Bariatric Procedures and Mechanisms of Weight Loss

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Aryan Modasi MD MSc FRCSC

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Disclosures

Nothing to Disclose

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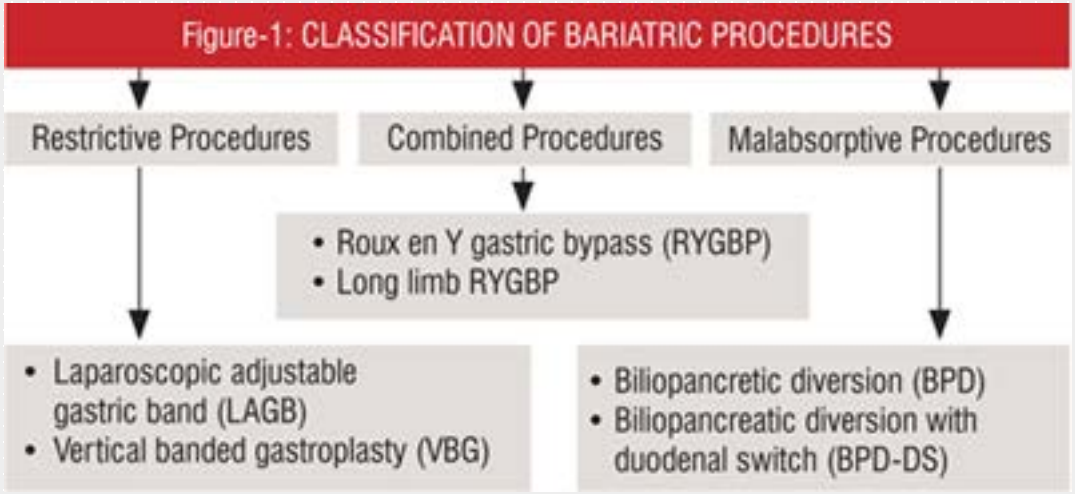
Traditional View

Restriction vs Malabsorption



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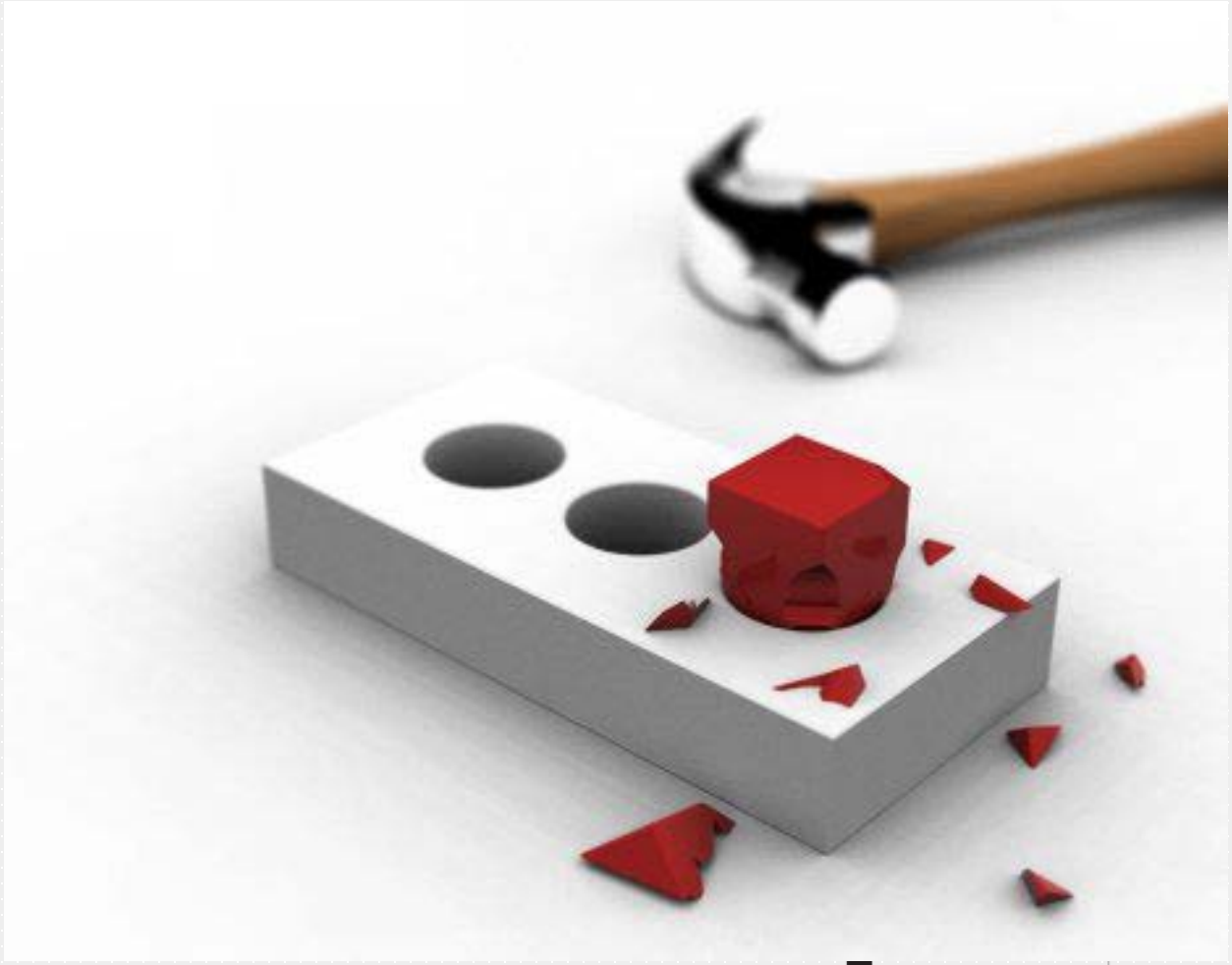
Traditional View



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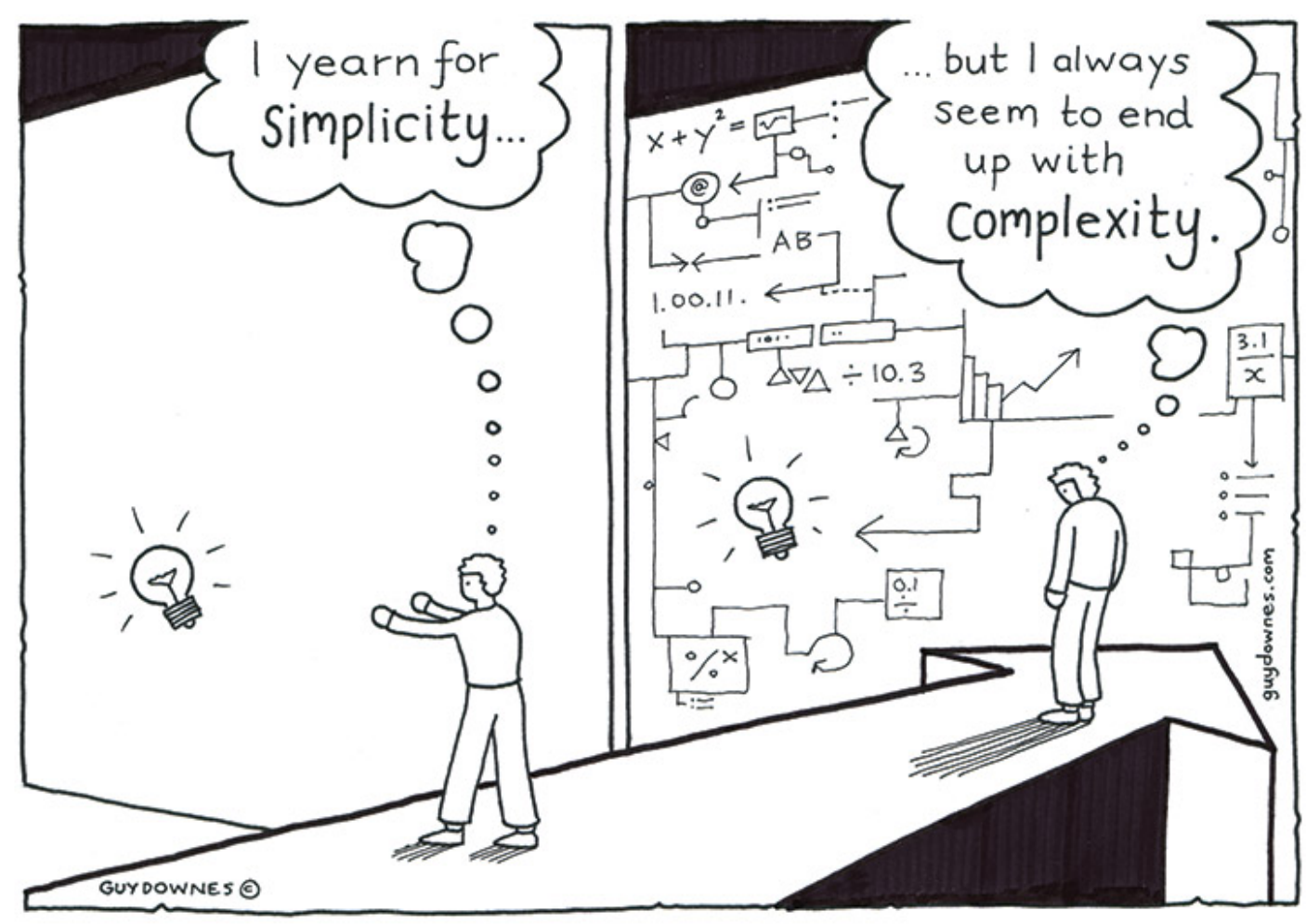
I yearn for Simplicity...
... but I always seem to end up with Complexity.
$x+y^2=$
AB
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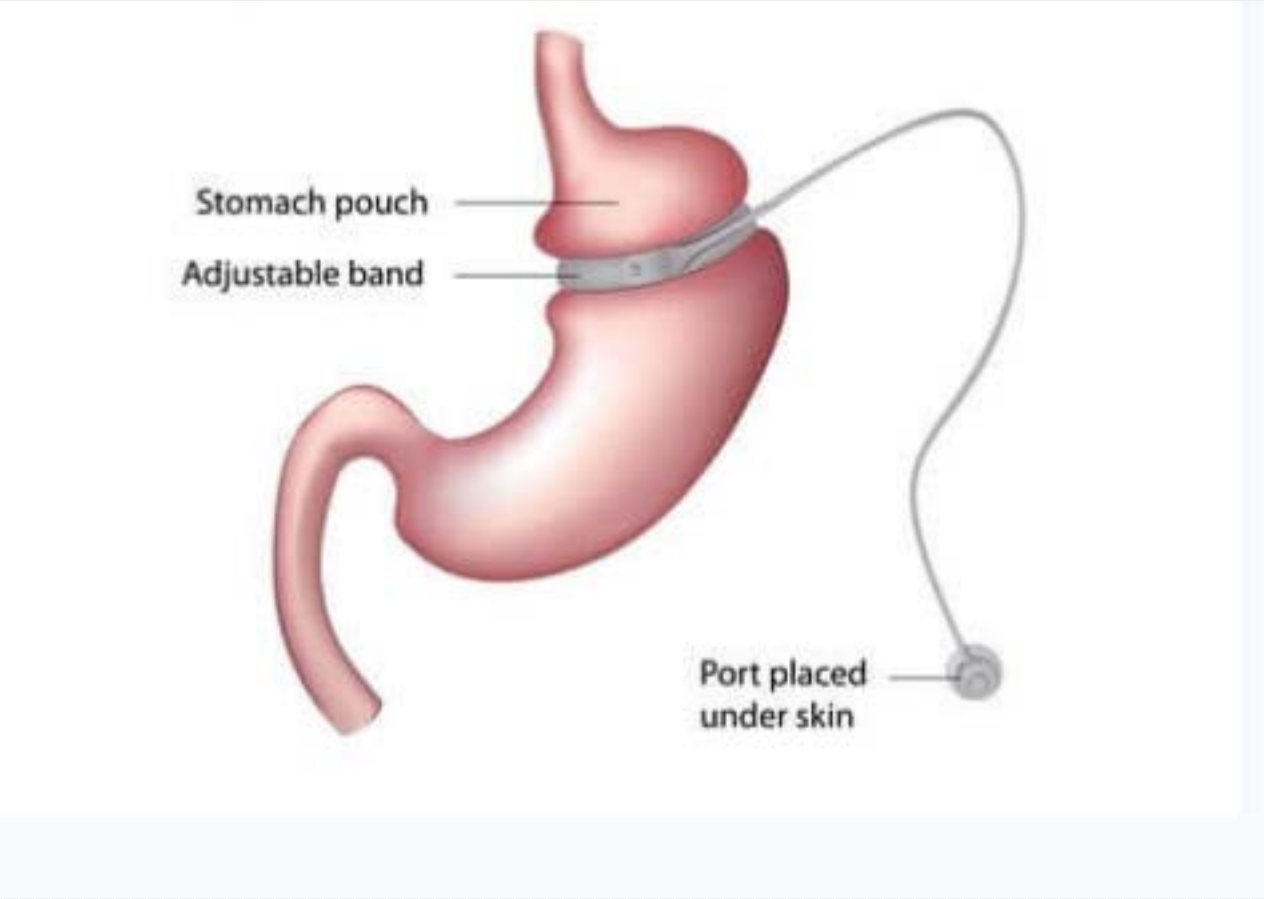
Adjustable Gastric Band



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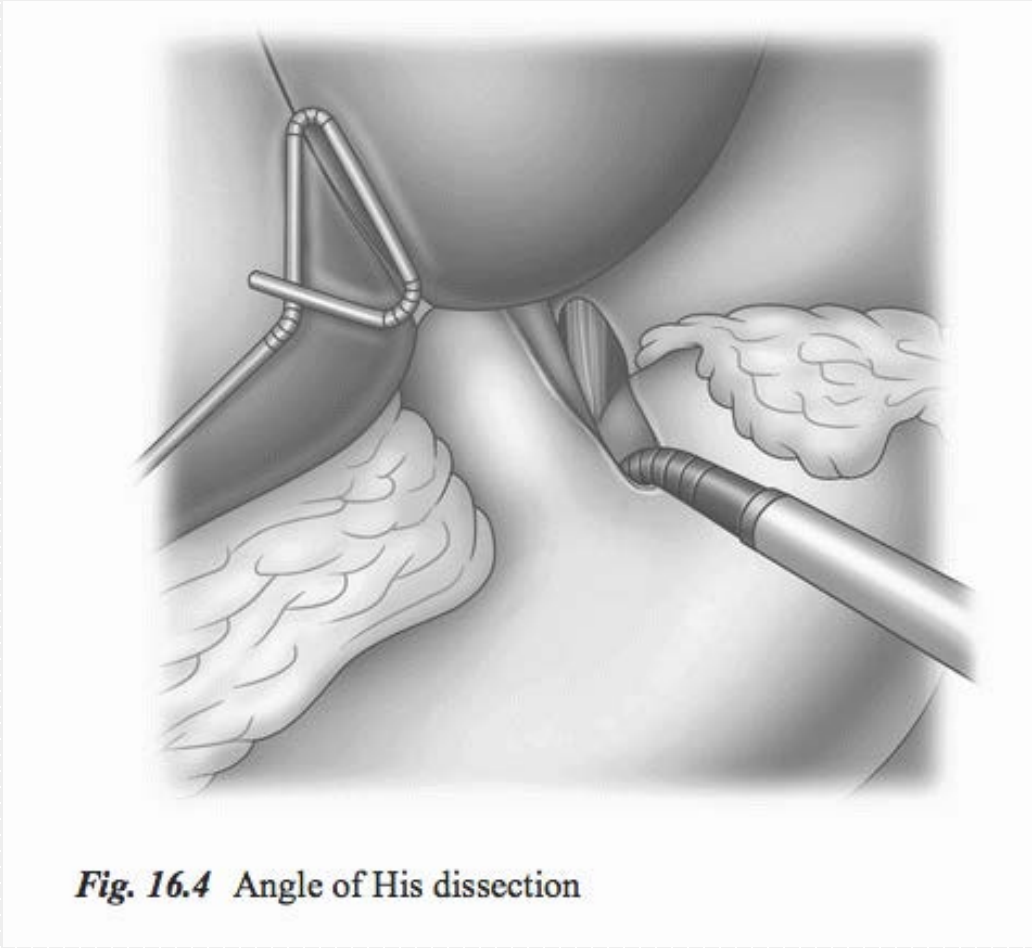

Fig. 16.4 Angle of His dissection

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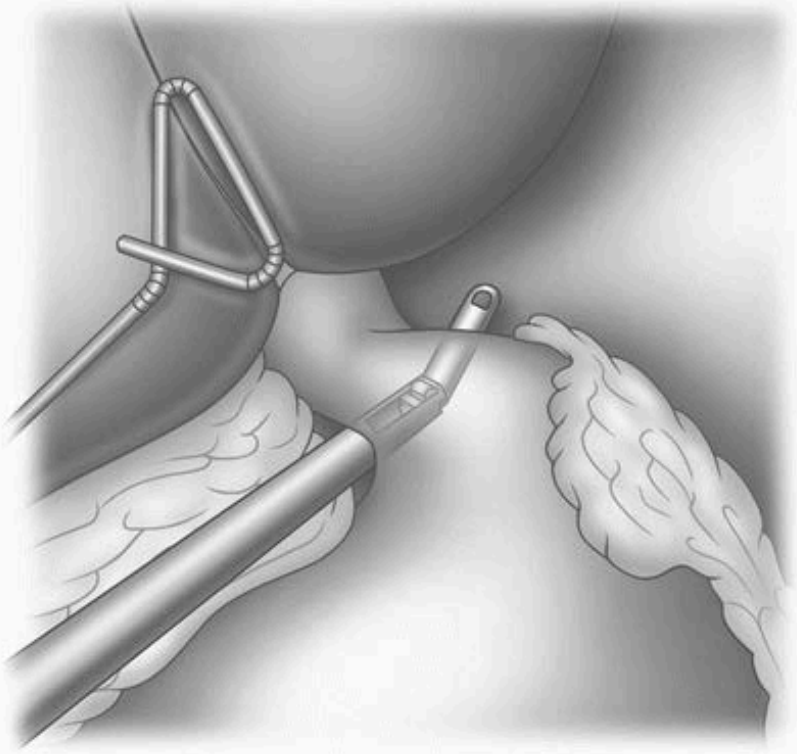

Fig. 16.5 Blunt articulating dissector used to create the posterior tunnel

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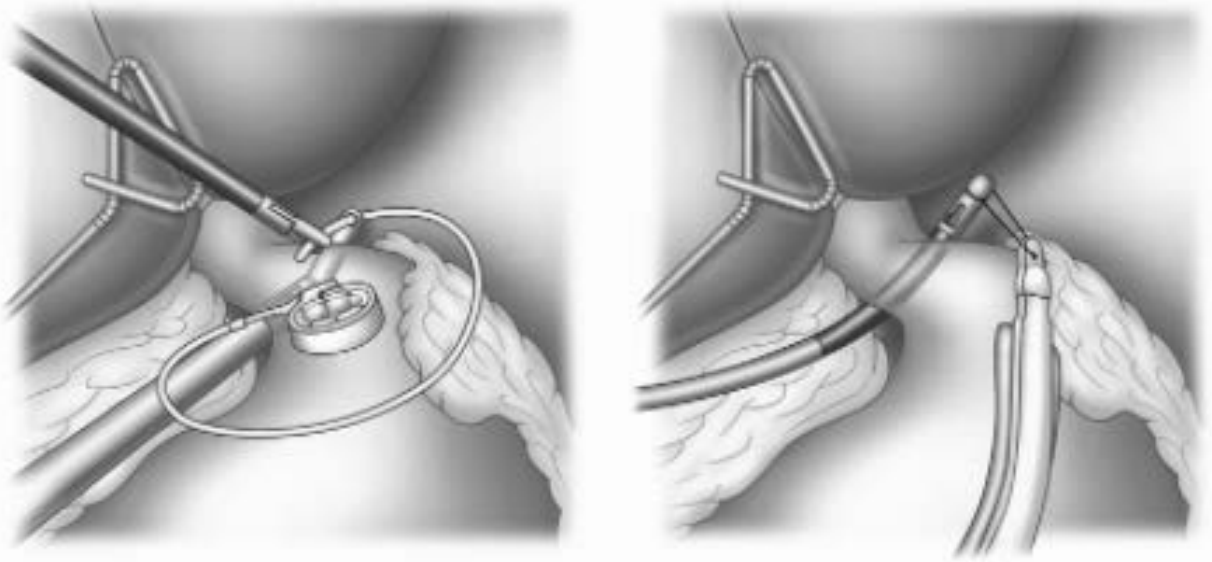

Fig. 16.8 Dissector tip holding the Lap-Band end tubing and Realize suture loop

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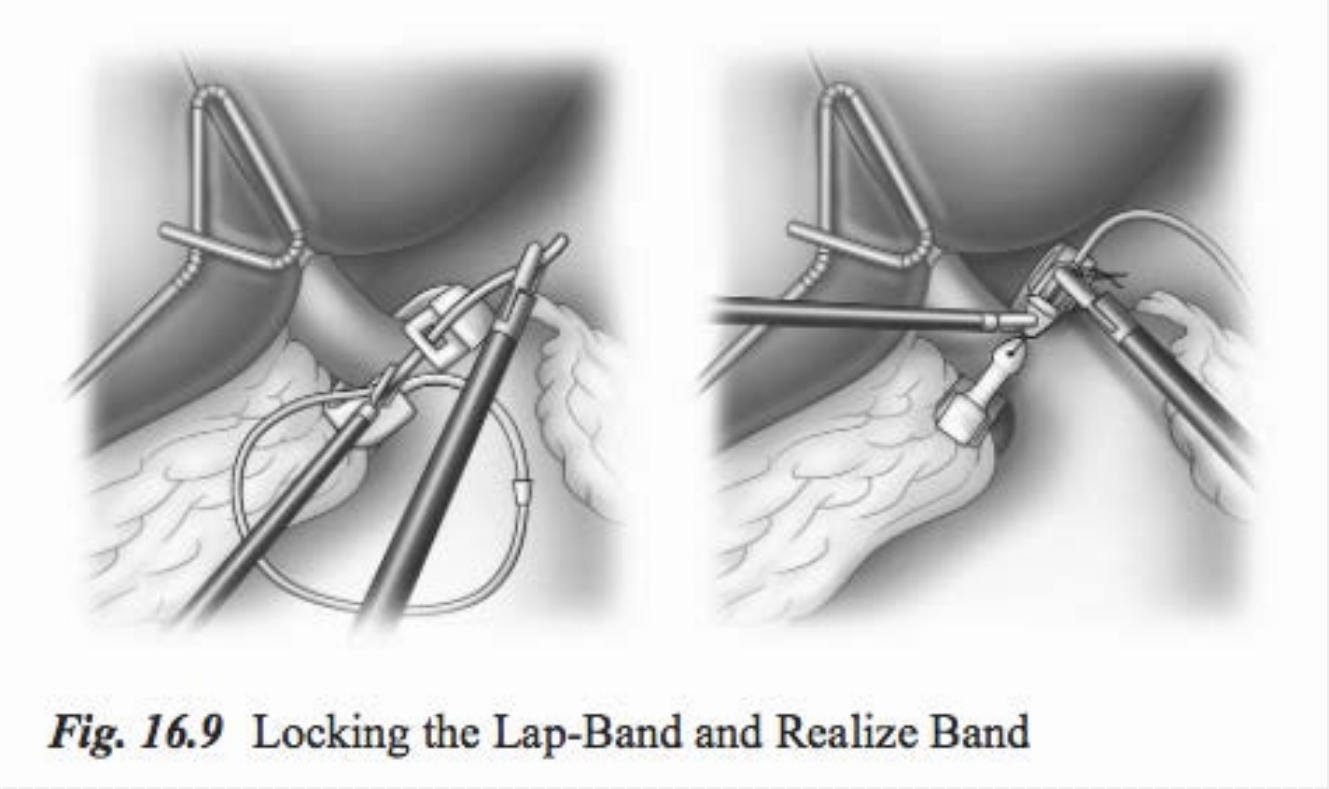

Fig. 16.9 Locking the Lap-Band and Realize Band

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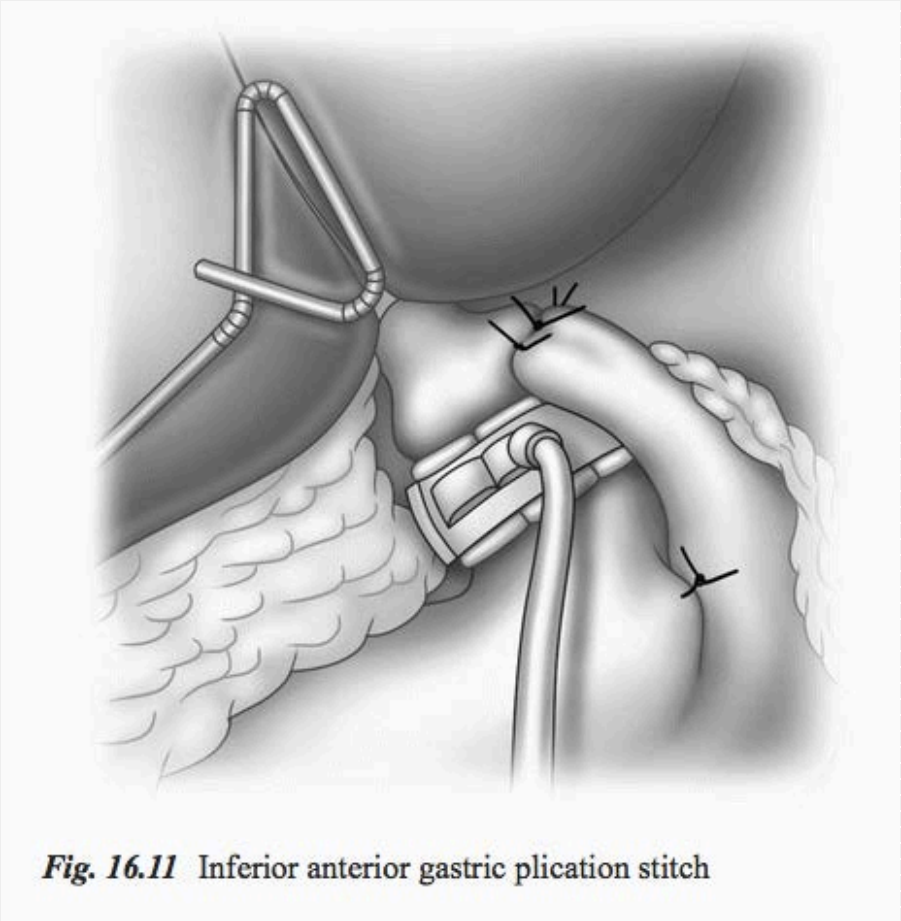

Fig. 16.11 Inferior anterior gastric plication stitch

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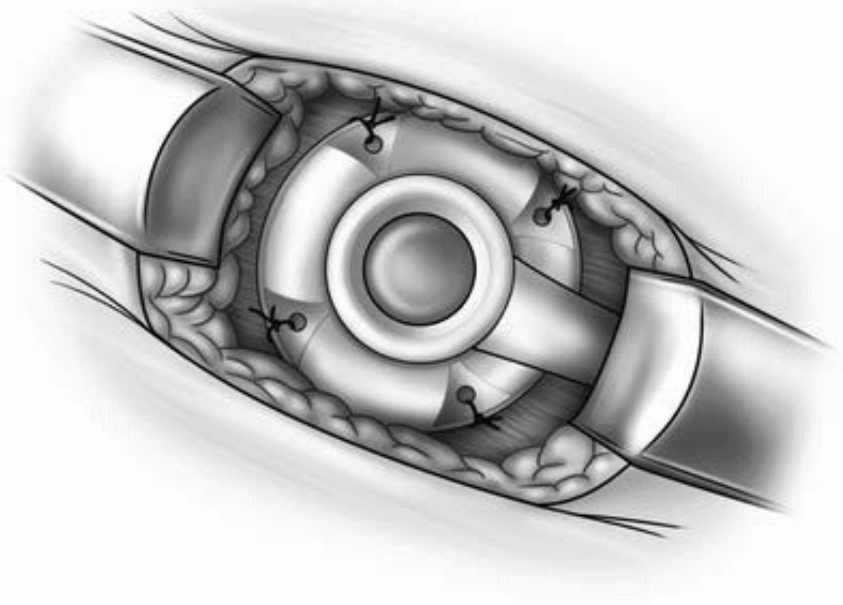

Fig. 16.12 Band access port fixation with four sutures in the anterior rectus muscle fascia

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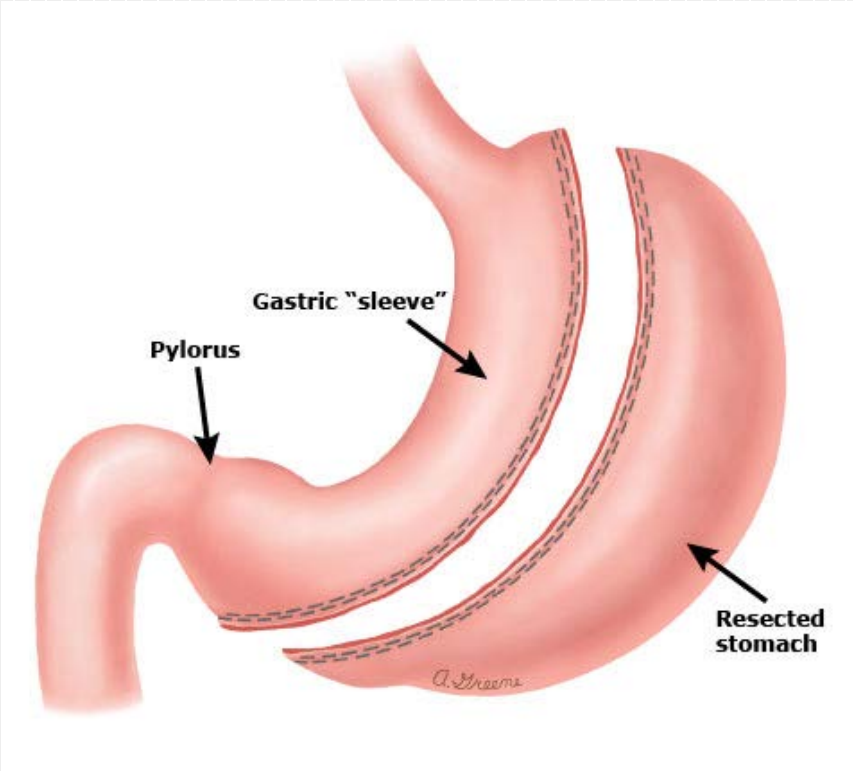
Sleeve Gastrectomy



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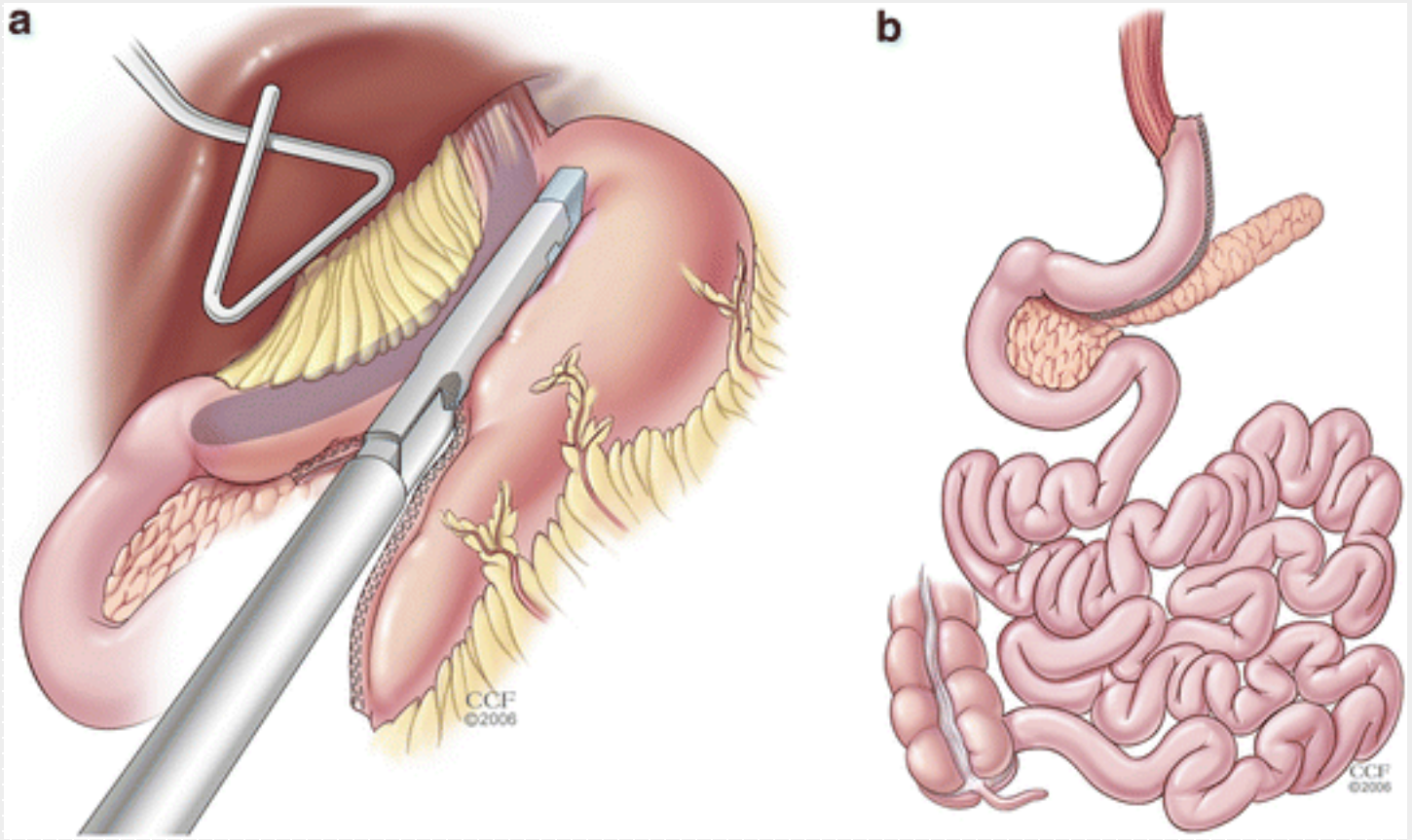

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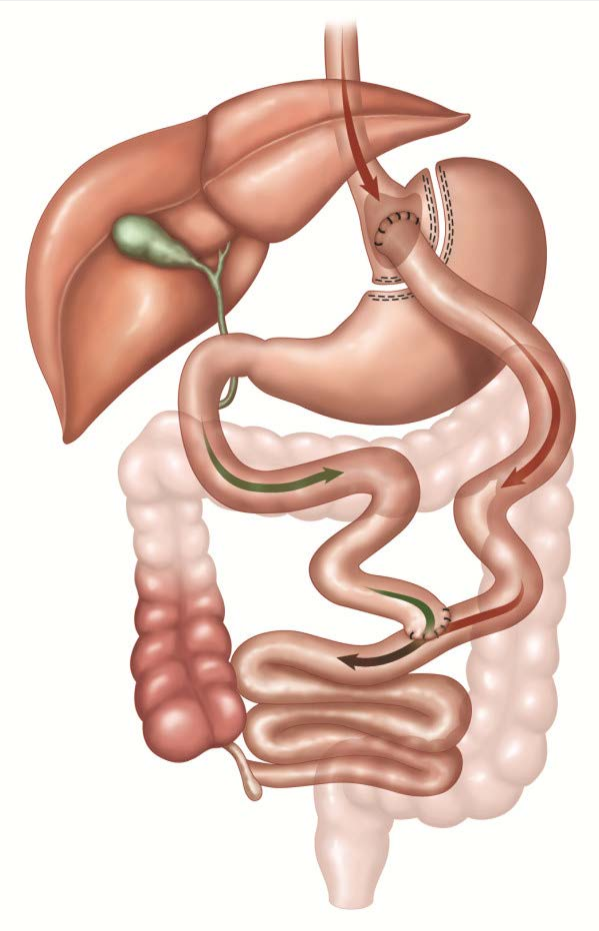
Roux-en-Y Gastric Bypass



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Roux-en-Y Gastric Bypass

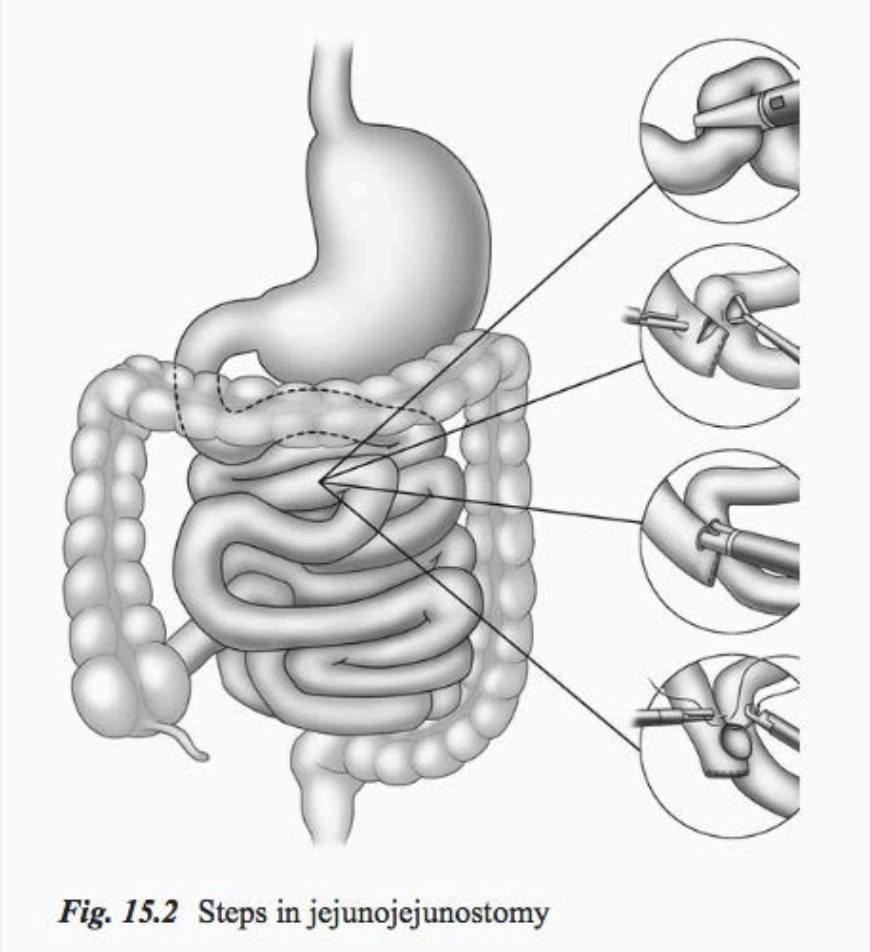

Fig. 15.2 Steps in jejunojejunostomy

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Roux-en-Y Gastric Bypass

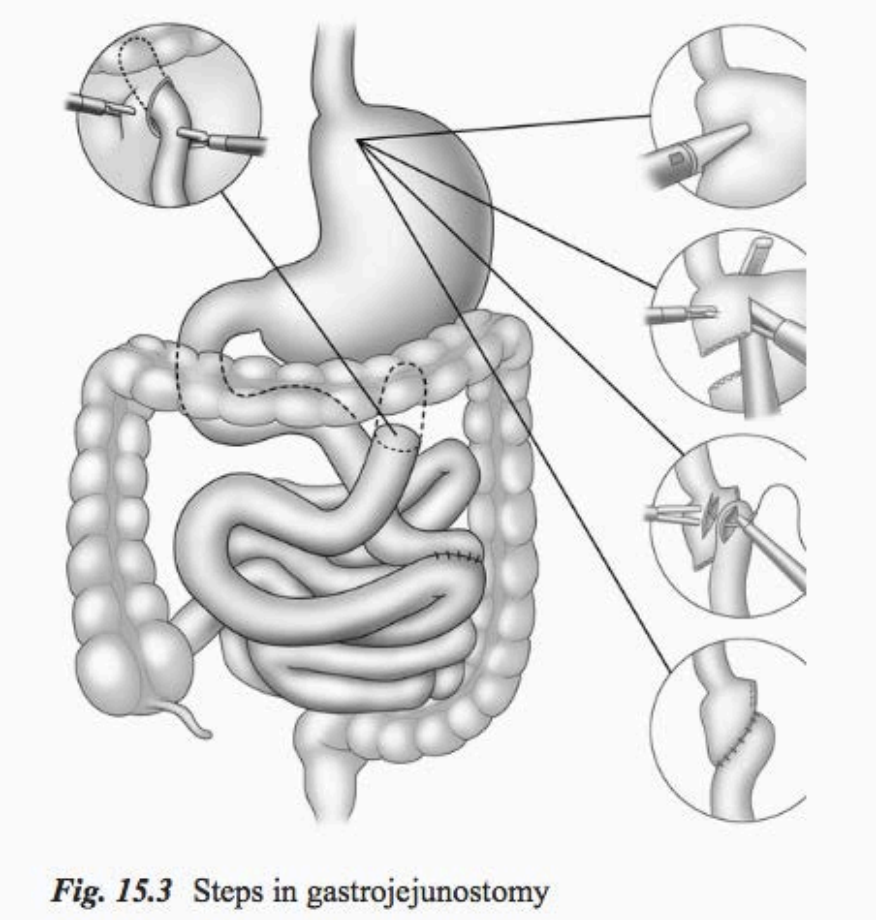

Fig. 15.3 Steps in gastrojejunostomy

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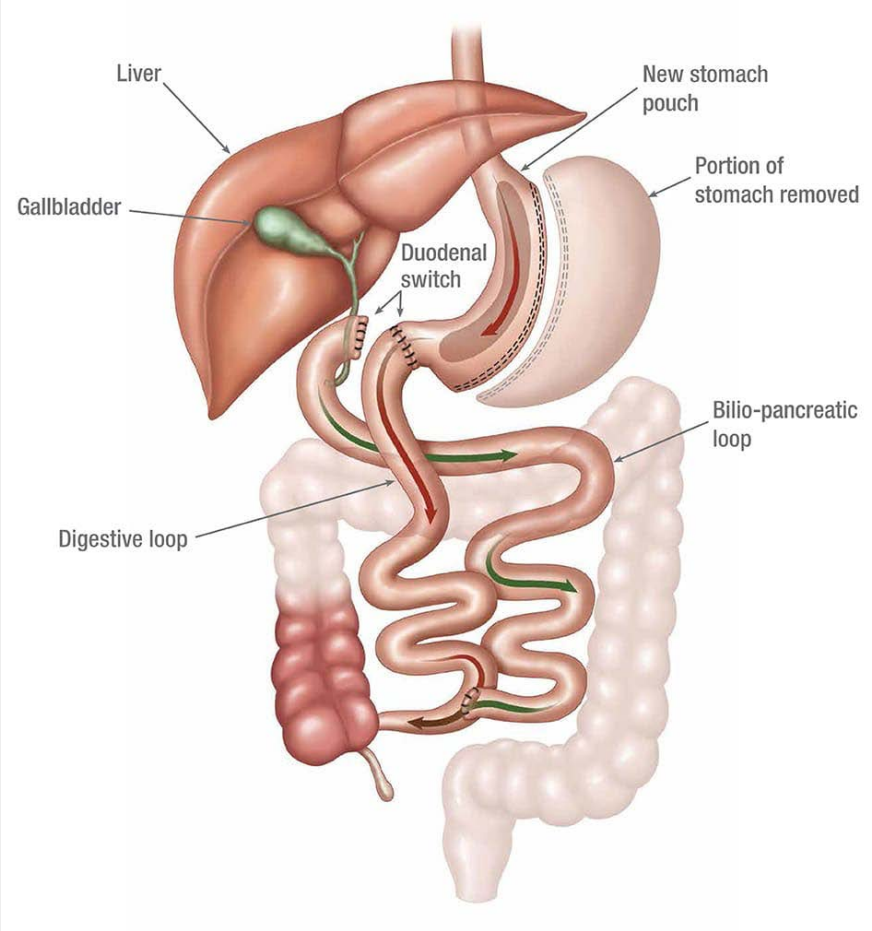
Duodenal Switch



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Mechanisms of Action

	Procedure				
Mechanism of action	**RYGB**	**LSG**	**LAGB**	**BPD**	**BPD-DS**
Malabsorption	+/−	−	−	+	+
Caloric restriction	+	+	+/−	+	+
Energy expenditure	+/−	−	−	+	+
Δ(delta)-eating behavior	+	+/−	−	?	?
Hormonal	+	+	−	+	+
Vagus nerve	?/−	?/−	?/−	?/−	?/−
Bile salts	+	+	+/−	+	+
Adipose tissue	+	+[a]	−	+	+
Microbiota	+/−	?	−	+/−	+/−
ß(beta)-cell function	+/−	?	−	+/−	+/−
Insulin sensitivity	+/−	+	+b	++	++

[b]Only related to weight loss

Trachta P et al. 2014

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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 → ↓ Energy Expenditure
    - Designed to preserve body weight

- Post Bariatric Surgery:
  - Conflicting data
  - No definite conclusions can be drawn

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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)

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Entero-Hormones

Table 5.2 Characteristics of the entero-hormones after bariatric operations

	Origin	Satiety	Glycemic control	GI motility	RYGB	LSG	LAGB	BPD	BPD-DS
GLP-1	L cells	↑	↑	↓	↑	↑	No Δ(delta)	↑	↑
GIP	K cells	No Δ(delta)	↑	No Δ(delta)	↓	Unknown	No Δ(delta)	↓	↓
PYY	L cells	↑	↑ or no Δ(delta)	↓	↑	↑ or no Δ(delta)	No Δ(delta)	↑	↑
Oxyntomodulin	L cells	↑	↑	↓	↑	↑	No Δ(delta)	↑	↑
CCK	I cells	↑	No Δ	↑	?	↑ or no Δ(delta)	Unknown	Unknown	Unknown
Ghrelin	Oxyntic	↓	No Δ	No Δ	↓	↓↓	No Δ(delta)	No Δ(delta)	↓↓

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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

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

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