REVIEW ARTICLE



Controversy Surrounding 'Mini' Gastric Bypass

Kamal K. Mahawar • William R. J. Carr • Shlok Balupuri • Peter K. Small

Published online: 8 October 2013 © Springer Science+Business Media New York 2013

Abstract Mini gastric bypass is a modification of Mason loop gastric bypass with a longer lesser curvature-based pouch. Though it has been around for more than 15 years, its uptake by the bariatric community has been relatively slow, and the procedure has been mired in controversy right from its early days. Lately, there seems to be a surge in the interest in this procedure, and there is now published experience with more than 5,000 procedures globally. This review examines the major controversial aspects of this procedure against the available scientific literature. Surgeons performing this procedure need to be aware of these controversies and counsel their patients appropriately.

Keywords Bariatric surgery · Mini gastric bypass · Omega loop gastric bypass · Obesity · Obesity surgery · Biliary reflux · Cancer

Abbreviations

MGB	Mini gastric bypass
RYGB	Roux-en-Y gastric bypass
PRISMA	Preferred Reporting Items for Systematic
	Reviews and Meta-Analyses
LRYGB	Laparoscopic Roux-en-Y gastric bypass
LMGB	Laparoscopic mini gastric bypass
GORD	Gastro-oesophageal reflux disease

Introduction

Mini gastric bypass (MGB) is a modification of Mason loop gastric bypass [1] with a longer lesser curvature-based pouch. Even though the first MGB procedure was performed more than 15 years ago in 1997 and reported in 2001 [2], its uptake by the bariatric community has been slow and controversial [3, 4]. Symptomatic biliary gastritis and oesophagitis, requiring revisional surgery, have been reported in the literature [5]. Others have expressed concern about the risk for gastric/ oesophageal cancer due to chronic alkaline reflux [3, 6, 7]. Concerns have also been expressed about reported complication rates and the extent of follow-up, with a recommendation to establish a registry of complications and revisional procedures [5].

At the same time, this procedure has its own unique advantages. It is an attractive bariatric procedure compared to the gold standard Roux-en-Y gastric bypass (RYGB) with one less anastomosis. Many thousands of these procedures have now been performed by different surgeons who believe it is a better alternative to RYGB due to shorter operative time, fewer sites for anastomotic leaks and internal herniation, shorter learning curve, ease of reversibility and revision with equivalent results in terms of weight loss and co-morbidity resolution [8–16].

This paper systematically examines the controversial aspects of this procedure on the basis of evidence available in the scientific literature in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.

Methods

An online search in PubMed, Embase, and Google Scholar was performed by two authors independently to identify all relevant clinical literature on MGB using key words 'mini gastric bypass', 'single anastomosis gastric bypass', 'omega loop gastric bypass', 'Mason loop gastric bypass', 'loop gastric bypass', 'bile reflux', 'Barrett's oesophagus', 'gastric cancer', and 'oesophageal cancer' in various combination. The articles were also obtained from the references of these articles. Last of this search was carried out on 30 April 2013. These articles were chosen as they either described significant experience with this

K. K. Mahawar (⊠) • W. R. J. Carr • S. Balupuri • P. K. Small Bariatric Unit, Sunderland Royal Hospital, Sunderland SR4 7TP, UK e-mail: kamal_mahawar@hotmail.com

procedure or otherwise made a useful contribution to the debate on this procedure. Figure 1 gives the PRISMA flow chart for article selection.

Results

There are a number of controversial aspects with this procedure. We discuss below each of these aspects against available published scientific literature.

Gastric and Oesophageal Bile Reflux

Fig. 1 PRISMA article selection

flow chart

First published opposition to MGB came from Fisher et al. [3]. Amongst other things, they pointed out the paper from McCarthy et al. [7], who studied endoscopic, chemical, and histological findings in 28 random patients with 'loop gastric bypass', 'loop gastric bypass plus entero-enterostomy' between the afferent and efferent loops, and 'Roux-en-Y gastric bypass'. Total bile acid levels for the three groups were as follows: 5, 092±1,673 µmol/L, 1,638±581 µmol/L, and 404±384 µmol/ L, respectively. The incidence of 'endoscopic' gastritis was 71 % in the standard loop bypass, 45 % in the enteroenterostomy group, and 13 % in the Roux-en-Y group. Histological abnormalities were present in 86 % of the patients who underwent standard loop bypass, in 91 % of those with an additional entero-enterostomy, and in 63 % of the Roux-en-Y group. There was poor correlation of symptoms and objective findings in this study. It is worth noting that a number of patients with RYGB also had histologically proven gastritis in this study. Others have observed similar findings [17].

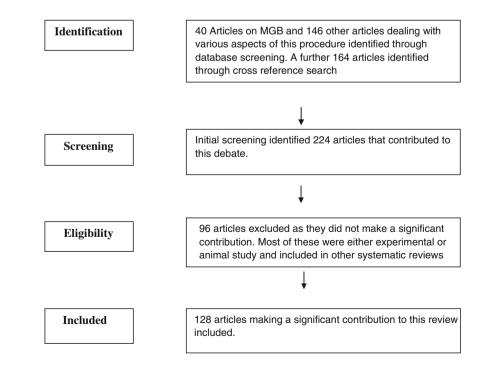
325

The study by McCarthy et al. [7] was published in 1985, long before the first MGB was performed and included patients who underwent Mason loop gastric bypass [1]. However, despite the obvious differences between the previously performed Mason's loop gastric bypass (high transverse gastric pouch based on the fundus) and the MGB (lesser curvature-based longer pouch), both of them are likely to lead to increased exposure of gastric mucosa to bile. It is however worth noting that the incidence of histological gastritis between the different groups in the study by McCarthy et al. [7] was not vastly different, and symptom correlation was poor.

Though duodenogastric bile reflux is a physiological phenomenon [18, 19], excessive duodenogastric biliary reflux can cause intestinal metaplasia and symptomatic gastritis/ oesophagitis [20–22]. A higher gastric and/or oesophageal concentration of bile is seen after surgical procedures like cholecystectomy [23–26], Billroth II gastrectomy [27–30] and, as McCarthy et al. [7] showed, after Mason's loop gastric bypass. Though there are no studies formally evaluating the bile acid levels in gastric pouch after MGB, it is likely that these patients will also have higher concentration of bile in the gastric pouch and oesophagus. However, we should be cautious in interpreting the significance of bile acid levels in the stomach in predicting symptomatic gastric/oesophageal reflux [31–34] or risk for gastric/oesophageal cancer. We examine these issues in the following paragraphs.

Symptomatic Biliary Reflux

The second published criticism of this procedure came from Johnson et al. [5]. They searched the databases of five medical



centres retrospectively to identify patients who needed surgical revision after a MGB procedure and found 32 patients. Indications for revisional surgery were gastrojejunostomy leak in 3, bile reflux in 20, intractable marginal ulcer in 5, malabsorption/malnutrition in 8, and weight gain in 2. Of these, 21 required conversions to RYGB, an additional 5 were planned for revision, 2 were treated with Braun enteroenterostomies, and 4 required one or more abdominal explorations. Three patients with anastomotic leaks required multiple operations to control sepsis, and two of them subsequently underwent conversion to RYGB. Five patients with intractable marginal ulcer had either undergone revision to RYGB (4) or planned for it (1). The most frequent complication reported in this study was bile reflux gastritis, which was seen in 20 patients. This necessitated conversion to RYGB in 14, and an additional 4 were planned for it. The remaining two of these patients were treated with Braun entero-enterostomies. The authors admit that the denominator from which these patients come is unknown, and significantly none of the patients in this study had biliary oesophagitis as their dominant problem. The authors of this paper reported that leaks following this procedure are more difficult to manage than those after Roux-en-Y reconstructions because of large volumes of biliary and pancreatic secretions.

On the other hand, surgeons who have significant experience with MGB have not reported symptomatic reflux to be a major problem after this procedure. Rutledge and Walsh reported significant improvement (85 %) in reflux-related symptoms in their series [8]. In Rutledge's earlier paper [2], only 0.6 % (six) of the patients had reflux postoperatively as opposed to 62 % of the patients preoperatively. None of the patients in the study by Carbajo et al. [13] had reflux symptoms postoperatively. They studied reflux with 24-h pH and manometry in conjunction with endoscopy at 12 and 18 months after surgery in their first 20 patients, and the results were normal in all. This, of course, does not measure bile reflux. Noun et al. [9] reported symptomatic biliary reflux in 4 patients (all revisional MGB) in their series of 1,000 patients. However, they believe that it is easily corrected by 'stapling the afferent loop just before the gastrojejunostomy and performing a latero-lateral jejuno-jejunostomy 70 cm distal to the gastrojejunostomy'. In the only available randomized controlled trial (RCT) of MGB vs RYGB [35], both procedures significantly improved the total Gastrointestinal Quality of Life Index (GIQLI) at 1 year to a similar extent. The same group reported [16] similar improvement in GIQLI score in their retrospective comparative analysis of RYGB and MGB over a period of 10 years. However, MGB patients had a better score in abdominal pain, but lower score in eating with pleasure and trouble with diarrhoea than RYGB patients. None of these authors have reported leaks following MGB to be particularly difficult.

Marginal Ulcer and Poor Follow-Up

Johnson et al. [5] claimed that reoperation rate for intractable marginal ulcers reported in the largest published series on MGB by Rutledge and Walsh [8] was 'clearly inaccurate and as a result of poor follow up and/or bias'. In their paper [8], Rutledge and Walsh reported persistent ulcers requiring revisional surgery in three patients; Johnson et al. [5] described five patients in their study who needed (or were waiting for) a conversion to RYGB for intractable marginal ulcers, and the authors suspected that many of these patients were operated by Rutledge. It is significant that Rutledge's study reports ulcers in 97 (4 %) patients, and their reported follow-up was 68 %. It is possible that some of these patients who developed marginal ulcers then presented to other centres and required revisional surgery. Reported marginal ulcer rate after MGB in the three big studies [8, 9, 16] of 0.6-4.0 % seems similar to that seen with RYGB [36-38]. All six patients in the report on 1,000 MGBs by Noun et al. [9] responded to PPI. Lee et al. [16] reported a 0.6 % rate of marginal ulcer in both MGB (n = 7/1, 163) and RYGB (3/494) patients in their retrospective comparative study of two procedures with similar improvements in Gastrointestinal Quality of Life scores at 5 years. They had a 56 % follow-up at 5 years. It is however obvious that some patients with marginal ulcers following MGB, like those undergoing RYGB, will need revisional surgery. It is unclear whether these ulcers after MGB are less responsive to medical management than those developing after RYGB. The point regarding poor follow-up is very valid and a general concern with all bariatric procedures, most of which are relatively new, and surgeons should think of ways to improve follow-up [39-43].

Long-term Risk for Gastric/Oesophageal Cancer due to Biliary Reflux

To the best of our knowledge, there has been no report of gastric or oesophageal cancers following MGB. However, in view of small numbers performed and relatively short follow-up available, this information is not of much clinical importance. In the absence of such a direct evidence that MGB is associated with a higher risk for gastric and/or oesophageal cancer, we examined incidence of Barrett's oesophagus and gastric and/or oesophageal cancers in other groups of patients (Billroth types I and II gastrectomy, cholecystectomy, and Mason's loop gastric bypass) who have a higher concentration of bile in stomach and/or oesophagus.

Bile Reflux and Barrett's Oesophagus

A recent systematic review [43] of 83 articles noted that in 'invivo studies', bile acid concentrations were higher in the oesophageal aspirates of patients with gastro-oesophageal reflux disease (GORD) than those of the controls, and bile acid infusions triggered GORD symptoms. The review also found that in the in vitro studies, bile acid stimulated squamous oesophageal cells and Barrett's epithelial cells to produce inflammatory mediators (e.g. IL-8 and COX-2) and caused oxidative stress, DNA damage and apoptosis. They also induced squamous cells to change their gene expression pattern to resemble intestinal-type cells and caused Barrett's cells to increase expression of intestinal-type genes.

Clinical studies however have not shown an unequivocal association between conditions with a higher gastric concentration of bile and Barrett's oesophagus. Nason et al. [44] found that risk for Barrett's oesophagus did not significantly vary with increasing concentrations of total or free bile acids in the stomach. Taha et al. [45] did not find increased prevalence or severity of reflux or Barrett's oesophagus in 140 patients who had undergone different types of gastric surgery (62 patients with vagotomy and gastroenterostomy, 21 with vagotomy and pyloroplasty, 15 gastro-enterostomy, 14 Billroth 1, and 28 Billroth II) compared to 100 patients with a normal stomach. One hundred nineteen of these 140 patients had bile gastritis, and 31 of these 119 also had biliary oesophagitis. Parrilla et al. [46], Avidan et al. [47], and Akiyama et al. [48] have also found that gastric surgery did not increase the risk for developing Barrett's oesophagus. Avidan et al. [47] concluded that gastric surgery for benign peptic ulcer disease was not a risk factor for either short- or long-segment Barrett's oesophagus. Akiyama et al. [48] did not find any association between distal gastrectomy and incidence or progression of Barrett's oesophagus. Champion et al. [49] further found that fasting bile acid concentrations in distal oesophagus did not distinguish patients with Barrett's oesophagus from those with reflux without Barrett's. They concluded that increased quantity of acid reflux was the single factor most characterizing patients with Barrett's oesophagus.

Partial Gastrectomy and Gastric Cancer

Partial gastrectomy, Billroth II in particular, is associated with bile reflux into the stomach [27–30]. This is believed by some to be the most logical explanation for higher incidence of gastric cancers reported in these patients [50–56]. This is not however a view shared by all [57–63]. Others have only found a long-term association. Lagergren et al. [64] found that the total number of observed gastric stump cancers was not higher than expected in their population-based cohort study of 18, 912 patients who had undergone distal gastrectomy for benign ulcer disease between 1964 and 2008 in Sweden. However, there was an increased risk for cancer in the gastric remnant 30 years or longer after gastric resection for benign disease. La Vecchia et al. [65] got similar results. They found that within 20 years after gastrectomy, the relative risk for gastric cancer was not significantly raised (RR=1.2, 95 % CI 0.5–2.8), but a positive association emerged after longer time intervals. The RR was 1.6 (95 % CI 0.7–4.1) after 20 to 29 years, and 3.5 (95 % CI 1.3–10.0) after 30 years or more. In their analysis, published in 1992, of the 12 prospective epidemiological studies, Lacaine et al. [66] found that 7 of them showed an increased risk for gastric cancer in patients operated on for peptic ulcer disease, while the remaining 5 studies did not. The authors believed that 'evidence was good enough to identify patients who underwent partial gastrectomy more than 20 years previously as a high-risk group for the development of carcinoma'. We examine below the possible factors behind this observed association.

Helicobacter pylori is a common etiologic agent for both gastric ulcers and gastric cancers, and patients with gastric ulcers are at an increased risk for gastric cancer [67]. Seoane et al. [68] found H. pylori in 100 % of gastrectomy patients who eventually developed stump cancer as opposed to 85 % of those who did not. This bacterium was first reported by Marshall and Warren in 1984 [69]. Rapid urease test came in 1987 [70], and the first eradication regime was suggested in 1988 [71]. It is worth noting that almost all of the studies [51–56] that show a significant association between partial gastrectomy and gastric cancer predate these important developments. The meta-analysis published by Tersmette et al. [72] that showed a relative risk of 1.46 for gastric cancer in postgastrectomy patients was also published in 1990. A more recent very large study [73] showed a 40 % risk reduction in resected gastric ulcer patients as opposed to their unoperated peers, who had double the risk for cancer compared to matched population.

Interestingly, the increase in risk for gastric cancer is associated with all kinds of benign ulcer surgeries [50, 74, 75] and not just Billroth II gastrectomy. This perhaps points towards H. pylori rather than bile reflux being the dominant factor in its pathogenesis. A slightly higher risk for gastric cancers seen with Billroth II gastrectomy as opposed to Billroth I gastrectomy by some authors [50, 72] may point to a synergistic role of bile and H. pylori in the pathogenesis of gastric stump cancer [76]. This is further reinforced by findings that H. *pylori*, either alone [77] or in conjunction with bile reflux [78], is an important factor in the pathogenesis of reflux gastritis after gastrectomy. Another important variable, which is whether biliary reflux facilitates or hampers survival of H. *pylori*, is a matter of debate, and this further complicates the issue [79-81]. In their comprehensive review of gastric stump carcinoma, Sitarz et al. [82] concluded that achlorhydria, hypergastrinaemia, bile reflux, Epstein-Barr virus, H. pylori, atrophic gastritis and polymorphisms in interleukin 1 beta and cyclooxygenase 2 all play a role in the pathogenesis of gastric stump cancer.

It is also worth noting that many studies from the Far East have not found a higher risk for cancer after Billroth types I and II gastrectomies [61, 83, 84]. Many surgeons there routinely use these methods to reconstruct anatomy after distal gastrectomy [85–90]. Since these populations report a higher incidence of gastric cancer as such, it is possible that removal of a significant part of the stomach more than compensates for the additional risks imposed by surgery.

Partial Gastrectomy and Oesophageal Cancer

Lagergren and Lindam [91] concluded that gastrectomy (Billroth II) for peptic ulcer disease did not increase the risk for oesophageal adenocarcinoma from their populationbased cohort study of 19,767patients who had undergone gastrectomy for peptic ulcer disease between 1964 and 2008 in Sweden. Birgisson et al. [92] found that previous gastric surgery was rarely found in patients with oesophageal cancer. They concluded that gastric surgery and its associated duodenogastroesophageal reflux do not play a role in the aetiology and rising incidence of adenocarcinoma of the oesophagus.

Lundegårdh et al. [75] found an increased risk for oesophageal cancer (relative risk of 2.2) in patients operated on for stomach ulcer. Alexandrou et al. [93] and Hashimoto et al. [94] found that a history of gastrectomy (especially the Billroth I type) is associated with more lower-third squamous cell oesophageal carcinomas. One suspects that the higher risk for squamous cell cancer seen in these studies would be due to factors other than biliary reflux. For example, smoking and alcohol have been implicated in the pathogenesis of both peptic ulcer disease and squamous cell carcinoma of the oesophagus.

Cholecystectomy and Gastric/Oesophageal Cancer

Cholecystectomy increases bile reflux into the stomach [23–25, 95, 96], but does not produce detectable difference in bile concentration in the oesophagus [97]. Cholecystectomized patients have not been found to be at a higher risk for gastric and oesophageal cancer [98–101]. It is possible that an association seen with oesophageal adenocarcinoma in some studies [102, 103] is due to the fact that symptomatic severe gallstone disease and adenocarcinoma of the oesophagus share some common risk factors such as obesity. Authors do not believe risk for oesophageal cancers to be a significant risk before offering cholecystectomy to their patients with symptomatic gallstone disease.

Mason's Loop Gastric Bypass

As discussed above, despite some differences, there are fundamental similarities between Mason's loop gastric bypass and MGB. The incidence of cancers in the historical cohort of loop gastric bypass patients can hence be used to get an idea of the risk for long-term gastric cancer with MGB.

Scozzari et al. [104] recently published a systematic review of all published cases of oesophago-gastric cancers after bariatric surgery. In the literature, they found 33 reported cancers after bariatric surgery. It is worth noting that only one of them was in the gastric pouch of a patient who had undergone Mason loop gastric bypass 26 years previously. In the absence of a known denominator, the relative risk is difficult to estimate. There were two further cancers reported in the bypassed stomach after Mason loop gastric bypass. There is currently insufficient data to comment on the overall risk for gastric cancer in the bypassed stomach after any type of gastric bypass. Though, it has been reported after both RYGB and Mason's bypass [104], some surgeons believe that gastric bypass actually protects against cancer in the bypassed stomach. An animal study [105] recently suggested that RYGB reduced the risk for cancer in an experimental rat model of dietary-induced carcinogenesis. It is also worth noting that Scozzari et al. [104] did not find any report of cancer after MGB, but this is probably insignificant in view of the small numbers and relatively short follow-up available for these patients.

Evidence Based on the Scientific Literature

At the present time, there are only a handful of studies describing any clinical experience with this procedure [2, 8-16,35, 106-117], but they present more than 5,000 published procedures with three groups reporting experience of at least a thousand patients [8, 9, 16]. There is only one RCT comparing laparoscopic Roux-en-Y gastric bypass (LRYGB) and laparoscopic mini gastric bypass (LMGB) from Taiwan, with 40 patients in each arm [35]. This RCT does not meet all the CONSORT guideline requirements, but does have robust follow-up. LRYGB had a significantly higher complication rate of 20% (eight patients) compared to 7.5% (three patients) with LMGB. There were two anastomotic leaks seen with LRYGB. Significantly higher number of patients achieved an excess weight loss of >50 % with LMGB (95 % vs 75 %), with no mortality in either group. The incidence of marginal ulcer was 5 % in the LMGB group, and 3 % in the LRYGB group. Paucity of level 1 evidence plagues the entire field of bariatric surgery, and more RCTs are needed to establish the evidence base for various bariatric procedures.

Malabsorption, Weight Loss, and Quality of Life

Though there is no published data on malabsorption after MGB, it is widely acknowledged that this procedure is more malabsorptive than RYGB. Many MGB surgeons believe that this procedure is more malabsorptive as it behaves a bit like biliopancreatic diversion with a biliopancreatic limb length of 2.0 m. This is in comparison to RYGB, where biliopancreatic limb length is typically only 50 cm, and the alimentary limb

length is 150 cm. These surgeons suggest that though the total bypassed small bowel is equal in both procedures, MGB is more malabsorptive as there is some potentially for digestion (due to salivary, gastric, and intestinal enzymes) and absorption (as food is in contact with small bowel) in the alimentary limb of a RYGB patient; this is not the case with MGB, where food is not in contact with the bypassed 2.0 m of the small bowel at all.

More malabsorption may explain why MGB patients have similar, if not superior, weight loss compared to RYGB patients, even though restrictive component provided by this procedure is lesser (due to a longer and bigger pouch). In our recently published systematic review on MGB [117], we observed an excess weight loss (EWL) of 76, 74.6, and 71 % at 12, 18–24, and 60 months, respectively. In the retrospective study of Lee et al. [16], the MGB patients had significantly better weight loss at 5 years (72.9 % vs 60.1 %, p value <005).

The automatic next question that then arises is whether this increased malabsorption translates into more diarrhoea and an adverse impact on quality of life. We have also examined quality of life with this procedure in our previously mentioned review [117] and generally found positive impact on quality of life after this procedure. Most authors [8, 13, 16] have reported improvement in patients' quality of life after this procedure. In the only available RCT of MGB and RYGB published by Lee's group [35], there was no significant difference in the Gastrointestinal Quality of Life Index score in the two groups 1 year after surgery, and the score was significantly higher compared to the preoperative score in both groups.

At the same time, higher malabsorption is a double-edged sword. Where, on one hand, it may mean better success in terms of weight loss, co-morbidity resolution, and less reliance on restriction (enabling patients to maintain a healthy diet), it also risks excessive malabsorption, requiring reversal of this procedure in some patients [8]. However, in these patients, ease with which this procedure can be reversed laparoscopically may prove beneficial.

Technical Ease, Learning Curve, and Complications

The proponents of this procedure claim that having one less anastomosis translates into technical ease, shorter learning curve, and lower short-term and long-term complication rates. In their 10-year retrospective experience of RYGB and MGB [16], Lee et al. observed a lower complication rate with MGB as compared to that with RYGB (1.8 % vs 3.2 %), but the difference was not significant statistically (*p* value 0.071). It is however noteworthy that MGB patients had statistically significant lower major bleeding (0.2 % vs 1.0 %, *p* value 0.016) and early bowel obstruction (0.1 % vs 0.8 %, *p* value 0.014) rates. Some MGB surgeons claim that one less anastomosis

will translate into lower leak rates with this procedure. However, one must note that leaks can also happen from staple lines elsewhere, and it is possible that decreased leak from one less anastomosis and division of omega loop will be offset by leaks from longer staple line on gastric pouch and remnant stomach. Indeed, Lee et al. [16] did not find a significant difference in leak rates between the two procedures (1.3 % with MGB compared to 1.4 % with RYGB, p value 0.836). In the RCT comparing RYGB and MGB published by the same group [35], RYGB took significantly longer to perform and had higher early complication rates (20 % vs 7.5 %). This RCT was however criticised for lack of authors' experience with RYGB. Further studies will be needed to clearly establish these trends towards superior early safety of this procedure. Though, it is conceivable that operative time and early complications are unlikely to be much different in the hands of 'experienced' RYGB surgeons, learning curve is a real issue with bariatric surgery, and procedures with shorter learning curve do tend to become very popular, sometimes even in the absence of long-term convincing data. Lee et al. [35] estimated the learning curve for MGB to be 30 cases less than that for RYGB. In addition, long-term risk for bowel obstruction and internal hernia appears to be lower with MGB. In their retrospective study, Lee et al. [16] found a significantly lower longterm incidence of bowel obstruction (0.1 % vs 0.8 %, p value 0.014) and internal hernia (0 % vs 0.4 %, p value 0.030). Finally, the absence of jejuno-jejunostomy may lend this procedure to easier revision and reversal compared to RYGB. So obviating the jejuno-jejunostomy may have some shortterm and long-term advantages. What remains slightly unclear is the cost at which these gains are achieved.

Name of the Procedure

There is also some controversy around the best name for this procedure. The name 'mini' gastric bypass does sound a bit promotional. Chakhtoura et al. [11] felt that omega loop bypass was a better name for it. We would have preferred the term 'modified loop gastric bypass' as the operation does have some similarities with the loop gastric bypass described by Mason, but there is now a body of scientific as well as grey literature which describes this procedure as mini gastric bypass. Changing the name at this stage will only create confusion and conflict in the minds of future researchers and patients.

Discussion

Bariatric surgeons around the world are constantly looking for safer and more effective options to the gold standard RYGB. Gastric banding, sleeve gastrectomy, and MGB have been invented on the back of these ideas. Even though adjustable gastric banding [118] and sleeve gastrectomy [119, 120] have not been around for around for much longer than MGB, they are now accepted as mainstream bariatric procedures [121, 122]. This is not withstanding the poor results experienced by some patients [123–126]. This has not been the case with MGB, which has been criticised almost since first being reported.

We have discussed all the controversial aspects of this procedure in some detail in this paper. We have attempted to present a balanced view of each controversial aspect and covered all the arguments in favour of and against this procedure comprehensively. It is not the purpose of this paper to take sides, and we hence leave the interpretation of our findings to the readers. Our aim is to inform the debate on this procedure and enable surgeons as well as their patients to choose wisely.

Our own interpretation of the literature is that biliary reflux will lead to a higher incidence of histological gastritis, but it does not always translate into adverse symptomatic outcome. With regards to gastric cancer, it would appear that *H. pylori* is a more important factor and probably an essential prerequisite. It would hence seem a sensible policy to screen for and eradicate *H. pylori* in all patients being considered for MGB. In our unit, we adopt this approach for all our RYGB patients as well. With a considerably longer gastric pouch, as opposed to the Mason loop gastric bypass, symptomatic biliary oesophageal reflux and oesophagitis do not appear to be major problems in published studies with this procedure.

We have not found a worryingly higher incidence of gastric/ oesophageal cancers reported in patients who underwent Mason loop gastric bypass, which is the closest surgical model of this procedure in our view. Any long-term risk for cancers will not be apparent for many decades. We suspect that any potential risk for gastric/oesophageal malignancy in these patients will be very low and outweighed by the benefits, some of which are shorter operative time, shorter learning curve, lower early complication rates, and lower early mortality. In addition, it is easier to revise and reverse compared to sleeve gastrectomy and RYGB.

It is also perhaps worth noting that procedures like gastric banding and sleeve gastrectomy are not free of these theoretical concerns either. Some patients may experience a higher incidence of gastro-oesophageal acid reflux [127, 128] after these procedures, which is a known etiologic factor for adenocarcinoma of the oesophagus. All bariatric procedures have their own unique advantages and shortcomings. The selection of appropriate bariatric procedure for a patient will clearly depend on a range of patient-related, surgeon-related, and procedure-related factors. Careful consideration of these factors by the surgical teams and a detailed discussion of pros and cons of various procedures should enable patients to make an appropriate informed choice.

Conclusion

MGB has been around for more than 15 years now, and many surgeons are routinely performing it. At the same time, there are several controversial aspects of this procedure that have discouraged a wider adoption of this procedure. This review comprehensively examines these aspects of this procedure against available evidence in the scientific literature. Surgeons performing this procedure need to be aware of these controversial aspects of this procedure, including the controversy surrounding the risk for gastric and oesophageal cancers, to be able to counsel their patients appropriately and enable them to make the most appropriate choice for them.

Conflict of interest None

References

- 1. Mason EE, Ito C. Gastric bypass in obesity. Surg Clin North Am. 1967;47:1345–52.
- Rutledge R. The mini-gastric bypass: experience with the first 1274 cases. Obes Surg. 2001;11(3):276–80.
- Fisher BL, Buchwald H, Clark W, et al. Mini-gastric bypass controversy. Obes Surg. 2001;11(6):773–7.
- Olchowski S, Timms MR, O'Brien P, et al. More on mini gastric bypass. Obes Surg. 2001;11(4):532.
- Johnson WH, Fernanadez AZ, Farrell TM, et al. Surgical revision of loop ("mini") gastric bypass procedure: multicenter review of complications and conversions to Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2007;3(1):37–41.
- Collins BJ, Miyashita T, Schweitzer M, et al. Gastric bypass. Why Roux-en-Y? A review of experimental data. Arch Surg. 2007;142(10):1000–3.
- McCarthy HB, Rucker RD, Chan EK, et al. Gastritis after gastric bypass surgery. Surgery. 1985;98:68–71.
- Rutledge R, Walsh TR. Continued excellent results with the minigastric bypass: six-year study in 2,410 patients. Obes Surg. 2005;15(9):1304–8.
- Noun R, Skaff J, Riachi E, et al. One thousand consecutive minigastric bypass: short- and long-term outcome. Obes Surg. 2012;22(5):697–703.
- Peraglie C. Laparoscopic mini-gastric bypass (LMGB) in the supersuper obese: outcomes in 16 patients. Obes Surg. 2008;18(9):1126– 9.
- Chakhtoura G, Zinzindohoué F, Ghanem Y, et al. Primary results of laparoscopic mini-gastric bypass in a French obesity-surgery specialized university hospital. Obes Surg. 2008;18(9):1130–3.
- Piazza L, Ferrara F, Leanza S, et al. A laparoscopic mini-gastric bypass: short-term single-institute experience. Updates Surg. 2011;63(4):239–42.
- Carbajo M, García-Caballero M, Toledano M, et al. Oneanastomosis gastric bypass by laparoscopy: results of the first 209 patients. Obes Surg. 2005;15(3):398–404.
- García-Caballero M, Valle M, Martínez-Moreno JM, et al. Resolution of diabetes mellitus and metabolic syndrome in normal weight 24–29 BMI patients with one anastomosis gastric bypass. Nutr Hosp. 2012;27(2):623–31.
- Kim Z, Hur KY. Laparoscopic mini-gastric bypass for type 2 diabetes: the preliminary report. World J Surg. 2011;35(3):631–6.

- Lee WJ, Ser KH, Lee YC, et al. Laparoscopic Roux-en-Y vs. minigastric bypass for the treatment of morbid obesity: a 10-year experience. Obes Surg. 2012;22(12):1827–34.
- Sinar DR, Flickinger EG, Park HK, et al. Retrograde endoscopy of the bypassed stomach segment after gastric bypass surgery: unexpected lesions. South Med J. 1985;78(3):255–8.
- Byrne JP, Romagnoli R, Bechi P, et al. Duodenogastric reflux of bile in health: the normal range. Physiol Meas. 1999;20(2):149–58.
- Fuchs KH, Maroske J, Fein M, et al. Variability in the composition of physiologic duodenogastric reflux. J Gastrointest Surg. 1999;3(4):389–95. discussion 395–6.
- Fiorucci S, Distrutti E, Di Matteo F, et al. Circadian variations in gastric acid and pepsin secretion and intragastric bile acid in patients with reflux esophagitis and in healthy controls. Am J Gastroenterol. 1995;90(2):270–6.
- Zhang Y, Yang X, Gu W, et al. Histological features of the gastric mucosa in children with primary bile reflux gastritis. World J Surg Oncol. 2012;10:27.
- 22. Matsuhisa T, Arakawa T, Watanabe T, et al. Relation between bile acid reflux into the stomach and the risk of atrophic gastritis and intestinal metaplasia: a multicenter study of 2283 cases. Dig Endosc. 2013;25(5):519–25. doi:10.1111/den.12030.
- Lorusso D, Misciagna G, Mangini V, et al. Duodenogastric reflux of bile acids, gastrin and parietal cells, and gastric acid secretion before and 6 months after cholecystectomy. Am J Surg. 1990;159(6):575–8.
- Kunsch S, Neesse A, Huth J, et al. Increased duodeno-gastroesophageal reflux (DGER) in symptomatic GERD patients with a history of cholecystectomy. Z Gastroenterol. 2009;47(8):744–8.
- Cabrol J, Navarro X, Simo-Deu J, et al. Evaluation of duodenogastric reflux in gallstone disease before and after simple cholecystectomy. Am J Surg. 1990;160(3):283–6.
- Atak I, Ozdil K, Yücel M, et al. The effect of laparoscopic cholecystectomy on the development of alkaline reflux gastritis and intestinal metaplasia. Hepatogastroenterology. 2012;59(113): 59–61.
- 27. Lee Y, Tokunaga A, Tajiri T, et al. Inflammation of the gastric remnant after gastrectomy: mucosal erythema is associated with bile reflux and inflammatory cellular infiltration is associated with *Helicobacter pylori* infection. J Gastroenterol. 2004;39(6):520–6.
- Lorusso D, Linsalata M, Pezzolla F, et al. Duodenogastric reflux and gastric mucosal polyamines in the non-operated stomach and in the gastric remnant after Billroth II gastric resection. A role in gastric carcinogenesis? Anticancer Res. 2000;20(3B):2197–201.
- Parrilla P, Lujan JA, Robles R, et al. Duodenogastric reflux quantification in peptic ulcer surgery: comparison between different surgical techniques. Surgery. 1993;113(1):43–7.
- Bechi P, Balzi M, Becciolini A, et al. Gastric cell proliferation kinetics and bile reflux after partial gastrectomy. Am J Gastroenterol. 1991;86(10):1424–32.
- Watson RG, Love AH. Intragastric bile acid concentrations are unrelated to symptoms of flatulent dyspepsia in patients with and without gallbladder disease and postcholecystectomy. Gut. 1987;28(2):131–6.
- Collins BJ, Crothers G, McFarland RJ, et al. Bile acid concentrations in the gastric juice of patients with erosive oesophagitis. Gut. 1985;26(5):495–9.
- Bost R, Hostein J, Valenti M, et al. Is there an abnormal fasting duodenogastric reflux in nonulcer dyspepsia? Dig Dis Sci. 1990;35(2):193–9.
- Schindlbeck NE, Heinrich C, Stellaard F, et al. Healthy controls have as much bile reflux as gastric ulcer patients. Gut. 1987;28(12): 1577–83.
- Lee WJ, Yu PJ, Wang W, et al. Laparoscopic Roux-en-Y versus mini-gastric bypass for the treatment of morbid obesity: a prospective randomized controlled clinical trial. Ann Surg. 2005;242(1): 20–8.

- 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.
- 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.
- Dallal RM, Bailey LA. Ulcer disease after gastric bypass surgery. Surg Obes Relat Dis. 2006;2(4):455–9.
- McVay MA, Friedman KE, Applegate KL, et al. Patient predictors of follow-up care attendance in Roux-en-Y gastric bypass patients. Surg Obes Relat Dis. 2012. doi:10.1016/j.soard.2012.11.005.
- 40. Lara MD, Baker MT, Larson CJ, et al. Travel distance, age, and sex as factors in follow-up visit compliance in the post-gastric bypass population. Surg Obes Relat Dis. 2005;1(1):17–21.
- Harper J, Madan AK, Ternovits CA, et al. What happens to patients who do not follow-up after bariatric surgery? Am Surg. 2007;73(2): 181–4.
- Shen R, Dugay G, Rajaram K, et al. Impact of patient follow-up on weight loss after bariatric surgery. Obes Surg. 2004;14(4):514–9.
- 43. McQuaid KR, Laine L, Fennerty MB, et al. Systematic review: the role of bile acids in the pathogenesis of gastro-oesophageal reflux disease and related neoplasia. Aliment Pharmacol Ther. 2011;34(2): 146–65.
- Nason KS, Farrow DC, Haigh G, et al. Gastric fluid bile concentrations and risk of Barrett's esophagus. Interact Cardiovasc Thorac Surg. 2007;6(3):304–7.
- 45. Taha AS, Angerson WJ, Morran CG. Reflux and Barrett's oesophagitis after gastric surgery—long-term follow-up and implications for the roles of gastric acid and bile in oesophagitis. Aliment Pharmacol Ther. 2003;17(4):547–52.
- Parrilla P, Liron R, Martinez de Haro LF, et al. Gastric surgery does not increase the risk of developing Barrett's esophagus. Am J Gastroenterol. 1997;92(6):960–3.
- Avidan B, Sonnenberg A, Schnell TG, et al. Gastric surgery is not a risk for Barrett's esophagus or esophageal adenocarcinoma. Gastroenterology. 2001;121(6):1281–5.
- Akiyama T, Inamori M, Akimoto K, et al. Gastric surgery is not a risk factor for erosive esophagitis or Barrett's esophagus. Scand J Gastroenterol. 2010;45(4):403–8.
- Champion G, Richter JE, Vaezi MF, et al. Duodenogastroesophageal reflux: relationship to pH and importance in Barrett's esophagus. Gastroenterology. 1994;107(3):747–54.
- Caygill CP, Hill MJ, Kirkham JS, et al. Mortality from gastric cancer following gastric surgery for peptic ulcer. Lancet. 1986;1(8487): 929–31.
- Lundegårdh G, Adami HO, Helmick C, et al. Stomach cancer after partial gastrectomy for benign ulcer disease. N Engl J Med. 1988;319(4):195–200.
- Fisher SG, Davis F, Nelson R, et al. A cohort study of stomach cancer risk in men after gastric surgery for benign disease. J Natl Cancer Inst. 1993;85(16):1303–10.
- Tersmette AC, Goodman SN, Offerhaus GJ, et al. Multivariate analysis of the risk of stomach cancer after ulcer surgery in an Amsterdam cohort of postgastrectomy patients. Am J Epidemiol. 1991;134(1):14–21.
- Domellöf L, Janunger KG. The risk for gastric carcinoma after partial gastrectomy. Am J Surg. 1977;134(5):581–4.
- Ovaska JT, Havia TV, Kujari HP. Risk of gastric stump carcinoma after gastric resection for benign ulcer disease. Ann Chir Gynaecol. 1986;75(4):192–5.
- Viste A, Bjørnestad E, Opheim P, et al. Risk of carcinoma following gastric operations for benign disease. A historical cohort study of 3470 patients. Lancet. 1986;2(8505):502–5.
- 57. Schafer LW, Larson DE, Melton 3rd LJ, et al. The risk of gastric carcinoma after surgical treatment for benign ulcer disease. A

population-based study in Olmsted County, Minnesota. N Engl J Med. 1983;309(20):1210-3.

- Fischer AB, Graem N, Jensen OM. Risk of gastric cancer after Billroth II resection for duodenal ulcer. Br J Surg. 1983;70(9): 552–4.
- Schnapka G, Hofstaedter F, Schwamberger K, et al. Gastric stump carcinoma following Billroth II resection for peptic ulcer disease. Comparison with cancer in non-operated stomach. Endoscopy. 1984;16(5):171–4.
- Pointner R, Schwab G, Königsrainer A, et al. Gastric stump cancer: etiopathological and clinical aspects. Endoscopy. 1989;21(3): 115–9.
- Tokudome S, Kono S, Ikeda M, et al. A prospective study on primary gastric stump cancer following partial gastrectomy for benign gastroduodenal diseases. Cancer Res. 1984;44(5):2208–12.
- Luukkonen P, Kalima T, Kivilaakso E. Decreased risk of gastric stump carcinoma after partial gastrectomy supplemented with bile diversion. Hepatogastroenterology. 1990;37 Suppl 2:171–3.
- Kivilaakso E, Hakkiluoto A, Kalima TV, et al. Relative risk of stump cancer following partial gastrectomy. Br J Surg. 1977;64(5):336–8.
- Lagergren J, Lindam A, Mason RM. Gastric stump cancer after distal gastrectomy for benign gastric ulcer in a population-based study. Int J Cancer. 2012;131(6):E1048–52.
- La Vecchia C, Negri E, D'Avanzo B, et al. Partial gastrectomy and subsequent gastric cancer risk. J Epidemiol Community Health. 1992;46(1):12–4.
- Lacaine F, Houry S, Huguier M. Stomach cancer after partial gastrectomy for benign ulcer disease. A critical analysis of epidemiological reports. Hepatogastroenterology. 1992;39(1):4–8.
- Hansson LE, Nyrén O, Hsing AW, et al. The risk of stomach cancer in patients with gastric or duodenal ulcer disease. N Engl J Med. 1996;335(4):242–9.
- Seoane A, Bessa X, Alameda F, et al. Role of *Helicobacter pylori* in stomach cancer after partial gastrectomy for benign ulcer disease. Rev Esp Enferm Dig. 2005;97(11):778–85.
- Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet. 1984;1(8390): 1311–5.
- Marshall BJ, Warren JR, Francis GJ, et al. Rapid urease test in the management of *Campylobacter pyloridis*-associated gastritis. Am J Gastroenterol. 1987;82(3):200–10.
- Marshall BJ, Goodwin CS, Warren JR, et al. Prospective doubleblind trial of duodenal ulcer relapse after eradication of *Campylobacter pylori*. Lancet. 1988;2(8626–8627):1437–42.
- 72. Tersmette AC, Offerhaus GJ, Tersmette KW, et al. Meta-analysis of the risk of gastric stump cancer: detection of high risk patient subsets for stomach cancer after remote partial gastrectomy for benign conditions. Cancer Res. 1990;50(20):6486–9.
- Bahmanyar S, Ye W, Dickman PW, et al. Long-term risk of gastric cancer by subsite in operated and unoperated patients hospitalized for peptic ulcer. Am J Gastroenterol. 2007;102(6):1185–91.
- Caygill CP, Knowles RL, Hall R. Increased risk of cancer mortality after vagotomy for peptic ulcer: a preliminary analysis. Eur J Cancer Prev. 1991;1(1):35–7.
- Lundegårdh G, Adami HO, Helmick C, et al. Risk of cancer following partial gastrectomy for benign ulcer disease. Br J Surg. 1994;81(8):1164–7.
- Leivonen M, Nordling S, Haglund C. Does *Helicobacter pylori* in the gastric stump increase the cancer risk after certain reconstruction types? Anticancer Res. 1997;17(5B):3893–6.
- Nagahata Y, Kawakita N, Azumi Y, et al. Etiological involvement of *Helicobacter pylori* in "reflux" gastritis after gastrectomy. Am J Gastroenterol. 1996;91(10):2130–4.
- Li XB, Lu H, Chen HM, et al. Role of bile reflux and *Helicobacter pylori* infection on inflammation of gastric remnant after distal gastrectomy. J Dig Dis. 2008;9(4):208–12.

- Nakagawara H, Miwa K, Nakamura S, et al. Duodenogastric reflux sustains *Helicobacter pylori* infection in the gastric stump. Scand J Gastroenterol. 2003;38(9):931–7.
- Bair MJ, Wu MS, Chang WH, et al. Spontaneous clearance of *Helicobacter pylori* colonization in patients with partial gastrectomy: correlates with operative procedures and duration after operation. J Formos Med Assoc. 2009;108(1):13–9.
- Tomtitchong P, Onda M, Matsukura N, et al. *Helicobacter pylori* infection in the remnant stomach after gastrectomy: with special reference to the difference between Billroth I and II anastomoses. J Clin Gastroenterol. 1998;27 Suppl 1:S154–8.
- Sitarz R, Maciejewski R, Polkowski WP, et al. Gastroenterostoma after Billroth antrectomy as a premalignant condition. World J Gastroenterol. 2012;18(25):3201–6.
- Nunobe S, Ohyama S, Miyata S, et al. Incidence of gastric cancer in the remnant stomach after proximal gastrectomy. Hepatogastroenterology. 2008;55(86–87):1855–8.
- 84. Tersmette AC, Giardiello FM, Offerhaus GJ, et al. Geographical variance in the risk of gastric stump cancer: no increased risk in Japan? Jpn J Cancer Res. 1991;82(3):266–72.
- Lee SW, Tanigawa N, Nomura E, et al. Benefits of intracorporeal gastrointestinal anastomosis following laparoscopic distal gastrectomy. World J Surg Oncol. 2012;10:267.
- Du J, Shuang J, Li J, et al. Totally laparoscopic Billroth II gastrectomy with a novel, safe, simple, and time-saving anastomosis by only stapling devices. J Gastrointest Surg. 2012;16(4):738–43.
- Lee J, Kim D, Kim W. Comparison of laparoscopy-assisted and totally laparoscopic Billroth-II distal gastrectomy for gastric cancer. J Korean Surg Soc. 2012;82(3):135–42.
- Oh SJ, Hong JJ, Oh CA, et al. Stapling technique for performing Billroth II anastomosis after distal gastrectomy. J Gastrointest Surg. 2011;15(7):1244–6.
- 89. Kang KC, Cho GS, Han SU, et al. Korean Laparoscopic Gastrointestinal Surgery Study (KLASS) Group. Comparison of Billroth I and Billroth II reconstructions after laparoscopy-assisted distal gastrectomy: a retrospective analysis of large-scale multicenter results from Korea. Surg Endosc. 2011;25(6):1953–61.
- Sah BK, Chen MM, Yan M, et al. Gastric cancer surgery: Billroth I or Billroth II for distal gastrectomy? BMC Cancer. 2009;9:428.
- Lagergren J, Lindam A. The risk of oesophageal adenocarcinoma after gastrectomy for peptic ulcer disease. Eur J Cancer. 2012;48(5): 749–52.
- 92. Birgisson S, Rice TW, Easley KA, et al. The lack of association between adenocarcinoma of the esophagus and gastric surgery: a retrospective study. Am J Gastroenterol. 1997;92(2):216–21.
- Alexandrou A, Davis PA, Law S, et al. Esophageal cancer in patients with a history of distal gastrectomy. Arch Surg. 2002;137(11):1238– 42.
- Hashimoto N, Inayama M, Fujishima M, et al. Esophageal cancer after distal gastrectomy. Dis Esophagus. 2006;19(5):346–9.
- Lorusso D, Pezzolla F, Linsalata M, et al. Duodenogastric reflux and gastric mucosal cell proliferation after cholecystectomy or Billroth II gastric resection. Gastroenterol Clin Biol. 1994;18(11):927–31.
- Wilson P, Jamieson JR, Hinder RA, et al. Pathologic duodenogastric reflux associated with persistence of symptoms after cholecystectomy. Surgery. 1995;117(4):421–8.
- Fein M, Bueter M, Sailer M, et al. Effect of cholecystectomy on gastric and esophageal bile reflux in patients with upper gastrointestinal symptoms. Dig Dis Sci. 2008;53(5):1186–91.
- Fall K, Ye W, Nyrén O. Risk for gastric cancer after cholecystectomy. Am J Gastroenterol. 2007;102(6):1180–4.
- Freedman J, Lagergren J, Bergström R, et al. Cholecystectomy, peptic ulcer disease and the risk of adenocarcinoma of the oesophagus and gastric cardia. Br J Surg. 2000;87(8):1087–93.
- Ge Z, Zhao C, Wang Y, et al. Cholecystectomy and the risk of esophageal and gastric cancer. Saudi Med J. 2012;33(10):1073–9.

- 101. Gustavsson S, Adami HO, Meirik O, et al. Cholecystectomy as a risk factor for gastric cancer. A cohort study. Dig Dis Sci. 1984;29(2):116–20.
- Freedman J, Ye W, Näslund E, et al. Association between cholecystectomy and adenocarcinoma of the esophagus. Gastroenterology. 2001;121(3):548–53.
- Lagergren J, Mattsson F. Cholecystectomy as a risk factor for oesophageal adenocarcinoma. Br J Surg. 2011;98(8):1133–7.
- 104. Scozzari G, Trapani R, Toppino M, et al. Esophagogastric cancer after bariatric surgery: systematic review of the literature. Surg Obes Relat Dis. 2013;9(1):133–42.
- Inoue H, Rubino F, Shimada Y, et al. Risk of gastric cancer after Roux-en-Y gastric bypass. Arch Surg. 2007;142(10):947–53.
- Rutledge R. Hospitalization before and after mini-gastric bypass surgery. Int J Surg. 2007;5(1):35–40.
- 107. Wang W, Wei PL, Lee YC, et al. Short-term results of laparoscopic mini-gastric bypass. Obes Surg. 2005;15(5):648–54.
- Noun R, Zeidan S. Laparoscopic mini-gastric bypass: an effective option for the treatment of morbid obesity. J Chir (Paris). 2007;144(4):301–4 [Article in French].
- Chevallier JM, Chakhtoura G, Zinzindohoué F. [Laparoscopic minigastric bypass]. J Chir (Paris). 2009;146(1):60–4.
- 110. Copăescu C, Munteanu R, Prala N, et al. Laparoscopic mini gastric bypass for the treatment of morbid obesity. Initial experience. Chirurgia (Bucur). 2004;99(6):529–39 [Article in Romanian].
- 111. Hu XG, Zheng CZ, Ji XR, et al. Short-term outcome of laparoscopic gastric bypass and minigastric bypass on obesity patients with type 2 diabetes mellitus. Zhonghua Wei Chang Wai Ke Za Zhi. 2009;12(6): 554–7 [Article in Chinese].
- 112. Guo X, Yin K, Zhuo GZ, et al. Efficacy comparison between 2 methods of laparoscopic gastric bypass surgery in the treatment of type 2 diabetes mellitus]. Zhonghua Wei Chang Wai Ke Za Zhi. 2012;15(11):1125–8 [Article in Chinese].
- Lee WJ, Lee YC, Ser KH, et al. Revisional surgery for laparoscopic minigastric bypass. Surg Obes Relat Dis. 2011;7(4): 486–91.
- 114. Lee WJ, Wang W, Lee YC, et al. Effect of laparoscopic mini-gastric bypass for type 2 diabetes mellitus: comparison of BMI > 35 and <35 kg/m2. J Gastrointest Surg. 2008;12(5):945–52.</p>

- 115. Lee WJ, Wang W, Lee YC, et al. Laparoscopic mini-gastric bypass: experience with tailored bypass limb according to body weight. Obes Surg. 2008;18(3):294–9.
- Noun R, Riachi E, Zeidan S, et al. Mini-gastric bypass by minilaparotomy: a cost-effective alternative in the laparoscopic era. Obes Surg. 2007;17(11):1482–6.
- 117. Mahawar KK, Jennings N, Brown J, Gupta A, Balupuri S, Small PK. "Mini" gastric bypass: systematic review of a controversial procedure. Obes Surg. 2003.
- 118. Kuzmak LI, Yap IS, McGuire L, et al. Surgery for morbid obesity. Using an inflatable gastric band. AORN J. 1990;51(5):1307–24. Erratum in: AORN J 1990; 51(6): 1573.
- 119. de Csepel J, Burpee S, Jossart G, et al. Laparoscopic biliopancreatic diversion with a duodenal switch for morbid obesity: a feasibility study in pigs. J Laparoendosc Adv Surg Tech A. 2001;11(2):79–83.
- 120. Milone L, Strong V, Gagner M. Laparoscopic sleeve gastrectomy is superior to endoscopic intragastric balloon as a first stage procedure for super-obese patients (BMI > or =50). Obes Surg. 2005;15(5):612–7.
- 121. Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2008. Obes Surg. 2009;19(12):1605–11.
- 122. Lomanto D, Lee WJ, Goel R, et al. Bariatric surgery in Asia in the last 5 years (2005–2009). Obes Surg. 2012;22(3):502–6. Erratum in: Obes Surg. 2012 Feb;22(2):345. Fah, Chin Kin [corrected to Chin, Kin-Fah].
- Himpens J, Cadière GB, Bazi M, et al. Long-term outcomes of laparoscopic adjustable gastric banding. Arch Surg. 2011;146(7):802–7.
- 124. Stroh C, Hohmann U, Schramm H, et al. Fourteen-year long-term results after gastric banding. J Obes. 2011;2011:128451.
- Weiner RA, Theodoridou S, Weiner S. Failure of laparoscopic sleeve gastrectomy—further procedure? Obes Facts. 2011;4 Suppl 1:42–6.
- Gautier T, Sarcher T, Contival N, et al. Indications and mid-term results of conversion from sleeve gastrectomy to Roux-en-Y gastric bypass. Obes Surg. 2013;23(2):212–5.
- 127. Chiu S, Birch DW, Shi X, et al. Effect of sleeve gastreetomy on gastroesophageal reflux disease: a systematic review. Surg Obes Relat Dis. 2011;7(4):510–5.
- Azagury DE, Varban O, Tavakkolizadeh A, et al. Does laparoscopic gastric banding create hiatal hernias? Surg Obes Relat Dis. 2013;9(1):48–52.