Disclosures

Nothing to Disclose
Traditional View

Restriction vs Malabsorption

Figure-1: CLASSIFICATION OF BARIATRIC PROCEDURES

- Restrictive Procedures
  - Roux en Y gastric bypass (RYGBP)
  - Long limb RYGBP
  - Laparoscopic adjustable gastric band (LAGB)
  - Vertical banded gastroplasty (VBG)

- Combined Procedures
  - Biliopancreatic diversion (BPD)
  - Biliopancreatic diversion with duodenal switch (BPD-DS)
Traditional View
I yearn for Simplicity...

...but I always seem to end up with Complexity.
Adjustable Gastric Band

- Stomach pouch
- Adjustable band
- Port placed under skin
Adjustable Gastric Band

Fig. 16.4 Angle of His dissection
Adjustable Gastric Band

Fig. 16.5 Blunt articulating dissector used to create the posterior tunnel
Fig. 16.8 Dissector tip holding the Lap-Band end tubing and Realize suture loop
Adjustable Gastric Band

Fig. 16.9 Locking the Lap-Band and Realize Band
Adjustable Gastric Band

Fig. 16.11 Inferior anterior gastric plication stitch
Adjustable Gastric Band

Fig. 16.12 Band access port fixation with four sutures in the anterior rectus muscle fascia
Sleeve Gastrectomy
Sleeve Gastrectomy
Roux-en-Y Gastric Bypass
Roux-en-Y Gastric Bypass

Fig. 15.2  Steps in jejunojejunostomy
Roux-en-Y Gastric Bypass

Fig. 15.3 Steps in gastrojejunostomy
Duodenal Switch
## Mechanisms of Action

<table>
<thead>
<tr>
<th>Mechanism of action</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RYGB</td>
</tr>
<tr>
<td>Malabsorption</td>
<td>+/−</td>
</tr>
<tr>
<td>Caloric restriction</td>
<td>+</td>
</tr>
<tr>
<td>Energy expenditure</td>
<td>+/−</td>
</tr>
<tr>
<td>Δ(delta)-eating behavior</td>
<td>+</td>
</tr>
<tr>
<td>Hormonal</td>
<td>+</td>
</tr>
<tr>
<td>Vagus nerve</td>
<td>?/−</td>
</tr>
<tr>
<td>Bile salts</td>
<td>+</td>
</tr>
<tr>
<td>Adipose tissue</td>
<td>+</td>
</tr>
<tr>
<td>Microbiota</td>
<td>+/−</td>
</tr>
<tr>
<td>β(beta)-cell function</td>
<td>+/−</td>
</tr>
<tr>
<td>Insulin sensitivity</td>
<td>+/−</td>
</tr>
</tbody>
</table>

*a Only related to weight loss

Trachta P et al. 2014
Malabsorption

- Significant in Duodenal Switch
  - Moderate in RYGB
    - ↑ Fecal Fat at 6 months (126%) and 12 months (87%)
      (Kumar R et al. 2011)
    - No sig. change in absorption of protein/carbohydrates
      - Overall change in combustible energy absorption only 6 – 11%
        (Odstrcil EA et al. 2010)
Caloric Restriction

• Important mechanism in immediate post-operative weight loss

  • Similar weight loss between operated and non-operated patients after 4 days of post-bypass diet
    • Only operated on patients had improvements in insulin sensitivity, secretion, and insulin-stimulating gut hormones (i.e. GLP-1)

  (Isbell JM et al. 2013)

• Other factors involved in long-term weight loss and glycemic control
Energy Expenditure

• Normal Diet:
  • Caloric restriction/weight loss $\rightarrow$ ↓ Energy Expenditure
    • Designed to preserve body weight

• Post Bariatric Surgery:
  • Conflicting data
  • No definite conclusions can be drawn
Change in Eating Behaviour

• Propensity towards high-fat vs lean food in obese patients

• Animal/human studies show preference of low fat diets, avoidance of calorie-dense diets post bariatric surgery
  
  (Thomas JR et al. 2008) & (Wilson-Pérez HE et al. 2013)

• Possible Mechanisms:
  
  • Change in taste acuity and cravings  (Delin CR et al. 1997)

  • Aversive symptoms by improper food choices (i.e. dumping syndrome with carbohydrates)
### Entero-Hormones

**Table 5.2** Characteristics of the entero-hormones after bariatric operations

<table>
<thead>
<tr>
<th></th>
<th>Origin</th>
<th>Satiety</th>
<th>Glycemic control</th>
<th>GI motility</th>
<th>RYGB</th>
<th>LSG</th>
<th>LAGB</th>
<th>BPD</th>
<th>BPD-DS</th>
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<tbody>
<tr>
<td>GLP-1</td>
<td>L cells</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>No Δ(delta)</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>GIP</td>
<td>K cells</td>
<td>No Δ(delta)</td>
<td>↑</td>
<td>No Δ(delta)</td>
<td>Unknown</td>
<td>No Δ(delta)</td>
<td>↓</td>
<td>↓</td>
<td></td>
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<tr>
<td>PYY</td>
<td>L cells</td>
<td>↑ or no Δ(delta)</td>
<td>↓</td>
<td>↑</td>
<td>↑ or no Δ(delta)</td>
<td>No Δ(delta)</td>
<td>↑</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>Oxyntomodulin</td>
<td>L cells</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>No Δ(delta)</td>
<td>↑</td>
<td>↑</td>
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<tr>
<td>CCK</td>
<td>I cells</td>
<td>↑</td>
<td>No Δ</td>
<td>↑</td>
<td>?</td>
<td>↑ or no Δ(delta)</td>
<td>Unknown</td>
<td>Unknown</td>
<td>Unknown</td>
</tr>
<tr>
<td>Ghrelin</td>
<td>Oxyntic</td>
<td>↓</td>
<td>No Δ</td>
<td>No Δ</td>
<td>↓</td>
<td>↓</td>
<td>No Δ(delta)</td>
<td>No Δ(delta)</td>
<td>↓</td>
</tr>
</tbody>
</table>
Glucagon-Like Peptide-1 (GLP-1)

• Released by L-cells of ileum/colon

• ↑Insulin secretion, ↓Glucagon secretion, ↓Gastric Emptying/GI Motility (ileal brake)

• Net effect: ↑Satiety, ↓Food Intake, Improved Glucose Homeostasis
Glucagon-Like Peptide-1 (GLP-1)

- Stimulated by nutrients/food in distal ileum
  - Explains rapid/durable hormonal/weight response after bypass (RYGB, DS)

- ↓Gastric transit time post LSG → ↑GLP-1 Secretion

- Liraglutide (Saxenda) → GLP-1 receptor agonist
Glucose-Dependent Insulinotropic Polypeptide (GIP)

- Secreted by K-cells of duodenum and proximal jejunum
  - Stimulated by nutrients in proximal small bowel
- ↑Insulin secretion, ↑Lipogenesis/Fat Deposition
- ↓Levels after bypass (RYGB, DS)
Peptide Tyrosine Tyrosine (PYY)

• Released by L-cells of ileum/colon (similar to GLP-1), and by the brain
  • Release proportional to caloric density of nutrients

• ↓Gastric Emptying/GI Motility (ileal brake), ↓Appetite

• ↑Levels post RYGB, DS, LSG
  • No change after LAGB
  • Levels normalize over time with LSG, not with RYGB
Oxyntomodulin

- Similar structure to GLP-1
  - Similar effects:
    - \( \uparrow \) Insulin secretion, \( \downarrow \) Glucagon secretion, \( \downarrow \) Gastric Emptying/GI Motility (ileal brake)

- \( \uparrow \) Levels after bypass (RYGB, DS)
Ghrelin

- Secreted primarily by oxyntic glands of gastric fundus

- Orexigenic effect on hypothalamus
  - ↑Appetite

- Inhibits insulin secretion
  - ↓Glucose homestasis

- Level post LSG < post RYGB
  - Reduction = improved glycemic control, ↑satiety
Bile Acids

- Regulators of energy balance, increase energy expenditure in brown adipose tissue
  - May also activate L-cells

- ↑Concentration post RYGB and LSG
  - 2º to ↓enterohepatic circulation → ↑conversion of cholesterol to bile acids

- ↑Levels associated with ↓rebound hyperphagia, improved glucose tolerance
Adipose Tissue

- ↑Peripheral fat assoc. with peripheral and hepatic insulin resistance

- ↑Visceral fat assoc. with:
  - ↑Pro-inflammatory cytokines (TNF, IL-6, Leptin)
  - ↓Anti-inflammatory cytokines (Adiponectin)
Leptin

- Secreted by white adipose tissue
  - Regulates energy balance by inhibiting hunger
    - Acts on hypothalamus (opposes Ghrelin)

- Leptin resistance and elevated levels in obese population

- Levels decrease with weight loss
  - Decreased levels found after all bariatric procedures
Adiponectin

- Produced by adipose tissue
- ↓Levels in obese population
  - Assoc. with insulin resistance and CAD
- ↑Levels post bariatric surgery
  - Improved insulin sensitivity
GI Microflora

• Animal/human studies demonstrate changes in GI microflora in obese population
  (Ley RE et al. 2005)

• Alteration of GI microflora in obese mice → weight loss
  (Cani PD et al. 2008)

• Weight loss shifts GI microflora to that of lean individuals
References


Questions?