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Bariatric Surgery and Its Metabolic Effects in Obese Patients with Type 2 Diabetes

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ABSTRACT

Bariatric/metabolic surgery is the most effective intervention for achieving substantial and durable weight loss in people with obesity and has profound, often rapid, effects on glycemic control in type 2 diabetes (T2D). Beyond calorie restriction and weight loss, procedures such as Roux-en-Y gastric bypass (RYGB), sleeve gastrectomy (SG), biliopancreatic diversion with duodenal switch (BPD-DS), and one-anastomosis gastric bypass (OAGB) remodel entero-insular signaling, bile-acid–FXR/TGR5 pathways, gut–brain–liver circuits, and the microbiome. These weight-independent mechanisms amplify insulin sensitivity, enhance β-cell function, and promote diabetes remission in a sizable subset of patients. Surgery also improves comorbidities—non-alcoholic fatty liver disease, obstructive sleep apnea, hypertension, dyslipidemia—and reduces incident cardiovascular events and mortality. However, benefits vary with procedure type, baseline β-cell reserve, diabetes duration, and adherence to postoperative nutrition and follow-up. Risks include peri-operative complications, micronutrient deficiencies, hypoglycemia, nephrolithiasis, alcohol use disorder, bone loss, and weight regain in a minority; careful selection, team-based peri-operative care, and long-term surveillance mitigate these. As potent anti-obesity/anti-diabetic pharmacotherapies (e.g., GLP-1 receptor agonists and multi-agonists) expand options, surgery retains a unique role when severe obesity, refractory hyperglycemia, or complications necessitate rapid, durable metabolic change. This review synthesizes mechanisms of glucose improvement after surgery; compares procedures and outcomes; summarizes effects on end organs; details peri-operative management; and proposes an integrated, precision framework that aligns surgical choice with phenotypes and uses adjunct lifestyle and pharmacotherapy to extend remission while minimizing risks.

Keywords: metabolic surgery; Roux-en-Y gastric bypass; sleeve gastrectomy; bile acids/incretins; diabetes remission

INTRODUCTION

Type 2 diabetes (T2D) most often develops on a background of excess adiposity and insufficient metabolic flexibility[1–3]. In this setting, insulin resistance (IR), ectopic lipid, and β-cell stress progressively erode glycemic control. Lifestyle change and medications lower glucose and cardiometabolic risk, yet many patients struggle to attain durable weight loss and sustained HbA1c targets. Bariatric now often termed metabolic surgery emerged as a powerful therapeutic option that improves glycemia rapidly and sometimes normalizes it before major weight loss occurs, implying mechanisms beyond energy deficit alone[4]. Over the last two decades, randomized trials and large registries have consolidated the role of surgery for patients with obesity (typically BMI ≥35 kg/m² with T2D, or BMI 30–34.9 kg/m² with inadequately controlled T2D in selected cases), demonstrating superior weight loss, higher rates of diabetes remission, reduced medication burden, and improved quality of life versus intensive medical therapy[5].

Four procedures dominate contemporary practice with differing anatomy and physiology. Roux-en-Y gastric bypass (RYGB) creates a small gastric pouch anastomosed to jejunum, excluding the duodenum/proximal jejunum from nutrient flow; this accelerates nutrient delivery to distal gut, augments incretin release (GLP-1, PYY), increases circulating bile acids, and modestly restricts intake[6]. Sleeve gastrectomy (SG) removes ~75–80% of the stomach along the greater curvature, reducing ghrelin production, accelerating gastric emptying, and enhancing distal gut hormone responses. Biliopancreatic diversion with duodenal switch (BPD-DS) couples a sleeve with long-limb intestinal bypass to produce the greatest malabsorption and weight loss, with powerful metabolic effects but higher nutritional risk. One-anastomosis gastric bypass (OAGB/MGB) uses a long gastric pouch with a single gastrojejunal anastomosis; metabolic outcomes approximate RYGB with technical differences and reflux considerations[6].

Why does surgery work so well for diabetes? Mechanisms can be grouped into weight-dependent and weight-independent effects. Weight loss decreases adipocyte size and lipolysis, lowering NEFA flux to liver and muscle, which improves hepatic and peripheral insulin signaling and reduces gluconeogenesis[7]. Ectopic fat regresses in liver, muscle, and pancreas, decompressing mitochondrial/ER stress and restoring insulin responsiveness. Appetite and food preference shift toward lower energy density and higher protein/fiber, supporting maintenance[8].

Weight-independent effects act early. Distal gut stimulation after RYGB/SG boosts incretins (GLP-1, PYY), improving β-cell function and satiety; bile-acid pools expand and shift, activating FXR/TGR5 signaling that enhances insulin sensitivity, energy expenditure, and GLP-1 secretion; intestinal gluconeogenesis and portal sensing modulate hepatic glucose output; vagal and brain reward circuits retune, reducing hedonic drive; and microbiome composition/function remodels toward metabolites that favor insulin sensitivity (e.g., SCFAs)[7]. Glucose homeostasis benefits from lower postprandial glycemia excursions, improved first-phase insulin secretion, and decreased hepatic glucose production—changes that can precede major weight loss by weeks[7].

Clinical results vary by procedure, patient phenotype, and disease duration. Shorter T2D duration and higher C-peptide (better β-cell reserve) predict remission; visceral adiposity, NAFLD, and severe IR often show large improvements[9]. RYGB generally induces more robust early glycemic change than SG at similar weight loss, while BPD-DS yields the highest remission rates at the cost of nutritional vigilance. Long-term, some patients experience weight regain and hyperglycemia relapse often linked to behavioral drift, anatomical adaptation, hormonal counter-regulation, or unaddressed psychosocial drivers highlighting the need for lifelong follow-up and adjunct therapies[9].

Safety is favorable in experienced centers: peri-operative mortality is low and serious complication rates compare with cholecystectomy or hysterectomy. Nonetheless, micronutrient deficiencies (iron, B12, folate, calcium/vitamin D), protein malnutrition (rare with SG/RYGB, higher with BPD-DS), hypoglycemia (post-bypass), small-bowel obstruction/internal hernia (RYGB), reflux (SG/OAGB), and bone loss warrant structured surveillance and supplementation[10]. Fertility increases post-surgery; pregnancy timing and nutrition require planning. Alcohol sensitivity may rise after RYGB; behavioral health support is integral[10].

As incretin-based pharmacotherapy (GLP-1 receptor agonists and GLP-1/GIP co-agonists) delivers medical weight loss approaching surgical ranges for some, questions arise about relative roles[11]. Rather than competition, a combinatorial model is emerging: surgery for rapid, durable metabolic change in appropriate candidates; advanced pharmacotherapy for others; and adjunct meds to prevent weight regain or treat relapse after surgery. Precision selection matching anatomy to phenotype, comorbidity, preferences, and local expertise can maximize benefit while minimizing risk[11]. This review synthesizes the mechanistic underpinnings of surgery's metabolic effects; compares procedures and outcomes; surveys end-organ benefits; outlines peri-operative management, risks, and follow-up; and proposes an integrated precision framework that positions surgery within modern diabesity care.

2. Surgical Procedures and Comparative Metabolic Outcomes

Roux-en-Y gastric bypass (RYGB). By creating a small gastric pouch and diverting nutrients to the jejunum, RYGB reduces stomach capacity, accelerates nutrient delivery to the distal intestine, and bypasses duodenum/proximal jejunum[12]. Early after surgery, fasting glucose and postprandial excursions decline sharply, often before significant weight loss[12]. GLP-1 and PYY surge, suppressing appetite and enhancing β-cell insulin secretion; bile-acid concentrations increase with altered composition, engaging FXR/TGR5. Average total weight loss (TWL) at 1–2 years is ~25–35%, with diabetes remission rates (off glucose-lowering meds, normoglycemia or near-normoglycemia) commonly 40–60% depending on definitions and baseline β-cell reserve. Long-term, partial relapse can occur, but many maintain reduced medication needs and improved cardiometabolic profiles[13].

Sleeve gastrectomy (SG). SG is technically simpler and now the most common procedure globally. Removal of the gastric fundus lowers ghrelin, speeds gastric emptying, and amplifies distal gut hormone responses, albeit typically less than RYGB. TWL averages ~20–30% at 1–2 years[14]. Diabetes remission is frequent though slightly lower than RYGB at matched weight loss in many series; however, SG avoids intestinal bypass and has lower risk of internal hernia and marginal ulcer. Reflux can worsen or newly develop; patient selection and hiatal hernia repair matter[14].

Biliopancreatic diversion with duodenal switch (BPD-DS) and single-anastomosis duodenal-ileal bypass (SADI-S). These provide the greatest weight loss and metabolic potency by combining sleeve with long-limb bypass that separates food from bile/pancreatic secretions until late in the ileum[14]. TWL often exceeds 35% with very high remission rates, but nutritional risk (protein malnutrition, fat-soluble vitamin deficiencies) is higher, necessitating rigorous supplementation and follow-up. SADI-S simplifies DS with comparable outcomes in emerging data[14].

One-anastomosis gastric bypass (OAGB/MGB). A long gastric pouch with a single gastrojejunal anastomosis yields weight loss and remission similar to RYGB; bile reflux and marginal ulcer risk require attention. Biliopancreatic limb length tailoring balances metabolic effect against malabsorption risk[15].

Comparative considerations. Procedure choice weighs metabolic potency, anatomic/functional risks, patient preferences (e.g., avoidance of intestinal bypass), GERD status, and center expertise. For severe T2D with long

duration but preserved C-peptide, RYGB/BPD-DS may maximize early glycemic gains; for patients with significant reflux, RYGB often outperforms SG; for high BMI with refractory diabetes/NAFLD, DS-variants offer strongest weight loss if follow-up is assured[16]. Regardless of procedure, structured nutrition, activity, and behavioral care determine durability.

3. Mechanisms of Glycemic Improvement: Beyond Weight Loss

Entero-insular axis. Distal nutrient delivery after RYGB/SG increases **GLP-1** and **PYY**, enhancing insulin secretion, slowing gastric emptying, and reducing appetite. Elevated GLP-1 improves β-cell responsiveness and may promote β-cell rest, while PYY augments satiety and reduces caloric intake changes evident within days[17].

Bile acids and FXR/TGR5 signaling. Surgery increases circulating bile acids and alters their composition. **FXR** activation in the ileum induces FGF19/15, which suppresses hepatic gluconeogenesis and regulates bile-acid synthesis; **TGR5** activation stimulates GLP-1 release and enhances energy expenditure in brown/beige adipose tissue. These pathways contribute to improved insulin sensitivity and lipid handling independent of weight loss[18].

Intestinal gluconeogenesis and portal sensing. RYGB/SG can upregulate intestinal gluconeogenesis; glucose sensed by the portal vein via vagal pathways signals the brain to suppress hepatic glucose production and improve insulin sensitivity a mechanism demonstrated in models and supported by human data[19].

Microbiome remodeling. Post-surgical shifts favor taxa that enhance short-chain fatty acid (SCFA) production and modify bile-acid deconjugation, influencing FXR/TGR5 and inflammatory tone. Reduced endotoxemia and altered microbial metabolites (indoles, phenolics) support barrier integrity and insulin sensitivity[20–23].

Adipose and hepatic lipid flux. Negative energy balance and hormonal changes rapidly reduce hepatic de novo lipogenesis and increase fatty-acid oxidation; intrahepatic triglyceride content declines within weeks, improving hepatic insulin sensitivity and fasting glucose. Adipose tissue becomes less inflamed and more insulin-responsive as adipocyte size shrinks; lipolysis normalizes, decreasing NEFA spillover[24–27].

Neuroendocrine and reward circuits. Vagal signaling, hypothalamic inflammation resolution, and dopamine reward recalibration reduce hedonic eating. Food preferences often shift toward protein-rich, less energy-dense options; alcohol sensitivity may increase post-bypass[28].

Net result. First-phase insulin secretion improves; postprandial glycemia blunts; hepatic glucose output falls; and peripheral insulin sensitivity rises—delivering remission in many and major medication reduction in most. These effects arise from the **integration** of gut hormones, bile acids, neural circuits, microbiome metabolites, and lipid flux changes, not from caloric restriction alone[29].

4. End-Organ Effects: Liver, Cardiovascular System, Kidneys, Sleep, and Fertility

Liver/NAFLD. Surgery reduces hepatic steatosis rapidly (weeks) and improves steatohepatitis/fibrosis over months to years. Mechanisms include decreased NEFA delivery, lowered de novo lipogenesis, increased insulin sensitivity, and bile-acid/FGF19 signaling that restrains gluconeogenesis and improves lipid handling[30]. Histologic improvement is most pronounced with larger weight loss and procedures with greater metabolic impact (RYGB, DS variants), though SG is also beneficial.

Cardiovascular risk. Blood pressure, triglycerides, and LDL-C/apoB typically decrease; HDL-C rises. Inflammation (CRP) and endothelial dysfunction markers fall. Large cohort studies associate surgery with reduced incident major adverse cardiovascular events and mortality versus usual care, likely due to combined weight, glycemic, and lipid improvements along with sleep apnea resolution[30].

Kidney. Albuminuria declines and eGFR stabilizes in many with diabetic kidney disease, aided by lower BP, improved glycemia, and reduced glomerular hyperfiltration. Nephrolithiasis risk may rise (oxalate) after malabsorptive procedures; hydration, calcium with meals, and oxalate moderation mitigate this[31].

Sleep and respiratory function. Obstructive sleep apnea severity drops as fat depots regress and airway tone improves; CPAP needs may diminish but reassessment is required to confirm resolution. Asthma control can also improve[31].

Bone and micronutrients. Bone mineral density can decline due to weight loss, altered calcium/vitamin D absorption, and secondary hyperparathyroidism most with bypass/DS. Routine supplementation, monitoring (25-OH-vitamin D, PTH), resistance training, and protein adequacy are essential[32].

Reproductive health. Fertility improves; menstrual regularity returns; PCOS features (hyperandrogenism, anovulation) soften. Contraception is advised for 12–18 months post-op during rapid weight loss. Pregnancy after stabilization is generally safe with appropriate nutritional surveillance; gestational diabetes risk falls but micronutrient vigilance is crucial[33].

Quality of life and mental health. Many report better mobility, pain, and psychosocial functioning. Screening for depression, eating disorders, and alcohol use is vital; behavioral care supports long-term success.

5. Peri-operative Management: Preparation, Medications, and Nutritional Care

Pre-operative assessment. Multidisciplinary evaluation includes glycemic control (optimized to reduce infection risk), sleep apnea screening, GERD assessment, NAFLD evaluation, micronutrient baseline (iron, B12, folate, vitamin D, calcium, thiamine), and lifestyle readiness. Pre-op very-low-calorie diets (2–4 weeks) shrink the liver and improve the operative view[34].

Medication adjustments. On surgery day and early post-op, insulin and secretagogues are reduced or paused to avoid hypoglycemia; metformin is typically held briefly then restarted; SGLT2 inhibitors are stopped pre-op to avoid euglycemic ketoacidosis and reintroduced later if needed. Antihypertensives and statins are titrated as weight and BP/lipids improve. Acid suppression is common after bypass to reduce marginal ulcer risk[35].

Diet progression. Standard pathways advance from clear liquids → full liquids/protein shakes pureed/soft regular textures over 3–6 weeks, with protein targets (~60–100 g/day), hydration (≥1.5–2 L/day), and mindful eating (small bites, thorough chewing, no drinking with meals). Dumping symptoms prompt slower eating and lower simple sugars.

Supplementation. Lifelong multivitamin/mineral supplementation is mandatory, tailored by procedure: iron with vitamin C, B12 (sublingual or injections), folate, calcium citrate (split doses) with vitamin D, +/- fat-soluble vitamins and trace elements for malabsorptive operations. Thiamine deficiency risk rises with vomiting or poor intake—low threshold for parenteral thiamine[36].

Activity and behavior. Early ambulation reduces VTE risk; progressive aerobic and resistance training preserves lean mass and bone, improves insulin sensitivity, and supports weight maintenance. Behavioral strategies—self-monitoring, structured meals, stimulus control, problem-solving, counter-grazing and liquid calorie drift.

Follow-up. Regular visits (e.g., 2 weeks; 3, 6, 12 months; then annually) track weight, HbA1c, lipids, micronutrients, kidney/liver function, bone health, and mental health. CGM can guide medication de-intensification and detect hypoglycemia.

6. Risks, Complications, and How to Mitigate Them

Early surgical risks include bleeding, leak, infection, VTE, and anesthetic complications uncommon in experienced centers. Procedure-specific issues: marginal ulcers and internal hernias (RYGB), reflux/strictures (SG), bile reflux (OAGB), and higher malnutrition risk (BPD-DS). Early recognition of tachycardia, fever, and abdominal pain triggers prompt evaluation[37].

Micronutrient deficiencies (iron, B12, folate, calcium/vitamin D, thiamine, fat-soluble vitamins) are the most common long-term problems. Prevention rests on routine supplements, annual labs, and dietitian support. Protein malnutrition is rare with SG/RYGB but possible with DS. Prioritize protein at meals, consider supplemental shakes, and treat steatorrhea contributing to losses[38].

Hypoglycemia (post-bypass) presents 1–3 hours after high-carbohydrate meals due to exaggerated incretin/insulin responses; management includes low-GI eating patterns, protein/fat pairing, acarbose, and, in refractory cases, diazoxide, calcium channel blockers, or revisional surgery[39].

Gallstones can form during rapid weight loss; ursodeoxycholic acid prophylaxis is sometimes used. Nephrolithiasis risk rises after malabsorptive procedures via hyperoxaluria; mitigation includes hydration, adequate calcium with meals, and moderating high-oxalate foods[40].

Bone loss and fractures: employ resistance training, adequate calcium/vitamin D, and bone density monitoring in high-risk patients. Alcohol use disorder risk increases post-RYGB due to faster absorption; counseling and screening are key[41]. Weight regain and diabetes relapse reflect behavioral, anatomical (dilated pouch/anastomosis), hormonal, or psychosocial factors. Management combines structured lifestyle, anti-obesity pharmacotherapy (e.g., GLP-1 RAs or multi-agonists), endoscopic therapies (pouch/anastomosis revision), or surgical revision when indicated[41].

Pregnancy after surgery generally has lower GDM and hypertensive disorder risk, but requires micronutrient monitoring (iron, B12, folate, calcium, vitamin D) and careful weight-gain targets; avoid pregnancy during the rapid weight loss phase[42].

Proactive education, standardized pathways, and rapid-response systems convert most risks into manageable issues, preserving the strong net clinical benefit of surgery.

7. Positioning Surgery in the Era of Potent Pharmacotherapy: Toward Precision Care

Modern anti-obesity agents, GLP-1 receptor agonists and GLP-1/GIP co-agonists achieve double-digit weight loss and significant HbA1c reductions for many. Where does surgery fit? **First**, surgery delivers the largest and most durable average weight loss, with rapid glycemic normalization, valuable for advanced T2D/NAFLD or imminent cardiovascular/renal complications. Second, patients who have plateaued on medications or who cannot tolerate them may benefit from surgical escalation. Third, combination strategies are emerging: pre-op pharmacotherapy to reduce operative risk and prime behavior; post-op pharmacotherapy to prevent weight regain or treat relapse, especially when hormonal counter-regulation or environmental drivers re-emerge.

Precision selection considers: (i) BMI and adiposity distribution; (ii) diabetes duration and β-cell reserve (C-peptide); (iii) GERD/hiatal hernia (favor RYGB); (iv) NAFLD/NASH severity (favor potent weight loss); (v) CKD and nephrolithiasis risk (caution with malabsorption); (vi) bone health; (vii) patient preferences and ability for long-term follow-up; and (viii) local surgical expertise. Predictive tools (e.g., diabetes remission scores incorporating age, HbA1c, meds, C-peptide) help set expectations.

Care model. A team-based program aligns surgery with nutrition, physical activity, behavioral therapy, and cardiometabolic medications. CGM-guided titration reduces hypoglycemia; lipid and BP management continue with statins/ACEi/ARB as indicated. NAFLD is monitored with non-invasive scores or imaging. Equity

matters facilitating access, time-off work, transportation, and culturally competent care improves uptake and outcomes.

Outcomes that matter to patients, energy levels, mobility, intimacy, and work productivity should be embedded in decision tools alongside HbA1c and weight charts. Shared decision-making balances metabolic efficacy, risks, lifestyle fit, and values to select the right therapy at the right time.

CONCLUSION

Bariatric/metabolic surgery is a metabolic reset, not merely a volume-reduction operation. By integrating weight loss with potent weight-independent mechanisms enhanced incretin and bile-acid signaling, remodeled gut–brain–liver circuits, rapid hepatic fat reduction, and microbiome shifts, it delivers superior glycemic improvement and remission for many people with T2D, while lowering cardiovascular, hepatic, renal, and sleep-related risks. Benefits are greatest when surgery is matched to phenotype, performed in experienced centers, and embedded in long-term, multidisciplinary care that includes nutrition, resistance training, behavioral support, micronutrient surveillance, and when useful, adjunct pharmacotherapy to prevent regain or manage relapse. As medical treatments strengthen, surgery remains indispensable for severe or complicated diabesity and as part of combination strategies. The practical mandate is precision: choose procedure and adjuncts based on anatomy, comorbidity, β-cell reserve, and patient preferences; monitor with CGM and metabolic panels; and intervene early on nutritional or behavioral drift. With this approach, surgery's metabolic effects can be harnessed safely and durably, shifting care from glucose management to disease modification and sustained risk reduction for people living with obesity-related T2D.

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