B.PHARM SEMESTER II | BP203T

HUMAN ANATOMY, PHYSIOLOGY AND PATHOPHYSIOLOGY II

(THEORY)

UNIT II

Gastrointestinal System: Structure, Function and Disorders

Short Notes as per PCI B.Pharm Syllabus

What is covered in this unit

UNIT II TOPICS

Part A – Anatomy and functions of the upper GIT: GIT wall, mouth and salivary glands, oesophagus, stomach (acid, pepsin, regulation by the vagus), small and large intestine.

Part B – Liver, pancreas, movements, digestion and absorption: liver, gallbladder and bile, pancreas, GIT movements, digestion, absorption and GI hormones.

Part C – Pathophysiology: inflammatory bowel disease, peptic ulcer, jaundice, hepatitis, typhoid, alcoholic and non-alcoholic fatty liver disease, GERD, pancreatitis and cirrhosis.

Part D – Clinical correlation: liver function tests and common investigations.

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PART A: ANATOMY AND FUNCTIONS OF THE UPPER GIT (MOUTH TO SMALL INTESTINE)

1. Introduction to the Gastrointestinal Tract

The **gastrointestinal tract (GIT)** or **alimentary canal** is a long muscular tube, about **9 metres** long in a living adult, running from the **mouth to the anus**.

Functions of the digestive system

- **Ingestion:** taking food into the mouth.
- **Secretion:** release of water, acid, enzymes, mucus and bile into the lumen (about 7 litres per day).
- **Motility (mixing and propulsion):** movements that crush, mix and push food forward.
- **Digestion:** mechanical and chemical breakdown of food into small absorbable units.

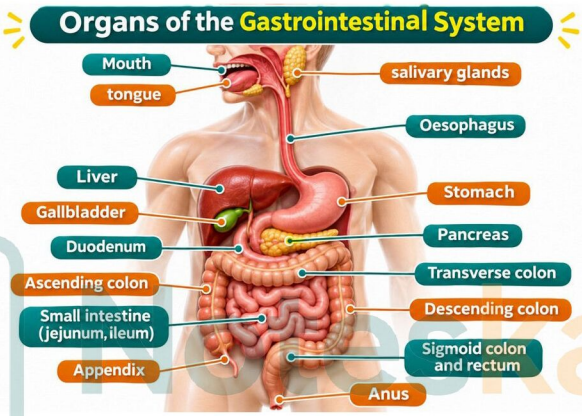

Figure 1: Organs of the gastrointestinal system

Parts of the GIT and accessory organs

Group	Organs
GIT (alimentary canal)	Mouth, pharynx, oesophagus, stomach, small intestine (duodenum, jejunum, ileum).
Accessory organs	Teeth, tongue, three pairs of salivary glands, liver, gallbladder, pancreas

Structure of the GIT wall

From the oesophagus to the anal canal, the wall has the **same four layers**, arranged from inside to outside.

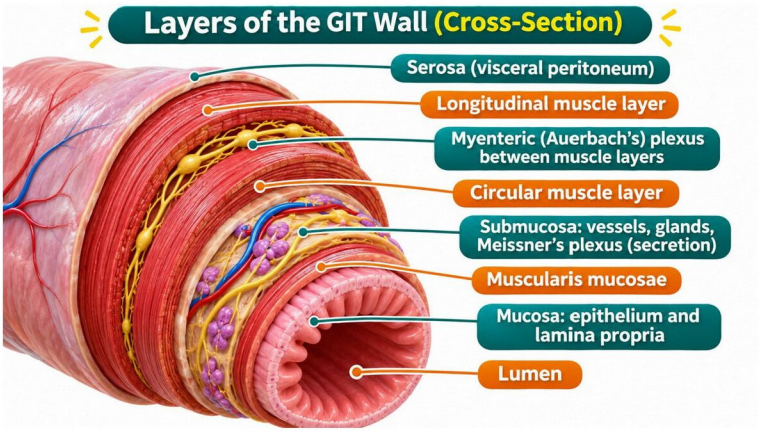

Figure 2: Layers of the GIT wall (cross-section)

2. Mouth, Salivary Glands and Swallowing

The **mouth (oral cavity)** contains the teeth, tongue and the openings of the salivary ducts.

Functions of the tongue and teeth

- **Tongue:** moves food, helps swallowing and speech, and has taste buds (sweet, sour, salty, bitter, umami).
- **Teeth:** 32 permanent teeth. Incisors cut, canines tear, premolars and molars grind food.

Salivary glands

Gland	Position	Duct opens	Type of secretion
Parotid (largest, about 25% of saliva)	In front of and below the ear	Opposite the upper second molar (Stensen's duct)	Serous (watery, rich in amylase)
Submandibular (about 70% of saliva)	Below the jaw	Beside the frenulum of the tongue (Wharton's duct)	Mixed (serous and mucous)
Sublingual (about 5%)	Under the tongue	Floor of the mouth (ducts of Rivinus)	Mainly mucous

Saliva

About **1 to 1.5 litres** of saliva are secreted daily.

Swallowing (deglutition)

Stage	Type	What happens
1. Oral stage	Voluntary	Tongue pushes the bolus back into the pharynx
2. Pharyngeal stage	Involuntary (reflex, centre in medulla)	Soft palate rises (seals the nose).
3. Oesophageal stage	Involuntary	Primary peristalsis (and secondary peristalsis if food is stuck) carries the bolus to the stomach in 5 to 9 seconds; the lower oesophageal sphincter (LES) relaxes to let food in.

3. Oesophagus and Stomach: Anatomy

Oesophagus

The oesophagus is a muscular tube about **25 cm** long that joins the pharynx to the stomach.

Stomach

The stomach is a J-shaped, expandable bag on the left side of the upper abdomen, under the diaphragm.

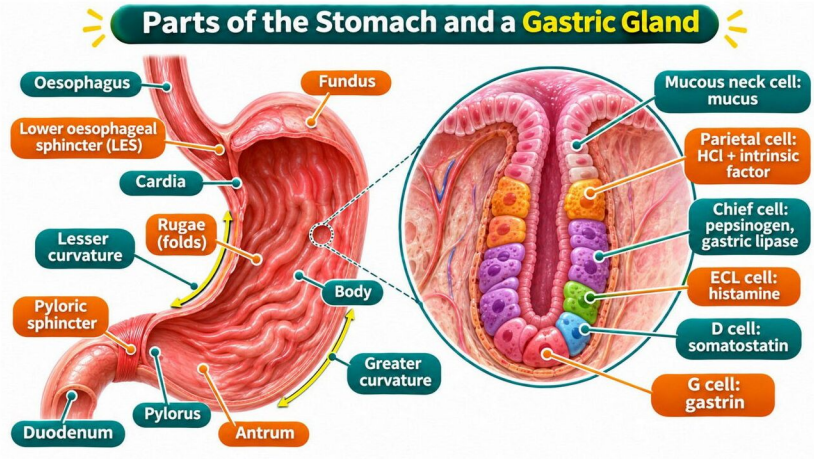

Figure 3: Parts of the stomach and a gastric gland

Cells of the gastric mucosa

Cell	Site	Secretion	Role
Mucous (surface and neck) cells	Surface and neck of glands	Mucus and bicarbonate	Protective alkaline layer on the mucosa
Parietal (oxyntic) cells	Body and fundus	**HCl** and **intrinsic factor**	Acid for digestion; intrinsic factor for vitamin B12 absorption
Chief (peptic) cells	Base of glands	**Pepsinogen** and gastric lipase	Protein and fat digestion
G cells	Antrum	**Gastrin**	Stimulates acid and motility

4. Gastric Secretion: Acid, Pepsin and Their Regulation

The stomach secretes about **2 to 3 litres of gastric juice** per day.

Mechanism of HCl production (parietal cell)

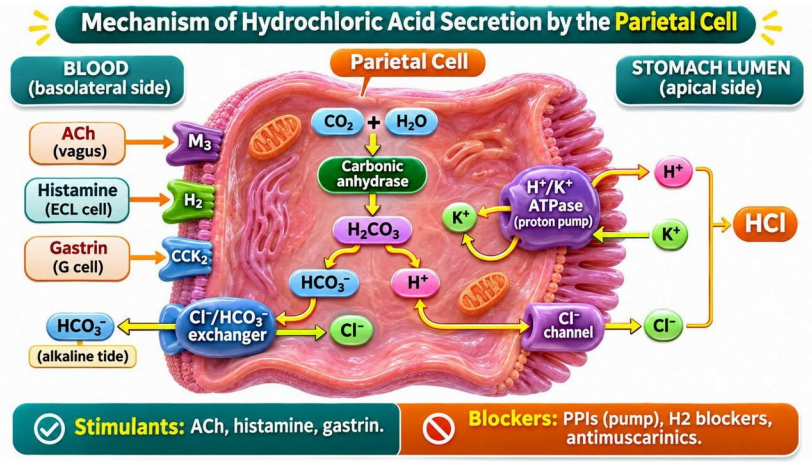

Figure 4: Mechanism of hydrochloric acid secretion by the parietal cell

- Inside the parietal cell, **carbonic anhydrase** combines CO_2 and H_2O to form H_2CO_3.
- **H^+ is pumped into the lumen** by the **H^+/K^+ ATPase (proton pump)** in exchange for K^+.
- **HCO_3^- leaves into the blood** in exchange for Cl^- (Cl^-/HCO_3^- exchanger).
- **Cl^- passes into the lumen** through channels and combines with H^+ to form **HCl**.

Functions of HCl

- Converts **inactive pepsinogen to active pepsin** and gives the acidic pH (1.5 to 3.5) that pepsin needs.
- Kills most swallowed bacteria; denatures proteins.
- Helps absorption of iron and calcium (keeps them soluble).
- Stimulates secretin release in the duodenum.

Role of pepsin in protein digestion

- **Pepsinogen** (inactive) is released by chief cells.
- **Pepsin** is an **endopeptidase**: it breaks the **internal peptide bonds** next to aromatic amino acids (phenylalanine, tyrosine, tryptophan) and leucine.
- It works best at **pH 1.5 to 2.5** and is **inactivated at pH above 5** (in the duodenum).
- Protein is changed to **large polypeptides, proteoses and peptones** (about 10 to 20% of total protein digestion).

Regulation of gastric acid secretion

Acid secretion is controlled in **three phases**.

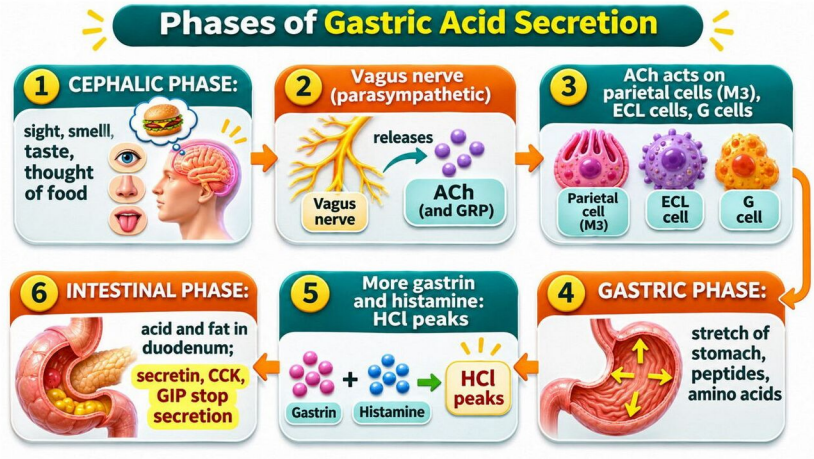

Figure 5: Phases of gastric acid secretion

Protection of the stomach from its own acid (mucosal barrier)

- A thick layer of **mucus with bicarbonate** keeps the pH at the cell surface near 7.
- **Tight junctions** between epithelial cells stop H^+ from leaking back.
- **Prostaglandins (PGE2)** increase mucus, bicarbonate and blood flow, and reduce acid.
- Rapid renewal of epithelial cells (every 3 to 5 days) and a rich blood supply.

5. Small Intestine

The small intestine is the **main site of digestion and absorption**.

Parts of the small intestine

Part	Length	Features

Adaptations for absorption (huge surface area, about 200 m^2)

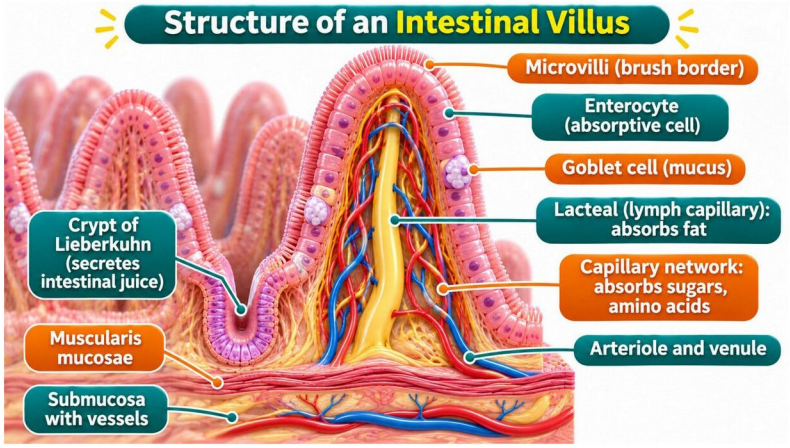

Figure 6: Structure of an intestinal villus

- **Circular folds (plicae circulares)**, **villi** (0.5 to 1 mm finger-like projections) and **microvilli (brush border)** increase the surface area about 600 times.
- Each villus has a **capillary network** (carries sugars, amino acids.
- **Crypts of Lieberkühn** at the base secrete intestinal juice and contain stem cells.

Intestinal juice (succus entericus)

About **1 to 2 litres per day**, pH 7.5 to 8.

Functions of the small intestine

- Completes digestion of carbohydrates, proteins and fats.
- Absorbs about **90% of nutrients and most of the water**.
- Secretes hormones (secretin, CCK, GIP, motilin) that control digestion.
- Immune defence (Peyer's patches, IgA) and mixing and propulsion of chyme.

6. Large Intestine

The large intestine is about **1.5 metres** long and 6 cm wide.

Parts

Part	Features
Caecum	Blind pouch in the right lower abdomen.
Colon	Ascending, transverse, descending and sigmoid parts.
Rectum	About 12 cm; stores faeces; stretch triggers the urge to defecate.
Anal canal	About 4 cm; **internal anal sphincter** (smooth muscle.

Functions

- **Absorption** of water (about 1.3 litres per day) and electrolytes (Na^+, Cl^-).
- **Gut bacteria (microbiota)** ferment undigested fibre to short-chain fatty acids (fuel for colon cells) and make **vitamin K and some B vitamins** (biotin, B12, folate).
- **Mucus secretion** to lubricate faeces.
- **Storage and defecation.**

PART B: LIVER, PANCREAS, MOVEMENTS, DIGESTION AND ABSORPTION

7. Liver

The liver is the **largest internal organ** (about **1.5 kg**), lying in the right upper abdomen under the diaphragm.

Structure: the liver lobule

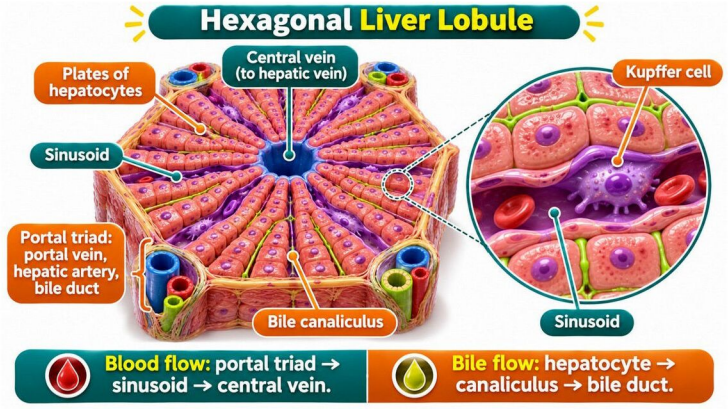

Figure 7: Hexagonal liver lobule

- The functional unit is the **hepatic lobule**, a hexagon with a **central vein** in the middle and a **portal triad** (portal vein branch, hepatic artery branch, bile duct) at each corner.
- **Hepatocytes** are arranged in plates.
- **Bile canaliculi** between hepatocytes collect bile and drain towards the bile duct (opposite to blood flow).
- **Stellate (Ito) cells** store vitamin A; they become collagen-making cells in fibrosis and cirrhosis.

Functions of the liver

Function	Details
Carbohydrate metabolism	Glycogenesis, glycogenolysis, gluconeogenesis; keeps blood glucose steady
Protein metabolism	Makes **albumin**, clotting factors (I, II, V, VII, IX, X).
Fat metabolism	Beta-oxidation, makes **cholesterol**, lipoproteins and bile acids; makes ketone bodies
Bile secretion	About 500 to 1000 mL per day; helps fat digestion and absorption.

8. Gallbladder and Bile

The **gallbladder** is a pear-shaped sac (7 to 10 cm, holds about **30 to 50 mL**) under the liver.

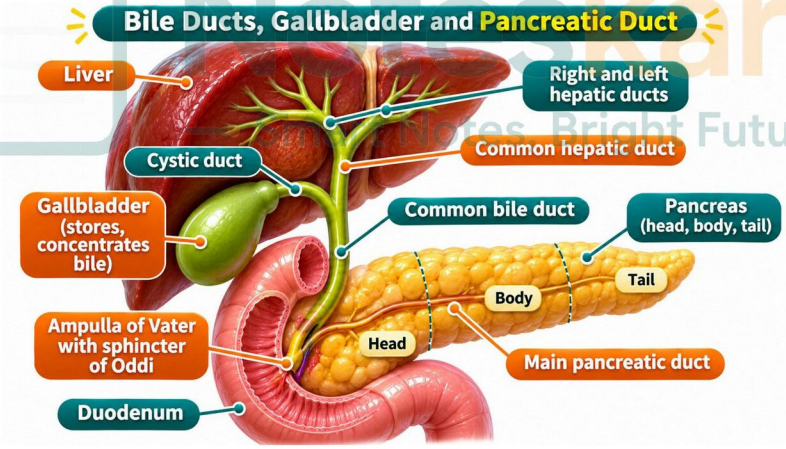

Figure 8: Bile ducts, gallbladder and pancreatic duct

Composition and functions of bile

Component	Role
Bile salts (sodium glycocholate, taurocholate)	**Emulsify fats**, form micelles for absorption of fats and fat-soluble vitamins (A, D, E, K); about 95% are reabsorbed in the ileum (**enterohepatic circulation**).
Bile pigments (bilirubin, biliverdin)	Waste product of haem; give colour to bile and faeces
Cholesterol and lecithin	Excretion of cholesterol; stabilise micelles
Bicarbonate	Neutralises acid chyme

Control of bile release

- **CCK (cholecystokinin)** from the duodenum (stimulated by fat and amino acids): contracts the gallbladder and relaxes the sphincter of Oddi.
- **Vagus nerve** (weak stimulation); **secretin** increases the watery, bicarbonate-rich part of bile.
- **Bile salts** returning to the liver stimulate more bile secretion (choleretic effect).

9. Pancreas

The pancreas is a soft, elongated gland (12 to 15 cm) behind the stomach with a **head** (in the C-loop of the duodenum), **body** and **tail**.

Part	Cells	Secretion

Pancreatic juice

Enzyme	Form released	Action
Trypsin	Trypsinogen (activated by enterokinase)	Breaks proteins at lysine and arginine; activates all other zymogens
Chymotrypsin	Chymotrypsinogen	Breaks proteins at aromatic amino acids
Carboxypeptidase	Procarboxypeptidase	Removes amino acids from the carboxyl end of peptides
Elastase	Proelastase	Digests elastin

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10. Movements of the Gastrointestinal Tract

Gut movements are carried out by smooth muscle.

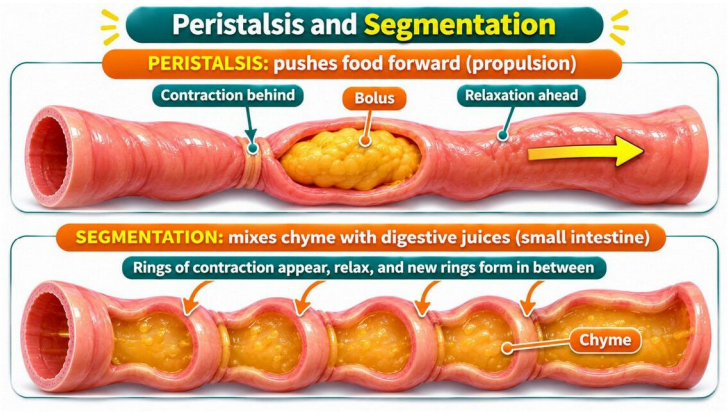

Figure 9: Peristalsis and segmentation

Region	Movement	Description and purpose
Mouth and pharynx	Mastication, swallowing	Chewing is voluntary and reflex; swallowing in three stages (oral, pharyngeal, oesophageal).
Oesophagus	**Peristalsis** (primary and secondary)	Wave of contraction behind the bolus and relaxation ahead of it; the LES relaxes for entry.

Region	Movement	Description and purpose
Stomach	Receptive relaxation, **mixing waves**, **gastric emptying**, hunger contractions	Fundus relaxes to accept food (vagovagal reflex). Mixing waves every 20 seconds churn food into chyme. The antrum pushes small amounts through the pylorus (retropulsion mixes further)
Small intestine	**Segmentation**, **peristalsis**, **migrating motor complex (MMC)**, pendular movements	Segmentation mixes chyme and exposes it to the wall.

Control of gastric emptying

- **Stomach factors (speed up):** volume of food, gastrin.
- **Duodenal factors (slow down):** acid, fat, hypertonic or high-protein chyme, stretch.
- **Order of speed:** carbohydrate empties fastest, then protein, then **fat slowest**.

11. Digestion of Nutrients

Digestion is the breakdown of large food molecules into small molecules that can be absorbed.

Summary of digestion at each site

Site	Enzyme / juice	Substrate	Product
Mouth	Salivary amylase	Starch	Maltose, dextrins
	Lingual lipase	Fat	Starts digestion
Stomach	HCl + **pepsin**	Proteins	Polypeptides, peptones
	Gastric lipase	Fat (short chain)	Fatty acids

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Carbohydrate digestion

Starch → (salivary and pancreatic amylase) → **maltose, maltotriose, limit dextrins** → (brush-border maltase, isomaltase) → **glucose**.

Protein digestion

Proteins → (pepsin) → **polypeptides** → (trypsin, chymotrypsin, elastase) → **oligopeptides** → (carboxypeptidase, aminopeptidase, dipeptidase) → **amino acids** (and a few di- and tripeptides absorbed whole).

Fat digestion

- **Emulsification:** bile salts break big fat droplets into tiny droplets (large surface area).
- **Lipolysis:** pancreatic lipase (with **colipase**) splits triglyceride at positions 1 and 3 into **2 free fatty acids and a 2-monoglyceride**.
- **Micelle formation:** bile salts surround the products, forming **mixed micelles** that carry them to the brush border for absorption.

12. Absorption of Nutrients

Absorption is the passage of digested products, water, vitamins and minerals from the lumen into blood or lymph.

Mechanisms of absorption

Mechanism	Features	Example
Simple diffusion	Down a concentration gradient; no carrier, no energy	Water (by osmosis), lipids, fatty acids, fat-soluble vitamins
Facilitated diffusion	Carrier protein, no energy	Fructose (GLUT5)
Primary active transport	Needs ATP, against gradient	Na^+/K^+ ATPase (drives all the others)
Secondary active transport (co-transport)	Uses the Na^+ gradient made by the pump	Glucose and galactose (SGLT1), amino acids

Absorption of each nutrient

Nutrient	Site	How it is absorbed	Route
Glucose and galactose	Duodenum and jejunum	**SGLT1** (Na^+ co-transport) then GLUT2 on the blood side	Capillaries → portal vein → liver
Fructose	Jejunum	Facilitated diffusion (GLUT5, GLUT2)	Capillaries → portal vein
Amino acids	Duodenum and jejunum	Na^+-dependent secondary active transport; di- and tripeptides by H^+-dependent PepT1	Capillaries → portal vein
Fats (monoglycerides, fatty acids)	Jejunum	Micelles carry them to the cell; they **diffuse in**, are re-made into triglycerides in the ER and packed with cholesterol and protein as **chylomicrons**	**Lacteals** → lymph → thoracic duct → blood (short-chain fatty acids go directly to the portal blood)

13. Gastrointestinal Hormones

Hormone	Source	Stimulus	Main actions
Gastrin	G cells (antrum)	Peptides, amino acids, stomach stretch, vagus	Increases HCl secretion and gastric motility; growth of mucosa
Secretin	S cells (duodenum)	Acid in the duodenum	Pancreatic **bicarbonate** and bile bicarbonate; inhibits gastric acid
Cholecystokinin (CCK)	I cells (duodenum, jejunum)	Fat and amino acids	Gallbladder contraction, **enzyme-rich pancreatic juice**, slows gastric emptying, satiety
Gastric inhibitory peptide (GIP, glucose-dependent insulinotropic peptide)	K cells (duodenum)	Glucose and fat	Releases **insulin**; inhibits gastric acid and motility

PART C: PATHOPHYSIOLOGY OF GASTROINTESTINAL AND LIVER DISORDERS

Pathophysiology explains how normal function is changed in disease.

14. Inflammatory Bowel Disease (IBD)

Definition

Inflammatory bowel disease is a group of **chronic, relapsing inflammatory disorders of the gut** with periods of flare-up and remission.

Causes and risk factors

- **Genetic:** family history; **NOD2/CARD15** gene (Crohn's), IL-23R and many others.
- **Immune:** abnormal, over-active mucosal immune response to normal gut bacteria (not an infection).
- **Environment:** smoking (worsens Crohn's, but **protects against UC**), Western diet, antibiotics in childhood, NSAIDs, stress, urban life.
- **Dysbiosis:** change in the gut microbiota, and a weak mucosal barrier.

Pathophysiology

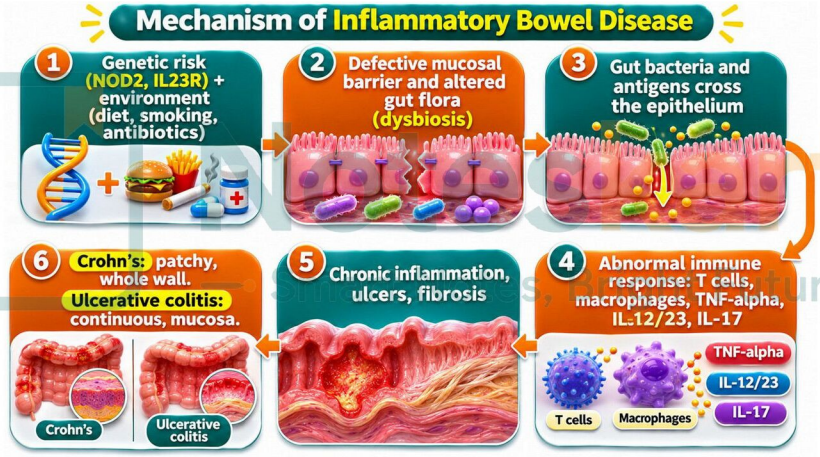

Figure 10: Mechanism of inflammatory bowel disease

- A leaky epithelial barrier lets bacteria and antigens enter the lamina propria.
- **Crohn's disease: Th1 and Th17** response (IL-12, IL-23, IFN-γ.
- **Ulcerative colitis: Th2-like** response (IL-5, IL-13) and TNF-α. Inflammation is limited to the **mucosa and submucosa**, with crypt abscesses and ulcers.
- Repeated injury and repair cause fibrosis, narrowing (stricture), loss of haustra (UC) and an increased risk of **colon cancer**.

Diagnosis and basis of treatment

- **Diagnosis:** colonoscopy with biopsy, **faecal calprotectin** (marks gut inflammation), CRP, ESR, blood count.
- **Aminosalicylates (5-ASA: mesalazine, sulfasalazine):** reduce mucosal inflammation, mainly in UC.
- **Corticosteroids (prednisolone, budesonide):** for flare-ups (not for long-term use).
- **Immunosuppressants:** azathioprine, 6-mercaptopurine, methotrexate (maintenance).

15. Peptic Ulcer Disease

Definition

A **peptic ulcer** is a **break in the mucosa** of the stomach or duodenum.

Causes

- **Helicobacter pylori infection:** present in about **95% of duodenal** and 70% of gastric ulcers.
- **NSAIDs and aspirin:** the next commonest cause.
- Others: smoking, alcohol, corticosteroids with NSAIDs, **Zollinger-Ellison syndrome** (gastrinoma), stress (critical illness), Crohn's disease.

Pathophysiology: imbalance of aggressive and defensive factors

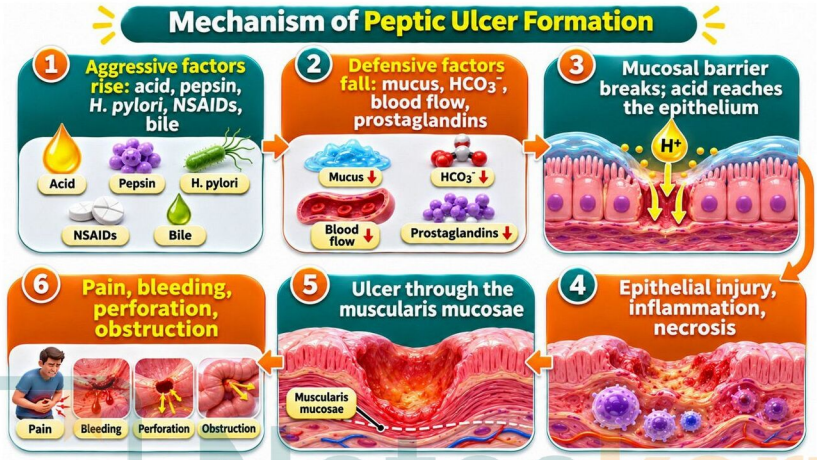

Figure 11: Mechanism of peptic ulcer formation

Aggressive factors (damage)	Defensive factors (protect)
HCl and pepsin	**Mucus layer** and bicarbonate
H. pylori and its toxins	**Prostaglandins (PGE2)**
NSAIDs (block prostaglandins)	Good **mucosal blood flow**
Bile reflux, alcohol, smoking	Epithelial renewal and tight junctions

Clinical features and complications

- **Symptoms:** burning epigastric pain, bloating, nausea, early satiety, belching.
- **Haemorrhage:** haematemesis (vomiting blood) and melaena (black tarry stool).
- **Perforation:** sudden severe pain, board-like abdomen, air under the diaphragm (anterior ulcers).
- **Gastric outlet obstruction** from scarring (vomiting of undigested food).

Diagnosis and basis of treatment

- **Diagnosis: upper GI endoscopy with biopsy**; **H. pylori tests** (urea breath test, stool antigen.
- **Stop NSAIDs**, smoking and alcohol.
- **Acid suppression: PPIs** (omeprazole), H2 blockers (ranitidine, famotidine), antacids.
- **H. pylori eradication (triple therapy for 14 days):** PPI + **clarithromycin** + **amoxicillin** (or metronidazole).

16. Jaundice

Definition

Jaundice (icterus) is a **yellow discolouration of the skin, sclera (white of the eye) and mucous membranes** caused by an excess of **bilirubin** in the blood (**hyperbilirubinaemia**).

Normal bilirubin metabolism

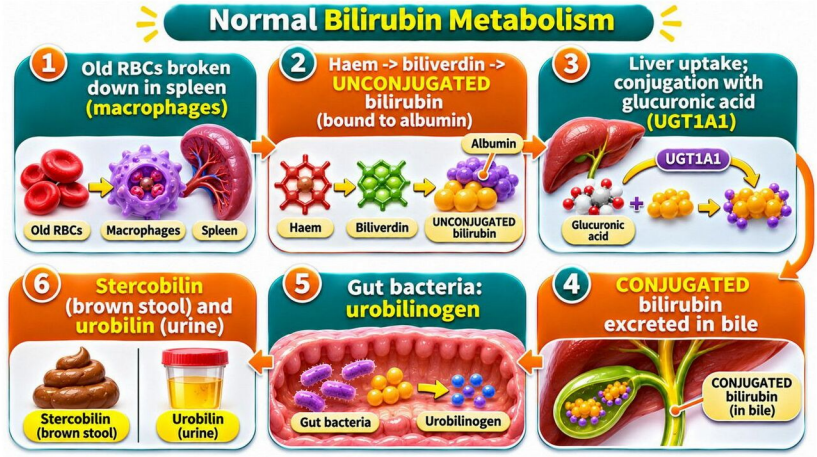

Figure 12: Normal bilirubin metabolism

Type	Properties
Unconjugated (indirect) bilirubin	Fat-soluble, **bound to albumin**, cannot be excreted in urine.
Conjugated (direct) bilirubin	Water-soluble (bound to glucuronic acid by UGT1A1 in the liver).

Types of jaundice and their pathophysiology

Type	Defect	Causes	Bilirubin
Pre-hepatic (haemolytic)	Too much red cell breakdown; the liver cannot clear all of the bilirubin.	Haemolytic anaemia, sickle cell, malaria, thalassaemia, G6PD deficiency, incompatible transfusion, neonatal haemolysis (Rh).	**Unconjugated** raised
Hepatic (hepatocellular)	Liver cells cannot take up, conjugate or excrete bilirubin	Viral hepatitis, alcohol, drugs (paracetamol), cirrhosis, Gilbert's, Crigler-Najjar, neonatal (physiological) jaundice	**Both** raised
Post-hepatic (obstructive, cholestatic)	Bile cannot reach the gut	**Gallstone** in common bile duct, carcinoma of head of pancreas, cholangiocarcinoma, biliary atresia, primary biliary cholangitis, strictures	**Conjugated** raised

Neonatal (physiological) jaundice

Seen in 60% of newborns on days 2 to 5.

17. Hepatitis

Definition

Hepatitis is **inflammation of the liver**.

Pathophysiology of viral hepatitis

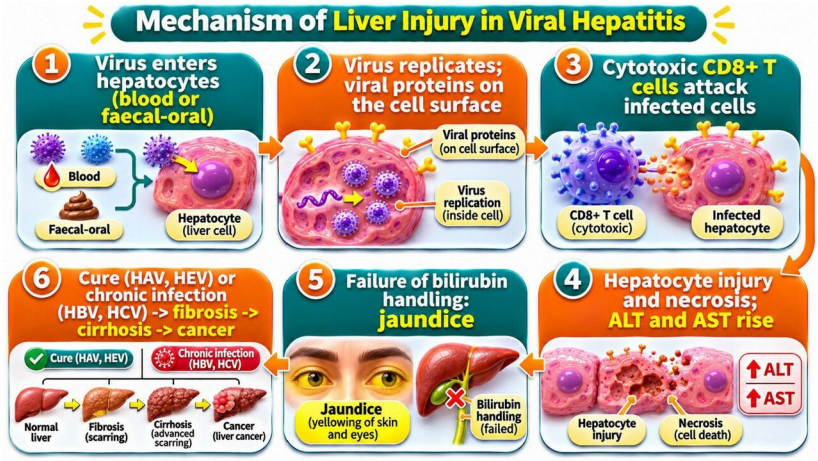

Figure 13: Mechanism of liver injury in viral hepatitis

- Most hepatitis viruses are **not directly toxic**.
- Release of **ALT and AST** from the dead cells raises their blood levels.
- If the immune system fails to clear the virus (HBV, HCV), **chronic inflammation** continues.

Clinical stages and features

- **Prodromal (pre-icteric) phase:** fever, malaise, loss of appetite, nausea, vomiting, joint pain, dislike of smoking.
- **Icteric phase:** jaundice, dark urine, pale stool, itching, tender enlarged liver; the fever subsides.
- **Recovery** over weeks, or **fulminant hepatic failure** (rare) with confusion (encephalopathy), bleeding and coma.
- **Chronic hepatitis:** may have no symptoms for years until cirrhosis (ascites, varices, encephalopathy) develops.

Diagnosis and basis of treatment

- **Liver function tests:** very high **ALT and AST** (often over 1000 IU/L in acute viral hepatitis), raised bilirubin, prolonged prothrombin time in severe disease.
- **Serology:** anti-HAV IgM, HBsAg and anti-HBc IgM, anti-HCV (then HCV RNA), anti-HEV IgM.
- **HAV and HEV:** supportive treatment (rest, fluids, nutrition, avoid alcohol and hepatotoxic drugs).
- **HBV: tenofovir or entecavir** (nucleoside analogues) and peginterferon; **vaccination** prevents infection.

18. Typhoid (Enteric Fever)

Definition

Typhoid fever is a systemic infection caused by **Salmonella enterica serotype Typhi** (Gram-negative, motile, non-lactose-fermenting rod).

Spread

Faecal-oral route: contaminated water and food (the **'5 Fs': Flies, Fingers, Faeces, Food, Fomites**), poor sanitation.

Pathophysiology

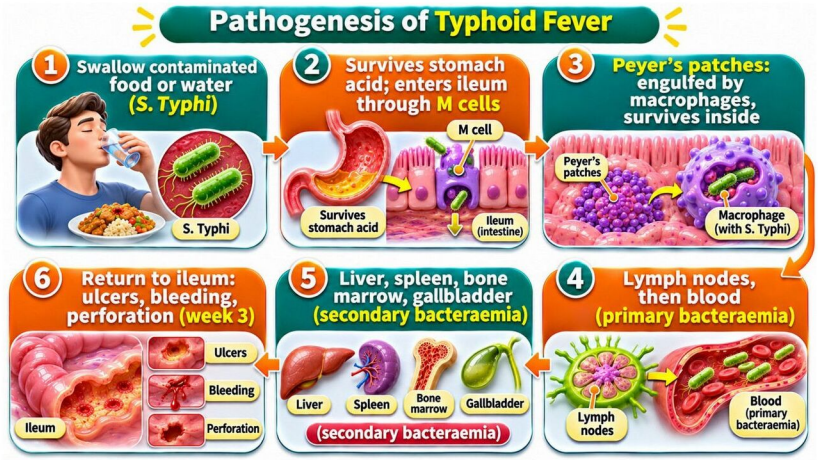

Figure 14: Pathogenesis of typhoid fever

- Bacteria survive stomach acid (more likely if the person is on antacids or PPIs.
- They are engulfed by **macrophages** but **survive and multiply inside them** (the **Vi capsular antigen** helps them to resist killing).
- They spread through lymph to the mesenteric nodes and then to the blood (**bacteraemia**).
- From the gallbladder, bacteria re-infect the intestine with bile.

Diagnosis and basis of treatment

- **Blood culture** (best in week 1; positive in 60 to 80%).
- **Widal test** (O and H antibodies; a rising titre; low specificity).
- **Antibiotics: azithromycin**, **ceftriaxone** (third-generation cephalosporin), cefixime; **ciprofloxacin** (resistance is now common); **chloramphenicol** historically.
- **Supportive:** fluids, antipyretics, soft food; **steroids (dexamethasone)** in severe typhoid with shock or delirium; surgery for perforation.

19. Alcoholic and Non-alcoholic Fatty Liver Disease

Fatty liver (hepatic steatosis) means that **more than 5% of the liver weight is fat (triglyceride)**.

Alcoholic liver disease (ALD)

Pathophysiology

- **Alcohol is mainly broken down in the liver** by **alcohol dehydrogenase (ADH)** to **acetaldehyde** (toxic), and then by aldehyde dehydrogenase (ALDH) to acetate.
- **High NADH/NAD⁺ ratio** stops fatty acid oxidation and gluconeogenesis.
- **Acetaldehyde** damages proteins and cell membranes and forms adducts; **CYP2E1** is induced and makes **free radicals (oxidative stress)**.
- Alcohol raises gut permeability, so **endotoxin (LPS)** reaches the liver.

Non-alcoholic fatty liver disease (NAFLD / MASLD)

Fat builds up in the liver in people who drink **little or no alcohol**.

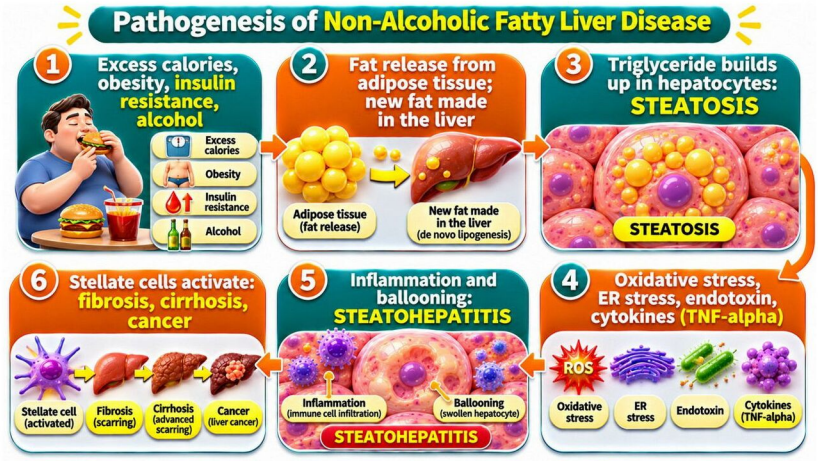

Figure 15: Pathogenesis of non-alcoholic fatty liver disease

Clinical features, complications and diagnosis

- Early disease usually has **no symptoms**; the liver is enlarged; fatigue and right upper abdominal discomfort.
- **Complications of cirrhosis:** ascites, oesophageal varices (bleeding), hepatic encephalopathy, hepatorenal syndrome, spontaneous bacterial peritonitis, hepatocellular carcinoma.
- **Diagnosis:** liver function tests, **ultrasound** (bright liver), **FibroScan** (elastography), FIB-4 score, **liver biopsy** (gold standard).

20. Gastro-oesophageal Reflux Disease (GERD)

GERD is the backward flow (reflux) of **stomach acid and contents into the oesophagus**, causing symptoms or damage (**reflux oesophagitis**).

Causes and pathophysiology

- **Weak or relaxed lower oesophageal sphincter (LES):** transient LES relaxations are the main mechanism.
- **Hiatus hernia:** part of the stomach slides above the diaphragm and weakens the barrier.
- **Delayed gastric emptying** and high stomach pressure (obesity, pregnancy).
- The oesophageal lining has **no mucus layer**, so acid and pepsin cause burning, inflammation and ulcers.

Features and treatment

- **Symptoms: heartburn** (burning behind the breastbone, worse on lying down or bending), regurgitation.
- **Complications:** oesophagitis, stricture (dysphagia), bleeding, Barrett's oesophagus, aspiration.
- **Lifestyle:** weight loss, small meals, no food 3 hours before bed.
- **Drugs: antacids and alginates**, H2 blockers, **PPIs (most effective)**, prokinetics (domperidone, metoclopramide).

21. Acute Pancreatitis

Acute pancreatitis is sudden **inflammation of the pancreas** caused by **premature activation of digestive enzymes inside the gland**, so the pancreas digests itself (**autodigestion**).

Causes (mnemonic I GET SMASHED)

- **Gallstones** (commonest) and **alcohol** (second commonest): together about 80% of cases.
- Hypertriglyceridaemia, hypercalcaemia, **drugs** (azathioprine, valproate, thiazides), ERCP, trauma, infection (mumps), scorpion sting, autoimmune disease.

Pathophysiology

- A stone blocking the ampulla, or alcohol, causes **ductal obstruction and acinar cell injury**.
- Trypsin activates other zymogens (elastase, phospholipase A2, lipase).
- Cytokines (TNF-α, IL-1, IL-6) cause a **systemic inflammatory response**, shock.

Features, diagnosis and treatment

- **Features:** severe **epigastric pain going through to the back**, relieved by leaning forward, nausea, vomiting, fever.
- **Diagnosis:** serum **amylase** (more than 3 times normal) and **lipase** (more sensitive and specific).
- **Treatment:** IV fluids, pain relief, early oral or tube feeding, oxygen.
- **Chronic pancreatitis** (usually alcohol) leads to permanent damage, **steatorrhoea** (lipase loss) and **diabetes** (islet loss).

22. Cirrhosis and Portal Hypertension

Cirrhosis is the **irreversible end-stage of chronic liver injury**: normal liver structure is replaced by **fibrous scar tissue and regenerating nodules**.

Pathophysiology

- Repeated injury activates **stellate cells**, which lay down **collagen**.
- Resistance to blood flow rises, causing **portal hypertension** (portal pressure above 10 mmHg).
- Loss of functioning hepatocytes causes **low albumin, poor clotting factors.**

Complications

Complication	Mechanism	Feature or treatment
Ascites	Portal hypertension, **low albumin**, and secondary hyperaldosteronism cause fluid leakage	Distended abdomen; salt restriction, **spironolactone and furosemide**, paracentesis
Oesophageal varices	Portosystemic collaterals form in the oesophagus and stomach	**Massive haematemesis**; propranolol (prevention), band ligation, octreotide, terlipressin
Hepatic encephalopathy	Ammonia and other toxins bypass the liver to the brain	Confusion, asterixis, coma; **lactulose**, rifaximin
Splenomegaly and hypersplenism	Congestion of the spleen	Low platelets and white cells

PART D: CLINICAL CORRELATION AND REFERENCE TABLES

23. Liver Function Tests (LFTs)

Test	Normal (approx.)	What it shows
ALT (SGPT)	7 to 56 IU/L	**Most specific for hepatocyte injury**; very high in viral and toxic hepatitis.
AST (SGOT)	10 to 40 IU/L	Hepatocyte injury (also muscle, heart); **AST:ALT above 2 in alcoholic hepatitis.**
ALP (alkaline phosphatase)	44 to 147 IU/L	**Cholestasis** and bile duct obstruction (also bone disease)
GGT	9 to 48 IU/L	Cholestasis; **raised by alcohol and enzyme-inducing drugs**

24. Common GI Symptoms and Investigations

Common investigations

Investigation	Use
Upper GI endoscopy (OGD)	Oesophagus, stomach, duodenum: ulcers, varices, biopsy for H. pylori
Colonoscopy	Colon and terminal ileum: IBD, polyps, cancer
Ultrasound abdomen	Liver (fatty liver), gallstones, biliary dilatation
ERCP / MRCP	Bile and pancreatic ducts: stones, strictures

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