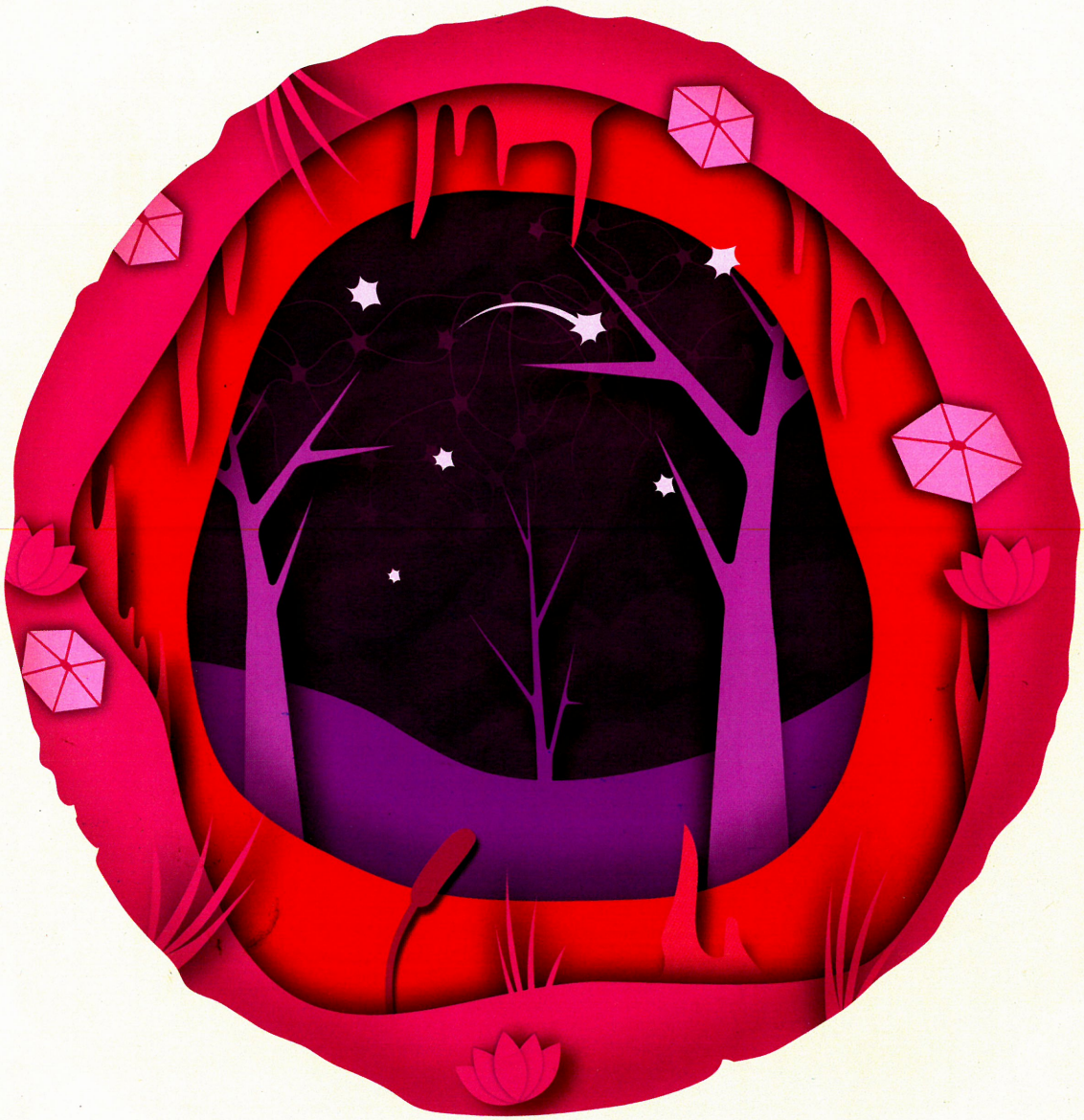
GENERAL MEDICINE 2

Marrow SS Medicine



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Dr. Rakesh Nair has over 10 years of experience teaching students for PG and SS entrance exams. He completed his MD in General Medicine from Stanley Medical College, his DM in Nephrology from GMC Thiruvananthapuram, and holds MRCP (UK), EBEEDM, and RCP (Ireland) certifications. He also serves as a Consultant Nephrologist at Welcare Hospital.

General Medicine

Volume - 2

MARROW
Super Speciality

Instructions

- The notes must be used in conjunction with the Marrow SS Medicine videos and should not be considered standalone material.

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- This set contains notes of all main videos published in the app until Jan 2025. You can find notes for any additional videos published after this date within the app under the videos section.

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PHYSIOLOGY OF ABSORPTION

----- Active space -----

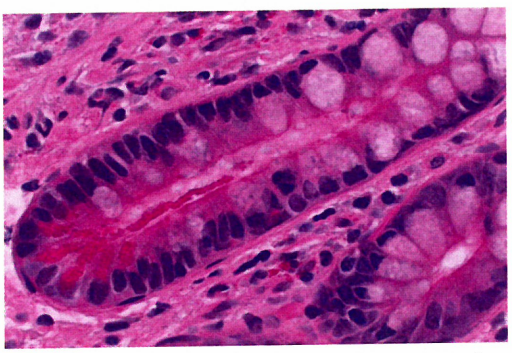
Introduction

00:00:28

Average length of small intestine : 500 cm
Average length of large intestine : 5 feet
Small intestine < 200cm : Short bowel syndrome.

Features of small intestine :

- Villi (absorption) and crypts (secretion).
- Continous renewal of intestinal epithelial cell occurs every 48-72 hours.
- Cylindrical structures surrounding villi are called Crypts of Lieberkuhn.
- Alpha defensins, lysozymes, phospholipase A2 are produced by Paneth cells.



Paneth cells, located at the base of the crypts of the small intestinal mucosa with bright red cytoplasmic granules.

- Luminal surface of small intestine has visible mucosal folds called Plica circularis.
- Interstitial cells of Kajal are pacemaker cells of small intestine.
- Brunner's gland are submucosal glands in duodenum which secretes bicarbonate rich alkaline juice
- macroscopic lymphoid aggregates in ileum are called Payer's patches.

Enterohepatic circulation

00:04:54

Fat (most calorie dense nutrient) absorption happens in the proximal small intestine (jejunum).
Fat absorption happens only by the formation of micelles.
Bile acids are required for micelle formation.

----- Active space -----

Enterohepatic circulation :

Primary bile acids : Cholic acid, chenodeoxycholic acid are formed in the cytoplasm of the liver (7 hydroxylase-α is the rate limiting enzyme

↓

Conjugation with glycine or taurine to form conjugated bile acids (bile salts)

↓

and part of deudenum

Formation of micelles and fat absorption

↓

Terminal Ileum

↓

95 % of the bile salts reabsorbed (enterohepatic circulation)

Remaining 5 % of the bile salts reach colon

↓

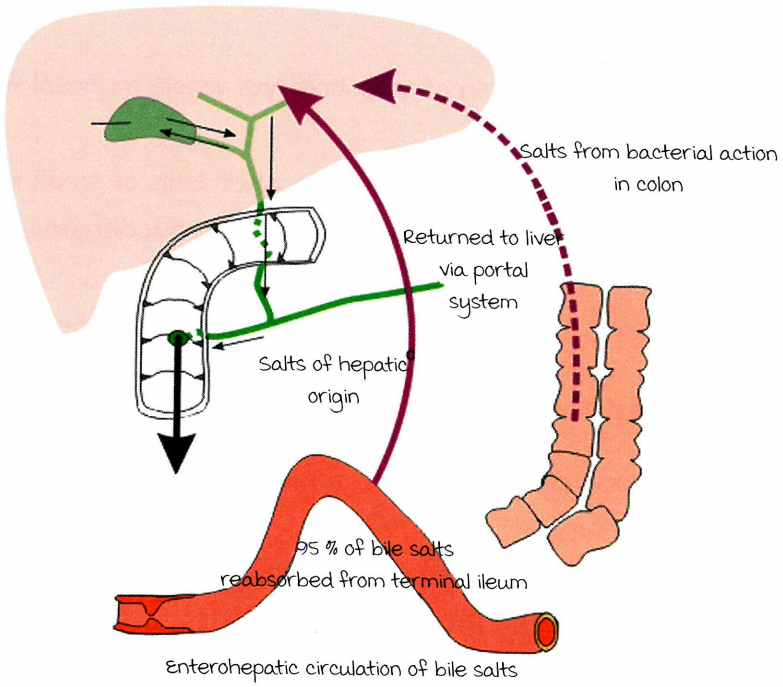
Deconjugated by bacteria in the colon

↓

Converted to secondary bile acids (deoxycholic acid and lithocholic acid)

Note:

- Bile acid pool : 4g
- Amount of bile acid produced per day : 500 mg
- Amount of bile acids excreted per day : 500 mg
- Bile reabsorption in the terminal ileum is via Na^{2+}-dependent active transport process



Enterohepatic circulation of bile salts

Bile acid defect : malabsorption of fats ⟶ Steatorrhea⟶ diarrhea

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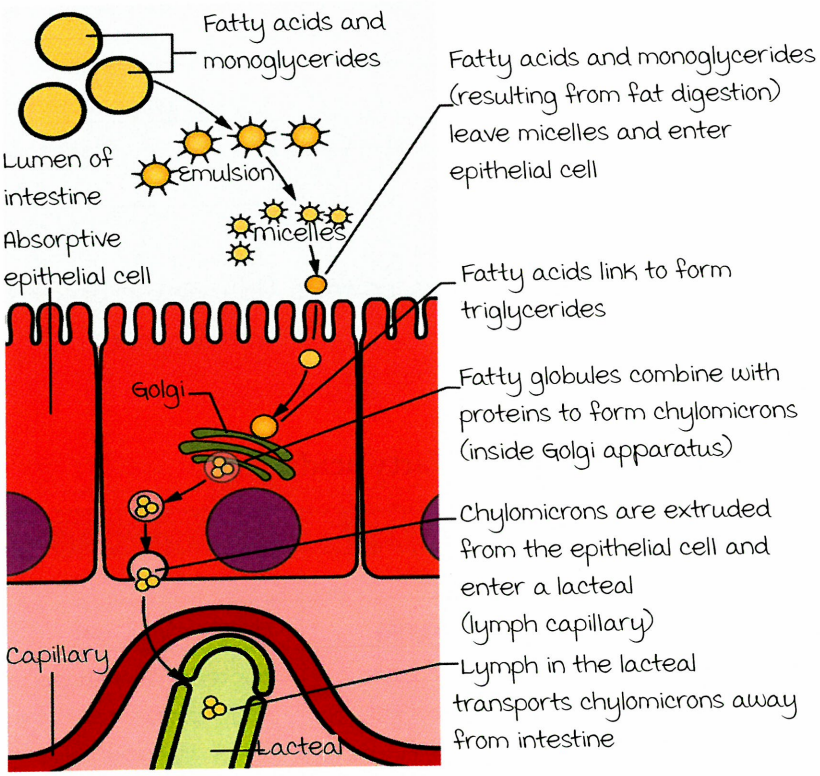
Steps of fat absorption

00:15:02



1. micelle formation
2. micelle absoprtion across the intestinal epithelial cells of jejunum.
3. Esterification : Fatty acids released from micelles linked to glycerol to form triglycerides.
4. Chylomicron formation in the golgi apparatus : Apoprotein (Apo B 48) combine with lipid to form apolipoprotein.
5. Chylomicrons are extruded from the epithelial cells and enter the lymphatics.
6. Transportation of chylomicrons away from the intestine.

Defects in lipid digestion and absorption in steatorrhea :

Phase : Process	Pathophysiologic defect	Disease example
Bile acid synthesis	Decreased hepatic function	Cirrhosis
Transport of bile acids	Detruction of the bile ducts	PBC, PSC
micelle formation	Deconjugation of the bile acids	Small intestinal bacterial overgrowth (SIBO)

----- Active space -----

Lipolysis formation	Decreased lipase secretion	Chronic pancreatitis
Mucosal uptake and reesterification	Mucosal dysfunction	Coeliac disease Whipple's disease Tropical sprue
Chylomicron formation	Absent betalipoproteins	Abetalipoproteinemia
Delivery from intestine	Abnormal lymphatics	Intestinal lymphangiectasia
Enterohepatic circulation	Abnormal ileal function	Crohn's disease TB

Note :

- Global malabsorption (Carbohydrates, proteins, fat malabsorption) is seen in :
  - Coeliac disease
  - Whipple's disease
  - ropical sprue

Comparision of different types of fatty acids

00:20:26

	Long chain	Medium-Chain (MCFA)	Short-Chain (SCFA)
Carbon chain length	12	8-12	<8
Present in diet	In large amounts	In small amounts	No
Origin	In diet as triglycerides	Only in small amounts in diet as triglycerides	Bacterial degradation in colon of non-absorbed carbohydrates to fatty acids (not in diet)
Primary site of absorption	Small intestine	Small intestine	Colon
Requires pancreatic lipolysis	yes	No	No
Requires micelle formation	Yes	No	No
Present in stool	Minimal	No	Substantial

Comparison of bile acid and fatty acid diarrhea

00:25:00

- Fatty acid diarrhea : Seen in extensive ileal disease resulting in steatorrhea which causes diarrhea when it reaches colon.
- Bile acid diarrhea : Seen in limited ileal disease where the defective bile acid reabsorption is compensated by hepatic synthesis. The bile acids reaching the colon cause diarrhea

	Bile acid diarrhea	Fatty acid diarrhea
Extent of ileal disease	Limited	Extensive
Ileal bile acid absorption	Reduced	Reduced
Fecal bile acid excretion	Increased	Increased

----- Active space -----

	Bile acid diarrhea	Fatty acid diarrhea
Fecal bile acid loss componsated by hepatic synthesis	Yes	No
Bile acid pool size	Normal	Reduced
Intraduodenal (bile acid)	Normal	Reduced
Steatorrhea	None or mild	> 20 g
Response to Cholestyramine (bile acid binding drug)	Yes	No
Response to low fat diet	No	Yes

Carbohydrates

00:29:29

Site of absorption : Proximal small intestine

Absorbed as monosacharides :

- Glucose, Galactose } Via SGLT-1 (Sodium dependent glucose transporter-1), secondary active transport
- Fructose : GLUT- 5 (facilitated diffusion)

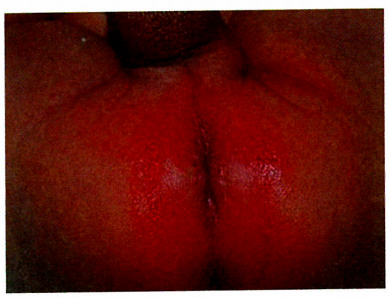
Note :

- SGLT- 2 : Predominently seen in PCT
- SGLT- 1 : Predominent in brush border of the small intestinal mucosa (jejunum).
- SGLT- 1 defect : Familial glucose galactose malabsorption syndrome (rare)

Lactose intolerance :

1. Primary lactose intolerance :

- Defect in lactase enzyme
- Child presents with failure to thrive, irritability, inability to gain weight, vomiting, watery diarrhea.



Tests to diagnose primary lactase deficiency :

- Stool pH : Acidic (presents as diaper dermatitis)
- Stool reducing substance : Positive

2. Secondary lactose intolerance : Associated with other diseases which affect the intestinal mucosal epithelial integrity

Adult : IBS like symptoms

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Protein malabsorption

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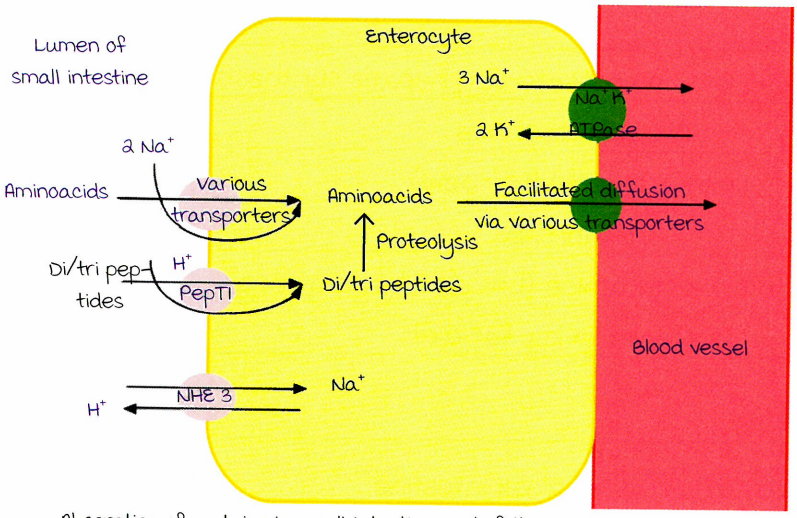

Absorption of proteins in small intestine and of then into blood cycle

- Enterokinase coverts trypsinogen → trypsin
- Proteolysis : pepsin / trypsin
- Enterokinase deficiency : Protein malabsorption is seen

- Neutral amino acid transporter defect : Hartnups disease (presents like pellagra with dermatitis, diarrhea, dementia).

- Dibasic aminoacid transporter defect : Cystinuria (COLA = Cystine, Ornithine, Lysine, Arginine in urine).

GASTRIC ANATOMY / GASTRITIS

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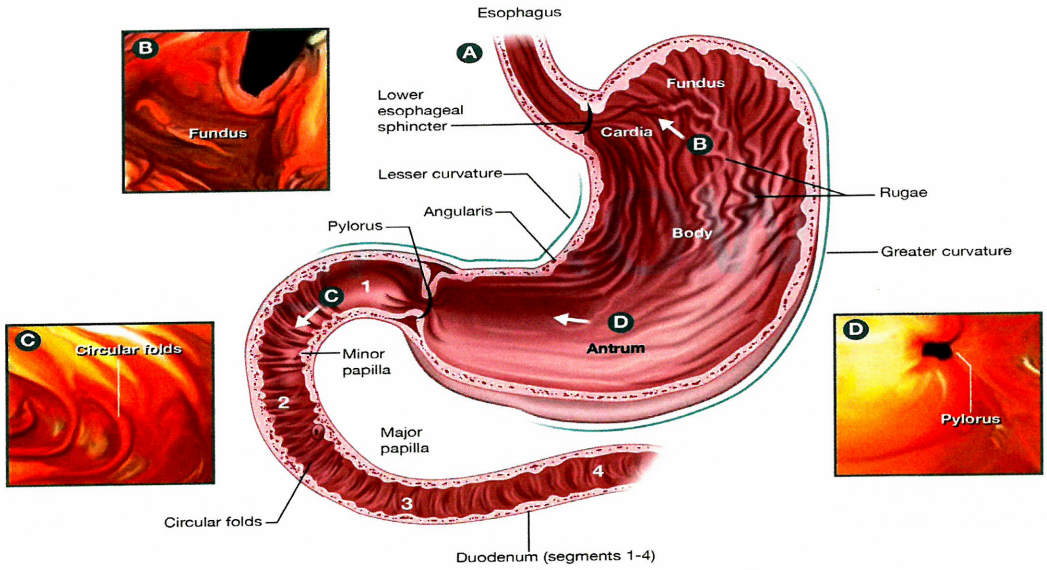
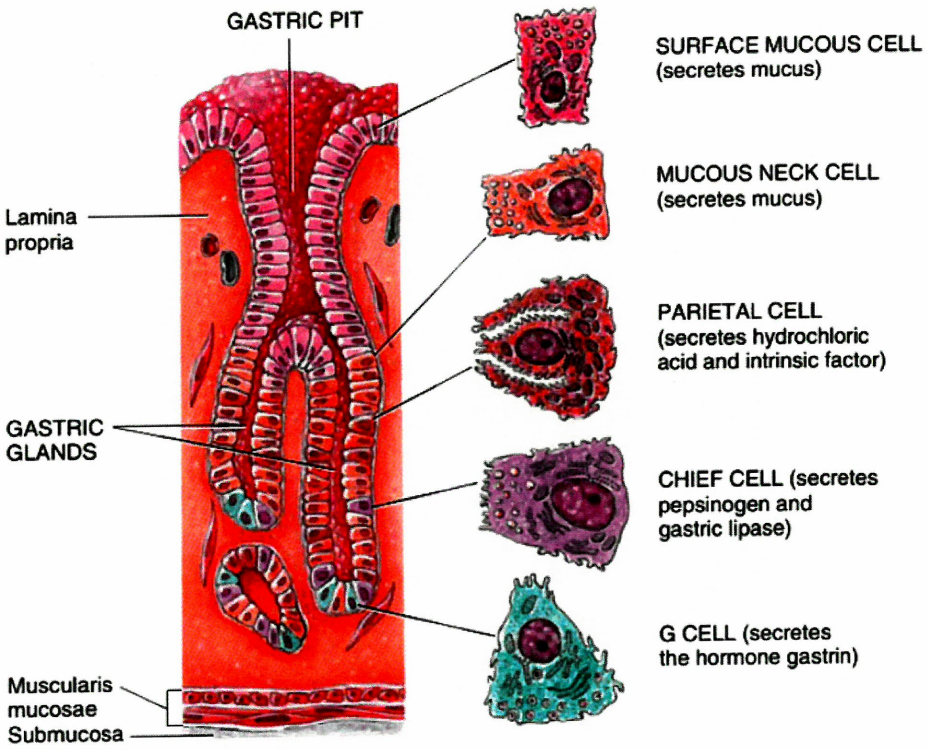
Anatomy

00:01:10



Parts of stomach



Anatomy of stomach

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Parts of stomach :

- Fundus
- Body/corpus : Contain rughe
- Cardia : Not defined correctly, belived to be a 3cm narrow passage between NK squamous epithelium of oesophagus and gastric epithelium
- Antrum : Below incisura angularis., with pylorus.

Fundus :

- Contains mucous and endocrine cells.
- ECL (enterochromaffin like cells) produces histamine.

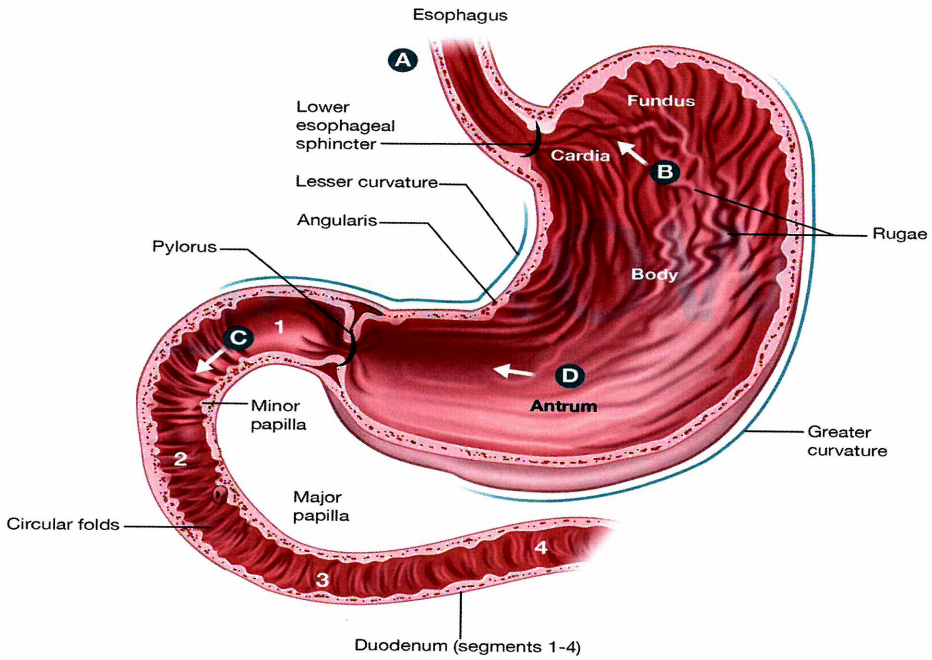
Body :

Contain oxyntic glands.

- Less number of mucous and endocrine cells.
- Oxyntic/parietal cells :
  - At the level of neck/isthmus.
  - Produces acid and IF.
- Chief cells which produce pepsinogen are present in the base.

Antrum/pylorus :

- Contains pyloric glands.
- Also contain G cells that produce gastrin.



Parts of stomach

Layers of gastroduodenal mucosal defence :

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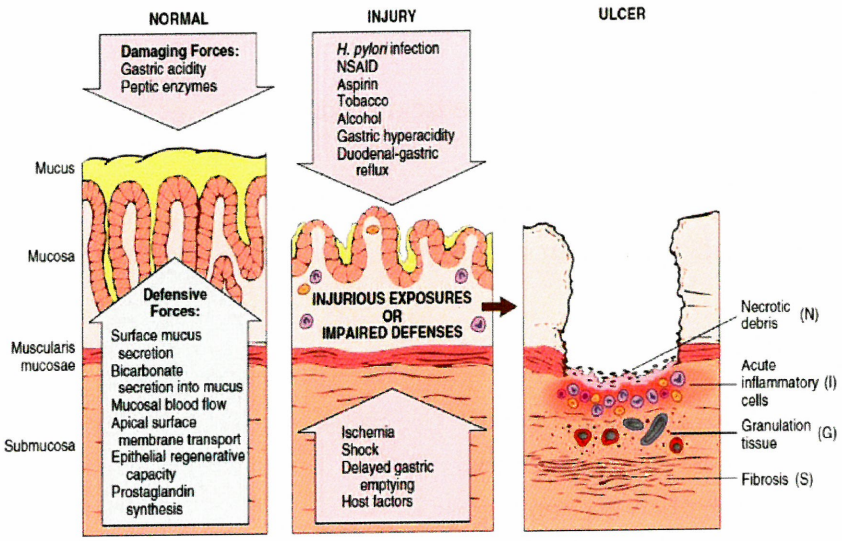
Luminal pH is 1 to 2.



Gastroduodenal mucosal defence

Layers :

1. Mucosa : Epithelial cells, coverd with mucus/gel.
    - Lamina propria.
    - Muscularis mucosa.
2. Submucosa : meissner's plexus.
3. Muscularis propria : Outer longitudinal, inner circular.
    - Between them lies the myenteric plexus.
4. Serosa.

Protective factors :

- Mucous secretion.
- Bicarbonate secretion.
- Regenerative capacity.

- 3 layers of gastroduodenal mucosal defence : Preepithelial, epithelial, subepithelial.
- Single most important factor in defence is prostaglandins.
- Pacemaker cells of GIT : Interstilial cells of Kajal.

Enteric nervous system :

- A.K.A. li4 3 =ttle brain.
- Intrinsic innervation : Auerbach plexus (motility), meissner's plexus (Intestinal secretion).
- Extrinsic innervation : ANS.

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Cooperative synergism :

- ECL → Histamine → Increases cAMP → Increase acid production.
- GRP (bombesin) → G cells → Gastrin → CCKB receptor parietal epithelial cells → Increase acid production.
- ACH → M3 (via Ca^{2+}) → Increase acid production.

Note :

CCK/PGs/Somatostatin/GIP : Decrease acid production.

BAO/MAO > 0.6 : Indicates gastrin secreting tumors like gastrinoma.

Acute gastritis

00:14:03

- Inflammation of gastric mucosa.
- M/c cause is NSAIDS > infections.
- Histology : Infiltration by neutrophils.
- No correlation between endoscopy and histology.

Acute phlegmonous gastritis :

- Dangerous acute gastritis.
- Progressive inflammation with thickening of the wall, necrosis and gas formation.
- Seen only in immunocompromised.
- Caused by streptococci > E.coli.
- Characterised by acute abdominal pain, vomiting and fever.
- Can lead to stomach wall necrosis.
- High mortality.

Chronic gastritis :

Superficial involvement with lymphocytes and plasma cells.

Can progress to atrophic gastritis → Intestinal metaplasia → Dysplasia → Carcinoma.

Types :

1. Type A gastritis :

- Autoimmune.
- HLA B8 and DR3.
- Associated with SLE, TI DM, Vitiligo.
- AMAG : Autoimmune metaplastic atrophic gastritis.
- Anti parietal cell antibodies present → Loss of acids and IF.
  - Achlorhydria, megaloblastic anemia seen.
  - No inhibitory factor for gastrin Hypergastrinemia.
  - Gasrs for gastrin secreting carcinoid tumor : mc seen malignancy.

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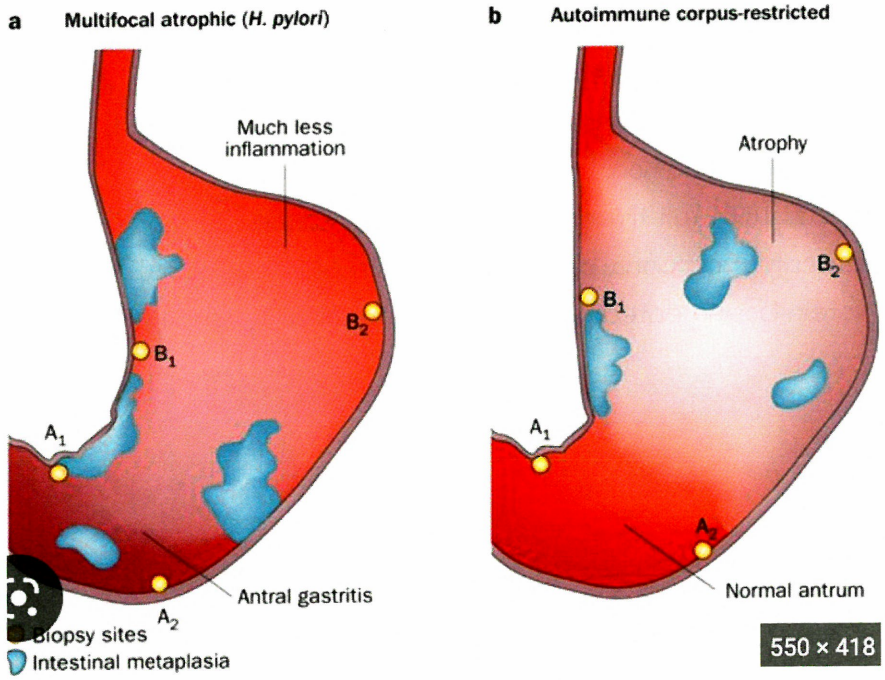
- Body is generally involved.
- Patchy atrophy and patchy metaplasia.
- Risk of Ca stomach is very less.



Features of chronic gastritis

2. Type B gastritis (EMAG) :

- m/c type.
- H.pylori related.
- Environmental metaplastic atrophic gastritis.
- Antral predominant gastritis → Pangastritis.
- Diffuse atrophy and significant metaplasia → High chance of adenocarcinoma of stomach.

Note :

- Eosinophilic gastritis presents with intestinal obstruction.
- Russel's body gastritis has pseudotumor endoscopy appearance.
- Crohns disease : Commonest cause of granulomatous gastritis.
- Lymphocytic gastritis is associated with coeliac disease.

----- Active space -----

Menetrier's disease :

- Hypertrophic gastritis.
- Foveolar hyperplasia with large tortuous mucosal folds which leaks proteins (protein losing gastropathy).
- Mediated by TGF alpha, linked with CMV infection in children.
- Body and fundus involvement.
- Hypoalbuminemia + edema + UGI symptoms.
- Premalignant condition.
- Endoscopy with full thickness biopsy is mandatory.
- Treatment : Monoclonal antibody against EGFR (cetuximab).

Note : Protein loosing enteropathy seen in intestinal lymphagiectasia.