Medicine

Marrow Edition 8

Volume - 1

MARROW

Instructions

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DIARRHEA

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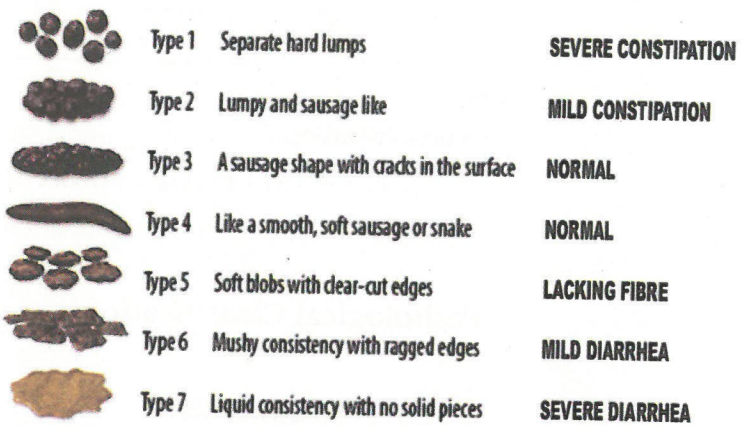
Definition :

- Stool water content >200 mL/24 hours.
- According to Bristol stool chart :
  - Type 1 & 2 : Constipation.
  - Type 6 & 7 : Diarrhea.



Bristol stool chart

Physiology of absorption :

1. Luminal.
2. Mucosal :
   - Small intestine (majority).
   - Maximum absorption.
3. Post mucosal.

Macronutrients for absorption :

- Fat :
  - Most calorie dense nutrient.
  - Most specific nutrient affected in malabsorption.
- Carbohydrates.
- Proteins.

Frequency Classification

00:03:00

	Acute	Persistent	Chronic
Duration	<2 weeks (Nuisance symptom)	>2 weeks	>4 weeks

ETIOLOGY

Acute diarrhea :

Infectious : Viral infection (m/c).

- m/c in adults : Norovirus.
- m/c in children : Rotavirus.
- Self-limiting.

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Chronic diarrhea :

D/d	Etiology
Osmotic	malabsorption
Secretory	• Tumor • Toxin
Inflammatory	Inflammatory bowel disease

Note :

Malassimilation :

- Maldigestion + malabsorption.
- Diminished intestinal digestion/absorption of one or more nutrients.

Pathological Classification

00:10:13

1. OSMOTIC DIARRHEA

Most consistent clinical symptom of malabsorption.

Steatorrhea :

Hallmark of malabsorption.

Definition : Passing of pale, bulky, fatty, malodorous, greasy stools.

Clinical presentation : Diarrhea (most consistent clinical finding).

Pathology :

↓ fat absorption in small intestine

↓ In colon

Unabsorbed macronutrients ↑ stool osmolality (osmotically active)

↓

Stool draws water from large intestinal epithelial cells

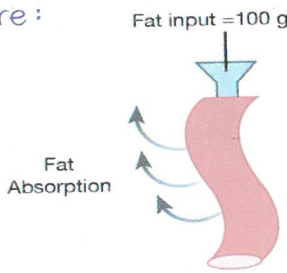
↓

Osmotic diarrhea

Investigations :

1. 72 hour fecal fat test : Gold standard.

Procedure :



Normal : <7 g/day

- >7 g/day for 3 days (D3, D4, D5)
- Stool fat excretion ≥7 %

} Steatorrhea

2. Qualitative stool fat :
   - Sudan III.
   - Sudan IV.
   - Oilred O.

2. SECRETORY DIARRHEA

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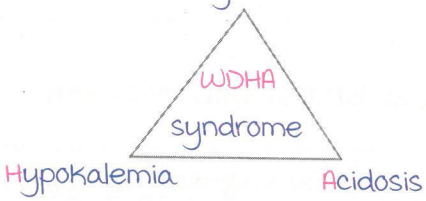
Etiology :

a. Toxins → ETEC, V. cholera (heat labile toxin)
a. Toxins → Enteropathogenic virus (rotavirus)

b. Tumours : VIPoma → Watery Diarrhoea



Note : No structural damage seen.

	Osmotic diarrhoea	Secretory diarrhoea
Stool osmotic gap	Increased (>100 mOsm/kg)	Normal/decreased (< 50 mOsm/kg). ↑ stool $Na^+ + K^+$ (By toxins).
Response to fasting	Improvement	No change
Stool pH	<5.5 (Fermentation of unabsorbed carbohydrates → Acids)	>6.0

Note : Stool osmotic gap

- measured osmolality (osmometer) − calculated osmolality
  → 290 − 2 (Stool $Na^+ + K^+$)
- Normal : 50–100 mOsm/kg.

3. FACTITIOUS DIARRHEA

Clinical presentation :

- Adolescent female.
- Chronic diarrhea.
- Fatigue.
- Weight loss.

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Findings :

Stool collection	Stool osmolality
Unsupervised (mixed with water)	very low (~ 16 mOsm/kg)
Supervised	Normal (~ 279)

Anatomical Classification

00:32:48

SMALL INTESTINAL V/S LARGE INTESTINAL DIARRHEA

		Small intestinal diarrhea	Large intestinal diarrhea
Etiology		• Osmotic. • Secretory : – ETEC. – Vibrio cholera.	• Infections : Invasive organisms (Shigella). • Inflammation : Ulcerative colitis.
Clinical presentation		Large (↓ nutrient absorption)	Small
Diarrhoea	Volume		
	Consistency	Watery	Watery
	Frequency/ urgency	↓	↑
	Pus/blood/ mucus	Absent	Present
	Tenesmus	Absent	Present
	Dyschezia	Absent	Present
Abdominal pain		Absent	Present
Fever		Absent	Present
Abdominal cramps		Present	Present
Bloating		Present	Present
Weight loss		Present (If persistent)	Rare
Vomiting		Maybe present	Rare
Steatorrhoea		Present	Absent

APPROACH TO DIARRHEA

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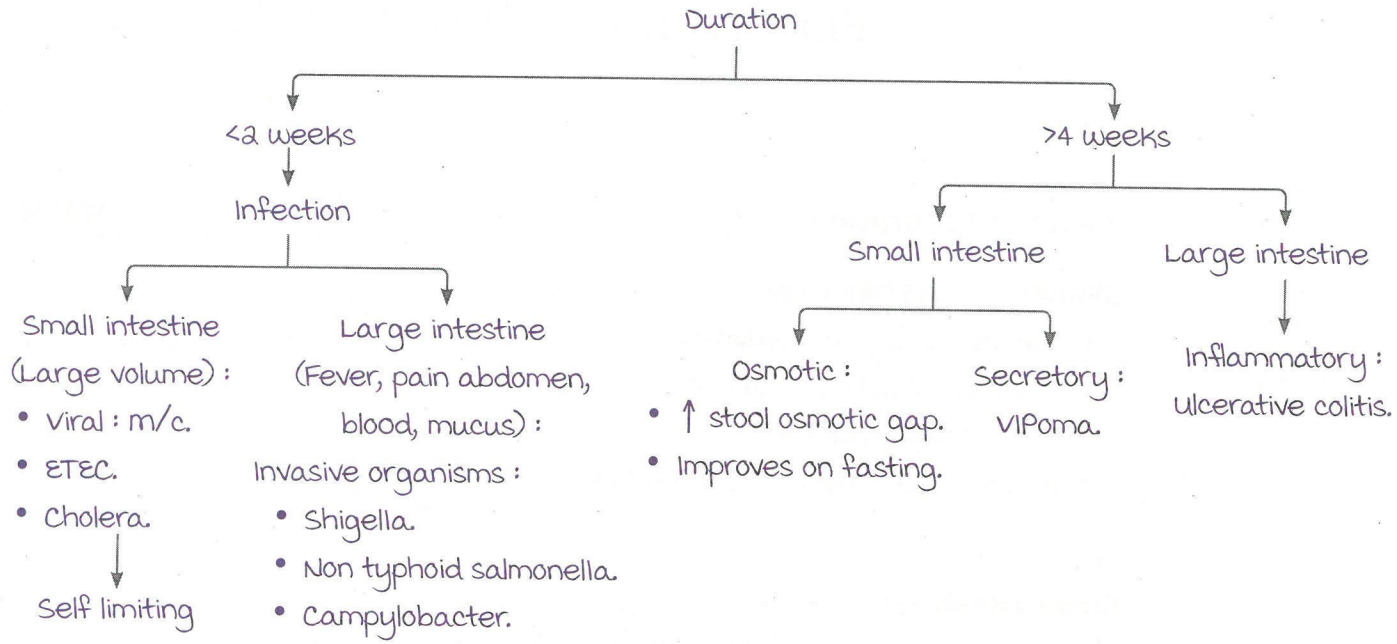

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PHYSIOLOGY OF GIT ABSORPTION AND SELECTIVE MALABSORPTION

Basics of Absorption

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STAGES OF ABSORPTION

1. Luminal : Enzymes mediated.
2. Small intestinal mucosal : Enterocyte.
   Villi → Brush borders contains enzymes.
3. Post mucosal : Mucosal epithelial cell → Into blood.

ANATOMY

Small intestine :

Proximal (2/5) : Maximum absorption (Jejunum > Duodenum).

Distal (3/5) : Absorption of Bile acid, Vit B_{12}, Mg^{2+} (Ileum).

Histology :

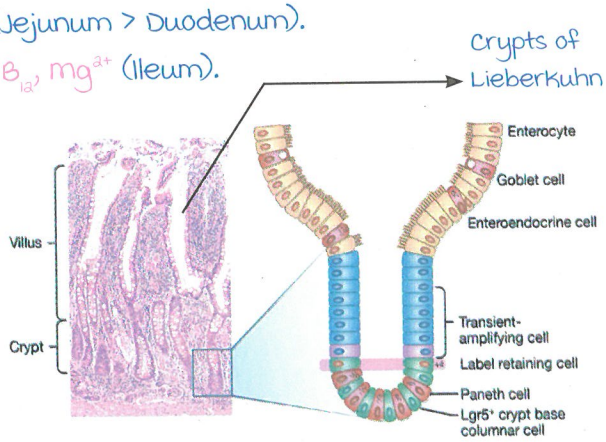
1. Mucosa :

a. Enterocyte :

- Villi (specific feature) :
  - Surrounded by crypts of Lieberkuhn.
  - Contains brush-border.
- Crypts :
  - Paneth cells (at base) : Produce immunological substances (alpha defensins, lysozymes, phospholipase A2).
  - Enteroendocrine cells.
  - Goblet cells.
- Continuous renewal : every 48–72hrs.

b. Lamina propria : Glands, blood vessels.

c. Muscularis mucosa : Tight attachment.



Small intestine mucosa with villi and crypts

Note :

Plica circularis : Visible mucosal folds on luminal surface of small intestine.

Interstitial cells of Cajal : Pacemaker cells of small intestine → Basal Electrical Rhythm.

2. Submucosa :

- Brunner's glands (In duodenum) → Secretes bicarbonate (Alkaline).
- Peyer's patches (In ileum).
  - Macroscopic lymphoid aggregates.
  - In typhoid : Hypertrophy.

3. Muscularis Propria.

4. Serosa.

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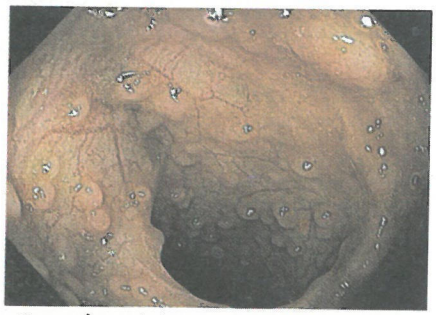


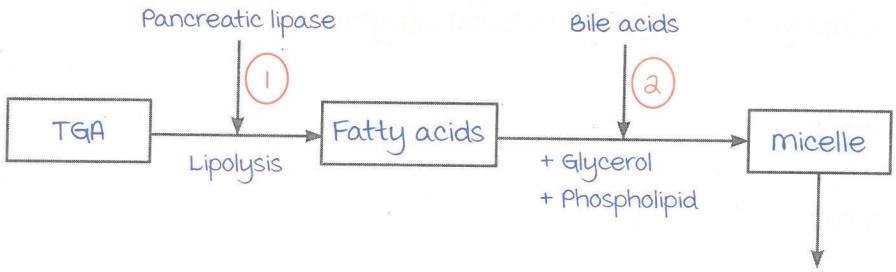
Peyer's patches on ileocolonoscopy

Fat Absorption

00:09:45

LUMINAL PHASE OF ABSORPTION

Mechanism :

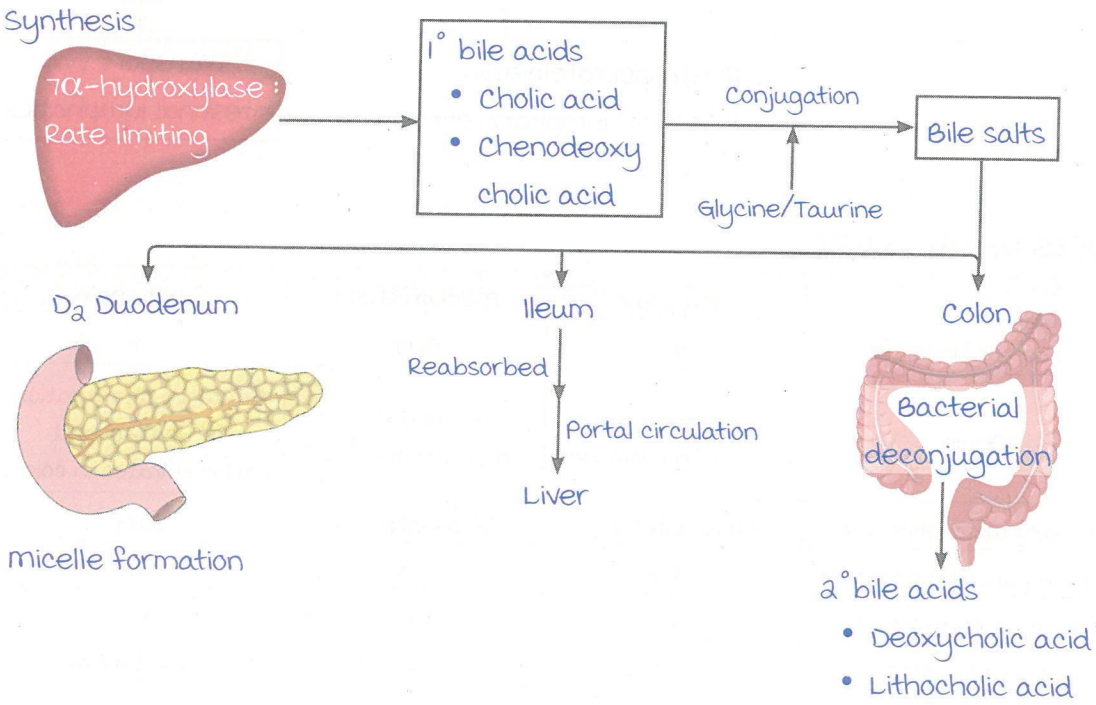


1. Chronic pancreatitis
2. Bile acid defect

Bile Acid Physiology :

- Formation & excretion : 500 mg.
- Body pool : 4g (maintained by enterohepatic circulation).

Enterohepatic circulation :



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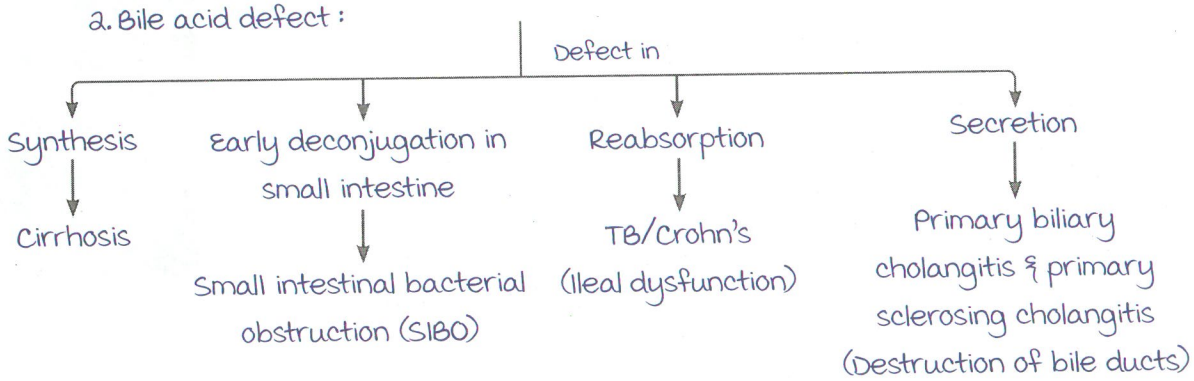
Defects in luminal absorption :

1. Chronic pancreatitis → Affects pancreatic lipase → Lipolysis affected.
2. Bile acid defect :



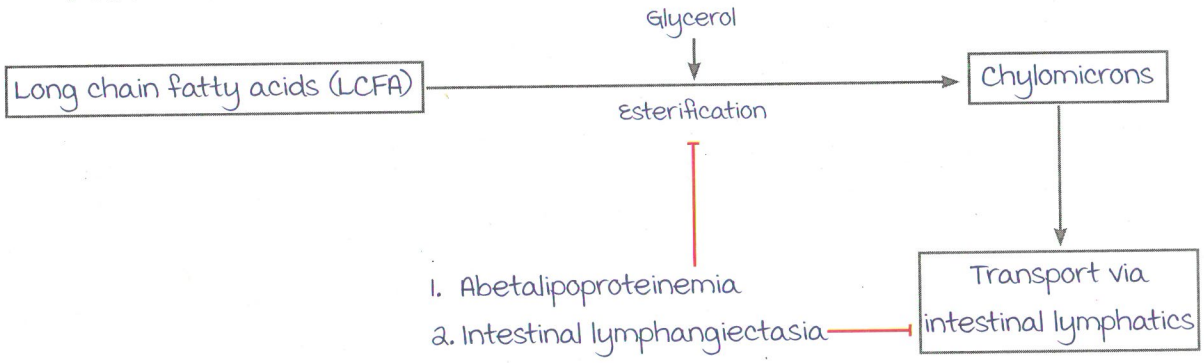
MUCOSAL PHASE OF ABSORPTION

Pathology : Defect in small intestinal mucosal integrity.

Etiology :

- Celiac disease.
- Whipple disease.
- Tropical sprue.

POST-MUCOSAL PHASE OF ABSORPTION



TYPES OF FATTY ACIDS

	Long chain	Medium chain	Short chain
Carbon chain length	>12	8–12	<8
Origin	Large quantity in diet as triglycerides	Small quantity in diet (Coconut oil)	Bacterial degradation of unabsorbed carbohydrates in colon.
Primary absorption site	Small intestine	Small intestine	Colon
Require pancreatic lipolysis	Yes	No	No
Require micelle formation	Yes	No	No
Present in stool	Minimal	No	Substantial

Carbohydrate Absorption

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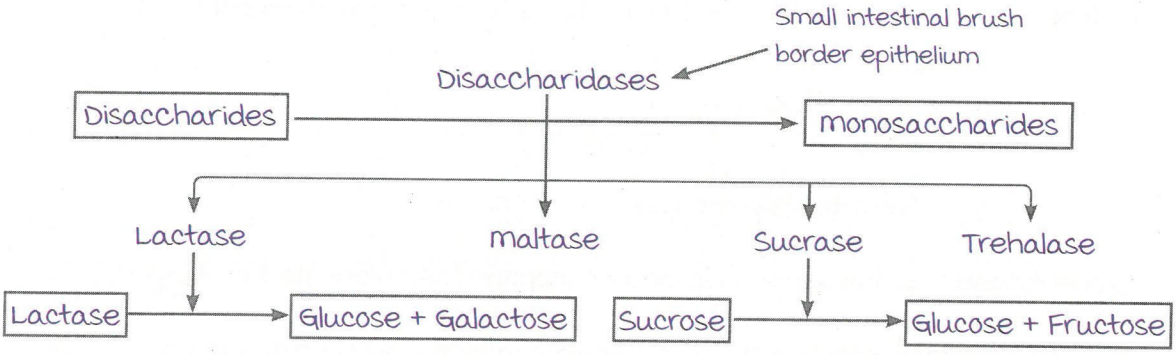
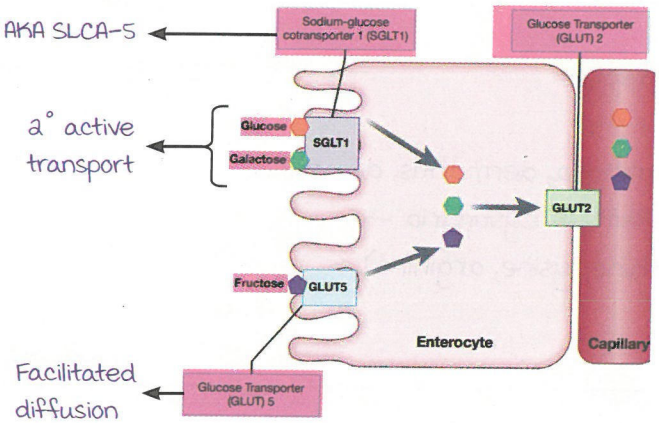
MECHANISM OF CARBOHYDRATE ABSORPTION

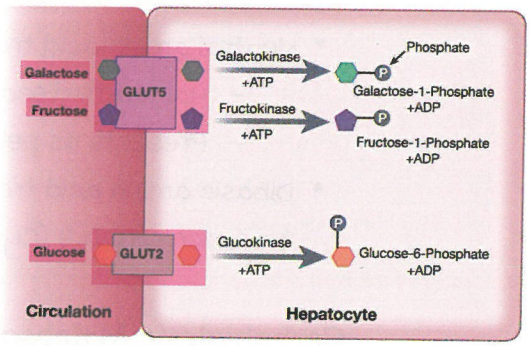


Receptors for absorption :

In the gut :



In the liver :



Note :

SGLT-1 defect → Familial glucose-galactose malabsorption syndrome.

DEFECTS IN CARBOHYDRATE ABSORPTION

Pathology	Etiology	Malabsorption
Lactase deficiency	• Congenital : Autosomal recessive. • Acquired : – Primary. – Secondary.	Selective.
Small intestinal mucosal defect	• Celiac disease. • Whipple's disease. • Tropical sprue.	Global.

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Proteins

00:34:00

Mechanism of absorption :

Endopeptidase : EnteroKinase (Produced by enterocyte brush border).

Trypsinogen → Trypsin

Trypsin → Activate chymotrypsin, elastase.

Exopeptidase : Carboxypeptidase and aminopeptidase (Activated by trypsin).

Transport in small intestinal mucosa : Sodium-amino acid co-transporters.

Defects in protein absorption :

Global malabsorption : Celiac disease, Whipple disease, Tropical sprue.

Selective malabsorption :

- EnteroKinase deficiency.
- Neutral amino acid transport defect :
  - Hartnup's disease.
  - Presents as pellagra : Diarrhoea, dermatitis, dementia.
- Dibasic amino acid transport defect → Cystinuria → Excretion of COLA (Cystine, ornithine, lysine, arginine).

Carbohydrate Malabsorption

00:39:45

LACTOSE INTOLERANCE (LACTASE DEFICIENCY)

Physiology :

Lactose (Dietary : Milk, cheese)

Lactase | Brush border

Glucose + Galactose

Etiology :

Congenital : Autosomal recessive (Absent enzyme from birth).

Acquired : ↓enzyme activity.

- Primary (m/c) : Genetic defect (allele persistence).
- Secondary :
  - Infections.
  - Irritable Bowel Syndrome (IBS).