# Bariatric Surgery for the Treatment of Type 2 Diabetes


**From:** http://www.purplemedicalblog.com/2012/11/can-bariatric-weight-loss-surgery.html

## Internal Medicine Grand Rounds – June 14, 2013

**Ildiko Lingvay, MD, MPH, MSCS**

Department of Internal Medicine – Division of Endocrinology

Department of Clinical Sciences

UT Southwestern Medical Center

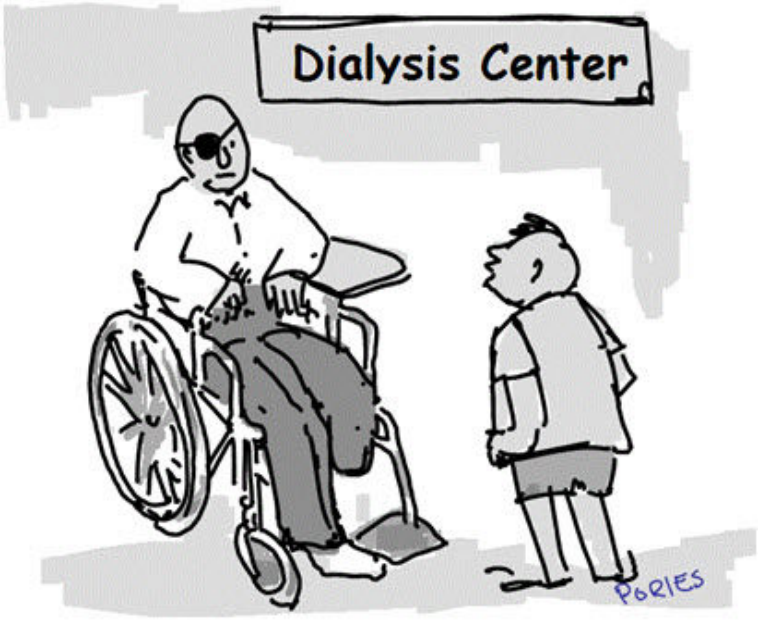
*This is to acknowledge that Dr. Ildiko Lingvay has disclosed that she does have financial interests or other relationships with commercial concerns related directly or indirectly to this program. Dr. Lingvay will be discussing off-label uses in her presentation*.

Ildiko Lingvay, MD, MPH, MSCS is an Assistant Professor in the Departments of Internal Medicine (Division of Endocrinology) and Clinical Sciences. Dr. Lingvay is a graduate of the inaugural class of the Clinical Scholars Program at UT Southwestern. Her clinical research is focused on the pathophysiology of type 2 diabetes and she has a particular interest in disease-modifying treatment interventions which can change the course of this progressive, chronic disease. Dr. Lingvay recently completed her NIH K23 Career Development Award for the evaluation of the role of pancreatic triglyceride accumulation in beta-cell dysfunction.

The purpose of this presentation is to review (1) the mechanisms leading to improvement in diabetes control following bariatric surgery, (2) the metabolic effectiveness of various bariatric procedures, (3) the current indications for bariatric surgery in patients with type 2 diabetes, (4) the proposed expanded indications for bariatric surgery, and (5) risks associated with bariatric surgery.

Educational Objectives:

1. Learn the various mechanisms implicated in the diabetes improvement following bariatric surgery;
2. Quantify the metabolic effects following different types of bariatric surgery;
3. Identify best candidates for bariatric surgery;
4. Evaluate the risk-benefit ratio of bariatric surgery.




*"Grandpa, why didn't you get a gastric bypass?"*

*Acknowledgements:* I would like to thank Dr. Perry Bickel for his relentless support, guidance, and leadership and Dr. Naim Maalouf for his helpful insight, advice, editorial help, and his friendship.

## 1. The Diabetes Epidemic

More than 8.3% of the US population is estimated to have diabetes, a chronic disease which carries a high morbidity and mortality burden, as well as a huge economic impact. The cost of medical expenditures in 2012 among people diagnosed with diabetes were 2.3 times higher than for people without diabetes (after adjusting for population age and sex differences)[1]. The estimated cost of diagnosed diabetes has increased by 41% from 2007 to 2012 alone, with the majority of this increase being accounted for by an increase in the cost of hospitalizations and care of diabetes-related complications[1].

The prevalence of diabetes in the US nearly tripled since 1980[2]. It is estimated that 25.8 million people in the US had diabetes in 2011. Unfortunately this trend is expected to continue, in which case by the year 2050 as many as 1 in 3 people in the US will have diabetes[3]. With such rapid growth in both prevalence of disease and disease-related cost of care, it is of utmost importance to intensify our efforts to find effective and safe interventions for the prevention, treatment, and ultimately cure of this disease. Successful interventions would also have to be implementable at large scale in order to reverse the devastating impact this disease already has in our society.

## 2. Pathophysiology and the Natural History of Type 2 Diabetes

Type 2 diabetes is the most common and fastest growing form of diabetes (90-95% of all cases – more than 23 million people in the US) and thus the primary driver behind the diabetes epidemic. There are two main pathophysiologic abnormalities in type 2 diabetes: (1) whole body insulin resistance and (2) inadequate beta-cell function, which cannot compensate for the prevailing insulin resistance.

Obesity is a major risk factor for diabetes, as it not only promotes end-organ insulin resistance, but is also thought to contribute to beta-cell failure through lipotoxicity[4]. It seems thus logical that a successful and durable intervention against diabetes would also target obesity.

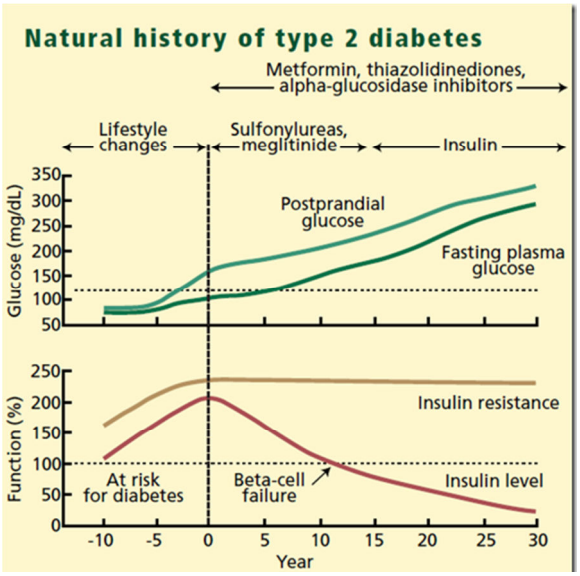



**Figure 1. The pathophysiological events throughout the natural history of type 2 diabetes.**

The natural course of type 2 diabetes is hallmarked by a progressive decline in beta-cell

function over time (Figure 1). Very early in the course of the disease there is hyperinsulinemia as beta-cells attempt to compensate for insulin resistance by increasing insulin production through increasing beta-cell function as well as mass. Over time beta-cell function and mass decline, leading to relative insulin deficiency which gradually progresses towards absolute insulin deficiency.

Current pharmacologic interventions for diabetes target hyperglycemia, but the durability of our available treatment interventions is limited[5], with disease stabilization, reversal, or cure all remaining aspirational goals. The limited durability is due to the fact that none of our pharmacological interventions are capable of changing the natural progressive course of the disease.

Though cure of diabetes is a goal of current research, investigators debate whether this term is even applicable for a chronic disease whose defining criteria is a continuous variable that can vary with transient physiologic or pathologic states. The current consensus is to use the term "remission" when referring to diabetes, and this has been defined as "partial" if the glycemic parameters (HbA1c and glucose level) are below diabetes diagnostic thresholds in the absence of active treatment for one year, and "complete" if these parameters are within normal range[6]. Unfortunately these definitions have not been uniformly adopted and clinical trials have used various arbitrary definitions of diabetes remission, thereby making comparison among trials very difficult.

**3. Effect of Bariatric Surgery on Type 2 Diabetes**

Bariatric surgery is the only known intervention which can effectively induce remission of diabetes. A large meta-analysis reported diabetes remission rates as high as 76.8% across all types of surgeries, ranging from 47.8% remission with the adjustable gastric banding (AGB) procedure, 83.7% with Roux-en-Y Gastric By-pass (RYGB) procedure, to 97.9% with biliopancreatic diversion (BPD)[7]. These impressive results have recently been replicated in two randomized controlled trials that only enrolled patients with type 2 diabetes. The first one, performed at Cleveland Clinic, randomized patients to usual diabetes care, RYGB, or sleeve gastrectomy (SG). After one year of follow-up, remission of diabetes (defined as HbA1c<6% with no medications) occurred in 0%, 42%, and 27%, respectively in the three groups[8]. In the second study, conducted in Italy, patients were randomized to usual care, RYGB, or BPD. After two years of follow-up, diabetes remission (defined as fasting glucose of <100 mg/dl and HbA1c<6.5% for one year in the absence of pharmacologic treatment) was achieved in 0% of patients in the medical group, 75% in the RYGB group, and 95% in the BPD group[9]. Additionally, the vast majority of patients who have not achieved remission have improvement in diabetes.

These high remission rates suggest that bariatric surgery exerts a significant disease modifying effect, reversing the natural course of type 2 diabetes, thus setting it apart from all pharmacological interventions currently in use for the treatment of diabetes.

**4. Mechanisms of Diabetes Remission after Bariatric Surgery**

The mechanisms through which bariatric surgery induces diabetes remission have not been completely elucidated. Weight loss, the logical candidate for this effect, starts within days, but significant weight loss (traditionally viewed as 5-10% of body weight) is usually achieved weeks to months after surgery. The vast majority of the improvement in diabetes is observed within the first few weeks after surgery, thus long before significant weight loss occurs. This observation led to the conclusion that other weight-independent mechanisms must be at play following surgery[10]. Postulated mechanisms include: (1) the hindgut hypothesis, which states that the delivery of undigested food to the distal small intestine stimulates the secretion of

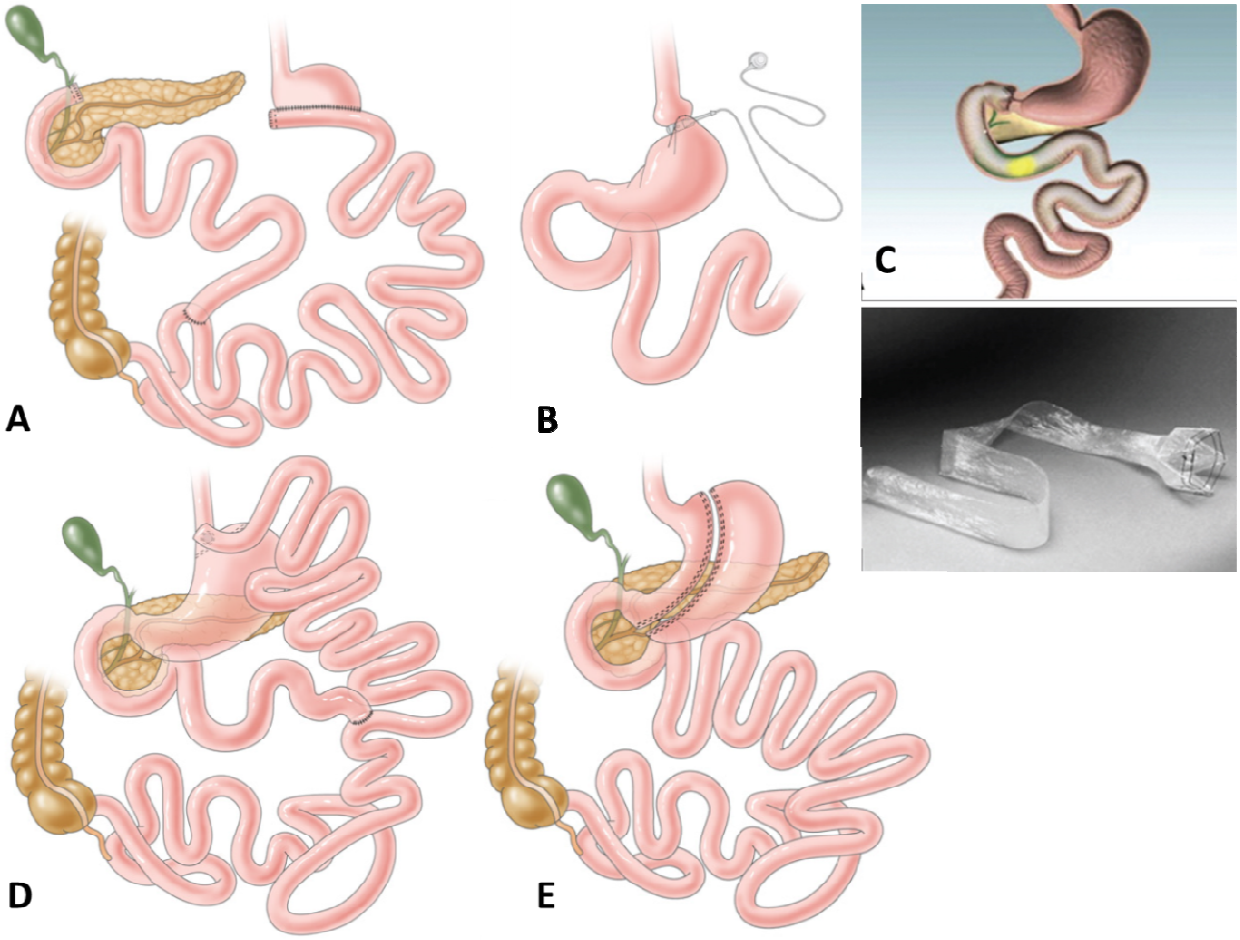



**Figure 2. Types of bariatric procedures. A. Biliopancreatic diversion (BPD); B. Adjustable gastric banding (AGB); C. Endoluminal intestinal liner; D. Roux-en-Y gastric by-pass (RYGB); E. Sleeve gastrectomy (SG).**

anti-diabetogenic hormones. Of these, glucagon-like peptide 1 (GLP-1), secreted by the L-cells, has been consistently demonstrated to increase immediately following surgery to have an exaggerated postprandial surge; b) the foregut hypothesis, which states that the exclusion of the proximal small intestine from nutrient flow leads to downregulation of yet unidentified diabetogenic signal(s); (c) impaired secretion of gastric hormones, including ghrelin, which control appetite; (d) changes in intestinal nutrient-sensing mechanisms regulating insulin sensitivity; (e) bile acid perturbations.

Numerous studies exist to support these weight-independent mechanisms, yet it is interesting to note that the strongest predictors of diabetes remission after bariatric surgery are greater weight loss, longer duration of diabetes, and lower insulin dose prior to surgery[11,12]. Furthermore, the weight-loss effectiveness of various bariatric procedures is closely related to their effectiveness at diabetes remission[7]. Rate of diabetes relapse after surgery is also predicted by weight, in this case weight regain[13]. Thus perhaps calorie restriction and weight-loss should be revisited as contributors to diabetes remission following bariatric surgery.

A good insight into the relative contribution of the various mechanisms to diabetes remission could be glimpsed from comparing and contrasting the different surgical procedures.

*Biliopancreatic diversion:* By far the most effective surgery, with diabetes remission rates exceeding 97%[7], is the BPD. This is the most complex bariatric procedure, involving the following intestinal rearrangements (Figure 2A): (1) creation of a small gastric pouch (200-500 cc) through a horizontal gastrectomy; (2) anastomosis of the new gastric outlet to the small intestine 250 cm from the ileocecal valve (alimentary channel); (3) resection of the main stomach body and pylorus; (4) anastomosis of the "biliopancreatic limb" (duodenum and jejunum) to the distal ileum (50 cm from the ileocecal valve), which results in (5) a short common channel. This procedure harnesses all mechanisms noted above, therefore explaining the extraordinarily high rate of diabetes remission following BPD. In order to separate the effect and relative contribution of each of the components employed in this surgery, we should evaluate results obtained with "simpler" procedures, which only harness one of the above mechanisms.

*Adjustable gastric banding:* The AGB procedure involves the placement of a silicone ring around the fundus of the stomach, creating a small gastric pouch (Figure 2B). Following the initial procedure patients undergo on average 6 adjustments to the size of the silicone ring to achieve optimal restriction. Its effect is thought to be strictly due to mechanical restriction which limits the amount of food that can be ingested at a given time. Patients limit their total daily caloric ingestion, an action reinforced by the negative effects of non-compliance. No significant hormonal changes have been noted to occur following this procedure. Among all bariatric surgeries it is the least effective at diabetes remission, has the highest reoperation and

failure rate, reasons for which its popularity is slowly decreasing. Nevertheless, its effects are far superior to medical treatment. Diabetes remission rate of 47.8%, and remission/improvement in 80.2% was noted in the largest meta-analysis[7]. The only long-term randomized controlled trial in patients with type 2 diabetes reported remission rates (defined as fasting glucose <126 mg/dl, HbA1c<6.2%, and no medication) of 73% in the AGB group compared with 13% in the medically treated group[14]. This study only enrolled patients with type 2 diabetes within 2 years of diagnosis, which is the likely explanation for the higher remission rates compared with the other reports. There are no long-term randomized trials directly comparing diabetes remission after AGB versus other bariatric procedures, but the existing body of literature indicates that the rate of diabetes remission after AGB is at least half of what is seen with RYGB. Since the only mechanisms harnessed by AGB is "mechanical restriction", this suggest that caloric restriction makes a relatively large contribution (approximately half of the effect seen with the more "potent" surgeries) to diabetes remission.

*Endoluminal intestinal liner:* The endo-luminal intestinal liner is an endoscopically placed device which is implanted in the duodenum (Figure 2C). A 60 cm impermeable liner is deployed, which prevents the contact of the food with the intestinal digestive juices, and delivers undigested food to the distal intestine. This procedure harnesses the hindgut and foregut hypothesis, having no restrictive component per se, nor any significant macronutrient malabsoption. This device is approved for a weight loss indication in Europe, Australia, and Israel, and is currently undergoing Phase 3 studies in the US - solely for a type 2 diabetes indication. An open-label randomized controlled trial of 6 months duration showed that patients with type 2 diabetes implanted with the device had a HbA1c lowering of 1.3% from a baseline of 8.4%, compared with the diet group which had a HbA1c lowering of 0.8%[15]. The device group lost 9.3 kg, versus 5.7 kg in the diet group. A single-group study evaluated 17 patients with diabetes during a 6 months implantation period[16]. Patients had a 1.4% lowering in their HbA1c (from baseline of 8.4%) and lost 12.7kg. The glycemic improvement occurred within one week after the procedure, with no further improvement thereafter in the glucose area under the curve after a standard meal test. There was also a significant post-prandial increase in GLP-1, and significant decrease in GIP and glucagon secretion after the standard meal. Interestingly, the GLP-1 levels returned promptly to baseline when studied 1 week after device explantation, indicating that the GLP-1 effect is directly induced by the device. These hormonal changes are thought to be the main determinant of the anti-diabetic effect seen with this device. There are no studies evaluating the effect of this procedure on caloric intake.

The device is only approved for up to a 1-year implantation period, and published studies only report data up to 6 months, therefore it is impossible to assess a true diabetes remission rate. A head-to-head comparison of the endo-luminal intestinal liner with any other bariatric procedure is not currently planned. While two completely distinct studies cannot be

compared head to head, it is interesting to note that in the Australian AGB study (mentioned above[14]) there was a 1.8% decrease in HbA1c (from baseline of 7.8%) and a 0.38% reduction in the diet group, contrasting with the EndoBarrier study which had a 1.3% HbA1c reduction (from a higher baseline of 8.4%) in the device group and 0.8% in the diet group. These contrasts between the two studies suggest – albeit indirectly – a much stronger anti-diabetic effect with the AGB versus the endo-luminal intestinal liner, which supports the conclusion that caloric restriction has a greater overall contribution to diabetes remission as compared to the surgery-induced changes in the incretin hormones.

*Roux-en-Y gastric by-pass:* RYGB is, conceptually speaking, a combination of the above two procedures. It involves the creation of a small gastric pouch (30 cc, approximately the same size as the gastric pouch created by the AGB) which is connected to a distal jejunum limb which was transected at approximately 30 cm from the ligament of Treitz. The rest of the stomach, duodenum, and first part of the jejunum are excluded from the alimentary channel and connect with it 75 to 150 cm from the ileocecal valve. Ingested food by-passes most of the stomach, duodenum and first part of the stomach – similarly to the endo-luminal intestinal liner. Diabetes remission rates of 83.8% are reported following RYGB, and improvement or remission occurs in 90.6% of patients[7]. Based on results seen with AGB and the endo-luminal intestinal liner, it is tempting to assume that just over half of the overall effect of RYGB on diabetes remission is due to restriction of caloric intake, while the rest is primarily due to hormonal changes induced by the intestinal by-pass. RYGB induces a major rise in postprandial GLP-1 level (more pronounced than the endo-luminal intestinal liner), while its effect on other incretin hormones are much smaller in magnitude (GIP, PYY, ghrelin) or counterintuitive (glucagon increases after surgery, an effect which might promote hyperglycemia)[17]. The rapid, consistent, and significant increase in GLP-1 induced by RYGB has been linked by many researchers to the rapid improvement in glycemia noted immediately after surgery. Yet this connection needs to be scrutinized for the following reasons: (1) the GLP-1 plasma levels achieved after RYGB[18] are supra-physiological but comparable to those seen in patients treated with GLP-1 agonists (Victoza and Byetta package insert) or patients undergoing insertion of an endoluminal intestinal liner[16], yet the glycemic effects are much less with the latter two, (2) there is no difference in the post-meal glucose response between AGB and RYGB if the caloric intake between groups is identical, despite a significant difference in GLP-1 between groups[18]. These findings suggest that the post-RYGB rise in GLP-1 is a secondary contributor to diabetes remission, and other mechanisms must predominate.

BPD is the most effective antidiabetes procedure, with remission rates above 97%[7]. This procedure entails several components, including gastric restriction and intestinal by-pass as seen with the RYGB, but also resection of the gastric body and a much shorter common alimentary channel. The latter induces significant malabsorption, with contributes to the caloric

deficit, weight loss, and diabetes remission, and is likely responsible for the difference in the rate of diabetes remission between RYGB and BPD. The extent of the contribution, if any, of the partial gastric resection to the antidiabetic effect of BPD is not known. It is intriguing that a newer bariatric procedure, SG, only employs a partial gastrectomy.

*Sleeve gastrectomy:* The SG, despite its simplicity, boasts metabolic efficacy comparable to that of RYGB. In this procedure the stomach is divided length-wise to create a sleeve-shaped conduit, while removing up to 75% of the stomach (fundus and body). The procedure was initially intended as a first step risk-reducing intervention in patients deemed too high-risk for more complex procedures like RYGB or BPD. Its popularity soared when its effectiveness was appreciated. Mechanistically this procedure remains a mystery. There is no mechanical restriction, no malabsorbtion, nor an intestinal by-pass, yet GLP-1 and PYY increase to levels similar to those seen in RYGB (Figure 3)[19]. These findings suggests the presence of additional mediator(s), perhaps secreted in the stomach, which are responsible for the stimulation of the incretin hormones, an effect traditionally attributed to the hindgut/foregut hypothesis.

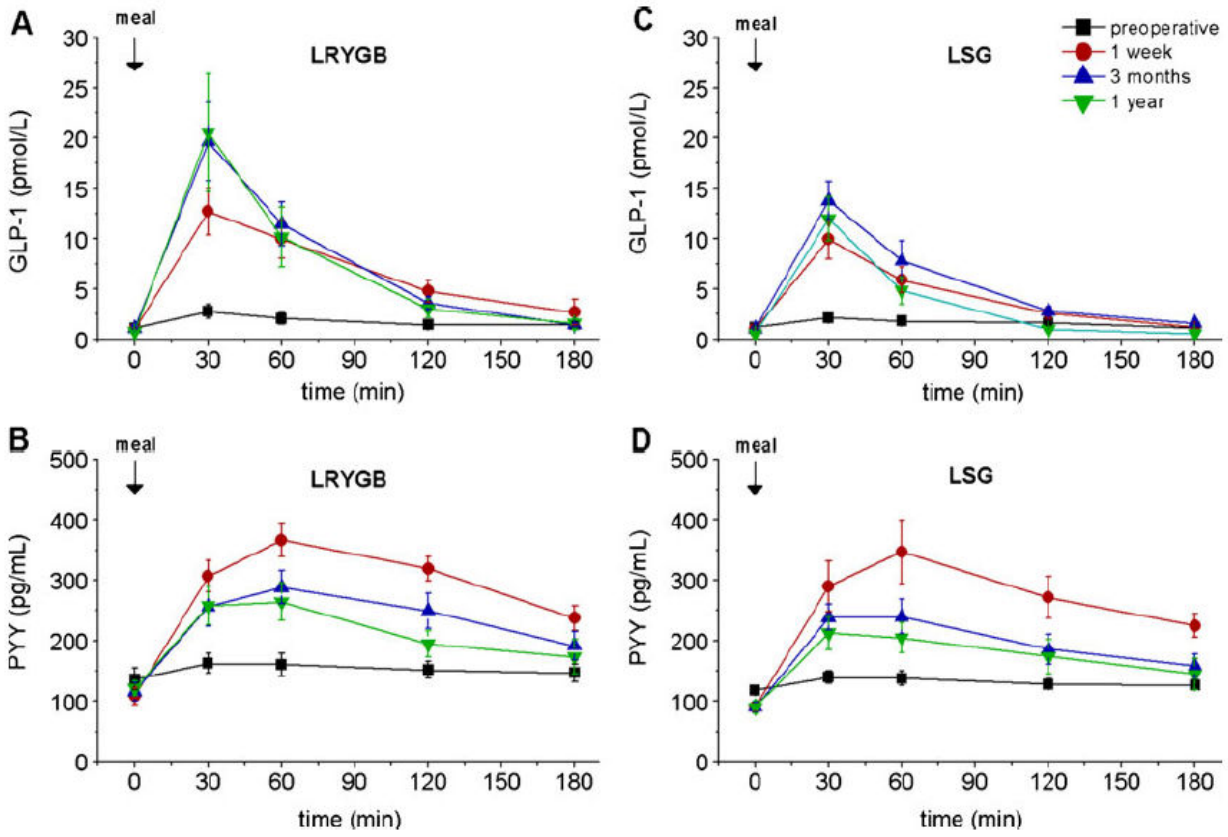



**Figure 3. Randomized controlled trial comparing the mechanisms of action of laparoscopic Roux-en-Y gastric by-pass (LRYGB) versus laparoscopic sleeve gastrectomy (LSG) in non-diabetic patients (Peterli, 2012). Results show comparable effects of the two procedures on incretin hormones, including GLP-1 and PYY.**

The mechanisms mentioned above – caloric restriction, hingut/foregut hypothesis, malabsorption - presumably make the biggest contribution to diabetes remission and have the best supporting data. It is important to acknowledge that the biology of these surgeries is far more complex and includes many other changes which are beyond the scope of this review. The surgical technique-based approach used above purposefully simplifies the changes which occur following bariatric surgery in order to illustrate the main mechanisms at play and highlight the big relative contribution of caloric restriction, which has traditionally been ignored or discarded as a mechanism for diabetes control.

In conclusion, several mechanisms contribute to the anti-diabetic effects seen with bariatric surgery, of which caloric restriction makes the biggest relative contribution (especially in the immediate postoperative period), followed by alterations in the intestinal hormone secretion (of which GLP-1 has the biggest increase), and lastly malabsoption (Figure 4). The mechanisms through which SG exerts its anti-diabetic effect remain elusive.

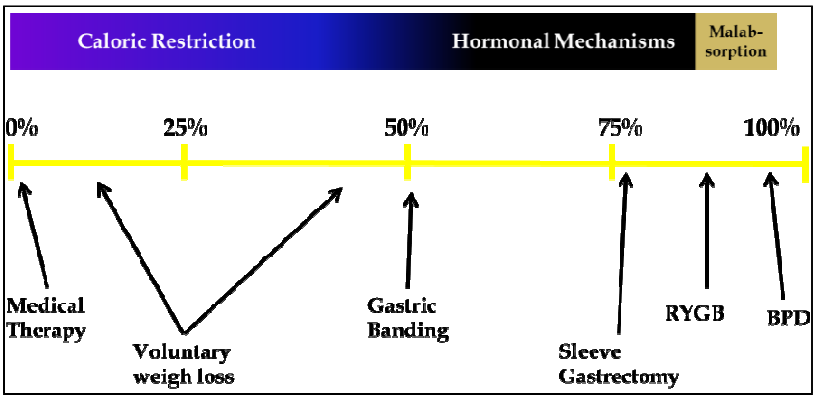



**Figure 4. Illustration of the relative contribution of various mechanisms to the anti-diabetic effect of bariatric surgery, as illustrated by the differential effectiveness of the various types of surgery. RYGB – Roux-en-Y gastric by-pass; BPD – biliopancreatic diversion.**

## 5. The Effect of Caloric Restriction on Type 2 Diabetes

To further explore the concept that caloric restriction is the main mediator of diabetes remission following bariatric surgery it is important to review the effects of caloric restriction in the absence of bariatric surgery. First, it is notable that the typical post RYGB or BPD diet is very restrictive. Patients consume a non-calorie containing liquid diet for a few days, followed by a very slow progression to a full liquid diet for two weeks. Afterwards patients are advised to gradually progress to a soft mechanical diet and eventually to a regular low calorie diet. A typical caloric intake in the first 2 weeks after surgery is approximately 500 kcal/day, and the recommendations are to maintain a daily caloric intake under 1,000 kcal thereafter. In contrast, progression to full diet is much faster after AGB, and a 1,000-1,500 kcal/day diet is recommended afterwards. To contrast the effect of caloric restriction alone on glycemic control, we reviewed studies which employed comparable caloric restrictions (fewer than 1000 kcal/day).

Taylor et all studied patients with type 2 diabetes who consumed 600 kcal/day for 8 weeks[20]. A gradual weight loss of 15 kg occurred. Interestingly, normalization of glycemia occurred within a week (Figure 5A), akin to bariatric surgery, and was maintained during the

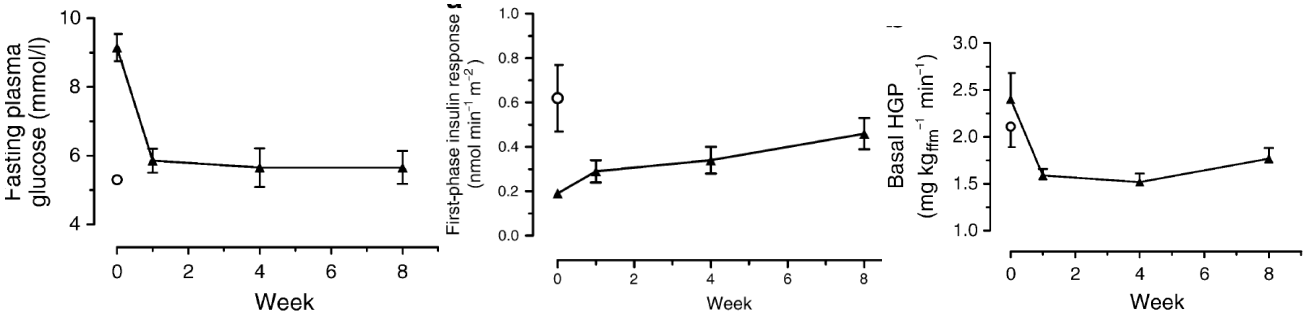



**Figure 5. Effect of 8-week treatment with a low calorie diet (600 kcal/day) on glycemic parameters. Open circles represent data from an age-, sex-, and weight-matched non-diabetic group. HGP - hepatic glucose output.**

entire duration of the study. A similar pattern of improvement was noted in basal hepatic glucose output (Figure 5C), which was attributed to a rapid decrease in hepatic triglyceride content. Furthermore, a slow persistent improvement in first phase insulin response (Figure 5B) was noted over the 8-week study duration, which was associated with a similar pattern reduction in pancreatic triglyceride content. The study concluded that caloric restriction alone can lead to significant changes in glycemia, an effect possibly mediated though a decrease in ectopic fat deposition and associated lipotoxicity.

Kelly et all studied patients with type 2 diabetes after 3 different periods of caloric restriction: first a 1 week of 800 kcal/day, followed by 2 months of 400 kcal/day, and a final 1 week of 800 kcal/day restriction[21]. They noted that approximately half of the total improvement in fasting glucose, hepatic glucose production, and insulin sensitivity occurred within a week of treatment, while fasting insulin secretion had a gradual, steady improvement over the 4-month total study duration. A similar rapid improvement in glycemia – immediate onset and majority of total effect occurring in the first 7-10 days – was also observed by Henry et al. using a 330 kcal/day intervention[22]. Evidence is strong that caloric restriction (below 1,000 kcal/day) leads to rapid improvement in glycemia, a fact well known to physicians several decades ago when such therapy was commonly employed for treatment of uncontrolled type 2 diabetes, usually during inpatient observation. These studies support the role of caloric restriction in glycemic control, but a head-to-head comparison with bariatric surgery is important in order to separate the role of caloric restriction from other bariatric surgery-induced changes.

Our group explored the relative contribution of caloric restriction versus other surgery-specific mechanisms on glycemic control in patients with type 2 diabetes[23]. The same patients underwent (1) a one-week period of treatment with the post-bariatric diet, followed by (2) a

several months-long period of re-equilibration, and finally (3) RYGB surgery along with a perfectly matched diet to the first study period. The two treatment periods were identical in all aspects (diet, activity level, fluid intake) except the actual surgery which was performed on day #2 (Figure 6). Detailed metabolic evaluation was undertaken before and after each one-week active intervention period. We found that the average daily glucose levels were in fact lower in the diet-only group due to an initial transient worsening in glycemia in the first 3 days postoperatively, presumably due to surgical stress (Figure 6). The improvement in the glucose excursion after a Mixed Meal (measuring post-challenge glucose tolerance) was identical during the two intervention periods (diet versus RYGB-plus-diet) (Figures 7). Our findings also support the hypothesis that caloric restriction is the major determinant of

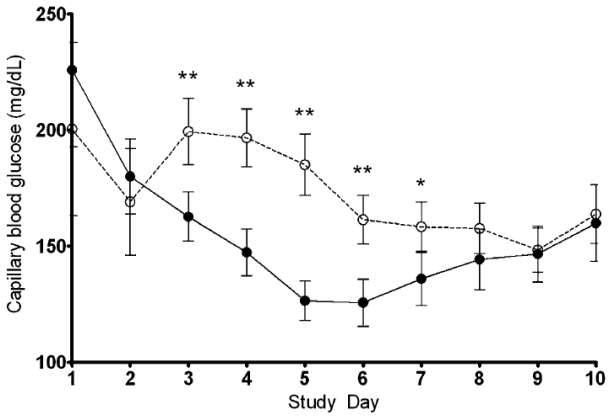



**Figure 6. The average daily glucose level during a diet-only intervention (solid circles and solid line) compared to RYGB plus matched-diet (open circles and dotted line)**

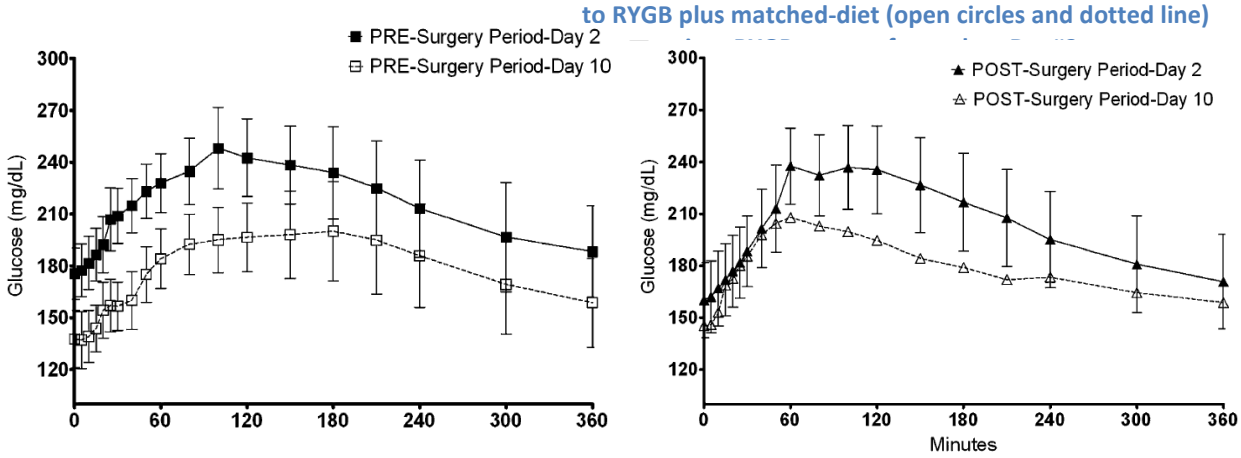



**Figure 7. Glucose excursion during a Mixed Meal Challenge test before (solid lines) and after (dotted lines) a diet-only intervention (Panel A) and RYGB with matched-diet (Panel B).**

diabetes remission - at least in the immediate post-operative period.

The same question was addressed by Bradley et al. using a different study design[18]. They compared metabolic changes in non-diabetic patients undergoing AGB (primarily a restrictive procedure) and RYGB (complex procedure involving gastric and intestinal by-pass) – while controlling for weight loss. Both groups were carefully studied when they reached a 20% weight loss, which was at 22 weeks for the AGB group and 16 weeks for the RYGB group. Their findings show that despite differences in post-prandial GLP-1 secretion, the improvement in oral glucose tolerance, insulin secretion, insulin sensitivity, and hepatic glucose output were all identical in

the two groups. They concluded that marked weight loss itself is primarily responsible for the metabolic effects of RYGB and AGB.

The concept of caloric restriction/weight loss as the main determinant of glycemic changes after bariatric surgery could also be tested in a model of weight re-gain after surgery. Understandably there are no interventional studies of such nature, but there is indirect supportive evidence from patients who have band failure or regain their weight for any reasons[13,24].

A key discrepancy remains unresolved: if caloric restriction per se results in comparable improvements in glycemia , why has the medical intervention arm in all randomized studies fared so poorly when compared to bariatric surgery[8,9,14,25]? The answer is the need to control appetite in order to successfully implement and sustain such major caloric restrictions. Compliance with any caloric restriction, especially so restrictions to below 1000 kcal/day, is notoriously poor and rarely sustained long term. Hence most diet studies are of short duration and many take place in a strictly supervised inpatient setting to ensure compliance. On the other hand, these dietary restrictions are readily adhered to by most patients undergoing bariatric surgery, strongly suggesting that in fact the main mechanism of action of bariatric surgery is in its ability to control caloric intake, which in turn exerts its positive effects on glycemic control (Figure 8). How does bariatric surgery control appetite is the key unanswered question, and the key to unlock a non-surgical population-based effective anti-diabetic and anti-obesity intervention. Until this puzzle is solved, bariatric surgery will continue to be undeniably the most effective disease-modifying treatment for type 2 diabetes and obesity.

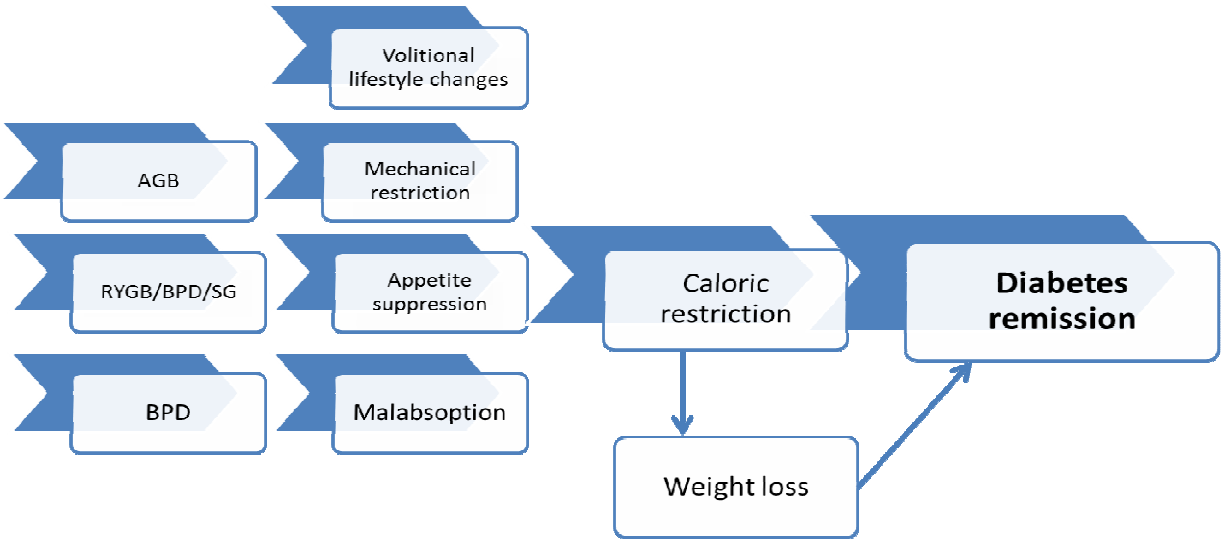



**Figure 8. Conceptual framework of the main diabetes remission pathway following bariatric surgery. AGB – adjustable gastric banding; RYGB – Roux-en-Y gastric by-pass; BPD – bliliopancreatic diversion; SG – sleeve gastrectomy.**

## 6. Patient Selection Criteria for Bariatric Surgery

Patient selection criteria are driven by payers and are based upon the results of an NIH consensus statement developed in 1991[26]. These include: (1) patients with a BMI>40kg/$m^2$ or (2) patients with BMI 35-40 kg/$m^2$ who have additional metabolic of physical obesity-related complications, and (3) no surgery recommended for children and adolescents. While a lot more information is now available about the effectiveness and complications of various bariatric procedures, over 20 years later these guidelines have not been updated and are still the ones primarily followed by insurance companies. On the other hand, these criteria still correctly identify the patient group whose risk to benefit ratio is the highest and thus likely to have the best long-term outcomes.

The American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic and Bariatric Surgery have developed in 2013 updated guidelines in an effort to inform the medical community and other stakeholders about the wealth of new information available regarding the effectiveness and safety of the bariatric procedures currently in use. The new guidelines advocate the expansion of eligibility for bariatric surgery to several specific populations, including mild obesity (BMI 30-35 kg/$m^2$) if the patient has type 2 diabetes or metabolic syndrome, adolescents who meet all medical and psychological criteria, as well as older patients (>45 years) who were previously considered a high-risk group. These guidelines were reported shortly after the FDA has granted an indication for the AGB for patients with a BMI 30-35 kg/$m^2$ and metabolic complications. This FDA decision was surrounded by much controversy due to the limited long-term data in this group, yet it was promptly adopted by the International Diabetes Federation.

Studies evaluating the effect of bariatric surgery in mild obesity (BMI 30-35 kg/$m^2$) report significant weight loss (>70% of the excess body weight), with up to 40% of patients attaining normal weight status (BMI<25 kg/$m^2$) after surgery[27-29]. Most studies that enrolled patients with BMI of 30-35 kg/$m^2$ and type 2 diabetes report remission rates below 50%[8,30], except one study which had an outstanding 88% remission rate[31].

A very interesting study, conducted in Asia, only enrolled patients with uncontrolled type 2 diabetes (HbA1c 9.1%) and a BMI<30 kg/$m^2$ (average BMI 26 kg/$m^2$). Patients lost an average of 14% of their body weight at one year after RYGB, while their HbA1c decreased to 6.8%[12]. Diabetes remission was predicted by a shorter duration of diabetes and higher C-peptide (indicating milder disease), but most importantly by baseline BMI and % weight loss. In the group with a baseline BMI in the lowest quartile (<24 kg/m2) there was an 18.5% rate of diabetes remission, contrasting with the group with the highest baseline BMI (28.6-30 kg/$m^2$)

who had a 52% rate of diabetes remission. These data suggest that the effectiveness of bariatric surgery diminishes progressively with lower baseline BMI. Still, even normal weight patients (BMI<25 kg/$m^2$) with type 2 diabetes experience significantly better results with bariatric surgery than would be anticipated with medical therapy.

Should bariatric surgery be offered to all patients with type 2 diabetes, regardless of BMI? The benefit-risk ratio diminishes rapidly as the baseline BMI decreases. Perhaps such options should be considered in selected patients, who are most committed to lifestyle changes, have a short duration of diabetes, and a higher beta-cell function reserve, in order to maximize the remission rate.

**7. Bariatric Surgery Benefits beyond Glycemia**

Bariatric surgery has proven benefits beyond glycemic control, which should be taken into account when the risk-benefit ratio for each individual is determined. These include improvements in (1) other metabolic abnormalities like hyperlipidemia (especially hypertriglyceridemia), hypertension, gout, non-alcoholic fatty liver disease, and polycystic ovarian disease, (2) abnormalities physically induced by the excess weight, such as osteoarthritis, sleep apnea, venous stasis disease, stress urinary incontinence, gastroesophagial reflux disease, and (3) other obesity-related abnormalities like migraine headaches, pseudotumor cerebri, asthma, and depression.

Improvements in weight, glycemia, and other metabolic parameters represent surrogate markers of overall morbidity, and therefore to fully appreciate the long term effect of bariatric surgery it is important to study its effect on hard end-points like micro- and macrovascular complications and mortality. It is reassuring that several studies have now shown reduction in all cause and cardiovascular mortality[32-34], as well as significant reductions in major macrovascular[35,36] and microvascular[37] diabetes related events.

The cost effectiveness analysis of bariatric surgery procedures has been scrutinized as there is a substantial upfront investment and treatment cost related to surgical complications. Yet this investment was shown to be balanced out as early as 5 years, especially in patients with diabetes, by savings related to medication costs, prevention of complications, and decrease in mortality (Figure 9)[38-40].

| | Total costs* | Remaining life-years | QALYs* | Cost-effectiveness ratio ($/QALY)† |
|---|---|---|---|---|
| Patients with newly diagnosed diabetes | | | | |
| No surgery (standard care) | $71,130 | 21.62 | 9.55 | |
| Bypass surgery | $86,665 | 23.34 | 11.76 | |
| Incremental (vs. no surgery) | $15,536 | 1.72 | 2.21 | $7,000 |
| Banding surgery | $89,029 | 22.76 | 11.12 | |
| Incremental (vs. no surgery) | $17,900 | 1.14 | 1.57 | $11,000 |
| Patients with established diabetes | | | | |
| No surgery | $79,618 | 16.86 | 7.68 | |
| Bypass surgery | $99,944 | 17.95 | 9.38 | |
| Incremental (vs. no surgery) | $20,326 | 1.09 | 1.70 | $12,000 |
| Banding surgery | $96,921 | 17.80 | 9.02 | |
| Incremental (vs. no surgery) | $17,304 | 0.94 | 1.34 | $13,000 |

*Costs and QALYs are discounted at a 3% annual rate. †Cost-effectiveness ratios are rounded to the nearest $1,000/QALY.

**Figure 9. Cost-effectiveness ratio of bariatric surgery compared to no surgery (from Hoerger, 2010). QALY – quality adjusted life year**

## 8. Limitations of Bariatric Surgery

If the benefits of bariatric surgery are so encompassing, and even extending to certain populations with a lower BMI (overweight and mild obesity), then why is bariatric surgery not offered to everyone?

First of all, bariatric procedures carry associated risks, which need to be carefully balanced against the anticipated benefits. The risks associated with bariatric surgery can be divided into perioperative risks and consequences of nutritional deficiencies.

Perioperative risks occur in approximately 10%[41,42] of patients and range from surgical complications, such as leaks, bleeding, intestinal obstruction, and wound infections, to medical complications like deep venous thrombosis and renal failure. Up to 2.5% of patients require a reoperation, most commonly seen after RYGB procedure. Mortality rates are low overall at 0.1% (Figure 9). These reports only consider events which occur within 30 days of surgery, and do not take into consideration later complications, of which the most common are marginal ulcer, intestinal obstruction, cholelithiasis, and nephrolithiasis. Of the three most commonly performed procedures in the US, AGB, SG, and RYGB, the latter is the most effective but also the one which carries the highest risks.

| Outcome | % (95% CI) Overall (N=15 275) | Laparoscopic Adjustable Gastric Band (n=5380) | Sleeve Gastrectomy (n=854) | Gastric Bypass (n=9041) | *P* Value[a] |
|---|---|---|---|---|---|
| Any complication | 7.3 (6.9-7.7) | 2.3 (1.9-2.7) | 5.9 (1.3-7.4) | 10.3 (9.7-11.0) | <.001 |
| Non–life-threatening | 4.7 (4.4-5.1) | 1.5 (1.2-1.8) | 3.6 (2.5-5.1) | 6.7 (6.2-7.3) | <.001 |
| Potentially life-threatening | 2.3 (2.0-2.5) | 0.78 (0.56-1.1) | 2.2 (1.3-3.5) | 3.1 (2.8-3.5) | <.001 |
| Permanently disabling | 0.21 (0.14-0.30) | 0.04 (0.01-0.13) | 0 | 0.33 (0.22-0.47) | <.001 |
| Fatal | 0.10 (0.6-0.16) | 0.04 (0.01-0.13) | 0 | 0.14 (0.08-0.25) | .09 |
| Combined serious complications[b] | 2.6 (2.3-2.8) | 0.86 (0.61-1.1) | 2.2 (1.2-3.2) | 3.6 (3.2-4.0) | <.001 |
| Surgical site | 5.9 (5.6-6.3) | 1.7 (1.4-2.1) | 3.6 (2.5-5.1) | 8.7 (8.1-9.3) | <.001 |
| Leak/perforation | 0.59 (0.47-0.72) | 0.07 (0.02-0.19) | 0.35 (0.07-1.0) | 0.92 (0.73-1.1) | <.001 |
| Anastomotic leak | 0.49 (0.36-0.64) | 0 | 0 | 0.49 (0.36-0.64) | |
| Perforation/other leak | 0.27 (0.20-0.37) | 0.07 (0.02-0.19) | 0.35 (0.07-1.0) | 0.39 (0.27-0.54) | .002 |
| Obstruction | 1.5 (1.3-1.7) | 0.26 (0.14-0.44) | 0.70 (0.26-1.5) | 2.4 (2.0-2.7) | <.001 |
| Small bowel obstruction | 0.49 (0.38-0.61) | 0 | 0.12 (0.01-0.66) | 0.81 (0.63-1.0) | <.001 |
| Stricture/other obstruction | 1.1 (0.93-1.3) | 0.26 (0.14-4.4) | 0.59 (0.19-1.4) | 1.6 (1.4-1.9) | <.001 |
| Infection | 3.2 (2.9-3.5) | 1.3 (1.0-1.6) | 2.5 (1.5-3.7) | 4.4 (4.0-4.8) | <.001 |
| Abdominal abscess | 0.45 (0.35-0.57) | 0.07 (0.02-0.19) | 0.47 (0.13-1.2) | 0.67 (0.52-0.87) | <.001 |
| Wound complication | 2.7 (2.5-3.0) | 0.84 (0.61-1.1) | 2.2 (1.3-3.5) | 3.9 (3.5-4.3) | <.001 |
| Port site infection | 0.30 (0.32-7.1) | 0.30 (0.32-7.1) | 0 | 0 | |
| Hemorrhage | 1.5 (1.3-1.7) | 0.13 (0.05-0.27) | 0.59 (0.19-1.4) | 2.3 (2.0-2.7) | <.001 |
| Medical complication | 1.5 (1.3-1.7) | 0.58 (0.39-0.82) | 1.4 (0.73-2.4) | 2.1 (1.8-2.4) | <.001 |
| Venous thromboembolism | 0.39 (0.30-0.50) | 0.11 (0.04-0.24) | 0.94 (0.41-1.8) | 0.50 (0.36-0.67) | <.001 |
| Cardiac | 0.10 (0.06-0.16) | 0.04 (0.01-0.13) | 0 | 0.14 (0.08-0.25) | .09 |
| Renal failure | 0.31 (0.23-0.41) | 0.07 (0.02-0.19) | 0 | 0.48 (0.34-0.61) | <.001 |
| Respiratory | 0.99 (0.84-1.2) | 0.35 (0.21-0.55) | 0.47 (0.13-1.2) | 1.4 (1.2-1.7) | <.001 |
| Utilization | | | | | |
| Reoperation | 1.7 (1.5-1.9) | 0.63 (0.44-0.88) | 0.59 (0.19-1.4) | 2.5 (2.2-2.8) | <.001 |
| Readmission | 4.0 (3.7-4.3) | 2.0 (1.6-2.4) | 5.5 (4.1-7.3) | 5.1 (4.6-5.6) | <.001 |
| Transfer | 0.14 (0.09-0.22) | 0 | 0.23 (0.03-0.84) | 0.22 (0.14-0.34) | .002 |
| Emergency department visit | 6.8 (6.4-7.2) | 3.1 (2.7-3.6) | 7.5 (5.8-9.5) | 8.9 (8.4-9.5) | <.001 |

**Figure 10. Rate of perioperative complications by type of surgery (from Brikmeyer, 2010).**

Nutritional deficiencies are specific to procedures which entail an intestinal by-pass, and include malabsobtion of iron, vitamin B12, calcium, vitamin D, and less commonly folic acid, vitamin B6, vitamin A, copper, selenium, and zinc. Routine supplementation and periodic monitoring of iron, B12, calcium, and vitamin D are recommended lifelong for all patients undergoing an intestinal by-pass procedure (RYGB or BPD). Symptom based evaluation is recommended for folic acid, vitamin B6, vitamin A, copper, selenium, and zinc, and replacement only if a deficiency is noted.

Second, there is a huge demand-capacity mismatch. Only about 1% of the eligible population is currently undergoing bariatric surgery[43]. With the proposed extension of the eligibility pool to patients with a lower BMI and age extremes, this percentage will be even lower. Whether this miss-match is due to patient preferences, funding limitations, or limited healthcare resources is ultimately a moot point, as this issue brings up a bigger problem: bariatric surgery is an individual approach to a public health problem! While this intervention remains by far the most effective approach to the treatment of obesity and diabetes, its

availability to less than 1% of those who need it makes it an impractical solution to our impending healthcare crises induced by the obesity and diabetes epidemic.

Bariatric surgery should be viewed as a gateway to enhancing our understanding of the pathophysiology of obesity and diabetes. Decoding the mechanisms leading to the metabolic benefits of bariatric surgery will hopefully lead to treatment options that can be deployed to all those in need.

## 9. Conclusions

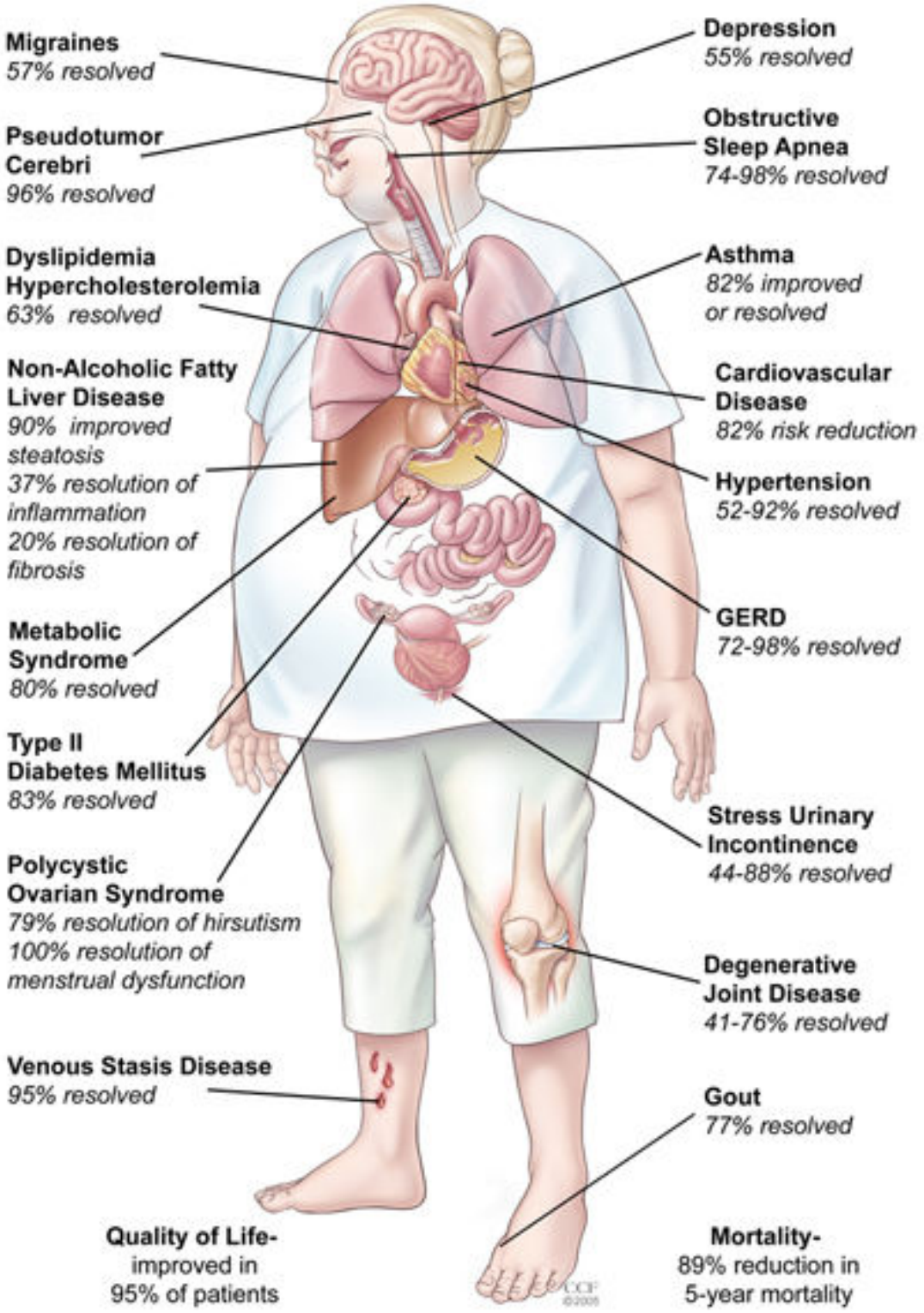
Bariatric surgery is the most effective treatment currently available for type 2 diabetes and the only one capable of significantly changing the course of the disease and induce remission. The primary mechanism through which bariatric surgery induces diabetes remission is caloric restriction, but the mechanisms through which bariatric surgery is capable of inducing long term adherence to caloric restriction are not known and should be the key research focus. Despite its undeniable effectiveness, a surgical procedure is not the answer to a disease epidemic. Continued research to unlock the mechanisms responsible for the long-term maintenance of caloric restriction following bariatric surgery is of critical importance.

## References:


1. **Economic costs of diabetes in the U.S. in 2012. *Diabetes care.* Apr 2013;36(4):1033-1046.**
2. **Number (in Millions) of Civilian, Noninstitutionalized Persons with Diagnosed Diabetes, United States, 1980–2010. 2011; http://www.cdc.gov/diabetes/statistics/prev/national/figpersons.htm. Accessed March 23, 2012, 2012.**
3. **Boyle JP, Honeycutt AA, Narayan KM, et al. Projection of diabetes burden through 2050: impact of changing demography and disease prevalence in the U.S. *Diabetes care.* Nov 2001;24(11):1936-1940.**
4. **Taylor R. The 2012 Banting Lecture Reversing the twin cycles of Type 2 diabetes. *Diabet Med.* Oct 17 2012.**
5. **Kahn SE, Haffner SM, Heise MA, et al. Glycemic durability of rosiglitazone, metformin, or glyburide monotherapy. *The New England journal of medicine.* Dec 7 2006;355(23):2427-2443.**
6. **Buse JB, Caprio S, Cefalu WT, et al. How do we define cure of diabetes? *Diabetes care.* Nov 2009;32(11):2133-2135.**
7. **Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. *Jama.* Oct 13 2004;292(14):1724-1737.**
8. **Schauer PR, Kashyap SR, Wolski K, et al. Bariatric Surgery versus Intensive Medical Therapy in Obese Patients with Diabetes. *The New England journal of medicine.* Mar 26 2012.**

9. Mingrone G, Panunzi S, De Gaetano A, et al. Bariatric Surgery versus Conventional Medical Therapy for Type 2 Diabetes. *The New England journal of medicine.* Mar 26 2012.
10. Rubino F, Schauer PR, Kaplan LM, Cummings DE. Metabolic surgery to treat type 2 diabetes: clinical outcomes and mechanisms of action. *Annual review of medicine.* 2010;61:393-411.
11. Kadera BE, Lum K, Grant J, Pryor AD, Portenier DD, DeMaria EJ. Remission of type 2 diabetes after Roux-en-Y gastric bypass is associated with greater weight loss. *Surg Obes Relat Dis.* May-Jun 2009;5(3):305-309.
12. Dixon JB, Chuang LM, Chong K, et al. Predicting the glycemic response to gastric bypass surgery in patients with type 2 diabetes. *Diabetes care.* Jan 2013;36(1):20-26.
13. Arterburn DE, Bogart A, Sherwood NE, et al. A multisite study of long-term remission and relapse of type 2 diabetes mellitus following gastric bypass. *Obesity surgery.* Jan 2013;23(1):93-102.
14. Dixon JB, O'Brien PE, Playfair J, et al. Adjustable gastric banding and conventional therapy for type 2 diabetes: a randomized controlled trial. *Jama.* Jan 23 2008;299(3):316-323.
15. Koehestanie P, Meesters B, Janssen I, Bouvy N, Greve J. Six month results of a multi-center, prospective, randomized trial of the endoscopic Duodenal-Jejunal Bypass Liner for the treatment of type 2 diabetes in obese subjects. Paper presented at: American Diabetes Association2012; Philadelphia, PA.
16. de Jonge C, Rensen SS, Verdam FJ, et al. Endoscopic Duodenal-Jejunal Bypass Liner Rapidly Improves Type 2 Diabetes. *Obesity surgery.* Mar 23 2013.
17. Bradley D, Magkos F, Klein S. Effects of bariatric surgery on glucose homeostasis and type 2 diabetes. *Gastroenterology.* Oct 2012;143(4):897-912.
18. Bradley D, Conte C, Mittendorfer B, et al. Gastric bypass and banding equally improve insulin sensitivity and beta cell function. *J Clin Invest.* Dec 3 2012;122(12):4667-4674.
19. Peterli R, Steinert RE, Woelnerhanssen B, et al. Metabolic and hormonal changes after laparoscopic Roux-en-Y gastric bypass and sleeve gastrectomy: a randomized, prospective trial. *Obesity surgery.* May 2012;22(5):740-748.
20. Lim EL, Hollingsworth KG, Aribisala BS, Chen MJ, Mathers JC, Taylor R. Reversal of type 2 diabetes: normalisation of beta cell function in association with decreased pancreas and liver triacylglycerol. *Diabetologia.* Oct 2011;54(10):2506-2514.
21. Kelley DE, Wing R, Buonocore C, Sturis J, Polonsky K, Fitzsimmons M. Relative effects of calorie restriction and weight loss in noninsulin-dependent diabetes mellitus. *J Clin Endocrinol Metab.* Nov 1993;77(5):1287-1293.
22. Henry RR, Scheaffer L, Olefsky JM. Glycemic effects of intensive caloric restriction and isocaloric refeeding in noninsulin-dependent diabetes mellitus. *J Clin Endocrinol Metab.* Nov 1985;61(5):917-925.
23. Lingvay I, Guth E, Islam A, Livingston E. Rapid Improvement of Diabetes After Gastric Bypass Surgery: Is It the Diet or Surgery? *Diabetes care.* Mar 25 2013.
24. Sjostrom L, Lindroos AK, Peltonen M, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *The New England journal of medicine.* Dec 23 2004;351(26):2683-2693.
25. Ikramuddin S, Korner J, Lee WJ, et al. Roux-en-Y gastric bypass vs intensive medical management for the control of type 2 diabetes, hypertension, and hyperlipidemia: the Diabetes Surgery Study randomized clinical trial. *Jama.* Jun 5 2013;309(21):2240-2249.
26. Gastrointestinal surgery for severe obesity. *Consensus statement / NIH Consensus Development Conference. National Institutes of Health Consensus Development Conference.* Mar 25-27 1991;9(1):1-20.

27. **Maggard-Gibbons M, Maglione M, Livhits M, et al. Bariatric surgery for weight loss and glycemic control in nonmorbidly obese adults with diabetes: a systematic review. *Jama.* Jun 5 2013;309(21):2250-2261.**
28. **Angrisani L, Cutolo PP, Formisano G, Nosso G, Santonicola A, Vitolo G. Long-Term Outcomes of Laparoscopic Adjustable Silicone Gastric Banding (LAGB) in Moderately Obese Patients With and Without Co-morbidities. *Obesity surgery.* Jul 2013;23(7):897-902.**
29. **Mercer RE, Michaelson SD, Chee MJ, Atallah TA, Wevrick R, Colmers WF. Magel2 is required for leptin-mediated depolarization of POMC neurons in the hypothalamic arcuate nucleus in mice. *PLoS genetics.* 2013;9(1):e1003207.**
30. **Zhu L, Mo Z, Yang X, et al. Effect of laparoscopic Roux-en-Y gastroenterostomy with BMI<35 kg/m(2) in type 2 diabetes mellitus. *Obesity surgery.* Oct 2012;22(10):1562-1567.**
31. **Cohen RV, Pinheiro JC, Schiavon CA, Salles JE, Wajchenberg BL, Cummings DE. Effects of gastric bypass surgery in patients with type 2 diabetes and only mild obesity. *Diabetes care.* Jul 2012;35(7):1420-1428.**
32. **Sjostrom L, Narbro K, Sjostrom CD, et al. Effects of bariatric surgery on mortality in Swedish obese subjects. *The New England journal of medicine.* Aug 23 2007;357(8):741-752.**
33. **Busetto L, Mirabelli D, Petroni ML, et al. Comparative long-term mortality after laparoscopic adjustable gastric banding versus nonsurgical controls. *Surg Obes Relat Dis.* Sep-Oct 2007;3(5):496-502; discussion 502.**
34. **Adams TD, Gress RE, Smith SC, et al. Long-term mortality after gastric bypass surgery. *The New England journal of medicine.* Aug 23 2007;357(8):753-761.**
35. **Johnson BL, Blackhurst DW, Latham BB, et al. Bariatric surgery is associated with a reduction in major macrovascular and microvascular complications in moderately to severely obese patients with type 2 diabetes mellitus. *J Am Coll Surg.* Apr 2013;216(4):545-556; discussion 556-548.**
36. **Romeo S, Maglio C, Burza MA, et al. Cardiovascular events after bariatric surgery in obese subjects with type 2 diabetes. *Diabetes care.* Dec 2012;35(12):2613-2617.**
37. **Heneghan HM, Cetin D, Navaneethan SD, Orzech N, Brethauer SA, Schauer PR. Effects of bariatric surgery on diabetic nephropathy after 5 years of follow-up. *Surg Obes Relat Dis.* Jan-Feb 2013;9(1):7-14.**
38. **Pollock RF, Chilcott J, Muduma G, Valentine WJ. Laparoscopic adjustable gastric banding vs standard medical management in obese patients with type 2 diabetes: a budget impact analysis in the UK. *Journal of medical economics.* 2013;16(2):249-259.**
39. **Picot J, Jones J, Colquitt JL, et al. The clinical effectiveness and cost-effectiveness of bariatric (weight loss) surgery for obesity: a systematic review and economic evaluation. *Health Technol Assess.* Sep 2009;13(41):1-190, 215-357, iii-iv.**
40. **Hoerger TJ, Zhang P, Segel JE, Kahn HS, Barker LE, Couper S. Cost-effectiveness of bariatric surgery for severely obese adults with diabetes. *Diabetes care.* Sep 2010;33(9):1933-1939.**
41. **Dimick JB, Nicholas LH, Ryan AM, Thumma JR, Birkmeyer JD. Bariatric surgery complications before vs after implementation of a national policy restricting coverage to centers of excellence. *Jama.* Feb 27 2013;309(8):792-799.**
42. **Birkmeyer NJ, Dimick JB, Share D, et al. Hospital complication rates with bariatric surgery in Michigan. *Jama.* Jul 28 2010;304(4):435-442.**
43. **Mechanick JI, Youdim A, Jones DB, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient-2013 update: cosponsored by american association of clinical endocrinologists, the obesity society, and american society for metabolic & bariatric surgery. *Endocr Pract.* Mar-Apr 2013;19(2):337-372.**


Migraines
57% resolved
Pseudotumor Cerebri
96% resolved
Dyslipidemia Hypercholesterolemia
63% resolved
Non-Alcoholic Fatty Liver Disease
90% improved steatosis
37% resolution of inflammation
20% resolution of fibrosis
Metabolic Syndrome
80% resolved
Type II Diabetes Mellitus
83% resolved
Polycystic Ovarian Syndrome
79% resolution of hirsutism
100% resolution of menstrual dysfunction
Venous Stasis Disease
95% resolved
Quality of Life-
improved in
95% of patients
Depression
55% resolved
Obstructive Sleep Apnea
74-98% resolved
Asthma
82% improved or resolved
Cardiovascular Disease
82% risk reduction
Hypertension
52-92% resolved
GERD
72-98% resolved
Stress Urinary Incontinence
44-88% resolved
Degenerative Joint Disease
41-76% resolved
Gout
77% resolved
Mortality-
89% reduction in
5-year mortality

| Preoperative Checklist for Bariatric Surgery* | |
|---|---|
| ✓ | Complete H & P (obesity-related co-morbidities, causes of obesity, weight BMI, weight loss history, commitment, and exclusions related to surgical risk) |
| ✓ | Routine labs (including fasting blood glucose and lipid panel, kidney function, liver profile, lipid profile, urine analysis, prothrombin time/INR, blood type, CBC) |
| ✓ | Nutrient screening with iron studies, $B_{12}$ and folic acid (RBC folate, homocysteine, methylmalonic acid optional), and 25 vitamin D (vitamins A and E optional); consider more extensive testing in patients undergoing malabsorptive procedures based on symptoms and risks |
| ✓ | Cardiopulmonary evaluation with sleep apnea screening (ECG, CXR, echocardiography if cardiac disease or pulmonary hypertension suspected; DVT evaluation if clinically indicated) |
| ✓ | GI evaluation (H pylori screening in high-prevalence areas; gallbladder evaluation and upper endoscopy if clinically indicated) |
| ✓ | Endocrine evaluation ($A_{1c}$ with suspected or diagnosed prediabetes or diabetes; TSH with symptoms or increased risk of thyroid disease; androgens with PCOS suspicion (total/bioavailable testosterone, DHEAS, $\Delta_4$-androstenedione); screening for Cushing's syndrome if clinically suspected (1 mg overnight dexamethasone test, 24-hour urinary free cortisol, 11 PM salivary cortisol) |
| ✓ | Clinical nutrition evaluation by RD |
| ✓ | Psychosocial-behavioral evaluation |
| ✓ | Document medical necessity for bariatric surgery |
| ✓ | Informed consent |
| ✓ | Provide relevant financial information |
| ✓ | Continue efforts for preoperative weight loss |
| ✓ | Optimize glycemic control |
| ✓ | Pregnancy counseling |
| ✓ | Smoking cessation counseling |
| ✓ | Verify cancer screening by primary care physician |
| *See text for abbreviations. | |

| Postoperative Checklist for Bariatric Surgery* | | | | | |
|---|---|---|---|---|---|
| Checklist Item | | LAGB | LSG | RYGB | BPDDS |
| *Early postoperative care* | | | | | |
| ✓ | monitored telemetry at least 24 hr if high risk for MI | ✓ | ✓ | ✓ | ✓ |
| ✓ | protocol-derived staged meal progression supervised by RD | ✓ | ✓ | ✓ | ✓ |
| ✓ | healthy eating education by RD | ✓ | ✓ | ✓ | ✓ |
| ✓ | multivitamin plus minerals (# tablets for minimal requirement) | 1 | 2 | 2 | 2 |
| ✓ | calcium citrate, 1200-1500 mg/d | ✓ | ✓ | ✓ | |
| ✓ | vitamin D, at least 3000 units/d, titrate to >30 ng/mL | ✓ | ✓ | ✓ | ✓ |
| ✓ | vitamin $B_{12}$ as needed for normal range levels | ✓ | ✓ | ✓ | ✓ |
| ✓ | maintain adequate hydration (usually >1.5 L/d PO) | ✓ | ✓ | ✓ | ✓ |
| ✓ | monitor blood glucose with diabetes or hypoglycemic symptoms | ✓ | ✓ | ✓ | ✓ |
| ✓ | pulmonary toilet, spirometry, DVT prophylaxis | ✓ | ✓ | ✓ | ✓ |
| ✓ | if unstable, consider pulmonary embolus (PE), intestinal leak (IL) | PE | PE | PE/IL | PE/IL |
| ✓ | if rhabdomyolysis suspected, check CPK | ✓ | ✓ | ✓ | ✓ |
| *Follow-up* | | | | | |
| ✓ | visits: initial, interval until stable, once stable (months) | 1,1-2,12 | 1,3-6,12 | 1,3,6-12 | 1,3,6 |
| ✓ | monitor progress with weight loss and evidence of complications each visit | ✓ | ✓ | ✓ | ✓ |
| ✓ | SMA-21, CBC/plt with each visit (and iron at baseline and after as needed) | ✓ | ✓ | ✓ | ✓ |
| ✓ | avoid nonsteroidal antiinflammatory drugs | ✓ | ✓ | ✓ | ✓ |
| ✓ | adjust postoperative medications | ✓ | ✓ | ✓ | ✓ |
| ✓ | consider gout and gallstone prophylaxis in appropriate patients | ✓ | ✓ | ✓ | ✓ |
| ✓ | need for antihypertensive therapy with each visit | ✓ | ✓ | ✓ | ✓ |
| ✓ | lipid evaluation every 6-12 months based on risk and therapy | ✓ | ✓ | ✓ | ✓ |
| ✓ | monitor adherence with physical activity recommendations | ✓ | ✓ | ✓ | ✓ |
| ✓ | evaluate need for support groups | ✓ | ✓ | ✓ | ✓ |
| ✓ | bone density (DXA) at 2 years | ✓ | ✓ | ✓ | ✓ |
| ✓ | 24-hour urinary calcium excretion at 6 months and then annually | ✓ | ✓ | ✓ | ✓ |
| ✓ | $B_{12}$ (annually; MMA and HCy optional; then q 3-6 months if supplemented) | ✓ | ✓ | ✓ | ✓ |
| ✓ | folic acid (RBC folic acid optional), iron studies, 25-vitamin D, iPTH | x | x | ✓ | ✓ |
| ✓ | vitamin A (initially and q 6-12 months thereafter) | x | x | optional | ✓ |
| ✓ | copper, zinc, and selenium evaluation with specific findings | x | x | ✓ | ✓ |
| ✓ | thiamine evaluation with specific findings | ✓ | ✓ | ✓ | ✓ |
| ✓ | consider eventual body contouring surgery | ✓ | ✓ | ✓ | ✓ |
| *see text for abbreviations; based on general obesity-related risks, GI functional anatomy, and clinical endpoints after specific bariatric surgical procedures. | | | | | |

**From: Clinical Practice Guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patients – 2013 update: cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic and Bariatric Surgery.**