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REVIEW

Guiding the non-bariatric surgeon through complications of bariatric surgery



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Gastric banding;
Fistula;
Hemorrhage

Summary Complications in bariatric surgery are varied; they are severe at times but infrequent. They may be surgical or non-surgical, and may occur early or late. The goal of this systematic review is to inform and help the attending physician, the emergency physician and the non-bariatric surgeon who may be called upon to manage surgical complications that arise after adjustable gastric band (AGB), sleeve gastrectomy (SG), or gastric bypass (BPG). Data from evidence-based medicine were extracted from the literature by a review of the Medline database and also of the most recent recommendations of the learned societies implicated. The main complications were classified for each intervention, and a distinction was made between early and late complications. Early complications after AGB include prosthetic slippage or perforation; SG can be complicated early by staple line leak or fistula, and BPG by fistula, stenosis and postoperative hemorrhage. Delayed complications of AGB include intragastric migration of the prosthesis, late prosthetic slippage and infection, while SG can be complicated by gastro-esophageal reflux, and BPG by anastomotic ulcer and internal hernia. The analysis of available data allowed us to develop decisional algorithms for the management of each of these complications.

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Introduction

The prevalence of obesity is steadily increasing throughout the world [1,2], having doubled in the last 30 years; it is no longer uniquely confined to rich countries. Today, 500 million

adults are obese and France, where 15% of adults are obese, has not escaped this pandemic [3].

Since 2005, the number of bariatric interventions carried out in France has multiplied by 3.4; more than 42,000 interventions were performed in 2013. Bariatric surgical activity is booming and its widespread application requires good quality management of surgical complications.

The HAS (High Authority for Health) has published recommendations, which were updated in 2009. Bariatric surgery involves two main types of intervention:

- strictly restrictive procedures include: adjustable gastric banding (AGB) (Fig. 1) and sleeve gastrectomy (SG) (Fig. 2);

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Technique of adjustable gastric banding

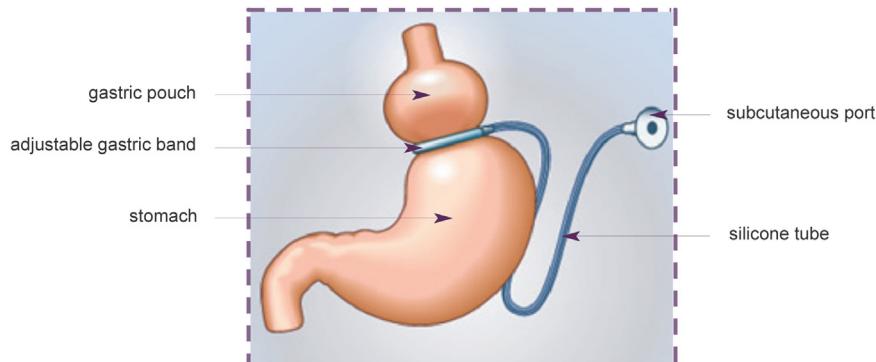


Figure 1. Schema of the adjustable gastric band (AGB).

From HAS-Obesity: surgical management in the adult, 2009. http://www.has-sante.fr/portail/upload/docs/application/pdf/2009-09/fiche_technique_anneau_gastrique_080909.pdf.

Technique of sleeve gastrectomy

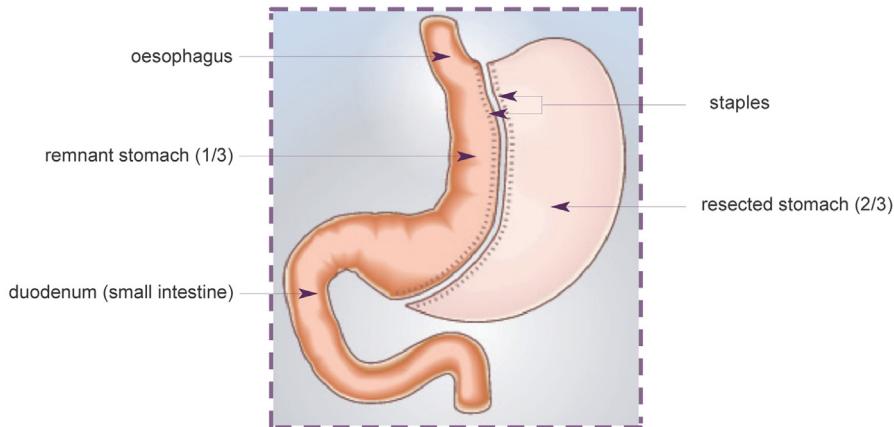


Figure 2. Schema of sleeve gastrectomy (SG).

From HAS-Obesity: surgical management in the adult, 2009. http://www.has-sante.fr/portail/upload/docs/application/pdf/2009-09/fiche_technique_gastrectomie_080909.pdf.

- mixed restrictive and malabsorptive procedures: the main variants are gastric bypass (GBP) [4] (Fig. 3) and bilio-pancreatic diversion (BPD). Complications of BPD have not been detailed in this systematic review in view of the low number of BPD performed in France in comparison with the other procedures.

Today, no single technique can objectively prevail over the others.

The AGB is the least aggressive intervention, and offers satisfactory weight loss that is dependent on a careful nutritional follow-up. This is a minimally invasive procedure and may be the first of several surgical procedures [5].

SG, whose use has expanded significantly in recent years, offers results that are intermediate between AGB and GBP; perioperative mortality rate is equally intermediate between AGB and GBP.

GBP offers the best initial weight loss and long-term weight stability [6,7]. The most commonly performed technique is the Roux-en-Y GBP. There is indeed a variant with a single anastomosis, called GBP en Omega or mini-GBP. This procedure has not, to date, been validated by the HAS nor recognized in the nomenclature [8].

Bariatric surgery has long been marginalized, and only recently has dedicated training of this discipline and its particularities been introduced. Therefore, many surgeons are

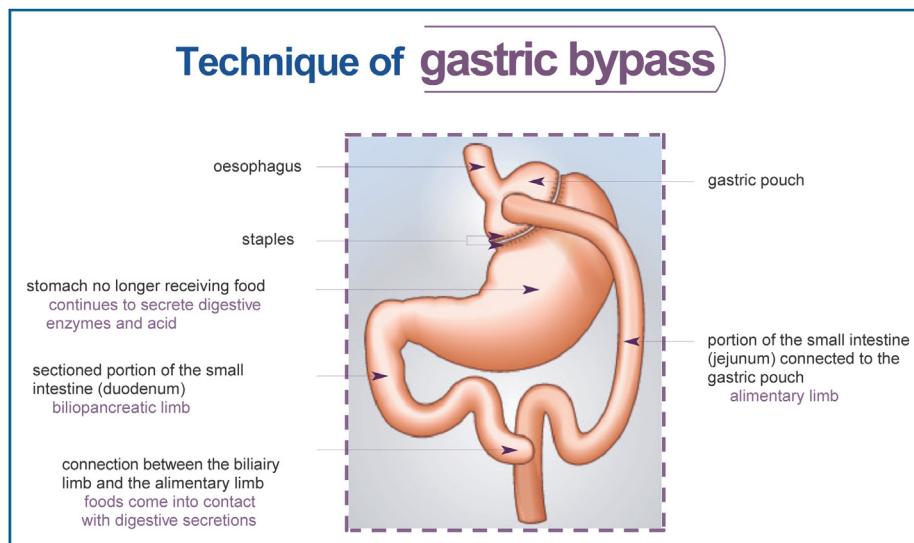


Figure 3. Schema of Roux-en-Y Gastric BYPASS (GBP).

From HAS-Obesity: surgical management in the adult, 2009. http://www.has-sante.fr/portail/upload/docs/application/pdf/2009-09/fiche_technique_bypass_080909.pdf

not well trained in the principles of surgical management of obese patients [9].

Non-surgical postoperative complications

Thrombo-embolic complications

The incidence of pulmonary embolism (PE) after bariatric surgery ranges from 0 to 0.6%; with the use of protective anticoagulation, mortality ranges from 0 to 0.4% [10–13]. Discrepancies of results between various authors may be explained by several phenomena:

- some procedures are performed by laparotomy, others by laparoscopy;
- reports often combine several different procedures (AGB, SG and GBP);
- finally, PE is often an epiphénomène related to other surgical complications.

The identified risk factors for PE are:

- age: there is a linear correlation between age and risk;
- BMI: there is also a linear correlation between BMI and risk;
- a prior history of thrombo-embolic disease;
- the occurrence of surgical complications.

No studies provide definitive recommendations on the optimal duration of postoperative thrombo-embolic prophylaxis. The French Society of Anesthesia and Reanimation (SFAR) suggests two daily injections of low-molecular-weight Heparin AF: the author should confirm, we can suggest, or at least point out the problem, but I do not think it is not up to us, alone, to intervene in his/her place (without exceeding 10,000 IU/d) for a ten-day period. Prophylactic efforts should be multimodal and early ambulation should be the rule. The combined use of intermittent pneumatic calf compression (IPCC) has also been proposed, but without evidence-based confirmation. Similarly, the wearing of elastic support hose for one month postoperatively has been proposed.

The incidence of postoperative thrombo-embolic complications is multiplied by a factor of 2 to 3 in obese

patients. The risk is further increased when the surgical approach is by laparotomy compared to the laparoscopic approach [10,11]. The development of multimodal approaches to the prevention of thrombo-embolic disease has reduced the prevalence of pulmonary embolism to less than 0.7% [10,11,14].

Respiratory complications

If pulmonary embolism is excluded from respiratory complications, the incidence of pulmonary complications (pneumonopathy, pleural effusion, etc.) ranges from 0.6% to 1.1% [15]. Laparoscopy probably plays a favorable role in reducing respiratory complications and, in 2013, their incidence after bariatric surgery was 1.6%, consisting mainly of atelectasis of the pulmonary bases, frequently associated with pneumonopathies [16]. Thus, whenever a CT is performed postoperatively in search of a complication, the radiologist should always scrutinize the pulmonary bases.

Nutritional complications

The possibility of vitamin deficiencies is largely related to the malabsorptive procedures and is potentiated by the induced loss of appetite. Deficiencies in vitamin D and B-12 are the most likely, which justifies systematic postoperative supplementation [17–20]. Vitamin D deficiency (due to decreased absorption) exposes the patient to the risk of early osteoporosis from secondary hyperparathyroidism. Finally, while GBP does not include a gastric resection, the reconfiguration of the alimentary and biliary circuit modifies the pH rendering gastric intrinsic factor inactive and impairing iron absorption, resulting in vitamin B-12 and iron deficiency [21].

Chronic vomiting after bariatric surgery, regardless of the cause, increases the risk of vitamin B-12 deficiency. Intravenous perfusion of dextrose and saline without vitamin supplementation may therefore lead to Gayet-Wernicke syndrome [22,23]. Finally, there is a risk of dumping syndrome (5–10%) and late hypoglycemia (<1%), especially after GBP but also after SG [24].

Postoperative surgical complications

After AGB

Safety is the principal advantage of AGB since its mortality is currently almost nil: less than 0.01% or actually zero in some series [7]. However, AGB has a higher morbidity with more early and late complications.

It is difficult and illogical to speak of global morbidity since early complications appear to be essentially related to lack of experience and the surgical learning curve; their prevalence decreases rapidly over the years of a surgeon's experience. However, late complications continue to increase over time [5,25,26].

Early

Esophageal and/or gastric perforation

The peri-gastric positioning of the gastric ring determines the upstream gastric volume, which is calibrated by use of a probe that includes a 15 cm³ balloon at its lower end. This probe is introduced by the anesthesiologist and advanced into view of the surgeon, then inflated and withdrawn until it is pulled snug against the cardia. However, its use remains optional.

Esophageal perforation may occur intraoperatively during the advancement of the calibration probe, but its diagnosis is often not made until the early postoperative period. Clinical degradation of the patient or evidence of leakage by upper GI series (UGIS) is confirmed by a CT scan with oral contrast. If perforation is diagnosed intraoperatively, the AGB should not be placed [1,27]. More typically, gastric perforation occurs intraoperatively during the creation of the retro-gastric tunnel. If perforation is diagnosed intraoperatively, the AGB should not be placed. Perforation may not be diagnosed until the postoperative UGIS is performed. In the meta-analysis conducted by the French National Agency for Health Evaluation (ANAES) in 2001, the incidence of gastric leak was 0.3% (15 cases out of 5237). In one series that compared the "pars flaccida" technique to the peri-gastric technique, the former indeed lowered the incidence of this complication to 0.3% [1]. In all cases of perforation or leak, re-operation is necessary.

Acute dilatation

Acute dilatation of the gastric pouch is a rare but extremely severe complication that may lead to gastric necrosis due to distension and diastatic ischemia. Its incidence ranges between 0.3% and 2.6% [28,29]. Dilatation occurs mainly when the AGB is positioned too low (in the lesser sac), is not anchored posteriorly resulting in early slippage and tipping with gastric prolapse leading to acute gastric distension. It is thus a posterior slippage. The UGIS objectively confirms this complication showing the AGB in a vertical position if the pouch prolapses posteriorly and in a horizontal position if the pouch prolapses anteriorly [27], with a large gastric air pocket and no passage of the contrast medium. Re-operation is obligatory with removal of the AGB, although some surgeons consider the possibility of repositioning the AGB [29].

Late complications

Late complications occur frequently after AGB. All complications described below may lead to the need for re-operation. The overall rate of complications related to the access port and connector tubing is about 5% [29].

Infection

Chronic purulent drainage should lead to suspicion of infected AGB, particularly due to gastric mural erosion with intragastric migration; this requires removal of the AGB. The infection rate of AGB at one year was 0.6% in the National Health Insurance Fund (CNAM) survey published in 2007 [30].

The incidence of access port infection is estimated at 1%. Typically, the access port is contaminated during transcutaneous puncture to modulate the tightening of the band. The infected access port leads to local inflammation with pus exiting along a trocar tract. The infected access port must then be removed along with any infected connector tubing. The resected access port and connector tubing are sent separately for bacterial culture. The remaining tubing is left in the peritoneal cavity. Local care of the access port site is then continued until healing. Once healing has been achieved, the tubing can be reconnected to a new access port via a laparoscopic approach.

Connector tubing rupture

The tubing connects the access port to the balloon of the AGB; the access port is implanted subcutaneously while the tubing leads into the abdominal cavity. This tract must ideally be created without kinking in order to avoid any risk of rupture or disconnection. The incidence of rupture or disconnection is about 1%. The main clinical sign is the patient's sensation that the AGB is no longer present or working with an arrest of excess weight loss (EWL). Opacification by injection of the access port with contrast shows a leakage of contrast medium at the point of tubing rupture. This complication has become increasingly rare since the connections have been placed at a distance from the passage through the abdominal muscle layers and since the creation of an oblique subcutaneous tubing trajectory has become the rule [29,31].

Pocket dilatation and late AGB slippage

Dilatation of the pouch with slippage of the AGB is the most frequent late complication, with 2% prevalence; it typically consists of delayed progressive dilatation of the gastric pouch due to late sliding of the AGB. The restrictive function of the AGB remains effective as long as the gastric pouch remains small. Excessive mobility of the AGB combined with large food boluses leads to progressive dilatation of the small gastric pouch. As the sensation of satiety is delayed, the quantity of ingested food increases. This vicious circle leads to progressive distension and the gastric wall slips through the AGB. This is, in essence, a trans-prosthetic gastric herniation [27]. Clinical signs are an arrest of EWL associated with a major worsening of comfort during eating ranging from heartburn to total alimentary intolerance. An UGIS confirms the diagnosis: the AGB appears to lie horizontal with an anterior trans-prosthetic gastric herniation [32].

This complication develops gradually, unlike early band slippage with posterior rotation. But in the absence of early diagnosis, the dilatation of the gastric pouch worsens progressively just as for anterior slippage and trans-prosthetic hernia. The situation can also develop into an acute complication of strangulated hernia through the AGB with a risk of gastric necrosis.

Technical modifications have significantly reduced the frequency of this complication:

- when the AGB was positioned through a posterior approach in the lesser sac, the prevalence of slippage, and thus of pouch dilatation, reached 20% of the cases [33,34];

- with peri-gastric positioning, there was slippage of the AGB in 10% of cases;
- when the AGB is positioned according to the currently recommended “pars flaccida” technique, the rate of slippage is reduced to less than 2%, as was found in a prospective randomized trial [35].

The heterogeneity of procedures and length of follow-up explain the differences between various rates reported in the literature.

Whenever there is an arrest of EWL, with or without heartburn and early satiety, the diagnosis of AGB slippage with pouch dilatation must be evoked. Only the UGIS allows confirmation of the slippage. Once the diagnosis is made, the gastric band must be loosened. Endoscopy should not be performed through a tight AGB. If the contrast medium does not cross the AGB narrowing, it is urgent to loosen it.

Erosion and intragastric migration

The prevalence of gastric wall erosion varies from 0.3% to 28% in various studies. In studies with high rates, erosion can be explained by technical errors but also by excessive tightening of the band [1,36,37]. The true incidence of this complication appears to be more in the range of 0.5 to 1% [29,38,39]. In the National Health Insurance Fund study, the one-year intragastric migration rate was 1.3% [30]. The causes of intragastric migration are mostly obscure. We must distinguish early-onset erosion (very rare) from late erosion (more frequent). Some patients are asymptomatic, but for the rest, the symptomatology is varied (epigastric pain, tachycardia, fever, digestive hemorrhage with hematemesis, dysphagia), with an arrest of EWL in most cases. While UGIS and CT scan affirm the diagnosis, the examination of choice is fiberoptic upper endoscopy. The logical treatment is removal of the AGB and most teams have more or less uniform approaches depending on the degree of AGB migration [5,29,40]. If more than 50% of the circumference of the AGB has migrated transmurally, endoscopic removal seems the best approach with over 80% success. In the event of failure, combined endoscopy-laparoscopy or laparoscopy alone should be performed for extraction of the AGB through an anterior gastrotomy. If less than 50% of the circumference of the AGB has migrated, symptomatology becomes an essential factor in the therapeutic decision. For asymptomatic patients, surveillance is necessary with inflation of the AGB and upper endoscopy every 2 to 3 months until the degree of

migration exceeds 50% and allows an endoscopic extraction. For symptomatic patients, semi-urgent extraction must be done by laparoscopy.

Esophageal dilation

If an AGB is overtightened, the gastric pouch dilates and accumulated upstream gastric content then progressively dilates the esophagus. But if loosening of the AGB restores normal anatomy, this is not a true “esophageal dilatation”. True dilatation is progressive and not simply a consequence of downstream obstruction. Indeed, esophageal dilatation is not always accompanied by dilatation of the proximal gastric pouch. This complication therefore poses the question of whether there is decompensation of pre-existing esophageal motor disorders after placement of AGB. The AGB may behave like a second lower esophageal sphincter (LES), with resting hypertonia and non-relaxation leading to symptomatology similar to that of achalasia involving the LES [41].

Dargent et al. [42] have proposed a classification of esophageal dilatation into four stages:

- stage 1: moderate dilatation due to excessive or prolonged AGB tightening (about 25% of all patients);
- stage 2: tonic dilatation with a certain degree of achalasia but with preserved esophageal motility (15%);
- stage 3: esophageal dilatation with AGB slippage and resultant dilation of the upper gastric pouch (0.5%);
- stage 4: atonic dilatation due to complete and irreversible achalasia.

The prevalence of esophageal dilatation is difficult to assess due to variability in the definition of this complication. However, it is estimated that 5% of cases of esophageal dilation require removal of the AGB [29]. When esophageal dilation is observed on UGIS, the AGB should be loosened for a period of three months and then re-evaluated. If the pouch returns to normal, a slight band tightening can be resumed, but persistence of the dilatation should lead to removal of the AGB [27].

Complications after SG

SG was initially described as the first operative stage of bilio-pancreatic diversion. Since the early 2000s, it has come into its own as a full-fledged bariatric intervention, and has been recommended since 2009. In 2013, 24,000 procedures

Table 1 Surgical complications after SG [6] (Figs. 4 and 5).

Early complications			
Leak/fistula	2.3%		
Stricture	0.7 to 4%		
Hemorrhage	1.5%		
Late complications			
Gastro-esophageal reflux	Up to 25% [81,82]		
		Fistula is the principal complication after SG; management is difficult and prolonged. Typically arises at the upper edge of the staple line. It has been widely reported in the literature According to its location, treatment can be endoscopic or surgical Bleeding is most commonly intraperitoneal rather than intra-luminal. Management is primarily surgical rather than endoscopic	
		Only severe pre-existing esophagitis and Barrett's esophagus are contra-indications to the performance of SG-S Postoperative gastro-esophageal reflux is often controlable with PPI medication	



Figure 4. CT: CT-scan showing a postoperative fistula after sleeve gastrectomy. Red arrow: leak of oral contrast agent.

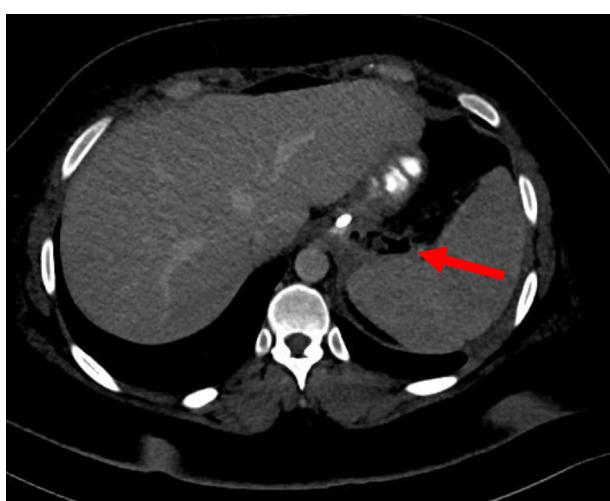


Figure 5. CT: CT-scan of a postoperative fistula after sleeve gastrectomy. Red arrow: pneumoperitoneum.

made SG the leading bariatric procedure in France, exceeding both GBP and AGB. The complications for this procedure are listed in Table 1 (Figs. 4 and 5) [43]. The main complication is leak or fistula from the top of the staple line with a prevalence of about 2%. With regard to the mechanism of complications after SG, some cases can be explained by the persistence of the pylorus (which creates intragastric hyperpressure) and others by the technique of SG. Hyperpressure probably plays a role in the genesis of fistulae and also failures due to dilatation of the neo-gastric tube in case of stenosis. Partial antrectomy could contribute to this gastric hyperpressure by altering gastric motility [44]. Finally, ischemia plays a role in fistulas that develop on the staple line [45].

Complications after Roux-en-Y GBP

Early complications

Digestive fistula

The main early complication of GBP is leak or fistula with an overall prevalence of 2–3.6%; in more recent series, the prevalence is close to 2% (Tables 2 and 3) [6,46].

Each suture line of the GBP is logically at risk of leak: the gastric pouch, the excluded stomach, the gastro-jejunostomy, the jejuno-jejunostomy and staple closures of small intestinal division sites. The most frequent site of leak

is the gastro-jejunostomy, which accounts for more than half of the fistulae.

Most authors recommend performing an UGIS during the first postoperative days. This examination makes it possible to detect a fistula at the gastro-jejunostomy but not at the jejun-jejunostomy at the foot of the biliary loop, situated 1.5 m distal to the proximal anastomosis. However, UGIS has the added advantage of simultaneously determining the absence of stenosis at one or the other anastomosis despite its lack of sensitivity and specificity for fistula screening.

The treatment of fistulas after GBP depends on their localization and their size, which determine their clinical repercussions. Fistulae at the jejun-jejunostomy at the foot of the loop (anastomosis between the alimentary limb and the biliary limb) are generally manifested by peritonitis with violent abdominal pain and signs of sepsis that require urgent surgical re-intervention [47–49].

This is also the case with most fistulae that occur at the site of distal gastric division, resulting from distension of the excluded stomach due to a downstream obstruction (see anastomotic stenosis). It should be noted that, in these patients, clinical examination through the obese abdominal wall is sometimes of little help in detecting postoperative peritonitis. Tachycardia is often the only clinical sign of a possible surgical complication. In fact, a tachycardia greater than 120/min requires an urgent exploratory laparoscopy in order to search for a postoperative complication [50]. In leaks from either site, the peritoneal contamination is rapid and massive because of the large amount of enteric flow at the level of the leak, or backed up in the stomach. Urgent surgery is always required for these fistulae with peritoneal toilet and reconstruction of the anastomosis, despite the context of peritonitis. Indeed, the thickness of the obese abdominal wall imposes certain pragmatism. The dogma of performing stomas in the setting of postoperative peritonitis from anastomotic fistula is barely compatible with safe management of the stomas in this context and can lead to the death of the patient or impose multiple re-interventions [49]. Each succeeding operation poses a challenge because, in the meantime, the patient loses little or no weight while rapidly becoming malnourished, and the parietal sequelae can rapidly exceed all therapeutic resources and, in turn, become life threatening [49]. For fistula of the distal stomach, a gastrostomy tube should be placed to decompress the excluded stomach and protect the repair; it can then possibly be useful at a later stage as access for enteric nutrition [48,51].

Fistulae originating from the gastric pouch suture line or the gastro-jejunostomy may be clinically evident at the outset, but are often insidious and sometimes even asymptomatic. If the patient is hemodynamically stable, conservative treatment is generally appropriate, while patients with systemic signs or hemodynamic or respiratory instability (tachycardia, tachypnea, oliguria, fever, pain) should undergo surgery. The aims of surgical re-intervention are precise identification of the site of leakage, lavage of the abdominal cavity, and effective drainage. The creation of a feeding jejunostomy should be considered in this context of postoperative peritonitis in a massively obese patient. Fistulas at the gastro-jejunostomy, if they are diagnosed early, are readily amenable to primary closure. In the case of late re-operation, it may be impossible to close the leak due to the poor quality of the surrounding tissues; their conversion to directed fistula is preferable [48].

For asymptomatic gastro-jejunal fistulas, treatment is conservative, keeping the patient on an empty stomach

Table 2 Early surgical complications after GBP.

Early complications		
Leak/fistula	1.9% [6]	The principal complication after GBP. The most common site of leakage is at the gastro-jejunostomy (Fig. 1) Reported prevalence varies from 1% to more than 20% Symptomatology and management differ according to the site of obstruction, just as for fistula/leak (see below)
Early postoperative obstruction/anastomotic stricture	< 1% to > 20% [7,48]	Most bleeding occurs in the immediate postoperative period and, in most cases, is due to intra-luminal or intraperitoneal bleeding from a staple line
Hemorrhage	1.7% [54,57]	

Table 3 Late surgical complications after GBP.

Late complications		
Anastomotic ulcer	1–7% [55,58,59]	Half of these arise in the first three months, and only 15% beyond two years
Bowel obstruction	1–6% [56,65,66,83]	In more than 60% of cases, obstruction is due to an internal hernia

**Figure 6.** Stricture of the gastro-jejunostomy (blue arrow).

with good drainage and appropriate nutritional support. For enteric nutrition in this situation, the insertion of a naso-jejunal tube should be preferred [52]. Antibiotics are not systematically recommended [49]. A peri-anastomotic collection must be subjected to good drainage and, if necessary, percutaneous drainage.

Early postoperative obstruction due to anastomotic stricture

Obstruction can occur at either the gastro-jejunostomy (Fig. 6) or the jejunoo-jejunostomy. The prevalence of strictures varies in the literature from less than 1% to more than 20%. This difference in rates can be explained by the heterogeneity of the series: laparotomy/laparoscopy, early/late stricture, failure to distinguish between gastro-jejunal and jejunoo-jejunostomy anastomotic stricture [51,53–55].

Symptoms and management differ according to location, just as for fistula.

**Figure 7.** Dilatation of the excluded stomach (blue arrow), a result of stricture at the jejunoo-jejunostomy.

Early obstruction due to stricture of the jejunoo-jejunostomy is extremely serious. Obstruction due to faulty anastomotic construction occurs early (day 1 or 2); it may be asymptomatic initially, but evolves very quickly. The key to treatment of this stricture is early diagnosis. The stricture (Fig. 7) causes obstruction, but after GBP, the obstruction is not betrayed by vomiting. Instead, the upstream jejunum and duodenum of the biliary limb dilate. But the biliopancreatic limb is a blind loop ending in the excluded distal stomach; it is shorter than the alimentary limb (70 cm versus 150 cm). It therefore distends much more rapidly and, if surgical re-intervention is not promptly performed, leads to distension of the excluded stomach, perforation, and then a generalized peritonitis with a very grim prognosis. Some authors, including Msika et al., have proposed systematic performance of an abdominal plain X-ray on day 1 in order to detect gaseous distension of the stomach, which would indicate stenosis at the foot of the loop. Surgical re-intervention

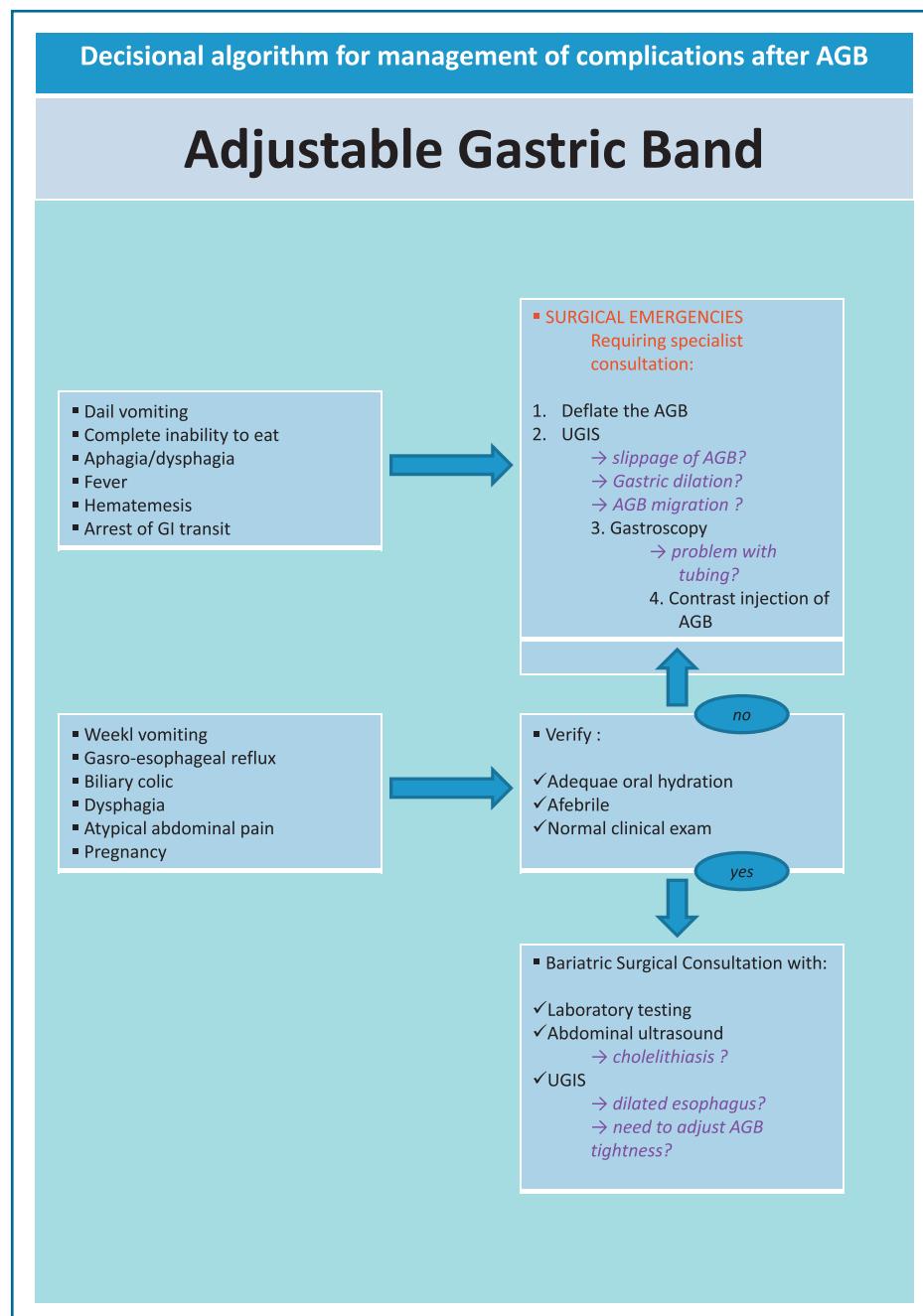


Figure 8. Decisinal algorithm for management of complications after AGB.

is always indicated [48], with reconstruction of the jejunoojejunostomy at the foot of the loop and placement of a decompressive tube gastrostomy.

Symptoms due to early stenosis of the gastro-jejunostomy appear later than after stenosis at the foot of the loop. They become evident in the first weeks after surgery and gastro-jejunal stricture must be considered when the patient presents with epigastric pain associated with progressive dysphagia and vomiting. Some predisposing mechanisms that should be suspected include the realization of a circular stapled anastomosis, an overlarge gastric pouch, and a pre-colic trajectory of the alimentary limb [51,56]. In 90% of cases, stenoses of the gastro-jejunostomy are treated endoscopically by pneumatic dilatation. The number of dilatation sessions is variable; the resort to surgery remains exceptional [51].

Hemorrhage

Most bleeding occurs in the immediate postoperative period and is either intra-luminal or intraperitoneal [54].

In the experience compiled by the American insurance company BLIS, the prevalence of hemorrhage after GBP was 1.7% [57]. Intraperitoneal hemorrhage is almost always the result of bleeding from a staple line. The diagnosis is obvious when blood issues from an abdominal drain, but if there is any doubt, exploratory laparoscopy is essential. The treatment is surgical with peritoneal toilet and realization of hemostasis.

Intra-luminal hemorrhages have several possible origins. For bleeding from the gastro-jejunostomy or the gastric pouch, the primary symptom is hematemesis, although associated melena is also possible. If bleeding occurs in the excluded stomach or at the jejunoojejunostomy, melena

is the primary symptom. The treatment of hemorrhage at the gastro-jejunostomy is upper endoscopy to identify the bleeding site, to evacuate clots and to perform a hemostatic gesture. For bleeding at the foot of the loop, intensive surveillance and possible transfusions are sufficient in most cases [51,53].

Anastomotic ulcer

Anastomotic ulcer occurs only at the gastro-jejunostomy. Its prevalence is between 1 and 7% [55–58]. Half of them occur within the first three months and only 15% after two years [59]. They are mainly observed in non-compliant patients who smoke and who still require proton pump inhibitor medications (PPI).

The pathophysiology is not yet clearly established. The two main predisposing factors seem to be the acidity of the gastric pouch (the acid pocket effect described in hiatal hernias), and alteration of the gastric mucosa after this intervention.

Other hypotheses have also been mentioned:

- absence of acid buffering by pancreatico-biliary secretions. This hypothesis seems false since similar rates of marginal ulcer have been reported after bilio-pancreatic derivation of the mini-GBP type, in which this buffering exists [60];
- use of non-absorbable suture material. The only evidence supporting this hypothesis in the literature is the study by Sacks et al. in 2006 [61]. However, in their study, the postoperative use of PPI was not specified. Other studies do not show any significant difference between the use of non-absorbable suture versus absorbable suture;
- tension on the anastomosis caused by the ante-colic trajectory of the alimentary limb, compared to the more-direct retro-colic trajectory. Only Lublin et al. in 2006 provided evidence to support this importance of this parameter [62]. In other older series, the postoperative prescription of PPI was not specified.

Symptoms of anastomotic ulcer are typically epigastric pain associated with dietary discomfort (hunger pangs), nausea/vomiting, and worsening of dietary tolerance. In some cases, the ulcer can be complicated by upper GI bleeding, a peptic stricture or even perforation.

The reference examination is upper endoscopy, which confirms the diagnosis of ulcer, allows biopsies in search of *Helicobacter pylori* infection, and finally allows monitoring of ulcer healing after treatment.

The treatment is essentially medical with the prescription of double dose PPI for three months. Peptic stricture is treated by PPI and iterative dilatations or, occasionally, by resection and reconstruction of the anastomosis if medical treatments fail.

Two particularities are worthy of note:

- an anastomotic ulcer can perforate into the excluded stomach. This may provoke another clinical sign: the regaining of weight. If PPI therapy fails to cure anastomotic ulcer, the treatment is surgical with resection and reconstruction of the anastomosis and resection of the fistulous tract extending into the excluded stomach [63];
- the development of an ulcer in the excluded stomach is extremely rare: 0.25% [27].

Delayed complications

Anastomotic ulcer

The modalities of management are similar to those for early anastomotic ulcer. Its occurrence may be quite delayed,

with up to 15% of ulcers occurring two years after surgery [59].

Anastomotic stricture

Late stricture involves only the gastro-jejunostomy. Two possible etiologies are:

- peptic stricture (cf. anastomotic ulcer);
- healing of a fistula with resultant stricture (see digestive fistula).

Obstruction

The prevalence of late obstruction ranges from 1 to 6% and is due to internal hernia in 60% of cases [64]. The late appearance of this complication is made possible by the patient's weight loss and, in consequence, the development of large potential spaces or passages between the small intestinal mesenteries, within which the intestine can incarcerate (Fig. 3). The principal site of internal herniation is the mesenteric window at the level of the jeuno-jejunostomy at the foot of the loop. Incarceration here causes pain that is often localized in the left flank. Herniation can also occur through Petersen's orifice (the space between the transverse mesocolon and an ante-colic alimentary limb). Finally, there can be trans-mesocolic herniation of the alimentary loop if there is a breach in the transverse mesocolon, which, like the others, enlarges with the patient's progressive weight loss.

Diagnosis of obstruction is often very difficult since most patients who underwent GBP do not vomit! Moreover, the obstruction is almost always intermittent, with clinical symptoms consisting only of repetitive bouts of pain in the same abdominal distribution without obvious cause [65]. Only a CT scan with oral contrast, performed during a bout of pain allows delineation of this internal hernia along with non-specific signs, such as distended small bowel loops or signs of mesenteric vascular engorgement (venous stasis) or sometimes an aspect suggesting a common mesentery with most or all of the small intestine lateralized to one side (usually to the left). However, false negatives are frequent and a normal CT should not contraindicate exploratory laparoscopy to confirm the diagnosis and close the mesenteric defects [66–70]. Many authors therefore recommend the closure of mesenteric defects during GBP, but this is not sufficient to completely prevent this complication since the problem tends to appear after significant weight loss, months and years after the intervention [64].

Cholecystolithiasis

The risk of a biliary complication after bariatric surgery increases from the third month to the second year, with a peak incidence between 6 and 18 months, corresponding to the period of maximal EWL. Indeed, while lithogenesis is multifactorial, obesity and the rapidity of weight loss are the main factors implicated in its development [71,72]. Elevated biliary cholesterol concentration and decreased gallbladder motility due to decrease in circulating cholecystokinin are both active in the lithogenic process [71,73].

The series by Coupaye et al. [72] investigated the occurrence of cholecystolithiasis six months after the completion of a GBP: the postoperative prevalence of vesicular stones was 30% at one year, with microlithiasis in all cases, mostly multiple, while half of the patients were asymptomatic.

No predisposing preoperative factors were found. Therefore, they recommended routine prevention of stone formation with ursodeoxycholic acid (UDCA) for a maximum of six months in all patients who underwent GBP without cholecystectomy [4].

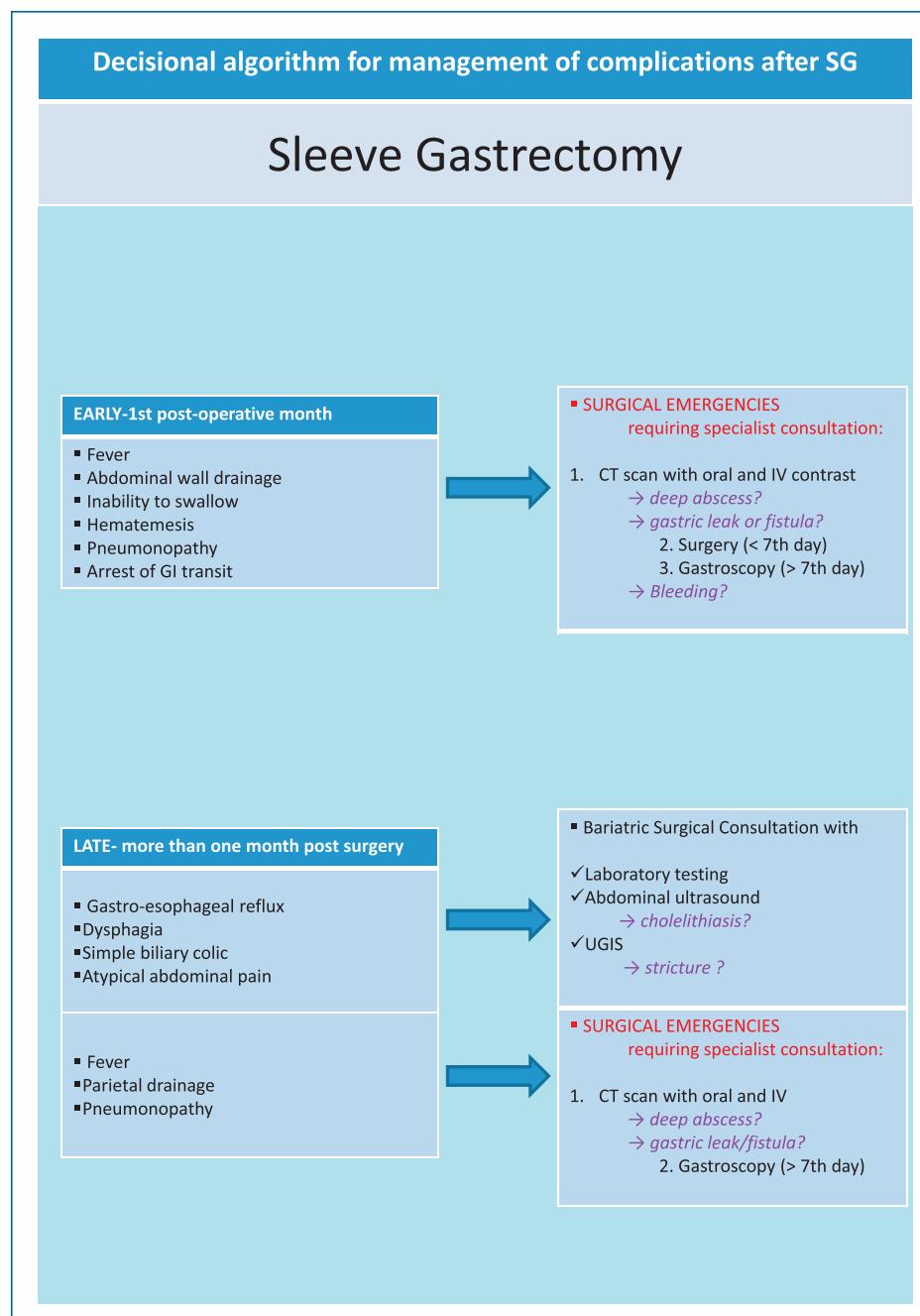


Figure 9. Decisional algorithm for management of complications after SG.

After GBP, the biliary tract is no longer accessible for endoscopic retrograde cholangiopancreatography (ERCP) by fiberoptic gastroscopy, which complicates management in patients who develop common duct stones. Most authors propose prophylactic cholecystectomy be performed subsequent to GBP in cases of known cholelithiasis (the HAS recommends abdominal ultrasound as part of the pre-operative assessment prior to GBP), but no formal guidelines have been made [74]. If cholelithiasis is not detected by preoperative ultrasound, the attitude is controversial. Some authors suggest prophylactic UDCA therapy treatment after GBP, while others advocate routine cholecystectomy at the time of GBP [75–77].

If gallstones migrate into the common bile duct, treatment is surgical with three possible options [51,76,77]:

- all laparoscopic: cholecystectomy and common duct exploration with stone extraction in one stage;
- the creation of a surgical gastrostomy to allow endoscopic management by ERCP sphincterotomy and stone extraction;
- temporary restoration of normal anatomy to allow access to the papilla for endoscopic sphincterotomy and stone extraction.

Particularities of the GBP en omega

The creation of a single gastro-jejunostomy two meters distal to the ligament of Treitz without a Roux-en-Y limb is a technically simple gesture, but associated with a mortality of 0.2% and morbidity of 5.5% [78].

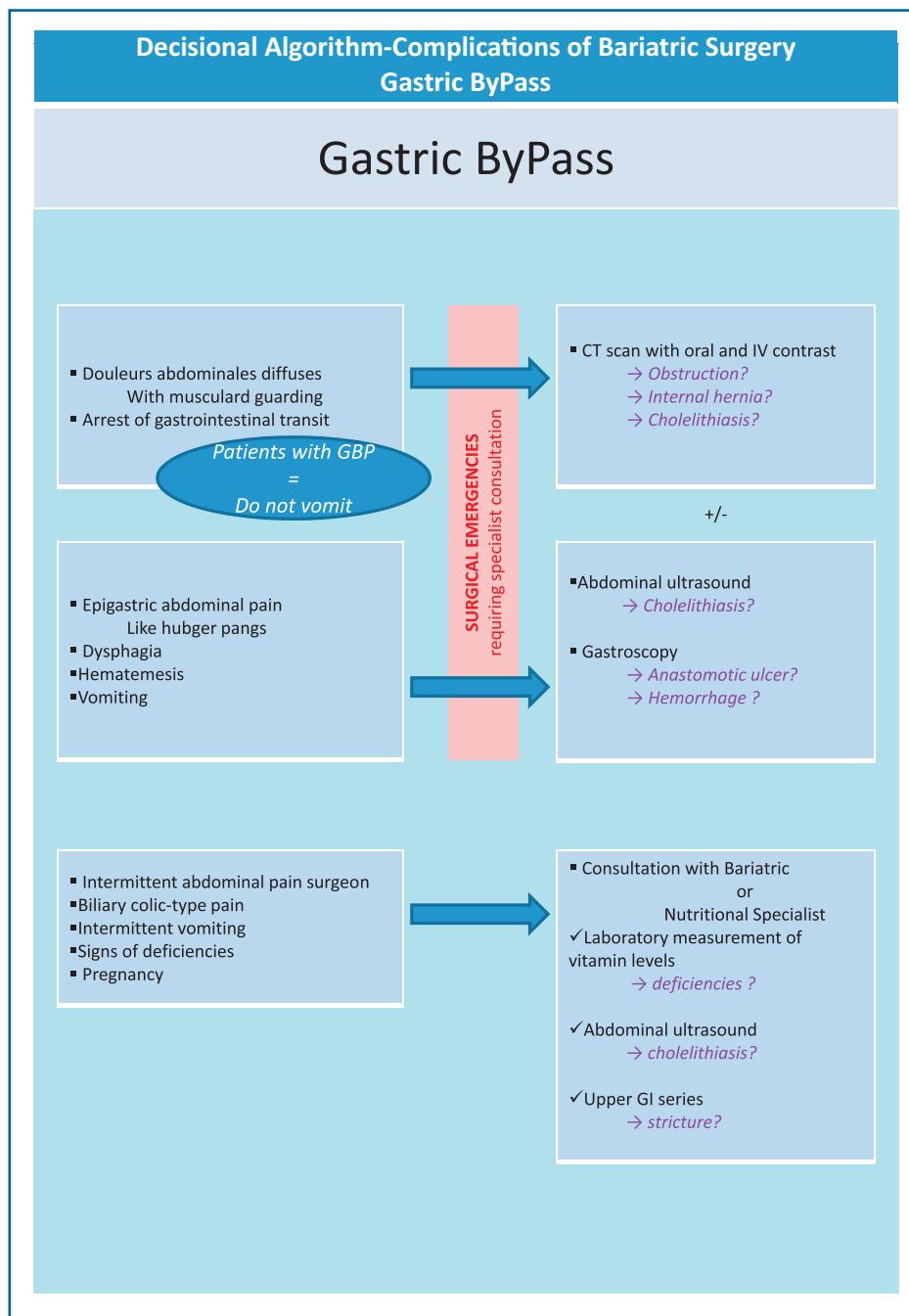


Figure 10. Decisional algorithm for management after gastric bypass.

The main early complications of GBP en omega are anastomotic fistulas and bleeding similar to those occurring after Roux-en-Y GBP. The particularity of fistula management is the need for re-operation, which can sometimes be avoided in Roux-en-Y GBP [79].

The main late complications are: anastomotic ulcer, biliary reflux, and malnutrition. The incidence of anastomotic ulcer is comparable to that observed in Roux-en-Y GBP. Biliary reflux is a specific complication of the omega loop construction that may require conversion to Roux-en-Y GBP. [8].

Summary of complications and decisional algorithm

Most complications after AGB are benign. Currently, the perioperative mortality associated with AGB placement is

almost zero. Severe complications include gastric perforation and gastric necrosis caused by acute distension: they have become extremely rare as techniques have evolved and operator experience has increased. The overall early morbidity is low, estimated at about 5%. AGB is a minimally invasive procedure, and can be succeeded by other surgical procedures if necessary. However, late complications are frequent and varied, coupled with the fact that it has only moderate bariatric efficacy; these factors make it a limited but rather interesting approach for the young adult or even the adolescent [80].

The use of SG has expanded significantly because it allows rapid major weight loss, is easier to perform than LGB, and seems to lead to fewer long-term vitamin deficiencies. The bariatric results of SG are intermediate between AGB and GBP and have an intermediate complication risk. However, SG's seeming ease of performance should not mask

its formidable early complication of gastric fistula at the top of the staple line. The fistula is of multifactorial origin although always arising in the same location (or is it a question of faulty surgical technique?); its management is long and difficult, often requiring multiple hospitalizations, multiple surgical resections and/or endoscopic procedures. GERD, whether arising *de novo* or an exacerbation of previous symptoms, is the most common delayed complication after SG, occurring in almost a quarter of cases.

GBP is the most thoroughly understood of modern bariatric interventions. Its complications are now well known and described. Nutritional and vitamin deficiencies arise inherently after GBP or any total gastrectomy or Roux-en-Y surgical montage. However, in the setting of bariatric surgery with GBP, nutritional deficiencies appear as an expected albeit undesirable effect of different procedures. The prevention of these deficiencies is an integral part of obesity management, with preoperative screening and correction of deficiencies, while most bariatric services recommend postoperative lifelong supplementation after GBP and bilio-pancreatic diversion. These rare deficiencies are mainly vitamin and mineral deficiencies. Protein deficiencies, after AGB, SG or GBP are exceptional [22]. But many patients opt for GBP despite the well-known risks of deficiency, because it has the best results for significant and durable EWL; its proven track record over more than 20 years outweighs the constraints related to lifelong vitamin therapy and the need for ongoing follow-up.

The following algorithms are designed to assist the attending physician, emergency physician and non-bariatric surgeon in managing any surgical complications after AGB, SG or GBP (Figs. 8–10). The specialist consultation corresponds to the opinion of a surgeon trained in bariatric surgery or belonging to a Specialized Center of Obesity Care (SCOC).

Conclusion

Bariatric surgery is legitimate with major, durable and reproducible results. The complications of each of these procedures are varied, potentially severe but fortunately rare. There is a correlation between the "aggressiveness" or technical difficulty of an intervention (postoperative morbidity and mortality) and its "effectiveness" (loss of excess weight). The mortality associated with AGB is essentially zero, but at the price of limited effectiveness and late morbidity that may require re-operations. GBP, the oldest intervention, offers the longest follow-up with the best compromise between morbidity, mortality, efficiency and risk. Complications, although rare, can be severe at either the early or late postoperative stage. SG is presented as the intermediate procedure for both weight loss and the risk of complications (predominantly fistula), the treatment of which belongs to a SCOC.

Disclosure of interest

The authors declare that they have no competing interest.

Key points

- Adjustable gastric band (AGB):
 - minimal morbidity and mortality;
 - limited efficacy (mean excess weight loss: 50% at 5 years),
 - high rate of late re-intervention,
 - late complications: intragastric band migration, esophageal dilation, tipping of the AGB prosthesis;
- Sleeve gastrectomy (SG):
 - technically easy to perform,
 - intermediate level of risk with intermediate weight loss results: mortality rate of 0.2% et EWL of 60% at 5 years,
 - fistula is rare (0.5–2%) with an interval to diagnosis of up to several months. Management of fistula is prolonged and requires a multidisciplinary approach (endoscopic, nutritional, surgical) most appropriate in a specialized center;
- Gastric bypass (GBP):
 - oldest intervention with the longest follow-up,
 - best compromise between weight loss results (mean EWL of 65% at 5 years) and nutritional deficiencies (long-term follow-up i.e. essential to detect the development of these deficiencies),
 - mortality rate of 0.5%.
 - Early surgical intervention is mandatory in case of tachycardia (> 120/min); a negative laparoscopic exploration does nothing to impair the postoperative results.
 - Any patient who presents with chronic vomiting is at risk to develop vitamin B-1 deficiency (whatever the bariatric procedure) and these patients must not receive dextrose infusions without vitamin supplementation in order to avoid Wernicke's encephalopathy.

References

- [1] Di Lorenzo N, et al. Laparoscopic adjustable gastric banding via pars flaccida versus perigastric positioning: technique, complications, and results in 2549 patients. *Surg Endosc* 2010;24:1519–23.
- [2] Ng M, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 2014;384:766–81.
- [3] HAS. Étude épidémiologique nationale sur le surpoids et l'obésité. (Étude « ObEpi-Roche »); 2012.
- [4] HAS. Obésité: prise en charge chirurgicale chez l'adulte; Recommandations de bonnes pratiques; 2009 www.has-sante.fr.
- [5] 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;257:87–94.
- [6] Zellmer JD, Mathias MA, Kallies KJ, Kothari SN. Is laparoscopic sleeve gastrectomy a lower risk bariatric procedure compared with laparoscopic Roux-en-Y gastric bypass? A meta-analysis. *Am J Surg* 2014;208:903–10 [discussion 909–910].
- [7] Chang SH, et al. The effectiveness and risks of bariatric surgery: an updated systematic review and meta-analysis, 2003–2012. *JAMA Surg* 2014;149:275–87.
- [8] Georgiadou D, et al. Efficacy and safety of laparoscopic mini gastric bypass. A systematic review. *Surg Obes Relat Dis* 2014;10:984–91.

- [9] Drouin P, Pointel JP, Gay G, Sauvanet JP, Debry G. [Is surgical treatment of major and recurrent obesity justified?]. Rev Prat 1976;26:2723–31.
- [10] Biertho L, et al. Perioperative complications in a consecutive series of 1000 duodenal switches. Surg Obes Relat Dis 2013;9:63–8.
- [11] Finks JF, et al. Predicting risk for venous thromboembolism with bariatric surgery: results from the Michigan Bariatric Surgery Collaborative. Ann Surg 2012;255:1100–4.
- [12] Kakarla VR, Nandipati K, Lalla M, Castro A, Merola S. Are laparoscopic bariatric procedures safe in superobese ($BMI \geq 50 \text{ kg/m}^2$) patients? An NSQIP data analysis. Surg Obes Relat Dis 2011;7:452–8.
- [13] Magee CJ, Barry J, Javed S, Macadam R, Kerrigan D. Extended thromboprophylaxis reduces incidence of postoperative venous thromboembolism in laparoscopic bariatric surgery. Surg Obes Relat Dis 2010;6:322–5.
- [14] Caruana JA, et al. Roux en Y gastric bypass by single-incision mini-laparotomy: outcomes in 3300 consecutive patients. Obes Surg 2011;21:820–4.
- [15] Nguyen NT, et al. Comparison of pulmonary function and post-operative pain after laparoscopic versus open gastric bypass: a randomized trial. J Am Coll Surg 2001;192:469–76 [discussion 476–467].
- [16] van Huisstede A, et al. Pulmonary function testing and complications of laparoscopic bariatric surgery. Obes Surg 2013.
- [17] Ledoux S, et al. Long-term evolution of nutritional deficiencies after gastric bypass: an assessment according to compliance to medical care. Ann Surg 2014;259:1104–10.
- [18] Coupaye M, et al. Comparison of nutritional status during the first year after sleeve gastrectomy and Roux-en-Y gastric bypass. Obes Surg 2014;24:276–83.
- [19] Verger EO, et al. Micronutrient and protein deficiencies after gastric bypass and sleeve gastrectomy: a 1-year follow-up. Obes Surg 2016;26:785–96.
- [20] Aron-Wisnewsky J, et al. Nutritional and protein deficiencies in the short term following both gastric bypass and gastric banding. PLoS One 2016;11:e0149588.
- [21] Caiazzo R, Torres F, Pattou F. La chirurgie bariatrique en 2015. Hepatogastroenterology 2015;569–81. <http://dx.doi.org/10.1684/hpg.2015.1181>.
- [22] Coupaye M, et al. Nutritional consequences of adjustable gastric banding and gastric bypass: a 1-year prospective study. Obes Surg 2009;19:56–65.
- [23] Tabbara M, Carandina S, Bossi M, et al. Obes Surg 2016;26:2843. <http://dx.doi.org/10.1007/s11695-016-2227-8>.
- [24] Berg P, McCallum R. Dumping syndrome: a review of the current concepts of pathophysiology, diagnosis, and treatment. Dig Dis Sci 2016;61:11–8.
- [25] Himpens J, et al. Long-term outcomes of laparoscopic adjustable gastric banding. Arch Surg 2011;146:802–7.
- [26] Polat F, Poyck PP, Dickhoff C, Gouma DJ, Hesp WL. Outcome of 232 morbidly obese patients treated with laparoscopic adjustable gastric banding between 1995 and 2003. Dig Surg 2010;27:397–402.
- [27] Chevallier JM, Pattou F. Chirurgie de l'obésité. Rapport présenté au 106^e congrès français de chirurgie. Paris: Association française de chirurgie; 2004.
- [28] Dargent J. Pouch dilatation and slippage after adjustable gastric banding: is it still an issue? Obes Surg 2003;13:111–5.
- [29] Chevallier JM, et al. Complications after laparoscopic adjustable gastric banding for morbid obesity: experience with 1000 patients over 7 years. Obes Surg 2004;14:407–14.
- [30] Chevallier JM, et al. Predictive factors of outcome after gastric banding: a nationwide survey on the role of center activity and patients' behavior. Ann Surg 2007;246:1034–9.
- [31] Suter M, Giusti V, Worreth M, Heraief E, Calmes JM. Laparoscopic gastric banding: a prospective, randomized study comparing the Lapband and the SAGB: early results. Ann Surg 2005;241:55–62.
- [32] ANAES. Chirurgie de l'obésité morbide; 2000.
- [33] Belachew M, Zimmermann JM. Evolution of a paradigm for laparoscopic adjustable gastric banding. Am J Surg 2002;184:21S–5S.
- [34] Favretti F, Ashton D, Busetto L, Segato G, De Luca M. The gastric band: first-choice procedure for obesity surgery. World J Surg 2009;33:2039–48.
- [35] 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;15:820–6.
- [36] Westling A, Bjurling K, Ohrvall M, Gustavsson S. Silicone-adjustable gastric banding: disappointing results. Obes Surg 1998;8:467–74.
- [37] Niville E, Dams A, Vlasselaers J. Lap-Band erosion: incidence and treatment. Obes Surg 2001;11:744–7.
- [38] Weiner R, et al. Outcome after laparoscopic adjustable gastric banding – 8 years experience. Obes Surg 2003;13:427–34.
- [39] Belachew M, Belva PH, Desaive C. Long-term results of laparoscopic adjustable gastric banding for the treatment of morbid obesity. Obes Surg 2002;12:564–8.
- [40] Owers C, Ackroyd R. A study examining the complications associated with gastric banding. Obes Surg 2013;23:56–9.
- [41] Greenstein RJ, Nissan A, Jaffin B. Esophageal anatomy and function in laparoscopic gastric restrictive bariatric surgery: implications for patient selection. Obes Surg 1998;8:199–206.
- [42] Dargent J. Esophageal dilatation after laparoscopic adjustable gastric banding: definition and strategy. Obes Surg 2005;15:843–8.
- [43] <http://stats.ath.sante.fr/mco/catalogmco.php>.
- [44] Rosenthal RJ, et al. International sleeve gastrectomy expert panel consensus statement: best practice guidelines based on experience of > 12,000 cases. Surg Obes Relat Dis 2012;8:8–19.
- [45] Perez M, et al. Does anatomy explain the origin of a leak after sleeve gastrectomy? Obes Surg 2014;24:1717–23.
- [46] Flum DR, et al. Perioperative safety in the longitudinal assessment of bariatric surgery. N Engl J Med 2009;361:445–54.
- [47] Gautier T, Sarcher T, Contival N, Le Roux Y, Alves A. Indications and mid-term results of conversion from sleeve gastrectomy to Roux-en-Y gastric bypass. Obes Surg 2013;23:212–5.
- [48] Ballesta C, Berindoague R, Cabrera M, Palau M, Gonzales M. Management of anastomotic leaks after laparoscopic Roux-en-Y gastric bypass. Obes Surg 2008;18:623–30.
- [49] Heyd B, Balique JG, Dehni N. Péritonites postopératoires. Rapport présenté au 112^e congrès français de chirurgie. Paris: Association française de chirurgie; 2010.
- [50] Kassir R, et al. Complications of bariatric surgery: presentation and emergency management. Int J Surg 2016;27:77–81.
- [51] Msika S. [Surgical treatment of morbid obesity by gastrojejunostomy bypass using laparoscopic roux-en-Y (gastric short circuit)]. J Chir (Paris) 2002;139:214–7.
- [52] Elke G, et al. Enteral versus parenteral nutrition in critically ill patients: an updated systematic review and meta-analysis of randomized controlled trials. Crit Care 2016;20:117.
- [53] Suter M, Paroz A, Calmes JM, Giusti V. European experience with laparoscopic Roux-en-Y gastric bypass in 466 obese patients. Br J Surg 2006;93:726–32.
- [54] Suter M, Donadini A, Calmes JM, Romy S. Improved surgical technique for laparoscopic Roux-en-Y gastric bypass reduces complications at the gastrojejunostomy. Obes Surg 2010;20:841–5.
- [55] Csendes A, Burgos AM, Burdiles P. Incidence of anastomotic strictures after gastric bypass: a prospective consecutive routine endoscopic study 1 month and 17 months after surgery in 441 patients with morbid obesity. Obes Surg 2009;19:269–73.
- [56] Facciano E, Iannelli A, Gugenheim J, Msika S. Internal hernias and nonclosure of mesenteric defects during laparoscopic Roux-en-Y gastric bypass. Obes Surg 2010;20:676–8.
- [57] Chebli JE, Schindler R. The results of a surgical complication protection program (BLIS, Inc.) for private pay bariatric patients in the U.S.: 2006–2011. Obes Surg 2012;22:1798–801.

- [58] 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;19:135–8.
- [59] Rasmussen JJ, Fuller W, Ali MR. Marginal ulceration after laparoscopic gastric bypass: an analysis of predisposing factors in 260 patients. *Surg Endosc* 2007;21:1090–4.
- [60] Chakhtoura G, et al. Primary results of laparoscopic mini-gastric bypass in a French obesity-surgery specialized university hospital. *Obes Surg* 2008;18:1130–3.
- [61] Sacks BC, 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;2:11–6.
- [62] Lublin M, McCoy M, Waldrep DJ. Perforating marginal ulcers after laparoscopic gastric bypass. *Surg Endosc* 2006;20:51–4.
- [63] Marmuse JP, Parenti LR. Gastric bypass. Principles, complications, and results. *J Visc Surg* 2010;147:e31–7.
- [64] Geubbels N, et al. Meta-analysis of internal herniation after gastric bypass surgery. *Br J Surg* 2015;102:451–60.
- [65] 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.
- [66] Iannuccilli JD, 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.
- [67] Stenberg E, et al. Closure of mesenteric defects in laparoscopic gastric bypass: a multicentre, randomised, parallel, open-label trial. *Lancet* 2016;387:1397–404.
- [68] Rondelli F, et al. Antecolic or retrocolic alimentary limb in laparoscopic Roux-en-Y gastric bypass? A meta-analysis. *Obes Surg* 2016;26:182–95.
- [69] Al Harakeh AB, Kallies KJ, Borgert AJ, Kothari SN. Bowel obstruction rates in antecolic/antegastric versus retrocolic/retrogastric Roux limb gastric bypass: a meta-analysis. *Surg Obes Relat Dis* 2016;12:194–8.
- [70] Rosas U, et al. Mesenteric defect closure in laparoscopic Roux-en-Y gastric bypass: a randomized controlled trial. *Surg Endosc* 2015;29:2486–90.
- [71] Wudel Jr LJ, et al. Prevention of gallstone formation in morbidly obese patients undergoing rapid weight loss: results of a randomized controlled pilot study. *J Surg Res* 2002;102:50–6.
- [72] Coupaye M, Ledoux S, Msika S. [Long-term management of patients after bariatric surgery]. *Presse Med* 2008;37:1007–14.
- [73] Sugerman HJ, et al. A multicenter, placebo-controlled, randomized, double-blind, prospective trial of prophylactic ursodiol for the prevention of gallstone formation following gastric-bypass-induced rapid weight loss. *Am J Surg* 1995;169:91–6 [discussion 96–97].
- [74] Warschikow R, et al. Concomitant cholecystectomy during laparoscopic Roux-en-Y gastric bypass in obese patients is not justified: a meta-analysis. *Obes Surg* 2013;23:397–407.
- [75] D'Hondt M, et al. Prophylactic cholecystectomy, a mandatory step in morbidly obese patients undergoing laparoscopic Roux-en-Y gastric bypass? *J Gastrointest Surg* 2011;15:1532–6.
- [76] Nagem R, Lazaro-da-Silva A. Cholezystolithiasis after gastric bypass: a clinical, biochemical, and ultrasonographic 3-year follow-up study. *Obes Surg* 2012;22:1594–9.
- [77] Nagem RG, Lazaro-da-Silva A, de Oliveira RM, Morato VG. Gallstone-related complications after Roux-en-Y gastric bypass: a prospective study. *Hepatobiliary Pancreat Dis Int* 2012;11:630–5.
- [78] Chevallier JM, et al. One thousand single anastomosis (omega loop) gastric bypasses to treat morbid obesity in a 7-year period: outcomes show few complications and good efficacy. *Obes Surg* 2015;25:951–8.
- [79] Rutledge R. The mini-gastric bypass: experience with the first 1274 cases. *Obes Surg* 2001;11:276–80.
- [80] Treadwell JR, Sun F, Schoelles K. Systematic review and meta-analysis of bariatric surgery for pediatric obesity. *Ann Surg* 2008;248:763–76.
- [81] Rosenthal RJ, et al. International Sleeve Gastrectomy Expert Panel Consensus Statement: best practice guidelines based on experience of >12,000 cases. *Surg Obes Relat Dis* 2012;8:8–19.
- [82] Stenard F, Iannelli A. Laparoscopic sleeve gastrectomy and gastroesophageal reflux. *World J Gastroenterol* 2015;21:10348–57.
- [83] Nguyen NT, Huerta S, Gelfand D, Stevens CM, Jim J. Bowel obstruction after laparoscopic Roux-en-Y gastric bypass. *Obes Surg* 2004;14:190–6.