Bonatti H, Sawyer RG, et al. Obesity and site-specific nosocomial infection risk in the intensive care unit. Surg Infect (Larchmt). 2009;10(2):137–42.
37. Winegar DA, Sherif B, Pate V, DeMaria EJ. Venous thromboembolism after bariatric surgery performed by Bariatric Surgery Center of Excellence Participants: analysis of the Bariatric Outcomes Longitudinal Database. Surg Obes Relat Dis. 2011;7(2):181–8.
38. Sapala JA, Wood MH, Schuhknecht MP, Sapala MA. Fatal pulmonary embolism after bariatric operations for morbid obesity: a 24-year retrospective analysis. Obes Surg. 2003;13(6):819–25.
39. Finks JF, English WJ, Carlin AM, Krause KR, Share DA, Banerjee M, et al. Predicting risk for venous thromboembolism with bariatric surgery: results from the Michigan Bariatric Surgery Collaborative. Ann Surg. 2012;255(6):1100–4.
40. Birkmeyer NJ, Finks JF, Carlin AM, Chengelis DL, Krause KR, Hawasli AA, et al. Comparative effectiveness of unfractionated and low-molecular-weight heparin for prevention of venous thromboembolism following bariatric surgery. Arch Surg. 2012;147(11):994–8.
41. American Society for M, Bariatric Surgery Clinical Issues C. ASMBS updated position statement on prophylactic measures to reduce the risk of venous thromboembolism in bariatric surgery patients. Surg Obes Relat Dis. 2013;9(4):493–7.
42. Birkmeyer NJ, Share D, Baser O, Carlin AM, Finks JF, Pesta CM, et al. Preoperative placement of inferior vena cava filters and outcomes after gastric bypass surgery. Ann Surg. 2010;252(2):313–8.
43. Dapri G, Cadiere GB, Himpens J. Reinforcing the staple line during laparoscopic sleeve gastrectomy: prospective randomized clinical study comparing three different techniques. Obes Surg. 2010;20(4):462–7.
44. Knapps J, Ghanem M, Clements J, Merchant AM. A systematic review of staple-line reinforcement in laparoscopic sleeve gastrectomy. JSLS. 2013;17(3):390–9.
45. Pilone V, Di Micco R, Monda A, Villamaina E, Forestieri P. Use of Tachosil(R) in bariatric surgery: preliminary experience in control of bleeding after sleeve gastrectomy. Minerva Chir. 2012;67(3):241–8.
46. D'Ugo S, Gentileschi P, Benavoli D, Cerci M, Gaspari A, Berta RD, et al. Comparative use of different techniques for leak and bleeding prevention during laparoscopic sleeve gastrectomy: a multicenter study. Surg Obes Relat Dis. 2014;10(3):450–4.
47. Dick A, Byrne TK, Baker M, Budak A, Morgan K. Gastrointestinal bleeding after gastric bypass surgery: nuisance or catastrophe? Surg Obes Relat Dis. 2010;6(6):643–7.
48. Bal BS, Finelli FC, Shope TR, Koch TR. Nutritional deficiencies after bariatric surgery. Nat Rev Endocrinol. 2012;8(9):544–56.
49. Landais A. Neurological complications of bariatric surgery. Obes Surg. 2014;24(10):1800–7.
50. Parikh M, Issa R, McCrillis A, Saunders JK, Ude-Welcome A, Gagner M. Surgical strategies that may decrease leak after laparoscopic sleeve gastrectomy: a systematic review and meta-analysis of 9991 cases. Ann Surg. 2013;257(2):231–7.
51. Kolakowski Jr S, Kirkland ML, Schuricht AL. Routine postoperative upper gastrointestinal series after Roux-en-Y gastric bypass: determination of whether it is necessary. Arch Surg. 2007;142(10):930–4. discussion 4.
52. Gonzalez R, Sarr MG, Smith CD, Baghai M, Kendrick M, Szomstein S, et al. Diagnosis and contemporary management of anastomotic leaks after gastric bypass for obesity. J Am Coll Surg. 2007;204(1):47–55.
53. Mohos E, Schmaldienst E, Richter D, Prager M. Examination of the efficacy and safety of intraoperative gastroscopic testing of the gastrojejunal anastomosis in laparoscopic Roux Y gastric bypass surgery. Obes Surg. 2011;21(10):1592–6.
54. Alasfar F, Chand B. Intraoperative endoscopy for laparoscopic Roux-en-Y gastric bypass: leak test and beyond. Surg Laparosc Endosc Percutan Tech. 2010;20(6):424–7.
55. Lee MG, Provost DA, Jones DB. Use of fibrin sealant in laparoscopic gastric bypass for the morbidly obese. Obes Surg. 2004;14(10):1321–6.
56. Jacobsen HJ, Nergard BJ, Leifsson BG, Frederiksen SG, Agajahni E, Ekelund M, et al. Management of suspected anastomotic leak after bariatric laparoscopic Roux-en-Y gastric bypass. Br J Surg. 2014;101(4):417–23.

57. Jurowich C, Thalheimer A, Seyfried F, Fein M, Bender G, Germer CT, et al. Gastric leakage after sleeve gastrectomy-clinical presentation and therapeutic options. Langenbecks Arch Surg. 2011;396(7):981–7.
58. Keren D, Eyal O, Sroka G, Rainis T, Raziel A, Sakran N, et al. Over-the-Scope Clip (OTSC) System for Sleeve Gastrectomy Leaks. Obes Surg. 2014.
59. Ritter LA, Wang AY, Sauer BG, Kleiner DE. Healing of complicated gastric leaks in bariatric patients using endoscopic clips. JSLS. 2013;17(3):481–3.
60. Singhal R, Kitchen M, Ndirika S, Hunt K, Bridgwater S, Super P. The "Birmingham stitch"—avoiding slippage in laparoscopic gastric banding. Obes Surg. 2008;18(4):359–63.
61. Eid I, Birch DW, Sharma AM, Sherman V, Karmali S. Complications associated with adjustable gastric banding for morbid obesity: a surgeon's guides. Can J Surg. 2011;54(1):61–6.
62. Snow JM, Severson PA. Complications of adjustable gastric banding. Surg Clin North Am. 2011;91(6): 1249–64, ix.
63. Labib PL, Agrawal S. An unusual case of significant weight loss following malposition of a laparoscopic adjustable gastric band. Obes Facts. 2012;5(4):625–8.
64. Parikh A, Alley JB, Peterson RM, Harnisch MC, Pfluke JM, Tapper DM, et al. Management options for symptomatic stenosis after laparoscopic vertical sleeve gastrectomy in the morbidly obese. Surg Endosc. 2012;26(3):738–46.
65. Dapri G, Cadiere GB, Himpens J. Laparoscopic seromyotomy for long stenosis after sleeve gastrectomy with or without duodenal switch. Obes Surg. 2009;19(4):495–9.
66. Goitein D, Matter I, Raziel A, Keidar A, Hazzan D, Rimon U, et al. Portomesenteric thrombosis following laparoscopic bariatric surgery: incidence, patterns of clinical presentation, and etiology in a bariatric patient population. JAMA Surg. 2013;148(4):340–6.
67. Pazouki A, Pakaneh M, Khalaj A, Tamannaie Z, Jangjoo A, Shapoori P, et al. Blood bezoar causing obstruction after laparoscopic Roux-en-Y gastric bypass. Int J Surg Case Rep. 2014;5(4):183–5.
68. Brolin RE. The antiobstruction stitch in stapled Roux-en-Y enteroenterostomy. Am J Surg. 1995;169(3): 355–7.
69. Singla S, Guenthart BA, May L, Gaughan J, Meilahn JE. Intussusception after laparoscopic gastric bypass surgery: an underrecognized complication. Minim Invasive Surg. 2012;2012:464853.
70. Elms L, Moon RC, Varnadore S, Teixeira AF, Jawad MA. Causes of small bowel obstruction after Roux-en-Y gastric bypass: a review of 2,395 cases at a single institution. Surg Endosc. 2014;28(5): 1624–8.
71. Brolin RE, Kella VN. Impact of complete mesenteric closure on small bowel obstruction and internal mesenteric hernia after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2013;9(6):850–4.
72. Scozzari G, Zanini M, Cravero F, Passera R, Rebecchi F, Morino M. High incidence of trocar site hernia after laparoscopic or robotic Roux-en-Y gastric bypass. Surg Endosc. 2014;28(10):2890–8.
73. Sherman V, Dan AG, Lord JM, Chand B, Schauer PR. Complications of gastric bypass: avoiding the Roux-en-O configuration. Obes Surg. 2009;19(8): 1190–4.
74. Csendes A, Burgos AM, Altuve J, Bonacic S. Incidence of marginal ulcer 1 month and 1 to 2 years after gastric bypass: a prospective consecutive endoscopic evaluation of 442 patients with morbid obesity. Obes Surg. 2009;19(2):135–8.
75. El-Hayek K, Timratana P, Shimizu H, Chand B. Marginal ulcer after Roux-en-Y gastric bypass: what have we really learned? Surg Endosc. 2012; 26(10):2789–96.

Thromboembolic Disease in the Bariatric Patient: Prevention, Diagnosis, and Management

4

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Key Points

- Approximately 370,000 patients are diagnosed with VTE annually and at least the same number of patients with VTE are undiagnosed.
- The majority of VTE are related to specific trigger events, often caused by acquired and/or inherited risk factors.
- Pulmonary embolism is the most common preventable cause of in-hospital death.
- VTE after bariatric surgery is relatively uncommon, with most studies reporting rates between 0.17 and 0.5 %.
- There appears to be an increasing trend in the portal venous thrombosis rate seen in sleeve gastrectomy patients, although the data are preliminary and inconclusive at the time of this publication.

4.1 Introduction

Patients admitted to the hospital for medical care and surgery occasionally suffer from venous thromboembolism (VTE), which is defined as deep vein thrombosis (DVT), pulmonary embolism (PE), or both. Significant morbidity and mortality are realized each year as approximately 300,000–600,000 individuals suffer from VTE, and some of those individuals will die from PE [1]. The overall age- and sex-adjusted annual incidence of VTE was 1.17 per 1000 (.48 per 1000 for DVT and .69 per 1000 for PE). It has been shown that the incidence of VTE increases with age, with rates increasing dramatically after 50 years of age [2]. Knowing that advanced age and obesity are risk factors for VTE, it is reasonable to assume that as the average age of the population and the obesity rate in the USA increase, there will be a considerable increase in the incidence of VTE.

Some estimates suggest that VTE causes more deaths each year than breast cancer, HIV, or motor vehicle crashes—illnesses or injuries that are well understood by most Americans. In contrast, a telephone survey taken by the American Public Health Association found that fewer than 1 in 10 Americans understood DVT, its symptoms, and risk factors [2]. In addition, clinicians were not ordering VTE prophylaxis of any form on a regular basis. One study in 2007 analyzed 1375 hospitalized non-orthopedic surgery patients in a prospective registry of 5451 patients with ultrasound confirmed deep vein thrombosis (DVT) from 183 hospitals in the USA. Compared to medical patients, surgical patients presented with a more occult clinical

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_4

picture and complained less often of extremity edema, extremity discomfort, or difficulty walking. Immobility within 30 days of DVT diagnosis, prior hospitalization within 30 days of DVT diagnosis, presence of an indwelling central venous catheter, obesity (BMI > 30 kg/m^2), and previous smoking were the most common VTE risk factors among surgical patients. Among surgical patients who developed DVT, some form of prophylaxis had been used in only 44 %. Once diagnosed with DVT, surgical patients received IVC filters more often than medical patients [3]. These facts were critical in the decision for the surgeon general to publish a call to action for the prevention of VTE in 2008.

Awareness of VTE and DVT prophylaxis has increased considerably since the release of the surgeon general's publication. However, there is no consensus on the approach one should utilize to prevent VTE. This is evident by the wide variation of philosophy and practice pertaining to VTE prophylaxis reported in the literature. One study describes current practice patterns of VTE screening and prophylaxis in high-risk bariatric surgery. Nearly all surgeons agree on risk factors that qualify patients as high risk, but only half routinely screen patients preoperatively. Nearly all surgeons use preoperative VTE chemoprophylaxis, but the duration of therapy varies [4].

In efforts to increase VTE awareness, prevention, and the use of chemoprophylaxis, the Centers for Medicare and Medicaid Services initiated a nonpayment policy for certain hospital-acquired conditions (HACs), so-called never events, in 2008; VTE is one of the diagnoses listed on the HAC list. A recent study calls into question the decision for such policy given that there are factors inherent to the bariatric patient population that cannot be influenced [5].

DVT and PE are easy to overlook because the signs and symptoms are often difficult to recognize. In many cases, clinical signs are not readily apparent. As many as 50 % of the cases of DVT are "silent," and sometimes the first symptom of DVT is a fatal PE. PE is often undiagnosed, and thus the true death rate is almost certainly substantially higher. The surgeon general's report cited that the diagnosis of PE is often missed and was correctly confirmed on autopsy in only 39 to 50 % of patients. In a review of autopsies performed after bariatric surgery, it was revealed that PE was the direct cause of death in 30 % of patients. However, 80 % of patients were noted to have PEs [6].

Roughly 30–40 % of those who have a DVT in a given year will suffer from a recurrent event within the next 10 years, with the risk being greatest in the first 2 years. Patients who were diagnosed with an initial "spontaneous" VTE were more likely to experience a recurrent VTE, provided that there were no inciting events such as trauma, surgery, or hormonal changes due to pregnancy, oral contraceptives, or hormone replacement. Patients with symptomatic PE tend to have a higher risk of recurrent VTE than those presenting with DVT symptoms alone. The recurrence in those who initially presented with PE is more likely to be another embolism (as opposed to DVT alone) [2].

Chronic venous insufficiency (CVI) and post-thrombotic syndrome have been shown to occur in over 50 % of VTE patients followed for 10 years, while 6 % developed severe disease. CVI occurs when the blood clot injures or destroys one or more of the venous valves that are located in the deep veins of the leg. When functioning properly, valves work against gravity to enable blood flow back to the heart. When valves are working improperly, individuals may experience long-term complications that include swelling, pain, discoloration, and, in severe cases, ulcers in the affected limb which tend to be permanent and irreversible. CVI has been found to cause a significant reduction in the quality of life, similar to the impact caused by chronic heart, lung, or arthritic disease [2].

The economic costs of treating VTE and its complications can be quite substantial. Treatment costs of a single VTE event range from $10,000 to over $16,000 per person. Consequently, each year over 2 billion dollars are spent on VTE treatment, attributable to costs associated with both new and recurrent events [7]. When looking at the economic impact of surgical procedures alone, one study reports excess hospital stay and charges of up to $10,000 per event, translating to

55 million dollars [8]. More research is needed on both the direct and indirect costs and how it affects individuals, families, and society at large. Many individuals suffering from the sequelae of VTE may find it difficult to remain productive members of the workforce, thus creating an even greater economic strain on family finances as well as the overall economy.

4.2 Mechanism of VTE

Within the highly regulated intrinsic and extrinsic pathways for hemostasis, a delicate balance is maintained between a controlled bleeding response toinjuryandactivitytopreventhypercoagulabilityand thrombosis (Fig. 4.1).

In 1856, Virchow (Fig. 4.2) proposed a triad of events leading to venous thrombosis. He proposed that stasis of blood flow, hypercoagulability of the blood, and damage to the vascular endothelium are associated with thrombosis (Fig. 4.3). It has since become increasingly clear that one or more pathophysiologic factors of Virchow's triad are a part of any risk leading to the development of DVT.

Patients who are hospitalized for acute medical illness and urgent or emergent major surgery are known to be associated with more than a tenfold increased risk for VTE. Most hospitalized patients have at least one risk factor, including immobility, cancer, infection, and/or surgery. When VTE prophylaxis is not used, studies have shown 16 to 55 % of medical and general surgery patients, and 40 to 60 % of patients requiring major orthopedic surgery develop thrombosis. It has been reported that approximately 10 % of hospital deaths are related to PE; many times this disease was not suspected before death [2].

The majority of DVT/PE events are related to specific, identifiable triggering events such as prolonged periods of immobility, hospitalization, major surgery, and trauma. Acquired and/or inherited risk factors are often present in patients who experience a triggering event leading to the development of a DVT or PE.

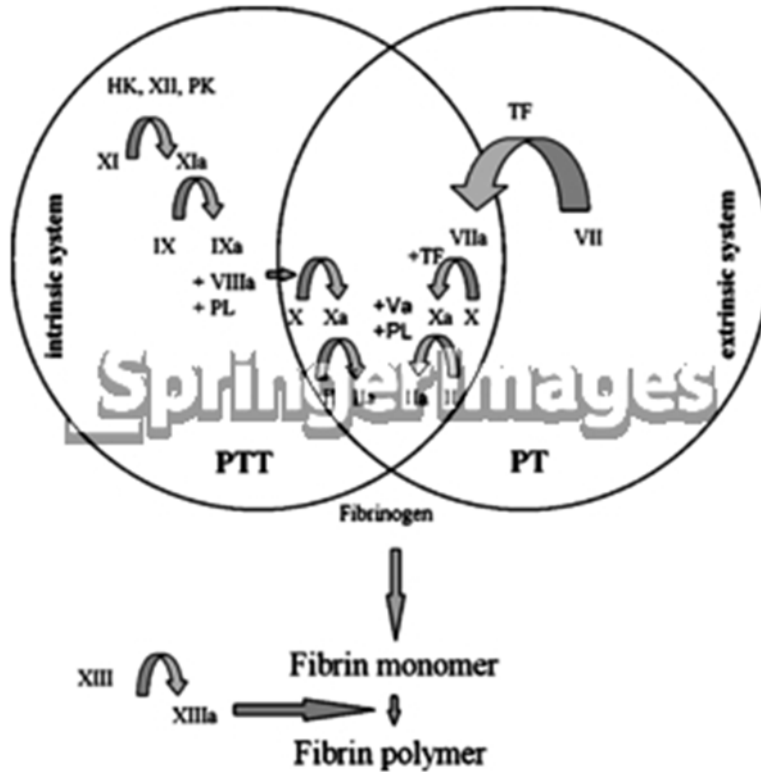

Fig. 4.1 Intrinsic and extrinsic coagulation pathways

Fig. 4.2 Rudolph Virchow proposed, in 1856, a triad of events (Fig. 4.3) that are necessary to the development of venous thrombosis. With permission from Jatoi I. Surgical Considerations in the Management of Primary Invasive Breast Cancer. In: Jatoi I, Kaufmann M, eds. Management of Breast Diseases, 2010. Springer, New York; pp. 227–241 [99] © Springer

4.3 Risk Factors for VTE

There are many inherited and acquired risk factors associated with VTE and recurrent VTE (Table 4.1). Strong genetic risk factors that lead to a hypercoagulable state include deficiencies in the anticoagulants antithrombin, protein C, and protein S. Moderate genetic risk factors include factor V Leiden, prothrombin G20210A, and non-type O blood (Table 4.2). Acquired risk factors include age, surgery, obesity, cancer, pregnancy, hormone-based contraceptives, hormone replacement, antiphospholipid syndrome, acute infection, immobilization, indwelling catheter use, paralysis, prolonged travel, smoking, hospitalization, reduced fibrinolysis, and acquired thrombophilia.

Plasma markers used to screen for inherited thrombophilia include factor V Leiden mutation, low protein C activity, low protein S activity, and free protein S deficiency. Markers for acquired thrombophilia include D-dimer elevation, fibrinogen elevation, elevation of coagulation factors VIII, IX, and XI, elevation of lupus anticoagulants and homocysteine level, and antithrombin III deficiency (Table 4.2). One study screened

Fig. 4.3 Virchow's triad

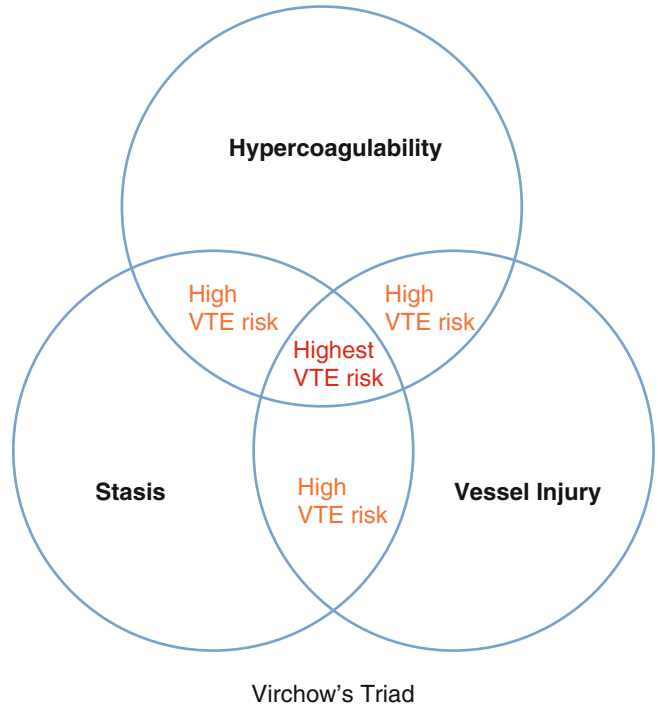

Table 4.1 Risk factors associated with VTE

Hypercoagulability	Hypercoagulability	Stasis (abnormal blood flow)	Vessel injury
Genetic:	**Acquired:**	• Immobility	• Surgery
• Factor V Leiden	• Malignancy	• Polycythemia	• Trauma
• Prothrombin G20210A	• Chemotherapy	• Atrial fibrillation	• Venipuncture
• Protein C and S deficiency	• Oral contraceptive use	• LV dysfunction	• Indwelling catheter
• Antithrombin III deficiency	• Hormonal replacement therapy	• Venous insufficiency	• Atherosclerosis
• Activated protein C resistance	• Pregnancy	• Varicose veins	• Hypertension
• Blood group non-O	• Heparin-induced thrombocytopenia	• Venous obstruction due to obesity and/or pregnancy	• Toxins (smoking)
• Single-nucleotide polymorphism (fibrinogen, factor V, factor XI, other factors)	• Obesity	• Bradycardia	
	• Advanced age (>50 years)	• Hypotension	
	• Antiphospholipid syndrome	**Turbulent blood flow:**	
	• Inflammation	• Heart valve disease or replacement	
	• Sepsis	• Atherosclerotic plaque	
	• Nephrotic syndrome		
	• Inflammatory bowel disease		

Table 4.2 Prevalence of familial and acquired thrombophilia

Condition	Prevalence in Caucasian population, %	Incidence of VTE, % (relative risk)	Incidence of recurrent VTE, % (relative risk)
Factor V Leiden	3–7	12–20 (4.3)	40–50 (1.3)
Prothrombin 20210A	1–3	3–8 (1.9)	15–20 (1.4)
Protein C deficiency	0.02–0.05	2–5 (11.3)	5–10 (2.5)
Protein S deficiency	0.001–1	1–3 (32.4)	5–10 (2.5)
Antithrombin III deficiency	0.02–0.04	1–2 (17.5)	2–5 (2.5)

bariatric surgery candidates and found that serologic markers occurred more frequently than what would be expected in the general population. Results included D-dimer elevation in 31 %, fibrinogen elevation in 40 %, factor VIII elevation in 50 %, factor IX elevation in 64 %, factor XI elevation in 50 %, and lupus anticoagulant in 13 % [9]. One study identified clinical markers of a hypercoagulable state using rotational thromboelastometry (ROTEM) in patients being prepared for bariatric surgery. ROTEM detects hyperfunctional changes to determine if a hypercoagulable state exists by looking at clot starting time, clot formation time to 20 mm, maximum clot firmness, and clot lysis. Metabolic and inflammatory markers, such as leptin, C-reactive protein, fibrinogen levels, and platelet count, were noted to be significantly higher in the high-risk patients and it was concluded that a hypercoagulable state is associated with central obesity and high fibrinogen levels [10].

4.3.1 Genetic Factors That Increase VTE Risk

Thrombophilia is an inherited blood clotting disorder caused by one or more genetic risk factors or mutations that make a person susceptible to VTE. The risks for VTE are much greater for those

individuals with thrombophilia compared to the population at large, particularly for those who also have another risk, such as surgery, hospitalization, or a prolonged bed stay or prolonged travel.

4.3.1.1 Antithrombin III, Protein C, and Protein S Deficiency

Mutations in the genes that produce protein C and its cofactor protein S are found in less than 1 % of the population, while deficiencies in the gene that produces antithrombin are found in roughly 1 in 5000 individuals. There is a tenfold increase in the risk of thrombosis in patients with deficiencies in the protein C, protein S, and antithrombin. The highest risk is seen in patients with antithrombin deficiency.

4.3.1.2 Factor V Leiden

Factor V Leiden is a relatively common mutation in the gene for clotting factor V and is resistant to inactivation by activated protein C, which leads to an increased risk of VTE. This genetic defect is most commonly found among Caucasians of European origin.

4.3.1.3 Prothrombin G20210A

Prothrombin G20210A is single-nucleotide polymorphism in the 3′ untranslated region of the prothrombin gene that leads to increased expression. Roughly 2–3 % of Caucasians have a mutation in the gene that produces prothrombin (clotting factor II). Approximately 6 % of all VTE patients have this mutation, which leads to a threefold increase in the risk of thrombosis.

4.3.1.4 Fibrinogen C10034T

Fibrinogen C10034T is a fibrinogen gamma-chain gene variant associated with increased venous thrombosis.

4.3.1.5 Non-O Blood Type

Certain blood types, especially when combined with certain genetic mutations, constitutes the most significant risk factor for formation of VTE; significantly higher than risk associated with factor V Leiden or prothrombin G20210A alone. Individuals with blood type O have lower von Willebrand factor (vWF) and factor VIII levels than non-O blood group individuals. While hemorrhagic diathesis can be seen in patients deficient in vWF, elevated vWF levels are associated with increased risk of VTE. A two-fold increased risk of a first DVT has been shown in patients with non-O blood type, and VTE recurrence has been associated with blood type B. Non-O blood type also strongly influenced the risk of thrombosis in patients who were factor V Leiden carriers [11].

An individual with a genetic mutation will not necessarily develop a VTE, and fewer than 10 % of those who carry the most common mutations will develop a detectable blood clot each year. At least one-third of patients diagnosed with a DVT will have at least one genetic mutation associated with increased VTE risk [2].

Taking a good family history is vital in a surgeon's effort to decrease the incidence of VTE in bariatric surgery patients. In almost all cases where there was a presence of an inherited hypercoagulable state, at least one of the parents also had the disorder, and there is a 50 % chance that a sibling or child will have the disorder as well. Other blood relatives, including aunts, uncles, and cousins, may also be affected.

4.3.2 Acquired Hypercoagulable States

Acquired hypercoagulable states make patients more susceptible to VTE and can be seen in patients undergoing surgery or requiring a prolonged hospitalization. More details of how acquired risk factors may affect bariatric surgery patients are reviewed below.

4.3.2.1 Obesity

A systematic review, as well as cohort and case-control studies, demonstrates that obesity doubles the risk compared to that which is seen for healthy weight individuals [12, 13]. One study demonstrated that VTE risk increases with increasing BMI and the associated excess risk is much greater after surgery than without surgery. During a 12-week period without surgery, the incidence rate of VTE per 1000 women with a BMI <25 was 0.10 and ≥25 was 0.19; the corre-

sponding rates in the 12 weeks following day and inpatient surgery were, respectively, about 4 and 40 times higher [14].

The inflammatory state associated with excess body fat and its associated comorbidities creates conditions that increase the risk for VTE. Excess adipose tissue causes hypoxia and increases delivery of inflammatory adipocytokines and free fatty acids (FFAs) to the liver, where coagulation factors are synthesized. FFAs can induce mitochondrial production of reactive oxygen species (ROS), which are cytotoxic and serve as signals to activate endothelial cells and initiate systemic coagulation [15]. Initially, inflammation is confined to the adipocytes, but excess activity overflows into the systemic circulation, where fatty infiltration of the liver, muscles, and vascular endothelium develops. Thus, an inflammatory process now begins in the peripheral tissues, which are not as well equipped to handle the subsequent cytotoxic effects [16]. Loss of body weight has been shown to reduce the concentrations of coagulation factors toward the normal range and improve fibrinolysis [17].

Insulin resistance associated with increasing BMI has been reported to increase the risk of VTE due to the overactivation of the renin-angiotensin system and elevated level of circulating FFAs, which interfere with insulin-mediated glucose uptake. The subsequent hyperglycemia can lead to ROS generation and oxidative stress, which can trigger systemic inflammation and further FFA production [18, 19].

The synergistic effects of obesity with other risk factors increase the VTE risk even further. One study analyzed the risk of obesity associated with oral contraceptives with or without factor V Leiden and found that the incidence of thrombosis was increased 4-fold in individuals taking hormone contraceptives, 7-fold in those with factor V Leiden, and 36-fold in individuals with both risk factors [20].

4.3.2.2 Race and Gender

For reasons that are not completely understood, African-Americans and Caucasians tend to have a greater VTE risk than those whose ethnic background is either Asian or Native American. O blood type is proportionally higher in African-Americans; thus one would expect that African-American individuals would have fewer VTEs. African-Americans have a 30 % higher risk than Caucasians, while Asian and Native Americans have a 70 % lower risk.

Studies demonstrate the risk of recurrent VTE to be higher among men than women [21]. Women have a higher incidence of DVT during their childbearing years, although this risk is still relatively low compared to risk levels for older men and women. However, after the age of 50, men are at greater risk than woman.

4.3.2.3 Age

A number of studies support an association between increasing age and a higher incidence of VTE. The incidence among children (under the age of 14) is quite low, at less than 1 per 100,000 population. Incidence rates increase relatively slowly until the age of 50, and then accelerates dramatically, surpassing 1000 per 100,000 population by the age of 80. The average annual rates of hospitalizations with a discharge diagnosis of DVT, PE, or VTE among adults were 152, 121, and 239 per 100,000 population, respectively. For VTE, the average annual rates were 60 per 100,000 population aged 18–39 years, 143 for persons aged 40–49 years, 200 for persons aged 50–59 years, 391 for persons aged 60–69 years, 727 for persons aged 70–79 years, and 1134 for persons aged ≥80 years [22].

4.3.2.4 Infection and Inflammatory Diseases

Respiratory tract, urinary tract, skin, intra-abdominal infections, and bacteremia diagnosed in hospital or treated in the community were associated with at least a twofold increase in VTE risk. The association was strongest within the first 2 weeks after onset of infection, gradually declining thereafter [23].

Patients with rheumatologic disease have an increased risk for VTE. A meta-analysis evaluating VTE risks in patients with inflammatory arthritis, vasculitis, and connective tissue diseases (including systemic lupus erythematosus (SLE), Sjögren's syndrome, inflammatory myositis, and

systemic sclerosis) demonstrated a threefold higher risk compared to the general population [24].

VTE risk appears to be increased in patients with inflammatory bowel disease (IBD), especially in those patients with trigger events; most often, the trigger event is a hospitalization. A population-based study identified a threefold increased risk for VTE [25]. Patients with IBD are also at an increased risk of recurrent VTE compared to patients without IBD [26]. At the time of a flare, however, this increase in risk was demonstrated to be much more prominent, with the risk being lower during non-hospitalized periods (6.4 per 1000 person-years) than during hospitalized periods (37.5 per 1000 person-years) [27].

4.3.2.5 Nonsteroidal Anti-Inflammatory Drugs

A systematic review and meta-analysis demonstrated a statistically significant increased risk of VTE among nonsteroidal anti-inflammatory drug (NSAID) users. Use of nonselective NSAIDs or cyclooxygenase-2-selective inhibitors (COX2Is) has been associated with an increased risk for VTE. In a population-based case-control study in northern Denmark, use of nonselective NSAIDs or COX2Is was associated with twofold or more increased risk of VTE. Current use was classified as new use (first-ever prescription redemption within 60 days before VTE diagnosis date) or long-term use. Compared to patients who did not use NSAIDs, there was an increased adjusted incidence rate ratio (IRR) associated with current nonselective NSAID and COX2I use with VTE. Recent users had substantially smaller increases than current users [28].

As indicated earlier, rheumatologic patients have a higher VTE risk, which is compounded even further in patients taking NSAIDs chronically for pain management.

4.3.2.6 Smoking

A meta-analysis involving approximately 4 million subjects, and more than 35,000 patients with VTE from 32 observational studies, found a slightly increased risk of VTE for smokers compared to nonsmokers. The risk was higher in studies adjusted for conventional cardiovascular risk factors, especially for BMI. The risk of developing VTE was greater for current smokers than for former smokers, and a dose-response relationship was found for daily smoking and pack-years smoked [29, 30]. A synergistic effect on VTE risk for smoking and oral contraceptive use was demonstrated in one study, reporting an odds ratio of developing VTE for oral contraceptive users of 3.90, which increased to 8.79 when current smoking was added [31].

4.3.2.7 Antiphospholipid Syndrome

The antiphospholipid syndrome is a relatively common acquired cause of venous thrombosis. Antiphospholipid antibodies recognize phospholipid-protein complexes such as β2-glycoprotein I (β2GPI), and prothrombin. These autoantibodies interfere with the physiological mechanisms of the coagulation cascade and fibrinolysis, thus leading to hypercoagulation. It also interferes with the function of platelets, monocytes, and endothelial cells. The protein C pathway is the most important natural anticoagulant pathway, activated in the presence of low concentrations of thrombin. The activated protein C (APC) exerts its anticoagulant effect through proteolytic inactivation of coagulation factors V and VIII. Diagnostic tests performed to confirm antiphospholipid syndrome include lupus anticoagulant, anticardiolipin, and anti-β2-glycoprotein I antibodies (anti-β2GPI) [32].

4.3.2.8 Oral Contraceptives/Hormonal Replacement Therapy

Women using oral contraceptives in their childbearing years and postmenopausal women using hormone therapy are at increased risk for VTE. It is well established that oral contraceptives (OCs) carry a risk of VTE, especially during the first year of use. Studies reveal that some combined OCs containing new-generation and anti-androgenic progestogen (desogestrel, gestodene, drospirenone, or cyproterone) have a higher risk of VTE than older drugs, such as levonorgestrel. Combined oral contraceptives that contain progestogen induce a more pronounced APC resistance than those containing levonorgestrel [33].

Oral contraceptives that contain both estrogen and progestin increase the risk of a blood clot by two- to eightfold. The risk may even be greater with patches that contain transdermal contraceptives, since the amount of estrogen absorbed can be up to 60 % higher.

Postmenopausal women undergoing hormonal therapy also have a higher risk of VTE, with recent large studies suggesting a two- to fourfold increase in risk, with even larger increases in risk for those on high doses of estrogen (greater than 1.25 mg/day) [34].

Women with thrombophilia who also are exposed to oral contraceptives, pregnancy, or hormonal therapy will face a considerably greater risk for VTE.

4.3.2.9 Pregnancy

Although pregnant women are not considered candidates for bariatric surgery, there are rare cases in which a negative pregnancy test was resulted on the day of surgery and then becomes positive within the ensuing postoperative period. Pregnancy increases the risk of DVT fivefold compared to nonpregnancy, with the risk being even greater in the postpartum period [2].

4.3.2.10 Malignancy

About 10 % of patients who present with VTE will have an occult cancer diagnosed within 2 years of the thrombotic episode. The diagnosis of malignancy was established within the first year of presentation of DVT in greater than 75 % of cases and more than 40 % of these cancers were found to be metastatic. Cohort studies and clinical trials suggest that the cancer risk of persons presenting with idiopathic unprovoked VTE is more than three times higher than patients with a provoked VTE, and these patients are typically diagnosed with cancer over the next 5 to 10 years [35].

Cancer patients receiving chemotherapy are at even higher risk. Cancer patients with VTE face much worse outcomes than those with cancer alone. The probability of death within 183 days of initial hospital admission is over 94 % for those with VTE and malignant disease, compared to less than 40 % for those with cancer alone.

The incidence of DVT/PE is substantially higher for cancer patients than for non-cancer patients across all types of major surgery. It is uncertain whether the incidence of VTE decreases to pre-cancer risks or if the risk remains increased in cancer survivors [36].

4.4 VTE Risk and Bariatric Surgery

Patients undergoing elective general surgery, including bariatric surgery, are at relatively low risk of VTE. Specifically, the incidence of VTE is approximately 0.03 % with inguinal hernia repair and laparoscopic cholecystectomy, 0.1 to 0.6 % with operations involving the abdominal wall or appendix, and up 1.7 % with major resections such as esophagectomy, hepatectomy, and splenectomy. Most large series report VTE rates after bariatric surgery of about 0.4 % [6].

Most studies report the highest risk for VTE within the first 3–4 weeks and up to 3 months postoperatively. However, there are some studies suggesting that VTE risk remains elevated for at least 6 months in the postoperative period [37–40].

Using data from nearly 74,000 patients, the Bariatric Outcomes Longitudinal Database (BOLD) demonstrated a VTE incidence of 0.42 % at 90 days, with a risk of approximately 1.5 % after open surgery compared to 0.34 % laparoscopically [41]. The Longitudinal Assessment of Bariatric Surgery (LABS) study reported a 30-day VTE rate of 0.4 % [43].

Analyzing the VTE events in 93 of 27,818 patients (0.33 %), the Michigan Surgery Collaborative (MBSC) reported a DVT rate of 0.21 %, a PE rate of 0.18 %, and both a DVT and PE in 0.06 % [44]. In this dataset there were eight VTE-associated deaths, giving a case fatality rate of 8.6 % and accounting for one-third of all deaths in the registry.

In a large, multi-institutional retrospective chart review of 4293 patients undergoing primary or revisional bariatric surgery over an 8-year period, 57 patients (1.3 %) had a VTE [46]. Pulmonary embolism occurred in 39 (0.9 %), and DVT

occurred 18 (0.4 %). Of note, no patients were denied surgery due to risk of VTE. Interestingly, of the patients with PE, 38.5 % had negative duplex studies of the lower extremities. There was only one VTE-related mortality in this study (0.02 %).

Gastric bypass was more likely to result in VTE events than LAGB (OR = 0.31). The incidence of VTE at 6 months for the different operations were as follows: LAGB 0.8 % (n = 616), laparoscopic RYGB 2.7 % (n = 5695), and open RYGB 3.3 % (n = 11,123).

4.4.1 Predictors of VTE Risk After Bariatric Surgery

Univariate and multivariate analysis of the aforementioned study revealed that age, BMI, open, and revisional surgery were predictive of VTE. Comparing different bariatric operations, VTE rates were as follows: 1.1 % of 2945 RYGB patients, 2.9 % of 709 VSG patients, 0.2 % of 467 LAGB patients, and 6.4 % of 171 revisional surgery patients [46].

In the MBSC study, significant risk factors for VTE complication included: previous history of VTE (OR 4.15, CI 2.42–7.08), male gender (OR 2.08, CI 1.36–3.19), operative time more than 3 hours (OR 1.86, CI 1.07–3.24), BMI category (per 10 units) (OR 1.37, CI 1.06–1.75), age category (per 10 years) (OR 1.25, CI 1.03–1.51), and most significant risk factor was procedure type, with duodenal switch carrying the highest risk for VTE [44].

In the BOLD study, the risk of VTE was greater in older patients (HR = 1.04), patients with a higher BMI (HR = 1.05), blacks versus whites (HR = 1.65), pulmonary hypertension (HR = 1.8), lower extremity edema (HR = 2.23), men (HR = 2.32), patients with a history of VTE (HR = 4.96), and prior inferior vena cava filter (HR = 7.66) [41]. The risk of VTE was greater in the patients undergoing gastric bypass than in those undergoing adjustable gastric banding (0.55 % versus 0.16 %). Also, VTE was more frequent when the procedure was performed using an open than a laparoscopic approach (1.54 % versus 0.34 %).

Multiple regression analysis from over 304,000 bariatric surgery patients in the National Inpatient Sample database demonstrated an overall VTE rate of 0.17 %, with a lower VTE rate seen in laparoscopic procedures compared to open procedures (0.13 to 0.45 %) [42]. Alcohol abuse (OR 8.7), open operation (OR 2.5), renal failure (OR 2.3), congestive heart failure (OR 2.0), male gender (OR 1.5), and chronic lung disease (OR 1.4) were associated with a higher rate of VTE.

4.4.1.1 Use of IVC Filters

There is no consensus on the use of IVC filters and what comprises a high enough risk to consider its use. There are studies suggesting that the use of IVC filters in patients undergoing bariatric surgery has been associated with a significantly higher risk for VTE and mortality [44]. However, other studies report safe use of IVC filters with low complication rates [48].

4.4.1.2 Duration of Procedure

There have been several studies looking at procedure time and the risk of VTE. Some of the risk may be associated with technical difficulty of the procedure (i.e., intestinal adhesions, presence of abdominal wall or hiatal hernia), but surgeon skill may play a significant role. One study demonstrated BMI as an independent predictor of operative time, and subsequently an increased incidence of complications and VTE [49]. Another study adjusted for surgeon characteristics and resident involvement, and found that slower surgeons had statistically significant higher rates of complications and VTE [50].

4.4.1.3 Procedure Type

The Michigan collaborative study mentioned in the previous section reported the following VTE rates among different bariatric operations: laparoscopic RYGB 0.65 %, open RYGB 1.04 %, LAGB 0.53 %, VSG 0.74 %, and DS 1.77 %.

In the BOLD study, the 90-day VTE event rates among the various procedures were as follows: LAGB 0.14 % (n = 29,384), RYGB 0.46 % (n = 39,350), sleeve gastrectomy 0.50 % (n = 1806), and BPD with DS 2.16 % (n = 647) [41].

A single-institution database review of 362 biliopancreatic diversion with duodenal switch (BPD-DS) patients found a VTE rate of 3.3 % (n = 12) [47]. Of these 12 patients, 8 presented with DVT, giving a DVT rate of 2.2 %. Four patients presented with PE, giving a PE rate of 1.1 %. All patients in this study received VTE chemoprophylaxis, which was continued for 14 days after discharge in . There were no VTE-related mortalities in this study. Operative time and length of hospital stay were identified as risk factors associated with postoperative VTE complications.

4.4.1.4 Prior History of VTE

Studies clearly demonstrate an increased risk of VTE in patients with a previous history of VTE [41, 44].

4.4.1.5 Impact of Surgical Complications

Complications after bariatric surgery are associated with prolonged hospitalizations and immobilization, and have been shown to play a significant role with increased risk for VTE. A multicenter retrospective analysis looking at patients who underwent bariatric surgery demonstrated a VTE incidence of 0.58 % within 6 months, with a strong association between VTE and surgical complications, and intensive care unit admissions. The majority of complications were anastomotic leaks, abscesses, and infections [38].

4.5 VTE Prevention: Diagnosis and Treatment

Despite significant advances in the prevention and treatment of VTE, pulmonary embolism remains the most common preventable cause of hospital death, responsible for many deaths each year in the USA. Thus, it is vital that efforts continue to be made to find the safest and most effective means of preventing and managing VTE. Practical approaches to the prevention of VTE in surgical bariatric patients are reviewed here.

According to the Agency for Healthcare Research and Quality, the prevention of VTE is the number one strategy to improve patient safety in hospitals [51]. As an example, as part of the Surgical Care Improvement Project, the Center for Medicare and Medicaid Services (CMS) now considers appropriate VTE prophylaxis to be a pay-for-performance quality measure for specific procedures (see Table 4.3) [52–54]. Effective and safe prophylactic measures are now available for most high-risk patients [55–58] and numerous evidence-based guidelines have been published for the prevention of VTE [59–61]. The American College of Chest Physicians clinical practice guidelines recommend VTE prophylaxis by surgical risk groups [60].

In the absence of appropriate prophylaxis, the incidence of asymptomatic DVT detected by objective diagnostic screening tests has ranged from 10 to 80 % in various hospitalized medical and surgical groups. From earlier studies, the incidence of fatal pulmonary embolism in the absence of prophylaxis was estimated to be 0.1 to 0.8 % in patients under-

Table 4.3 Hospital Quality Alliance/Centers for Medicare & Medicaid Services (CMS) Surgical Care Improvement quality measures for perioperative VTE prevention

General surgery	Any of the following:
	• Low-dose unfractionated heparin (LDUH)
	• Low-molecular-weight heparin (LMWH)
	• Factor Xa inhibitor (fondaparinux)
	• LDUH or LMWH or factor Xa inhibitor (fondaparinux) combined with IPC or GCS
Excluded populations:	
• Patients less than 18 years of age	
• Patients who have a length of stay >120 days	
• Burn patients	
• Patients with procedures performed entirely by laparoscope	
• Patients enrolled in clinical trials	
• Patients who are on warfarin prior to admission	
• Patients whose ICD-9-CM principal procedure occurred prior to the date of admission	
• Patients whose total surgery time is less than or equal to 60 min	
• Patients who stayed less than or equal to 3 calendar days postoperatively	
• Patients with contraindications to both mechanical and pharmacological prophylaxis	

From Specifications Manual for National Hospital Inpatient Quality Measures. Available at www.qualitynet.org

going elective general surgery, which includes bariatric surgery, 2 to 3 % in patients having elective total hip replacement, and 4 to 7 % of patients undergoing surgery for a fractured hip [59].

These estimates are likely lower today because of the increasing use of early ambulation and shorter lengths of hospitalization. However, the incidence of VTE, and in particular fatal pulmonary embolism, remains excessively high, even after hospital discharge.

Most bariatric surgery patients are considered at high risk for VTE given the prevalence of risk factors that promote VTE, including obesity, obstructive sleep apnea/hypoventilation syndrome, and exposure to general anesthesia.

4.5.1 Prevention of VTE

There are two approaches to the prevention of fatal pulmonary embolism:

- Primary prophylaxis: Either drugs or physical methods that are effective for preventing DVT.
- Secondary prevention: Early detection and treatment of subclinical venous thrombosis by screening postoperative patients with objective tests that are sensitive for the presence of DVT.

However, no single screening method has found universal acceptance for secondary prevention [62, 63]. Accordingly, primary prophylaxis is preferred in most clinical circumstances; it is more cost effective than treatment of complications once they occur [64]. Secondary prevention with screening is reserved for patients in whom primary prophylaxis is either contraindicated or shown to be ineffective.

4.5.1.1 Primary Prophylaxis

Early and frequent ambulation is preferred in surgical patients at very low risk of VTE as a solo measure in the general population.

4.5.1.2 Intermittent Pneumatic Compression

Intermittent pneumatic compression (IPC) prevents venous thrombosis by enhancing blood flow in the deep veins of the legs, thereby preventing venous stasis [65]. IPC also reduces plasminogen activator inhibitor-1 (PAI-1), thereby increasing endogenous fibrinolytic activity [66].

4.5.1.3 Graduated Compression Stockings

Graduated compression stockings (GCS) alone can help prevent DVT, but when combined with other prophylactic methods appear to improve rates of DVT prevention.

4.5.1.4 Inferior Vena Cava Filter

The only widely accepted and validated indications for vena cava filter placement in patients with thromboembolism are an absolute contraindication to therapeutic anticoagulation, and failure of anticoagulation when there is acute proximal venous thrombosis.

4.5.2 Selecting a Pharmacologic Agent

Combined pharmacologic and mechanical methods (usually intermittent pneumatic compression), rather than either method alone, should be considered in surgical patients assessed to be at very high risk of VTE. Some considerations on the choice of chemoprophylaxis agent are as follows:

- LMWH and fondaparinux are preferred over unfractionated heparin (UFH) or other pharmacologic agents, particularly in high-risk surgical patients, due to their proven efficacy in this population.
- Low-dose UFH is a reasonable alternative to LMWH for surgical patients in whom there is a contraindication to LMW heparin (e.g., renal insufficiency) or for patients in whom cost is an issue.

- Warfarin may be considered as an alternative to LMWH and UFH when delayed prophylaxis is desired.
- Direct thrombin and factor Xa inhibitors may be alternatives to LMWH. These agents have not been compared with UFH or aspirin.
- Aspirin can be considered for orthopedic patients who have undergone a total hip or knee replacement and are not candidates for other anticoagulants. It is not considered the best option for prophylaxis in bariatric surgery.

There is considerable variability among bariatric surgeons in the approach to thromboprophylaxis because of a lack of consensus regarding the optimal strategy for this population [67–69]. Most bariatric surgeons use early and frequent postoperative ambulation, pneumatic compression devices, and subcutaneous unfractionated or LMWH [70]. The current American Society of Bariatric and Metabolic Surgeons (ASMBS) guidelines regarding VTE prophylaxis state that all bariatric patients may receive mechanical prophylaxis and should undergo early postoperative ambulation [6].

Illustrating the wide variations in VTE protocol, one paper described in detail the prevention protocols at two major academic bariatric programs [46]. At one institution, VTE prophylaxis included intraoperative subcutaneous injection of 5000 units of unfractionated heparin and application of pneumatic compression devices intra- and postoperatively. Patients also routinely received LMWH 40 units twice daily, unless the BMI was above 50 kg/m^2, in which case the dose was increased to 60 units twice daily and extended 2 weeks postoperatively. Chemoprophylaxis was also extended postoperatively in patients with lymphedema or pulmonary hypertension and those who were wheelchair bound. At the other institution patients were given unfractionated heparin 5000 units preoperatively and twice daily for the first 24 hours, and then LMWH 40 units twice daily until discharge. Patients at high risk for VTE due to lymphedema, pulmonary hypertension, or non-ambulatory functional status received an IVC filter prior to the operation. Between the two centers, there was no difference statistically in VTE rates. The mean time to VTE diagnosis was 24 days. For 8 patients VTE was diagnosed during the hospital stay, and 17 of the other 49 patients experienced a VTE event despite being on extended chemoprophylaxis for 2–4 weeks after discharge.

Patients who are considered at a higher level of risk for VTE, such as patients with hypercoagulable disorders, history of previous VTE, or body mass index greater than 60 kg/m^2, may be managed with extended administration of venous thromboembolism prophylaxis because of surgeon preference; there is no consensus regarding indications for extended prophylaxis or duration of therapy for patients undergoing bariatric surgery [71–73]. The prophylaxis is started before surgery and continued at least until the patient is fully ambulating or the VTE risk is deemed to be acceptably low.

4.5.3 Diagnosis of VTE

When approaching the patient with suspected DVT of the lower extremity, it is important to appreciate that only a minority of patients actually have the disease and will require anticoagulation. This illustrates the importance of using validated algorithms to evaluate patients with suspected DVT, along with objective testing to establish the diagnosis. Given the potential risks associated with lower extremity DVT that is not treated (e.g., fatal pulmonary emboli) and the potential risk of anticoagulation in a patient who does not have a DVT (e.g., life-threatening bleeding), accurate diagnosis is essential.

The classic presentation of DVT includes swelling, pain, and erythema of the involved extremity. There is not necessarily a correlation between the location of symptoms and the site of thrombosis. Symptoms in the calf alone are often the presenting manifestation of significant proximal vein involvement, while some patients with whole leg symptoms are found to have isolated calf vein DVT.

Phlegmasia cerulea dolens is an uncommon form of massive proximal (e.g., iliofemoral) venous thrombosis of the lower extremities associated with a high degree of morbidity and mortality. Signs and symptoms include sudden severe leg pain with swelling, cyanosis, edema, venous gangrene, compartment syndrome, and arterial compromise, often followed by circulatory collapse and shock. Delay in treatment may result in death or loss of the patient's limb.

The initial laboratory evaluation in patients with venous thrombosis should include a complete blood count and platelet count, coagulation studies (e.g., prothrombin time, activated partial thromboplastin time), renal and liver function tests, and urinalysis. Any abnormality observed on initial testing should be investigated aggressively.

4.6 D-Dimer

The utility of measuring D-dimer, a degradation product of cross-linked fibrin, has been extensively studied for the diagnosis of both DVT and pulmonary embolus. D-dimers are detectable at levels greater than 500 ng/mL of fibrinogen equivalent units in nearly all patients with VTE. The finding of elevated D-dimer concentrations alone is insufficient to establish the diagnosis of venous thromboembolism, because elevated D-dimer levels are not specific for VTE and are commonly present in hospitalized patients and surgical patients. In general, it is a sensitive test but lacks specificity and is therefore only useful when negative (i.e., cutoff value <500 ng/mL).

4.6.1 Diagnostic Tests

4.6.1.1 Contrast Venography

Venography is **not** recommended as an initial screening due to patient discomfort and difficulty in obtaining an adequate study.

4.6.1.2 Impedance Plethysmography

Impedance plethysmography requires a patient to lie still while a thigh cuff is inflated. The change in blood volume at the calf is measured from the impedance of the calf as determined by electrodes wrapped around it [74]. After rapid deflation of the cuff, the proportional change of impedance over the subsequent 3 seconds is used to measure venous outflow obstruction in a manner similar to spirometry. At present, however, many facilities have neither the equipment nor skilled personnel to perform impedance plethysmography, while the availability of ultrasonography is more widespread.

4.6.1.3 Compression Ultrasonography

A more direct approach to the diagnosis of DVT involves use of compression ultrasonography [75]. The chronicity of the thrombus may be inferred from the echogenicity of the clot because older clots appear more echodense [76, 77].

4.6.1.4 Magnetic Resonance Venography

The diagnostic accuracy of magnetic resonance venography (MRV) is comparable to that of contrast venography, although outcome data are lacking. In addition, the present high cost of MRV makes it unlikely that it will gain prominence as a noninvasive test for DVT. However, MRV is a useful approach when contrast venography is required but precluded because of allergy to contrast material.

4.6.1.5 Computed Tomography

Experience is increasing with the use of computed tomography (CT) for establishing the presence of DVT. Most active investigation protocols image the pulmonary arteries and the subdiaphragmatic deep veins (including the legs) during the same sitting, ideally with no additional contrast medium or venipuncture beyond what is required for a CT pulmonary angiogram [78]. In some reports, CT venography has performed in a manner comparable to ultrasonography in the detection of femoropopliteal venous thrombosis [79]. At present, the use of CT in this setting remains experimental, although the technique holds potential for simplifying the diagnosis of VTE in the future.

4.6.2 Treatment of VTE

The primary objectives of treatment of VTE are to prevent and/or treat the following complications:

- Prevent further clot extension.
- Prevent acute pulmonary embolism.
- Reduce the risk of recurrent thrombosis.
- Treat massive iliofemoral thrombosis with acute lower limb ischemia and/or venous gangrene (i.e., phlegmasia cerulea dolens).
- Limit the development of late complications, such as the post-thrombotic syndrome, chronic venous insufficiency, and chronic thromboembolic pulmonary hypertension.

Anticoagulant therapy is indicated for patients with DVT, since pulmonary embolism may occur in untreated individuals, most often within days or weeks of the event.

4.6.2.1 Initial Therapy

The following recommendations for the treatment of acute venous thromboembolic disease are in accord with the 2012 ACCP evidence-based clinical practice guidelines for antithrombotic and thrombolytic therapy [80, 81].

- Patients with DVT or pulmonary embolism should be treated acutely with LMWH, fondaparinux, unfractionated intravenous heparin, or adjusted-dose subcutaneous heparin.
- Minimal elements for early discharge and/or outpatient therapy with LMW heparin or fondaparinux are listed in Table 4.4.
- When UFH is used, the dose should be sufficient to prolong the activated partial thromboplastin time (aPTT) to 1.5 to 2.5 times the mean of the control value, or the upper limit of the normal aPTT range.
- Treatment with LMWH, fondaparinux, or UFH should be continued for at least 5 days overlap with oral anticoagulation with a vitamin K antagonist.
- For most patients, warfarin should be initiated simultaneously with the heparin, at an initial oral dose of approximately 5 mg/day. In elderly patients and in those at high risk of bleeding or who are undernourished, debilitated, or have heart failure or liver disease, the starting dose should be reduced. The heparin product can be discontinued on day 5 or 6 if the INR has been therapeutic for two consecutive days.
- For patients receiving UFH, ACCP Guidelines suggest that platelet counts be obtained regularly to monitor for the development of thrombocytopenia. The heparin product should be stopped if any one of the following occurs: a precipitous or sustained fall in the platelet count, or a platelet count <100,000/μL.
- The use of thrombolytic agents, surgical thrombectomy, or percutaneous mechanical thrombectomy in the treatment of venous thromboembolism must be individualized. Patients with hemodynamically unstable PE or massive iliofemoral thrombosis (i.e., phlegmasia cerulea dolens), and who are also at low risk to bleed, are the most appropriate candidates for such treatment.
- Inferior vena caval filter placement is recommended when there is a contraindication to, or a failure of, anticoagulant therapy in an individual with, or at high risk for, proximal vein thrombosis or PE. It is also recommended in patients with recurrent thromboembolism despite adequate anticoagulation, for chronic recurrent embolism with pulmonary hyperten-

Table 4.4 Minimal requirements for early hospital discharge or outpatient therapy of venous thromboembolic disease

The responsible physician must ensure that all of the following conditions apply:
The patient is ambulatory and in stable condition, with normal vital signs
There is a low a priori risk of bleeding in the patient
Severe renal insufficiency is not present
There is a practical system in place for the following:
Administration of LMWH and/or warfarin with appropriate monitoring**, and**
Surveillance and treatment of recurrent VTE and bleeding complications

Adapted from Hyers, TM, Agnelli, G, Hull, RD, et al. Antithrombotic therapy for venous thromboembolic disease. Chest 2001; 119:176S. (Sixth ACCP Consensus Conference on Antithrombotic Therapy)

sion, and with the concurrent performance of surgical pulmonary embolectomy or pulmonary thromboendarterectomy.
- Oral anticoagulation with a vitamin K antagonist should prolong the INR to a target of 2.5 (range: 2.0 to 3.0). If the use of a vitamin K antagonist is contraindicated or inconvenient, long-term therapy can be undertaken with either adjusted-dose unfractionated heparin, LMWH, fondaparinux, or rivaroxaban.

4.6.2.2 Duration of Treatment

The duration of anticoagulation therapy varies with the clinical setting, as well as with patient values and preferences.

- Patients with a first thromboembolic event in the context of a reversible or time-limited risk factor (e.g., trauma, surgery) should be treated for 3 months.
- Patients with a first idiopathic thromboembolic event should be treated for a minimum of 3 months. Following this, all patients should be evaluated for the risk/benefit ratio of long-term therapy.
- In patients with a first isolated unprovoked or provoked episode of distal DVT, 3 months of anticoagulant therapy, rather than indefinite therapy, appears to be sufficient.
- Most patients with advanced malignancy should be treated indefinitely or until the cancer resolves.

4.7 Special Circumstances

4.7.1 Portal and Superior Mesenteric Vein Thrombosis

In recent years, sleeve gastrectomy has increased in popularity with bariatric surgery patients and surgeons, and is now the most common bariatric surgery procedure performed in the USA. In one series of 1713 laparoscopic sleeve gastrectomies, 17 patients (1 %) developed portal vein thrombosis after an uncomplicated operation [82]. Of the 17 patients, 16 were women, 8 had a history of smoking, 7 used oral contraceptives, and 2 had a family history of deep vein thrombosis of the lower limbs. Ultimately, seven patients tested positive for thrombophilia. Symptoms presented at a median of 15 days after surgery (range 8–43) with abdominal pain in most cases. One case required emergency laparotomy and splenectomy because of an active bleeding hematoma with massive portomesenteric vein thrombosis. Eleven patients presented with thrombosis of the superior mesenteric vein and ten patients presented with concomitant thrombosis of the splenic vein.

A 2014 study revealed that at least 67 % of bariatric surgery patients in the State of Michigan undergo a laparoscopic sleeve gastrectomy [83]. With the increasing trend towards performing sleeve gastrectomy, there have been unpublished reports from the MBSC suggesting that the incidence of portal vein thrombosis (PVT) after laparoscopic sleeve gastrectomy may be surpassing the incidence of DVT. Further investigation is required before any conclusions can be drawn from these reports. There are published reports on the incidence of PVT (see Table 4.5), but none clearly demonstrating an increasing trend at this time.

In a retrospective, multicenter study of 5706 patients who had laparoscopic bariatric surgery, 17 (0.3 %) developed portomesenteric vein thrombosis, 16 after VSG, and 1 following LAGB [84]. Seven patients were women, the mean age was 38 years, and the mean body mass index was 44.3 kg/m^2. All patients received mechanical and pharmacological VTE prophylaxis. None of the patients had a known coagulopathy prior to surgery. Of note, two of the seven women took oral contraceptives, and they did not stop taking them prior to surgery. All 17 patients underwent a formal hematological work-up for evaluation of hypercoagulability after hospital discharge following the thrombotic event. Three patients (17.6 %) were abnormal, with two having factor V Leiden deficiency and one having protein S, protein C, and methylenetetrahydrofolate reductase deficiencies.

The median time to presentation was 10.1 days, and new-onset epigastric pain was

Table 4.5 Portal venous thrombosis after bariatric surgery: Summary of case reports

Author	Procedure	Total	PVT	SMVT	SVT	Hematologic evaluation
Bellanger, 2010 [85]	VSG	3	2	2	2	Negative for all 3
Berthet, 2009 [86]	VSG	1	1	1	1	Factor V Leiden deficiency
Calmes, 2002 [87]	LAGB	1	1	0	1	Negative
Denne, 2005 [88]	LRYGB	1	1	0	0	Not reported
Gandhi, 2010 [89]	LRYGB	1	0	1	0	Not reported
Hughes, 2014 [90]	VSG	1	1	1	0	Not reported
James, 2009 [91]	LRYGB	7	4	6	1	Not reported for all
Johnson, 2005 [92]	LRYGB	1	0	1	0	Protein S deficiency
Pigeyre, 2008 [93]	LRYGB	1	1	1	1	Protein S deficiency
Pineda, 2013 [94]	VSG	1	0	1	0	Negative
Singh, 2010 [95]	VSG	1	0	1	0	Negative
Rosenberg, 2012 [96]	VSG	1	1	0	0	Not reported
Sonpal, 2004 [97]	RYGB	1	0	1	0	Negative
Swartz, 2004 [98]	LRYGB	3	0	3	0	Not reported for 2, negative in 1

VSG (laparoscopic) vertical sleeve gastrectomy, *LAGB* laparoscopic adjustable gastric banding, *LRYGB* laparoscopic Roux-en-Y gastric bypass, *RYGB* (open) Roux-en-Y gastric bypass, *PVT* portal vein thrombosis, *SMVT* superior mesenteric vein thrombosis, *SVT* splenic vein thrombosis

present in all patients. All patients were treated by anticoagulation, and three required surgery: laparoscopic splenectomy due to infarct and abscess for one patient and laparotomy for two patients (with necrotic small-bowl resection for one of these patients). There were no deaths in this series.

4.8 Conclusion

In summary, bariatric surgery patients are at risk of suffering from potentially fatal VTE, and there are effective prevention strategies. VTE risk factors that have been shown to have predictive significance may include procedure type, open versus laparoscopic approach, increasing BMI, increasing age, male gender, prior history of VTE, smoking, use of hormonal therapy, prolonged operative time, immobility, chronic lung disease, obesity hypoventilation and pulmonary hypertension, alcohol abuse, renal failure, and congestive heart failure [6, 41, 42, 44–46].

There is not one perfect or accurate risk prediction model available, and it is extremely important to consider multiple VTE risk factors when preparing patients for bariatric surgery.

References

1. Beckman MG, Hooper WC, Critchley SE, Ortel TL. Venous thromboembolism: a public health concern. Am J Prev Med. 2010;38(4 Suppl):S495–501.
2. Office of the Surgeon General (US), National Heart, Lung, and Blood Institute (US). The Surgeon General's Call to Action to Prevent Deep Vein Thrombosis and Pulmonary Embolism. Rockville (MD): Office of the Surgeon General (US). 2008.
3. Seddighzadeh A, Zurawska U, Shetty R, Goldhaber SZ. Venous thromboembolism in patients undergoing surgery: low rates of prophylaxis and high rates of filter insertion. Thromb Haemost. 2007;98(6):1220–5.
4. Pryor 2nd HI, Singleton A, Lin E, Lin P, Vaziri K. Practice patterns in high-risk bariatric venous thromboembolism prophylaxis. Surg Endosc. 2013;27(3):843–8.
5. Lidor AO, Moran-Atkin E, Stem M, Magnuson TH, Steele KE, Feinberg R, Schweitzer MA. Hospital-acquired conditions after bariatric surgery: we can

predict, but can we prevent? Surg Endosc. 2014. [Epub ahead of print].
6. The American Society for Metabolic and Bariatric SurgeryClinical Issues Committee. ASMBS updated position statement on prophylactic measures to reduce the risk of venous thromboembolism in bariatric surgery patients. Surg Obes Relat Dis. 2013;9:493–7.
7. Spyropoulos AC, Lin J. Direct medical costs of venous thromboembolism and subsequent hospital readmission rates: an administrative claims analysis from 30 managed care organizations. J Manag Care Pharm. 2007;13:475–86.
8. Mukherjee D, Lidor AO, Chu KM, Gearhart SL, Haut ER, Chang DC. Postoperative venous thromboembolism rates vary significantly after different types of major abdominal operations. J Gastrointest Surg. 2008;12(11):2015–22.
9. Overby DW, Kohn GP, Cahan MA, Galanko JA, Colton K, Moll S, Farrell TM. Prevalence of thrombophilias in patients presenting for bariatric surgery. Obes Surg. 2009;19(9):1278–85.
10. Taura P, Rivas E, Martinez-Palli G, Blasi A, Holguera JC, Balust J, Delgado S. Lacy AM Clinical markers of the hypercoagulable state by rotational thrombelastometry in obese patients submitted to bariatric surgery. Surg Endosc. 2014;28(2):543–51.
11. Franchini M, Makris M. Non-O blood group: an important genetic risk factor for venous thromboembolism. Blood Transfus. 2013;11(2):164–5.
12. Ageno W, Matteo ND, et al. Association between the metabolic syndrome, its individual components, and unprovoked venous thromboembolism: results of a patient-level meta-analysis. Arterioscler Thromb Vasc Biol. 2014;34:2478–85.
13. Allman-Farinelli MA. Obesity and venous thrombosis: a review. Semin Thromb Hemost. 2011;37(8):903–7.
14. Parkin L, Sweetland S, Balkwill A, Green J, Reeves G, Beral V. Body mass index, surgery, and risk of venous thromboembolism in middle-aged women: a cohort study. Circulation. 2012;125:1897–904.
15. Görlach A. Redox regulation of the coagulation cascade. Antioxid Redox Signaling. 2005;7(9-10):1398–404.
16. O'Rourke RW. Inflammation, obesity, and the promise of immunotherapy for metabolic disease. Surg Obes Relat Dis. 2013;9:609–16.
17. Lindahl B, Nilsson TK, Jansson JH, Asplund K, Hallmans G. Improved fibrinolysis by intense lifestyle intervention. A randomized trial in subjects with impaired glucose tolerance. J Intern Med. 1999;246:105–12.
18. Van Schouwenburg IM, Mahmoodi BK, Veeger NJGM, Bakker SJL, Meijer K, et al. Insulin resistance and risk of venous thromboembolism: results of a population-based cohort study. J Thromb Haemost. 2012;10:1012–8.
19. Kalupahana NS, Moustaid-Moussa N. The renin-angiotensin system: a link between obesity, inflammation and insulin resistance. Obes Rev. 2012;13:136–49.
20. Pomp ER, le Cessie S, Rosendaal FR, Doggen CJ. Risk of venous thrombosis: obesity and its joint effect with oral contraceptive use and prothrombotic mutations. Br J Haematol. 2007;139(2):289–96.
21. Fang C, Cohen HW, Billett HH. Race, ABO blood group, and venous thromboembolism risk: not black and white. Transfusion. 2013;53(1):187–92.
22. Venous Thromboembolism in Adult Hospitalizations—United States. 2007–2009 Centers for Disease Control and Prevention. MMWR Weekly. 2012;61(22):401–4.
23. Schmidt M, Horvath-Puho E, Thomsen RW, Smeeth L, Sørensen HT. Acute infections and venous thromboembolism. J Intern Med. 2012; 271(6):608–18.
24. Lee JJ, Pope JE. A meta-analysis of the risk of venous thromboembolism in inflammatory rheumatic diseases. Arthritis Res Ther. 2014;16:435.
25. Grainge MJ, West J, Card TR. Venous thromboembolism during active disease and remission in inflammatory bowel disease: a cohort study. Lancet. 2010;375 (9715):657–63.
26. Novacek G, Weltermann A, Sobala A, et al. Inflammatory bowel disease is a risk factor for recurrent venous thromboembolism. Gastroenterology. 2010;139(3):779–87.
27. Freeman HJ. Venous thromboembolism with inflammatory bowel disease. World J Gastroenterol. 2008;14(7):991–3.
28. Ungprasert P, Srivali N, Wijarnpreecha K, Charoenpong P, Knight EL. Non-steroidal anti-inflammatory drugs and risk of venous thromboembolism: a systematic review and meta-analysis. Rheumatology. 2014. [Epub ahead of print].
29. Cheng Y, Liu Z, et al. Current and former smoking and risk for venous thromboembolism: a systematic review and meta-analysis. PLoS Med. 2013;10(9): e1001515.
30. Zhang G, Xu X, Su W, Xu Q. Smoking and risk of venous thromboembolism: a systematic review. Southeast Asian J Trop Med Public Health. 2014;45(3):736–45.
31. Pomp ER, Rosendaal FR, Doggen CJ. Smoking increases the risk of venous thrombosis and acts synergistically with oral contraceptive use. Am J Hematol. 2008;83:97–102.
32. Sikara MP, Grika EP, Vlachoyiannopoulos PG. Pathogenic Mechanisms of Thrombosis in Antiphospholipid Syndrome (APS) "Thrombophilia" Edited by Andrea Luigi Tranquilli. InTech; Published 09 Nov 2011. p. 226.
33. Vandenbroucke JP, et al. Oral contraceptives and the risk of venous thrombosis. N Engl J Med. 2001;344 (20):1527–35.
34. Increased risk of thromboembolism in newer oral contraceptives. Published in Health News and Evidence 14 Feb 2013.
35. Hettiarachchi RJ, Lok J, Prins MH, et al. Undiagnosed malignancy in patients with deep vein thrombosis: incidence, risk indicators, and diagnosis. Cancer. 1998;83:180–5.

36. Sorensen HT, Mellemkjaer L, Steffensen FH, Olsen JH, Nielsen GL. The risk of a diagnosis of cancer after primary deep venous thrombosis or pulmonary embolism. N Engl J Med. 1998;338:1169–73.
37. Steele KE, Schweitzer MA, Prokopowicz G, Shore AD, Eaton LC, Lidor AO, Makary MA, Clark J, Magnuson TH. The long-term risk of venous thromboembolism following bariatric surgery. Obes Surg. 2011;21(9):1371–6.
38. Celik F, Bounif F, Fliers JM, Kersten BE, van Dielen FM, Cense HA, Brandjes DP, van Wagensveld BA, Janssen IM, van de Laar AW, Gerdes VE. The impact of surgical complications as a main risk factor for venous thromboembolism: a multicenter study. Obes Surg. 2014;24(10):1603–9.
39. Froehling DA, Daniels PR, Mauck KF, Collazo-Clavell ML, Ashrani AA, Sarr MG, Petterson TM, Heit JA. Incidence of venous thromboembolism after bariatric surgery: a population-based cohort study. Obes Surg. 2013;23(11):1874–9.
40. Zurawska U, Parasuraman S, Goldhaber S. Prevention of pulmonary embolism in general surgery patients. Circulation. 2007;115:e302–7.
41. Winegar DA, Sherif B, Pate V, DeMaria EJ. Venous thromboembolism after bariatric surgery performed by Bariatric Surgery Center of Excellence Participants: analysis of the Bariatric Outcomes Longitudinal Database. Surg Obes Relat Dis. 2011;7(2):181–8.
42. Masoomi H, Buchberg B, Reavis KM, Mills SD, Stamos M, Nguyen NT. Factors predictive of venous thromboembolism in bariatric surgery. Am Surg. 2011;77(10):1403–6.
43. Flum DR, Belle SH, King WC, et al. Perioperative safety in the longitudinal assessment of bariatric surgery. N Engl J Med. 2009;361:445–54.
44. Finks JF, English WJ, Carlin AM, Krause KR, Share DA, Banerjee M, Birkmeyer JD, Birkmeyer NJ, Michigan Bariatric Surgery Collaborative, Center for Healthcare Outcomes and Policy. Predicting risk for venous thromboembolism with bariatric surgery: results from the Michigan Bariatric Surgery Collaborative. Ann Surg. 2012;255(6):1100–4.
45. Finks JF, Kole KL, Yenumula PR, English WJ, Krause KR, Carlin AM, Genaw JA, Banerjee M, Birkmeyer JD, Birkmeyer NJ, Michigan Bariatric Surgery Collaborative, from the Center for Healthcare Outcomes and Policy. Predicting risk for serious complications with bariatric surgery: results from the Michigan Bariatric Surgery Collaborative. Ann Surg. 2011;254(4):633–40.
46. Jamal MH, Corcelles R, Shimizu H, Kroh M, Safdie FM, Rosenthal R, Brethauer SA, Schauer PR. Thromboembolic events in bariatric surgery: a large multi-institutional referral center experience. Surg Endosc. 2014. [Epub ahead of print].
47. Rezvani M, et al. Venous thromboembolism after laparoscopic biliopancreatic diversion with duodenal switch: analysis of 362 patients. Surg Obes Relat Dis. 2014;10(3):469–73.
48. Vaziri K, Devin Watson J, Harper AP, Lee J, Brody FJ, Sarin S, et al. Prophylactic inferior vena cava filters in high-risk bariatric surgery. Obes Surg. 2011;21(10):1580–4.
49. Chan MM, Hamza N, Ammori BJ. Duration of surgery independently influences risk of venous thromboembolism after laparoscopic bariatric surgery. Surg Obes Relat Dis. 2013;9(1):88–93.
50. Reames BN, Bacal D, Krell RW, Birkmeyer JD, Birkmeyer NJ, Finks JF. Influence of median surgeon operative duration on adverse outcomes in bariatric surgery. Surg Obes Relat Dis. 2014. [Epub ahead of print].
51. Shojania KG, Duncan BW, McDonald KM, et al. Making health care safer: a critical analysis of patient safety practices. Report/Technology Assessment No. 43. Rockville, MD: Agency for Healthcare Research and Quality. Available at www.ahrq.gov/clinic/ptsafety/. Accessed 03 Jan 2002.
52. Specifications Manual for National Hospital Inpatient Quality Measures. www.qualitynet.org. Accessed 11 Mar 2009.
53. Sutedjo JL, Ng RK, Piazza G, Goldhaber SZ. Medicare's new regulations for deep vein thrombosis as a "never event": wise or worrisome? Am J Med. 2009;122:975.
54. Passman MA. Mandated quality measures and economic implications of venous thromboembolism prevention and management. Am J Surg. 2010;199:S21.
55. Clagett GP, Reisch JS. Prevention of venous thromboembolism in general surgical patients. Results of meta-analysis. Ann Surg. 1988;208:227.
56. Collins R, Scrimgeour A, Yusuf S, Peto R. Reduction in fatal pulmonary embolism and venous thrombosis by perioperative administration of subcutaneous heparin. Overview of results of randomized trials in general, orthopedic, and urologic surgery. N Engl J Med. 1988;318:1162.
57. Leizorovicz A, Haugh MC, Chapuis FR, et al. Low molecular weight heparin in prevention of perioperative thrombosis. BMJ. 1992;305:913.
58. Nurmohamed MT, Rosendaal FR, Büller HR, et al. Low-molecular-weight heparin versus standard heparin in general and orthopaedic surgery: a meta-analysis. Lancet. 1992;340:152.
59. Geerts WH, Pineo GF, Heit JA, et al. Prevention of venous thromboembolism: the seventh ACCP conference on antithrombotic and thrombolytic therapy. Chest. 2004;126:338S.
60. Geerts WH, Bergqvist D, Pineo GF, et al. Prevention of venous thromboembolism: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest. 2008;133:381S.
61. Lyman GH, Khorana AA, Falanga A, et al. American Society of Clinical Oncology guideline: recommendations for venous thromboembolism prophylaxis and treatment in patients with cancer. J Clin Oncol. 2007;25:5490.

62. Meyer CS, Blebea J, Davis Jr K, et al. Surveillance venous scans for deep venous thrombosis in multiple trauma patients. Ann Vasc Surg. 1995;9:109.
63. Schellong SM, Beyer J, Kakkar AK, et al. Ultrasound screening for asymptomatic deep vein thrombosis after major orthopaedic surgery: the VENUS study. J Thromb Haemost. 2007;5:1431.
64. Hull RD, Hirsh J, Sackett DL, Stoddart GL. Cost-effectiveness of primary and secondary prevention of fatal pulmonary embolism in high-risk surgical patients. Can Med Assoc J. 1982;127:990.
65. Roberts VC, Sabri S, Beeley AH, Cotton LT. The effect of intermittently applied external pressure on the haemodynamics of the lower limb in man. Br J Surg. 1972;59:223.
66. Comerota AJ, Chouhan V, Harada RN, et al. The fibrinolytic effects of intermittent pneumatic compression: mechanism of enhanced fibrinolysis. Ann Surg. 1997;226:306.
67. Gould MK, Garcia DA, Wren SM, et al. Prevention of VTE in nonorthopedic surgical patients: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;1412:e227S.
68. Service GJ, Thompson GB, Service FJ, et al. Hyperinsulinemic hypoglycemia with nesidioblastosis after gastric-bypass surgery. N Engl J Med. 2005;353:249.
69. Barba CA, Harrington C, Loewen M. Status of venous thromboembolism prophylaxis among bariatric surgeons: have we changed our practice during the past decade? Surg Obes Relat Dis. 2009;5:352.
70. Wu EC, Barba CA. Current practices in the prophylaxis of venous thromboembolism in bariatric surgery. Obes Surg. 2000;10:7.
71. Hamad GG, Choban PS. Enoxaparin for thromboprophylaxis in morbidly obese patients undergoing bariatric surgery: findings of the prophylaxis against VTE outcomes in bariatric surgery patients receiving enoxaparin (PROBE) study. Obes Surg. 2005;15:1368.
72. Raftopoulos I, Martindale C, Cronin A, Steinberg J. The effect of extended post-discharge chemical thromboprophylaxis on venous thromboembolism rates after bariatric surgery: a prospective comparison trial. Surg Endosc. 2008;22:2384.
73. Sapala JA, Wood MH, Schuhknecht MP, Sapala MA. Fatal pulmonary embolism after bariatric operations for morbid obesity: a 24-year retrospective analysis. Obes Surg. 2003;13:819.
74. Hull R, Taylor DW, Hirsh J, et al. Impedance plethysmography: the relationship between venous filling and sensitivity and specificity for proximal vein thrombosis. Circulation. 1978;58:898.
75. Donnelly R, Hinwood D, London NJ. ABC of arterial and venous disease. Non-invasive methods of arterial and venous assessment. BMJ. 2000;320:698.
76. Peter DJ, Flanagan LD, Cranley JJ. Analysis of blood clot echogenicity. J Clin Ultrasound. 1986;14:111.
77. O'Shaughnessy AM, FitzGerald DE. Organization patterns of venous thrombus over time as demonstrated by duplex ultrasound. J Vasc Invest. 1996;2:75.
78. Garg K, Mao J. Deep venous thrombosis: spectrum of findings and pitfalls in interpretation on CT venography. AJR Am J Roentgenol. 2001;177:319.
79. Garg K, Kemp JL, Wojcik D, et al. Thromboembolic disease: comparison of combined CT pulmonary angiography and venography with bilateral leg sonography in 70 patients. AJR Am J Roentgenol. 2000;175:997.
80. Kearon C, Kahn SR, Agnelli G, et al. Antithrombotic therapy for venous thromboembolic disease: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest. 2008;133:454S.
81. Kearon C, Akl EA, Comerota AJ, et al. Antithrombotic therapy for VTE disease: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;141:e419S.
82. Salinas J, et al. Portomesenteric vein thrombosis after laparoscopic sleeve gastrectomy. Surg Endosc. 2014;28(4):1083–9.
83. Reames BN, Finks JF, Bacal D, Carlin AM, Dimick JB. Changes in bariatric surgery procedure use in Michigan, 2006–2013. JAMA. 2014;312(9):959–61.
84. Goitein D, et al. Portomesenteric thrombosis following laparoscopic bariatric surgery: incidence, patterns of clinical presentation, and etiology in a bariatric patient population. JAMA Surg. 2013;148(4):340–6.
85. Bellanger DE, Hargroder AG, Greenway FL.Mesenteric venous thrombosis after laparoscopic sleeve gastrectomy. Surg Obes Relat Dis. 2010;6(1):109–11.
86. Berthet B, et al. Portal vein thrombosis due to factor 2 leiden in the post-operative course of a laparoscopic sleeve gastrectomy for morbid obesity. Obes Surg. 2009;19(10):1464–7.
87. Calmes JM, et al. Band infection with splenoportal venous thrombosis: an unusual but severe complication of gastric banding. Obes Surg. 2002;12(5):699–702.
88. Denne JL, Kowalski C. Portal vein thrombosis after laparoscopic gastric bypass. Obes Surg. 2005;15(6):886–9.
89. Gandhi K, et al. Mesenteric vein thrombosis after laproscopic gastric sleeve procedure. J Thromb Thrombolysis. 2010;30(2):179–83.
90. Hughes DL, et al. Mesenteric ischaemia secondary to portomesenteric venous thrombosis, 2 weeks post laparoscopic sleeve gastrectomy in a cirrhotic patient. BMJ Case Rep. 2014.
91. James AW, et al. Portomesenteric venous thrombosis after laparoscopic surgery: a systematic literature review. Arch Surg. 2009;144(6):520–6.
92. Johnson CM, et al. Mesenteric venous thrombosis after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2005;1(6):580–2. discussion 582-3.
93. Pigeyre M, et al. Laparoscopic gastric bypass complicated by portal venous thrombosis and severe neurological complications. Obes Surg. 2008;18(9):1203–7.

94. Pineda L, Sarhan M, Ahmed L. Superior mesenteric vein thrombosis after laparoscopic sleeve gastrectomy. Surg Laparosc Endosc Percutan Tech. 2013;23(4):e162–3.
95. Singh P, et al. Acute mesenteric vein thrombosis after laparoscopic gastric sleeve surgery for morbid obesity. Surg Obes Relat Dis. 2010;6(1):107–8.
96. Rosenberg JM, et al. Portal vein thrombosis following laparoscopic sleeve gastrectomy for morbid obesity. JSLS. 2012;16(4):639–43.
97. Sonpal IM, et al. Mesenteric venous thrombosis after gastric bypass. Obes Surg. 2004;14(3):419–21.
98. Swartz DE, Felix EL. Acute mesenteric venous thrombosis following laparoscopic Roux-en-Y gastric bypass. JSLS. 2004;8(2):165–9.
99. Jatoi I. Surgical considerations in the management of primary invasive breast cancer. In: Jatoi I, Kaufmann M, editors. Management of breast diseases. New York: Springer; 2010. p. 227–41.

Hemorrhage after Bariatric Surgery: Evaluation and Management

5

Ivan Alberto Zepeda Mejia and Tomasz Rogula

5.1 Introduction and Definitions

Extraluminal bleeding is bleeding outside of the gastrointestinal tract into the peritoneal cavity. Gastrointestinal bleeding, also referred to as intraluminal bleeding, occurs inside the lumen of the gastrointestinal tract. Their clinical presentations differ depending on the location. Early bleeding is defined as occurring perioperatively within 24 h of surgery while late bleeding occurs after the first 24 h. Management is based on a combined approach that may include conservative treatment, medical interventions, endoscopy, or surgical procedures.

5.2 Prevalence

Bleeding in the early postoperative period after gastric bypass surgery is reported in 0.94–3.9 % of cases [1–4]. The incidence of bleeding in sleeve gastrectomy ranges from 1.2 to 5.6 % [5–7]. Biliopancreatic diversion with duodenal switch has a higher risk of bleeding, up to 5–10 %. Laparoscopic gastric band placement has been reported to have a rate of bleeding as low as 0.005 % [8]. Postoperative bleeding increases hospital stay, mortality rate, and morbidity [1, 3, 9].

Clinical and surgical factors that may increase bleeding risk include: liver cirrhosis and hepatosplenomegaly, type 2 diabetes [10, 11], undiagnosed clotting factor deficiencies and acquired bleeding disorders, previous abdominal surgeries [1], age > 60 years [12, 13], super-obese status (BMI > 50 kg/m^2), LRYGB operation [14], and the use of chronic anticoagulants [15].

LMWH is often used before and after bariatric surgery to lower the incidence of VTE and presents a similar risk of hemorrhage when compared to other types of VTE prophylaxis used in these high risk patients [16]. Dose-adjusted unfractioned heparin (UH) is more efficacious than fixed dose of UH in preventing VTE and does not have a higher bleeding rate [17].

Laparoscopic Roux-en-Y gastric bypass has a higher rate of postoperative bleeding compared to open RYGB [3, 18]. Laparoscopic sleeve gastrectomy has a higher incidence of postoperative bleeding compared to LRYGB [19, 20]. However, LRYGB has less intraoperative bleeding than open RYGB [21]. No difference in bleeding incidence between robotic and laparoscopic gastric bypass has been reported [22].

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_5

5.3 Clinical Presentation

The clinical presentation of bleeding varies depending on the location of the bleeding vessel. In RYGB, the most common sites of bleeding are the gastrojejunostomy staple-line (for stapled anastomoses), the gastric remnant or other visceral vessels injured during the procedure. In RYGB, intraluminal bleeding after surgery presents with signs and symptoms of upper or lower gastrointestinal bleed (hematemesis, melena, or hematochezia). Early bleeding is frequently located at the anastomotic staple-line or from the gastric remnant. Later bleeding may be secondary to ulceration near the gastrojejunal anastomosis.

Up to 0.5–2.3 % of bowel obstructions after gastric bypass may be caused by intraluminal blood clots, especially at the jejunojejunostomy (JJ) [23, 24]. Differential diagnosis with other causes of JJ obstruction causes are: internal hernia, adhesions, bowel kinking and mesocolon window scarring [25]. Such obstruction can potentially increase intraluminal pressure and lead to gastrojejunostomy blow-out which may results in further complications of gastric necrosis, pancreatitis, biliary stasis, sepsis, and multi-organ failure [26]. Bleeding from the gastric remnant may remain occult for a prolonged period of time and present its symptoms late. Obviously, intraluminal bleeding will not manifest in increases drain output, if intraperitoneal drains have been placed. Other more subtle presentations of chronic bleeding include iron deficiency anemia or heme-positive stool [1, 27, 28].

Intraperitoneal bleeding has a higher incidence compared to luminal bleeding [20]. Nearly 73 % of early bleeding will present in the first 24 h. It may present with clinical signs of hemodynamic instability that needs to be treated as a surgical emergency. It can present with symptoms of hypovolemic shock, including tachycardia to 100–120 bpm or more, hypotension, oliguria, and drop in hematocrit and hemoglobin

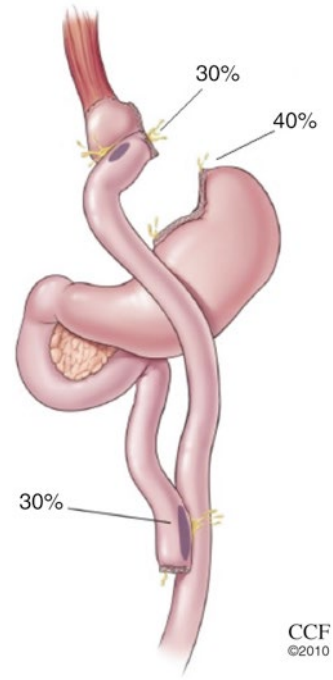

Fig. 5.1 Most common sites of bleeding after gastric bypass. © Cleveland Clinic, with permission

[1, 3, 29]. The potential sites and causes of intraperitoneal bleeding include the various staple lines depicted in Fig. 5.1. Mesenteric vessels and iatrogenic injury to the viscera or other structures in the abdomen such as the spleen, falciform ligament, liver, and trocar lesions to the inferior epigastric vessels in the abdominal wall when accessing the abdomen are also common sites if bleeding [1].

Extraluminal bleeding can less commonly present as late bleeding, in some cases up to several months after surgery. Some symptoms can be confusing, like sub-obstruction symptoms with weight loss, vomiting, abdominal pain, and nausea.

Special attention must be given to injuries to the abdominal aorta that can occur when entering the abdomen in any laparoscopic surgery. Intraabdominal injuries have occurred in all three techniques used to place the first trocar [30–34].

The overall risk of aortic injury with trocar placement in LRYGB is 0.043 % and up to 0.091 % when an optical trocar is used [35]. All techniques have pros and cons and the individual surgeon's personal experience in each type of entry is of great importance to prevent this complication [35] (Fig. 5.1)

Other signs and symptoms found in both intraluminal and intraperitoneal bleeding include hypotension, dizziness, weakness or shortness of breath, hypoactive bowel movement sounds, fever, and abdominal discomfort or abdominal hematoma [1].

5.4 Diagnosis and Management

The diagnosis of hemorrhage can be challenging due to altered postsurgical gastrointestinal anatomy. There is no standard therapeutic strategy defined for diagnosis and management. Clinical presentation and timing of bleeding will dictate the most appropriate diagnostic and therapeutic strategy. In some cases, the source of bleeding can be identified based on the clinical presentation without the need of endoscopy or imaging studies. For instance, hematemesis in a gastric bypass patient strongly suggests bleeding from a proximal source such as the gastric pouch or gastrojejunostomy. Melena, on the other hand, usually comes from a bleeding source at the jejunojejunostomy or gastric remnant [3].

After suspicion of hemorrhage is established, a careful physical examination should be performed. Hematocrit/hemoglobin should be drawn and close monitoring of vital signs initiated, including heart rate, blood pressure, urine output, respiratory rate, and pulse oximetry. The systolic blood pressure may not decrease significantly until 25–40 % of blood volume is lost, so other signs of instability must be taken into consideration [36]. Frequent clinical assessments should be made. Attention should be paid to symptoms such as: pain level, shortness of breath and lethargy and objective symptoms such as tachycardia, changed mental status, decreased urine output, decreased/increased respiration, abdominal distension, peritoneal signs, color and volume drain output, bleeding at port sites or bruising, bloody/black stools, bloody vomiting, signs of intestinal obstruction (from occluding blood clots), and jaundice (from absorbing blood or hemophilia).

- *In the hemodynamically stable patient*: Immediate resuscitation fluids (crystalloid or PRBC) and close monitoring (transfer to ICU if judged necessary). The recommendations and suggestions of the 2015 practice guidelines for perioperative blood management of the American Society of Anesthesiologists are as follows [37]:
  - The determination whether hemoglobin concentrations between 6 and 10 g/dL justify or require red blood cell transfusions should be based on potential ongoing bleeding (rate and magnitude), intravascular volume status, signs of organ ischemia, and adequacy of cardiopulmonary reserve.
  - Red blood cells should be administered unit-by-unit, when possible, with interval reevaluation. The maximal surgical blood order schedule should be used, in accordance with your institutional policy.
  - Anticoagulants should be reversed, if previously used. Urgent reversal for warfarin requires Prothrombin Complex Concentrate (PCC) while vitamin K may be used for non-urgent reversal.
  - Treatment of excessive bleeding: start by obtaining a full platelet count and a test of platelet function, if available, in patients with suspected drug induced platelet dysfunction. Platelet transfusion may be indicated despite an apparently adequate platelet count or, in the absence of a platelet count, if there is known or suspected platelet dysfunction and in surgical or

obstetric patients. Platelet transfusion is rarely indicated when platelet count is known to be greater than 100×10^9/l and is usually indicated when the count is less than 50×10^9/l in the presence of excessive bleeding.

- Obtain coagulation tests (PT/INR and aPTT) before transfusion of fresh frozen plasma (FFP)—if results are normal, FFP should not be used. FFP may be indicated for excessive microvascular bleeding in the presence of an INR > 2.0 in the absence of heparin, in urgent reversal of warfarin when no PCCs are available, for correction of excessive microvascular bleeding secondary to coagulation factor deficiency in patients transfused with more than one blood volume (approximately 70 mL/kg) and when PT or INR and aPTT cannot be obtained in a timely fashion.
- Assess fibrinogen levels before the administration of cryoprecipitate, if possible. Such evaluation is indicated when a test of fibrinogen activity indicates fibrinolysis, when the fibrinogen concentration is less than 80–100 mg/dL in the presence of excessive bleeding, as an adjunct in massively transfused patients when fibrinogen concentrations cannot be measured in a timely fashion, and for patients with congenital fibrinogen deficiencies. Whenever possible, decisions regarding patients with congenital fibrinogen deficiencies should be made in consultation with the patient's hematologist
- Desmopressin and topical hemostatics such as fibrin glue or thrombin gel can be used. Consider the use of antifibrinolytics if fibrinolysis is documented or suspected and if these agents are not already being used.
- PCCs may be used in patients with excessive bleeding and increased INR.
- Consider recombinant activated factor VII when traditional options for treating excessive bleeding have been exhausted.

A formula that helps calculate the drop in hematocrit with the use of the estimated blood loss is available [38]:

$$\text{EBV}\left(\text{estimated blood volume}\right) = \text{weight}\left(\text{kg}\right) \times \text{Average blood volume}$$

EBV for Adult Men is 75 mL/kg and for Adult Women 65 mL/kg

$$\text{Allowable Blood Loss} = \left[\text{EBV} \times \left(H_i - H_f\right)\right] / H_i$$

$$\left(H_i = \text{initial hematocrit and } H_f = \text{final lowest hematocrit accepted}\right).$$

If the patient remains hemodynamically stable, endoscopic inspection of the gastrojejunostomy should be considered. Thermal coagulation or epinephrine injections via therapeutic endoscopy have been successful in the management of bleeding at the gastrojejunostomy and jejunojejunostomy [3, 39, 40]. This procedure is effective for both late bleeding and early bleeding in patients who are hemodynamically stable.

EGD for early bleeding should optimally be performed under general anesthesia, in the operating room and with endotracheal intubation [10, 41]. Endoscopy has shown to control acute bleeding from the gastrojejunal anastomosis; endoscopic management of jejunojejunostomy hemorrhage has also been described [39, 40]. When the source of the gastrointestinal hemorrhage in a gastric bypass patient is not visualized endoscopically, the gastric remnant or the duode-

num should be suspected [27]. The bypassed stomach of the RYGB patient is inaccessible by conventional endoscopy, so an alternative method of access must be utilized. These approaches include laparoscopic transgastric endoscopy in which a laparoscopic trocar is surgically placed into the gastric remnant and serves as a conduit for passage of the flexible endoscope [10], percutaneous endoscopic gastrostomy [42] and retrograde double balloon endoscopy [43, 44]. A more complex method of gastric remnant access involves the use of a double-balloon endoscope to achieve retrograde endoscopy. This technique is particularly technically challenging and requires a specially trained endoscopist to perform [45, 46].

- *Hemorrhage in an unstable patient*: Unstable vital signs, such as hypotension, persistent severe tachycardia, and drop in hematocrit of 10 % or continuous dropping after transfusion indicates the need for urgent surgical intervention. Also, frank hematemesis or bright red blood per rectum within the first 6 h after surgery with a decline in hematocrit indicates active bleeding which will most likely require surgical intervention [2]. The objective of the reoperation is to identify the bleeding source, decompress the lumen from blood and blood clots, and control the bleeding. The patient has to be immediately resuscitated with fluids and blood products must be given as needed. It is advisable to use a combined management approach with intraoperative endoscopy to manage gastrointestinal bleeding with care to avoid disrupting a newly created anastomosis. Surgical management consists of either laparoscopy or laparotomy. If the patient is profoundly hypotensive, laparoscopy is relatively contraindicated.

5.5 Prevention

- Confirm Blood Type (based on two or more independently collected samples): Order a "Type and Screen" for every patient who is undergoing bariatric surgery. The specimen may be drawn up to 30 days in advance of surgery.
- Thorough examination through the patient's history and on his preoperative laboratory results in the preoperative assessment. Investigate the patient's history and his family's history of bleeding. If any suspicion of higher risk for bleeding, he should be referred to a hematology specialist and receive a more thorough investigation. Afterwards, the patient should return as an outpatient, with recommendations of preventions for bleeding, if needed.
- Mechanical and chemical prevention, making sure to obtain hemostasis in all staple-line edges.
- Consider reinforcing the staple-line in sleeve gastrectomy using bovine pericardium, synthetic polyester or glycolide/trimethylene or oversewing. This may enhance homeostasis and reduce bleeding incidence when compared to no reinforcement [47–49].
- Using the correct staple height for a given tissue is one of the most important factors to limit this complication [50]. Use thicker staple sizes for the stomach and thinner staple sizes for the small bowel [18].
- The use of routine peritoneal drainage after RYGB does not provide clear benefits [51, 52]. Normal serous drainage in a patient with suspected bleeding does not rule out the possibility of intraperitoneal hemorrhage (Fig. 5.2).

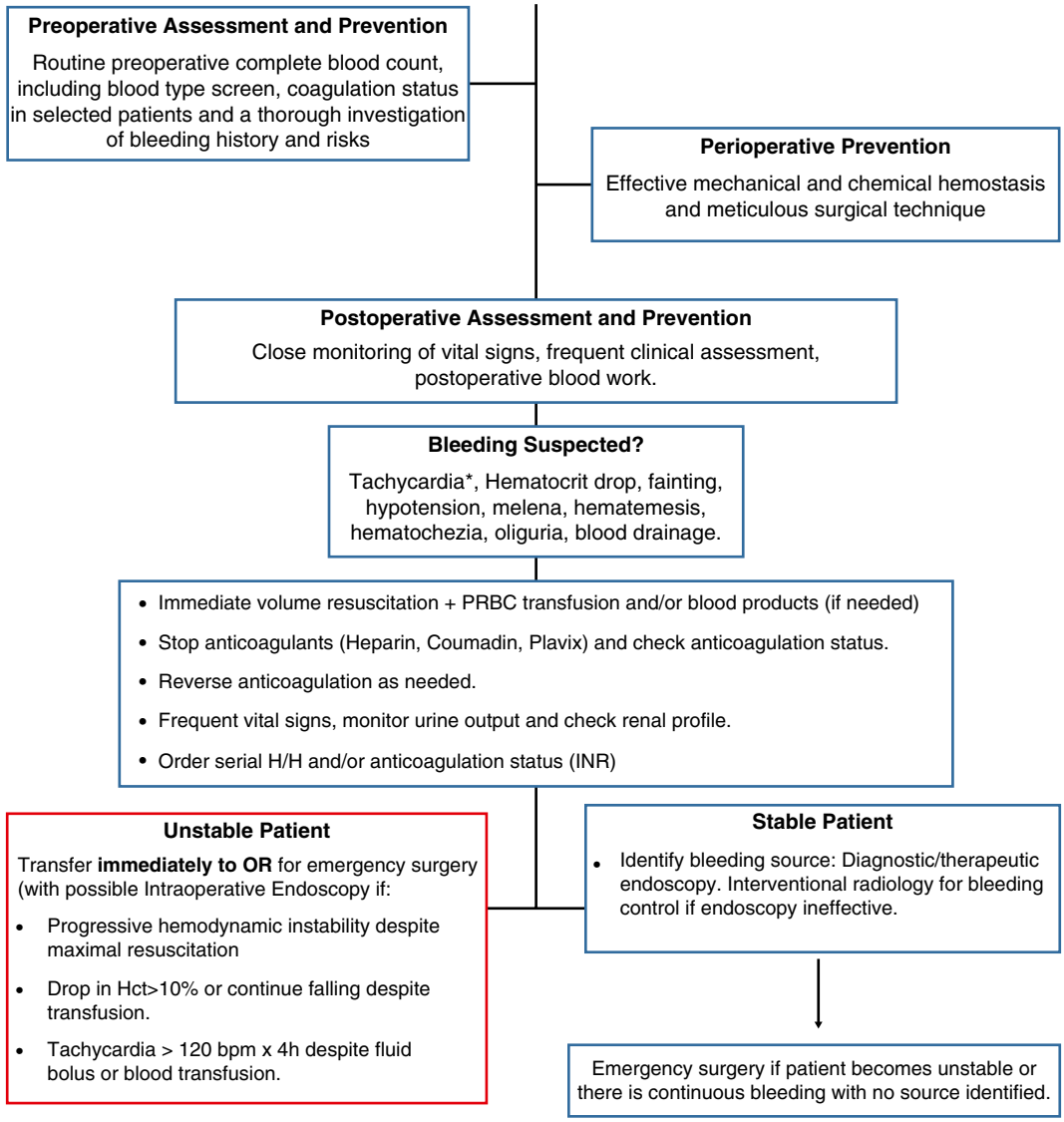

Fig. 5.2 Proposed algorithm for management of hemorrhage

References

1. Heneghan HM, Meron-Eldar S, Yenumula P, Rogula T, Brethauer SA, Schauer PR. Incidence and management of bleeding complications after gastric bypass surgery in the morbidly obese. Surg Obes Relat Dis. 2012;8(6):729–35.
2. Nguyen NT, Rivers R, Wolfe BM. Early gastrointestinal hemorrhage after laparoscopic gastric bypass. Obes Surg. 2003;13(1):62–5.
3. Dick A, Byrne TK, Baker M, Budak A, Morgan K. Gastrointestinal bleeding after gastric bypass surgery: nuisance or catastrophe? Surg Obes Relat Dis. 2010;6(6):643–7.
4. Higa KD, Boone KB, Ho T. Complications of the laparoscopic Roux-en-Y gastric bypass: 1,040 patients--what have we learned? Obes Surg. 2000;10(6):509–13.
5. Bransen J, Gilissen LPL, van Rutte PWJ, Nienhuijs SW. Costs of leaks and bleeding after sleeve gastrectomies. Obes Surg. 2015;25(10):1767–71.
6. Frezza EE, Reddy S, Gee LL, Wachtel MS. Complications after sleeve gastrectomy for morbid obesity. Obes Surg. 2009;19(6):684–7.
7. Brethauer SA, Hammel JP, Schauer PR. Systematic review of sleeve gastrectomy as staging and primary

bariatric procedure. Surg Obes Relat Dis. 2009;5(4): 469–75.
8. Schauer PR, Schirmer BD, Brethauer S. Minimally invasive bariatric surgery. New York: Springer Science & Business Media; 2008. p. 370. Chapter 24; 516 p.
9. Rosenthal RJ, Szomstein S, Kennedy CI, Soto FC, Zundel N. Laparoscopic surgery for morbid obesity: 1,001 consecutive bariatric operations performed at The Bariatric Institute, Cleveland Clinic Florida. Obes Surg. 2006;16(2):119–24.
10. Rabl C, et al. Early and late abdominal bleeding after Roux-en-Y gastric bypass: sources and tailored therapeutic strategies. Obes Surg. 2011;21:413–20.
11. Campos GM, et al. Spectrum and risk factors of complications after gastric bypass. Arch Surg. 2007;142: 969–75. discussion 976.
12. Varela JE, Wilson SE, Nguyen NT. Outcomes of bariatric surgery in the elderly. Am Surg. 2006;72:865–9.
13. Bakhos C, Alkhoury F, Kyriakides T, Reinhold R, Nadzam G. Early postoperative hemorrhage after open and laparoscopic roux-en-y gastric bypass. Obes Surg. 2009;19:153–7.
14. Parikh J, et al. Is high BMI associated with specific complications after laparoscopic Roux-en-Y gastric bypass? Am Surg. 2007;73:959–62.
15. Mourelo R, Kaidar-Person O, Fajnwaks P, Roa PE, Pinto D, Szomstein S, Rosenthal RJ. Hemorrhagic and thromboembolic complications after bariatric surgery in patients receiving chronic anticoagulation therapy. Obes Surg. 2008;18(2):167–70.
16. Birkmeyer NJO, Finks JF, Carlin AM, Chengelis DL, Krause KR, Hawasli AA, et al. Comparative effectiveness of unfractionated and low-molecular-weight heparin for prevention of venous thromboembolism following bariatric surgery. Arch Surg. 2012;147(11) :994–8.
17. Ikesaka R, Delluc A, Le Gal G, Carrier M. Efficacy and safety of weight-adjusted heparin prophylaxis for the prevention of acute venous thromboembolism among obese patients undergoing bariatric surgery: a systematic review and meta-analysis. Thromb Res. 2014;133(4):682–7.
18. Podnos YD, Jimenez JC, Wilson SE, Stevens CM, Nguyen NT. Complications after laparoscopic gastric bypass: a review of 3464 cases. Arch Surg. 2003; 138(9):957–61.
19. Topart P, Becouarn G, Ritz P. Comparative early outcomes of three laparoscopic bariatric procedures: sleeve gastrectomy, Roux-en-Y gastric bypass, and biliopancreatic diversion with duodenal switch. Surg Obes Relat Dis. 2012;8(3):250–4.
20. Weiner RA, El-Sayes IA, Theodoridou S, Weiner SR, Scheffel O. Early post-operative complications: incidence, management, and impact on length of hospital stay. A retrospective comparison between laparoscopic gastric bypass and sleeve gastrectomy. Obes Surg. 2013;23(12):2004–12.
21. Schauer PR, Schirmer BD, Brethauer S. Minimally invasive bariatric surgery. New York: Springer Science & Business Media; 2008. p. 275. Cap.21.5; 516 p.
22. Bailey JG, Hayden JA, Davis PJB, Liu RY, Haardt D, Ellsmere J. Robotic versus laparoscopic Roux-en-Y gastric bypass (RYGB) in obese adults ages 18 to 65 years: a systematic review and economic analysis. Surg Endosc. 2014 Feb;28(2):414–26.
23. Mala T, Søvik TT, Schou CF, Kristinsson J. Blood clot obstruction of the jejunojejunostomy after laparoscopic gastric bypass. Surg Obes Relat Dis. 2013;9(2):234–7.
24. Rogula T, Yenumula PR, Schauer PR. A complication of Roux-en-Y gastric bypass: intestinal obstruction. Surg Endosc. 2007;21(11):1914–8.
25. Gunabushanam G, Shankar S, Czerniach DR, Kelly JJ, Perugini RA. Small-bowel obstruction after laparoscopic Roux-en-Y gastric bypass surgery. J Comput Assist Tomogr. 2009;33(3):369–75.
26. Torrens AS, Born PW, Naver L. Gastric blow-out: complication after obesity surgery. Ugeskr Laeger. 2009;171(49):3624–5.
27. Braley SC, Nguyen NT, Wolfe BM. Late gastrointestinal hemorrhage after gastric bypass. Obes Surg. 2002;12(3):404–7.
28. Huang CS, Forse RA, Jacobson BC, Farraye FA. Endoscopic findings and their clinical correlations in patients with symptoms after gastric bypass surgery. Gastrointest Endosc. 2003;58(6):859–66. http://www.giejournal.org/article/S0016510703023101/abstract.
29. Bellorin O, Abdemur A, Sucandy I, Szomstein S, Rosenthal RJ. Understanding the significance, reasons and patterns of abnormal vital signs after gastric bypass for morbid obesity. Obes Surg. 2011;21(6):707–13.
30. Azevedo JLMC, Azevedo OC, Miyahira SA, Miguel GPS, Becker OM, Hypólito OHM, et al. Injuries caused by Veress needle insertion for creation of pneumoperitoneum: a systematic literature review. Surg Endosc. 2009;23(7):1428–32.
31. Deziel DJ, Millikan KW, Economou SG, Doolas A, Ko ST, Airan MC. Complications of laparoscopic cholecystectomy: a national survey of 4,292 hospitals and an analysis of 77,604 cases. Am J Surg. 1993; 165(1):9–14.
32. Kazemier G, Hazebroek EJ, Lange JF, Bonjer HJ. Needle and trocar injury during laparoscopic surgery in Japan. Surg Endosc. 1999;13(2):194.
33. Champault G, Cazacu F, Taffinder N. Serious trocar accidents in laparoscopic surgery: a French survey of 103,852 operations. Surg Laparosc Endosc. 1996;6(5):367–70.
34. Sharp HT, Dodson MK, Draper ML, Watts DA, Doucette RC, Hurd WW. Complications associated with optical-access laparoscopic trocars. Obstet Gynecol. 2002;99(4):553–5.
35. Sundbom M, Hedberg J, Wanhainen A, Ottosson J. Aortic injuries during laparoscopic gastric bypass for morbid obesity in Sweden 2009–2010: a nationwide survey. Surg Obes Relat Dis. 2014;10(2):203–7.
36. Cogbill, TH. Abnormal operative and postoperative bleeding. In: Current surgical therapy. 10th Edition, Cameron, JH. Philadelphia, PA: 2010; Elsevier; p.1090.

37. American Society of Anesthesiologists Task Force on Perioperative Blood Management. Practice guidelines for perioperative blood management: an updated report by the American Society of Anesthesiologists Task Force on Perioperative Blood Management. Anesthesiology. 2015;122(2):241–75.
38. http://ether.stanford.edu/calc_mabl.html
39. Nguyen NT, Longoria M, Chalifoux S, Wilson SE. Gastrointestinal hemorrhage after laparoscopic gastric bypass. Obes Surg. 2004;14(10):1308–12.
40. Moretto M, Mottin CC, Padoin AV, Berleze D, Repetto G. Endoscopic management of bleeding after gastric bypass -- a therapeutic alternative. Obes Surg. 2004;14(5):706.
41. Santo MA, Pajecki D, Riccioppo D, Cleva R, Kawamoto F, Cecconello I. Early complications in bariatric surgery: incidence, diagnosis and treatment. Arq Gastroenterol. 2013;50(1):50–5.
42. Gill KR, McKinney JM, Stark ME, Bouras EP. Investigation of the excluded stomach after Roux-en-Y gastric bypass: the role of percutaneous endoscopy. World J Gastroenterol. 2008;14(12):1946–8. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2700407/.
43. Tagaya N, Kasama K, Inamine S, Zaha O, Kanke K, Fujii Y, et al. Evaluation of the excluded stomach by double-balloon endoscopy after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2007;17(9):1165–70.
44. Sakai P, Kuga R, Safatle-Ribeiro AV, Faintuch J, Gama-Rodrigues JJ, Ishida RK, et al. Is it feasible to reach the bypassed stomach after Roux-en-Y gastric bypass for morbid obesity? The use of the double-balloon enteroscope. Endoscopy. 2005;37(6):566–9.
45. Flickinger EG, Sinar DR, Pories WJ, Sloss RR, Park HK, Gibson JH. The bypassed stomach. Am J Surg. 1985;149(1):151–6.
46. Sinar DR, Flickinger EG, Park HK, Sloss RR. Retrograde endoscopy of the bypassed stomach segment after gastric bypass surgery: unexpected lesions. South Med J. 1985;78(3):255–8.
47. D'Ugo S, Gentileschi P, Benavoli D, Cerci M, Gaspari A, Berta RD, et al. Comparative use of different techniques for leak and bleeding prevention during laparoscopic sleeve gastrectomy: a multicenter study. Surg Obes Relat Dis. 2014;10(3):450–4.
48. Shah SS, Todkar JS, Shah PS. Buttressing the staple line: a randomized comparison between staple-line reinforcement versus no reinforcement during sleeve gastrectomy. Obes Surg. 2014;24(12):2014–20.
49. Choi YY, Bae J, Hur KY, Choi D, Kim YJ. Reinforcing the staple line during laparoscopic sleeve gastrectomy: does it have advantages? A meta-analysis. Obes Surg. 2012;22(8):1206–13.
50. Schauer PR, Schirmer BD, Brethauer S. Minimally invasive bariatric surgery. New York: Springer Science & Business Media; 2008. p. 292. Chapter 21.7; 516 p.
51. Dallal RM, Bailey L, Nahmias N. Back to basics--clinical diagnosis in bariatric surgery. Routine drains and upper GI series are unnecessary. Surg Endosc. 2007;21(12):2268–71.
52. Liscia G, Scaringi S, Facchiano E, Quartararo G, Lucchese M. The role of drainage after Roux-en-Y gastric bypass for morbid obesity: a systematic review. Surg Obes Relat Dis. 2014;10(1):171–6.

Enteric Leaks after Gastric Bypass: Prevention and Management

6

Cheguevara Afaneh and Gregory F. Dakin

Key Points

- Enteric leaks typically present with abdominal pain, tachycardia, fever, and tachypnea.
- Some weak evidence suggests that the use of buttressing materials may aid in preventing the occurrence of a leak; however, this is far from definitive.
- Upper GI or CT scans may be used to diagnose a leak.
- Surgical exploration is mandatory in most cases, unless the patient is hemodynamically stable and the leak is well-contained.
- Management includes *nil per os*, broad-spectrum antibiotic therapy, total parenteral nutrition, and adequate drainage.

6.1 Introduction

Obesity remains an epidemic disease that plagues the Western world [1, 2]. Bariatric surgery has proven to be the most effective tool in combating obesity and its associated comorbidities [3]. Bariatric surgery affords patients a reduction in morbid obesity-related medical comorbidities, improvements in quality of life, as well as reduction in overall mortality. Laparoscopic Roux-en-Y gastric bypass (LRYGB) is one of the most commonly performed bariatric procedures to treat morbid obesity (Fig. 6.1) [4]. This has been deemed safe and effective for the treatment of morbid obesity. Nevertheless, this procedure is technically demanding and carries a risk of several complications. One of the most feared complications is an anastomotic leakage. The overall reported incidence of this complication in both open RYGB and LRYGB ranges from 1 to 5.6 % [2, 5–7].

Development of an enteric leak can lead to devastating morbidity as well as significantly increased mortality [8]. Early diagnosis may significantly reduce morbidity and mortality, although the host immunoreactivity triggering the inflammatory response probably plays a larger role than timing of treatment [9]. Patients who develop an enteric leak require additional diagnostic tests, longer duration of hospitalizations, intensive care unit support, prolonged ventilator support, and even additional surgical interventions in some instances. The most common site of an enteric leak following Roux-en-Y gastric bypass (RYGB) is the gastrojejunostomy.

The purpose of this chapter is to discuss the presentation and predilection of patients with an enteric leak after RYGB for morbid obesity; assess various methods to prevent enteric leaks;

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_6

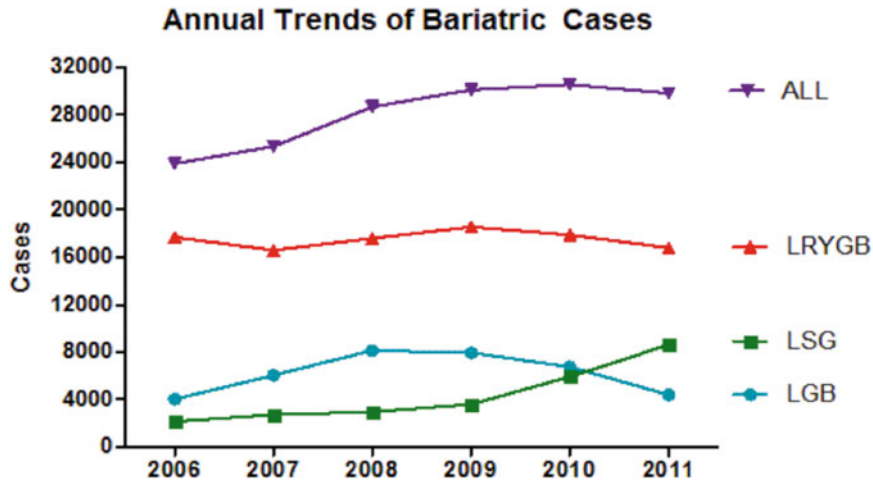

Fig. 6.1 Population-based trends of various bariatric surgery procedures from California, Florida, and New York. The graph demonstrates the number of cases performed for laparoscopic Roux-en-Y gastric bypass (LRYGB), laparoscopic sleeve gastrectomy (LSG), and laparoscopic gastric band placement (LGB) over the last 5 years in California, Florida, and New York

and offer several treatment options to manage these patients.

6.2 Etiology of an Enteric Leak

An anastomotic leak is a disruption of the normal acute healing process, leading to a defect in the new enteric connection. The basic tenets of any anastomotic technique include avoiding excess tension, maintaining adequate blood flow, and providing adequate oxygenation to avoid ischemia [10]. Ischemia may occur secondary to excess mechanical tension, excessive dissection, or preexisting comorbidities, such as atherosclerosis, diabetes mellitus, or coronary artery disease. Other factors that may contribute include previous history of chemotherapy, prior radiation exposure or administration, and use of glucocorticoids.

Patient factors have been implicated in the etiology of enteric leaks. Several studies have identified individual risk factors for major complications following RYGB. Livingston et al. found male gender, revisional procedures, advanced age, and increasing weight to be predictors of major complications after RYGB [11]. Gonzalez and colleagues validated BMI > 50 kg/m^2 and revisional procedures to be independent risk factors for postoperative complications [12]. Nguyen et al. confirmed male gender, advanced age, as well as surgeon inexperience (fewer than 75 cases), to have a negative impact on postoperative outcomes following LRYGB [13]. In a study by Fernandez and colleagues of 3000 patients undergoing bariatric procedures, male patients, advanced age, increased weight and those with multiple comorbidities were at increased risk for developing an anastomotic leak [6].

6.3 Presentation and Diagnosis

Over the last two decades, bariatric surgeons have become adept at performing the RYGB. Nevertheless, enteric leaks remain a feared complication in the morbidly obese postoperative patient. Enteric leaks present with some element of peritonitis or sepsis. Clinical signs and symptoms may include fever, tachycardia, nausea, vomiting, abdominal pain, tachypnea, shortness of breath, and/or altered mental status. Laboratory data may demonstrate a leukocytosis. It is imperative that the clinician have a high index of suspicion, as these patients are often difficult to examine given their girth and body

habitus. Additionally, many of these patients have multiple comorbidities that may confound the diagnosis. Nevertheless, some patients may be completely asymptomatic. In these cases, additional diagnostic modalities should be investigated.

Some patients may require additional investigative studies to diagnose an enteric leak. Some centers advocate the use of routine postoperative upper gastrointestinal series (UGIS). The sensitivity of detecting a leak is fairly variable, ranging from 22 to 75 % [14]. The variability in sensitivity is multifactorial, attributable to the low quality of radiologic imaging, limited radiologist clinical experience, premature timing of the test, and initial postoperative anastomotic edema of the gastrojejunostomy. Another imaging modality is helical computed tomography (CT) scan. CT scanning has high specificity and low rate of false-negativity for enteric leaks. The major drawbacks to using CT scanning in place of UGIS as a first radiographic test is the higher cost, weight limit of the table, availability of the machine, as well as aperture of the machine. Nevertheless, CT scanning may be necessary as UGIS is not sensitive or specific enough. Furthermore, the clinician must maintain a high index of suspicion, even in the face of a negative UGIS or even a CT scan sometimes.

6.4 Prevention of Enteric Leaks

Any large series of RYGB will report a certain percentage of patients experiencing an enteric leak. The majority of leaks are probably not solely due to technical error, but rather multifactorial, as previously discussed. Various surgical techniques have been reported to decrease the incidence of an enteric leak, including hand-sewing the anastomosis, use of a linear stapler, use of a circular stapler, or some combination of the aforementioned techniques. Moreover, technical modifications have also included oversewing staple lines, reinforcing staple lines, the use of fibrin glue, or the use of other tissue sealants (Table 6.1). At our institution, we perform an intraoperative leak test to assess for any potential leaks. This test is performed with a methylene blue dye instilled into the gastric pouch while the Roux limb is obstructed. Any blue extravasation is considered a positive test for a leak. The test can also be performed with air and the anastomosis can be submerged in irrigation fluid. Any bubbling noted would be considered a positive test as well. Data regarding the efficacy of this technique in preventing enteric leaks are predominantly retrospective, but highly suggestive of being helpful in identifying intraoperative leaks [15, 16].

Table 6.1 Preventative considerations for enteric leaks

Anatomic considerations
Avoid overdissection of blood supply
Divide greater omentum
Score the mesentery
Retrocolic position
Stapling considerations
Linear reinforcement with buttressing material
Reinforce staple line with sutures
Fibrin-sealant reinforcement

6.4.1 Blood Supply and Tension

Maintaining adequate blood supply is essential for prevention of anastomotic ischemia, necrosis, and failure. Careful and meticulous dissection of the left gastric artery branches to the pouch should be preserved. Minimal dissection of the lesser curve and avoiding excessive dissection of the phrenoesophageal ligament will help maintain adequate blood supply to the gastric pouch. Mobilizing the esophagus at the hiatus is another technique to increase esophageal length and decrease tension on the pouch. These points are especially important for patients undergoing a revisional bariatric procedure, i.e., those who previously underwent a sleeve gastrectomy or previously had an adjustable gastric band.

Avoiding tension from the root of the mesentery of the Roux limb facilitates a tension-free anastomosis. The proximal jejunum should be run caudally to a point of tension-free mesenteric mobility, typically between 50 and 100 cm distal to the ligament of Treitz. We routinely divide the

greater omentum to within 2 cm from the transverse colon, which is especially helpful in patients with significant intraabdominal fat. The omentum is then divided parallel to the transverse colon to allow an obstructed path of the Roux limb to the gastric pouch in an antecolic, antegastric fashion. This allows confirmation of a gastric pouch with minimal to no tension. If there is any doubt, the mesentery can be scored to further decrease the tension. An alternative approach is a Roux limb placed in a retrocolic position which generally is a shorter path to the proximal gastric pouch and can be a very useful in high-BMI or male patients with a predominance of intra-abdominal and mesenteric fat.

6.4.2 Surgical Techniques

Several techniques have evolved in creation of the gastric pouch and the jejunojejunostomy in patients undergoing RYGB. The choice of anastomotic technique is largely a function of surgeon preference and institutional familiarity. Initial studies over a decade ago by Gonzalez et al. found no difference in leak rate for patients undergoing LRYGB between the hand-sewn, linear-stapled, and circular-stapled anastomosis for the gastric pouch (no leaks occurred in all 87 patients studied), although stricture rates occurred more frequently in the circular-stapled group (31 %) compared to the hand-sewn (3 %) and linear-stapled (0 %) groups ($P<0.01$) [17]. Bendewald and colleagues compared a series of 882 consecutive patients undergoing LRYGB for morbid obesity [18]. Three different techniques were performed for creation of the gastrojejunostomy, including hand-sewn, use of a linear stapler, and use of a 25 mm circular stapler. On multivariate analysis, the authors found leak rates of 1.1 % in the hand-sewn group, 1.0 % in the linear stapler group, and no leaks in the circular stapler group ($p=0.48$) and concluded that there was no difference in outcomes with respect to anastomotic technique. Stricture rates were also not significantly difference in this study (6.1 % vs. 6.0 % vs. 4.3 %, respectively; $p=0.66$). Giordano and colleagues conducted a meta-analysis of 1321 patients from eight studies and compared the linear-stapled to the circular-stapled anastomosis during gastrojejunostomy for LRYGB [19]. The primary endpoints were gastrojejunal leak and stricture rates. No technique was superior to the other with respect to leak rate; however, the linear-stapled anastomosis demonstrated a significantly lower risk of stricture (relative risk [RR]: 0.34; 95 % confidence interval [CI]: 0.12–0.93; $p=0.04$). Wound infection (RR: 0.38; 95 % CI: 0.22–0.67; $p=0.0008$) and operative times ($P<0.0001$) were significantly lower with the linear stapler technique as well.

6.4.3 Staple Line Reinforcement

Various tools are available in the bariatric surgeon's armamentarium to prevent adverse intraoperative and postoperative events, including enteric leaks and bleeding (Fig. 6.2). One of these tools is treated bovine pericardial strips for staple line buttressing. These were first introduced in 1994 in the field of thoracic surgery to decrease the incidence and duration of air-leaks following lung resections [20]. The first major application of this type of product in bariatric surgery was to decrease the incidence of extraluminal bleeding using a linear stapler buttressed with this material. Angrisani et al. performed a prospective randomized control trial of 98 patients undergoing LRYGB for morbid obesity [21]. Fifty patients were randomized to the treated bovine pericardial strips for use with the linear stapler, while the remaining 48 patients had non-buttressed staple lines. The gastrojejunostomy was performed using a circular stapler, but the gastric transection was still performed with a linear stapler. Although the authors focused on extraluminal bleeding, which was significantly lower in the bovine pericardial strip group based on operative time and number of clips used ($P<0.01$), the number of positive methylene blue leak tests was 6/48 in the non-buttressed group and 0 in the bovine pericardial strip group ($P<0.0001$).

Another prospective randomized control trial using a slightly different product, polyglycolic acid (PGA) and trimethylene carbonate, demonstrated the superiority of the bioabsorbable staple line material during LRYGB for morbid obesity

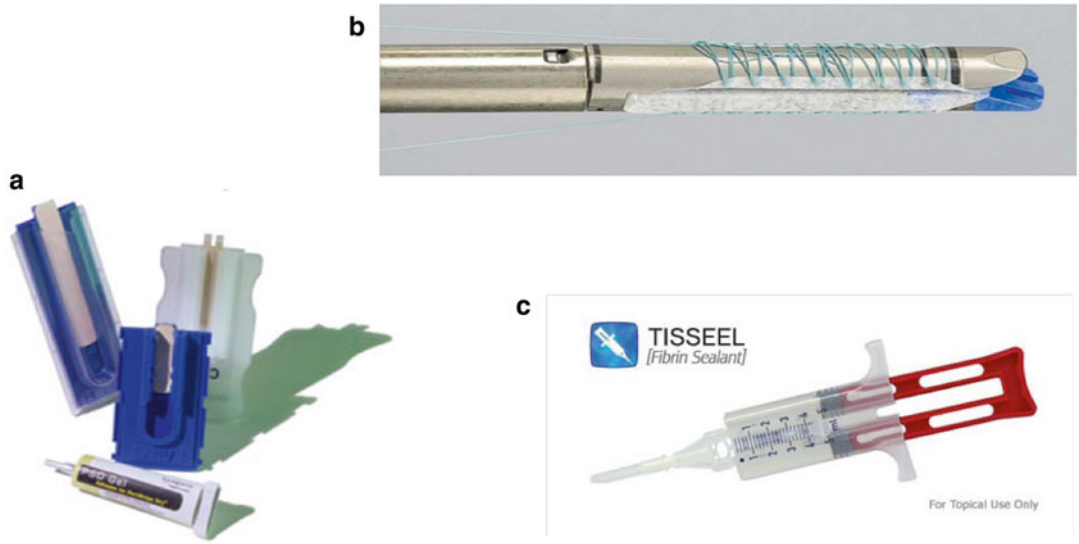

Fig. 6.2 Anastomotic buttressing materials. *Panel A* shows the bovine pericardial strips. *Panel B* depicts the absorbable polymer membrane (Bioabsorbable Seamguard, W.L. Gore, Flagstaff, Arizona, USA). *Panel C* illustrates the fibrin sealant (Tisseel, Baxter Healthcare Corp. ©, Deerfield, IL)

with respect to staple line bleeding [22]. In this study, there were no leaks in either group, but there was one positive methylene blue leak test in the non-buttressed group. Three patients in the non-buttressed group developed gastrogastric fistulas; however, this was not statistically significant ($p=0.20$).

Circular stapler reinforcement has also been investigated with respect to enteric leak rates, bleeding, and stricture rates. In a study by Jones and colleagues of 393 patients undergoing LRYGB, the use of bioabsorbable staple line reinforcement (PGA and trimethylene carbonate) for circular staplers was investigated [23]. In this study, 138 consecutive patients underwent bioabsorbable staple line reinforcement for the circular stapler and these were compared to a series of 255 patients without circular staple line reinforcement. There was no significant difference in anastomotic leak rate or bleeding in the buttressed versus the non-buttressed groups (0.7 % vs. 1.9 %, respectively; $p=0.34$, and 0.7 % vs. 1.1 %, respectively; $p=0.64$). However, the incidence of stricture was significantly higher without the use of a bioabsorbable staple line reinforcement material (9.3 % vs. 0.7 %, respectively; $p=0.0005$). Ibele et al. reported on a series of 81 consecutive patients who underwent circular stapled anastomoses using a nonabsorbable buttressing material (bovine pericardium strip) [24]. These patients were compared to a series of 419 patients who underwent circular stapled anastomoses without buttressing material. The leak rate was significantly higher in the buttressed group (4.9 % vs. 0.7 %, respectively; $p=0.02$). Moreover, one staple line failure occurred in the buttressed group compared to none in the non-buttressed group. The authors concluded that caution should be taken when using the buttressing material for circular stapled anastomoses, given the staple line failure and enteric leaks.

Another option to reinforce staple lines includes the use of a fibrin sealant. The sealant forms an insoluble polymerized matrix that stabilizes as it adheres to the edges of the gastrojejunal anastomosis, which effectively hinders fibrinolysis by inhibition of the plasminogen–plasmin cascade. This theoretically creates an impermeable seal along the anastomosis. In a large study by Sapala and colleagues of 738 patients, the effects of vapor-heated fibrin sealant was investigated to assess the efficacy in anastomotic leaks at the gastrojejunostomy for patients

undergoing RYGB for morbid obesity [25]. A total of 1 mL of vapor-heated fibrin glue was applied to the anastomosis. Two patients developed a leak (0.3 %) compared to their historical control leak rate of 0.9 %. Interestingly, the anastomotic leaks did not occur at the fibrin-sealed gastrojejunostomy sites. Furthermore, no gastrogastric fistulas occurred. In another study of 480 undergoing RYGB for morbid obesity, 120 patients had fibrin sealant applied to the gastrojejunal anastomosis [26]. None of the patients in the fibrin sealant group developed a leak, while 8 of the remaining 360 patients developed an enteric leak requiring either re-operation, a drainage procedure, or long-term parenteral nutrition. A prospective multicenter, randomized trial of 320 patients studying the use of fibrin sealant to prevent major complications following LRYGB demonstrated no significant difference in rate of anastomotic leaks with fibrin sealant application (1 leak in fibrin sealant group vs. 3 leaks in control group) [27]. The early complication rate was not significantly different between the two groups ($P > 0.05$). However, patients without fibrin sealant application had a significantly higher reintervention rate for early postoperative complications ($p = 0.016$). There were six patients with gastrojejunal stenosis in each group.

6.5 Management of Enteric Leaks

Management of enteric leaks is dependent on several factors, including the severity and location of the leak. Nevertheless, the mainstay of treatment is surgery and other interventions can be considered on a case-by-case basis. Broad-spectrum antibiotic therapy should be initiated immediately and, dependent on the extent of the leak, the patient should be taken back to the operating room for either diagnostic laparoscopy or exploratory laparotomy.

Csendes et al. developed a classification system of leaks following RYGB [28]. The presence of a leak was evaluated by three parameters, including timing of appearance after surgery, severity of the leak (two types), and exact location of the leak (Table 6.2). In this manner leaks can be properly described and treatment algorithms can be tailored for each individual type of leak.

Table 6.2 Classification of leaks [28]

Parameter	Description
Timing	Early: 1–4 days after surgery
	Intermediate: 5–9 days after surgery
	Late: ≥10 days after surgery
Severity	Type I: localized leak, minimal systemic inflammation, small drainable collection
	Type II: systemic inflammation, large collection ± air fluid level, requires careful drain care
Location:	Type 1: Gastric pouch Type 2: Gastrojejunal anastomosis
	Type 3: Jejunal stump Type 4: Jejunojejunal anastomosis
	Type 5: Excluded stomach Type 6: Duodenal stump (in resectional bypass)
	Type 7: Blind end biliary jejunal limb after laparoscopic surgery

Three important goals when managing enteric leaks need to be achieved. First, wide and adequate drainage needs to be performed to clean the abdominal cavity of any contamination and avoid further complications. Second, correct the underlying defect. This typically involves suturing the perforation after properly debriding the edges. This may be difficult if significant inflammation has already set-in and the tissue planes are dense or difficult to ascertain. In some cases, this is not feasible and wide drainage may be the staple of treatment. Defects in the jejunojejunostomy are typically more amenable to repair and rarely warrant anastomotic revision. Finally, the gastric remnant should be decompressed via a gastrostomy tube to avoid gastric dilatation from the imminent ileus from the contamination. Moreover, the gastrostomy tube can be used for enteral access, bypassing the area of leak if that so happens to be the gastrojejunostomy. Patients are monitored in an Intensive Care Unit. Postoperative care should be aimed at managing the sepsis with broad-spectrum antibiotics, *nil per os*, and nutritional support with total parenteral nutrition. Patients may or may not need ventilator support, which can generally be weaned in

the postoperative period. These patients may be at a higher risk of a venothromboembolic event, thus adequate mechanical and chemical prophylaxis should be maintained. Patients who fail to show signs of improvement should undergo repeat imaging with CT scan. Any collections should be drained via percutaneous radiographic modalities whenever feasible. Prior to resuming an oral diet, patients should undergo repeat UGI study to confirm that the leak has sealed.

Nonoperative treatment should only be considered in hemodynamically stable patients with contained or controlled leaks (Table 6.3) [5, 8]. The classic patient has little to no signs or symptoms of a leak and has an abnormal UGI study demonstrating a contained leak. These patients generally undergoing an additional imaging study, such as a CT scan to assess for any additional collections in the abdominal cavity. Most contained leaks can be access and drained by interventional radiologic techniques. Nonoperative management consists of nothing by mouth, broad-spectrum antibiotics, total parenteral nutrition, and adequate drainage. Many of these leaks resolve with 1–2 weeks. Repeat UGI studies are performed to assess for resolution of the leak. Patients may then be advanced to an oral diet once complete resolution of the leak is documented.

Durak and colleagues reported on a series of 1133 patients who underwent primary RYGB at a single institution [29]. The overall incidence of clinically apparent enteric leak after RYGB was 1.5 %. The most common site for enteric leak was at gastrojejunal anastomosis (13/17 or 76 %), followed by the gastric pouch (2/17 or 11 %), gastric remnant (1/17 or 6 %), and the jejunojejunostomy (1/17 or 6 %). All patients had a negative methylene blue leak test at the time of the original procedure. All but one patient was symptomatic, and that patient presented with acute kidney injury. The most common presenting signs and symptoms were abdominal pain, tachycardia and fever. Twelve of the 17 patients had abnormal radiographic studies, either UGI or CT, while the remainder had normal UGI or CT findings. In 13 patients (76 %), primary closure of the leak was performed with gastrostomy tube placement, and wide drainage. In three patients, wide drainage was performed with or without gastrostomy tube placement; and in one patient, no leak was detected intraoperatively and wide drainage was performed with or without gastrostomy tube placement. All patients received closed suction drains and broad-spectrum antibiotic therapy. One patient in this cohort died.

Table 6.3 Nonoperative management of enteric leaks

Clinical criteria
Hemodynamically stable
Well-contained leaks
No signs of peritonitis
Management
Nil per os
Broad-spectrum antibiotics
Total parenteral nutrition
Appropriate drainage

Recently, Jacobsen and colleagues reported on a series of 6030 patients who underwent LRYGB [30]. Sixty-four patients (1.1 %) developed a significant leak; 45 patients (70 %) were treated surgically, while 19 patients (30 %) were treated nonoperatively. The nonoperative treatment group had milder symptoms that presented, on average, 4 days later than those treated surgically (10 vs. 6.5 days, respectively; $p=0.03$). The most common symptoms included tachycardia and abdominal pain. All patients were treated with antibiotic therapy. The most common location for a leak was at the gastrojejunal anastomosis or along the staple line at the gastric pouch. Closure of the mesenteric defect had no association with the leak rate (1.1 % without closure vs. 1.0 % with closure, $p=0.85$). Twenty-patients underwent early operation (5 days or fewer after LRYGB), and 20 of the 22 patients had successful suturing of the defect. Five of these patients had a gastrostomy tube placed in the gastric remnant as the etiology of their early leak was obstruction. One patient with leak at the distal esophagus was treated with endoscopic stenting. Late leaks (>5 days after LRYGB) occurred in 23 patients and was treated with operative drainage in 19 patients and gastrostomy tube placement in the gastric remnant in 15 patients. Thirteen patients had suture repair of the defect and/or

omentoplasty. Two patients, treated at an outside hospital, underwent endoscopic stenting for a leak at the gastrojejunostomy. Two deaths occurred in the entire cohort of leaks (3 %), both with late leaks. The authors reported worse outcomes for those patients who experienced symptoms for more than 24 h prior to reoperation, compared to those undergoing reoperation within 24 h of developing symptoms.

Investigational and non-traditional techniques, such as use of fibrin sealant application and endoscopic interventions, have also been considered in the treatment of enteric leaks. Victorzon and colleagues reported on their series of 645 consecutive LRYGB procedures [31]. Six patients (0.93 %) developed a leak at the gastrojejunostomy; four occurred in primary LRYGB, while two occurred in revisional LRYGB. Four patients underwent endoscopic fibrin sealant injection; three of these patients required repeated injections. The patient undergoing only one fibrin sealant injection had a self-expandable metal stent placed. Time to closure of the enteric leak was 21 days or less in all patients who underwent endoscopic fibrin sealant injection. In another large series of 836 patients who underwent primary LRYGB, eight patients (0.95 %) developed a leak [32]. Five patients were treated nonoperatively, three of which underwent endoscopic fibrin sealant injection. Two of these three were successful.

6.5.1 Chronic Leaks

Chronic leaks represent a difficult problem to manage for the bariatric surgeon. Chronic leaks can be defined as those persisting or occurring >30 days after the index procedure despite optimal surgical and medical management. Currently, there are no specific guidelines regarding management and treatment. Most management strategies focus on maintaining control of the leak; minimizing peritoneal contamination; and optimizing nutrition to heal. Some have advocated the use of stents in this case. In this method, the stent may serve to prevent or greatly diminish further peritoneal contamination. Avoiding further chemical and bacterial peritonitis can optimize potential for recovery and promote healing. Moreover, use of a stent may allow for enteral or oral nutrition. Stent migration remains an issue, as it has been reported to range between 17 and 58 % [33, 34].

The data regarding the use of stents to manage chronic leaks and strictures following bariatric surgery have been conflicting. In a series of 21 patients at the Mayo clinic presenting with either chronic strictures or chronic anastomotic leaks after bariatric surgery (sleeve gastrectomy, RYGB, or biliopancreatic diversion/duodenal switch), patients were treated self-expanding metal stents [35]. Stent placement was successful in 4 patients (19 %). Those patients with endoscopic success included two patients with chronic anastomotic strictures following RYGB, one patient with an esophagopleural fistula, and one patient with a leak from a sleeve gastrectomy. Three patients died and the remaining 14 patients were treated successfully by reoperation. On the other hand, Eubanks et al. reported on a series of 19 patients who underwent stent placement following postoperative leaks [11], fistulas [2], or strictures [6, 33]. Successful closure was accomplished in 16 patients (84 %). However in this series, the stent migration rate was 58 %. Puli and colleagues reported on a meta-analysis of 67 patients with chronic anastomotic or staple line leaks treated by self-expandable metal stents [34]. The pooled proportion of successful closure was 88 % and stent retrieval was successful in 92 % of cases.

6.5.2 Jejunojejunostomy Leaks

Jejunojejunostomy leaks are significantly more devastating than gastrojejunal leaks. Part of the issue with these distal leaks is the delay in diagnosis. Typically, patients with these leaks do not immediately manifest until after peritonitis has developed and patients are clinically unstable. Moreover, screening radiographic tests are not routinely performed to assess for these leaks. Thus, an early diagnosis can only be made on a clinical basis unless a CT scan is obtained based on a high index of suspicion. Treatment of these

leaks always requires surgical intervention. Lee and colleagues reported on a series of 3828 gastric bypasses [36]. The overall jejunojejunostomy leak rate was 0.5 %, compared to 2.7 % for gastrojejunal leaks. Ten patients had UGIS performed, 9 were read as normal. The median time to detection from surgery of the jejunojejunostomy leak, compared to a gastrojejunal leak, was significantly longer (4 vs. 2 days, respectively; $p=0.037$). The mortality from a jejunojejunostomy leak was 40 %, which was significantly higher than gastrojejunal leaks (9 %; $p=0.005$). The false negative rate of UGIS in detecting jejunojejunostomy leaks may contribute to the higher mortality rates.

6.6 Conclusion

Enteric leaks following RYGB are one of the most devastating complications, associated with significant morbidity and one of the most common causes of mortality. Prompt diagnosis and treatment is integral in optimizing outcomes. Technical consideration and buttressing materials may aid in preventing this complication. Fibrin sealant has also been used with some anecdotal success. Management is predominantly surgical, with closure of the defect, debridement, and placement of a gastrostomy tube in the remnant stomach as the three main principles. Endoscopic and percutaneous treatments are largely adjunctive, but may play an important role in well-contained leaks in patients without hemodynamic instability. Prevention of this complication is of utmost significance to ensure optimal patient outcomes.

References

1. Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. JAMA. 2004;292(14):1724–37.
2. Sjostrom L, Narbro K, Sjostrom CD, et al. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med. 2007;357(8):741–52.
3. Mingrone G, Panunzi S, De Gaetano A, et al. Bariatric surgery versus conventional medical therapy for type 2 diabetes. N Engl J Med. 2012;366(17):1577–85.
4. Afaneh C, Giambrone G, Eskreis-Winkler J, et al. A three-state analysis of bariatric procedures: trends and outcomes. Gastroenterology. 2014;146(5):S1095–6.
5. Gonzalez R, Sarr MG, Smith CD, et al. Diagnosis and contemporary management of anastomotic leaks after gastric bypass for obesity. J Am Coll Surg. 2007;204(1):47–55.
6. Fernandez Jr AZ, DeMaria EJ, Tichansky DS, et al. Experience with over 3,000 open and laparoscopic bariatric procedures: multivariate analysis of factors related to leak and resultant mortality. Surg Endosc. 2004;18(2):193–7.
7. Fullum TM, Aluka KJ, Turner PL. Decreasing anastomotic and staple line leaks after laparoscopic Roux-en-Y gastric bypass. Surg Endosc. 2009;23(6):1403–8.
8. Hamilton EC, Sims TL, Hamilton TT, et al. Clinical predictors of leak after laparoscopic Roux-en-Y gastric bypass for morbid obesity. Surg Endosc. 2003;17(5):679–84.
9. Al-Sabah S, Ladouceur M, Christou N. Anastomotic leaks after bariatric surgery: it is the host response that matters. Surg Obes Relat Dis. 2008;4(2):152–8.
10. Gonzalez R, Nelson LG, Gallagher SF, Murr MM. Anastomotic leaks after laparoscopic gastric bypass. Obes Surg. 2004;14(10):1299–307.
11. Livingston EH, Ko CY. Assessing the relative contribution of individual risk factors on surgical outcome for gastric bypass surgery: a baseline probability analysis. J Surg Res. 2002;105(1):48–52.
12. Gonzalez R, Bowers SP, Venkatesh KR, Lin E, Smith CD. Preoperative factors predictive of complication postoperative management after Roux-en-Y gastric bypass for morbid obesity. Surg Endosc. 2003;17(12):1900–4.
13. Nguyen NT, Rivers R, Wolfe BM. Factors associated with operative outcomes in laparoscopic gastric bypass. J Am Coll Surg. 2003;197(4):548–57.
14. Quartararo G, Facchiano E, Scaringi S, Liscia G, Lucchese M. Upper gastrointestinal series after Roux-en-Y gastric bypass for morbid obesity: effectiveness in leakage detection. A systematic review of the literature. Obes Surg. 2014;24(7):1096–101.
15. Haddad A, Tapazoglou N, Singh K, Averbach A. Role of intraoperative esophagogastroenteroscopy in minimizing gastrojejunostomy-related morbidity: experience with 2,311 laparoscopic gastric bypasses with linear stapler anastomosis. Obes Surg. 2012;22(12):1928–33.
16. Sekhar N, Torquati A, Lutfi R, Richards WO. Endoscopic evaluation of the gastrojejunostomy in laparoscopic gastric bypass. A series of 340 patients without a postoperative leak. Surg Endosc. 2006;20(2):199–201.
17. Gonzalez R, Lin E, Venkatesh KR, Bowers SP, Smith CD. Gastrojejunostomy during laparoscopic gastric bypass: analysis of 3 techniques. Arch Surg. 2003;138(2):181–4.
18. Bendewald FP, Choi JN, Blythe LS, Selzer DJ, Ditslear JH, Mattar SG. Comparison of hand-sewn, linear-stapled, and circular-stapled gastrojejunostomy

in laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2011;21(11):1671–5.
19. Giordano S, Salminen P, Biancari F, Victorzon M. Linear stapler technique may be safer than circular in gastrojejunal anastomosis for laparoscopic Roux-en-Y gastric bypass: a meta-analysis of comparative studies. Obes Surg. 2011;21(12):1958–64.
20. Hazelrigg SR, Boley TM, Naunheim KS, et al. Effect of bovine pericardial strips on air leak after stapled pulmonary resection. Ann Thorac Surg. 1997;63(6):1573–5.
21. Angrisani L, Lorenzo M, Borrelli V, Ciannella M, Bassi UA, Scarano P. The use of bovine pericardial strips on linear stapler to reduce extraluminal bleeding during laparoscopic gastric bypass: prospective randomized clinical trial. Obes Surg. 2004;14(9):1198–202.
22. Miller KA, Pump A. Use of bioabsorbable staple reinforcement material in gastric bypass: a prospective randomized clinical trial. Surg Obes Relat Dis. 2007;3(4):417–22.
23. Jones WB, Myers KM, Traxler LB, Bour ES. Clinical results using bioabsorbable staple line reinforcement for circular staplers. Am Surg. 2008;74(6):462–7.
24. Ibele A, Garren M, Gould J. Effect of circular staple line buttressing material on gastrojejunostomy failure in laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2010;6(1):64–7.
25. Sapala JA, Wood MH, Schuhknecht MP. Anastomotic leak prophylaxis using a vapor-heated fibrin sealant: report on 738 gastric bypass patients. Obes Surg. 2004;14(1):35–42.
26. Liu CD, Glantz GJ, Livingston EH. Fibrin glue as a sealant for high-risk anastomosis in surgery for morbid obesity. Obes Surg. 2003;13(1):45–8.
27. Silecchia G, Boru CE, Mouiel J, et al. The use of fibrin sealant to prevent major complications following laparoscopic gastric bypass: results of a multicenter, randomized trial. Surg Endosc. 2008;22(11):2492–7.
28. Csendes A, Burgos AM, Braghetto I. Classification and management of leaks after gastric bypass for patients with morbid obesity: a prospective study of 60 patients. Obes Surg. 2012;22(6):855–62.
29. Durak E, Inabnet WB, Schrope B, et al. Incidence and management of enteric leaks after gastric bypass for morbid obesity for a 10-year period. Surg Obes Relat Dis. 2008;4(3):389–93.
30. Jacobsen HJ, Nergard BJ, Leifsson BG, et al. Management of suspected anastomotic leak after bariatric laparoscopic Roux-en-Y gastric bypass. Br J Surg. 2014;101(4):417–23.
31. Victorzon M, Victorzon S, Peromaa-Haavisto P. Fibrin glue and stents in the treatment of gastrojejunal leaks after laparoscopic gastric bypass: a case series and review of the literature. Obes Surg. 2013;12(10):1692–7.
32. Brolin RE, Lin JM. Treatment of gastric leaks after Roux-en-Y gastric bypass: a paradigm shift. Surg Obes Relat Dis. 2013;9(2):229–33.
33. Eubanks S, Edwards CA, Fearing NM, et al. Use of endoscopic stents to treat anastomotic complications after bariatric surgery. J Am Coll Surg. 2008;206(5):935–8.
34. Puli SR, Spofford IS, Thompson CC. Use of self-expandable stents in the treatment of bariatric surgery leaks: a systematic review and meta-analysis. Gastrointest Endosc. 2012;75(2):287–93.
35. Puig CA, Waked TM, Baron Sr TH, Wong Kee Song LM, Gutierrez J, Sarr MG. The role of endoscopic stents in the management of chronic anastomotic and staple line leaks and chronic strictures after bariatric surgery. Surg Obes Relat Dis. 2014;10(4):613–7.
36. Lee S, Carmody B, Wolfe L, et al. Effect of location and speed of diagnosis on anastomotic leak outcomes in 3828 gastric bypass cases. J Gastrointest Surg. 2007;11(6):708–13.

7 Enteric Leaks After Sleeve Gastrectomy: Prevention and Management

Monica Sethi and Manish Parikh

Key Points

- Leaks after LSG are rare (2–3 %), but may cause significant morbidity.
- Utilizing bougie ≥40 Fr may decrease leak rate without affecting overall weight loss up to 36 months postoperatively.
- For patients who develop leak after LSG, nutritional support and source control are cornerstones of management, including laparoscopic drainage and washout and feeding jejunostomy tube, if necessary.
- Most leaks resolve with endoscopic stenting.
- In rare cases, surgery (resection with Roux-en-Y esophagojejunostomy or placement of Roux limb to the fistula) is required for definitive management.

7.1 Introduction

Laparoscopic sleeve gastrectomy (LSG) involves a stapled vertical transection of the stomach and creation of a tubular alimentary channel along the stomach's lesser curvature, calibrated along an orogastric bougie (Fig. 7.1). Perhaps one of the most significant changes in bariatric surgery over the past decade is the growing popularity of the LSG. For instance, the University HealthSystem Consortium data reveals an increase in LSG from 0.9 % in 2008 to 36.5 % in 2012 [1]. Reasons for this increase include:

- Short-term weight loss comparable to that of the gastric bypass (60–70 % excess weight loss by 3 years)
- Improvement in insurance coverage for the LSG
- Favorable complication profile compared to the gastsric bypass
- Less required postoperative follow-up compared to gastric banding

Surgeons experienced with LSG report that the most common complications include leak, hemorrhage, stenosis, spleen/liver injury, portal vein thrombosis, and reflux [2]. This chapter focuses on leak after LSG, with a particular focus on prevention and management.

7.2 Presentation and Diagnosis

7.2.1 Incidence and Presentation

The rate of staple-line leaks after LSG varies in the literature, but is generally between 1.1 and 5.3 % of cases [3]. A systematic review of 9991 LSG

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_7

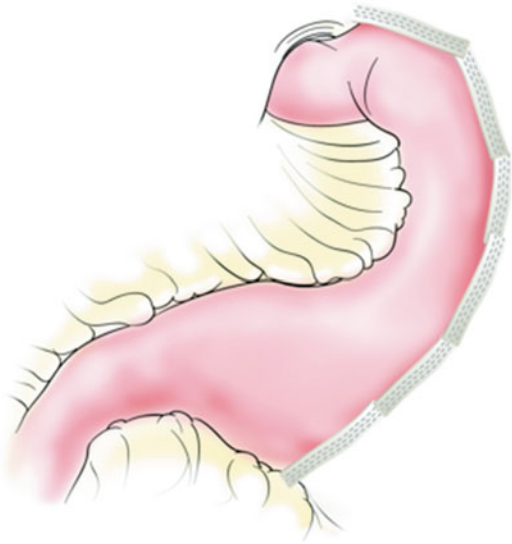

Fig. 7.1 Laparoscopic sleeve gastrectomy. Reprinted with permission from Parikh M, Gagner M, Pomp A. Laparoscopic Duodenal Switch. In: Nguyen NT, De Maria EJ, Ikramuddin S, Hutter MM. eds. The SAGES Manual: a Practical Guide to Bariatric Surgery. Springer, New York, 2008;109–129 [38] © Springer

reported a leak rate of 2.2 % [4]. The mortality rate from leaks after LSG is 0.11 % [4]. The vast majority of leaks (75–89 %) occur proximally, near the gastroesophageal junction [5].

Leaks present at a mean of 7 days postoperatively, but can present as late as 120 days postoperatively [3]. The majority of leaks present after patients are discharged home from the hospital; therefore close follow-up in the immediate postoperative period is critical after LSG. Rosenthal et al. proposed a classification system for leak after LSG based on timing: acute leak (within 7 days postoperatively), early leak (within 1–5 weeks postoperatively), late leak (greater than 6 weeks postoperatively), and chronic leak (after 12 weeks) [6].

Staple-line leak after LSG can present with many clinical scenarios, ranging from a stable patient with mild abdominal pain to a patient with manifestations of systemic inflammatory response syndrome (SIRS—see Table 7.1) to a patient with sepsis and multiorgan failure. A high index of suspicion is important, as early intervention is the key to successful management of these patients [7].

Table 7.1 SIRS criteria based on Metabolic and Bariatric Surgery Accreditation and Quality Improvement Program (MBSAQIP)

SIRS criteria
Presence of two of the following:
• Temperature >100.4 ° F or <96.9 ° F • WBC > 12,000 or <4000 or >10 % bands • HR >90 bpm • RR >20, PaCO2 < 32 • Gap acidosis

SIRS = Systemic inflammatory response syndrome; WBC = white blood count; HR = heart rate; RR = respiratory rate

7.2.2 Diagnostic Study

Abdominal computed tomography (CT) scan with oral and intravenous contrast is the diagnostic study of choice for most patients suspected of having leak. CT findings may range from blips of extraluminal air to frank contrast extravasation (Figs. 7.2 and 7.3). Esophagrams may also be used to diagnose leak; however it may be normal despite the presence of leak.

Since leaks often present after patient discharge from the hospital, the value of immediate postoperative upper GI studies has been debated. Studies have demonstrated the lack of association between routine postoperative swallow study and leak [8]. Similarly, intraoperative leak tests fail to detect leak, unless due to a stapler misfire or other

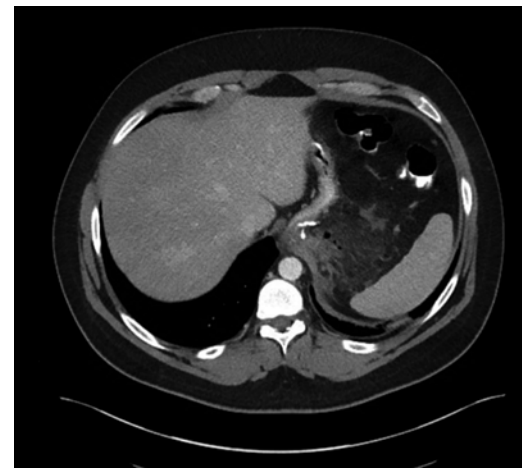

Fig. 7.2 Blips of air around staple line in patient POD#9 after LSG. This resolved with intravenous antibiotics

Fig. 7.3 CT scan POD#8 showing extraluminal fluid collection consistent with leak

technical error. A normal intraoperative leak test and a normal postoperative swallow study do not preclude the development of staple-line leak after LSG. Despite this, many surgeons still favor these tests and perform them routinely.

Some surgeons also advocate for routine drain placement after LSG. However, this has fallen out of favor as leaks present nearly a week after LSG, and leaving a drain in for this duration is unnecessary in a vast majority of LSG cases. In 2013, 39 % of surgeons left a drain in the abdominal cavity after LSG, and this number continues to decline [2]. If a drain is left in place, however, postoperative leak test with methylene blue may be effective in diagnosing leak. Some surgeons have also used this method during follow-up to monitor the progress of the fistula [9, 10].

7.3 Prevention of Leak After LSG

Leak after LSG can occur for a variety of reasons. Possible factors include patient-level factors that predispose to leak. Other factors may be related to the technical aspects of LSG construction, inadequate oxygenation with subsequent ischemia, or thermal injury [11].

7.3.1 Patient Characteristics

Certain patient factors may be associated with increased leak rate. Benedix et al. retrospectively reviewed 103 leaks in 5400 LSG cases (1.9 %) performed over a 6-year period in order to identify factors that increase the risk of leak [12]. They found that higher body mass index (BMI), male gender, presence of sleep apnea, conversion to laparotomy, longer operative time, year of procedure, and intraoperative complications significantly increased leak rate. On multivariate analysis, however, only operative time and year of procedure maintained a significant association with leak.

Superobese patients (BMI >50 kg/m^2) may have a higher incidence of leak, as is the case in gastric bypass. A systematic review of 4888 LSG found the leak rate to be 2.9 % among the superobese versus 2.2 % in those with a preoperative BMI <50 kg/m^2, but this was not statistically significant [5]. Another study found type 2 diabetes to be an independent risk factor for the development of leak ($p<0.01$) [13].

Sakran et al. found an association between previous bariatric surgery and increased likelihood of leak ($p<0.005$). Leaks developed in 44 out of 2834 LSG (1.5 %). Eleven patients (25 %) had a

prior silastic ring vertical gastroplasty or LAGB, versus 10 % of non-leaks, implying a threefold increased risk of leak in patients with previous bariatric surgery [3].

7.3.2 Technical Factors

In a retrospective review of 529 cases with 0 % leak rate, Bellanger et al. discussed the technical principles for decreasing enteric leakage after LSG [14]. A key point mentioned is to position the tip of the stapler to give a distance of one and a half times the width of the bougie at the area of the incisura angularis (Fig. 7.4). Other technical principles included positioning the stapler to leave 1 cm of gastric tissue lateral to the angle of HIS to avoid stapling too close to the esophagus in the area of the cardia (Fig. 7.5), allowing adequate compression of the gastric tissue with the stapling device, and thorough visual inspection of the staple line after procedure completion [14].

Sakran et al. proposed that heat-producing instruments may cause thermal injury to the sleeve, leading to leak. Additionally, aggressive dissection near the posterior aspect of the upper sleeve may cause devascularization, increasing susceptibility to leak. They propose that dissection in this area should be kept to a minimum and the final staple fire should be directed away from the esophagus and to the left of the gastroesophageal junction [3].

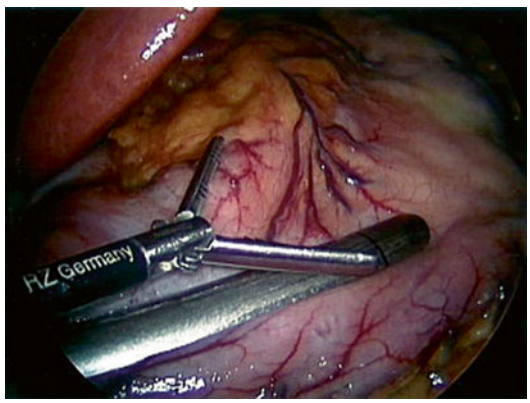

Fig. 7.4 First application of stapler one and a half times the distance from the bougie. Reprinted with permission from Bellanger et al. Laparoscopic sleeve gastrectomy, 529 cases without a leak: short-term results and technical considerations. Obesity Surgery 2011;21:146–50 [14] © Springer

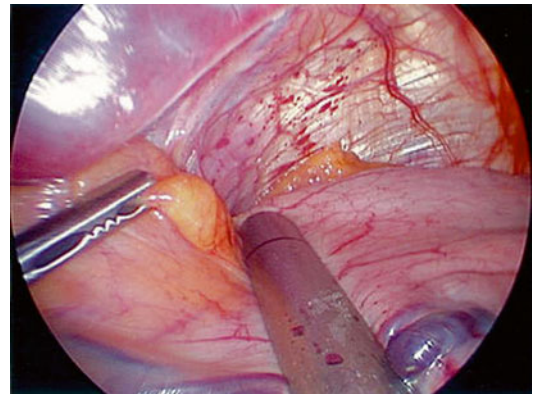

Fig. 7.5 Application of stapler lateral to periesophageal fat pad. Reprinted with permission from Bellanger et al. Laparoscopic sleeve gastrectomy, 529 cases without a leak: short-term results and technical considerations. Obesity Surgery 2011;21:146–50 [14] © Springer

7.3.3 Systematic Review and Meta-Analysis of Factors That Contribute to Leak (Table 7.2)

Technical aspects of LSG, including bougie size used to calibrate the sleeve, distance from the pylorus where the stapling begins, height of stapler used to transect the stomach, and the role of buttressing material on the staple line, may affect leak rate. Debate exists whether the creation of tighter (i.e., smaller) sleeves results in higher leak rate (Fig. 7.6) [15].

In a meta-analysis of 9991 LSG, various technical aspects of performing LSG were analyzed [4]. Bougie size was <40 Fr in the majority (69 %) of patients, LSG transection began ≥5 cm from the pylorus in 68 % of patients, and some form of buttressing was used in 82 % (Fig. 7.7). All leaks were analyzed based on bougie size, the distance from the pylorus, the use of buttressing, and the type of buttressing (Fig. 7.8).

Due to the fact that there are multiple factors that may contribute to leak, a general estimating equation (GEE) model was then created utilizing the variables of bougie size (<40 Fr, 40–49 Fr, ≥50 Fr), distance from the pylorus (<5 cm, ≥5 cm), and the use of buttressing (bioabsorbable,

Table 7.2 GEE (general estimating equation) model adjusting for the effect of bougie size, distance from pylorus, and the use of buttressing or sutures on leak rate while controlling for age and BMI

	Unadjusted			Adjusted		
	OR	95 % CI	*p*-Value	OR	95 % CI	*p*-Value
Bougie size						
<40 Fr (reference)	–			–		
40–49 Fr	0.69	[0.41, 1.16]	0.161	0.53	[0.37, 0.77]	0.0009
≥50 Fr	0.37	[0.18, 0.73]	0.0041	0.40	[0.15, 1.07]	0.068
Distance to pylorus						
<5 cm (reference)	–			–		
≥5 cm	1.16	[0.60, 2.25]	0.659	1.30	[0.81, 2.09]	0.279
Use of buttressing/sutures						
Bioabsorbable (reference)	–			–		
No buttressing, no sutures	1.00	[0.37, 2.69]	0.997	1.06	[0.49, 2.30]	0.873
Non-absorbable buttressing	1.78	[1.17, 2.72]	0.0075	2.01	[0.87, 4.68]	0.104
No buttressing, sutures only	1.95	[1.25, 3.02]	0.0031	2.87	[1.21, 6.84]	0.017
Age						
Mean age < 40	–					
Mean age 40–44	0.78	[0.51, 1.19]	0.250	0.83	[0.54, 1.27]	0.392
Mean age 45+	0.51	[0.27, 0.98]	0.044	0.57	[0.31, 1.03]	0.061
BMI						
Mean BMI < 45	–					
Mean BMI 45–49	1.82	[0.99, 3.32]	0.052	1.81	[1.21, 2.71]	0.0041
Mean BMI 50+	1.44	[0.73, 2.84]	0.296	1.96	[1.16, 3.34]	0.012

(OR = odds ratio; CI = confidence interval)

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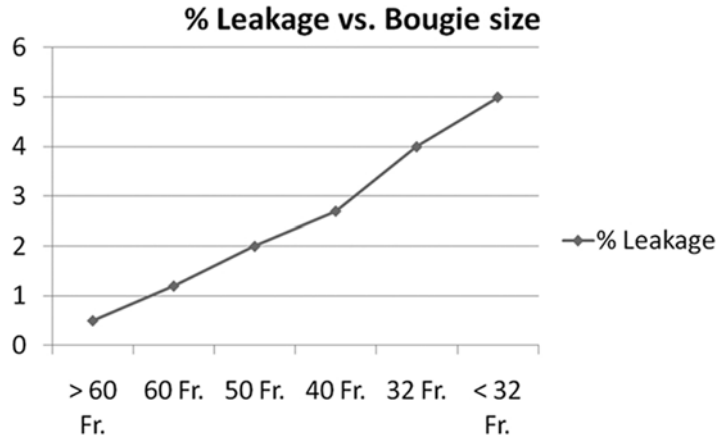

Fig. 7.6 Percentage of leakage versus bougie size. On the *x*-axis, a bougie size in French and on the *y*-axis leakage rate in percentage. Reprinted with permission, Gagner M. Leaks after sleeve gastrectomy are associated with smaller bougies. Prevention and treatment strategies. Surg Laparoscopic Endosc Percutan Tech 2010;20:166–169 [15] © Wolters Kluwer Health

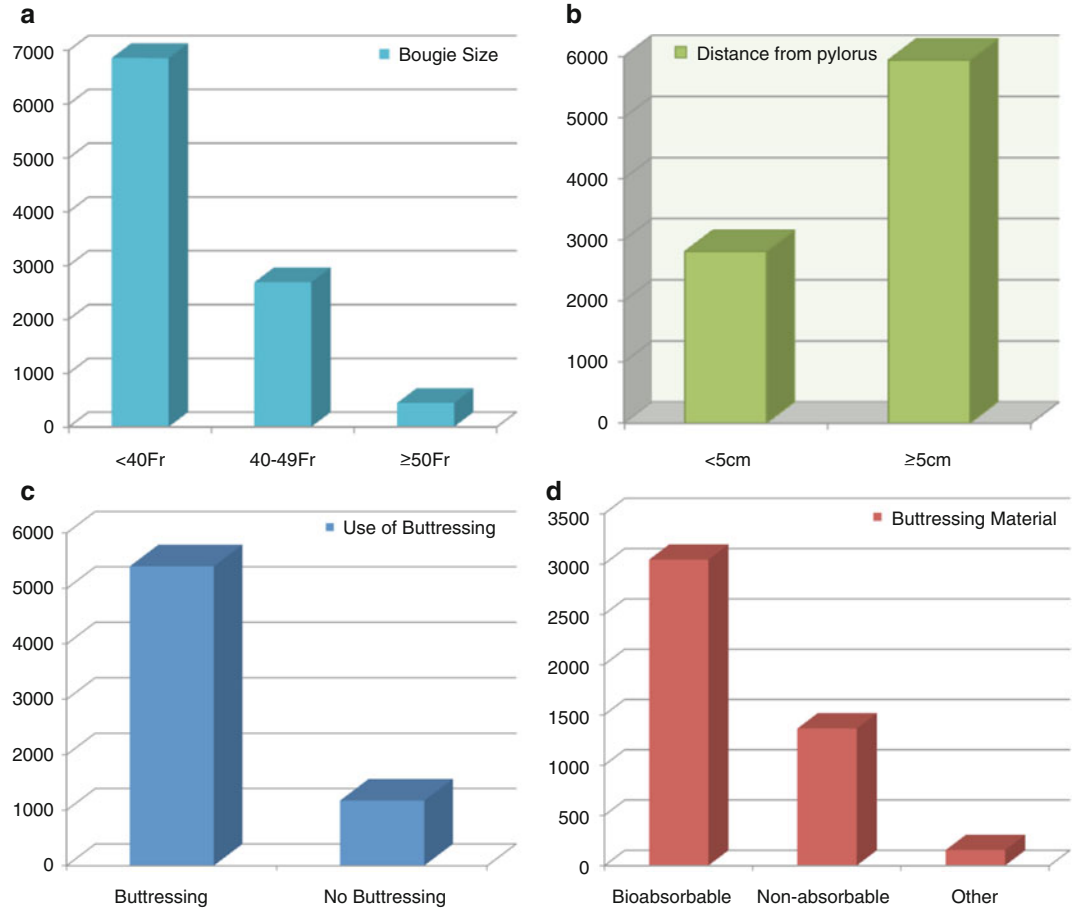

Fig. 7.7 Most common techniques used for LSG. Reprinted with permission from Parikh M, Issa R, McCrillis A, et al. Surgical strategies that may decrease leak after laparoscopic sleeve gastrectomy. Ann Surg 2013;257:231–237 [4] © Wolters Kluwer Health

non-absorbable, other, none) while controlling for age and BMI. The GEE model revealed that the risk of leak after LSG decreased by using a bougie ≥40 Fr (OR 0.53 [0.37–0.77], $p=0.0009$; see Table 7.2). Distance from pylorus did not impact leak rate ($p=0.279$). The use of bioabsorbable buttressing did not impact leak rate ($p=0.104$). However suturing alone (without buttressing) increased leak (OR 2.87 [1.21–6.84], $p=0.017$). BMI>50 also increased leak rate (OR 1.96 [1.16–3.34], $p=0.012$). A linear repeated measures regression model was used to compare weight loss between bougie size <40 Fr and bougie size ≥40 Fr and found no difference in weight loss up to 3 years (70.1 % mean EWL; $p=0.273$) (Fig. 7.9). Based on this study, one of the most important technical factors that may decrease leak is utilizing bougie ≥40 Fr.

The vast majority of surgeons utilize reinforcement when performing LSG [2, 4]. Reinforcement options include buttressing material (absorbable and non-absorbable) as well as oversewing. Oversewing techniques include a running baseball-type stitch throughout the staple line and invagination of the staple line. Buttressing has been shown to decrease bleeding along the staple line [16].

However the impact of buttressing on leak rate is controversial. The meta-analysis by Parikh (9991 LSG) did not show decreased leak with buttressing [3]. Another systematic review (4881 LSG) also failed to show a difference [17]. On the other hand, one retrospective multicenter

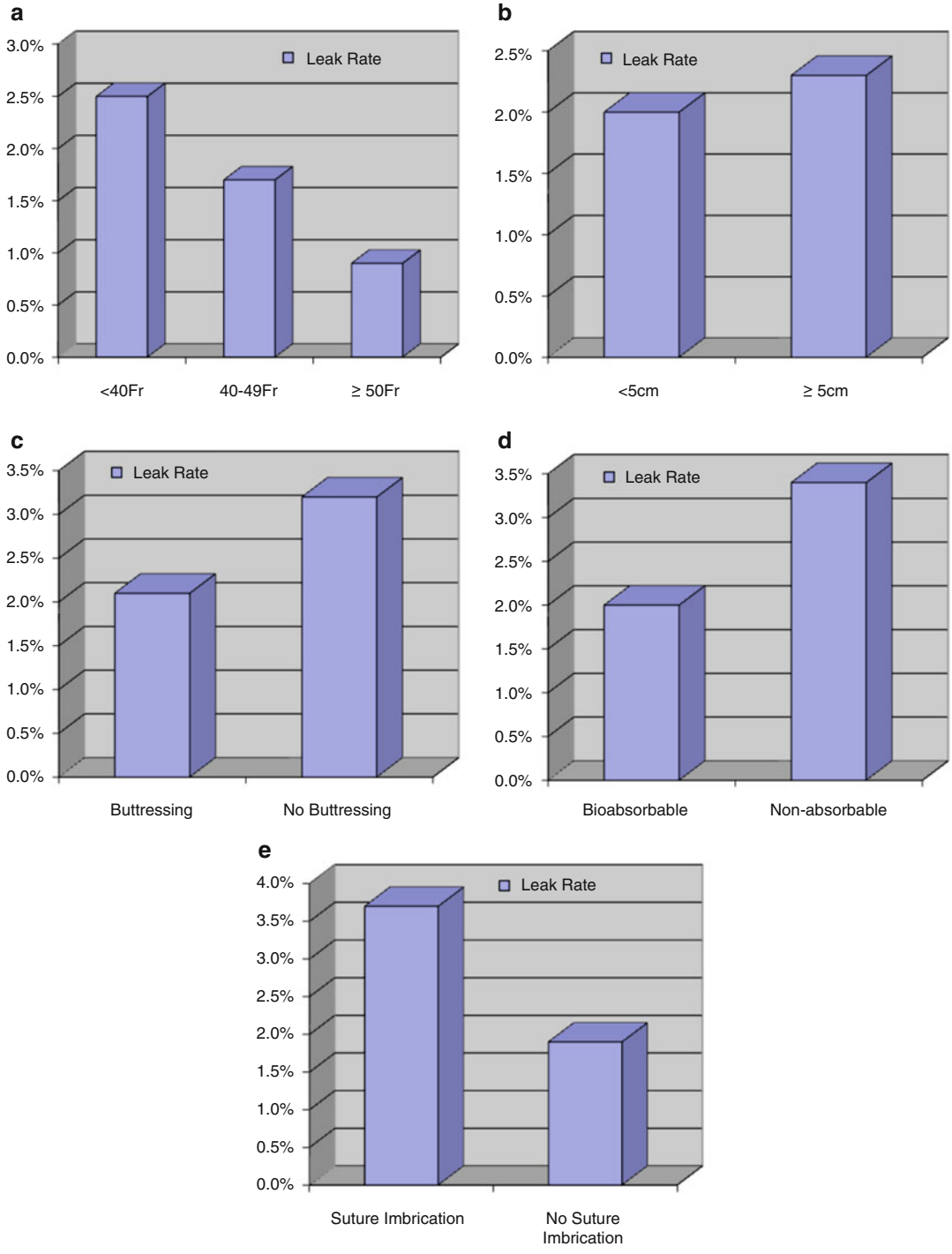

Fig. 7.8 Effect of technique on leak rate. Reprinted with permission from Parikh M, Issa R, McCrillis A, et al. Surgical strategies that may decrease leak after laparoscopic sleeve gastrectomy. Ann Surg 2013;257: 231–237 [4] © Wolters Kluwer Health

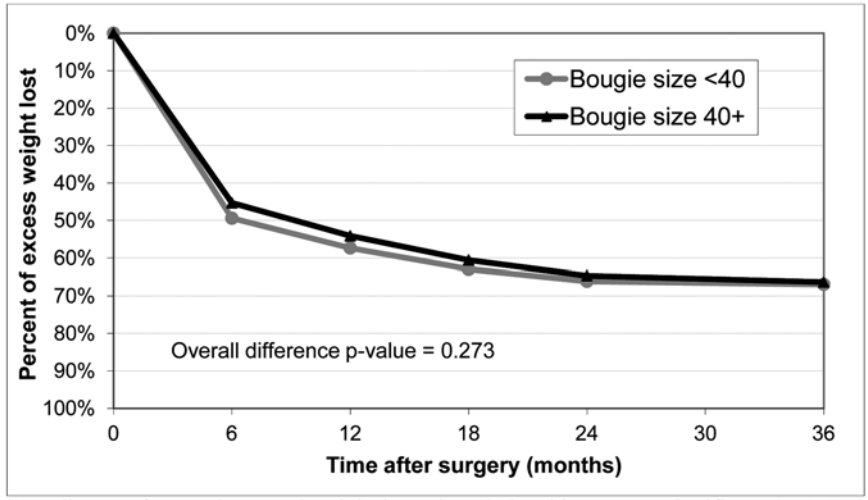

Fig. 7.9 Linear repeated measures regression model comparing weight loss between bougie size < 40 Fr and ≥ 40 Fr. Reprinted with permission from Parikh M, Issa R, McCrillis A, et al. Surgical strategies that may decrease leak after laparoscopic sleeve gastrectomy. Ann Surg 2013;257:231–237 [4] © Wolters Kluwer Health

study that analyzed multiple types of staple-line reinforcement in 1162 LSG found a significantly decreased leak rate among LSG reinforced with bovine pericardium relative to other types of staple-line reinforcement and no reinforcement (0.3 % vs. 2.8 %, $p < 0.01$) [13].

A more recent systematic review was performed by Gagner et al. comparing no reinforcement, oversewing, nonabsorbable bovine pericardial strips, and absorbable polymer membrane (APM) staple-line reinforcement [18]. Leak rates ranged from 1.09 % in the APM group to 3.3 % in the bovine pericardium group, with APM having a significantly lower leak rate than other groups ($p < 0.05$). However, this review did not control for other technical factors such as bougie size.

7.3.4 Other Factors

Some authors propose that early gastric decompression for at least 24 h postoperatively may decrease intragastric pressure and therefore prevent leak. In a prospective randomized study on gastric decompression with a nasogastric tube, there was no difference in leak rate between the groups [19]. However this study was likely underpowered with only 75 patients per treatment group.

7.4 Management of Leak After LSG

The approach to managing LSG leak has evolved as surgeons gain more experience with leak. Early intervention is the key to successful management of these patients. Treatment options depend on the clinical scenario and range from intravenous antibiotics and nutritional support to endoscopic interventions including stenting to surgical interventions including gastrectomy with Roux-en-Y esophagojejunostomy or fistula-jejunostomy. Sepsis control and nutritional support are cornerstones of management, but specific treatments should be based on a patient's clinical presentation and timing of the leak [20]. With the evolution of endoscopic stents to treat leaks, the majority of leaks may be treated without definitive surgery [21].

We favor a treatment algorithm based on the presence of SIRS. Generally, patients with SIRS (Table 7.2) or peritonitis benefit from immediate reoperation with laparoscopic washout, and placement of a large-bore drain (e.g., 19 Fr Blake), with or without placement of a feeding jejunostomy. Stable patients without systemic illness can be treated non-operatively, with percutaneous image-guided drainage, antibiotics, and parenteral hyperalimentation. After drainage, we routinely utilize upper GI series to demonstrate the anatomy of the

leak. Then, endoscopic stenting is the treatment of choice to manage the leak.

7.4.1 Endoscopic Intervention

Endoscopic stent placement was originally utilized in the management of anastomotic leak after esophagectomy, and has been adapted to treat enteric leak after LSG. The stent provides a temporary seal of the leak while also allowing oral intake during the process of healing. Stents may also aid in the correction of the sleeve axis in cases of gastric torsion or twist [19]. Generally, stents should be placed in hemodynamically stable patients after any intraabdominal collection has been drained by either laparoscopy or percutaneous CT-guidance (Fig. 7.10).

The use of endoscopic stents to treat LSG leaks is well established in the literature; however, most of the studies on this topic suffer from small sample sizes. Additionally, the lack of standardized stent timing and treatment limits meaningful comparison between studies. Nonetheless, current data suggest that stents are safe and effective in treating proximal leaks after LSG.

In a recent retrospective study, 17 LSG patients with leak underwent endoscopic stenting with self-expandable metal stents [22]. The median duration of stent placement was 42 days, and stenting was successful in treating 13 (76 %) leaks. This study also found that shorter duration between LSG and time of stent placement was associated with improved outcomes. In a similar study, Simon et al. used self-expanding metal stents to treat patients with enteric leaks after LSG, with a mean stent duration of 6.4 weeks and a 78 % success rate [23]. The authors of this study advocate for early (<3 weeks) stent placement as it decreases healing time. There is little consensus on the ideal size and type of stent in treating leak after LSG or the duration of the stent, but most authors recommend a period of 6–8 weeks prior to stent removal.

Another study with six patients with leaks stented with Hanarostent demonstrated an 84 % success rate [24]. In contrast, Tan et al. reported eight cases of endoscopic stenting for leak after LSG, with only a 50 % success rate due to stent-related complications [9]. Complications included stent migration, hematemesis, and gastric obstruction from kinking at the proximal aspect of the stent. Other possible causes of stent failure include erosion, as well as patient intolerance with nausea, vomiting, drooling, early satiety, retrosternal discomfort, and exacerbation of reflux symptoms. Table 7.3 summarizes the current literature regarding endoscopic stents and leaks after LSG.

Additional endoscopic methods have been reported in treating leak. Some have reported

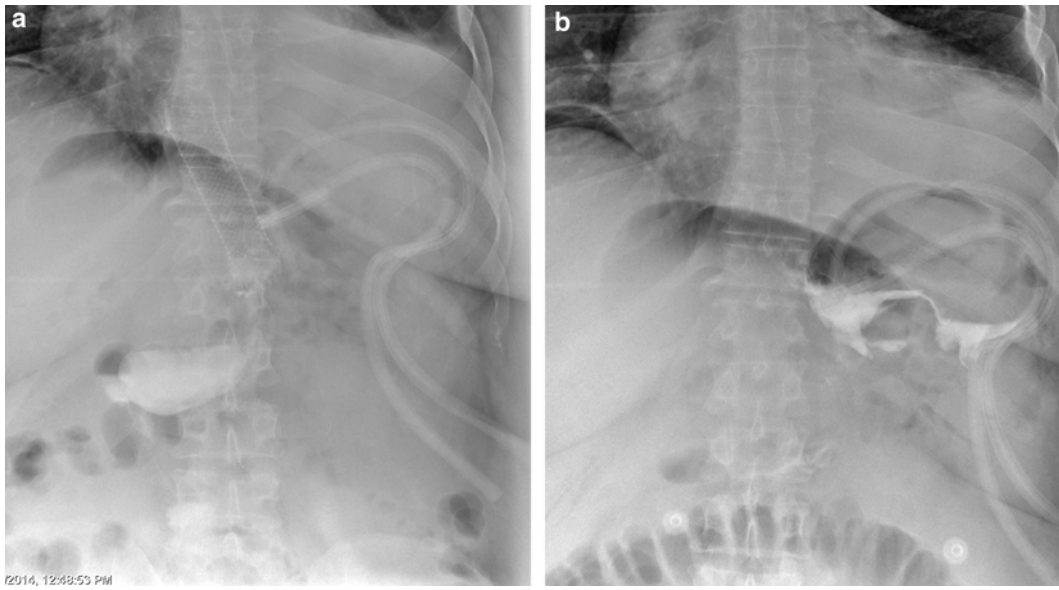

Fig. 7.10 Example of LSG leak treated with stent

using endoscopic internal drainage with pigtail stents [28]. In one study, three stents on average were placed in each of 21 patients, with a 95 % success rate at a mean of 55.5 days postoperatively [29]. Overall, pigtail stents were found to require fewer procedures per patient, were better tolerated, and had lower morbidity-mortality than self-expanding metal stents.

Another study reported successful management of late (16 months post-op) LSG leak with a 10 mm over-the-scope metallic clip [30]. In another case, a patient with a leak refractory to multiple attempts at endoscopic stenting and drainage was successfully treated endoscopically by placing a vascular plug in the fistula and stenting over the plug [31]. Lastly, Oshiro et al. reported on their success with percutaneous transesophageal gastro-tubing (PTEG) in treating two patients with refractory leak [32].

While imperfect, of all the endoscopic treatment options available, stents are most commonly used and have been associated with the most success.

7.4.2 Surgical Interventions

Surgical management of leak after LSG has two main indications:

- Source control in a systemically ill or septic patient.
- Salvage treatment in chronic or refractory leaks that have failed endoscopic management.

Patients exhibiting SIRS or overt signs of sepsis benefit from laparoscopic drainage of the contaminated peritoneal fluid. A well-placed large-bore surgical drain along the staple line also helps maintain source control. We have found in our experience that surgical drainage/washout leads to quicker resolution of SIRS than percutaneous drainage or intravenous antibiotics alone. A feeding jejunostomy tube can also be placed at this time. Usually the leaks present too late to directly repair the defect. Another well-described surgical option is to place a t-tube into the defect to help establish drainage [33].

Surgery has also been described for successful management of chronic leak (>12 weeks). Roux-en-Y reconstruction with resection of the leak site is the most common treatment option in proximal chronic leaks, because it resects the pathology and converts the high-pressure system with distal obstruction of a gastric sleeve to the lower pressure system of a Roux-en-Y gastric bypass [34, 35]. A more recently described option that avoids resection is placement of a Roux limb to the defect to avoid gastrectomy and its attendant complications. This is done with a one-layer anastomosis utilizing a running absorbable monofilament suture (Fig. 7.11a–c) [36]. Chour et al. propose this technique at an early stage to prevent chronic morbidity and increased hospitalization associated with chronic leak [37]. However there was a small leak reported in 3/6 (50 %) patients. Most surgeons advise waiting at least 12 weeks before definitive surgical management to avoid dense adhesions [2]. Even in these scenarios, surgery for definitive treatment of LSG leak can have substantial morbidity [27].

7.4.3 Algorithm (Fig. 7.12)

We recommend a treatment algorithm based on the clinical presentation of the patient, specifically the presence of SIRS. Patients suspected of having a leak should undergo abdominal imaging via CT with IV and PO contrast. If there is radiographic evidence of leak, the patient should be assessed for SIRS (Table 7.1). If SIRS is present, we recommend surgical drainage and consideration of placement of a feeding jejunostomy tube. Primary repair is attempted only in the immediate post-op period (<48 h). If the patient does not have SIRS, image-guided percutaneous drainage should be used to drain any collection.

After resolution of SIRS, we perform esophagram to delineate the anatomy of the leak. Next, an endoscopic covered stent can be placed. Anecdotally, we have had more success with shorter (100 mm) and wider stents (23–25 mm); however there is no definitive literature regarding ideal stent size. We reserve surgery (gastrectomy with Roux-en-Y esophagojejunostomy) for those patients with ongoing morbidity from chronic leak.

Table 7.3 Summary of data regarding endoscopic stent placement after LSG leaks

Study Year	*n* (stented leaks)	Time to leak presentation	Stent type	Stent duration (days)	Time to healing	Success rate	Additional findings
Alazmi 2014 [22]	17	n/a	UltraFlex + polyFlex 18 × 150 mm self-expandable metal stent	42	n/a	76 %	• Shorter duration between gastrectomy and time of stent placement was associated with improved outcomes. Persistent leaks were treated with conversion to RYGB.
Sakran 2012 [3]	11	Mean 7 days	Unnamed endoscopic stents	n/a	40 day	55 %	• Routine intra- and postoperative to rule out leaks are superfluous. • Management options should be based on patient disposition.
Corona 2013 [25]	6	Range 1–7 days	Wallflex fully covered esophageal stent	30	n/a	100 %	• An algorithmic approach to treatment based on the eligibility for percutaneous drainage is beneficial in treating leak after LSG.
Simon 2013 [23]	9	Mean 11 day, range 2–29 days	Hanarostent 18 × 170 mm	45	141 day	78 %	• Early (<3 weeks after leak diagnosis) stent placement as it decreases healing time.
Nguyen 2010 [26]	3	Range 7 days–9 months	Alimax-E 22 × 120 mm covered stent	63	n/a	100 %	• Endoscopic stenting was safe and effective in treating both early and late leaks.
Tan 2010 [9]	8	n/a	n/a	n/a	n/a	50 %	• Stents were removed for complications and patient intolerance. • The authors now reserve stents for use in patients who failed other management.

Table 7.3 (continued)

Study Year	*n* (stented leaks)	Time to leak presentation	Stent type	Stent duration (days)	Time to healing	Success rate	Additional findings
de Aretxabala 2011 [20]	4	Range 3–25 days	Unnamed covered stents	42	21–240 day	100 %	• Management should be tailored to patient presentation. • Stents are effective, but sepsis control and nutritional support are cornerstones of treatment.
Moskowicz 2013 [27]	6	Mean 5.3 days	n/a	n/a	n/a	60 %	• Stenting alone was associated with a high failure rate, but salvage was achieved in by Ovesco clip + stent.

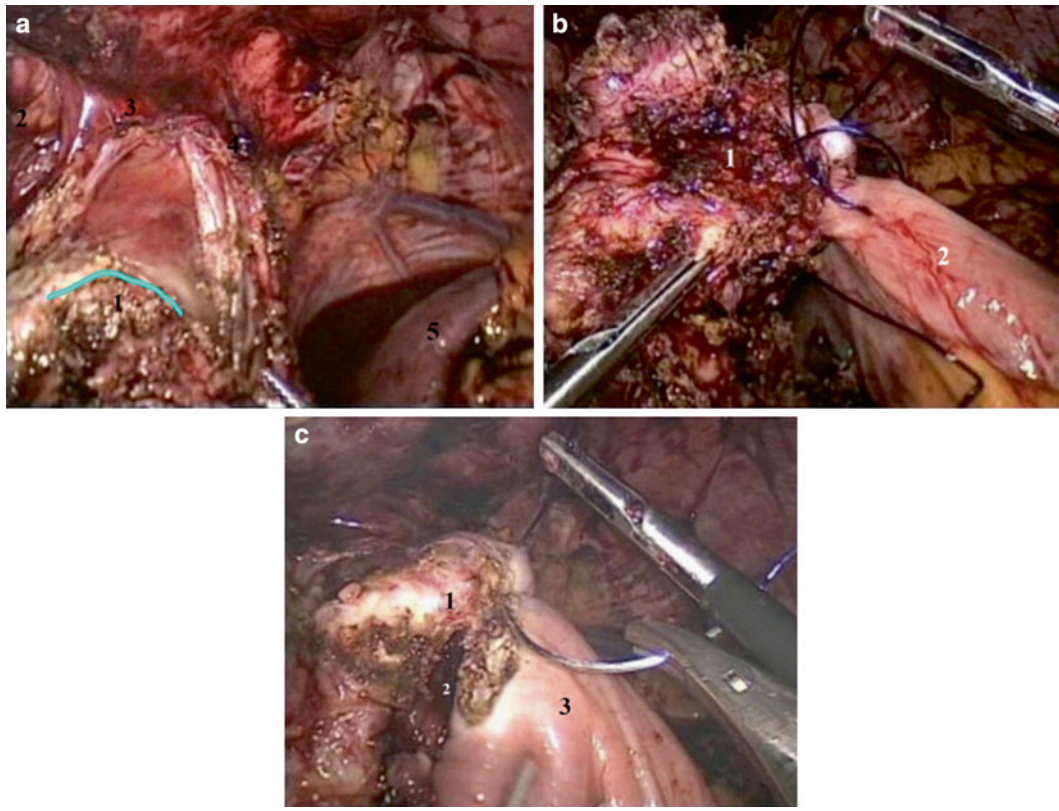

Fig. 7.11 Fistulojejunostomy surgical technique. (**a**) The hiatal region with the chronic fistula of the proximal sleeve. (1) Edge of the defect; (2) left lobe of the liver; (3) right crus; (4) left crus; (5) spleen. (**b**) Posterior anastomosis between the defect and the Roux limb. (1) Chronic fistula. (2) Roux limb. (**c**) Anterior anastomosis after opening the small bowel lumen of the Roux limb. (1) Edge of the defect; (2) nasogastric tube; (3) Roux limb. Reprinted with permission from van de Vrande S, Himpens J, El Mourad H, Debaerdemaeker R, Leman G. Management of chronic proximal fistulas after sleeve gastrectomy by laparoscopic Roux-limb placement. Surgery for Obesity and Related Diseases 2013;9:856–61 [36] © Elsevier

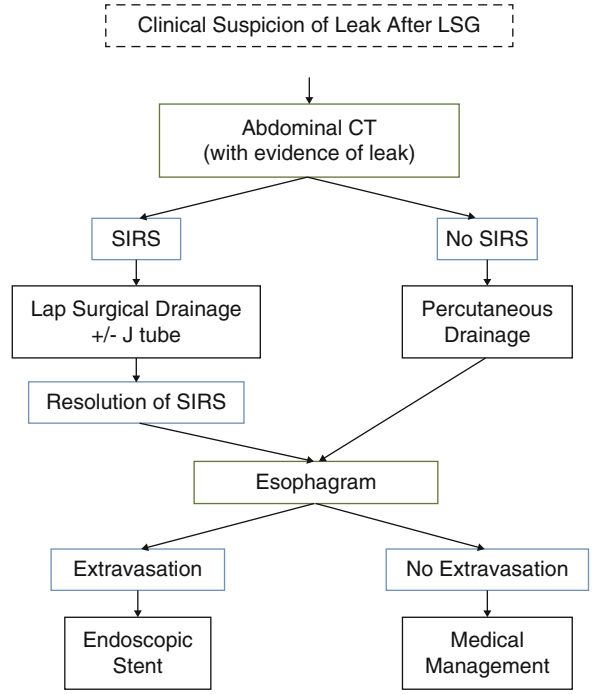

Fig. 7.12 Treatment algorithm for leak after LSG

7.5 Conclusion

Although rare, leaks after LSG may result in significant morbidity. Intraoperative techniques such as using a bougie size ≥40 French may decrease the rate of leak. Cornerstones of management include sepsis control and nutritional support, including laparoscopic washout, drainage, and placement of a jejunostomy tube, if necessary. Fortunately, most leaks resolve with endoscopic stenting alone. Surgical treatment (resection with Roux-en-Y esophagojejunostomy or fistula-jejunostomy) is occasionally needed in patients with chronic leaks refractory to endoscopic treatment.

References

1. Nguyen NT, Nguyen B, Gebhart A, Hohmann S. Changes in the makeup of bariatric surgery: a national increase in use of laparoscopic sleeve gastrectomy. J Am Coll Surg. 2013;216(2):252–7.
2. Gagner M, Deitel M, Erickson AL, Crosby RD. Survey on laparoscopic sleeve gastrectomy (LSG) at the Fourth International Consensus Summit on Sleeve Gastrectomy. Obes Surg. 2013;23(12):2013–7.
3. Sakran N, Goitein D, Raziel A, Keidar A, Beglaibter N, Grinbaum R, et al. Gastric leaks after sleeve gastrectomy: a multicenter experience with 2,834 patients. Surg Endosc. 2013;27(1):240–5.
4. Parikh M, Issa R, McCrillis A, Saunders JK, Ude-Welcome A, Gagner M. Surgical strategies that may decrease leak after laparoscopic sleeve gastrectomy: a systematic review and meta-analysis of 9991 cases. Ann Surg. 2013;257(2):231–7.
5. Aurora AR, Khaitan L, Saber AA. Sleeve gastrectomy and the risk of leak: a systematic analysis of 4,888 patients. Surg Endosc. 2012;26(6):1509–15.
6. Rosenthal RJ, International Sleeve Gastrectomy Expert P, Diaz AA, Arvidsson D, Baker RS, Basso N, et al. International Sleeve Gastrectomy Expert Panel Consensus Statement: best practice guidelines based on experience of >12,000 cases. Surg Obes Relat Dis. 2012;8(1):8–19.
7. Dallal RM, Bailey L, Nahmias N. Back to basics--clinical diagnosis in bariatric surgery. Routine drains and upper GI series are unnecessary. Surg Endosc. 2007;21(12):2268–71.
8. Mittermair R, Sucher R, Perathoner A, Wykypiel H. Routine upper gastrointestinal swallow studies

after laparoscopic sleeve gastrectomy are unnecessary. Am J Surg. 2014;207(6):897–901.
9. Tan JT, Kariyawasam S, Wijeratne T, Chandraratna HS. Diagnosis and management of gastric leaks after laparoscopic sleeve gastrectomy for morbid obesity. Obes Surg. 2010;20(4):403–9.
10. Jen S, Simillis C, Efthimiou E. A very challenging leak from a sleeve gastrectomy. Surg Obes Relat Dis. 2013;9(4):e56–9.
11. Burgos AM, Braghetto I, Csendes A, Maluenda F, Korn O, Yarmuch J, et al. Gastric leak after laparoscopic-sleeve gastrectomy for obesity. Obes Surg. 2009;19(12):1672–7.
12. Benedix F, Benedix DD, Knoll C, Weiner R, Bruns C, Manger T, et al. Are there risk factors that increase the rate of staple line leakage in patients undergoing primary sleeve gastrectomy for morbid obesity? Obes Surg. 2014;24:1610–6.
13. D'Ugo S, Gentileschi P, Benavoli D, Cerci M, Gaspari A, Berta RD, et al. Comparative use of different techniques for leak and bleeding prevention during laparoscopic sleeve gastrectomy: a multicenter study. Surg Obes Relat Dis. 2014;10(3):450–4.
14. Bellanger DE, Greenway FL. Laparoscopic sleeve gastrectomy, 529 cases without a leak: short-term results and technical considerations. Obes Surg. 2011;21(2):146–50.
15. Gagner M. Leaks after sleeve gastrectomy are associated with smaller bougies. Prevention and treatment strategies. Surg Laparosc Endosc Percutan Tech. 2010; 20:166–9.
16. Consten EC, Gagner M, Pomp A, Inabnet WB. Decreased bleeding after laparoscopic sleeve gastrectomy with or without duodenal switch for morbid obesity using a stapled buttressed absorbable polymer membrane. Obes Surg. 2004;14(10):1360–6.
17. Knapps J, Ghanem M, Clements J, Merchant AM. A systematic review of staple-line reinforcement in laparoscopic sleeve gastrectomy. JSLS. 2013;17(3):390–9.
18. Gagner M, Buchwald JN. Comparison of laparoscopic sleeve gastrectomy leak rates in four staple-line reinforcement options: a systematic review. Surg Obes Relat Dis. 2014;10(4):713–23.
19. Rossetti G, Fei L, Docimo L, Del Genio G, Micanti F, Belfiore A, et al. Is nasogastric decompression useful in prevention of leaks after laparoscopic sleeve gastrectomy? A randomized trial. J Invest Surg. 2014; 27(4):234–9.
20. de Aretxabala X, Leon J, Wiedmaier G, Turu I, Ovalle C, Maluenda F, et al. Gastric leak after sleeve gastrectomy: analysis of its management. Obes Surg. 2011;21(8):1232–7.
21. Casella G, Soricelli E, Rizzello M, Trentino P, Fiocca F, Fantini A, et al. Nonsurgical treatment of staple line leaks after laparoscopic sleeve gastrectomy. Obes Surg. 2009;19:821–6.
22. Alazmi W, Al-Sabah S, Ali DA, Almazeedi S. Treating sleeve gastrectomy leak with endoscopic stenting: the kuwaiti experience and review of recent literature. Surg Endosc. 2014;28(12):3425–8.
23. Simon F, Siciliano I, Gillet A, Castel B, Coffin B, Msika S. Gastric leak after laparoscopic sleeve gastrectomy: early covered self-expandable stent reduces healing time. Obes Surg. 2013;23(5):687–92.
24. Fukumoto R, Orlina J, McGinty J, Teixeira J. Use of Polyflex stents in treatment of acute esophageal and gastric leaks after bariatric surgery. Surg Obes Relat Dis. 2007;3(1):68–72.
25. Corona M, Zini C, Allegritti M, Boatta E, Lucatelli P, Cannavale A, et al. Minimally invasive treatment of gastric leak after sleeve gastrectomy. Radiol Med. 2013;118(6):962–70.
26. Nguyen NT, Nguyen XM, Dholakia C. The use of endoscopic stent in management of leaks after sleeve gastrectomy. Obes Surg. 2010;20(9):1289–92.
27. Moszkowicz D, Arienzo R, Khettab I, Rahmi G, Zinzindohoue F, Berger A, et al. Sleeve gastrectomy severe complications: is it always a reasonable surgical option? Obes Surg. 2013;23(5):676–86.
28. Pequignot A, Fuks D, Verhaeghe P, Dhahri A, Brehant O, Bartoli E, et al. Is there a place for pigtail drains in the management of gastric leaks after laparoscopic sleeve gastrectomy? Obes Surg. 2012;22(5):712–20.
29. Donatelli G, Ferretti S, Vergeau BM, Dhumane P, Dumont JL, Derhy S, et al. Endoscopic Internal Drainage with Enteral Nutrition (EDEN) for treatment of leaks following sleeve gastrectomy. Obes Surg. 2014;24(8):1400–7.
30. Dakwar A, Assalia A, Khamaysi I, Kluger Y, Mahajna A. Late complication of laparoscopic sleeve gastrectomy. Case Rep Gastrointest Med. 2013;2013:136153.
31. Kim Z, Kim YJ, Kim YJ, Goo D, Cho J. Successful management of staple line leak after laparoscopic sleeve gatrectomy with vascular plug and covered stent. Surg Laparosc Endosc Percutan Tech. 2011;21:e206–8.
32. Oshiro T, Saiki A, Suzuki J, Satoh A, Kitahara T, Kadoya K, et al. Percutaneous transesophageal gastrotubing for management of gastric leakage after sleeve gastrectomy. Obes Surg. 2014;24(9):1576–80.
33. El Hassan E, Mohamed A, Ibrahim M, Margarita M, Al Hadad M, Nimeri AA. Single-stage operative management of laparoscopic sleeve gastrectomy leaks without endoscopic stent placement. Obes Surg. 2013;23(5):722–6.
34. Thompson III CE, Ahmad H, Lo Menzo E, Szomstein S, Rosenthal RJ. Outcomes of laparoscopic proximal gastrectomy with esophagojejunal reconstruction for chronic staple line disruption after laparoscopic sleeve gastrectomy. Surg Obes Relat Dis. 2014;10(3):455–9.
35. Nedelcu A, Skalli M, Deneve E, Fabre J, Nocca D. Surgical managemnt of chronic fistula after sleeve gastrectomy. Surg Obes Relat Dis. 2013;9:879–84.

36. van de Vrande S, Himpens J, El Mourad H, Debaerdemaeker R, Leman G. Management of chronic proximal fistulas after sleeve gastrectomy by laparoscopic Roux-limb placement. Surg Obes Relat Dis. 2013;9(6):856–61.
37. Chour M, Alami RS, Sleilaty F, Wakim R. The early use of Roux limb as surgical treatment for proximal postsleeve gastrectomy leaks. Surg Obes Relat Dis. 2014;10(1):106–10.
38. Parikh M, Gagner M, Pomp A. Laparoscopic duodenal switch. In: Nguyen NT, De Maria EJ, Ikramuddin S, Hutter MM, editors. The SAGES manual: a practical guide to bariatric surgery. New York: Springer; 2008. p. 109–29.

8 Work-Up of Abdominal Pain in the Gastric Bypass and Vertical Sleeve Gastrectomy Patient

Adrian Dobrowolsky, Pornthep Prathanvanich, and Bipan Chand

Key Points

- A low threshold of suspicion and early diagnosis of complications is a very important thing in management of acute abdominal pain after bariatric surgery.
- Clinical manifestations, especially those of intra-abdominal septic complications, differ from standard descriptions in the non-obese patient.
- The cause of abdominal pain following LRYGB or LSG can be divided into anatomical and functional related disease and common or uncommon cause. Therefore physicians should develop a basic anatomic, clinical, and surgical understanding of these common procedures.

8.1 Introduction

According to the American Society for Metabolic and Bariatric Surgery (ASMBS), the number of bariatric procedures in the USA has doubled over the past 6 years. In the USA alone in 2008, 220,000 patients underwent bariatric surgery, with over 344,000 bariatric procedures performed worldwide the same year [1].

Over 90 % of bariatric operations are now performed laparoscopically; the most common operations are the laparoscopic Roux-en-Y gastric bypass (LRYGB), laparoscopic sleeve gastrectomy (LSG), and laparoscopic adjustable gastric band (LAGB) [1]. Nguyen et al. [2] reviewed the clinical data obtained from the University Health System Consortium database of 60,738 bariatric procedures between 2008 and 2012 and found that LSG rose from the third most popular procedure to the second, with an increase from 0.9 to 36.3 % total procedures. Alternatively, the use of LAGB decreased from 23.8 to 4.1 % and LRYGB fell from 66.8 to 56.4 % of total procedures. Open gastric bypass decreased from 8.6 to 3.2 % in 2012.

On average, 15–30 % of patients will visit the emergency room or require readmission within 3 years of gastric bypass [3, 4]. Nausea, vomiting, dehydration, benign abdominal pain, and wound issues account for over half of all emergency room visits or readmissions. Abdominal pain is the primary complaint in 20–45 % of these cases [3, 4]. Acute abdominal pain following bariatric surgery requires a low threshold of suspicion of organic causes. A failure of early diagnosis of complications may result in significant morbidity and mortality. Clinical manifestations, especially those of intra-abdominal septic

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_8

Table 8.1 Etiology of abdominal pain after LRYGB or LSG

	Common cause	Uncommon cause
1. Small bowel-related disease	– Internal hernia – Petersen's hernia – Mesocolic hernia – Mesenteric hernia – Incisional hernia – Trocar site hernia	– Adhesive small bowel obstruction – Intussusception – Stenosis or leak of the jejunojejunostomy
2. Gastric pouch and gastric remnant-related disease	– Leak – Ulcer disease (marginal ulcer and ulcer in remnant stomach) – Stenosis – Gastrogastric fistula – Gastroesophageal reflux disease – Hiatus hernia	
3. Biliary disease	– Gallstone (GS)	– Cholecystitis – Choledocholithiasis – Cholangitis – GS pancreatitis – Sphincter of Oddi dysfunction
4. Functional disorders	– Constipation, diarrhea – Irritable bowel syndrome – Dumping syndrome	– Esophageal motility disorders
5. Behavioral and nutritional disorders	– Maladaptive eating behavior: Overeating, rapid eating – Food intolerance – Micronutrient deficiencies	– Bacterial overgrowth in the defunctionalized stomach or small intestine
6. Other		– Omental torsion and/or infarction – Superior mesenteric syndrome – Median arcuate syndrome

complications, differ from standard presentations in the non-obese patient. Therefore, it is imperative that physicians develop a basic anatomic, clinical, and surgical understanding of these common procedures. The differential diagnosis of abdominal pain after LRYGB or LSG is diverse and presents diagnostic and therapeutic challenges. The etiologies can be divided into anatomic and functional diseases with common and uncommon causes as illustrated in Table 8.1.

This chapter focuses on the work-up of abdominal pain after LRYGB or LSG while subsequent chapters will discuss complications related to LAGB and the management of specific bariatric procedure-related complications.

8.1.1 Clinical Work-Up of Bariatric Surgical Complications

When a bariatric surgery patient presents with acute abdominal pain, evaluation should follow a stepwise approach. To begin, one must obtain a detailed history and perform a standard physical examination with special attention to the patient's vital signs. Since a complete evaluation should focus on bariatric surgery-related complications, consultation with a bariatric surgeon should be obtained early in the course of the evaluation. Ideally, abdominal pain work-up should involve the original surgeon as patients are not always aware of the details of their procedure, and variability in surgical technique is ubiquitous. An understanding of the specific bariatric procedure and its potential complications is essential to reveal the diagnosis [5–8] (Fig. 8.1).

Given the broad differential diagnosis in the stable and non-peritoneal patient, diagnostic algorithms must be guided by clinical history and physical exam but should remain flexible. A careful dietary and food history along with serum chemistries, complete blood count, and vitamin levels may reveal behavioral or nutritional causes of pain that are often easily treated. For example, patients may not sense satiety until the gastric pouch has distended to an uncomfortable and

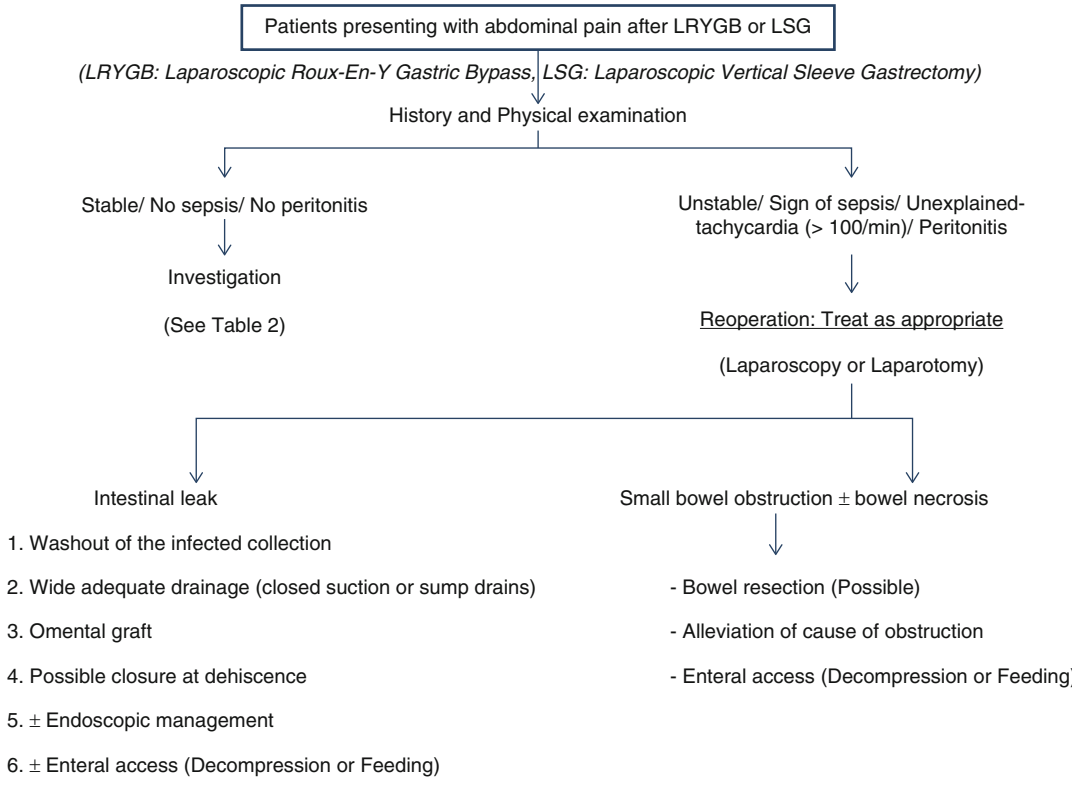

Fig. 8.1 Algorithm of patients presenting with abdominal pain after LRYGB or LSG

often painful state. Small, frequent meals may alleviate these problems, although patients typically learn to recognize early signs of discomfort in the months following surgery and adjust their eating habits accordingly. Additionally, hygroscopic foods such as rice, bread, and pastas should be limited as they are common culprits for uncomfortable gastric distention.

Also common to both gastric bypass and gastric sleeve patients is dehydration. The secondary effects of decreased fluid intake may lead to constipation with lower abdominal colicky pain being the common presenting complaint. Improved hydration and laxative use should alleviate these symptoms.

In any postoperative patient, common causes for abdominal pain may be as simple as a seroma or hematoma formation either in the subcutaneous tissues or the intra-abdominal compartment. Alternatively, one may develop a local wound infection, which has been reported in 3 % of LRYGB versus over 6 % of open RYGB patients [9]. These present with skin-level erythema, fluctuance, induration, pain, and possibly fevers or chills with leukocytosis on laboratory work-up. Local wound care sufficiently treats these; however, if there is a deep or intra-abdominal abscess, image-guided catheter placement versus surgical washout may be warranted.

Additional clues towards obtaining a diagnosis may be obtained through appropriate diagnostic testing (Fig. 8.1 and Table 8.2). Most patients will require abdominal X-rays, upper gastrointestinal (GI) contrast studies, and upper GI endoscopy as useful tests to provide a diagnosis in most cases. If a diagnosis cannot be identified, computerized tomography (CT) imaging of the abdomen and pelvis with intravenous and oral contrast may be indicated. If CT is non-diagnostic, ultrasound or esophageal manometry may be considered depending on the clinical presentation.

Table 8.2 Treatment for stable/no sepsis/no peritonitis patients presenting with abdominal pain after LRYGB or LSG (continue)

1. Complete history and physical examination, focusing on type of operation and presenting symptoms
2. Diagnostic (possible therapeutic) endoscopy
3. Laboratory: Full set of blood work such as CBC, coagulations, liver function, amylase
4. Diagnostic imaging such as acute abdominal series, upper gastrointestinal (GI) contrast study, abdominal ultrasound (US), computer tomography (CT) scan abdomen and pelvis with IV/oral contrast
5. Diagnostic laparoscopy

The diagnostician must consider the possibility of sepsis caused by anastomotic leak or from necrotic small bowel due to an internal hernia. An intra-abdominal infection from a leaking anastomosis is the most common cause of mortality within the first 12 weeks after surgery [10]. Fever, hypotension, tachycardia, tachypnea, decreased urine output, and hypoxia (with tachycardia being the most sensitive sign [11] should alert the physician to a possible bariatric surgery-related cause of sepsis. In a review by Bellorin et al., an anastomotic leak was likely to be present in patients with sustained tachycardia above 120 beats per minute (bpm) whereas bleeding complications were revealed by cyclical spikes of tachycardia usually less than 120 bpm [12]. The delay in onset between peritonitis and reoperation is the most important determinant of morbidity and mortality.

In general, when evaluating and managing patients who present with abdominal pain, some general guidelines may be observed:

1. Avoid placing the severely obese patient in a fully supine position during evaluation to minimize possible respiratory embarrassment caused by excess abdominal mass.
2. Should endotracheal intubation become necessary, ensure that the anesthesiologist is appropriately trained and aware of potential intubation difficulties [13, 14].
3. Nasogastric or orogastric intubation should be performed only if necessary, and care should be taken to avoid injury due to the altered anatomy of the upper GI tract.
4. Prolonged use of drugs that may induce gastric mucosal damage (NSAIDs, ASA, and steroids) should be avoided if possible.
5. The possibility of thiamine deficiency, due to vomiting, acute or chronic malnutrition, or altered eating habits, must be considered. If fluid replacement is indicated, start infusing non-glucose-containing solutions (normal saline or Ringer lactate), and administer thiamine before infusing glucose to avoid an acute onset of Wernicke's syndrome [15, 16].

The possibility of acute cholecystitis or symptomatic choledocholithiasis should be considered in any patient presenting with right upper quadrant pain after bariatric surgery. A recent meta-analysis revealed that cholecystectomy was subsequently performed in 6.8 % of all LRYGB patients, as compared to 1–5 % of the general population. Of the 6.8 %, 5.3 % were for biliary colic or biliary dyskinesia and 1 % due to cholecystitis [17]. Ultrasound will diagnose gallstones with an accuracy of more than 95 % and nuclear cholescintigraphy will diagnose acute cholecystitis with an accuracy of more than 90 % [18]. More sophisticated endoscopic and laparoscopic-assisted interventions to study the biliary tree or remnant stomach may be necessary in patients suspected of having disease in these organ systems, again keeping in mind the post-surgical anatomic alterations.

If a diagnosis is still not made after taking a full history, lab studies, and imaging, strong consideration should be made for diagnostic laparoscopy. This will allow for diagnosis of some pathologies like internal hernia which may not be evident even after a thorough preoperative work-up. In the very stable patient with a more chronic presentation, conservative therapies including acid suppression medications, smoking cessation, and NSAID avoidance should be considered as adjunctive management. If such conservative therapy fails after 4–8 weeks, diagnostic laparoscopy will likely be required to assess for a potential intra-abdominal source [19]. Unfortunately some may experience persistent pain despite exhaustive work-up and pain management consultation can often provide relief for these patients.

8.2 Procedure-Specific Complications

8.2.1 Small Bowel Obstruction After Bariatric Surgery

The incidence of small bowel obstruction (SBO) following open bariatric surgery has been reported to range from 1 to 5 % [20]. Similar rates have been reported with the laparoscopic approach (0.6–3.9 %) [21]. In a recent review of nearly 10,000 laparoscopic gastric bypasses, Martin et al. reported an overall incidence of 3.6 % [22]. Patients may present with severe intermittent diffuse abdominal pain lasting hours without a relationship to food. Bilious emesis is common, with obstipation being a less common finding, as these are usually proximal obstructions. Unlike open bariatric procedures where adhesive disease is the most common cause of obstruction, SBO after laparoscopic bariatric surgery is caused primarily by non-adhesive disease. An internal hernia is widely recognized as one of the most frequent causes of SBO (>50 %) in bariatric patients. Additionally, abdominal wall hernias may also cause pain in obese patients [22]. Understandably, it may be difficult to identify small incisional or trocar site hernias in an obese patient due to the limitations of physical examination in this population. There are three classic locations where SBO due to internal herniation can occur after LRYGB: Petersen's space (between the Roux limb's mesentery and transverse mesocolon in a retrocolic bypass), at the transverse mesocolon defect (for a retrocolic bypass), and at the jejuno-jejunostomy. Nasogastric decompression may be ineffective on a substantial portion of the gastrointestinal tract (gastric remnant, biliopancreatic limb) and prolonged non-operative management may be futile and dangerous. It is critical to remember that internal hernia often presents with abdominal pain but without bowel obstruction; the pain is caused by bowel ischemia secondary to venous outflow occlusion.

Other causes of post-bypass surgery SBO involve the formation of mesocolic defect strictures around the Roux limb (in retrocolic gastric bypass only), anastomotic strictures, intussusception, and volvulus of the gastric sleeve or the bowel distal to the Roux limb at the J-J anastomosis in LRYGB.

The patient's diagnosis is based on clinical presentation, radiologic imaging (upper gastrointestinal series or CT), and upper endoscopy. CT scan is an extremely effective diagnostic tool in the bypass population as it can reveal dilatation due to obstruction in the Roux limb, the gastric remnant, or the biliopancreatic limb; in a patient with internal hernia, it may even show a mesenteric "swirl" sign. CT scan has a sensitivity ranging from 78 to 100 % and specificity of 80–90 % [23, 24]. The cardinal signs of obstruction are proximally dilated bowel (usually including the esophagus and gastric pouch), distally collapsed bowel (distal small bowel and colon), and a transition point somewhere in between. Internal herniation is typically represented by the herniated bowel seen as fluid-filled dilated loops of small bowel situated at the left upper quadrant associated with a proximally dilated esophagus/gastric pouch/gastrojejunostomy and distally decompressed small bowel [25]. The high frequency of negative imaging may be due to the fact that CT scans may not be obtained during an episode of incarceration or that incarceration of a short segment of the biliopancreatic limb may not cause recognizable small bowel dilation. For these reasons, severe abdominal pain in a patient with prior gastric bypass is strongly suggestive of internal hernia and mandates surgical exploration unless a clear alternative diagnosis is established.

Laparoscopic exploration should always include evaluation of bowel viability and a retrograde examination of the bowel starting from the ileocecal valve. In the case of positive identification of an internal hernia, reduction of the herniated bowel should be performed, followed by closure of the mesenteric defect. Incarceration, which in many cases is transient, may not be found at exploration, but closure of defects nonetheless achieves good results with relief of pain in the majority of patients [26]. In a series of 13 patients who underwent exploratory laparoscopy for pain after gastric bypass,

there were two negative laparoscopies, while a diagnosis was made in 85 % including internal hernia in four [19]. These data support a low threshold for exploration for unexplained abdominal pain after bariatric surgery.

Acute dilatation of the gastric remnant after LRYGB is potentially a catastrophic event resulting from the closed-loop obstruction that follows obstruction of the biliopancreatic limb (BPL). This is due to the large volume of digestive secretions accumulating in the upper digestive tract, with possible evolution to gastric wall necrosis and/or perforation. Severe epigastric pain and hypovolemic shock (evidenced by tachycardia) in conjunction with gastric dilatation on a plain abdominal X-ray or CT scan are diagnostic. Gastric remnant obstruction can lead to rapid clinical deterioration with blowout of the staple line and hemodynamic instability. Percutaneous remnant gastrostomy decompression will decompress the dilated remnant and temporize the situation, while laparoscopic or open exploration will allow for formal management of the underlying cause of BPL obstruction.

8.2.2 Laparoscopic Sleeve Gastrectomy

8.2.2.1 Staple-Line Leak

In the literature, the incidence of gastric leak after laparoscopic sleeve gastrectomy (LSG) ranges from 0 to 7 % [7, 8]. Most leaks appear in the proximal third of the stomach, close to the esophagogastric (EG) junction or near the angle of His. Burgos et al. reported that 85 % of leaks occur in the proximal third of the stomach and only 14 % in the distal third [27]. Etiologies may include poor staple-line configuration or devascularization. Due to this potential complication, many authors suggest resecting at least 2 cm away from the EG junction.

The signs and symptoms of a patient who develops a leak are similar to other types of abdominal infections. However the clinical presentation of gastric leak ranges from an asymptomatic patient (identified by an imaging study) to localized or generalized peritonitis. A septic patient may have pain, fever, tachycardia, tachypnea, persistent hiccoughs, and pain in the left shoulder. This may lead to septic shock, multiorgan failure, and, if undiagnosed or untreated, death. Abdominal plain X-rays, contrast studies, as well as measuring drain fluid amylase may assist in the diagnosis. In order to increase sensitivity, abdominal computerized tomography (CT) scan with oral Gastrografin contrast should be performed. Additionally, CT scan provides additional information in regard to fluid collections or abscess formation or the presence of subdiaphragmatic free intraperitoneal air.

Surgical intervention should focus on sepsis control, prevention of abdominal recontamination, and nutritional support via enteral or parenteral access and is detailed in later chapters.

8.2.3 Laparoscopic Roux-En-Y Gastric Bypass

8.2.3.1 Anastomotic Leak

The incidence ranges from 0 to 6.1 % [6]. Patients having undergone laparoscopic Roux-en-Y gastric bypass (LRYGB) that have a gastrointestinal leak present similarly to those with a leak after LSG. Diagnosis can be confirmed with Gastrografin swallow and CT scan. Emergent surgical treatment should be considered in hemodynamically unstable patients with severe, persistent symptoms. Unlike LSG, the site of intestinal leakage may be more of a challenge to diagnose given the different regions of the GI tract that are operated upon and the lack of contrast opacification of the entire system once bypassed.

8.2.3.2 Marginal Ulcer

This refers to a peptic ulcer on the mucosa near the site of the gastrojejunal anastomosis. It can occur early (1–3 months) or late after an LRYGB. It is located either directly on the anastomosis (50 %) or just distal to this on the jejunum (40 %) [28]. Its reported incidence ranges between 0.3 and 16 %, and several risk factors are known including type of suture used (absorbable vs. nonabsorbable), patient age, history of previous

gastric surgery, preoperative diabetes, coronary artery disease or peptic ulcer disease, and the use of nonsteroidal anti-inflammatory medications or tobacco [29, 30]. In a large cohort study, prior or current tobacco use remained the only independent risk factor for ulcer persistence after treatment [31]. The most common presenting symptom is pain (63 %) followed by bleeding (24 %), but perforation can occur. Pouch ulceration heals with proton pump inhibitors and/or sucralfate along with cessation of NSAID intake and smoking. In patients with a large pouch, ulcer recurrence with medical therapy alone is common and consideration should be given to a reduction of the pouch size with excision of the refractory ulcer. The incidence of a perforated marginal ulcer after LRYGB is ≤1 %. The clinical picture is similar to any other visceral perforation: severe epigastric pain, tachycardia, fever, and leukocytosis, with free intra-abdominal air on plain radiographs or CT scan. Surgical management is required and can be performed by laparoscopy or laparotomy. Treatment consists of omental patch or revision of the anastomosis as required [32, 33]. A gastrostomy tube in the excluded stomach should be considered for enteral nutrition, and high-dose PPI therapy is indicated as well as eradication of *H. pylori* if present.

8.2.3.3 Gastrogastric Fistula

Before linear cutting staplers were used to divide the remnant stomach from the gastric pouch, undivided RYGB had a reported incidence of gastrogastric fistulae of up to 50 % [34]. With divided RYGB, the incidence has fallen to 0–6 % [34, 35]. Patients may present with nausea and vomiting (often bilious given the connection to the duodenum) as well as with epigastric pain. A marginal ulcer is often found as a result of excessive gastric acid refluxing through the fistula and bathing the anastomosis. Proposed mechanisms of gastrogastric fistulae include staple migration, inflammation from foreign material, and local tissue ischemia from the staple line [34, 36]. Because visualization of the fistula through endoscopy has a lower sensitivity, the test of choice is an upper gastrointestinal series where contrast may be visualized traversing into the gastric remnant and into the duodenal sweep.

Superior Mesenteric Artery Syndrome

While superior mesenteric artery (SMA) syndrome is a rare disorder, one must keep in mind that patients with rapid weight loss are at risk for this. Patients present with atypical postprandial pain, nausea, vomiting, and even further weight loss due to an acute angulation of the SMA and aorta on the third portion of the duodenum. The mechanism is thought to be loss of the supportive fatty tissue around the SMA. Endoscopy is utilized to rule out other pathology but CT angiography is the most sensitive test to show obstruction of the third portion of the duodenum and an acute angulation of the SMA [37]. Even rarer is the phenomenon of median arcuate ligament syndrome in which the diaphragm impinges on the celiac artery and plexus causing postprandial pain, nausea, vomiting, and weight loss similar to SMA syndrome. However CT shows the impingement on the celiac artery and ultrasound identifies elevated velocities at the level of the celiac artery [38].

8.3 Conclusion

There are multiple causes of abdominal pain after LRYGB or LSG, some of which are specific to the operation while others are common to all patients. It is an important problem that presents significant diagnostic and therapeutic challenges. A clear understanding of the pathogenesis of each complication will help guide physicians in their diagnosis and reduce overall postoperative morbidity and mortality.

References

1. Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2008. Obes Surg. 2009;9(12):1605–11.
2. Nguyen NT, Nguyen B, Gebhart A, Hohmann S. Changes in the makeup of bariatric surgery: a national increase in use of laparoscopic sleeve gastrectomy. J Am Coll Surg. 2013;216:252–7.

3. Cho M, Kaidar-Person O, Szomstein S, Rosenthal RJ. Emergency room visits after laparoscopic Roux-en-Y gastric bypass for morbid obesity. Surg Obes Relat Dis. 2008;4(2):104–9.
4. Kellogg TA, Swan T, Leslie DA, Buchwald H, Ikramuddin S. Patterns of readmission and reoperation within 90 days after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5(4):416–23.
5. Gagner M, Deitel M, Kalberer BA, et al. The second international consensus summit for sleeve gastrectomy, March 19–21, 2009. Surg Obes Relat Dis. 2009;5:476–85.
6. Monkhouse SJ, Morgan JD, Norton SA. Complications of bariatric surgery: presentation and emergency management—a review. Ann R Coll Surg Engl. 2009; 91(4):280–6.
7. Lalor PF, Tucker ON, Szomstein S, et al. Complications after laparoscopic sleeve gastrectomy. Surg Obes Relat Dis. 2008;1:33–8.
8. Aurora AR, Khaitan L, Saber AA. Sleeve gastrectomy and the risk of leak: a systematic analysis of 4,888 patients. Surg Endosc. 2012;26:1509–15.
9. Podnos YD, Jimenez JC, Wilson SE, Stevens CM, Nguyen NT. Complications after laparoscopic gastric bypass: a review of 3464 cases. Arch Surg. 2003; 138(9):957–61.
10. Smith MD, Patterson E, Wahed AS, et al. Thirty-day mortality after bariatric surgery:independently adjudicated causes of death in the longitudinal assessment of bariatric surgery. Obes Surg. 2011;21(11):1687–92.
11. Gagnière J, Slim K. Don't let obese patients be discharged with tachycardia after sleeve gastrectomy. Obes Surg. 2012;22(9):1519–20.
12. Bellorin O, Abdemur A, Sucandy I, Szomstein S, Rosenthal RJ. Understanding the significance, reasons and patterns of abnormal vital signs after gastric bypass for morbid obesity. Obes Surg. 2011; 21(6):707–13.
13. Loder WA. Airway management in the obese patient. Crit Care Clin. 2010;26(4):641–6.
14. Kristensen MS. Airway management and morbid obesity. Eur J Anaesthesiol. 2010;27(11):923–7.
15. Kazemi A, Frazier T, Cave M. Micronutrient-related neurologic complications following bariatric surgery. Curr Gastroenterol Rep. 2010;12(4):288–95.
16. Galvin R, Bråthen G, Ivashynka A, Hillbom M, Tanasescu R, Leone MA. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. Eur J Neurol. 2010;17(12):1408–18.
17. Warschkow R, Tarantino I, Ukegjini K, et al. Concomitant cholecystectomy during laparoscopic roux-en-Y gastric bypass in obese patients is not justified: a meta-analysis. Obes Surg. 2013;23(3): 397–407.
18. Tucker ON, Fajnwaks P, Szomstein S, Rosenthal RJ. Is concomitant cholecystectomy necessary in obese patients undergoing laparoscopic gastric bypass surgery? Surg Endosc. 2008;22(11):2450–4.
19. Pitt T, Brethauer S, Sherman V, et al. Diagnostic laparoscopy for chronic abdominal pain after gastric bypass. Surg Obes Relat Dis. 2008;4:394–8.
20. Srikanth MS, Keskey T, Fox SR, Oh KH, Fox ER, Fox KM. Computed tomography patterns in small bowel obstruction after open distal gastric bypass. Obes Surg. 2004;14(6):811–22.
21. Elms L, Moon RC, Varnadore S, Teixeira AF, Jawad MA. Causes of small bowel obstruction after roux-en-Y gastric bypass: a review of 2,395 cases at a single institution. Surg Endosc. 2014;28(5):1624–8.
22. Martin MJ, Beekley AC, Sebesta JA. Bowel obstruction in bariatric and nonbariatric patients: major differences in management strategies and outcome. Surg Obes Relat Dis. 2011;7(3):263–9.
23. Iannuccilli JD, Grand D, Murphy BL, et al. Sensitivity and specificity of eight CT signs in the preoperative diagnosis of internal mesenteric hernia following Roux-en-Y gastric bypass surgery. Clin Radiol. 2009; 64:373–80.
24. Lockhart ME, Tessler FN, Canon CL, et al. Internal hernia after gastric bypass: sensitivity and specificity of seven CT signs with surgical correlation and controls. AJR Am J Roentgenol. 2007;188:745–50.
25. Quigley S, Colledge J, Mukherjee S, Patel K. Bariatric surgery: a review of normal postoperative anatomy and complications. Clin Radiol. 2011;66:903–14.
26. Gandhi AD, Patel RA, Brolin RE. Elective laparoscopy for herald symptoms of mesenteric/internal hernia after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5(2):144–9.
27. Burgos AM, Braghetto I, Csendes A, et al. Gastric leak after laparoscopic-sleeve gastrectomy for obesity. Obes Surg. 2009;19:1672–7.
28. Azagury DE, Abu Dayyeh BK, Greenwalt IT, Thompson CC. Marginal ulceration after Roux-en-Y gastric bypass surgery: characteristics, risk factors, treatment, and outcomes. Endoscopy. 2011; 43(11):950–4.
29. Sacks BC, Mattar SG, Qureshi FG, Eid GM, Collins JL, Barinas-Mitchell EJ, Schauer PR, Ramanathan RC. Incidence of marginal ulcers and the use of absorbable anastomotic sutures in laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2011; 2(1):11–6.
30. Scheffel O, Daskalakis M, Weiner RA. Two important criteria for reducing the risk of postoperative ulcers at the gastrojejunostomy site after gastric bypass: patient compliance and type of gastric bypass. Obes Facts. 2011;4 Suppl 1:39–41.
31. El-Hayek K, Timratana P, Shimizu H, Chand B. Marginal ulcer after Roux-en-Y gastric bypass: what have we really learned? Surg Endosc. 2012;26(10):2789–96.
32. Binenbaum SJ, Dressner RM, Borao FJ. Laparoscopic repair of a free perforation of a marginal ulcer after Roux-en-Y gastric bypass: a safe alternative to open exploration. JSLS. 2007;11(3):383–8.

33. Wendling MR, Linn JG, Keplinger KM, Mikami DJ, Perry KA, Melvin WS, Needleman BJ. Omental patch repair effectively treats perforated marginal ulcer following Roux-en-Y gastric bypass. Surg Endosc. 2013;27(2):384–9.
34. Capella JF, Capella RF. Gastro-gastric fistulas and marginal ulcers in gastric bypass procedures for weight reduction. Obes Surg. 1999;9(1):22–7. discussion 28.
35. Carrodeguas L, Szomstein S, Soto F, et al. Management of gastrogastric fistulas after divided roux-en-Y gastric bypass surgery for morbid obesity: analysis of 1,292 consecutive patients and review of literature. Surg Obes Relat Dis. 2005;1(5):467–74.
36. Stanczyk M, Deveney CW, Traxler SA, McConnell DB, Jobe BA, O'Rourke RW. Gastro-gastric fistula in the era of divided roux-en-Y gastric bypass: strategies for prevention, diagnosis, and management. Obes Surg. 2006;16(3):359–64.
37. Goitein D, Gagne DJ, Papasavas PK, et al. Superior mesenteric artery syndrome after laparoscopic roux-en-Y gastric bypass for morbid obesity. Obes Surg. 2004;14(7):1008–11.
38. Richards NG, Neville RF, Sidawy AN, Brody FJ. Celiac artery compression after a gastric bypass. Surg Laparosc Endosc Percutan Tech. 2014; 24(2):e66–9.

9 Workup of Abdominal Pain or Vomiting in the Gastric Band Patient

Ann M. Rogers, Cheickna Diarra, and Shaukat A. Gulfaraz

Key Points

- Nausea, vomiting, and abdominal pain are common in band patients.
- Some causes of these symptoms are easily treated in the outpatient setting.
- Heightened awareness of the more urgent or emergent band complications may be potentially life-saving.
- Special expertise is helpful in expeditiously and appropriately evaluating and treating patients with band-related emergencies, so whenever possible, trained bariatric surgeons should be involved in their care.

9.1 Introduction

Gastric band placement has been a commonly performed operation for weight loss around the world. Several types of nonadjustable bands were placed in the past, but because of the nature and severity of the ensuing complications, these operation have been largely abandoned in modern bariatric practice. Attention is focused in this chapter on the evaluation and management of abdominal pain, vomiting, and other complications related specifically to adjustable gastric bands.

The adjustable gastric band procedure became very popular in the USA during the decade after the FDA approved the Lap-Band® in 2001 [1]. Reasons for its popularity included the technical ease and safety of placement, promising short term weight loss data, and a perception by patients of noninvasiveness and potential reversibility. However, with medium-term data, band patients have been found to have issues with inadequate weight loss, weight regain, and frequent complications requiring reoperation or system removal. Because of this, the use of adjustable gastric banding has significantly decreased in many bariatric centers. Nonetheless, bariatric surgeons and emergency departments will continue to see patients who have bands in situ and who are having symptoms potentially related to their band.

There are two major brands of gastric bands currently approved for use in this country: the Lap-Band® (Apollo Endosurgery, Austin, TX) and the Realize Band® (Ethicon, Cincinnati, OH) with various subtypes differing in size, shape and maximum fill volume. Two other bands are used in Europe: the Heliogast band and the Midband, both of which also have adjustable internal balloons. While these bands are not approved for use in the USA, they could come to the attention of surgeons in this country if patients with such bands travel to the USA and require care.

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_9

9.1.1 Normal Radiographic Findings in Band Patients

On plain abdominal radiographs or on fluoroscopic examinations, the band should be visible just below the gastroesophageal junction and should point towards the left shoulder at roughly a 30–45° angle (Fig. 9.1).

During a contrast swallow there should be roughly a 3–4 mm stream of contrast seen on fluoroscopy going through the band (Figs. 9.2 and 9.3).

9.2 Dysphagia, Gastroesophageal Dilatation and Dysmotility

Dysphagia, or difficulty swallowing, is a common symptom for band patients and may suggest a number of different pathologies. Dysphagia is differentiated from odynophagia, or pain on swallowing, and globus sensation, or a sense of a lump in the throat, particularly after swallowing. Any or all of these symptoms may be present at one time or another in band patients, and the leading cause is an overly tightened band [2]. A band may be too tight even without the radiologic findings of dilated esophagus or gastric pouch, and the diagnosis is fairly easily made in a patient who has recently had an adjustment in which

Fig. 9.1 Normal postoperative plain abdominal film with Lap-Band®. The band is visible in the upper half of the image, just to the left of the patient's spine, while the access port is visible in the lower half, to the right of the spine

Fig. 9.2 Normal immediate postoperative upper gastrointestinal swallow study after band placement

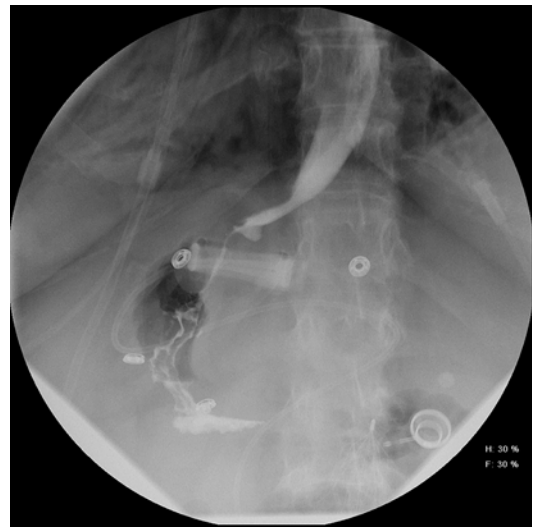

Fig. 9.3 Normal postoperative swallow study of band in patient with *situs inversus* associated with Kartagener's Syndrome

fluid was added to the band. Usually a patient will report that symptoms started soon after a band fill, and the problem may be simply treated with band deflation and watchful waiting.

Patients may often report having a bolus of food become stuck at the level of the band, and that they have waited with discomfort for the bolus to pass. Often such boli do eventually pass spontaneously but sometimes have to be brought up through regurgitation, be it natural or induced. Food bolus impaction was reported as the most common cause of emergency department visits for band patients in an Australian series, representing 32.1 % of band patients seen urgently [3]. In such a presentation, the band should be deflated, which more often than not allows the bolus to pass. This can be accomplished by locating the access port by palpation and entering the center of the port with a non-coring Huber needle, then aspirating out as much saline as possible. If deflating the band is inadequate to allow passage of a stuck food bolus, endoscopy may be required to either retrieve the obstructing item or push it through the band distally. Foods that more commonly lead to obstruction or globus sensation in band patients include fibrous, poorly chewed meats and sticky "white" foods such as bread, pasta and rice. The most common symptoms of a stuck food bolus in the outpatient setting include hiccups, "productive burping" (bringing up the bolus unbidden), and "sliming." Sliming refers to bubbling mucus that comes up the throat in response to a stuck bolus.

Other symptoms of a too-tight band include heartburn, signs of nocturnal reflux such as nighttime cough, regurgitation onto the pillow, or frank aspiration. Patients are often loathe to report that their band is too tight for fear that this will lead to deflation and that they will therefore gain weight. Sometimes a too-tight band is not brought to the surgeon's attention until a patient presents with aspiration pneumonia.

More insidious onset of dysphagia can come about from cycles of stuck food and vomiting, after which the portion of stomach passing through the band becomes edematous. In such a situation, what might have been a good level of restriction becomes too tight, and patients often need to accommodate by regressing to a liquid diet or "slider" foods that pass through the band more easily. Advancing once more to a regular diet, particularly if the patient eats too quickly or does not chew carefully, may again cause stuck food and the need for regurgitation. Because of this, the first presentation of a too-tight band may be weight gain; patients are hungry and because they have difficulties with bulkier foods, they will turn to those items that pass more easily through the band, such as ice cream. Ongoing cycles of turning to slider foods, followed by attempts at more solid intake with concomitant vomiting, will frequently lead to dilation of the gastric pouch above the band, and eventually to esophageal dilatation. Such dilatation can be easily seen on upper gastrointestinal (UGI) swallow studies (Figs. 9.4 and 9.5).

Fig. 9.4 Upper GI swallow study in a patient with pouch and esophageal dilatation

The anatomy seen on such studies can be confusing to both the radiologist and the surgeon. It is not uncommon to see a report of a "hiatal hernia" above the band. In order to spare patients unnecessary and potentially harmful reoperations, esophagogastric dilation should be care-

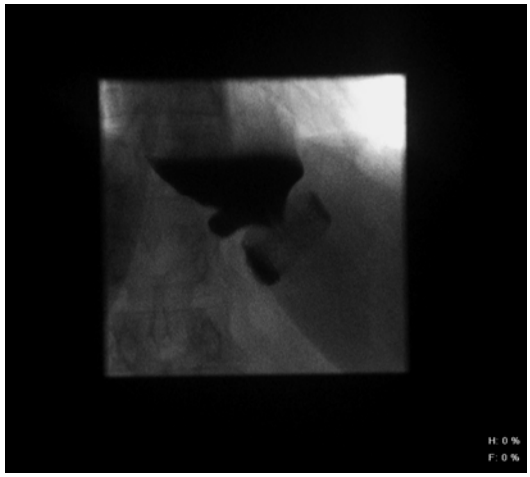

Fig. 9.5 Upper GI swallow study in a patient with pouch and esophageal dilation. Note gastroesophageal junction can be seen below the level of the diaphragm

Fig. 9.6 Herniation of gastric pouch above the diaphragm

fully differentiated from true hiatal herniation of the pouch as seen below (Fig. 9.6).

Gastric and esophageal dilatation are common in band patients [4], particularly those who eat forcefully against the band. Cycles of overeating, stuck food and vomiting lead to fatigue of the esophageal musculature and alteration of the normal peristaltic function to the point that the esophagus can become a flaccid holding pen—in essence a second stomach in the chest above the band. When this happens, patients will frequently experience some regurgitation, but much of their ingested food will remain in the esophagus and slowly pass through the band like sand through an hourglass. Patients will be confused, therefore, by what seems to be the counterintuitive coexistence of vomiting and weight gain. Such patients must be counseled that further tightening of the band will only exacerbate the problem.

As stated previously, the treatment in such situations would include band deflation, followed by a waiting period of several weeks, followed by possible repeat imaging, and if the dilatation is improved, a slow band refill process. In cases such as this, it frequently happens that a good point of restriction is reached at a lower fill level than previously, so patients should be counseled not to perseverate about absolute quantities of fill but on satiety and restriction only.

Esophagitis and gastroesophageal reflux are not uncommon after gastric banding [4], and may present as abdominal or chest pain, dysphagia, or night cough. There are conflicting reports of the band either improving or worsening heartburn in patients, but in the majority of band patients simple heartburn is amenable to treatment with medications. Severe, unrelenting heartburn should be investigated, as it may be a sign of a more serious diagnosis. Cases of erosive esophagitis with hemorrhage have been seen with a band that is too tight, even in the absence of other pathology such as prolapse.

Esophageal dysmotility after banding is another entity that may be noted on UGI studies (Figs. 9.7 and 9.8).

If contrast or motility studies were not performed preoperatively, and in most practices they are not, it will not be possible to definitively determine if the patient had baseline altered motility prior to band placement or if such dysmotility developed secondary to the presence of the band. Dysmotility may be seen to improve on

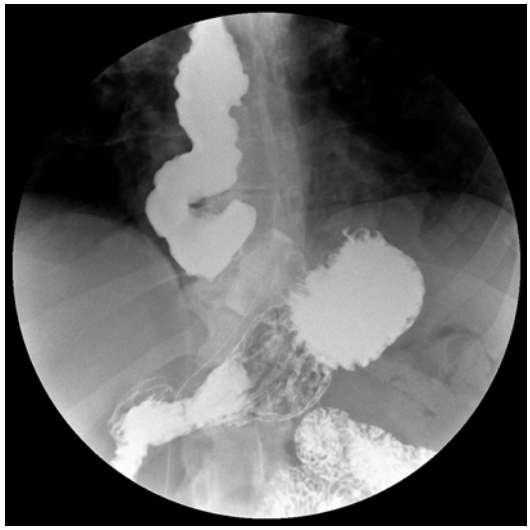

Fig. 9.7 Pouch and esophageal dilation, with pouch herniated above the diaphragm, and with tertiary waves of esophageal contraction

Fig. 9.8 Pouch and esophageal dilation, with associated tortuosity of esophagus from forceful eating against the band

imaging studies after band deflation, but it is important to be aware that it may return with ongoing fills. In some patients, dysmotility makes it impossible to achieve a good point of restriction without causing dysphagia.

Patients should be made aware that after band deflation, food will generally pass more easily. Because of this, patients who have been suffering with a too-tight band may, after deflation, celebrate their renewed ability to eat, and therefore may rapidly gain or regain weight. This can be avoided by protein shake meal replacements, mindful eating with reasonable portion sizes and good food choices, and separating liquid from solid foods during meals, i.e., revisiting their preoperative dietary counseling. A good analogy for band patients is that they must not think of their band as an "air bag." When driving, one should not wait for the air bag to deploy in order to know to slow down; just as one should drive at a proper speed and stay within the lane markings rather than waiting for external cues, patients must learn to preselect their portion sizes, chew carefully, and eat slowly. By this analogy, stuck food or vomiting should not be the cue that tells band patients to slow down and eat mindfully.

On the topic of band deflation, bariatric programs need practitioners who are adept at band adjustments. Such adjustments may be done in a variety of ways, according to surgeon or practitioner preference. This may include such techniques as following a prescribed volume algorithm for a given band while accessing the band, with or without local anesthetic, on an examination table; filling the band under fluoroscopic imaging with the patient in a sitting or standing position while swallowing contrast material; or filling the band with the patient in the sitting position while drinking water and tightening the band to the point where the water passes slowly. However the adjustment is done, it should always be done with a Huber non-coring needle to minimize the risk of injury to the silicone diaphragm; this can happen with other needles that may remove a core of the diaphragm during needle passage, thus allowing the fluid to slowly leak out of the access port and thus rendering the system nonfunctional.

9.3 Gastric Prolapse

Prolapse of a portion of the stomach up through the band may also be referred to as a "slippage" or "slipped band." It is helpful to remember that the band is not actually slipping down onto the distal stomach, but that the stomach itself is prolapsing upward through the band, usually secondary to forceful vomiting. This is the most common intra-abdominal complication of banding [2] and one that may require an abdominal operation for resolution. Prolapse was a more common complication in the early days of banding, when a perigastric approach was used [5]. The now-preferred pars flaccida approach, in which the gastrohepatic ligament is entered, the medial aspect of it is held within the band–stomach complex, and entry into the lesser sac is avoided, has significantly decreased the risk of prolapse. While the majority of surgeons perform a gastrogastric plication over the anterior portion of the band in order to prevent prolapse, there are surgeons who avoid this step, claiming it takes additional time and does not prevent prolapse. These results remain controversial [6]. In an acute presentation of prolapse, a patient generally has had an episode of acute vomiting or retching, followed by complete or near-complete obstruction to passage of food or even liquids at the level of the band. As with a band that is simply too tight, there may be associated heartburn, or signs of nocturnal reflux. With such symptoms, the band should be completely deflated. If deflation leads to complete resolution of symptoms, it is possible that the patient simply had a too-tight band. However, it is also possible for a prolapse to completely reduce upon band deflation.

On plain films or fluoroscopy, a band prolapse will be demonstrated by a change in the angle of the band such that it no longer points toward the left shoulder. This comes about from pressure of the prolapsed stomach pushing down on the band (Fig. 9.9).

In extreme cases, a prolapse will present as an "O" sign in which the lumen of the band is clearly visible on an A-P projection (Fig. 9.10).

An UGI swallow study with contrast is the best way to image many band complications, including prolapse. This may show pouch dilatation or eccentric pouch dilatation with or without esophageal dilatation, delayed passage of contrast material, flattening or reverse angulation of the band itself, and excessive pouch overhanging the band (Figs. 9.11 and 9.12).

Acute gastric prolapse generally requires hospital admission and resuscitation, and may

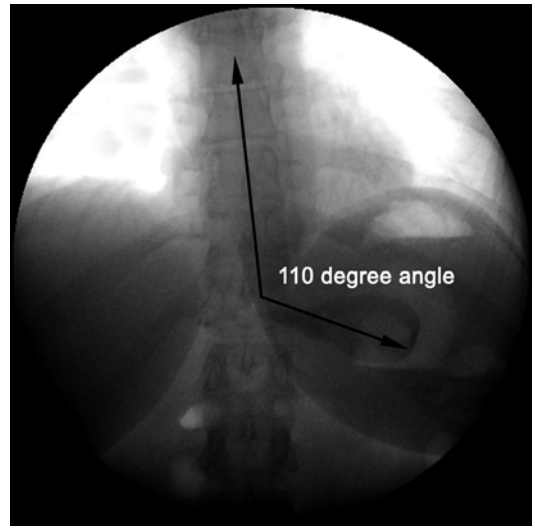

Fig. 9.9 Expansion of the usual 45° angle of band, which no longer points toward the left shoulder. This patient was found to have a gastric prolapse

Fig. 9.10 Complete posterior prolapse causing extreme angulation of the band

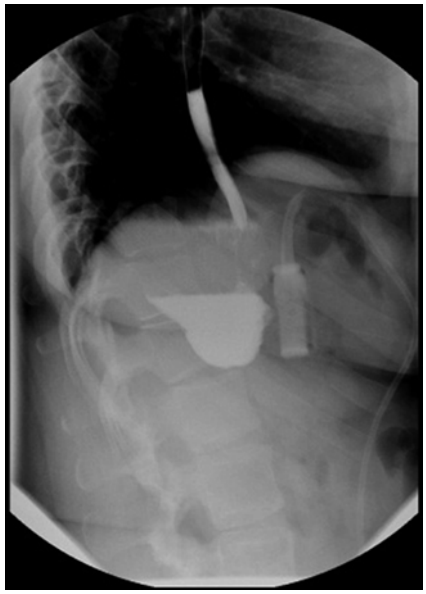

Fig. 9.11 Contrast study showing side-view of patient from Fig. 9.10, with posterior prolapse

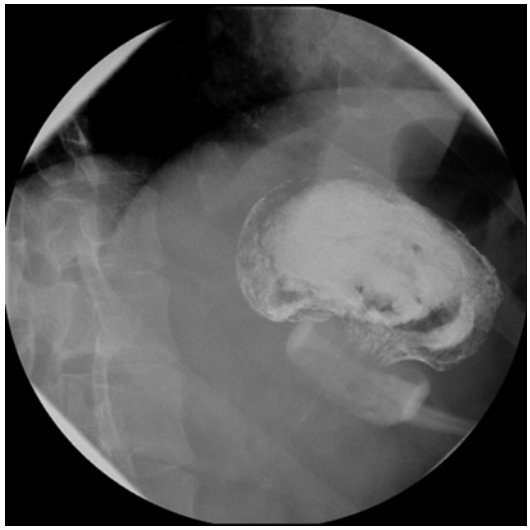

Fig. 9.12 Upper GI contrast study of patient with large anterior prolapse

require operative intervention. Indications for operation include ongoing obstruction and abdominal pain even after complete deflation of the band. Pain implies the possibility of gastric ischemia, which may progress to frank necrosis. This can lead to the need for resection and if untreated can potentially lead to perforation and death [7].

Operative treatment of symptomatic, unreduced gastric prolapse can generally be accomplished laparoscopically. Gentle downward traction on the stomach may reduce it through the band, although the presence of adhesions and gastric edema may prevent this. Sometimes takedown of the area of gastrogastric suturing, if present, may facilitate gastric reduction. If this is unsuccessful, an attempt may be made to unbuckle the band; however, this can be exceedingly difficult with certain band types. If unbuckling is successful, there are various options including simply leaving the band unbuckled with the decision made to come back at a later date to replace and rebuckle the band. Another option is to replace the band in an appropriate position above the prolapsed portion of stomach and again perform gastric plication with interrupted sutures toward the greater curvature. In cases where the prolapse cannot be reduced and the band cannot be unbuckled, it may be sharply transected and removed or replaced. Band replacement may be done through an entirely new retrogastric instrument passage, or by suturing a new band to the transected old band and pulling it through the retrogastric tract, then assuring good position by placing the new band cephalad to the formerly prolapsed segment. It is important to assure that the newly buckled band is not too tight around the stomach, by assuring that a smooth grasper passes easily between the band and the stomach. This can also be confirmed with intraoperative gastroscopy. Note that if there is any sign of gastric necrosis or perforation, the band system should be entirely removed and the ischemic area addressed in an appropriate fashion.

Gastric prolapse may also come to the surgeon's attention insidiously, after many months of mild symptoms suggestive of a too-tight band. Prolapse not associated with vomiting or abdominal pain may be treated in a more elective fashion, but in many cases will ultimately come to the need for surgical treatment.

9.4 Erosion

Band erosion into the stomach has an estimated incidence of 1–6 % [8–10]. While the stomach is the most common site, erosion into nearby organs has also been reported, including the liver, transverse colon, duodenum, jejunum, celiac axis, renal hilum, and spleen [8, 11]. Erosions have been classified as occurring early (<6 months) or late (>6 months) [12]. Early erosions are rare and are likely a result of unrecognized gastric or esophageal injury at the time of band placement. Whether early or late, band erosion is generally an indolent process, although there have been case reports of more urgent presentation, such as with massive upper gastrointestinal hemorrhage and circulatory collapse, or with complete circumferential necrosis of the gastric wall separating the pouch from the distal stomach, requiring total gastrectomy and splenectomy for sepsis [13, 35].

Late band erosion is thought to have a variety of potential associated causes [14] including the use of nonsteroidal anti-inflammatory drugs (NSAIDs), alcohol, and tobacco, which are thought to contribute to hyperacidity and gastric mucosal irritation [15]. Another hypothesis is related to gastric wall injury during band placement or tight anterior fixation, especially around the band buckle [8, 9, 16]. The buckle may act as a lead point for gastric erosion, possibly inducing an inflammatory response in already-injured serosa [16]. Another hypothesis is that band overinflation, in association with frequent forceful vomiting, results in microscopic areas of trauma; one of this group's patients experienced band erosion several weeks after a bleeding mucosal injury from swallowing a sharp, poorly chewed portion of pizza crust. Relative differences in pressure between the balloon and the gastric wall may lead to areas of ischemia and a propensity for erosion. Because of this, it is believed that newer band models designed as high volume, low pressure systems are less likely to cause erosions compared to older low volume, high pressure models [8, 17].

Finally, operative technique may play a role in the incidence of band erosion. The original perigastric method, involving more extensive dissection around the gastric wall, was associated with increased risk of gastric injury, band migration, and erosion. At this time, the pars flaccida technique is the preferred route for band placement, and since its adoption there has been a significant decrease in the rates of both erosion and prolapse [18]. Nonetheless, most band erosions are likely multifactorial events.

A variety of symptoms may raise suspicion for band erosion, including epigastric pain, loss of restriction, weight regain, frequent vomiting, fever, or port site infection [8, 19, 20]. Inability to adequately adjust the band after several attempts may also suggest an erosion [16] although it may also be indicative of tubing puncture (Fig. 9.13).

Although emphysematous gastritis has been reported in the setting of band erosion [21], peritonitis is generally absent. In most cases, erosions occur progressively over time, allowing formation of a fibrotic capsule around the band and decreasing the risk of intra-abdominal leakage of gastric contents. This lack of signs of peritonitis may cause a delay in diagnosis.

There have been case reports of band erosion demonstrated with clear extravasation of contrast on a swallow study as seen below.

In addition, on rare occasions CT can delineate the band inside the gastric wall. In general,

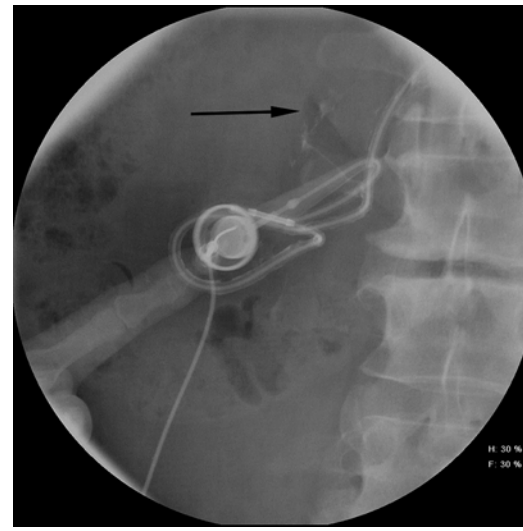

Fig. 9.13 Contrast extravasation from proximal portion of port tubing secondary to puncture, seen upon fluoroscopic band access

Fig. 9.14 Abdominal CT scan showing very subtle tissue edema in a band patient with erosion

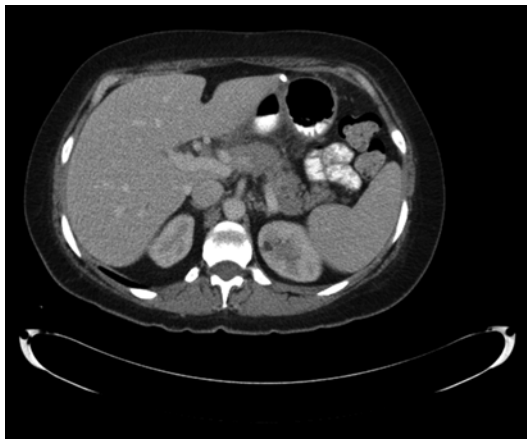

Fig. 9.15 The same patient with a small amount of fluid and subtle inflammatory stranding in the lesser sac and along band tubing

however, neither CT nor UGI will reliably diagnose erosion [17] (Figs. 9.14, 9.15, and 9.16).

If erosion is suspected, the best diagnostic modality is upper endoscopy (Figs. 9.17 and 9.18).

It is important to adequately retroflex the endoscope to fully visualize the extent of the erosion. According to the Nocca erosion classification scheme, in stage 1, a small part of the band is visible through a defect in the gastric mucosa; in stage 2, there is partial migration (50 %) of the band into the gastric lumen; and in stage 3, there is complete intragastric migration of the band [19]. There are reports of such intragastric migration, with peristaltic passage of the entire band and associated tubing into the small bowel, in one of which there was small bowel obstruction, and in the other local necrosis of the small bowel where the band had lodged [22, 23].

Fig. 9.16 Upper GI swallow study, read as "anticipated postoperative changes after laparoscopic gastric band" in a patient with erosion of about ¼ the band diameter into the gastric lumen. No extravasation of contrast is seen

Band erosion has also been diagnosed in the setting of upper or lower gastrointestinal hemorrhage [24]. Because it is an implanted medical device, the presence of a band system must always be kept in mind while generating a differential diagnosis of more common abdominal conditions. Several treatment options exist for band erosion. The choice of treatment depends on surgeon preference, patient hemodymanic stability, availability of surgical and endoscopic equipment and expertise, and extent and area of band erosion. There is general agreement that once band erosion is diagnosed, the band should be removed, and this may be accomplished via laparoscopy, laparotomy, endoscopy, or a combined laparoscopic and endoscopic approach. With an abdominal approach the band is removed, followed by any combination of primary repair of the gastric wall defect if visible, application of an

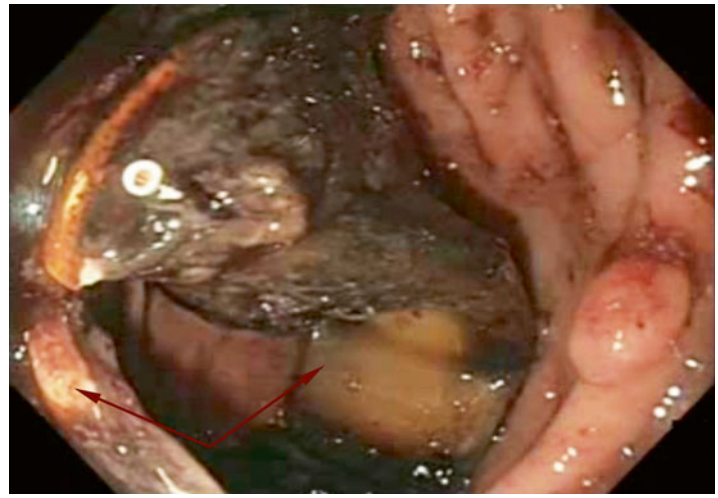

Fig. 9.17 Eroded band almost entirely within gastric lumen. *Yellow arrows* show cut edges of the transected band prior to attempted removal

Fig. 9.18 Endoscopic view of grasper drawing buckle of band upward to extract the band perorally

omental patch, methylene blue or endoscopic leak test, drain placement, or nasogastric tube decompression [22, 25]. Complete endoscopic removal of an eroded gastric band is an option in selected cases, but requires endoscopic skill and availability of specialized tools. In particular, a cutting wire is placed around a section of the eroded band, the band is transected, and it is then retrieved with a grasper through a working channel of the endoscope. Another approach includes laparoscopic creation of a distal gastrotomy, division of the eroded portion of band, and either transgastric or peroral removal of the accessible band components. The gastrotomy site is then either closed primarily or a gastrostomy tube can be inserted and removed a few weeks postoperatively [14].

Placement of another band at the time of removal of an eroded band has been reported [26], as has band replacement at a later date, but these options are at this time considered inadvisable given a very high rate of recurrent erosion, reported from 17 to 40 % [17, 27]. Conversion to another bariatric procedure at the time of eroded band explantation is also controversial. General consensus is to remove the band, allow time for the inflammatory process to resolve, and then proceed with a conversion. This also allows for appropriate patient-reeducation on a different bariatric procedure.

Along with erosion involving the band portion of the system, there are multiple reports of erosion of band tubing into a variety of organs, such as the small bowel [8]. As noted previously, solid organs are not immune from such erosion, adding complexity to band removal in this setting.

9.5 Abdominal Wall Pain

There are several potential causes for abdominal wall pain in patients who have undergone adjustable gastric banding, beyond simple postoperative incisional pain. Most patients will experience transient discomfort during band adjustments because of the necessity of using a Huber needle to access the port. A small number of patients will develop or may already have an aversion to needles, which will generally make them suboptimal band patients. This can sometimes be alleviated with premedication or with the use of local anesthetic, but a small number of patients will find that they simply cannot or will not tolerate band adjustments, thus rendering the system less than functional.

Note that because port access can sometimes be difficult depending on the thickness of the abdominal wall, where the port and tubing are placed, patient compliance, and other issues including provider inexperience, there is a small but real risk of tubing puncture during the procedure. Puncture of the tubing will render the system unfillable; this will present as rapid loss of restriction after a recent fill, and can be confirmed if immediate reaccess of the port fails to retrieve any fluid. It can be definitively diagnosed with a fluoroscopic study in which the port is accessed with a Huber needle and contrast dye is instilled; in the event of a tubing puncture, a blush of escaped dye will be noted in the surrounding tissues. Such a finding indicates that the port and a segment of tubing will need to be operatively replaced; because of this, it is key to have as much of the tubing as possible be placed in an intra-abdominal location so that it will not be punctured during adjustments. Of note, some leaks may be at the level of the band balloon, which can be inadvertently punctured during gastrogastric suturing, or may be inadvertently torn by a grasper during operative manipulation. Again, this can be diagnosed by fluoroscopic band fill, with a blush at the level of the band, and if present will necessitate replacement of the band system (Fig. 9.19).

Fig. 9.19 Fluoroscopic band fill showing blush of contrast escaping the band balloon. At operation there was a tear found in the balloon of the exact shape and size of the type of smooth grasper used during its placement

Some patients will report ongoing or new-onset pain at the access port site, and may report movement or angulation of the port as well. This may be related to suboptimal fixation of the access port on the anterior rectus fascia, or breaking of a previously placed anchoring suture, which can allow for movement of the port and may even result in complete flip of the port, rendering it inaccessible. Attachment of the port to the fascia has been addressed a number of different ways in the literature, with some surgeons using no form of fixation whatsoever and simply depositing the port into a subcutaneous pocket [28]. The majority of surgeons have affixed Lap-Band® ports to the anterior rectus fascia with strong, braided or single-stranded interrupted sutures using the existent suture holes in the port, although a mechanical port fixation device is available for the Lap-Band®. The Realize Band® is affixed to the underlying fascia with the built-in port clips that are deployed with its own mechanical port fixation device. There are also reports of attaching the port to a small segment of

mesh and placing this against or physically affixing the mesh to the fascia in order to prevent port flips [29, 30], but no technique has proven 100 % effective in preventing this complication.

Port site infection will generally present not only with pain in the area, but with erythema and sometimes frank drainage. In the early postoperative period, this may represent as a superficial infection and can be treated with a course of antibiotics; some such infections may require longer courses in order to completely resolve. With infections that fail to resolve, the access port should be removed and the band "orphaned", meaning the tubing is allowed to fall back into the abdominal cavity for later retrieval after the infection is completely cleared. At this time, the tubing is retrieved laparoscopically and the access port is placed in a different location. As discussed previously, port site infections that occur remotely from the initial placement should be assumed to arise on the basis of a band erosion.

An unusual but reported cause of port site pain is frank port disconnection, with passage of the tubing into the peritoneal cavity [31]. This also presents with weight gain and an inability to adjust the band. This complication seems to have been more common with earlier iterations of the LapBand, and requires temporary port removal during laparoscopic tubing retrieval, reattachment of the tubing to the port, and re-siting the port on the fascia.

Another potential cause of abdominal wall pain, as with any laparoscopic procedure, is port site hernia. In patients who are still obese, these may be difficult to appreciate on physical examination alone and are most commonly diagnosed with CT scanning. The most likely site of such herniation would be where the band tubing traverses the abdominal wall through a 15 mm trocar site that may not be routinely closed (Fig. 9.20).

Patients with very little subcutaneous tissue may develop thinned skin over a prominent access port; the port may actually erode through the skin, thus rendering the system no longer sterile (Fig. 9.21). This risk can be avoided in certain patients by using low-profile access ports that can be separately ordered.

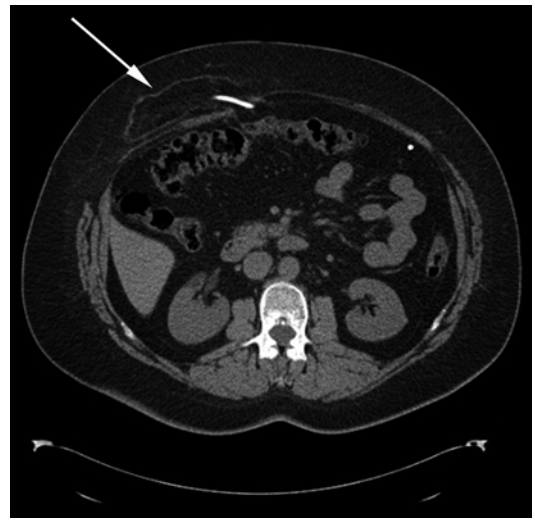

Fig. 9.20 Symptomatic herniation of omentum around the access port, passing through the tubing port site

Fig. 9.21 Visible access port three days after port placement, related to injury from sudden lifting of a large-screen television set

9.6 Undifferentiated Abdominal Pain with or Without Vomiting in Gastric Band Patients

When adjustable gastric band patients present to an emergency department with complaints of abdominal pain and/or vomiting, the band should be deflated prior the performance of radiologic studies. Even if there is a low index of suspicion that the band is the cause of the pain, it is helpful to simply take the band out of the picture while the workup proceeds. This will also decrease the risk of aspiration in the setting of bowel obstruction, and will potentially facilitate nasogastric tube placement if needed.

The presence of band fill should always be taken into consideration whenever such a patient is planning to undergo upper endoscopy, elective surgical procedures wherein postoperative nausea and vomiting might be expected, in the setting of gastroenteritis, if chemotherapy is to be administered, and in pregnant patients. Some patients ask if SCUBA diving or high altitude exposure will affect their band; as liquid is not compressible, and as most air is removed from the system during initial placement or adjustments, the odds of change in pressure affecting the band are negligible.

Some patients report increased feelings of gassiness after placement of an adjustable gastric band. This is most likely related to air swallowing while eating, and an altered ability to eructate with a band in place. Massive gastric distention has been reported [32] and responds well to nasogastric tube decompression and promotility agents. Counseling on chewing carefully, eating slowly, and possibly avoiding carbonated beverages is likely to resolve the problem.

9.7 The Odd and Unusual

Less frequent complications of adjustable gastric band placement have been reported, and despite their rarity, surgeons who place such bands must be aware of their existence. One dreaded complication is perforation of the stomach or esophagus during the blind passage of an instrument in the retrogastric space. Prior upper abdominal surgery, particularly in the region of the hiatus, or a history of upper abdominal radiation may predispose to such a risk. The key is to recognize and address this complication at the time of surgery.

It is certainly possible to injure the spleen in any operation involving work at the angle of His. Similarly, injury to the left hemidiaphragm is possible during band placement, and a report of tension pneumothorax after such an injury is cautionary [23]. Bowel obstruction from band tubing has been reported [33] but physical examination and radiologic studies may sometimes be unrevealing. Figure 9.22 shows CT evidence of obstruction related to the tubing.

Similarly, a case of mesenteric ischemia due to band tubing wrapped around the mesenteric root has been reported [34]. Band tubing that has become disconnected has also been implicated as a cause of visceral pain mistaken for acute appendicitis [31]. Given such reports, diagnostic laparoscopy should be considered a useful diagnostic and therapeutic tool in any band patient with significant unexplained abdominal pain.

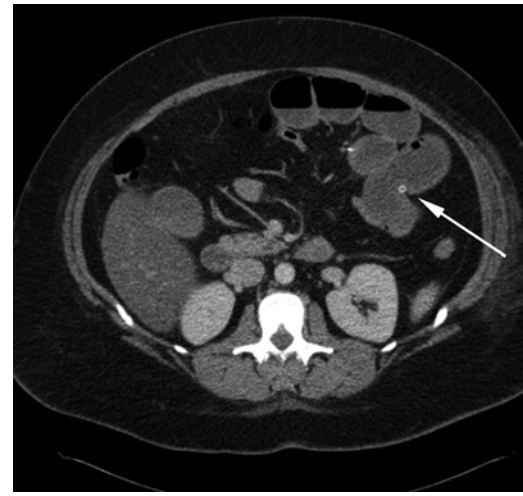

Fig. 9.22 Patient with high-grade small bowel obstruction, found to have closed loop twisted around band tubing

9.8 Conclusion

Because the adjustable gastric band has been a prevalent weight loss option in the last 20 years, despite its current waning popularity there continue to be vast numbers of band patients in this country and abroad. At some point many if not all emergency rooms and surgical services will need to provide care for such patients if they present with abdominal pain, nausea or vomiting. The subtleties of the workup and evaluation of these patients make this an area of specialization and in general such patients should be cared for by physicians with bariatric expertise. Even physicians who have not themselves ever placed a band will need to know how such bands work, what the likely diagnoses may be at presentation, and what the appropriate management entails.

References

1. http://www.accessdata.fda.gov/cdrh_docs/pdf/p000008a.pdf.
2. Allen JW. Laparoscopic gastric band complications. Med Clin N Am. 2007;91:485–97.
3. Freeman L, Brown WA, Korin A, et al. An approach to the assessment and management of the laparoscopic adjustable gastric band patient in the emergency department. EMA. 2011;23:186–94.
4. Mittermeir R, Aigner F, Obermuller S. High complication rate after Swedish adjustable gastric banding in younger patients ≤ 25 years. Obes Surg. 2009;19:446–50.
5. Fielding GA, Ren CJ. Laparoscopic adjustable gastric band. Surg Clin N Am. 2005;85:129–40.
6. Ponce J. Laparoscopic adjustable gastric banding: technique and outcomes. In: Nguyen NT et al., editors. The ASMBS textbook of bariatric surgery: Volume I: Bariatric surgery. New York: Springer Science + Business Media; 2015. doi:10.1007/978-1-4939-1206-3_16.
7. Fragkouli K, Mitselou A, Vougiouklakis T. Death-related gastric necrosis after laparoscopic adjustable gastric banding in the early post-operative period. Diagn Pathol. 2010;5:68.
8. Manatakis DK, Terzis I, Kyriazanos ID, et al. Simultaneous gastric and duodenal erosions due to adjustable gastric banding for morbid obesity. Case Rep Surg. 2014;146980:1–4.
9. Snyder B, Wilson T, Mehta S, et al. Past, present, and future: critical analysis of use of gastric bands in obese patients. Diabetes Metab Syndr Obes. 2010;3:55–65.
10. Suter M, Giusti V, Heraief E, et al. Band erosion after laparoscopic gastric banding: occurrence and results after conversion to Roux-en-Y gastric bypass. Obes Surg. 2004;14(3):381–6.
11. Cintolo JA, Levine MS, Huang S, et al. Intraluminal erosion of laparoscopic gastric band tubing into duodenum with recurrent port-site infection. J Laparoendosc Adv Surg Tech A. 2012;22(6):591–4.
12. Di Lorenzo N, Lorenzo M, Furbetta F, et al. Intragastric gastric band migration: erosion: an analysis of multicenter experience on 177 patients. Surg Endosc. 2013;27(4):1151–7.
13. Rao AD, Ramalingam G. Exsanguinating hemorrhage following gastric erosion after laparoscopic adjustable gastric banding. Obes Surg. 2006;16:1675–8.
14. El-Hayek K, Timrata P, Brethauer SA, et al. Complete endoscopic/transgastric retrieval of eroded gastric band: description of a novel technique and review of the literature. Surg Endosc. 2013;27:2974–9.
15. Abu-Abeid S, Szold A. Laparoscopic management of Lap-Band® erosion. Obes Surg. 2001;11:87–9.
16. Meir E, Van Baden M. Adjustable silicone gastric banding and band erosion: personal experience and hypotheses. Obes Surg. 1999;9:191–3.
17. Brown WA, Egberts KJ, Franke-Richard D, et al. Erosions after laparoscopic adjustable gastric banding: diagnosis and management. Ann Surg. 2013;257(6):1047–52.
18. Stroh C, Hohmann LL, Schromm H, et al. Fourteen-year long-term results after gastric banding. J Obes. 2011;128451:1–6.
19. Nocca D, Frering V, Gallix B, et al. Migration of adjustable gastric banding from a cohort study of 4236 patients. Surg Endosc. 2005;19:947–50.
20. Eid I, Birch DW, Sharma AM, et al. Complications associated with adjustable gastric banding for morbid obesity: a surgeon's guide. Can J Surg. 2011;54(1):61–6.
21. Su MZ, Munro WS. Gastric emphysema secondary to laparoscopic gastric band erosion. Int J Surg Case Rep. 2014;5:727–30.
22. Bueter M, Thalheimer A, Meyer D, et al. Band erosion and passage, causing small bowel obstruction. Obes Surg. 2006;16:1679–82.
23. Hady HR, Dadan J, Soldatow M, et al. Complications after laparoscopic gastric banding in own material. Wideochir Inne Tech Malo Inwazyjne. 2012;7(3):166–74.
24. Kirshtein B, Lantsberg L, Mizrahi S, et al. Bariatric emergencies for non-bariatric surgeons: complications of laparoscopic gastric banding. Obes Surg. 2010;20:1468–78.
25. Yoon CI, Pak KH, Kim SM. Early experience with diagnosis and management of eroded gastric bands. J Korean Surg Soc. 2012;82(1):18–27.
26. Niville E, Dams A, Vlasselaers J. Lap-band erosion: incidence and treatment. Obes Surg. 2001;11:744–7.
27. Chisholm J, Kitan N, Toouli J. Gastric band erosion in 63 cases: endoscopic removal and rebanding evaluated. Obes Surg. 2011;21(11):1676–81.

28. Wellborn JC, Wellborn SH, Wellborn T. Technique for nonfascial fixation of the laparoscopic adjustable gastric band access port. SOARD. 2010;6:429–33.
29. Randhawa S, Ghai P, Bhoyrul S. Port fixation during gastric banding: 4-year outcome using a synthetic mesh. SOARD. 2013;9:296–9.
30. Piorkowski JR, Ellner SJ, Mavanur AA, et al. Preventing port site inversion in laparoscopic adjustable gastric banding. SOARD. 2007;3:159–62.
31. Kirshtein B, Avinoach E, Mizrahi S, et al. Presentation and management of port disconnection after laparoscopic gastric banding. Surg Endosc. 2009;23:272–5.
32. Shayani V, Sarker S. Diagnosis and management of acute gastric distention following laparoscopic adjustable gastric banding. Obes Surg. 2004;14:702–4.
33. Jacob BP, Vine AJ. Abdominal pain 1 month after adjustable gastric banding: an unusual complication caused by connecting tubing. SOARD. 2010;6:554–6.
34. Daetwiler S, Adamina M, Schob O. Intractable abdominal pain following laparoscopic adjustable gastric banding. Obes Surg. 2005;15:1341–3.
35. Landen S, Majerus B, Delugeau V. Complications of gastric banding presenting to the ED. Am J Emer Med. 2006;23:368–70.

Internal Hernias: Prevention, Diagnosis, and Management

10

Britney Corey and Jayleen Grams

Key Points

- Internal hernia is a serious and potentially life-threatening complication of LRYGB, and one must maintain a high index of clinical suspicion for internal hernia in any patient status post LRYGB who presents with intermittent or acute signs or symptoms of small bowel obstruction.
- Techniques that may reduce the incidence of internal hernia after LRYGB should be used. In our practice, we implement the following: (a) antecolic antegastric positioning of the Roux limb, (b) counterclockwise rotation of the alimentary limb, (c) nondivision of the small bowel mesentery unless necessary, (d) orientation of the stapled end of the Roux limb toward the left upper quadrant, (e) omental division and placement on each side of the Roux limb, (f) a 40-cm biliopancreatic limb, and (g) routine closure of both Petersen's and mesomesenteric defects with a running nonabsorbable suture.
- Patients who have an internal hernia may present with signs or symptoms that are nonspecific and include abdominal pain, nausea or vomiting, and abdominal bloating. CT imaging can be helpful, but negative results may be found in 20 % of patients who have an internal hernia. Thus, radiologic imaging cannot exclude the presence of an internal hernia.
- Whether based on clinical suspicion or radiographic evidence, the management of internal hernias is operative repair. This is usually feasible using a laparoscopic approach. Any mesenteric defects should be closed, even when discovered incidentally during reoperation for other reasons.
- Early operation in patients with concerning symptoms is crucial, since delay in management increases morbidity and mortality.

10.1 Introduction

Since its initial introduction in 1994, laparoscopic Roux-en-Y gastric bypass (LRYGB) has continued to be the preferred bariatric operation due to its effectiveness and durability [1]. During the preceding years of open gastric bypass, common complications included wound complications and incisional hernias [2, 3]. The incidence of both of these was reduced with the laparoscopic approach. Surprisingly, the incidence of small bowel obstruction after LRYGB was increased, with the most common cause being internal hernia

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_10

accounting for 42–61 % of cases [2–6]. Although the anatomy of the operation had not appreciably changed during the conversion from open to the laparoscopic approach, it has been theorized that reduced adhesions with the laparoscopic approach resulted in the increased incidence of internal hernias [7, 8]. Retrospective studies report an internal hernia incidence of 0.3–6.2 % [9–13]. A recent study at our home institution demonstrated an overall internal hernia rate of 5 % [4]. Still, it is difficult to know the true incidence of internal hernia since many studies either have a relatively short follow-up period or do not report the follow-up interval. Furthermore, many patients may not return to the same surgeon or hospital system when presenting with a complication from bariatric surgery.

The definition of an internal hernia is "the protrusion of a viscus, most commonly small bowel, through a peritoneal or mesenteric aperture, resulting in its encapsulation within another compartment" [14]. Internal hernias may occur due to congenital defects or idiopathic mesenteric or omental defects, but many are iatrogenic as is the case for the potential mesenteric spaces created during the Roux-en-Y gastric bypass. The Roux-en-Y gastric bypass creates two or three potential spaces, depending on variation in technique (Fig. 10.1). A retrocolic Roux limb tunnels through a defect in the transverse colon mesentery, usually to the left of the middle colic vessels, and in retrogastric position to the cardiac pouch. This defect within the transverse mesocolon can enlarge over time, allowing small bowel to herniate through this space, and is the location of a mesocolic hernia. The alternative operative technique places the Roux limb anterior to the transverse colon, in antecolic fashion, and thereby eliminates the potential for a mesocolic hernia. The second potential space for an internal hernia is called Petersen's hernia, named after the German surgeon Dr. Walther Petersen who first described it in 1900. It is defined by the Roux limb mesentery and transverse mesocolon and retroperitoneum, and is created when bringing the Roux limb to the cardiac pouch to form the gastrojejunostomy [15]. It is present in both antecolic and retrocolic positioning of the Roux limb, and an internal hernia may occur on either side of the Roux limb. A third potential hernia space is created where the mesentery of the Roux limb meets with the mesentery of the biliopancreatic limb at the jejunojejunostomy. A hernia in this space is called a mesomesenteric hernia. It should also be noted that any potential gap between intestinal loops could allow internal hernias to form in spaces unrelated to the mesentery. For example, Paroz et al. reported on a new internal hernia site in the space between the two jejunal limbs at the site of the jejunojejunostomy called a jejunojejunal hernia (Fig. 10.2) [16].

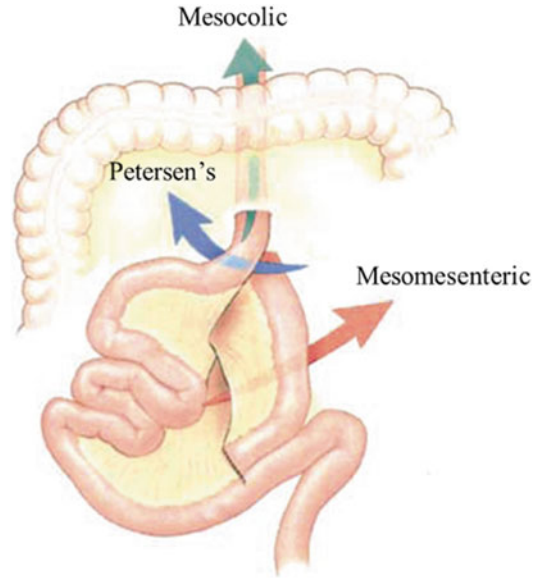

Fig. 10.1 Sites of potential internal hernia defects following LRYGB, including the mesocolic window or retrocolic tunnel (*green arrow*), Petersen's defect (*blue arrow*), and mesomesenteric or distal anastomosis defect (*red arrow*). With kind permission from Comeau E, Gagner M, Inabnet WB, Herron DM, Quinn TM, Pomp A. Symptomatic internal hernias after laparoscopic bariatric surgery. Surg Endosc. 2005;19:34–9 [38]. © Springer

10.2 Prevention

An internal hernia can be a devastating complication of LRYGB, and there has been considerable interest in operative techniques to minimize their occurrence. The two major areas of debate are antecolic vs. retrocolic positioning of the Roux limb and closure vs. nonclosure of the mesenteric

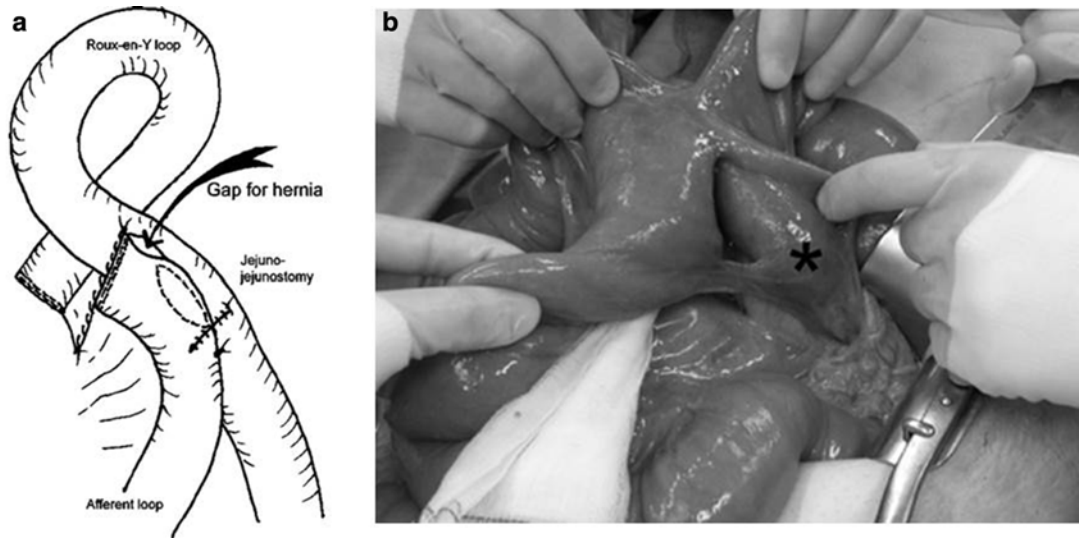

Fig. 10.2 (**a**) Schematic drawing of the jejunojejunostomy showing the location of a new type of internal hernia reported by Paroz et al. (**b**) Intraoperative photograph demonstrating the gap that has developed between the two jejunal loops. The *asterisk* denotes the end of the biliopancreatic limb. This type of internal hernia does not involve a mesenteric defect. With permission from Paroz A, Calmes JM, Romy S, Giusti V, Suter M. A new type of internal hernia after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2009;19:527–30 [16]. © Springer

defects. Regarding positioning of the Roux limb, most studies support bringing the Roux limb anterior to the transverse colon [6, 17, 18]. This has the obvious advantage of eliminating one of the potential sites of internal hernia, the mesocolic defect. Koppman et al. combined data of all LRYGB cases performed at their institution with those identified in a Medline search of the published literature to review small bowel obstruction after LRYGB in 9527 patients [6]. The overall incidence of small bowel obstruction was 3.6 % and internal hernia was the most common cause accounting for 42 % of the obstructions. When data were stratified according to position of the Roux limb, the rate of internal hernia was significantly higher after retrocolic vs. antecolic placement (2.4 % vs. 0.3 %, respectively; $p<0.0001$). A study by Escalona et al. also demonstrated a significantly higher internal hernia rate with the retrocolic vs. antecolic technique (9.3 % vs. 1.8 %, respectively; $p<0.001$), and the retrocolic position was identified as a risk factor for internal hernia on multivariate analysis ($p<0.001$) [18]. Advocates of the retrocolic approach have suggested that careful defect closure may result in a decreased internal hernia rate [19]. However, it is notable that all of the mesenteric defects were closed in both the retro- and ante-colic technique in the Escalona et al. study, yet there was still a fivefold decrease in internal hernia rate with antecolic positioning of the Roux limb [18]. Thus, overall the literature favors an antecolic gastric bypass with a retrocolic Roux limb being acceptable in patients whose anatomy does not allow the creation of a tension-free antecolic Roux limb [6, 19].

Current studies also favor the complete closure of all mesenteric defects created during LRYGB [9, 11, 12, 20–22]. Four studies comparing nonclosure with closure of mesenteric defects reported a significantly decreased number or incidence of internal hernias after closure [11, 20–22]. Iannelli et al. described an overall internal hernia incidence of 1.6 % [20]. When stratified by nonclosure or closure of mesenteric defects, there was a decrease in the rate of internal hernia from 1.9 to 0.6 %, respectively [20]. A second study from de la Cruz-Muñoz et al. demonstrated an overall incidence of 1.8 %, and a greater number of patients developed internal hernia with nonclosure of the mesenteric defect at the jejunojejunostomy ($p<0.001$) [21]. However, the authors did not report the denominator for the number of

patients in each group, so that the incidence for each group could not be calculated. Brolin and Kella saw a decrease in internal hernia rate from 2.6 to 0.5 % after changing their practice to closure of the mesenteric defect ($p=0.056$) [22]. Although the studies by de la Cruz-Muñoz et al. and Brolin and Kella support the closure of the mesomesenteric defect alone, other studies have reported significant rates of internal hernia at the mesocolic and Petersen's defects [10, 11]. Bauman et al. examined 1047 patients in their practice and found an internal hernia rate of 6.2 % at Peterson's space and 0.7 % at the mesomesenteric site [11]. The rate of internal hernia at Peterson's space decreased to 0 % after changing their practice to closure of this defect. Although several techniques have been described, the most common technique for closure of potential hernia sites is a running nonabsorbable suture, in either simple or purse-string fashion [9, 10, 23, 24]. Those who oppose closure of the defects argue that improper closure may cause tension on the anastomosis, hematomas, or injury to the mesenteric blood vessels [19]. This highlights the importance of taking care to close defects using only superficial closely spaced sutures of the mesentery to avoid injury. Given the data presented above, it must be noted that internal hernias still occur in patients who have their mesenteric defects closed but at a lower incidence. This may be due to improper closure, incomplete closure from tearing of the mesentery, or reduction in intra-abdominal fat as significant weight loss occurs, allowing the hernia spaces to expand [20].

As mentioned previously, it is difficult to know the true incidence of internal hernia with any technique, since patients may be lost to follow-up and there are different follow-up intervals between compared groups. For both antecolic vs. retrocolic positioning of the Roux limb and closure vs. nonclosure of mesenteric defects, the data cited have compared a change in technique from an earlier to later practice. This results in an inherently shorter follow-up interval for antecolic and closure of potential hernia sites groups. The Koppman et al. study reviewed papers with a range of overall follow-up from 4 to 43 months, and follow-up interval was not reported in 7 of 17 studies included [6]. The Escalona et al. study notes a median follow-up of 16 months. Neither report what the follow-up interval was for the antecolic vs. retrocolic groups separately. Similarly, the follow-up interval for patients with nonclosure vs. closure of mesenteric defects was not reported in the Iannelli et al. or de la Cruz-Muñoz et al. studies [20, 21]. The de la Cruz-Muñoz et al. group did indicate the percentage of patients at 1- and 5-year follow-up was 62 % and 60 % vs. 37 % and 30 % with nonclosure or closure, respectively [21]. The Brolin and Kella study reported a mean follow-up of 100 ± 12 months vs. 40 ± 14 months for nonclosure vs. closure groups, respectively [22]. Thus, the lower internal hernia rate in these studies could in part be attributed to the shorter follow-up interval for patients in the antecolic and closure of mesenteric defects groups. However, most literature supports the interval from LRYGB to development of internal hernia to be less than 1–3 years [4, 9, 10, 20, 22].

A recent study at our institution demonstrated a significant decrease in the rate of internal hernias with antecolic positioning of the Roux limb and closure of the mesenteric defects [4]. Like many practices, ours evolved from retrocolic positioning and nonclosure to an antecolic Roux limb and closure of both mesenteric defects. Our internal hernia rate decreased from 8.4 to 3.8 % ($p=0.005$). Median length of overall follow-up was 56 months (range, 13–113). When stratified by technique, median follow-up for the nonclosure of defects group was 73 months (range 17–113) and 41 months (range 13–90) for the closure group ($p=0.001$) [4]. Overall median time to develop an internal hernia was 22.6 months (range 3–103) months, and it was longer in the nonclosure group [33.5 months (range 10–103) vs. 16.6 months (range 3–72), respectively; $p<0.001$] supporting that we are capturing more internal hernias with longer follow-up intervals. Whether a decreased internal hernia rate using the antecolic positioning of the Roux limb with closure of defects technique would persist given a comparable length of follow-up remains to be determined.

Other surgical techniques have been proposed to decrease the incidence of internal hernia. In their study, Quebbeman and Dallal changed the orienta-

tion of the end of the Roux limb so that it faced the greater curvature of the stomach and the rate of internal hernia decreased from 9 to 0.5 % [25]. The authors theorized that the decrease in internal hernias was due to the Roux limb mesentery lying on the right side of the ligament of Treitz, with better apposition of the two mesenteries. Nandipati et al. demonstrated that rotating the Roux limb counterclockwise allowed the jejunojejunostomy to be located on the left side of the abdomen, allowing the jejunojejunostomy to lie in its more natural position on the left side of the mesenteric axis [26]. The overall internal hernia rate was 4.7 %. When stratified by rotation of the Roux limb, there was a significant decrease in the incidence of internal hernias with counterclockwise vs. clockwise rotation (0.7 % vs. 6.9 %, respectively; $p=0.0018$). According to the authors, counterclockwise rotation also makes the mesenteric defect easier to close completely. Other methods described include minimal division or nondivision of the small bowel mesentery, division of the omentum with tucking the bisected omentum to each side of the Roux limb, creation of a long jejunojejunostomy, placement of the jejunojejunostomy above the colon in the left upper quadrant, and a shorter biliopancreatic limb [11, 13, 17, 20, 27].

Because an internal hernia is a potentially devastating complication, we recommend using the strategies discussed above to minimize the occurrence. In our practice, we implement the following techniques: (a) antecolic antegastric positioning of the Roux limb, (b) counterclockwise rotation of the alimentary limb, (c) nondivision of the small bowel mesentery unless necessary, (d) orientation of the stapled end of the Roux limb toward the left upper quadrant, (e) omental division and placement on each side of the Roux limb, (f) a 40-cm biliopancreatic limb, and (g) routine closure of both Petersen's and mesomesenteric defects with a running nonabsorbable suture (Figs. 10.3 and 10.4).

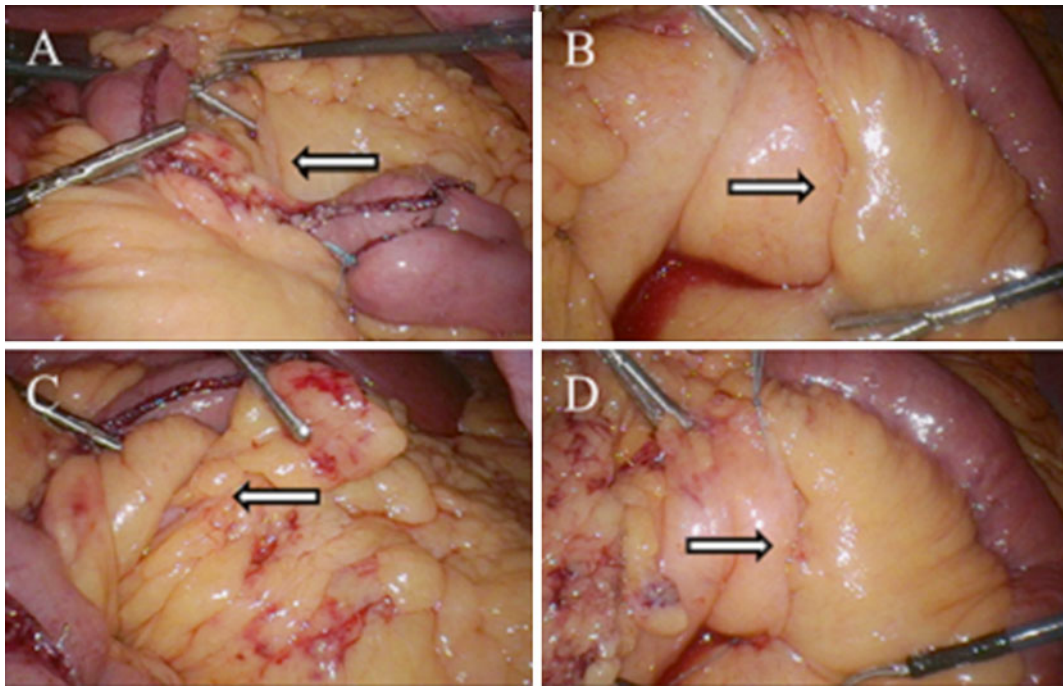

Fig. 10.3 Intraoperative photos during LRYGB demonstrating (**a**) the leaflets of mesocolon and Roux limb mesentery (Petersen's defect) as seen from the left side of the patient (*arrow*), (**b**) Petersen's defect as viewed from the right side of the patient (*arrow*), (**c**) Peterson's space as viewed from the left with *arrow* demonstrating fat and small bowel attempting to herniate through the defect (*arrow*), (**d**) closure of Petersen's defect from the patient's right side with running, nonabsorbable suture (*arrow*). Photos kindly provided by Dr. Richard Stahl

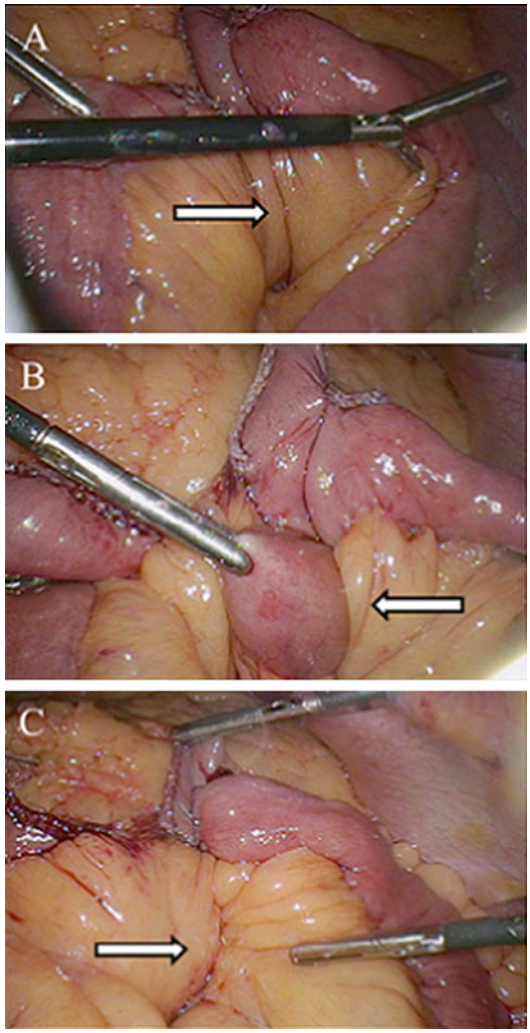

Fig. 10.4 Intraoperative photos during LRYGB demonstrating (**a**) the mesenteric defect created at the jejunojejunal anastomosis (*arrow*), (**b**) bowel herniating through this site (*arrow*), and (**c**) closure of the mesomesenteric defect with running, nonabsorbable suture (*arrow*). Photos kindly provided by Dr. Richard Stahl

10.3 Diagnosis

Diagnosing an internal hernia in a patient after LRYGB can be challenging, since many patients with a symptomatic internal hernia have nonspecific complaints of abdominal pain, nausea, and vomiting [4, 9, 11]. The clinical history of the patient often contributes to the differential diagnosis. While internal hernias may occur at any time following an operation, patients who have recently undergone their operation are more likely to suffer from an anastomotic leak or adhesive disease [28]. Anastomotic strictures may also present with postprandial fullness, nausea and vomiting, and abdominal pain [6]. Patients who have greater weight loss and are at least 1–3 years from LRYGB are thought to be at highest risk for internal hernia. Greater weight loss is thought to result in enlargement of mesenteric defects due to loss of intraperitoneal fat, thereby increasing the risk of internal hernia [28]. Abdominal examination and laboratory evaluation may be unrevealing as well [9, 29]. Imaging can be very helpful in diagnosing an internal hernia and is best performed when the patient is having symptoms, since some internal hernias may spontaneously reduce and recur, leading to intermittent pain [30]. It is crucial to remember that imaging may be negative in 20 % of patients [4, 9].

Findings consistent with the presence of internal hernia on upper GI series and CT imaging have been described in Roux-en-Y gastric bypass patients [28, 31]. In the Blachar et al. study, there was considerable overlap in a comparison of findings on upper GI series in patients with adhesions vs. internal hernia as the cause of small bowel obstruction, leading the authors to conclude that a specific cause of small bowel obstruction could not be made using this modality [28]. Findings of small bowel obstruction and distended small bowel segments > 2.5 cm were both present in 100 % of patients with adhesions vs. internal hernia. A diagnosis of internal hernia was favored with the finding of a cluster of dilated loops of small bowel located in the left upper or middle abdomen, which remained high in the abdomen with the patient in erect position. Ahmed et al. found that upper GI series had a positive finding suggestive of internal hernia in 65 % of their patients [31]. The four most recurring findings were dilated fluid-filled loops of small bowel, redundant Roux limb in the lesser sac, a preponderance of small bowel loops in the left upper quadrant, and slow emptying of contrast with prolonged transit times.

CT imaging has emerged as the preferred imaging modality in gastric bypass patients who are having symptoms of small bowel obstruction.

There are many reasons for this. First, CT imaging typically has less technical difficulties than may be encountered when performing an upper GI series on a patient with obesity, such as difficult positioning or poor image quality [31, 32]. CT imaging often provides more rapid diagnostic information in the acute setting, and it is more widely available since some facilities may not have qualified staff available to perform upper GI series at night and on weekends. Additionally, upper GI series is a dynamic study. Review and interpretation by the surgeon is dependent on the images captured by the radiology team. In contrast, CT images are more readily interpreted by surgeons, and review by a bariatric surgeon may improve the diagnostic yield [33]. Finally, CT imaging is more sensitive and specific than other reported imaging techniques.

A number of CT findings have been described in the literature: (a) swirled appearance of the vessels and fat at the mesenteric root or "swirled mesentery" (Fig. 10.5), (b) a mushroom shape of the herniated mesenteric root or "mushroom sign" (Fig. 10.6), (c) tubular or round shape of the distal mesenteric fat closely surrounded by bowel loops known as the "hurricane eye" (Fig. 10.7), (d) findings of small bowel obstruction including dilated small bowel, dilated Roux limb, finding of a transition point to nondilated or collapsed bowel, or dilated biliopancreatic limb (Fig. 10.8), (e) small bowel other than duodenum behind the superior mesenteric artery or vein (Fig. 10.9), (f) displaced jejunojejunostomy (Fig. 10.10), (g) clustered loops of small intestine (Fig. 10.11), (h) altered course of the superior mesenteric artery or vein, (i) distended gastric remnant (Fig. 10.12), (j) widening of the jejunojejunostomy, and (k) engorgement of mesenteric lymph nodes [30, 32, 34]. Marchini et al. reviewed CT images from 34 patients who had documented internal hernias at the time of exploration. The most common CT finding was clustered small bowel loops (79.4 %), followed by small bowel obstruction (73.5 %), swirled mesentery (64.7 %), altered course of the superior mesenteric artery or vein (61.8 %), and the finding of a transition point (58.8 %) [32]. Both Lockhart et al. and Iannuccilli et al. identified swirled mesentery as the best single predictive sign with sensitivity of 61–100 % and specificity of 67–94 % [30, 34]. The degree of swirl was important with a median amount of swirl <90° in patients found to have mesenteric swirl but no internal hernia at exploration; in cases of ≥270° mesenteric swirl, all patients were found to have an internal hernia [30]. The mushroom sign and hurricane eye each had low sensitivity but high specificity. Iannuccilli et al. also reviewed CT images for engorged mesenteric lymph nodes. This finding had moderate sensitivity (44–89 %) and high specificity (90–100 %) for the presence of an internal hernia, presumably due to lymphatic obstruction from mesenteric torsion [34]. Other CT findings have been described for mesocolic internal hernias such as clustered loops of small intestine cephalad to the transverse colon in the left upper quadrant and a high position of the jejunojejunostomy at the level of the hernia defect or the gastrojejunostomy [35].

While radiographic imaging studies can be very useful, we again emphasize that 20 % of patients had no CT evidence of internal hernia in two separate studies (4. 9). Thus, normal radiological studies do not exclude the presence of an internal hernia. If the patient is not sick, a second imaging test could be ordered. In the study by Ahmed et al., CT imaging was negative for findings of internal hernia in three patients, and two of these patients went on to have upper GI series. In both patients, upper GI series had findings suggestive of internal hernia [31]. However, delay in the diagnosis of an internal hernia can have fatal consequences, and it is crucial to have a high level of clinical suspicion and early involvement of a general surgeon familiar with the care of bariatric patients [8, 29, 33]. Signs and symptoms of internal hernia are related to whether the hernia is incarcerated or strangulated. As signs and symptoms progress, there is a higher risk of small bowel ischemia and/or perforation and a significantly higher risk of mortality. Early diagnosis and intervention is critical. If clinical suspicion remains high in the absence of diagnostic evidence, early intervention with operation is indicated for diagnosis and management.

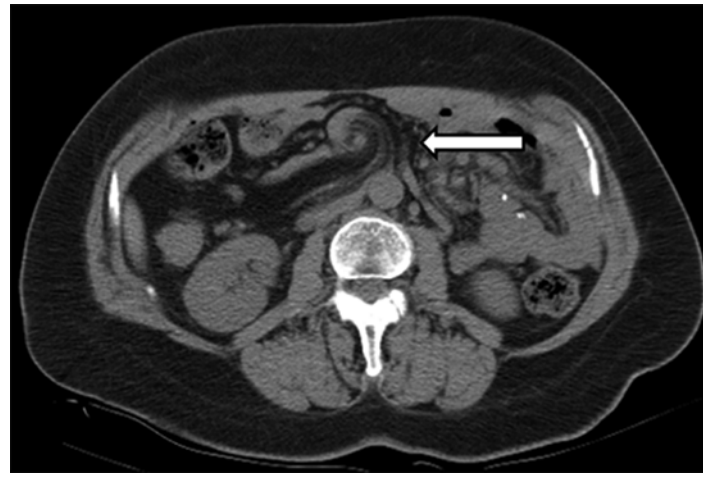

Fig. 10.5 CT scan with *arrow* pointing to the swirled appearance of mesenteric fat or vessels at the root of the mesentery, referred to as the "mesenteric swirl" sign

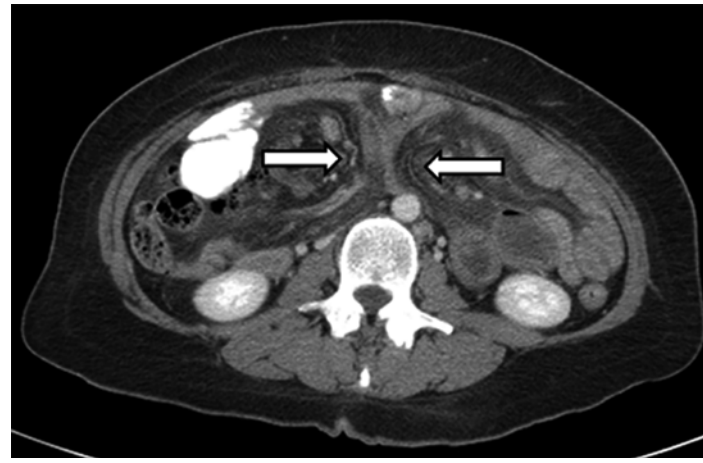

Fig. 10.6 *Arrows* point to the "mushroom sign," a typical appearance of an internal hernia on CT scan due to crowding or stretching of the vessels of the mesenteric root as they travel through the hernia

Fig. 10.7 The "hurricane eye" sign refers to the tubular shape of the distal mesenteric fat with surrounding bowel (*arrows*). Reprinted with permission from Iannuccilli JD, Grand D, Murphy BL, Evangelista P, Roye GD, Mayo-Smith W. Sensitivity and specificity of eight CT signs in the preoperative diagnosis of internal mesenteric hernia following Roux-en-Y gastric bypass surgery. Clin Radiol. 2009;64:373–80 [34]. © Elsevier

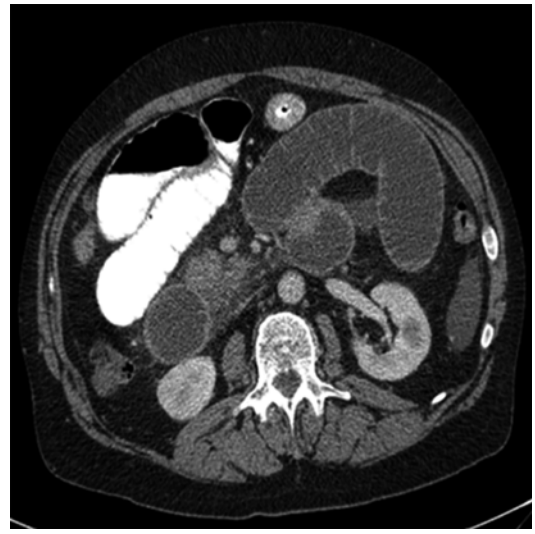

Fig. 10.8 Evidence of small bowel obstruction on CT imaging in a patient with previous LRYGB. The collapsed Roux limb is seen passing anterior to the dilated small bowel

Fig. 10.9 A CT scan demonstrating small bowel behind the superior mesenteric artery. The *arrows* point to a segment of bowel which is thin and stretched. Reprinted with permission from Iannuccilli JD, Grand D, Murphy BL, Evangelista P, Roye GD, Mayo-Smith W. Sensitivity and specificity of eight CT signs in the preoperative diagnosis of internal mesenteric hernia following Roux-en-Y gastric bypass surgery. Clin Radiol. 2009;64:373–80 [34]. © Elsevier

Fig. 10.10 *Arrow* points to the right sided jejunojejunal anastomosis in a patient with an internal hernia who underwent CT imaging. This is concerning for internal hernia as this anastomosis is routinely made on the left side of the abdomen. Reprinted with permission from Iannuccilli JD, Grand D, Murphy BL, Evangelista P, Roye GD, Mayo-Smith W. Sensitivity and specificity of eight CT signs in the preoperative diagnosis of internal mesenteric hernia following Roux-en-Y gastric bypass surgery. Clin Radiol. 2009;64:373–80 [34]. © Elsevier

Fig. 10.11 Clustered loops of small bowel marked with an *arrow* on CT scan. An internal hernia was confirmed at the time of operative exploration

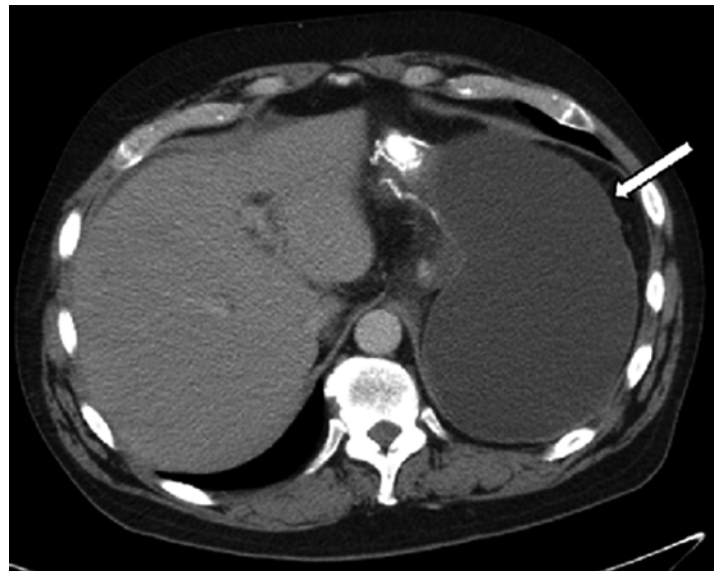

Fig. 10.12 A dilated, fluid-filled gastric remnant shown on CT scan. Internal hernia was confirmed at operative exploration

10.4 Management

Whether based on clinical suspicion or radiographic evidence, the management of internal hernias is operative repair. Operative intervention may be elective, urgent, or emergent depending on the clinical status of the patient. Regarding elective management, patients with intermittent abdominal pain and/or nausea may have episodic internal hernias that spontaneously reduce, and operation should be recommended [9, 36]. Gandhi et al. reported that small bowel obstruction due to internal hernia is typically preceded by symptoms of intermittent obstruction. They called these symptoms "herald symptoms," comprising intermittent abdominal pain associated with bloating and nausea suggestive of transient small bowel obstruction [36]. They identified 11 patients who had these herald symptoms, and they recommended elective operation to all 11 patients [36]. Nine patients agreed to operation and all were found to have an internal hernia. The two patients who initially refused operation later underwent emergent operation for small bowel obstruction and both were found to have an internal hernia. Small bowel volvulus was found in 4

of these 11 patients, including 3 of the 9 who underwent elective operation. Thus, even under these "elective" circumstances, operative intervention should be expeditious.

If possible, operative management of patients with an internal hernia should be attempted through a laparoscopic approach. Higa et al. demonstrated that the majority of patients with internal hernias could be successfully reduced and repaired using three or four of the original laparoscopic port sites, especially in the elective setting [9]. Of 63 patients with an internal hernia, only 5 patients required open repair for severe bowel distention, peritonitis, confusing anatomy, or enteric spillage. Of 26 cases of internal hernia, Elms et al. repaired all of them laparoscopically [12]. In our own practice, which includes surgeons who favor both laparoscopic and open approaches, 39 cases (86.7 %) were repaired laparoscopically [4]. A case report of a laparoscopic single-incision repair of internal hernia defects has also been described [37].

Regardless of the operative approach used, the conduct of the operation includes running the entire small bowel, starting at a fixed point, such as the ligament of Treitz or the terminal ileum [23]. It is usually easiest to identify and start at the ileocecal valve then to proceed from distal to proximal in running the small bowel, since the distal portion of the intestine will be decompressed, easier to manipulate, and usually in normal anatomic position. If starting at the ligament of Treitz, it can be difficult to sort out the Roux limb from the biliopancreatic limb from the common channel and to determine which portion of the small intestine has herniated, especially since this bowel is typically dilated and more easily injured. All potential hernia sites need to be examined, and after reduction they should be closed with a nonabsorbable suture in running or purse-string fashion. Importantly, when mesenteric defects are found incidentally at the time of another surgical procedure, they should be closed.

The literature suggests that internal hernia may present with acute small bowel obstruction requiring emergency operation in 40–50 % of patients [4, 9]. The most important decision to be made in the management of a suspected internal hernia is when to proceed to the operating room, since a delay in diagnosis can quickly become life threatening [4, 9, 36]. Gandhi et al. found that some element of delay occurred in virtually all patients who developed severe complications after operation for small bowel obstruction [36–38]. Three patients required resection of small bowel for an internal hernia with strangulation and prolonged ICU stays, and all had a delay of >48 h from admission to another medical center to operation. In our study, the single mortality was in a patient who presented in extremis and fulminant liver failure after being hospitalized elsewhere with the incorrect diagnosis [4]. Thus, the most important principle guiding management of internal hernia after LRYGB is to maintain a high index of clinical suspicion and to have a low threshold for operative intervention. Again, whether based on clinical suspicion or radiographic evidence, the management of internal hernias is operative repair.

References

1. Wittgrove AC, Clark GW, Tremblay LJ. Laparoscopic gastric bypass, Roux-en-Y: preliminary report of five cases. Obes Surg. 1994;4:353–7.
2. Nguyen NT, Goldman C, Rosenquist CJ, Arango A, Cole CJ, Lee SJ, Wolfe BM. Laparoscopic versus open gastric bypass: a randomized study of outcomes, quality of life, and costs. Ann Surg. 2001;234:279–91.
3. Jones KB, Afram JD, Benotti PN, Capella RF, Cooper CG, Flanagan L, Hendrick S, Howell LM, Jaroch MT, Kole K, Lirio OC, Sapala JA, Schuhknecht MP, Shapiro RP, Sweet WA, Wood MH. Open versus laparoscopic Roux-en-Y gastric bypass: a comparative study of over 25,000 open cases and the major laparoscopic bariatric reported series. Obes Surg. 2006;16:721–7.
4. Obeid A, McNeal S, Breland M, Stahl R, Clements RH, Grams J. Internal hernia after laparoscopic Roux-en-Y gastric bypass. J Gastrointest Surg. 2014;18:250–6.
5. Husain S, Ahmed AR, Johnson J, Boss T, O'Malley W. Small-bowel obstruction after laparoscopic Roux-en-Y gastric bypass: etiology, diagnosis, and management. Arch Surg. 2007;142:988–93.
6. Koppman JS, Li C, Gandsas A. Small bowel obstruction after laparoscopic Roux-en-Y gastric bypass: a review of 9527 patients. J Am Coll Surg. 2008;206:571–84.

7. Champion JK, Williams M. Small bowel obstruction and internal hernias after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2003;13:596–600.
8. Garza Jr E, Kuhn J, Arnold D, Nicholson W, Reddy S, McCarty T. Internal hernias after laparoscopic Roux-en-Y gastric bypass. Am J Surg. 2004;188:796–800.
9. Higa KD, Ho T, Boone KB. Internal hernias after laparoscopic Roux-en-Y gastric bypass: incidence, treatment and prevention. Obes Surg. 2003;13:350–4.
10. Ahmed RA, Rickards G, Husain S, Johnson J, Boss T, O'Malley W. Trends in internal hernia incidence after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2007;17:1563–6.
11. Bauman RW, Pirrello JR. Internal hernia at Petersen's space after laparoscopic Roux-en-Y gastric bypass: 6.2 % incidence without closure–a single surgeon series of 1047 cases. Surg Obes Relat Dis. 2009;5:565–70.
12. Elms L, Moon RC, Varnadore S, Teixeira AF, Jawad MA. Causes of small bowel obstruction after Roux-en-Y gastric bypass: a review of 2395 cases at a single institution. Surg Endosc. 2013;28:1624–8.
13. Ortega J, Cassinello N, Sánchez-Antúnez D, Sebastián C, Martínez-Soriano F. Anatomical basis for the low incidence of internal hernia after a laparoscopic Roux-en-Y gastric bypass without mesenteric closure. Obes Surg. 2013;23:1273–80.
14. Blachar A, Federle MP. Internal hernia: an increasingly common cause of small bowel obstruction. Semin Ultrasound CT. 2000;23:174–83.
15. Faria F, Preto J, Oliveira M, Pimenta T, Baptista M, Costa-Maia J. Petersen's space hernia: a rare but expanding diagnosis. Int J Surg Case Rep. 2011;2:141–3.
16. Paroz A, Calmes JM, Romy S, Giusti V, Suter M. A new type of internal hernia after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2009;19:527–30.
17. Cho M, Pinto D, Carrodeguas L, Lascano C, Soto F, Whipple O, Simpfendorfer C, Gonzalvo JP, Zundel N, Szomstein S, Rosenthal RJ. Frequency and management of internal hernias after laparoscopic antecolic antegastric Roux-en-Y gastric bypass without division of the small bowel mesentery or closure of mesenteric defects: review of 1400 consecutive cases. Surg Obes Relat Dis. 2006;2:87–91.
18. Escalano A, Devaud N, Pérez G, Crovari F, Boza C, Viviani P, Ibáñez L, Guzmán S. Antecolic versus retrocolic alimentary limb in laparoscopic Roux-en-Y gastric bypass: a comparative study. Surg Obes Relat Dis. 2007;3:423–7.
19. Miyashiro LA, Fuller WD, Ali MR. Favorable internal hernia rate achieved using retrocolic, retrogastric alimentary limb in laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2010;6:158–62.
20. Iannelli A, Buratti MS, Novellas S, Dhaman M, Amor IB, Sejo E, Facchiano E, Addeo P, Gugenheim J. Internal hernia as a complication of laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2007;17:1283–6.
21. de la Cruz-Muñoz N, Cabrera JC, Cuesta M, Hartnett S, Rojas R. Closure of mesenteric defect can lead to decrease in internal hernias after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2011;7:176–80.
22. Brolin RE, Kella VN. Impact of complete mesenteric closure on small bowel obstruction and internal mesenteric hernia after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2013;9:850–4.
23. Aghajani E, Jacobsen HJ, Nergaard BJ, Hedenbro JL, Leifson BG, Gislason H. Internal hernia after gastric bypass: a new and simplified technique for laparoscopic primary closure of the mesenteric defects. J Gastrointest Surg. 2012;16:641–5.
24. Walker AS, Bingham JR, Causey MW, Sebesta JA. Mesenteric irritation as a means to prevent internal hernia formation after laparoscopic gastric bypass surgery. Am J Surg. 2014;207:739–41.
25. Quebbemann BB, Dallal RM. The orientation of the antecolic Roux limb markedly affects the incidence of internal hernias after laparoscopic gastric bypass. Obes Surg. 2005;15:766–70.
26. Nandipati KC, Lin E, Husain F, Srinivasan J, Sweeney JF, Davis SS. Counterclockwise rotation of Roux-en-Y limb significantly reduces internal herniation in laparoscopic Roux-en-Y gastric bypass (LRYGB). J Gastrointest Surg. 2012;16:675–81.
27. Madan AK, Menzo EL, Dhawan N, Tichansky DS. Internal hernias and nonclosure of mesenteric defects during laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2009;19:549–52.
28. Blachar A, Federle MP, Pealer KM, Ikramuddin S, Schauer P. Gastrointestinal complications of laparoscopic Roux-en-Y gastric bypass surgery: clinical and imaging findings. Radiology. 2002;223:625–32.
29. Filip JE, Mattar SG, Bowers SP, Smith CD. Internal hernia formation after laparoscopic Roux-en-Y gastric bypass for morbid obesity. Am Surg. 2002;68:640–3.
30. Lockhart ME, Tessler FN, Canon CL, Smith JK, Larrison MC, Fineberg NS, Roy BP, Clements RH. Internal hernia after gastric bypass: sensitivity and specificity of seven CT signs with surgical correlation and controls. Am J Roentgenol. 2007;188:745–50.
31. Ahmed RA, Rickards G, Johnson J, Boss T, O'Malley W. Radiological findings in symptomatic internal hernias after laparoscopic gastric bypass. Obes Surg. 2009;19:1530–5.
32. Marchini AK, Denys A, Paroz A, Romy S, Suter M, Desmartines N, Meuli R, Schmidt S. The four different types of internal hernia occurring after laparoscopic Roux-en-Y gastric bypass performed for morbid obesity: are there any multidetector computed tomography (MDCT) features permitting their distinction? Obes Surg. 2011;21:506–16.

33. Onopchenko A. Radiological diagnosis of internal hernia after Roux-en-Y gastric bypass. Obes Surg. 2005;15:606–11.
34. Iannuccilli JD, Grand D, Murphy BL, Evangelista P, Roye GD, Mayo-Smith W. Sensitivity and specificity of eight CT signs in the preoperative diagnosis of internal mesenteric hernia following Roux-en-Y gastric bypass surgery. Clin Radiol. 2009;64:373–80.
35. Reddy SA, Yang C, McGinnis LA, Seggerman RE, Garza E, Ford III KL. Diagnosis of transmesocolic internal hernia as a complication of retrocolic gastric bypass: CT imaging criteria. Am J Roentgenol. 2007;189:52–5.
36. Gandhi AD, Patel RA, Brolin RE. Elective laparoscopy for herald symptoms of mesenteric/internal hernia after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5:144–9.
37. Tymitz K, Steele K, Schweitzer M. Laparoscopic single-incision repair of internal hernia defects using an intracorporeal suturing technique. Surg Obes Relat Dis. 2011;7:778–80.
38. Comeau E, Gagner M, Inabnet WB, Herron DM, Quinn TM, Pomp A. Symptomatic internal hernias after laparoscopic bariatric surgery. Surg Endosc. 2005;19:34–9.

Marginal and Peptic Ulcers: Prevention, Diagnosis, and Management

11

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Key Points

- Marginal ulcers are ulcers that occur at or near the gastrojejunostomy in patients following Roux-en-Y gastric bypass. They occur after 0.6–25 % of laparoscopic Roux-en-Y gastric bypasses [3].
- The exact pathophysiology of marginal ulcers remains unknown, but theories include smoking, nonsteroidal anti-inflammatory drug use, *Helicobacter pylori* infection, inappropriate surgical anatomy, or foreign body reaction to suture or staples.
- Diagnosis of marginal and peptic ulcers relies heavily on endoscopy. Endoscopic treatments are also important for ulceration due to reversible causes.
- Treatment of marginal ulcers is first medical. Smoking and NSAID cessation are necessary. Eradication of *H. pylori* infection should be completed. High dose proton pump inhibitors, H2-blockade, or sucralfate therapy should be initiated.

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11.1 Introduction

Bariatric and metabolic surgery continues to increase in use throughout the world, and especially the USA, as a treatment for obesity and its comorbidities. The American Society for Metabolic and Bariatric Surgery estimates that about 180,000 bariatric surgeries were completed in the USA in 2013 [1]. This is a gradual increase from about 160,000 surgeries completed in 2011. Of those surgeries completed in 2013, about 35 % were Roux-en-Y gastric bypasses. This is a decrease in the percentage compared to previous years but still a substantial number. Laparoscopic Roux-en-Y gastric bypass (LRYGB) techniques and results were first reported in 1994 by Wittgrove et al. and since then have continued to increase in use due to successful weight loss with minimal morbidity and mortality [2].

Marginal ulceration can occur after any operation in which the small intestine is anastomosed to the stomach. A marginal ulcer is defined as an ulceration at or near the gastrojejunostomy and may also be referred to as an anastomotic ulcer or ischemic ulcer. Initial experience with marginal ulceration occurred in patients who underwent partial gastrectomy with gastrojejunostomy reconstruction, such as antrectomy with Billroth II reconstruction. Roux-en-Y gastric bypass (RYGB) is now far more common than Billroth II reconstruction.

D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_11

11.2 Incidence

The reported incidence of marginal ulceration with LRYGB varies significantly. In a systematic review, Coblijn et al. found the incidence of MU to range from 0.6 to 25 % [3]. Rasmussen et al. in a retrospective review of 260 consecutive patients had a total of 19 patients (7 %) develop symptomatic marginal ulcers (MU) [4]. These were diagnosed using endoscopy on patients who presented with epigastric pain, persistent nausea or vomiting, or gastrointestinal hemorrhage. The study only evaluated patients with epigastric symptoms and did not include any asymptomatic patients.

Csendes et al. performed a prospective study, completing endoscopy on 441 gastric bypass patients 1 month after surgery and repeat endoscopy at 17 months after surgery [5]. Of these, 315 patients completed the study with endoscopy at 17 months. All patients underwent endoscopy without consideration for symptoms. Twenty-five of the 441 patients (5.6 %) who underwent endoscopy at 1 month were found to have MUs. These patients had undergone a mixture of open resectional RYGB and LRYGB with the remnant stomach left in place. A total of 360 patients underwent open resectional RYGB with 15 developing MUs at 1 month (4.1 %) and 81 patients underwent LRYGB with 10 developing MUs at 1 month (12.3 %). At 17 months after surgery, only two marginal ulcers were found. One was a recurrent ulcer and the second a new ulcer.

Marginal ulcers occur in both the early and late postoperative periods. The early postoperative period is defined as within 3 months of surgery and the late postoperative period occurs 12 months after surgery [5]. Csendes et al. found most marginal ulcers 1 month following gastric bypass and very few ulcers at repeat endoscopy at 17 months post-operation [5]. Dallal et al. followed 201 consecutive laparoscopic gastric bypass surgeries [6]. Seven patients developed marginal ulcer with development of symptoms between 3 and 14 months postoperatively.

Another study followed gastric bypass patients who developed symptoms that required upper endoscopy. 328 symptomatic patients underwent endoscopy and 112 patients were found to have MUs. Fifty-nine of the patients developed symptoms within 12 months after surgery and 53 of the patients developed symptoms more than 12 months after surgery. Specifically, MUs were diagnosed within 3 months of surgery in 30 %, between 4 and 12 months from surgery in 23 %, and after 12 months from surgery in 47 % of patients [6]. With the large amount of variability found in the literature concerning their time of presentation following surgery, marginal ulcers should always remain on the differential diagnosis for a patient presenting with epigastric pain, nausea, vomiting, or gastrointestinal hemorrhage.

11.3 Presentation

Marginal ulcers present with a constellation of symptoms [7]. Epigastric pain is by far the most common presenting symptom, occurring in more than half of patients with MUs [8, 9]. The pain can be associated with dysphagia or nausea and vomiting. Both dysphagia and nausea and vomiting may present as independent symptoms as well. Rarer presenting symptoms include increasing dyspepsia and reflux. Some patients may begin to regain weight if eating certain high calorie foods helps to relieve the pain. This may also be an indicator of a fistula from the gastric pouch to the gastric remnant. Conversely, some patients will present with failure to thrive due to pain with eating or obstructive symptoms from edema associated with the ulceration. Significantly, patients may develop marginal ulcers and remain asymptomatic. In the previously mentioned report by Csendes, they found 7 patients out of 25 who had marginal ulcers were without symptoms [5].

As marginal ulcers progress, additional complications may occur, such as bleeding, perforation, or obstruction. In some patients, these may be the presenting symptoms of the ulcer [7]. In a review of 777 patients found to have MUs, 117 (15.1 %) presented with bleeding as their main symptom [3]. Another series of 103 patients found bleeding as the presenting symptom in 24 % of patients [10]. Bleeding from MUs may occur from erosion of the ulcer

into vasculature of the Roux limb or erosion completely through the bowel wall into surrounding vasculature, such as the splenic artery [11].

Perforation represents an extremely serious presentation or complication of MUs. The symptoms will be that of perforated viscus, including tachycardia, fever, and severe epigastric pain. Leukocytosis will likely occur and free air will be seen on plain films or computed tomography [9]. Felix et al., in their review of 3430 LRYGB patients, found that perforated MUs occurred in 35 (1 %) of patients [12]. With early recognition and prompt surgical therapy, most cases of perforated MUs can be treated laparoscopically [13].

11.4 Pathophysiology

While factors predisposing patients to MUs have not been completely elucidated, the origin is likely multifactorial [14]. They typically occur on the jejunal side of the gastrojejunal anastomosis. The jejunum, unlike the duodenum, does not typically see acidic fluid in its normal anatomic orientation and lacks protective mechanisms against acid, such as mucin and bicarbonate [15]. An increase in acid production, the breakdown of mucosal defenses, or nuances about the surgical procedure may all be potential contributing factors.

Tobacco use has been studied extensively as a possible cause of MUs. In peptic ulcer disease, it has been shown to increase acid production through several mechanisms, decrease mucosal prostaglandin synthesis, and impair ulcer healing [16]. In LRYGB patients with MUs, evidence has not been as clear. Wilson et al. reviewed endoscopic findings of LRYGB patients who presented with upper gastrointestinal symptoms. In those patients with MUs, cigarette smoking significantly increased the risk with an adjusted odds ratio of 30.6 [17]. Another series confirmed these results when they found smoking increased the risk of MUs with an odds ratio of 2.5 [10]. Despite conflicts in the literature of the role of smoking on MU formation, tobacco use may increase the risk of perforation of MUs [12]. El Hayek et al. found that history of tobacco use or current use did not increase the risk of developing MUs but did predispose to non-healing ulcers [7]. Rasmussen et al. contradict these results in their series of 260 patients with only one patient using tobacco developing MUs [4].

Alcohol use has been postulated to contribute to the formation of MUs. Many studies have failed to find a correlation. In their study of 112 patients with MUs, El Hayek et al. did not find that alcohol was a significant risk factor [7]. Though it is likely alcohol has some contribution to ulceration, more studies are needed to show the effect.

Another modifiable risk factor for the development of MUs is the use of nonsteroidal anti-inflammatory drugs (NSAIDs). In the non-bariatric population, NSAIDs have long been identified as a risk factor for the development of ulcers as they reduce the mucosal defense to acid through inhibition of cyclo-oxygenase 1. With these known risks, the American Society for Metabolic and Bariatric Surgery (ASMBS) recommends avoiding NSAIDs postoperatively in all bariatric surgery patients, not just LRYGB patients [18]. NSAID use does not guarantee ulcer formation. Garrido et al. evaluated 118 patients following LRYGB. Ten of these patients used NSAIDs intermittently, and none of these patients went on to develop MUs [19]. Rasmussen et al. only showed a small percentage, 11 %, of the patients with MUs used NSAIDs [4]. When patients develop ulcers, using NSAIDs likely increases the risk of perforation [12]. NSAIDs should be avoided in all LRYGB patients to prevent the development of MUs and discontinued in all patients who develop MUs.

Since the work of Dr. Barry Marshall and Dr. Robin Warren, *Helicobacter pylori* has been a suspected cause for all foregut mucosal ulcerations. The mechanism by which *H. pylori* causes or contributes to MU formation is still under investigation. It is known that *H. pylori* leads to a cytokine mediated inflammatory response, leading to gastritis, intestinal metaplasia, and ulcer formation. Its prevalence within the general population varies by geographic location, socioeconomic status, and age. In developing countries, the prevalence of infection is estimated at greater

than 80 % and ranging from 20 to 50 % in industrialized countries. Papasavas and colleagues studied LRYGB patients preoperatively in Pennsylvania and found an *H. pylori* incidence of 22.4 %, which is similar to the rate seen in non-obese patients [20]. Rasmussen et al. showed that *H. pylori* infection may predispose LRYGB patients to MU formation. However, in their series, active *H. pylori* infection was not found postoperatively in patients with MUs. They postulate that MUs form postoperatively after LRYGB due to preoperative injury to gastric mucosa [4]. This is confirmed by other studies that do not find active *H. pylori* infection in LRYGB patients who develop MUs [21]. Hartin et al. followed 183 consecutive patients, 125 of whom were not tested for *H. pylori* and 58 patients who were tested and treated if indicated. No difference was found in the ulceration rate between the groups, but perforations were slightly higher in patients who were not tested for *H. pylori*. Of the perforations, half were found to have *H.* pylori [22]. With these studies, though far from conclusive, the ASMBS recommends preoperative screening for *H. pylori* for patients in areas with high prevalence.

Aside from modifiable risk factors as potential causes of MUs, the nuances of LRYGB may predispose certain patients to develop MUs. Techniques for creating the gastric pouch, suture material used during the anastomosis, and variations in the path of the Roux limb have all been hypothesized as potential technical causes contributing to MU formation.

Gastric surgery prior to development of the Roux-en-Y gastric bypass was plagued by an increase in ulceration rates. Mason et al. theorized that gastric acid secretion was under antral control and that an enlarged gastric pouch that contained large portions of the body or fundus would prevent sufficient acid from reaching the antrum, thereby creating a loop of increased stimulation [23]. Once smaller gastric pouches were created (less than 50 ml), the rate of MUs decreased significantly [24]. In modern LRYGB, a pouch of about 20–30 ml should exclude most of the parietal cell mass of the body and fundus. However, this may not be true in all patients. Siilin and colleagues analyzed the mucosa of the anastomotic rings after creating gastrojejunostomies with a circular stapler. They attempted to create a pouch that measured 4 × 3 cm for a goal volume of 30 ml. In all 23 patients, parietal cells were found within the gastric mucosa included in the staple line [25]. This coincides with other twenty-first century studies that have found an "acid pocket" within the proximal cardia that escapes the buffering effect of meals. It was found to be present in all types of patients; but in those with gastroesophageal reflux disease, the acid pocket was found to be longer and migrate proximally towards the gastroesophageal junction [26]. If parietal cell mass is more proximal in some patients, then acid production within the gastric pouch would likely predispose these patients to development of MUs. Scintigraphy studies have shown this acid pocket and its location within the cardia (Fig. 11.1). Hedberg and his colleagues showed that patients with MUs had lower pH levels within their pouches compared to controls. Six patients with MUs approximately 5 years after gastric bypass were compared to six control patients also approximately 5 years from surgery who did not have MUs. The median

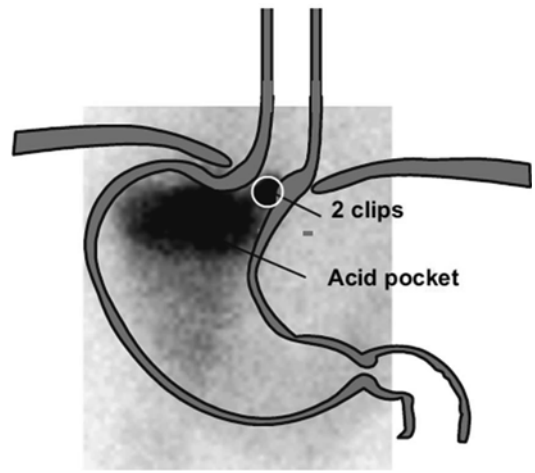

Fig. 11.1 Scintigraphic image showing parietal cell burden, the acid pocket, in the cardia in a healthy individual. Reprinted with permission from Beaumont H, Bennink RJ, de Jong J, Boeckxstaens GE. The position of the acid pocket as a major risk factor for acidic reflux in healthy subjects and patients with GORD. Gut. 2010;59(4):441–51 [47]. © BMJ Publishing Group

proportion of time with pH less than four in the study group was 96 %, whereas the control group median proportion of time with pH less than four was 20 % [27]. Acid secretion likely has some role in the creation of MUs due to the fact that most ulcerations will resolve simply with acid suppression medications [4].

An additional finding from Mason and his colleagues was that gastrin levels are low in most patients after gastric bypass [23]. This low gastrin level would correlate well with the decreased gastric acid stimulation in patients following LRYGB. For those who continue to have low pH within the gastric pouch, it is possible that the acid production occurs from vagal stimulation. Again, the acid production, whether from gastrin or vagal innervation, would respond to proton pump inhibition with medications [14].

As the gastric bypass was slowly being developed, several surgeons created the gastric pouch simply by stapling the stomach and not transecting the pouch from the remainder of the stomach [28]. While eliminating a potential location for leak, this led to an increased incidence of gastrogastric fistula creation [29]. Capella and Capella found that gastric pouches left stapled in continuity or only partially transected led to a gastrogastric fistula rate of 49 % in 189 patients. When they transitioned to complete division of the pouch from the remnant stomach, the rate dropped to 2.6 % in the next 188 patients. Despite this decrease in gastrogastric fistula rate, the incidence of marginal ulceration between the two groups did not change significantly. This is in contrast to Carrodeguas and colleagues who had an incidence of marginal ulceration of 4.2 % within their series of 1292 LRYGB patients. They divided the gastric pouch from the gastric remnant. In the 15 patients (1.2 %) who developed gastrogastric fistulas, the rate of marginal ulceration was 53.3 % [30]. Patel et al. also found an increased incidence of MUs in patients with gastrogastric fistulas. In 2282 patients who had undergone RYGB, a mixture of open and laparoscopic procedures, 122 patients (5.3 %) were found to develop MUs. Of those patients, 28 (22.9 %) were found to have gastrogastric fistulas [31]. Increased jejunal mucosal acid exposure due to the gastrogastric fistula likely increases the risk of marginal ulceration.

Much of the variability in techniques of LRYGB exists in the creation of the gastrojejunal anastomosis. Described techniques include a stapled anastomosis, hand-sewn anastomosis, sewn anastomosis with nonabsorbable suture, sewn anastomosis with absorbable suture [28]. Some of these techniques may predispose LRYGB patients to marginal ulceration. Capella and Capella transitioned from stapled gastrojejunostomies to hand-sewn gastrojejunostomies after they adopted division of the gastric pouch from the gastric remnant [29]. In the stapled gastrojejunostomies, the rate of marginal ulceration remained similar to the rate of ulceration prior to division of the pouch, 5.1 %. Once they began to complete the anastomoses with silk suture, only 5 patients (1.6 %) in 306 developed MUs. They went one step further and switched to absorbable suture from silk. In the next 97 patients with absorbable sutures, no symptomatic marginal ulceration had occurred with follow-up of 10 months.

Sacks and colleagues similarly modified their anastomotic technique. After using a linear stapler to create the gastrojejunostomy, the common gastroenterotomy was closed using an inner layer of running nonabsorbable suture followed by a second seromuscular layer of nonabsorbable suture. They transitioned to an inner layer of absorbable suture [32]. With this change, the rate of MUs decreased from 2.6 %, 28 of 1095 LRYGB patients, to 1.3 %, 29 of 2190 LRYGB patients (Fig. 11.2).

Suture used to reinforce stapled gastrojejunal anastomoses may also predispose LRYGB patients to the development of MUs. Vasquez et al. transitioned from an outer reinforcing suture layer of nonabsorbable suture to absorbable suture. Both groups had a 25-mm circular stapled gastrojejunal anastomosis. Use of absorbable suture had fewer MUs, 2 patients of 84 LRYGB patients (2.3 %), versus 31 patients out of 231 (13.4 %) with nonabsorbable suture [33].

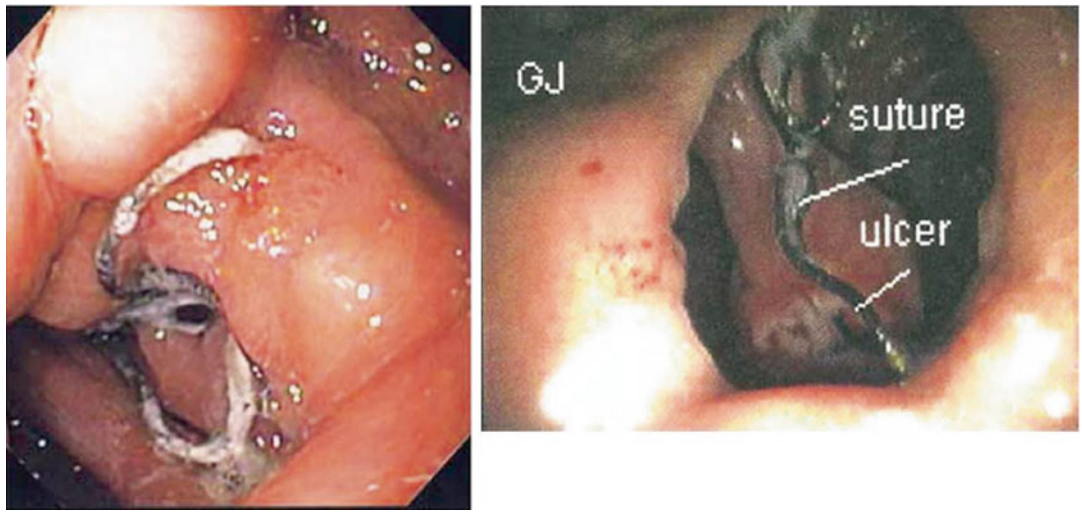

Fig. 11.2 Marginal ulcer seen on endoscopy with suture material within the ulcer base; GJ = gastrojejunostomy. Reprinted with permission from Sacks BC, Mattar SG, Qureshi FG, Eid GM, Collins JL, Barinas-Mitchell EJ, Schauer PR, Ramanathan RC. Incidence of marginal ulcers and the use of absorbable anastomotic sutures in laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2006;2(1):11–6 [32]. © Elsevier

Along with variations in techniques for creating the gastrojejunostomy, two variations exist for positioning the Roux limb during a LRYGB. Both antecolic and retrocolic Roux limb positions are used routinely. The debate over the best position continues, mostly around the incidence of internal hernias. The variation may also contribute to a higher incidence of MUs. Ribeiro-Parenti and her colleagues examined two large cohorts of patients using the antecolic position first, followed by the retrocolic technique, with 572 and 570 consecutive patients, respectively [34]. In their 1142 patients, 46 patients developed symptomatic MUs (4 %). Patients in the antecolic group were more likely to develop MUs and more likely to develop those ulcers early, within 3 months of surgery. Also, 2 patients with MUs in the antecolic group went on to perforate while no patients in the retrocolic group did. The two cohorts were comparable in regards to other risks for MUs, such as tobacco use and NSAIDs. The difference in MU rates between the two Roux positions may be due to more tension on the anastomosis, with an antecolic Roux limb possibly resulting in mucosal ischemia.

11.5 Diagnosis

The diagnosis of MUs, as with all complications, requires a high level of suspicion. LRYGB patients who present with new onset epigastric pain should be considered for MUs. However, internal hernias, stenosis, adhesive bowel obstructions, biliary disease, or cardiac disease may all present with epigastric pain. For those MUs that present with nausea or vomiting; internal hernias, stenosis, adhesive bowel obstructions and biliary disease must again be considered. If bleeding is the initial symptom, then bleeding from the large bowel may also require evaluation. With a long differential diagnosis accompanying the usual presentation of epigastric pain, nausea, vomiting, or bleeding, a systematic and careful evaluation is required [9] (Fig. 11.3). For patients that do not present acutely and develop insidious symptoms, the bariatric support staff are important in helping to make the diagnosis of MU. Registered dieticians and primary care providers will see patients more postoperatively than surgeons to assist in dietary changes and alterations to medication

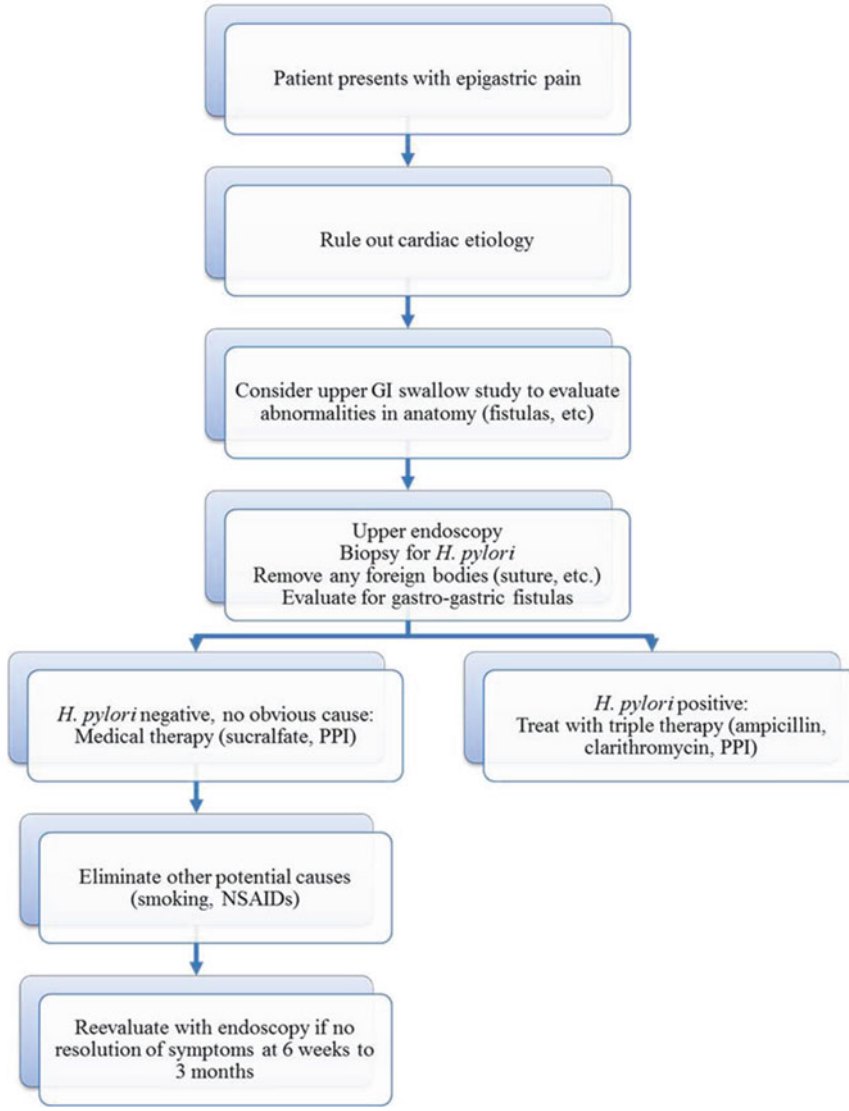

Fig. 11.3 Diagnostic flowchart for marginal ulceration following laparoscopic Roux-en-Y gastric bypass

regimens. As such, bariatric surgeons should ensure that those working with their patients are aware of the complications and the need to report these symptoms for further evaluation [35].

For MUs presenting acutely, such as with perforation, resuscitation and possibly emergent surgery may usurp any other diagnostics. For patients who are stable with suspicion of MUs, radiographic evaluation is the next step in evaluation as clinical evaluation in the obese patient is usually difficult. Upper gastrointestinal (UGI) examinations using fluoroscopy can be very helpful in the diagnosis of epigastric abdominal pain, evaluating for leaks or obstruction. MUs may also be seen. They will likely appear as a small focal collection of contrast within the ulcer that remains as the luminal contrast continues distally (Fig. 11.4). It will be located near the gastrojejunal anastomosis, either on the anastomosis or in the jejunum distally. While not completely diagnostic, UGI does serve as an initial first step in the workup of MUs and will help to rule out other diagnoses but may not always be necessary [36].

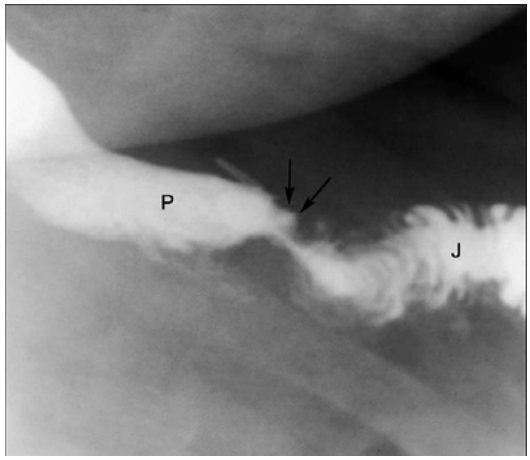

Fig. 11.4 Upper gastrointestinal fluoroscopic spot image with oral contrast showing retained contrast within the ulcer base (*arrows*). Patient is in the left posterior oblique position; P = pouch, J = jejunum. Reprinted with permission from Carucci LR, Turner MA. Radiologic evaluation following Roux-en-Y gastric bypass surgery for morbid obesity. Eur J Radiol. 2005;53(3):353–65 [36]. © Elsevier

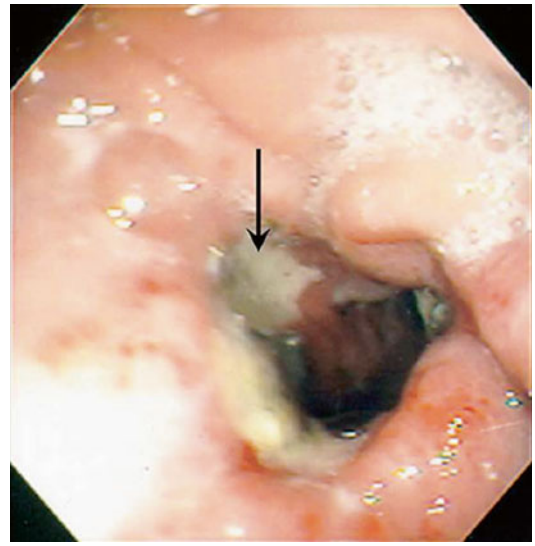

Fig. 11.5 Marginal ulcer (*arrow*) seen just beyond gastrojejunostomy 1 month following laparoscopic gastric bypass. Reprinted with permission from Gumbs AA, Duffy AJ, Bell RL. Incidence and management of marginal ulceration after laparoscopic Roux-Y gastric bypass. Surg Obes Relat Dis. 2006;2(4):460–3 [41]. © Elsevier

UGI will also assist in the diagnosis of potential causes of MUs. Gastrogastric fistulas can sometimes be seen on UGI as a communication between the gastric pouch and the excluded stomach. Computed tomography (CT) is usually a mainstay of evaluation for postoperative bariatric patients, particularly in the acute care setting within the emergency department. Findings on CT will likely be within normal limits unless oral contrast is given. With oral contrast, the findings will be similar to UGI. Complications of MUs may also be seen with CT. Free air or extravasation of contrast may be seen with perforation. Gastrogastric fistulas may also be seen as contrast traversing from the gastric pouch to the excluded stomach [37]. CT will also help to eliminate other potential causes of epigastric pain, nausea, or vomiting, such as internal hernias or leaks.

After radiographic evaluation that may show findings concerning for MUs or eliminate other potential diagnoses endoscopy remains the gold standard for the diagnosis of MUs. It also serves as a method of treatment. On endoscopy, the ulcer will be visible at the anastomosis or on the jejunal mucosa of the Roux limb (Fig. 11.5). Etiology of the ulcer may also be seen on endoscopy, such as suture material within the ulcer. Histologic and testing of biopsied surrounding mucosa may also help to eliminate *H. pylori* as a potential etiology.

11.6 Prevention and Management

As with all surgical complications, prevention of the complication is better than eventual treatment. Because of the multiple causes of MUs that have been postulated, strategies for prevention vary. At a minimum, the ASMBS recommends smoking cessation counseling prior to surgery [18]. In our practice, patients must abstain from all tobacco products for 3 months prior to any bariatric surgeries. Many patients who wish to undergo bariatric surgery will present with joint disease and will likely be taking NSAIDs for pain and inflammation management. This presents a difficult situation for health care providers of patients who desire LRYGB. NSAIDs will increase the risk of developing MUs, but without them, the patients may not ambulate as much as

is necessary to prevent other complications, such as venous thromboembolism. Attempts should be made to limit the amount of NSAIDs that patients take preoperatively and cessation of NSAID therapy should follow LRYGB as soon as possible. For postoperative LRYGB patients who present with injuries where NSAIDs would be used as treatment, alternative therapies should be attempted.

H. pylori infection in bariatric patients has a reported prevalence of 24–67 % in the literature [38]. While its causal relationship with peptic ulcer disease is well known, its exact relationship with MUs is still debatable. As such, many bariatric programs will screen for *H. pylori* infection prior to LRYGB. Hartin and colleagues found that not testing for *H. pylori* did not increase the incidence of MU formation but did confer a greater risk of perforation if the patients went on to develop ulcers [22]. While *H. pylori* infection may not directly cause ulcers, its presence has been linked to epigastric symptoms following LRYGB. Ramaswamy et al. found that patients were more likely to complain of dyspepsia symptoms without MUs if infected with *H. pylori* [38]. Based on this, they recommended testing for *H. pylori* infection as eradication may decrease postoperative foregut symptoms and MU formation. Contrary to this, Papasavas and colleagues found no difference in MU rate [20]. Currently, the ASMBS suggests testing for *H. pylori* in regions where there is a high prevalence of infection [18]. Most of the USA has a relatively low prevalence. Patients from developing countries who desire LRYGB would likely benefit from preoperative screening for *H. pylori.* Testing for infection may be completed noninvasively through the urease breath test or stool antigen study. Invasive testing requiring endoscopy requires biopsies and also evaluates for urease or microscopic visualization of the organisms. Patients who present with epigastric symptoms following surgery should also be tested for *H. pylori* and undergo eradication if positive. The most commonly recommended initial regimen includes a proton pump inhibitor (PPI), amoxicillin, and clarithromycin. Regimens for the eradication of *H. pylori* have been designed for patients of normal body mass index. Morbidly obese patients may not respond to the same regimen [39]. If eradication is needed, confirmation should be completed.

Intraoperative prevention of ulceration includes ensuring that the gastric pouch is appropriately sized to exclude as much parietal cell mass as possible. The pouch should be divided from the gastric remnant, not just stapled in continuity, to decrease the risk of gastrogastric fistula formation. Absorbable sutures should be used for the inner layer of the gastrojejunal anastomosis, and the Roux limb must be under no tension to prevent any possible ischemia at the anastomosis.

Postoperative medication regimens, such as the use of PPIs for MU prevention, vary by institution. The ASMBS currently has no recommendations about the use of PPIs postoperatively for the prevention of MUs [18]. No conclusive evidence exists for the use of PPIs or H2 blockers, such as ranitidine, postoperatively, except in cases of eradication of *H. pylori.* One study by D'Hondt and his colleagues showed no difference in LRYGB patients who underwent a short course of omeprazole 20 mg daily for 1 month after surgery than in those who did not. Both formed MUs at the same rate. A difference was seen in patients who had undergone *H. pylori* eradication prior to surgery. For those patients, use of prophylactic low-dose PPI therapy did decrease the rate of MU formation [40]. Garrido et al. showed this as well after following 118 LRYGB patients who were placed on esomeprazole for 60 days after surgery. Nine patients (7.6 %) still developed MUs despite therapy [19]. With the lack of conclusive evidence, each individual institution and surgeon should implement a plan for postoperative medication use to prevent MUs.

Once a MU has been diagnosed, medical treatment remains the mainstay of treatment. Multiple studies have been completed showing that medication alone will adequately treat the majority of MUs. In a study of 26 patients who developed MUs following LRYGB, all of the MUs resolved following high-dose PPIs [41]. Others recommend use of sucralfate to assist in the healing of

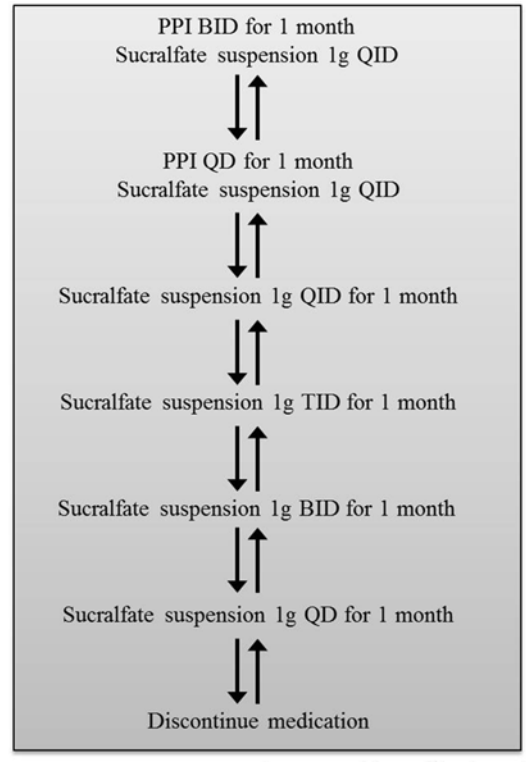

Fig. 11.6 Example treatment algorithm for slow taper from sucralfate and proton pump inhibitor for the treatment of marginal ulceration. Adapted with permission from Dallal RM, Bailey LA. Ulcer disease after gastric bypass surgery. Surg Obes Relat Dis. 2006;2(4):455–9 [6]. © Elsevier

the MUs (Fig. 11.6). A suspension of the sucralfate is created using warm water and the large tablets. Theoretically, this will coat the ulcer base, no matter the cause, and assist in healing. No conclusive evidence exists for the use of sucralfate or PPIs or a combined therapy [6]. Sucralfate may help to heal ulcers not associated with acid, as is possible with MUs. When an MU is suspected, beginning high dose PPI for acid suppression with or without the use of sucralfate is the beginning step in treatment and to prevent complications.

For patients with symptoms concerning for marginal ulceration, endoscopy remains an essential diagnostic tool. Upper endoscopy also serves as a therapeutic tool depending on the specific pathophysiology of the ulceration in each patient. For patients who present with ulceration due to foreign body reaction from suture, endoscopy can be used to remove the suture from the lumen of the bowel. Endoscopic graspers are used to place the suture under tension and the suture is transected using endoshears [42]. Similar methods can be used to remove staples from the mucosa that are causing foreign body reaction. Gastrogastric fistulas may also be found using endoscopy. Advances in endoscopic technology have allowed for treatment of gastrogastric fistulas with the use of over-the-scope clips (Ovesco Endoscopy AG, Tubingen, Germany) and endoscopic suture devices. While these devices do require advanced training and general anesthesia, their use does not require the risks of laparoscopic or open reoperation. Endoscopy remains essential for the diagnosis of MUs and will treat many different causes of ulceration that will not resolve with medical therapy.

For patients who fail to resolve with medical therapy and endoscopic treatments are not available or have been unsuccessful, reoperation with revision of the gastrojejunostomy is the next step. Revision of the gastrojejunostomy has been successful in treating intractable MUs. These operations have been found to be safe and effective,

with appropriate patient selection [43]. Patients should be cleared for surgery medically. Nonsurgical causes of MUs should be addressed, such as smoking cessation, *H. pylori* infection, and NSAID use. Once these have been completed, and the MUs are confirmed to be unresponsive to medical therapy, then reoperation should be discussed. After lysis of adhesions, which may be extensive and difficult, the gastrojejunostomy is dissected circumferentially. The gastric pouch is stapled proximal to the anastomosis. Additional staple firings may be necessary to decrease the size of the pouch if it is greater than 20–30 ml in volume. This is done to exclude as many parietal cells as possible to decrease acid production within the pouch [44]. The Roux limb is then transected just distal to the anastomosis. Intraoperative endoscopy is helpful to ensure that all of the ulcerations are contained within the resected segment. The anastomosis is then recreated using either stapled or hand-sewn technique. The new gastrojejunostomy should be free of tension and have sufficient blood supply to heal without recurrence of the ulcerations [45]. Revision of the gastrojejunostomy can be completed laparoscopically or open. As with most foregut surgery, visualization of the field is usually better with laparoscopy, but conversion to open if required for patient safety or complications is always appropriate.

Reoperation may also be necessary for other causes of intractability, such as the presence of a gastrogastric fistula. If the gastrojejunostomy shows no abnormalities and the gastric pouch is appropriately sized, then simple transection of the gastrogastric fistula may lead to resolution of the MU. This is completed by resecting the fistula tract, which usually connects to the fundus of the stomach. The fundus of the stomach is also resected to ensure adequate distance between the gastrojejunostomy and the gastric remnant [31]. For complications of MUs, such as strictures, reoperation with revision of the gastrojejunostomy, pouch, or Roux limb may also be necessary. Treatment of perforated MUs remains surgical. For clinically unstable patients presenting with increasing epigastric abdominal pain and free air seen on imaging, concerning for perforation, emergent surgical intervention is warranted.

If all medical therapies and endoscopic or surgical interventions have been exhausted without healing of the ulceration, reversal of the LRYGB may occasionally be required. This is rarely indicated, but in a select few patients, it is the only option to improve quality of life and symptoms. Chousleb and colleagues described eight patients who underwent reversal of gastric bypass. Three of the patients had intractable MUs. All of the operations were done open. One of the patients subsequently required conversion back to gastric bypass due to gastric atony [46]. Consideration of conversion to another bariatric surgery, such as sleeve gastrectomy, can be considered. If reversal is required, this should be performed at centers with extensive revisional bariatric experience.

11.7 Peptic Ulcers Following Bariatric Surgery

Ulcer formation is not limited to patients who undergo laparoscopic Roux-en-Y gastric bypass. Any operations where the stomach remains are at risk for developing peptic ulcers. Sleeve gastrectomy and biliopancreatic diversion with duodenal switch (BPD/DS) are two other forms of bariatric surgery performed in the USA. In both of these surgeries peptic ulcers may occur. Following LRYGB, ulceration may occur in the gastric remnant or the duodenum. These ulcers occur similarly to peptic ulcers in nonsurgical patients. The likely pathophysiology is NSAID use and *H. pylori* infection.

Patients will likely present with epigastric pain with meals and may complain of reflux symptoms. Bleeding may occur and present with melena or hematemesis. Prolonged ulceration may lead to obstruction causing nausea and vomiting. In peptic ulcers within the gastric remnant of LRYGB patients, bleeding or epigastric pain will likely be the presenting symptom.

Diagnosis of peptic ulcers in sleeve gastrectomy and BPD/DS patients usually requires endoscopy. All portions of the alimentary tract where peptic ulcers may form can be visualized.

Evaluation of the gastric remnant in LRYGB patients is much more difficult. Double balloon endoscopy will sometimes allow visualization of the duodenum and the gastric remnant. Many times, laparoscopic assisted endoscopy is required to evaluate the remnant. For this procedure, the gastric remnant is brought to the anterior abdominal wall and an endoscope is placed through the abdominal wall.

Once diagnosed, treatment of peptic ulcers in bariatric patients is similar to the treatment of non-bariatric patients. Endoscopy plays a key role, allowing for clip placement or epinephrine injection. Ulcers may require surgical intervention for complications or intractability. Perforation requires surgical intervention. Obstruction requires intervention. If unable to be treated with endoscopic dilation, conversion to other bariatric procedures may be necessary, such as sleeve gastrectomy being converted to LRYGB.

References

1. Estimate of bariatric surgery numbers. [Internet] 1 Mar 2014 [cited 1 Nov 2014]. Available at: http://asmbs.org/resources/estimate-of-bariatric-surgery-numbers.
2. Wittgrove AC, Clark GW. Laparoscopic gastric bypass, Roux-en-Y: Preliminary report of five cases. Obes Surg. 1994;4(4):353–7.
3. Coblijn UK, Goucham AB, Lagarde SM, Kuiken SD, van Wagensveld BA. Development of ulcer disease after Roux-en-Y gastric bypass, incidence, risk factors, and patient presentation: a systematic review. Obes Surg. 2014;24(2):299–309.
4. Rasmussen JJ, Fuller W, Ali MR. Marginal ulceration after laparoscopic gastric bypass: an analysis of predisposing factors in 260 patients. Surg Endosc. 2007;21(7):1090–4.
5. Csendes A, Burgos AM, Altuve J, Bonacic S. Incidence of marginal ulcer 1 month and 1 to 2 years after gastric bypass: a prospective consecutive endoscopic evaluation of 442 patients with morbid obesity. Obes Surg. 2009;19(2):135–8.
6. Dallal RM, Bailey LA. Ulcer disease after gastric bypass surgery. Surg Obes Relat Dis. 2006;2(4):455–9.
7. El-Hayek K, Timratana P, Shimizu H, Chand B. Marginal ulcer after Roux-en-Y gastric bypass: what have we learned? Surg Endosc. 2012;26(10):2789–96.
8. Abell TL, Minocha A. Gastrointestinal complications of bariatric surgery: diagnosis and therapy. Am J Med Sci. 2006;331(4):214–8.
9. Campanile FC, Boru CE, Rizzello M, et al. Acute complications after laparoscopic bariatric procedures: update for the general surgeon. Langenbecks Arch Surg. 2013;398(5):669–86.
10. Azagury DE, Abu Dayyeh BK, Greenwalt IT, Thompson CC. Marginal ulceration after Roux-en-Y gastric bypass surgery. Endoscopy. 2011;43(11):950–4.
11. Sidani S, Akkary E, Bell R. Catastrophic bleeding from a marginal ulcer after gastric bypass. JSLS. 2013;17(1):148–51.
12. Felix EL, Kettelle J, Mobley E, Swartz D. Perforated marginal ulcers after laparoscopic gastric bypass. Surg Endosc. 2008;22(10):2128–32.
13. Bramkamp M, Muller MK, Wildi S, Clavien PA, Weber M. Perforated ulcer at the gastrojejunostomy: Laparoscopic repair after Roux-en-Y gastric bypass. Obes Surg. 2006;16(11):1545–7.
14. Al Harakeh AB. Complications of laparoscopic Roux-en-Y gastric bypass. Surg Clin N Am. 2011;91(6):1225–37.
15. Needleman B. Gastric bypass: Marginal ulceration. In: Ngyuen N et al., editors. The SAGES manual: a practical guide to bariatric surgery. New York, NY: Springer; 2008.
16. Maity P, Biswas K, Roy S, Banerjee RK, Bandyopadhyay U. Smoking and the pathogenesis of gastroduodenal ulcer—recent mechanistic update. Mol Cell Biochem. 2003;253(1-2):329–38.
17. Wilson JA, Romagnuolo J, Byrne TK, Morgan K, Wilson FA. Predictors of endoscopic findings after Roux-en-Y gastric bypass. Am J Gastroenterol. 2006;101(10):2194–9.
18. Mechanick JI, Youdim A, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient—2013 Update: Cosponsored by American Association of Clinical Endocrinologists, the Obesity Society, and American Society for Metabolic and Bariatric Surgery. Surg Obes Relat Dis. 2013;9(2):159–91.
19. Garrido Jr AB, Rossi M, Lima Jr SE, Brenner AS, Gomes Jr CA. Early marginal ulcer following Roux-en-Y gastric bypass under proton pump inhibitor treatment—prospective multicentric study. Arq Gastroenterol. 2010;47(2):130–4.
20. Papasavas PK, Gagne DJ, Donnelly PE, et al. Prevalence of *Helicobacter pylori* infection and value of preoperative testing and treatment in patients undergoing laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2008;4(3):383–8.
21. Yang CS, Lee WJ, Wang HH, Huang SP, Lin JT, Wu MS. The influence of *Helicobacter pylori* infection on the development of gastric ulcer in symptomatic patients after bariatric surgery. Obes Surg. 2006;16(6):735–9.
22. Hartin Jr CW, ReMine DS, Lucktong TA. Preoperative bariatric screening and treatment of *Helicobacter pylori*. Surg Endosc. 2009;23(11):2531–4.
23. Mason EE, Munns JR, Kealey GP, et al. Effect of gastric bypass on gastric secretion.1977. Surg Obes Relat Dis. 2005;1(2):155–60.

24. Sapala JA, Wood MH, Sapala MA, Flake TM. Marginal ulcer after gastric bypass: a prospective 3-year study of 173 patients. Obes Surg. 1998;8(5):505–16.
25. Siilin H, Wanders A, Gustavsson S, Sundbom M. The proximal gastric pouch invariably contains acid-producing parietal cells in Roux-en-Y gastric bypass. Obes Surg. 2005;15(6):771–7.
26. Gilmore MM, Kallies KJ, Mathiason MA, Kothari SN. Varying marginal ulcer rates in patients undergoing laparoscopic Roux-en-Y gastric bypass for morbid obesity versus gastroesophageal reflux disease: is the acid pocket to blame? Surg Obes Relat Dis. 2013;9(6):862–6.
27. Hedberg J, Hedenstrom H, Nilsson S, Sundbom M, Gustavsson S. Role of gastric acid in stomal ulcer after gastric bypass. Obes Surg. 2005;15(10): 1375–8.
28. Baker MT. The history and evolution of bariatric surgical procedures. Surg Clin N Am. 2011;91(6): 1181–201.
29. Capella JF, Capella RF. Gastro-gastric fistulas and marginal ulcers in gastric bypass procedures for weight reduction. Obes Surg. 1999;9(1):22–7.
30. Carrodeguas L, Szomstein S, Soto F, et al. Management of gastrogastric fistulas after divided Roux-en-Y gastric bypass surgery for morbid obesity: analysis of 1292 consecutive patients and review of literature. Surg Obes Relat Dis. 2005;1(5): 467–74.
31. Patel RA, Brolin RE, Gandhi A. Revisional operations for marginal ulcer after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5(3):317–22.
32. Sacks BC, Mattar SG, Qureshi FG, et al. Incidence of marginal ulcers and the use of absorbable anastomotic sutures in laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2006;2(1):11–6.
33. Vasquez JC, Overby DW, Farrell TM. Fewer gastrojejunostomy strictures and marginal ulcers with absorbable suture. Surg Endosc. 2009;23(9):2011–5.
34. Ribeiro-Parenti L, Arapis K, Chosidow D, Marmuse JP. Comparison of marginal ulcer rates between antecolic and retrocolic laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2015;25(2):215–21.
35. Benson-Davies S, Quiqley DR. Screening postoperative bariatric patients for marginal ulcers. J Am Diet Assoc. 2008;108(8):1369–71.
36. Carucci LR, Turner MA. Radiologic evaluation following Roux-en-Y gastric bypass surgery for morbid obesity. Eur J Radiol. 2005;53(3):353–65.
37. Griffith PS, Birch DW, Sharma AM, Karmali S. Managing complications associated with laparoscopic Roux-en-Y gastric bypass for morbid obesity. Can J Surg. 2012;55(5):329–36.
38. Ramaswamy A, Lin E, Ramshaw BJ, Smith CD. Early effects of *Helicobacter pylori* infection in patients undergoing bariatric surgery. Arch Surg. 2004;139(10): 1094–6.
39. Abdullahi M, Annibale B, Capoccia D, et al. The eradication of *Helicobacter pylori* is affected by body mass index (BMI). Obes Surg. 2008;18(11):1450–4.
40. D'Hondt MA, Pottel H, Devriendt D, Van Rooy F, Vansteenkiste F. Can a short course of prophylactic low-dose proton pump inhibitor therapy prevent stomal ulceration after laparoscopic Roux-en-Y gastric bypass? Obes Surg. 2010;20(5):595–9.
41. Gumbs AA, Duffy AJ, Bell RL. Incidence and management of marginal ulceration after laparoscopic Roux-Y gastric bypass. Surg Obes Relat Dis. 2006;2(4):460–3.
42. Freeza EE, Herbert H, Ford R, Wachtel MS. Endoscopic suture removal at gastrojejunal anastomosis after Roux-en-Y gastric bypass to prevent marginal ulceration. Surg Obes Relat Dis. 2007;3(6):619–22.
43. Behrns KE, Smith CD, Kelly KA, Sarr MG. Reoperative bariatric surgery—lessons learned to improve patient selection and results. Ann Surg. 1993;218(5):646–53.
44. Madan AK, DeArmond G, Ternovits CA, Beech DJ, Tichansky DS. Laparoscopic revision of the gastrojejunostomy for recurrent bleeding ulcers after past open revision gastric bypass. Obes Surg. 2006; 16(12):1662–8.
45. Nguyen NT, Hinojosa MW, Gray J, Fayad C. Reoperation for marginal ulceration. Surg Endosc. 2007;21(11):1919–21.
46. Chousleb E, Patel S, Szomstein S, Rosenthal R. Reasons and operative outcomes after reversal of gastric bypass and jejunoileal bypass. Obes Surg. 2012;22(10):1611–6.
47. Beaumont H, Bennink RJ, de Jong J, Boeckxstaens GE. The position of the acid pocket as a major risk factor for acidic reflux in healthy subjects and patients with GORD. Gut. 2010;59(4):441–51.

Gastrointestinal Obstruction in the Bypass Patient

12

Ahmad Elnahas and Allan Okrainec

Key Points

- Appreciation of RYGB anatomy is necessary to properly diagnose and manage bowel obstructions in the bypass patient. A good understanding of the index procedure and onset of symptoms will assist in the accurate diagnosis and treatment strategy.
- Early obstruction usually occurs at the level of the jejunojejunostomy and is seen more commonly with stapled closure and patients with ventral hernias. Late obstruction can occur several months after surgery and is usually due to internal herniation or adhesive disease.
- Clinical presentation and radiological tests are often nonspecific or misinterpreted. Therefore, there should be a low threshold for diagnostic laparoscopic evaluation in postoperative bypass patients.
- Most bowel obstructions following RYGB may be treated with a laparoscopic approach; conversion to open surgery may be advised in cases that are technically challenging.

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12.1 Introduction

12.1.1 Small Bowel Obstructions in the Bypass Patient

Small bowel obstruction (SBO) is a well-known complication of bariatric surgery, with a reported incidence between 1 and 11 % following RYGB [1–5]. In order to properly diagnose and treat SBO after Roux-en-Y gastric bypass (RYGB), a thorough understanding of the surgical anatomy is imperative. Postoperative SBO can be associated with considerable morbidity and mortality if not recognized and treated promptly [6].

Laparoscopic RYGB has been associated with a higher incidence of postoperative SBO compared to open surgery due in part to a relative paucity of adhesion formation [7]. The leading cause of SBO after laparoscopic RYGB is internal herniation, while the most common cause after open RYGB is intra-abdominal adhesion formation [1, 2]. SBO following RYGB can be described based on the timeline of presentation after surgery. Early SBO occurs within 30 days of surgery, while late SBO manifests after 30 days [6]. Causes of early SBO include postoperative ileus and jejunojejunal anastomotic stricture, usually due to technical error [2, 7]. Other causes of early SBO include anastomotic edema, intraluminal clot, angulation of the Roux limb, and incarcerated abdominal wall hernias [6]. Late SBO often results from internal hernias, ventral hernias, or adhesive disease [2, 6].

D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_12

Presenting symptoms of bowel obstruction following RYGB can be nonspecific and elusive. The most common presentation includes abdominal pain, followed by nausea, vomiting, bloating, and dysphagia [8]. Roux limb obstruction usually presents with nausea, bloating, and epigastric abdominal pain that is temporarily relieved by emesis [4]. Biliopancreatic limb obstruction leads to gastric remnant distension and usually presents with nausea, bloating, tachycardia, hiccups, and shoulder pain but not vomiting [4]. Common channel obstruction will often present with a combination of the above symptoms. Given the small size of the gastric pouch, it is rare for obstructed patients to present with large amounts of emesis. With the exception of gastrogastric fistula, bilious vomiting in a post-RYGB patient indicates a common channel obstruction at or beyond the jejunojejunal anastomosis until proven otherwise [4, 9]. The time of onset of SBO following RYGB can vary considerably with median times ranging from 21 days to 24 weeks [2, 10].

Laboratory analysis is rarely useful in the work-up for suspected bowel obstruction after RYGB, although lipase may be elevated in those with a biliopancreatic limb obstruction [4]. Diagnosis of SBO in non-bariatric patients is usually evident on abdominal X-rays. However, most RYGB patients, especially those with internal hernias, do not demonstrate typical signs on plain films [5]. Computed tomography (CT) imaging with oral and intravenous contrast is considered a necessary component in the work-up of SBO in the bypass patient. However, the sensitivity of CT for diagnosing bowel obstruction after RYGB has been reported to be lower (51.1 %) compared to the general population (80–90 %) [8]. Diagnostic laparoscopy is therefore considered mandatory in the setting of a suspected postoperative bowel obstruction in a bypass patient despite normal imaging [5].

Delaying treatment for bowel obstruction in the bypass patient can result in progression to bowel necrosis. The potential for extensive bowel resection in this scenario underscores the importance of achieving a prompt diagnosis in this population to prevent significant morbidity. Studies have demonstrated that a low threshold for surgical exploration translates to a lower bowel resection rate in cases of bowel obstruction [8]. The majority of SBO after laparoscopic RYGB can be managed laparoscopically [4]; however the safety and feasibility of this approach are usually based on the extent of bowel dilation (i.e., working space) and the site of bowel obstruction (i.e., etiology) [6, 11]. Previous literature has demonstrated that laparoscopic management of post-RYGB bowel obstruction is associated with a high rate of conversion to open procedure [8]. Conversion to open may be necessary if laparoscopic evaluation of bowel pathology or viability proves to be technically difficult, as in the case of distal obstruction with concomitant severe bowel dilation. In general, the risk of delayed intervention in a patient with suspected bowel obstruction more than offsets the risks of a negative laparoscopic exploration, which usually has minimal morbidity [8]. There is no clear acceptable negative diagnostic laparoscopy rate in this population; however a rate of 2–10 % has been reported in several studies [4, 12].

Physicians and surgeons involved with postoperative management of bariatric surgery patients should be familiar with RYGB anatomy and the potential causes and treatment of bowel obstructions. Laparoscopy can be safely performed for the management of SBO by an experienced surgeon with advanced laparoscopic skills in a carefully selected patient population [6].

12.2 Intraluminal Obstructions

12.2.1 Gastrojejunal Stricture (Stomal Stenosis)

Gastrojejunal (GJ) stricture is one of the most common complications after RYGB, with a reported incidence ranging from 3 to 27 % in the literature [13, 14]. With experience and standardization of anastomotic technique, the

incidence has decreased substantially to less than 5 % [13]. The etiology of these strictures can be multifactorial and risk factors include local ischemia, gastric acid hypersecretion (i.e., large pouch size), chronic ulcers (NSAIDS, smoking), subclinical anastomotic leaks, suture material, and surgical technique [15].

Symptoms of a GJ stricture include postprandial epigastric pain and frequent emesis of partially digested solids, followed by progressive inability to tolerate food [15]. Features of malnutrition, such as failure to thrive, can be common in patients with late-forming strictures [16]. The average time of symptom onset ranges from 32 to 82 days after surgery [16]. A GJ stricture is most commonly diagnosed in the first 90 days after surgery and would be very unusual to see before 2 weeks postoperatively [17].

A GJ stricture can be diagnosed using an upper gastrointestinal (UGI) study or an upper endoscopy (Fig. 12.1). The definition of a stricture is variable, but most surgeons consider a clinically significant stricture present if an endoscope cannot pass through or the stoma is less than 10 mm in diameter [16].

Post-bypass GJ strictures can be endoscopically graded into four groups [19]:

- Grade I: Mild stenosis—10.5 mm endoscope can be passed
- Grade II: Moderate stenosis—8.5 mm pediatric endoscope can be passed
- Grade III: Severe stenosis—only a guide wire can be passed
- Grade IV: Complete/near-complete obstruction—non-traversable

A pooled analysis by Markar et al. found that 21 mm stapled GJ anastomoses were associated with an increased symptomatic stricture rate compared to 25 mm anastomoses [20]. However, no significant weight loss difference was found between the two groups [20]. Their study concluded that the 25 mm circular stapler would reduce the risk of GJ symptomatic stricture while providing adequate weight loss [20]. A hand-sewn technique using the linear stapler with transverse

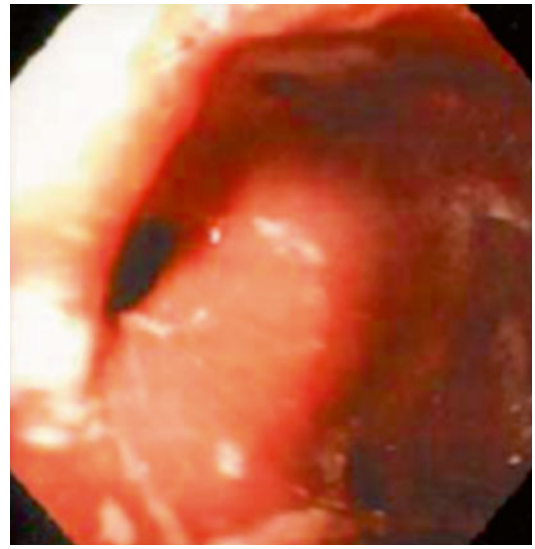

Fig. 12.1 Stomal stenosis on endoscopy. With permission from Go MR, Muscarella P, 2nd, Needleman BJ, Cook CH, Melvin WS. Endoscopic management of stomal stenosis after Roux-en-Y gastric bypass. Surg Endosc. 2004;18(1):56–9 [18]. © Springer

enterotomy closure has also been associated with a lower stricture rate compared to a vertical longitudinal closure or the 21 mm circular stapler [14, 16, 21]. In general, ischemic strictures are more frequently reported with circular stapled rather than hand-sewn anastomoses [16]. The use of a circular stapler has also been found to be associated with stricture recurrence [17]. However, proponents of circular stapled anastomoses argue that stoma sizes remain relatively constant compared with linear stapled or hand-sewn stomas, which can dilate over time [16].

The treatment of GJ strictures has varied from non-operative endoscopic dilation to open or laparoscopic surgical revision of the gastrojejunostomy combined with medical management to prevent future recurrence [13, 19]. The first-line treatment for a GJ stricture is usually endoscopic balloon dilation, which has been shown to be a very effective strategy. Early endoscopic intervention is important in RYGB patients with GJ strictures in order to alleviate symptoms and avoid complications such as dehydration and metabolic derangement. A shorter interval from

surgery to initial dilation has been associated with a higher likelihood of success and, accordingly, a lower likelihood of requiring revisional surgery. Early strictures are very responsive to endoscopic dilation because they usually result from simple mucosal overgrowth [16]. In fact, most patients require only a single dilation when presenting early after surgery or less than 90 days.

Serial balloon dilation every 2–3 weeks to a maximum of 15 mm has been shown to resolve symptoms with an overall success rate of over 80 % [14]. Although the optimal maximal dilation size still remains to be determined, overdilation should be avoided to prevent complications such as perforation and preserve weight loss [15]. Ryskina et al. found that balloon dilation up to 15 mm was not associated with reduced weight loss at 6 or 12 months following surgery. Approximately 3–8 % of patients with GJ strictures, most with late strictures, require more than three dilations for resolution [17]. These patients may benefit from placement of a feeding tube in the gastric remnant to allow for caloric supplementation [19].

Perforation is the greatest concern after endoscopic dilatation. Large series published on post-RYGB GJ stricture dilatation have demonstrated a 0.6–2.2 % perforation rate [16]. To minimize the risk of perforation, some surgeons will not dilate a stricture more than 3 mm (or 9 Fr) during a single session. However, studies have not demonstrated an increased risk of perforation due to balloon size or number of dilations [14]. Some surgeons also believe that the more rigid Savary-Gilliard bougies offer a better, more durable dilation than the more pliable pneumatic balloons and should be used following initial balloon dilation [19]. Intralesional steroid injections have been reported for cases of refractory strictures. Steroids are thought to prevent cross-linking of collagen and thus prevent fibrotic healing. However, the role of steroid injections is still not defined as some studies have found that they provide no added benefit [14]. For patients with GJ strictures refractory to endoscopic therapy, operative revision of the anastomosis can be very effective, with a success rate of more than 95 % [13]. However, these revisional procedures are often technically challenging and complex. Most refractory anastomotic strictures are thought to be due to large-volume gastric pouches, which result in excessive acid production. These situations ultimately require major downsizing of the proximal gastric pouch to less than 10 mL in volume to help ensure that the pouch contains only gastric cardia and excludes all acid-producing gastric mucosa [13].

12.2.2 Jejunojejunal Stricture

Jejunojejunal (JJ) stricture is one of the leading causes of SBO in the early postoperative period, with an incidence of 0.4–1.2 % and a mean interval to presentation of 10–15 days [2, 3, 9]. Jejunojejunal narrowing usually occurs in the Roux limb portion of the anastomosis because of technical error when too much tissue is taken using a double-stapling technique with a linear stapler [2]. A large study demonstrated that stapled closure of the common enterotomy resulted in a significantly higher rate of JJ obstruction compared with hand-sewn closure [4]. Brolin also reported that bowel obstruction can occur at the afferent limb of the jejunojejunostomy after open RYGB and advocated placement of an "anti-obstruction suture" to prevent kinking at the level of the anastomosis [22]. The majority of JJ strictures present with nonspecific clinical symptoms and signs suggestive of partial SBO. However, these types of strictures can be easily diagnosed with UGI series or CT imaging [2]. Postoperative edema can also cause early JJ obstruction that tends to be partial and responds well with conservative management [9]. Narrowing of the jejunojejunostomy due to incorrect stapling may ultimately require creation of a new side-to-side anastomosis proximal to the obstruction site [11].

12.2.3 Intussusception

Retrograde intussusception post-RYGB is usually located at the jejunojejunostomy, progressing

from distally to proximally (i.e., antiperistaltic), rather than the more common proximal to distal direction (i.e., isoperistaltic) [23]. This postoperative complication is quite rare, with a reported incidence of less than 0.6 % in bypass patients [24].

Intussusception usually occurs after significant weight loss and its cause appears to be multifactorial. Most reports of retrograde intussusception have described an absence of any definable lead point [25]. However, staple lines, sutures, and adhesions have been proposed as possible lead points [26].

Although the exact mechanism is not yet clear, motility disturbances are believed to be the most likely cause of intussusception after RYGB [24]. In RYGB, the distal jejunum is separated from the duodenal pacemaker and disrupts the propagation of the natural pacemaker into the Roux limb [24]. As a result, ectopic pacemakers arise in the Roux limb, which can generate pacesetter potentials in both distal and proximal directions [24]. Manometric studies have confirmed that patients after RYGB have a high incidence of motility disorders secondary to these ectopic pacemakers [27]. It is hypothesized that an ectopic pacemaker can create a peristaltic contractile wave that reaches the jejunojejunostomy at the same time as a normal peristaltic wave from the duodenum, producing a high-amplitude peristaltic wave in the proximal channel that engulfs the bowel distal to it [27]. Motility disorders can also contribute to "Roux stasis syndrome," a condition characterized by chronic abdominal pain, nausea, and vomiting [24].

The clinical presentation of intussusception can vary, ranging from chronic intermittent abdominal pain to sudden severe intractable pain consistent with complete obstruction and bowel ischemia [23]. The most common presentation is vague abdominal pain (100 %) followed by vomiting (40 %) and bloody stools (20 %) [26]. This type of complication appears to present more commonly late after surgery and in those patients with substantial weight loss [28].

Plain abdominal films are usually unreliable in the evaluation of intussusception [25]. Contrast-enhanced CT is diagnostic in most cases, and a characteristic "target sign" at the site of the intussusception is usually pathognomonic [23]. CT findings also include a dilated gastric remnant consistent with an obstructed biliopancreatic limb [28].

Patients with evidence of intussusception require immediate surgery to rule out bowel ischemia. Therefore, there should be a low threshold for laparoscopic exploration in suspected cases. The options for surgical management include reduction alone, reduction with enteropexy, and resection of the JJ with reconstruction of the anastomosis [28]. Simper et al. found a 100 % recurrence rate associated with reduction alone [23]. However, Varban et al. demonstrated that reduction with or without enteropexy could achieve equivalent morbidity and low recurrence compared with resection [28]. Most authors would recommend an *en bloc* resection of the affected segment and reconstruction of the anastomosis given the high prevalence of bowel infarction and risk of perforation [25, 26].

12.2.4 Bezoar

12.2.4.1 Hemobezoar (Intraluminal Blood Clot)

Acute postoperative bleeding after LRYGB is estimated to occur in approximately 3 % of patients [29]. Accordingly, obstruction due to an intraluminal clot is exceedingly rare [4]. Intraluminal bleeding is usually self-limiting and likely from the GJ anastomosis or from the staple lines of the gastric remnant. The JJ can also bleed or act as a locus for clot formation from the passing blood [30]. Staple line bleeding in laparoscopic RYGB has been shown to be three times more likely than in open RYGB. Although bleeding from anastomotic staple lines may be appreciated during surgery, the lumen of the gastric remnant cannot be visualized after the pouch is constructed.

The most common symptom in this type of obstruction is a sense of impending doom, which is likely associated with acute gastric remnant dilation. Tachycardia is reported to be the most common sign in these patients. As with any early

postoperative acute mechanical SBO, immediate treatment to avoid serious complications is required.

Placement of a gastrostomy tube is necessary to decompress the dilated gastric remnant and to permit enteral access in the postoperative period. Intraoperative endoscopy to suction the clot may be difficult given the distance required to reach the anastomosis. Endoscopic insufflation may also further dilate the bowel and lead to perforation. An enterotomy to evacuate the blood clot may be useful if the common channel appears to be completely collapsed. Anastomotic revision may be necessary if the anastomosis appears to be disrupted from the obstruction [11].

12.2.4.2 Phytobezoar

Phytobezoars are retained concretions of undigested fruit or vegetable fibers in the GI tract [31]. These are the most common foreign body of the gastrointestinal tract with SBO being the most commonly associated complication [32]. Nonetheless, SBO due to bezoar formation after RYGB is rather uncommon. Management can include chemical dissolution (i.e., cellulase), endoscopy (i.e., fragmentation and flushing), or surgical evacuation [31]. Complete impaction may require an enterotomy to remove the impacted phytobezoar, which can be done laparoscopically. Bowel resection is rarely indicated in these situations [32]. It is important to provide patients with appropriate nutritional counseling and/or psychiatric evaluation in the postoperative setting to help avoid future recurrences.

12.3 Extraluminal Obstructions

12.3.1 Incarcerated Ventral Hernia

Obesity, or more specifically central adiposity, is associated with an increased risk of umbilical and incisional hernia [33]. Although many bariatric surgeons have reported their experience with bowel obstruction from incarcerated abdominal wall hernias, there is still no consensus as to the optimal treatment of ventral hernia in bypass candidates. Ventral hernia in patients presenting for RYGB surgery has posed a therapeutic dilemma for two main reasons: the high recurrence rate after primary repair (~50 %) and the potential of mesh infection from contamination during surgery [33].

Umbilical hernias smaller than 3–4 cm in diameter can be closed primarily at the end of an RYGB using transabdominal sutures but can still have a recurrence rate of over 20 % [33]. If omentum is present in the sac, it should not be reduced. Instead, a rent can be made between the hernia and transverse colon to allow sufficient access to run the small bowel. If a hernia is found completely reduced, patients have an increased risk of developing SBO postoperatively. Some surgeons would advocate performing a sleeve gastrectomy over an RYGB in this setting, to avoid the potentially devastating complications associated with obstructed bypass patients. There is evidence that concomitant repair of ventral hernias with biological mesh can be a safe and effective alternative in these cases [33].

In a series reported by Cho et al. where closure of port-site abdominal fascia was not routine, the hernia incidence was 0.14 % [2]. Larger trocar size and cutting trocars have been associated with the development of port-site hernias [4]. Fortunately, dilating trocars have decreased the need for fascial closure of trocar sites <12 mm in diameter [4]. In a review by Koppman et al., port-site herniation resulted in bowel obstruction in 0.3 % of patients [4]. Hernias within the preperitoneal spaces have been reported, prompting some to suggest that peritoneal closure should be incorporated during port-site closure. A full-thickness closure can be performed safely using port-site closing devices [11].

12.3.2 Internal Hernia

A full discussion of internal hernia is provided in Chapter 11. However, a brief overview of this pathology as it leads to bowel obstruction is also included here. The incidence of internal hernia after LRYGB had been reported to be 1–4 % [12, 34, 35]. Antecolic, antegastric LRYGB approach significantly reduces the incidence of internal hernia compared to the retrocolic, retrogastric

approach because it eliminates the mesocolic defect [1, 2]. The predisposing factor for the development of internal hernia is that mesenteric fat is lost quickly with weight reduction and enlarges the surgically created mesenteric defects [11]. As well, the mesentery can end up tearing or loosening at the level of the sutures in cases where the defects have been closed [34]. There are several possible defects that can lead to internal herniation (Fig. 12.2).

1. **Transmesocolic**—Small bowel, usually the Roux limb, herniates through a surgically created defect in the mesocolon (only seen with a retrocolic approach). The transition point is usually proximal to the jejunojejunostomy at the level of the mesocolic window [36].
2. **Petersen's hernia**—Small bowel herniates behind the Roux limb mesentery through an opening anterior to the transverse mesocolon. It is the most frequent type found in patients with antecolic antegastric RYGB. CT imaging demonstrates a sac-like cluster of small bowel loops displaced in the left mid-abdomen [36].
3. **Mesojejunal**—Small bowel herniates through the mesenteric defect at the jejunojejunostomy. A large cluster of dilated bowel can be seen adjacent to the jejunojejunal anastomosis, typically pressed against the anterior abdominal wall without overlying omental fat [36].
4. **Jejunojejunal**—Small bowel herniates through the interjejunal space between the biliopancreatic staple line and a suture adjacent to the jejunojejunostomy. This hernia is usually a result of surgical technique rather than rapid weight loss. A transition point may be seen around the anastomosis [36].

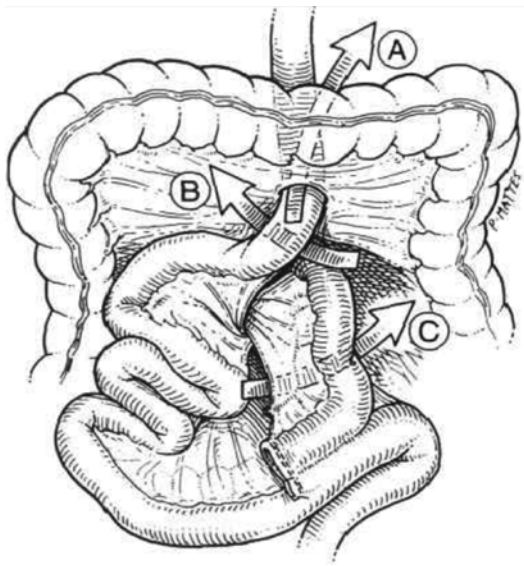

Fig. 12.2 Types of internal hernias. With permission from Higa KD, Ho T, Boone KB. Internal hernias after laparoscopic Roux-en-Y gastric bypass: incidence, treatment and prevention. Obes Surg. 2003;13(3):350–4 [12]. © Springer

Various intraoperative strategies can be employed to prevent internal hernias, such as meticulous closure of all mesenteric defects and Petersen's space, proper anastomotic orientation of the Roux limb (i.e., "right oriented"), nondivision of the small bowel mesentery, division of omentum only when too thick, and use of an antecolic approach [1]. Closure of defects using running nonabsorbable sutures should also be performed to decrease the rate of internal herniation [1]. Rodriguez et al. showed that it is better not to divide the mesentery after identifying a decrease in internal hernia rate from 15.5 to 1 % with this approach [5].

The timing of internal herniation is highly variable but typically symptoms present some months following surgery after substantial weight loss [36]. Clinical presentation can be quite vague and may be acute or chronic [34]. Nausea, vomiting, and intermittent epigastric abdominal pain radiating to the back are common symptoms of an internal hernia [11]. While symptoms of internal hernia may result from bowel obstruction, the pain is more commonly caused by bowel ischemia due to venous congestion. Signs of peritonitis suggest an acute abdomen that requires an emergent exploration.

Plain films can be unremarkable even in the presence of complete obstruction secondary to an internal hernia [12]. Owing to the altered bowel anatomy after an RYGB, diagnosis of SBO on CT can also be quite challenging and more likely to miss internal hernias [7, 8]. Early series have

reported that 20 % of patients with symptomatic internal hernias have completely normal contrast studies [12]. The most commonly encountered "subtle sign" on imaging is an abundance of small bowel or cluster of dilated bowel in the left upper quadrant [8]. Other CT findings suggestive of an internal hernia include the jejunojejunostomy staple line superior to the transverse colon, along with stretching or congestion of the mesenteric vessels. In the case of volvulus, the mesenteric vasculature may be seen to spiral, resulting in a "whirl" or "swirl" sign [4]. This sign was the best indicator of internal hernia after RYGB with an average sensitivity of 74 % and specificity of 83 % [35].

Laparoscopic evaluation may be safe in the absence of severe bowel dilation [3]. Principles of management include running the bowel starting at the terminal ileum and moving proximally, reduction of herniated bowel, and closure of all potential internal spaces using continuous non-absorbable suture [5]. Repair can be safely performed through previous laparoscopic trocar sites. An additional trocar can be placed in the lower abdomen to facilitate locating the terminal ileum and running the distal collapsed small bowel. If laparoscopy proves challenging, conversion to open surgery is always advised.

12.3.3 Adhesive Bands

Adhesions are the second most common cause of SBO in respective series by Abasbassi et al. with an incidence of 2.1 % [1]. The incidence is less than 1 % in most laparoscopic RYGB studies and 1.3–5 % in open RYGB studies [4]. CT imaging can be quite effective in the diagnosis of adhesive obstructions [8].

12.4 Gastric Remnant Dilatation due to Biliopancreatic Limb Obstruction

Acute gastric remnant dilatation is a rare postoperative complication of RYGB occurring in less than 0.8 % of cases [37]. It is generally the result of an obstruction at the level of the biliopancreatic limb or common channel [2]. It may occur due to obstruction of the distal anastomosis due to technical error, kinking, or other extrinsic compression. Obstruction of the biliopancreatic limb proximal to the common lumen will result in a dilated gastric remnant filled with fluid but not air [9]. The typical presenting symptoms include left upper quadrant abdominal pain, hiccups, and persistent tachycardia. Prompt surgical intervention is necessary since the gastric remnant cannot be decompressed with a nasogastric tube and acute gastric distension can result in staple line rupture or gastric wall perforation. This type of complication can significantly increase postoperative morbidity and mortality. Several reports have demonstrated that percutaneous gastrostomy tube placement by interventional radiology can be an effective method for temporizing decompression and potentially avoiding reoperation [38, 39]. Surgical management consists of gastrostomy tube placement for decompression and relief of the distal obstruction [37].

12.5 Roux-en-O Configuration

Roux-en-O misconstruction is a very rare but potentially devastating complication. This occurs when the proximal divided jejunum (i.e., biliopancreatic limb) is incorrectly identified as the distal jejunum and anastomosed to the gastric pouch [40]. The segment of the bowel connecting the excluded stomach to the gastric pouch thus creates a blind "O" loop (Fig. 12.3).

Strategies to prevent misconstruction at the time of surgery include correctly identifying the ligament of Treitz, keeping the biliopancreatic limb relatively short so that it will not reach the gastric pouch; and marking limbs shortly after jejunal transection with a clip or suture. In almost all cases, patients with a Roux-en-O present with chronic or protracted bilious vomiting. The presence of bilious vomiting in the absence of a mechanical common limb obstruction should always raise suspicion of misconstruction [40]. If not diagnosed early, this form of misconstruction can lead to severe malnutrition due to poor oral intake.

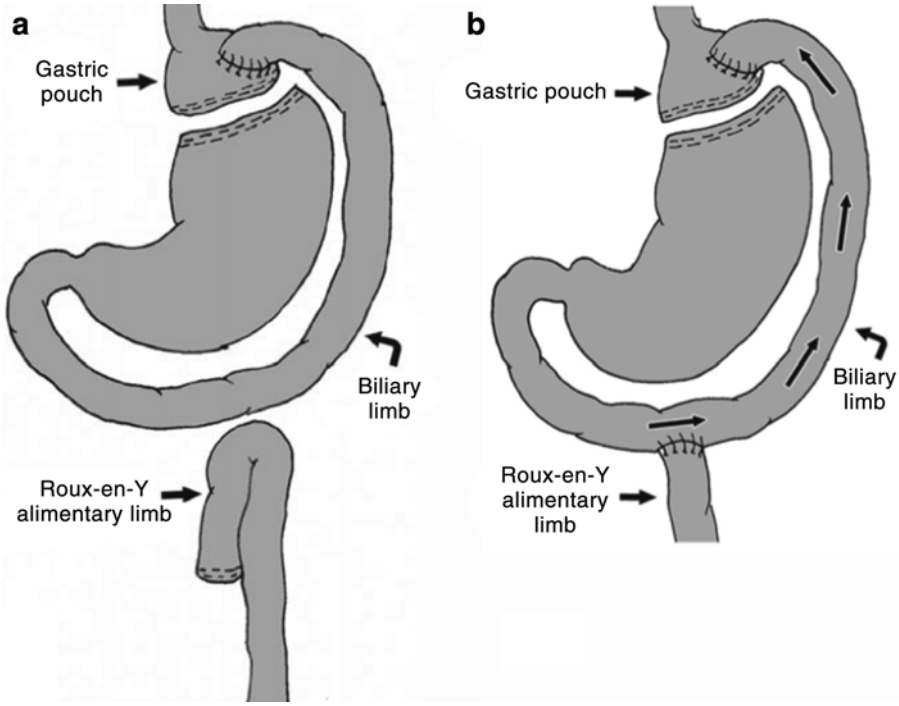

Fig. 12.3 (**a**) Disconnected. (**b**) Connected Roux-en-O misconstructions. With permission from Mitchell MT, Gasparaitis AE, Alverdy JC. Imaging findings in Roux-en-O and other misconstructions: rare but serious complications of Roux-en-Y gastric bypass surgery. AJR Am J Roentgenol. 2008;190(2):367–73 [40]. © American Roentgen Ray Society

Roux-en-O misconstruction can be considered either connected or disconnected to the distal jejunum. Postoperative diagnosis may be elusive, especially with connected Roux-en-O misconstruction since contrast fluoroscopy, CT, and abdominal plain films may appear normal [41]. The treatment for this complication is ultimately surgical exploration, either open or laparoscopically, to clarify the anatomy and reconstruct the anastomoses [41].

References

1. Abasbassi M, Pottel H, Deylgat B, Vansteenkiste F, Van Rooy F, Devriendt D, et al. Small bowel obstruction after antecolic antegastric laparoscopic Roux-en-Y gastric bypass without division of small bowel mesentery: a single-centre, 7-year review. Obes Surg. 2011;21(12):1822–7.
2. Cho M, Carrodeguas L, Pinto D, Lascano C, Soto F, Whipple O, et al. Diagnosis and management of partial small bowel obstruction after laparoscopic antecolic antegastric Roux-en-Y gastric bypass for morbid obesity. J Am Coll Surg. 2006;202(2):262–8.
3. Nguyen NT, Huerta S, Gelfand D, Stevens CM, Jim J. Bowel obstruction after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2004;14(2):190–6.
4. Koppman JS, Li C, Gandsas A. Small bowel obstruction after laparoscopic Roux-en-Y gastric bypass: a review of 9527 patients. J Am Coll Surg. 2008;206(3):571–84.
5. Rodriguez A, Mosti M, Sierra M, Perez-Johnson R, Flores S, Dominguez G, et al. Small bowel obstruction after antecolic and antegastric laparoscopic Roux-en-Y gastric bypass: could the incidence be reduced? Obes Surg. 2010;20(10):1380–4.
6. Shimizu H, Maia M, Kroh M, Schauer PR, Brethauer SA. Surgical management of early small bowel obstruction after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2013;9(5):718–24.
7. Gunabushanam G, Shankar S, Czerniach DR, Kelly JJ, Perugini RA. Small-bowel obstruction after laparoscopic Roux-en-Y gastric bypass surgery. J Comput Assist Tomogr. 2009;33(3):369–75.
8. Husain S, Ahmed A, Johnson J. Small-bowel obstruction after laparoscopic Roux-en-Y gastric bypass. Arch Surg. 2007;142(10):988–93.

9. Lewis CE, Jensen C, Tejirian T, Dutson E, Mehran A. Early jejunojejunostomy obstruction after laparoscopic gastric bypass: case series and treatment algorithm. Surg Obes Relat Dis. 2009;5(2):203–7.
10. Champion JK, Williams M. Small bowel obstruction and internal hernias after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2003;13(4):596–600.
11. Rogula T, Yenumula PR, Schauer PR. A complication of Roux-en-Y gastric bypass: intestinal obstruction. Surg Endosc. 2007;21(11):1914–8.
12. Higa KD, Ho T, Boone KB. Internal hernias after laparoscopic Roux-en-Y gastric bypass: incidence, treatment and prevention. Obes Surg. 2003;13(3): 350–4.
13. Cusati D, Sarr M, Kendrick M, Que F, Swain JM. Refractory strictures after Roux-en-Y gastric bypass: operative management. Surg Obes Relat Dis. 2011;7(2):165–9.
14. Ukleja A, Afonso BB, Pimentel R, Szomstein S, Rosenthal R. Outcome of endoscopic balloon dilation of strictures after laparoscopic gastric bypass. Surg Endosc. 2008;22(8):1746–50.
15. Catalano MF, Chua TY, Rudic G. Endoscopic balloon dilation of stomal stenosis following gastric bypass. Obes Surg. 2007;17(3):298–303.
16. Yimcharoen P, Heneghan H, Chand B, Talarico JA, Tariq N, Kroh M, et al. Successful management of gastrojejunal strictures after gastric bypass: is timing important? Surg Obes Relat Dis. 2012;8(2):151–7.
17. Ryskina KL, Miller KM, Aisenberg J, Herron DM, Kini SU. Routine management of stricture after gastric bypass and predictors of subsequent weight loss. Surg Endosc. 2010;24(3):554–60.
18. Go MR, Muscarella 2nd P, Needleman BJ, Cook CH, Melvin WS. Endoscopic management of stomal stenosis after Roux-en-Y gastric bypass. Surg Endosc. 2004;18(1):56–9.
19. Goitein D, Papasavas PK, Gagne D, Ahmad S, Caushaj PF. Gastrojejunal strictures following laparoscopic Roux-en-Y gastric bypass for morbid obesity. Surg Endosc. 2005;19(5):628–32.
20. Markar SR, Penna M, Venkat-Ramen V, Karthikesalingam A, Hashemi M. Influence of circular stapler diameter on postoperative stenosis after laparoscopic gastrojejunal anastomosis in morbid obesity. Surg Obes Relat Dis. 2012;8(2):230–5.
21. Mueller CL, Jackson TD, Swanson T, Pitzul K, Daigle C, Penner T, Urbacg D, Okrainec A. Linear-stapled gastrojejunostomy with transverse hand-sewn enterotomy closure significantly reduces strictures for laparoscopic roux-en-y gastric bypass. Obes Surg. 2013;1302–08.
22. Brolin RE. The antiobstruction stitch in stapled Rouxen-Y enteroenterostomy. Am J Surg. 1995;169: 355–7.
23. Simper SC, Erzinger JM, McKinlay RD, Smith SC. Retrograde (reverse) jejunal intussusception might not be such a rare problem: a single group's experience of 23 cases. Surg Obes Relat Dis. 2008;4(2):77–83.
24. Daellenbach L, Suter M. Jejunojejunal intussusception after Roux-en-Y gastric bypass: a review. Obes Surg. 2011;21(2):253–63. PubMed.
25. McAllister MS, Donoway T, Lucktong TA. Synchronous intussusceptions following Roux-en-Y Gastric Bypass: case report and review of the literature. Obes Surg. 2009;19(12):1719–23.
26. Edwards MA, Grinbaum R, Ellsmere J, Jones DB, Schneider BE. Intussusception after Roux-en-Y gastric bypass for morbid obesity: case report and literature review of rare complication. Surg Obes Relat Dis. 2006;2(4):483–9.
27. Ver Steeg K. Retrograde intussusception following Roux-en-Y gastric bypass. Obes Surg. 2006;16(8): 1101–3.
28. Varban O, Ardestani A, Azagury D, Lautz DB, Vernon AH, Robinson MK, et al. Resection or reduction? The dilemma of managing retrograde intussusception after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2013;9(5):725–30.
29. Awais O, Raftopoulos I, Luketich JD, Courcoulas A. Acute, complete proximal small bowel obstruction after laparoscopic gastric bypass due to intraluminal blood clot formation. Surg Obes Relat Dis. 2005;1(4):418–22.
30. Mala T, Sovik TT, Schou CF, Kristinsson J. Blood clot obstruction of the jejunojejunostomy after laparoscopic gastric bypass. Surg Obes Relat Dis. 2013;9(2): 234–7.
31. Roy M, Fendrich I, Li J, Szomstein S, Rosenthal RJ. Treatment option in patient presenting with small bowel obstruction from phytobezoar at the jejunojejunal anastomosis after Roux-en-Y gastric bypass. Surg Laparosc Endosc Percutan Tech. 2012;22(4):e243–5.
32. Sarhan M, Shyamali B, Fakulujo A, Ahmed L. Jejunal Bezoar causing obstruction after laparoscopic Roux-en-Y gastric bypass. J Soc Laparoendosc Surg. 2010;14(4):592–5.
33. Eid GM, Mattar SG, Hamad G, Cottam DR, Lord JL, Watson A, et al. Repair of ventral hernias in morbidly obese patients undergoing laparoscopic gastric bypass should not be deferred. Surg Endosc. 2004;18(2): 207–10.
34. Iannelli A, Buratti MS, Novellas S, Dahman M, Amor IB, Sejor E, et al. Internal hernia as a complication of laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2007;17(10):1283–6.
35. Parakh S, Soto E, Merola S. Diagnosis and management of internal hernias after laparoscopic gastric bypass. Obes Surg. 2007;17(11):1498–502.
36. Kawkabani Marchini A, Denys A, Paroz A, Romy S, Suter M, Desmartines N, et al. The four different types of internal hernia occurring after laparascopic Roux-en-Y gastric bypass performed for morbid obesity: are there any multidetector computed tomography (MDCT) features permitting their distinction? Obes Surg. 2011;21(4):506–16.
37. Han SH, White S, Patel K, Dutson E, Gracia C, Mehran A. Acute gastric remnant dilation after laparoscopic Roux-en-Y gastric bypass operation in

long-standing type I diabetic patient: case report and literature review. Surg Obes Relat Dis. 2006;2(6): 664–6.
38. López-Tomassetti Fernández EM, Arteaga González I, Diaz-Luis H, Carrillo Pallares A. Obstruction of the bypassed stomach treated with percutaneous drainage: an alternative treatment for selected cases. Obes Surg. 2008;18:233–6.
39. Hamoui N, Crookes PF, Kaufman HS. Percutaneous gastric drainage as a treatment for small bowel obstruction after gastric bypass. Obes Surg. 2007;17: 1411–2.
40. Mitchell MT, Gasparaitis AE, Alverdy JC. Imaging findings in Roux-en-O and other misconstructions: rare but serious complications of Roux-en-Y gastric bypass surgery. AJR Am J Roentgenol. 2008;190(2): 367–73.
41. Sherman V, Dan AG, Lord JM, Chand B, Schauer PR. Complications of gastric bypass: avoiding the Roux-en-O configuration. Obes Surg. 2009;19(8): 1190–4.

Food Intolerance in the Sleeve Patient: Prevention, Evaluation, and Management

13

Gregg H. Jossart

13.1 Introduction

Laparoscopic sleeve gastrectomy has emerged as an acceptable surgical option for the treatment of morbid obesity. The technical ease of this procedure relative to anastomotic procedures like Roux-en-Y gastric bypass is appealing. There is no foreign body, no anastomosis, and no intestinal bypass. The long-term risk profile is appealing as the risks of foreign body and intestinal bypass complications are eliminated. The preservation of the pylorus and the resection of most of the stomach may also offer hormonal and motility benefits that are not yet well understood. The seemingly absent technical difficulties of this procedure can be misleading. The gastric staple line is the longest of all the procedures and staple line dehiscence and fistula formation is a constant concern. Durable weight loss is also a concern. While the sleeve gastrectomy is generally a low-morbidity procedure, technical efforts to make the pouch smaller to improve weight loss and reinforcing the staple line to reduce dehiscence may yield an increase in obstructive type side effects and complications. Indeed, much of the early morbidity of a sleeve gastrectomy is related to dysphagia. It is generally more difficult to manage than with a gastric bypass because it is usually functional and not related to a discrete anastomotic narrowing or stricture. Moreover, postoperative problems from a suboptimal technique may not present for months or years after surgery, so it is difficult to promote one technical preference over another. This chapter aims to provide a framework to navigate the management of sleeve gastrectomy patients with food intolerance, dysphagia, and obstruction. Special consideration is given to preventive management.

13.2 History

Sleeve gastrectomy has emerged as an acceptable procedure for almost any bariatric patient. During the open bariatric surgery era, it formed the restrictive component of the duodenal switch [1]. The advent of laparoscopic bariatric surgery facilitated the sleeve gastrectomy as a first-stage, lower risk option in high-risk patients [2]. It has since proven to be a reasonable single-stage option for the lower BMI group of patients and for patients with unique contraindications to adjustable gastric banding or intestinal bypass procedures [3, 4]. The technique of sleeve gastrectomy has not been well standardized. The earliest patients tended to have larger pouches and less dissection at the hiatus. As the pouch

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_13

volume has decreased, the pouch has become a higher resistance gastric tube and the LES may be anatomically compromised by the disruption of the phreno-esophageal ligament [5]. This may lead to more reflux dysphagia and food intolerance. Narrowing at the angularis and inadequate resection of the fundus may create a spectrum of symptoms such as reflux, cough, vomiting, reduced satiety, dysphagia, and food intolerance [6]. One must also be aware that results are not static. Indeed, pouch malformations and thoracic migration leading to persistent regurgitation can occur over time [7, 8].

13.3 Sleeve Anatomy and Technique

The entire technique of laparoscopic sleeve gastrectomy is beyond the scope of this chapter and can be reviewed in this author's chapter describing the surgical technique [9]. However, it is worth mentioning key technical details that will reduce the incidence of postoperative food intolerance and dysphagia-related symptoms. Technical errors can lead to obstruction which in turn present as food intolerance, dysphagia, and related symptoms. It is critical to mobilize distally to within 1–2 cm of the pylorus and divide any adhesions that may tether the pylorus. This mobilization will allow for optimal placement of the first 1–2 staple cartridges and provide adequate resection of the antrum without creating stenosis at the angularis. The choice to mobilize less and create a bigger antrum may inadvertently lead to placing the end of the longer staple cartridge (60 mm) to close to the bougie at the level of the angularis and relative stenosis leading to obstructive symptoms may be created. It is also critical to mobilize the fundus and cardia off of the left crus so that adequate exposure is gained for the final staple cartridge. Failure to do so may lead to a retained fundus or cardia which can function like an atonic pouch or diverticulum and create obstructive type symptoms [6, 7]. The hiatus must always be examined for an obvious hiatal hernia or even laxity. How much to dissect and what type of repair are still controversial [10, 11]. This author probes anteriorly and along the left crus for any evidence of a sliding hernia and if present proceeds with a circumferential dissection and closure both anteriorly and posteriorly as well as a cardiopexy to reduce migration and/or recurrence. Inadequate detection of a hiatal hernia or failure to repair may dramatically increase postoperative reflux and regurgitation symptoms and lead to an erosive esophagitis with associated dysphagia. When stapling, it is important to create a uniform pouch. An hourglass-shaped pouch with a relatively larger antrum and fundus and a narrow incisura may lead to chronic food intolerance. It is important to apply the stapler, then rotate the stomach, and verify that the staple line will be equidistant both anteriorly and posteriorly from the lesser curvature. Any excessive traction anteriorly or posteriorly could lead to stenosis at any area along the staple line [12]. An omentopexy is recommended by some authors as the antrum and angularis can tend to coil, kink, or form an intermittent volvulus with associated vomiting problems; this may theoretically be improved if the stomach is tethered to the divided edge of omentum [13–16].

13.4 Preoperative Management

Most bariatric surgeons have a well-established protocol or checklist for patients in the week or two before surgery. Preoperative weight loss of anywhere from 5 to 20 % is often recommended as this can tend to reduce visceral fat, liver size, and peri-gastric fat. The enhanced exposure may allow for the construction of a more uniform sleeve and reduce the chance of leaving a retained fundus or a relatively larger cardia region. A mild bowel preparation to reduce the incidence of postoperative constipation and obstipation may reduce the early symptoms of food intolerance and bloating. A detailed reflux history and endoscopy, if indicated, are important as patients with active esophagitis or gastritis may have more dysphagia-type symptoms in the early weeks after surgery. It is reasonable to start a PPI medication 1 week prior to surgery and continue it for 1 month postoperative as many patients can

develop some level of esophagitis in the early postoperative period. This early postoperative esophagitis can lead to dysphagia. Reports have documented that up to 40 % of bariatric patients have existing GERD, esophagitis, and/or hiatal hernias and are at risk for an exacerbation of their reflux if it is not addressed at the time of surgery [17–19]. Routine versus selective endoscopy is controversial [20, 21]. Most of the findings at endoscopy are esophagitis and gastritis and are well treated with empiric antacids. Barrett's dysplasia, adenocarcinoma, and GIST tumors are rare but more common after age 50. Routine endoscopy after age 50 in all patients undergoing sleeve gastrectomy is a reasonable approach to rule out the above-noted findings. Helicobacter pylori may be prevalent in up to 10 % of patients preoperatively and may contribute or be indicative of active gastritis which may lead to postoperative food intolerance [22]. It is important to diagnose and treat preoperatively, or, if present on the postoperative pathology report, treat in the first few months after surgery.

The patient's current list of prescription medications and supplements should be carefully reviewed for what is mandatory. Many patients have a polypharmacy of either large, numerous tablets or unnecessary supplements that could lead to intermittent episodes of esophageal or gastric obstruction in the early postoperative period. Preoperative consent issues should be discussed with the patient in detail. Generally, the patient chooses the sleeve gastrectomy for its favorable safety profile and they tend not to anticipate problems with food intolerance, dysphagia, and obstruction. Chronic diabetics should be aware that they may have underlying gastroparesis and this may yield more dysphagia, vomiting, slower recovery, and a possible need for endoscopy and pyloric Botox® injection. All patients should be aware that the rescue procedure for a sleeve gastrectomy with severe obstruction may be a Roux-en-Y gastric bypass. The procedure is not reversible, only modifiable. The presence of Barrett's and its potential for progression are relative or even absolute contraindications for a sleeve gastrectomy as it is unlikely that the sleeve pouch could be used as a conduit for an esophagogastrectomy. Barrett's metaplasia or mild dysplasia should be treated with endoscopic ablation methods prior to offering any weight loss surgery. If well ablated, a sleeve gastrectomy may be an acceptable option. The last consent issue unique to the sleeve is that of reflux. Patients should understand that reflux and hiatal hernia problems may occur in all procedures and require additional operations or conversions to treat. Routinely recommending a gastric bypass over a sleeve gastrectomy for reflux issues is reasonable, but the surgeon must inform the patient of the risks of ulcers and bowel obstructions that can occur with the gastric bypass.

13.5 Perioperative Management

Most bariatric anesthesia protocols are well established and preemptive use of antiemetics upon arrival to the preoperative area and minimal use of narcotics tend to reduce the problems of early postoperative nausea. Additional medications that can reduce nausea and early vomiting include IV antacids, IV or sublingual benzodiazepines, IV or oral metoclopramide, or serotonin 5-HT3 receptor antagonist. Cardiospasm can occur shortly after surgery and create the sensation of chest tightness or pain and prevent the consumption of oral liquids. The sublingual anticholinergic hyoscyamine can reduce these spasms dramatically. Oral liquids are usually started the morning after surgery and patients are educated to start consuming 1 ounce (30 ml) every 10 min to achieve a goal of 6 ounces per hour. Any patient who can drink 6 ounces per hour of various liquids is safe for discharge. Patients not able to consume 6 ounces per hour may require additional days in the hospital. They may also require additional evaluation to include an esophagram or endoscopy. Upon discharge, patients should consume up to 60 ounces of liquids daily including the recommended protein drinks. They should drink when sitting or standing and not while in a supine position. They should not be supine within 30 min of drinking. It is reasonable to recommend they

elevate the head of the bed 30° for the first few weeks to help reduce the chance of early postoperative aspiration. After 2 weeks, patients may progress to soft foods for 2 weeks and then regular foods at 1 month.

13.6 Managing Early Food Intolerance, Dysphagia, and Vomiting

The altered gastric anatomy following a laparoscopic sleeve gastrectomy will induce some upper gastrointestinal symptoms. It is important to be able to recognize what is normal for a postoperative sleeve patient and what needs further diagnostic and therapeutic intervention. Carabotti et al. [23] reported on 97 sleeve gastrectomy patients who self-administered the validated Rome III Criteria symptom questionnaire for upper gastrointestinal symptoms. 95.6 % of patients were satisfied with the sleeve and would undergo it again; yet 91.9 % complained of upper gastrointestinal symptoms. Postprandial distress syndrome (early satiation, epigastric pain) occurred in 59 % of patients at a median follow-up of 13 months. Dysphagia was present in 19.7 % of patients. This study confirms that dysphagia, early satiation, and epigastric pain are common in the sleeve patient. It is part of the restrictive nature of the operation. What should not be common and should be evaluated quickly is severe or progressive dysphagia and repetitive vomiting or intolerance of most liquids and solids.

Early dysphagia or vomiting either will be present immediately in the hospital or can occur in the first month after surgery. It is important to carefully review with the patient what they are trying to consume, how fast, and if they are using any of their discharge medications (anticholinergics, proton pump inhibitors, antiemetics). Teach patients to call early or have a bariatric nurse or dietitian contact the patient in the first week after surgery to verify that they are consuming liquids well and have not developed a vomiting problem. Many patients will advance to soft and regular foods too early or try to eat too much too fast. It is critical not to ignore or delay the treatment of any vomiting problems as a patient can develop a B1 deficiency and severe neurological complications if left untreated. Generally, if basic recommendations regarding the use of medications or modifying the liquids they are consuming do not work, the patient may need to present to the emergency room for urgent evaluation. The differential diagnosis, tests, and treatment options are detailed in Table 13.1. Mild dysphagia, limiting the intake of liquids in the first few weeks, may be related to esophageal or gastric spasms. Usually, these respond well to anticholinergics and benzodiazepines. If they do not respond well, an obstruction

Table 13.1 Differential diagnosis, diagnostic options, and treatment options for sleeve gastrectomy patients with progressive dysphagia, vomiting, and food intolerance

Diagnosis	Test	Treatment
Esophageal spasm	Upper GI	Hyoscyamine, lorazepam
Cardiospasm	Upper Gi	Hyoscyamine, lorazepam
Stasis esophagitis	Endoscopy	PPI
Regurgitant esophagitis	Endoscopy	Dietary counseling
Esophageal ulcer	Endoscopy	PPI
Gastritis	Endoscopy	PPI
Stenosis	Upper GI	Endoscopic dilation
Pylorospasm	Upper GI	Endoscopic Botox injection
Gastroparesis	Upper GI, history	Endoscopic Botox injection
Improper alimentation	Negative upper GI and endoscopy	Dietary counseling

Comment: Dietitian and detailed oral intake history may be most helpful. Be present at upper GIs. Always provide IV fluids with multivitamin, thiamine, and folate

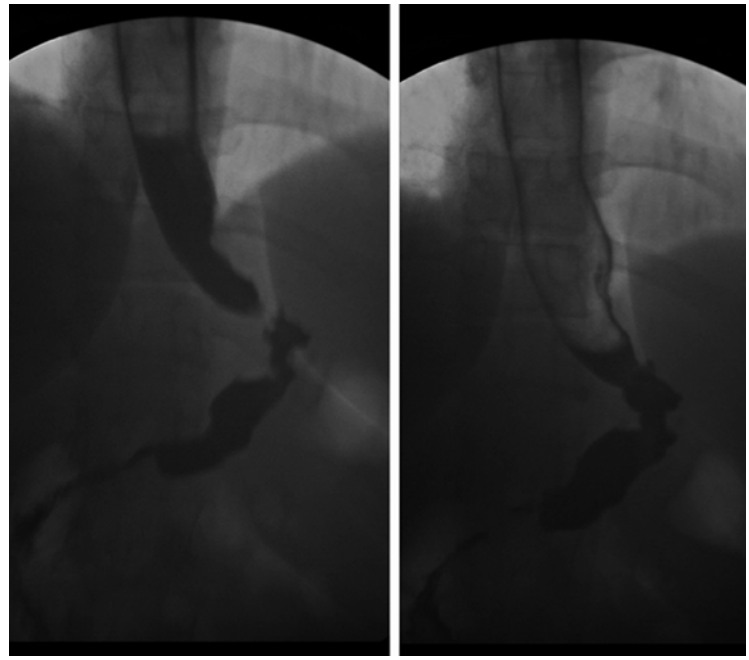

Fig. 13.1 Sleeve gastrectomy patient 3 weeks postoperative with chest tightness and poor oral intake. First image (*left*) reveals a narrow area in the proximal stomach. After a second drink of barium, the narrow area (*image on right*) opens up consistent with a spasm. Treatment was hyoscyamine three times a day. Symptoms resolved in 5 days

or stenosis may be present. An upper GI is relatively simple to perform and is excellent as a noninvasive method of diagnosing whether a patient is having spasms (Fig. 13.1) versus a fixed obstruction such as stenosis at the lower esophageal sphincter, at the incisura, or at the pylorus. It is wise to be present at the study as a radiologist is rarely experienced with these fine anatomic details. Areas that look like stenosis often prove to be spasms that relax after waiting for 10–20 s. If the upper GI proves only spasm, additional dietary counseling may be adequate. However, some patients may have much greater difficulty and an endoscopy may yield additional information and be therapeutic. Endoscopy can diagnose esophagitis which may occur from stasis due to a more distal obstruction or from regurgitation from eating too much or too fast. Esophageal ulcers would be documented and treated appropriately. Any areas of obstruction can be dilated either with just passage of the scope for patients who are in the first few weeks after surgery or balloon dilation for those who are more than a few weeks postoperative. If pyloric obstruction is suspected from the upper GI, a pyloric Botox® injection can be both diagnostic and therapeutic (Fig. 13.2). Pyloric obstruction or spasm may be more common in the more severe diabetics. Generally, the endoscopy with the associated inflation of the pouch is often therapeutic for patients with more severe spasms not responsive to medications. Rarely, a CT scan is indicated for vomiting and dysphagia-related problems. If the upper GI and endoscopy do not prove useful, a patient may have extrinsic obstruction from a hematoma or abscess that may require drainage or antibiotics. These types of extrinsic problems can be diagnosed and treated via CT scan.

Once objective studies have been completed and anatomical obstructions have been ruled out, another round of education with the patient is necessary to get them through this period of food intolerance. Patients may need to maintain a liquid diet for more than 2 weeks. Any patient who is vomiting frequently or cannot tolerate their vitamins may need to be admitted and supported with IV fluids, multivitamin, thiamine, and folate. This should be rare provided that the abovementioned diagnostic and therapeutic maneuvers have been done.

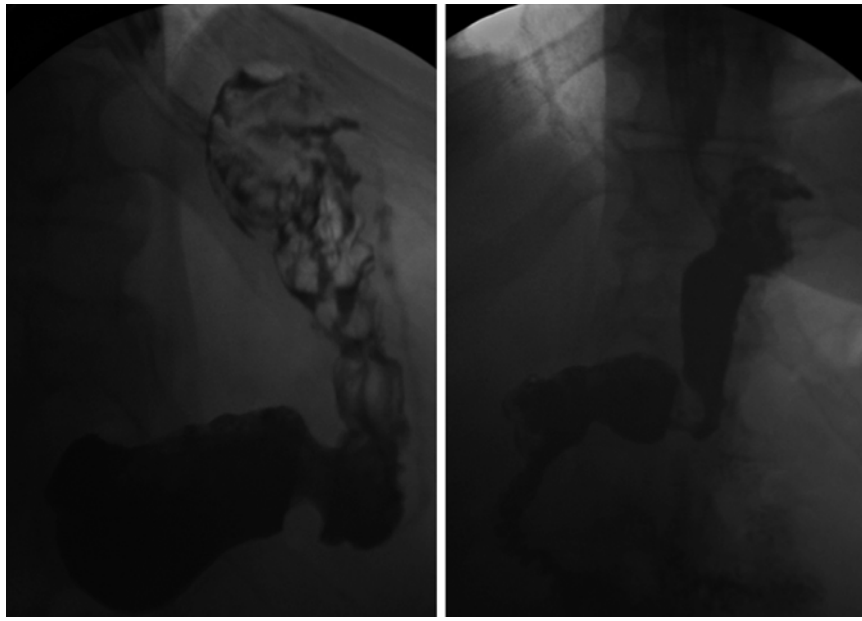

Fig. 13.2 Sleeve gastrectomy patient 30 days postoperative and cannot advance to soft foods without vomiting. He had diabetes for more than 5 years. The upper GI on the left reveals a lack of gastric emptying. He underwent an endoscopy which was negative. Botox® was injected into the pylorus. His symptoms resolved and a follow-up upper GI (*right*) revealed prompt gastric emptying

13.7 Stenosis and Volvulus

Symptoms of stenosis include regurgitation, progressive dysphagia, and even sialorrhea (drooling). Any patient who reports difficulty with liquids or worsening dysphagia should be evaluated before they get to the point of constant vomiting and drooling. As stated earlier, the upper GI and endoscopy are the diagnostic and therapeutic methods most appropriate for any sleeve patient presenting with a possible stenosis. Figure 13.3 is an excellent example of a patient who did not have signs of immediate obstruction but over a year developed worsening reflux and vomiting problems due to both a retained fundus and a relative narrowing at the angularis. The upper GI easily made the diagnosis.

The reported incidence of stenosis ranges from 0.1 to 3.9 % [24, 25]. Patients usually present with vomiting and obstructive symptoms in the first few weeks after surgery but can present as late as 27 months after surgery [25]. True stenosis with a fixed narrowing is more likely to present early after surgery. Functional stenosis due to torsion or prolapse at the angularis may present early or even a year or more after surgery as a progressive vomiting syndrome.

Burgos et al. [24] reported on 5 patients with gastric stenosis (all at the angularis) in a series of 717 patients. Treatment with single-balloon dilation was successful in one and with rigid Savary dilators in three others. One patient was converted to a Roux-en-Y gastric bypass after two Savary dilator attempts. While the occurrence of stenosis was only 0.69 %, the authors did note that in two of the five patients, the staple line was oversewn.

Ogra and Kini [25] reported 26 of 857 sleeve patients with stenosis (3.03 %). Three of the 26 had proximal gastric stenosis that responded well to a 20 mm balloon dilation. The other 23 patients had narrowing at the angularis and only 7 of those responded to balloon dilation. The nine patients with stenosis at the angularis that failed balloon dilation went on to additional procedures.

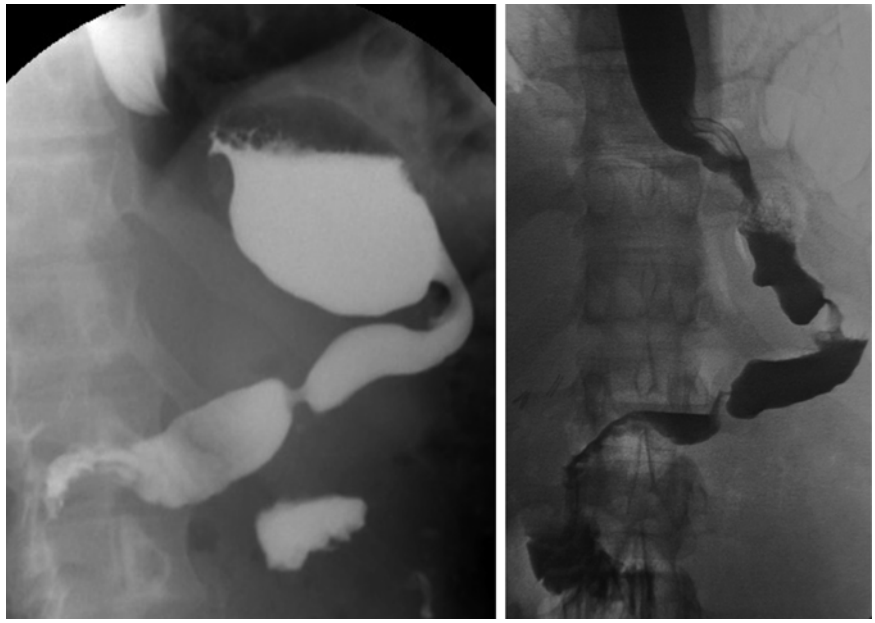

Fig. 13.3 Patient 2 years post-sleeve gastrectomy with chronic vomiting, reflux, and aspiration symptoms. Endoscope passed easily through the region of the angularis and pylorus. Preoperative upper GI (*image on left*) reveals a large retained fundus with relative narrowing at the angularis. Laparoscopic resection of the retained fundus resolved the obstructive symptoms (*postoperative image on right*)

Six underwent dilation with an achalasia balloon to 30 mm and three underwent placement of a self-expanding metal stent. Seven patients underwent achalasia balloon dilation first and only two of these needed to go on to stenting. The endoscope passed through all of these "stenosis" sites prior to dilation suggesting that the primary problem was torsion or prolapse at a relatively narrow angularis. The surgical description did not mention an omentopexy.

Vilallong and Himpens [26] reported on the laparoscopic management of persistent strictures after sleeve gastrectomy. Sixteen of 812 patients required surgical treatment. Endoscopic treatment was not attempted because the endoscope passed through the entire sleeve and the stenosis was deemed "functional." This was a complex group of patients, many with prior operations. The reason for treatment included dysphagia in all 16 patients plus, reflux in 8 patients, cachexia in 1, and eructation in 1. Seromyotomy was performed in 14 of the patients but had a leak rate of 35.7 %. Two patients underwent a wedge resection with a good result and three were converted to a Roux-en-Y gastric bypass.

Gastric volvulus has been described as a case report [27] presenting immediately after a sleeve gastrectomy. The patient underwent an antrectomy and a gastroileal anastomosis and did well. It is worthwhile to emphasize the key technical steps that may prevent nearly all of the stenosis and volvulus type of complications [9, 16]. Stapling too close to the angularis with a 60 mm stapler with a soft or absent bougie while simultaneously attempting to leave a larger antrum (>4 cm from pylorus) may create a relative narrowing at the angularis that can develop into an ischemic fixed stenosis or act as a lead point for torsion and prolapse. Directing the staple tip slight away from the angularis (tangential firing) and leaving a small "elbow" of gastric tissue may prevent this problem. In addition, posterior fixation of the angularis and antrum to the retroperitoneal tissue and omentum with a few sutures may also prevent most of the torsion and volvulus problems that seemingly present and are published as "stenosis."

References

1. Hess DS, Hess DW. Biliopancreatic diversion with a duodenal switch. Obes Surg. 1998;8(3):267–82.
2. Ren C, Patterson E, Gagner M. Early results of laparoscopic biliopancreatic diversion with duodenal switch: a case series of 40 consecutive patients. Obes Surg. 2000;10(6):514–23. discussion 524.
3. Lee C, Cirangle P, Jossart G. Vertical Gastrectomy for morbid obesity in 216 patients: report of two-year results. Surg Endosc. 2007;21(10):1810–6.
4. Bellanger DE, Greenway FL. Laparoscopic sleeve gastrectomy, 529 cases without a leak: short term results and technical considerations. Obes Surg. 2011;21:146–50.
5. Braghetto I, Korn O, Valladares H, Gutierrez L, Csendes A, Debandi A, et al. Laparoscopic sleeve gastrectomy: surgical technique, indications and clinical results. Obes Surg. 2007;17(11):1442–50.
6. Toro J, Lin E, Patel A, Davis S, Sanni A, Urrego H. Association of radiographic morphology with early gastroesophageal reflux disease and satiety control after sleeve gastrectomy. J Am Coll Surg. 2014;219(3):430–8.
7. Keidar A, Shussman N, Elazary R, Rivkind AI, Mintz Y. Dilated upper sleeve can be associated with severe postoperative gastroesophageal dysmotility and reflux. Obes Surg. 2010;20(2):140–7.
8. Baumann T, Grueneberger J, Pache G, Kuesters S, Marjanovic G, Kulemann R. Three-dimensional stomach analysis with computed tomography after laparoscopic sleeve gastrectomy: sleeve dilation and thoracic migration. Surg Endosc. 2011;25:2323–9.
9. Nguyen N, Scott-Connor C. The SAGES manual. Volume 2: Advanced laparoscopy and endoscopy. 3rd ed. New York: Springer; 2012. Chapter 4, Laparoscopic sleeve gastrectomy.
10. Daes J, Jimenez M, Said N, Dennis R. Improvement of gastroesophageal reflux sytmptoms after standardized laparoscopic sleeve gastrectomy. Obes Surg. 2014;24:536–40.
11. Mahawar K, Carr W, Jennings N, Balupuri S, Small P. Simultaneous sleeve gastrectomy and hiatus hernia repair: a systematic review. Obes Surg. 2015; 25:159–66.
12. Parikh A, Alley J, Peterson R, Harnisch M, Pfluke J, Tapper D. Management options for symptomatic stenosis after laparoscopic vertical sleeve gastrectomy in the morbidly obese. Surg Endosc. 2012;26:738–46.
13. Santoro S. Technical aspects in sleeve gastrectomy. Obes Surg. 2007;17(11):1534–5.
14. Afaneh C, Costa R, Pomp A, Dakin G. A prospective randomized controlled trial assessing the efficacy of omentopexy during laparoscopic sleeve gastrectomy in reducing postoperative gastrointestinal symptoms. Surg Endosc. 2014; [Epub ahead of print].
15. Abdo A, Averbach A. Omentopexy in vertical sleeve gastrectomy might be associated with reduced short and long term morbidity. SAGES 2014. http://www.sages.org/wp-content/uploads/posters/53281.jpeg?de989e.
16. Zachariah S, Chang P, Se En Ooi A, Hsin M, Yiu Kin Wat J, Huang C. Laparoscopic sleeve gastrectomy for morbid obesity: 5 years experience from an Asian center of excellence. Obes Surg. 2013;23:939–46.
17. Ayazi S, Hagen JA, Chan LS, et al. Obesity and gastroesophageal reflux: quantifying the associated between body mass index, esophageal acid exposure, and lower esophageal sphincter status in a large series of patients with reflux symptoms. J Gastroinest Surg. 2009;13(8):1440–7.
18. Dutta SK, Arora M, Kireet A, Bashandy H, Gandsas A. Upper gastrointestinal symptoms and associated disorders in morbidly obese patients: a prospective study. Dig Dis Sci. 2009;54(6):1243–6.
19. Csendes A, Burdiles P, Rojas A, Henriquez A. Pathological gastroesophageal reflux in patients with severe, morbid and hyper obesity. Rev Med Chil. 2001;129(9):1038–43.
20. Humphrey L, Meredith H, Morgan J. Detection of asymptomatic adenocarcinoma at endoscopy prior to gastric banding justifies routine endoscopy. Obes Surg. 2012;22:594–6.
21. Schgit A, Coblijn U, Lagarde S. Is esophagogastroduodenoscopy before Roux-en-Y gastric bypass or sleeve gastrectomy mandatory? Surg Obes Relat Dis. 2014;10(3):411–7.
22. Almazeedi S, Al-Sabah S, Alshammari D. The impact of Helicobacter pylori on the complications of laparoscopic sleeve gastrectomy. Obes Surg. 2014;24:412–5.
23. Carabotti M, Silecchia G, Greco F, Leonetti F, Piretta L, Rengo M. Impact of laparoscopic sleeve gastrectomy on upper gastrointestinal symptoms. Obes Surg. 2013;23(10):1551–7.
24. Burgos A, Csendes A, Braghetto I. Gastric stenosis after laparoscopic sleeve gastrectomy in morbidly obese patients. Obes Surg. 2013;23:1481–6.
25. Ogra R, Kini G. Evolving endoscopic management options for symptomatic stenosis post-laparoscopic sleeve gastrectomy for morbid obesity: experience at a large bariatric surgery unit in New Zealand. Obes Surg. 2014; [epub ahead of print].
26. Villalonga R, Himpens J, van de Vrande S. Laparoscopic management of persistent strictures after laparoscopic sleeve gastrectomy. Obes Surg. 2013;23:1655–61.
27. Del Castillo Dejardin D, Sabench Pereferrer F, Hernandez Gonzalez M, Blanco Blasco S, Vilanova A. Gastric volvulus after sleeve gastrectomy for morbid obesity. Surgery. 2012;153(3):431–3.

14 Gallstones and Common Bile Duct Stones in the Bariatric Surgery Patient: Surgical and Endoscopic Management

Dana A. Telem and Eric M. Pauli

Key Points

- Biliary disease in preoperative patients and those who have undergone gastric specific operations may be managed by standard methods.
- Routine evaluation of the CBD is recommended in patients planning to undergo or who have undergone RYGB or DS prior to or at the time of cholecystectomy.
- Choledocholithiasis in the RYGB or DS patient poses a technical challenge. Management is dependent on local expertise, anatomic constraints, urgency of procedure, and need for further endoscopic or operative intervention.
- Endoscopic and laparoscopic options should be the first-line approach.
- Operative management via choledochotomy or drainage procedure remains the standard of care for those in whom endoscopic management or transcystic exploration has failed.

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14.1 Introduction

Gallstones are a common condition present in approximately 12 % of the adult population. While many people are asymptomatic, a significant percentage will develop clinical manifestations consistent with biliary disease. Symptomatic cholelithiasis results in over 600,000 hospitalizations and 500,000 operations annually—making cholecystectomy one of the most commonly performed operative procedures in the USA. Choledocholithiasis complicates 10–15 % of these cases [1]. Although many risk factors have been identified which increase the likelihood of developing gallbladder disease, two of the major causes are obesity and rapid weight loss [2, 3]. As such, one would anticipate bariatric patients to be at higher risk for biliary disease both pre- and postoperatively. This chapter explores management of biliary disease in the bariatric patient. Specifically, it addresses issues unique to the bariatric patient related to diagnosis and timing of cholecystectomy and management of common bile duct stones. Particular focus is placed on the patient status post-Roux-en-Y gastric bypass (RYGB) and duodenal switch (DS).

14.2 Clinical Presentation and Diagnostic Work-Up

Patients with biliary disease may have variable presentations. While right upper quadrant pain following ingestion of a fatty meal remains the

D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_14

typical pain associated with biliary colic or cholecystitis, many patients present atypically. Presentation with epigastric pain, mid-back pain, or vague abdominal pain is not uncommon. Acuity and timing of presentation also vary. For preoperative patients a detailed history and physical should be performed to assess for biliary symptoms. If positive, a work-up consisting of ultrasound should ensue. Routine preoperative screening in asymptomatic patients is not mandatory and typically left to the discretion of the operating surgeon [4, 5].

For postoperative patients clinical diagnosis may be challenging. Presentation with abdominal pain following bariatric surgery may represent a myriad of diagnoses, several of which can be life threatening. Biliary disease following a bariatric procedure must be distinguished from potentially life-threatening conditions such as anastomotic or staple line leak, slippage of gastric band with incarcerated stomach, marginal ulcer, and internal hernia. The choice of imaging modality should be based on acuity of presentation, timing of and prior bariatric procedure, clinical examination, and laboratory analysis. If a high index of suspicion for biliary disease exists, ultrasound remains the imaging modality of choice to assess for cholelithiasis and cholecystitis. The sensitivity of ultrasound, however, for detecting common bile duct (CBD) stones remains low at less than 25 % [6, 7].

Proper identification of patients with choledocholithiasis is central for assistance with perioperative planning and option assessment for access to the biliary tree. For patients with clinical symptoms consistent with choledocholithiasis and/or abnormal liver function tests (LFT) or a dilated biliary tree on imaging CBD evaluation should be performed. Evaluation can be performed preoperatively with imaging modalities including magnetic resonance cholangiopancreatography (MRCP) or endoscopic ultrasound (EUS). Alternatively, intraoperative evaluation with cholangiogram or ultrasound may also be performed [8–11].

The question arises as to whether routine evaluation of the CBD is necessary in patients with a low index of suspicion for choledocholithiasis. For gastric specific operations, namely sleeve gastrectomy (SG) and adjustable gastric banding (AGB) or older vertical (or horizontal) banded gastroplasty (VBG), the approach to assessment of the biliary tree should not differ from any other patient and is left to the discretion of the clinician. For patients who are planning to undergo or have undergone RYGB or duodenal switch (DS), consideration should be made for routine CBD evaluation. It is estimated that asymptomatic choledocholithiasis is identified in up to 5 % of patients on routine cholangiogram [12]. Given the challenge of postoperative management of the biliary tree secondary to anatomic alterations, early identification of choledocholithiasis in RYGB or DS patients is ideal for operative planning and maximization of intervention options.

14.3 Management

14.3.1 Preoperative Patients

14.3.1.1 Symptomatic Cholelithiasis and Acute Cholecystitis

For patients with symptomatic biliary disease, laparoscopic cholecystectomy is recommended before or at the time of bariatric procedure. Timing of cholecystectomy depends on severity and presentation of symptoms. If clinical symptoms allow, cholecystectomy should be performed at the time of bariatric surgery. While operative time may be increased, performing the operation simultaneously avoids a second operation. No increased risk of short- or long-term morbidity is conferred by performing a concurrent cholecystectomy [13, 14]. For patients scheduled to undergo RYGB or DS, preoperative or intraoperative CBD evaluation is recommended.

14.3.1.2 Asymptomatic Cholelithiasis

Controversy exists regarding performance of prophylactic cholecystectomy for asymptomatic cholelithiasis at the time of bariatric surgery. Prior to the adoption of laparoscopy, cholecystectomy was commonly performed during open bariatric procedures. Recent

literature, however, questions the necessity of this practice. The risk of developing symptomatic biliary disease requiring cholecystectomy following a bariatric procedure ranges from 6 to 10 %. No increased patient morbidity or mortality has been demonstrated between cholecystectomy performed at the time of or after bariatric procedure [15–17]. Given the low frequency of postoperative cholecystectomy without increased patient morbidity, current data does not support routine prophylactic cholecystectomy at the time of bariatric surgery. This is reflected by national trends which demonstrate a significant decrease in performance of concomitant cholecystectomy over the past decade, from 26.3 % in 2001 to 3.7 % in 2008 [18].

14.3.1.3 Choledocholithiasis

Patients with common bile duct stones identified prior to or at the time of their bariatric operation are managed similarly to nonbariatric patients. CBD stones identified preoperatively by imaging studies or intraoperatively by cholangiogram may be managed by endoscopic, operative, or percutaneous approaches. Timing and modality utilized depend on several factors including size and location of stone, patient anatomy, surgeon comfort level and preference, hospital resources, planned bariatric operation, and clinical presentation. Management options include the following:

Endoscopic

Endoscopic retrograde cholangiopancreatography (ERCP) is the most frequently performed and preferred modality for the management of choledocholithiais—specifically for retained stones following cholecystectomy. The purpose of this procedure is to both extract the stones and drain the biliary tree. For this procedure, an endoscopy is performed with a side-viewing endoscope (duodenoscope) to the second portion of the duodenum. The ampulla is identified and a sphincterotomy is performed. The bile duct is then cleared and a biliary stent may or may not be placed. In experienced hands, the success rate of ERCP is 85–90 %. Factors predictive of failure include CBD stone greater than 2 cm and unfavorable anatomy such as the ampulla situated within a diverticulum [10, 19]. Complications of sphincterotomy and stone extraction occur in approximately 10 % of cases. These include bleeding (2 %), perforation (1 %), cholangitis (2 %), pancreatitis (2 %), and bile duct injury (<1 %). The mortality rate following ERCP with sphincterotomy is approximately 1 % [20].

In the absence of cholangitis, ERCP may be non-urgently performed preoperatively or postoperatively. While no increased patient morbidity is conferred based on timing of ERCP, preoperative performance is recommended in case the procedure is unsuccessful and operative management is warranted [10, 19]. For patients undergoing RYGB or DS preoperative or intraoperative ERCP is preferred. Postoperative anatomic challenges increase the technical difficulty and significantly reduce the success rate of postoperative ERCP (without transgastric assistance) even in the most experienced of hands [21].

Operative

Operative management of choledocholithiasis may represent surgeon preference or may be required for CBD stones that are not amenable to endoscopic retrieval. Surgical methods vary and may be attempted by both a laparoscopic or open approach. In addition, hybrid or "rendezvous" techniques have been described to facilitate performance of a previously unsuccessful ERCP. Prior to initiation of any of these approaches, a repeat intraoperative cholangiogram (IOC) should be performed in patients with preoperative identification of a CBD stone as many will pass spontaneously [10]. Additionally, an attempt should first be made to clear the CBD by administering intravenous glucagon and/or flushing the bile duct with isotonic sodium chloride if small stones or sludge are demonstrated on the cholangiogram. This may be successful in up to 20 % of cases precluding the necessity for further intervention [22].

(a) Transcystic Common Bile Duct Exploration

In patients with a gallbladder, this is the preferred method to clear the CBD of stones during laparoscopic cholecystectomy. For patients undergoing RYGB or DS with CBD stone identified intraoperatively, this is a valuable option. While several techniques have

been described, the principle of the procedure follows. After identification of CBD stones on IOC, a soft hydrophilic guide wire is passed into the CBD through the cholangiogram catheter under fluoroscopic guidance. Once the position of the wire in the CBD is confirmed, dilation of the cystic duct is performed. Following adequate dilation, CBD stone extraction may be attempted under fluoroscopic guidance or with the use of a flexible choledochoscope. Completion cholangiography is required to ensure adequate clearance of the duct and identify any potential injuries. In well-selected patients in experienced hands, the success rate for this approach is 80–95 %. Failure is typically secondary to unfavorable cystic duct anatomy and presence of large or impacted stones [23–25].

(b) Choledochotomy

Traditionally, open choledochotomy has been the standard of care for the treatment of choledocholithiasis in ducts greater than 4–6 mm. While laparoscopy is technically challenging, it may be considered particularly in cases where the CBD is dilated greater than 10 mm [26]. Nonetheless, choledochotomy remains a viable option in situations where both ERCP and laparoscopy have failed. Choledochotomy is typically performed by placing two traction sutures on either side of the intended choledochotomy incision on the CBD distal to the cystic duct. The anterior wall of the CBD is then opened longitudinally for a distance of approximately 1–1.5 cm and the CBD cleared. Once cleared, closure of the CBD is performed with a monofilament absorbable suture. Closure traditionally occurs over a t-tube; however in larger diameter ducts with low risk of stricture this step may be omitted at the surgeons' discretion [10].

(c) Drainage Procedures

Transduodenal sphincteroplasty entails a retrograde approach to the exploration and clearance of the CBD. This is the preferred procedure for smaller caliber ducts. In this procedure, a sphincterotomy is performed at the 11-o'clock mark in order to avoid the pancreatic duct and carried for a distance of approximately 1 cm. Advantages of this procedure are that it avoids a choledochotomy and it facilitates drainage [10].

Choledochoduodenostomy is the most commonly employed drainage procedure. The anastomosis can be performed either in a side-to-side or end-to-side fashion. One complication that can ensue from this procedure is reflux of food particles into the CBD resulting in obstruction. As such, *choledochojejunostomy* may be performed either in continuity or as a Roux-en-Y loop [10, 27].

(d) Laparo-Endoscopic "Rendezvous" for Difficult Bile Duct Cannulation

For patients with failed ERCP secondary to difficult cannulation or large impacted stones, antegrade transcystic cannulation can be performed either as a one-step or a two-step procedure. For the procedure a guide wire is introduced via the cholangiography catheter and advanced into the duodenum under fluoroscopic guidance (Fig. 14.1). The visible end of the guide wire is then snared and pulled through the operating channel of the duodenoscope. A sphincterotome is then mounted over the guide wire and ad vanced to complete the cannulation of the bile duct.

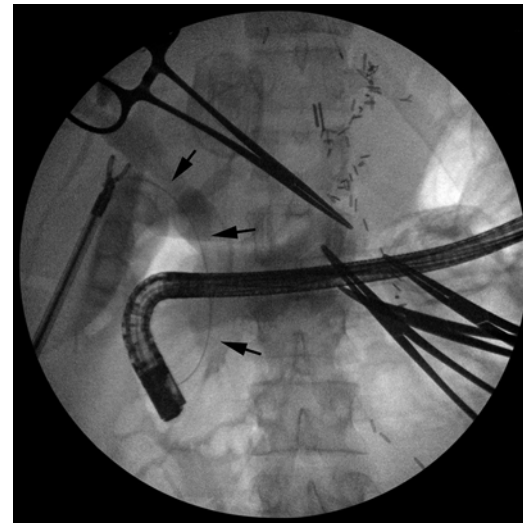

Fig. 14.1 Fluoroscopic image of a "rendezvous" transgastric assisted ERCP. *Arrows* denote the guide wire being passed from a cholangiogram catheter, down the common bile duct, and into the duodenoscope

In addition to its reported success rate of up to 88 %, this technique may reduce the incidence of postoperative ERCP pancreatitis and pancreatic injury [28, 29].

Percutaneous. Percutaneous management of choledocholithiasis is traditionally performed by interventional radiology via a transhepatic approach. This approach is indicated for patients in whom ERCP has failed and/or are poor operative candidates. To perform this procedure, confirmation of choledocholithiasis is performed via a percutaneous transhepatic cholangiogram. Once confirmed an external biliary catheter is placed. The tract is then dilated over 2–6 weeks by placement of progressively larger catheters until a 16 French size is reached. Once dilation is complete, the CBD stones are then extracted. The success rate for this procedure is reported between 75 and 96 %. The associated morbidity and mortality rate of this procedure are 10 % and 1 %, respectively. Major complications include bleeding, duct injury, bile leakage, and cholangitis [30, 31].

14.3.2 Postoperative Patients

For patients who present with symptomatic biliary colic or cholecystitis, management is the same. Laparoscopic cholecystectomy should be performed with timing based on acuity of presentation, clinical exam, and patient preference. For the patient status post-RYGB or DS, preoperative or intraoperative evaluation of the CBD is recommended. Routine inspection of limbs and potential spaces for occult internal hernia should also be considered at the time of cholecystectomy.

For patients with choledocholithiasis, management options remain the same in patients who have undergone gastric specific operations such as SG, AGB, or VBG. For patients who have undergone AGB, the band may require deflation prior to ERCP to accommodate the endoscope. Previously described surgical management options remain the same for patients who have undergone RYGB or DS. Endoscopic management, however, is technically challenging secondary to anatomic constraints. Accessing the duodenum is difficult via standard endoscopic approaches secondary to the length of the alimentary limb. As such, novel techniques have been developed and employed to facilitate duct clearance via ERCP. The remainder of this chapter focuses on endoscopic CBD management options specific to this patient population.

14.3.2.1 Management of Choledocholithiasis in RYGB and DS Patients

Surgically Assisted ERCP

Transgastric Access

Surgically assisted ERCP, generally performed laparoscopically, involves the creation of a transluminal access point to permit subsequent endoscopic access to the ampulla. For RYGB patients, the remnant stomach is the preferred location because it is generally easily accessed, is defunctionalized (creating a low risk of significant postoperative leak), and places the side-viewing duodenoscope in a standard position for ERCP. This is the most widely reported method in the literature [32–45].

Access to the body of the remnant stomach varies by author with some preferring more proximal access and some preferring more distal access [32, 34, 37, 38, 45]. It is important, however, not to place access too close to the pylorus as this can make endoscopic navigation and endoscope stability more difficult. Several methods of access have been described, including the creation of a gastrotomy with direct placement of an ethylene oxide gas-sterilized endoscope through the abdominal wall. Alternatively, a 15 mm laparoscopic trocar can be placed through the abdominal wall and into the remnant gastrotomy [37, 45]. A non-sterile endoscope can subsequently be passed into the stomach through a sterile ultrasound probe cover placed onto the trocar itself.

Standard ERCP methods are then used to clear the common bile duct. One notable difference is the endoscopist's position; because the patient is supine for laparoscopy, the endoscopist's position is reversed making cannulation more challenging. In this circumstance a "rendezvous" technique, as

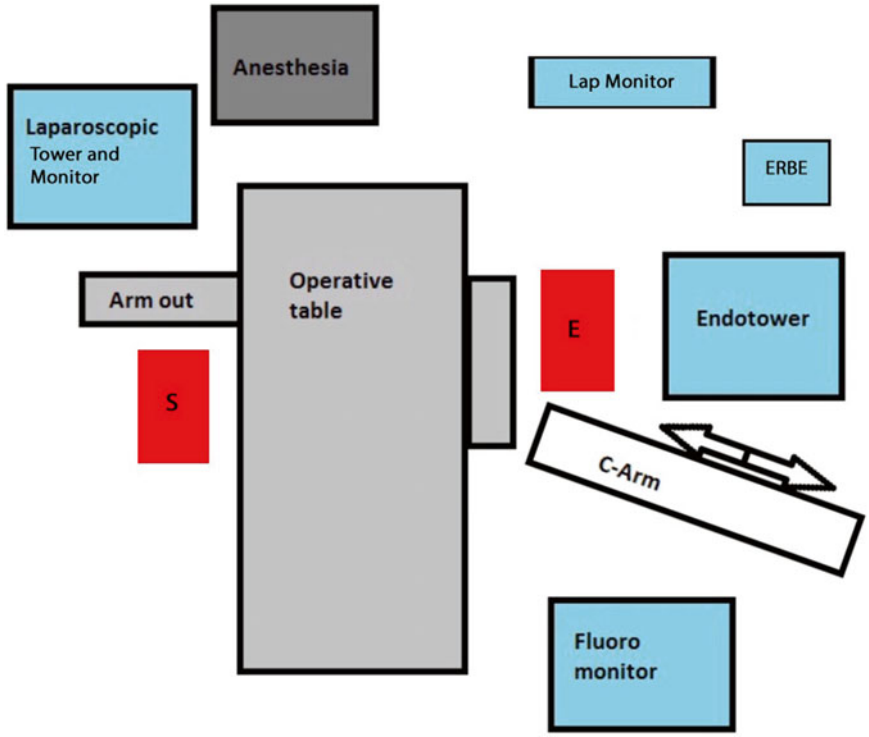

Fig. 14.2 Recommended room setup for transgastric assisted ERCP (E = endoscopist, S = surgeon)

described earlier, can greatly facilitate ERCP (Fig. 14.1) [46]. At the conclusion of the procedure the gastrotomy can be closed with sutures, resected with a stapler, or converted into a gastrostomy. Gastrostomy formation permits repeat transabdominal access to the remnant stomach without the need for additional surgery. This is the preferred method when additional endoscopic biliary interventions are anticipated.

Transgastric ERCP has a high technical success rate. A recent literature review of 113 patients undergoing transgastric ERCP noted technical success in 112 (98.8 %) with a complication rate (7.2) similar to ERCP alone [45]. The sole failure was due to an impacted stone in the ampulla [34]. While there is added morbidity (3.6 %) from laparoscopic access to the remnant stomach (leak, wound infection), many of these patients require an additional surgical procedure (most notably cholecystectomy) that can be conducted simultaneously under a single anesthetic setting. Lysis of adhesions and reduction of internal hernias are also common interventions performed [40, 47]. While this method is quite beneficial, it can require significant coordination between the surgeon, the endoscopist, and the ancillary staff. Adequate room setup to optimize both laparoscopic and endoscopic interventions is important (Fig. 14.2).

Transjejunal Access

In lieu of a gastrotomy, the small bowel can be accessed in the biliopancreatic limb in both RYGB and DS patients to permit retrograde access to the ampulla [47, 48]. For DS patients, this is the preferred route of access for endoscopic CBD access. Access to the BP limb means that endoscopic visualization of the ampulla will be retrograde, and ERCP can be conducted with either a side-viewing or forward-viewing scope. At the conclusion of the procedure, the enterotomy can be closed with sutures, turned into a stapled entero-enterostomy, or converted into a large-caliber jejunostomy. There are only case reports of this technique in the literature, which have all been successful [47, 48].

No case series are described to report the technical success rate or complications.

Per-Oral ERCP

In patients with RYGB anatomy, direct access to the ampulla with a duodenoscope is an extreme technical challenge. This is due to a combination of factors including the distance that needs to be traversed to reach the ampulla due to proximal gastric division (40 cm esophagus, 5–10 cm gastric pouch, 100–150 cm alimentary limb, 50–80 cm biliopancreatic limb) as well as the fact that safely navigating the small bowel with side-viewing duodenoscope is challenging even over short distances.

To address these issues, multiple alternative means of accessing the ampulla with forward-viewing endoscopes have been described. Even when successful in reaching the ampulla, such methods of bile duct clearance are hampered by several technical factors; the ampulla is approached from the distal duodenum at an upward angle, there is no channel elevator to facilitate cannulation, and traditional accessories for ERCP (sphincterotomes, balloons, stents) may be too short for the scope or angled inappropriately to permit easy bile duct access or be too wide to fit through the accessory channel. Some of these limitations, however, can be overcome with sufficient technical prowess. The literature supports several methods of per-oral access to the biliary tree in bariatric patients.

ERCP with a Standard Duodenoscope

As noted above, patients with gastric specific operations can undergo ERCP via standard methods. For RYGB patients, a 33 % technical success rate with this method has been reported [49]. Because of this low success rate, this method is rarely utilized and should be considered only in patients with very short bypass limbs and no other viable options for clearing the duct.

ERCP via Push Enteroscopy

ERCP utilizing a push enteroscope or a pediatric colonoscope has also been described. Navigation with these types of scopes is time consuming and requires frequent loop reduction maneuvers, external pressure to prevent loop formation, and changing patient position. Most series describing this method unfortunately include both bariatric and nonbariatric patients [50, 51]. When considering just bariatric patients, the reported success rate is only 45 % [51].

ERCP via Balloon-Assisted Enteroscopy

Balloon-assisted endoscopic methods utilize high-volume, low-pressure balloons and overtubes to permit small bowel stabilization on the endoscope. Single-balloon techniques have one balloon attached to an overtube while double-balloon methods have two balloons (one on an overtube, one on the endoscope insertion tube). These methods have a proven track record of deep intubation of the small bowel for a variety of endoscopic interventions. They have gained favor in the bariatric population for their ability to navigate the long limbs of the RYGB anatomy to access the bile ducts without the need for surgery. Technical success rates in reaching the ampulla with balloon-assisted methods range from 55 to 100 % with an 83–100 % chance of biliary orifice cannulation once there [52–60]. Therapeutic success of 77–100 % is reported if the biliary orifice is able to be cannulated [52–60]. When taken together, these methods are overall of low risk, but have a higher technical failure rate than the surgical and hybrid methods described above. They are still not universally available and require a skilled endoscopist.

ERCP via Spiral Enteroscopy

Spiral endoscopes have a rotating overtube with a helical design that pleat or sleeve the small bowel onto the endoscope to permit forward motion. While not widely available, early reports have indicated a modest success rate (64–77 %) at reaching the biliary orifice [60–62].

Percutaneous Access to the Biliary Tree

Any method of percutaneous access to the gastrointestinal tract can theoretically be used to access the biliary tree. This includes percutaneous gastrostomy tubes, jejunostomy tubes, cholecystostomy tubes, and transhepatic tubes. Following track maturation and upsizing, flexible endoscopes can be used to access the biliary tract (directly or indirectly) to manage disease processes.

Novel Therapies on the Horizon

Gastro-Gastric Fistula Formation

The exploitation of a preexisting gastro-gastric fistula (GGF) to permit per-oral endoscopic interventions in the excluded portions of the RYGB foregut has been described. Due to the close proximity of the gastric pouch and the proximal remnant stomach, some authors have proposed the intentional endoscopic creation of a GGF as a means of accessing the excluded stomach. Under fluoroscopic or endoscopic ultrasound guidance, needle access is obtained from the pouch into the remnant stomach and a guide wire is passed. Over-the-wire balloon dilation and/or enteral stent placement then follows. Endoscopic interventions (include ERC) can then be conducted via this GGF tract. The advantage of this method is that a standard duodenoscope can be used to reach the ampulla without the need for surgical incisions or a gastrostomy and the full array of ERCP accessories are at the disposal of the endoscopist. Obvious disadvantages include the creation of an acute perforation of the pouch and remnant stomach (with the inherent risk of leak), the possibility of long GGF persistence following the intervention, and stent removal (with the risks of marginal ulcer formation, weight regain, and recrudescence of diabetes due to the presence of food within the stomach and duodenum). Novel methods of perforation and fistula closure, including over-the-scope clips and endoscopic suturing devices, may negate some of the risks of intentional GGF creation and may ultimately make this method a viable endoscopic option.

Percutaneous Cholelithectomy

Percutaneous transhepatic access and cholangioscopy have an established track record for CBD clearance in the RYGB patient (as described above). More recently, it has been recognized that in some patients with a gallbladder in situ a percutaneous cholecystostomy tube can both decompress the acutely obstructed CBD and permit an access route for complete removal of all gallstones. Following cholecystostomy tube placement, wire access is obtained through the CBD via the cystic duct. The cystic duct can be dilated to permit larger instruments to be passed into the CBD to clear the duct and perform a sphincteroplasty. Subsequently, standard choledochoscopic methods can be used to remove gallstones from the gall bladder itself, negating the need for subsequent cholecystectomy. This method requires several favorable factors including the presence of a gallbladder, favorable cystic duct anatomy, and size-appropriate gallstones. Multiple interventions are required but this method may be beneficial in poor operative candidates.

Device-Assisted Endoscopy

Several investigators have described case reports of transprosthetic endoscopy to permit immediate access and therapy via the remnant stomach. Wire access to the remnant stomach is first obtained via double balloon-assisted PEG method or via trans-abdominal ultrasound [63]. The tract is subsequently dilated to 20 mm, and T-anchors and/or stents are placed to secure the tract (Fig. 14.3). ERCP via the stent is then performed using a standard duodenoscope and accessories. The stent is then removed and converted to a gastrostomy tube at the conclusion of the procedure.

Endoscopic Ultrasound-Assisted Biliary Access

Utilizing EUS methods, Weilert and colleagues gained wire access from the gastric pouch into the left lateral segment biliary tree. A wire was able to be advanced across the ampulla in six consecutive patients which permitted successful biliary intervention in all six, four using an antegrade transgastric, transhepatic method and two using a retrograde DBE technique that was able to successfully rendezvous with their antegrade-placed wire [64].

14.4 Choosing the Route of CBD Clearance

Because of the variety of options available, no single method of CBD access following RYGB is considered the gold standard. Table 14.1 provides an overview of the five major methods of bile duct

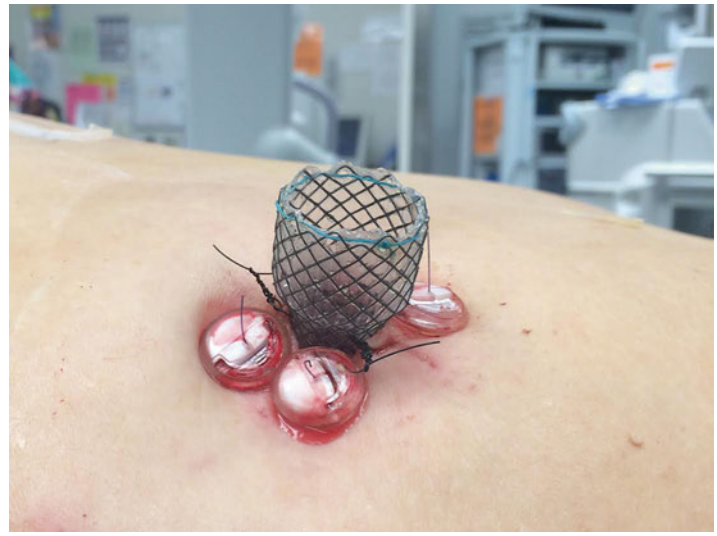

Fig. 14.3 Gastrointestinal T anchors (*white buttons*) and percutaneous fully covered stent placement into remnant stomach to permit subsequent transprosthetic-ERCP

clearance. The bariatric surgeon, in conjunction with the endoscopist, must determine which method has the highest chance of success and the lowest risk of morbidity for the individual patient. Factors to consider include the following:

14.4.1 Local Expertise

Facilities with significant expertise in balloon enteroscopy may make BE-ERC their first-line therapy for CBD stones in RYGB patients. Conversely facilities without enteroscopes (push or balloon assisted) may choose an alternative route of access. In such locations, LA-ERC is often the procedure of choice because the instrumentation needed for both the surgical portions and the endoscopic portions is standard.

14.4.2 Limb Length

The likelihood of successful endoscopic intervention decreases as the limb length increases. A review of the operative report to confirm the alimentary and biliopancreatic limb lengths is recommended to assess the likelihood of endoscopic success. Patients with longer alimentary limbs (>100–150 cm) may be preferential candidates for a surgical assisted ERC rather than a purely endoscopic means.

14.4.3 Urgency of the Procedure

The urgency of the procedure can be divided into two considerations: the need to decompress the bile duct and the need to clear the duct of stones. Many of the methods described above (percutaneous gastric access with delayed ERC for example) would be an inappropriate choice for the patient needing urgent CBD decompression/clearance as the tract generally requires a period for upsizing and maturation. Similarly, a patient with asymptomatic stones and a higher surgical risk would be an ideal candidate for this as G-tube formation, upsizing, and trans-gastric ERC can all be performed serially under sedation without the need for surgical intervention.

14.4.4 Need for Repeat Intervention

In patients with an anticipated need for multiple biliary interventions (e.g., multiple or large stones or a known bile duct stricture), a route that permits

Table 14.1 Overview of the five main methods of CBD clearance in RYGB and DS patients

Method	Pros	Cons	Pitfalls	Ideal patient
Double-balloon ERCP	• Non-operative	• Repeat intervention difficult • Technically challenging • High expertise required • Time consuming	• Long limb lengths • Internal hernia • DS patient	• Known limb length • Prior cholecystectomy • Local expertise • Repeat intervention not anticipated
Transgastric ERCP	• High success rate • Single operative procedure • Facilitates repeat intervention • Standard ERCP equipment	• Gastrotomy complications • Increased operative time • Surgical morbidity	• Difficult remnant access • Two-team coordination • DS patient	• Undergoing cholecystectomy • Anticipated repetitive CBD interventions • No anticipated issue with gastric access
Transcystic CBD exploration	• Single team • Single operative procedure • No visceral incisions	• Technically challenging • Prolonged operative times	• Large or impacted stones • Unfavorable cystic duct anatomy (i.e., sharp angulation, spirality)	• Undergoing cholecystectomy • Favorable cystic duct anatomy • Small stone(s)
Choledochotomy	• Definitive operative therapy	• T-tube complications • CBD stricture • High level of expertise	• CBD <4–6 mm • Laparoscopy challenging	• Failed endoscopic and/or transcystic exploration • Dilated duct • Good operative candidate
Percutaneous (*transhepatic*)	• Non-operative • Immediate duct decompression	• Staged dilation • Multiple procedures • Percutaneous drain	• Small ducts • Cirrhosis	• Prior cholecystectomy with failed ERCP • Failed ERCP and/or transcystic exploration • Poor operative candidate

easy reaccess to the bile ducts is preferable. A method that leaves an indwelling catheter (G-tube, J-tube, or PTC tube) at the conclusion of the intervention meets this requirement.

14.4.5 Need for Another Surgical Procedure

Patients who require a simultaneous surgical procedure, such as reduction of an internal hernia, should have the CBD cleared by one of the surgical means listed above. Some include the need for subsequent cholecystectomy in this group. Thus, for patients with CBD stones, the cholecystectomy and CBD clearance occur under a single anesthetic setting. This can be accomplished via a pure surgical method (laparoscopic/open cholecystectomy and laparoscopic/open CBD exploration) or via surgical assisted ERC (e.g., laparoscopic cholecystectomy and laparoscopic assisted transgastric ERC).

References

1. Digestive diseases in the United States: Epidemiology and Impact—NIH Publication No. 94–1447, US Government Printing Office, NIDDK, 1994.
2. Liddle RA, Goldstein RB, Saxton J. Gallstone formation during weight-reduction dieting. Arch Intern Med. 1989;149:1750–3.
3. Everhart JE. Contributions of obesity and weight loss to gallstone disease. Ann Intern Med. 1993;119:1029–35.

4. Papasavas PK, Gagné DJ, Ceppa FA, Caushaj PF. Routine gallbladder screening not necessary in patients undergoing laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2006;2(1):41–6.
5. Almazeedi S, Al-Sabah S, Alshammari D. Routine trans-abdominal ultrasonography before laparoscopic sleeve gastrectomy: the findings. Obes Surg. 2014;24(3):397–9.
6. Lichtenbaum RA, McMullen HF, Newman RM. Preoperative abdominal ultrasound may be misleading in risk stratification for presence of common bile duct abnormalities. Surg Endosc. 2000;14(3):254–7.
7. Einstein DM, Lapin SA, Ralls PW, Halls JM. The insensitivity of sonography in the detection of choledocholithiasis. AJR Am J Roentgenol. 1984;142(4):725–8.
8. Petrov MS, Savides TJ. Systematic review of endoscopic ultrasonography versus endoscopic retrograde cholangiopancreatography for suspected choledocholithiasis. Br J Surg. 2009;96(9):967–74.
9. Varghese JC, Liddell RP, Farrell MA, Murray FE, Osborne H, Lee MJ. The diagnostic accuracy of magnetic resonance cholangiopancreatography and ultrasound compared with direct cholangiography in the detection of choledocholithiasis. Clin Radiol. 2000;55(7):579.
10. Lahmann BE, Adrales G, Schwartz RW. Choledoc holithiasis—principles of diagnosis and management. Curr Surg. 2004;61(3):290–3.
11. Shamiyeh A, Lindner E, Danis J, Schwarzenlander K, Wayand W. Short-versus long-sequence MRI cholangiography for the preoperative imaging of the common bile duct in patients with cholecystolithiasis. Surg Endosc. 2005;19(8):1130–4.
12. Collins C, Maguire D, Ireland A, Fitzgerald E, O'Sullivan GC. A prospective study of common bile duct calculi in patients undergoing laparoscopic cholecystectomy: natural history of choledocholithiasis revisited. Ann Surg. 2004;239(1):28–33.
13. Obeid NR, Kurian MS, Ren-Fielding CJ, Fielding GA, Schwack BF. Safety of laparoscopic adjustable gastric banding with concurrent cholecystectomy for symptomatic cholelithiasis. Surg Endosc. 2014; 29(5):1192–7.
14. Desbeaux A, Hec F, Andrieux S, Fayard A, Bresson R, Pruvot MH, Mulliez E. Risk of biliary complications in bariatric surgery. J Visc Surg. 2010;147(4):e217–20.
15. Warschkow R, Tarantino I, Ukegjini K, Beutner U, Güller U, Schmied BM, Müller SA, Schultes B, Thurnheer M. Concomitant cholecystectomy during laparoscopic Roux-en-Y gastric bypass in obese patients is not justified: a meta-analysis. Obes Surg. 2013;23(3):397–407.
16. Caruana JA, McCabe MN, Smith AD, Camara DS, Mercer MA, Gillespie JA. Incidence of symptomatic gallstones after gastric bypass: is prophylactic treatment really necessary? Surg Obes Relat Dis. 2005;1(6):564–7.
17. Villegas L, Schneider B, Provost D, Chang C, Scott D, Sims T, Hill L, Hynan L, Jones D. Is routine cholecystectomy required during laparoscopic gastric bypass? Obes Surg. 2004;14(2):206–11.
18. Worni M, Guller U, Shah A, Gandhi M, Shah J, Rajgor D, Pietrobon R, Jacobs DO, Ostbye T. Cholecystectomy concomitant with laparoscopic gastric bypass: a trend analysis of the nationwide inpatient sample from 2001 to 2008. Obes Surg. 2012;22(2):220–9.
19. Lai EC, Mok FP, Tan ES, Lo CM, Fan ST, You KT, et al. Endoscopic biliary drainage for severe acute cholangitis. N Engl J Med. 1992;326(24):1582–6.
20. Leese T, Neoptolemos JP, Carr-Locke DL. Successes, failures, early complications and their management following endoscopic sphincterotomy: results in 394 consecutive patients from a single centre. Br J Surg. 1985;72(3):215–9.
21. Lopes TL, Baron TH. Endoscopic retrograde cholangiopancreatography in patients with Roux-en-Y anatomy. J Hepatobiliary Pancreat Sci. 2011;18(3):332–8.
22. Hamouda AH, Goh W, Mahmud S, Khan M, Nassar AH. Intraoperative cholangiography facilitates simple transcystic clearance of ductal stones in units without expertise for laparoscopic bile duct surgery. Surg Endosc. 2007;21(6):955–9.
23. Motson RW, Wetter LA. Operative choledochoscopy: common bile duct exploration is incomplete without it. Br J Surg. 1990;77(9):975–82.
24. DePaula AL, Hashiba K, Bafutto M. Laparoscopic management of choledocholithiasis. Surg Endosc. 1994;8(12):1399–403.
25. Martin IJ, Bailey IS, Rhodes M, O'Rourke N, Nathanson L, Fielding G. Towards T-tube free laparoscopic bile duct exploration: a methodologic evolution during 300 consecutive procedures. Ann Surg. 1998;228(1):29–34.
26. Lee HM, Min SK, Lee HK. Long-term results of laparoscopic common bile duct exploration by choledochotomy for choledocholithiasis: 15-year experience from a single center. Ann Surg Treat Res. 2014;86(1):1–6.
27. Escudero-Fabre A, Escallon Jr A, Sack J, Halpern NB, Aldrete JS. Choledochoduodenostomy. Analysis of 71 cases followed for 5 to 15 years. Ann Surg. 1991;213(6):635–42. discussion 643–4.
28. Swahn F, Nilsson M, Arnelo U, Löhr M, Persson G, Enochsson L. Rendezvous cannulation technique reduces post-ERCP pancreatitis: a prospective nationwide study of 12,718 ERCP procedures. Am J Gastroenterol. 2013;108(4):552–9.
29. Noel R, Enochsson L, Swahn F, Löhr M, Nilsson M, Permert J, Arnelo U. A 10-year study of rendezvous intraoperative endoscopic retrograde cholangiography during cholecystectomy and the risk of post-ERCP pancreatitis. Surg Endosc. 2013;27(7):2498–503.
30. Stokes KR, Falchuk KR, Clouse ME. Biliary duct stones: update on 54 cases after percutaneous transhepatic removal. Radiology. 1989;170(3 Pt 2):999–1001.
31. Szulman C, Giménez M, Sierre S. Antegrade papillary balloon dilation for extrahepatic bile duct stone clearance: lessons learned from treating 300 patients. J Vasc Interv Radiol. 2011;22(3):346–53.
32. Peters M, Papasavas PK, Caushaj PF, et al. Laparoscopic transgastric endoscopic retrograde cholangiopancreatography for benign common bile duct stricture after Roux-en-Y gastric bypass. Surg Endosc. 2002;16:1106.

33. Pimentel RR, Mehran A, Szomstein S, Rosenthal R. Laparoscopy-assisted transgastrostomy ERCP after bariatric surgery: case report of a novel approach. Gastrointest Endosc. 2004;59:325–8.
34. Ceppa FA, Gagné DJ, Papasavas PK, Caushaj PF. Laparoscopic transgastric endoscopy after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2007;3:21–4.
35. Nakao FS, Mendes CJ, Szego T, Ferrari AP. Intrao perative transgastric ERCP after a Roux-en-Y gastric bypass. Endoscopy. 2007;39 suppl 1:E219–20.
36. Patel JA, Patel NA, Shinde T, et al. Endoscopic retrograde cholangiopancreatography after laparoscopic Roux-en-Y gastric bypass: a case series and review of the literature. Am Surg. 2008;74:689–93. discussion 693–4.
37. Nguyen NT, Hinojosa MW, Slone J, et al. Laparoscopic transgastric access to the biliary tree after Roux-en-Y gastric bypass. Obes Surg. 2007;17:416–9.
38. Roberts KE, Panait L, Duffy AJ, et al. Laparoscopic-assisted transgastric endoscopy: current indications and future implications. JSLS. 2008;12:30–6.
39. Dapri G, Himpens J, Buset M, et al. Video. Laparoscopic transgastric access to the common bile duct after Roux-en-Y gastric bypass. Surg Endosc. 2009;23:1646–8.
40. Gutierrez JM, Lederer H, Krook JC, et al. Surgical gastrostomy for pancreatobiliary and duodenal access following Roux en Y gastric bypass. J Gastrointest Surg. 2009;13:2170–5.
41. Peeters G, Himpens J. A hybrid endo-laparoscopic therapy for common bile duct stenosis of a choledocho-duodenostomy after a Roux-en-Y gastric bypass. Obes Surg. 2009;19:806–8.
42. Sebastian JJ, Resa JJ, Penã E, et al. Laparoscopically assisted ERCP in a case of acute cholangitis in a patient with biliopancreatic diversion with distal gastric preservation. Obes Surg. 2009;19:250–2.
43. Badaoui A, Malherbe V, Rosiere A, De Ronde T. ERCP by laparoscopic transgastric access and cholecystectomy at the same time in a patient with gastric bypass who was seen with choledocholithiasis. Gastrointest Endosc. 2010;71:212–4.
44. Bertin PM, Singh K, Arregui ME. Laparoscopic transgastric endoscopic retrograde cholangiopancreatography (ERCP) after gastric bypass: case series and a description of technique. Surg Endosc. 2011; 25:2592–6.
45. Richardson JF, Lee JG, Smith BR, et al. Laparoscopic transgastric endoscopy after Roux-en-Y gastric bypass: case series and review of the literature. Am Surg. 2012;78(10):1182–6.
46. Juza RM, Haluck RS, Rogers AM, et al. Antegrade wire, rendezvous cannulation of the biliary tree may reduce the incidence of post-ERCP pancreatitis. Surg Endosc. 2014;28:S336.
47. Lopes TL, Clements RH, Wilcox CM. Laparoscopy-assisted ERCP: experience of a high-volume bariatric surgery center (with video). Gastrointest Endosc. 2009;70:1254–9.
48. Lopes TL, Clements RH, Wilcox CM. Laparoscopy-assisted transjejunal ERCP in a patient with Roux-en-Y reconstruction following partial gastrectomy. J Laparoendosc Adv Surg Tech A. 2010;20:55–8.
49. Hintze RE, Adler A, Veltzke W, Abou-Rebyeh H. Endoscopic access to the papilla of Vater for endoscopic retrograde cholangiopancreatography in patients with billroth II or Roux-en-Y gastrojejunostomy. Endoscopy. 1997;29:69–73.
50. Elton E, Hanson BL, Qaseem T, Howell DA. Diagnostic and therapeutic ERCP using an enteroscope and a pediatric colonoscope in long-limb surgical bypass patients. Gastrointest Endosc. 1998;47:62–7.
51. Wright BE, Cass OW, Freeman ML. ERCP in patients with long-limb Roux-en-Y gastrojejunostomy and intact papilla. Gastrointest Endosc. 2002;56:225–32.
52. Aabakken L, Bretthauer M, Line PD. Double-balloon enteroscopy for endoscopic retrograde cholangiography in patients with a Roux-en-Y anastomosis. Endoscopy. 2007;39(12):1068–71.
53. Raithel M, Dormann H, Naegel A, et al. Double-balloon-enteroscopy-based endoscopic retrograde cholangiopancreatography in post-surgical patients. World J Gastroenterol. 2011;17(18):2302–14.
54. Emmett DS, Mallat DB. Double-balloon ERCP in patients who have undergone Roux-en-Y surgery: a case series. Gastrointest Endosc. 2007;66(5):1038–41.
55. Parlak E, Cicek B, Disibeyaz S, et al. Endoscopic retrograde cholangiography by double balloon enteroscopy in patients with Roux-en-Y hepaticojejunostomy. Surg Endosc. 2010;24(2):466–70.
56. Neumann H, Fry LC, Meyer F, et al. Endoscopic retrograde cholangiopancreatography using the single balloon enteroscope technique in patients with Roux-enanastomosis. Digestion. 2009;80(1):52–7.
57. Wang AY, Sauer BG, Behm BW, et al. Single-balloon enteroscopy effectively enables diagnostic and therapeutic retrograde cholangiography in patients with surgically altered anatomy. Gastrointest Endosc. 2010;71(3):641–9.
58. Itoi T, Ishii K, Sofuni A, et al. Single-balloon enteroscopy-assisted ERCP in patients with Billroth II gastrectomy or Roux-en-Y anastomosis (with video). Am J Gastroenterol. 2010;105(1):93–9.
59. Saleem A, Baron TH, Gostout CJ, et al. Endoscopic retrograde cholangiopancreatography using a single-balloon enteroscope in patients with altered Roux-en-Y anatomy. Endoscopy. 2010;42(8): 656–60.
60. Shah RJ, Smolkin M, Ross AS, et al. A multi-center, U.S. experience of single balloon, double balloon, and rotational overtube enteroscopy-assisted ERCP in long limb surgical bypass patients. Gastrointest Endosc. 2010;71:AB134.
61. Lennon AM, Kapoor S, Khashab M, et al. Spiral assisted ERCP is equivalent to single balloon assisted ERCP in patients with Roux-en-Y anatomy. Dig Dis Sci. 2012;57(5):1391–8.

62. Wagh MS, Draganov PV. Prospective evaluation of spiral overtube-assisted ERCP in patients with surgically altered anatomy. Gastrointest Endosc. 2012;76(2):439–43.
63. Baron TH, Song LM. Percutaneous assisted transprosthetic endoscopic therapy (PATENT): expanding gut access to infinity and beyond. Gastrointest Endosc. 2012;76(3):641–4.
64. Weilert F, Binmoeller KF, Marson F, et al. Endoscopic ultrasound-guided anterograde treatment of biliary stones following gastric bypass. Endoscopy. 2011;43:1105–8.

Management of Abdominal Wall Hernias in the Bariatric Patient

15

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Abbreviations

BMI	Body mass index
BPD/D	Biliopancreatic diversion with duodenal switch
ePTFE	Expanded polytetrafluoroethylene
LAGB	Laparoscopic adjustable gastric band
RYGB	Roux-en-Y gastric bypass
VSG	Vertical sleeve gastrectomy

Key Points

- Should not we change the (derogatory) term "morbid obesity" to "medically complicated obesity," which will be more acceptable to patients? Would you want to be called "morbid?"
- Bariatric surgery should at least be considered and offered to appropriately obese patients who meet criteria for a bariatric surgical intervention who have an abdominal wall hernia.
- A laparoscopic VSG, RYGB, BPD/DS, and, maybe in highly selected patients, an LAGB prior to repair of a large or complex hernia in patients with medically complicated obesity can yield substantial weight loss resulting in a potentially technically easier successful hernia repair.
- The question whether the hernia should be repaired before, during, or after bariatric surgery depends on multiple variables—size of defect, need for a true abdominal wall reconstruction, symptoms, etc.
- Many abdominal wall hernia repairs can be combined with an abdominoplasty after weight loss induced first by bariatric surgery.

Obesity and abdominal wall hernias go hand in hand with one another; obesity predisposes to the development of incisional hernias after a prior abdominal operation as well as predisposing to umbilical hernias. Therefore, the problem of abdominal wall hernia is a very relevant discussion in patients being considered for bariatric surgery [1]. Equally important in this discussion is the patient presenting for treatment of an abdominal wall hernia who fulfills the criteria for medically complicated class 2 obesity (BMI>35 kg/m^2), but who has never been approached about considering bariatric surgery and who may not have even considered bariatric surgery as an important intervention not only to optimize the outcome (and safety) of repair of their hernia but also in terms of their overall health. The population of patients with abdominal wall hernias in the setting of obesity has been largely ignored by many of

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_15

our health care providers and even by all-too-many general surgeons. This chapter addresses this topic of abdominal wall hernias in the patient with medically complicated obesity (please, let's stop using the terms "morbid" obesity and use a more deferential term that is better accepted and understood by patients), both from the standpoint of a bariatric surgeon and from the standpoint of a general surgeon performing repairs of abdominal wall hernias. Remember, we as "surgeons" should believe that we are really "physicians who can also operate," and we should first address the overall health of the patient and only then address the surgical problems. Therefore, this chapter addresses the following topics:

15.1 Should We Actively Suggest and Offer Bariatric Surgery to the Patient with an Abdominal Wall Hernia Who Is Markedly Overweight?

In the patient planning to undergo bariatric surgery who also has an abdominal wall hernia (small or large hernia), should the hernia be repaired before, during, or after the bariatric surgery?

15.2 Abdominal Wall Hernia in a Patient with Unaddressed Medically Complicated Obesity

Obesity and especially severe obesity predispose to the formation of incisional hernias after any form of celiotomy, approaching an incidence of 10–12 %. With our frightening national increase in the prevalence of obesity, the occurrence of incisional hernias is increasing, and the association of obesity in patients presenting to a primary care provider or surgeon for repair of an abdominal wall hernia is likewise all too common. With this common association currently, we should ask the following: Does obesity predispose to a greater risk of recurrence of the hernia after repair? The answer, of course, is "yes," but we should also acknowledge that the risk increases with the severity of obesity, and the risk is extremely high in those patients with severe obesity [2, 3]. These data need to be considered actively when considering an elective repair of an incisional hernia (or even a large umbilical hernia) in the patient with concomitant, medically complicated obesity.

In addition to the elective repair of an abdominal wall hernia in an obese patient, the physician (as well as surgeon) should consider the entire well-being of the patient. If we see a patient with a straightforward incisional hernia who has a body mass index (BMI) ≥35 *but* who also has some combination of sleep apnea, hypertension, diabetes mellitus, dyslipidemia, and degenerative joint disease, shouldn't we and their primary care provider also entertain bariatric surgery in itself, even independent of their abdominal wall hernia? Our approach should be to address the overall health of the patient and not just the hernia. Indeed, performing a bariatric procedure first may convert an otherwise non-repairable hernia or one with inordinate operative risk and risk of recurrence to a much more manageable abdominal wall reconstruction—and the patient will benefit both medically and in their quality of life by the weight loss induced by the bariatric procedure. This concept and other technical considerations in the management of abdominal wall hernias are addressed below.

15.3 Abdominal Wall Hernias in Patients to Undergo Bariatric Surgery

The presence of an abdominal wall hernia can complicate markedly not only the technical aspects of the planned bariatric operation but also potentially which bariatric procedure is best. Considerations for the bariatric surgeon are the following: (1) Should the bariatric surgery be performed *before*, *during*, or *after* repair of the abdominal wall hernia? (2) Which bariatric procedure is best—Roux-en-Y gastric bypass (RYGB), vertical sleeve gastrectomy (VSG), laparoscopic adjustable gastric band (LAGB), or biliopancreatic diversion with duodenal switch (BPD/DS)? We will address each question separately.

15.3.1 Bariatric Surgery Before, During, or After Hernia Repair

There are pros and cons regarding the timing of the bariatric procedure which affect the risk of hernia recurrence, complications of the bariatric operation, and risk of complications related to simultaneous or staged hernia repair. Other considerations include the need for potential aesthetic surgery, such as abdominoplasty.

15.3.1.1 Simultaneous Bariatric Surgery and Hernia Repair

Ideally, repair of the abdominal wall hernia at the time of bariatric surgery would seem most appropriate, but other considerations are noteworthy (Table 15.1). Simultaneous bariatric and hernia procedures would require only one anesthetic, one penetration of the coelomic cavity, and only one convalescence (absence from work, usual lifestyle, etc.). Because bariatric surgery requires a celiotomy, albeit via minimal access, the possibility for a sublay, prosthetic-based repair is very attractive; indeed, no one would realistically condone a non-prosthetic-based herniorrhaphy in the patient with medically complicated obesity. Overall cost would also be markedly less with one operation and one hospitalization. In addition, by repairing the hernia simultaneously, the possibility of the hernia incarcerating, strangulating, or causing a small bowel obstruction prior to definitive repair would be prevented (see below, bariatric procedure first).

Table 15.1 Simultaneous bariatric surgery and abdominal wall herniorrhaphy

Pros	Cons
One anesthetic, one convalescence	Risk of infection of prosthetic-based herniorrhaphy
Less overall cost	Possible need for open hernia repair
Less absence from work	Need for concomitant full abdominal wall reconstruction[a]
Possible dual-laparoscopic procedures	More difficult hernia repair
Prevents complications of hernia (obstruction, pain) while awaiting hernia repair	Greater risk of hernia recurrence
	Fewer options for type of hernia repair
	Need for future abdominoplasty

[a]Requiring tissue transfers, rotational flaps, etc.

In contrast, there are several potentially compelling reasons to avoid a simultaneous procedure. First, all bariatric procedures except an LAGB open the gut with the potential risk of intraperitoneal/wound bacterial contamination, thereby risking infection of the prosthesis used to repair the hernia. This consideration may be especially pertinent if the herniorrhaphy requires an open approach and/or a true abdominal wall reconstruction necessitating tissue transfer (component separation, skin/subcutaneous advancement flaps, wide sublay on onlay repairs with the prosthetic "patching" of the defect in the subcutaneous space, as opposed to being covered by musculofascial autogenous tissues, etc.). In addition, many surgeons maintain that repair of the abdominal wall hernia, especially if large, is much easier in patients after substantial weight loss and that the risk of recurrence is less. Also, the type of hernia repair possible may be limited in the morbidly obese for both technical reasons or because of concerns about wound complications or prosthetic-based infections. If a future abdominoplasty is probable, a second operation under general anesthesia will be necessary, and the hernia repair may very well have been easier at the time of abdominoplasty. Finally, in some cases, with the gain of intra-abdominal domain after substantial weight loss, the use of mesh may potentially be avoided altogether for smaller hernias due to the development of extreme laxity of the abdominal wall.

15.3.1.2 Staged Repair—Bariatric Surgery First, Hernia Repair Later

Many surgeons argue that a staged approach is best for multiple reasons (Table 15.2). First, the morbidity and possibly mortality of any abdominal operation are increased, especially for formal abdominal wall reconstructions that require more than just a laparoscopic sublay herniorrhaphy in patients with medically complicated obesity. Second, the hernia operation after weight loss is technically easier, more options are available

Table 15.2 Bariatric surgery FIRST, abdominal wall herniorrhaphy SECOND

Pros	Cons
Healthier patient at the time of herniorrhaphy[a]	Hernia-related pain/ symptoms persist
Easier herniorrhaphy[a]	Hernia can enlarge, incarcerate, strangulate, or cause small bowel obstruction[b]
More options for herniorrhaphy	Hernia may interfere with a bariatric procedure
More skin to use for herniorrhaphy[a]	More adhesions at the time of herniorrhaphy
Can do simultaneous abdominoplasty	
Less chance for hernia recurrence[a]	

[a]Weight much less
[b]While awaiting weight loss, especially if bariatric procedure required mobilization of bowel from within the hernia

(more skin to advance, less poorly vascularized subcutaneous fat, possibility of avoiding the use of mesh), and often the herniorrhaphy and an abdominoplasty (for redundant lower abdominal skin and subcutaneous tissue after the bariatric-induced weight loss) can be performed simultaneously. Finally, the risk of hernia recurrence is less after substantial weight loss.

But others will acknowledge arguments against bariatric surgery prior to hernia repair (Table 15.2). Most importantly, the strongest arguments include the following question—How should we manage the patient who is very symptomatic from their hernia? We know that a certain percentage of patients with an abdominal wall hernia, especially incisional hernias, develop complications such as incarceration, strangulation, or enlargement of the hernia; moreover, these hernias themselves can cause a small bowel obstruction independent of incarceration/strangulation. There is also concern that the reduction of hernia contents (omentum/bowel) that is often necessary when performing RYGB or BPD/DS without definitive repair may predispose to future hernia complications such as incarceration with possible obstruction or strangulation. Other considerations include the possibility of the bariatric operation increasing the difficulty of the hernia operation or that a very large hernia may necessitate an open bariatric operation or may prevent the possibility of a laparoscopic approach. Finally, repairing the hernia first may allow a later laparoscopic, minimally invasive bariatric operation.

15.3.1.3 What Are the Data?

Does Obesity Predispose to Recurrence of Abdominal Wall Herniorrhaphy?

The answer is "yes." Although we all as surgeons "know" this by experience, there are studies which document this increase in recurrence. Possibly the most visible study is that of the large prospective study of laparoscopic incisional herniorrhaphy by Heniford and colleagues [2]. Unfortunately, there are no other good studies addressing the incidence of recurrent hernia after a primary incisional hernia repair in patients with BMI $\geq$ 35. The remainder of this chapter addresses the bariatric patient with an abdominal wall hernia.

Incisional hernias develop in ~20–25 % of patients undergoing open bariatric surgery. Thus, obesity predisposes to hernia formation after any celiotomy, and many patients we see (or should see) with medically complicated obesity have an abdominal wall hernia and/or have a recurrent abdominal wall hernia. Therefore, the association of medically complicated obesity and abdominal wall hernia is not at all uncommon, and especially so for umbilical hernias.

Considerations When Approaching the Bariatric Patient Who Has an Abdominal Wall Hernia (Table 15.3)

First, can the operation be performed laparoscopically or maybe can a specific bariatric procedure, such as LAGB or laparoscopic VSG, be possible even if a laparoscopic RYGB or BPD/DS is not possible? Many abdominal wall hernias involve the periumbilical and lower abdominal region rather than the uppermost abdomen (e.g., after gynecologic procedures or colonic problems like distal colectomies); therefore, access to the supracolic space laparoscopically may be possible even if safe access to the infracolic space

Table 15.3 Repair of abdominal wall hernia at the time of bariatric surgery

Can the bariatric procedure be accomplished laparoscopically? • Access • Gastric sleeve, band, RYGB, BPD/DS • Need to reduce incarcerated bowel or sizeable length of bowel
Size of hernia defect • Less than versus greater than 4 cm[a]
Reason for hernia repair • Intermittent small bowel obstruction • Pain
Type of repair needed • Patch type repair (sublay, onlay, or prosthetic reinforcement) • Laborer/high-performance athlete/? Younger patient requiring abdominal wall reconstruction, not "patch-type repair"
Will an open bariatric operation be required?

[a]Risk of direct hernia-related serious complications

(jejunoileum) is not possible or technically feasible. Similarly, when non-obstructed small bowel fills the hernia, avoiding reduction of the non-obstructed bowel may be safer than reducing the bowel to perform an RYGB or BPD/DS. Indeed, two studies by Eid et al. [4] and Datta and colleagues [5] showed that construction of an RYGB requiring reduction of non-obstructed bowel from the hernia defect was associated with hernia-related complications within the following 6 months postoperatively while awaiting the appropriate weight loss before repairing the hernia in a planned stage approach.

Second, how big (or rather how "small") is the hernia defect? Many surgeons believe that the smaller the defect, the greater the risk of incarceration/strangulation/bowel obstruction. While there is no objective size, 4 cm seems a reasonable cutoff and may affect how the surgeon deals with a small hernia. Indeed, the indications for hernia repair may also affect how the surgeon should approach the patient. The patient with a small, incarcerated, painful hernia or a history of recurrent, intermittent small bowel obstruction related apparently to the hernia often requires repair of the hernia first or simultaneously at the time of the bariatric procedure.

Next, which type of hernia repair is indicated? If the patient is a candidate for a laparoscopic sublay repair ("patching" the defect) without planning an autogenous reapproximation of the fascial defect, this can often be done at the time of bariatric surgery. The concern, of course, is that when the celomic cavity is exposed to the enteric lumen (RYGB, BPD/DS, and even VSG), the risk of prosthetic infection increases, and at least some consideration may be given to LAGB or VSG with the thought that LAGB is a clean case, and contamination with VSG is minimal.

A number of studies have addressed the topic of simultaneous, prosthetic-based repairs at the time of bariatric surgery. In the era of open bariatric surgery, concomitant placement of an alloplastic prosthesis was often problematic. Herbert et al. reported that 3 of 16 patients (19 %) undergoing open RYGB developed a mesh-based infection [6]. Similarly, the authors have had several mesh infections after simultaneous bariatric and alloplastic-based hernia repairs. In contrast, Schuster and colleagues found no mesh infections in their 11 patients [7].

For laparoscopic bariatric procedures, results of simultaneous bariatric procedures combined with prosthetic-based herniorrhaphy procedures have been more encouraging. Eid and colleagues reported no mesh infections in 12 patients undergoing RYGB and laparoscopic herniorrhaphy, but all hernias in their series were small defects [4]. Bonatti et al. combined LAGB with laparoscopic herniorrhaphy successfully [8]. Probably, many such simultaneous repairs go unreported.

For patients with very large hernias that will require a formal abdominal wall reconstruction using tissue transfers, a more conservative approach seems more appropriate. With these patients, combining a bariatric procedure, other than LAGB or possibly VSG, with a complex abdominal wall reconstruction utilizing an alloplastic prosthesis seems potentially risky; an infected prosthesis may/usually requires its removal and would leave a very difficult hernia to repair in the future. Despite this warning, Chang et al. [9] have reported six patients in whom an anterior component separation (without any prosthetic reinforcement) was used successfully to

repair the hernia defect by allowing a primary, "tension-free" autogenous fascial reapproximation combined with a bariatric procedure with no recurrent hernias at a mean follow-up of 3.5 years. Their lack of any prosthetic-based reinforcement of the fascial closure in a patient with medically complicated obesity who already has formed an abdominal wall hernia and who is undergoing a weight loss-inducing procedure (itself associated with a high risk of hernia formation), would have to be considered less than ideal by most herniologists.

One recent report by Carbonell and colleagues [10] deserves some comment in this regard. Classically, surgeons have shied away from placing permanent, alloplastic prostheses to repair hernias in type 3 (contaminated) and usually even in type 2 (clean-contaminated) procedures (like the bariatric procedures of RYGB, BPD/DS, and even VSG). Carbonell and colleagues reviewed their experience in 100 patients with types 2 and 3 wounds using the more recent large-pore, light- to medium-weight meshed prosthetics with surprisingly good outcomes at 1 year in terms of serious mesh infections (none required explantation as opposed to the older small pore/heavy weight meshed prostheses or the expanded polytetrafluoroethylene (ePTFE)-based prostheses). Similarly, Israelsson and colleagues have placed large-pore/lightweight prostheses at the time of creation of an enterostoma (ileostomy, urostomy, colostomy) to "prevent" parastomal hernias; their success has been impressive, and no mesh infections have occurred in 75 patients [11]. These two reports may serve to expand the indications for simultaneous bariatric and abdominal wall herniorrhaphies using the newer large-pore alloplastic meshes. Moreover, many surgeons (including us) do believe that these newer meshed prostheses will granulate through when involved in a mesh infection as opposed to the older, small-pore, heavyweight polypropylene prosthetics, although we know of no good reported data to support our anecdotal observations!

Finally, some discussion of the use of bioprostheses and synthetic absorbable prostheses is appropriate. Fear of mesh infection when using permanent, alloplastic prostheses has generated considerable interest and controversy. The concept of "repairing" an abdominal wall hernia with one of these nonpermanent prostheses as a laparoscopic "patch," while attractive in theory, does not seem to the authors or to most herniologists as a viable option for *definitive* repair, especially for the larger defects. In contrast, their use when placed as a patch to span the defect as a *temporizing* measure to obliterate the defect while weight loss from the bariatric procedure takes place may prove a reasonable alternative, particularly if the bariatric procedure must be done as an open operation. Indeed, some of the newer bioprosthetics maintain effective integrity for 6–9 months (and thus effectively patch the hernia defect for the postoperative duration of maximal weight loss), and are an attractive albeit expensive option for a subset of these difficult situations (e.g., big hernia in superobese patients).

Staged Repair: Bariatric Procedure First/ Abdominal Wall Herniorrhaphy Later

The arguments for a staged approach are that the hernia repair is easier, the patient is healthier, the recurrence rate of the hernia is less, the wound-related morbidity of an abdominal wall reconstruction is less, and the hernia repair can be combined with an abdominoplasty after the weight loss that occurs with the bariatric procedure. Newcombe et al. used such a staged approach in 27 patients with very large hernias (mean: 200 cm^2) [12]. Outcomes were excellent with no hernia recurrences at 2-year follow-up. The experience of the senior author (MGS) is similar in 17 patients; at ≥5-year follow-up, there have been two recurrences, one related to a mesh-based infection.

There is also quite a robust experience with combined body contouring (abdominoplasty) and both abdominal wall herniorrhaphy and abdominal wall reconstruction, albeit most often in patients after prior open bariatric surgery who develop an incisional hernia. Although many of the details of the characteristics, size of the hernia, and prosthetic materials used are lacking and follow-up is usually too short, the results are very encouraging [3, 12–15]. Most hernia defects appear to have been small (<50 cm^2), but several authors have described true abdominal wall reconstructions using alloplastic prostheses combined with an abdominoplasty with good results.

15.4 Summary

The combination of need for bariatric surgery and repair of an abdominal wall hernia is not uncommon. Small hernias that do not preclude a laparoscopic bariatric approach are probably best managed either by a concomitant laparoscopic hernia (patch) repair using a large-pore/light- to middle-weight protected meshed prosthesis or a longer persisting biologic or synthetic prosthesis that maintains integrity for ≥6 months. For the larger hernias containing omentum or bowel, probably a better approach would be to perform a laparoscopic VSG and not an RYGB or BPD/DS, so as not to have to reduce to hernia contents, allow an appropriate weight loss to occur, and then perform a definitive abdominal herniorrhaphy or abdominal wall reconstruction later after substantial weight loss. For the unusual situation where an open bariatric procedure is necessary and the access must be through the hernia defect, the best approach (at least to these authors) is to perform the best bariatric procedure and to obliterate the hernia defect either by a primary autogenous fascial closure reinforced either with a large-pore/light- to medium-weight prosthesis placed as an onlay or probably more safely to patch the defect with a sublay of a longer persisting bioprosthesis or with a synthetic absorbable prosthesis that persists longer than the typical more rapidly absorbed polyglactin prosthetics.

Acknowledgement The authors are greatly appreciative of the administrative support provided by Deborah I. Frank in the preparation and submission of this chapter.

References

1. Sarr MG. Abdominal wall reconstruction in the morbidly obese patient. In: Nahabedian M, Bhanot P, editors. Abdominal wall reconstruction. Woodbury, CT: Cine-Med; 2014. p. 201–6.
2. Heniford BT, Park A, Ramshaw BJ, Voeller G. Laparoscopic repair of ventral hernias: nine years' experience with 850 consecutive hernias. Ann Surg. 2003;238(3):391–9. discussion 9–400.
3. Raftopoulos I, Courcoulas AP. Outcome of laparoscopic ventral hernia repair in morbidly obese patients with a body mass index exceeding 35 kg/m^2. Surg Endosc. 2007;21(12):2293–7.
4. Eid GM, Mattar SG, Hamad G, Cottam DR, Lord JL, Watson A, et al. Repair of ventral hernias in morbidly obese patients undergoing laparoscopic gastric bypass should not be deferred. Surg Endosc. 2004;18(2):207–10.
5. Datta T, Eid G, Nahmias N, Dallal RM. Management of ventral hernias during laparoscopic gastric bypass. Surg Obes Relat Dis. 2008;4(6):754–7.
6. Herbert GS, Tausch TJ, Carter PL. Prophylactic mesh to prevent incisional hernia: a note of caution. Am J Surg. 2009;197(5):595–8. discussion 8.
7. Schuster R, Curet MJ, Alami RS, Morton JM, Wren SM, Safadi BY. Concurrent gastric bypass and repair of anterior abdominal wall hernias. Obes Surg. 2006;16(9):1205–8.
8. Bonatti H, Hoeller E, Kirchmayr W, Muhlmann G, Zitt M, Aigner F, et al. Ventral hernia repair in bariatric surgery. Obes Surg. 2004;14(5):655–8.
9. Chang EI, Foster RD, Hansen SL, Jazayeri L, Patti MG. Autologous tissue reconstruction of ventral hernias in morbidly obese patients. Arch Surg. 2007;142(8):746–9. discussion 9–51.
10. Carbonell AM, Criss CN, Cobb WS, Novitsky YW, Rosen MJ. Outcomes of synthetic mesh in contaminated ventral hernia repairs. J Am Coll Surg. 2013;217(6):991–8.
11. Janes A, Cengiz Y, Israelsson LA. Experiences with a prophylactic mesh in 93 consecutive ostomies. World J Surg. 2010;34(7):1637–40.
12. Newcomb WL, Polhill JL, Chen AY, Kuwada TS, Gersin KS, Getz SB, et al. Staged hernia repair preceded by gastric bypass for the treatment of morbidly obese patients with complex ventral hernias. Hernia. 2008;12(5):465–9.
13. Iljin A, Szymanski D, Kruk-Jeromin J, Strzelczyk J. The repair of incisional hernia following Roux-en-Y gastric bypass-with or without concomitant abdominoplasty? Obes Surg. 2008;18(11):1387–91.
14. Downey SE, Morales C, Kelso RL, Anthone G. Review of technique for combined closed incisional hernia repair and panniculectomy status post-open bariatric surgery. Surg Obes Relat Dis. 2005;1(5):458–61.
15. Saxe A, Schwartz S, Gallardo L, Yassa E, Alghanem A. Simultaneous panniculectomy and ventral hernia repair following weight reduction after gastric bypass surgery: is it safe? Obes Surg. 2008;18(2):192–5. discussion 6.

Band Prolapse: Diagnosis and Management

16

Abraham Krikhely, Elana Gluzman, and Danny A. Sherwinter

16.1 Diagnosis

Obesity continues to have a high prevalence in the USA and worldwide. The CDC estimated that the prevalence of obesity in the USA in 2011–2012 in adults was 34.9 % and in youth was 16.9 %. Of the adult population 6.4 % had a BMI > 40. These rates have not changed significantly since 2003–2004 [1]. Bariatric surgery remains an important tool in the battle against obesity. Numerous studies have shown that bariatric surgery is more effective than intensive medical therapy at weight loss and at reversal of medical comorbidities such as diabetes [2–5].

Although currently waning in popularity, the laparoscopic adjustable gastric band was one of the most popular bariatric surgeries performed over the past two decades. The first laparoscopic placement of an adjustable silicone gastric band was reported by Belachew et al. in 1993 [6]. The Lap-Band® (Apollo Endosurgery, Austin, TX) was approved for use in the USA in 2001 and the Realize® band (Ethicon, Cincinnati, OH) in 2008 [7]. Gastric banding was found to provide effective weight loss and reduction of medical comorbidities and its popularity grew. It was particularly attractive to patients who were averse to getting operations involving gastric stapling and would otherwise not have considered bariatric surgery. Though the gastric band can have complications, these are usually not life threatening. One of the more common late complications is the gastric band prolapse, also known as a slip, which is the focus of this chapter.

Band prolapse refers to the herniation of any portion of the gastric wall through the band in a proximal direction, with caudal migration of the band. Because the cross-sectional area of the stomach at the gastric body is greater than at the angle of His, complete outflow obstruction can result. The prolapse can be an anterior prolapse of the anterolateral fundus or a posterior prolapse of the posterior fundus. This differs from gastric pouch/esophageal dilatation (GED) in which there is concentric enlargement of the gastric pouch and/or esophagus resulting from stretching of the gastric wall. GED is readily treated with band decompression and rarely results in obstruction and does not require surgical intervention [8]. Both entities though are thought to arise from poor food processing, patient overeating, and/or surgeon overfilling of the band [9].

Band prolapse has been noted since the early days of the band era and several modifications in the technique were introduced to reduce this tendency. Initially, the band had been placed using the perigastric approach, which was associated

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_16

with a high rate of prolapse, especially of the posterior type [9]. In this technique, a tunnel was created 3 cm below the gastroesophageal junction from the lesser curve, around the stomach, and through the lesser sac. With the advent of the pars flaccida technique, the rate of prolapse decreased substantially [10–12]. With the pars flaccida technique, the pars flaccida is incised as is the peritoneum along the medial edge of the right crus and a tunnel is gently bluntly created through the retroperitoneal attachments of the proximal stomach. In a prospective randomized controlled trial, Dixon and O'Brien described a 16 % slip rate with the perigastric approach compared to a 4 % slip rate with the pars flaccida approach [13]. This was mainly a function of eliminating the posterior prolapse. Though substantially less common with the pars flaccida technique, posterior prolapse has since been reported [14]. The pars flaccida technique is now the approach generally adopted worldwide.

Another key aspect of surgical technique which can affect slip rates is the gastro-gastric plication: the anterior fundus and cardia under the band are plicated to the gastric pouch above the band, starting as far posterolaterally as possible to minimize the chances of anterior prolapse [12, 14]. Different surgeons describe varied techniques for this plication with some advocating no plication without an increase in prolapse rates [15].

Gastric band prolapse can typically present with any combination of food intolerance, nausea, vomiting, dysphagia, or worsening GERD. The patient may exhibit signs of volume depletion. Tachycardia and mild hypotension should resolve with resuscitation, as should a leukocytosis, a mildly elevated lactate, and abnormal BUN/Cr. Peritonitis, persistent abdominal pain, or lab abnormalities that are markedly abnormal or refractory to resuscitation should raise suspicion for gastric ischemia, volvulus, or perforation, which are rare but reported complications of a prolapse [16]. Marescaux postulates that a herniated gastric pouch can have decreased blood flow as a result of both the strangulating pressure from the band and the increased transmural pressure from the excessively dilated pouch. After the band has been deflated and resuscitation has begun, imaging may be obtained.

The mainstay of diagnosis is radiography. On plain films and upper GI contrast swallow, a normally positioned band appears as a rectangle and lies in an 8 o'clock to 2 o'clock orientation (Fig. 16.1). The φ (phi) angle is also measured to determine whether the band is correctly positioned. This is the angle formed on frontal radiographs between the vertical axis of the spine and the bottom of the gastric band. In a correctly positioned band, the φ angle is less than 58° (Fig. 16.2a) [17]. In a gastric band prolapse, the eccentrically dilated pouch presses the band caudally, creating an orientation that is closer to a 10 o'clock to 4 o'clock orientation or a φ angle that is greater than 58°. These are findings most commonly associated with an anterior prolapse, in which the band rotates to a horizontal position (Fig. 16.2b). The "O sign" is another radiographic finding that has been described, more commonly associated with a posterior prolapse in which the band rotates vertically; here the band is seen in a forward-facing position and has an O-shaped appearance (Fig. 16.3a) [18]. Oral contrast may demonstrate a pouch that is eccentri-

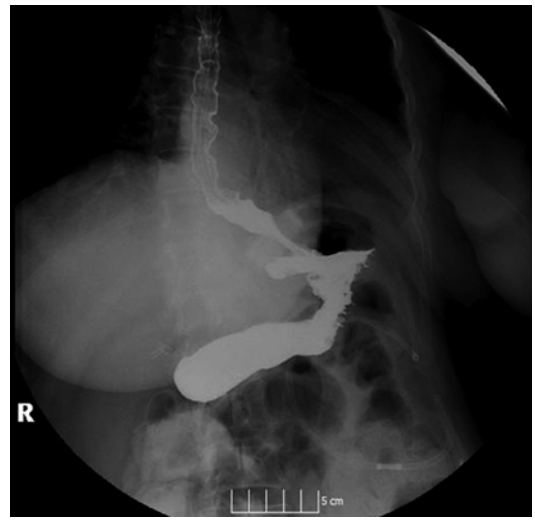


Fig. 16.1 A normally positioned gastric band with an 8 o'clock to 2 o'clock orientation and passage of oral contrast

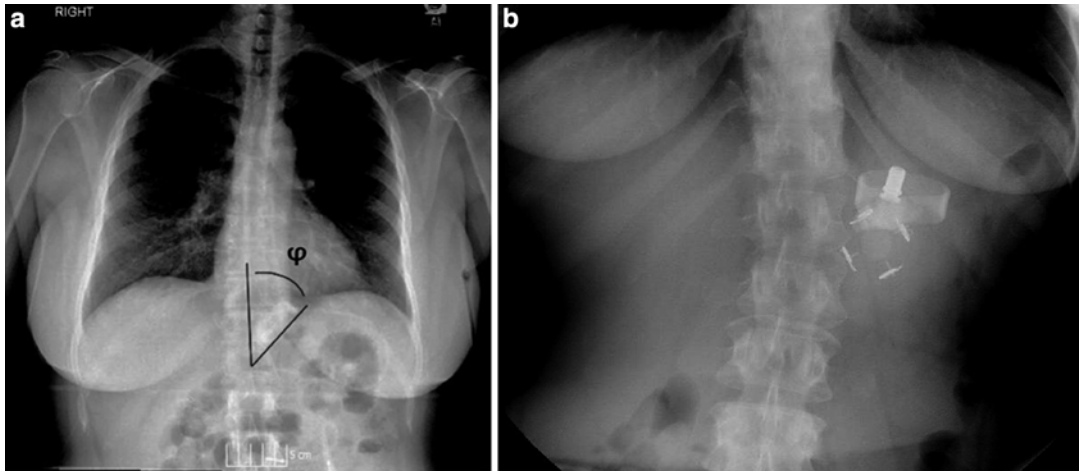


Fig. 16.2 (**a**) φ (phi) angle is measured to determine whether the band is correctly positioned. This is the angle formed on frontal radiographs between the vertical axis of the spine and the bottom of the gastric band. (**b**) Prolapse exhibiting a large φ (phi) angle and 10 o'clock to 4 o'clock orientation

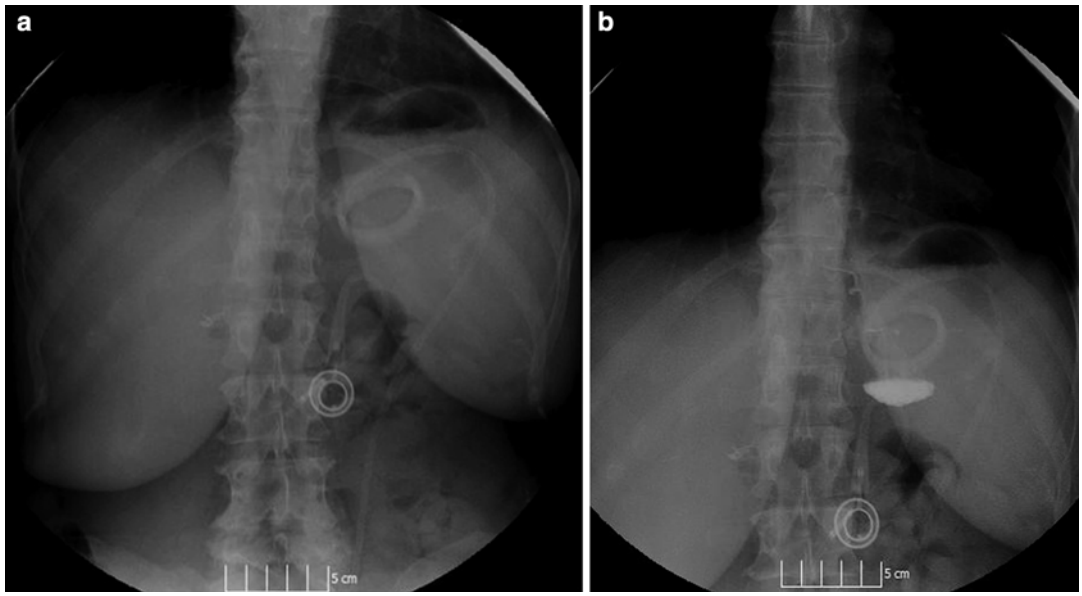


Fig. 16.3 (**a**) "O" sign suggestive of a posterior gastric band prolapse. (**b**) "O" sign with pooled contrast and air bubble

cally dilated, contains an air fluid level, and has abnormal emptying (Fig. 16.3b). A CT scan generally should not be necessary but can be useful to rule out other pathology if clinical signs and symptoms are more complex (Fig. 16.4). Endoscopy can also be used to diagnose gastric band prolapse, but is more invasive than radiologic modalities.

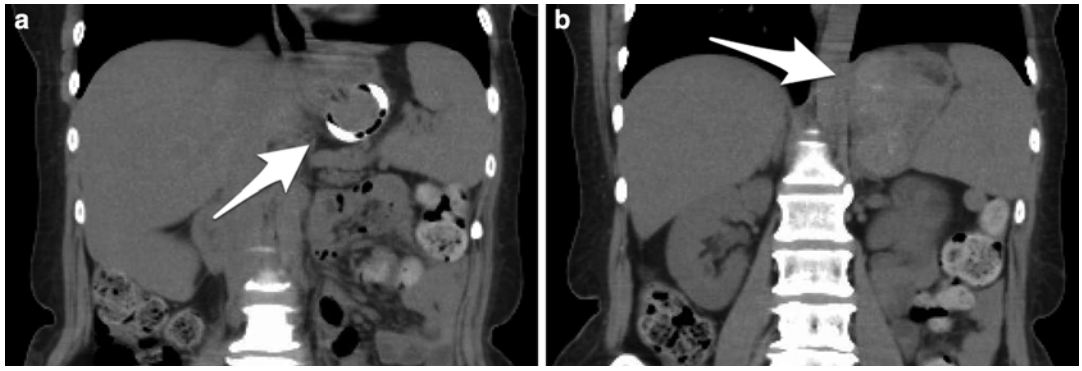


Fig. 16.4 CT scan demonstrates a vertically rotated gastric band (O sign) and a large posterior pouch

16.2 Management

The natural history of untreated prolapse remains unclear. Although this question is inadequately addressed in the literature, this author's experience suggests that patients with delayed follow-up or who refuse intervention do not appear to be at a higher risk of gastric compromise or the need for emergent intervention. Despite this, we continue to advise all patients found to have a prolapse to undergo some form of treatment to potentially avoid the catastrophic complication of progressive prolapse, leading to gastric ischemia, necrosis, and gastric perforation [19].

Patients who present acutely with a severely prolapsed band, abdominal pain, and obstruction and who do not respond to band decompression require emergent surgical intervention. Our practice in these rare cases is to remove the band and all of its associated components. Under these acute circumstances we do not routinely give any other revisional options to the patient. The reason for this is that we have found that the prolapsed section of stomach is edematous and friable and not in optimal condition to be manipulated and stapled. Also, these patients are often dehydrated and nutritionally depleted and thus not in optimal condition to undergo a revisional bariatric procedure. The primary focus in these patients, as with any surgical emergency patients, should emphasize lifesaving maneuvers; additional bariatric surgery should be considered on an elective, interval basis only. The remaining section of this chapter deals with patients presenting with a more chronic form of prolapse.

Management of chronic LAGB prolapse involves one of the four options:

1. A conservative non-operative approach
2. Band removal
3. Band revision (re-banding)
4. Band removal with conversion to an alternate bariatric procedure

16.3 Conservative Treatment

Nonsurgical management should be the first step in managing patients who present with prolapse. In our practice nonsurgical or conservative management includes band decompression and commencement of a strict liquid diet for a month. This is then followed by re-imaging and slow re-inflation of the band if the prolapse has resolved. Although the efficacy of this approach for patients presenting with a true prolapse has been questioned by some [20], the authors routinely follow this algorithm with acceptable results. The theory behind this approach is that since food impactions due to inadequate food processing led to obstruction with herniation and incarceration of the proximal stomach above the band [9], decompression and thereby relaxation of the "hernia ring" followed by a liquid diet will allow the pouch to spontaneously reduce. Some groups

have also reported on a technique using endoscopy and gastric insufflation to reduce the prolapse with reasonable outcomes [21].

If conservative management is successful, patients must be aggressively counseled and strongly encouraged to stick with a multidisciplinary nutritional, psychological, and medical approach. This last point cannot be overstated. Revisional surgery should only be considered as a last resort and this must be made very clear to the patient: "there is no surgery safer than no surgery."

16.4 Surgical Treatment

If conservative management fails, surgical treatment becomes the only remaining option. Helping patients choose between band removal, re-banding, or an alternative bariatric procedure is difficult and has traditionally been more based on patient preference and surgeon bias than published evidence. This is primarily due to the fact that data on LAGB prolapse and its optimal management is limited by small sample sizes and widely varying results.

Despite initial hopes that the LAGB would lead to durable lifestyle changes and by extension weight loss maintenance even after explantation, the literature suggests that patients opting for band explantation do not retain their weight loss and comorbidity resolution. In a report by Aarts et al. [22], not only had all 21 patients who had their band removed not been able to maintain their weight loss but their median weight had actually increased after 5 years of follow-up. This led them to recommend that all band explantations should be combined with a revisional bariatric operation.

Some have advocated an algorithmic approach to choosing a revisional procedure. In patients who were initially successful with the band but then experienced band complications such as prolapse, another restrictive procedure can be considered, e.g., re-banding or revisional laparoscopic sleeve gastrectomy (re-LSG). Patients who failed to achieve their weight loss goals however may instead be better served with a revisional laparoscopic Roux-en-Y gastric bypass (re-LRYGB) because of the added malabsorptive component [23]. This has also been the author's practice. Future studies will be required to definitively answer this question.

Because of extensive adhesion formation and fibrotic reaction surrounding the band, any revisional bariatric operation is difficult and often tedious, requiring advanced laparoscopic skills and an excellent knowledge of UGI anatomy. Revisional bariatric surgery should only be undertaken by experts and only after an extensive discussion with the patient regarding risks. The consent process should include a discussion of bleeding, gastric or esophageal perforation, and injury to the vagus nerve. These injuries may lead to significant morbidity and even death.

Another very important issue related to revisional bariatric surgery is managing patient expectations as far as weight loss and complication rates. Patients often have unrealistic expectations that their results and risk with revisional surgery will mimic those of their friends who have undergone primary bariatric surgery. Pros and cons must be weighed very carefully and discussed at length with each individual patient. Although true about any bariatric surgery, this is particularly true for revisional bariatric surgery. Lastly, we specifically discuss the paucity of data available regarding revisional surgery and encourage patients to consider this carefully before deciding on revisional surgery.

16.5 Technique

All revisional surgery involves the following common steps.

1. Lysis of adhesions between the liver, the anterior gastric wall, and the capsule of the band: These are often extensive.
2. Repair of hiatal hernia which is particularly common in patients with prolapse.
3. Unbuckling of the band.
4. The region of the gastric band capsule consists of fibrotic tissue which must be divided and dissected off of the gastric serosa.
5. Dissection of and clearance of the angle of His with visualization of the left crus.

Dismantling the wrap and resecting it or at least disrupting the band capsule is the most difficult and delicate part of revisional surgery. Some of the tricks and tips we use include the following:

1. The use of L-hook electrosurgery: The band is made of silicone and therefore is very amenable to the use of hook electrosurgery to lyse adhesions using the band as a baffle. The electrical current will not pass through the band, thereby protecting underlying structures (Fig. 16.5).
2. Adhesiolysis can be performed aggressively outside of the band ring. Other than where the wrap envelopes the band, anterolaterally and to the left of the buckle, the stomach/esophagus will always be contained within the band ring. This may be especially helpful as the surgeon comes around the buckle medially just adjacent to the caudate lobe and on the right crus.
3. The hiatus is an extremely important landmark and is most often a virgin surface just cephalad to the adhesions formed around the band (Fig. 16.6). Recognizing the hiatus helps keep dissection away from important structures such as the esophagus and IVC.
4. We use extreme care in releasing the gastrogastric wrap and use sharp dissection and electrocautery exclusively in areas that are clearly translucent, i.e., fibrous capsule (Fig. 16.7a). If a segment of tissue is suspected of being the gastric wrap, we tend to divide it using a stapler rather than taking the risk of injury and leak (Fig. 16.7b).
5. Unbuckling of the band can be very helpful. Doing this without damaging the band is important if the plan is to reuse the band. The original Lap-Band® and Realize® bands were significantly more difficult to unbuckle as compared to the current AP band, where a simple traction-countertraction maneuver suffices.
6. Stripping the capsule can be tedious, but is particularly important for patients undergoing revisional sleeve or bypass. Some authors feel that this stripping must be circumferential and not just on the anterior surface (Figs. 16.8, 16.9, 16.10, and 16.11).
7. Leaving the band in situ may help identify the posterior aspect of the capsule. Even in the scarred posterior retroperitoneal attachments of the proximal stomach where the anatomy is obscured, the band can be easily palpated and then, as described above, electrocautery used on the band to divide the capsule in a very safe manner.

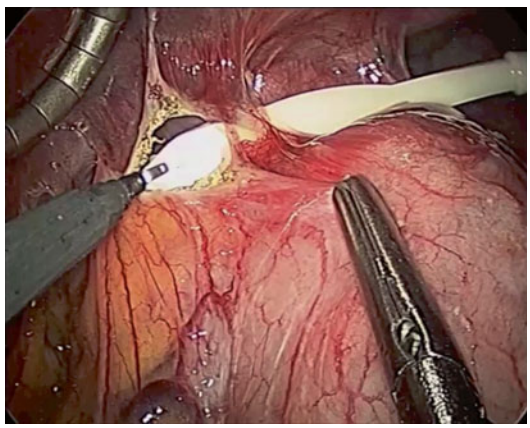


Fig. 16.5 The hook electrocautery can be used to lyse adhesions directly overlying the band using the band as a baffle

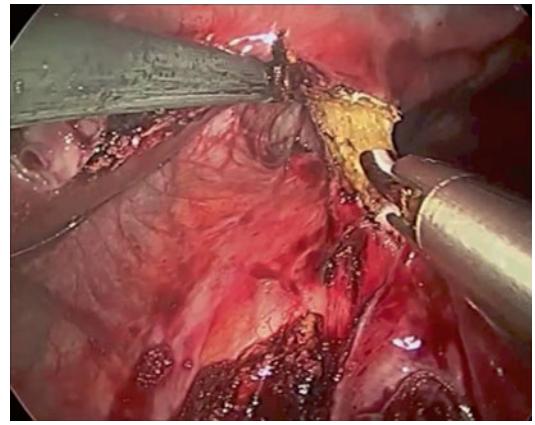
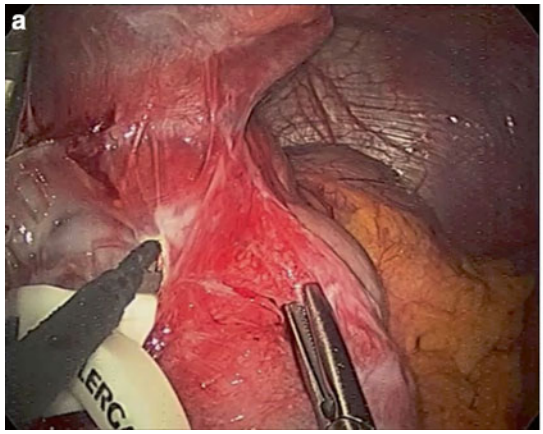


Fig. 16.6 Identifying the hiatus is an extremely important landmark and is most often a virgin surface just cephalad to the adhesions formed around the band

16.5.1 Re-banding

As described above, the evidence for re-banding is lacking and results vary in the literature. In a recent paper, Suter [24] described his experience



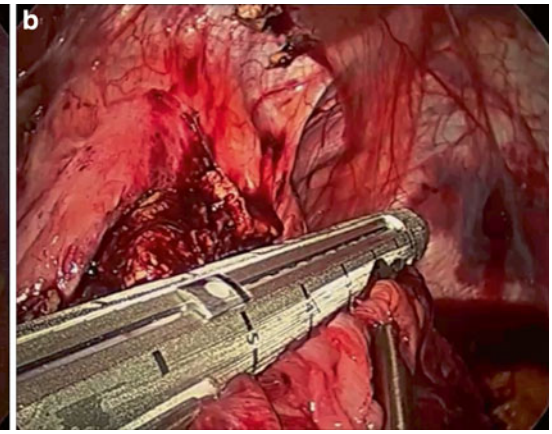


Fig. 16.7 (**a**) Extreme care should be exercised in releasing the gastro-gastric wrap. Sharp dissection and electrocautery should only be sued in areas that are clearly translucent, i.e., fibrous capsule. (**b**) Any area even suspected of being gastric wrap, we tend to divide using a stapler rather than taking the risk of gastric injury

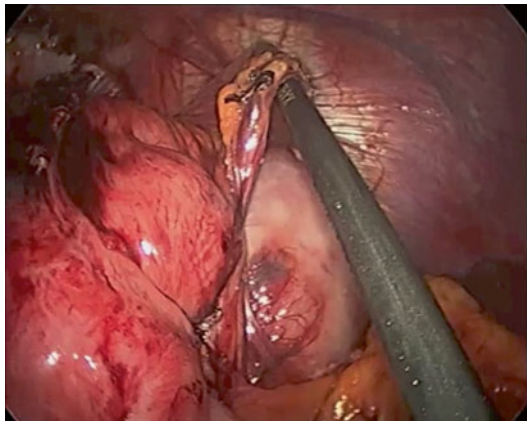


Fig. 16.8 Stripping the capsule can be tedious, but is particularly important for patients undergoing revisional sleeve or bypass. This stripping must be circumferential and not just on the anterior surface

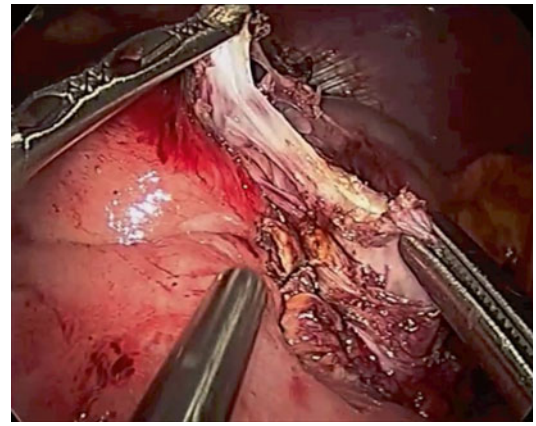


Fig. 16.10 There is often an adhesive band extending around the prolapsed segment of stomach which also must be divided

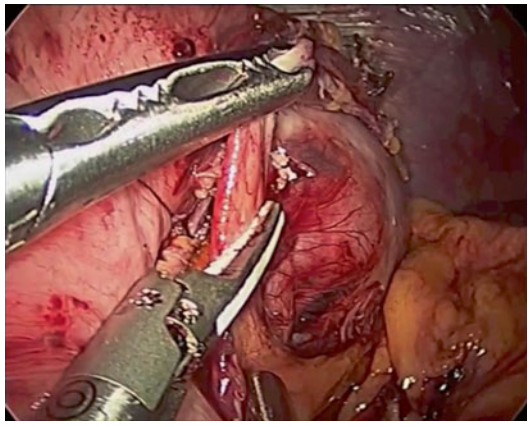


Fig. 16.9 Stripping of the capsule then proceeds laterally

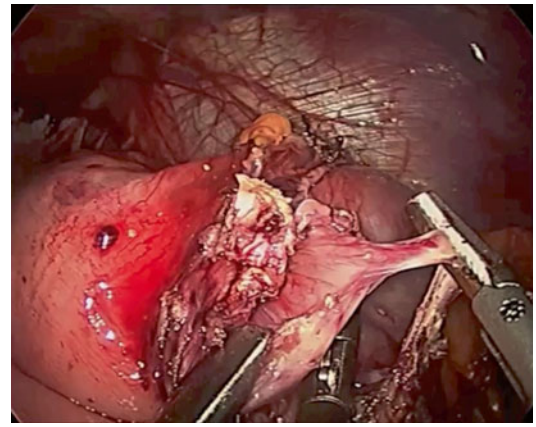


Fig. 16.11 Finally the capsule is stripped posterolaterally to complete the freeing of the prolapsed stomach

with a small number of patients (9 patients) undergoing re-banding after prolapse. After a mean follow-up of 20 months, 6 (66.6 %) had insufficient weight loss and only 2 (22 %) went on to lose further weight. More than half of these patients required further surgery, including band removal in three and conversion to gastric bypass in two. He concluded that re-banding for prolapse produces disappointing results in midterm follow-up.

Ponce et al. [25] reported their experience with re-banding for prolapse in 40 consecutive patients. They found that after a mean follow-up of 17.6 months, their mean percentage of excess weight loss after band repositioning was 48.1 % (range 18.2–77.4 %) with only 5 % subsequently explanted. They also noted successful comorbidity resolution of DM, HTN, and sleep apnea in 60 %, 65 %, and 72 % of patients, respectively. They concluded that re-banding results in preservation of most of the initial weight loss and comorbidity resolution.

Foletto et al. [26] reviewed 29 patients undergoing re-banding for prolapse. Mean BMI at the time of revisional surgery was 34.3 and at 1-, 2-, and 3-year follow-up the mean BMI were 36.3, 37.13, and 33.5, respectively. Two patients (7 %) experienced recurrence of the prolapse and five (17 %) required additional revisional surgery including re-banding, band removal, biliopancreatic diversion, and sleeve gastrectomy. The authors concluded that re-banding was a reasonable approach to patients with a prolapsed band with good outcomes.

Riele et al. [27] reported on 81 patients who underwent re-banding for prolapse. They concluded that re-banded patients had similar results when compared to patients who had undergone primary banding. As expected though, they found that re-banding unsuccessful LAGB patients resulted in poor long-term weight loss results.

After completion of the lysis of adhesions common to all revisional procedures (see above) re-banding requires a repassage of the band into the proper location. It is not uncommon that due to the fibrotic reaction caused by the band the standard tunnel site will have become obliterated, thus preventing the band from achieving the standard Phi angle. In the author's experience this has not had a negative impact on patient success following band replacement. We also have noted that even in smaller patients who had previously been managed with a standard-sized band (AP-S), the fibrosis and scarring present at reoperation necessitates use of the large band (AP-L). Again in the author's experience, this has not affected outcomes but requires an extensive preoperative discussion with the patient regarding differences in adjustment volumes and schedules.

Issues to be discussed with patients specific to re-banding include the fact that the band adjustment strategy may be very different from what they had previously experienced, requiring higher or lower volumes of saline as compared to the initial band. In addition, expected weight loss may not materialize; patients who experienced good results after their initial band placement may be disappointed after the re-banding surgery.

16.5.2 Conversion to an Alternate Bariatric Procedure

In patients who elect to have their band converted to an alternate bariatric procedure, their choices include conversion of the band to a laparoscopic sleeve (LSG) or classically to a gastric bypass (LRYGB). A third option of conversion to a BPD-DS is described in the literature and is steadily gaining adherents.

The importance of optimizing adhesiolysis to flatten out the stomach and return it back to its anatomical shape cannot be overstated. The key to this lies in dividing the band capsule entirely not just anteriorly but also posteriorly where even after the band itself is divided the capsule retains its shape and rigidity and will hold the stomach in its prolapsed position preventing correct stapling of the pouch or sleeve.

16.5.2.1 Revisional Laparoscopic Sleeve Gastrectomy (re-LSG)

Literature regarding the results of re-LSG performed for band prolapse is not readily available. In most reports, prolapse as an indication for revision is lumped together with other indications for

revision most notably weight loss failure [28]. A retrospective review of 90 band to sleeve revisions was reported by Yazbek et al. [29]. The indication for revision in the majority of these cases was inadequate weight loss (IWL) but one-third were performed for prolapse. All but two of these cases were performed in one stage. Mean postoperative %EWL was 54.1 % after a mean follow-up of 2 years. Comorbidity resolution or improvement was noted in 82.3 %, 64.2 %, 44.4 %, and 53.1 % of patients with OSA, DM, hyperlipidemia, and HTN, respectively. There was however an almost tenfold increase in complications following re-LSG which included leaks (5.5 %) and gastric hematomas (4.4 %). No specific data were provided to determine if outcomes and/or complications differed between the IWL group and the prolapsed group. They concluded that re-LSG is an effective revisional procedure albeit with an increased complication rate as compared to primary sleeves.

A recent systematic review, however, concluded otherwise. Elnahas et al. [30] looked at revisional surgery performed for failed LAGB (not specifically prolapse) and found that re-LSG was an inadequate revisional procedure when compared to re-LRYGB and re-BPD-DS.

As primary LSG has become more popular, the volume of centers making re-LSG available to their band patients requiring revisional surgery is naturally expanding. We expect reports in the literature to grow in magnitude and finally allow some definitive statements to be made regarding the efficacy and safety of this operation for patients presenting with band prolapse.

The authors routinely perform extensive capsule division to preclude leaving large areas of pouch behind leading to poor weight loss or complaints of food stasis in the pouch. Other complications of leaving the capsule intact are the potential for a significant twist or stapling multiple layers of gastric wall, any of which may lead to an increased tendency for bleeding and/or leak. We therefore recommend a dissection down to serosa and a complete division of the capsule.

Although the authors routinely advocate a single-stage band-to-LSG in patients undergoing revision, specifically for patients with prolapse, consideration should be given a two-step approach. We have found that allowing the stomach time to return to its natural anatomical shape prior to performing a sleeve gastrectomy may ultimately result in more symmetric sleeve anatomy. The clinical significance of this, however, remains unknown.

16.5.2.2 Revisional RYGB (re-LRYGB)

Revision to a gastric bypass after the removal of the gastric band is considered by many surgeons to be the gold standard. This may be particularly appealing in the patient with weight loss failure or weight regain who is still struggling with morbid obesity and its complications. Many view this as a natural choice for revision because in a patient that had demonstrated failure with a restrictive operation the bypass will add malabsorption and hormonal change.

Literature describing revision from band to the bypass has demonstrated safety and good weight loss. However, most papers have a relatively small sample size, do not report results of prolapse separately, and have poor long-term follow-up. Results in the literature have also been widely variable and may in part be due to differences in surgical technique and learning curve [31–34].

A number of recent larger series have described good results and safety with a one-stage or a two-stage revision. Hii et al. [35] presented 82 patients with revision from band to bypass. Patients required revision due to inadequate weight loss (51 %), slippage (13 %), esophageal dilation (13 %), dysphagia/reflux (12 %), and erosion (8 %). Single-stage conversion to gastric bypass was possible in 78 % of patients. The pouch was created 6 cm below the gastroesophageal junction. Usually this was below the level of the previous band. If a staple line was required to be placed at the level of the band capsule, the authors peeled the capsule off the stomach. In constructing the gastrojejunostomy, the authors used circular stapler, linear stapler, and hand-sewn techniques. The alimentary limb was 100–150 cm. There were no perioperative mortalities and one anastomotic leak. Stricture rate was 19.5 % mainly attributed to hand-sewn technique and the use of a 21 mm circular stapler. The circular stapler was

also associated with a 26 % wound infection rate. Ulcer rate was 2.4 %. The ten patients who had preoperative symptoms of reflux and dysphagia had resolution of their symptoms. EWL at 1 year was 50 % (with 68 % follow-up).

In the largest series to date looking at revisions from band to bypass with a sample size of 257 patients, Emous et al. show that re-LRYGB is safe and effective [32]. A two-stage approach was used for patients with infection, erosion, migration, or poor tissue quality. A two-stage approach was also employed for those who had slippage and whose symptoms did not resolve with band deflation and for those who did not want to wait for a planned one-stage operation. In this series, the pouch extended below the scar tissue from the gastric band, the capsule was inconsistently removed, and a linear stapler was used for the gastrojejunostomy. Leak rate with the one-stage group was 2.3 % and with the two-stage group was 4.5 %. At 29-month follow-up, EWL was 53 % with the one-stage group and 67 % with the two-stage group. There were no deaths, seven leaks, two intra-abdominal abscesses, seven gastric ulcers, and two strictures; there were no statistically significant differences between the one- and two-stage groups. With median 29-month follow-up, EWL was 53 and 67 % with the one- and two-stage groups, which was not statistically significant.

Aarts et al. [36] presented a retrospective series of 195 patients who were revised from band to bypass in a single operation. Twenty-two of these patients had required revision due to band slippage. They excluded patients who had a known band migration; in these cases, they removed the band in the initial setting and brought the patients back later for a revision. Their technique included a linear stapled gastrojejunostomy, a 100–150 cm Roux limb, and a pouch just extending below the scar tissue from the gastric band. Post-op complications included two leaks, two strictures, and one abscess at the gastrojejunostomy anastomosis. EWL was 63 % at 3 years (with 64 % patient follow-up) and 53 % at 5 years (with 15 % patient follow-up).

There are a number of technical considerations with the gastric pouch in a revision case. Some surgeons remove the capsule while others leave it intact. Many surgeons start the creation of the pouch below the scar tissue from the gastric band, although some surgeons go above. The concern for starting the staple line distal to the capsule is that the pouch may be larger than desired, thereby leading to inadequate weight loss. On the other hand, creating the pouch above the capsule requires division and dissection of the capsule and anastomosing in a scarred and fibrotic area which may increase the risk of leak. Circular stapler, linear stapler, and hand-sewn gastrojejunostomy have all been described. There are insufficient published data to definitively draw any conclusions regarding either the timing of surgery (one stage vs. two stages), excision of the capsule, location of the staple line, or type of gastrojejunostomy. Further studies with randomization, larger sample size, and longer follow-up are needed to help determine the best timing and technique for re-LRYGB for patients with lap band prolapse.

Comparing the different revisional options, Müller et al. [37] compared re-banding to re-LRYGB in 74 consecutive patients (re-banding 44 and re-LRYGB 30) for failed LAGB during a median follow-up of 36 months. Patients who underwent re-LRYGB had significantly better weight loss than patients who had undergone a re-banding operation. In addition they found that almost half of the re-banding group needed a further operative revision, as compared to only 20 % in the re-LRYGB group. They concluded that LRYGB is a better rescue treatment than re-banding and should thus be the preferred choice after failed laparoscopic gastric banding. Indications for band revision included prolapse but no specific data regarding the number and outcomes of this group of patients is provided in the paper.

In a recent systematic review, Coblijn et al. [38] compared patients undergoing revision of their bands to either re-LS or re-LRYGB. They were unable to reach a recommendation between these two procedures, although re-LRYGB seemed to have better long-term weight loss, but this was mainly because the data on re-LSG was lacking in long-term outcomes. Prolapse as an

indication for revision was present in only a very small minority of patients [65 (16.8 %) re-LRYGB and 2 (1 %) re-LSG] absolutely precluding drawing any conclusions.

In summary, lap band prolapse is a very-well-described complication following LAGB. Limited and variable data preclude definitive recommendations regarding the optimal treatment, timing, and ideal revisional choice for these complex patients.

References

1. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. JAMA. 2014;311(8):806–14.
2. Parikh M, Pachter L, et al. Randomized pilot trial of bariatric surgery versus intensive medical weight management on diabetes remission in type 2 diabetic patients who do NOT meet NIH criteria for surgery and the role of soluble RAGE as a novel biomarker of success. Ann Surg. 2014;260(4):617–22.
3. Schauer P, Bhatt D, et al. Bariatric surgery versus intensive medical therapy for diabetes—3-year outcomes. N Eng J Med. 2014;370(21):2002–13.
4. Mingrone G, Rubino F, et al. Bariatric surgery versus conventional medical therapy for type 2 diabetes. N Eng J Med. 2012;366(17):1577–85.
5. Dixon J, O'Brien P, et al. Adjustable gastric banding and conventional therapy for type 2 diabetes: a randomized controlled trial. JAMA. 2008;299(3):316–23.
6. Belachew M, Legrand MJ, Defechereux TH, et al. Laparoscopic adjustable sili-cone gastric banding in the treatment of morbid obesity. A preliminary report. Surg Endosc. 1994;8(11):1354–6.
7. Snow J, Severson P. Complications of adjustable gastric banding. Surg Clin North Am. 2011;91(6):1249–64. ix.
8. Moser F, Horgan S, et al. Pouch enlargement and band slippage: two different entities. Surg Endosc. 2006; 20:1021–9.
9. O'Brien P, Dixon J. Weight loss and early and late complications—the international experience. Am J Surg. 2002;184(6):42S–5.
10. Fielding G, Duncombe J. Clinical and radiological follow-up of laparoscopic adjustable gastric bands, 1998 and 2000: a comparison of two techniques. Obes Surg. 2005;15(5):634–40.
11. Fielding G, Allen J. A step-by-step guide to placement of the LAP-BAND adjustable gastric banding system. Am J Surg. 2002;184:26S–30.
12. Ren C, Fielding G. Laparoscopic adjustable gastric banding: surgical technique. J Laparoendosc Adv Surg Tech. 2003;13(4):257–63.
13. O'Brien PE, Dixon JB, Laurie C, Anderson M. A prospective randomized trial of placement of the laparoscopic adjustable gastric band: comparison of the perigastric and pars flaccida pathways. Obes Surg. 2005;15(6):820–6.
14. Sherwinter D, Powers C, Geiss A. Posterior prolapse: an important entity even in the modern age of the pars flaccida approach to lap-band placement. Obes Surg. 2006;16(10):1312–7.
15. Mizrahi S, Avinoah E. Technical tips for laparoscopic gastric banding: 6 years' experience in 2800 procedures by a single surgical team. Am J Surg. 2007;193(2):160–5.
16. Marescaux J, Lunca S, et al. Late gastric prolapse with pouch necrosis after laparoscopic adjustable gastric banding. Obes Surg. 2005;15(4):571–5.
17. Sonavane S, Menias C, et al. Laparoscopic adjustable gastric banding: what radiologists need to know. RadioGraphics. 2012;32:1161–78.
18. Pieroni S, Sommer E, et al. The "O" sign, a simple and helpful tool in the diagnosis of laparoscopic adjustable gastric band slippage. Am J Roentgenol. 2010;195(1):137–41.
19. Lunca S, Vix M, Rikkers A, et al. Late gastric prolapse with pouch necrosis after laparoscopic adjustable gastric banding. Obes Surg. 2005;15(4):571–5.
20. Moser F, Gorodner MV, Galvani CA, Baptista M, Chretien C, Horgan S. Pouch enlargement and band slippage: two different entities. Surg Endosc. 2006;20(7):1021–9. Epub 2006 May 13.
21. Kang SH, Kim KC, Kim KH. Endoscopic treatment of gastric band prolapse. Obes Surg. 2014;24(6):954–7.
22. Aarts EO, Dogan K, Koehestanie P, Janssen IM, Berends FJ. What happens after gastric band removal without additional bariatric surgery? Surg Obes Relat Dis. 2014;10(6):1092–6.
23. Zundel N, Hernandez JD. Revisional surgery after restrictive procedures for morbid obesity. Surg Laparosc Endosc Percutan Tech. 2010;20(5):338–43.
24. Suter M. Laparoscopic band repositioning for pouch dilatation/slippage after gastric banding: disappointing results. Obes Surg. 2001;11(4):507–12.
25. Ponce J, Fromm R, Paynter S. Outcomes after laparoscopic adjustable gastric band repositioning for slippage or pouch dilation. Surg Obes Relat Dis. 2006;2(6):627–31.
26. Foletto M, Bernante P, Busetto L, Pomerri F, Vecchiato G, Prevedello L, Famengo S, Nitti D. Laparoscopic gastric rebanding for slippage with pouch dilation: results on 29 consecutive patients. Obes Surg. 2008;18(9):1099–103.
27. te Riele WW, van Santvoort HC, Boerma D, van Westreenen HL, Wiezer MJ, van Ramshorst B. Rebanding for slippage after gastric banding: should we do it? Obes Surg. 2014;24(4):588–93.
28. Noel P, Schneck AS, Nedelcu M, Lee JW, Gugenheim J, Gagner M, Iannelli A. Laparoscopic sleeve gastrectomy as a revisional procedure for failed gastric banding: lessons from 300 consecutive cases. Surg Obes Relat Dis. 2014;10(6):1116–22.
29. Yazbek T, Safa N, Denis R, Atlas H, Garneau PY. Laparoscopic sleeve gastrectomy (LSG)-a good

bariatric option for failed laparoscopic adjustable gastric banding (LAGB): a review of 90 patients. Obes Surg. 2013;23(3):300–5.
30. Elnahas A, Graybiel K, Farrokhyar F, Gmora S, Anvari M, Hong D. Revisional surgery after failed laparoscopic adjustable gastric banding: a systematic review. Surg Endosc. 2013;27(3):740–5.
31. Emous M, et al. Conversion of failed laparoscopic adjustable gastric banding to Roux-en-Y gastric bypass is safe as a single-step procedure. Surg Endosc. 2014;16.
32. Spivak H, et al. Laparoscopic revision from LAP-BAND to gastric bypass. Surg Endosc. 2007;21(8):1388–92.
33. Apers J, et al. Perioperative outcomes of revisional laparoscopic gastric bypass after failed adjustable gastric banding and after vertical banded gastroplasty: experience with 107 cases and subgroup analysis. Surg Endosc. 2013;27(2):558–64.
34. Marin-Perez P, Rosenthal R, et al. Outcomes after laparoscopic conversion of failed adjustable gastric banding to sleeve gastrectomy or Roux-en-Y gastric bypass. Br J Surg. 2014;101(3):254–60.
35. Hii MW, Lake AC, Kenfield C, Hopkins GH. Laparoscopic conversion of failed gastric banding to Roux-en-Y gastric bypass: short-term follow-up and technical considerations. Obes Surg. 2012;22(7):1022–8.
36. Aarts E, Janssen I, et al. Revisional surgery after failed gastric banding: results of one-stage conversion to RYGB in 195 patients. Surg Obes Relat Dis. 2014;10(6):1077–83.
37. Müller MK, Attigah N, Wildi S, Hahnloser D, Hauser R, Clavien PA, Weber M. High secondary failure rate of rebanding after failed gastric banding. Surg Endosc. 2008;22(2):448–53.
38. Coblijn UK, Verveld CJ, van Wagensveld BA, Lagarde SM. Laparoscopic Roux-en-Y gastric bypass or laparoscopic sleeve gastrectomy as revisional procedure after adjustable gastric band—a systematic review. Obes Surg. 2013;23(11):1899–914.

Band Erosion: Surgical and Endoscopic Management

17

Paul Thodiyil and Petros Benias

Key Points

- Erosion of adjustable gastric bands is now uncommon.
- Erosion typically presents with weight regain and loss of restriction.
- Upper endoscopy is the diagnostic tool of choice.
- Treatment involves band explantation and staged conversion of another band, sleeve gastrectomy, or a gastric bypass.
- Such revision will likely return the patient to expected weight loss trajectory.

17.1 Introduction

Erosion of an adjustable gastric band refers to its migration into the gastric lumen. It renders the band ineffective, unable to create a restrictive effect, with a resultant weight regain or a failure to lose additional weight. With an average reported incidence of 1.46 %, erosion is uncommon. However, because of the significant clinical implications, it is important to understand this complication and define preventive and treatment strategies [1].

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17.2 Pathophysiology

The etiology of erosions remains unclear. Erosion of prosthetic material into the stomach has previously been well described with the Angelchik prosthesis and the banded gastroplasties [2–6], with erosion rates in the vertical banded gastroplasty ranging from 1.2 to 4 %. These typically occur after a 3-year lag period. The histological characteristics of the pseudocapsule surrounding the band are similar in those with and without erosion [7], suggesting erosions do not represent an idiosyncratic response.

The possibility of an infection of the band progressing to erosion is probably a factor in those patients with gastric perforations, interval cholecystectomy, or early postoperative access port infection. Primary postoperative infection of the access port sites has been reported to be as high as 16.5 % in patients developing erosions [8], compared to the expected rate of 0.36 % [9], supporting an etiological role for port site infections in the development of erosions. However, late port infections, in contrast to primary postoperative infection, are more likely a manifestation than a cause of erosion.

Focal pressure of the band on the gastric wall, caused by anchoring the pouch to the crura or incorporating the buckle of the band, has also been implicated as a potential cause. Other risk factors for the development of erosions include

D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_17

use of the early 10 cm band (4.1 %) or use of the perigastric technique in band placement (6.6 %) [8]. In contrast, the risk with the Lap-Band AP Small™ is 0.9 % and the pars flaccida placement technique 1.1 % [8]. The location of the erosion in the largest published series was in the region of the buckle of the band, followed by the anterior aspect of the lesser curve and to a lesser extent on the high posterior gastric wall [8]. Other series report the majority of erosions along the greater curvature [10].

17.3 Epidemiology

The incidence of erosions after a primary laparoscopic adjustable gastric band is 0.2–4 % [8, 11–18] with a higher incidence (6.8–7.6 %) [19] after revision of previous weight loss procedures to LAGB. There is no difference in age, gender, initial weight, or BMI between a background cohort without erosion compared to those developing erosion [8]. The median interval to erosion was 33 months (range: 11–170 months) in one of the largest series to date [8]. Di Lorenzo, in a retrospective multicenter review of 117 erosions in 6,839 patients report a different time pattern of erosions with 41.8 %, 17.9 %, 21.4 %, 22.6 %, 3.4 %, and 2.2 % presenting at 6–12, 24, 36, 48, 60, and 72 months after band placement respectively. Suter et al. reported a mean interval of 22.5 months from primary band placement [19].

Most gastric band series have reported an early cluster of erosions. The reason for the early cluster is probably related to a number of factors, including the learning curve [8, 17] and initial adoption of the perigastric technique (Fig. 17.1) [20]. An aspect of the operative technique where an attempt was made to achieve complete anterior fixation by suturing right up to and including the buckle was probably contributory. The buckle area was frequently noted to be the lead point of the erosion (unpublished personal communication). The prevalence of erosions fell once the buckle was excluded from the gastrogastric sutures, a trend that started before the transition from a perigastric approach to the pars flaccida technique.

17.4 Clinical Presentation

The clinical manifestations of band erosion are summarized in Table 17.1. The characteristic features of band erosion include of a loss of satiety, weight gain, and a need for a steadily increasing band fill volume in an effort to control appetite. In this series, initial appearance of erosion

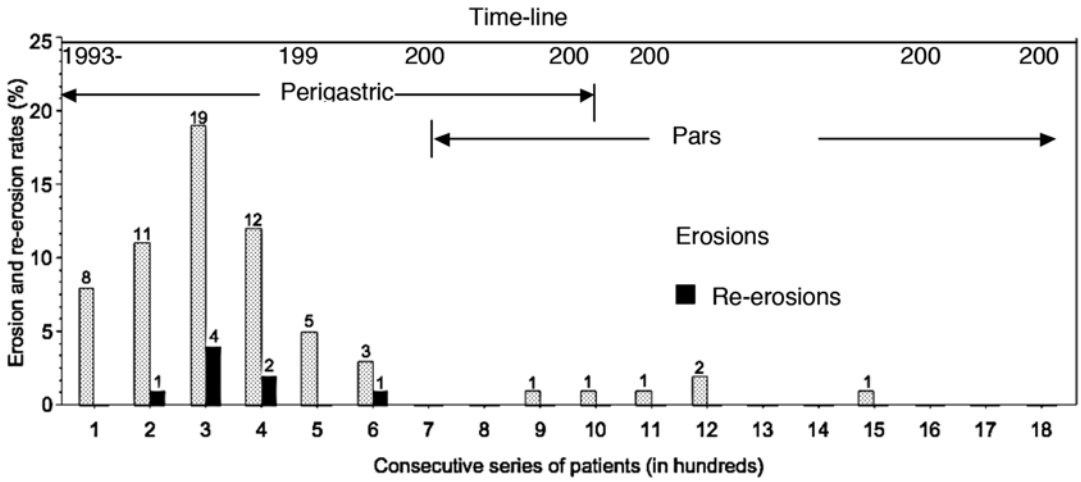

Fig. 17.1 This shows the distribution of erosions by consecutive series of hundred cases of LAGB Bands performed. Most erosions occurred early in the learning curve, peaking at a rate of 19 %, but now falling to below 1 %

Table 17.1 Summary of clinical manifestations of band erosion [20]

Symptom	Percentage (%)
Satiety	
Loss of satiety	83
No change in satiety	14.1
Weight gain	
Weight regain	64
Weight Loss maintained	27
Abdominal pain	
Site: Epigastric	45
Left upper quadrant	35
Epigastric, Left upper quadrant	25
Left flank	5
Port Fluid Characteristics	
Need for increasing band volume	78
Missing volume	22
Gastric fluid	19
Air on aspiration (>1 ml)	2
Port Issues [8]	
Spontaneous infection	21
Extrusion of port	3
Port site tenderness	1
Vomiting	22
Anemia	14
Heartburn	10
Hematemesis	5
Dysphagia	3

occurred at a mean time interval 24.5 months (SD 20.7, range 1.3–95.5 months) after band placement [20] and 4.5 ± 3.7 months before diagnosis [8]. Those with a perigastric approach present later compared to the pars flaccida approach (43 months vs. 20 months) [8].

The patient with an eroded band complains of a gradual loss of satiety and subsequent weight regain [20]. The loss of satiety typically results in the managing physician steadily adding fluid to the band during a lead period that averages of 36 months prior to endoscopic diagnosis of erosion. This is associated with a mean weight gain of 2.3 BMI units, starting at an average 9 months prior to endoscopic confirmation of the diagnosis of erosion [20]. Late, spontaneous infection of the access port may occur in 21 % of patients and should invariably lead to a search for band erosion using endoscopy. While most patients present with a range of benign symptoms such as decreased satiety, about 5 % present to the emergency room with hematemesis [21–23].

17.5 Diagnosis

A diagnostic workup to look for erosion should be prompted by the appearance of the abovementioned clinical features. The long delay between the onset of symptoms and eventual diagnosis is in keeping with its characteristically benign and insidious presentation. Gastroscopy remains the definitive diagnostic tool. While the diagnosis is confirmed by intragastric visualization of the LAGB at endoscopy (Fig. 17.2a), radiologic imaging, especially with a barium meal or a CT scan, can occasionally reveal pathognomonic features (Fig. 17.2b) [24]. However, sensitivity of an imaging is very poor at only 8 %.

The diagnosis of erosion is sometimes made intraoperatively, in 5 % of patients who either undergo emergent surgery for hematemesis or for removal of a prolapsed band. Endoscopic diagnosis is typically made at a median 32.2 (range 24–58) months from band placement, with the interval from first clinical manifestation to eventual endoscopic diagnosis a median of 10.7 (range 1.6–27.8) months.

17.6 Management

There are two issues to be considered in the management of band erosion. The first relates to the management of the erosion itself while the second concerns the choice of a subsequent weight loss procedure. The eroded band may be removed surgically or endoscopically, while the options for the subsequent obesity intervention range from primary or staged band replacement to use of an alternate bariatric procedure such as a sleeve gastrectomy or gastric bypass. Clearly, it is also an option to pursue dietary and behavioral management alone, if the patient is uninterested in or unable to undergo further surgery.

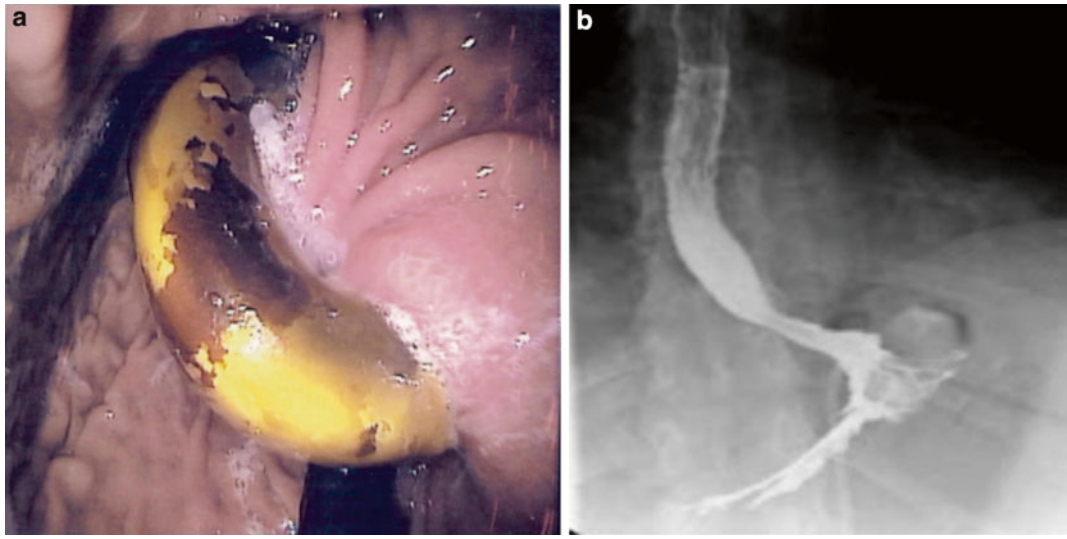

Fig. 17.2 Figure (**a**) shows a band eroding into the gastric lumen, losing its ability to cause restriction. Figure (**b**) shows the rare instance with contrast outlining part of an intraluminal band. It is unusual to see a diagnostic barium meal even in the presence of complete erosion into the stomach. Courtesy of P. E. O'Brien, MD

17.7 Surgical Explantation

Surgical explantation of an eroded band can be achieved laparoscopically, utilizing previous port sites one of which will typically be a 15 mm port. Despite the frequent presence of adhesions between the left lobe of the liver and the anterior gastric wall, the left subcostal area is often free of adhesions. A CO_2 pneumoperitoneum to 15 mmHg can be safely achieved using a 5 mm left subcostal optical trocar. Under direct vision, using a 5 mm 30-degree laparoscope, a 15 mm port is placed about 15 cm beneath the xiphisternum just to the left of the midline. Two 5 mm ports are then introduced, one at the left subcostal anterior axillary line and the second at the right subcostal and mid-clavicular lines. It is expected that there will be significant adhesions between the left lobe of the liver and the anterior gastric wall, which will require careful mobilization. This can be facilitated by use of a Nathanson liver retractor and a steep reverse Trendelenburg position.

With the patient in reverse Trendelenburg position, the tubing of the lap band is identified and divided as it enters the peritoneal cavity from the access port. The proximal end can be used as a guide to the band and its buckle. It is traced through the inflammatory mass or adhesions to the upper anterior gastric wall. Using an L-hook cautery or ultrasonic shears, the pseudocapsule and gastrogastric sutures are divided to expose the lap band. If the anterior portion of the band is completely intragastric, a small gastrotomy will need to be created anteriorly just below the buckle in order to allow access to the intraluminal band. When a portion of the band is still extragastric anteriorly, simply incising the pseudocapsule will expose the band. Using endoshears, the exposed band is divided thus permitting its removal from within the stomach. The gastric defect in the stomach can be closed with interrupted 2–0 absorbable sutures. The band and tubing are then removed from the peritoneal cavity through the 15 mm port. Copious irrigation is carried out and integrity of the closure is confirmed by testing for leaks while insufflating the stomach with air using an orogastric tube or gastroscope. The gastrotomy repair site may be reinforced with fibrin sealant. Additionally, an omental patch may be applied to the area.

A careful inspection should be made of the intraperitoneal tubing prior to conclusion of the procedure as secondary extra-gastric sites of additional erosion may exist [25]. This author

(PT) has operated on one patient with intragastric band erosion where upon retrieving the band and tubing, a 2" segment of tubing within an adjacent inflammatory mass on the transverse colon was noted to be stained yellow. Assuming this to be due to erosion of the tubing into the transverse colon, the two exit points of the tubing in this inflammatory mass were sutured using interrupted 2–0 polyglactin suture with an uneventful postoperative course. The incidence of such secondary or extra-gastric erosions remains unknown.

After completion of the exploration, the access port is then removed by dividing its anchoring sutures and applying traction to the port and its attached tubing. The access port site is irrigated and then either left open or loosely closed.

The procedure is generally well-tolerated, although 11 % of patients will develop a superficial surgical site infection [8], usually involving the port through which the port is removed or the site of the access port. Less common complications include wound abscess (4 %) and gastric fistula (2 %) [8]. In our series there were no deaths either in the perioperative period or in the period of follow up (median 44 months after band replacement and 2.5 months after explant only) [20].

17.8 Endoscopic Explantation

The endoscopic approach is less commonly used for removal of an eroded band. Before endoscopic removal of an eroded band can be attempted there are two prerequisites. First, the access port must be removed surgically, leaving the connecting tube in the peritoneal cavity. This procedure can be performed with minimal anesthesia and is usually well-tolerated. Second, the band must show at least 50 % effacement [26, 27]. If this is not the case, the erosion can be given time to mature. Periodic endoscopic surveillance will document progression of the intragastric migration.

The most commonly applied technique for cutting the band endoscopically is the so called butter-wire technique in which a 0.035" wire is threaded around the eroded band and pulled out through the mouth. In this manner a long wire is placed around the band and both ends can be fed into the crank handle of a mechanical lithotripter. Controlled pressure is applied incrementally until the band material cracks and is transected [28]. The eroded band can then be completely removed in two pieces through the oropharynx with the use of a standard metal snare. It is not necessary to perform any closure after the band is removed as the tract is often formed slowly over many months and there is a cicatricial response. Free perforation into the peritoneal cavity is thus exceedingly rare.

The endoscopic removal of an adjustable band is a minimally invasive technique, which can be performed in a single outpatient session, providing a simple solution to a potentially serious complication. Successful endoscopic removal can be achieved in between 80 and 95 % of patients who meet the criteria noted above [18, 29, 30]. The largest series of endoscopic band removal [18] described 50 cases with successful removal in 46 (92 %), requiring 1–5 endoscopies (median of 1) prior to removal. The median duration of the procedure was 46 min with a range of 17–118 min [18]. Failure to remove the eroded band or other intraoperative events lead to conversion to a formal surgical procedure in the operating room [30].

There is no established algorithm for endoscopic management due in part to its relatively low incidence. Where the erosion is early, with less than 50 % effacement, endoscopic monitoring for progressive effacement may not be without risk especially major hematemesis [23]. Endoscopic needle knife gastrotomy over the partially eroded band has been described as a novel technique to hasten intraluminal effacement [31]. While rare, complications from endoscopic band removal may occur, including bleeding or other adverse events. For this reason, it is advisable to undertake the procedure in the operating theater rather than the endoscopy suite [30, 32]. Pneumoperitoneum requiring surgical decompression [29] and gastric fistula or subcutaneous abscess at the site of the access port have been reported as possible complications of endoscopic explantation. Pain, and sometimes port

site infection, can be observed and often managed conservatively.

Endoscopic explantation has not gained widespread popularity outside a small number of centers, in part because of delays in definitive treatment for band effacement and consequent need for multiple endoscopies and anesthesia in addition to the required operative procedure for removal of the access port [33].

17.9 Management of Obesity after Band Explantation

There are several approaches to the management of obesity after band explantation for erosion, ranging from band replacement or other bariatric operation to medical and behavioral therapy.

17.9.1 Explantation with Synchronous Band Replacement

This approach is not recommended, and is now only of historic significance. This approach may result in re-erosion rates as high as 40 % [8], with mean time to re-erosion of 45 months. The patients who did not experience re-erosion were able to achieve weight loss and return to their previous weight loss trajectory.

17.9.2 Explantation with Staged Band Replacement

In programs that offer the adjustable gastric band as their preferred weight loss surgical option, staged laparoscopic band replacement is the standard approach [8, 34, 35]. The band is replaced after an interval of 3 or more months. Of 43 patients such treated in a 2013 series, 4 patients (9 %) went on to develop re-erosion after a mean of 31.2 ± 4 months [8]. The main disadvantages of delayed band replacement are the additional requirement of a second operative procedure and the weight gain that occurs in the interval before replacement, a median of 3.7 BMI units at 6 months [20]. However, these concerns are offset by a much lower re-erosion rate compared to primary replacement.

Patients who only had an explant or were waiting for a band replacement continued to put on weight, amounting to 3.7 BMI units above their weight at diagnosis and occurring during a median period of 6.3 months after explant, and resulting in a net loss of only 5.7 BMI units (CI: 4–7.6) from initial BMI. However, the percentage of excess weight loss is not statistically different between those treated for erosion by explantation and band replacement and a corresponding cohort of patients who did not develop erosion [8].

17.9.3 Explantation with Conversion to Roux-En-Y Gastric Bypass

Suter et al. [19] reported a series of 11 patients with eroded bands who underwent either explantation and immediate conversion to RYGB (nine patients) or explantation followed by a staged RYGB (two patients). The mean resultant EWL was 65.1 % with almost 70 % of patients having EWL greater than 50 %. Perioperative morbidity consisted of one leak requiring reoperation and four wound infections, and is worse compared to one-stage revisions to RYGB for band failures other than erosions [36].

17.9.4 Explantation with Conversion to Sleeve Gastrectomy

The safety and feasibility of converting failed, but non-eroded, adjustable gastric bands to sleeve gastrectomy is well described [37]. The two-stage approach has morbidity similar to a primary sleeve gastrectomy (17 % vs. 11 %) with similar 2-year excess weight loss [38].

Park and co-authors [39] reported a series of nine patients undergoing revision of eroded bands to sleeve gastrectomy. Six of these were staged revision after a minimum of 3 months while three were revised to sleeve at the same time. While there was no mortality, there was

major morbidity with one stenosis and two proximal leaks, the latter requiring total gastrectomy. At 19 months follow-up, EWL was 86 % compared to the pre-banding weight. Whether lengthening the interval between explantation and conversion to sleeve results in reduction of major morbidity remains to be seen.

There are no studies comparing staged and synchronous revision of eroded bands to either sleeve gastrectomy or gastric bypass. Revision to either sleeve or gastric bypass results in similar morbidity and excess weight loss [40].

17.10 Conclusion

Erosions of adjustable gastric bands have become uncommon. Its presentation is insidious with a clinical course characterized by weight gain and loss of restriction and rarely by significant hematemesis. Upper endoscopy is the preferred diagnostic tool. Surgical explantation remains the preferred option with endoscopic explantation in selected patients. Staged band replacement allows resumption of weight loss with a low risk of re-erosion, while conversion to sleeve gastrectomy or gastric bypass results in effective weight loss. A staged approach may improve morbidity associated with the latter options.

References

1. Egberts K, Brown WA, O'Brien PE. Systematic review of erosion after laparoscopic adjustable gastric banding. Obes Surg. 2011;21:1272–9. doi:10.1007/s11695-011-0430-1.
2. Holt JB, Castiglione CL, Trowbridge PE. Immediate and long-term results of vertical banded gastroplasty for morbid obesity. Conn Med. 1987;51:638–42.
3. Asbun HJ, Alvarez A, Calabria RP, Jacobsen DC. Simplified perioperative management in vertical banded gastroplasty. Int Surg. 1992;77:248–50.
4. Naslund E, Backman L, Granstrom L, Stockeld D. Seven year results of vertical banded gastroplasty for morbid obesity. Eur J Surg. 1997;163:281–6.
5. Moreno P, et al. Band erosion in patients who have undergone vertical banded gastroplasty: incidence and technical solutions. Arch Surg. 1998;133:189–93.
6. Toppino M, et al. Outcome of vertical banded gastroplasty. Obes Surg. 1999;9:51–4.
7. Lattuada E, et al. Histologic study of tissue reaction to the gastric band: does it contribute to the problem of band erosion? Obes Surg. 2006;16:1155–9. doi:10.1381/096089206778392338.
8. Brown WA, et al. Erosions after laparoscopic adjustable gastric banding: diagnosis and management. Ann Surg. 2013;257:1047–52. doi:10.1097/SLA.0b013e31826bc21b.
9. Chapman AE, et al. Laparoscopic adjustable gastric banding in the treatment of obesity: a systematic literature review. Surgery. 2004;135:326–51. doi:10.1016/S0039.
10. Cherian PT, Goussous G, Ashori F, Sigurdsson A. Band erosion after laparoscopic gastric banding: a retrospective analysis of 865 patients over 5 years. Surg Endosc. 2010;24:2031–8. doi:10.1007/s00464-010-0899-z.
11. Ren CJ, Weiner M, Allen JW. Favorable early results of gastric banding for morbid obesity: the American experience. Surg Endosc. 2004;18:543–6. doi:10.1007/s00464-003-8931-1.
12. O'Brien PE, Dixon JB. Weight loss and early and late complications--the international experience. Am J Surg. 2002;184:42S–5.
13. Niville E, Dams A, Vlasselaers J. Lap-Band erosion: incidence and treatment. Obes Surg. 2001;11:744–7.
14. Sarker S, Herold K, Creech S, Shayani V. Early and late complications following laparoscopic adjustable gastric banding. Am Surg. 2004;70:146–9. discussion 149–150.
15. Dargent J. Laparoscopic surgery in morbid obesity: adjustable-ring gastroplasty. 320 cases. Ann Chir. 1999;53:467–71.
16. Silecchia G, et al. Laparoscopic adjustable silicone gastric banding: prospective evaluation of intragastric migration of the lap-band. Surg Laparosc Endosc Percutan Tech. 2001;11:229–34.
17. Di Lorenzo N, et al. Intragastric gastric band migration: erosion: an analysis of multicenter experience on 177 patients. Surg Endosc. 2013;27:1151–7. doi:10.1007/s00464-012-2566-z.
18. Chisholm J, Kitan N, Toouli J, Kow L. Gastric band erosion in 63 cases: endoscopic removal and rebanding evaluated. Obes Surg. 2011;21:1676–81. doi:10.1007/s11695-011-0468-0.
19. Suter M, Giusti V, Heraief E, Calmes JM. Band erosion after laparoscopic gastric banding: occurrence and results after conversion to Roux-en-Y gastric bypass. Obes Surg. 2004;14:381–6.
20. Thodiyil P, Anderson M, Dixon JB, O'Brien PE. Obesity Surgery Society of Australia and New Zealand, Conference (OSSANZ), New Zealand. 2005.
21. Aarts EO, et al. Intragastric band erosion: experiences with gastrointestinal endoscopic removal. World J Gastroenterol. 2015;21:1567–72. doi:10.3748/wjg.v21.i5.1567.
22. Torab FC, Hefny AF, Taha M, Abou-Rebyeh H, Abu-Zidan FM. Delayed life-threatening upper gastrointestinal bleeding as a complication of laparoscopic

adjustable gastric banding: case report and review of the literature. Asian J Surg. 2012;35:127–30. doi:10.1016/j.asjsur.2012.04.026.
23. Campos J, et al. Hypovolemic shock due to intragastric migration of an adjustable gastric band. Obes Surg. 2007;17:562–4.
24. Hainaux B, et al. Intragastric band erosion after laparoscopic adjustable gastric banding for morbid obesity: imaging characteristics of an underreported complication. AJR Am J Roentgenol. 2005;184:109–12.
25. Cintolo JA, Levine MS, Huang S, Dumon K. Intraluminal erosion of laparoscopic gastric band tubing into duodenum with recurrent port-site infections. J Laparoendosc Adv Surg Tech A. 2012;22:591–4. doi:10.1089/lap.2012.0132.
26. Khamaysi I, et al. Endoscopic removal of partially migrated intragastric bands following surgical gastroplasty: a prospective case series. Obes Surg. 2015;25:942–5. doi:10.1007/s11695-015-1629-3.
27. Regusci L, et al. Gastroscopic removal of an adjustable gastric band after partial intragastric migration. Obes Surg. 2003;13:281–4. doi:10.1381/096089203764467216.
28. Kedia P, Jacob B, DiMaio CJ. Mechanical lithotriptor-assisted endoscopic removal of an eroded gastric lap band. Gastrointest Endosc. 2015;81:1256–7. doi:10.1016/j.gie.2014.08.018.
29. Neto MP, et al. Endoscopic removal of eroded adjustable gastric band: lessons learned after 5 years and 78 cases. Surg Obes Relat Dis. 2010;6:423–7. doi:10.1016/j.soard.2009.09.016.
30. Mozzi E, et al. Treatment of band erosion: feasibility and safety of endoscopic band removal. Surg Endosc. 2011;25:3918–22. doi:10.1007/s00464-011-1820-0.
31. Campos JM, et al. Small erosion of adjustable gastric band: endoscopic removal through incision in gastric wall. Surg Laparosc Endosc Percutan Tech. 2010;20:e215–7. doi:10.1097/SLE.0b013e3181fec9eb.
32. Lattuada E, et al. Band erosion following gastric banding: how to treat it. Obes Surg. 2007;17:329–33.
33. O'Brien P. Comment on: endoscopic removal of eroded adjustable gastric band: lessons learned after 5 years and 78 cases. Surg Obes Relat Dis. 2010;6:427–8. doi:10.1016/j.soard.2009.11.001.
34. Niville E, Dams A, Van Der Speeten K, Verhelst H. Results of lap rebanding procedures after Lap-Band removal for band erosion -- a mid-term evaluation. Obes Surg. 2005;15:630–3. doi:10.1381/0960892053923860.
35. Vertruyen M, Paul G. 11-cm Lap-Band system placement after history of intragastric migration. Obes Surg. 2003;13:435–8.
36. Fournier P, et al. Laparoscopic Roux-en-Y gastric bypass for failed gastric banding: outcomes in 642 patients. Surg Obes Relat Dis. 2015. doi:10.1016/j.soard.2015.04.007.
37. Bernante P, et al. Feasibility of laparoscopic sleeve gastrectomy as a revision procedure for prior laparoscopic gastric banding. Obes Surg. 2006;16:1327–30. doi:10.1381/096089206778663797.
38. Silecchia G, et al. Laparoscopic sleeve gastrectomy as a revisional procedure for failed laparoscopic gastric banding with a "2-step approach": a multicenter study. Surg Obes Relat Dis. 2014;10:626–31. doi:10.1016/j.soard.2013.10.017.
39. Park YH, Kim SM. Laparoscopic sleeve gastrectomy as revisional surgery for adjustable gastric band erosion. J Laparoendosc Adv Surg Tech A. 2014;24:593–600. doi:10.1089/lap.2013.0584.
40. Gonzalez-Heredia R, et al. Revisions after failed gastric band: sleeve gastrectomy and Roux-en-Y gastric bypass. Surg Endosc. 2015;29:2533–7. doi:10.1007/s00464-014-3995-7.

18 Vertical Banded Gastroplasty: Evaluation and Management of Complications

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18.1 Introduction

Bariatric surgery is associated with a significant reduction of weight and associated comorbid conditions resulting in improved quality and prolongation of life. Even though the mortality and morbidity associated with bariatric operations has decreased over time, more complex bariatric operations are associated with increased incidence of complications compared to simpler operations. Therefore, surgical innovators have been in constant search of operations that will improve the health and well-being of the patients without an increase in surgery related complications. In the post jejunoileal bypass era, various forms of gastroplasties and gastric bypasses were designed. One such operation was called the vertical banded gastroplasty (VBG) and was described by Mason in 1982 [1]. In this operation, a vertical pouch was created along the lesser curvature of the stomach using a non-cutting stapler. This resulted in a stomach that was partitioned without actually dividing it and had a capacity of 50 ml. No bowel was bypassed and the stomach was not transected. Thus, it was thought to be a simpler bariatric operation that was also fast and technically easier to perform and with fewer associated operative complications than the Roux-en-Y gastric bypass (RYGB). Postoperative malabsorption, dumping, and marginal ulceration were also avoided.

The original procedure consisted of creating a non-transected 50 ml pouch around a 32 French Ewald tube. A band of Marlex mesh was also placed around the pouch outlet with the hope of providing long-lasting weight loss from a fixed stoma. At the time, one of the prevailing beliefs regarding weight regain after gastroplasty or RYGB was stoma dilation, and the hope was that the Marlex mesh would not allow the gastric outlet in a VBG to dilate with time. However, the Mason VBG had an undivided stomach, which was prone to staple line breakdown resulting in a gastro-gastric fistula and weight regain. It was also performed by laparotomy with the associated wound-related complications such as ventral hernia. In 1993, MacLean et al. [2] described division of the stomach to overcome the problem of staple line breakdown and in 1994 Hess et al. [3] reported a laparoscopic approach. With the advent of the laparoscopic adjustable gastric band (LAGB), the frequency of this once very popular purely restrictive procedure declined considerably and currently very few VBGs are performed.

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_18

However, in a relatively recent study, Scozzari et al. [4] reported favorable results of the laparoscopic MacLean type VBG and found that the excess weight loss (EWL) percentages at 3, 5, and 10 years were 65.0 %, 59.9 %, and 59.8 %, respectively. The resolution and/or improvement rate for comorbidities were 47.5 % for hypertension, 55.6 % for diabetes, 75 % for sleep apnea, and 47.4 % for arthritis. Mean Moorehead-Ardelt Quality of Life Questionnaire and BAROS values were 1.4 and 3.8, respectively. Intraoperative complication rate and conversion rate were each 0.9 %. Early postoperative complication rate was 4.2 % and early reoperation rate was 0.5 %. They had no mortality. Late complications occurred in 14.7 % of patients, and 10.0 % of the patients underwent revisional surgery. Most reoperations were for weight regain, though other causes included severe dysphagia, outlet stricture unresponsive to endoscopic dilation, severe reflux, and gastro-gastric fistula. The rate of revision surgery increased during the follow-up period to 10 % at 10 years. Of note, their patients were highly selected as sweet-eaters and patients with a hiatal hernia over 3 cm were excluded [4].

Bekheit et al. [5] have also reported excellent weight loss results in a cohort of 150 patients with a mean preoperative BMI of 47 ± 8.4 kg/m^2, as 60 % of patients achieved >50 % EWL with a low rate of revision to other operations (<5 %). Their late complications such as staple line dehiscence, stomal stenosis, and mesh erosion rates were also low. However, other authors have described significantly lower weight loss after VBG compared to RYGB.

In a prospective randomized trial comparing VBG to RYGB, Sugerman et al. [6] enrolled 20 patients in each treatment arm and found that at 3 years post surgery, VBG patients had 37 ± 20 % EWL compared to 64 ± 19 % EWL for patients who had RYGB. The difference was even greater for sweet eaters who underwent a VBG compared to those who underwent a RYGB. The study was closed at 9 months because weight loss strongly favored the RYGB.

In another study by Marsk et al. [7], 21 % of patients who underwent VBG required reoperation. Reasons included: staple line disruption, vomiting or food intolerance, inadequate weight loss, band erosion, and an enlarged opening. They also described early complications including leak, bleeding, and pulmonary embolism, but VBG operations are rarely practiced these days and therefore most surgeons are unlikely to encounter these early postoperative complications. Nonetheless, because of the popularity of the VBG in the 1980s and 1990s, there are many surviving patients with a previous VBG and surgeons will still encounter patients suffering from one or more of the long-term complications. Several other authors have reported poor weight loss and high long-term complication rates after VBG [8, 9].

18.2 Management of Complications

The common late complications of VBG include food intolerance that is manifested by nausea and vomiting, gastroesophageal reflux disease, failure to lose adequate weight or weight regain and occasionally excess weight loss. Many of the symptoms are related to maladaptive eating habits, gastric outlet obstruction, staple line dehiscence, or band erosion. Nausea and vomiting may also be due to bowel obstruction from incarcerated bowel in a ventral hernia, if the initial access was obtained via laparotomy.

Management of all of these VBG-related complications requires a detailed history and investigations to clarify the nature of the complication as well as the suitability of the patient to undergo a reoperation. A dietary history including the quantity and type of food consumed is important, as is obtaining a psychological evaluation. An esophagogastroduodenoscopy (EGD) [10] and upper gastrointestinal X-ray series with contrast is routine. In addition, pH studies and esophageal manometry may be indicated based on the nature of the symptoms. Plain abdominal X-ray series or computerized tomography may also be needed if a bowel obstruction is suspected. Overall, complication rates of revising a VBG are high and the operation should not be taken lightly by either the surgeon or the patient [11].

18.2.1 Nausea and Vomiting

Dietary intolerance may be a result of maladaptive eating patterns, esophageal dysmotility, gastric outlet obstruction, or a bowel obstruction. The investigations mentioned above will help identify the cause. Patients with chronic nausea and vomiting may also suffer from nutritional deficiencies as well as electrolyte abnormalities. These should be identified and corrected. Particular care should be taken to supplement thiamine prior to placing the nutritionally depleted patient on dextrose-rich solutions in order to prevent Wernicke's encephalopathy.

While maladaptive eating behavior may respond to nutritional counseling, often anatomic or physiologic abnormalities will need surgical correction. Gastric outlet obstruction because of the Marlex mesh or the Silastic band is unlikely to respond to balloon dilation. In such instances, conversion to another bariatric operation may be considered. Occasionally if the patient has severe malnutrition, a reversal may be indicated.

18.2.2 Gastroesophageal Reflux Disease

As a result of maladaptive eating behavior, gastric outlet obstruction, or a hiatal hernia, patients may present with severe gastroesophageal reflux disease (GERD) and may have severe esophagitis. GERD should be differentiated from nausea and vomiting. Dietary counseling associated with the use of proton pump inhibitors would be first line treatment. Diagnosing and treating an *H. pylori* infection should also be done. Manometry to rule out esophageal dysmotility and pH testing to quantify the degree of acid reflux will also help in planning a revision operation.

If the problem is primarily related to outlet obstruction, any of the current bariatric operations that relieves outflow problems will also relieve the symptoms and help with further weight loss. Concomitant repair of the hiatal hernia, if present, should also be carried out. In most instances, severe GERD is best treated by a conversion to a RYGB. In a small series of eight patients with quantifiable severe reflux, VBG conversion to RYGB resulted in near-normalization of acid reflux parameters with a decrease in DeMeester score from 58.1 to 15.9, total time with pH < 4.0 was reduced from 18.4 to 3.3 % and need for proton pump inhibitor medication was eliminated [12].

18.2.3 Weight Regain

Weight regain in a patient with a VBG could either be related to maladaptive eating habits or disruption of the staple line. In the case of staple line disruption, the patient may report weight regain after initially good results. A history of being able to eat larger quantities of food after the initial period of restriction is common. Staple line disruption can be demonstrated by performing an upper gastrointestinal series with barium as well as endoscopy. If there is no staple line disruption, and poor eating behaviors are identified, a dietary consult and re-engagement in physical activity should be the first-line approach.

A variety of surgical approaches have been described to address staple line disruption including redoing the VBG, adding an adjustable band, conversion to a sleeve gastrectomy, RYGB, biliopancreatic diversion or a biliopancreatic diversion with duodenal switch.

18.2.4 Conversion of VBG

In comparing patients who underwent restoration for failed VBG to another VBG, Marsk et al. [7] reported that out of 104 patients needing revision, 31 underwent repeat VBG, of whom ten needed a secondary revision procedure. In contrast, 49 patients who underwent conversion to a RYGB, required no additional revision surgery.

In another study, Van Gemert et al. [13] estimated that 56 % of the patients who received an initial VBG would require revision after a 12-year period compared to 12 % after initial RYGB. Of the patients who underwent a re-VBG, 68 % would require further revision surgery over a

5-year period with no further revision surgery after conversion to a RYGB. The re-VBG series included patients in whom a new staple line was created with or without transection, or a new band was applied around the stoma, or the band size was corrected. Therefore, restoration of VBG is not recommended, as conversion to another bariatric procedure has demonstrated superior results.

Gonzalez et al. [14] have described five key steps in converting a VBG to RYGB. These include: identification of the band; delineation of the extent of the pouch; division of the stomach; preparation of the Roux limb; and completion of the gastro-jejunostomy. When converting to a RYGB, care must be taken to not leave behind a blind stomach pouch. These revision operations can be successfully performed laparoscopically and the stapled partition can be excised or taken down using a linear stapler through a gastrotomy along the greater curvature. Endoscopic visualization or placing ports within the stomach to directly visualize the staple line and then dividing it has also been described.

Adding a laparoscopic adjustable gastric band (LAGB) to a failed VBG has been described [15]. However, due to the overall decline in popularity of the LAGB, this approach should only be considered very selectively and may be considered for patients who had good weight loss with VBG and were satisfied with their quality of life, but weight regain occurred due to staple line dehiscence.

With increasing popularity of the sleeve gastrectomy (SG), failed VBGs have also been converted to SG [16, 17]. Although Berende [16] reported satisfactory weight loss and resolution of comorbid conditions after conversion to a SG, they also reported high complication rates, including bleeding and leaks, and cautioned that such operations should only be performed in tertiary care centers.

Conversion to duodenal switch (DS) has been described and involves taking down the staple line using a linear cutter stapler through a gastrotomy at the greater curvature and then performing a SG. After the SG is completed, the duodeno-ileostomy and the ileo-ileostomy are performed in standard fashion. In a series of 16 patients who underwent open conversion from a VBG to a DS, Keshishian et al. [18] reported good comorbidity resolution and very satisfactory weight loss. However, patients who underwent conversion to DS were susceptible to leaks along the gastrogastrostomy [19]. A minimally invasive approach for converting the VBG to a DS has also been described [20].

Conversion to biliopancreatic diversion (BPD) may offer some unique advantages. In this operation, if the stapled partition has failed, the chance of creating a blind pouch is low and there may be no need to excise the mesh or band around the gastric outlet, if badly scarred. In a series of 10 patients who underwent conversion from VBG to a BPD, median EWL was excellent at 55 % and perioperative morbidity and mortality was low [21].

Complication rates after conversion of VBG are quite high and therefore should be performed by experienced surgeons and with appropriate informed consent. Apers et al. [22] converted 107 patients laparoscopically to a RYGB of which 21 had a prior VBG and the remaining had a prior LAGB. Conversion rate from laparoscopy to open surgery was 38 % for the VBG group. Early complications occurred in 24 % and included pouch leakage, torsion of the alimentary limb that required revision of the anastomosis, stomach remnant leakage re-bleeding, pulmonary embolism, and sepsis/bilateral pneumonia. The reoperation rate for the VBG subgroup was 36 %. In another study in which 23 VBG patients were converted to a RYGB, good weight loss was achieved, but the overall complication rate was 43.5 % including a mortality. Major complications included anastomotic leak (8.7 %), incisional hernia (13 %), fistula (8.7 %), respiratory failure (8.7 %), and perforation (4.4 %) [23].

18.2.5 Band Erosion

Erosion of mesh or a silastic band is often detected on endoscopy and endoscopic extraction techniques have been described by Karmali et al. [24]. In a series of nine patients, the dual channel

scope and conventional endoscopic scissors were used to retrieve the bands per os and patients were discharged the same day. For a partially eroded band, stents can sometimes facilitate complete erosion and subsequent endoscopic retrieval. Such techniques are preferable to open or laparoscopic approaches due to the dense scarring of the mesh to the stomach and liver.

18.3 Summary

Patients who have undergone VBG in the past may still present to bariatric practices for complications such as nausea, vomiting gastroesophageal reflux disease inadequate or excess weight loss and band erosion. A careful history and physical examination supplemented by additional endoscopic and radiologic investigations can help elicit the etiology of the symptoms. Several operative approaches have been described and should be thoughtfully considered as the complication rate associated with these operations is not trivial. In carefully selected patients who have undergone appropriate nutritional education, revision to other bariatric operations is often accompanied by resolution of the symptoms.

References

1. Mason EE. Vertical banded gastroplasty for obesity. Arch Surg. 1982;117(5):701–6.
2. MacLean LD, Rhode BM, Forse RA. A gastroplasty that avoids stapling in continuity. Surgery. 1993;113(4):380–8.
3. Hess DW, Hess DS. Laparoscopic vertical banded gastroplasty with complete transection of the staple-line. Obes Surg. 1994;4(1):44–6.
4. Scozzari G, Toppino M, Famiglietti F, Bonnet G, Morino M. 10-year follow-up of laparoscopic vertical banded gastroplasty: good results in selected patients. Ann Surg. 2010;252(5):831–9.
5. Bekheit M, Katri K, Salam WN, Ezzat T, El Kayal el S. Rejecting the demise of vertical-banded gastroplasty: a long-term single-institute experience. Obes Surg. 2013;23(10):1604–10.
6. Sugerman HJ, Starkey JV, Birkenhauer R. A randomized prospective trial of gastric bypass versus vertical banded gastroplasty for morbid obesity and their effects on sweets versus non-sweets eaters. Ann Surg. 1987;205(6):613–24.
7. Marsk R, Jonas E, Gartzios H, Stockeld D, Granstrom L, Freedman J. High revision rates after laparoscopic vertical banded gastroplasty. Surg Obes Relat Dis. 2009;5(1):94–8.
8. Baltasar A, Bou R, Arlandis F, Martinez R, Serra C, Bengochea M, et al. Vertical banded gastroplasty at more than 5 years. Obes Surg. 1998;8(1):29–34.
9. del Amo DA, Diez MM, Guedea ME, Diago VA. Vertical banded gastroplasty: is it a durable operation for morbid obesity? Obes Surg. 2004;14(4):536–8.
10. Clapp B, Yu S, Sands T, Wilson E, Scarborough T. Preoperative upper endoscopy is useful before revisional bariatric surgery. JSLS. 2007;11(1):94–6.
11. Cariani S, Nottola D, Grani S, Vittimberga G, Lucchi A, Amenta E. Complications after gastroplasty and gastric bypass as a primary operation and as a reoperation. Obes Surg. 2001;11(4):487–90.
12. Ekelund M, Oberg S, Peterli R, Frederiksen SG, Hedenbro JL. Gastroesophageal reflux after vertical banded gastroplasty is alleviated by conversion to gastric bypass. Obes Surg. 2012;22(6):851–4.
13. van Gemert WG, van Wersch MM, Greve JW, Soeters PB. Revisional surgery after failed vertical banded gastroplasty: restoration of vertical banded gastroplasty or conversion to gastric bypass. Obes Surg. 1998;8(1):21–8.
14. Gonzalez R, Gallagher SF, Haines K, Murr MM. Operative technique for converting a failed vertical banded gastroplasty to Roux-en-Y gastric bypass. J Am Coll Surg. 2005;201(3):366–74.
15. Dargent J. Lap banding as a redo surgery: "restriction over restriction" may be a relevant bariatric strategy. Obes Surg. 2009;19(9):1243–9.
16. Berende CA, de Zoete JP, Smulders JF, Nienhuijs SW. Laparoscopic sleeve gastrectomy feasible for bariatric revision surgery. Obes Surg. 2012;22(2):330–4.
17. Iannelli A, Schneck AS, Ragot E, Liagre A, Anduze Y, Msika S, et al. Laparoscopic sleeve gastrectomy as revisional procedure for failed gastric banding and vertical banded gastroplasty. Obes Surg. 2009;19(9):1216–20.
18. Keshishian A, Zahriya K, Hartoonian T, Ayagian C. Duodenal switch is a safe operation for patients who have failed other bariatric operations. Obes Surg. 2004;14(9):1187–92.
19. Greenbaum DF, Wasser SH, Riley T, Juengert T, Hubler J, Angel K. Duodenal switch with omentopexy and feeding jejunostomy--a safe and effective revisional operation for failed previous weight loss surgery. Surg Obes Relat Dis. 2011;7(2):213–8.
20. Jain-Spangler K, Portenier D, Torquati A, Sudan R. Conversion of vertical banded gastroplasty to stand-alone sleeve gastrectomy or biliopancreatic diversion with duodenal switch. J Gastrointest Surg. 2013;17(4):805–8.
21. Daskalakis M, Scheffel O, Theodoridou S, Weiner RA. Conversion of failed vertical banded gastroplasty to biliopancreatic diversion, a wise option. Obes Surg. 2009;19(12):1617–23.

22. Apers JA, Wens C, van Vlodrop V, Michiels M, Ceulemans R, van Daele G, et al. Perioperative outcomes of revisional laparoscopic gastric bypass after failed adjustable gastric banding and after vertical banded gastroplasty: experience with 107 cases and subgroup analysis. Surg Endosc. 2013;27(2):558–64.
23. Bolton J, Gill RS, Al-Jahdali A, Byrns S, Shi X, Birch DW, et al. Endoscopic revision (StomaphyX) versus formal surgical revision (gastric bypass) for failed vertical band gastroplasty. J Obes. 2013;2013:108507.
24. Karmali S, Snyder B, Wilson EB, Timberlake MD, Sherman V. Endoscopic management of eroded prosthesis in vertical banded gastroplasty patients. Surg Endosc. 2010;24(1):98–102.

Inadequate Weight Loss after Gastric Bypass and Sleeve Gastrectomy

19

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Key Points

- Obesity is a chronic disease and initial surgical treatment will not provide durable results in some patients. Corrective and conversion procedures should be offered to appropriate patients with inadequate weight loss or weight regain after bariatric surgery.
- There is a large amount of data regarding reoperative bariatric surgery but it generally consists of single institution, retrospective studies. Large prospective studies or national data registries may help to define the role of reoperative bariatric surgery for inadequate weight loss.
- Weight regain can occur after every bariatric procedure. The causes for inadequate weight loss or weight regain are often multifactorial. Anatomic, behavioral, psychological, and medical factors should be evaluated to determine candidates for reoperative bariatric surgery.
- Weight regain after sleeve gastrectomy can be effectively treated with conversion to gastric bypass or duodenal switch.
- Weight regain after gastric bypass poses a more difficult challenge, but selected patients can benefit from pouch or anastomotic revisions or limb lengthening procedures.

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19.1 Gastric Bypass

19.1.1 Epidemiology and Etiology

It is estimated that 5–15 % of the patients fail to lose an adequate amount of weight after gastric bypass, despite precise surgical technique and regular follow-up [1, 2]. Several factors associated with inadequate weight loss (IWL) include older age [3, 4], black race [5, 6], male sex [7], being married [5], greater initial weight and BMI [3, 5, 7], diabetes mellitus [3, 7, 8], other obesity associated diseases [9], physical inactivity after surgery [10], larger gastric pouch [11], poor follow-up after surgery [12, 13], and insurance status [12].

In a study by Campos et al. [14] where 310 of 361 patients had follow-up data at 12 months, greater initial BMI, presence of diabetes, open surgical approach and larger pouch size were associated with IWL (defined as EWL ≤ 40 % [7, 14]) on univariate analysis. Association of black race with IWL nearly achieved statistical significance ($p = .06$) on univariate analysis. However, on multivariate analysis only two variables, presence of diabetes and larger pouch size, were independently associated with IWL. In

D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_19

addition, the use of insulin replacement to control diabetes had a stronger association with IWL [14].

Inadequate weight loss in patients with type 2 diabetes (T2DM) has several potential causes. Most patients with type 2 diabetes mellitus use exogenous insulin and/or oral hypoglycemic agents which increase circulating levels and/or sensitivity to insulin. This may result in less weight loss due to the anabolic effects of insulin that promote lipogenesis, stimulation of triglyceride synthesis, adipocyte differentiation, and muscle synthesis [15–17]. Other causes of weight gain in patients with diabetes include a defensive increase in caloric intake to manage hypoglycemic episodes, reduction of urinary glucose losses, and a direct effect of insulin on the distal renal tubule resulting in sodium and water retention [17–24].

Another important aspect of gastric bypass is the degree of gastric restriction offered by the small pouch size. While the issue is still somewhat controversial, some authors have shown an inverse correlation between initial size of the gastric pouch and EWL [11, 14]. Seventy percent of surgeons in North America create a gastric pouch by measuring distance from the estimated location of the gastroesophageal junction to a variable distance in the lesser curvature of the stomach, and/or by the number of vessels in the lesser curvature; only about one-fifth use a sizing balloon [25]. This technical variability, along with variations in patient anatomy, may result in variable pouch sizes that could potentially affect long-term weight loss.

In other studies, greater initial weight and BMI have been associated with IWL after gastric bypass [3, 5, 7, 26]. In a study by Ma et al., 377 of 494 patients completed 12 months follow-up and initial weight and BMI were inversely associated with percentage of weight loss ($p < .001$) [3]. Factors that may be responsible for this finding in various studies are higher rate of obesity-associated diseases with greater weight and BMI and the issue of using excess weight loss as a measure of success, particularly as it applies to patients with higher BMI [14].

Black race has been associated with IWL in various studies [5, 6, 27]. Responsible factors that may explain IWL in black patients are: variation in body composition, fat distribution, resting energy expenditure, and thermogenesis [28]. A higher prevalence of diabetes [29], the cultural and social environment [30], and the definition of ideal body weight that possibly underestimates ideal body weight in black patients may also contribute to these findings [30].

19.1.2 Medical Management

Inadequate weight loss or weight regain are common indications for revisional surgery after bariatric surgery. Adequate weight loss has been consistently associated with behavioral and diet modifications in addition to the surgery, and a committed exercise regimen. It is imperative to evaluate eating habits and exercise routines prior to committing to reoperative bariatric surgery. A medical and behavioral evaluation within a multidisciplinary bariatric program is essential. This should include a referral for psychological and nutritional evaluation to identify an underlying psychiatric condition and/or a maladaptive eating behavior. Active involvement in bariatric support groups can be beneficial as well [31].

There are currently several medications approved for a weight loss indication in the USA including lorcaserin, naltrexone/buproprion, phentermine/topirimate, liraglutide, and orlistat [32]. While there are few data at this point regarding their use after bariatric surgery, they may prove to be a valuable adjunctive treatment for the post-bariatric patient who is struggling to maintain or achieve their desired weight loss.

19.1.3 Surgical Management

As with any chronic medical or surgical therapy used in the treatment of a chronic disease, some patients who undergo bariatric surgery will be complete responders, some will be partial responders or nonresponders, and some will have disease recurrence after initial therapeutic success. Viewing the treatment of obesity with

bariatric surgery in this way is consistent with every other chronic disease paradigm but there is still a clear bias against this way of thinking as evidenced by "one per lifetime" insurance policies for bariatric surgery or lack of coverage for additional treatment or surgical therapy after weight regain. Based on the current evidence, it is clear that reoperative bariatric surgery can be beneficial in carefully selected patients (Table 19.1) [33]. However, these patients must be thoroughly evaluated by a multidisciplinary program in order to assess the cause for their poor response to their primary gastric bypass surgery. Indications for corrective surgery after gastric bypass include inadequate weight loss, weight regain, or recurrence of weight-related comorbid conditions [33]. Evaluating the post-gastric bypass anatomy with endoscopy and contrast studies plays an important role in determining the optimal revisional procedure [34]. Whenever possible, it is also important to obtain previous operative notes to identify the limb lengths and positions as part of the surgical planning.

Endoscopic management to augment gastric restriction by reducing the pouch and gastrojejunal stomal size is a safe corrective procedure, and has been shown not only to arrest weight gain [35], but also attain short-term weight loss [36–38]. However, the published studies are mostly small non-controlled series and numerous devices utilized for this approach are commercially unavailable.

Indications for surgical revision of the pouch or gastrojejunostomy include significant pouch or stoma dilatation (Fig. 19.1), presence of gastrogastric fistula with inadequate weight loss or persistence of marginal ulceration [39, 40]. Various definitions have been used to define a "dilated" or large gastric pouch or gastrojejunostomy. While it is still unclear what the definition should be, in our practice we define a pouch larger than 5×5 cm or containing a large amount of fundus to be enlarged. We consider a gastrojejunostomy more than 2 cm in greatest diameter to be large as well, but these are arbitrary cutoffs and need to be placed in the context of the patient's clinical course and overall evaluation. Since anatomic evaluations are not routinely performed for patients who are maintaining their weight loss, it is unclear how many patients with a "large" pouch or stoma by these criteria are able to achieve long-term success and this needs further study.

Another corrective option is surgical placement of an adjustable or nonadjustable band around a gastric pouch to add additional gastric restriction [41–43]. While this has been shown to be a safe option, the utility of this type of adjunctive treatment is not clear. Like primary gastric banding procedures, there is considerable variability in reported outcomes utilizing the adjustable gastric band for additional weight loss after gastric bypass.

Other corrective surgical options include lengthening of the biliopancreatic limb to increase the malabsorptive component, or lengthening of the Roux limb to increase the bypass component. Duodenal switch as a conversion procedure for patients with inadequate weight loss after gastric bypass has been reported but is technically challenging and not widely accepted due to the risk associated with this conversion procedure. Currently, there are only feasibility data in the literature regarding this approach and no data regarding the long-term risks and benefits [44].

Improved weight loss after reoperative surgery has been reported by many authors, but the current evidence to support these strategies is limited to mostly single institution retrospective case series [45]. The lack of prospective data and the heterogeneity of the published data for revisional bariatric surgery can be partially attributed to the difficulties in getting access to care for these patients. Since many patients do not have coverage for revisional bariatric procedures or have limited options for reoperative surgery, there are relatively few large study cohorts in the literature. This is in stark contrast to available coverage for reoperative surgery provided by major national plans and state employee health plans for other surgical specialties (orthopedics, cardiac surgery) [46].

Table 19.1 Selected papers reporting gastric bypass conversions for inadequate weight loss

Author	*N*	Primary procedure (s)	Revisional procedure (s)	Follow-up duration (range)	Complications	Leaks	30-day mortality	Preoperative BMI (at primary procedure)	Pre-revision BMI	Pre-revision weight loss	Post-revision weight loss	Interval from primary operation–revision
Thompson, et al. (35) 2013	TORe ($n=50$) or sham procedure ($n=27$)	RYGB	Endoscopic sutured transoral outlet reduction (TORe)	6 months	1 pulmonary edema immediately post-procedure		0		37.6 ± 4.9 in –TORe group 38.6 ± 6.2 in control group	73.2 ± 20.5 in –TORe group ($n=50$) 73.7 ± 21.5 in control group ($n=26$)	15.9 ± 20.90 in TORe group ($n=43$) 7.7 ± 20.18 in Control group ($n=26$)	58.8 ± 25.7 months ($n=48$) in TORe group 67.5 ± 24.5 ($n=27$) in control group
Leitman, et al. (36) 2010	64	RYGB	Endoscopic plication and revision of the gastric pouch (EPRGP)	5.8 (3–12) months	2 (3 %) intraoperative complications (equipment failure), 1 observed for bleed (no transfusion)		0	48.5	39.5	nadir BMI 31	7.3 kg (0–31)	5 years
Himpens, et al. (37) 2012	88	LRYGB (with and without prior VBG or AGB)	Distal RYGB, Fobi ring around pouch, bypass reconstruction, LSG, plication	48 (18–122) months	Overall reoperation rate: 7.3 %, overall severe complication rate: 20.7 %, overall leak rate 12.1 %	12.10 %	0	42.7 ± 19.7 (33.0–56.6)	39.1 ± 11.3 (30.8–51.8)	12.4 ± 9.3 % (−1.0–29.1)	Post-revision BMI 29.6 ± 12.4 (18.0–45.5)	3.0 years (1.5–8.0)
Irani, et al. (43) 2011	43	RYGB	Salvage banding	26 ± 14 (6–66) months	12 adverse events: 1 enterotomy requiring band removal; 1 SBO, 1 GI bleed, 3 esophageal dilations resolved with band deflation, 1 minor port leak, 1 port flip, 1 band slip, 1 case of persistent dysphagia, and 2 cases of intragastric band migration		0	50.4 (35–60)	43.3 (34–60)	17 % EWL	Post-LAGB BMI: 33.8 (25–47); 38 % EWL from LAGB; 55 % cumulative (initial + revisional) EWL	223 ± 154 months

Author	*N*	Primary procedure (s)	Revisional procedure (s)	Follow-up duration (range)	Complications	Leaks	30-day mortality	Preoperative BMI (at primary procedure)	Pre-revision BMI	Pre-revision weight loss	Post-revision weight loss	Interval from primary operation–revision
Rawlins et al. (45) 2011	29	RYGB	distal RYGB	1–5 years	Short-term: 0 leaks, 4 DVTs, 10 SSIs; Long-term: 1 partial SBO, 6 ventral incisional hernias, 9 w/ albumin <3, 6 required TPN, 1 reversed	0	0	57.9 (38–81)	48.1 (35–67)	26.6 % (0–46 %) EWL	60.9 % (39–83 %) EWL at 1 year; 68.8 % (53–91 %) EWL at 5 years	
Parikh et al. (59) 2007	12	RYGB	BPD-DS	11 (2–37) months	6 (4 strictures, 1 metabolic acidosis, 1 wound complication)	0	0	53.9 (40.7–66.0)	40.7 (33.2–46.0)	42 % (8–63 %) EWL; lowest BMI after primary RYGB: 31.6 (23.3–39.0)	62.7 % (18.8–96.2 %) EWL at 11 months 79.4 % (48.3–98.1 %) overall	NR
Dapri et al. (60) 2011	4	RYGB	LSG	11 ± 12.8 months	1 GG fistula	NR	0	43.2 ± 8	37.3 ± 6.6	27.5 ± 11.8 % EWL; 26.5 ± 12 % EBMIL	59.3 ± 31.5 % EWL; 42.3 ± 34.5 % EBMIL	36.7 ± 15.6 months

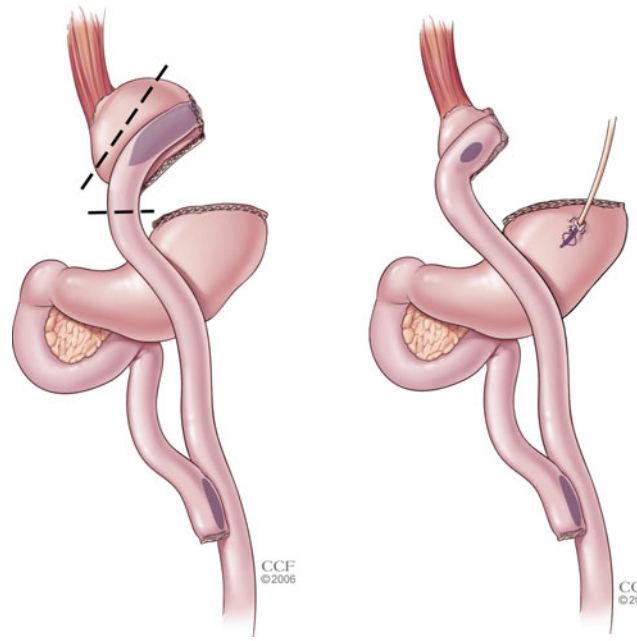

Fig. 19.1 Revision of a large gastric pouch or dilated gastrojejunostomy for weight gain after gastric bypass can be achieved with resection of the gastrojejunal complex and dilated pouch. A new, smaller gastrojejunostomy is then created. A gastrostomy tube can be placed in the gastric remnant at the surgeon's discretion

19.2 Sleeve Gastrectomy

19.2.1 Epidemiology and Etiology

Laparoscopic sleeve gastrectomy is still relatively new to bariatric surgery and has been widely utilized as a primary procedure for about 10 years. There is a growing body of long-term weight loss data in the literature but the true incidence of inadequate weight loss or weight regain after LSG is still not clear. In a study by Cesana et al., about 5 % of LSG patients required reoperation with a mean follow-up 21.1 ± 9.7 months (range 6–57 months) [47]. In another study, weight regain of 10 kg from nadir was observed in 19.2 %, i.e., in 5 of the 26 patients during the 5 year follow-up. In the weight regain group, the first year %EWL was comparable to the adequate weight loss group, however the %EWL significantly decreased by the second year in the weight regain group [48]. Like RYGB, the etiology of weight regain after LSG is multifactorial and likely involves anatomic, behavioral, socioeconomic, and psychological components. There are currently few published data that can help identify the right patient for the right bariatric operation, so bariatric surgeons rely on experience, clinical judgment, and patient preference to drive these decisions.

Dilation of the gastric lumen, particularly the gastric fundus, is a common imaging finding in patients with weight regain after LSG. This may be attributed to a lack of adequate calibration at the time of the primary procedure or a natural process of stomach tissue to dilate and become more compliant over time. Patients' behavioral issues, eating habits, and lack of adherence to the post-surgical diet recommendations may also contribute to this problem [47].

19.2.2 Medical Management

As with RYGB, some patients may benefit from continued medical therapy after LSG. While hunger often disappears for several months after LSG, it inevitably returns and some patients may benefit from medication to control appetite long-term. With a variety of FDA-approved medication for the treatment of obesity available in the USA, these may play an important adjunctive role in the long-term management of some sleeve gastrectomy

patients. Further research is necessary to better define the role of medical therapy for patients with IWL or weight regain after sleeve.

19.2.3 Surgical Management

A subset of patients with IWL after sleeve gastrectomy may benefit from additional surgical therapy if their weight loss or comorbidity improvement is suboptimal. In a recently published report by Sieber et al., 8 of 68 patients (11.8 %) underwent reoperative surgery due to IWL after sleeve gastrectomy [49]. However, similar to any bariatric procedure, the patient must be evaluated by a multidisciplinary team to determine the cause of weight regain. Surgical options include placement of an adjustable band over the proximal sleeve, re-sleeve gastrectomy (corrective), or conversion to gastric bypass or duodenal switch.

In one study of patients who had a LSG over a 60 French Bougie, placement of an adjustable band due to inadequate weight loss after sleeve gastrectomy resulted in a 78 lb weight loss within 9 months, corresponding to an EWL of 57 % [50]. Overall, though, there are not strong data to support this approach and it is not commonly used.

In a study from Italy, 11 of 201 patients (5.4 %) who regained weight after laparoscopic sleeve gastrectomy underwent laparoscopic re-sleeve gastrectomy with a significant decrease in mean BMI and increase in mean percentage of EWL at 1 year follow-up [47]. Rebibo et al. compared 15 patients who underwent re-sleeve gastrectomy to 30 patients who underwent primary sleeve gastrectomy, and the leak rate for the former group was 13 % (2/15), with less weight loss [51]. Dapri et al. reported a leak in one of seven patients who underwent re-sleeve gastrectomy [52]. However, two series of patients who underwent re-sleeve gastrectomy report no post-procedural leaks [53, 54]. This approach is typically reserved for patients with a dilated sleeve or fundus who refuse conversion to a bypass procedure.

In a study from Austria, 8 out of 73 patients underwent conversion procedure from a laparoscopic sleeve gastrectomy to laparoscopic Roux-en-y gastric bypass (five of the eight were for weight regain). None of these five patients were found to have significant sleeve dilatation. After conversion, a mean weight reduction of 15.2 ± 8 kg (range, 6–25 kg) was achieved within a follow-up from 1 to 52 months [55]. In a group of high risk, high BMI patients, Cottam et al. showed that a second stage RYGB can result in continued weight loss after LSG. One hundred twenty-six patients with mean BMI of 65 underwent LSG with an overall EWL of 46 % at 1 year. Thirty-six patients underwent a conversion procedure to RYGB 1 year after the LSG. That subgroup of patients had a mean BMI of 49 at the time of the conversion and this decreased to a mean BMI of 39 six months after conversion to RYGB with continued improvement in comorbidity status [56]. This study demonstrated the utility of LSG as a risk management strategy in high BMI patients. On the other hand, there will be a subset of these patients who can maintain long-term weight loss after LSG. In a long-term follow-up study of the same patient group, Eid et al. showed that 69 of those patients who did not return for the second stage bypass procedure were able to maintain 48 % EWL and good comorbidity improvement 6–8 years after LSG [57].

These studies highlight why the sleeve gastrectomy has become so popular: It is an effective primary operation but leaves the surgeon several safe and effective options for conversion for patients who do not achieve sufficient weight loss or have weight regain over time.

In a study by Carmeli et al., 19 patients underwent a conversion procedure after sleeve gastrectomy due to IWL (nine underwent duodenal switch and ten underwent gastric bypass). Duodenal switch yields a greater weight loss than gastric bypass, but both are feasible and effective conversion procedures after failed sleeve gastrectomy [58]. The two major advantages of duodenal switch as the conversion procedure are the avoidance of entrance into the area of scarred stomach, and revisability of the malabsorptive component (altering common channel length). The same group from Israel favored gastric bypass as the conversion procedure compared to

duodenal switch if the patient had a high operative risk, vitamin deficiency, prior small bowel resection, improvement in diabetes and hypertension after sleeve gastrectomy, or BMI less than 50 prior to the sleeve gastrectomy [58].

In summary, the series for re-sleeve gastrectomy are small non-controlled studies with short follow-up and do not provide strong support to utilize this approach in clinical practice unless the patient refuses all other options. In our opinion, patients who have failed LSG should be evaluated for a conversion to RYGB or DS depending on their comorbidities, BMI, eating behavior, and surgical risk.

References

1. Salem L, Jensen CC, Flum DR. Are bariatric surgical outcomes worth their cost? A systematic review. J Am Coll Surg. 2005;200(2):270–8. PubMed: 15664103.
2. Christou NV, Look D, MacLean LD. Weight gain after short- and long-limb gastric bypass in patients followed for longer than 10 years. Ann Surg. 2006;244(5):734–40. PubMed: 17060766.
3. Ma Y, Pagoto SL, Olendzki BC, Hafner AR, Perugini RA, Mason R, et al. Predictors of weight status following laparoscopic gastric bypass. Obes Surg. 2006;16(9):1227–31. PubMed: 16989709.
4. Dunkle-Blatter SE, St Jean MR, Whitehead C, Strodel III W, Bennotti PN, Still C, et al. Outcomes among elderly bariatric patients at a high-volume center. Surg Obes Relat Dis. 2007;3(2):163–70. PubMed: 17331804.
5. Lutfi R, Torquati A, Sekhar N, Richards WO. Predictors of success after laparoscopic gastric bypass: a multivariate analysis of socioeconomic factors. Surg Endosc. 2006;20(6):864–7. PubMed: 16738971.
6. Anderson WA, Greene GW, Forse RA, Apovian CM, Istfan NW. Weight loss and health outcomes in African Americans and whites after gastric bypass surgery. Obesity (Silver Spring). 2007;15(6):1455–63. PubMed: 17557983.
7. Melton GB, Steele KE, Schweitzer MA, Lidor AO, Magnuson TH. Suboptimal weight loss after gastric bypass surgery: correlation of demographics, comorbidities, and insurance status with outcomes. J Gastrointest Surg. 2008;12(2):250–5. PubMed: 18071836.
8. Carbonell AM, Wolfe LG, Meador JG, Sugerman HJ, Kellum JM, Maher JW. Does diabetes affect weight loss after gastric bypass? Surg Obes Relat Dis. 2008;4(3):441–4. PubMed: 18065289.
9. Jamal MK, DeMaria EJ, Johnson JM, Carmody BJ, Wolfe LG, Kellum JM, et al. Impact of major co-morbidities on mortality and complications after gastric bypass. Surg Obes Relat Dis. 2005;1(6):511–6. PubMed: 16925280.
10. Evans RK, Bond DS, Wolfe LG, Meador JG, Herrick JE, Kellum JM, et al. Participation in 150 min/wk of moderate or higher intensity physical activity yields greater weight loss after gastric bypass surgery. Surg Obes Relat Dis. 2007;3(5):526–30. PubMed: 17903772.
11. Roberts K, Duffy A, Kaufman J, Burrell M, Dziura J, Bell R. Size matters: gastric pouch size correlates with weight loss after laparoscopic Roux-en-Y gastric bypass. Surg Endosc. 2007;21(8):1397–402. PubMed: 17332953.
12. Gould JC, Beverstein G, Reinhardt S, Garren MJ. Impact of routine and long-term follow-up on weight loss after laparoscopic gastric bypass. Surg Obes Relat Dis. 2007;3(6):627–30. PubMed: 17950045.
13. Harper J, Madan AK, Ternovits CA, Tichansky DS. What happens to patients who do not follow-up after bariatric surgery? Am Surg. 2007;73(2):181–4. PubMed: 17305299.
14. Campos GM, Rabl C, Mulligan K, Posselt A, Rogers S, Westphalen A, et al. Factors associated with weight loss after gastric bypass. Arch Surg. 2008;143(9):877–83. PubMed: 18794426.
15. Flier J, Maratos-Flier E. Energy homeostasis and body weight. Curr Biol. 2000;10(6):R215–7. PubMed: 10744985.
16. Kahn BB, Flier JS. Obesity and insulin resistance. J Clin Invest. 2000;106(4):473–81. PubMed: 10953022.
17. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33) [published correction appears in Lancet. 1999; 354 (9178): 602]. Lancet. 1998;352(9131):837–53. PubMed: 9742976.
18. Purnell JQ, Weyer C. Weight effect of current and experimental drugs for diabetes mellitus: from promotion to alleviation of obesity. Treat Endocrinol. 2003;2(1):33–47. PubMed: 15871553.
19. Carver C. Insulin treatment and the problem of weight gain in type 2 diabetes. Diabetes Educ. 2006;32(6):910–7. PubMed: 17102158.
20. Eldor R, Stern E, Milicevic Z, Raz I. Early use of insulin in type 2 diabetes. Diabetes Res Clin Pract. 2005;68 Suppl 1:S30–5. PubMed: 15955372.
21. Rosenstock J, Hassman DR, Madder RD, Brazinsky SA, Farrell J, Khutoryansky N, Repaglinide Versus Nateglinide Comparison Study Group, et al. Repaglinide versus nateglinide monotherapy: a randomized, multicenter study. Diabetes Care. 2004;27(6):1265–70. PubMed: 15161773.

22. Marbury T, Huang WC, Strange P, Lebovitz H. Repaglinide versus glyburide: a one-year comparison trial. Diabetes Res Clin Pract. 1999;43(3):155–66. PubMed: 10369424.
23. Vasudevan AR, Balasubramanyam A. Thiazolidinediones: a review of their mechanisms of insulin sensitization, therapeutic potential, clinical efficacy, and tolerability. Diabetes Technol Ther. 2004;6(6):850–63. PubMed: 15684639.
24. Yki-Järvinen H. Thiazolidinediones. N Engl J Med. 2004;351(11):1106–18. PubMed: 15356308.
25. Madan AK, Harper JL, Tichansky DS. Techniques of laparoscopic gastric bypass: on-line survey of American Society for Bariatric Surgery practicing surgeons. Surg Obes Relat Dis. 2008;4(2):166–73. PubMed: 18069071.
26. Alvarado R, Alami RS, Hsu G, Safadi BY, Sanchez BR, Morton JM, et al. The impact of preoperative weight loss in patients undergoing laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2005;15(9):1282–6. PubMed: 16259888.
27. Buffington CK, Marema RT. Ethnic differences in obesity and surgical weight loss between African-American and Caucasian females. Obes Surg. 2006;16(2):159–65. PubMed: 16469217.
28. Gallagher D, Albu J, He Q, Heshka S, Boxt L, Krasnow N, et al. Small organs with a high metabolic rate explain lower resting energy expenditure in African American than in white adults. Am J Clin Nutr. 2006;83(5):1062–7. PubMed: 16685047.
29. Egede LE, Dagogo-Jack S. Epidemiology of type 2 diabetes: focus on ethnic minorities. Med Clin North Am. 2005;89(5):949–75. viii. [PubMed: 16129107].
30. Albert MA, Torres J, Glynn RJ, Ridker PM. Perspective on selected issues in cardiovascular disease research with a focus on black Americans. Circulation. 2004;110(2):e7–12. doi:10.1161/01.CIR.0000135583.40730.21. PubMed: 15249517.
31. Fischer JE, Bland, KI, Callery MP. Mastery of surgery (Volume 1). Part VII: the gastrointestinal tract. p. 988.
32. Pucci A, Finer N. New medications for treatment of obesity: metabolic and cardiovascular effects. Can J Cardiol. 2015;31(2):142–52. Pubmed: 25661549.
33. Brethauer SA, Kothari S, Sudan R, Williams B, English WJ, Brengman M, et al. Systematic review on reoperative bariatric surgery. Surg Obes Relat Dis. 2014;10(5):952–72. PubMed: 24776071.
34. Brethauer SA, Nfonsam V, Sherman V, Udomsawaengsup S, Schauer PR, Chand B. Endoscopy and upper gastrointestinal contrast studies are complementary in evaluation of weight regain after bariatric surgery. Surg Obes Relat Dis. 2006;2(6):643–8. Pubmed: 17020823, Discussion 9–50.
35. Thompson CC, Chand B, Chen YK, Demarco DC, Miller L, Schweitzer M, et al. Endoscopic suturing for transoral outlet reduction increases weight loss after Roux-en-Y gastric bypass surgery. Gastroenterology. 2013;145(1):129–37. Pubmed: 23567348.
36. Leitman IM, Virk CS, Avgerinos DV, Patel R, Lavarias V, Surick B, et al. Early results of trans- oral endoscopic plication and revision of the gastric pouch and stoma following Roux-en-Y gastric bypass surgery. JSLS. 2010;14(2):217–20. Pubmed: 20932372.
37. Himpens J, Coromina L, Verbrugghe A, Cadiere GB. Outcomes of revisional procedures for insufficient weight loss or weight regain after Roux-en-Y gastric bypass. Obes Surg. 2012;22(11):1746–54. Pubmed: 22990874.
38. Dakin GF, Eid G, Mikami D, Pryor A, Chand B, American Society for Metabolic and Bariatric Surgery (ASMBS) Emerging Technology and Procedures Committee. Endoluminal revision of gastric bypass for weight regain — a systematic review. Surg Obes Relat Dis. 2013;9(3):335–42. Pubmed: 23561960.
39. Yimcharoen P, Heneghan H, Chand B, Talarico JA, Tariq N, Kroh M, et al. Successful management of gastrojejunal strictures after gastric bypass: is timing important? Surg Obes Relat Dis. 2012;8(2):151–7. Pubmed: 21441074.
40. Heneghan HM, Yimcharoen P, Brethauer SA, Kroh M, Chand B. Influence of pouch and stoma size on weight loss after gastric bypass. Surg Obes Relat Dis. 2012;8(4):408–15. Pubmed: 22055390.
41. Shimizu H, Annaberdyev S, Motamarry I, Kroh M, Schauer PR, Brethauer SA. Revisional bariatric surgery for unsuccessful weight loss and complications. Obes Surg. 2013;23(11):1766–73. Pubmed: 23828032.
42. Vijgen GH, Schouten R, Bouvy ND, Greve JW. Salvage banding for failed Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2012;8(6):803–8. Pubmed: 23041427.
43. Irani K, Youn HA, Ren-Fielding CJ, Fielding GA, Kurian M. Midterm results for gastric banding as salvage procedure for patients with weight loss failure after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2011;7(2):219–24. Pubmed: 21130044.
44. Keshishian A, Zahriya K, Hartoonian T, Ayagian C. Duodenal switch is a safe operation for patients who have failed other bariatric operations. Obes Surg. 2004;14(9):1187–92. Pubmed: 15527632.
45. Rawlins ML, Teel D, Hedgcorth K, Maguire JP. Revision of Roux-en-Y gastric bypass to distal bypass for failed weight loss. Surg Obes Relat Dis. 2011;7(1):45–9. Pubmed: 21111688.
46. National Conference of State Legislatures [home page on the Internet]. State employee health benefits. http://www.ncsl.org/issues-research/health/state-employee-health-benefits-ncsl.aspx#Agencies.
47. Cesana G, Uccelli M, Ciccarese F, Carrieri D, Castello G, Olmi S. Laparoscopic re-sleeve gastrectomy as a treatment of weight regain after sleeve gastrectomy. World J Gastrointest Surg. 2014;6(6):101–6. PubMed: 24976903.

48. Bohdjalian A, Langer FB, Shakeri-Leidenmühler S, Gfrerer L, Ludvik B, Zacherl J, et al. Sleeve gastrectomy as sole and definitive bariatric procedure: 5-year results for weight loss and ghrelin. Obes Surg. 2010;20(5):535–40. PubMed: 20094819.
49. Sieber P, Gass M, Kern B, Peters T, Slawik M, Peterli R. Five-year results of laparoscopic sleeve gastrectomy. Surg Obes Relat Dis. 2014;10(2):243–9. Pubmed: 24139922.
50. Greenstein AJ, Jacob BP. Placement of a laparoscopic adjustable gastric band after failed sleeve gastrectomy. Surg Obes Relat Dis. 2008;4(4):556–8. Pubmed: 18586565.
51. Rebibo L, Fuks D, Verhaeghe P, Deguines JB, Dhahri A, Regimbeau JM. Repeat sleeve gastrectomy compared with primary sleeve gastrectomy: a single-center, matched case study. Obes Surg. 2012;22(12):1909–15. Pubmed: 23001573.
52. Dapri G, Cadière GB, Himpens J. Laparoscopic repeat sleeve gastrectomy versus duodenal switch after isolated sleeve gastrectomy for obesity. Surg Obes Relat Dis. 2011;7(1):38–43. Pubmed: 21115409.
53. Noel P, Nedelcu M, Nocca D, Schneck AS, Gugenheim J, Iannelli A, et al. Revised sleeve gastrectomy: another option for weight loss failure after sleeve gastrectomy. Surg Endosc. 2014;28(4):1096–102. PubMed: 24170068.
54. Iannelli A, Schneck AS, Noel P, Ben Amor I, Krawczykowski D, Gugenheim J. Re-sleeve gastrectomy for failed laparoscopic sleeve gastrectomy: a feasibility study. Obes Surg. 2011;21(7):832–5. Pubmed: 20924713.
55. Langer FB, Bohdjalian A, Shakeri-Leidenmuhler S, Schoppmann SF, Zacherl J, Prager G. Conversion from sleeve gastrectomy to Roux-en-Y gastric bypass – indications and outcome. Obes Surg. 2010;20(7):835–40. Pubmed: 20393810.
56. Cottam D, Qureshi FG, Mattar SG, Sharma S, Holover S, Bonanomi G, et al. Laparoscopic sleeve gastrectomy as an initial weight-loss procedure for high-risk patients with morbid obesity. Surg Endosc. 2006;20(6):859–63. Pubmed: 16738970.
57. Eid GM, Brethauer S, Mattar SG, Titchner RL, Gourash W, Schauer PR. Laparoscopic sleeve gastrectomy for super obese patients: forty-eight percent excess weight loss after 6 to 8 years with 93% follow-up. Ann Surg. 2012;256(2):262–5. Pubmed: 22791102.
58. Carmeli I, Golomb I, Sadot E, Kashtan H, Keidar A. Laparoscopic conversion of sleeve gastrectomy to a biliopancreatic diversion with duodenal switch or a Roux-en-Y gastric bypass due to weight loss failure: our algorithm. Surg Obes Relat Dis. 2015;11(1):79–85. Pubmed: 25304833.
59. Parikh M, Pomp A, Gagner M. Laparoscopic conversion of failed gastric bypass to duodenal switch: technical considerations and preliminary outcomes. Surg Obes Relat Dis. 2007;3(6):611–8. Pubmed: 17936087.
60. Dapri G, Cadiere GB, Himpens J. Laparoscopic conversion of Roux-en-Y gastric bypass to sleeve gastrectomy as first step of duodenal switch: technique and preliminary outcomes. Obes Surg. 2011;21(4):517–23. Pubmed: 20838919.

Failed Weight Loss after Lap Band Surgery

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George A. Fielding

Key Points

- It is easy to blame the patient, their food choices, their lack of exercise, their failure to follow up, but as Buchwald recently wrote, as often as not the operation has failed hem [1]. Our job is to continue to help them find another solution for their obesity, usually with another procedure.
- In many facets of surgery, lesser is considered better if the outcomes are similar. Then, if the lesser procedure fails, one can move on to more complicated approaches.
- The first reason band patients fail at weight loss is that they either do not attend for follow-up, or do not get adjusted when they do.
- At the end of the day, the band determines portion size and desire to eat, while the patient determines what they eat.
- Patients eat fast, food gets stuck, they loosen the band, they like how it feels, then they do not come back. They will regain every pound they have lost.

The next group of causes for failure of weight loss involves technical issues with the band.

- If a patient who has a slip has done well, they will continue to do well after band revision.
- Band erosions always present with weight regain, despite tightening the band.
- If a patient presents with weight regain, and describes return of hunger it is incumbent on their surgeon to check for these various causes, rather than assuming the band has failed, as these mechanical problems are easily remedied.
- In recent years sleeve gastrectomy has become the favored conversion procedure. In the short term, it has had great success. There are the same concerns of increased risk as seen with the bypass, but gradually it has become accepted to do removal and sleeve at the same time.

Failure of weight loss is the bane of bariatric surgery. All bariatric surgeries work. They can all fail, and do.

The Roux-en-Y gastric bypass is considered the gold standard of bariatric surgery, the mark against which all other operations are compared. In the last few years there have been two papers looking at the long-term success of gastric bypass. The first, by Kelvin Higa, looked at his series from the 1990s. A total of 242 patients were operated on between 1998 and 1999 [2]. Office follow-up was 7 % at 10 years. Telephone follow-up was 19 % at 10 years. As a group, the available patients had an excess weight loss (EWL) of 57 %. It turned out that 86 (35 %) had one or more complications during follow-up,

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_20

including internal hernia in 16 % and gastrojejunal stenosis in 4.9 %. In this group 65 (33 %) failed to achieve EWL greater than 50 %. Only 136 (51 %) had nutritional testing at least once after postoperative year 1. Of these 136 patients, only 24 (18 %) had remained nutritionally intact.

This is a bypass paper from a fabulous surgeon, with one of the biggest bypass experiences in the world. It reflects the reality, and the difficulty of maintaining long-term follow-up in bariatric patients—you will hardly ever see them again, they are often malnourished and they will often regain weight.

More recently, Obeid reviewed a similar cohort of patients from NYU [3]. He studied 328 gastric bypass patients done at NYU between 2000 and 2004. After 10 years, he had found 134, or 46 % of these patients. They had 59 % EWL. Almost exactly as with Higas's group, 35 % failed to achieve 50 % EWL after 10 years. Furthermore, 20 % had a BMI greater than 40 kg/m^2, and if their starting BMI was greater than 50 kg/m^2, it was 39 %. Of these, 9 % had revisional surgery. With regard to complications requiring surgery, 12.8 % had internal hernia, 6 % small intestinal obstruction and 3 % incisional hernia. Most of these complications occurred at around 3 years after surgery. Once again, as in Higa's study, 87 % had at least one major vitamin or mineral deficiency.

The similarity between these two papers is striking. Yes, bypass works very well for many patients, but about one-third fail, and success comes at considerable cost. However, in the big picture, it is a successful procedure. The fact that one-third do not achieve a sustained weight loss that we consider satisfactory does not prevent surgeons performing this procedure in huge numbers, as it helps many, many people.

It is hard to imagine a more successful bariatric procedure in terms of weight loss than biliopancreatic diversion, with or without duodenal switch, especially when using Scopinaro's original 50 cm common channel. Yet it can fail, and does. Years ago I published a paper about placing lap bands over gastric pouches in failed BPDs [4]. Gagner similarly published on resleeving the pouch [5]. Back in the mid-1990s, when we were doing BPDs, it was almost inconceivable that we would have to do this with this most aggressive operation. But we did. All operations work and they can all fail. It was the metabolic consequences that staunched my enthusiasm for BPD, not the occasional failure. In recent times, BPD has had a second coming, as revisional procedure for failed gastric sleeves.

It is easy to blame the patient, their food choices, their lack of exercise, their failure to follow up, but as Buchwald recently wrote, as often as not the operation has failed them [1]. Our job is to continue to help them find another solution for their obesity, usually with another procedure, which leads us to the laparoscopic adjustable gastric band, or LAGB. The LAP-BAND® (Apollo Endosurgery, Austin, TX) was the first commercially available LAGB in the USA. The band was developed by Vern Vincent, then at Inamed, who expanded on the permanent band used by Kuzmak, making it adjustable and suitable for laparoscopic placement. The new band was first placed by Belachew in Belgium in 1993, closely followed by Favretti in Italy, and Himpens in France [6, 7]. Its appeal was obvious. Placement of a band involved little dissection, it was adjustable, it could easily be removed if it failed, and, in many cases, it worked very well. It appealed to many patients who simply would not agree to any of the more aggressive procedures that involved cutting and rerouting organs, as well as significant nutrient deficiencies [8].

The band became very popular in Europe, then Mexico, then Australia, after O'Brien, who had a long experience in gastric bypass, visited Belachew and Favretti and brought the technique back home. He taught me how to place the band, and most importantly, the philosophy of regular, frequent band adjustments, aimed at enhancing satiety, and most important, reducing the relentless hunger and the constant urge to eat, that bedevil obese patients.

As a morbidly obese man myself, with multiple comorbid conditions, and on 11 medications, it seemed a pipedream that I could be not hungry. I started doing bands in 1995, heard over and over that patients were less hungry, saw many of them lose a lot of weight. Eventually, in 1999, I had my

own LAGB placed by Paul O'Brien. For me and many others, the pipedream came true. If a band is well-adjusted, patients are less hungry, have less urge to eat, eat less, and lose weight. If the band is kept adjusted, they do not gain weight. That is the essence of success with a band: adjust it correctly, and keep it there. Dixon stressed the importance of not over-tightening the band, of keeping the band and the patient in the "green zone," not too loose and not too tight. He also showed clearly that the band reduced hunger [9, 10].

One of the confounding issues with the band is the variability of outcomes around the world. Some centers have great results, others in the same city not. There has been a great diminution in band placement over recent years, partly as a result of this variability. To me, having done bands for 20 years, and having had one for nearly 16 years, I fully understand its variability, even day to day. Before I discuss possible reasons for band failure, and need for further surgery it is important to present a few long-term studies of band outcomes, and assess the impact of this reduction in band placement.

In many facets of surgery, lesser is considered better if the outcomes are similar. Then, if the lesser procedure fails, one can move on to more complicated approaches. The classic example of this is cardiac disease, where there is usually a steady progression from statins to stents, to more stents, to CABG, to redo CABG. Likewise with knee and hip joint surgery, going from resurfacing to joint replacement, to repeat joint replacement if needed. These progressions are not considered failures, but rather continued steps in the treatment of a chronic disease. Obesity is a chronic disease. It seems logical to consider such a progression in our treatment. This is why the LAGB appealed to so many patients, and their doctors. As we have seen, the same applies to BPD, bypass, and most certainly to sleeve gastrectomy. Many of them will need revision. With the band, its initial safety and effectiveness made it a perfect first operation, providing the outcomes were equitable with the more complex procedures.

There is little doubt that the band is safe. In a 2012 study of UHC academic hospitals, Nguyen et al. reviewed 10,151 bands placed between 2007 and 2009 [11]. Mean length of stay was 1.2 days. Morbidity was 3.0 % and mortality only 0.03 %. Over a 3-year period band revision occurred in 0.76 % and explantation in 0.87 %. Gould, in a study of 32,509 bariatric patients between 2005 and 2007, of which 21 % were bands and 79 % a mix of open and laparoscopic bypasses, found mortality very low in both: 0.02 % for band and 0.09 % for bypass. However there was a significant difference in inpatient complications: 1.6 % for band and 4 % for bypass ($p<0.01$) [12]. Saunders reviewed 1 year readmissions in a high volume bariatric center and showed readmission after a band of 12.7 % and 24.2 % for a bypass [13].

Buchwald was the first to show that the outcomes were equitable [14]. He did a meta-analysis of 22,094 patients in 136 studies, and found the band had 61.6 % EWL with 0.1 % mortality, compared to the bypass with 68 % EWL and 0.5 % mortality. The BPD had 70 % EWL, with 1 % mortality. The difference between 61 and 68 % EWL in an average patient with a BMI of 47 kg/m^2 is about 15 pounds. Is a 35 year old 5' 4", 300 pound woman who loses all her comorbidities, gets a better job and loves her new life really less a success if she ends up 185 pounds instead of 170?

These results were mid-term. As we all know, it is the long-term that matters. Data on bypasses over 10 years show EWL around 57 %. At NYU, Weichman reviewed 2909 patients with an average BMI of 45 kg/m^2, who had bands between 2000 and 2008 [15]. There was an operative mortality of 0.06 %. At 7 years they had maintained a 47 % EWL. O'Brien, in a series of 3227 bands between 1994 and 2011, had 714 patients at longer than 10-year follow-up [16]. There had been no perioperative mortality for primary placement or any revisions. He also found 47 % EWL in this group. Only 5.6 % had their bands removed.

The band is safe, and it often works. Why does it sometimes fail? The mechanism of weight loss after a band is two-pronged. The objective is to adjust the band to achieve loss of hunger and increased satiety. It needs the patient to attend regularly, and the surgeon or their team to make these visits available, then adjust the band as

needed. Then, the patient has to eat slowly, slower than they ever thought. If either of these components is missing, the patient will regain weight.

Ever since O'Brien, Dixon, and I started teaching about the band, it has been all about the adjustments. Patients want the band and they want it to work. For it to work, it needs to be adjusted. It certainly does not fit the usual surgical model, where an operation is done, a follow-up visit or two are made, and the patient is discharged. We still see patients once a month for about 18 months then less frequently, but still regularly, up to 5 years, then yearly or as needed. I tell patients we are titrating their medicine, much like adjusting their blood pressure meds, perhaps needing to add another one if they need more. It becomes a rhythm of their life in the first couple of years, coming once a month for an adjustment.

On average, if they do 12 visits in the first year, they will get six adjustments. If they are losing weight, usually about 2 lbs a week, and they are not hungry, they do not need a fill. One cardinal sign is if the patient wakes up and craves breakfast. For some reason, well-adjusted band patients are never hungry in the morning. Furthermore, due to the diurnal functioning of the esophagus, they get full much quicker in the morning. Likewise, if they are snacking after dinner they are too loose.

So the first reason band patients fail at weight loss is that they either do not attend for follow-up, or do not get adjusted when they do. It is a lot of work, for both parties. Patients need to find the time to attend clinic. Surgeons need to work out dealing with the caseload in their office. As an example, at NYU we do about 1200 bariatric cases a year. Many are still bands. We do about 1000 band adjustments a month. The majority are in our main office, but many are done at satellite clinics, where we rent space on a weekly or monthly basis, around the periphery of New York. This makes it easier for patients to attend, so they do.

In the first year, patients need these regular adjustments, as they quite quickly adapt to the level of tension on the band. If they wait another month, they will be too loose, and they will regain weight. Seeing band patients every 3 months in the first 2 years after surgery is an exercise in futility. If they are seen, say they are still hungry and eating too much, yet are told to change their eating habits, rather than having their band adjusted, that too is a waste of time. What is more, they become discouraged, see little point returning, and do not. Then a few years later they turn up, and everyone agrees the band does not work. It was never given a chance to.

As a reflection of this, a study from the Netherlands reviewed 201 patients given bands between 1995 and 2003 [17, 18]. Of these, 193 were longer than 14 years. Nearly half, 46 %, still had their bands. They were only seen six times in the first 2 years, and once a year after that. It is surprising that any had done well. This is reflected in the poor weight loss compared to their patients who had gastric bypass. A similar study from Finland, of 60 patients at 14 years, with 100 % follow-up, showed that 52 % still had their bands, and they had 49 % EWL. The main difference between these two studies and many like them, and data from O'Brien in Australia and us at NYU is the incidence of band removal. I will discuss that later. The striking similarity is that in those patients who kept their bands, the weight loss in all these studies is the same, about 50 % EWL long-term.

At NYU, we have been referred 441 patients who had their band elsewhere, and were unhappy with their outcome [19]. Many had been told to have their bands removed, but preferred not to. Of these, 293 needed a band revision, and 26 patients had band removal and conversion to another procedure. The remaining 222 patients were treated by band adjustment, dietary counseling, and behavioral counseling about how to live with a band.

The most important points they were taught included the variability of band tightness due to stress, that it is always tighter in the morning, to eat smaller bites, and most important, to eat slowly, waiting between the bites. We also discussed that they would not become malnourished by eating such small meals, and that if you are not hungry, do not eat. If you are hungry, try hard to eat something healthy. At the end of the day, the band determines portion size and desire to eat,

while the patient determines what they eat. If most of what they eat is healthy, they will do well.

When they presented, these patients had lost an average of 12.2 % EWL. After only 1 year, they had a further 24.5 % EWL after following this advice.

Once a patient has a band and it is adjusted, it is quite tight. You simply cannot eat at the rate you used to. You cannot live as if you do not have a band. You cannot eat at the same rate as other people at the table, ever. It takes about 20 s for food to go down the esophagus, into the pouch and through the band. If you eat quicker than that, the bites pile up above the band, stretching the esophagus. This causes a reflex to gag and regurgitate that is terribly unpleasant, and embarrassing in social settings. This is the second reason bands fail. Patients do not eat slowly enough.

I constantly stress this, from the very first consultation. Every visit, I make patients look at a clock and see how long 25 s really is. I advise them to get a 30-s timer and watch the sand fall. I tell them to put their knife and fork down, put the sandwich or the slice of pizza down between every bite. Let it go down. I have heard more nonsense about foods people cannot eat with a band than I ever thought possible. If you eat slowly, in most cases it is fine. If you do not, food gets stuck and you are in the bathroom bringing it up. As I tell every patient, I have had a band for all these years, and if I eat at the same rate as my slender wife, I puke. It is not negotiable. The problem is that many people do not like being told what to do, least of all by a thing. So they eat fast, vomit, and come in to have the band loosened, which renders them hungry, and able to eat easily, so they start regaining weight. Or they switch to soft food like ice cream that goes down easily, and gain weight. It is all about eating slowly. If I eat slowly, I eat less than my slender wife, I'm satisfied, and I keep the weight off.

This is all nonnegotiable. It will never change. It is why weight loss curves with a band are dead straight at about 50 % EWL for years and years. It never changes, as long as it remains adjusted.

One of the common catch-cries about the sleeve gastrectomy, which I hear every day, especially from patients who have had their bands removed to have a sleeve, is that it is so much easier to eat with a sleeve than with a band. Yes it is, especially after a couple of years, and that is why many of them are now dealing with rapid, total weight regain. If the band is kept adjusted, and patients eat slowly, they do not regain weight. If it is loosened due to the discomfort from eating too fast, they all regain weight.

The third cause of weight gain is a combination of the first two. Patients eat fast, food gets stuck, they loosen the band, they like how it feels, then they do not come back. They will regain every pound they have lost. If they do come back, and start again, they will re-lose the weight. More often, they feel the band has not worked, and prefer to convert to another procedure.

The next group of causes for failure of weight loss involve technical issues with the band. Of these, by far the most common is slipped band, often in combination with pouch dilatation or hiatal hernia. The main issue with a slipped band is reflux, which is usually severe, causing loss of sleep, inability to eat due to dysphagia, and at its worst, aspiration pneumonia. The diagnosis is easily made by an esophagram. The first step after diagnosis is to loosen the band, which leads to relief of symptoms, and immediate weight gain.

Over the last 20 years, the main efforts in the development of best technique for a lap band placement have been directed at reducing the incidence of slipped band. The first was the move to pars flaccida technique [20, 21]. Then in 2003 Dolan wrote of the importance of looking for and repairing hiatal hernias at the time of band placement [22]. In 2008, Gulkarov reviewed our experience at NYU, demonstrating a much-reduced need for reoperation if a hiatal hernia is repaired at the primary operation [23]. Between July 2001 and August of 2006, 1298 patients underwent a lap band and a further 520 patients underwent band with concurrent hiatal hernia repair. The mean initial weight and BMI were 128 kg (range 71.1–245.7 kg) and 45.4 kg/m^2 (range 28–75 kg/m^2). Average follow-up for the band and band/HHR groups was 24.8 and 20.5 months, respectively. Rate of reoperation for HHR alone, or with band slip or concentric pouch dilatation, for band and band/HHR groups was 5.6 % and 1.7 %,

respectively ($p<0.001$). Total reoperation rate for slip, HHR and pouch dilatation was 7.9 % and 3.5 %, respectively ($p<0.001$). There was no significant difference in rate of slip repair alone between the two groups: 2.3 % and 1.7 %, respectively ($p<0.44$).

The importance of doing a hernia repair if one is seen, even a very small defect, became obvious to us. It is so important to reduce the chance of severe reflux which will require band loosening. The constant high pressure from the band will gradually dilate the pouch and blow open any weakness in the hiatus, eventually leading to a symptomatic hiatal hernia.

I believe the second cause of slip is excessively tightening the band. Loss of hunger and increased satiety is a wonderful thing for an obese patient. Not surprisingly, many feel that more is better. It is important to resist the entreaties to over-tighten the band. If patients are doing well, losing weight, and feeling well, do not tighten the band. If a band is too tight, it leads to pouch dilatation and to slipped band. The enemy of good is better.

The big decision facing a surgeon confronted with a slipped band is whether to fix it, or remove the band. I will discuss what to do upon removing the band later. This decision is of paramount importance. It is confounded by the fact that patients with a slip lose a lot of weight, love that fact, and are nervous that if they present for relief of their reflux, the band will be removed. As a result, they often present late, with a big slip. This can present technical difficulties during band revision.

The first step when facing a slip is to empty the band. I always warn the patient that they will be starving hungry within 24 h, and that they will regain a lot of weight, much of which is water. I then see them at 2 weeks and repeat the esophagram. Many times, the band will have returned to a completely normal position, and one can start re-tightening the band. If it is improved but not completely better, I repeat the process, often adding appetite suppression medications such as phentermine for the next 2 weeks. If it has not improved they need revision of their band. If the band is left empty, all patients will regain all their lost weight. The return of hunger, and no restriction of intake, is impossible to overcome.

With the surge of popularity of the sleeve gastrectomy, many surgeons opt to remove the band, and, pending insurance approval, do a sleeve gastrectomy at the time. Alternatively, they will remove the band and then enroll the patient in the required 6 months follow-up prior to insurance approval for a subsequent procedure. Much of this decision is predicated on the belief that band revision is technically challenging, and that patients will not be able to keep their weight off after revision.

Band revision surgery can indeed be challenging, largely due to the thick fibrous ring that forms around the band, especially posteriorly around the left crus of the diaphragm. Like all operations, it is a matter of step by step. After insufflation of the abdomen, I place a liver retractor and three 5 mm ports, using the old scars. If the patient had a single incision band placement, I gain access in the left subcostal area, and assess whether extra ports need to be placed, or it can be done through the periumbilical incision.

The first step is to divide all adhesions to the liver, to completely expose the hiatus and enable full liver retraction. I then start around the buckle of the band, using a diathermy hook to divide all the adhesions to the buckle. Once it is free, the band can be rotated easily, and also used as a retractor. Then I sharply divide the plane between the gastrogastric sutures over the band. The band is then pulled to the right to allow further division posteriorly. Once the gastrogastric suture plane has been divided, I fully mobilize the fundus off the left crus of the diaphragm, to visualize the gastroesophageal junction. I then assess whether there is a hiatal hernia, and if so, repair it anteriorly with figure-8 sutures of 0-polypropylene. I then unlock the band, slide it up to its correct position, relock it and redo gastro gastric sutures.

Yes, it can be difficult, but it is very worthwhile for many of these patients to be able to keep their bands, especially those who have done well.

In an effort to assess this problem, Beitner reviewed our experience with band revisions at NYU [24]. A retrospective analysis was conducted of adult patients who underwent lap band

surgery from January 1, 2001 to June 30, 2009 at NYU. Patients who required revision for pouch-related problems including band slippage, pouch dilation, and hiatal hernia were studied. Of 3876 patients, 390 patients had revision. Of the 411 patients (10.6 %) who underwent band revision for a pouch-related problem, 12 were converted to another bariatric procedure after revision and nine subsequently had the band removed.

Within the study period, the reoperation rate for first-generation bands was 15.6 % whereas only 3.5 % for second generation bands. This may be due to pressure differentials that can lead to vomiting and, therefore, slip and pouch dilation. In our practice, these bands have been superseded by lower pressure, higher volume bands (AP bands and Realize C bands), with the expectation of lower overall revision rates.

Reoperation occurred at a mean of 33.7 ± 33.3 months after the primary procedure. Mean operating time was 67.0 ± 30.5 min and length of hospital stay was 1.1 ± 0.9 days. In 252 patients (64.6 %), the band was able to be repositioned. In the remaining 109 patients (27.9 %), the band required replacement, and 29 patients (7.4 %) had hiatal hernia repair alone.

The procedure-related mortality was 0 %. Early (30-day) complications occurred in 0.5 %, late complications (erosion) in 0.5 %, and 29 patients (7.4 %) required a second revision. For patients undergoing revision, the initial weight was 124.1 ± 21.3 kg and BMI was 44.8 ± 6.1 kg/m^2. At reoperation, weight was 89.2 ± 20.5 kg, BMI was 32.3 ± 6.5 kg/m^2 and %EWL was 54.1 ± 21.8 %. Twelve months post-revision, weight was 92.2 ± 20.2 kg, BMI was 33.3 ± 6.4 kg/m^2, and %EWL was 48.8 ± 22.7 %. Weight was 92.4 ± 19.9 kg, BMI was 33.5 ± 6.2 kg/m^2, and %EWL was 47.5 ± 22.9 % 24 months post-revision.

The most important finding came after analyzing the impact of pre-revision weight loss on weight outcomes after revision, Patients were divided into three groups according to their %EWL at reoperation: less than 40 % (n = 93), 40–69 % (n = 187), and more than 70 % (n = 90). Patients with %EWL of less than 40 % at reoperation still had %EWL of less than 40 % 2 years after revision, whereas patients with better weight loss, %EWL of more than 40 % at reoperation, maintained a %EWL of more than 40 % over the 2-year period after revision.

This is the crux of the problem. If a patient who has a slip has done well, they will continue to do well after band revision. If they have had more mediocre outcomes, that will not change. It is the latter group where one should consider converting them to another procedure, should they wish to.

Beitner also compared the revised patients to non-revised patients, and there was no difference in weight loss between the two groups at 5 years, with both groups having 49 % EWL.

The majority of patients who required revision for a slip required only one revision. The need for a second revision was low. In this series, 7.4 % of patients required a revision and only 0.75 % of the study cohort required more than one reoperation. Slip was the most common cause for both the first and second reoperations, but recurrent slip occurred only in 0.26 % of the entire population.

In stark contrast, Lim reported that 21.7–35.5 % of LAGB patients will require revision or removal of either the band or the port [25]. Manganiello reported a higher incidence of second revision after band repositioning than after band replacement (55 % vs. 0 %) [26]. In contrast, our rate of second revision was similar for band repositioning (7.1 %) and replacement (6.4 %).

The reoperation rate of 10.6 % over a 9-year experience might be explained by several factors. Slips are corrected by loosening the band before the need for reoperation arises. The lower reoperation rates may also be attributed to aggressive evaluation and repair of hiatal hernias at primary banding, as discussed.

Furthermore, band revision is the best option when faced with slip, pouch dilation, or hiatal hernia, because it is significantly safer than conversion to another bariatric procedure.

In studies of conversion of LAGB to Roux-en-Y gastric bypass, the incidence of early and late complications was as high as 12–17% and 2–23 %, respectively [27]. Moreover, late reoperations occurred in up to 20 %, and between 1.8 and

4.3 % of procedures attempted laparoscopically required conversion to an open approach [28, 29]. Conversion to biliopancreatic diversion with or without duodenal switch had a complication rate of 6.3 % and 6.4 % in the series by Dapri et al. [30] and Dolan and Fielding [31]. The reoperation rate after conversion to biliopancreatic diversion was 20.6 % in Dapri's series. The reported complication rate after conversion of band to sleeve gastrectomy varies from 0 % in small series (ten or fewer patients) to 16.7 % [32].

Band revision surgery has lower morbidity than conversion to these other procedures. Even combining the morbidities of primary and revisional band operations, it is safer to undergo gastric banding than any of the other primary bariatric procedures.

My final assessment of the management of slips is that it is best to revise a band if the patient has done well, and that it can be done safely. Conversely, if they have not had success, I support converting them to another procedure. It is very important to maintain contact with the patient after revision, to allow proper band adjustment.

Lap band erosion is a potent cause of weight regain after band surgery. It is also a great enigma. Some would have us believe that most bands will erode, and, in small numbers, would seem to have the data to support that [33]. On the contrary, many other surgeons simply do not see it at alarming rates. At NYU, two separate reviews of a large number of patients at different time points over 8 years showed 0.4 % of 2437 patients, and 0.2 % of 2909 patients [34, 35]. There must be some difference in surgical technique to explain the higher incidence in some hands. Perhaps the gastrogastric sutures are too tight, or there was trauma from excision of the fat pad. Perhaps the band was left too tight for too long. Perhaps it was bad luck. What we do know is that it certainly does happen. Two studies have shown a significant difference in band erosion between original bands placed by the old perigastric technique, and new lower pressure bands placed by pars flaccida technique [36].

Brown reviewed 2986 patients with band placed between 1994 and 2010 [37]. They found 100 erosions in 85 patients (2.85 %) at a median time of 33 months from initial surgery to the erosion (range 11–170 months). Her important finding was that the rate of erosion was highest when the band was placed by the perigastric approach at 6.77 %. Since the adoption of the pars flaccida approach, the rate of erosion has dropped to 1.07 %. Early on in their experience, they replaced the band at the same sitting, and 13 of these patients (15.3 %) had two erosions, and one patient had three erosions. They now do delayed replacement of the band. The band has been successfully replaced in 56 patients. It has been explanted in 27 patients and two patients were converted to other bariatric procedures. The weight loss in patients who had a LAGB reinserted after erosion was not significantly different from the background cohort.

In a similar study, Singhal searched electronic databases for publications focusing solely on laparoscopic adjustable gastric banding with at least 500 patients and a minimum follow-up period of 2 years [37]. Multivariate meta-analyses were conducted separately for the pars flaccida group, the perigastric group, and the combined overall group to pool the average rates of both erosion and slippage for each paper included. The inclusion criteria were met by 19 studies. The mean rates of erosion and slippage were 1.0 % and 4.9 %, respectively. The results demonstrated a statistically significant overall correlation between erosion and slippage rates ($r=0.48$, $p=0.032$). A very strong correlation between erosion and slippage was found if the perigastric technique of placement was used ($r=0.99$, $p<0.001$). However, this correlation was not statistically significant where the pars flaccida technique of insertion was used ($r=0.34$, $p=0.38$). It would appear that the change to lower pressure bands, placed by the pars flaccida technique has greatly reduced the incidence of band erosion.

Band erosion typically presents with weight regain, despite tightening the band. It may occasionally be associated with an infection at the port. Diagnosis is by endoscopy, although sometimes it is easily seen on esophagram, with contrast swirling around the band. The band should be removed, and the gastric defect sutured. I use

2–0 polydioxanone to repair the defect. In the past, I put new bands in at the time, but several of these re-eroded. I now wait 6 months and either redo the band, or do a sleeve gastrectomy, which can be done through relatively normal tissue.

Park demonstrated the difficulty in performing concurrent eroded band removal and sleeve gastrectomy [38]. They reported on nine female patients with eroded bands from March 2011 to February 2013. Six patients underwent a staged procedure, and the other three underwent a single-stage revision. Among the six staged patients, eroded bands had been removed by laparoscopy in four and by endoscopy in two without complications. Their LSGs were performed at a median of 4.4 months after band removal. Another three patients underwent single-stage revision. No mortality occurred. However, there was one stenosis and two proximal leaks. After a mean follow-up of 19.1 months, all nine patients exhibited weight loss. The mean pre- and post-LSG BMIs were 34.0 ± 4.4 and 25.6 ± 2.1 kg/m^2, respectively. Revisional LSG resulted in a further median %EWL of 28.0 % (range, 7.9–68.9 %) versus weight at time of band removal.

Port and tubing problems account for a large portion of patients who have weight gain. Once the fluid goes, there is no restriction, and return of hunger and ability to eat. This is easily diagnosed by measuring the fill in the port, injecting fluid and remeasuring, sometimes in a week or two if one suspects a slow leak. Virtually all are at the port, either a crack in the tubing where it bends to enter the abdomen, or a needle stick to the tubing during adjustment. Rarely, there can be a leak in the band. At NYU, port problems occurred in 3 %. Most can be fixed under local anesthetic with sedation. If the port is not the problem, then laparoscopy is performed, the system injected with methylene blue, and the band leak identified. In the latter situation, a new band is placed. This is a safe surgery, and patients are discharged the same day.

If a patient presents with weight regain, and describes return of hunger it is incumbent on their surgeon to check for these various causes, rather than assuming the band has failed, as these mechanical problems are easily remedied. The patient can then continue on their weight loss journey with the band.

Lap band pseudoachalasia is a potent cause of severe reflux and band intolerance, but is much rarer than pouch dilatation, band slippage, or expansion of an existing hiatal hernia.

Burton evaluated 123 band patients who had adverse events or poor weight loss [39]. They had high-resolution video manometry and were compared with 30 patients who had successful LAGB results and 56 preoperative patients.

Five pathophysiologic patterns were identified: transhiatal enlargement ($n=40$), subdiaphragmatic enlargement ($n=39$), no abnormality ($n=30$), aperistaltic esophagus ($n=7$), and intermittent gastric prolapse ($n=3$). Esophageal motility disorders were more common in symptomatic and preoperative patients than in patients with successful LAGB outcomes ($P=.01$). Significant differences between patients with successful outcomes and symptomatic patients included the length of the high-pressure zone above the band ($P<.005$), peristaltic velocity ($P<.005$), frequency of previous surgery ($P=.01$), and lower esophageal sphincter tone ($P=.05$). Video manometry identified abnormalities in three-quarters of symptomatic patients in whom conventional contrast swallow had not been diagnostic. Seven of these patients had pseudoachalasia.

The research team then went on to assess the role of the lower esophageal contractile segment (LECS) in these patients [40]. An intact LECS during normal swallows was more frequent in patients with a successful LAGB outcome than in symptomatic patients (95 % vs. 43 %; $P<.005$). The rate of hypotensive swallows in symptomatic patients increased after removing all fluid from the gastric band (30 % vs. 17 %; $P=.002$). An intact LECS in 70 % of normal swallows defined normal motility in patients who had undergone LAGB.

A team from the NYU assessed whether emptying, rather than removing, a gastric band will resolve hypotensive swallowing [41]. The key question was whether the band should be removed if pseudoachalasia developed. The research team retrospectively reviewed the clinical, manometric, and radiologic data of 6 female patients

(age range, 37–55 years) in whom dysphagia or heartburn had developed and in whom manometric studies showed aperistalsis following LAGB. Fluid in the gastric bands was completely removed in five patients, and the band itself was removed in one patient. Reversibility of esophageal aperistalsis was then assessed.

Five patients (four who had removal of the fluid from the band and one who had surgical removal of the band) underwent manometry. Of these, two patients had a partial return of peristalsis, one had normal peristalsis, and two others had continued aperistalsis but showed clinical improvement. Another patient had improvement of radiologic esophageal dilation but declined repeat manometry. The findings suggest that achalasia-like esophageal aperistalsis may be reversible.

In a French study, 11 of 20 patients (55 %) with esophageal motility disorders fit the manometric criteria for an achalasia-like disorder, with a mean esophagogastric junction (EGJ) resting pressure of 32.1 cm H_2O and an EGJ relaxation pressure of 24.2 cm H_2O [42]. Nine of the 11 patients underwent band removal, with symptom resolution. The remaining two patients underwent band deflation. Manometric control after band removal showed both a decrease in resting and relaxation EGJ pressures (mean of 9.5 cm H_2O and 6.5 cm H_2O, respectively) and a recovery of wave contractions in 88 % of cases. Four patients underwent revision surgery due to weight regain and had successful outcomes.

In another study, 5 of 257 band patients presented with megaesophagus at a mean time of 32 months [43]. Preoperative esophageal manometry findings were normal in four of these five patients, and one patient had a nonspecific motility disorder. Megaesophagus partially improved in all of the patients after band deflation, but all patients required band removal because of persistent symptoms.

The simple approach to pseudoachalasia is to remove the band, which will result in full regaining of weight. The more effective long-term approach might be to loosen the band for 6 weeks and then assess the esophageal diameter via an esophagram. If findings come back normal, which is usually the case, then the band can be gradually retightened. If findings are abnormal, then band removal with concurrent RYGB may be the best option.

If, after all these issues are addressed, the patient has failed weight loss, it is appropriate to offer them another procedure. There is now substantial published experience in bypass, sleeve and BPD after failed band.

Until recently, with the surge of interest in the sleeve, gastric bypass was the preferred method of conversion. Kothari was one of the first to present this [44]. His group, led by Sugerman, placed 36 bands between 1996 and 1998, and 14 were removed and converted to gastric bypass, at a median time of 38.2 months. Median follow-up after conversion to gastric bypass was 8.3 months. Nineteen percent excess weight loss occurred after LAP-BAND placement. Forty-three percent excess weight loss occurred after conversion to gastric bypass ($P = .025$).

Mognol presented 70 patients with a median BMI of 45 ± 11 who underwent attempted laparoscopic conversion of LAGB to an RYGBP [45]. Indications for conversion were insufficient weight loss or weight regain after band deflation for gastric pouch dilatation in 34 patients (49 %), inadequate weight loss in 17 patients (25 %), symptomatic proximal gastric pouch dilatation in 15 patients (20 %), intragastric band migration in three patients (5 %), and psychological band intolerance in one patient. 3 of 70 patients (4.3 %) had to be converted to a laparotomy because of severe adhesions. Mean operative time was 240 ± 40 min (range 210–280). Mean hospital length of stay was 7.2 days. Early complication rate was 14.3 % (10/70). Late major complications occurred in six patients (8.6 %). There was no mortality. Median excess body weight loss was 70 ± 20 %. 60 % of patients achieved a BMI of <33 with mean follow-up 18 months.

Spivak, in an experience of 1400 lap bands, converted 33 patients to gastric bypass because of inadequate weight loss and/or complications, at a mean 28.2 months after the original gastric banding [30]. The mean BMI at the time of revision was 42.8 kg/m^2 (range 33.1–50.0; SD 4.8). The mean revision operative time was 105 min (range

85–175), and the mean hospital stay was 2.8 days (range 1–10). After conversion to RYGBP, mean BMI decreased to 33.9 kg/m^2 at 6 months ($p<0.001$) and 30.7 kg/m^2 (range 22–39.6; SD = 5.3) at 12 months or more of followup (average = 15.7 months; $p<0.0001$).

These are just three of the many papers addressing this topic. They all show good early success. All authors also point out that this revision surgery is more difficult. This is confirmed in a large study [46]. In a review of 66,303 patients who underwent RYGB, including 3132 patients (5 %) who had RYGB after removal of LAGB, it was shown that patients who had RYGB after a band removal were at greater risk for intraoperative complications (odds ratio [OR], 2.3; $P=.002$), postoperative complications (OR, 8.0; $P<.001$), and reoperations or reinterventions (OR, 6.0; $P<.001$) and had an increased length of hospital stay.

In 2004, Dolan published on my experience with band removal and conversion to BPD or BPD-DS, both open and laparoscopic, in 85 of 1439 patients (5.9 %), most commonly for persistent dysphagia and recurrent slippage [31]. The removal rate and slippage rate decreased from 10.8 and 14.2 to 2.8 and 1.3 %, respectively, following introduction of the pars flaccida technique. Fifteen of 27 patients with previous open vertical banded gastroplasty (VBG) required removal of the band. Mean percentage excess weight loss 12 months following open BPD, laparoscopic BPD, open BPD-DS, and laparoscopic BPD-DS was 44, 37, 35, and 28 %, respectively.

In recent years sleeve gastrectomy has become the favored conversion procedure. In the short term, it has had great success. There are the same concerns of increased risk as seen with the bypass, but gradually it has become accepted to do removal and sleeve at the same time. In the USA, this is even more important due to insurance issues. Barrett has recently published a typical series [47]. Thirty-two patients underwent single-stage revision from LAGB to LSG, with a control group of 64 matched primary sleeve patients. The most common indication for revision was insufficient weight loss (62.5 %). Operative time for revision and control groups was 134 and 92 min, respectively ($p<0.0001$). Hospital stay was 3.2 and 2.6 days, respectively ($p=0.02$). Overall, the 30-day complication rate for revision and control patients was 14.7 and 6.3 %, respectively ($p=0.20$). There were no leaks, one stricture (3.1 %) in the revision group, and one reoperation for bleeding in the control group (1.6 %). For patients with BMI >30 kg/m^2 at surgery, change in BMI at 12 months for revision and control was 8.8 and 11.6 kg/m^2, respectively ($p=0.02$). They also noticed something we have seen at NYU, that weight loss is greater in those who undergo primary LSG compared to those who undergo LSG as conversion from band.

At NYU, Obeid has recently published our series of 80 band conversions to sleeve [48]. The first 20 were done as two stage, and the last 60 as one stage. There was no difference in OR time or adverse events between the two groups.

In another study that compared primary sleeve gastrectomy ($n=259$) with sleeve gastrectomy plus band removal ($n=46$), the complication rate was 8 % in the primary-sleeve group and 9 % in the band-removal group [49]. The fistula rates were 3 % and 4 %, respectively ($P=.56$).

Failure of weight loss, and weight regain, certainly happens with the band, as it does with all the other procedures. There is still a big role for the band in managing morbid obesity. Many patients simply will not come for any other procedures. When many surgeons stopped doing bands, the overall number of bariatric procedures performed in the USA fell dramatically. The slack was not taken up by sleeve gastrectomy. There is no doubt that the subset of female patients, with a BMI under 50, who attend regularly for adjustments do best with the band. It is also a useful tool for revision of failed gastric bypass [50]. In an attempt to further delineate who should have a band, Sethi looked at pre-visit maximum weight loss by any other means as an indicator of likely success with the band at NYU [51] Patients who lost more than 50 lbs on their own, then regained it, did best, and were also least likely to fail. A full 70 % of these patients achieved greater than 40 % EWL at 2 years, and only 2 % had less than 20 % EWL.

References

1. Buchwald H. Revisional metabolic/bariatric surgery: a moral obligation. Surg Obes Relat Dis. 2014; 10(6):1019–21.
2. Higa K, Ho T, Tercero F, et al. Laparoscopic Roux-en-Y gastric bypass: 10-year follow-up. Surg Obes Relat Dis. 2011;7(4):516–25.
3. Obeid NR, Concors S, Schwack BF, et al. Long-term outcomes in Roux-en-Y gastric bypass patients: 10–13 year data. Surg Obes Relat Dis. In press.
4. Slater GH, Fielding GA. Combining laparoscopic adjustable gastric banding and biliopancreatic diversion after failed bariatric surgery. Obes Surg. 2004;14(5):677–82.
5. Gagner M, Rogula T. Laparoscopic re-operative sleeve gastrectomy for poor weight loss after biliopancreatic diversion with duodenal switch. Obes Surg. 2003;13(4):649–54.
6. Belachew M, Legrand MJ, Vincent V. History of Lap-Band: from dream to reality. Obes Surg. 2001;11(3): 297–302.
7. Favretti F, Cadière GB, Segato G, et al. Laparoscopic placement of adjustable silicone gastric banding: early experience. Obes Surg. 1995;5(1):71–3.
8. Ren CJ, Cabrera I, Rajaram K, et al. Factors influencing patient choice for bariatric operation. Obes Surg. 2005;15(2):202–6.
9. Colles SL, Dixon JB, O'Brien PE. Hunger control and regular physical activity facilitate weight loss after laparoscopic adjustable gastric banding. Obes Surg. 2008;18(7):833–40.
10. Dixon AF, Dixon JB, O'Brien PE. Laparoscopic adjustable gastric banding induces prolonged satiety: a randomized blind crossover study. J Clin Endocrinol Metab. 2005;90:813–9.
11. Nguyen N, et al. Outcome of laparoscopic adjustable gastric banding and prevalence of band revision and explantation at academic centers: 2007–2009. Surg Obes Relat Dis. 2012;8:724–8.
12. Gould JC, Campos G, et al. Perioperative safety and volume: outcomes relationships in bariatric surgery: a study of 32,000 patients. J Am Coll Surg. 2011;213:771–7.
13. Saunders J, Schmidt H, et al. One-year readmission rates at a high volume bariatric surgery center: laparoscopic adjustable gastric banding, laparoscopic gastric bypass, and vertical banded gastroplasty-Roux-en-Y gastric bypass. Obes Surg. 2008;18(10):1233–40.
14. Buchwald H, Estok R, Fahrbach K, et al. Trends in mortality in bariatric surgery: a systematic review and meta-analysis. Surgery. 2007;142(4):621–32.
15. Weichman K, Ren C, Kurian M, et al. The effectiveness of adjustable gastric banding: a retrospective 6-year U.S. follow-up study. Surg Endosc. 2011;25: 397–403.
16. O'Brien PE, Macdonald L, Anderson M, et al. Long-term outcomes after bariatric surgery: fifteen-year follow-up of adjustable gastric banding and a systematic review of the bariatric surgical literature. Ann Surg. 2013;257:87–94.
17. Suter M, Calmes JM, Paroz A, Giusti V. A 10-year experience with laparoscopic gastric banding for morbid obesity: high long-term complication and failure rates. Obes Surg. 2006;16(7):829–35.
18. Aarts EO, Dogan K, Koehestanie P, et al. Long-term results after laparoscopic adjustable gastric banding: a mean fourteen year follow-up study. Surg Obes Relat Dis. 2014;10(4):633–40.
19. Victorzon M, Tolonen P. Mean fourteen-year, 100% follow-up of laparoscopic adjustable gastric banding for morbid obesity. Surg Obes Relat Dis. 2013; 9(5):753–7.
20. Obeid N, Malick W, Baxter A, et al. Weight loss outcomes among patients referred after primary bariatric surgery. Am J Surg. In press.
21. Fielding GA, Duncombe JE. Clinical and radiological follow-up of laparoscopic adjustable gastric bands, 1998 and 2000: a comparison of two techniques. Obes Surg. 2005;15:634–40.
22. O'Brien PE, Dixon JB, Laurie C, et al. A prospective randomized trial of placement of the laparoscopic adjustable gastric band: comparison of the perigastric and pars flaccida pathways. Obes Surg. 2005; 15(6):820–6.
23. Dolan K, Finch R, Fielding G. Laparoscopic gastric banding and crural repair in the obese patient with a hiatal hernia. Obes Surg. 2003;13(5):772–5.
24. Gulkarov I, Wetterau M, Ren CJ, et al. Hiatal hernia repair at the initial laparoscopic adjustable gastric band operation reduces the need for reoperation. Surg Endosc. 2008;22:1035–41.
25. Beitner M, Ren-Fielding C, Kurian M, et al. Sustained weight loss after gastric banding revision for pouch-related problems. Ann Surg. 2014;260(1):81–6.
26. Lim RB, Blackburn GL, Jones DB. Benchmarking best practices in weight loss surgery. Curr Probl Surg. 2010;47:79–174.
27. Manganiello M, Sarker S, Tempel M, et al. Management of slipped adjustable gastric bands. Surg Obes Relat Dis. 2008;4:534–8.
28. Schouten R, Japink D, Meesters B, et al. Systematic literature review of reoperations after gastric banding: is a stepwise approach justified? Surg Obes Relat Dis. 2011;7:99–109.
29. Topart P, Becouarn G, Ritz P. One-year weight loss after primary or revisional Roux-en-Y gastric bypass for failed adjustable gastric banding. Surg Obes Relat Dis. 2009;5:459–62.
30. Mognol P, Chosidow D, Marmuse JP. Laparoscopic conversion of laparoscopic gastric banding to Roux-en-Y gastric bypass: a review of 70 patients. Obes Surg. 2004;14:1349–53.
31. Dapri G, Cadiere GB, Himpens J. Laparoscopic conversion of adjustable gastric banding and vertical banded gastroplasty to duodenal switch. Surg Obes Relat Dis. 2009;5:678–83.

32. Dolan K, Fielding G. Bilio pancreatic diversion following failure of laparoscopic adjustable gastric banding. Surg Endosc. 2004;18:60–3.
33. Foletto M, Prevedello L, Bernante P, et al. Sleeve gastrectomy as revisional procedure for failed gastric banding or gastroplasty. Surg Obes Relat Dis. 2010;6:146–51.
34. Romy S, Donadini A, Giusti V, et al. Roux-en-Y gastric bypass vs gastric banding for morbid obesity: a case-matched study of 442 patients. Arch Surg. 2012;147(5):460–6.
35. Carelli AM, Youn HA, Kurian MS, et al. Safety of the laparoscopic adjustable gastric band: 7-year data from a U.S. center of excellence. Surg Endosc. 2010;24(8):1819–23.
36. Kurian M, Sultan S, Garg K, et al. Evaluating gastric erosion in band management: an algorithm for stratification of risk. Surg Obes Relat Dis. 2010;6(4): 386–9.
37. Brown WA, Egberts KJ, Franke-Richard D, et al. Erosions after laparoscopic adjustable gastric banding: diagnosis and management. Ann Surg. 2013;257(6):1047–52.
38. Singhal R, Bryant C, Kitchen M, et al. Band slippage and erosion after laparoscopic gastric banding: a meta-analysis. Surg Endosc. 2010;24(12):2980–6.
39. Park YH, Kim SM. Laparoscopic sleeve gastrectomy as revisional surgery for adjustable gastric band erosion. J Laparoendosc Adv Surg Tech A. 2014; 24(9):593–600.
40. Burton PR, Brown WA, Laurie C, et al. Criteria for assessing esophageal motility in laparoscopic adjustable gastric band patients: the importance of the lower esophageal contractile segment. Obes Surg. 2010;20: 316–25.
41. Burton PR, Brown WA, Laurie C, et al. Pathophysiology of laparoscopic adjustable gastric bands: analysis and classification using high-resolution video manometry and a stress barium protocol. Obes Surg. 2010;20:19–29.
42. Khan A, Ren-Fielding C, Traube M. Potentially reversible pseudoachalasia after laparoscopic adjustable gastric banding. J Clin Gastroenterol. 2011; 45:775–9.
43. Arias IE, Radulescu M, Stiegeler R, et al. Diagnosis and treatment of mega-esophagus after adjustable gastric banding for morbid obesity. Surg Obes Relat Dis. 2009;5:156–9.
44. Robert M, Golse N, Espalieu P, et al. Achalasia-like disorder after laparoscopic adjustable gastric banding: a reversible side effect? Obes Surg. 2012;22: 704–11.
45. Kothari SN, DeMaria EJ, Sugerman HJ, et al. Lapband failures: conversion to gastric bypass and their preliminary outcomes. Surgery. 2002;131(6):625–9.
46. Spivak H, Beltran OR, Slavchev P, et al. Laparoscopic revision from LAP-BAND to gastric bypass. Surg Endosc. 2007;21(8):1388–92.
47. Worni M, Ostbye T, Shah A, et al. High risks for adverse outcomes after gastric bypass surgery following failed gastric banding: a population-based trend analysis of the United States. Ann Surg. 2013; 257:279–86.
48. Barrett AM, Vu KT, Sandhu KK, et al. Primary sleeve gastrectomy compared to sleeve gastrectomy as revisional surgery: weight loss and complications at intermediate follow-up. J Gastrointest Surg. 2014;18(10): 1737–43.
49. Obeid NR, Schwack BF, Kurian MS, et al. Single-stage versus 2-stage sleeve gastrectomy as a conversion after failed adjustable gastric banding: 30-day outcomes. Surg Endosc. 2014;28(11):3186–92.
50. Rebibo L, Mensah E, Verhaeghe P, et al. Simultaneous gastric band removal and sleeve gastrectomy: a comparison with front-line sleeve gastrectomy. Obes Surg. 2012;22:1420–6.
51. Irani K, Youn HA, Ren-Fielding CJ, et al. Midterm results for gastric banding as salvage procedure for patients with weight loss failure after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2011;7(2):219–24.

Post-Gastric Bypass Hypoglycemia: Diagnosis and Management

21

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21.1 Introduction

Symptomatic hyperinsulinemic hypoglycemia after Roux-en-Y gastric bypass (RYGB) is an uncommon complication that is a challenge to successfully treat. The syndrome is suspected when RYGB subjects have postprandial neuroglycopenic symptoms (reduced cognition, weakness, fatigue, warm sensation, slurred speech, hypoglycemia unawareness/loss of consciousness) that are associated with an abnormally low serum glucose level [1, 2]. Glucose is the sole source of fuel to the brain, and symptoms and signs of central nervous system malfunction are detectable at a plasma glucose around 54 mg/dL [3]. Persistent or unrecognized hypoglycemia can evolve to progressively severe symptoms such as confusion, loss of consciousness, seizures, coma, and even death [3].

The prevalence of symptomatic hyperinsulinemic hypoglycemia after RYGB is estimated to be between 0.3 and 1 % [4, 5], while asymptomatic postprandial hypoglycemia may have a prevalence as high as 72 % after ingestion of large amounts of carbohydrate [6]. Symptomatic hyperinsulinemic hypoglycemia was described many years ago and is known to most general surgeons as part of the commonly described "dumping syndrome" after gastric resection [1, 7, 8]. After RYGB, mild symptoms related to postprandial hypoglycemia may be beneficial to some patients as they learn to avoid dense foods loaded with simple carbohydrates. In the vast majority of patients, these hypoglycemic symptoms can be managed with dietetic modification and medications, without resorting to revisional surgery [1]. However, in a small subset, the symptoms of hyperinsulinemic hypoglycemia are severe enough to be life-threatening and do not resolve with dietary changes or medical therapy. In this chapter, we discuss the current understanding of the pathophysiology of hyperinsulinemic hypoglycemia syndrome and present a diagnostic strategy and options for nonsurgical and surgical treatment.

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_21

21.2 Pathophysiology of Hyperinsulinemic Hypoglycemia

The precise etiology of symptomatic postprandial hyperinsulinemic hypoglycemia after RYGB is still under scrutiny. However, most experts agree that the anatomic changes of RYGB which allow delivery of the food bolus to the mid small bowel while bypassing most of the stomach and duodenum, leads to changes in glucose kinetics, changes in multiple glucose regulatory mechanisms, as well as gastrointestinal and pancreatic hormones levels involved in glucose homeostasis (Table 21.1) [9–19]. Though these anatomic and physiologic changes are beneficial in the majority of obese patients, both in diabetics and nondiabetics [20–22], they are thought to be the major

Table 21.1 Factors involved in glycemic control and changes after RYGB

Factors affecting glucose regulation	Author	Journal
Calorie restriction/Negative energy balance	Lips [65] Isbell [66] Lingvay [67] Laferrere [33] Swarbrick [68]	Clin Endocrinol, 2014 Diabetes Care, 2010 Diabetes Care, 2013 J Clin Endocrinol Metab, 2008 Diabetologia, 2008
Decrease in fat mass	Olbers [69] Tamboli [70] Miller [71] Immonen [72]	Ann Surg, 2006 Obesity, 2010 Diabetes Obes Metab, 2011 J Hepatol, 2014
Decrease in lipotoxicity (adipocytokines)	Lin [73] Malin [74] Geloneze [75]	Diabetes, 2007 Diabetes Obes Metab, 2014 Obes Surg, 2001
Changes in hepatic glucose production	Immonen [72] Camastra [76] Dunn [77] Bojsen-Moller [78]	J Hepatol, 2014 Diabetologia, 2011 Diabetes Care, 2012 Diabetes, 2014
Changes in hepatic insulin clearance	Bojsen-Moller [79]	J Clin Endocrinol Metab, 2013
Changes in insulin resistance	Camastra [76] Dunn [77] Bojsen-Moller [79]	Diabetologia, 2011 Diabetes Care, 2012 Diabetes, 2014
Altered glucose kinetics	Rodieux [9]	Obesity, 2008
Altered postprandial gut hormone levels (GLP-1, GIP, PYY, Ghrelin, etc.)	Rodieux [9] Nannipieri [80] Thaler [81] Cummings [82]	Obesity, 2008 J Clin Endocrinol Metab, 2013 Endocrinology, 2009 N Engl J Med, 2002
Altered postprandial pancreatic hormone levels (Insulin, Glucagon, PPP)	Campos [40] Umeda [83] Kashyap [84]	Surg Obes Relat Dis, 2014 Obes Surg, 2011 Int J Obes, 2010
Changes in pancreatic beta-cell function	Kashyap [84] Weiss [85] Ferrannini [86]	Int J Obes, 2010 Diabetes, 2014 Diabetes Care, 2009
Changes in resting and meal-induced energy expenditure	Rabl [87] Das [88]	Surgery, 2014 Am J Clin Nutr, 2003
Changes in gut microbiota	Liou [89] Vrieze [90] Sweeney [91]	Sci Transl Med, 2013 Gastroenterology, 2012 Best Pract Res Clin Gastroenterol, 2014
Changes in enterohepatic recirculation and bile acid composition	Sweeney [91] Patti [92] Pournaras [93]	Best Pract Res Clin Gastroenterol, 2014 Obesity, 2009 Endocrinology, 2012
Altered gastric emptying, nutrient intake, particle size and absorption	Carswell [94]	Obes Surg, 2014

factors responsible for recalcitrant symptoms of hyperinsulinemic hypoglycemia.

It is important to note that earlier reports trying to elucidate the pathophysiology of the condition have suggested that, in addition to the physiologic changes described above, some patients may have developed increased pancreatic beta-cell mass or nesidioblastosis [23]. These authors hypothesized that RYGB patients with hyperinsulinemic hypoglycemia had an abnormal increase in pancreatic beta-cell mass as a result of chronic beta-cell stimulation by increased postprandial GLP-1 levels [17, 23–25]. In an often quoted publication in the *New England Journal of Medicine*, Service et al. [23] reported that the histologic findings of pancreatectomy specimens of patients with RYGB-related hypoglycemia as having characteristics of nesidioblastosis, including "islet cell enlargement, beta-cells budding off ductular epithelium, and islets in apposition to ducts" [5]. Based on that information, those authors and other centers then have offered subtotal or total pancreatectomy as a surgical solution for medically refractory hyperinsulinemic hypoglycemia [23–26]. However, the initial findings of the Service study have not been corroborated by other experts in the field of pancreatic beta cell replication. Meier et al. [27] demonstrated convincingly that the patients in the original publication did not have increased islet hyperplasia, greater beta-cell turn over, or greater relative beta-cell area. They showed that the original study conclusions were due to an incorrect interpretation of the pathologic findings related to an inappropriate choice of control group. In the study by Service et al., the control group was autopsy specimens from patients with pancreatic cancer, which are expected to have altered pancreatic beta-cell function and morphology [27]; and also a BMI of 33.2 to 36.3, thus substantially less obese than the index subjects before gastric bypass (BMI, 44.4 to 62.5). When Meier et al. reevaluated the same specimens from the Service study using a different control group (pancreas autopsy specimens from obese and lean subjects without pancreatic disease), they found that the pathological findings in RYGB subjects were equivalent to samples taken from obese and lean controls, thus demonstrating that there are no inherent changes in pancreatic beta-cell mass related to RYGB [27].

One aspect of pathophysiology in these patients, which has not been well studied are the changes in post-RYGB counter-regulatory mechanisms to hypoglycemia. In normal homeostasis, the body protects itself with a series of physiologic and neuroendocrine regulatory measures to maintain serum glucose levels roughly between 65 and 125 mg/dL [3]. The standard counter-regulatory mechanisms activated to respond to hypoglycemia involve multiple systems and depend on the degree of hypoglycemia. Serum glucose levels below approximately 70 mg/dL are associated with a reduction in endogenous insulin secretion and increased pancreatic glucagon production, which in turn upregulates hepatic glycogenolysis and gluconeogenesis. Serum glucose levels below 65 mg/dL promote sympathetic nervous system activation with the release of adrenaline, growth hormone, and cortisol. Prolactin, antidiuretic hormone (ADH), aldosterone, and atrial natriuretic peptide (ANP) are also released, although their contribution to glucose homeostasis is uncertain. These homeostatic mechanisms are likely also affected by the anatomic and physiologic changes, which occur after RYGB and possibly play a role in the subjects prone to develop symptomatic hyperinsulinemic hypoglycemia.

The alterations in postprandial glucose kinetics, glucose regulatory mechanisms and gastrointestinal and pancreatic hormones levels after RYGB have been extensively studied in an effort to explain diabetes remission as well as refractory RYGB-related hypoglycemia. RYGB patients have increased postprandial insulin, glucagon-like peptide-1 (GLP-1), and polypeptide YY (PYY) levels and a greater postprandial suppression of ghrelin [9]. GLP-1 is an incretin which has been identified as playing a crucial role in postprandial insulin secretion [9, 28–30] and is secreted by the L-cells of the ileum during nutrient ingestion [28]. After RYGB, patients experience postprandial increase in beta-cell secretion of insulin that is accompanied by a markedly increased secretion of GLP-1 [12, 28, 31–33]. In addition, it has been noted that there is no increase in pancreatic GLP-1 receptors in

patients with RYGB-related hypoglycemia, suggesting that the pathophysiology is different from that of insulinoma [34]. Insulin response to a meal has a distinct response with a pattern of a rapid rise and peak in post-RYGB patients and this effect is exaggerated in patients who have symptomatic hypoglycemia as compared to those who are asymptomatic [29]. In addition, glucose kinetics are also altered as plasma glucose peaks earlier and higher in post-RYGB patients and are associated with lower plasma glucose nadirs [9, 35]. One recent study was performed in which patients who were administered a GLP-1 receptor blocker had significantly higher blockage of postprandial insulin secretion [29]. In fact, blockade of the GLP-1 receptor may be a potential treatment option for patients with refractory RYGB-related hypoglycemia [35].

It is clear that the etiology of symptomatic hyperinsulinemic hypoglycemia is not only due to GLP-1-stimulated insulin secretion [29]. One study found that GLP-1 was normalized after reversal of RYGB, however, hyperinsulinemic hypoglycemia persisted [32]. Other incretins, such as GIP may also play a role in post-RYGB hypoglycemia, as it has been shown to be similarly increased postprandially [31–33, 36]. Additionally, insulin sensitivity is also improved after surgery, although this result is not immediately present and requires significant weight loss to manifest [12, 37]. Nevertheless, most patients with hyperinsulinemic hypoglycemia syndrome present with symptoms once significant weight loss has occurred and thus insulin resistance is decreased.

Current evidence suggests that changes in postprandial glucagon levels do not play a role in postprandial hypoglycemia. While some have hypothesized that a lack of glucagon response to profound hypoglycemia could be attributed to the known glucagonostatic effect of the elevated GLP-1 levels [31] and that the disruption of this physiologic feedback mechanism could contribute to hypoglycemia [38], others studying glucagon levels in RYGB patients found a paradoxical increase in glucagon during OGTT after RYGB [33]. This finding has been also corroborated by our group in which five patients with well-documented hyperinsulinemic hypoglycemia syndrome had no impairment in postprandial glucagon levels and no inherent inappropriate glucagon to insulin secretion [39].

These important recent findings support the hypothesis that the pathophysiology of RYGB-related hyperinsulinemic hypoglycemia is associated with the reversible anatomic and physiologic alterations produced by RYGB and not with inherent changes in pancreatic beta-cell mass or function. This hypothesis has been tested by the documentation of normalization of glucose kinetics, abolition of neuroglycopenic episodes and normalization of postprandial levels of gastrointestinal and pancreatic hormones when a meal test is done through the excluded portion of the stomach [40, 41]. Hyperinsulinism and hypoglycemic symptoms have been shown to persist if the meal test is performed orally via the RYGB anatomy. These case series have also documented resolution of postprandial, symptomatic hyperinsulinemic hypoglycemia after RYGB in most patients after reversal to either normal anatomy or a modified sleeve gastrectomy. While RYGB reversal, as detailed below, may provide an effective surgical approach to treat this condition, much research is still underway to precisely delineate the differences between patients who are prone to developing the syndrome and those who are not. Identifying the precise mechanisms may lead to a less invasive treatment than surgical RYGB reversal.

21.3 Diagnosis

There are no clear criteria for the diagnosis of RYGB-related hyperinsulinemic hypoglycemia as the recognition of this syndrome has been evolving over the past 10 years. Here, we detail a reasonable approach to identify these patients and a diagnostic algorithm to clarify the diagnosis and rule out other causes of hypoglycemia.

The first step is recognition of the symptoms in a patient who has had RYGB. Typically, the episodes of hypoglycemia are a late complication, occurring 1–4 years after the initial surgery. As discussed later in this chapter, the symptoms can overlap with those of "dumping syndrome." A careful

history should be performed, including what symptoms are occurring, whether they coincide with a low blood sugar, what time of day they occur, whether they occur when fasting or postprandial, what foods trigger symptoms, and whether symptoms resolve with food intake. Typically, patients with RYGB-related hypoglycemia will describe symptoms beginning 1–2 h after a meal with minimal fasting symptoms. High carbohydrate intake is often a trigger for symptoms. Symptoms should improve within 15 min of food intake, but then may recur again an hour later. After many recurrent episodes of hypoglycemia, the symptoms may lessen as the patient develops hypoglycemia unawareness.

In order to diagnose hypoglycemia of any cause, it is important to identify Whipple's triad. Whipple's triad includes (1) the presence of classic hypoglycemia symptoms, (2) a low plasma glucose (not capillary glucometer reading) at the time of symptoms, and (3) resolution of symptoms with food intake. Therefore, laboratory testing must begin with documentation of low plasma glucose during a symptomatic episode, typically less than 55 mg/dL. Although patients may be provided with a glucometer to test capillary glucose during symptomatic episodes, these readings should not be considered diagnostic. In the case of RYGB-related hypoglycemia, symptoms are typically postprandial, so it makes sense to provoke symptoms in order to document hypoglycemia in a controlled setting. Although mixed meal tests are sometimes used, we typically perform an oral glucose tolerance test (OGTT) to assist in making the diagnosis (Fig. 21.1). The OGTT provides a higher carbohydrate load and is more likely to induce hypoglycemia and related symptoms. This provocative test can be performed on an outpatient basis and consists of administration of 50–100 g of oral glucose after a 12 h fast. The serum glucose, insulin, and C-peptide are measured at the start of the test and every 30 min thereafter for 2–3 h after administration. A typical pattern in RYGB-related hypoglycemia is a rapid rise in glucose, insulin, and C-peptide in the first 30 min, followed by a rapid decline. Although the insulin and C-peptide levels often remain elevated or inappropriately normal at the time of the hypoglycemia, they should demonstrate a rapid decrease. Persistent hyperinsulinism is not consistent with reactive hypoglycemia. Fasting hyperinsulinemia with fasting hypoglycemia would also not be expected, and would prompt consideration of alternate causes of hypoglycemia, such as insulinoma. Importantly, fasting hyperinsulinemia with normal or elevated fasting glucose is indicative of insulin resistance or diabetes, not insulinoma. A failure to see a coincident elevation in C-peptide levels would raise concern that the hypoglycemia was induced by exogenously administered insulin. Consultation with an endocrinologist is recommended if an alternate cause is considered [42]. The OGTT test should be considered confirmatory for RYGB-related hyperinsulinemic hypoglycemia if glucose levels are less than 55 mg/dL and the patient developed symptoms during testing consistent with their described ambulatory symptoms. Importantly, it should be noted that the OGTT will induce hypoglycemia in up to 12.5 % of control patients and up to 72 % of RYGB patients [6, 43]. Therefore, the lab results should be interpreted with consideration of the clinical presentation of the individual patient.

Several algorithms for the diagnosis of hypoglycemia involve additional testing to rule out other causes, including insulinoma. It is reasonable to exclude alternate causes of hypoglycemia in RYGB patients when the testing is relatively straightforward and noninvasive. A TSH will screen for hyperthyroidism and an early morning cortisol will screen for adrenal insufficiency. Liver and kidney function should be examined to rule out contributions from severe renal insufficiency or liver disease. However, exclusion of insulinoma is a much more involved process. Insulinoma should be considered if fasting hypoglycemia and hyperinsulinemia are present or if symptoms are not clearly postprandial in nature. However, careful consideration of the expense and the invasiveness of a complete insulinoma rule out should take place if the patient has classic RYGB-related postprandial hyperinsulinemic hypoglycemia symptoms and testing.

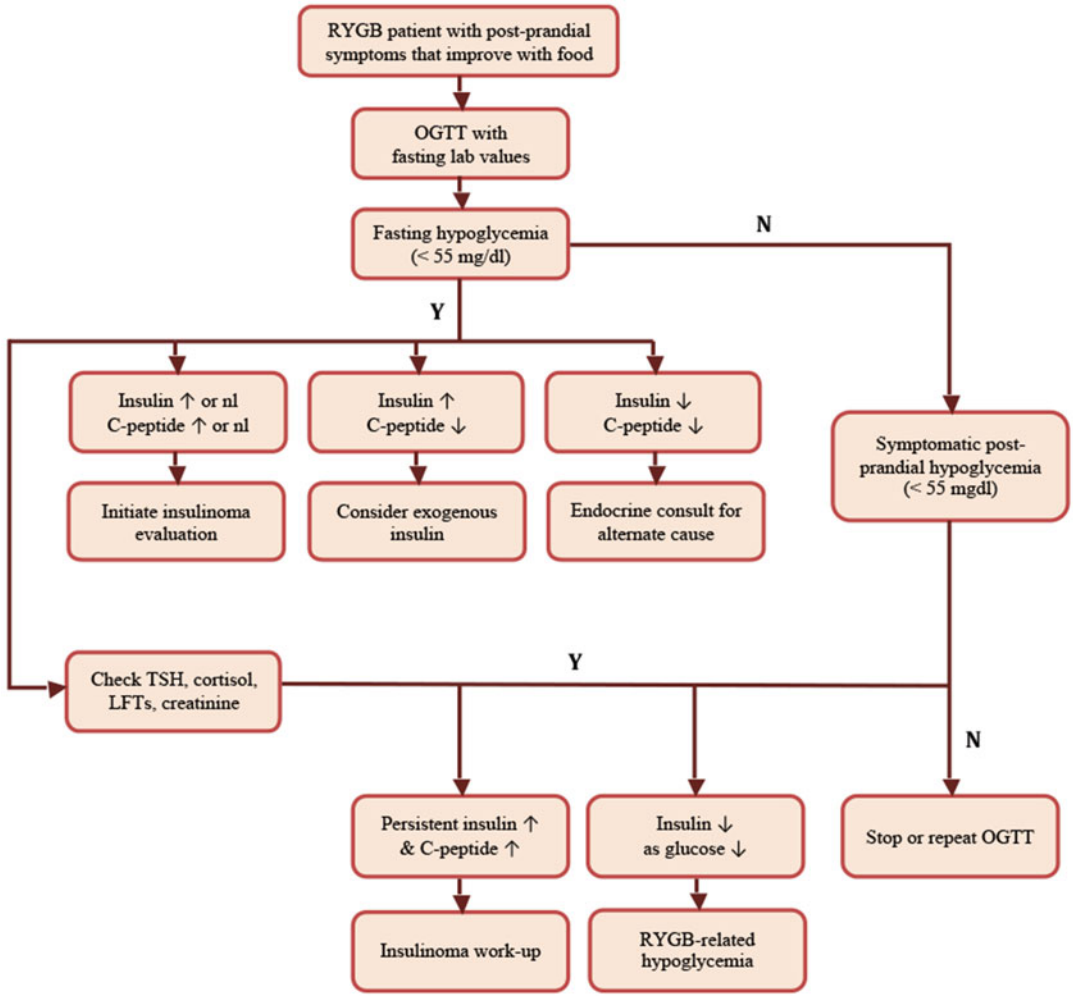

Fig. 21.1 Algorithm for diagnosis of RYGB-related hypoglycemia

Ruling out an insulinoma involves performing a 72-h diagnostic fast. In hyperinsulinemic hypoglycemia, this test should be negative as the hypoglycemia in these patients follows the consumption of food. However, if hypoglycemia and symptoms are not induced with OGTT or a shorter 12 h fast, then a prolonged fast would be indicated. This testing requires inpatient admission and careful coordination to manage the needs for frequent blood draws. The serum glucose, insulin, C-peptide, and proinsulin are measured every 6 h or every 2 h if the glucose level drops below 60 mg/dL. The test is stopped if the patient's glucose levels drop below 45 mg/dL with the development of neuroglycopenic symptoms. The patient is then tested for serum insulin, C-peptide, proinsulin, beta-hydroxybutyrate, and sulfonylurea levels. One mg of glucagon is administered and the serum glucose levels are checked at 10, 20 and 30 min [44].

A computed tomography scan of the abdomen and pelvis should be obtained to evaluate for masses in the pancreas if fasting or persistent hyperinsulinemia with hypoglycemia is seen. Some surgeons will also evaluate the pancreas with endoscopic ultrasound. If there is high suspicion, calcium-stimulated arterial angiography can be used to identify more precisely the location of an insulin-secreting pancreatic lesion.

21.4 Management

21.4.1 Diet Modification

Dietary modification is extremely effective and should be the mainstay of therapy for most patients with RYGB-related postprandial hyperinsulinemic hypoglycemia [4, 45]. Most patients naturally alter their own diets over time to avoid the unpleasant symptoms associated with this syndrome. Patients should be encouraged to eat a low-carbohydrate, high-protein diet with appropriate amounts of complex carbohydrates and fat for calories [1, 46]. Simple sugars such as candy or soda should be avoided [1]. Eating smaller, more frequent meals can also help to alleviate symptoms [1, 4, 46], but this must still be combined with a low carbohydrate intake or the patient will just have recurrent symptoms throughout the day. Lying supine for 30 min after a meal can minimize symptoms of dizziness and syncope [1, 46].

21.4.2 Continuous Glucose Monitoring Therapy

With recurrent hypoglycemia associated with hypoglycemia unawareness, consultation with an endocrinologist is recommended to consider use of a continuous glucose monitoring system (CGMS). With this technology, patients will have warning of hypoglycemia and can treat appropriately before severe cognitive impairment.

21.4.3 Pharmacologic Therapy

Approximately 3–5 % of patients will have more severe symptoms of hyperinsulinemic hypoglycemia that will not resolve with dietary modification alone [1]. A variety of pharmacologic agents have been used to alleviate refractory symptoms.

Alpha-glucosidase inhibitors, such as acarbose, were first shown to ameliorate the symptoms of dumping syndrome in 1979 [47]. The drug serves to slow the absorption of glucose from the small intestine, reducing the direct stimulation of the pancreatic beta-cells due to acute hyperglycemia. A randomized, double-blind trial showed significantly lower peak plasma glucose, insulin, and gastric inhibitory polypeptide (GIP) levels when compared with a placebo [47]. Therapeutic dosing ranges from 50 to 100 mg administered two or three times daily 30 min prior to a meal. Acarbose has also been shown to be effective in treating severe hyperinsulinemic hypoglycemia in many reports [48–52]; however, others have noted limited improvement in symptoms [1]. The use of acarbose may be limited by diarrhea and flatulence [1].

Diazoxide was first developed as an antihypertensive medication, but was found to inhibit insulin secretion from pancreatic beta-cells. It does this by opening the ATP-sensitive K^+ channels causing hyperpolarization and ultimately eliminating the influx of Ca^{2+} thus stopping the secretion of insulin. The inhibition of insulin also leads to an increase in glucose production from the liver [49]. One case reports describes a patient with RYGB-related hyperinsulinemic hypoglycemia who failed both surgical therapy with a subtotal pancreatectomy, as well as medical therapy with octreotide, voglibose, and diet modification, but had successful treatment of severe nocturnal hypoglycemia with administration of diazoxide [49]. Side effects can include facial flushing, edema, and weight increase. Diazoxide administration also can lead to hyperglycemia.

Somatostatin is a peptide hormone secreted by the gastric antrum, duodenum, and pancreas which inhibits the release of many other gastrointestinal hormones, including insulin and glucagon. Additionally, it reduces the rate of gastric emptying, slows intestinal transit time, as well as reducing motility, absorption of nutrients, and splanchnic blood flow [46]. In one randomized, double-blind trial comparing the somatostatin analogue, octreotide acetate, to placebo, it was found to completely prevent the development of both vasomotor and gastrointestinal symptoms of "dumping syndrome" [53]. Overall, five randomized, controlled trials have been conducted confirming the efficacy of octreotide [46]. The dosing is 50–100 μg administered subcutaneously

three times a day, 30 min prior to a meal. Side effects can be significant and include nausea, abdominal pain, flatulence and diarrhea. Long-term administration of octreotide is associated with gall bladder dysfunction and increased risk of diabetes. Octreotide can be highly effective in preventing the symptoms of both early and late "dumping syndrome" in over 90 % of patients [1, 46]. Somatostatin analogues have also shown efficacy in reducing postprandial hyperinsulinemic hypoglycemia in recent case reports [54, 55].

Verapamil, a calcium channel blocker, has been used combined with various other agents to treat RYGB-related hyperinsulinemic hypoglycemia [1, 48]. Sustained-release verapamil, dosed from 120 to 240 mg per day has been shown to provide complete resolution of vasomotor symptoms in one study [56].

21.4.4 Endoluminal Therapies

Some providers have attributed "intractable dumping syndrome," which clinically may appear indistinguishable from postprandial hyperinsulinemic hypoglycemia, to rapid emptying of the gastric pouch through a dilated gastrojejunal anastomosis. They have proposed an endoscopic tightening procedure to delay emptying of the gastric pouch in an effort to alleviate symptoms [57]. The endoscopic procedure begins with measuring the gastrojejunal anastomosis as well as the pouch size. The mucosa of the anastomosis is ablated using an argon plasma coagulator. The EndoCinch suturing system (CR Bard, Murray Hill, NJ) is then used to place endoscopic sutures to plicate the anterior and posterior aspects of the anastomotic ring together with a goal anastomotic lumen of less than 1 cm. Fibrin glue is then applied to the sutures areas. All six patients in this series reported complete resolution of symptoms lasting for a median follow-up of over 600 days. The limitations of this study, however, include a lack of documentation of postprandial hyperinsulinemic hypoglycemia and subsequent objective resolution as all patient results are obtained from clinical interview only.

Another group used the StomaphyX device (EndoGastric Solutions, Redmond, WA) to plicate the gastrojejunostomy in 42 patients with "severe dumping syndrome" [58]. This device places 3–0 polypropylene "H" fasteners circumferentially at 1–2 cm intervals from just proximal to the anastomosis to the gastroesophageal junction in order to cinch down the anastomosis as well as the size of the pouch. They report complete resolution of symptoms in 71 % of patients and improvement of symptoms in all patients. Again, the limitations of this study are related to their definition of "dumping syndrome" and preoperative and postoperative documentation of objective factors. It is unclear whether these two studies can be applied to patients with RYGB-related hyperinsulinemic hypoglycemia.

21.4.5 Pancreatectomy

Pancreatectomy has been described by various authors as a potentially curative surgical intervention for RYGB-related hyperinsulinemic hypoglycemia for the past 20 years; however, as described above, there are many complicating factors which make this technique a poor candidate for appropriate therapy. Multiple case series have been reported of patient with postprandial hyperinsulinemic hypoglycemia after RYGB who were then treated with subtotal or distal pancreatectomy [23–26]. Unfortunately, in all three series, most of the treated patients had no resolution of symptoms or had recurrence of symptomatic hypoglycemia at 1 year [23–25]. At least 25 percent of patients in another study experienced zero benefit from partial pancreatectomy [59]. The overall recurrence of symptoms after partial pancreatectomy has been reported as high as 87 % with a median time to recurrence of 16 months [59]. Therefore, in some cases, the patients ultimately underwent total pancreatectomy to resolve symptoms, but resulting in brittle diabetes.

It seems clear then that pancreatectomy neither addresses the underlying pathophysiology

leading to hypoglycemia nor leads to resolution of symptoms in most patients. Additionally, sub-total or total pancreatectomy has elevated perioperative morbidity and may lead to brittle diabetes and other dysfunction related to extensive or complete pancreatic resection. Thus, pancreatectomy should not be offered as therapeutic option in these patients.

21.4.6 Reversal of Gastric Bypass

Reversal of a RYGB to normal anatomy was first described in 2006 [60]. Multiple groups had previously described the conversion of RYGB to other bariatric procedures and documented the feasibility of performing revisions laparoscopically. The indication for this first reversal was incapacitating "dumping syndrome" and the patient had complete resolution of symptoms in addition to maintaining her pre-reversal weight loss.

Postprandial hyperinsulinemic hypoglycemia refractory to diet modification or medical therapy is an indication for reversal of gastric bypass to either normal anatomy or modified sleeve gastrectomy. Preoperative evaluation as described above should be performed to confirm the diagnosis. An esophagogastroduodenoscopy can be performed to confirm pouch size and anatomy and to rule out other pathology. The decision to reverse to normal anatomy or a modified sleeve gastrectomy is complex and based on individual patient characteristics including prior history of gastroesophageal reflux before RYGB.

Prior to consideration of a reversal procedure, we recommend laparoscopic placement of a gastrostomy tube in the gastric remnant followed by a Meal Tolerance Test (MTT) through the excluded gastric route and through the RYGB anatomy to document normalization of glucose and GI and pancreatic hormones postprandial kinetics [40]. The patient is also encouraged to use the gastrostomy tube for feedings and this allows for confirmation that the patient's postprandial hypoglycemia symptoms will resolve upon surgical reversal. Feeding via a gastrostomy tube prior to surgery can also help avoid refeeding syndrome in cases where the patient is malnourished or has had significant weight loss [61].

The technique has been described in detail previously [40]. The patient is placed in the modified lithotomy position after general anesthesia with an endotracheal tube has been obtained. The abdomen can be entered using either direct trocar insertion with or without Veress insufflation or with the Hasson technique. Five or six trocars should be placed at the surgeon's discretion to aid in dissection.

The procedure begins with dissection of adhesions around the alimentary limb, gastric pouch and gastric remnant. The gastrojejunostomy is identified and dissected circumferentially. The alimentary limb and the common channel should be measured for length estimates. A linear stapler is fired across the gastric pouch just proximal to the gastrojejunal anastomosis (Fig. 21.2). Care should be taken to avoid injuring the left gastric artery as it is the only blood supply to the gastric

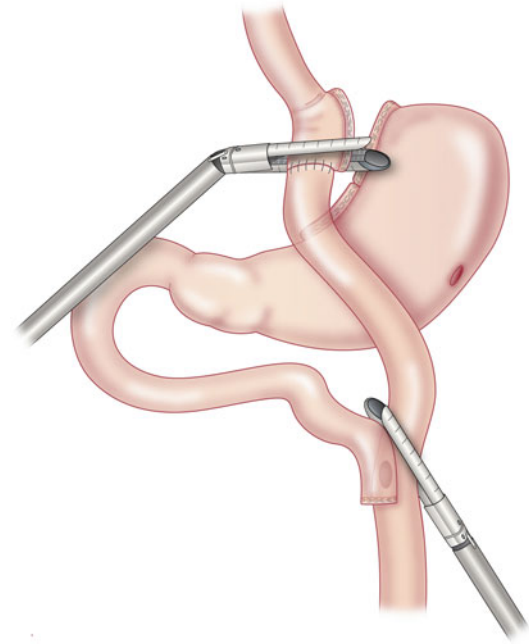

Fig. 21.2 Division of gastrojejunostomy and alimentary limb using linear staplers. With permission from Campos GM, Ziemelis M, Paparodis R, Ahmed M, Davis DB. Laparoscopic reversal of Roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. Surg Obes Relat Dis. 2014;10(1):36–43. doi:10.1016/j.soard.2013.05.012. Epub 2013 Jun 29 [95]. © Elsevier

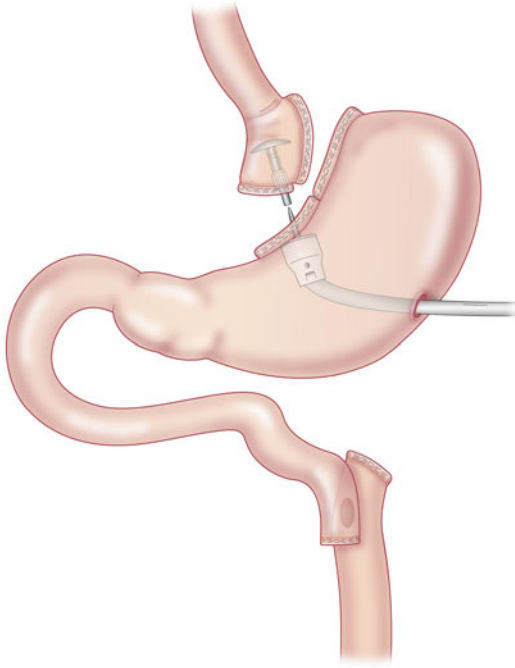

Fig. 21.3 Gastrogastric anastomosis using a 25 mm oral anvil and a 4.8 mm, 25 mm circular stapler via the gastric remnant. With permission from Campos GM, Ziemelis M, Paparodis R, Ahmed M, Davis DB. Laparoscopic reversal of Roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. Surg Obes Relat Dis. 2014;10(1):36–43. doi:10.1016/j.soard.2013.05.012. Epub 2013 Jun 29 [95]. © Elsevier

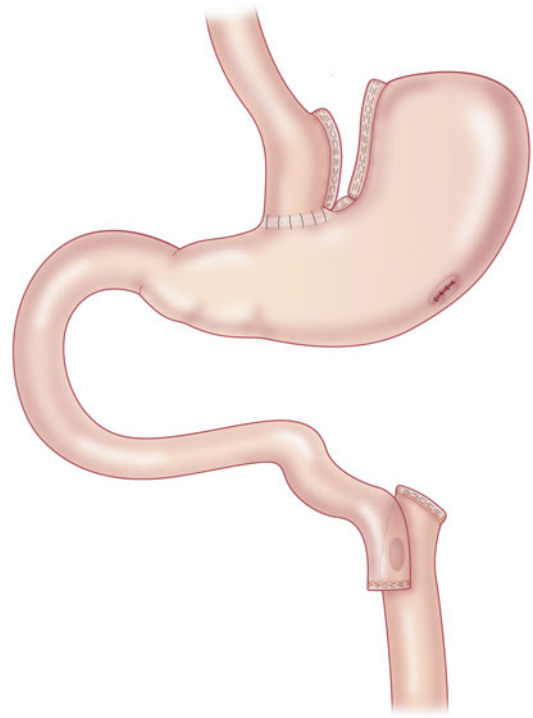

Fig. 21.4 Completed reversal to normal anatomy. With permission from Campos GM, Ziemelis M, Paparodis R, Ahmed M, Davis DB. Laparoscopic reversal of Roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. Surg Obes Relat Dis. 2014;10(1):36–43. doi:10.1016/j.soard.2013.05.012. Epub 2013 Jun 29 [95]. © Elsevier

pouch. In general, dissection should be kept to a minimum to avoid complications.

There are multiple methods which can be used to then create the gastrogastrostomy. We prefer to use a trans-oral anvil technique to create a circular-stapled anastomosis as previously described (Fig. 21.3) [62]. A small gastrotomy is made in the midline of the gastric pouch staple line using a harmonic scalpel. Another gastrotomy large enough to accommodate the circular stapler is also made in the anterior body of the gastric remnant along the greater curvature. The 90 cm polyvinyl chloride (PVS) delivery tube is then inserted into the patient's mouth and delivered through the gastrotomy on the gastric pouch. Once the 25-mm anvil has been exteriorized through the gastric pouch staple line, the delivery tube is removed and the stapler is inserted through the gastric remnant gastrostomy. The anastomosis is completed by joining the stapler and anvil. We typically use a 4.8 mm staple height to create our gastrogastrostomy. Reinforcing sutures can be placed at the corners of the anastomosis to reduce tension. Once the stapler and anvil are removed, the gastrostomy on the gastric body can be closed with multiple firings of a linear stapler. The gastrogastrostomy should be examined with a leak test (Fig. 21.4). Other papers have described a completely hand-sewn gastrogastrostomy in a single layer with running polydioxanone (PDS) suture or with a linear stapler using a running PDS suture to close the common enterotomy [41, 61].

If the patient has elected to undergo a reversal of a gastric bypass to modified sleeve gastrectomy, the sleeve gastrectomy can be performed after the creation of the gastrogastrostomy. In this

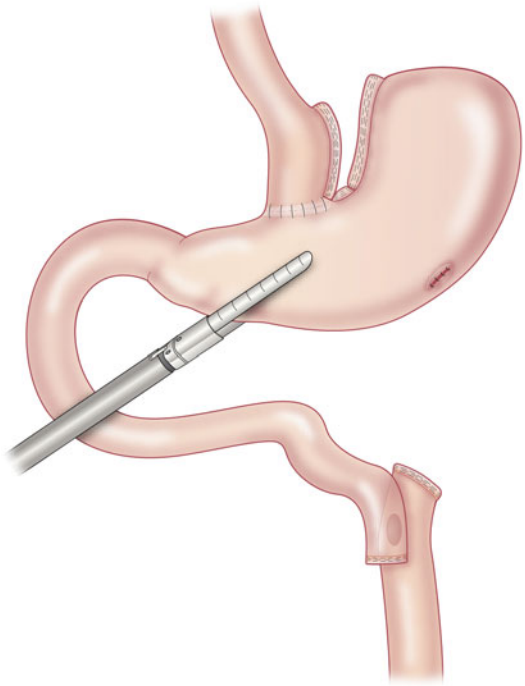

Fig. 21.5 Initial stapler placement for creation of a modified sleeve gastrectomy. With permission from Campos GM, Ziemelis M, Paparodis R, Ahmed M, Davis DB. Laparoscopic reversal of Roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. Surg Obes Relat Dis. 2014;10(1):36–43. doi:10.1016/j.soard.2013.05.012. Epub 2013 Jun 29 [95]. © Elsevier

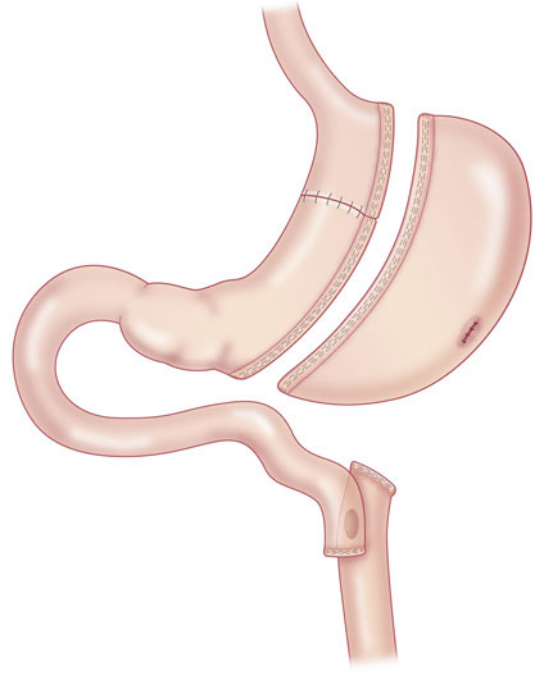

Fig. 21.6 Completed reversal to modified sleeve gastrectomy with removal of the excised portion of stomach. With permission from Campos GM, Ziemelis M, Paparodis R, Ahmed M, Davis DB. Laparoscopic reversal of Roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. Surg Obes Relat Dis. 2014;10(1):36–43. doi:10.1016/j.soard.2013.05.012. Epub 2013 Jun 29 [95]. © Elsevier

case, the gastric remnant needs to be completely dissected free from all adhesions. The gastroepiploic arcade is divided about 4 cm from the pylorus. A 12 mm (36 Fr) gastroscope is inserted under direct visualization through the gastrogastric anastomosis and the sleeve is then created using serial loads of a linear stapler (Fig. 21.5). We typically use 4.8 mm height staples on gastric antrum and 3.5 mm height staples on the gastric body and fundus. Care should be taken to avoid coming too close to the gastrogastric anastomosis with the sleeve staple line to decrease the risk of leak (Fig. 21.6).

Depending on the length of the common channel from the jejunojejunostomy to the ileocecal valve, the alimentary limb can be either saved or sacrificed. The alimentary limb is first divided from the jejunojejunostomy using a linear stapler. If the common channel is longer than 3 m, we typically resect the alimentary limb from its mesentery and remove it from the patient. If there is concern of the patient's length of small bowel, the alimentary limb can be saved by dividing the biliopancreatic limb from the jejunojejunostomy and anastomosing the distal aspect of the biliopancreatic limb to the proximal aspect of the alimentary limb, thus restoring the full length of the small intestine. This anastomosis can be created using either stapled or hand-sewn techniques. If the alimentary limb is saved, the mesenteric defect should be closed to prevent internal herniation of the small intestine [61].

On postoperative day number one, an upper GI radiologic examination is performed under fluoroscopic guidance using gastrografin followed by thin barium. Once the imaging has been reviewed

and no anastomotic leak is observed, the patient is started on a clear liquid diet. They are then discharged home once they are tolerating a pureed diet.

In our experience, we performed the laparoscopic reversal to either normal anatomy ($n=3$) or sleeve gastrectomy ($n=5$) in eight patients. Six of these patients had hyperinsulinemic hypoglycemia as their indication for surgery. All patients had a laparoscopic gastrostomy tube placed 8 to 12 weeks prior to reversal and had documented resolution of postprandial hypoglycemia on OGTT when fed via the G-tube. Our average operating time was 175 min (range 125–230 min) and our average length of hospital stay was 3 days (range 2–5 days). There were two early postoperative complications, including hemorrhage requiring blood transfusion and a superficial surgical site infection, and one late complication of a trocar site hernia. At mean 2 years follow-up (range 12 to 32 months), all patients had resolution of their hypoglycemia symptoms. Continuous glucose monitoring documented a decrease in the number of hypoglycemic events from 18.5 ± 12.4 to 1.5 ± 1.9 per week ($p=0.05$).

Another retrospective analysis looked at nine patients with hyperinsulinemic hypoglycemia who had reversal of a gastric bypass to either normal anatomy or sleeve gastrectomy with complete recovery of hypoglycemia documented on OGTT [41]. The authors also selected out the patients whose glucose metabolism normalized with a preoperative gastrostomy tube placed in the gastric remnant. Of note, they recorded a higher rate of complications with three staple line leaks with the addition of sleeve gastrectomy to the procedure. There have also been reports of patients undergoing reversal of RYGB to normal anatomy without improvement in symptoms [32]. In this report, unfortunately, neither patient underwent preoperative gastrostomy tube placement to confirm resolution of symptoms and their post-reversal hypoglycemia remained severe and refractory to medical management. These conflicting results suggest that further investigation into the etiology of hyperinsulinemic hypoglycemia is of paramount importance.

21.5 Dumping Syndrome

The alterations in anatomy and glucose metabolism after gastrectomy (including RYGB) can sometimes lead to a poorly defined constellation of various symptoms known as "dumping syndrome" [2, 46] which was first described by Mix in 1922 [63]. The incidence ranges anywhere from 15 to 76 % [46, 64]. Early dumping syndrome (75 %) is more common than late dumping syndrome (25 %) with only a few patients having symptoms of both [46] and the etiologies are thought to differ. Early dumping syndrome occurs within 15 to 30 min of eating and is thought to be related to the gastrointestinal response to a hyperosmolar food bolus. It is characterized by sudden onset of vasomotor symptoms, such as weakness, faintness, palpitations, diaphoresis, and an intense desire to lie down [7, 53]. This is followed by gastrointestinal symptoms such as abdominal bloating, cramping pain, and diarrhea which is attributed to the acute distention of the small intestine [1, 53]. Late dumping occurs 1.5 to 3 h after eating and is associated with hypoglycemia related to excessive insulin production [7]. Its symptoms are characterized by systemic, vascular symptoms that are related to hypoglycemia; these include diaphoresis, tachycardia, confusion, syncope, and neuroglycopenia [1, 53]. Some have proposed that dumping syndrome may partially contribute to weight loss after RYGB as it provides a negative reinforcement that deters patients from eating energy-dense foods [64]. Hyperinsulinemic hypoglycemia is often described as a severe form of late dumping syndrome [23], however most agree that hyperinsulinemic hypoglycemia as a unique and well-described syndrome which likely overlaps with "late dumping syndrome."

21.6 Conclusions

RYGB-related hyperinsulinemic hypoglycemia can cause severe and incapacitating symptoms which will become more common as the number of patients undergoing bariatric surgery increase. The etiology of the syndrome is complex and has

not been fully elucidated. One likely component is that the anatomic and physiologic changes related to RYGB lead to changes in the neurohormonal regulation of glucose metabolism including ability of the subject to mount a counter-regulatory response to hypoglycemia. Diet modification and medical therapies should be first line treatment for postprandial hyperinsulinemic hypoglycemia. However, if refractory hypoglycemia with neuroglycopenic symptoms persists despite maximal medical therapy, and a gastrostomy tube inserted in the gastric remnant confirms improvement of symptoms, reversal of the gastric bypass to either normal anatomy or modified sleeve gastrectomy can alleviate symptoms and improve quality of life.

References

1. Ukleja A. Dumping syndrome: pathophysiology and treatment. Nutr Clin Pract. 2005;20(5):517–25.
2. Ceppa EP, Ceppa DP, Omotosho PA, et al. Algorithm to diagnose etiology of hypoglycemia after Roux-en-Y gastric bypass for morbid obesity: case series and review of the literature. Surg Obes Relat Dis. 2012;8(5):641–7.
3. Choudhary P, Teh MM, Amiel SA. Methods of assessment of counterregulation to hypoglycaemia. In: Roden M, editor. Clinical diabetes research: methods and techniques. Hoboken, NJ: John Wiley & Sons, Ltd.; 2007.
4. Kellogg TA, Bantle JP, Leslie DB, et al. Postgastric bypass hyperinsulinemic hypoglycemia syndrome: characterization and response to a modified diet. Surg Obes Relat Dis. 2008;4(4):492–9.
5. Service FJ, Natt N, Thompson GB, et al. Noninsulinoma pancreatogenous hypoglycemia: a novel syndrome of hyperinsulinemic hypoglycemia in adults independent of mutations in Kir6.2 and SUR1 genes. J Clin Endocrinol Metab. 1999;84(5):1582–9.
6. Roslin M, Damani T, Oren J, et al. Abnormal glucose tolerance testing following gastric bypass demonstrates reactive hypoglycemia. Surg Endosc. 2011; 25(6):1926–32.
7. Deitel M. The change in the dumping syndrome concept. Obes Surg. 2008;18(12):1622–4.
8. Sawyers JL. Management of postgastrectomy syndromes. Am J Surg. 1990;159(1):8–14.
9. Rodieux F, Giusti V, D'Alessio DA, et al. Effects of gastric bypass and gastric banding on glucose kinetics and gut hormone release. Obesity (Silver Spring). 2008;16(2):298–305.
10. Mingrone G, Castagneto-Gissey L. Mechanisms of early improvement/resolution of type 2 diabetes after bariatric surgery. Diabetes Metab. 2009;35(6 Pt 2):518–23.
11. Laferrere B. Do we really know why diabetes remits after gastric bypass surgery? Endocrine. 2011;40(2):162–7.
12. Anderwald C-H, Tura A, Promintzer-Schifferl M, et al. Alterations in gastrointestinal, endocrine, and metabolic processes after bariatric Roux-en-Y gastric bypass surgery. Diabetes Care. 2012;35(12):2580–7.
13. Caiazzo R, Zerrweck C, Verhaeghe R, et al. Gastric bypass and glucose metabolism. Diabetes Metab. 2009;35(6 Pt 2):528–31.
14. Korner J, Inabnet W, Conwell IM, et al. Differential effects of gastric bypass and banding on circulating gut hormone and leptin levels. Obesity (Silver Spring). 2006;14(9):1553–61.
15. Rubino F, Gagner M, Marescaux J. Surgical treatment of type 2 diabetes mellitus. Lancet. 2001;358(9282):668–9.
16. Bikman BT, Zheng D, Pories WJ, et al. Mechanism for improved insulin sensitivity after gastric bypass surgery. J Clin Endocrinol Metab. 2008;93(12):4656–63.
17. Cummings DE. Gastric bypass and nesidioblastosis—too much of a good thing for islets? N Engl J Med. 2005;353(3):300–2.
18. Korner J, Inabnet W, Febres G, et al. Prospective study of gut hormone and metabolic changes after adjustable gastric banding and Roux-en-Y gastric bypass. Int J Obes (Lond). 2009;33(7):786–95.
19. Baggio LL, Drucker DJ. Biology of incretins: GLP-1 and GIP. Gastroenterology. 2007;132(6):2131–57.
20. Adams TD, Gress RE, Smith SC, et al. Long-term mortality after gastric bypass surgery. N Engl J Med. 2007;357(8):753–61.
21. Buchwald H, Estok R, Fahrbach K, et al. Weight and type 2 diabetes after bariatric surgery: systematic review and meta-analysis. Am J Med. 2009;122(3): 248–56.
22. Adams TD, Pendleton RC, Strong MB, et al. Health outcomes of gastric bypass patients compared to nonsurgical, nonintervened severly obese. Obesity (Silver Spring). 2010;18(1):121–30.
23. Service GJ, Thompson GB, Service FJ, et al. Hyperinsulinemic hypoglycemia with nesidioblastosis after gastric-bypass surgery. N Engl J Med. 2005;353(3):249–54.
24. Clancy TE, Moore FD, Zinner MJ. Post-gastric bypass hyperinsulinism with nesidioblastosis: subtotal or total pancreatectomy may be needed to prevent recurrent hypoglycemia. J Gastrointest Surg. 2006;10(8):1116–9.
25. Patti ME, McMahon G, Mun EC, et al. Severe hypoglycaemia post-gastric bypass requiring partial pancreatectomy: evidence for inappropriate insulin secretion and pancreatic islet hyperplasia. Diabetologia. 2005;48(11):2236–40.
26. Alvarez GC, Faria EN, Beck M, et al. Laparoscopic spleen-preserving distal pancreatectomy as treatment for nesidioblastosis after gastric bypass surgery. Obes Surg. 2007;17(4):550–2.
27. Meier JJ, Butler AE, Galasso R, et al. Hyperinsulinemic hypoglycemia after gastric bypass surgery is not accompanied by islet hyperplasia or increased beta-cell turnover. Diabetes Care. 2006;29(7):1554–9.

28. Verhaeghe R, Zerrweck C, Hubert T, et al. Gastric bypass increases postprandial insulin and GLP-1 in nonobese minipigs. Eur Surg Res. 2014;52(1–2):41–9.
29. Salehi M, Prigeon RL, D'Alessio DA. Gastric bypass surgery enhances glucagon-like peptide 1-stimulated postprandial insulin secretion in humans. Diabetes. 2011;60(9):2308–14.
30. Korner J, Bessler M, Inabnet W, et al. Exaggerated glucagon-like peptide-1 and blunted glucose-dependent insulinotropic peptide secretion are associated with Roux-en-Y gastric bypass but not adjustable gastric banding. Surg Obes Relat Dis. 2007;2007(3):6.
31. Rabiee A, Magruder JT, Salas-Carrillo R, et al. Hyperinsulinemic hypoglycemia after Roux-en-Y gastric bypass: unraveling the role of gut hormonal and pancreatic endocrine dysfunction. J Surg Res. 2011;2011(167):2.
32. Lee CJ, Brown T, Magnuson TH, et al. Hormonal response to a mixed-meal challenge after reversal of gastric bypass for hypoglycemia. J Clin Endocrinol Metab. 2013;98(7):E1208–12.
33. Laferrere B, Teixeira J, McGinty J, et al. Effect of weight loss by gastric bypass surgery versus hypocaloric diet on glucose and incretin levels in patients with type 2 diabetes. J Clin Endocrinol Metab. 2008;93(7):2479–85.
34. Reubi JC, Perren A, Rehmann R, et al. Glucagon-like peptide (GLP-1) receptors are not overexpressed in pancreatic islets from patients with severe hyperinsulinaemic hypoglycaemia following gastric bypass. Diabetologia. 2010;53(12):2641–5.
35. Salehi M, Gastaldelli A, D'Alessio DA. Blockage of glucagon-like peptide 1 receptor corrects postprandial hypoglycemia after gastric bypass. Gastroenterology. 2014;146(3):669–80.
36. Goldfine AB, Mun EC, Devine E, et al. Patients with neuroglycopenia after gastric bypass surgery have exaggerated incretin and insulin secretory responses to a mixed meal. J Clin Endocrinol Metab. 2007;92(12):4678–85.
37. Campos GM, Rabl C, Peeva S, et al. Improvement in peripheral glucose uptake after gastric bypass surgery is observed only after substantial weight loss has occurred and correlates with the magnitude of weight loss. J Gastrointest Surg. 2010;14(1):15–23.
38. Patti ME, Goldfine AB. Hypoglycemia after gastric bypass: the dark side of GLP-1. Gastroenterology. 2014;146(3):605–8.
39. Davis DB, Khoraki J, Campos GM. Recalcitrant hypoglycemia with neuroglycopenia after Roux-en-Y gastric bypass is caused by postprandial changes in glucose, gut and pancreatic hormone responses to altered feeding route, not nesidioblastosis. Scientific Papers Session. 31st Annual Meeting. American Society for Bariatric and Metabolic Surgery. Boston, MA; 2014.
40. Campos GM, Ziemelis M, Paparodis R, et al. Laparoscopic reversal of Roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. Surg Obes Relat Dis. 2014;10(1):36–43.
41. Vilallonga R, van de Vrande S, Himpens J. Laparoscopic reversal of Roux-en-Y gastric bypass into normal anatomy with or without sleeve gastrectomy. Surg Endosc. 2013;27(12):4640–8.
42. Cryer PE, Axelrod L, Grossman AB, et al. Evaluation and management of adult hypoglycemic disorders: an Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2009;94(3):709–28.
43. Lev-Ran A, Anderson RW. The diagnosis of postprandial hypoglycemia. Diabetes. 1981;30(12):996–9.
44. Service FJ. Hypoglycemic disorders. N Engl J Med. 1995;332(17):1144–52.
45. Mordes JP, Alonso LC. Evaluation, medical therapy, and course of adult persistent hyperinsulinemic hypoglycemia after Roux-en-Y gastric bypass surgery: a case series. Endocr Pract. 2014;1–34 (Epub ahead of print).
46. Scarpignato C. The place of octreotide in the medical management of the dumping syndrome. Digestion. 1996;57 Suppl 1:114–8.
47. McLoughlin JC, Buchanan KD, Alam MJ. A glycoside-hydrolase inhibitor in treatment of dumping syndrome. Lancet. 1979;314(8143):603–5.
48. Moreira RO, Moreira RB, Machado NAM, et al. Postprandial hypoglycemia after bariatric surgery: pharmacological treatment with verapamil and acarbose. Obes Surg. 2008;18(12):1618–21.
49. Arao T, Okada Y, Hirose A, et al. A rare case of adult-onset nesidioblastosis treated successfully with diazoxide. Endocr J. 2006;53(1):95–100.
50. Ritz P, Vaurs C, Bertrand M, et al. Usefulness of acarbose and dietary modifications to limit glycemic variability following Roux-en-Y gastric bypass as assessed by continuous glucose monitoring. Diabetes Technol Ther. 2012;14(8):736–40.
51. Valderas JP, Ahuad J, Rubio L, et al. Acarbose improves hypoglycaemia following gastric bypass surgery without increasing glucagon-like peptide 1 levels. Obes Surg. 2012;22(4):582–6.
52. Frankhouser SY, Ahmad AN, Perilli GA, et al. Post-gastric-bypass hypoglycemia successfully treated with alpha-glucosidase inhibitor therapy. Endocr Pract. 2013;19(3):511–4.
53. Geer RJ, Richards WO, O'Dorisio TM, et al. Efficacy of octreotide acetate in treatment of severe postgastrectomy dumping syndrome. Ann Surg. 1990;212(6):678–87.
54. de Heide LJ, Laskewitz AJ, Apers JA. Treatment of severe postRYGB hyperinsulinemic hypoglycemia with pasireotide: a comparison with octreotide on insulin, glucagon, and GLP-1. Surg Obes Relat Dis. 2014;10(3):e31–3.
55. Myint KS, Greenfield JR, Farooqi IS, et al. Prolonged successful therapy for hyperinsulinaemic hypoglycaemia after gastric bypass: the pathophysiological role of GLP1 and its response to a somatostatin analogue. Eur J Endocrinol. 2012;166(5):951–5.
56. Tabibian N. Successful treatment of refractory post-vagotomy syndrome with verapamil (Calan SR). Am J Gastroenterol. 1990;85(3):328–9.

57. Fernandez-Esparrach G, Lautz DB, Thompson CC. Peroral endoscopic anastomotic reduction improves intractable dumping syndrome in Roux-en-Y gastric bypass patients. Surg Obes Relat Dis. 2010;6(1):36–40.
58. Leitman IM, Virk CS, Avgerinos DV, et al. Early results of trans-oral endoscopic plication and revision of the gastric pouch and stoma following Roux-en-Y gastric bypass surgery. JSLS. 2010;14(2):217–20.
59. Vanderveen KA, Grant CS, Thompson GB, et al. Outcomes and quality of life after partial pancreatectomy for noninsulinoma pancreatogenous hypoglycemia from diffuse islet cell disease. Surgery. 2010;148(6):1237–45.
60. Himpens J, Dapri G, Cadiere GB. Laparoscopic conversion of the gastric bypass into a normal anatomy. Obes Surg. 2006;16(7):908–12.
61. Dapri G, Cadiere GB, Himpens J. Laparoscopic reconversion of Roux-en-Y gastric bypass to original anatomy: technique and preliminary outcomes. Obes Surg. 2011;21(8):1289–95.
62. Campos GM, Jablons D, Brown LM, Ramirez RM, et al. A safe and reproducible anastomotic technique for minimally invasive Ivor Lewis oesophagectomy: the circular-stapled anastomosis with the trans-oral anvil. Eur J Cardiothorac Surg. 2010;37(6):1421–6.
63. Mix CL. "Dumping stomach" following gastrojejunostomy. Surg Clin North Am. 1922;2:617–22.
64. Banerjee A, Ding Y, Mikami DJ, et al. The role of dumping syndrome in weight loss after gastric bypass surgery. Surg Endosc. 2013;27(5):1573–8.
65. Lips MA, de Groot GH, van Klinken JB, et al. Calorie restriction is a major determinant of the short-term metabolic effects of gastric bypass surgery in obese type 2 diabetic patients. Clin Endocrinol (Oxf). 2014;80(6):834–42.
66. Isbell JM, Tamboli RA, Hansen EN, et al. The importance of caloric restriction in the early improvements in insulin sensitivity after Roux-en-Y gastric bypass surgery. Diabetes Care. 2010;33(7):1438–42.
67. Lingvay I, Guth E, Islam A, et al. Rapid improvement in diabetes after gastric bypass surgery: is it the diet or surgery? Diabetes Care. 2013;36(9):2741–7.
68. Swarbrick MM, Stanhope KL, Austrheim-Smith IT, et al. Longitudinal changes in pancreatic and adipocyte hormones following Roux-en-Y gastric bypass surgery. Diabetologia. 2008;51(10):1901–11.
69. Olbers T, Bjorkman S, Lindroos A, et al. Body composition, dietary intake, and energy expenditure after laparoscopic Roux-en-Y gastric bypass and laparoscopic vertical banded gastroplasty: a randomized clinical trial. Ann Surg. 2006;244(5):715–22.
70. Tamboli RA, Hossain HA, Marks PA, et al. Body composition and energy metabolism following Roux-en-Y gastric bypass surgery. Obesity (Silver Spring). 2010;18(9):1718–24.
71. Miller GD, Carr JJ, Fernandez AZ. Regional fat changes following weight reduction from laparoscopic Roux-en-Y gastric bypass surgery. Diabetes Obes Metab. 2011;13(2):189–92.
72. Immonen H, Hannukainen JC, Iozzo P, et al. Effect of bariatric surgery on liver glucose metabolism in morbidly obese diabetic and non-diabetic patients. J Hepatol. 2014;60(2):377–83.
73. Lin E, Phillips LS, Ziegler TR, et al. Increases in adinopectin predict improved liver, but not peripheral, insulin sensitivity in severly obese women during weight loss. Diabetes. 2007;56(3):735–42.
74. Malin SK, Bena J, Abood B, et al. Attenuated improvements in adinopectin and fat loss characterize type 2 diabetes non-remission status after bariatric surgery. Diabetes Obes Metab. 2014;16(12):1230–8.
75. Geloneze B, Tambascia MA, Pareja JC, et al. Serum leptin levels after bariatric surgery across a range of glucose tolerance from normal to diabetes. Obes Surg. 2001;11(6):693–8.
76. Camastra S, Gastaldelli A, Mari A, et al. Early and longer term effects of gastric bypass surgery on tissue-specific insulin sensitivity and beta cell function in morbidly obese patients with and without type 2 diabetes. Diabetologia. 2011;54(8):2093–102.
77. Dunn JP, Abumrad NN, Breitman I, et al. Hepatic and peripheral insulin sensitivity and diabetes remission at 1 month after Roux-en-Y gastric bypass surgery in patients randomized to omentectomy. Diabetes Care. 2012;35(1):137–42.
78. Bojsen-Moller KN, Dirksen C, Jorgensen NB, et al. Increased hepatic insulin clearance after Roux-en-Y gastric bypass. J Clin Endocrinol Metab. 2013;98(6):E1066–71.
79. Bojsen-Moller KN, Dirksen C, Jorgensen NB, et al. Early enhancements of hepatic and later of peripheral insulin sensitivity combined with increased postprandial insulin secretion contribute to improved glycemic control after Roux-en-Y gastric bypass. Diabetes. 2014;63(5):1725–37.
80. Nannipieri M, Baldi S, Colligiani D, et al. Roux-en-Y gastric bypass and sleeve gastrectomy: mechanisms of diabetes remission and role of gut hormones. J Clin Endocrinol Metab. 2013;98(11):4391–9.
81. Thaler JP, Cummings DE. Minireview: hormonal and metabolic mechanisms of diabetes remission after gastrointestinal surgery. Endocrinology. 2009;150(6):2518–25.
82. Cummings DE, Weigle DS, Frayo RS, et al. Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. N Engl J Med. 2002;346(21):1623–30.
83. Umeda LM, Silva EA, Carneiro G, et al. Early improvement in glycemic control after bariatric surgery and its relationships with insulin, GLP-1, and glucagon secretion in type 2 diabetic patients. Obes Surg. 2011;21(7):896–901.
84. Kashyap SR, Daud S, Kelly KR, et al. Acute effects of gastric bypass versus gastric restrictive surgery on beta-cell function and insulinotropic hormones in severely obese patients with type 2 diabetes. Int J Obes (Lond). 2010;34(3):462–71.
85. Weiss R. Effects of Roux-en-Y gastric bypass on β-cell function. Diabetes. 2014;63(4):1171–3.

86. Ferrannini E, Mingrone G. Impact of different bariatric surgical procedures on insulin action and beta-cell function in type 2 diabetes. Diabetes Care. 2009;32(3):514–20.
87. Rabl C, Rao MN, Schwarz JM, et al. Thermogenic changes after gastric bypass, adjustable gastric banding or diet alone. Surgery. 2014;156(4):806–12.
88. Das SK, Roberts SB, McCrory MA, et al. Long-term changes in energy expenditure and body composition after massive weight loss induced by gastric bypass surgery. Am J Clin Nutr. 2003;78(1):22–30.
89. Liou AP, Paziuk M, Luevano JM, Machineni S, et al. Conserved shifts in the gut microbiota due to gastric bypass reduce host weight and adiposity. Sci Transl Med. 2013;5(178):178ra41.
90. Vrieze A, Van Nood E, Holleman F, et al. Transfer of intestinal microbiota from lean donors increases insulin sensitivity in individuals with metabolic syndrome. Gastroenterology. 2012;143(4):913–6.
91. Sweeney TE, Morton JM. Metabolic surgery: action via hormonal milieu changes, changes in bile acids or gut microbiota? A summary of the literature. Best Pract Res Clin Gastroenterol. 2014;28(4):727–40.
92. Patti ME, Houten SM, Bianco AC, et al. Serum bile acids are higher in humans with prior gastric bypass: potential contribution to improved glucose and lipid metabolism. Obesity (Silver Spring). 2009;17(9):1671–7.
93. Pournaras DJ, Glicksman C, Vincent RP, et al. The role of bile after Roux-en-Y gastric bypass in promoting weight loss and improving glycaemic control. Endocrinology. 2012;153(8):3613–9.
94. Carswell KA, Vincent RP, Belgaumkar AP, et al. The effect of bariatric surgery on intestinal absorption and transit time. Obes Surg. 2014;24(5):796–805.
95. Campos GM, Ziemelis M, Paparodis R, Ahmed M, Davis DB. Laparoscopic reversal of Roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. Surg Obes Relat Dis. 2014;10(1):36–43. doi:10.1016/j.soard.2013.05.012. Epub 2013 Jun 29.

Nutritional Complications and Emergencies

22

Samuel Szomstein and David M. Nguyen

Key Points

- Long-term or lifelong follow-up of postoperative bariatric patients is critical in successful management as nutritional supplementation does not necessarily prevent deficiencies.
- While type of surgical procedure is a major consideration, nutritional deficiencies can result from patient factors including postoperative diet tolerance, behavioral modifications, and level of adherence to supplementation.
- Preoperative screening of nutritional status with appropriate treatment is imperative in bariatric postoperative success.

22.1 Preoperative and Postoperative Considerations

Nutritional complications and emergencies following bariatric surgery are complex in nature due to both patient factors and post-surgical physiology. Postoperative nutritional status depends upon nutritional status preceding obesity surgery, dietary modifications, food intolerance or overindulgence, and noncompliance with multivitamin supplementation, which increases the rate of postoperative deficiency twofold [1]. Both the malabsorptive and restrictive features of bariatric surgery affect the severity of complications. However, nutritional deficiencies commonly associated with a malabsorptive or restrictive procedure are not exclusive to the respective procedure type. In addition, recognition of certain deficiencies must be prompt, since delayed diagnosis and treatment may lead to adverse and potentially permanent health consequences.

Bariatric surgery success is largely dependent on nutritional management (i.e., routine monitoring, nutritional counseling, and behavior modification) in the preoperative as well as the postoperative phase. Given the high prevalence of preexisting deficiencies in the obese population, preoperative screening of nutritional deficiency serves as a critical component of the bariatric workup.

Although increasingly questioned, restriction and malabsorption have historically been considered the primary mechanisms driving metabolic improvements after bariatric surgery [2]. Weight loss appears also to be due to increased satiety by gut hormones, and malnutrition is intricately influenced by these hormones. In gastric bypass and duodenal switch procedures, nutrient

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_22

deficiency may be proportional to the length of the absorptive area bypassed. Vitamin B complexes along with iron, calcium, and vitamin D are commonly deficient after Roux-en-Y gastric bypass (RYGB). Biliopancreatic diversion with or without duodenal switch (BPD/DS) increases the risk of protein, fat-soluble vitamins A, D, E, and K, and as well as calcium deficiency. Folate deficiency is commonly reported following adjustable gastric banding (AGB) [3]. Increasing evidence of hypovitaminosis has also been reported after laparoscopic sleeve gastrectomy (SG), particularly with the B-complex vitamins [4].

Vitamins and minerals are involved in enzymatic reactions for homeostasis, metabolism, and neuronal functions, including long-term weight management. Nutritional supplementation does not always prevent nutritional deficiencies as these are seen in both supplemented and non-supplemented patients. Thus, long-term biochemical follow-up of post-bariatric patients is essential for early recognition and treatment of nutritional deficiencies. In general, at least 100 % of the recommend daily dose of multivitamins-/minerals is needed in AGB, and 200 % of the daily value for RYGB, BPD with or without duodenal switch, and SG [5].

This section describes key aspects in the nutritional care of bariatric patients in hopes of increasing awareness of potential nutritional complications commonly seen after obesity surgery. Specific macronutrient and micronutrient deficiencies and related complications are discussed with postoperative management (as summarized in Table 22.1). Emphasis is placed on preoperative screening and early recognition.

22.2 B_1 (Thiamine)

Thiamine, a water-soluble vitamin, is found abundantly in meat and grain products. A deficiency of thiamine is commonly referred to as beriberi. Bariatric beriberi, a condition preceding the more severe Wernicke's encephalopathy, Korsakoff's syndrome, and Wernicke–Korsakoff syndrome, has its highest prevalence within the first three months following a bariatric operation. Preoperative and postoperative prevalence are reported to be as high as 29 and 49 % respectively [6]. Preoperative deficiency is most commonly seen in African Americans and Hispanics. Etiologic factors are multifaceted and include non-adherence to supplementation, food intolerance, rapid weight loss, and poor hydration. In small number of cases where thiamine substitution is ineffective in clinical resolution, this may be caused by intestinal bacterial overgrowth, which results in malabsorption of thiamine [7]. AGB and laparoscopic sleeve gastrectomy are risk factors for such a deficiency as recurrent vomiting may persistently occur after these procedures. Intravenous glucose infusion before vitamin B_1 administration also presents risk to beriberi.

The fact that the body's store of thiamine is only 30 mg with a half-life of 9–18 days explains why severe depletion can occur in such a short period with a very rapid onset of symptoms. Because the clinical presentation of beriberi is highly variable, regular laboratory evaluation of whole blood thiamine levels may be helpful for its diagnosis. Early postoperative factors leading to thiamine deficiency include nausea, food intolerance, and decreased oral intake with vomiting and constipation. This condition is historically classified into wet beriberi and dry beriberi. Dry beriberi involves the central and peripheral nervous system whereas the wet form involves the cardiovascular system. Patients with dry beriberi exhibit symptoms of paraplegia, ataxia, and peripheral neuritis, typically with an onset 4 weeks or more after operation.

Thiamine levels are measured by erythrocyte transketolase activity assay or by urine and plasma thiamin levels. It should be noted that serum thiamine responds to dietary supplementation but poorly reflects total body stores. The best single test to assess whole body thiamine is whole blood (or RBC) thiamine. Due to practical constraints of availability, post-bariatric patients displaying signs and symptoms of beriberi may begin treatment without laboratory confirmation. In patients with symptoms suggestive of nervous system involvement, head CT or MRI should be performed.

Table 22.1 Clinical manifestations and treatment of severe nutritional deficiency following bariatric surgery

Nutrient	Clinical manifestations	Treatment for severe deficiency
B_1 (thiamine)	Dry beriberi, wet beriberi, Wernicke's encephalopathy, Korsakoff's syndrome, Wernicke–Korsakoff syndrome	Wernicke encephalopathy/acute psychoses: intravenous thiamine 500 mg three times per day up to 5 days followed by intravenous thiamine 250 mg daily
Vitamin B_6	Sideroblastic anemia, dermatitis, glossitis, angular cheilitis, conjunctivitis, neuropathy, hyperhomocystinemia	Oral vitamin B_6 up to 100 mg daily
Folic acid (B_9)	Macrocytic, megaloblastic anemia, palpitations, irritability, no neurologic abnormalities	Up to 5 mg folic acid per day orally up to 3 months
B_{12} (cobalamin)	Pernicious anemia, myelosis funicularis	Intramuscular vitamin B_{12} 1000 μg daily for 1 week followed by weekly injections of 1000 μg for 1 month. Monthly intramuscular 1000 μg for life is recommended
Iron	Anemia, fatigue, irritability, pallor, brittle nails, Plummer–Vinson syndrome, restless legs syndrome	High molecular intravenous ferric carboxymaltose 1000 mg followed by intravenous 500 mg up to the calculated dose weekly[a]
Vitamin D	Osteomalacia, osteoporosis, arthralgia, myalgia, fasciculation, depression	50,000–150,000 IU vitamin D3 orally per day with oral calcitriol
Calcium	Oral, perioral, and acral paresthesias; petechiae; tetany; cardiac arrhythmias; intermittent QT prolongation; neuromuscular hyperexcitability	DEXA T-score of <2.5, intravenous bisphosphonates
Vitamin A	Nyctalopia, xerophthalmia, pruritis, dry hair	Corneal lesions: intramuscular vitamin A 50,000–100,000 IU followed by 50,000 IU per day intramuscularly for 2 weeks
Vitamin E	Spinocerebellar ataxia, dysarthria, anemia, retinopathy	Oral vitamin E 800–1200 IU per day
Vitamin K	Petechiae, hematoma (at surgical site), delayed blood clotting; osteoporosis, heavy menstrual	Intramuscular vitamin K 10 mg followed by 2 mg orally per week
Zinc	Taste abnormalities, hypogeusia, alterations in sense of smell, hair loss, glossitis, eye and skin lesions, poor wound healing	Oral 60 mg zinc sulfate twice a day with 1 mg copper given for each 8–15 mg zinc received
Copper	Normochromic anemia, myelopathy peripheral neuropathy, optic neuropathy	2.4 mg copper sulfate mixed in 100 mL of normal saline intravenously over 4 h daily for 5 days
Selenium	Cardiac myositis, hypothyroidism, goiter	Oral selenium up to 200 mcg daily
Magnesium	Muscle contractions and fasciculations, osteoporosis, tetany seizures, coronary spasms, cardiac arrhythmia, hypocalcemia, hypokalemia	4 g in 250 mL dextrose 5 % water intravenous infusion daily with no more than 3 mL per minute
Protein	Decreased lean tissue mass, weakness, hair loss, generalized edema	Parenteral nutrition; surgical revision

[a]Iron dosage based on hemoglobin levels and body weight

Hemoglobin (g/dL)	Body weight < 70 kg	Body weight ≥ 70 kg
≥10.0	1000 mg	1500 mg
7.0–10.0	1500 mg	2000 mg
<7.0	2000 mg	2500 mg

While many surgeons suggest following patients after surgery with whole blood thiamine levels at 6–12 month intervals surveillance for 3 years, consensus in monitoring guidelines for thiamine deficiency is lacking. If patients present with symptoms of Wernicke encephalopathy or acute psychoses, inpatient intravenous thiamine under close monitoring is recommended with a dose of 500 mg three times per day up to 5 days. This should be followed by intravenous thiamine 250 mg daily until clinical improvement of oculomotoric symptoms [8, 9]. Water-soluble vitamins and magnesium deficiency must be treated simultaneously.

Anamnestic and gait disorders resulting from severe beriberi are seen to be irreversible in over 50 % of patients. Central nervous system damage may even lead to coma from delayed treatment. Subclinical cases in patients with high suspicions of thiamine deficiency or early symptoms of neuropathy can be treated with 100 mg of oral thiamine daily until symptoms resolve. Bariatric candidates with preoperative marginal thiamine level can be given 100 mg oral thiamine twice daily until levels are normalized (10–64 μg/L) [10]. For prevention after surgery, standard multivitamins are sufficient. When recurrent vomiting is experienced postoperatively, oral thiamine 100 mg twice daily for 1 month is needed. Although deficiency is rare, these key points of thiamine deficiency and treatment are essential for bariatric surgeons in both large and small volume centers.

22.3 Vitamin B_6

Vitamin B_6 is found in a wide range of food sources from meats to vegetables. The active form of vitamin B_6 is pyridoxal 5′-phosphate (PLP), which is vital in amino acid, glucose, and lipid metabolism. Vitamin B_6 is mainly absorbed in the jejunum and ileum through passive diffusion. B_6 deficiency is characterized by dermatitis, glossitis, angular cheilitis, conjunctivitis, neuropathy from impaired sphingosin synthesis, and impaired heme synthesis resulting in sideroblastic anemia. Vitamin B_6 deficiency should be considered when there is unresolved anemia in pre- and post-bariatric patients. The preoperative and postoperative prevalence remain poorly understood, since this deficiency is considered rare and thus the vitamin is not routinely measured. Vitamin supplementation appears to be effective in maintaining B_6 levels before and after obesity surgery. However, serum PLP levels may not be representative of vitamin B_6 status in patients on standard recommended multivitamin with normal PLP levels [11]. RBC glutamic pyruvate transaminase as a marker for B_6 instead of serum PLP has demonstrated deficiency in post-bariatric patients, suggesting greater dosage of the recommended amount may be required. Oral vitamin B_6 up to 100 mg daily may be used to treat deficiency.

22.4 Folic Acid

A water-soluble vitamin and member of the vitamin B group, folic acid (often referred to as folate, its anion form) is also known as vitamin B_9 and plays critical roles in prevention of congenital neural tube defects. Rich sources of folate can be found in vegetables, particularly spinach and fruits, as well as in grains and liver. Deficiency may occur due to low intake and poor adherence to supplementation rather than from malabsorption, as folate is well-absorbed through both the small intestine and colon. Thus, deficiency can simply be corrected by oral supplementation [12]. Patients should also be educated that certain medications such as anticonvulsants, oral contraceptives, and cancer agents can cause folate deficiency.

The prevalence of folate deficiency is relatively low, up to 10 % preoperatively and 38 % postoperatively. Folate deficiency is particularly common in female bariatric patients during pregnancy [12, 13]. Unlike thiamine and zinc where body stores may last for years, folate stores are minimal and deficiency can appear early in the postoperative phase.

Macrocytic anemia, palpitations, irritability, hostility, and fatigue are clinical abnormalities seen with folate deficiency. Deficiency is detected

by measuring red blood cell (RBC) folate levels. Serum folate can vary and reflects dietary intake rather than providing a true representation of whole-body folate status. RBC folate has proven to be a more appropriate and sensitive marker since red blood cells store 95 % of circulating folate [14].

The recommended dosage to treat deficiency after obesity surgery is up to 5 mg per day orally for up to 3 months. Gastric bypass patients on this regimen have shown to maintain normal serum levels while clear deficiency is seen in non-adherent patients. Prevention in the postoperative period is achieved by a routine multivitamin preparation consisting of at least 800 μg of folic acid daily, particularly in women of childbearing age. In contrast to iron and vitamin B_{12}, folate contained in multivitamin preparations is sufficient to alleviate deficiency. Persistent deficiency indicates poor adherence to prescribed supplementation. It should be mentioned that excessive folic acid supplementation is a risk for vitamin B_{12} deficiency as clinical signs and symptoms can be masked with persistence of neurologic injury. Thus, supplementation greater than 1000 mg per day is not recommended. In cases of high suspicion, homocysteine is the most sensitive marker for folate deficiency. Additionally, folate deficiency can result from vitamin B_{12} deficiency as B_{12} is essential in the production of active tetrahydrofolic acid [15]. Abstinence from alcohol is encouraged during treatment for deficiency as alcohol interferes with folate absorption.

22.5 B_{12} (Cobalamin)

Vitamin B_{12} (cobalamin), found in fish, meat, and dairy, is critical for DNA synthesis. Deficiency can lead to both hematologic and neuropsychiatric pathologies. After iron deficiency, Vitamin B_{12} deficiency is the second most common cause of anemia in deficient post bariatric patients. Due to the gastric and intestinal mechanisms of gastric acid and intrinsic factor in vitamin B_{12} absorption, deficiency after gastric band or sleeve gastrectomy is rare. However, the prevalence of B_{12} deficiency may be higher in malabsorptive procedures; deficiency has been noted in up to 35 % of patients 5 years after RYGB and up to 62 % of patients 2 years after BPD-DS [16]. These procedures are characterized by incomplete digestion and decreased production of hydrochloric acid, which is necessary for B_{12} separation from food sources. Preoperative deficiency has been reported in up to 18 % [17]. Although human body storage of B_{12} is adequate to maintain levels for years, vegetarians or vegans, obese patients commonly on proton pump inhibitors (or H_2 blockers) and metformin, and individuals with intestinal bacterial overgrowth are particularly prone to becoming deficient. Obesity is associated with a 4.3 fold increased in risk of B_{12} deficiency. Each unit increase in BMI was demonstrated to result in an increased risk of 1.24 [18].

Early symptoms of B_{12} deficiency include paresthesias and numbness of the limbs. Concentration disturbances and depression may also manifest early. Symptoms may ultimately irreversibly progress to unsteady gait and dementia. The American Society for Metabolic and Bariatric Surgery (ASMBS) recommends annual B_{12} screening in post-bariatric patients who underwent procedures excluding the lower stomach. In healthy individuals with no history of obesity surgery, only 10 mcg of 500 mcg oral B_{12} supplement is absorbed. Due to the fact that intrinsic factor and gastric acid are produced in the lower stomach, gastric bypass patients require oral crystalline B_{12} supplementation.

Serum B_{12} measurement is known to inaccurately detect deficiency in up to 30 %, and thus, high suspicion is required in the presence of symptoms and signs [19]. Serum homocysteine is the test of choice due to its high sensitivity. The methylmalonic acid serum test is also excellent, reported to be greater than 98 % specific and sensitive for B_{12} deficiency, and can be used in combination with the serum homocysteine test. For therapy, the timing and dosage of vitamin B_{12} are wide ranging. Treatment must account for the fact that symptoms manifest when body stores (5 mg) of B_{12} decrease to 10 % of normal. Vitamin B_{12} intramuscular injection of 1000 μg daily for 1 week followed by weekly injections of 1000 μg

for 1 month is recommended for rapid repletion in post-bariatric patients. Monthly 1000 μg injection for life could be recommended, and neurologic disease may be irreversible [20]. Preoperative B_{12} deficient patients may follow the abovementioned dosage for repletion preceding surgery. For maintenance postoperatively from RYGB and BPD/DS, the ASMBS recommends intramuscular 1000 μg per month or crystalline oral 350–500 μg per day for 3 months after surgery. In B_{12} vitamin deficient patients on folate acid supplementation, there should be critical awareness that excessive folic acid supplements can mask B_{12} deficiency. This can lead to exacerbation of neurological deterioration.

22.6 Iron

In general, iron deficiency is the most common nutritional deficiency with 9–16 % of adult women in the general population affected. This trace element is abundantly found in red meat, poultry, and leaf vegetables. Dissociation of the duodenum from chyme, food intolerance of red meat, decreased gastric acid, and anemia of chronic disease are contributing factors. Iron deficiency is found in up to 18 % of preoperative bariatric patients. Preoperative and postoperative deficiency was shown to be statistically significantly more common in men (35.5–40.7 %) than in women (14–19.1 %) [10]. As men have higher levels of cytokine and leptin than women, this contributes to hepcidin synthesis that results in decreased iron absorption. Iron deficiency is considered a long-term complication from obesity surgery that appears frequently in 20–49 % of patients. Two-year postoperative prevalence is demonstrated to be 17 % after LSG and 30 % after vertical banded gastroplasty (VBG), BPD, or RYGB. After 5 years, the prevalence rises to 45 % from RYGB and BPD [21]. In addition, many superobese patients and 51 % of menstruating women who underwent RYBG are also found to be iron deficient [22].

Signs and symptoms of iron deficiency are non-pathognomonic in nature. These include anemia, fatigue, irritability, pallor, brittle nails, Plummer–Vinson syndrome, and restless leg syndrome. Screening consists mainly of serum ferritin, serum iron, and total iron binding capacity (TIBC). It should be mentioned that ferritin, an acute phase reactant, is elevated with active disease or inflammation, including the common cold. Additional laboratory indexes can include hemoglobin and hematocrit levels. Therapy for iron deficiency involves iron supplements. The type of supplement depends upon the severity and the required speed of improvement. Parenteral iron administration (e.g., high molecular intravenous ferric carboxymaltose) is recommended for rapid responses in post-bariatric treatment. Infusion should begin with 1000 mg, then 500 mg up to the calculated dose weekly. This iron dose strategy is based on hemoglobin levels and body weight and has been shown by Evstatiev et al. to be safe and effective [23]. Due to dissociation of the duodenum from bypass surgery, oral iron is likely to be relatively ineffective, and may be associated with abdominal pain, nausea, and diarrhea. Of note, oral contraceptives can reduce blood loss in menstruating females and may be a helpful adjunct in treatment. In women with preexisting use of oral contraceptives, they may have lower requirements for iron supplementation. As preventative maintenance following obesity surgery, oral iron supplementation with oral ferrous sulfate 300 mg two times per day is used in many programs, although organic or chelated iron formulations may be better absorbed and tolerated. In low risk patients-men and postmenopausal women-, using complete multivitamins with 36 mg of ferrous sulfate may be sufficient [24]. Serum iron and TIBC should be screened at 6 months postoperatively and then annually.

22.7 Vitamin D

While not abundant in food sources, sun exposure on human skin produces vitamin D3 and supplies 90 % of vitamin D. Vitamin D is paramount in calcium and bone metabolism, regulating parathyroid hormone functions. Preoperative and postoperative vitamin D deficiencies appear in 68

and 80 % of patients respectively as secondary hyperparathyroidism (PTH) is frequent after bariatric surgery. It is reported to be common in malabsorptive procedures, appearing in BPD-DS patients after 1 year, and to a lesser extent in SG and RYGB patients. Although SG is considered a pure restrictive procedure, vitamin D deficiency can still occur. Up to 53 % of patients are found to have increased serum PTH post-obesity surgery [25]. Despite receiving vitamin D supplementation after gastric bypass surgery, vitamin D deficiency with hyperparathyroidism is seen to continue to 50 % of patients [26]. The reasons remain unknown but may be attributed to various factors in dietary intake, season of the year, and socioeconomic status. Deficiency of vitamin D will in turn lead to decreased calcium absorption. This may result in low calcitriol, which favors fat accumulation.

Signs and symptoms of vitamin D deficiency include osteomalacia, osteoporosis, arthralgia, myalgia, fasciculation, and depression. Considering the high prevalence of preoperative deficiency, all candidates for obesity surgery should undergo 25-hydroxy vitamin D screening. Awareness should be noted that serum calcium may be low or normal with a decrease in serum phosphorus and increase in serum alkaline phosphatase. There is no agreement on recommendations for vitamin D therapy after bariatric surgery as few evidence-based regimens exist.

With severe deficiency, 50,000–150,000 IU of vitamin D3 per day can be sufficient with oral calcitriol if necessary. Some studies have recommended 5000 IU per day and 50,000 IU 2 times per day for prophylaxis and maintenance in RYGB and BPD patients correspondingly [27, 28]. Proper dosage may widely vary in each individual patient, and 25-hydroxy vitamin D levels should be monitored 2 weeks after initiation. These levels should be repeated up to every 3 months in the first year after surgery. Suggested supplementation for prevention after surgery is generally 400–800 U per day of oral vitamin D2 or D3. For patients who underwent malabsorptive procedures, vitamin D levels of $\geq$100 nmol/L have been demonstrated to be effective in prevention of secondary hyperparathyroidism [29]. Rigorous vitamin D supplementation is an important concern preceding and following bariatric surgery as incidences remain high.

22.8 Calcium

The human body has more calcium than any other mineral as 99 % of reserves are stored in bones. Dietary sources include dairy products, leafy vegetables, and fish with edible soft bones. Out of 1000 mg of intake, only 400 mg is absorbed passively in the ileum and jejunum, and actively by 1,25 OH vitamin D in the duodenum in the presence of acid. The incidence of both preoperative and postoperative calcium deficiency is 10 % [30]. Postoperative anatomical changes, i.e., the bypass of the duodenum, the relatively short common channel in distal RYGB or BPD, and including decreased mixing of bile salts, can lead to malabsorption of calcium and vitamin D.

Acute hypocalcemia can manifests as paresthesia of the limbs and oral cavity, and progress to tetany. Cardiac arrhythmia is a serious complication and must be acutely recognized. Long-term deficiency presents with increased risk for bones fractures and osteoporosis from low bone density. Thus, clinical signs and symptoms may suddenly become apparent when skeletal calcium stores become depleted.

In determining calcium status, serum calcium levels have limited value due to serum calcium being regulated by PTH-vitamin D. Hypoalbuminemia, common after bariatric surgery, may simulate hypocalcemia because of the high affinity between albumin and calcium. Calcium excretion in 24-h urine along with alkaline phosphatase may be performed at 6 month intervals to assess calcium levels. Bone turnover and mineral density can be also measured by serum PTH and the DEXA test. Some authors recommend spine and hip DEXA scans for osteoporosis monitoring in post-RYGB and BPD patients at baseline then after 2 years [31]. In terms of prevention and treatment following obesity surgery, calcium supplementations (calcium citrate) can be up to 2000 mg daily,

especially when serum PTH is increased [32]. Calcium carbonate has shown to absorb poorly in low acid environments (e.g., gastric bypass). An important consideration: oral calcium is known to hinder intestinal absorption of copper, iron, and zinc. A DEXA T-score of less than 2.5 may warrant intravenous bisphosphonates when calcium deficiency continues to persist after exhausting oral supplementations.

22.9 Vitamin A

Vitamin A is a fat-soluble vitamin existing in numerous forms. The two different types of vitamin A are preformed vitamin A (retinols) in animal products and pro-vitamin A in plant-based products with beta-carotene being the most common. The difference is the high saturated fat and cholesterol content in animal preformed vitamin A. The active form for vitamin A is retinol. In dietary supplements, retinyl acetate or palmitate (preformed vitamin A) and beta-carotene (pro-vitamin A) are generally the usual forms. It plays an intricate role in healthy skin, bones, mucus membranes, the retina, and is important in the prevention of low-density lipoprotein oxidation (atherosclerosis prevention). Obese individuals are shown to have lower serum concentrations of vitamin A compared to the normal weight population [33]. This has been attributed to inadequate dietary intake along with tobacco smoking. Low carotenoid and alpha-tocopherol serum levels have been demonstrated to be associated with impaired glucose metabolism and insulin resistance.

Vitamin A is absorbed in the proximal jejunum and requires micelle formation with conjugated bile acids. In BPD-DS, the prevalence has been reported to be as high as 50 % after 1 year and 70 % after 4 years [34, 35]. The BPD-DS and RYGB (up to 11 % prevalence) may result in bile acid deficiency due to a short common channel. A rare occurrence, bile acid deconjugation may occur from bacterial overgrowth in the jejunum. Night blindness (nyctalopia) is typically the first clinical sign of deficiency. Other manifestations include xerophthalmia, pruritis, decreased immunity, and dry hair. Severe progression can lead to complete blindness. Plasma retinol is the most common biomarker for vitamin A deficiency. However, plasma retinol levels do not decline until liver stores of vitamin A are exceedingly low, and serum retinol is negatively affected by iron deficiency, reducing mobilization of vitamin A from the liver. Retinol levels may decrease in the presence of inflammation as retinol binding protein (RBP) is a negative acute-phase reactant. A history of night blindness has proven effective in detection preoperatively and postoperatively as ocular findings are suggestive of the diagnosis [36]. Vitamin A deficiency should be suspected in any patient with a history of intestinal surgery and unexplained vision problems.

In severe deficiency without corneal changes, up to 25,000 IU per day orally for 2 weeks is recommended. In the presence of corneal lesions, 50,000–100,000 IU intramuscular followed by 50,000 IU per day intramuscularly for 2 weeks is recommended by the ASMBS. There is no preventive recommendation for this fat-soluble vitamin as toxicity may lead to liver damage, diplopia, headache, and vomiting when intake exceeds 100,000 IU in 6 months. Iron and copper deficiency are known to impair resolution of vitamin A deficiency.

22.10 Vitamin E

Similar to other fat-soluble vitamins in passive small intestine absorption, vitamin E is commonly found in vegetable oils, margarine, nuts, and leafy greens. Deficiency is considered rare and uncommon after bariatric surgery with an unknown prevalence. Some data have suggested that deficiency occurs more commonly after RYGB compared to BPD-DS or AGB. Reduced plasma alpha-tocopherol has been detected in up to 22 % of RYGB patients [37]. The screening test for vitamin E is plasma alpha-tocopherol. There is a positive correlation of serum alpha-tocopherol with serum cholesterol and obesity. Neurological problems of ataxia, dysarthria, lower limb areflexia, and peripheral neuropathy can arise from vitamin E deficiency. Evidence-based data

on prevention and treatment of vitamin E deficiency following bariatric surgery is scarce or unknown, however, recommendations range from 800 to 1200 IU orally per day [38].

22.11 Vitamin K

Vitamin K, a fat-soluble nutrient, is a key component in protein production for clotting factors and osteocalcin homeostasis in bones. The most common dietary sources are green vegetables and dark berries. Intestinal bacteria in the colon (e.g., *Escherichia coli*) can synthesize vitamin K_2, a subtype of vitamin K. The distal jejunum and ileum are the main sites of absorption for dietary vitamin K. Similar to vitamin B_1, vitamin K has a short half-life and low body stores.

Manifestations of clinical deficiency include bleeding and osteoporosis. While rates of preoperative deficiency remain unknown, initial data on postoperative prevalence shows deficiency is common in BPD-DS patients after 1 year and RYGB patients to a lesser extent. A case report has been published demonstrating the potential severity of vitamin K deficiency: purpura fulminans leading to disseminated intravascular coagulation which was postulated to result from decreased absorption of vitamin K-dependent proteins C, S, and antithrombin in a gastric bypass patient [39].

Prothrombin time (PT) is a common screening tool for vitamin K deficiency. However, PT has been shown to underdetect deficiency due to its low sensitivity. Additional laboratory indexes include increased des-gamma-carboxy prothrombin (DCP) and decreased plasma phylloquinone. Enzyme-linked immunosorbent assays (ELISAs) and high-performance liquid chromatography (HPLC) can be utilized for their high sensitivities, however, these tests are expensive and may not be readily available. As there is no general recommendation for maintenance, deficiency should be treated with an initial 10 mg intramuscular injection followed by 2 mg orally per week in BPD and RYBG patients [40]. In cases where prothrombin time does not improve, vitamin K deficiency can be ruled out.

22.12 Zinc

Zinc is an essential mineral and important for gene expression and enzymatic reactions. The synthesis and functions of insulin by tyrosine kinase activity are dependent on zinc. Zinc can also be applied to the skin to improve wound healing. In natural food sources, oysters and red meat are found to have the highest zinc per serving. Prolonged total parenteral nutrition has often led to zinc deficiency. Early signs and symptoms of zinc deficiency are hair loss, taste abnormalities, glossitis, fatigue, and eye and skin lesions. The association of zinc and obesity is shown with low zinc plasma level and high urinary zinc excretion and serum insulin. In obese candidates for bariatric surgery, prevalence is 28 %. As zinc is mainly absorbed in the duodenum with minimal body stores, prevalence after BPD-DS is found to be up to 91 %, and up to 33 % and 12 % after RYBG and LSG respectively [41, 42]. Increased urinary zinc excretion is reported after surgery; however, the mechanism remains unknown.

Although no reliable method exists for determining zinc status, plasma zinc is commonly used for screening in deficiency. This is due to albumin being the primary binding protein for zinc. Thus, albumin levels are necessary for accurate interpretation. There are no generally accepted recommendations for the prevention of zinc deficiency. However, obese patients who had bariatric surgery should be prescribed lifelong multivitamin supplementation for prevention. For treatment of deficiency, 60 mg elemental zinc (zinc sulfate) twice a day with 1 mg copper given for each 8–15 mg zinc received is recommended by the ASMBS. It is of importance for oral zinc to be taken 1 h before breakfast as zinc interferes with iron and copper absorption [43]. Large doses of zinc may result in hypochromic anemia from copper deficiency.

22.13 Copper

Copper is found abundantly in shellfish and organ meats (liver, kidneys). Absorbed by the stomach and proximal gut, it is involved in intestinal iron

absorption, red blood cell formation with iron, and the synthesis of norepinephrine. Studies on hypocupremia following obesity surgery are increasingly recognized. A 5-year follow-up study demonstrated copper deficiency prevalence to be in 23.6 % after BPD and 1.9 % after RYGB [44]. Preoperative prevalence is still unidentified. Other risk factors include the increased use of antacids and zinc supplementation. Zinc supplementation of greater than 50 mg is known to impede intestinal copper absorption as zinc overload can result in copper myelopathy.

Symptoms of hypocupremia can be neurological (myeloneuropathy, unsteady gait, paresthesia) and hematological (normochromic anemia). These symptoms may appear years (mean 11.4 years) following bariatric surgery [45]. Commonly misinterpreted and similar to vitamin B_{12} and iron deficiency, copper deficiency should be considered when there is unexplained anemia, neurologic deficits or poor wound healing. Currently, routine copper screening following surgery is not common as there are no general guidelines. Laboratory tests for copper deficiency are blood copper and ceruloplasmin levels. However, these levels can be falsely elevated in the presence of inflammation. Recent studies have indicated that copper/zinc superoxide dismutase to be a superior biomarker in terms of sensitivity, but reference values are not currently established.

Long-term zinc and copper supplementation (2 mg oral copper gluconate daily) for prevention or maintenance after obesity surgery should be considered with 1 mg copper for each 8–15 mg zinc dose. Treatment for severe copper deficiency is suggested with 2.4 mg copper sulfate mixed in 100 mL of normal saline, intravenously infusing over 4 h daily for 5 days [31].

22.14 Selenium

Seafoods and organ meats are dietary trace sources rich in selenium. Selenium is involved in thyroid hormone metabolism and protects cells from oxidative damage. Published literature for this overlooked mineral is rare, but recent data shows 58 % of morbidly obese preoperative bariatric patients to be selenium deficient [46]. Postoperative deficiency is reported in up to 20 % of bariatric patients, especially after malabsorptive procedures such as BPD-DS [47].

Cardiac myositis may be seen in selenium deficiency. In severe cases, cardiomyopathy (in combination with Coxsackie virus infection—Keshan disease) may result in heart failure. Only one published case report describes cardiomyopathy due to selenium deficiency 9 months following BPD [48]. Conversely, high selenium levels (>100 μg/dl) are associated with neurological symptoms, hair loss, and gastrointestinal symptoms.

No recommendations exist for screening after obesity surgery. However, it is imperative to obtain selenium levels as part of a panel for postoperative patients with anemia or signs of cardiomyopathy. While up to 70 mcg of selenium may be used for prevention, oral selenium up to 200 mcg daily for 6 months is recommended for deficiency treatment.

22.15 Magnesium

Magnesium is amply found in leafy green vegetables. It is involved in muscle contraction and cardiac excitability. Along with calcium, extracellular magnesium is essential for neuromuscular activity. 1,25-dihydroxyvitamin D stimulates the absorption of magnesium in the jejunum and ileum. As magnesium is excreted in urine, the kidneys are mainly responsible for serum magnesium regulation. PTH increases reabsorption while hypercalcemia and hypermagnesemia inhibit reabsorption. Hypomagnesemia can result from vitamin D deficiency. Diseases commonly associated with obesity—hyperlipidemia, diabetes mellitus, and hypertension—have been correlated with hypomagnesemia. Studies have suggested that proton pump inhibitors may lead to hypomagnesemia after bariatric surgery [49].

Signs of deficiency are vomiting, irritability, and weakness which may progress to muscle contractions, osteoporosis, tetany seizures, coronary spasms, and cardiac arrhythmia. Since only 1 % of magnesium is found in extracellular fluid,

serum magnesium may not accurately represent total body stores, underdetecting magnesium deficits. Preoperative and postoperative prevalence have been documented to be 35 and 32 % respectively [50] but data are conflicting. Some reports have identified significant hypokalemia following gastric bypass (up to 2.4 %) [51]. However, serum magnesium is still is useful biomarker in severe deficiency. Urinary magnesium monitoring can be utilized in cases of high suspicion. For supplementation, magnesium citrate 300 mg daily is recommended and has the highest bioavailability. Severe deficiency is treated by 4 g in 250 mL dextrose 5 % water intravenous infusion daily.

22.16 Protein

With pepsinogen secreted in the stomach, cholecystokinin and enterokinase are activated as chyme enters the intestines. This allows further activation of pancreatic trypsin and carboxypolypeptidase. Small intestinal peptidases allow additional breakdown of protein where most of the digestion and absorption occur. The association between protein malnutrition (serum albumin level < 3.5 mg/dL) and malabsorptive procedures is well-known as lean tissue mass loss can be greater than body fat lost, especially within 6 months post-operation [28]. Secondary lactase deficiency and intolerance to protein rich food has shown to develop in 50 % of RYGB patients [52]. The prevalence of protein deficiency after 2 years from distal RYGB, proximal RYGB, and BPD is 13, 5, and up to 18 %, respectively [53, 54]. Protein malnutrition is a frequent cause of readmission in these patients. In particular, malnutrition is commonly demonstrated in distal RYBG due to the combination of the small gastric pouch and the long Roux limb [55].

Protein malnutrition is commonly associated with B_{12}, folate, iron, zinc, thiamine, B_6, and copper deficiency. Additionally, disturbances may be seen in sodium, potassium, magnesium, and phosphorus levels. Early symptoms of protein malnutrition include weakness, hair loss, and, in severe cases, generalized edema from increased carbohydrate intake resulting in hypoalbuminemia. Preoperative and postoperative screening and diagnostic tests of protein status consist of serum albumin, total protein, transferrin, and lymphocyte count. Increased awareness is required in interpreting serum albumin as it is a negative acute phase reactant in the presence of inflammation. Other options are dual X-ray absorptiometry (DEXA) and body impedance assessment (BIA) in measuring fat mass and body composition, but these tests tend to overestimate values in the preoperative and postoperative bariatric population [53, 56].

Consensus guidelines recommend daily protein intake of up to 120 g after RYBG, 90 g following BPD/DS, and 1.1 g/kg of ideal body weight after LSG with aerobic exercises to prevent loss of lean tissue mass [28, 57]. When vomiting or food intolerance is absent, a high protein liquid diet and eventual progression to a regular diet may be sufficient. In treating protein malnutrition, oral supplementation of up to 12 g of leucine daily has been demonstrated to stimulate protein synthesis [58]. Enteral feeding may be necessary if required. In severe cases, hospitalization and parenteral nutrition must be initiated with consideration of revisional surgery, namely elongating the common channel and adjusting the length of intestinal limbs to improve protein digestion and absorption. All underlying behavioral and mechanical causes must be ruled out as well.

22.17 Pregnancy

Pregnancy in the postoperative bariatric patient presents issues. The benefits of bariatric surgery prior to conception include the decreased risk of hypertensive disorders and gestational diabetes mellitus, and improved fertility. The American College of Obstetricians and Gynecologists recommends a minimum of 12 months after obesity surgery before conception to avoid the rapid weight loss. Studies have indicated no differences in preterm deliveries, complications, and cesarean delivery for patients conceiving before or after the 1 year mark after RYBG or

LAGB, or after the 1 year mark compared to non-obese controls [59, 60]. Other studies have reported spontaneous abortion and premature birth rates of 29 % after LAGB and 18 % after LRYGB within 2 years post-operation [61, 62]. Presently, data are conflicting when assessing the risks affecting pregnancy in post-bariatric patients. In any case, conception before 12 months requires close monitoring of the patient and fetus.

In particular, vitamin K deficiency represents a major concern for pregnant postoperative patients and their newborns. Reported cases of neonatal intracranial bleeding in 5 infants have been described in women who underwent bariatric surgery before conception. These cases were attributed to vitamin K deficiency following bariatric surgery [63]. Additionally, the protein status must be monitored in pregnant patients, especially in the period of rapid weight loss. Protein supplementation is recommended in expecting individuals who are not gaining weight with fetal growth below the 50th percentile. Irrespective of the procedural type, broad evaluation with high suspicion is necessary in detection of macronutritional and micronutritional deficiency. This should be followed by calcium, vitamin D, ferritin, and iron monitoring every trimester. Standard supplementation for pregnant individuals includes multivitamin (i.e., folic acid) with vitamin D, calcium citrate, iron, and vitamin B_{12}. Parenteral nutrition is considered when nutritional status does not improve by the oral route. Some groups recommend only initiating nutritional assessments of pregnant post-bariatric patients based on clinical grounds.

22.18 Other Considerations

In combination with nutritional supplementation, pharmacological therapy represents a major aspect of postoperative care. While antidiabetic medication usage decreases following obesity surgery, pain medications along with proton pimp inhibitors, H2 receptor antagonists, and antibiotics are still significantly prescribed. Thus far, few data have been published regarding measured absorption and bioavailability of drugs and nutrients following bariatric surgery in a controlled metabolic setting. Altered nutritional intake has been proposed to be chiefly responsible for nutritional status alterations as opposed to malabsorption. Studies would ideally need to include radioisotope labeling in measuring drug and nutrient intake. Gastric and intestinal adaptation (compensatory response) after bariatric surgery is still unclear when considering the long-term effects of surgery. Consideration of trace mineral deficiency must also be applied to presumably restrictive-only procedures such as LAGB and LSG.

References

1. Brolin RE, Gorman RC, Milgrim LM, Kenler HA. Multivitamin prophylaxis in prevention of post-gastric bypass vitamin and mineral deficiencies. Int J Obes. 1991;15(10):661–7.
2. Stefater MA, Wilson-Pérez HE, Chambers AP, Sandoval DA, Seeley RJ. All bariatric surgeries are not created equal: insights from mechanistic comparisons. Endocr Rev. 2012;33(4):595–622.
3. Gasteyger C, Suter M, Calmes JM, Gaillard RC, Giusti V. Changes in body composition, metabolic profile and nutritional status 24 months after gastric banding. Obes Surg. 2006;16(3):243–50.
4. van Rutte PW, Aarts EO, Smulders JF, Nienhuijs SW. Nutrient deficiencies before and after sleeve gastrectomy. Obes Surg. 2014;24(10):1639–46.
5. Buchwald H, Avidor Y, Braunwald E, Jensen MD, Pories W, Fahrbach K, et al. Bariatric surgery: a systematic review and meta-analysis. JAMA. 2004; 292(14):1724–37.
6. Carrodeguas L, Kaidar-Person O, Szomstein S, Antozzi P, Rosenthal R. Preoperative thiamine deficiency in obese population undergoing laparoscopic bariatric surgery. Surg Obes Relat Dis. 2005;1(6):517–22.
7. Lakhani SV, Shah HN, Alexander K, Finelli FC, Kirkpatrick JR, Koch TR. Small intestinal bacterial overgrowth and thiamine deficiency after Roux-en-Y gastric bypass surgery in obese patients. Nutr Res. 2008;28(5):293–8.
8. Rudnicki SA. Prevention and treatment of peripheral neuropathy after bariatric surgery. Curr Treat Options Neurol. 2010;12(1):29–36.
9. Sechi G, Serra A. Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. Lancet Neurol. 2007;6(5):442–55.
10. Flancbaum L, Belsley S, Drake V, Colarusso T, Tayler E. Preoperative nutritional status of patients undergoing Roux-en-Y gastric bypass for morbid obesity. J Gastrointest Surg. 2006;10(7):1033–7.

11. Turkki PR, Ingerman L, Schroeder LA, Chung RS, Chen M, Russo-Mcgraw MA, et al. Thiamin and vitamin B6 intakes and erythrocyte transketolase and aminotransferase activities in morbidly obese females before and after gastroplasty. J Am Coll Nutr. 1992;11(3):272–82.
12. Mallory GN, Macgregor AM. Folate status following gastric bypass surgery (the great folate mystery). Obes Surg. 1991;1(1):69–72.
13. Madan AK, Orth WS, Tichansky DS, Ternovits CA. Vitamin and trace mineral levels after laparoscopic gastric bypass. Obes Surg. 2006;16(5):603–6.
14. Carmel R, Green R, Rosenblatt DS, Watkins D. Update on cobalamin, folate, and homocysteine. Hematol Am Soc Hematol Educ Program. 2003;62–81.
15. Shane B, Stokstad EL. Vitamin B_{12}-folate interrelationships. Annu Rev Nutr. 1985;5:115–41.
16. Skroubis G, Sakellaropoulos G, Pouggouras K, Mead N, Nikiforidis G, Kalfarentzos F. Comparison of nutritional deficiencies after Roux-en-Y gastric bypass and after biliopancreatic diversion with Roux-en-Y gastric bypass. Obes Surg. 2002;12(4):551–8.
17. Aasheim ET, Johnson LK, Hofsø D, Bøhmer T, Hjelmesæth J. Vitamin status after gastric bypass and lifestyle intervention: a comparative prospective study. Surg Obes Relat Dis. 2012;8(2):169–75.
18. Pinhas-Hamiel O, Doron-Panush N, Reichman B, Nitzan-Kaluski D, Shalitin S, Geva-Lerner L. Obese children and adolescents: a risk group for low vitamin B_{12} concentration. Arch Pediatr Adolesc Med. 2006;160(9):933–6.
19. Sumner AE, Chin MM, Abrahm JL, Berry GT, Gracely EJ, Allen RH, et al. Elevated methylmalonic acid and total homocysteine levels show high prevalence of vitamin B_{12} deficiency after gastric surgery. Ann Intern Med. 1996;124(5):469–76.
20. Herrmann W, Obeid R. Causes and early diagnosis of vitamin B_{12} deficiency. Dtsch Arztebl Int. 2008;105(40):680–5.
21. Obinwanne KM, Fredrickson KA, Mathiason MA, Kallies KJ, Farnen JP, Kothari SN. Incidence, treatment, and outcomes of iron deficiency after laparoscopic Roux-en-Y gastric bypass: a 10-year analysis. J Am Coll Surg. 2014;218(2):246–52.
22. Brolin RE, Gorman JH, Gorman RC, Petschenik AJ, Bradley LJ, Kenler HA, et al. Are vitamin B_{12} and folate deficiency clinically important after roux-en-Y gastric bypass? J Gastrointest Surg. 1998;2(5):436–42.
23. Evstatiev R, Marteau P, Iqbal T, Khalif IL, Stein J, Bokemeyer B, et al. FERGIcor, a randomized controlled trial on ferric carboxymaltose for iron deficiency anemia in inflammatory bowel disease. Gastroenterology. 2011;141(3):846–53.
24. Brolin RE, Gorman JH, Gorman RC, Petschenik AJ, Bradley LB, Kenler HA, et al. Prophylactic iron supplementation after Roux-en-Y gastric bypass: a prospective, double-blind, randomized study. Arch Surg. 1998;133(7):740–4.
25. Diniz Mde F, Diniz MT, Sanches SR, Salgado PP, Valadão MM, Araújo FC, et al. Elevated serum parathormone after Roux-en-Y gastric bypass. Obes Surg. 2004;14(9):1222–6.
26. Carlin AM, Rao DS, Yager KM, Genaw JA, Parikh NJ, Szymanski W. Effect of gastric bypass surgery on vitamin D nutritional status. Surg Obes Relat Dis. 2006;2(6):638–42.
27. Goldner WS, Stoner JA, Lyden E, Thompson J, Taylor K, Larson L, et al. Finding the optimal dose of vitamin D following Roux-en-Y gastric bypass: a prospective, randomized pilot clinical trial. Obes Surg. 2009;19(2):173–9.
28. Heber D, Greenway FL, Kaplan LM, Livingston E, Salvador J, Still C, et al. Endocrine and nutritional management of the post-bariatric surgery patient: an Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2010;95(11):4823–43.
29. Hewitt S, Søvik TT, Aasheim ET, Kristinsson J, Jahnsen J, Birketvedt GS, et al. Secondary hyperparathyroidism, vitamin D sufficiency, and serum calcium 5 years after gastric bypass and duodenal switch. Obes Surg. 2013;23(3):384–90.
30. Brethauer SA, Chand B, Schauer PR. Risks and benefits of bariatric surgery: current evidence. Cleve Clin J Med. 2006;73(11):993–1007.
31. Mechanick JI, Youdim A, Jones DB, Garvey WT, Hurley DL, McMahon MM, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient—2013 update: cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. Obesity (Silver Spring). 2013;21 Suppl 1:S1–27.
32. Mechanick JI, Kushner RF, Sugerman HJ, Gonzalez-Campoy JM, Collazo-Clavell ML, Spitz AF, et al. American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery medical guidelines for clinical practice for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient. Obesity (Silver Spring). 2009;17 Suppl 1:S1–70.
33. Neuhouser ML, Rock CL, Eldridge AL, Kristal AR, Patterson RE, Cooper DA, et al. Serum concentrations of retinol, alpha-tocopherol and the carotenoids are influenced by diet, race and obesity in a sample of healthy adolescents. J Nutr. 2001;131(8):2184–91.
34. Clements RH, Katasani VG, Palepu R, Leeth RR, Leath TD, Roy BP, et al. Incidence of vitamin deficiency after laparoscopic Roux-en-Y gastric bypass in a university hospital setting. Am Surg. 2006;72(12):1196–202. discussion 1203–4.
35. Zalesin KC, Miller WM, Franklin B, Mudugal D, Rao Buragadda A, Boura J, et al. Vitamin a deficiency after gastric bypass surgery: an underreported postoperative complication. J Obes. 2011;2011. pii:760695.

36. Pereira S, Saboya C, Ramalho A. Impact of different protocols of nutritional supplements on the status of vitamin A in class III obese patients after Roux-en-Y gastric bypass. Obes Surg. 2013;23(8):1244–51.
37. Ueda N, Suzuki Y, Rino Y, Takahashi T, Imada T, Takanashi Y, et al. Correlation between neurological dysfunction with vitamin E deficiency and gastrectomy. J Neurol Sci. 2009;287(1–2):216–20.
38. Koch TR, Finelli FC. Postoperative metabolic and nutritional complications of bariatric surgery. Gastroenterol Clin North Am. 2010;39(1):109–24.
39. Bersani I, De Carolis MP, Salvi S, Zecca E, Romagnoli C, De Carolis S. Maternal-neonatal vitamin K deficiency secondary to maternal biliopancreatic diversion. Blood Coagul Fibrinolysis. 2011;22(4):334–6.
40. Allied Health Sciences Section Ad Hoc Nutrition Committee, Aills L, Blankenship J, Buffington C, Furtado M, Parrott J. ASMBS allied health nutritional guidelines for the surgical weight loss patient. Surg Obes Relat Dis. 2008;4(5 Suppl):S73–108.
41. Gehrer S, Kern B, Peters T, Christoffel-Courtin C, Peterli R. Fewer nutrient deficiencies after laparoscopic sleeve gastrectomy (LSG) than after laparoscopic Roux-Y-gastric bypass (LRYGB)–a prospective study. Obes Surg. 2010;20(4):447–53.
42. Ernst B, Thurnheer M, Schmid SM, Schultes B. Evidence for the necessity to systematically assess micronutrient status prior to bariatric surgery. Obes Surg. 2009;19(1):66–73.
43. Olivares M, Pizarro F, Ruz M, de Romaña DL. Acute inhibition of iron bioavailability by zinc: studies in humans. Biometals. 2012;25(4):657–64.
44. Balsa JA, Botella-Carretero JI, Gómez-Martín JM, Peromingo R, Arrieta F, Santiuste C, et al. Copper and zinc serum levels after derivative bariatric surgery: differences between Roux-en-Y gastric bypass and biliopancreatic diversion. Obes Surg. 2011;21(6):744–50.
45. Lazarchick J. Update on anemia and neutropenia in copper deficiency. Curr Opin Hematol. 2012;19(1):58–60.
46. Kimmons JE, Blanck HM, Tohill BC, Zhang J, Khan LK. Associations between body mass index and the prevalence of low micronutrient levels among US adults. MedGenMed. 2006;8(4):59.
47. Shankar P, Boylan M, Sriram K. Micronutrient deficiencies after bariatric surgery. Nutrition. 2010; 26(11–12):1031–7.
48. Boldery R, Fielding G, Rafter T, Pascoe AL, Scalia GM. Nutritional deficiency of selenium secondary to weight loss (bariatric) surgery associated with life-threatening cardiomyopathy. Heart Lung Circ. 2007; 16(2):123–6.
49. Luk CP, Parsons R, Lee YP, Hughes JD. Proton pump inhibitor-associated hypomagnesemia: what do FDA data tell us? Ann Pharmacother. 2013;47(6):773–80.
50. Dalcanale L, Oliveira CP, Faintuch J, Nogueira MA, Rondó P, Lima VM, et al. Long-term nutritional outcome after gastric bypass. Obes Surg. 2010;20(2):181–7.
51. Crowley LV, Seay J, Mullin G. Late effects of gastric bypass for obesity. Am J Gastroenterol. 1984;79(11): 850–60.
52. Song A, Fernstrom MH. Nutritional and psychological considerations after bariatric surgery. Aesthet Surg J. 2008;28(2):195–9.
53. Chaston TB, Dixon JB, O'Brien PE. Changes in fat-free mass during significant weight loss: a systematic review. Int J Obes (Lond). 2007;31(5):743–50.
54. Moizé V, Andreu A, Rodríguez L, Flores L, Ibarzabal A, Lacy A, et al. Protein intake and lean tissue mass retention following bariatric surgery. Clin Nutr. 2013;32(4):550–5.
55. Levinson R, Silverman JB, Catella JG, Rybak I, Jolin H, Isom K. Pharmacotherapy prevention and management of nutritional deficiencies post Roux-en-Y gastric bypass. Obes Surg. 2013;23(7):992–1000.
56. Andreu A, Moizé V, Rodríguez L, Flores L, Vidal J. Protein intake, body composition, and protein status following bariatric surgery. Obes Surg. 2010;20(11): 1509–15.
57. Snyder-Marlow G, Taylor D, Lenhard MJ. Nutrition care for patients undergoing laparoscopic sleeve gastrectomy for weight loss. J Am Diet Assoc. 2010;110(4):600–7.
58. Layman DK. Protein quantity and quality at levels above the RDA improves adult weight loss. J Am Coll Nutr. 2004;23(6 Suppl):631S–6.
59. Dao T, Kuhn J, Ehmer D, Fisher T, McCarty T. Pregnancy outcomes after gastric-bypass surgery. Am J Surg. 2006;192(6):762–6.
60. Dixon JB, Dixon ME, O'Brien PE. Birth outcomes in obese women after laparoscopic adjustable gastric banding. Obstet Gynecol. 2005;106(5 Pt 1):965–72.
61. Weiss HG, Nehoda H, Labeck B, Hourmont K, Marth C, Aigner F. Pregnancies after adjustable gastric banding. Obes Surg. 2001;11(3):303–6.
62. Patel JA, Patel NA, Thomas RL, Nelms JK, Colella JJ. Pregnancy outcomes after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2008;4(1): 39–45.
63. Eerdekens A, Debeer A, Van Hoey G, De Borger C, Sachar V, Guelinckx I, et al. Maternal bariatric surgery: adverse outcomes in neonates. Eur J Pediatr. 2010;169(2):191–6.

Excessive Skin after Massive Weight Loss: Body Contouring and Bariatric Surgery

23

Nikki Burish and Peter J. Taub

Key Points

- The field of bariatric surgery has grown appreciably over the past decade, with procedures designed and developed to induce weight loss and improve or eliminate obesity related comorbidities. As such, a new patient population has emerged: the massive weight loss patient.
- Despite the positive aesthetic and functional benefits of body contouring procedures, massive weight loss patients are at an increased risk for complications, highlighting the importance of developing standard operating room protocols with a focus on perioperative patient safety and utilization of a multidisciplinary team approach.
- Comprehensive informed consent is imperative to ensure patient understanding and realistic expectations. Many body contouring procedures may take months to recover from and years to produce final results.
- Body contouring after bariatric surgery is an exciting field that continues to grow and evolve, ultimately requiring a dedication to one's self and surgeon in order to optimize outcomes and minimize complications in this challenging patient population.

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23.1 Introduction

Obesity is a complex condition with significant physical, psychological, and social effects, which touch virtually all ages and socioeconomic groups. It is pervasive throughout the United States and other Westernized countries. Today, roughly two out of three (69 %) American adults are overweight or obese and one out of three (36 %) is obese [1]. Even more alarming is the prevalence of childhood obesity, with one out of three children and adolescents ages 2–19 overweight or obese, and one out of six being obese. Since early obesity increases the likelihood of adult obesity, these numbers will likely continue to rise.

Obesity is defined as a body mass index (BMI) greater than 30 kg/m^2, with morbid obesity defined as a BMI > 40 kg/m^2 (Table 23.1). Mortality rates of such individuals are double that of a normal weight individual [2]. Although obesity should be considered an avoidable chronic disease in its own right, it is a significant risk factor for other chronic diseases including coronary artery disease, arthritis, high blood pressure, high cholesterol, diabetes, obstructive sleep apnea, and asthma, amongst various others, making it a complex health issue to address [3].

D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_23

Table 23.1 Classification of obesity

Body mass index (BMI) kg/m^2	Obesity classification obesity class
<18.5	Underweight
18.5–24.9	Normal weight
25.0–29.9	Overweight
30.0–34.9	Obesity Class I
35.0–39.9	Obesity Class II
≥40	Morbid/Extreme Obesity Class III

As obesity has become an epidemic leading to a decreased quality of life, the number of patients electing to undergo bariatric surgery has increased substantially. As a result, a new patient population has emerged: the massive weight loss patient. Massive weight loss (MWL) results in loss of soft-tissue volume with increased laxity, ptosis, and redundant skin. This is a population of patients presenting with diverse and severe contour deformities—different from those ever encountered previously by the plastic surgeon. As such, the demand for body contouring due to excessive skin after massive weight loss has dramatically increased, and will likely increase further.

The field of bariatric surgery has grown appreciably over the past decade, with multiple different procedures developed to induce weight loss and improve or eliminate obesity-related comorbidities. The National Institutes of Health states that surgery is the most effective therapy for long-term significant weight loss in morbidly obese patients [4]. Current guidelines recommend bariatric surgery in those with a body mass index of 40 kg/m^2 or higher and for people with a BMI of higher than 35 kg/m^2 who have significant comorbidities [5].

Several options for bariatric surgery exist including Roux-en-Y gastric bypass, sleeve gastrectomy, biliopancreatic diversion with or without duodenal switch and increasingly less common adjustable gastric band (Fig. 23.1). These procedures work through a combination of restriction, malabsorption, and hormonal changes to achieve weight loss. While most bariatric patients achieve good-to-excellent weight loss, one-third will achieve massive weight loss; each such patient will have unique combinations of nutritional and aesthetic implications with regard to reconstructive body contouring surgery.

23.2 Impact of Bariatric Procedures

It is important to remember that nutritional deficiencies are common in the MWL patient. Bariatric procedures utilizing any degree of malabsorption can impair the body's stores of critical vitamins and minerals that are vital for wound healing [6]. These include vitamins such as B_{12}, iron, calcium, vitamins A and D, zinc, protein, and thiamine. Therefore, nutritional issues should be addressed with each patient preoperatively in order to optimize them for their body contouring procedures and recovery thereafter. Such body contouring procedures should only be considered at a time remote from the index procedure to allow the patient to regain a stable level of nutritional resources. Usually, this is a year or more following surgery.

Significant weight loss may have a large impact on skin's elasticity. In obese patients, skin is under prolonged tension, with the resultant breakdown of many of the elastin fibers. When weight is lost, the excess skin remains, as the skin's ability to retract back into place has been lost. MWL after bariatric surgery frequently results in redundant, amorphous and excess skin in most areas of the body, including the face, breasts, abdomen, arms, thighs, and buttocks. This can lead to rashes, infections, and musculoskeletal pain. Performing daily activities may become difficult and clothes may not fit properly, ultimately leading to embarrassment and further psychological issues.

Factors affecting skin elasticity can be broken down into two categories: those within the patient's immediate control and those that are not. Aging and genetics, neither of which is controllable, play a large role in the skin's cosmesis and appearance. Smoking, on the other hand, is a modifiable risk factor, which decreases arterial and capillary blood flow. Vessel constriction can lead to damage of connective tissues causing collagen breakdown at an accelerated rate as well

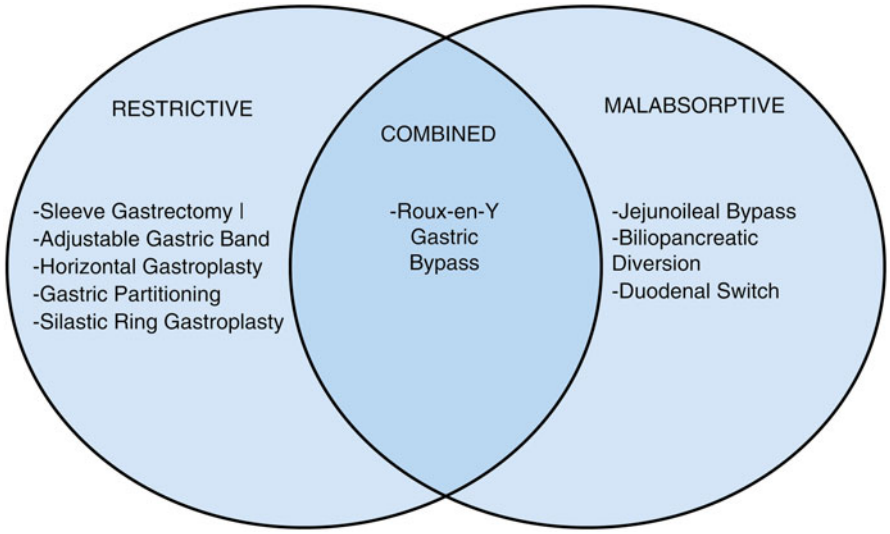

Fig. 23.1 Bariatric surgical options

as direct damage to skin fibroblasts (cells that form collagen and elastin). Diabetes mellitus can also contribute to loss of skin elasticity and tone due to dehydration and poor circulation as well as increased sensitivity to sun and UV light.

23.3 Preoperative Consultation

The preoperative evaluation is an important opportunity to address a variety of factors that are crucial in MWL patients. Body contouring surgery can produce results that improve both physical and psychological health, including greater exercise capabilities and physical tolerances which improve self-esteem and confidence. However, these operations are associated with high complication rates secondary to potential persistent obesity, nutritional deficiencies, weight-related comorbidities, and inelastic and poor quality tissue. Therefore, patient selection and practical considerations must not be overlooked.

When selecting patients to undergo postbariatric body contouring procedures, it is imperative to select individuals committed to leading a healthy lifestyle with dedication to proper nutrition and physical health. It is well known that the complications and risks in the postbariatric patients are indeed higher than that of the general population, making it critical that the surgeon spend adequate and ample time with the patients prior to their surgery in order to manage expectations.

For best results, the patient's BMI should be less than 35 kg/m^2, preferably less than 30 kg/m^2, with a stable weight. Many people believe that there is no true BMI cutoff for body contouring surgery after MWL but rather a continuum, where increasing BMI correlates with increasing complications and suboptimal aesthetic results [7]. Since the patient's height is a constant variable, the weight is the only manageable variable with regard to the BMI. Ideally, the patient should maintain a stable weight with fluctuations of no more than five pounds over a period of 3–6 months prior to elective procedures. Such a weight plateau is usually reached 12–18 months following bariatric surgery, depending on the type of bariatric surgery obtained. Patients who have undergone Roux-en-Y gastric bypass or sleeve gastrectomy tend to lose weight more quickly than those who have had an adjustable gastric band placed. Band patients may take up to several years to reach their lowest weight. Superior outcomes are achieved in those who are closer to their ideal body weight and are able to maintain a stable weight.

Each patient requires an individualized nutrition plan, tailored to the physiologic impact of their bariatric operation and their overall physical health. Since micronutrient and occasionally macronutrient deficiencies exist in the MWL patient population, a preoperative laboratory workup should be initiated several weeks to months prior to body contouring surgery, allowing ample time to correct any deficiencies that may be present (Table 23.2) [8]. Although patients may appear to be at a stable weight, this does not equate to a *healthy* weight. Vitamin and mineral deficiencies are often still present. Patients and surgeons must be made aware that overall nutritional optimization is essential for the best aesthetic outcome and uncomplicated recovery.

Managing comorbidities such as diabetes and cardiovascular disease is of paramount importance in this patient population. Additionally, smoking cessation is critical since smokers incur wound infection rates as high as 12 %, compared to 2 % in nonsmokers [9]. In diabetic patients, several factors can negatively influence wound healing including blood glucose levels, poor circulation, and neuropathy. The surgeon should work closely with the patient's primary care physician or endocrinologist as appropriate to achieve smoking cessation and optimize blood glucose control prior to any elective or major surgical procedure.

Table 23.2 Preoperative labs and studies for the massive weight loss patient

Preoperative labs[a]	Preoperative imaging/ studies
Complete blood count	Chest radiograph
Coagulation studies (pt/ INR/ptt)	EKG
Comprehensive metabolic panel	CT Abdomen[b]
Nutrition markers: Albumin Pre-albumin Micronutrients Iron Folate Vitamin B1 Vitamin B12 Calcium Vitamin D	

[a]Preoperative labs should be drawn at least 4 weeks prior to surgery to allow adequate time to address/correct any abnormalities

[b]CT Abdomen can be beneficial if the patient has a history or concern for hernias

Because previous abdominal operation suggests the possibility of occult abdominal wall hernias, special attention should be paid to surgical scars. Any abdominal incisions should be palpated both supine and sitting or standing to best evaluate the presence of a hernia. For an abdominoplasty, supraumbilical scars such as a Kocher incision can present problems with the blood supply of the superior flap, difficult dissection in the scar area, and/or patient dissatisfaction with the scar still visible postoperatively [10].

A history of venous thromboembolism including deep vein thrombosis or pulmonary embolism is the greatest single risk factor for future venous thromboembolism, the most potentially lethal morbidity after body contouring surgery [11]. Other risk factors include hormonal therapy/contraceptives, history of cancer or other hypercoaguable state, large bone fractures and immobility. High-risk patients should be properly assessed, treated and cleared by specialized physicians prior to elective body contouring procedures.

Patients should have a realistic expectation about outcomes after body contouring procedures for MWL. These are not procedures for treating obesity. They are designed to remove the excess skin and subcutaneous tissue and smooth the remaining skin. In essence, patients are trading excess tissue for long incisions that may or may not heal as perfectly as they like. Important concepts to emphasize to patients prior to surgery are the presence of scars, the lack of effect on regions outside those being treated, the potential for recurrence of skin laxity, the magnitude of recovery, and the risk of wound healing complications, which may require further operations. Postoperative management is equally important. Many of these procedures are major procedures that may take months to recover from and years to produce final results. As with most things in life, these results do not come without hard work and dedication to one's self and the surgical process.

Ultimately, the MWL patients are a unique group of individuals that present multiple challenges for medical and ancillary practitioners involved in their care. A multidisciplinary approach is essential in order to achieve the best and most sustainable outcomes. The composition of this team may vary, but often includes a psychiatrist, a dietician, an exercise physiologist, a bariatric surgeon, and a plastic surgeon. Lifelong follow-up and surveillance will be of utmost importance to ensure continued weight loss success and to achieve the best aesthetic outcomes with the highest degree of patient and surgeon satisfaction.

23.4 Preoperative Examination

The profile of a MWL patient is unique in that nearly every part of the body can be affected. The physical exam should follow a discussion of the issues regarding the medical and surgical history. Attention should be paid to previous surgeries and the resultant scars. The physician should focus on the skin to determine its overall tone, quality, laxity and redundancy. The location of adherent rolls/folds, lipodystrophy, and regional adiposity should also be evaluated. Some areas may be damp, erythematous, or ulcerated. This should be documented.

The most common area of concern is typically the abdomen due to its central location and affect on clothing. Features to consider include existing scars, striae, and hernias. Perhaps most important is the amount and direction of excess tissue. Nearly all patients have excess tissue in the vertical direction. This may be addressed with a traditional abdominoplasty. Patients with excess laxity in the horizontal direction, as well, may be better addressed with a fleur-de-lis abdominoplasty.

The extremities represent another area of concern. Excess tissue in the upper arm becomes a concern if the patient wears short sleeve shirts. Excess tissue over the medial thighs is more of a concern with ambulation. If lower extremity procedures are to be considered, a thorough vascular examination should be performed. Distal pulses, presence of varicosities, the quality of the lower extremity skin, and range of motion of the joints must all be examined. Other areas, such as the face, may have minimal functional impairments but significant psychological concerns.

The Pittsburgh Rating Scale relies on a point-based rating system for severity of deformities in the MWL patient by anatomic region [12] and correlates severity to the type of treatment necessary. An assessment of overall body composition with bio-impedance analysis may also be used. A thorough physical examination will better prepare the plastic surgeon to determine the optimal procedures appropriate for each patient.

23.5 Staging Procedures

As noted, MWL patients often present with multiple areas of concern. A complete series of body contouring procedures may require several operations and therefore several years to complete. In order to maximize patient satisfaction, it is essential to understand and determine the patient's priorities. If reasonable, these areas can be focused on first.

Patients with a higher starting BMI are more likely to require a staged approach than those with a lower BMI. Although aggregate minor complication rates are higher than in single procedure cases, there is no significant increase in complications on a per-procedure basis [13]. Proper staging is essential, as each procedure may have an impact on adjacent areas of the body. In most cases, a period of at least 3 months is necessary between stages. A clear advantage of the staged approach is that it provides an opportunity to revise recurrent skin laxity or defects after previous procedures.

In appropriately selected patients, if the surgery can be completed in a safe and timely fashion, multiple body contouring procedures may be combined into a single stage. When selecting procedures to combine, those that result in opposing vectors of tension should be avoided (Table 23.3). Length of operating time, surgeon fatigue, and financial burden on the patient must be taken into account when deciding the best plan for each patient.

23.6 Body Contouring Procedures

23.6.1 Abdominal Surgery

Abdominoplasty is one of the most commonly performed aesthetic surgical procedures. In 1899, Kelly was one of the first surgeons to attempt to correct excess abdominal skin and fat, describing an operation involving a mid-abdominal incision [14]. Over the past 100 years, abdominoplasty has evolved as new techniques and operations have been developed. With regard to excess skin of the abdomen, there are multiple approaches that can be taken. Factors to consider include zones of adherence (Fig. 23.2), prior scars, presence and size of pannus or mons pubis, laxity and flaccidity of skin above and below the umbilicus, flaring of ribs, diastasis of rectus muscles, and existing hernias. Large incisional, umbilical, or ventral hernias may require complex repair before performing an abdominoplasty for aesthetic improvement. After thorough examination, categorization of each patient according to his or her treatable soft tissue layers of skin, fat and muscle can be made and the proper abdominoplasty technique utilized.

Table 23.3 Combination body contouring procedures

Safe/Favorable combination of procedures	Unfavorable combinations that require caution
Abdominoplasty and liposuction	Lower body lift and upper body lift
Abdominoplasty and breast enhancement	Lower body lift and vertical thigh lift
Abdominoplasty and lower body lift	Upper body lift and brachioplasty
Abdominoplasty and brachioplasty	
Lower body lift and brachioplasty	
Lower body lift and breast enhancement	
Upper body lift and breast enhancement	
Brachioplasty and breast enhancement	
Brachioplasty and thigh lift	
Breast enhancement and thigh lift	

23.6.1.1 Traditional Abdominoplasty

Traditional abdominoplasty is appropriate for the patient with excess skin and fat above and below the umbilicus, periumbilical hooding or diastasis rectus. Abdominoplasty allows for full access to correct abdominal wall musculoaponeurotic laxity. When performing traditional abdominoplasty, vascular zones I to III must be taken into account (Fig. 23.3). This is especially important when upper abdominal scars are present. Zone 1 is almost always interrupted by flap elevation in abdominoplasty, leaving zone 3 as the only remaining blood supply. A traditional abdominoplasty typically includes a lower abdominal incision (usually spanning from one hip to the other, ~7 cm above vulvar commissure or penis), skin flap undermining from the symphysis pubis to the xiphoid process, midline musculoaponeurotic fascial plication, translocation of the umbilicus, and dermolipectomy of the lower half of the flap to remove excess skin and fat in a vertical vector [9]. In the traditional abdominoplasty, the superior resection margin is above the level of the umbilicus.

23.6.1.2 Limited Abdominoplasty/ Mini-abdominoplasty

This technique was first described by Wilkinson and Swartz in 1986 [15] and refined further in 1987 by Greminger [16]. Patients with limited skin and subcutaneous fat located predominantly below the umbilicus who do not have significant diastasis recti, are ideal candidates for a mini-abdominoplasty. This procedure is less invasive than its traditional counterpart, involving a smaller incision with a smaller area of abdominal wall exposed. Undermining and plication of the fascia is only done up to the level of the umbilicus. The surgeon may choose to "float the umbilicus" by releasing it at its base to allow for further undermining of the abdominoplasty flap. The umbilicus is subsequently pulled inferiorly to eliminate laxity in the upper abdomen. Disadvantages of the mini-abdominoplasty include a potentially low umbilical position and abnormal umbilical shape. Recovery is usually more rapid and compares to that of liposuction.

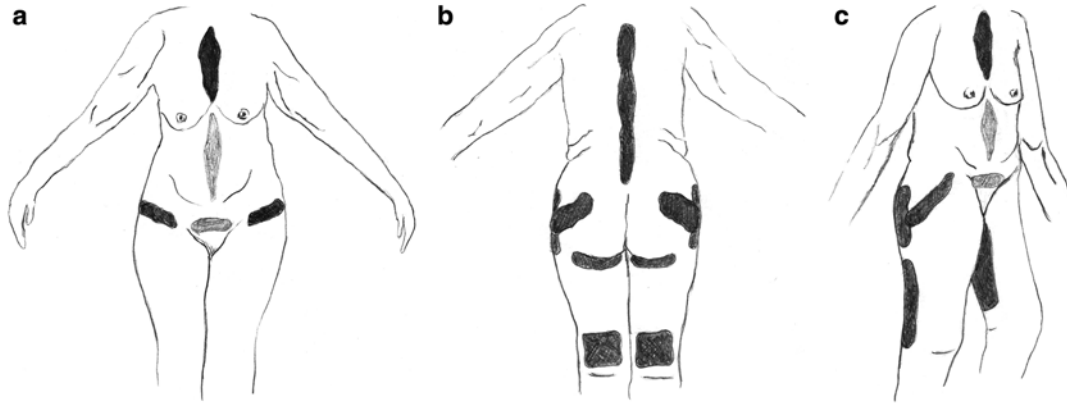

Fig. 23.2 (**a–c**) Fascial zones of adherence in the massive weight loss patient

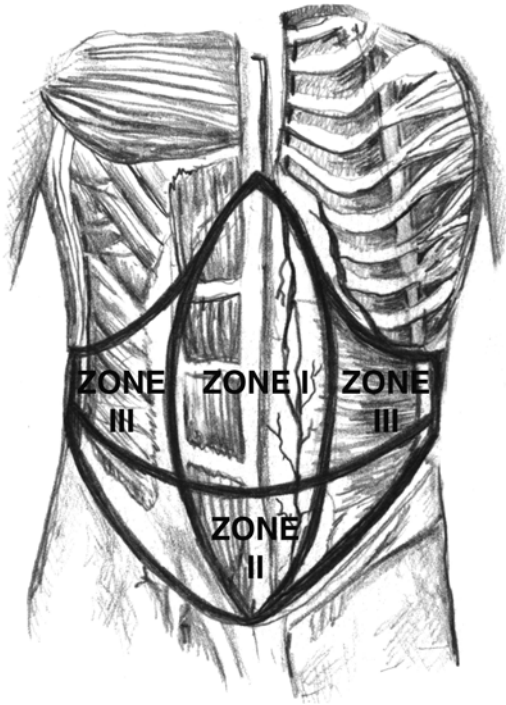

Fig. 23.3 Three Zones of blood supply to the abdomen. Zone I: deep superior and deep inferior epigastric arteries. Zone II: epigastric arcade, superficial external pudendal, superficial inferior epigastric and superficial circumflex iliac arteries. Zone III: lumbar, musculophrenic and lower intercostal arteries

23.6.1.3 Fleur-de-lis Abdominoplasty

In the MWL patient, traditional abdominoplasty techniques often fail to correct the complex contour deformities. There is often excess skin and soft tissue in the vertical direction as well as the horizontal direction. To address this concern, an additional vertical scar is needed [9]. The fleur-de-lis abdominoplasty (Fig. 23.4) was first popularized by Dellon in 1985 [17]. This technique utilizes an inverted "T" incision in order to facilitate the removal of circumferential lower truncal excess. The end result leaves the patient with a midline incision, similar to that of an exploratory laparotomy, in addition to a traditional abdominoplasty scar. The vertical limb must extend to the level of the xiphoid to minimize residual laxity and protrusion of upper abdominal skin and soft tissue. The presence of a preexisting vertical scar should make one consider this approach if there is also significant horizontal laxity. Dissection and undermining should be limited to that which is required to perform central plication, as extensive undermining past the xiphoid process can compromise the blood supply of the flaps. It is critical that the surgeon acknowledge that the full amount of horizontal resection *and* the full amount of vertical resection may not be possible. Movement of the abdominal flaps in both vectors should be considered prior to final resection [9]. It is important to counsel patients undergoing this procedure about the increased complication rates, including wound-healing concerns at the confluence of the incisions. This technique, however, can help eliminate both horizontal and vertical excess when appropriate.

23.6.1.4 Monsplasty

The mons may exhibit both horizontal and vertical excess and is almost always ptotic in the

Fig. 23.4 Preoperative photograph of a good candidate for a Fleur-de-lis type abdominoplasty. Notice the excess skin in both the vertical and horizontal directions

MWL patient. Vertically, a wedge excision may be incorporated to reduce the width of the mons. For proper shaping, tissue must be thinned to match the thickness of the abdominal flap. The mons superficial fascial system must be suspended up to the abdominal wall and superficial Scarpa's fascia. Care must be taken not to suspend the mons too high, as this may lead to displacement of the clitoris or urethral meatus.

23.6.1.5 Panniculectomy

A panniculectomy is indicated in the patient with a large amount of abdominal skin and soft tissue with a high BMI. While it would be optimal to have the patient lose weight prior to a more definitive abdominoplasty, there is a subset of patients who are too big to exercise and have complications related to the excess tissue. The goal of this procedure is to alleviate a major impediment to allowing the patient to exercise and become a possible candidate for bariatric surgery in the future. The procedure involves a wedge excision of affected tissue with no undermining, plication, or umbilicoplasty.

23.6.2 Breast Procedures

Correction of breast deformity after MWL can be extremely difficult, with recurrent ptosis and residual deformities difficult to avoid. The breast deformities seen following MWL include volume loss, ptosis (grade 3 or 4), distortion, asymmetry, medialization of the nipple, lateral chest wall laxity, and axillary fat roll extension to the posterior torso (Fig. 23.5). Descriptions of reduction mammoplasties can be seen as early as Paulus of Aegina (625–690 AD), but it was not until the late nineteenth century that attention was turned to correcting ptosis of the breast [18].

Beyond typical breast changes of glandular tissue loss and ptosis, MWL patients tend to present with more asymmetrical volume loss and flatness of the upper pole than the typical breast reduction candidate. Skin laxity is very apparent and the degree of excess skin can be significant. Some degree of asymmetry almost always occurs following reduction mammoplasty and/or mastopexy. Many patients benefit from liposuction with autologous fat grafting in addition to excisional surgery. Proper planning is required when the decision is made to use an

Fig. 23.5 Massive weight loss patient with loss of volume and resultatnt ptosis of bilateral breasts

implant in conjunction with parenchymal modifications. In this patient population, multiple procedures are frequently required to achieve acceptable aesthetic results.

23.6.2.1 Breast Reduction

For larger, broad-based, and severely ptotic breasts, standard breast reduction techniques that utilize either Wise-pattern/McKissock or vertical skin patterns may be performed (Fig. 23.6). Wise pattern resection technique, with an inverted T-scar using an inferior pedicle, is most often preferred for this patient population. However, this approach can lead to square and flattened breasts with a high risk of bottoming out [19]. Although the vertical techniques avoid the inframammary scar, it is difficult with severe glandular ptosis to adequately reduce the excess skin between the areola and inframammary fold.

Mastopexy

The history of mastopexy closely parallels that of breast reduction since both procedures attempt to alter the skin envelope and shape of the breast.

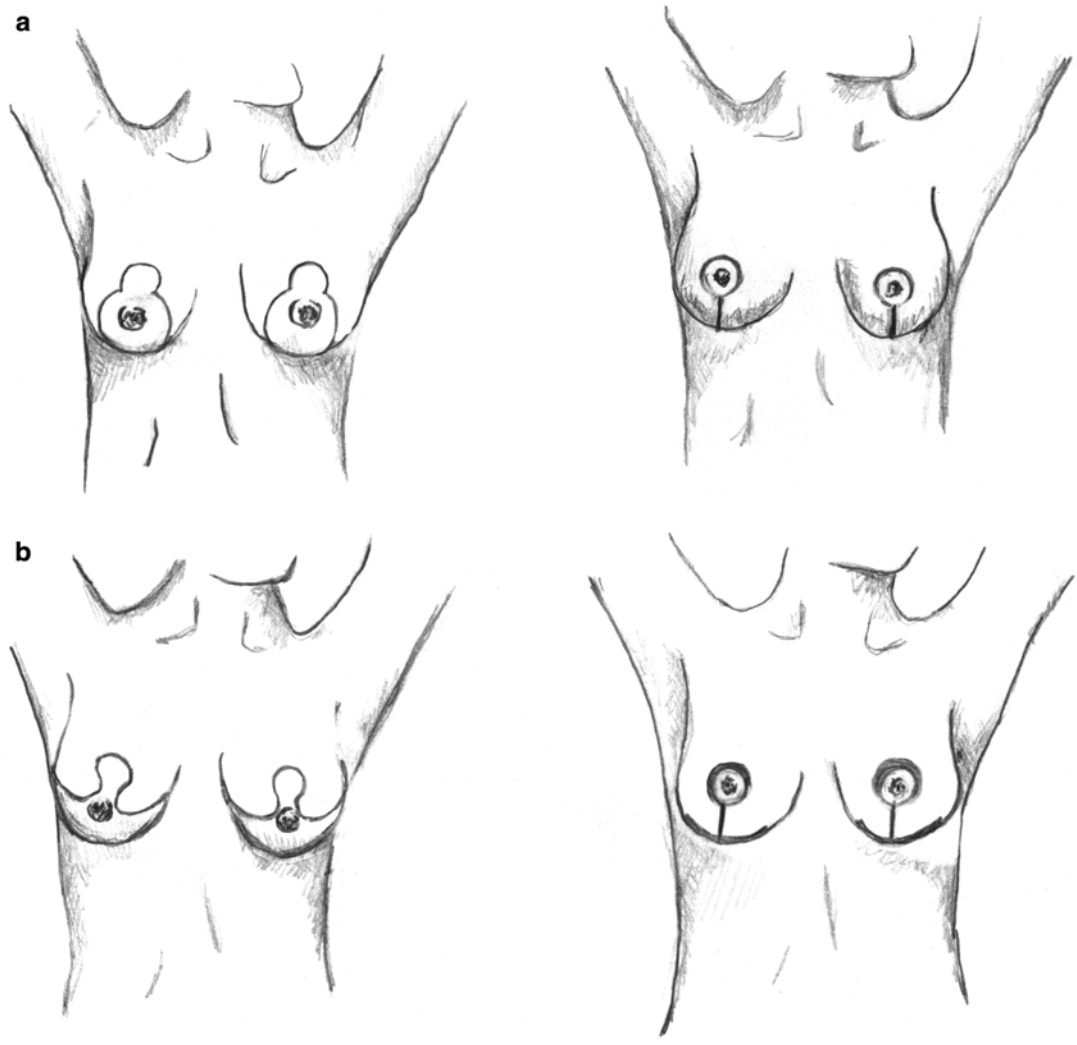

Fig. 23.6 Surgical procedures for breast reduction. (**a**) Design of vertical skin resection pattern and resultatnt scar, (**b**) Design of Wise or anchor resection pattern and resultant scar

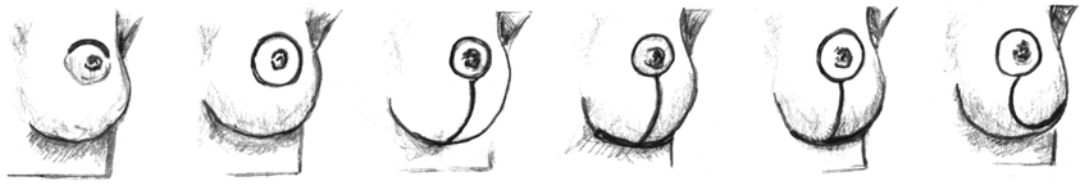

Fig. 23.7 Surgical procedures for mastopexy-primary pattern of scars. (**a**) Periareolar lift (crescent), (**b**) Circumareolar lift (Benelli, Donut), (**c**) Circumvertical lift (lollipop), (**d**) Anchor lift, (**e**) Anchor with areolar reduction lift, (**f**) B Mastopexy (Regnault)

Most of the essential technical elements of the mastopexy had been developed by the 1930s. Involutional changes caused by MWL exacerbate the laxity of the suspensory ligaments (Cooper's ligaments) and skin envelope, causing descent of the breast tissue and nipple-areolar complex (NAC). To properly correct these changes, elevation of the breast parenchyma is necessary. Mastopexy attempts to lift and reshape the breasts without removing much volume. The NAC must be transposed and the redundant skin envelope must be removed. Most mastopexies involve elevation of the breast mound using suspension techniques. Guidelines for the different types of mastopexies can be combined and modified for each individual patient (Fig. 23.7).

Autoaugmentation

After MWL, breasts change dramatically with a large reduction in the volume of fat. This reduced volume of fat results in poor shape and projection. Due to the major loss of volume and the inelasticity of the skin, preservation of autologous tissue can become important and offer another option for augmentation. This concept implies repositioning of available breast tissue rather than resection. Rubin described a mammoplasty technique for these patients involving de-epithelialization, total parenchymal reshaping, and dermal suspension sutures to the chest wall. With this technique, the tissue of the prominent axillary skin fold, typically found in patients with MWL, is used to increase the upper pole volume of the breast [20]. An inferiorly based parenchymal flap, with superiorly based NAC flap, provides central breast projection. The inferior flap is de-epithelialized, with the option of extension into the lateral chest wall and axilla, and placed behind the superior flap. Suspension sutures support the inferior flap and prevent ptosis from recurring. This technique helps to preserve the volume of the deflated breast and reduce the lateral axillary roll deformity in patients with MWL (Fig. 23.8). Regardless of the technique used, the aesthetic goal is to achieve adequate projection, superior fullness, correctly positioned NAC, and bilateral symmetry with a well-defined inframammary fold.

23.6.3 Extremity Procedures

23.6.3.1 Brachioplasty

MWL patients can present with excess skin in their upper arms that may extend into the posterior axilla, down onto the chest wall and distally below the elbow. Rarely, liposuction alone will provide adequate contouring. Correa-Iturraspe and Fernandez first described arm reduction surgery, mainly brachioplasty in 1954 [20]. Since then, many advances and modifications have been made. More recently, in 1995, Lockwood introduced a technique that involved fixation of the superficial facial system suspension, anchoring the arm flap to the axillary fascia [21]. Suturing of the superficial facial system results in a smoother contour and finer scar. As these procedures have become more refined, results have continued to improve.

Multiple techniques for brachioplasty exist including the limited technique, traditional, and extended techniques. After MWL, the arm and lateral chest demonstrate a great deal of variation. A thorough evaluation of each patient should be performed to assess the degree of skin laxity, tone, quality and extension. If the patient is found to have considerable fat excess throughout the

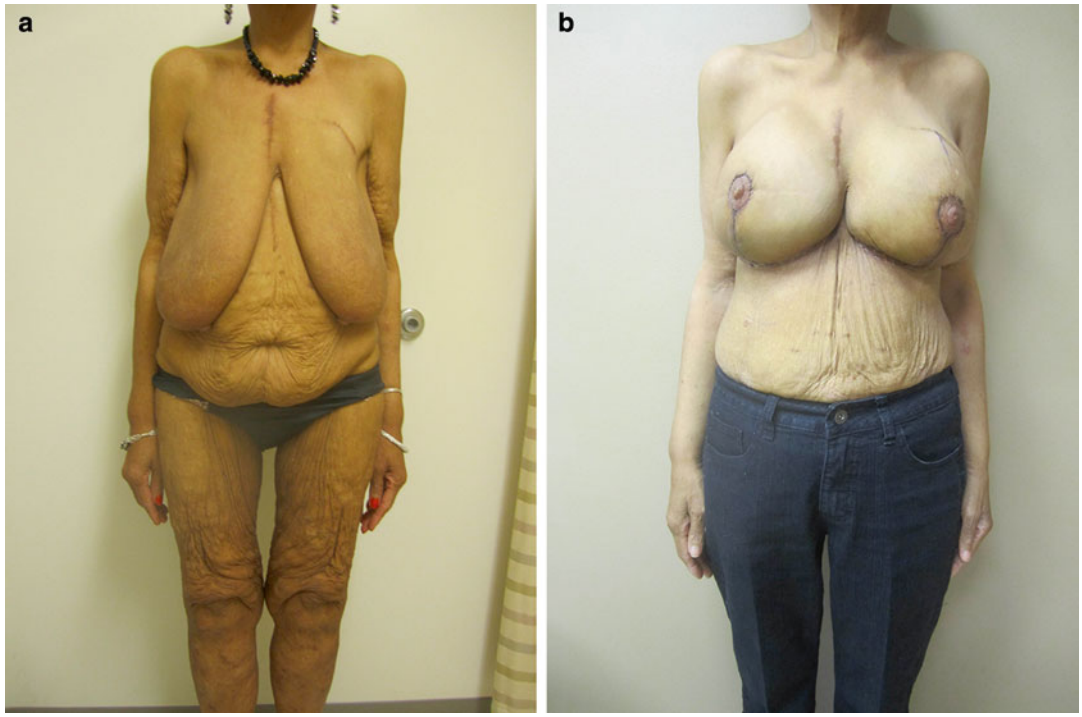

Fig. 23.8 (**a**) Preoperative frontal veiw of massive weight loss patient with deflated breasts with excess skin and ptosis (**b**) postoperative photograph of the same patient status post breast reduction with autoaugmentation reconsturction

upper arm, they may require a staged contouring procedure with liposuction as a first stage. This helps to debulk, contour, and loosen the subcutaneous tissue plane, allowing for an easier flap dissection. Ultimately, the degree of skin laxity and excess skin determines what type of approach should be undertaken.

Traditional Technique

The traditional technique is best performed on patients with redundancy of the entire upper arm. The scar in this technique extends from the axilla to the elbow and addresses both longitudinal and transverse skin excess (Fig. 23.9). The incision is often in the antebrachial groove, with deepening of the incision to the fascia of the overlying muscle and neurovascular structures [22]. When performing the resection, rotation of the skin and fat toward the proposed incision line and toward the axilla helps with residual excess near the elbow [23]. The majority of patients feel that the medial placement of the scar in the bicipital groove is the most aesthetically and functionally pleasing. While this sufficiently hides the scar, care must be taken to avoid injury to the medial brachial and antebrachial cutaneous nerves, which descend in the arm near the basilic vein, anterior and medial to the brachial artery (Fig. 23.10). These cutaneous nerves provide pain and pressure sensation to the medial arm and elbow. Injury to the nerves can result in neuropathic pain that extends into the forearm.

Limited Incision Brachioplasty

The ideal patient for the limited technique has mild to moderate amount of fat and skin excess in the upper one third of the arm with isolated skin excess in the longitudinal direction. The excision in this technique, an ellipse created in the crease of the underarm in the anteroposterior direction, is limited to the axillary region [24]. This is a less invasive procedure and is often complemented with liposuction to contour the upper arm.

Fig. 23.9 Preoperative photograph of a massive weight loss patient who is a candidate for the traditional technique bilateral brachioplasty due to skin excess in both the longitudinal and transverse directions

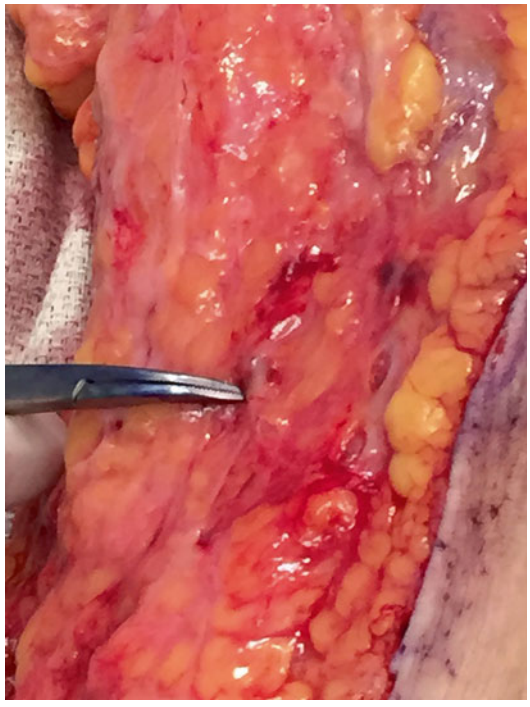

Fig. 23.10 Exposure of the medial brachial cutaneous nerve which should be preserved during brachioplasty

23.6.3.2 Extended Arm Lift

Many patients presenting for brachioplasty also have skin excess of their lateral chest wall with a "bat wing" deformity. These patients can benefit from an extended arm lift procedure. This procedure is similar to the traditional brachioplasty, except the incision is extended along the arm down the lateral chest wall to the level of the inframammary fold. Preoperative marking is critical because once the patient is lying supine on the operating table, it becomes very difficult to judge the amount of excess skin and fat to be removed without pulling the breast laterally. To avoid contracture, a Z-plasty may be used in the axilla. A drain is usually placed from the axilla down to the distal chest wall.

Complications with brachioplasty occur less frequently than with other body contouring procedures, even in the MWL population. The most vexing side effect is an unsightly scar, most commonly near the elbow. While several techniques exist to manage excess skin of the arms and axilla, and newer techniques continue to evolve, meticu-

lous physical exam and knowledge of the anatomy enhances the ability to perform the safest, most aesthetically pleasing contouring procedure.

23.6.3.3 Upper Body Lift

Many patients have extensive skin rolls of the back and lateral chest that cannot be thoroughly addressed with an extended brachioplasty. These patients are good candidates for an upper body lift (UBL). The main goal of an UBL is to correct the horizontal skin excess that exists on the posterior trunk and lateral chest wall. The thorax is comprised of anterior and posterior zones of adherence that prevent the overlying skin from movement during weight fluctuations. The lateral aspect of the inframammary fold is less adherent, which can result in vertical decent, leading to a poorly defined fold [9]. In these patients, a transverse excision can be combined with brachioplasty or merged with a mastopexy scar to fully contour the upper body. Although zones of adherence and position of skin laxity and fat rolls will dictate scar placement posteriorly, attempts should be made to conspicuously hide the scar in the bra-line, if possible. If a lower back lift is also to be performed, the UBL is best carried out at a different time due to the opposing lines of tension. This can lead to suboptimal tissue resection, vascular compromise and banding across the back.

23.6.3.4 Thighplasty

The thighs can be as variable as the upper arms in the MWL patient in terms of excess skin versus residual fat. Similarly, there are a variety of types of thigh lifts, differentiated by the excisional technique used to remove excess skin, fat and tissue. When significant thigh laxity exists, a thigh lift alone or in combination with liposuction and a lower body lift can dramatically correct laxity and improve overall shape and contour of the MWL thigh.

Lewis first described the medial thigh lift in 1957 [25]. Refinements made by Lockwood in 1988 [26], with a vertical vector of pull, included fascial anchoring. However, this traditional approach was limited to patients with minimal to moderate laxity and insufficiently addressed the greater degree of excision necessary for the MWL patient. As such, to address the needs of these patients, a myriad of technical variations to reorient the vector of pull from vertical to horizontal, with and without staging liposuction, have been described [27]. Medial and circumferential thigh laxity can be addressed through a proximal thigh lift procedure, with scars hidden in the groin creases, or a vertical thigh lift procedure, with more extensive excisions.

Proximal Thigh Lift

The proximal thigh lift is designed ideally to address only the proximal medial thigh, with the excision tapering into the abdomen anteriorly and infragluteal fold posteriorly. This procedure is limited to those patients with proximal medial thigh skin that has lost its elasticity due to weight fluctuations. Although small amounts of improvement can be seen in the distal medial thigh, this is not the procedure of choice for patients with circumferential thigh laxity extending from the pubic area down to, or past the knee.

Vertical Thigh Lift

The vertical thigh lift procedure is a much more powerful and useful tool in tightening and shaping the thigh compared to the proximal thigh lift [28]. It is the procedure of choice for patients with significant skin laxity and poor skin quality and excess. The incision extends from the origins of the gracilis down to the knee, and may be shortened or extended beyond, if necessary. It is important to inform patients about the extent of the scar that accompanies this procedure (Fig. 23.11). Concomitant liposuction is often performed first, with the goal to remove the majority of the subcutaneous fat from the resection area. Suspension of the superficial facial system in the thigh to the Colles fascia system in the groin is performed to assure superior contour outcomes. This procedure provides circumferential thigh tightening and elimination of skin laxity from the groin to the knee, and therefore is the procedure of choice for the MWL patient.

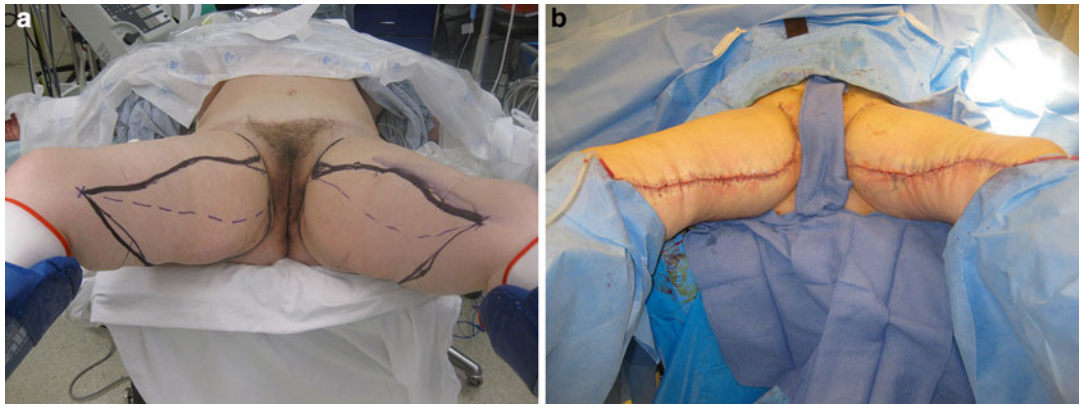

Fig. 23.11 Intraoperative photos of vertical thigh lift procedure. (**a**) Intraoperative markings (**b**) primary closure of vertical thigh lift incisions immediately postoperatively on operating room table

23.6.3.5 Lower Body Lift

Massive weight loss patients appropriate for lower body lift include those individuals with excessive tissue laxity of the lower abdomen, buttocks and thighs who ideally have reached a normal, steady BMI. In the lower body lift, the abdomen, thigh and lower back are approached as one unit. Ancillary procedures including autologous buttock augmentation may also be included, as many MWL patients experience substantial volume loss in the gluteal region. This combination of procedures frequently leads to superior functional and aesthetic outcomes as compared to single procedures. Liposuction can be added to help improve contour in the region of the lateral thighs and aid in lifting of the outer thighs.

Incisions for the belt lipectomy are often made to hide the scar in undergarments or swimwear. To carry out these procedures, a low transverse mark is made at the level of the pubic symphysis and lengthened out on either side to the iliac crest. The posterior incision is carried out along the upper portion of the buttocks. The amount of tissue resection from the posterior body is always conservative in the midline secondary to the strong zones of adherence present in this region of the body.

Performing a belt lipectomy without additional attention to gluteal reconstruction may result in a flat buttock with lack of waist definition [29]. If autologous gluteal augmentation is to be performed, the posterior incisions need to be low enough to allow for rotation of tissue low into the buttock region. When this procedure is simultaneously performed, the amount of posterior tissue resection is reduced to accommodate the flap volume that will be rotated into the pockets created over the gluteal muscles. Additionally, autologous fat transfer may be used in select patients as an adjunct to enhance the buttock shape. Many of these patients do not have adequate volume at the donor sites for fat harvest. Nonetheless, it is a clinically effective technique that may play a vital role in contouring.

Attention is then turned to areas for concurrent liposuction, including the outer thighs, if appropriate. The posterior incisions are tapered anteriorly into the abdominoplasty markings if a circumferential belt lipectomy is also planned. In addition to lower back lift, gluteal augmentation, and abdominoplasty, thighplasty may also be performed, constituting a total lower body lift procedure.

23.6.4 Face and Neck Procedures

Removing excess skin around the face and neck in patients after MWL is a similar procedure to those done for non-massive weight loss patients. However, there is often increased skin laxity and decreased elasticity in the MWL patient, requir-

ing more aggressive volume augmentation and skin resection. Common problem areas encountered after MWL include the midface, jowls, and neck. There can be loss of volume in the midface, bowing of the submental angle, and an excess of laxity in the neck. Excessive facial fat atrophy coupled with redundant skin and underlying supporting tissue leads to loss of facial contours [30]. A multiplanar rhytidectomy ("facelift") can address both skin laxity and volume deficiency in order to restore the desired youthful neck and facial contours. The incisions are kept within the hairline superiorly, junction of the ear and face laterally, and postauricular posteriorly. The addition of fat grafting to the deficient malar areas can further restore lost volume and improve the appearance of the face. The neck can be addressed at the same time to reposition lax skin and remove excess skin. If there is excess skin around the eyes, this may be addressed with either upper and/or lower blepharoplasty.

23.7 Complications

Despite the positive aesthetic and psychosocial outcomes of body contouring procedures, MWL patients are at an increased risk for complications secondary to potential nutritional deficiencies, persistent obesity, and decreased tissue quality including damage to the extracellular matrix [31]. While complications may be minor or major, minor ones occur most frequently in this patient population and as such, should be discussed throughly during the informed consent discussion [32]. Although overall complication rates are higher in this patient population, complications are generally accepted by patients because of the dramatic aesthetic and functional benefits that come with these procedures.

Wound dehiscence is the most common complication in body contouring procedures, with reported rates as high as 60 % [33]. This remains true whether single or multiple procedures are performed at once. Increasing the number of simultaneous procedures appears to increase the incidence of wound dehiscence [13]. Wound dehiscence can occur early in the postoperative period or later. Early dehiscence is often due to inappropriate patient movement/mobilization. Patient and nursing team education is very beneficial to minimize this risk. Dehiscence later in the wound healing process is most often due to an underlying seroma. In general, wound dehiscence is most commonly managed with local wound care and/or debridement of dead tissue to optimize healing and reduce infection [34]. Smaller wounds will often heal by secondary intention, whereas larger wounds, once cleaned and debrided, may require an operative closure.

Seromas pose the second most common complication of body contouring following MWL, with reported rates averaging 15 % [35]. Seromas are fluid collections that develop under the skin and may result in undermining, large cavities, drainage and chronic wounds. Shear forces, which are more common in the obese patient population, can exacerbate them. To minimize the risk of seroma formation, close attention must be paid when performing superficial dissection near any lymph node basin. Dissection in this area can disrupt lymphatic channels, which increases the risk of lymphoceles [35]. Preserving a thin layer of fat may help diminish the risk of seroma by maintaining a thin layer of lymphatic drainage. Intraoperative measures including hypothermia avoidance to prevent the patient's body temperature from dropping below 36 degrees Celsius has demonstrated a decreased incidence of seroma formation [36]. Closed drains are often placed intraoperatively to drain any fluid that may develop after surgery. Additional interventions include progressive tension sutures and three-point suturing of the superficial facial system to the deep fascia. Fibrin tissue sealants and doxycycline are other modalities that have been used with inconclusive evidence.

Scarring may develop as a result of poor wound healing, fat necrosis, or infection. Excess skin laxity in MWL patients predisposes them to dog-ears and widened scars along their incisions. Additionally, hypertrophic and keloid scarring may occur. Many of these scars can be treated with intra-lesional steroid injections and/or excised under local anesthesia in the office.

Larger areas of scar due to fat necrosis or infection may require additional trips to the operating room. Scar migration is another issue that may arise and require further operations to correct. Scar migration is most noticeable in the extremities and may occur as a result of poor skin quality, poor preoperative planning, or failure to fix the tissue to the underlying superficial fascial system. When planning the various scar placements over the body, it is important to remain cognizant of the zones of adherence in order to minimize scar migration.

Fat and skin necrosis complicates approximately 6–10 % of postbariatric body contouring procedures [37]. Individuals who are current smokers or have recently quit have a much higher rate of occurrence due to the interruption of blood supply to the skin. More limited, partial thickness necrosis can often be treated with local wound care including wet to dry dressing changes, topical bacitracin or silver sulfadiazine. For large areas of fat necrosis or significant undermining, operative debridement and/or readvancement of skin flaps may be necessary to achieve a satisfactory aesthetic result [35].

Due to the extensive nature of body contouring surgery, postoperative bleeding may occur. Patients with hypertension or taking blood thinners (including herbal remedies) are at increased risk. Additionally, patients undergoing these procedures often have underlying vitamin deficiencies, namely, iron deficiency anemia, Vitamin B_{12} (cobalamin) and fat-soluble vitamin deficiency; all of which may lead to blood clotting disorders and subsequent hematoma formation. Active bleeding usually occurs within 24 h of surgery. It may lead to anemia. In some cases, blood transfusion may be necessary. In general, the diagnosis is usually clinically evident. Warm compresses, aspiration, or immediate return to the operating room to coagulate bleeding vessels may be necessary depending on the severity of the bleeding and/or hematoma formation.

Infection is another complication that may occur. Although the rate of postoperative infections is low (1–2 %), infections are more common in the postbariatric patient than in non-bariatric patients undergoing cosmetic body contouring [38]. Obesity is linked to alterations in the cutaneous microcirculation and macrocirculation. Low perfusion accompanying obesity and diabetes, as well as modified collagen structure and function, leads to deficiencies in wound healing mechanisms.

Common bacteria isolated are those found in the skin flora including *Staph. aureus, Corynebacterium* and *Streptococcus*. Methicillin-resistant *staph aureus* (MRSA), which has become increasingly prevalent in the hospital and community, should also be considered. If infection is suspected, wound cultures should be obtained to help direct antimicrobial treatment. In the setting of failed antibiotics and continued cellulitis, there should be a high suspicion for *Candida*. To minimize infections perioperatively, antibiotics are often given and continued until drains are removed. Serious and life threatening infections are extremely rare, and although infrequent, may require additional operations.

Nerve injury with resulting neuropathy is a rare but potentially debilitating complication of body contouring procedures. This may occur due to direct nerve laceration, traction injury, or patient positioning leading to stretch or compression of the involved nerve. Knowledge of anatomy and adequate padding and positioning are critical to minimize nerve injury. Depending on the extent of injury, patients may have temporary paresthesias, or in rare cases, neuropraxia (diminished or complete block of conduction across a nerve segment). Expectant management is most often used, utilizing massage, prescription medications and physical therapy. This frequently results in improvement over time. In the case of persistent symptoms, consultation with neurological services, nerve conduction studies and electromyography may be necessary.

The risks of major life threatening complications after postbariatric body contouring procedures are very low and although mortality is exceedingly rare, it cannot be ignored as a potential complication. Less common but serious complications include deep venous thrombosis, pulmonary embolism and myocardial infarction. Thromboembolic complications have been

reported with an incidence of up to 9.7 % in patients undergoing body-contouring procedures [39]. Conservative measures for venous thromboembolism include early postoperative ambulation and placement of sequential compression devices. Additionally, medical management may be warranted. A general administration of low molecular weight heparin has been associated with a reduction of thromboembolic complications down to the range of 0–2.9 %.

23.8 Conclusion

The goal of body contouring procedures is to address the patient's aesthetic and body image concerns in a safe, effective and definitive manner. As the number of body contouring procedures increases, so does the necessary time to recover from these surgeries. The majority of MWL patients are extremely pleased with their functional and aesthetic outcomes. Due to the excessive amounts of subcutaneous tissue and skin laxity, it is challenging to achieve exact symmetry. Preoperative and postoperative photographs are often beneficial to remind patients of where they started, and where they have come. Recovery from these procedures can be a long and arduous process; one that requires a lifetime of commitment to a truly life changing transformation.

Body contouring surgery for the MWL patient has become an established field of surgery, which requires a dedicated and comprehensive approach to optimize outcomes and minimize complications in this challenging patient population. There are many additional techniques that can be utilized to accomplish the goals and results outlined throughout this chapter. The skilled surgeon must appreciate the uniqueness of each individual patient and their dynamic anatomies must be taken into account and respected.

References

1. Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. JAMA. 2012;307:491–7.
2. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. JAMA. 2005;293(15):1861–7.
3. Dietz WH. Childhood weight affects adult morbidity and mortality. J Nutr. 1998;128:411S–4.
4. National Institutes of Health, Poires W. Bariatric surgery for severe obesity. http://www.niddk.nih.gov/health-information/health-topics/weight-control/bariatric-surgery-severe-obesity/Pages/bariatric-surgery-for-severe-obesity.aspx. Accessed 1 Apr.
5. North American Association for the Study of Obesity and the National Heart, Lung and Blood Institute. The practical guide: identification, evaluation, and treatment of overweight and obesity in Adults (NIH Publication No. 00–4084). Bethesda, Md: National Institutes of Health. 2000.
6. Kenkel, J. A review of bariatric surgery procedures. Plast Reconstr Surg. 2006;117 (1):8S-13S.
7. Borud LJ, Lin SJ, Rezak KM. Atlas of body contouring after weight loss. Bariatric Times. 2011;8(6):11.
8. Kenkel, J. The physicologic impact of bariatric surgery on the massive weight loss patient. Plast Reconstr Surg. 2006;117 (1):14S–16S.
9. Barclay L, Vega CP. Smoking increases risk of wound infections even for simple wounds. Ann Surg. 2003; 238:1–5. 6–8.
10. Friedland JA, Maffi TR. Abdominoplasty. Plast Reconstr Surg. 2008;121(4):1–11.
11. Shermak MA. Body contouring. Plast Reconstr Surg. 2012;129(6):963e–78.
12. Rubin P. Principles of plastic surgery after massive weight loss. In: Charles H. Thorne, editor. Grabb and Smith's Plastic Surgery. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014.
13. Cood D, Michaels V J, Gusenoff JA, Purnell C, Friedman T, Rubin JP. Multiple procedures and staging in the massive weight loss population. Plast Reconstr Surg. 2010;125(2):691–8.
14. Kelly HA. Report of gynecological cases (excessive growth of fat). Johns Hopkins Med J. 1899;10:197.
15. Wilkinson TS. Limited abdominoplasty techniques applied to complete abdominal repair. Aesthetic Plast Surg. 1994;18:49.
16. Greminger RF. The mini-abdominoplasty. Plast Reconstr Surg. 1987;79:356.
17. Dellon AL. Fleur-de-lis abdominoplasty. Aesthetic Plast Surg. 1985;9(1):27–32.
18. Eisenhardt SU, Nienhueser H, Braig D, Penna V, Bannasch H, Torio-Padron N. Comparison of the Rubin dermal suspension sutures and total parenchymal reshaping technique with a traditional inverted T-Scar reduction mammaplasty technique using a superior pedicle. Aesthetic Plast Surg. 2013; 37(6):1153–60.
19. Antony K, Yegiyants S, Danielson K, Wisel S, Morris D, Dolezal R, Cohen M. A matched cohort study of superomedial pedicle vertical scar breast reduction (100 breasts) and traditional inferior pedicle Wise-pattern reduction (100 breasts): An outcomes study over 3 years. Plast Reconstr Surg. 2013;132(5):1069.

20. Correa-Iturraspe M, Fernandez JC. Dermolipectomia braquial. Prensa Med Argent. 1954;34:2432.
21. Lockwood T. Brachioplasty with superficial fascial system suspension. Plast Reconstr Surg. 1995; 96(4):912–20.
22. Shermak MA. Technical refinements in upper arm contouring. Plast Reconstr Surg. 2010;126:1365–9.
23. Downey SE. Brachioplasty and upper trunk contouring. In: Charles H. Thorne, editor. Grabb and Smith's plastic surgery. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014. p. 707–12.
24. Trussler AP, Rohrich RJ. Limited incision medial brachioplasty: technical refinements in upper arm contouring. Plast Reconstr Surg. 2008;121(1):305–7.
25. Lewis JR. The thigh lift. J Int Coll Surg. 1957;27: 330–4.
26. Lockwood TE. Fascial anchoring technique in medial thigh lifts. Plast Reconstr Surg. 1988;82:299–304.
27. Gusenoff JA, Coon D, Nayar H, Kling RE, Rubin JP. Medial thigh lift in the massive weight loss population: outcomes and complications. Plast Reconstr Surg. 2015;135(1):98–106.
28. Hunstad JP, Repta R. Grabb and Smith's plastic surgery. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014. p. 696–706.
29. Centeno RF, Mendieta CG, Young VL. Gluteal contouring in the massive weight loss patient. Clin Plast Surg. 2008;35:73–91. discussion 93.
30. Shrivastava P, Aggarwal A, Khazanchi RK. Body contouring surgery in a massive weight loss patient: an overview. Indian J Plast Surg. 2008;41(Supp l):S114–29.
31. Atiyeh BS, Ibrahim A. Body contouring following bariatric surgery and massive weight loss: post-bariatric body contouring. Co-Editors: Atiyeh B, Costaglioia M, 1st ed. Bentham Science Publishers; Beijing, China 2012.
32. Shermak MA, Chang D, Magnuson TH, Schweitzer MA. An outcomes analysis of patients undergoing body contouring surgery after massive weight loss. Plast Reconstr Surg. 2006;118:1026–31.
33. Michaels JV, Coon D, Rubin JP. Complications in postbariatric body contouring: postoperative management and treatment. Plast Reconstr Surg. 2011; 127(4):1693–700.
34. Brolin RE, Kenler HA, Gorman RC, Cody RP. The dilemma of outcome assessment after operations for morbid obesity. Surgery. 1989;105:337–46.
35. Michaels JV, Coon D, Rubin JP. Complications in post bariatric body contouring: strategies for assessment and prevention. Plast Reconstr Surg. 2011;127:1352–7.
36. Coon D, Michaels J, Gusenoff JA, Chong T, Purnell C, Rubin JP. Hypothermia and complications in postbariatric body contouring. Plast Reconstr Surg. 2012;130(2):443–8.
37. Coon D, Gusenoff JA, Kannan N, El Khoudary SR, Naghshineh N, Rubin JP. Body mass and surgical complications in the postbariatric reconstructive patient: analysis of 511 cases. Ann Surg. 2009;249(3): 397–401.
38. American Society of Plastic Surgeons. 2012 statistics. http://www.plasticsurgery.org/Media/Statistics.html. Accessed 1 Apr 2015.
39. Kitzinger HB, Cakl T, Wenger R, et al. Prospective study on complications following a lower body lift after massive weight loss. J Plast Reconstr Aesthet Surg. 2013;66:231–8.

24 Psychological Complications After Bariatric Surgery (Eating Disorders, Substance Abuse, Depression, Body Image, etc.)

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Key Points

- Up to 20 % of bariatric surgery patients fail to lose the expected amount of weight and psychological factors may be responsible.
- While no specific presurgical psychological factors have been identified that predict poor outcome from surgery, several postoperative psychological factors warrant concern and further research.
- Patients are not only interested in losing weight, but experiencing significant improvements in health, physical functioning and improvements in quality-of-life such as their self-esteem, intimate and social relationships as well as career functioning.
- Mental health professionals play a crucial role in preparing patients for surgery and addressing postoperative concerns to improve overall outcome and "success" from surgery.
- Cognitive-behavior therapy (CBT) interventions have demonstrated effectiveness in addressing a number of postoperative challenges including: disordered eating, mood and anxiety disturbances, body image, self-esteem, and improved interpersonal functioning.

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24.1 Introduction

There is now a significant body of research confirming that bariatric surgery is the treatment of choice for morbid obesity [1–3]. Many positive psychological benefits are associated with bariatric surgery, such as reduction in depression, improvements in body image and enhancement of various markers of quality of life. These improvements are maintained in some studies for over 5 years [4–9]. However, it has also been reported that up to 15–20 % of surgical patients fail to achieve a significant amount of weight loss [3] and psychological factors are often suggested as a possible contributing factor [10].

While few, if any, published studies suggest an *increase* in rates of psychopathology such as anxiety or depression as a result of bariatric surgery, this may be due to the fact that most patients with severe psychopathology are excluded from bariatric surgery. Most outcome studies include carefully screened populations of patients largely free of major psychological problems [11, 12]. While the number of patients who report negative psychological outcomes from surgery is small, it is important to make continued efforts to try to understand and predict which variables and which patients might experience such an outcome.

Postoperative bariatric patients often encounter report symptoms of depression, anxiety and other forms of emotional distress following

D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_24

surgery. When one considers the dramatic changes in behavior and functioning that bariatric surgery entails, this should not be surprising. Some reasons that individual patients may experience depression, anxiety or other psychological symptoms include: difficulty adjusting to changes around eating, changes in intimate romantic and social relationships and dissatisfaction with body image [13–19]. Previous authors have suggested that these concerns and others be addressed prior to surgery and that the possibility that such challenges may occur after surgery should be reviewed with patients beforehand [20, 21].

24.2 Postoperative Disordered Eating

Many studies have documented the prevalence of eating disorders among individuals seeking bariatric surgery. The prevalence of binge eating disorder (BED) in obese patients before bariatric surgery has been reported to be as high as 49 % [12]. The percentage of individuals who meet full criteria for a DSM Eating Disorder at the time of surgery may be lower, in part because many bariatric programs delay or deny patient surgery on the basis of the presence of an eating disorder.

The most common form of disordered eating reported among those seeking bariatric surgery is binge eating. BED has been described as the consumption of an objectively large amount of food within a brief period (generally 2 h or less) combined with a subjective report of a loss of control during the overeating episode [22, 23]. Studies vary as to their methodology for establishing the presence of binge eating and BED, with some studies relying upon on clinical interviews, DSM criteria, or various eating disorder questionnaires or combinations of these assessment methods.

Research on the impact of presurgical eating disorders, including binge eating, on postsurgical weight loss is variable. In a recent article, Livhits et al. [24] reviewed 20 studies that reported on the relationship between preoperative binge eating and postoperative weight loss ($n = 2661$) with 417 patients identified as binge eaters. Three studies reported that patients with preoperative binge eating lost more weight postoperatively than those without binge eating; 13 studies reported no association and 4 studies reported a negative association. Follow-up time and methods for assessing binge eating varied widely which may account for some of the variability in these results.

A growing body of literature suggests that *postoperative* disordered eating may have a greater negative impact on surgical outcome [12, 25–27]. It is important to note that the full criteria of BED, as defined by the DSM, are difficult to achieve following bariatric surgery since volume restriction precludes the intake of an "exceedingly large amount of food" in less than a 2-h period. Instead, the diagnosis of "binging" behavior after bariatric surgery must focus less on the absolute quantity of food consumed during an episode and more on subjective feelings of distress and loss of control of eating [26, 28, 29].

In a recent review of eating pathology after bariatric surgery, Marino et al. [30] concluded that while the development of classical eating disorders after bariatric surgery is a rare occurrence, sub-syndromal eating disorders are far more common. The authors recommend that an additional nomenclature to classify such behavior needs to be developed and studied further. Kalarchian et al. observed no binge episodes in patients 4 months status post bariatric surgery [31], however, 46 % of patients have reported either objective or subjective binge eating at longer follow-up [26]. Therefore, one must consider that the presence of binge eating may increase with time and distance from the date of surgery and longer-term monitoring and inquiry about such behavior may be warranted.

Other forms of disordered eating that have received investigation following bariatric surgery include grazing and Night Eating Syndrome (NES). Grazing refers to the consumption of smaller amounts of food over extended periods of time [32]. Grazing, like binging, often involves feeling unable to control one's behavior and as such can be considered to be quite similar to binge eating but over a more extended period of time. Night Eating Syndrome refers to the con-

sumption of 35 % or more of daily calories after the evening meal, often accompanied by frequent nocturnal awakenings during which patients snack as a means of returning to sleep [33]. Postoperative grazing and "uncontrolled eating" in which patients reported a loss of control during the consumption of a large amount of food has been associated with diminished weight loss following bariatric surgery [34]. The research on NES is inconclusive, in part due to the variability in its definition and measurement, and warrants further study [34, 35].

The presence of postoperative eating disorders and their effect on long-term outcome from bariatric surgery highlights the need for ongoing follow-up with these patients to continually assess for the presence of such problems and to provide counseling and interventions when necessary. As in the nonsurgical weight loss population, cognitive behavior therapy (CBT) interventions have been found helpful in modifying eating disorders and disordered eating in the bariatric population.

24.3 Mood and Anxiety Disorders

Depression is common in candidates for bariatric surgery with rates as high as 25–35 % [9, 36, 37]. Bariatric surgery results in significant positive effects on mood and quality of life in the initial years following surgery [9, 12, 15, 38, 39]. Less clear are the longer-term effects of bariatric surgery on mood. Given that the majority of weight lost from surgery occurs during the first 2 years, it makes sense that improvements in mood would occur during this early period. Further, there is evidence that the amount of weight loss is proportional to improvement in mood [40, 41], but it is difficult to determine which factor is the cause and which is the effect.

There are mixed data regarding the impact of a previous history of psychiatric disorders on surgical outcome. Some researchers have found that the presence of mood and anxiety disorders prior to surgery is not a contraindication for surgery, and could in fact be prognostic of a positive outcome [40, 42, 43]. However, other studies suggest it may [44–48] adversely affect outcomes.

As is the case with disordered eating, the *reoccurrence* of anxiety and depressive disorders *following* surgery might have a stronger impact on weight loss than presurgery diagnoses [47]. De Zwaan et al. recently studied the course of preoperative and postoperative anxiety and depressive disorders using face-to-face interviews with 107 obese bariatric patients. They found that the point prevalence of depressive disorders decreased significantly after surgery whereas the point prevalence of anxiety disorders did not [48]. Additionally, as with other studies, the presence of a postoperative depressive disorder was associated with lower weight loss at 24–36 months [47]. Postoperative anxiety disorder was not associated with the amount of weight loss at any time. The Swedish Obese Subjects (SOS) study [49] also showed that the depressive subscale scores on the HADS worsened over time after significant initial improvement. There is also the suggestion that the *severity* of the disorder rather than the type of disorder may be more relevant for outcome following bariatric surgery [12].

It could be suggested that after the bulk of the weight is lost, the novelty of weight loss begins to fade and patients are now faced with life after dramatic weight loss leading to the recurrence of depressive symptoms. Many patients describe this immediate post-op time period as the "honeymoon phase," where weight loss is rapid and continuous. Once again, this speaks to the urgent need to discuss patient expectations prior to surgery [20] so they are prepared for the eventual slow-down of weight loss and the resumption of life after surgery.

24.4 Bariatric Surgery and Suicide

Attention should be paid to recent research suggesting a relationship between bariatric surgery and increased rates of suicide [50–56]. These findings are troubling as the reasons for an excess of suicides among bariatric surgery patients remain unknown. While the absolute suicide rate

among bariatric patients is still quite low, it remains higher than in the general population. Factors responsible for this increase may include the emotional burden of severe obesity as well as a history of major depression. An excellent summary of these factors can be found in Wadden's 2007 review [33]. As suggested by Tindle et al., it may be possible that presurgical psychological distress is exacerbated by suboptimal results of surgery or inadequate improvement in quality of life [56]. Addressing patient expectations and definitions of success from surgery are thus quite important [20, 57]. Tindle's group further suggests that the influence of body image [58], and recurrence of psychiatric disorders and susceptibility to substance abuse [59] may also be related to these higher suicide rates. While more research in this area is needed, it is clear that the need for identifying higher-risk patients prior to surgery and monitoring their progress, mood and functioning after surgery is important.

24.5 Sexual Abuse

Some researchers have suggested a connection between medical and surgical outcomes and a previous history of sexual abuse [60–62]. Kral stated that in his experience, "the most critical 'psychological/psychiatric' predictor of negative outcome has been a history of abuse, whether sexual or other." [63].

Steinig's extensive review on the impact of sexual abuse on weight loss following bariatric surgery yielded mixed results [64]. In this review, the authors report on 13 studies that examined rates of sexual abuse among bariatric patients and 8 studies that investigated the effects of sexual abuse on surgery outcome. In all studies, patients initially lost weight following surgery. However, three studies demonstrated significantly reduced weight loss among sexually abused patients. The authors note that significant methodological differences among these studies make it difficult to draw conclusions. For example, almost none of the 13 studies provided a detailed definition of sexual abuse. Additionally, the studies varied considerably in their choice of tools to measure sexual abuse. Interestingly, the 3 studies documenting the highest prevalence rates of sexual abuse used the Childhood Trauma Questionnaire (CTQ) [65, 66], which the authors indicate provides a more detailed interrogation of sexual abuse as compared to a standard clinical interview.

It is possible that the more detailed the inquiry regarding sexual abuse, the greater the rate of abuse discovered. Additionally, actual rates of sexual abuse among bariatric patients could be far higher as patients might conceal experiences of sexual abuse during the preoperative psychological evaluation for fear that it may jeopardize their candidacy for surgery. It is known that posttraumatic stress disorder (PTSD) secondary to childhood sexual abuse is common in the morbidly obese population and may be underreported during the preoperative psychological evaluation to evaluators unknown to the patient. In one study of 340 weight loss surgery candidates assessed for childhood maltreatment, 32 % reported sexual abuse [67]. One study noted a sudden onset of PTSD symptoms after major weight loss in patients with a history of sexual abuse [68].

Steinig and colleagues note in their review a tendency for slower weight loss after bariatric surgery in subjects who have been sexually abused. However, there is no evidence that sexually abused patients do worse over the long term. The authors suggest that this speaks against the widely held assumption that sexually abused patients might fight against weight loss (sabotage) as a mechanism of self-protection against further attention although this may vary among individuals.

Based upon these findings, there is little evidence to support the exclusion of individuals with a history of sexual abuse from having bariatric surgery. The authors suggest the advice of Grothe [20] that patients should be questioned for their views on possible positive aspects of being obese to predict and prevent any negative psychological implication of the results of the bariatric procedure. Further research on the complex relationship between sexual abuse and outcome from bariatric surgery is clearly needed.

24.6 Substance Abuse Disorders

The literature on postoperative substance abuse largely focuses on the effects of alcohol. This author did not identify any studies demonstrating an increased use of other substances following bariatric surgery. While early articles hypothesized a mechanism of "addiction transfer," whereby patients replaced the consumption of food with the consumption of alcohol, this concept has been largely dismissed in favor of research demonstrating the changes in the body's absorption of alcohol particularly in the case of the Roux-en-Y gastric bypass (RYGB) procedure [69].

Specifically, serum ethanol levels reach their peak much sooner and take longer to return to zero than compared to nonsurgical controls [59, 70]. In addition, individuals have reported more rapid onset of the intoxicating effects of ethanol after surgery and that these effects are experienced after consuming fewer drinks [71]. Ethanol is metabolized partially in the stomach by the gastric ADH enzyme, which is reduced during gastrectomy, thus increasing serum ethanol levels [72]. Similar alterations in the ethanol pharmacokinetics are demonstrated with Vertical Sleeve Gastrectomy (VSG) [73]. No studies have been conducted to demonstrate similar changes following laparoscopic adjustable gastric banding (LAGB).

In a study of 51 patients, Suzuki and colleagues found no association between weight loss following surgery and the development of an alcohol use disorder (AUD) or other Axis I diagnoses [74]. However, significantly more current AUDs were reported by individuals with a lifetime history of AUD compared to those without a lifetime AUD, and by individuals undergoing Roux-en-Y gastric bypass (RYGB) compared to those undergoing LAGB. The authors concluded that individuals with a lifetime history of AUD may be at increased risk for relapsing after surgery. While none of the study participants met criteria for an AUD at the time of surgery, about 10 % met criteria for a current AUD 2–5 years after surgery, which is comparable to the prevalence found in the general population. Since the majority (83.3 %) of those meeting criteria for an AUD after surgery had a lifetime history of AUD, these cases represent relapses rather than the novel development of an AUD after surgery.

There is the suggestion of a connection between binge eating disorder (BED) and AUD among individuals seeking bariatric surgery. Some morbidly obese individuals with higher lifetime prevalence of AUD and BED may reduce their consumption of alcohol because eating or binging provides sufficient rewards that were previously provided by alcohol [75]. This is consistent with reports that highly palatable foods produce effects in the brain and brain chemistry that are similar to that produced by substances of abuse [76–78]. These results suggest that patients with a history of AUD should be informed of their potentially greater risk of relapse given the significant changes in alcohol metabolism particularly in the case of RYGB and VSG.

Similarly, in a recent prospective cohort study of 2458 participants across 10 US hospitals, King et al. found that the prevalence of AUD was greater in the second postoperative year than the year prior to surgery or in the first postoperative year. Additionally, it was associated with male sex, younger age and numerous preoperative variables including a history of AUD and choice of RYGB procedure [79]. The authors suggest that since the significant increase in postoperative AUD was observed in those undergoing RYGB primarily during the second postoperative year, an increase in alcohol sensitivity combined with resumption of higher level of alcohol consumption during the second year is likely responsible. The authors did not find a significant association between preoperative mental health, depressive symptoms, binge eating or past-year treatment of psychological or emotional problems and postoperative AUD. However, they did note that worse postoperative mental health and postoperative treatment for psychiatric or emotional problems were significantly associated with AUD. This again suggests the need for close monitoring and provision of mental health services following surgery, especially among those with a history of AUD who undergo RYGB and possibly VSG.

Currently, there is little empiric evidence that bariatric surgery increases the risk of substance

use or other addictive behaviors, but additional research is certainly warranted [80]. Many surgical practices either deny or postpone surgery for candidates with active substance abuse at the time of the presurgical psychological evaluation, likely for fear that such behavior impacts judgment or is a predictor of poor impulse control or other factors that will adversely affect compliance and outcome. A significant number of individuals with a past history of substance abuse present for surgery and there is little evidence to suggest that these individuals do worse than other candidates in terms of weight loss. However, these results must be interpreted with caution as most such individuals are eliminated from candidacy from surgery.

What is clear is that patients with a history of substance use or abuse need to be counseled prior to surgery about the potential for relapse after surgery, perhaps especially for those having RYGB or VSG. This psychologist has noted that during the presurgical evaluation, many patients with a history of substance use and abuse believe that their eating behavior changed following their termination from using substances, specifically that eating took the place of the use of their former drug. This phenomenon of increased eating is commonly described by individuals following their termination from cigarette smoking, but is also acknowledged in the case of alcohol and other substances. While biochemical causes of this behavior are being explored, behavioral causes must be considered as well. It has been this author's experience that many patients reported that twelve-step programs they have attended actually *encouraged* attendees to substitute sugar and other food-items in favor of alcohol if necessary to maintain their sobriety, thereby training such behavior.

Because of the prevalence of this behavior and many patients' belief that food has taken the place of previous substances, this issue should be addressed prior to surgery. While it remains uncertain if patients with histories of substance abuse are at greater risk of relapse following bariatric surgery, it is prudent to make patients aware of the possibility that relapse could occur and, if so, to immediately bring it to the attention of the bariatric team and/or other health professionals.

24.7 Body Image

Modern Western culture denigrates excess weight and stigmatizes obese individuals [81]. Among the severely obese, impaired body image is commonly observed [82, 83]. Risk factors for poor body image among the obese include the severity of obesity and female gender [84]. Most studies demonstrate improved body image following weight loss surgery [58, 85–87], however there are some inconsistencies that may be due to combining surgery types within studies as well as with the various methods used to assess body image. Improvements in body image following massive weight loss occur may be due to reductions in depression rather than to the percentage of weight lost [8, 88].

More than half of post-bariatric patients report that excess skin is a negative consequence of surgery [89]. Studies from other cosmetic procedures suggest that body image improves postoperatively [90–93]. Kinzl and colleagues noted that patients who achieved minor weight loss were more content with their appearance than patients who achieved more substantial weight loss [94]. Some authors have found that excess skin could interfere with additional weight loss or actually lead to weight regain [95].

In a study of 252 patients who underwent gastric bypass between 2003 and 2009, Kitzinger and colleagues found that that 90 % of women and 88 % of men felt their appearance was at least satisfactory or better after the massive weight loss [96]. However, 96 % reported loose and hanging skin, 27 % reported recurring itching beneath the excess skin, 70 % reported occasional itching and only 3 % reported no itching. Intertriginous dermatitis was described as recurring in 54 %, intermittent in 41 % and absent in only 4.7 %. A third of patients reported difficulty doing sports as a result of the excess skin, with only 4.2 % reporting no discomfort during physical activity. A majority (65 %) of patients reported some difficulty finding appropriately fitting clothing. As might be expected, women were more critical in their evaluations of individual body parts. Most patients (89 %) were informed about the possibility of needing body-contouring surgery either by their surgeon, other physicians

or the Internet. The reported discontent with body image was associated with a desire for body contouring surgery in 75 % of women and 68 % of men in this study. Sarwer and colleagues [97] showed that very overweight women are still not content with their body image even after massive weight loss.

In a study of 62 consecutive patients undergoing sleeve gastrectomy using the body image questionnaire (BIQ-20) to assess body image and the Patient Health Questionnaire (PHQ-9) to assess depression, there was an overall improvement in body image at 1-year follow-up [98]. However, there was no relationship found between body image and postoperative weight. The authors note that there are likely a number of variables that increase obese individuals' susceptibility to body image problems, many of which have yet to be identified. Some possible risk factors that have been identified include gender and physical appearance [84], traits like perfectionism or self-esteem [99]. The possibility exists that alterations in lower gut hormones after bariatric surgery may modulate body image, but we are far from knowing how. The authors comment that: "the most important improvement in body image seems to be due to the initial bariatric procedure, which may be enhanced by body contouring." It is important to note that body contouring leads to dissatisfaction with other parts of the body, suggesting that as patients become closer to their ideal, their ideal may shift [100].

In a study of 160 patients who underwent gastric bypass surgery, Steffen and colleagues found that the greater the patients' BMI at the time completing the postoperative questionnaire, the more likely they were to be dissatisfied with excess skin [101]. Additionally, their findings were consistent with previous findings in noting an inverse relationship between the time elapsed since surgery and the desire for contouring surgery [102], suggesting that patients may come to accept the excess skin over time. Of the 160 patients in this study, 32 had contouring surgery since their weight loss surgery. Most, but not all, reported greater satisfaction after contouring surgery. The authors suggest that this dissatisfaction may be due to perioperative complications or to scarring and other esthetic consequences from contouring surgery. No specific predictors of interest in body contouring surgery could be identified. The authors importantly comment that in the same way that bariatric surgery programs encourage patients to have realistic weight loss expectations, so too should they discuss the likely body image changes patients might experience with massive weight loss and the potential role of contouring surgery to address the excess skin after bariatric surgery.

In their study of 98 patients having body-contouring surgery after gastric bypass surgery as compared to a matched control-group of 102 patients without body contouring, Modaressi and colleagues demonstrated that gastric bypass surgery improves health related quality of life (HRQoL), HRQoL improvement is directly related to weight loss, and that body contouring surgery further improves HRQoL in comparison to gastric bypass surgery alone.

Self-esteem is the most affected aspect of HRQoL, especially in women between 35 and 64 [103]. Despite improvement in self-esteem after bariatric surgery, it still remained low after the weight loss. However self-esteem was further enhanced with body contouring. Interestingly, sexual activity is the only domain where the majority of patients experienced no change after gastric bypass and only minimal improvement after body contouring. The authors suggest the explanation offered by Herpetz [39] that partners have some difficulty adapting to their new image following weight loss. The authors conclude that given the significant additional improvements in HRQoL and satisfaction with their post-body contouring surgery despite major scars, that patients be informed about the potential benefits of undergoing body contouring surgery. They further suggested that body contouring may represent an intervention that improves psychosocial functioning that could serve to further strengthen the weight loss produced by bariatric surgery, an idea previously discussed by Kalarchian and colleagues [104].

24.8 Additional Psychological Challenges

Much of the research on psychological outcomes after bariatric surgery focuses on symptoms that are measured by clinical questionnaires or that have established criteria such as DSM psychiatric disorders. For example, investigations of "depression" generally rely upon instruments such as the BDI-II and related self-report measures or the definition of Major Depressive Disorder as defined by the DSM ascertained either by clinical interview or structured interviews such as the SCID. Similarly, the impact of surgery on quality of life is measured by any one of tens of instruments which may or may not comprehensively assess quality of life. However, this psychologist and many others are certainly familiar with numerous psychological struggles that bariatric patients encounter that either do not have formal names or do not reach a level of significance that presently warrants a DSM diagnoses [13, 57, 105]. Consider the following vignettes:

1. A male patient who has lost over 100 pounds since having gastric bypass surgery becomes committed to maintaining his weight loss through regular physical activity. He is now participating in organized running events and bicycle races in his local and extended community, some of which require him to travel. While he is quite excited about the improvements in his physical functioning, his new activities, and the new relationships he has made with others who share his interests, it has put considerable strain on his relationship with his wife. While happy for her husband and the improvements in his health, she does not share his enjoyment of physical activity and misses the man who used to watch television with her and engage in more sedentary activities. They are spending an increasing amount of time apart, which is further increasing the demands on her time to care for their children. While the patient is not experiencing a diagnosable mood or anxiety disorder, he is experiencing significant turmoil in his marriage and is feeling more distant from his wife and family.
2. A female patient who has been married for over 20 years to a man she met when they were both in high school. While never madly in love, she has always been committed to him and their two children and the life they share. She has now lost over 70 pounds since having bariatric surgery. Friends, coworkers, and acquaintances are reacting quite differently towards her as a result of the dramatic change in her appearance as well as changes in her level of self-confidence. She finds herself attracted to one of her male work associates, who feels similarly about her, and they have had a number of lunches together in recent weeks. She feels conflicted but is greatly enjoying the attention from this work associate, as she has never experienced this kind of attention from her husband or anyone else. She feels guilty about her behavior but acknowledges that she is no longer attracted to her husband and has not been for some time. She is thrilled with the attention from her coworkers and others, but is anxious about the conflict that has resulted from the changes in her body and behavior and what acting upon her desires could result.
3. A woman has lost over 100 pounds since surgery. While her friends were initially excited for her, she notices them becoming more distant. The patient recognizes that she is being invited out by her friends to go to dinner and other social events less often and conversations regarding eating and weight loss often exclude her. One friend commented: "You don't understand what it's like to struggle with eating and weight. You won your battle, we're still fighting ours." This was particularly hurtful to the patient, as she had struggled with her weight for over 25 years before having the surgery and continues to experience challenges eating and with her new body. While happy with her successful weight loss, she is greatly disturbed by the effects this has had on her social life.

Sogg and Gorman describes an extensive array of the many interpersonal, social, work-related, and other challenges faced by patients following bariatric surgery like those described above [13]. This remains a relatively new area of study and there is little research on this topic [15].

Many patients struggle with increased attention following dramatic weight loss and it may take considerable time for their self-concept to become consistent with their actual appearance [106]. Following surgery, the patient's body often changes faster than their self-perception. Patients often experience feelings of awkwardness in navigating social situations that would be simple for others, such as accepting compliments. Social skills are learned by experience and many formerly obese people have avoided social situations or engaged in a limited number of situations so that they have never developed such skills. This often leads to social discomfort or social anxiety.

Similarly, some patients report feelings of anger toward those who now treat them better since they have lost weight [14, 15]. Sogg and Gorman describe the widespread discrimination and prejudice against obese individuals at work, within their families and strangers, as well as medical professionals, including those who work with the obese have been found to hold such prejudice [13].

Many patients have difficulty coping with questions regarding how they lost the weight and whether or not they have had bariatric or cosmetic surgery. Others struggle to cope with constant questions about how much weight they have lost. Frequently noted is the issue of anger resulting from patients recognizing improvements in their treatment by friends and family as their weight continues to decline. Particularly troubling are patient reports that others may interpret their having surgery as "cheating" or "taking the easy way out." Obesity is one of the few diseases where the patient is blamed for their plight, resulting from the belief that the weight gain was strictly due to laziness or lack of willpower [107–109]. While much research refutes this notion [107, 110–112], patients themselves often hold these beliefs which compromises their own self esteem making it potentially more difficult to defend their decision to have surgery to others [33, 37, 104, 113].

Intimate relationships bring additional challenges. Very close friends of the patient may feel jealous of their weight loss and may make comments attempting to be supportive, but coming across as sarcastic or demeaning [13, 105]. Such intimate relationships may be compromised if the patient's former weight or eating behavior was a means of inclusion in the social group [105] as demonstrated in the third vignette detailed earlier. Although the bariatric patient is quite able to discuss the struggles of being overweight and controlling one's eating despite having weight loss surgery, the perception of their social group is the key factor in being able to maintain these relationships.

Romantic relationships bring additional challenges. Most patients report positive benefits of weight loss on their confidence and willingness to pursue sexual relationships, however, concern over body image remains high. This is true among those patients who have not dated in many years, however, it is even higher among those who have been obese for all of their adult lives and may have had little or no sexual experiences at all. These patients are aware that they are physically adults, but mentally they may feel all of the awkwardness and anxiety of an adolescent exploring their sexuality for the very first time.

There are also changes within marital relationships as highlighted in the vignettes above. Much research supports the notion that marital relationships that were strong before surgery are likely to stay that way, however, marriages that were not strong are more likely to become increasingly unstable [16, 36, 114, 115]. Improvements in the bariatric patient's self-esteem and self-worth may decrease the patient's willingness to tolerate perceived mistreatment [14]. Issues of jealousy or anxiety about the patient's desire to terminate the relationship may surface [14, 115, 116]. Changes in role functioning within such relationships may also occur [14, 16, 17, 116].

As previously discussed, special attention should be paid to many of these concerns in

patients who have previously experienced sexual abuse. Many patients discuss the anxiety of becoming more "visible" [14] and for those who have experienced sexual abuse this anxiety may be more significant. Patients with histories of sexual abuse may find such increased attention to be threatening [14, 36] and this needs to be addressed especially if the patient considers the extra weight to be a defense against unwanted attention [104, 117, 118]. This is particularly important given the potential for diminished weight loss and other complications noted in such studies among patients with such histories [62, 119].

24.9 The Importance of the Mental Health Professional on the Bariatric Team

The role of the mental health professional on the bariatric team is diverse and includes interventions before and after surgery [120]. The initial contact is typically made during the presurgical psychological evaluation. Most bariatric surgery programs require patients to undergo psychological evaluation prior to surgery, and this practice is generally recommended by bariatric accrediting organizations [13, 20, 33, 78, 120, 121].

The role of the evaluation is complex and is often misconstrued as a pass-fail examination to determine eligibility for surgery [120, 122]. While there are infrequent circumstances when the mental health professional recommends against surgery, it is more likely that severe psychological issues would result in postponement of surgery until these concerns have been adequately addressed. This author can recall several situations where a patient who was requested to address certain concerns prior to surgery later acknowledged that they were not initially ready for surgery now having the knowledge of how challenging the postoperative behavior changes were.

Although no definitive psychological markers have been discovered that predict a poor outcome from surgery, there is indication that the *number* of psychological risk factors may be predictive of poor outcome [10]. Additionally, as instruments are developed to measure additional surgical outcomes, specific psychological factors may yet demonstrate an impact on overall outcomes from surgery.

It is important to consider that the presurgical psychological evaluation may be a patient's first and only interaction with a mental health professional. It is reasonable to suggest that having a positive interaction with a mental health professional prior to surgery could lead a patient to be more inclined to consult a mental health professional after surgery if the need arose. This willingness to seek out mental health services could lead to improvements in overall outcomes from surgery.

The role of the mental health professional becomes increasingly diversified following surgery. Excess weight loss (EWL) remains the primary outcome measure used to define "success" from bariatric surgery. As this author has discussed elsewhere [57], success from the patient's point of view likely extends beyond weight loss. Patients do not have surgery solely to lose weight, but rather to be able to enjoy improvements in their health and changes in their functioning and quality of life that losing weight allows. It is certainly the experience of this psychologist and many others working in the field that patients are expecting improvements in their physical functioning, intimate and social relationships, self-esteem, and career functioning as a result of their weight loss. As discussed, this journey is not always a smooth one [13, 105].

As patients struggle to make desired changes in their functioning, it is possible for them to experience feelings of frustration and depression and possibly even question if the surgery and the resulting weight loss was worth it [57, 123]. A mental health professional with experience in addressing these issues can prove invaluable in helping these patients make the complete transition to "success," beyond simply losing a significant amount of weight [13, 20].

In addition to working with individual patient, a number of bariatric programs offer postoperative support groups for their patient population, suggesting that patients are interested in ongoing

support [124]. In one study with a nonsurgical group, the single most highly valued aspect of treatment was the provision of continuing care, followed by support group [125].

24.10 Treatment of Psychological Concerns Following Bariatric Surgery

Cognitive-behavior therapy (CBT) is a short-term, problem-focused therapy derived from the science and theory of learning and cognition. CBT approaches to treatment and evaluation is guided by principles of empirical science. The origins of CBT are in the science of learning theory [126]. CBT interventions have great empirical support and have demonstrated effectiveness in treating a variety of psychological disorders including eating disorders [127, 128]. CBT interventions have been effective in improving the eating behavior of postoperative bariatric patients [129, 130]. This is of great importance as previously discussed research that suggests that postoperative disordered eating or eating disorders can jeopardize outcomes from bariatric surgery.

While the great majority of studies on CBT in the bariatric population have focused on modifying postoperative eating behavior, additional research has addressed anxiety [131], depressive symptomology [130], and a combination of variables [132]. Given the previously discussed concerns regarding alcohol abuse and suicide following bariatric surgery, it would be advisable for bariatric programs to address these concerns with prospective surgery candidates and certainly to monitor them following surgery. While current research suggests that there are no definitive preoperative psychological diagnoses that impact outcome in terms of excess weight loss, future research should seek to identify patients who may be vulnerable to postoperative substance abuse and suicide and to investigate the possible benefits of CBT interventions in these individuals.

CBT could be helpful in improving outcomes following bariatric surgery in both the short and long-term. As discussed earlier, there are a considerable number of behavior changes that these patients must undergo for a successful outcome following surgery. Immediately following surgery, most patients are appropriately focused on following dietary recommendations and making marked changes in their eating behavior such as changing the manner in which they eat and the quantity of food consumed. Patients need to learn to eat slower, chew more thoroughly and avoid drinking and eating simultaneously. It is this author's experience that most patients incorporate the required changes in eating behavior such that they become the "new normal." As an example, over time, many patients will require less concentration and focus to chew more slowly as this behavior has been become more automated. Similarly, patients may no longer put a beverage on the table during meals having become accustomed to no longer drinking when eating.

Over the longer term, challenges change and become more varied. Maintaining compliance with recommended behavioral changes is critical. In their interviews of 100 gastric bypass patients 7 years following surgery, Cook and Edwards [133] identified six key habits common among patients who had maintained at least 74 % of their initial weight loss at long-term follow up. These included eating three balanced meals and two snacks daily, drinking water and avoiding carbonated beverages, sleeping an average of 7 h per night, exercising regularly, and taking personal responsibility for weight control. In another study, subjects who engaged in self-monitoring postoperatively were less likely to regain any weight after bariatric surgery [134]. Predictors of significant postoperative weight regain include indicators of baseline food urges, decreased well-being, and concerns over addictive behaviors. Others have identified similar habits that contribute to greater weight loss and maintenance [135].

Cognitive-behavioral interventions have been found to be effective for improving body image [136–139]. This approach utilizes a number of elements common to cognitive-behavioral interventions including education, exposure and desensitization, identifying and corrective cognitive errors, modifying self-defeating behaviors and relapse prevention. Cognitive behavioral

interventions have been found effective with obese individuals [140] as well as with average-weight women [138, 141, 142]. There is reason to believe that such interventions could be beneficial to bariatric patients following significant weight loss and further research in this area is needed.

As previously discussed, there may be substantial challenges in patients' social and intimate romantic relationships after surgery as well. Patients may benefit from individual or marital counseling to address changes and complications in relationships. There is also a growing body of research demonstrating the benefits of CBT delivered in a group format following bariatric surgery for a variety of concerns [124, 143].

24.11 Conclusion

Bariatric surgery is the most effective intervention for the treatment of morbid obesity. The benefits of bariatric surgery include improvements in a number of measures of health, physical functioning, quality of life and psychological improvements. However, a significant minority of patients fails to lose the expected amount of weight following surgery and many patients regain weight in the coming years. Psychological and behavioral factors have been found to contribute to these suboptimal outcomes. No specific presurgical psychological factors that contribute to poor outcomes has been consistently identified, however, there is evidence that the *number* of presurgical psychological risk factors may be important. Additionally, the role of a history of sexual and other abuse warrants further investigation. While specific presurgical psychological risk factors that predict poor outcomes may remain elusive, postsurgical psychological factors that contribute to poor outcomes are more clear. Postsurgical disordered eating patterns such as binge eating or grazing and depression have been shown to diminish weight loss following bariatric surgery. Additionally, patients often look beyond the amount of weight lost in assessing whether or not bariatric surgery has been successful [57]. As an example, while bariatric surgery may lead to improvements in body image, most patients express an interest in body contouring to further improve their appearance and satisfaction. Following bariatric surgery and dramatic weight loss, many patients hope to make changes in their social, intimate romantic and professional lives. There are often a number of challenges in this process, including some potential unexpected negative consequences of being thinner. Of concern, there are studies that demonstrate a relationship between bariatric surgery and substance abuse and reveal higher than expected rates of suicide. While the rates of postoperative substance abuse and suicide are quite small, these areas warrant further investigation. Cognitive behavior therapy has been shown to be a powerful and effective modality for addressing a number of these postoperative challenges including: disordered eating and eating disorders, depression and anxiety, body image, and changes in interpersonal and social relationships.

References

1. Sjostrom L, Narbro K, Sjostrom CD, Karason K, Larsson B, Wedel H, et al. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med. 2007;357:741–52.
2. Buchwald H, Avidor Y, Braunwald E, Jensen MD, Pories W, Fahrbach K, et al. Bariatric surgery: a systematic review and meta-analysis. JAMA. 2004;292:1724–37.
3. Maggard MA, Shugarman LR, Suttorp M, Maglione M, Sugerman HJ, Livingston EH, et al. Meta-analysis: surgical treatment of obesity. Ann Intern Med. 2005;142:547–59.
4. Bastis JA, Romero-Corral A, Collazo-Clavell ML, Sarr MG, Somers VK, Brekke L, et al. Effect of weight loss on predicted cardiovascular risk: change in cardiac risk after bariatric surgery. Obesity. 2007;15(3):772–83.
5. Kushner RF, Noble CA. Long-term outcome of bariatric surgery: an interim analysis. Mayo Clin Proc. 2006;81(10 Suppl):S46–51.
6. Sjostrom L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004;351(26):2683–93.
7. Sjostrom L, Gummesson A, Sjostrom CD, Narbro K, Peltonen M, Wedel H, et al. Effects of bariatric surgery on cancer incidence in obese patients in

Sweden (Swedish obese subjects study): a prospective, controlled intervention trial. Lancet Oncol. 2009;10(7):653–62.
8. Kolotkin RL, Crosby RD, Gress RE, Hunt SC, Adams TD. Two-year changes in health related quality of life in gastric bypass patients compared with severely obese controls. Surg Obes Relat Dis. 2009;5(2):250–6.
9. Sarwer DB, Wadden TA, Fabricatore AN. Psychosocial and behavioral aspects of bariatric surgery. Obes Res. 2005;13:639–48.
10. Kinzl JF, Schrattenecker M, Traweger C, Mattesich M, Fiala M, Biebl W. Psychological predictors of weight loss after bariatric surgery. Obes Surg. 2006;16(12):1609–14.
11. Rowe JL, Downey JE, Faust M. Psychological and demographic predictors of successful weight loss following silastic ring vertical staples gastroplasty. Psychol Rep. 2000;86:1028–36.
12. Herpertz S, Kielmann R, Wolf AM, Hebebrand J, Senf W. Do psychosocial variables predict weight loss or mental health after obesity surgery? A systematic review. Obes Res. 2004;12(10):1554–69.
13. Sogg S, Gorman MJ. Interpersonal changes and challenges after weight-loss surgery. Prim Psychiatry. 2008;15(8):61–6.
14. Bocchieri LE, Meana M, Fisher BL. Perceived psychosocial outcomes of gastric bypass surgery: a qualitative study. Obes Surg. 2002;12(6):781–8.
15. Bocchieri LE, Meana M, Fisher BL. A review of psychosocial outcomes of surgery for morbid obesity. J Psychosom Res. 2002;52(3):155–65.
16. Rand CS, Kowalske K, Kuldau JM. Characteristics of marital improvement following obesity surgery. Psychosomatics. 1984;25(3):221–23, 26.
17. Hafner RJ. Morbid obesity: effects on the marital system of weight loss after gastric restriction. Psychother Psychosom. 1991;56(3):162–6.
18. Rand CS, Kuldau JM, Robbins L. Surgery for obesity and marriage quality. JAMA. 1982;247(10):1419–22.
19. Ortega J, Fernandez-Canet R, Alvarez-Valdeita S, Cassinello N, Baguena-Puigcerver MJ. Predictors of psychological symptoms in morbidly obese patients after gastric bypass surgery. Surg Obes Relat Dis. 2012;8:770–6.
20. Grothe KB, Dubbert PM, O'Jile JR. Psychological assessment and management of the weight loss surgery patient. Am J Med Sci. 2006;331(4):201–6.
21. Sogg S, Mori DL. Revising the Boston Interview: incorporating new knowledge and experience. Surg Obes Relat Dis. 2008;4(3):455–63.
22. Spitzer RL, Devlin M, Walsh BT, Hasin D, Wing R, Marcus M, et al. Binge eating disorder: a multisite field trial of the diagnostic criteria. In J Eat Disord. 1992;11(3):191–203.
23. Yanovski SZ. Binge eating disorder: current knowledge and future directions. Obes Res. 1993;1:306–24.
24. Livhits M, Mercado C, Yermilov I, Parikh JA, Dutson E, Mehran A, et al. Preoperative predictors of weight loss following bariatric surgery: systematic review. Obes Surg. 2012;22(1):70–89.
25. Beck NN, Mehlsen M, Stoving RK. Psychological characteristics and associations with weight outcomes two years after gastric bypass surgery: postoperative eating disorder symptoms are associated with weight loss outcomes. Eat Behav. 2012;13(4):394–7.
26. Kalarchian MA, Marcus MD, Wilson GT, Labouvie EW, Brolin RE, LaMarca LB. Binge eating among gastric bypass patients at long-term follow-up. Obes Surg. 2002;12(2):270–5.
27. Burgmer R, Grigutsch K, Zipfel S, Wolf AM, de Zwaan M, Husemann B, et al. The influence of eating behavior and eating pathology on weight loss after gastric restriction operations. Obes Surg. 2005;15(5):684–91.
28. Hsu LKG, Betancourt S, Sullivan SP. Eating disturbances before and after vertical banded gastroplasty: a pilot study. Int J Eat Dis. 1996;19:23–34.
29. Hsu LKG, Betancourt S, Sullivan SP. Eating disturbances and outcome of gastric bypass surgery: a pilot study. Int J Eat Dis. 1997;21:385–90.
30. Marino JM, Ertelt TW, Lancaster K, Steffen K, Peterson L, de Zwaan M, et al. The emergence of eating pathology after bariatric surgery: a rare outcome with important clinical implications. Int J Eat Dis. 2012;45(2):179–84.
31. Kalarchian MA, Wilson GT, Brolin RE, Bradley L. Effects of bariatric surgery on binge eating and related psychopathology. Eat Weight Disord. 1999;4(1):1–5.
32. Saunders R. "Grazing": a high-risk behavior. Obes Surg. 2004;14:98–102.
33. Wadden TA, Sarwer DB, Fabricatore AN, Jones L, Stack R, Williams NS. Psychological and behavioral status of patients undergoing bariatric surgery: what to expect before and after surgery. Med Clin N Am. 2007;91(3):451–69.
34. Colles SL, Dixon JB, O'Brien PE. Grazing and loss of control related to eating: two high-risk factors following bariatric surgery. Obesity. 2008;16(3):615–22.
35. Colles SL, Dixon JB. Night eating syndrome: impact on bariatric surgery. Obes Surg. 2006;16:811–20.
36. Wadden TA, Sarwer DB, Womble LG, Foster GD, McGuckin BG, Schimmel A. Psychosocial aspects of obesity and obesity surgery. Sug Clin North Am. 2001;81(5):1001–24.
37. Wadden TA, Butryn ML, Sarwer DB, Fabricatore AN, Crerand CE, Lipschutz PE, et al. Comparison of psychosocial status in treatment-seeking women with class III vs. class I–II obesity. Obesity. 2006;14 Suppl 3:90S–8.
38. van Hout GC, Boekestein P, Fortuin FA, Pelle AJ, van Heck GL. Psychosocial functioning following bariatric surgery. Obes Surg. 2006;16(6):787–94.

39. Herpertz S, Kielmann R, Wolf AM, Langkafel M, Senf W, Hebebrand J. Does obesity surgery improve psychosocial functioning? A systematic review. Int J Obes Relat Metab Disord. 2003;27(11):1300–14.
40. Thonney B, Pataky Z, Badel S, Bobbioni-Harsch E, Golay A. The relationship between weight loss and psychosocial functioning among bariatric surgery patients. Am J Surg. 2010;199(2):183–8.
41. Karlsson J, Sjostrom L, Sullivan M. Swedish obese subjects (SOS)-an intervention study of obesity. Two-year follow-up of health-related quality of life (HQRL) and eating behavior after gastric surgery for severe obesity. Int J Obes Relat Metab Disord. 1998;22:113–26.
42. Clark MM, Balsiger BM, Sletten CD, Dahlman KL, Ames G, Williams DE, et al. Psychosocial factors and 2-year outcome following bariatric surgery for weight loss. Obes Surg. 2003;13:739–45.
43. Averbukh Y, Heshka S, El-Shoreya H, Flancbaum L, Geliebter A, Kamel S, et al. Depression score predicts weight loss following Roux-en-Y gastric bypass. Obes Surg. 2003;13:833–6.
44. Kalarchian MA, Marcus MD, Levine MD, Soulakova JN, Courcoulas AP, Megan SC, Wisinski BS. Relationship of psychiatric disorders to 6-month outcomes after gastric bypass. Surg Obes Relat Dis. 2008;4:544–9.
45. Legenbauer T, de Zwaan M, Benecke A, Muhlhans B, Petrak F, Herpertz S. Depression and anxiety: their predictive function for weight loss in obese individuals. Obes Facts. 2009;2:227–34.
46. Semanscin-Doerr DA, Windover A, Ashton K, Heinberg LJ. Mood disorders in laparoscopic sleeve gastrectomy patients: does it affect early weight loss? Surg Obes Relat Dis. 2010;6:191–6.
47. Legenbauer T, de Zwaan M, Petrak F, Herpertz S. Influence of depressive and eating disorders on short- and long-term course of weight after surgical and non-surgical weight loss treatment. Compr Psychiatry. 2011;53:301–11.
48. De Zwaan M, Enderle J, Wagner S, Muhlhans B, Ditzen B, Gefeller O, Mitchell JE, Muller A. Anxiety and depression in bariatric surgery patients: a prospective, follow-up study using structured clinical interviews. J Affect Disord. 2011;133:61–8.
49. Karlsson J, Taft C, Ryden A, Sjostrom L, Sullivan M. Ten-year trends in health-related quality of life after surgical and conventional treatment for severe obesity: the SOS intervention study. Int J Obes. 2007;31:1248–61.
50. Adams TD, Gress RE, Smith SC, Halverson RC, Simper SC, Rosamond WD, et al. Long-term mortality after gastric bypass surgery. N Engl J Med. 2007;357:753–61.
51. Goldfeder L, Ren C, Gill J. Fatal complications of bariatric surgery. Obes Surg. 2006;16:1050–6.
52. Omalu BI, Ives DG, Buhari AM, Lindner JL, Schauer PR, Wecht CH, et al. Death rates and causes of death after bariatric surgery for Pennsylvania residents, 1995 to 2004. Arch Surg. 2007;142(10):923–9.
53. Mitchell J, Lancaster K, Burgard M, et al. Long-term follow-up of patients' status after gastric bypass. Obes Surg. 2001;11:464–8.
54. Carpenter KM, Hasin DS, Allison DB, Faith MS. Relationships between obesity and DSM-IV major depressive disorder, suicide ideation, and suicide attempts: results from a general population study. Am J Public Health. 2000;90:251–7.
55. Omalu BI, Cho P, Shakir AM, Agumadu UH, Rozin L, Kuller LH, et al. Suicides following bariatric surgery for the treatment of obesity. Surg Obes Relat Dis. 2005;1:447–9.
56. Tindle HA, Omalu B, Courcoulas A, Marcus M, Hammers J, Kuller LH. Risk of suicide after long-term follow-up from bariatric surgery. Am J Med. 2010;123(11):1036–42.
57. Huberman WL. The importance of pursuing the patient's definition of success following weight loss surgery: strategies and considerations for the bariatric team. Bariatric Times. 2013;10(10):1, 14–21.
58. Hrabosky J, Masheb R, White M, Rothschild B, Burke-Martindale C, Grilo C. A prospective study of body dissatisfaction and concerns in extremely obese gastric bypass patients: 6- and 12-month postoperative outcomes. Obes Surg. 2006;16:1615–21.
59. Hagedorn JC, Encarnacion B, Brat GA, Morton JM. Does gastric bypass alter alcohol metabolism? Surg Obes Relat Dis. 2007;3:543–8.
60. Felitti VJ. Long-term medical consequences of incest, rape, and molestation. South Med J. 1991;84:328–31.
61. King TK. Sexual abuse and obesity: implications for the treatment of obesity. Med Health R I. 1997;80:364–6.
62. Ray EC, Nickels MW, Sayeed S, Sax HC. Predicting success after gastric bypass: the role of psychosocial and behavioral factors. Surgery. 2003;134(4):555–63.
63. Kral JG. Patient selection for treatment of obesity. Surg Obes Relat Dis. 2005;2:126–32.
64. Steinig J, Wagner B, Shang E, Dolemeyer R, Kersting A. Sexual abuse in bariatric surgery candidates—impact on weight loss after surgery: a systematic review. Obes Rev. 2012;13:892–901.
65. Bernstein DP, Fink L, Handelsman L, Foote J, Lovejoy M, Wenzel K, et al. Initial reliability and validity of a new retrospective measure of child abuse and neglect. Am J Psychiatry. 1994;151:1132–6.
66. Bernstein DP, Ahluvalia T, Pogge D, Handelsman L. Validity of the Childhood Trauma Questionnaire in an adolescent psychiatric population. J Am Acad Child Adolesc Psychiatry. 1997;36:340–8.
67. Grilo CM, Masheb RM, Brody M, Toth C, Burke-Martindale CH, Rothschild BS. Childhood maltreatment in extremely obese male and female bariatric surgery candidates. Obes Res. 2005;13:123–30.

68. Collazo-Clavell ML, Clark MM, McAlpine DE, Jensen MD. Assessment and preparation of patients for bariatric surgery. Mayo Clin Proc. 2006; 81(Suppl):S11–7.
69. Sogg S. Alcohol misuse after bariatric surgery: epiphenomenon or "Oprah" phenomenon? Surg Obes Relat Dis. 2007;3(3):366–8.
70. Klockhoff H, Naeslund I, Jones AW. Faster absorption of ethanol and higher peak concentration in women after gastric bypass surgery. Br J Clin Pharmacol. 2002;54:587–91.
71. Ertelt TW, Mitchell JE, Lancaster K, Crosby RD, Steffen KJ, Marino JM. Alcohol abuse and dependence before and after bariatric surgery: a review of the literature and report of a new data set. Surg Obes Rel Dis. 2008;4:647–50.
72. Frezza M, Buda A, Terpin MM, Benvenuti AS, Burra P, Casini A, et al. Gastrectomy, lack of gastric first pass metabolism of ethanol and alcohol liver disease. Results of a multicenter study. Ital J Gastroentereol Hepatol. 1997;29:243–8.
73. Maluenda F, Csendes A, De Aretxabala X, Poniachik J, Salvo K, Delgado I, et al. Alcohol absorption modification after laparoscopic sleeve gastrectomy due to obesity. Obes Surg. 2010;20:744–8.
74. Suzuki J, Haimovici F, Chang G. Alcohol use disorders after bariatric surgery. Obes Surg. 2012;22: 201–7.
75. Barry D, Clarke M, Petry NM. Obesity and its relationship to addictions: is overeating a form of addictive behavior. Am J Addict. 2009;18:439–51.
76. Del Parigi A, Chen K, Salbe AD, Reiman EM, Tataranni PA. Are we addicted to food? Obes Res. 2003;11:493–5.
77. Volkow ND, Wise RA. How can drug addiction help us understand obesity? Nat Neurosci. 2005;8: 555–60.
78. Wang GJ, Volkow ND, Logan J, Pappas NR, Wong CT, Zhu W, et al. Brain dopamine and obesity. Lancet. 2001;357:354–7.
79. King WC, Chen JY, Mitchell JE, Kalarchian MA, Steffen KJ, Engel SG, et al. Prevalence of alcohol use disorders before and after bariatric surgery. JAMA. 2012;307(23):2516–25.
80. Sarwer DB, Fabricatore AN, Jones-Corneille LR, Allison KC, Faulconbridge LN, Wadden TA. Psychological issues following bariatric surgery. Prim Psychiatry. 2008;15(8):50–5.
81. Giel KE, Thiel A, Teufel M, Mayer J, Zipfel S. Weight bias in work settings—a qualitative review. Obes Facts. 2010;3:33–40.
82. Stunkard AJ, Wadden TA. Psychological aspects of severe obesity. Am J Clin Nutr. 1992;55:524S–32.
83. Foster GD, Wadden TA, Vogt RA. Body image in obese women before, during, and after weight loss treatment. Health Psychol. 1997;16:226–9.
84. Schwartz MB, Brownell KD. Obesity and body image. Body Image. 2004;1:43–56.
85. Adami GF, Meneghelli A, Bressani A, Scopinaro N. Body image in obese patients before and after stable weight reduction following bariatric surgery. J Psychosom Res. 1999;46:275–81.
86. Sarwer DB, Wadden TA, Moore RH, Eisenberg MH, Raper SE, Williams NN. Changes in quality of life and body image after gastric bypass surgery. Surg Obes Relat Dis. 2010;6:608–14.
87. Madan AK, Beech BM, Tichansky DS. Body esteem improves after bariatric surgery. Surg Innov. 2008; 15:32–7.
88. Masheb RM, Grilo CM, Burke-Martindale CH, Rothschild BS. Evaluating oneself by shape and weight is not the same as being dissatisfied about shape and weight: a longitudinal examination in severely obese gastric bypass patients. Int J Eat Disord. 2006;39:716–20.
89. Sarwer DB, Thompson JK, Mitchell JE, Rubin JP. Psychological considerations of the bariatric surgery patient undergoing body contouring surgery. Plast Reconstr Surg. 2008;121(6):423e–34.
90. Sarwer DB. The psychological aspects of cosmetic breast augmentation. Plast Reconstr Surg. 2007; 120(7 Suppl 1):110S–7.
91. Sarwer DB, Brown GK, Evans DL. Cosmetic breast augmentation and suicide. Am J Psychiatry. 2007;164(7):1006–13.
92. Sarwer DB, Crerand CE. Body image and cosmetic medical treatments. Body Image. 2004;1:99–111.
93. Sarwer DB, Crerand CE. Body dysmorphic disorder and appearance enhancing medical treatments. Body Image. 2008;5(1):50–8.
94. Kinzl JF, Traweger C, Trefalt E, Biebl W. Psychosocial consequences of weight loss following gastric banding for morbid obesity. Obes Surg. 2003;13:105–10.
95. Zuelzer HB, Baugh NG. Bariatric and body-contouring surgery: a continuum of care for excess and lax skin. Plast Surg Nurs. 2007;27:3–13.
96. Kitzinger HB, Abayev S, Pittermann A, Karle B, Bohdjalian A, Langer FB, Prager G, Frey M. After massive weight loss: patients' expectations of body contouring surgery. Obes Surg. 2012;22(4):544–8.
97. Sarwer DB, Wadden TA, Foster GD. Assessment of body image dissatisfaction in obese women: specificity, severity, and clinical significance. J Consult Clin Psychol. 1998;66:651–4.
98. Teufel M, Rieber N, Meile T, Giel KE, Sauer H, Hunnemeyer K, Enck P, Zipfel S. Body image after sleeve gastrectomy: reduced dissatisfaction and increased dynamics. Obes Surg. 2012;22(8): 1232–7.
99. Rosenberger PH, Henderson KE, Grilo CM. Correlates of body image dissatisfaction in extremely obese female bariatric surgery candidates. Obes Surg. 2006;16:1331–6.
100. Song AY, Rubin JP, Thomas V, Dudas JR, Marra KG, Fernstrom MH. Body image and quality of life

in post massive weight loss body contouring patients. Obesity. 2006;14:1626–36.
101. Steffen KJ, Sarwer DB, Thompson JK, Mueller A, Baker AW, Mitchell JE. Predictors of satisfaction with excess skin and desire for body contouring after bariatric surgery. Surg Obes Relat Dis. 2012;8: 92–7.
102. Gusenoff JA, Messing S, O'Malley W, Langstein HN. Temporal and demographic factors influencing the desire for plastic surgery after gastric bypass surgery. Plast Reconstr Surg. 2008;121:2120–6.
103. Modarressi A, Balague N, Huber O, Chilcott M, Pittet-Cuenod B. Plastic surgery after gastric bypass improves long-term quality of life. Obes Surg. 2013;23(1):24–30.
104. Kalarchian MA, Marcus MD, Levine MD, Courcoulas AP, Pilkonis PA, Ringham RM, Soulakova JN, Weissfeld LA, Rofey DL. Psychiatric disorders among bariatric surgery candidates: relationship to obesity and functional health status. Am J Psychiatry. 2007;164(2):328–34.
105. Huberman WL. Through thick and thin: the emotional journey of weight loss surgery. Niskayuna: Graphite Press; 2012.
106. Ogden J, Clementi C, Aylwin S. The impact of obesity surgery and the paradox of control: a qualitative study. Psychol Health. 2006;21(2):273–93.
107. Andreyeva T, Puhl RM, Brownell KD. Changes in perceived weight discrimination among Americans, 1995–1996 through 2004–2006. Obesity. 2008;16(5):1129–34.
108. Crandall CS. Prejudice against fat people: ideology and self-interest. J Pers Soc Psychol. 1994;66(5): 882–94.
109. Friedman JM. Modern science versus the stigma of obesity. Nat Med. 2004;10(6):563–9.
110. Bouchard C. The biological predisposition to obesity: beyond the thrifty genotype scenario. Int J Obes (Lond). 2007;31(9):1337–9.
111. Farooqui IS, O'Rahilly S. Genetic factors in human obesity. Obes Rev. 2007;8 Suppl 1:37–40.
112. Wardle J, Carnell S, Haworth CM, Plomin R. Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. Am J Clin Nutr. 2008;87(2):398–404.
113. Chen E, Bocchieri-Ricciardi L, Munoz D, Fischer S, Katterman S, Roehrig M. Depressed mood in class III obesity predicted by weight-related stigma. Obes Surg. 2007;17(5):673–5.
114. Applegate KL, Friedman KE, Grant JP. Assessments of relationship satisfaction and stability one year after weight loss surgery: a prospective study [abstract]. Surg Obes Relat Dis. 2006;2(3):310.
115. Kinzl JF, Trefalt E, Fiala M, Hotter A, Biebl W, Aigner F. Partnership, sexuality, and sexual disorders in morbidly obese women: consequences of weight loss after gastric banding. Obes Surg. 2001;11(4):455–8.
116. Andrews G. Intimate saboteurs. Obes Surg. 1997; 7(5):445–8.
117. Felitti VJ. Childhood sexual abuse, depression, and family dysfunction in adult obese patients: a case control study. South Med J. 1993;86(7):732–6.
118. Wiederman MW, Sansone RA, Sansone LA. Obesity among sexually abused women: an adaptive function for some? Women Health. 1999;29(1):89–100.
119. Clark M, Hanna B, Jai J, Graszner KM, Krochta JG, McAlpine DE, et al. Sexual abuse survivors and psychiatric hospitalization after bariatric surgery. Obes Surg. 2007;17(4):465–9.
120. Huberman WL. One Psychologist's 7-year experience in working with surgical weight loss: the role of the mental health professional. Prim Psychiatry. 2008;15(8):42–7.
121. Bauchowitz AU, Gonder-Frederick LA, Olbrisch ME, Azarbad L, Ryee MY, Woodson M, et al. Psychosocial evaluation of bariatric surgery candidates: a survey of present practices. Psychosom Med. 2005;67(5):825–32.
122. Franks SF, Kaiser KA. Predictive factors in bariatric surgery outcomes: what is the role of the preoperative psychological evaluation? Prim Psychiatry. 2008;15(8):74–83.
123. Gallagher S. Taking the weight off with bariatric surgery. Nursing. 2004;34(3):58–63.
124. McVay MA, Friedman KE. The benefits of cognitive behavioral groups for bariatric surgery patients. Bariatric Times. 2012;9(9):22–8.
125. Latner JD, Stunkard AJ, Wilson GT, Jackson ML. The perceived effectiveness of continuing care and group support in the long-term self-help treatment of obesity. Obesity. 2006;14(93):464–71.
126. Craske MG. Cognitive-behavioral therapy. Washington DC: American Psychological Association; 2010.
127. Fairburn CG. Overcoming binge eating. New York: Guilford Press; 1995.
128. Fairburn CG, Marcus MD, Wilson GT. Cognitive-behavioral therapy for binge eating and bulimia nervosa: a comprehensive treatment manual. In: Fairburn CG, Wilson FT, editors. Binge eating: nature, assessment and treatment. New York: Guilford Press; 1993. p. 361–4040.
129. Ashton K, Drerup M, Windover A, Heinberg L. Brief, four-session group CBT reduces binge eating behaviors among bariatric surgery candidates. Surg Obes Relat Dis. 2009;5:257–62.
130. Leahey TM, Crowther JH. A cognitive-behavioral mindfulness group therapy intervention for the treatment of binge eating in bariatric surgery patients. Cog Beh Prac. 2008;15:364–75.
131. Berman D. A case of clinical anxiety following successful bariatric surgery: a cognitive-behavior therapy approach. Bariatric Times. 2013;10(8):10–3.
132. Cassin SE, Sockalingam S, Wnuk S, Strimas R, Royal S, Hawa R, Parikh SV. Cognitive behavioral therapy for bariatric surgery patients: preliminary evidence for feasibility, acceptability, and effectiveness. Cog Beh Prac. 2013;20:529–43.
133. Cook CM, Edwards CE. Success habits of long-term gastric bypass patients. Obes Surg. 1999;9:80–2.

134. Odom J, Zalesin KC, Washington TL, Miller WW, Hakmeh B, Zaremba DL, et al. Behavioral predictors of weight regain after bariatric surgery. Obes Surg. 2010;20:349–56.
135. Hernandez-Estefania R, Gonzalez-Lamuno D, Garcia-Ribes M, Garcia-Fuentes M, Cagigas JC, Ingelmo A, Escalante C. Variables affecting BMI evolution at 2 and 5 years after vertical banded gastroplasty. Obes Surg. 2000;10:160–6.
136. Cash TF. Body-image therapy: a program for self-directed change. New York: Guilford Press; 1991.
137. Cash TF. What do you see when you look in the mirror?: helping yourself to a positive body image. New York: Bantam Books; 1995.
138. Cash TF, Grant JR. The cognitive-behavioral treatment of body-image disturbances. In: Van Hasselt V, Hersen M, editors. Sourcebook of psychological treatment manuals for adults. New York: Plenum Press; 1995. p. 567–614.
139. Jarry JL, Ip K. The effectiveness of stand-alone cognitive-behavioral therapy for body image: a meta-analysis. Body Image. 2005;2(4):317–31.
140. Rosen JC, Orosan P, Reiter J. Cognitive behavioral body image therapy for negative body images in obese. Beh Ther. 1995;26:25–42.
141. Dworkin SH, Kerr BA. Comparison of interventions for women experiencing body image problems. J Clin Consult Psychol. 1987;34:136–40.
142. Butters JW. Cash TF Cognitive-behavioral treatment of women's body image dissatisfaction. J Consult Clin Psychol. 1987;55:889–97.
143. Saunders R. Post-group therapy for gastric bypass patients. Obes Surg. 2004;14:1128–31.

Medical Malpractice in the Twenty-First Century

25

Daniel Cottam

25.1 Introduction

Litigation involving medical malpractice is a worldwide problem that affects all branches of medicine. A quick search of PubMed will demonstrate papers being published in every first world country and many developing ones about the causes of malpractice claims and practical advice to avoid being involved in a lawsuit. The issues surrounding malpractice claims and bariatric surgery in particular are remarkable similar regardless of the surgeons country of origin. This chapter attempts to focus on causes of malpractice claims, avoidance of malpractice claims, appropriate responses to malpractice claims once the claim has been filed, and trends in malpractice claims in the USA over the last decade with a special focus on bariatric claims in that time span.

25.2 Causes of Malpractice Claims

Perhaps the most obvious cause of malpractice claims is the adverse event. As any bariatric surgeon knows not all adverse events can be avoided. Adverse events happen more frequently with complex surgical cases in sicker patients. Bariatric surgery for the most part is a very complex surgical case that usually is performed on sicker patients. With hundreds of thousands of these high risk surgical procedures being performed annually throughout the world there are literally thousands of adverse events that could generate litigation.

As these are known risks general surgeons increasingly have left the bariatric surgical population to specialist bariatric surgeons. In this high risk population bariatric surgeons are increasingly being asked to perform general surgical procedures such as gallbladder, colon, and hernia surgery on a patient population deemed too risky for the average general surgeon. This decreases the general surgeons risk but increases the potential risk for the bariatric surgeon as more and more of these high risk patients are concentrated in fewer practices. If the common rules regarding lawsuits and general surgeons are applied to bariatric practices, then there are approximately 1.6 lawsuits per 1000 cases. This means most bariatric surgeons will be subjected to some type of lawsuit every 3–4 years. These numbers may be startling to some, yet they are a decade old and no longer hold true. In fact, as the specialty of bariatric surgery has evolved and deaths and complications have decreased, malpractice rates have also fallen across the country.

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9_25

Currently there are a wide variety of safe procedures offered around the world. Yet as surgeons push the boundaries of what is safe they have accepted more difficult patients and offering a wide array of not only primary procedures but revisional bariatric procedures as well. Many of these "non traditional" bariatric procedures present great difficulties in defining the standard of care. The "standard of care" is typically defined as the care that a reasonable bariatric surgeon would provide under similar circumstances. In many instances where adverse events occur during these "nontraditional" procedures there may not be another similar surgeon performing this surgery for hundreds of miles. This means that the standard of care for "nontraditional" surgery might reach some consensus nationwide but could be lacking in a given state, region, or country.

To combat these issues ASMBS and IFSO have developed consensus statements to help surgeons ascertain what a surgical norm might be on a given question. These can indeed be very useful yet it is impossible for a consensus statement to establish the "standard of care" in a rapidly changing field filled with surgeons of vastly different training and experience. Guidelines should always be used in light of a surgeon's judgement and clinical experience.

Finding experts who understand the differences between state, national, or international definitions of standard of care can be difficult. It is even more difficult for juries and patients to understand. When confronted with these problems the surgeons involved in a lawsuit should make every effort to make sure their "expert" refrains from mentioning anecdotes from their own practice or present the standard of care of a teaching hospital the same as the standard of care of a community hospital. The ASMBS has tried to help lawyers (both plaintiff and defense) by publishing consensus guidelines for expert witnesses.

25.3 The Culture of Accountability

Perhaps no trend in bariatric surgery since 2004 has been more controversial than the various center of excellence programs set up not only in the USA but around the world. Entry into these centers of excellence programs varies by country, but each shares many common characteristics. The first of these characteristics is clinical decision support. This means that practice systems are created to avoid the errors that cause malpractice cases before they begin. A bariatric example of this would be each time a patient presents with nausea or vomiting the practice protocol dictates that thiamine is given to reduce the chance that Wernicke–Korsakoff syndrome happens.

Another feature of centers of excellence programs relate to the requirement that programs must be comprehensive in nature. This means that no longer does a program only do surgery, often as part of the staff there is dietary, psychologic, exercise, as well as support groups. This multispecialty approach to bariatric surgery helps to identify problems earlier. The comprehensive approach to medical disease processes reduces claims frequency and amounts across specialty lines.

25.4 Common Mistakes

A brief mention should be made of general practice patterns that will help bariatric surgeons avoid lawsuits and win those that are brought to trial. The first is documentation. All notes should be legible and timed and dated. Operative reports should be dictated on the day of surgery to prevent speculation by the plaintiffs' attorneys. Surgeons should be active participants in their patients' care and see their patients regardless of their resident's or other specialist's involvement. Office staff should be trained that all communication should be documented whenever possible.

Perhaps nothing is more important to the success of the surgeon in a lawsuit than the documentation of the patient's education process and a patient subsequent noncompliance. Most comprehensive centers have classes, tests, and one-on-one visits with numerous staff members to document the fact that the patients understand the risks of bariatric surgery and who to call if an emergency happens. This information alone will

often discourage plaintiff's attorneys from filing a lawsuit since they can see that it will be an uphill battle regardless of the adverse event.

The most important document in the education process is the informed consent. This is one area that bariatric surgeons excel in and it is very rare to find a lawsuit that hinges on informed consent. Typical templates for informed consent include risks and benefits of the specific procedure in question, a place to indicate all questions have been answered to the patient's satisfaction, a picture of the proposed procedure, and places to have the prospective patient initial on all important points. Special mention should be made to indicate that there are unknown risks that cannot be totally mitigated despite applying current standards of care.

Currently, all centers of excellence require a pathway for call coverage when the operating surgeon is away from their practice. Breakdown of this pathway is a common cause of malpractice. Too often surgeons leave town in the hands of untrained professionals which results in litigation when complications arise. This "drop the baton" occurs far too often and is entirely avoidable if surgeons communicate with covering physicians.

Another entirely avoidable mistake relates to delay in care. Too often surgeons forget what how important vital signs and labs really are. If there are abnormalities in these common measurements, they should always be addressed. Surgeons who ignore these facts do so at their own risk. Nothing is easier for a plaintiff's attorney to litigate than a delay in diagnosis because a lab or vital sign has been ignored.

25.5 Complication Management

In spite of surgeons efforts adverse events happen and patient will experience leaks, abscess, aspirations, thromboembolic events, bowel obstructions, ulcers, and death. Proper conduct of adverse events is of utmost importance. Communication with the patient or their family daily demonstrates compassion. It is important to remember that regardless of the surgeons comfort levels with ICU care no patient or family is comfortable with this care and the ICU is very intimidating. To prevent wild speculation on the part of the family that often generates lawsuits it is important to give patients and their families a pathway to reach the surgeon of record. Having a daily briefing or giving out one's cell phone number will allow the surgeon to form bonds with family members that will be valuable in the event that things do not turn out favorably. It is important to remember that in the event of the worst outcome—death—that it is the family members who sue.

25.6 Actions to Take Once You Are Named in a Lawsuit

Once being named in a lawsuit you should attempt to pick your lawyer wisely. The law does not move quickly and there are mountains of documents to process. The surgeon should actively participate in the process of collecting documents and make sure your attorney understands the issues at stake. The surgeons should take the time to help the defense counsel pick an appropriate expert and go to any deposition that you can. This tends to have moderating effect on plaintiff's witnesses. Take the time to teach your attorney what the best practices in medicine are, so they can ask appropriate question both in depositions and in trials. Then once they understand the case make sure they are representing your interests and not the insurance company. Too often lawyers, for convenience, recommend settling a case that is entirely winnable. This may have detrimental effects on the surgeon down the road. Settled claims can make it very difficult to move from state to state and even harder to find employment. Often the surgeon's malpractices rates will skyrocket for trivial amounts of money. Many surgeons have been dropped by malpractice carriers for a single judgment less than 20,000 dollars. At trial juries want to believe the surgeons are honest and

forthright. This results in jury verdicts in favor of the defense in excess of 80 %. If at in point during a case a surgeon believes his appointed attorneys are not meeting their needs, they should retain private counsel.

Most good attorneys will prepare surgeons for their deposition. However, if the attorney does not prepare you, then you should take the time to prepare yourself. At deposition or trial nobody should know the chart better than the surgeons being sued. Nobody should understand the literature better than that surgeon on that day. A corollary to that involves specific preparation for the plaintiff's attorney style. Preparation is key to effective depositions. If you act unsure of yourself in your deposition, this will come across to jurors as well. Many malpractice cases have been lost by surgeons who were too arrogant to take the time to review their own charts, and the literature related to an event.

When in a deposition or trial, always answer questions politely. Whenever a document is referenced, take the time to review it before you answer. Do not ever provide more information than is asked and never speculate or provide multiple theories for what happened. Often plaintiffs' attorneys will try and confuse you with multiple similar sounding questions. If you think you have already answered a question, simply say, "I have answered that question already." If they ask a question without an answer say, "I have no answer for that question please rephrase it." Do not ever offer to rephrase it for them. Many times plaintiff attorney's depositions or questions at a trial are nothing more than an attempt to find you at fault for adverse outcomes. Do not make their job easy for them.

25.7 Conclusion

The good news is that the tide is changing. No longer are malpractice premiums rising. We are doing a better job both at treating patients and avoiding lawsuits. In order to continue these trend surgeons must treat every adverse event as a chance of litigation and document accordingly. By doing so not only will our patients benefit from a higher standard of care but the surgeon will benefit by lowering their malpractice risk.

Suggested Reading

1. Mello MM, Studdert DM, Kachalia A. The medical liability climate and prospects for reform. JAMA. 2014;312(20):2146–55. doi:10.1001/jama.2014.10705.
2. Weber CE, Talbot LJ, Geller JM, Kuo MC, Wai PY, Kuo PC. Obesity and trends in malpractice claims for physicians and surgeons. Surgery. 2013;154(2):299–304. doi:10.1016/j.surg.2013.04.026. Epub 2013 Jun 15.
3. Weber CE, Talbot LJ, Geller JM, Kuo MC, Wai PY, Kuo PC. Comparing 20 years of national general surgery malpractice claims data: obesity versus morbid obesity. Am J Surg. 2013;205(3):293–7. doi:10.1016/j.amjsurg.2012.10.023. discussion 297. Epub 2013 Jan 17.
4. Jena AB, Seabury S, Lakdawalla D, Chandra A. Malpractice risk according to physician specialty. N Engl J Med. 2011;365(7):629–36. doi:10.1056/NEJMsa1012370.
5. Dallal RM, Pang J, Soriano I, Cottam D, Lord J, Cox S. Bariatric-related medical malpractice experience: survey results among ASMBS members. Surg Obes Relat Dis. 2014;10(1):121–4. doi:10.1016/j.soard.2013.04.015. Epub 2013 Jun 11.
6. Cottam D, Lord J, Dallal RM, Wolfe B, Higa K, McCauley K, Schauer P. Medicolegal analysis of 100 malpractice claims against bariatric surgeons. Surg Obes Relat Dis. 2007;3(1):60–6. discussion 66–7. Epub 2006 Dec 27.
7. Casey BE, Civello Jr KC, Martin LF, O'Leary JP. The medical malpractice risk associated with bariatric surgery. Obes Surg. 1999;9(5):420–5.

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D.M. Herron (ed.), *Bariatric Surgery Complications and Emergencies*,
DOI 10.1007/978-3-319-27114-9