Bariatric Surgery for Obesity and Type 2 Diabetes
Sangeeta R. Kashyap, MD
Sunday, March 6, 2016
11:30 a.m. – 12:15 p.m.

Obesity has reached epidemic levels in the world and accelerates the development of metabolic diseases including Metabolic Syndrome, Pre-diabetes and Type 2 Diabetes. Obesity is strongly linked to insulin resistance and subsequent hyperinsulinemia that promotes the mitogenic effects of insulin in the development of various cancers, CAD and NAFLD/NASH. This presentation will provide an overview of the therapeutic benefits of various bariatric surgery procedures for the treatment of severe obesity and type 2 diabetes. Data from the Swedish Obesity Study (1,2) that compared bariatric procedures to conventional lifestyle therapy demonstrate reduction in mortality of 30% related to reduction in cancer and CAD deaths as well as, 10 year durable reduction in body weight and co-morbidities of obesity following surgery. Data from the Cleveland Clinic STAMPEDE trial (3) and others(4,5) demonstrate the superiority of bariatric procedures to intensive medical therapy for the treatment of type 2 diabetes in moderate and severe obesity. Other topics covered include mechanisms of action of bariatric surgery to restore beta cell function in type 2 diabetes and determinants of remission vs. non-remission of diabetes post-surgery.

Improvement of glycemic control following bariatric surgery must be considered in the setting of weight independent mechanisms (ie. Incretin hormone stimulation, bile acids, gut microbiota)(6,7) and those related to massive weight loss with subsequent effects on reduction of lipotoxicity, ectopic lipids and adipokines. In some cases, a dramatic lowering in glucose levels has been observed days to weeks following surgery, before major weight loss but in the setting of enforced caloric restriction (8,9). Persistent cases of diabetes after this initial period gradually improve in parallel with weight loss(10). Patients who undergo RYGB and BPD procedures improve sooner and maintain glucose control for longer periods than do patients treated by banding or sleeve gastrectomy procedures. However compared with patients following a very low calorie diet who achieved similar weight loss, patients who underwent RYGB have increased secretion of insulin, c-peptide, glucagon like peptide (GLP-1) and gastric inhibitory peptide (GIP) after oral glucose challenge consistent with an incretin effect(8). Over-stimulation of the incretin axis (primarily GLP-1) has been noted to underlie exaggerated prandial insulin responses leading to post-RYGB hypoglycemia (11) that may be reversed by gastrostomy tube feeds (12), suggesting that altered nutrient-intestinal transit rather than β-cell hyperplasia (13) may be responsible for this under-recognized condition.

References:


Metabolic Effects of Bariatric Surgery

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Associate Professor of Medicine
Cleveland Clinic Lerner College of Medicine
Department of Endocrinology, Diabetes and Metabolism

ADA Post-graduate course 2016, San Francisco

Objectives

• Recognize the efficacy and safety of bariatric surgery for type 2 diabetes from randomized clinical trials
• Identify the mechanisms of action (GI hormones/physiology) of bariatric surgery on glucose metabolism
• Identify clinical and metabolic determinants of diabetes remission following surgery

Etiology of Obesity-Complex and Multifactorial

Genetic and epigenetic influences-70%
Acquired: 30%
  Increased caloric intake
  Biological influences of hormones (leptin, adiponectin etc.)
  Gut microbes
  Imbalance of signals related to energy regulation (gut/adipose vs. hypothalamic)

Presenter Disclosure Information

In compliance with the accrediting board policies, the American Diabetes Association requires the following disclosure to the participants:

Sangeeta Kashyap
Research Support: Ethicon/Covidean
Employee: none
Board Member/Advisory Panel: none
Stock/Shareholder: none
Consultant: Ethicon
Other: na

Morbid Obesity in the 1700’s

Etiology of Obesity-Complex and Multifactorial

Regulation of Food Intake

Central Signals

External factors

Stimulate

NPP
Oxynt-A
AGRP
Dynorphin galanin

Inhibit

CCK
GLP-1
Apo-A-IV
Vagal afferents
Insulin
Leptin
Cortisol

Food characteristics
Lifestyle behaviors
Environmental cues

Peripheral signals

Gastrointestinal tract
Peripheral organs
Adipose tissue
Adrenal glands
Intake

Food

Glucose

Environmental cues

Dr. Hunterian Museum, Royal College of Surgeons, London UK

Brain

NPY
AGRP
dynorphin
galanin

Orexin-A
dynorphin

Stimulate

α-MSH
CRH/UCN
GLP-1
CART
NE
5-HT

Inhibit

External factors

Emotions
Food characteristics
Lifestyle behaviors
Environmental cues

Peripheral signals

Gastrointestinal tract
Peripheral organs
Adipose tissue
Adrenal glands
Intake

Food

Glucose
Characterization of \( \beta \)-cell Failure in T2DM From UKPDS

Factors associated with progression of pre-diabetes to diabetes:
- Elevated FPG and increase in FPG
- High BMI
- Weight gain
- Younger age
- High plasma insulin levels
- Decreased insulin response to glucose
- Hypertension
- Poor \( \beta \)-cell function
- Choice of treatment

Illustration of coefficient of \( \beta \)-cell failure over time in relation to A1C:

- Elevated FPG and increase in FPG
- High BMI
- Weight gain
- Younger age
- High plasma insulin levels
- Decreased insulin response to glucose
- Hypertension
- Poor \( \beta \)-cell function
- Choice of treatment

A1C (%)

\[ \text{Coefficient of failure} = 0.47 \text{ A1C \%}/\text{year} \]

\[ R^2 = 0.95 \]

Lipotoxicity

Adipocytes represent a storage depot for energy (i.e., fat). When the capacity of adipocytes to store fat is exceeded, there is an overflow of fat to:

- Muscle → insulin resistance
- Adipocyte → macrophage TNF\( \alpha \)
- Liver → HGP (GN); Steatosis
- Pancreas → insulin secretion
- Arteries → atherosclerosis

COMPARISON OF BARIATRIC OPERATIONS:
The resolution of diabetes is “dose related”

\[ n = 22,094 \text{ patients; 2738 citations 1990-2002} \]

<table>
<thead>
<tr>
<th>Banding</th>
<th>Gastric Bypass</th>
<th>Duodenal Switch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excess weight loss</td>
<td>47.5%</td>
<td>61.6%</td>
</tr>
<tr>
<td>Operative mortality</td>
<td>0.1%</td>
<td>0.5%</td>
</tr>
<tr>
<td>Resolution of diabetes</td>
<td>47.8%</td>
<td>83.6%</td>
</tr>
</tbody>
</table>

JAMA 2004;292:1724-1737

Weight loss in the SOS

Figure 1. Mean Weight Change Percentages From Baseline for Controls and the 3 Surgery Groups Over 20 Years in the Swedish Obese Subjects Study

Buchwald, Avidor, Braunwald, Jensen, Pories, Farbach, Schoelles

JAMA 2012;307(1):56-65
Bariatric Surgery is Associated with a Reduced Mortality: the SOS Study

Sjostrom L. NEJM 2007: 357-741-752

- 30% lower risk of dying
- MI: 25 in control, Group 13 in the Surgery group
- Cancer: 47 in the control group, 29 in the surgery group


“SOS STUDY”

- 30-60yrs
- A1C $\geq$ 7.0%
- A1C = 9.2 $\pm$1.5%
- BMI = 45.2
- DM = 6.1 yrs
- R-Y Bypass, Biliopancreatic diversion
- Fasting BG $<$100mg/dL & A1c $<$6.5% ± drug Rx for 2 yrs

Italian Study  Cleveland Study  Ikramuddin et al.

- 20-60yrs
- A1C $\geq$ 7.0%
- A1C = 8.7 $\pm$1.5%
- BMI = 36.8
- DM = 8.5 yrs
- R-Y Bypass, Sleeve Gastrectomy
- A1C<6.0% for 1 yr

Baseline Surgery

Primary endpoint

Fasting BG $<$100mg/dL & A1c<6.5% ± drug Rx for 2 yrs

Success rate of achieving HbA1c $\leq$ 6%

Secondary

- Change in fasting plasma glucose (FPG)
- Change in BMI
- Change in lipids, blood pressure, hs-CRP
- Change in medications
- Safety and adverse events

Endpoints

Bariatric Surgery vs. Intensive Medical Rx for T2DM:

Bariatric Surgery and Type 2 Diabetes


STAMPEDE Trial: Flow of Patients

- HbA1c $>$7.0%
- BMI 27-43 kg/m2
- Age 20-60 years

218 patients screened

150 randomized

- 50 Intensive medical therapy alone
- 50 Medical therapy plus gastric bypass
- 50 Medical therapy plus sleeve gastrectomy

8 withdrew consent
2 Lost to follow-up

Population for 3-Year Analysis surgery: 49

91% retention
Primary and Secondary Endpoints at 36 Months

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Medical Therapy (n=40)</th>
<th>Bypass (n=48)</th>
<th>Sleeve (n=49)</th>
<th>P Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c ≤ 6%</td>
<td>9%</td>
<td>37.5%</td>
<td>24.9%</td>
<td>&lt;0.001</td>
<td>0.912</td>
</tr>
<tr>
<td>HbA1c ≤ 6% (without DM meds)</td>
<td>0%</td>
<td>35.4%</td>
<td>20.4%</td>
<td>&lt;0.001</td>
<td>0.002</td>
</tr>
<tr>
<td>HbA1c ≥ 7%</td>
<td>40%</td>
<td>64.6%</td>
<td>63.3%</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>Change in FPG (mg/dL)</td>
<td>-6</td>
<td>-85.5</td>
<td>-46</td>
<td>0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>Relapse of glycemic control</td>
<td>80%</td>
<td>23.8%</td>
<td>50%</td>
<td>0.03</td>
<td>0.34</td>
</tr>
<tr>
<td>% change in HDL</td>
<td>+4.6</td>
<td>+34.7</td>
<td>+35.0</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>% change in TG</td>
<td>-21.5</td>
<td>-45.9</td>
<td>-31.5</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>% change in CIMT</td>
<td>0.048</td>
<td>0.013</td>
<td>0.017</td>
<td>0.36</td>
<td>0.49</td>
</tr>
</tbody>
</table>

1 Gastric Bypass vs Medical Therapy; 2 Sleeve vs Medical Therapy

Cardiovascular Medications at Baseline and Month 36

<table>
<thead>
<tr>
<th>CV medications – number (%)</th>
<th>Medical Therapy (n=40)</th>
<th>Bypass (n=48)</th>
<th>Sleeve (n=49)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>None</td>
<td>0 (0)</td>
<td>3 (6.3)</td>
</tr>
<tr>
<td></td>
<td>1 - 2</td>
<td>19 (47.5)</td>
<td>17 (35.4)</td>
</tr>
<tr>
<td></td>
<td>≥ 3</td>
<td>21 (52.5)</td>
<td>28 (58.3)</td>
</tr>
<tr>
<td>Month 36</td>
<td>None</td>
<td>1 (2.5)</td>
<td>33 (68.8)*</td>
</tr>
<tr>
<td></td>
<td>1 - 2</td>
<td>18 (45)</td>
<td>14 (29.2)</td>
</tr>
<tr>
<td></td>
<td>≥ 3</td>
<td>21 (52.5)</td>
<td>1 (2.1)</td>
</tr>
</tbody>
</table>

* P value <0.05 with Medical Therapy group as comparator

Quality of Life

<table>
<thead>
<tr>
<th>Component</th>
<th>Medical Therapy</th>
<th>Bypass</th>
<th>Sleeve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Role limitations</td>
<td>-4</td>
<td>-10</td>
<td>-11</td>
</tr>
<tr>
<td>General Health</td>
<td>-6</td>
<td>-6</td>
<td>-11</td>
</tr>
<tr>
<td>Physical functioning</td>
<td>0</td>
<td>0</td>
<td>-0.2</td>
</tr>
<tr>
<td>Physical health components</td>
<td>0</td>
<td>0</td>
<td>-0.1</td>
</tr>
<tr>
<td>Mental health components</td>
<td>-2</td>
<td>-4</td>
<td>-4</td>
</tr>
<tr>
<td>Social functioning</td>
<td>-2</td>
<td>-2</td>
<td>-4</td>
</tr>
</tbody>
</table>

Adverse Events through 36 Months

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Medical Therapy (n=43)</th>
<th>Bypass (n=50)</th>
<th>Sleeve (n=49)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI complications</td>
<td>2 (5)</td>
<td>13 (26)</td>
<td>5 (10)</td>
</tr>
<tr>
<td>Re-op</td>
<td>0</td>
<td>2 (4)</td>
<td>2 (4)</td>
</tr>
<tr>
<td>Stroke</td>
<td>0</td>
<td>0</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Retinopathy</td>
<td>0</td>
<td>1 (2)</td>
<td>2 (4)</td>
</tr>
<tr>
<td>Nephropathy</td>
<td>4 (9)</td>
<td>7 (14)</td>
<td>5 (10)</td>
</tr>
<tr>
<td>Foot ulcers</td>
<td>0</td>
<td>2 (4)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Excessive weight gain</td>
<td>7 (16)</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Summary

- Bariatric surgery was more effective than intensive medical therapy in achieving glycemic control (HbA1c ≤ 6.0%) with weight loss as the primary determinant of this outcome.
- Many surgical patients achieved glycemic control without use of any diabetic medications (particularly insulin).
- Metabolic syndrome components (HDL, triglycerides, glucose, BMI) showed greater improvement after surgery. Marked improvement in quality of life.
Diabetes Complications?

- Retinal data was negative at 2 years (Singh R et al. ADA scientific sessions, oral presentation 2014).
- Nephropathy/CKD: No worsening of GFR but improvement--Pilot study (N=15, Cr >1.3mg/dl) demonstrate no change in proteinuria, measured GFR (iothalamate testing), Cystatin C, or β2 microglobulin 1 year following surgery (Nanavatil et al. AJD, in press).
- Fractures- Bone density drops by 10%; 31% in surgical and 25% in medical. (Magrabi A et al. ADA scientific sessions, 2013).

Limitations

- Single-center trial – multicenter studies needed to determine if results can be generalized.
- Duration of Follow-up limited to 3 years so far.
- Larger studies will need to determine potential benefit on cardiovascular events and diabetes related microvascular complications.
- Sample size insufficient to detect uncommon but significant complications of surgery or medical Rx.

Bariatric surgery versus non-surgical treatment for obesity: a systematic review and meta-analysis of randomised controlled trials

BMJ Oct 22, 2013

Victoria J. Glynn junior researcher1, Matthias Briel assistant professor1, Deepak L. Bhatt professor2, Sangita Khoshyap associate professor of medicine3, Philip H Schauer medical director, professor of surgery4, Gertrude Mengrone professor5, Heiner C Bucher director, Alan J Nordrann associate professor1

- 11 studies, 796 patients, BMI 27-53
- Surgery superior to med Rx
  - Wt. loss, HbA1c, T2DM remission, TG, HDL, remission of metabolic syndrome, QOL, medication reduction
  - No difference in BP or LDL
  - No CV events or death after surgery
  - Anemia (15%), Reoperation (8%)

Mortality Rates (%) of 8 Procedures in Diabetics, 2008-2012

- CABG
- Infrainguinal Bypass
- Lap Colectomy
- Lap Cholecystectomy
- LRYGB
- Knee Arthroplasty
- Lap Hysterectomy

Mortality Rates of LRYGB in 1000

- Hyperinsulinemic hypoglycemia
- Osteomalacia/osteoporosis and bone fractures
- Kidney stone (calcium oxalate)
- Other vitamin and mineral deficiencies (iron, B12, vitamin d)
Risk of Selected Nutritional Deficiencies Resulting from Bariatric Surgery

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>LAGB</th>
<th>RYGB</th>
<th>BPD/DS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macronutrients</td>
<td>✓✓</td>
<td>✓✓</td>
<td>✓✓</td>
</tr>
<tr>
<td>Thiamine⁴</td>
<td>✓</td>
<td>✓</td>
<td>✓✓</td>
</tr>
<tr>
<td>Iron³</td>
<td>✓✓</td>
<td>✓✓</td>
<td>✓✓</td>
</tr>
<tr>
<td>Vitamin B₁₂</td>
<td>✓✓</td>
<td>✓✓</td>
<td>✓✓</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>✓✓</td>
<td>✓✓</td>
<td>✓✓</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>✓</td>
<td>✓✓</td>
<td>✓✓</td>
</tr>
</tbody>
</table>

LAGB = Laparoscopic adjustable gastric banding; RYGB = Roux-en-Y gastric bypass; BPD/DS = biliopancreatic bypass/duodenal switch

Nutritional Monitoring for the Bariatric Surgical Patient

- Routine
  - CBC
  - Chemistry profile
  - Liver function tests
  - Lipid panel
  - HbA₁c

- Micronutrients
  - Iron, TIBC, Saturation
  - Ferritin
  - Folate
  - Vitamin B₁₂
  - 25 (OH) vitamin D
  - PTH
  - DEXA scan

Routine Vitamin & Mineral Supplementation for RYGB Patients

<table>
<thead>
<tr>
<th>Supplement</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multivitamin-mineral or prenatal</td>
<td>1-2 daily</td>
</tr>
<tr>
<td>Calcium citrate³ with vitamin D⁵</td>
<td>1,200-2,000 mg/day + 400-800 U/day</td>
</tr>
<tr>
<td>Elemental iron⁶</td>
<td>40-65 mg/day</td>
</tr>
<tr>
<td>Vitamin B₁₂</td>
<td>≥350 µg/day orally or 1,000 µg/mo intramuscularly or 3,000 µg every 6 mo intramuscularly or 500 µg every week intranasally</td>
</tr>
</tbody>
</table>

³ Depending on dietary calcium intake
⁴ Daily dose determined by serum 25 (OH) vitamin D
⁵ For postmenopausal women
⁶ A separate dose of elemental iron is required for RYGB patients

Glucose Tolerance Following RYGB in Type 2 Diabetes

Effect of Duodenal-Jejunal Exclusion in a Non-obese Animal Model of Type 2 Diabetes


- Can exclusion of duodenum and jejunum control diabetes independent from weight loss?
- GJB 8 rats compared to sham, pair fed, controls
- OGT and ITT performed one week post-op
- No significant difference in food intake or weight gain between groups

Proposed Mechanisms

- Negative caloric balance and weight loss
- Improvement in insulin sensitivity and “lipotoxicity”; mobilization of ectopic lipids.
- Stimulation of the entero-insular axis and subsequent effects of B-cell function (early weight independent effects)
- Foregut hypothesis
- Hindgut hypothesis

- >40% reduction AUC with glucose tolerance testing in GJB group
- Better insulin sensitivity with GJB
- Exclusion of duodenum and jejunum directly controls DM2 independent of wt loss or treatment of obesity

Insulin Secretion RYGB

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>C Peptide (ng/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>30</td>
<td>4</td>
</tr>
<tr>
<td>60</td>
<td>6</td>
</tr>
<tr>
<td>90</td>
<td>8</td>
</tr>
</tbody>
</table>

Pre-RYGB

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>C Peptide (ng/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>-1</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>90</td>
<td>10</td>
</tr>
</tbody>
</table>

Post-RYGB

Glucagon Like Peptide (GLP-1)

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>GLP-1 (pg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
</tr>
</tbody>
</table>

Pre-RYGB

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>GLP-1 (pg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
</tr>
</tbody>
</table>

Post-RYGB

Clinical Outcomes at 2 years

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Medical Therapy</th>
<th>Bypass</th>
<th>Sleeve</th>
<th>P Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c &lt;6% (12m)</td>
<td>6.25</td>
<td>4.4%</td>
<td>26.32%</td>
<td>0.02</td>
<td>0.19</td>
</tr>
<tr>
<td>HbA1c &lt;6% (24m)</td>
<td>5.9%</td>
<td>33.3%</td>
<td>10.5%</td>
<td>0.09</td>
<td>1.00</td>
</tr>
<tr>
<td>Change in HbA1c (%)</td>
<td>-1.1</td>
<td>-3.1</td>
<td>-2.5</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Change in BMI (kg/m2)</td>
<td>-0.2</td>
<td>-8.7</td>
<td>-8.2</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Change in Total Body Fat (%)</td>
<td>1.0</td>
<td>-10.6</td>
<td>-7.7</td>
<td>0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>Change in Truncal Fat (%)</td>
<td>0.9</td>
<td>-15.9</td>
<td>-10.1</td>
<td>0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>Change in Leptin</td>
<td>-14864</td>
<td>-11155</td>
<td>-16211</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

1: Gastric Bypass vs Medical Therapy, 2: Sleeve vs Medical Therapy
Gastric Bypass Normalizes Post Prandial Glucose Metabolism


Glucose Measures During Mixed Meal Tolerance Test

Baseline

Month 24

Glucose (mg/dl)

0
50
100
150
200
250
300

Medical
RYGB
Sleeve

Insulin Measures During Mixed Meal Tolerance Test

Baseline

Month 24

Insulin (uU/dmL)

0
20
40
60
80
100

Minutes

A.

Increase in Pancreatic β-cell Function

B.

Increase in Insulin Sensitivity

Absolute Change in Insulin Sensitivity During the Mixed Meal Tolerance Test

\[ \text{Absolute Change in Insulin Sensitivity} = \left( \frac{\text{AUC ISR}}{\text{AUC Glucose}} \right) \times \text{Matsuda Index} \]

Physiologic effects of Gastric Bypass on Classical Pathways of Glucose Metabolism. Goldfine A B, and Patti M E Diabetes 2014;63:1454-1456

Non-remission of DM

• Result of impaired incretin stimulation of residual beta cell function

• OR

• Weight/fat related effects of insulin sensitivity

• Does weight regain following surgery impact recurrence of DM?

Results: Adiponectin

Non-remitters showed blunted response to bariatric surgery

Malin, Kashyap et al. Diabetes, Obesity And Metabolism. 2014 Dec;18(12):1200-4
Summary Points

- Diabetes remission characteristic of bariatric surgery (RYGB>Sleeve Gastrectomy) and linked to improvements in post prandial glucose metabolism.
- Weight independent effects related to GLP-1 specific effects to increase insulin secretion
- For those who initially achieved remission but relapse subsequently, consider targeting additional weight/fat loss and adiponectin raising interventions. (diet/exercise/metformin/TGTL2)
- Continuous surveillance for micro- and macro-vascular complications post-surgery is Key!!

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Sangeeta R. Kashyap, MD
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Julianne Filton, RN, BSN
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