SHORT BOWEL SYNDROME AND HIGH OUTPUT STOMA

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SHORT BOWEL SYNDROME

Normal Small Bowel Length

275-840 cm

Short bowel syndrome: < 200 cm in continuity of small intestine

Intestinal failure (IF): depends on the anatomy, integrity and adaptive potential of the small bowel remnant

INTESTINAL FAILURE

Reduction in GUT function (⬇ small intestinal absorptive area) below the minimum required to maintain nutrient, electrolye and hydration status for health and growth

- **Requires IV or enteral supplementation**

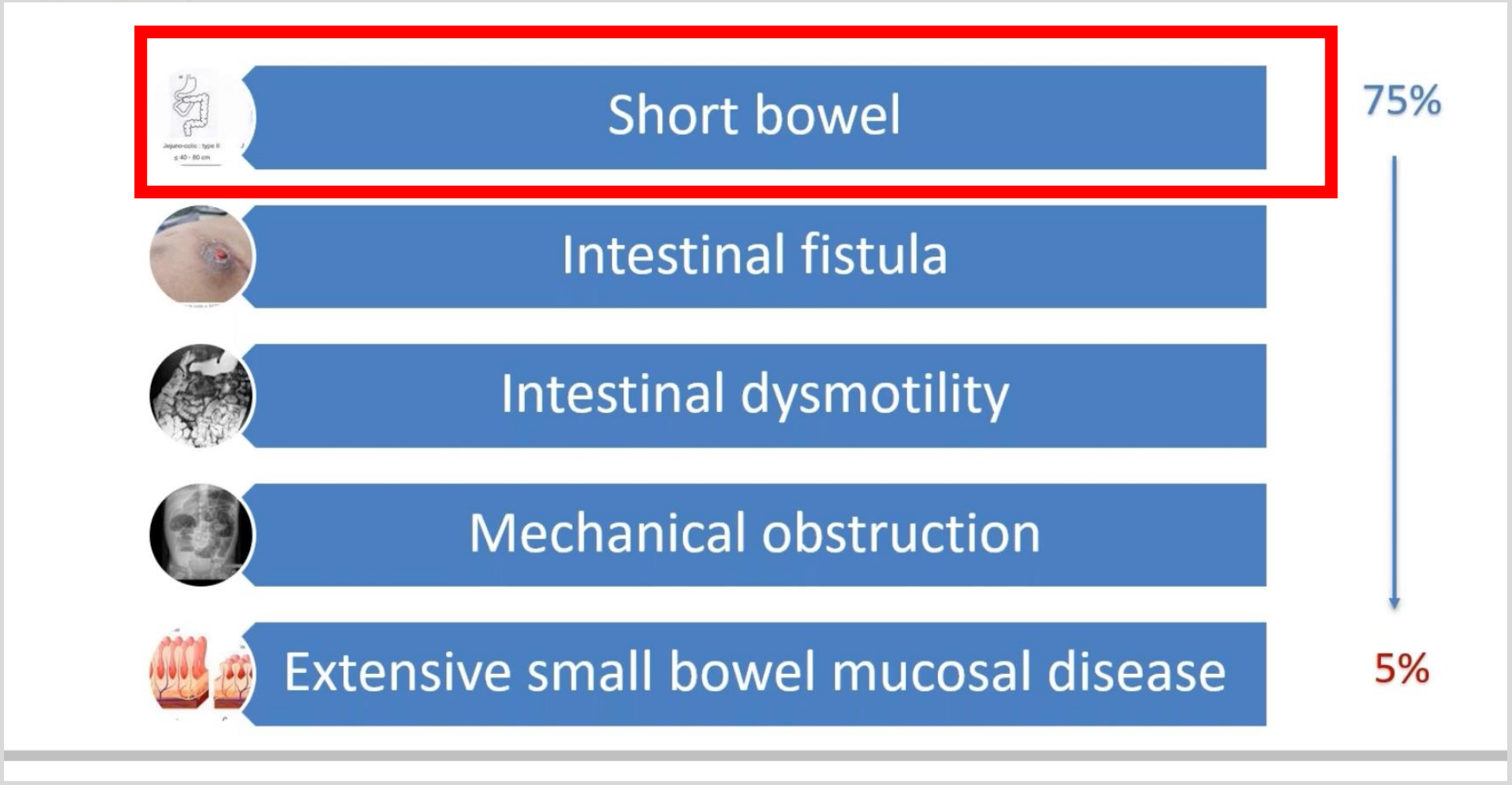
Intestinal insufficiency = NO IV support required

Classification of Intestinal failure

Functional (time)

- Type 1: self-limiting
  - Post-operative
- Type 2: weeks-months
  - EC fistula, diversion
- Type 3: years
  - SBS, dysmotility

Causes of Intestinal failure



Short Bowel Syndrome - Causes

- **Massive resection**
  - Trauma
  - Bowel obstruction
  - CD
  - Vascular catastrophe

- **Disease associated loss of absorption**
  - CD
  - Radiation
  - Mesenteric ischemia

Children

- NEC
- Congenital
  - mid-gut volvulus
  - Atresia

Determinants of Intestinal function

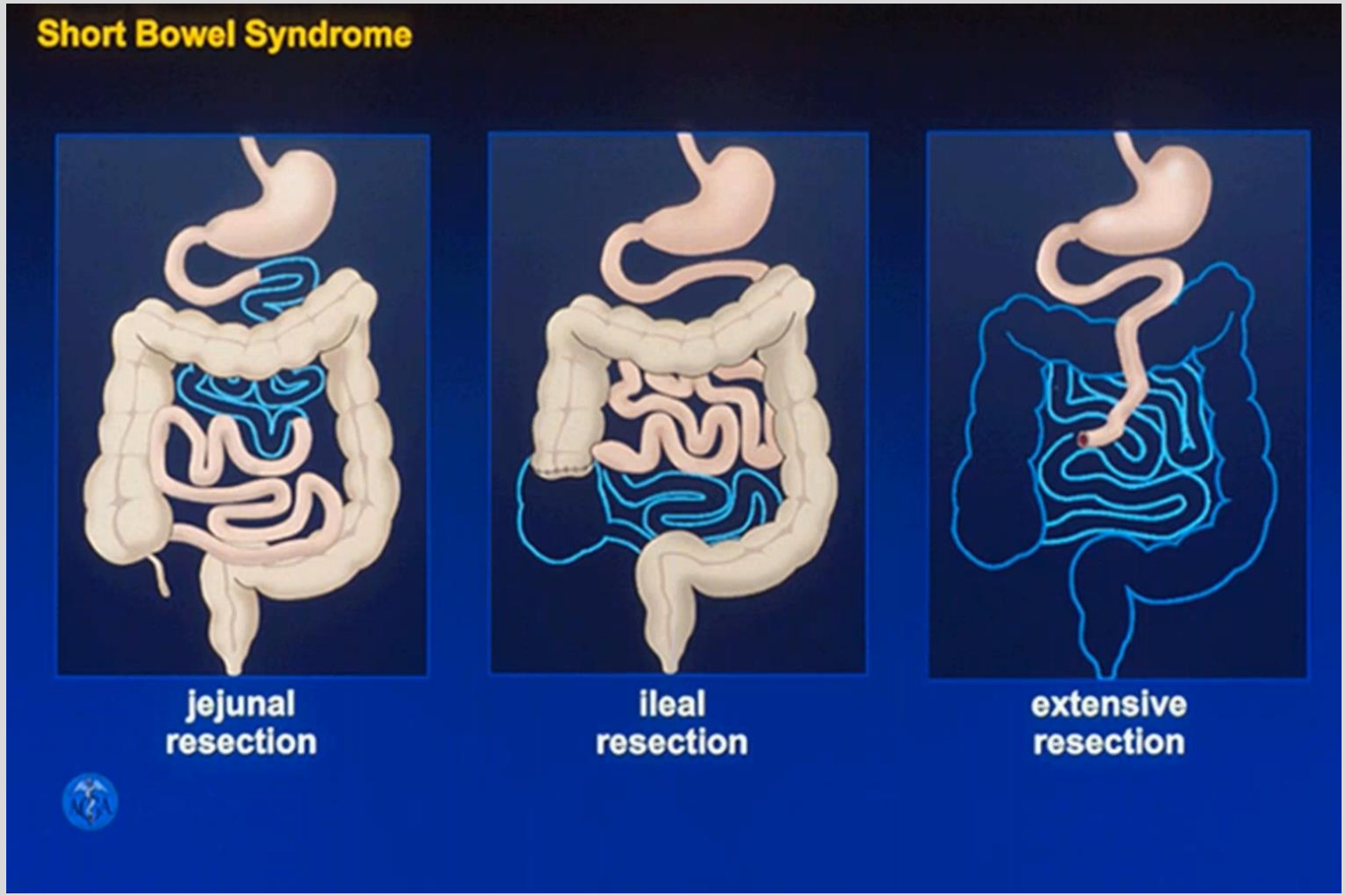
1. Small intestine length

- Normal = 480cm (wide range 300 – 800cm)
- SBS < 200cm
- Presence of colon in continuity
  - ➢Mitigates functional impairment
  - ➢Half colon = 50cm small bowel
- **Evaluating residual bowel left after surgery very important**
  - ➢Intra-operatively or on cross sectional imaging

Determinants of Intestinal function

2. Site of intestinal resection

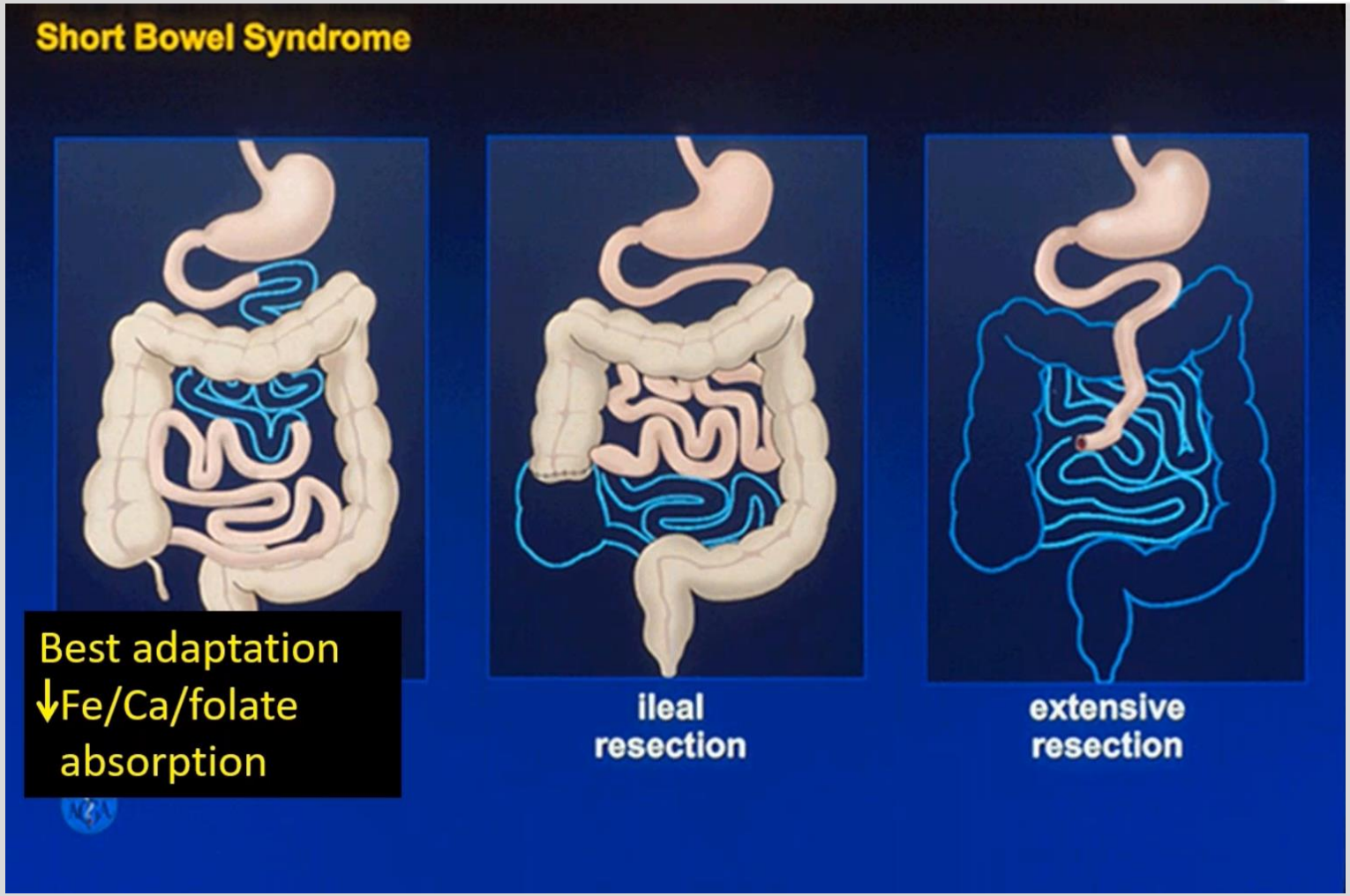
Sites of Intestinal resection



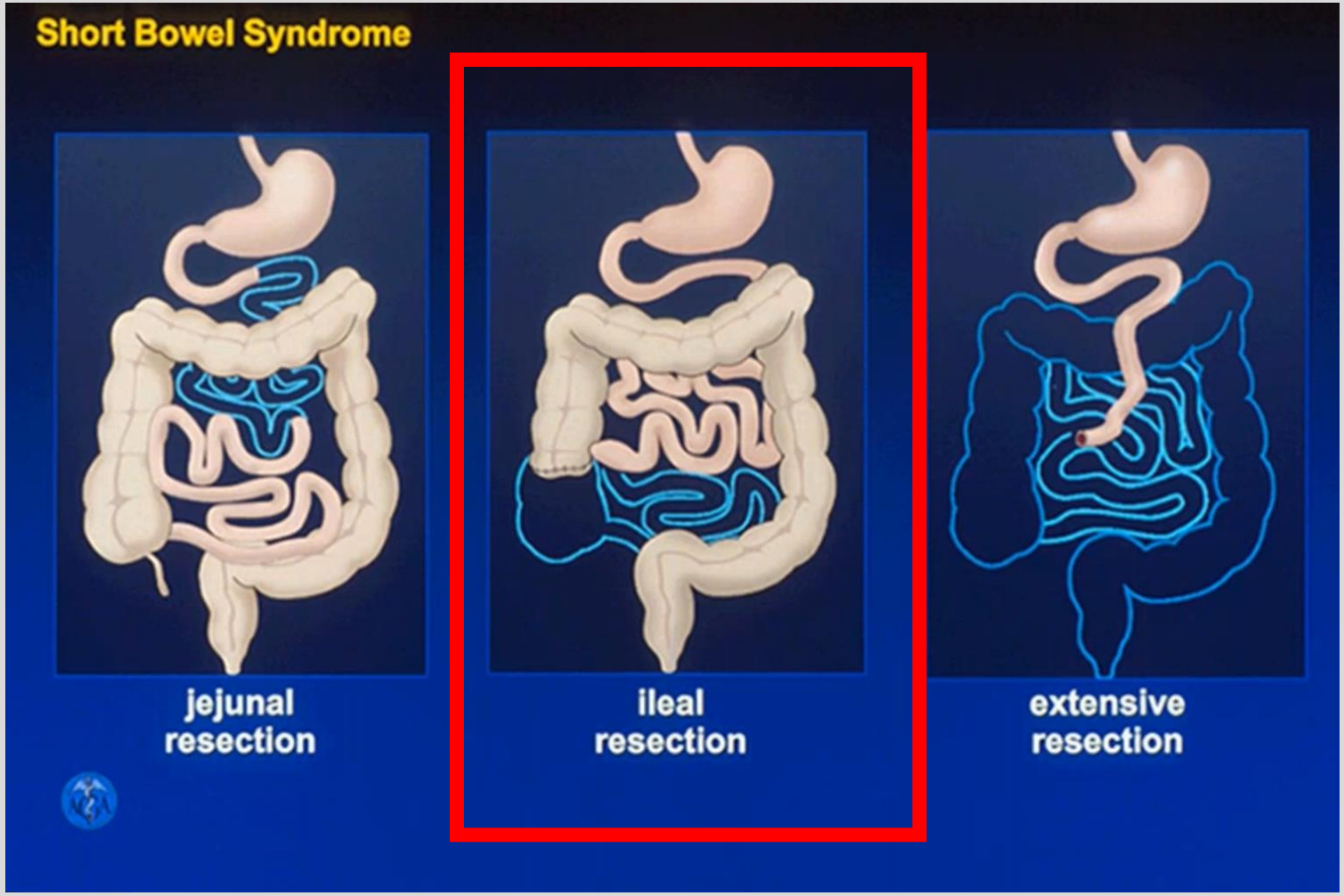
JEJUNAL RESECTION

Jejunum:

- **Primary absorptive area for macro and micronutrients**
- ✓ Long villi
- ✓ Large absorptive surface
- ✓ Highly concentrative digestive enzymes
- ✓ Active transport with carrier proteins
- **Wide intercellular junctions**
- ✓ Fluid absorption/secretion



ILEAL RESECTION



Bile Acid absorption

>100cm disruption

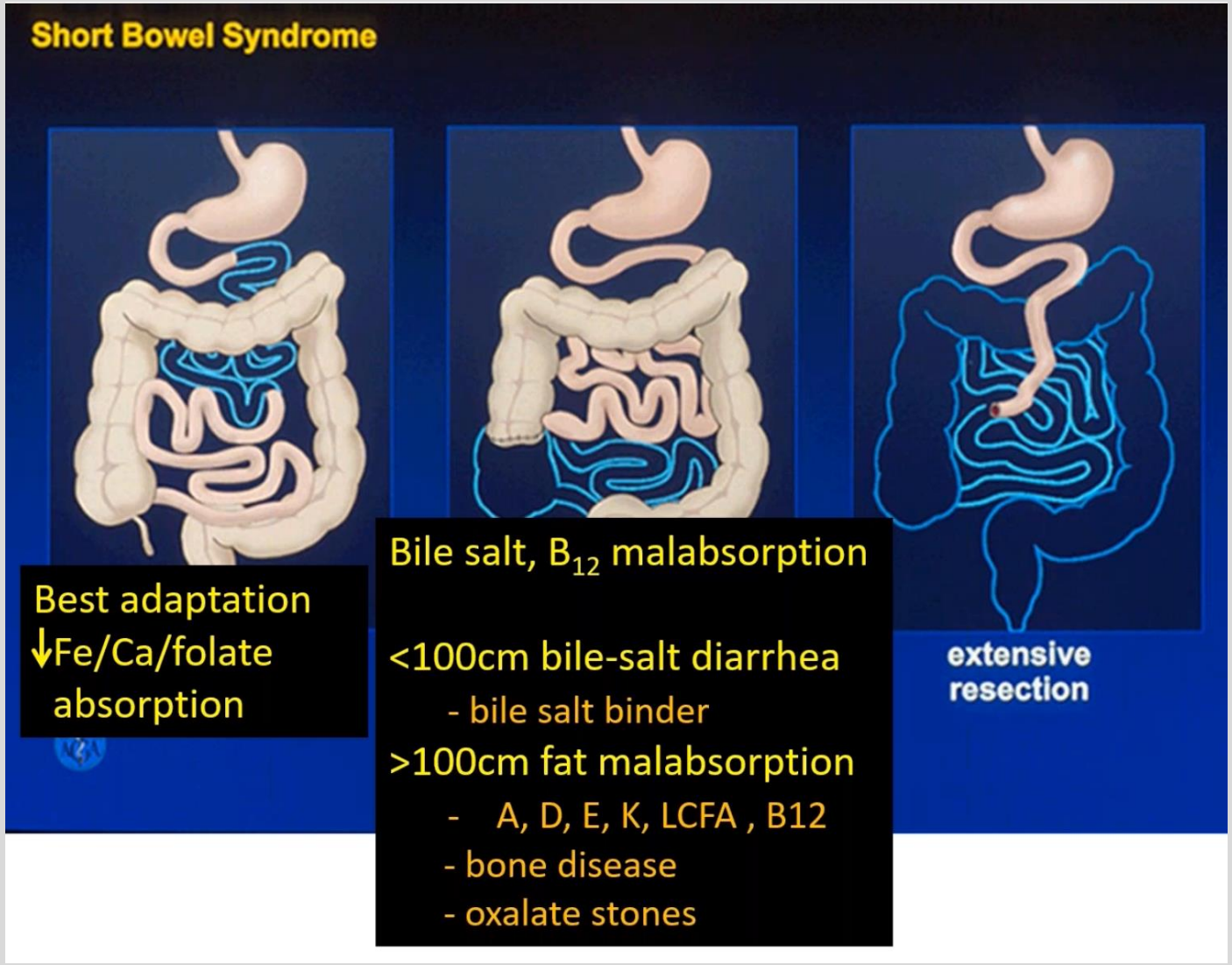
Disruption in enterohepatic circulation

↓

Bile Acid loss > Compensatory hepatic absorption

↓

Bile Acid deficiency



Features of ileum

Fluid absorption

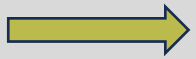
- Tight intercellular junctions
- Less water and sodium flux
- Active transport of NaCL
  - ➢ ++ Fluid reabsorption
  - ➢ Concentration of ileal contents

ILEAL Brake

- Unabsorbed lipids reaching ileum causes delayed gastric emptying
- Decreased transit time = Increased nutrient absorption
- Mediated by GUT hormones – GLP-2

Loss of IC valve

- **IC Valve** : regulates passage of fluid and nutrients from ileum to colon

Decreases intestinal transit  Increased absorption

- **++ SIBO** – Fat malabsorption and diarrhoea

- **Children**: Associated with difficulty weaning from TPN

Influence SBS on gastric secretion and pancreatic function

Loss of negative feedback for inhibiting gastric secretion and acid production

↓

Hypergastrinemia

Gastric hypersecretion

- Increased secretions entering proximal bowel →

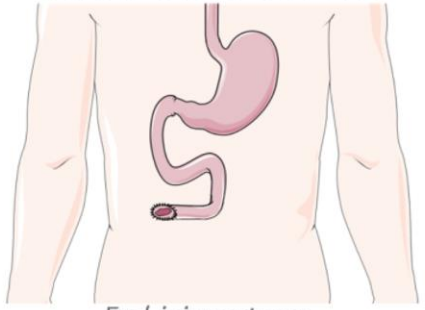
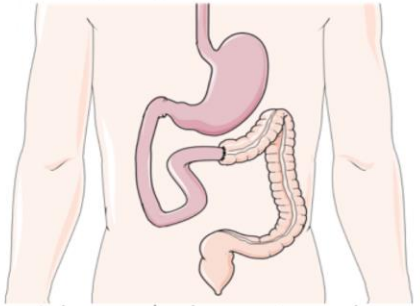
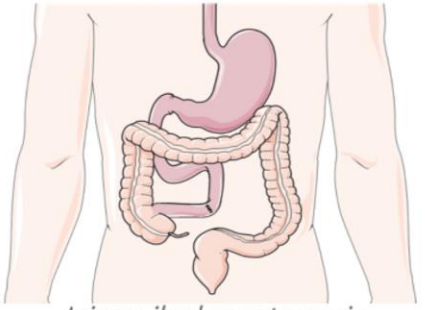
↓ pH
++ Fluid loss
Peptic complications

Patients have normal pancreatic and biliary secretions

Loss of colon

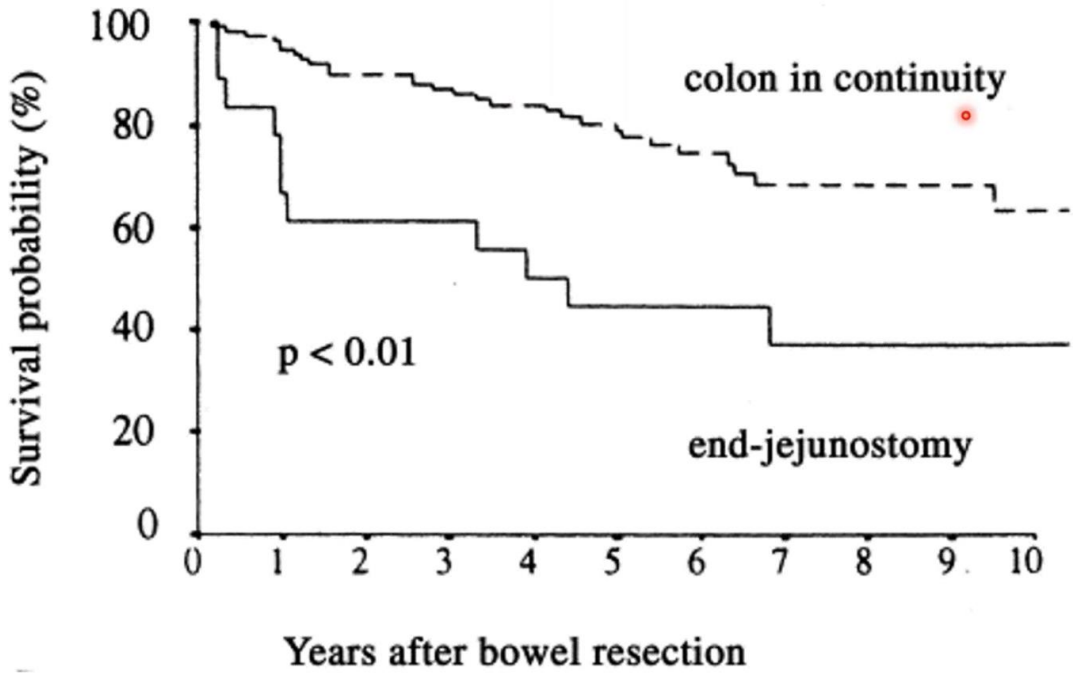
- Half Colon = 50 % small intestine

- Slowest transit and tight IC juction
  - ➢++ efficiency water and sodium absorption
  - ➢ Absorbs most of 1 – 1.5L/day that reaches the colon
  - ➢Can absorb up to 7L of water a day!
    - 700mmol of Na
    - 40mmmol of K
  - ➢Absorption of short chain fatty acids
  - ➢Colonic bacteria – absorb fermented carbohydrates

	COLON-IN-CONTINUITY		
	ANATOMY TYPE 1	ANATOMY TYPE 2	ANATOMY TYPE 3
	 *End-jejunostomy*	 *Jejuno-colonic anastomosis*	 *Jejuno-ileal anastomosis*
ETIOLOGY	**IBD > ISCHEMIA**	**ISCHEMIA > IBD**	**ISCHEMIA SURGICAL ADVERSE EVENTS**
PREVALENCE	**50 – 80 %**	**20 – 50 %**	**<10 %**
INTESTINAL FAILURE THRESHOLD	**100 cm**	**65 cm**	**30 cm**

Legend: IBD = inflammatory bowel disease

Reconnect Colon When Possible



Messing et al. Gastroenterology 1999;117:1043

INTESTINAL ADAPTATION

- Remaining small bowel undergoes macro- and microscopic changes to increase absorptive capacity

	Functional changes	Structural changes
Jejunum	**+ (better if in-continuity)**	**±**
Ileum	**+++**	**+++**

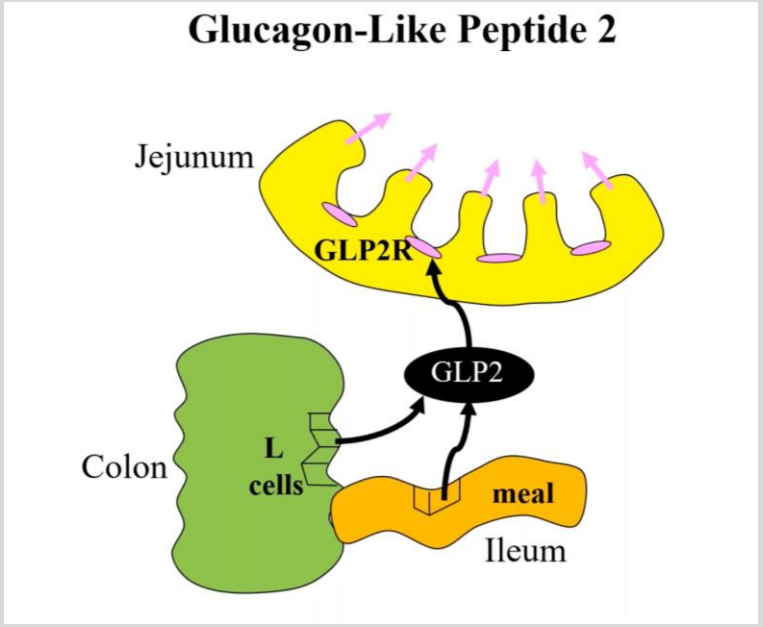
- Changes in GUT microbiota (esp loss of IC valve)
  - Loss of bacterial diversity
  - ++ Lactobacillus - **SIBO**

INTESTINAL ADAPTATION

- Nutrients in lumen = Release of trophic GUT hormones

Promote adaptation

GLP-2 = promote intestinal growth



- Gastrin
- Affect motility: Gastric emptying and intestinal transit (Loss of ileal break)

Clinical presentation of SBS

Diarrhoea/ Dehydration

- **High Output Stoma**
- Diarrhoea
- AKI – Renal stones
  (calcium oxalate)

Protein – calorie malnutirtion

- Weight loss > 10%
- Hypoproteinemia
- Oedema/Anasarca

Electrolyte and micronutrient deficiencies – Site of absorption

- Hypokalemia
- Calcium
- Magnesium
- B12
- Fat Soluble vitamins

Metabolic Bone disease

- Osteomalacia
- Osteoperosis
- Hyperparathyroidism

Hepatobiliary – TPN related

- Hepatic steatosis and Cholestasis
- Cholelithiasis (Cholesterol stones)

Oesophagitis/PUD

D-Lactic Acidosis - Lactobacillus

Diarrhoea - Multi-factorial

- ↓ Absorptive area
- ↓ Intestinal transit
- Hypersecretion
- Pancreatic exocrine dysfunction and fat malabsorption
- SIBO

High Output Stoma

- **HOS = Fluid, electrolyte and nutritional complications**

- Incidence : 16-30% of ileostomies
  - 7% require ongoing treatment

- 60-day ileostomy re-admission rates = 14%
  - **40% due to dehydration**

- Less common in colostomy patients
  - Exception proximal colostomy (extensive small bowel resection

Impact of HOS

- Increased hospital LOS
- Increased re-admission rates
- Impacts nutritional state
- Risk of renal failure
- Difficulty protecting skin

High Output Stoma

- Normal Ileostomy output: 600 – 1200ml/24 hours
- **>1500ml/24 hours for 48-72 hours**
  - ➢1L start worrying about dehydration
  - ➢>1,5L start worrying about malnutrition
  - ➢>2L You will need TPN

High Output Stoma - Physiology

- Loss of normal daily intestinal secretions
  - **Loss of Sodium and water**
- Excess consumption of hypotonic fluid
- Gastric acid hypersecretion
- Rapid GI transit – adverse impact on nutrition, drug absorption

Large patient variation in view of intestinal adaptation

Why is the output high

- **Short bowel**
  - How much do you need
  - Which is the best bit
- **Diseased bowel**
  - Crohn's
  - Radiation
  - Partial obstruction
- **Increased motility**

Sepsis in HOS

- SBS is the commonest underlying cause of HOS….*But*
- Sepsis drives stoma
- Sepsis should be actively looked for in every pt with HOS
- Ileus most common symptom with an anastomotic leak
- Does **not** have to be intra abdominal sepsis
- If abscess present - inflammatory markers are very unreliable

Cause of HOS

Box 1 High-output stoma

Early (<3 weeks of formation)

- Abdominal sepsis/ileus.
- Drug related
  - Prokinetic drugs (eg, metoclopramide).
  - Low cortisol.
  - Opiate withdrawal (eg, codeine phosphate).
- Enteritis (*Clostridium difficile*)

Late (chronic more than 3 weeks)

- Short bowel—jejunostomy <200 cm
- Intermittent/partial obstruction (strictures).
- Other less common causes:
- Recurrent disease.
- Internal fistula.
- Small bowel diverticula.
- Coeliac disease.
- Thyrotoxicosis.

SBS is the most common cause of HOS

Sepsis is the most comment acute precipitant of HOS

- **Incl non-abdominal sepsis**

↓

Hypoalbuminema

↓

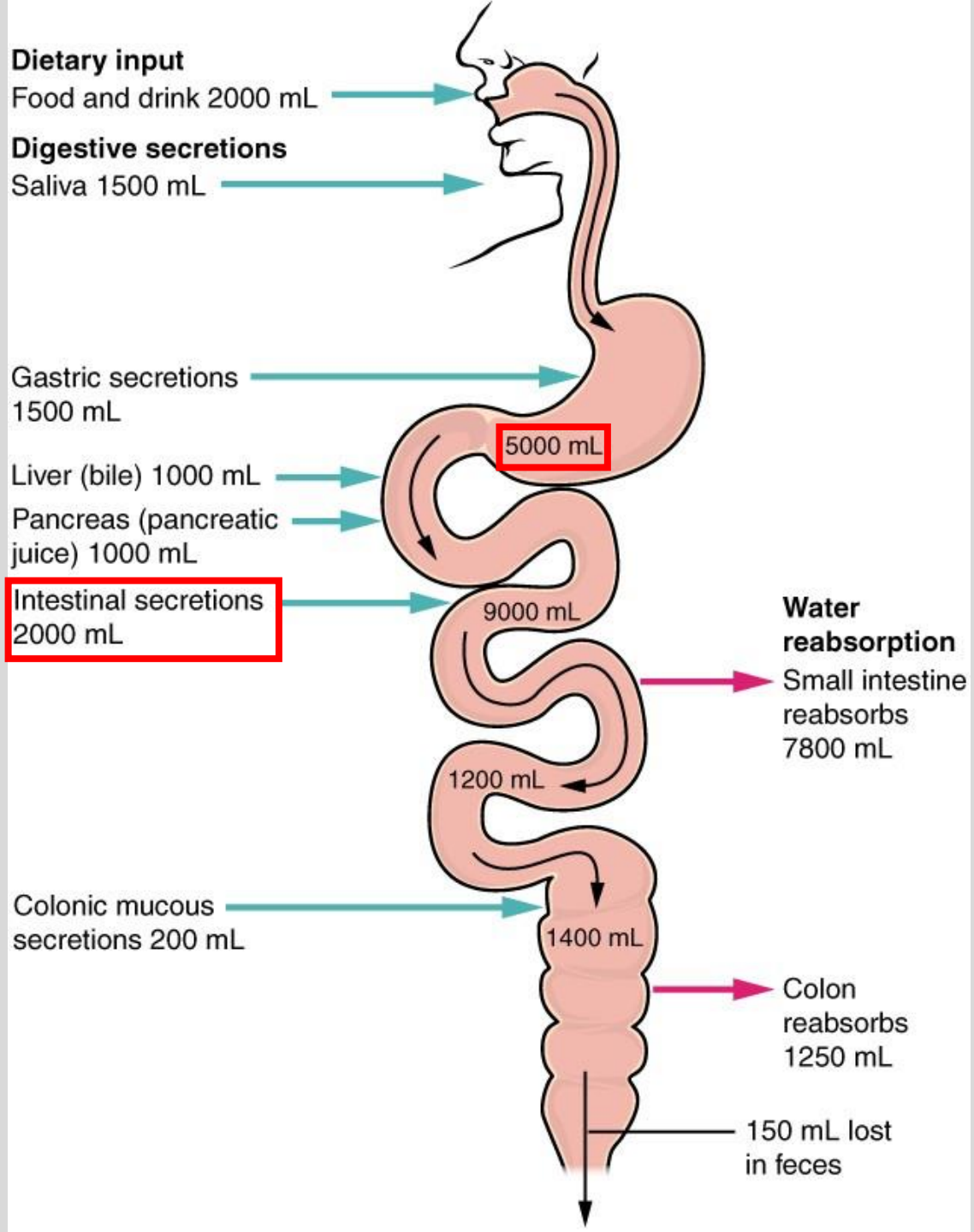
Oedema of stoma

↓

Outlet obstruction

HOS - Management

- Correct **dehydration** and **electrolyte imbalance**
- **Reduce output**
  - Pharmacological
  - Non-Pharmacological
- Support **nutrition**
- Identify and treat **underlying case of HOS**


Dietary input
Food and drink 2000 mL
Digestive secretions
Saliva 1500 mL
Gastric secretions
1500 mL
5000 mL
Liver (bile) 1000 mL
Pancreas (pancreatic
juice) 1000 mL
Intestinal secretions
2000 mL
9000 mL
Water
reabsorption
Small intestine
reabsorbs
7800 mL
1200 mL
Colonic mucous
secretions 200 mL
1400 mL
Colon
reabsorbs
1250 mL
150 mL lost
in feces

Movement of Na from plasma into jejunal lumen

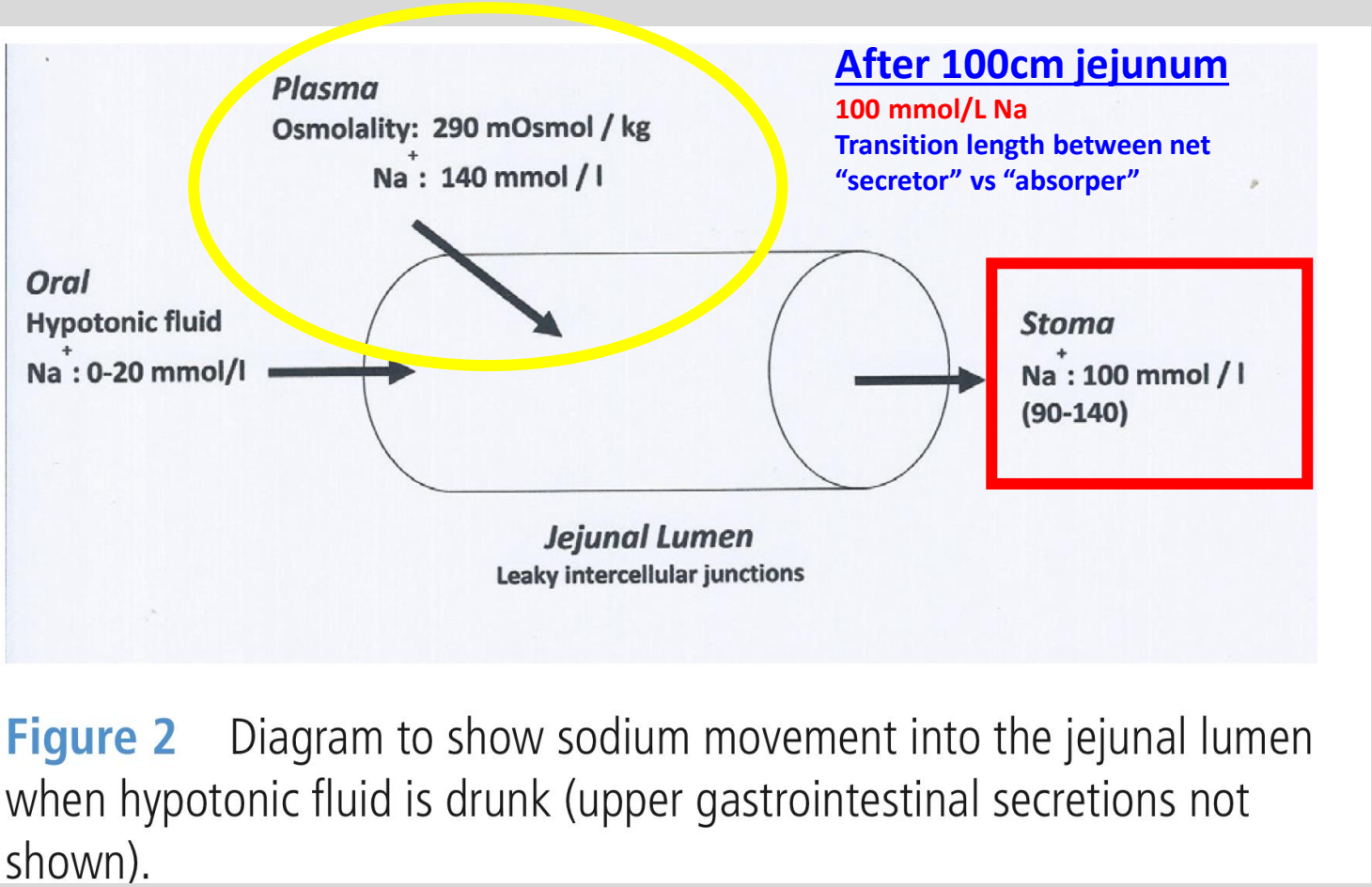
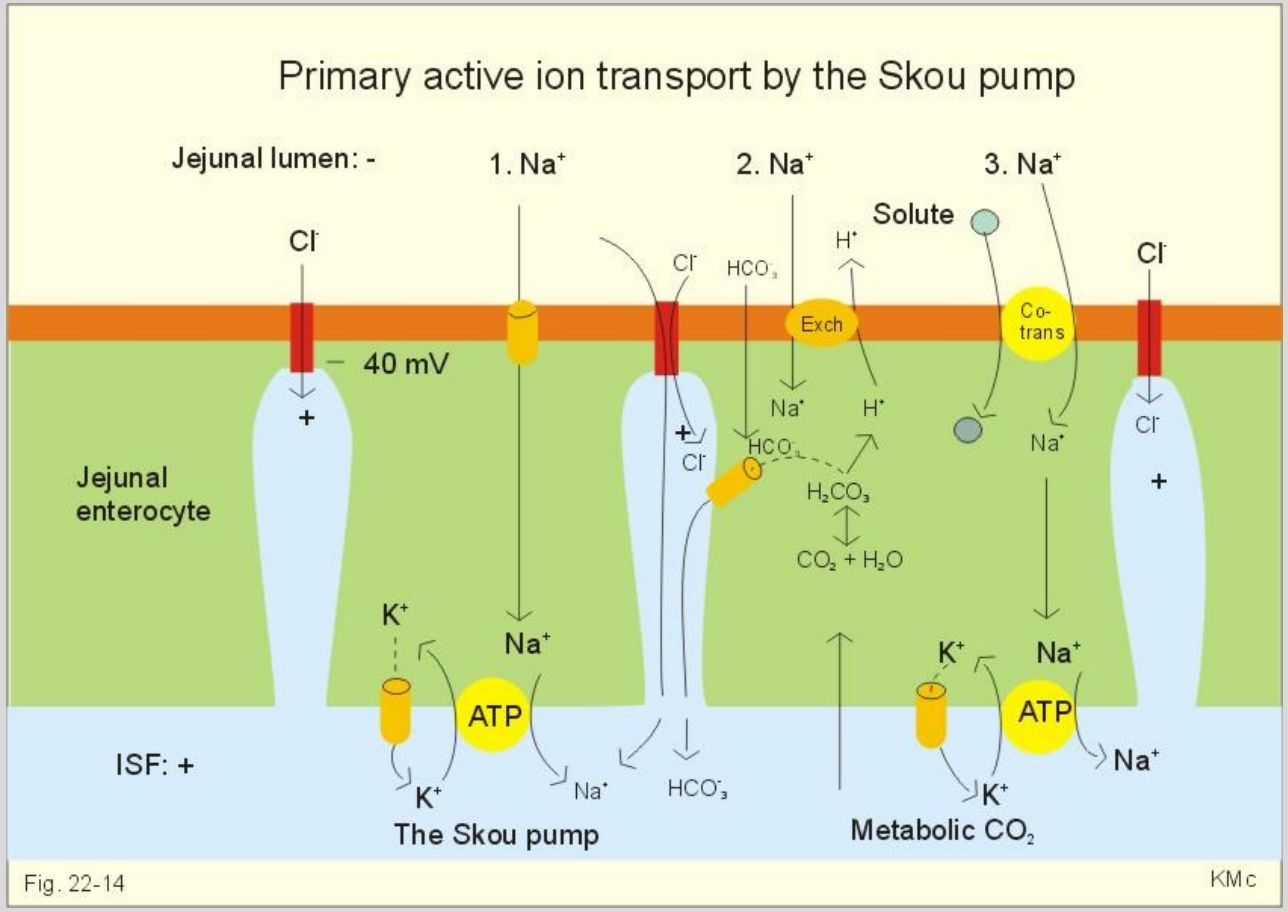


Figure 2 Diagram to show sodium movement into the jejunal lumen when hypotonic fluid is drunk (upper gastrointestinal secretions not shown).

Active sodium transport in Jejunum

- H2O absorption in the jejunum follows **sodium**
- Na concentration of the luminal content needs to be **>100mmol/l** otherwise net flow of fluid into the lumen
- ORS needs a Na concentration >100mmol



Thirst reflex

- Thirst centre located in the anterior wall of 3rd ventricle
- Thirst centre is distinct from hypothalamic osmoreceptors (responsible for ADH release)
- **Set point for ADH release is lower than thirst**
- Initial response from the body to dehydration is to activate **RAAS** release **ADH**
  - Causes reabsorption of sodium and water in the kidney
- When serum osmolality rises despite maximal resorption of free water by the kidneys under the action of ADH, thirst is **activated**
- **Thirst is a later adaptation to dehydration**

High Output Stoma

↓ Na

↓ water

Concentration sodium <u>unchanged</u>

↓

Delayed activation THIRST

↓

PROFOUND DEHYDRATION

++ Risk of AKI

ORS

- St Marks solution
- 1L of water
- 20g of glucose (six tablespoons)
- 3.5g of sodium chloride (one level 5-mL teaspoon)
- 2.5g of sodium bicarbonate (one heaped 2.5-mL teaspoon)
- May need to add Orange squash etc to make palatable

Na 50 mmol/L

Dietary Effects on Output – Fluids

Ingredient	ORS with Mg	St Mark's
Water	1000ml	1000ml
Salt	¾ teaspoon	1 teaspoon
Citric Acid	½ teaspoon	
Sodium Bicarbonate (baking soda)	¼ teaspoon	½ teaspoon
Glucose / Sugar	6 level teaspoons	6 level teaspoons
Magnesium effervescent tablet (Slow Mag Fizzy)	1 tablet	
Optional: Sugar free cordial such as Slimsy or LowCal	To taste	To taste

*90 mmol Na/L

Fluid restriction

- What goes in must come out
- Dry mouth from Immodium
- Constant water drinking
- Flushes luminal content out of ileostomy

- 500-1000ml ORS
- IV fluids
- A drink or 2 of patients choice
- Avoid caffeine

How do we reduce the output?

- This is the incorrect target
- Aim:

ABSORPTION

- **Drugs to decrease bowel motility**
- **Immodium/Loperamide – 250ml fluid reduction**
- **Opiates – Codeine Phosphate**

SECRETIONS

- **PPI (watch out for worsening hypomagnemia) - 500ml fluid reduction**
- **Octreotide (effect does not last)**

Slow transit

- **Loperamide**
  - 30min before meals
  - 2-8 tablets 6hrly
  - Dry mouth
  - At high doses QT interval, Torsades other ventricular arrythmia
- **Codeine Phosphate**
  - 30-60mg TDS
  - Addictive
  - Variable effect in combination with Loperamide

Cost
10 tablets of Adco Prodium R7.60
10 tablets of Imodium R88.10

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Oesophagitis/PUD

D-Lactic Acidosis - Lactobacillus

Correct electrolyte disturbances

- **HYPMAGNESEMIA**

➢ 45% patients with HOS

➢Reduction in absorption surface

  - Terminal ileum and Colon
  - Chelation with fatty acids

➢**Other:** ↓ **K** ↓ **PO4**

  - often refractory until Mg deficit corrected

Site specific Absorption

- **Vitamin B12**
  - Absorbed in TI

  > **60% TI resected:** risk for B12 def

  Bypassing stomach – lack intrinsic factor

- **Bile Acid**
  - Re-absorbed in TI

  ➢ **100cm TI resected** = Bile acid deficiency

  ➢ Fat malabsorption – steatorrhoea, malnutrition and fat soluble vit deficiency

Nutrition - Principles

- Close involvement of dietitian important in **ALL** patients
- Optimizing nutrition critical in short and long term (esp if future surgery planned)
- Rapid GI transit - ↓ absorption of all nutrients
- Improving nutrition may decrease HOS (250ml)
- **HOS > 2l per day = TPN**

Enteral Nutrition

- Preferred route
- Enteral nutrition = ⇧ **Absorption**
- Less infectious complications (eg line sepsis)
- Less bowel atrophy and adaptation

Compared to Oral feeding

	Exclusive ENTERAL	Combined Oral + Enteral
Protein absorption	↑ 15%	↑ 5%
Lipid absorption	↑ 28%	↑ 18%
Energy absorption	↑ 17%	↑ 10%

Indications for TPN in HOS

- Immediate post op – SBS
- High Output Fistulae (>500ml in fed state)
- **High Output Stoma (>2000ml)**
- **Already malnourished state**
- Supplemental PN

Eating-related behaviour to improve HOS

Consume more in the day/ less in the evening

- Positive effect on stoma volume and consistency

Avoiding fluids with meals

- Drink fluids in-between meals
- Improves output volume and consistency

Avoid hot/spicy foods

Meals

- Chew well
- Small, regular meals
  ≥ 6 snacks or meals/ day
- Eat slower
- Increase salt intake

Flatulence

- Chewing gum
- Drinking with a straw

Impact of fat intake

High fat-intake

↓

stimulate pancreaticobiliary secretions

↓

↑ Volume in intestine

↓

Watery an HOS

- **100cm TI resected = Bile acid deficiency**

↓

Fat malabsorption and steatorrhea

↓

Low fat diet may be beneficial

Nutritional requirements HOS

Carbohydrates

- Increase simple carbohydrates
  - ✓white rice, white bread,
  - ✓Pasta, marshmallows

Protein

High quality protein at each meal

Reduce fiber intake

- Avoid fresh fruit and vegetables
- Consider psyllium husk (Fybogel)
- Fibre particles absorb water and bulk up stool/enteric content

Oxalate

- Limit in patients with a colon
- Ensure adequate urine output

Decrease motility	Reduce secretions	Diet	Supplemental vitamins
• Loperamide • Lomotil/liquid opiates • Schedule before meals	• PPI • Octreotide (if exogenous losses > 3L)	• Separate solids and liquids • Remove hyper and hypotonic fluids • Small bowel stoma -> higher salt • Colon -> reduced fat, complex CHO	• Ileal resection: B12 • Fat soluble vitamins • Ca, Zn, Mg

When nothing works

- Gastroscopy for duodenal biopsies
- MRE to rule out partial obstructions
- Biopsies of the mucosa
- Distal ORS
- Distal feeding
- Refeeding enteroclysis

Conclusion

- Short bowel syndrome is associated with numerous short and long term complications that significantly affects patient's quality of life
- Knowing the length of residual bowel post-operatively is important
- Presence of colon-in continuity mitigates some of the complicates of SBS
- Ongoing bowel operations in patients with CD and SBS needs to be carefully considered
- Sepsis, fluid and nutritional management in patients developing HOS needs to carefully managed
- Critical importance of effective functioning of MDT

References/ Thank you

1. Assistance with lecture slides

- **Professor Adam Boutall** – Head of Colorectal unit at UCT/GSH
- **Anna-Lena du Toit** – Dietitian and head of Dietetics department at GSH

2. **American College of Gastroenterology** education lecture series on Short Bowel Syndrome (https://gi.org/education/)

3. **UpToDate.com**

4. Review articles and guidelines:

- Nightingale JMD, **How to manage a high output stoma. BMJ**, 2022; 13:140 - 151
- Cuerda C etal, **ESPEN Practical guideline: Clinical nutrition in chronic intestinal failure**
- HERPC Guideline for the medical management of high output stoma
- **University Hospitals of Leicester NHS Trust: Guidance for the Management of Adult patients with High Output Stoma**