BP203T · B.PHARM THEORY

BP203T Human Anatomy, Physiology and Pathophysiology II

Unit II — Gastrointestinal System

Pills & Python Notes

From-zero teaching standard: every technical term is defined before use.

Syllabus coverage: Unit II (12 hours).

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CHAPTER 7: ANATOMY OF THE GI TRACT AND THE STOMACH

Unit II · ~3 hours

7.1 Introduction

Every oral medicine you will ever dispense travels through the gastrointestinal tract — and the GI tract decides how much of the drug is absorbed, how fast, and whether food changes the effect. A pharmacist who understands the stomach's acid, the intestine's absorptive surface, and gut movements can counsel correctly on "take before food / after food / with water". This chapter covers the GI tract's plan and the stomach in detail, exactly as the syllabus requires: acid production, its parasympathetic regulation, and pepsin's role in protein digestion.

Learning objectives — after studying this chapter, you should be able to:

- list the organs of the GI tract in order and state the four layers of the GI wall;
- describe the anatomy of the stomach (regions, curvatures, sphincters);
- name the four cell types of gastric glands and their secretions;
- explain the mechanism of HCl production by parietal cells;
- describe the three phases of gastric regulation (cephalic, gastric, intestinal) including parasympathetic control;
- explain how pepsinogen is activated and pepsin's role in protein digestion;
- link stomach physiology to pharmacy (antacids, PPI action sites — in general terms).

7.2 Definitions

Scientific definitions:

- **Gastrointestinal (GI) tract:** the continuous muscular tube from mouth to anus (mouth, pharynx, oesophagus, stomach, small intestine, large intestine) that digests food and absorbs nutrients.
- **Digestion:** the mechanical and chemical breakdown of food into molecules small enough for absorption.
- **Absorption:** the movement of digested nutrients from the gut lumen into blood or lymph.
- **Peristalsis:** wave-like muscular contractions that push contents forward along the GI tract.
- **Sphincter:** a ring of muscle guarding the junction between two GI regions, controlling one-way flow.

Simple explanation: The GI tract is a **9-metre food-processing factory** (in adults, roughly). Food enters at the mouth (receiving dock), is churned and chemically treated in the stomach (mixing tank with acid), nutrients are absorbed in the small intestine (warehouse dispatch), water is reclaimed in the large intestine (recycling unit), and waste exits at the anus (dispatch gate).

7.3 Key Terminology

Term	Meaning	Simple explanation
Lumen	The hollow space inside the GI tube	"The tunnel food travels through"
Mucosa	Innermost lining layer	"Wallpaper" — secretes and absorbs
Submucosa	Connective tissue layer with vessels/nerves	"Wiring and plumbing layer"
Muscularis externa	Two muscle layers (circular + longitudinal)	"The pump" — moves food
Serosa	Outer protective covering	"Outer wrapping"
Chyme	Semi-liquid partially digested food leaving the stomach	"Food soup"
Parietal cell	Gastric gland cell secreting HCl and intrinsic factor	"Acid factory"
Chief cell	Gastric gland cell secreting pepsinogen	"Enzyme store"
G cell	Pyloric cell secreting gastrin (hormone)	"Acid-demand signal"
Pepsin	Active protein-digesting enzyme of the stomach	"Protein scissors"

7.4 Detailed Explanation

7.4.1 The GI tract in order + wall layers

Organs in order: mouth → pharynx → **oesophagus** → **stomach** → **small intestine** (duodenum → jejunum → ileum) → **large intestine** (caecum → colon → rectum) → anal canal → anus. Accessory organs (not in the tube but pouring juices in): **salivary glands, liver, gallbladder, pancreas**. **Four wall layers (inside → out): mucosa** (epithelium + glands — secretion/absorption) → **submucosa** (vessels, nerves) → **muscularis externa** (inner circular + outer longitudinal smooth muscle — motility) → **serosa** (outer peritoneal cover).

Memory hook: Wall layers inside→out: "**M**ucosa, **S**ubmucosa, **M**uscularis, **S**erosa" — "**M**y **S**tomach **M**oves **S**moothly".

7.4.2 The stomach — anatomy you can sketch

- **Location/shape:** J-shaped muscular sac in the upper-left abdomen, below the diaphragm; about 25 cm long, holding ~1–1.5 litres.
- **Regions: fundus** (dome above the oesophageal opening), **body** (large middle part), **pylorus** (narrow lower part ending at the **pyloric sphincter**, which meters chyme into the duodenum).
- **Curvatures: lesser curvature** (short inner curve, right side) and **greater curvature** (long outer curve, left side).
- **Openings: cardiac (lower oesophageal) sphincter** at the top (prevents reflux of acid into the oesophagus) and **pyloric sphincter** at the bottom.
- **Folds:** inner lining thrown into **rugae** (folds that flatten as the stomach fills); surface dotted with **gastric pits** leading to **gastric glands**.

How to sketch: Draw a J-shape → label fundus (top dome), body (middle), pylorus (narrow bottom) → mark cardiac sphincter (top) and pyloric sphincter (bottom) → label lesser (inner) and greater (outer) curvatures → show rugae as wavy lines inside.

7.4.3 Gastric glands — four cells, four jobs

Cell	Location	Secretes	Job
Parietal (oxyntic) cell	Body/fundus glands	**HCl** + intrinsic factor	Acid for digestion; intrinsic factor for vitamin B12 absorption
Chief (peptic) cell	Body/fundus glands	**Pepsinogen** (inactive)	Raw material for protein digestion
Mucous neck cell	Neck of glands	Mucus + bicarbonate	Protective barrier against acid
G cell	Pyloric glands	**Gastrin** (hormone)	Signals parietal cells to make more acid

Gastric juice: ~2–3 litres/day of acidic fluid (pH ~1.5–2.5) containing HCl, pepsinogen, mucus, intrinsic factor, and water.

7.4.4 Acid production — the parietal cell mechanism (exam favourite)

Steps (memorise in order):

1. Inside parietal cell: $CO_2 + H_2O \rightarrow H^+ + HCO_3^-$ (enzyme: carbonic anhydrase)
2. H^+ is pumped into the gland lumen by H^+/K^+ ATPase (the "proton pump") in exchange for K^+
3. Cl^- follows H^+ into the lumen through chloride channels
4. Result in lumen: $H^+ + Cl^- = HCl$
5. HCO_3^- passes into blood (the "alkaline tide")

Stimulants of acid (3 receptors on the parietal cell): ACh (via muscarinic receptors — parasympathetic/vagus), **gastrin** (via CCK2 receptors), **histamine** (via H2 receptors). All three converge to activate the proton pump. **Inhibitors: somatostatin** (from D cells), **secretin**, **GIP**, prostaglandins.

Why acid? (1) converts pepsinogen → pepsin; (2) kills most swallowed microbes; (3) denatures (unfolds) proteins so enzymes can attack them; (4) helps absorb iron and calcium.

> **Analogy:** The parietal cell is a **chemical factory**: raw materials (CO_2 + water) enter, the carbonic-anhydrase machine splits them, the proton-pump conveyor pushes H^+ out one door while Cl^- slips out beside it — and HCl rolls off the line.

Pharmacy relevance: Proton-pump inhibitors block the H^+/K^+ ATPase (the final step above); **H2 blockers** block the histamine receptor; **antacids** simply neutralize acid already made; **anticholinergics** reduce the vagal (ACh) drive. One mechanism diagram explains four drug classes.

7.4.5 Regulation of gastric secretion — three phases

1. **Cephalic phase** (~30% of secretion): sight, smell, taste, or *thought* of food → brain → **vagus nerve (parasympathetic)** → ACh → parietal + G cells. ("The mouth waters before the first bite.")
2. **Gastric phase** (~60%): food distends the stomach → stretch receptors + peptides activate vagal reflexes and **gastrin** release → more acid and pepsinogen.
3. **Intestinal phase** (inhibitory overall): acidic, fatty chyme entering the duodenum triggers **secretin, CCK, and GIP** → slow gastric emptying and reduce acid ("the intestine tells the stomach to slow down").

Flowchart:

```
See/smell food → Vagus (parasympathetic) → ACh → parietal cell → HCl  (cephalic)
Food stretches stomach → gastrin → parietal cell → more HCl          (gastric)
Acid/fat in duodenum → secretin/CCK/GIP → ↓ acid, ↓ emptying         (intestinal)
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Key takeaway (syllabus point): Parasympathetic (vagal) stimulation is the main *neural* driver of acid — which is why stress and vagal tone influence acidity, and why vagotomy (cutting the vagus, a historical ulcer surgery) reduced acid.

7.4.6 Pepsin and protein digestion (syllabus point)

- Chief cells release inactive **pepsinogen** (inactive = safe; it cannot digest the stomach itself).
- **HCl activates pepsinogen → pepsin** (acid removes a blocking peptide; pepsin then activates more pepsinogen — autocatalysis).
- **Pepsin** breaks proteins into **peptides** (short chains) — it starts protein digestion; it works best in strong acid (pH ~2).
- The stomach's **mucus–bicarbonate barrier** protects its own wall from being digested — when this barrier fails, ulcers form (see Chapter 11).

> **Analogy:** Pepsinogen is a pair of **safety scissors with the cover on**; stomach acid removes the cover, turning it into working scissors that snip protein chains.

India context: "Acidity" complaints are among the commonest pharmacy counter queries in India; explaining the vagal/meal-related pattern helps counsel on meal timing and why antacids give quick but short relief.

7.5 Common Confusions

- **Pepsinogen vs pepsin:** pepsinogen = inactive zymogen from chief cells; pepsin = active enzyme after HCl activation.
- **Parietal vs chief cells:** parietal = **P** for **P**roton pump (HCl); chief = **C** for **C**hymosin-like enzyme store (pepsinogen).
- **Gastrin vs secretin:** gastrin (stomach) *stimulates* acid; secretin (duodenum) *inhibits* acid and stimulates bicarbonate.
- **Cardiac sphincter** has nothing to do with the heart — "cardiac" here means "near the heart end" of the stomach.

7.6 Common Mistakes

- Saying the stomach absorbs most nutrients — it absorbs very little (some water, alcohol, aspirin); absorption is the small intestine's job.
- Writing that HCl digests food — HCl *activates pepsin* and kills microbes; **pepsin** digests protein.
- Placing acid production in chief cells — acid comes from **parietal** cells.

7.7 Mnemonics

- Gastric cells: "**P**arietal = **P**roton pump (acid); **C**hief = **C**uts protein (pepsinogen); **G** = **G**astrin (go signal); **M**ucous = **M**ucus (mask/protect)".
- Acid stimulants: "**A**ll **G**ood **H**earts pump acid" — **A**Ch, **G**astrin, **H**istamine → proton pump.

7.8 One-Minute Revision

- GI order: mouth → pharynx → oesophagus → stomach → small intestine → large intestine → anus.
- Wall: mucosa → submucosa → muscularis externa → serosa.
- Stomach: fundus, body, pylorus; cardiac + pyloric sphincters; lesser/greater curvatures; rugae.
- Cells: parietal (HCl + intrinsic factor), chief (pepsinogen), mucous (protection), G (gastrin).
- HCl: $CO_2 + H_2O \rightarrow H^+$ (via H^+/K^+ ATPase) + Cl^-; stimulated by ACh, gastrin, histamine.
- Regulation: cephalic (vagal), gastric (distension + gastrin), intestinal (secretin/CCK/GIP inhibit).
- Pepsinogen --(HCl)--> pepsin → digests proteins to peptides.

7.9 Practice Questions

MCQs

1. HCl in the stomach is secreted by: (a) chief cells (b) parietal cells (c) G cells (d) mucous cells. **Answer: (b)**
2. Pepsinogen is activated by: (a) bile (b) HCl (c) secretin (d) mucus. **Answer: (b)**
3. The parasympathetic transmitter stimulating acid secretion is: (a) norepinephrine (b) acetylcholine (c) dopamine (d) serotonin. **Answer: (b)**
4. The proton pump is: (a) Na^+/K^+ ATPase (b) H^+/K^+ ATPase (c) Ca^{2+} pump (d) Cl^- channel. **Answer: (b)**
5. Gastrin is secreted by: (a) parietal cells (b) chief cells (c) G cells (d) D cells. **Answer: (c)**

Short-answer (2–3 marks)

1. Draw and label the regions of the stomach.
2. Tabulate the four gastric gland cells with secretions.
3. Describe the three phases of gastric secretion.
4. How is pepsinogen activated? What is pepsin's role?

Long-answer (5–10 marks)

1. Describe the mechanism of HCl production by the parietal cell and its regulation through the parasympathetic system.
2. Describe the anatomy of the stomach and the functions of gastric juice components.

Application-based

1. A patient takes antacids for "acidity" but gets relief for only an hour. Using the regulation flowchart, explain why acid keeps returning and what longer-acting approaches target.
2. Why are proton-pump inhibitors taken before breakfast? (Think about when pumps are activated.)

Viva

1. Which cells secrete HCl and pepsinogen?
2. Name the three stimulants of the parietal cell.
3. What is chyme?
4. Which sphincter prevents acid reflux into the oesophagus?

CHAPTER 8: SMALL INTESTINE AND LARGE INTESTINE

Unit II · ~2 hours

8.1 Introduction

If the stomach is the mixing tank, the small intestine is the factory floor where digestion is completed and almost all absorption happens — across a surface area the size of a tennis court, packed into your abdomen by millions of tiny folds. The large intestine then reclaims water and forms the waste. Pharmacists use this daily: most oral drugs are absorbed in the small intestine, and constipation/diarrhoea medicines act on the large intestine. This chapter covers both.

Learning objectives — after studying this chapter, you should be able to:

- name the three parts of the small intestine and their lengths/functions;
- describe villi, microvilli, and crypts and explain how they aid absorption;
- list the brush-border enzymes and what each digests;
- describe the parts and functions of the large intestine;
- explain the defecation reflex;
- relate intestinal physiology to drug absorption and laxative action (general).

8.2 Definitions

Scientific definitions:

- **Villus (plural villi):** finger-like projection of the small-intestinal mucosa containing blood capillaries and a lymph vessel (lacteal); the main absorption unit.
- **Microvillus:** microscopic projection on each absorptive cell's surface, forming the **brush border** that carries digestive enzymes.
- **Peristalsis:** progressive wave of contraction that propels contents forward.
- **Segmentation:** rhythmic local contractions that mix chyme with juices without net forward movement.
- **Defecation:** the reflex emptying of faeces from the rectum.

Simple explanation: The small intestine multiplies its surface like a **towel folded into pleats, then into fringes, then into microscopic threads** — folds (plicae) → villi → microvilli — reaching ~200 m^2 for absorption. Segmentation is the **washing-machine agitation** (mixing); peristalsis is the **conveyor belt** (moving forward).

8.3 Key Terminology

Term	Meaning	Simple explanation
Duodenum	First ~25 cm of small intestine ("12 finger-breadths")	"Mixing bowl" — receives bile + pancreatic juice
Jejunum	Middle ~2.5 m	Main absorption zone
Ileum	Last ~3.5 m	Absorbs B12, bile salts; meets large intestine at ileocecal valve
Lacteal	Lymph vessel inside each villus	"Fat drain" — carries absorbed fats
Ileocecal valve	Guard between ileum and caecum	"One-way gate" preventing backflow
Haustra	Pouch-like segments of the colon	"Sausage-link pouches"
Taenia coli	Longitudinal muscle bands of the colon	"Drawstrings" that bunch the colon into haustra
Faeces	Undigested residue + water + bacteria + cells	The final waste

8.4 Detailed Explanation

8.4.1 Small intestine — three parts

Total length ~6 metres (in a living person; longer after death when muscle relaxes).

1. **Duodenum** (~25 cm, C-shaped around the pancreatic head): chyme from the stomach meets **bile** (from liver/gallbladder) and **pancreatic juice**; most chemical digestion happens here. Receives secretions via the **hepatopancreatic ampulla (of Vater)** guarded by the **sphincter of Oddi**.
2. **Jejunum** (~2.5 m): thick-walled, rich blood supply; the main site of **digestion and absorption** of carbohydrates, proteins, fats, vitamins.
3. **Ileum** (~3.5 m): completes absorption — **vitamin B12** (with intrinsic factor) and **bile salts** (recycled to the liver: **enterohepatic circulation**); ends at the **ileocecal valve**.

8.4.2 The absorption surface — folds within folds

1. **Plicae circulares** (circular folds) — large folds of mucosa + submucosa.
2. **Villi** (~1 mm finger-like; ~4–5 million) — each with capillaries (absorb sugars, amino acids → blood) and a **lacteal** (absorbs fats → lymph).
3. **Microvilli** (brush border) — on each absorptive cell; carry **brush-border enzymes**.

Together they create an enormous absorptive area (~200 m^2 — about a tennis court).

Brush-border enzymes table:

Enzyme	Substrate → Product
Maltase	Maltose → glucose + glucose
Sucrase	Sucrose → glucose + fructose
Lactase	Lactose → glucose + galactose
Peptidases (aminopeptidase, dipeptidase)	Peptides → amino acids
(Pancreatic enzymes arrive from the duct: amylase, lipase, trypsin — see Ch. 9)	

Clinical note (general): Lactase deficiency (common in many Indian adults) leaves lactose undigested → gas, bloating, diarrhoea after milk — "lactose intolerance". Recognise the pattern; refer/dietary counsel generally.

8.4.3 Large intestine — parts and functions

Length ~1.5 m, wider than the small intestine. Parts: **caecum** (pouch; **appendix** attached) → **ascending colon** → **transverse colon** → **descending colon** → **sigmoid colon** → **rectum** → **anal canal** (with internal involuntary + external voluntary sphincters). **Functions:** (1) absorb **water and electrolytes** (about 1–1.5 L/day), converting liquid chyme to solid faeces; (2) house **gut bacteria** that ferment fibre, make some **vitamin K and B vitamins**, and produce gas; (3) store faeces in the rectum; (4) expel via **defecation**. **Defecation reflex:** faeces stretch the rectum → spinal reflex contracts rectal muscle and relaxes the internal anal sphincter → urge felt; the **external sphincter is voluntary** — you choose when. (This is why toilet training is learning voluntary control.)

> **Analogy:** The large intestine is the **recycling plant**: it squeezes the last water out of the waste stream, lets bacteria compost the fibre, and bales the remainder for disposal.

Pharmacy relevance: Laxatives act here — bulk-forming (fibre holds water), osmotic (draw water in), stimulant (increase peristalsis). **Antidiarrhoeals** slow motility, giving more time for water absorption. Oral **iron** and most drugs are absorbed higher up, in the small intestine.

India context: Fibre-rich Indian diets (whole grains, vegetables, lentils) support colonic health; low-fibre, refined diets are linked with constipation — a common counselling point at the pharmacy counter.

8.5 Common Confusions

- **Villi vs microvilli:** villi = visible finger-like projections of mucosa; microvilli = microscopic projections on individual cells (the brush border).
- **Duodenum vs jejunum vs ileum order and jobs:** duodenum = mixing/digestion; jejunum = main absorption; ileum = B12 + bile salts.
- **Lacteal vs capillary in a villus:** lacteal (lymph) takes **fats**; capillaries (blood) take **sugars and amino acids**.

8.6 Common Mistakes

- Saying absorption mainly occurs in the stomach or large intestine — it is the **small intestine** (jejunum mainly).
- Writing that the large intestine digests food — it absorbs **water/electrolytes**; digestion is essentially finished before it.
- Confusing peristalsis (propulsion) with segmentation (mixing).

8.7 Mnemonics

- Small intestine order: "**D**ine **J**oyfully **I**nside" — **D**uodenum → **J**ejunum → **I**leum.
- Ileum absorbs: "**B**12 and **B**ile" (both start with B, both at the Bottom end).

8.8 One-Minute Revision

- Small intestine ~6 m: duodenum (25 cm, digestion), jejunum (2.5 m, absorption), ileum (3.5 m, B12 + bile salts).
- Surface: plicae → villi (capillary + lacteal) → microvilli (brush-border enzymes) ≈ 200 m^2.
- Brush border: maltase, sucrase, lactase, peptidases.
- Large intestine ~1.5 m: caecum → ascending → transverse → descending → sigmoid → rectum → anus.
- Functions: water/electrolyte absorption, bacterial fermentation (vitamin K), faeces storage, defecation.
- Defecation: rectal stretch → spinal reflex; external sphincter voluntary.

8.9 Practice Questions

MCQs

1. The main site of nutrient absorption is the: (a) stomach (b) duodenum (c) jejunum (d) colon. **Answer: (c)**
2. Lacteals absorb: (a) glucose (b) amino acids (c) fats (d) water. **Answer: (c)**
3. Vitamin B12 is absorbed in the: (a) duodenum (b) jejunum (c) ileum (d) colon. **Answer: (c)**
4. The brush border carries: (a) bile (b) digestive enzymes like lactase (c) mucus only (d) hormones. **Answer: (b)**
5. The voluntary sphincter in defecation is the: (a) internal anal (b) external anal (c) pyloric (d) ileocecal. **Answer: (b)**

Short-answer (2–3 marks)

1. Name the three parts of the small intestine with length and one function each.
2. How do villi and microvilli aid absorption?
3. List the parts of the large intestine and its four functions.

Long-answer (5–10 marks)

1. Describe the anatomy of the small intestine and explain the structural adaptations for absorption.
2. Describe the anatomy and functions of the large intestine, including the defecation reflex.

Application-based

1. A patient gets bloating and diarrhoea after drinking milk but not after eating curd. Explain using brush-border enzymes.
2. Why do stimulant laxatives act on the colon rather than the stomach? (motility + water absorption logic)

Viva

1. Which part of the small intestine receives bile and pancreatic juice?
2. What is the lacteal?
3. Name two brush-border enzymes.
4. What does the large intestine absorb?

CHAPTER 9: SALIVARY GLANDS, PANCREAS AND LIVER

Unit II · ~2 hours

9.1 Introduction

Three organs outside the GI tube pour their juices into it — saliva, pancreatic juice, and bile. Without them, digestion fails: no saliva, and starch digestion never starts; no pancreatic juice, and fats and proteins pass undigested; no bile, and fats cannot be absorbed (nor can fat-soluble vitamins A, D, E, K). This chapter covers each gland's anatomy and functions, as the syllabus requires.

Learning objectives — after studying this chapter, you should be able to:

- name the three pairs of salivary glands and describe saliva's composition and functions;
- describe the pancreas as a dual gland (exocrine + endocrine) and list pancreatic enzymes;
- describe the liver's gross anatomy and its digestive/metabolic functions;
- explain bile formation, storage, composition, and functions;
- state the pharmacy relevance (e.g., pancreatitis, gallstones, jaundice — general recognition).

9.2 Definitions

Scientific definitions:

- **Salivary glands:** exocrine glands pouring saliva into the mouth — three pairs: parotid, submandibular, sublingual.
- **Pancreas:** a dual (mixed) gland — **exocrine** part secretes digestive juice into the duodenum; **endocrine** part (islets of Langerhans) secretes insulin and glucagon into blood.
- **Liver:** the largest internal organ and largest gland; produces bile and performs metabolic, storage, and detoxification functions.
- **Bile:** a yellow-green alkaline fluid made by the liver, stored in the gallbladder, that emulsifies fats.

Simple explanation: These are the **catering suppliers** of the food factory: salivary glands = the **pre-wash station** (moisten + start starch digestion), pancreas = the **main chemical supplier** (enzymes for all three food types + bicarbonate), liver = the **multipurpose plant** (makes bile detergent, processes nutrients, detoxifies).

9.3 Key Terminology

Term	Meaning	Simple explanation
Amylase	Starch-digesting enzyme	"Starch scissors"
Emulsification	Breaking large fat globules into tiny droplets	"Dishwash liquid action on grease"
Acinus	Grape-like cluster of enzyme-secreting pancreatic cells	"Enzyme grape bunch"
Islets of Langerhans	Endocrine cell clusters in the pancreas	"Hormone islands"
Hepatic lobule	Hexagonal functional unit of the liver	"Liver's building block"
Kupffer cells	Liver macrophages	"Liver's security guards"
Gallbladder	Pear-shaped sac storing/concentrating bile	"Bile warehouse"
Portal vein	Carries nutrient-rich blood from gut to liver	"Delivery highway to the liver"

9.4 Detailed Explanation

9.4.1 Salivary glands and saliva

Three pairs: parotid (largest, in front of the ear — swells in mumps), **submandibular** (under the jaw), **sublingual** (under the tongue). Together they make ~1–1.5 litres of saliva daily. **Composition:** ~99.5% water + **salivary amylase (ptyalin)** + **lysozyme** (antibacterial enzyme) + mucus + salts (bicarbonate) + a little urea. **Functions:** (1) moistens food and binds it into a **bolus** for swallowing; (2) **amylase** starts starch digestion (starch → maltose); (3) **lysozyme** kills some mouth bacteria; (4) dissolves food chemicals for taste; (5) cleanses the mouth and teeth. **Regulation:** mainly **parasympathetic** ("rest and digest" — watery saliva flows at the sight/smell of food); sympathetic gives small amounts of thick saliva ("dry mouth in fear").

India context: Mumps (parotid swelling) is a familiar childhood illness; dry mouth is a common complaint with many medicines (anticholinergic effect) — pharmacists counsel on hydration and sugar-free gum.

9.4.2 The pancreas — a dual gland

- **Location/shape:** soft, leaf-shaped gland ~15 cm long, lying **behind the stomach**, head tucked in the duodenal C-loop, tail toward the spleen.
- **Exocrine part (~99%): acini** secrete **pancreatic juice** (~1–1.5 L/day) into the duodenum via the pancreatic duct. Contents:
- **Bicarbonate (HCO_3^-)** — neutralizes acidic chyme (protects duodenum; gives enzymes their working pH).
- **Pancreatic amylase** — starch → maltose.
- **Pancreatic lipase** — fats → fatty acids + monoglycerides (the main fat digester).
- **Trypsinogen/chymotrypsinogen** (activated to trypsin/chymotrypsin in the duodenum) — proteins → peptides. (Released inactive = safe; activated only in the gut by **enterokinase**.)
- **Procarboxypeptidase, nucleases** — finish protein/nucleic-acid digestion.
- **Endocrine part (~1%): islets of Langerhans** — alpha cells (glucagon), beta cells (insulin), delta cells (somatostatin). (Hormone detail belongs to Unit IV/endocrine, but know the islets sit in the pancreas.)

Clinical note (general): If pancreatic enzymes activate *inside* the pancreas, they digest the gland itself — **pancreatitis** (severe upper abdominal pain). Chronic alcohol use and gallstones are known risk factors (general knowledge). Long-standing pancreatic damage can lead to diabetes (beta-cell loss).

9.4.3 The liver — anatomy and functions

- **Gross anatomy:** largest internal organ (~1.5 kg); sits in the right upper abdomen under the diaphragm; **four lobes** (right, left, caudate, quadrate); **porta hepatis** on the underside where the portal vein, hepatic artery, and bile duct enter/leave; functional unit = **hepatic lobule** (hexagonal, with central vein).
- **Blood supply (dual): hepatic portal vein** (~75% — nutrient-rich blood from the gut) + **hepatic artery** (~25% — oxygen). Blood leaves via hepatic veins to the inferior vena cava.
- **Functions table (exam favourite):**

Category	Functions
Bile production	Makes ~500 mL–1 L bile/day for fat emulsification
Metabolism	Converts glucose ↔ glycogen (storage); makes plasma proteins (albumin, clotting factors); processes fats and amino acids
Storage	Glycogen, iron, copper, vitamins A, D, B12
Detoxification	Converts ammonia → urea; metabolizes drugs, alcohol, toxins (**first-pass metabolism** — key pharmacy concept)
Defence	Kupffer cells (macrophages) remove old red cells, bacteria, debris
Blood	Foetal blood-cell formation; removes worn-out red cells; makes bile pigments from haemoglobin

Bile — composition and functions: water + **bile salts** + bile pigments (**bilirubin**, from old red cells — gives faeces/urine colour) + cholesterol + phospholipids. Stored and concentrated in the **gallbladder**; released when fatty chyme triggers **CCK**. Functions: (1) **emulsifies fats** (breaks big globules into droplets for lipase); (2) aids absorption of **fat-soluble vitamins A, D, E, K**; (3) **alkaline** — neutralizes stomach acid in the duodenum; (4) excretes bilirubin, cholesterol, some drugs.

Pharmacy relevance: The liver's **first-pass metabolism** decides how much of an oral drug reaches the blood — the basis of bioavailability and why some drugs can't be given orally. Bile-salt deficiency (liver/gallbladder disease) causes fatty stools and poor vitamin A/D/E/K absorption — pharmacists recognise the pattern and refer.

India context: Gallstones and fatty liver are increasingly common in India with changing diets; "liver tonics" are popular but evidence for most is limited — pharmacists should counsel on proven measures (weight, alcohol, diet) rather than unproven products.

9.5 Common Confusions

- **Exocrine vs endocrine pancreas:** exocrine = juice via **ducts** to the duodenum; endocrine = hormones via **blood** (islets).
- **Bile vs pancreatic juice:** bile (liver) **emulsifies** fat but digests nothing — it has **no enzymes**; pancreatic juice has the **enzymes**.
- **Gallbladder vs liver:** the liver **makes** bile; the gallbladder only **stores and concentrates** it.
- **Trypsinogen vs trypsin:** inactive (−ogen) form made by pancreas; activated to trypsin in the duodenum by enterokinase.

9.6 Common Mistakes

- Saying bile digests fat — bile **emulsifies**; **lipase** digests.
- Placing insulin/glucagon secretion in the exocrine pancreas — they come from the **islets** (endocrine).
- Writing that the liver stores bile — the **gallbladder** stores it.

9.7 Mnemonics

- Salivary glands: "**P**arotid = **P**re-ear (biggest); **S**ubmandibular = **S**ub-jaw; **S**ublingual = **S**ub-tongue" — "**P**lease **S**erve **S**aliva".
- Pancreatic enzymes: "**A**mylase (st**A**rch), **L**ipase (**L**ipids), **T**rypsin (pro**T**ein)" — "**ALT** — the pancreas handles ALT foods".
- Bile functions: "**E**mulsify, **A**bsorb vitamins (A,D,E,K), **A**lkalinize, **E**xcrete" ("**EAAE** — bile does EAAE work").

9.8 One-Minute Revision

- Salivary glands: parotid, submandibular, sublingual; saliva = water + amylase (starch) + lysozyme + mucus; parasympathetic-driven.
- Pancreas: behind stomach; exocrine acini → bicarbonate + amylase + lipase + trypsinogen (activated by enterokinase); endocrine islets → insulin/glucagon.
- Liver: ~1.5 kg, 4 lobes, porta hepatis; dual blood supply (portal vein + hepatic artery).
- Liver functions: bile, metabolism, storage (glycogen, iron, A/D/B12), detoxification (first-pass), Kupffer-cell defence, plasma proteins.
- Bile: no enzymes; emulsifies fat; absorbs A/D/E/K; alkaline; excretes bilirubin. Stored in gallbladder, released by CCK.

9.9 Practice Questions

MCQs

1. The largest salivary gland is the: (a) sublingual (b) submandibular (c) parotid (d) buccal. **Answer: (c)**
2. Bile is produced by the: (a) gallbladder (b) liver (c) pancreas (d) duodenum. **Answer: (b)**
3. Trypsinogen is activated by: (a) HCl (b) enterokinase (c) bile (d) secretin. **Answer: (b)**
4. Which is NOT a function of bile? (a) fat emulsification (b) protein digestion (c) vitamin K absorption (d) bilirubin excretion. **Answer: (b)**
5. Insulin is secreted by: (a) acini (b) islets of Langerhans (c) Kupffer cells (d) hepatocytes. **Answer: (b)**

Short-answer (2–3 marks)

1. Name the salivary glands and list three functions of saliva.
2. Tabulate the pancreatic enzymes with their substrates.
3. List six functions of the liver.
4. What is emulsification? Why does it matter?

Long-answer (5–10 marks)

1. Describe the pancreas as a dual gland: exocrine secretions and endocrine islets.
2. Describe the gross anatomy of the liver and its functions. Add a note on bile.

Application-based

1. A patient who had the gallbladder removed is advised to avoid very fatty meals. Explain using bile physiology.
2. Why is "first-pass metabolism" in the liver important when choosing between oral and injectable forms of a drug?

Viva

1. Which gland makes bile and which stores it?
2. Name the three pairs of salivary glands.
3. What activates trypsinogen?
4. What are Kupffer cells?

CHAPTER 10: MOVEMENTS OF THE GIT, DIGESTION AND ABSORPTION OF NUTRIENTS

Unit II · ~2 hours

10.1 Introduction

Food's journey takes 24–72 hours, powered by coordinated muscle movements and completed by enzymes — ending with glucose, amino acids, and fatty acids entering the blood. This chapter joins the whole story: how food moves (deglutition → peristalsis → segmentation → mass movements), how each nutrient is digested step by step, and how each is absorbed. It is the chapter that turns separate organs into one working system — and explains why "take on an empty stomach" or "take with food" instructions exist.

Learning objectives — after studying this chapter, you should be able to:

- describe the three stages of deglutition (swallowing);
- differentiate peristalsis, segmentation, and mass movements;
- trace carbohydrate, protein, and fat digestion from mouth to intestine in flowcharts;
- explain the absorption of glucose, amino acids, fatty acids, water, and vitamins;
- state the pharmacy relevance (food–drug timing, ORS principle — in general terms).

10.2 Definitions

Scientific definitions:

- **Deglutition:** swallowing — moving the bolus from mouth to stomach in three stages.
- **Mass movement:** strong, slow peristaltic waves of the colon moving contents toward the rectum (1–3 times daily).
- **Migrating motor complex (MMC):** cyclical waves of motility sweeping the stomach and small intestine clean between meals ("housekeeper waves").
- **Emulsification:** bile's breaking of large fat globules into tiny droplets.
- **Micelle:** tiny bile-salt-coated droplet carrying digested fats to the absorptive surface.

Simple explanation: The gut moves food the way a **factory line** does: swallowing = loading the belt, peristalsis = the belt moving forward, segmentation = the mixing paddles, mass movements = the end-of-day sweep pushing waste to the exit, and the MMC = the night-shift cleaner between meals.

10.3 Key Terminology

Term	Meaning	Simple explanation
Bolus	Chewed, saliva-moistened food mass	"Food ball" ready to swallow
Chyme	Acidic semi-liquid leaving the stomach	"Food soup"
Epiglottis	Flap covering the windpipe during swallowing	"Traffic policeman" — food goes to food pipe, not windpipe
Brush border	Microvilli with enzymes on absorptive cells	"Final checkout counter"
Chylomicron	Lipoprotein carrying fats into lymph	"Fat parcel" for delivery
SGLT1	Sodium–glucose transporter absorbing glucose	"Glucose taxi" (needs sodium as co-passenger)

10.4 Detailed Explanation

10.4.1 Deglutition — three stages of swallowing

1. **Oral (voluntary):** tongue pushes the bolus to the pharynx.
2. **Pharyngeal (involuntary):** soft palate rises (closes nose), **epiglottis** covers the larynx (closes windpipe), pharyngeal muscles squeeze the bolus into the oesophagus. Breathing pauses briefly.
3. **Oesophageal (involuntary): peristalsis** carries the bolus down; the **lower oesophageal sphincter** relaxes to admit it to the stomach, then closes to prevent reflux.

Clinical note (general): A weak lower oesophageal sphincter lets acid flow back — **gastro-oesophageal reflux (heartburn)**. Recognise the pattern (burning behind the breastbone after meals/lying down); refer persistent cases.

10.4.2 Types of GIT movements

Movement	Where	What happens	Purpose
Peristalsis	Oesophagus → rectum	Wave of contraction behind + relaxation ahead	Propulsion (forward movement)
Segmentation	Small intestine (mainly)	Rhythmic local squeezes	Mixing chyme with juices
Mass movements	Colon	Strong slow waves (1–3/day, often after meals — **gastrocolic reflex**)	Push faeces toward rectum
MMC (housekeeper)	Stomach + small intestine, between meals	Sweeping waves every ~90 min	Clear debris, bacteria, undigested bits
Tonic contractions	Sphincters	Sustained partial contraction	Guard junctions (one-way flow)

10.4.3 Digestion of nutrients — three flowcharts

Carbohydrates:

> Mouth: salivary amylase → starch → maltose (little time to act)
> Stomach: acid stops amylase; no carb digestion here
> Duodenum: pancreatic amylase → maltose/maltotriose
> Brush border: maltase → glucose; sucrase → glucose + fructose; lactase → glucose + galactose
> Absorption: glucose/galactose via SGLT1 (with Na^+); fructose by facilitated diffusion

Proteins:

> Stomach: pepsinogen --(HCl)--> pepsin → proteins → peptides
> Duodenum: trypsin/chymotrypsin/carboxypeptidase → smaller peptides
> Brush border: peptidases → amino acids, di/tripeptides
> Absorption: amino acids via Na^+-linked transporters into blood

Fats:

Mouth/stomach: almost none (gastric lipase is minor)
Duodenum: bile EMULSIFIES fat globules → tiny droplets
pancreatic lipase → fatty acids + monoglycerides
→ form MICELLES with bile salts
Absorption: micelles deliver to brush border → inside cell rebuilt to triglycerides
→ packed into CHYLOMICRONS → lacteal → lymph → blood

Key takeaway: Carbs → **glucose** (blood); proteins → **amino acids** (blood); fats → **chylomicrons** (lymph first, then blood). Water, salts, and most vitamins are absorbed along the small intestine; **B12** in the ileum (with intrinsic factor); **fat-soluble vitamins A, D, E, K** need bile/micelles.

10.4.4 Absorption summary table

Nutrient	Final form absorbed	Absorbed into	Site
Carbohydrate	Glucose, galactose, fructose	Blood (SGLT1/diffusion)	Jejunum
Protein	Amino acids, small peptides	Blood	Jejunum
Fat	Fatty acids/monoglycerides → chylomicrons	Lymph (lacteal)	Jejunum/ileum
Water	Water	Blood	Small + large intestine
Iron, calcium	Ions	Blood	Duodenum/jejunum
Vitamin B12	B12–intrinsic factor complex	Blood	Ileum
Vitamins A, D, E, K	With micelles	Lymph	Small intestine

Pharmacy relevance: Food–drug timing follows this physiology — drugs absorbed like nutrients compete or cooperate with food (general principle; specific advice comes from the drug's own literature). **ORS (oral rehydration solution)** works on the SGLT1 principle: glucose + sodium together pull water into the body — which is why ORS, not plain water, treats dehydration in diarrhoea (general mechanism, no formula quoted).

India context: ORS is one of India's great public-health successes against childhood diarrhoeal dehydration — every pharmacist should know *why* it works (sodium–glucose co-transport), not just that it does.

10.5 Common Confusions

- **Peristalsis vs segmentation:** peristalsis = forward propulsion; segmentation = stationary mixing.
- **Micelle vs chylomicron:** micelle = bile-salt droplet *delivering* fats to the gut wall (in the lumen); chylomicron = lipoprotein *carrying* rebuilt fats away in lymph (inside the body).
- **Emulsification vs digestion:** emulsification (bile) is physical breaking-up; digestion (lipase) is chemical cutting.

10.6 Common Mistakes

- Saying fats are absorbed into blood capillaries — they go via **lacteals into lymph** as chylomicrons.
- Writing that protein digestion ends in the stomach — it only *starts* there; it finishes at the brush border.
- Claiming the stomach digests carbohydrates — salivary amylase is destroyed by stomach acid; carb digestion resumes in the duodenum.

10.7 Mnemonics

- Fat pathway: "**E**mulsify → **D**igest → **M**icelle → **C**hylomicron" ("**E**at **D**inner **M**aking **C**haat").
- Movements: "**P**eristalsis **P**ushes; **S**egmentation **S**tirs; **M**ass **M**ovements **M**arch waste out".

10.8 One-Minute Revision

- Swallowing: oral (voluntary) → pharyngeal (epiglottis guards airway) → oesophageal (peristalsis).
- Movements: peristalsis (propel), segmentation (mix), mass movements (colon sweep), MMC (between-meal housekeeper).
- Carbs: amylase → brush border → glucose → SGLT1 → blood.
- Proteins: pepsin → pancreatic proteases → brush-border peptidases → amino acids → blood.
- Fats: bile emulsifies → lipase digests → micelles deliver → chylomicrons → lymph.
- B12 in ileum; A/D/E/K need bile; water in small + large intestine.

10.9 Practice Questions

MCQs

1. Fats are absorbed mainly as chylomicrons into the: (a) blood capillaries (b) lacteals (c) portal vein (d) bile duct. **Answer: (b)**
2. Glucose is absorbed via: (a) simple diffusion (b) SGLT1 with sodium (c) lacteals (d) pinocytosis. **Answer: (b)**
3. The epiglottis prevents food from entering the: (a) oesophagus (b) trachea (c) stomach (d) pharynx. **Answer: (b)**
4. Micelles are formed by: (a) proteins (b) bile salts + digested fats (c) glucose (d) enzymes. **Answer: (b)**
5. Mass movements occur mainly in the: (a) oesophagus (b) stomach (c) colon (d) duodenum. **Answer: (c)**

Short-answer (2–3 marks)

1. Describe the three stages of deglutition.
2. Differentiate peristalsis and segmentation.
3. Trace the digestion and absorption of fats with a flowchart.

Long-answer (5–10 marks)

1. Describe the digestion and absorption of carbohydrates, proteins, and fats with flowcharts.
2. Describe the movements of the GIT and their functions.

Application-based

1. Explain, using SGLT1, why ORS (salt + sugar solution) rehydrates better than plain water in diarrhoea.
2. A patient on a fat-free diet develops night blindness over time. Connect this to bile and micelles. (vitamin A is fat-soluble)

Viva

1. What is the difference between a micelle and a chylomicron?
2. Where is vitamin B12 absorbed?
3. Name the "housekeeper" wave of the gut.
4. Which enzyme starts starch digestion, and where?

CHAPTER 11: PATHOPHYSIOLOGY OF GASTROINTESTINAL DISORDERS

Unit II · ~3 hours

11.1 Introduction

GI complaints — acidity, ulcers, jaundice, diarrhoea, fatty liver — fill Indian pharmacy counters daily. This chapter covers the six disorder groups in the syllabus: what each is, what goes wrong (pathophysiology, in general terms), typical features to *recognise*, and when to *refer*. You will not diagnose or prescribe — you will recognise patterns, counsel on lifestyle and adherence, and send the patient to a doctor at the right time.

Learning objectives — after studying this chapter, you should be able to:

- define IBD and differentiate Crohn's disease from ulcerative colitis;
- explain the pathophysiology of peptic ulcer (acid–pepsin vs mucosal defence);
- classify jaundice and explain bilirubin handling in simple terms;
- describe the types and transmission of hepatitis;
- describe typhoid's cause, transmission, and typical features;
- differentiate alcoholic from non-alcoholic fatty liver disease and describe the progression to cirrhosis (general);
- list red flags needing urgent referral for each.

11.2 Definitions (quick glossary)

Term	Simple meaning
Ulcer	A break in the lining (mucosa) exposing deeper layers
Inflammation	The body's defensive response: redness, swelling, pain, heat, loss of function
Cirrhosis	Late-stage liver scarring where normal tissue is replaced by fibrous bands
Steatosis	Abnormal fat accumulation inside liver cells ("fatty liver")
Faeco-oral transmission	Spread when stool contaminates food/water that is then swallowed

11.3 The Six Disorder Groups

11.3.1 Inflammatory bowel diseases (IBD)

- **What:** chronic, relapsing inflammation of the intestines. Two main types:
- **Comparison table (exam favourite):**

Feature	Crohn's disease	Ulcerative colitis
Site	Any part of GI tract (mouth → anus), most often terminal ileum	**Colon and rectum only**, starting at the rectum
Depth	**Transmural** (full wall thickness)	**Mucosal** (inner lining only)
Pattern	**Skip lesions** (patchy — diseased areas between normal ones)	**Continuous** (no skip areas)
Complications (general)	Fistulas (abnormal connections), strictures (narrowing)	Higher long-standing cancer vigilance; toxic megacolon in severe flares
Typical features	Abdominal pain, diarrhoea, weight loss, sometimes mouth-to-anus lesions	Bloody diarrhoea, urgency, abdominal cramps

- **Pathophysiology (general):** an abnormal immune response against the gut's own microbes in genetically susceptible people → chronic inflammation → ulcers, bleeding, malabsorption.
- **Pharmacist's role:** recognise chronic bloody diarrhoea + weight loss as referral-worthy; counsel on adherence to long-term medicines; advise smoking avoidance in Crohn's (general lifestyle counsel); never treat chronic bloody diarrhoea with over-the-counter antidiarrhoeals alone — refer.

11.3.2 Peptic ulcer

- **What:** a break (ulcer) in the lining of the **stomach (gastric ulcer)** or **duodenum (duodenal ulcer)** — sometimes the lower oesophagus.
- **Pathophysiology:** an imbalance between **aggressive factors** (acid, pepsin, *H. pylori* infection, NSAIDs, smoking, alcohol) and **defensive factors** (mucus–bicarbonate barrier, blood flow, prostaglandins). Key causes: ***Helicobacter pylori*** infection (a bacterium living in stomach mucus) and **NSAID use** (they block protective prostaglandins).
- **Typical features (general):** burning/gnawing upper abdominal pain related to meals (gastric ulcer: worse with food; duodenal ulcer: pain eased by food, worse on empty stomach — general patterns), nausea, bloating; **alarm features**: vomiting blood, black tarry stools (melaena), unexplained weight loss, persistent vomiting.
- **Pharmacist's role: refer alarm features immediately**; counsel NSAID users to report stomach pain and never ignore black stools; advise on meal regularity, avoiding smoking/alcohol excess, and completing *H. pylori* treatment courses (adherence counselling).

11.3.3 Jaundice

- **What:** yellow discolouration of skin, eyes, and mucous membranes due to excess **bilirubin** in blood.
- **Bilirubin story (simple):** old red blood cells are broken down (mainly in spleen/liver) → **unconjugated bilirubin** (not water-soluble) travels bound to albumin → liver **conjugates** it (makes it water-soluble) → excreted in **bile** → gives stool its brown colour; some is reabsorbed (**enterohepatic circulation**).
- **Classification (exam favourite):**

Type	Where the fault is	Examples (general)	Clue pattern
Pre-hepatic (haemolytic)	Too much bilirubin made	Excess red-cell breakdown	Unconjugated bilirubin rises
Hepatic (hepatocellular)	Liver can't process	Hepatitis, liver injury	Both fractions rise; liver enzymes raised
Post-hepatic (obstructive)	Bile can't flow out	Gallstones, tumours blocking bile duct	Pale/clay stools, dark urine, itching

- **Typical features:** yellow eyes/skin, dark urine, pale stools (obstructive), itching, fatigue.
- **Pharmacist's role:** any new jaundice needs **medical evaluation** — refer; in newborns, jaundice is common but must be assessed by a doctor (never reassure blindly); counsel against alcohol and self-medication with "liver tonics" of unproven value.

11.3.4 Hepatitis

- **What: inflammation of the liver**, most often due to viruses (A, B, C, D, E); also drugs, alcohol, autoimmune causes.
- **Virus table (exam favourite):**

Virus	Main transmission	General notes
Hepatitis A	Faeco-oral (contaminated food/water)	Usually acute, self-limiting; vaccine-preventable
Hepatitis B	Blood, sexual contact, mother-to-child	Can become chronic → cirrhosis/liver cancer risk; vaccine-preventable
Hepatitis C	Blood (transfusions before screening, shared needles)	Often chronic; curable with modern medicines (general)
Hepatitis D	Only with hepatitis B co-infection	Worsens B
Hepatitis E	Faeco-oral (water-borne)	Like A; can be severe in pregnancy (general)

- **Typical features (general):** fatigue, loss of appetite, nausea, right-upper-abdominal discomfort, jaundice, dark urine; acute vs **chronic** (lasting months–years, may silently progress).
- **Pharmacist's role:** counsel on vaccination (A and B) where available; safe food/water and hygiene advice; strict adherence counselling for chronic hepatitis medicines; safe disposal of sharps; refer all suspected cases.

India context: Hepatitis B vaccination is part of India's Universal Immunization Programme — pharmacists can remind families about completing the schedule.

11.3.5 Typhoid

- **What:** a systemic bacterial infection caused by ***Salmonella* Typhi** (and close relatives causing paratyphoid).
- **Pathophysiology (general):** ingested via **contaminated food/water (faeco-oral)** → invades intestinal lymphoid tissue (Peyer's patches) → enters blood → spreads to liver, spleen, bone marrow → sustained infection.
- **Typical features (general):** prolonged **step-ladder fever** (rising daily), headache, abdominal pain, constipation *or* diarrhoea, **rose spots** (faint pink rash) in some, enlarged spleen; complications can include intestinal bleeding/perforation in severe untreated cases.
- **Pharmacist's role:** suspect in prolonged fever with abdominal symptoms — **refer for testing** (blood culture); counsel on completing the full antibiotic course (stopping early breeds resistance and relapse); prevention: safe water, hygienic food, vaccination where advised.

India context: Typhoid remains common in India where water sanitation is poor; the monsoon season often brings a rise in cases — a familiar exam and counselling topic.

11.3.6 Alcoholic and non-alcoholic fatty liver disease

- **What: steatosis** — excess fat stored in liver cells. **Alcoholic (AFLD):** due to chronic heavy alcohol use. **Non-alcoholic (NAFLD):** in people who drink little/no alcohol — linked with obesity, diabetes, and metabolic syndrome.
- **Spectrum of progression (general):** simple steatosis (fat only, often reversible) → **steatohepatitis** (fat + inflammation) → **fibrosis** (scarring) → **cirrhosis** (severe scarring, liver failure risk). Stopping alcohol / reducing weight can halt or partly reverse early stages (general).
- **Comparison table:**

Feature	Alcoholic fatty liver disease	Non-alcoholic fatty liver disease
Cause	Chronic heavy alcohol intake	Obesity, insulin resistance, diabetes, dyslipidaemia
Who	Heavy drinkers	Often overweight/diabetic individuals
Key advice	Complete abstinence from alcohol	Weight reduction, diet, exercise, diabetes control
Progression	Same spectrum: steatosis → hepatitis → fibrosis → cirrhosis	Same spectrum

- **Typical features (general):** often **no symptoms** early (found on scans or blood tests); later: fatigue, right-upper-abdominal discomfort, enlarged liver; cirrhosis brings jaundice, swelling, bleeding tendencies.
- **Pharmacist's role:** counsel empathetically on alcohol reduction/cessation (no judgment); support weight/diabetes management; warn that "liver tonics" don't reverse scarring — proven measures do; refer abnormal liver reports for medical follow-up.

11.4 Common Confusions

- **Crohn's vs ulcerative colitis:** Crohn's = anywhere, transmural, skip lesions; UC = colon only, mucosal, continuous.
- **Gastric vs duodenal ulcer pain pattern:** gastric often worse *with* food; duodenal often eased by food, worse when empty (general patterns, not rules).
- **Hepatitis A/E vs B/C:** A and E = faeco-oral (food/water); B, C, D = blood/body fluids.
- **Jaundice vs hepatitis:** jaundice is a *sign* (yellow colour); hepatitis is a *disease* (liver inflammation) that can *cause* jaundice — not synonyms.
- **Steatosis vs cirrhosis:** steatosis = fat accumulation (early, reversible); cirrhosis = end-stage scarring (largely irreversible).

11.5 Common Mistakes

- Treating chronic bloody diarrhoea as simple diarrhoea with OTC medicines — it needs medical evaluation (possible IBD).
- Ignoring black tarry stools or vomiting blood in ulcer patients — these are emergencies.
- Calling all jaundice "hepatitis" — jaundice has pre-hepatic and obstructive causes too.
- Advising alcohol "in moderation" to someone with alcoholic liver disease — the advice is abstinence.

11.6 Mnemonics

- Crohn's vs UC: "**C**rohn's = **C**an occur anywhere, **C**obblestone skip lesions, **C**omplete wall; **U**lcerative **C**olitis = **U**niform **C**olon, **C**ontinuous, **C**loser to surface (mucosal)".
- Hepatitis transmission: "**A** and **E** = **A**te it (food/water); **B**, **C**, **D** = **B**lood".
- Fatty liver spectrum: "**S**teatosis → steato**H**epatitis → **F**ibrosis → **C**irrhosis" ("**S**lowly **H**eading **F**or **C**irrhosis").

11.7 One-Minute Revision

- IBD: Crohn's (anywhere, transmural, skip) vs UC (colon only, mucosal, continuous); chronic bloody diarrhoea = refer.
- Peptic ulcer: acid/pepsin + H. pylori/NSAIDs overpower mucosal defence; alarm = blood vomit, black stools.
- Jaundice: pre-hepatic (too much made) / hepatic (liver can't process) / post-hepatic (bile blocked); any new jaundice = refer.
- Hepatitis: A/E faeco-oral; B/C/D blood; B and C can be chronic; vaccines for A and B.
- Typhoid: Salmonella Typhi, faeco-oral, step-ladder fever, rose spots; complete the antibiotic course.
- Fatty liver: alcoholic (stop alcohol) vs non-alcoholic (weight/diabetes); spectrum steatosis → fibrosis → cirrhosis.

11.8 Practice Questions

MCQs

1. Skip lesions and transmural inflammation occur in: (a) ulcerative colitis (b) Crohn's disease (c) peptic ulcer (d) typhoid. **Answer: (b)**
2. The commonest infectious cause of peptic ulcer is: (a) E. coli (b) H. pylori (c) Salmonella (d) Shigella. **Answer: (b)**
3. Hepatitis B is mainly transmitted by: (a) contaminated water (b) blood and body fluids (c) air (d) mosquitoes. **Answer: (b)**
4. Pale stools, dark urine, and itching suggest: (a) haemolytic jaundice (b) obstructive jaundice (c) hepatitis A (d) typhoid. **Answer: (b)**
5. Typhoid is caused by: (a) Vibrio cholerae (b) Salmonella Typhi (c) Hepatitis E virus (d) E. coli. **Answer: (b)**
6. Simple hepatic steatosis means: (a) liver scarring (b) fat accumulation in liver cells (c) bile duct block (d) liver cancer. **Answer: (b)**

Short-answer (2–3 marks)

1. Differentiate Crohn's disease from ulcerative colitis.
2. Explain the aggressive vs defensive factors in peptic ulcer formation.
3. Classify jaundice with one example each.
4. Tabulate the five hepatitis viruses with transmission.

Long-answer (5–10 marks)

1. Describe the pathophysiology of peptic ulcer disease and its typical features, including alarm signs.
2. Write short notes on: (a) IBD, (b) jaundice types, (c) typhoid, (d) fatty liver disease spectrum.

Application-based

1. A regular NSAID user reports black tarry stools. What is your immediate action and why?
2. A customer asks for "something for jaundice" at the counter. How do you respond?

Viva

1. Which two factors most commonly cause peptic ulcer?
2. How does hepatitis A spread? How does hepatitis C spread?
3. What is the difference between steatosis and cirrhosis?
4. Name the three types of jaundice.

UNIT II INFOGRAPHIC CONCEPT — "The GI System at a Glance"

Design note for PDF builder: single-page visual summary.

- **Centre:** GI tract silhouette (mouth → anus) with the three accessory glands (salivary, liver, pancreas) attached; arrow showing food flow direction.
- **Left panel:** stomach close-up — four cell types (parietal/HCl, chief/pepsinogen, G/gastrin, mucous) with the acid-regulation flowchart (vagus → ACh → HCl; gastrin; secretin/CCK inhibit) and "pepsinogen --(HCl)--> pepsin".
- **Right panel:** absorption map — villus cross-section (capillary = sugars/amino acids → blood; lacteal = fats → lymph); nutrient table mini-version (carbs→glucose, protein→amino acids, fat→chylomicrons); brush-border enzymes.
- **Bottom strip:** six disorder groups as icons with one-line tags: IBD (Crohn's vs UC), Peptic ulcer (H. pylori/NSAIDs), Jaundice (3 types), Hepatitis (A/E food-water; B/C blood), Typhoid (faeco-oral, step-ladder fever), Fatty liver (alcoholic vs NAFLD → cirrhosis spectrum).
- **Footer line:** "Mouth moistens · Stomach churns · Small intestine absorbs · Large intestine reclaims water".

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