Notable Grand Rounds
of the
Michael & Marian Ilitch
Department of Surgery

Wayne State University
School of Medicine

Detroit, Michigan, USA

Dr. Roozbeh Mansour

PRINCIPLES AND PRACTICE OF BARIATRIC SURGERY

September 28, 2022

About Notable Grand Rounds

These assembled papers are edited transcripts of didactic lectures given by mainly senior residents, but also some distinguished attending and guests, at the Grand Rounds of the Michael and Marian Ilitch Department of Surgery at the Wayne State University School of Medicine.

Every week, approximately 50 faculty attending surgeons and surgical residents meet to conduct postmortems on cases that did not go well. That “Mortality and Morbidity” conference is followed immediately by Grand Rounds.

This collection is not intended as a scholarly journal, but in a significant way it is a peer reviewed publication by virtue of the fact that every presentation is examined in great detail by those 50 or so surgeons.

It serves to honor the presenters for their effort, to potentially serve as first draft for an article for submission to a medical journal, to let residents and potential residents see the high standard achieved by their peers and expected of them, and by no means least, to contribute to better patient care.

David Edelman, MD
Program Director
The Detroit Medical Center

and

Professor of Surgery
Wayne State University School of Medicine

Principles and Practice of Bariatric Surgery

Roozbeh Mansour, MD, FACS

Assistant Professor of Surgery
Wayne State University
School of Medicine

Surgical Grand Rounds
September 28, 2022

This paper is based on the author's Surgical Grand Rounds presentation on September 28, 2022, with an addendum contributed by Dr. Alyssa Stroud.

Introduction

This paper is organized in three parts:

1. Physiology (covering the epidemiology and causes of obesity, endohormonal control of energy metabolism, anatomical changes of bariatric surgery relating to the axis of energy metabolism, postoperative dietary and metabolic changes and comorbidity resolution);

2. Complications; and

3. Recent developments in bariatric surgery.

Part 1: Physiology

Obesity as an Epidemic

Adult obesity has doubled in the past 30 years and tripled in the childhood age range. 67% of Americans are overweight (defined as having a BMI of 25 and above) and 35%—over 116 million people—are morbidly obese.

In 1985, obesity statistics were few because it was not considered that much of a problem, although the Midwest had already reached between 10 and 15% morbid obesity (BMI over 30) in the population. By 1990, more statistics were being gathered and more places showed rates

up to 15%. By 1996, the rate in some areas was up to 20%; in 2000, over 20%; in 2004, over 25%; and in 2009, over 40%.

Today, no state in the country has less than 20% morbid obesity. Eleven states have less than 30%, three states and the District of Columbia less than 25%, 20 states plus Guam and Puerto Rico have 30 to 35%, and 16 states including Michigan have 35% and above (the Midwest has always had the highest level of morbid obesity in the country since statistics began to be gathered).

Comorbidities

The comorbidities that accompany obesity are:

- Hypertension
- Lipid disorders
- Diabetes
- Coronary artery disease
- Pulmonary hypertension
- Asthma
- Hypoventilation syndromes
- Obstructive sleep apnea
- Gallstones
- NASH (Non-alcoholic steatohepatitis)
- Urinary incontinence
- Gastroesophageal reflux
- Arthritis
- Low back pain
- Infertility/Menstrual problems
- Obstetrical complications
- DVT and thromboembolism
- Depression
- Immobility
- Breast/Bowel/Prostate/Uterine cancer
- Venous stasis disease/ulcers
- Intertrigo
- Accident proneness

Causes

Known causes include genetic problems such as Prader-Willis and Angelman Syndromes (though we have not yet seen any patients with those syndromes at our institution). The prevalence of obesity in parents, and twin studies, provide clear evidence of a genetic factor. In addition, there are well-known behavioral and environmental factors—overeating, poor diet (**Fig. 1** shows how portion sizes have inflated over the years for a representative selection of fast foods), and lack of exercise.

Energy imbalance is the main reason for obesity. Junk food is relatively cheap, tempting, hygienic, and easy to find. Our lives are sedentary. We don't walk, we don't ride bikes, we don't ride horses. We drive everywhere, we take elevators and escalators rather than the stairs. Manual jobs are easier through mechanical aids and automation, including robots.

A third factor behind obesity is neurohormonal dysregulation, discovered when bypass surgery began to be performed in the 1970s and suspected even before that in duodenal switch operations. The natural regulation of body weight is a homeostatic process where energy is stored and weight is gained in times of plenty versus times of famine when energy is expended, weight is lost, and appetite is induced.

The system that controls appetite and weight gain/loss is very complex. It involves food sensory inputs, neural stimulation of the vagal nerves, the modulation of neurotransmitters, and peripheral endohormones, A great deal remains unknown about some of these components, whose signals are integrated in the hypothalamus to regulate eating behavior.

The endohormones affect the metabolic axis and change stress (cortisol) levels and affect the cardiovascular system. See **Fig. 2** on the next page.

Fig. 1. Portion size inflation in fast foods.
Source: Hensrud, DD and Klein, S. *Mayo Clinic Proceedings*. 2006. 11: s5-10

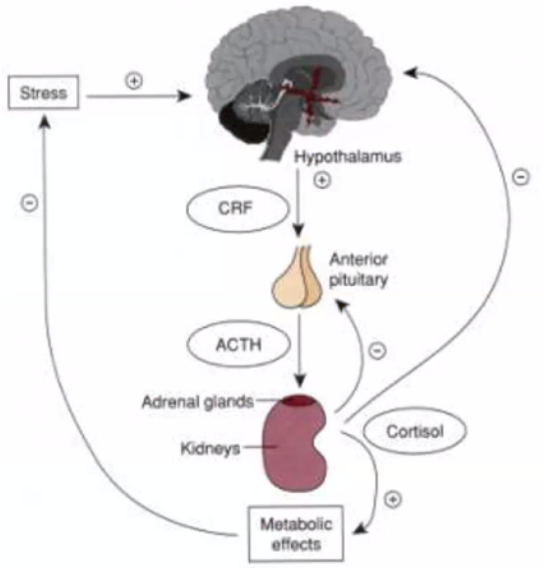

Fig. 2. The Metabolic Axis

The Neurohormonal Axis of Energy Balance: The Players

Leptin, ghrelin, peptide YY, and GLP-1/insulin are the neurohormones known to work through hypothalamus mediators to affect appetite. Other hypothalamus mediators include POMC (proopiomelanocortin), Agouti-related peptide (AGRP), and neuropeptide Y (NPY). Metabolic syndrome also plays a part.

It was first described in the 1920s aa a constellation of abnormalities that can lead to type 2 diabetes and atherosclerotic heart disease, though none of the abnormalities is by itself all that dramatic.

The abnormalities include:

- Fasting hyperglycemia > 100 mg/dl or TX
- Hypertension > 130/85 or TX
- Centripetal Obesity: Waist >40 (35 for women) inches
- Decreased HDL Cholesterol < 40mg/dl or TX
- Elevated Triglycerides > 150 mg/dl or TX

a. LEPTIN

Leptin was first isolated in 1950 from obese rats. Circulating lepton level correlates directly to BMI —the higher the leptin level, the higher the BMI. Leptin is the primary signal sent from adipose tissue to tell the brain that the body has enough energy.

People with high circulating leptin levels (the signal for too much fat storage) are theorized to be leptin resistant: They store fat, the signal is sent that there is too much fat, but the body is desensitized to the signal so they keep eating. The structure is so similar to members of the cytokine family that it is called adipokine.

Leptin acts like a local paracrine signal and contributes directly to metabolic syndrome—though not everybody (not even super morbidly obese people) has metabolic syndrome. And not everybody has a high leptin level, which is why some obese people respond to diet and exercise while others with morbid obesity and these derangements do not.

Leptin raises blood pressure by increasing sympathetic activity. It contributes to endothelial dysfunction, increases vascular tone, and exerts strong pro-inflammatory and immuno-stimulatory effects.

b. GHRELIN

Ghrelin was identified by Kanagawa in 1999 in Osaka, Japan. It is made up of 28 amino acids, is well preserved across species, and is expressed mainly in the stomach but can be found in other tissues. It elicits growth hormone (GH) secretion via growth hormone secretagogue receptor (GHS-R). Administration of ghrelin stimulates food intake and weight gain.

Fig. 3 (next page) shows the structure of ghrelin. At first it was confusing because the levels did not always correlate to expected levels. Eventually it was realized that there are two forms of it in the circulation, and it is the active form that is acylated.

Fig. 4 (next page) places ghrelin in the context of the metabolic axis. The vagus nerve clearly has

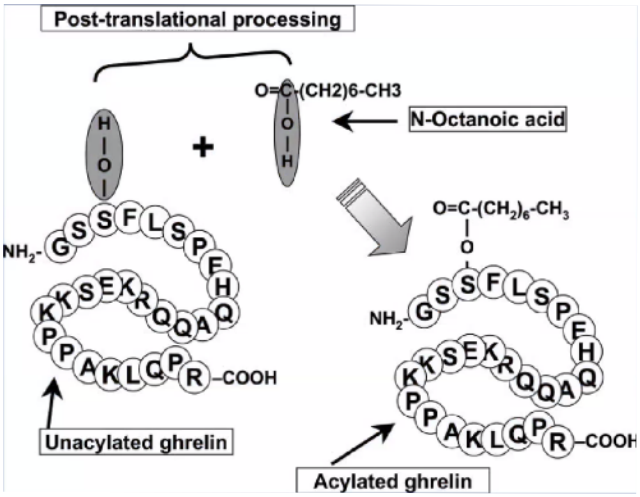

Fig. 3. Structure of ghrelin

a strong effect on the axis: gastric distension, gastric ghrelin. High levels of adipokines have an effect on adipose tissue and trophic effects on tumors. They increase activity of B and T cells, not necessarily in a good way—being more active, they are in a hyperinflammatory state, decreasing insulin secretion from the pancreas, decreasing the exocrine secretion, and affecting the heart.

Ghrelin is higher preprandially—the level rises with hunger. It is not known whether ghrelin initiates the feeling of hunger or hunger stimulates the secretion of ghrelin. It also regulates the duration of ingestion—someone with higher, or more persistently high, levels of ghrelin will tend to eat for a longer time before feeling full. It influences the secretion and motility of the stomach, which obviously has effects on appetite. Energy storage is adipogenic.

c. and d. PEPTIDE YY and GLP-1
Peptide YY (PYY) and glucagon-like protein (GLP-1) comprise what is known as the *ileal brake*. If the volume of undigested nutrients reaching the terminal ileum exceeds some threshold, the terminal ileum sends signals to several organs, including the brain, saying essentially that there are enough undigested nutrients to conclude that satiety has been reached. The ileum thus puts the brake on appetite.

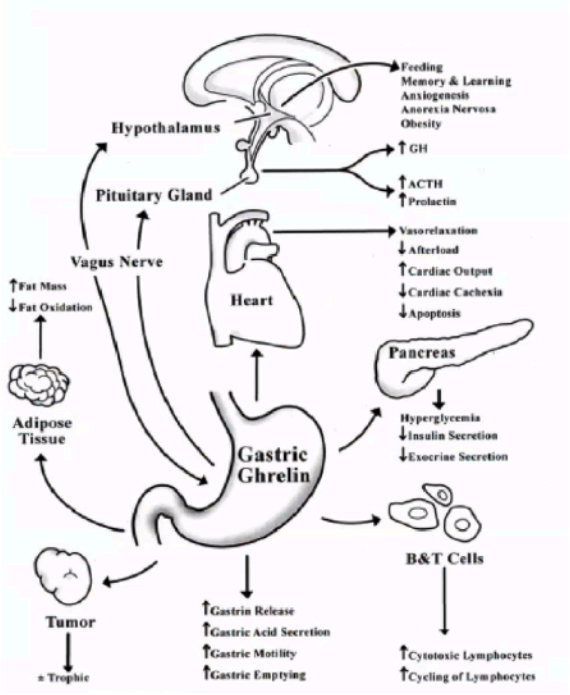

Fig. 4. Physiological actions of gherkin

It is thought that PYY, GLP-1, and probably other hormones not yet identified are responsible for the ileal brake. They are secreted by L cells in the terminal ileum. PYY tends to stop ingestion and improve nutrient utilization. GLP-1 is an incretin, which stimulates release of insulin from beta cells. Perhaps because they have longer intestines, obese individuals have less PYY/GLP-1 release.

Fig. 5 (next page) shows how those factors work and how two diabetes medications (Byetta and Januvia) work on those factors. Obesity and type 2 diabetes work hand-in-hand. At least we know these two enzymes are mediators and we have medications that induce them. Those medications help with weight loss too, albeit temporarily.

Inserting PYY and GLP-1, and their effect on the hypothalamus, in the metabolic axis (**Fig. 6**, next page) reveals only that the imbalance between leptin and ghrelin is indicative of morbid obesity.

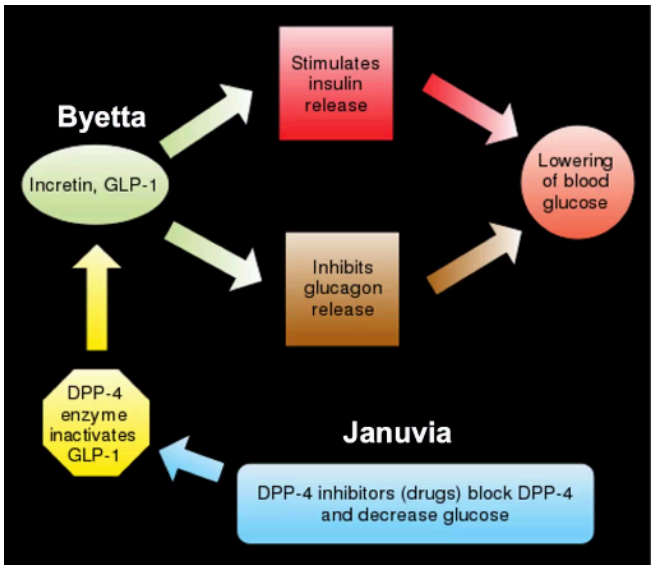

Fig. 5. Effect of 2 diabetes medications on PYY and GLP-1

It remains unknown whether the imbalance of the hormones causes obesity or obesity causes the hormonal imbalance, but there is clearly a correlation.

Will these modifications affect hunger or behavior (a lot of these hormones work on the brain for behavioral responses) or energy imbalance? Do these modifications cure obesity and its related co-morbid diseases?

The biggest endocrine organ in the in the body is the small bowel. We don't know the function of many of the hormones it secretes, but it has the biggest number of mediators and transmitters in the neurohormonal system, so it makes sense that altering the small bowel would affect this axis more than anything else.

Surgeries that Modify the Neurohormonal Axis of Energy Metabolism

Can we cure morbid obesity surgically? Current surgeries that we use to try to do so include the traditional switch (**Fig. 7**), sleeve gastrectomy (**Fig. 8**), gastric banding (**Fig 9**), and Roux-en-Y gastric bypass (**Fig. 10**) (all on next page).

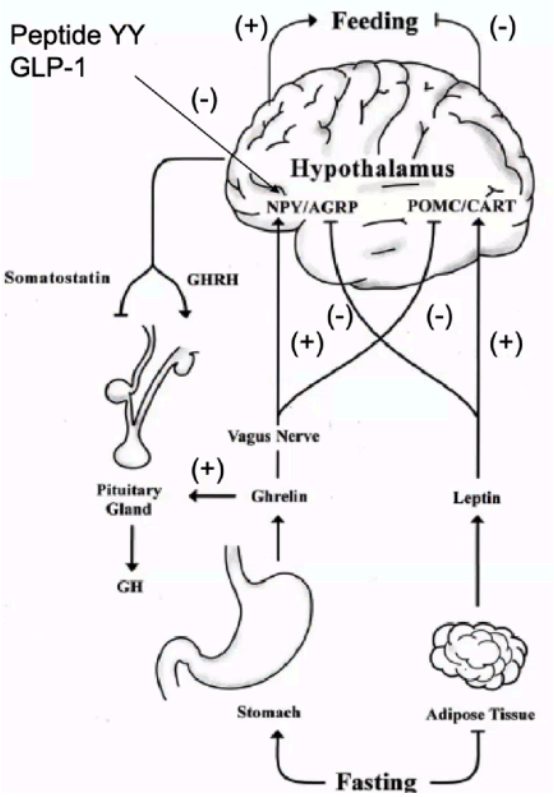

Fig. 6. PYY and GLP-1 effect on the hypothalamus in the metabolic axis

The major differences between the traditional switch and bypass is that the pylorus is in place, there is no pouch, and the excess stomach has been resected. It is called the duodenal switch because the division is right at the first part of the duodenum.

Sleeve gastrectomy was done as a part of the switch in the time of open surgery. It was very morbid to do an open duodenal switch on someone with a BMI of 60, so the protocol was (1) gastrectomy, (2) wait six months for the patient to lose weight, (3) do the switch. Some patients lost enough weight in the six months that they never needed the duodenal switch.

Gastric bypass is still the gold standard of bariatric surgery. It is easier and has good outcomes, but this is the one that has both restrictive and metabolic and most of our studies are

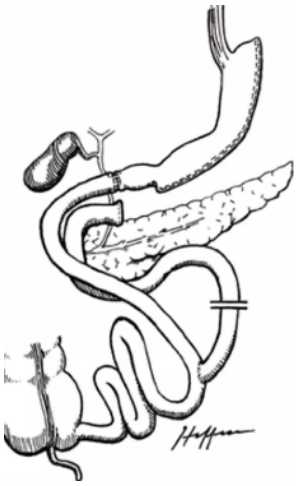

Fig. 7. Biliopancreatic diversion (BPD): Partial gastrectomy with duodenal switch

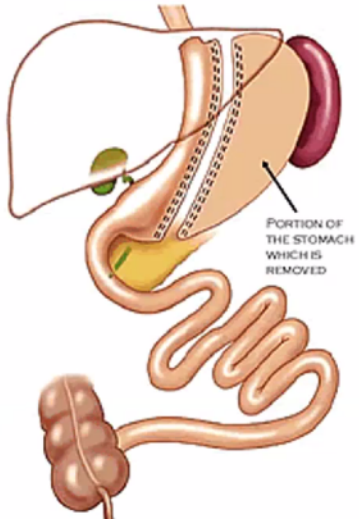

Fig. 8. Sleeve gastrectomy

Source: Laparoscopic Association of San Francisco

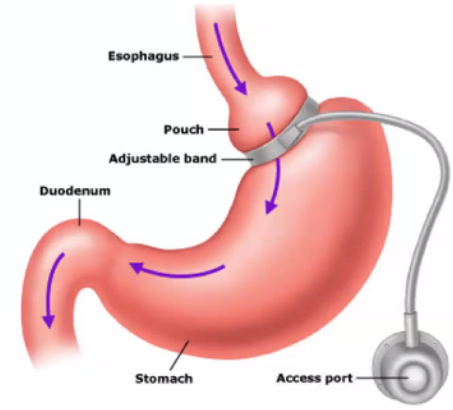

Fig. 9. Adjustable gastric banding

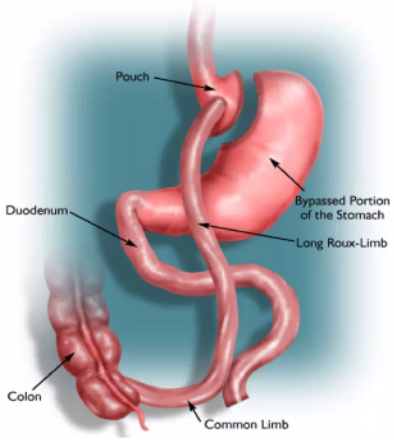

Fig. 10. Roux-en-Y divided gastric bypass

based on this, because it is not as dramatic as a switch with the side effects and it is not only restrictive, like some other operations—gastric bypass, obviously.

Gastric banding does not modify the axis and is no longer used much, though some people benefit from it. It is not alone in losing effect over time and it has a lot of complications. It is purely restrictive and does not affect the axis much.

Roux-en-Y gastric bypass does affect the axis, in ways not fully understood, and most studies are inconsistent.[1] Following the procedure, ghrelin may be elevated, unchanged, or lower. Postprandial levels, which should decrease, were found in some studies to be blunted or exagger-

1 Saliba, J. *et al*. *Current Opinion in Clinical Nutrition & Metabolic Care 2009*. 12:515-521.

ated. But seven out of eight studies had only looked at total ghrelin, not the active form alone, and it is the active form that affects the axis. A recent study demonstrated for active acylated ghrelin showed decreased basal and no change in postprandial levels—the patients tended not to get very hungry so did not eat as much.

GLP-1 starts to go up one month to four years after Roux-en-Y gastric bypass because the body's homeostasis mechanism tries to get it back to its former state and some of these hormones go back to their previous levels. This is why, in most cases, the surgeries lose their strong effect over time. Medical weight loss patients do not have this problem. It correlates to people with diabetes resolution: Patients who gain more GLP-1 after bariatric surgery are more likely to be cured of their diabetes. It is thought to be a major player in type 2 diabetes and some medications have been formulated accordingly.

Peptide YY's postprandial response is blunted in obese individuals. After bypass, the postprandial response goes up like a normal (non-obese) patient. It plateaus after a year. **Fig. 11** below shows two groups of gastric bypass patients—one with good response, the other with poor response to the bypass, and their correlation to levels of peptide YY and GLP1. The lower the peptide YY or GLP1, the lower the response to gastric bypass, thus confirming that the procedure is more metabolic than restrictive.

Sleeve is mostly reductive or restrictive, but it does have some hormonal effects. Active ghrelin goes down after bypass. It decreases appetite. The ileal brake system (peptide YY and GLP1) gets exaggerated after gastric bypass as a result of increased nutrient presentation to the distal ileum and there is increase in the vagal stretch receptors that stimulate the hypothalamus to experience satiety because the stomach is smaller (that is the case also with sleeve).

Getting Back to Normal?

The main goal in treating obesity is to go from comorbidity, disease, inactivity, embarrassment, and early death to a healthy, active, engaged, enhanced quality, and longer life. The fact is that patients who have these two operations have complications. That is why duodenal switches are no longer needed. An ABSITE question that still comes up is: "What is the most effective bariatric surgery for diabetes?" The answer is: The duodenal switch. "Then why don't we do it?" Because we have to deal with its other consequences, which include:

1. *Protein deficiency*
   - Up to 15% of post-op patients are clinically deficient
   - Hypoalbuminemia, alopecia, edema
   - 1.2 g/kg/day post op requirement

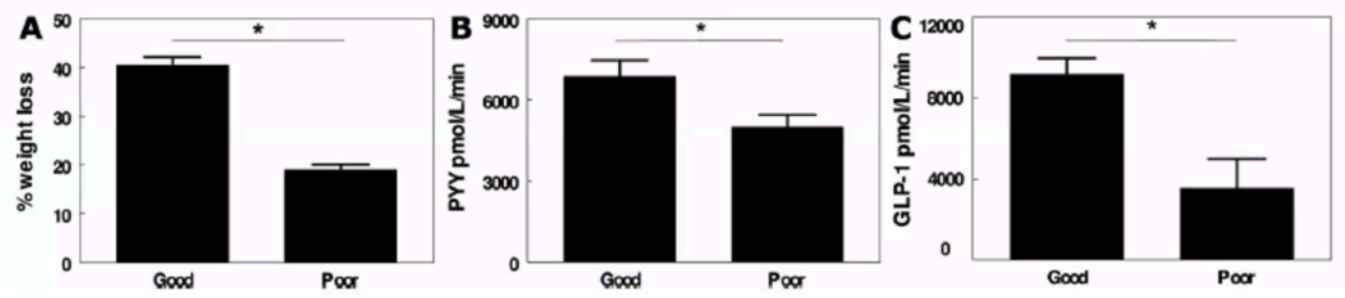

Fig. 11. Hormonal responses to good vs. poor outcomes of gastric bypass. A: Total body weight loss after gastric bypass surgery in good and poor weight loss groups; B and C: PYY and GLP-1 responses after a standard meal of 400 kcal in the good and poor weight loss groups. *$P < 0.05$.

2. *Dumping syndrome*
   - Early: nausea, vomiting, bloating, dizziness, weakness, fatigue, diarrhea
   - Late: weakness, dizziness, sweating
   - High osmotic load into the intestines
   - TX: diet modification, somatostatin, distal pancreatectomy for severe hypoglycemia

3. *Vitamin and mineral deficiencies* in iron, vitamin BI2, vitamin BI (thiamine), folate, calcium, and selenium/zinc: Bypass patients need to take all of these, life-long.

The Good News

The good news, compiled from 361 primary studies involving 85,048 patients in all,[2] is, first, that the procedures are relatively safe. (See **Table 1 below and Table 2** on the next page.)

Second, they are often a cure for type 2 diabetes. A major study published in 2016 in the *Journal of Diabetes—Endocrinology* included bariatric surgery in the algorithm for the first time, and recommended that patients likely to develop type 2 diabetes should have prophylactic surgery early before comorbidities set in.

Overall, a meta-analysis of the various studies found an 85% cure rate at 1-month follow-up and 85-90% at 15-year follow-up. For patients with BMI < 35: the rate was 90%. Bypass patients

	Death ≤30 days				*Death >30 days to 2 years*			
Surgery type	*t*	n/N	%	*Meta-analysis, Mean (95% CI)*	*t*	n/N	%	*Meta-analysis, Mean (95% CI)*
Total	475	310/84,931	0.4	0.28 (0.22-0.34)**	140	155/19,928	0.8	0.35 (0.12-0.58)**
Gastric banding								
Open	16	4/1,319	0.3	0.18 (0.00-0.49)	1	0/35	0.0	0.00 (0.00-3.93)
Laparoscopic	87	19/17,644	0.1	0.06 (0.01-0.11)	27	1/5,145	0.0	0.00 (0.00-0.08)
Gastroplasty								
Open	63	27/7,768	0.3	0.33 (0.15-0.51)	14	2/606	0.3	0.23 (0.00-0.86)
Laparoscopic	19	3/1,652	0.2	0.21 (0.00-0.48)	7	0/299	0.0	0.00 (0.00-0.86)
Gastric bypass								
Open	66	51/9,677	0.5	0.44 (0.25-0.64)	24	34/3,171	1.1	0.69 (0.03-1.35)**
Laparoscopic	76	42/19,610	0.2	0.16 (0.09-0.23)	34	8/6,682	0.1	0.09 (0.00-0.18)
BPD/DS								
Open	24	53/5,588	0.9	0.76 (0.29-1.23)**	6	29/1,719	1.7	0.85 (0.00-1.97)
Laparoscopic	5	4/539	0.7	1.11 (0.00-2.70)*		—		—
Revision/reoperation								
Open	22	17/1,134	1.5	0.96 (0.09-1.82)**	3	0/74	0.0	0.00 (0.00-3.14)
Laparoscopic	9	0/221	0.0	0.00 (0.00-1.47)	2	1/56	1.8	1.65 (0.00-6.25)
Restrictive								
Open	82	32/9,203	0.3	0.30 (0.15-0.46)	16	2/660	0.3	0.22 (0.00-0.84)
Laparoscopic	114	23/19,503	0.1	0.07 (0.02-0.12)	39	2/5,621	0.0	0.01 (0.00-0.09)
Restrictive/malabsorptive								
Open	83	61/12,263	0.5	0.41 (0.24-0.58)	24	34/3,171	1.1	0.69 (0.03-1.35)**
Laparoscopic	76	42/19,610	0.2	0.16 (0.09-0.23)	34	8/6,682	0.1	0.09 (0.00-0.18)
Malabsorptive								
Open	24	53/5,588	0.9	0.76 (0.29-1.23)**	6	29/1,719	1.7	0.85 (0.00-1.97)
Laparoscopic	5	4/539	0.7	1.11 (0.00-2.70)*	—	—	—	—

%, percent of patients with mortality; *BPD/DS*, biliopancreatic diversion/duodenal switch; *CI*, confidence interval; *n*, number of patients with mortality; *N*, number of patients in groups reporting mortality; *t*, number of treatment groups reporting mortality.
*$P < .10$ for test of heterogeneity of outcome.
**$P < .01$ for test of heterogeneity of outcome.

Table 1. Short term versus long term death after bypass and band.

[2] Buchwald, H et al. Surgery. 2007. 142(4): 621-35.

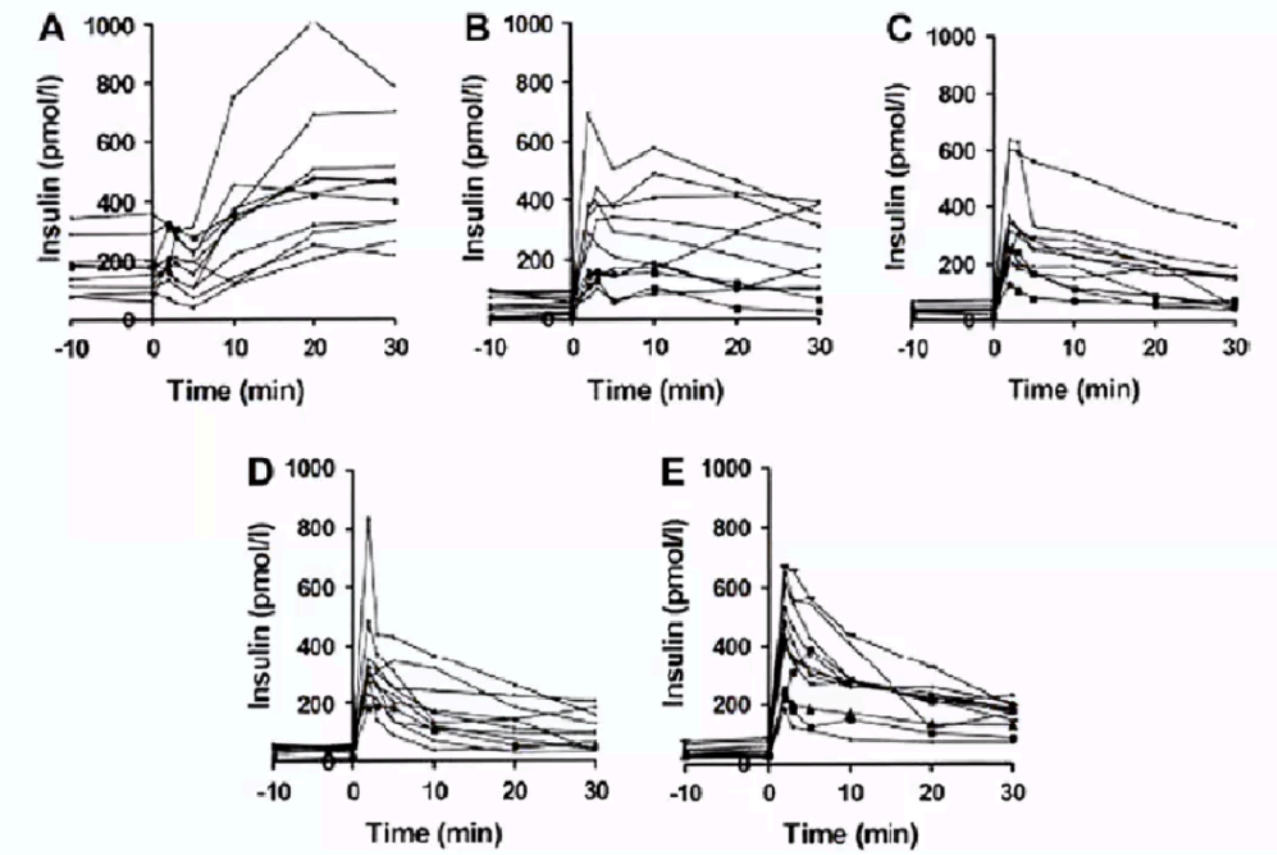

Fig. 12. Recovery of the first phase insulin response after gastric bypass.
Source: Polyzogopoulou EV, Kalfarentzos F, Vagenakis AG, et al. Restoration of euglycemia and normal acute insulin response to glucose in obese subjects with type 2 diabetes following bariatric surgery. Diabetes 2003;52:101.

also had greater glycemic control than weight loss independently (medical, banding).[3] **Fig. 12** (next page) shows recovery of the first phase insulin response after gastric bypass. Insulin response after intravenous glucose infusion in normal control subjects (E) and patients with type 2 diabetes, before gastric bypass (A), at 3 months (B), 6 months (C), and 12 months (D) after gastric bypass. At 3, 6, and 12 months after bypass, the insulin response drops close to that of a non-obese patient without diabetes (E).

	Death < 30 d			Death > 30d to 2 yr		
Surgery Type	n	N	%	n	N	%
Laparoscopic Gastric Banding	19	17644	0.01	1	5145	0.0
Laparoscopic Gastric Bypass	42	19610	0.2	8	6682	0.1

Table 2. Death following bariatric surgery
Source: Buchwald, H *et al*. *Surgery*. 2007. 142(4): 621-35.

[3] Thaler and Cummings. Endocrinology. 2009. 150: 2518-25

Author	Diabetes mellitus	HTN	GERD	Sleep apnea	Elevated cholesterol/ lipids	Joint pain	Stress urinary incontinence
DeMaria et al. (17)	93	52	95			76	88
Rutledge (13)	92	90	77	90	93/100	72	81
Schauer et al. (8)	100	88	96	93	96/86	88	89
Wittgrove and Clark (9)	98	92	98	98	97/99	90	97

Abbreviations: HTN, hypertension; GERD, gastroesophageal reflux disease.

Table 3. Percent Improvement or Resolution of Obesity Comorbidities After Laparoscopic Gastric Bypass
Source: Sugerman, HJ and Nguyen, NT, eds. *Management of Morbid Obesity*, 2006. p. 147.

Several reasons have been proposed to explain why type 2 diabetes is cured. One is that the patient starves, so loses weight. It is possible of course to starve without bypass, but most people can't stick to a starvation diet. Another is that ghrelin blocks adiponectin, insulin signaling, and insulin secretion. A lower intestine hypothesis based on the ileal brake (GLP-1/PYY) and an upper intestinal hypothesis, mostly regarding sleeve gastrectomy, have also been proposed to account for the cure .

Table 3 above, based on multiple studies, shows the resolution rates for different comorbidities. Diabetes resolution of 100% was reported in one of the studies. Clearly, bypass has a very good effect on comorbidity resolution.

Fig. 13 presents a comparison of weight trends over 15 years among non-surgical, gastric bypass, band, and gastroplasty patients.

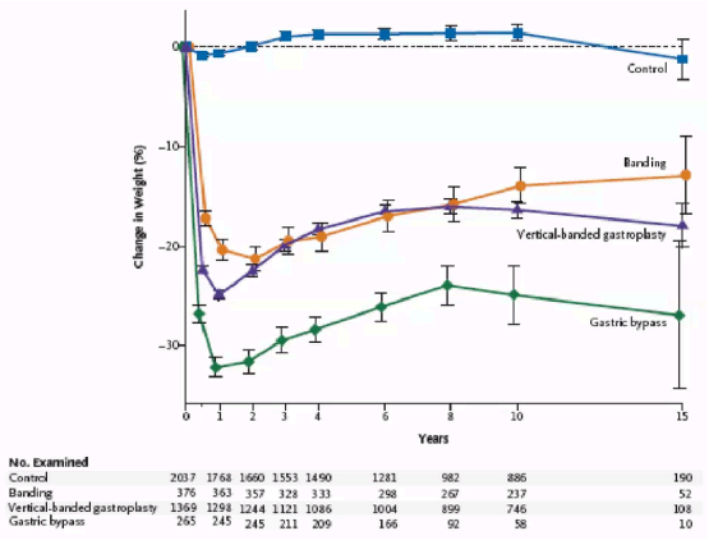

Fig. 13. Mean percent weight change during a 15-year period in the control group and the surgery group, according to the method of barlatric surgery.

I bars denote 9596 confidence intervals.

Summary
Bariatric surgery can cure some of the deadly problems. Even after 15 years, some of the co-morbidities are gone, even if the patient regains some weight. The physiology is far more complex than "You eat too much, so you're fat," and "You're fat, so you have diabetes." Stated more formally: Malabsorptive and reductive bariatric surgery affords the morbidly obese patient the best chance to cure deadly co-morbid diseases.

The physiology of morbid obesity and bariatric surgery is a complex web of genetic, behavioral, and neurohormonal factors. It is a web we are just beginning to understand.

Part 2: Long-term Complications

Long-term complications of bariatric surgery are mostly related to bypass because of the malabsorptive component, and some are specific to band:

- Protein and vitamin deficiencies and electrolyte imbalances.
- Hypocalcemia (uncommon, but if it occurs it may require a bypass reversal).
- Chronic abdominal pain, nausea, vomiting (even without any reason some people have to have their bypasses reversed because of that, or they had sleeve and have to convert to something else).
- Dumping syndrome and chronic hypoglycemia. Dumping syndrome can be persistent even if diet is controlled.
- Staple line and/or anastomotic (marginal) ulcers.
- Gasro-gastric fistula.
- Internal hernias (any person with gastric bypass history with abdominal pain. needs a CT scan because a missed internal hernia can lead to dead bowel).
- Intussusception.
- Strictures.
- Band complications (General surgeons need to know how to deal with bands because many have been placed and are likely to present at any time).

Specific examples from actual cases include a mesenteric swirl in a bypass patient (**Fig. 14**). The mesenteric swirl is especially malignant in bypass patients and should be operated upon. Any internal hernia needs prompt attention in any small bowel resection. Bypass patients lose fat from the mesentery, develop a hernia and an internal hernia defect and the swirl results. But note that this is not just a swirl. Most of the anastomoses—if they're done with staples or clips placed for further studies—show up on CT. The JJ should be seen in the left upper quadrant. A CT of a patient with abdominal pain with a history of bypass showing JJ in the right *lower* quadrant might indicate internal hernia.

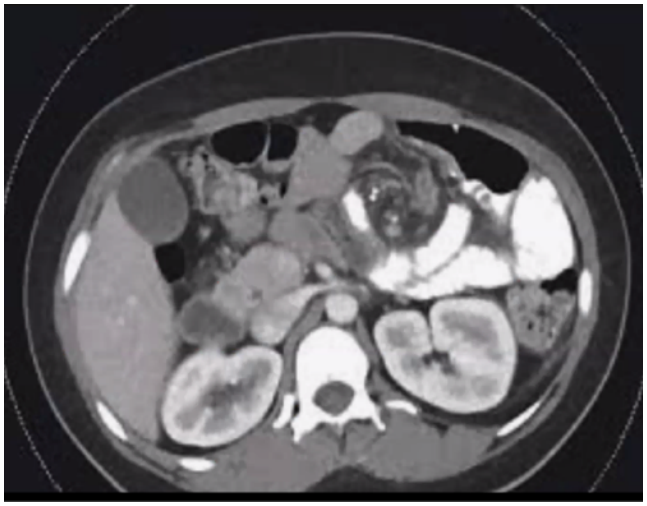

Fig. 14. Internal hernia

Be aware that the remnant stomach should be collapsed. If it is full of fluid or has gas in it, there is some problem past the JJ—an obstruction or an internal hernia, which can be deadly in these patients. The hernias are classified into various types of retro-Roux and Petersons Defect, sometimes seen in retro-colic bypasses. Hernias can cause volvulization, ischemia, and sepsis.

Treatment, clearly, is surgical. In the anti-colic operation, the Roux limb is brought over the colon, leaving a space that can potentially cause a retro-Roux hernia. It is sometimes misidentified as Peterson's Defect. With retro-Roux, Peterson's hernia can break through the mesentery, the true retro room behind the Roux limb, resulting in a jejuno-jejunostomy mesenteric defect, which should be treated like any other small bowel obstruction.

Fig. 15 (next page) shows a gastrojejunostomy on what is loosely termed a marginal ulcer, but it is always on the jejunal side. The presence of a pouch indicates a gastric (peptic) ulcer. It is true that bypass decreases acid therefore it treats a lot of problems; however, this part of small bowel is 150 centimeters closer to the contents of the

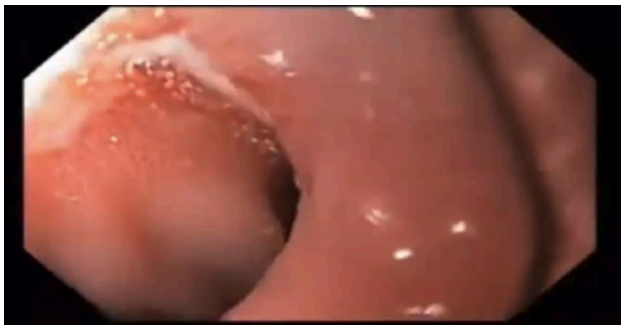

Fig. 15. Marginal ulcer

GI tract than it was before bypass, so even a small amount of acid may affect it and cause ulcers. For this reason, PPIs are still indicated for three months post-operatively because the small bowel is no longer getting the quantity of bicarbonate from the pancreas that it used to get.

Staple line ulcers can occur with the sleeve, anastomotic ulcers (usually marginal, on the jejunal side) with bypass. Smoking and NSAIDs are the most common reasons for seemingly inexplicable ulcers, but they can occur for no apparent reason. They may require endoscopic intervention, medications, and sometimes an anastomosis may need revision. Surgery is clearly indicated when there are acute complications such as bleeding and perforation. Fistula and stricture are chronic complications.

In post-gastric bypass patients an upper GI may show what you see in **Fig. 16**. There's a fistula, because that part of stomach should be excluded. So it's a gastro-gastric fistula. Usually it is after having long-term peptic or marginal ulcers, or gastric ulcers, which chronically find their way to the other side, that will separate it. Patients may complain: "I'm regaining weight. I was doing everything right. I had belly pain for a while. Now the pain is better, but I'm gaining weight and my diabetes is back." This is basically a reversal of bypass, and a telltale sign, even if they don't have pain, even if they don't have ulcers any more, because people put them on PPIs. They don't have pain any more but there is a fistula therefore the bypass is bypassed and now you

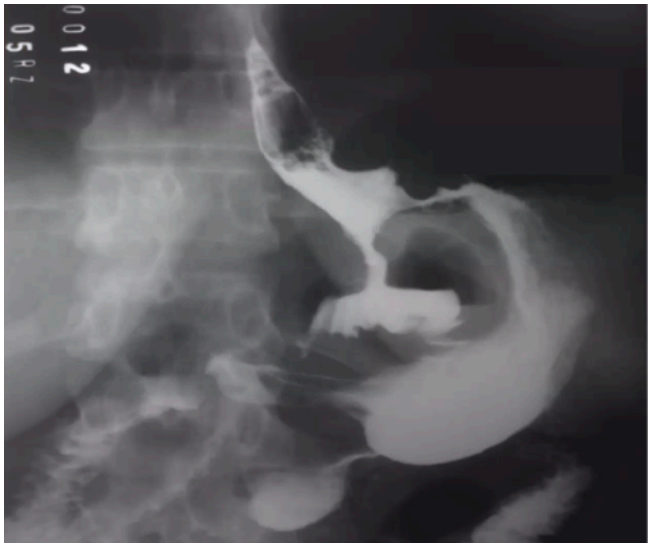

Fig. 16. Gastro-gastric fistula

have everything creeping back up. If an upper GI or endoscopy reveals a gastro-gastric fistula, it must be treated.

Fig. 17 shows an anastomosis (a leak) with air and fluid surrounding it. It could be acute, it could be chronic following an abscess and showing up later, or could be delayed from an ulcer. Most commonly seen chronically is a marginal ulcer that perforates.

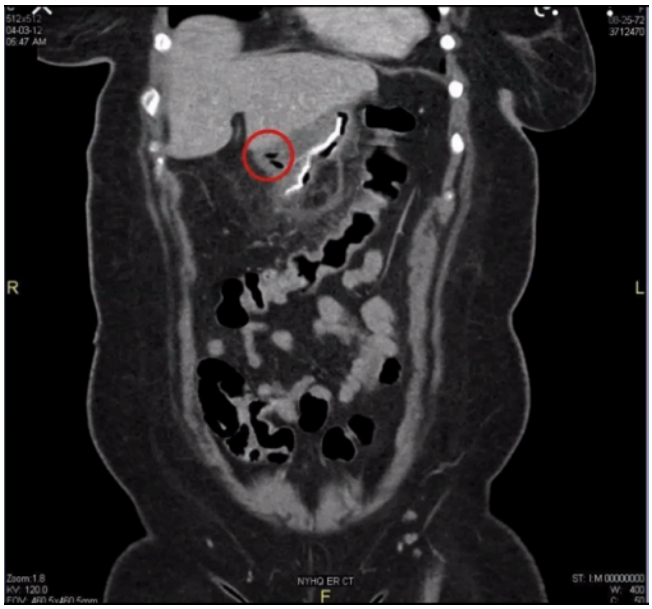

Fig. 17. Anastomotic leak

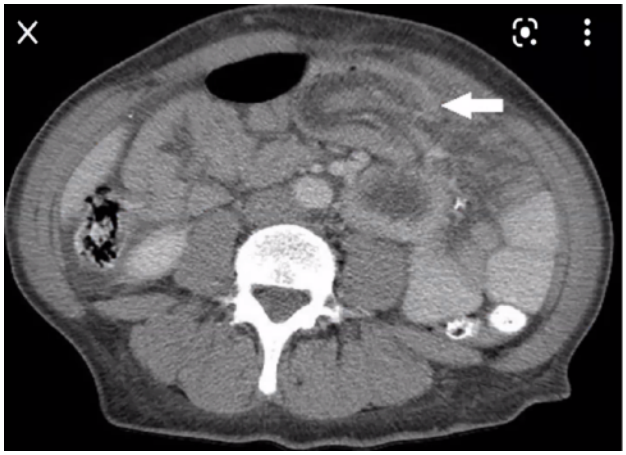

Fig. 18. Intussusception

Fig. 18 above is an intussusception, commonly seen following gastric bypass, especially in cases with a wide JJ anastomosis. Whether it is acute with symptoms or chronic, it must be addressed because there may be an obstruction, or ischemia. Recurrent intussusception may need revision but sometimes pexy may solve the problem.

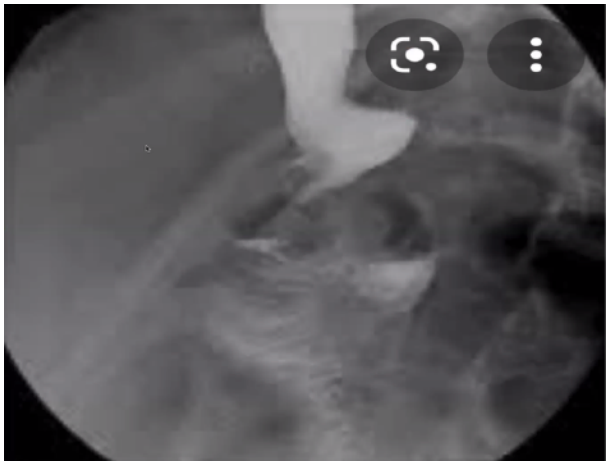

Fig. 19. Anastomotic stricture

Fig. 19 above is a bypass patient. The esophagus, pouch, and Roux limb are visible. The patient has dysphasia. It is a stricture, an anastomosis, not uncommon, especially with people who have marginal ulcers. Surgeons who hand sew, do not use a standardized bougie, or use small size EEA staplers (to increase wight loss) tend to cause more strictures. Ballooning is a possible solution, or it may be necessary to operate.

Fig. 20 is important. A patient who presents at a community hospital with this will die unless the surgeons there are prepared to act. The lap-banded patient is vomiting profusely, his creatinine is elevated, he is dehydrated. The band has slipped. If the angle does not point towards the patient's right hip, it has slipped. If the patient does not begin to recover, the band needs to come out.

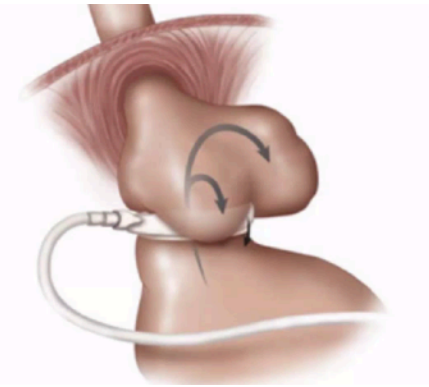

Fig. 20. Slipped band

Fig. 21 is an endoscopy of a lap-band patient who presented with chronic abdominal pain. Erosion has found its way all the way into the stomach. In endoscopic resection of the band, the surgeon should remove only what is visible and leave the hole because there is a capsule around it which will prevent any leak.

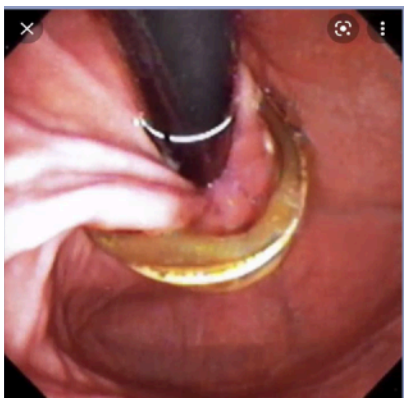

Fig. 21. Band erosion

Addendum: Recent Developments in Bariatric Surgery

by

Alyssa Stroud, MD

PGY 5 resident and
2023 Minimally Invasive Bariatric Fellow
Wayne State University School of Medicine

Two new procedures were introduced relatively recently: The Single Anastomosis Duodeno-ileal Switch (SADI-S) and One Anastomosis Gastric Bypass (OAGB).

a. **SADI-S**

In a way, SADI-S, simplifies the biliopancreatic diversion with duodenal switch (BPD-DS) procedure. It is both a malabsorptive and restrictive procedure. The components of the operation are a vertical sleeve gastrectomy followed by a transection of the proximal duodenum about three to four centimeters distal to the pylorus. A loop duodenal ileostomy is then created about 300 centimeters from the ileocecal valve.

The procedure is intended to address the complexities and the limitations of BPD DS, which tend not to result in significant weight loss and can even result in weight re-gain. BPD DS is also lengthy and technically difficult, and may lead to significant malabsorptive complications and nutritional deficiencies.

The left-hand panel in **Fig. A-1** below depicts the classic BPD DS. It shows an anastomosis at the duodenal ileostomy. A distal entero enterostomy creates the BP limb and the common channel. In contrast, in SADI-S, a sleeve gastrectomy is followed by a single anastomosis—the duodenal ileostomy, which is about 300 centimeters of common limb proximal to the TI.

Outcomes data for SADI-S are still limited to two studies. One was a prospective multi-center study designed to evaluate the weight loss, comorbidity resolution, and 1-year nutritional out-

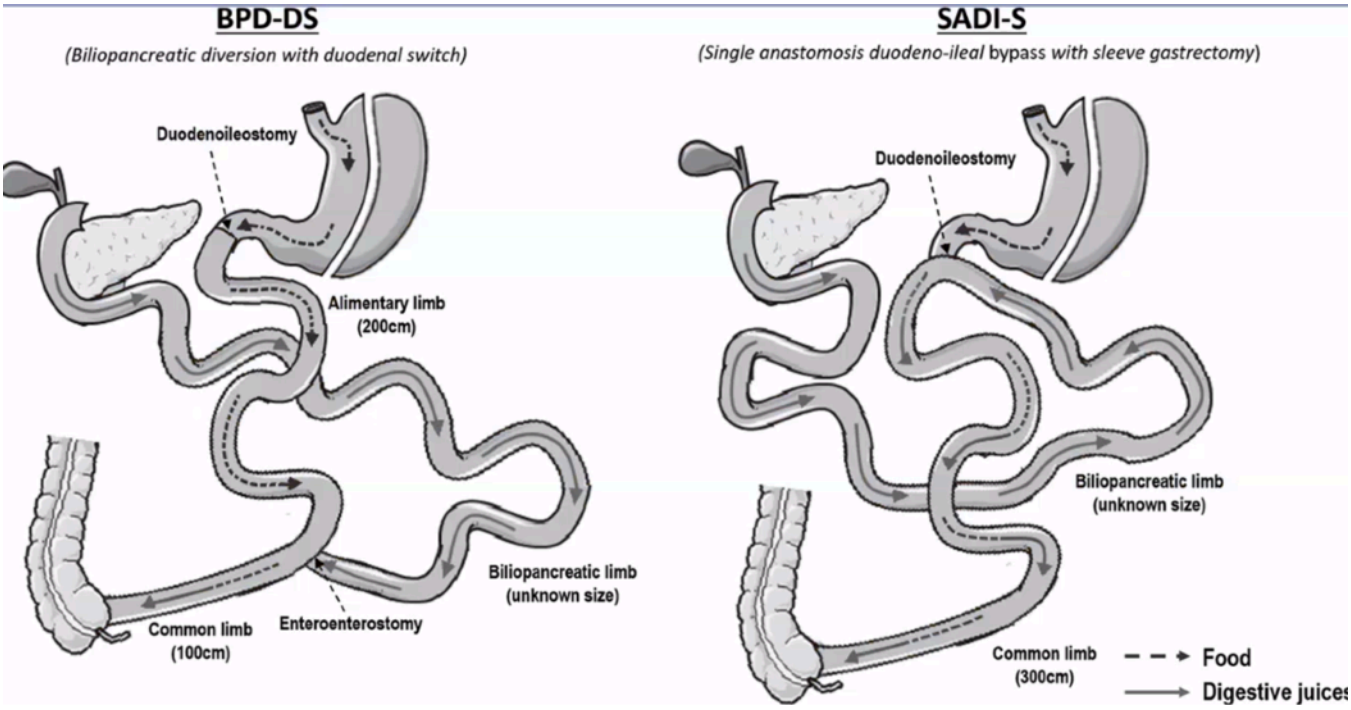

Fig. A-1. Single Anastomosis Duodenal Switch vs. Biliopancreatic Diversion with Duodenal Switch

comes of SADI-S. It ran from October 2014 through January 2017 and involved 120 patients at six sites across the US. The primary endpoint was weight loss as seen in percent excess weight loss, percent total body weight loss and final BMI at 1 year.

The study found that at 12 months, SADI-S patients showed significantly reduced BMI when compared to baseline (46.8 ‡ 5.8 vs 29.8 4.4, P < 0.001 respectively).

Secondary end points included resolution of obesity associated co-morbidities. The study found:

- 96.3% of the patients had a resolution of type 2 diabetes by 12 months with a mean A1C
- reduction from 7.8 ‡ 1.6 to 5.3 ‡ 0.7
- reductions in hyperlipidemia, sleep apnea, and hypertension at 12 months
- gastroesophageal reflux disease satisfaction and quality of life (SF-36) scores were significantly
- higher at 12 months postprocedure (P < 0.001 in all cases)
- There were abnormalities of parathyroid hormone and vitamin D at 1 year with all other
- nutritional markers being not significantly different at 1 year from baseline

An early comparison of 54 SADI-S and 54 RYGB patients showed the groups had statistically similar weight loss at 18 months (39.6 versus 41% total weight loss, respectively). Analysis of 53 SG patients and 53 SADI-S patients with 300-cm absorptive limbs (all with a 40-Fr SG) showed early weight loss, which seems to be related to the SG component, while the intestinal component extended the period of ongoing weight loss by several months, thus, increasing the total %EWL significantly.

Complication rates were noted to be similar between the 2 groups, with most complications being related to the SG component of each procedure.

The study center's matched-cohort analysis comparing 61 SADI-S patients (40-Fr SG/300-cm absorptive limb) with 61 BPD-DS patients (40-Fr SG/150-cm Roux limb/150- cm common channel) at 2-year follow-up showed %EWL and rate of complications was statistically identical between the 2 procedures, but the mean operative time for SADI-S was significantly shorter (70 6 14 versus 137 6 36 min)

The American Society of Metabolic and Bariatric Surgery (ASMBS) concluded that SADI-S provides for similar outcomes to those reported after classic DS and should therefore be endorsed, similar to the ASMBS' endorsement of the predicate procedure of BPD-DS; and its conclusion from the current review was that the currently available peer-reviewed literature does not suggest outcomes will differ substantially from those seen with classic DS.

b. OAGB

The other procedure is called a one anastomosis gastric bypass (OAGB) or a mini gastric bypass. This is also a malabsorptive and restricted procedure. It may be thought of as a mini or simplified version of a Roux-en-Y. It too is a single anastomosis. A gastric pouch is followed by a gastrojejunostomy approximately 200 centimeters from the ligament of Treitz.

OAGB is promoted as a technically easier and safer alternative to the Roux-en-Y gastric bypass, and in 2011, it compromised 1.5% of all bariatric surgeries. So there's actually quite a bit of publications on this— 69 publications with over 30,000 patients. The mean operative time is a little bit shorter, but it ranges from 86 to 110 minutes (one study actually said that their average was 36 minutes.)

The current literature on OAGB comprises 69 publications with over 38,000 patients. Findings have included:

- Mean operative times ranging from 86 to 110 minutes, varying with obesity class
- Early complication rates range from 3.5 - 7.5%
    - One prospective, randomized study comparing OAGB with RYGB found significantly lower early complication rates with OAGB (7.5% versus 20%, respectively)
    - Mortality was rare (<0.5%) and comparable to other bariatric operations)
- Weight loss outcomes appear to be comparable to RYGB with most series reporting 68.6% to 85% %EWL at 5-year follow up
    - One 10-year retrospective series reported higher %EWL with OAGB vs. TYGB at 5 years (72.9% versus 60.1%, p<0.5)
    - Some studies have demonstrated superior weight loss with OAGB compared with sleeve gastrectomy, while others have reported similar weight loss between the two
- Initial evidence suggests OAGB is similar to RYBG in its ability to induce remission of T2DM
    - Some series have reported diabetes remission rates as high as 93.2% at 6-year follow up
    - Several studies have shown a favorable impact of OAGB on hypercholesterolemia, hypertriglyceridemia, hypertension, GED, and OSA

-

The long-term complications are similar to Roux-en-Y gastric bypass—more marginal ulceration, bile reflux, hypoabsorption, and protein calorie malnutrition, and of course, internal hernias. The study commented that malabsorption and nutritional deficiencies could be more severe due to the longer bypass limb length (200 centimeters in OAGB, 50-100 in RYGB. Internal hernia rates have been reported to be lower in some studies and the rates of bile reflux are anywhere from 0.9 to 4%.

The ASMBS concluded that OAGB has a short operative time, low complication rate, and excellent weight-loss outcomes, but that most of the studies were retrospective and lack long-term follow-up, which limits the current evidence particularly in regards to the long-term nutritional deficiencies due to the hypoabsorptive nature of the procedure and issues specific to the loop formation of the loop gastroenterostomy which includes bile reflux and its potential long-term effects on carcinogenesis.

* * *