

Liver and Gastric Function Tests.

? 1. TESTS FOR LIVER FUNCTION (LFTs)

Liver function tests evaluate **synthetic capacity, excretory function, detoxification, and liver cell integrity.**

? A. Tests for Hepatocellular Integrity (Damage)

? 1. Aminotransferases

- **ALT (Alanine transaminase)**
 - More specific for liver damage
 - Markedly elevated in viral hepatitis
- **AST (Aspartate transaminase)**
 - Also elevated in muscle injury
 - AST:ALT >2 ? **Alcoholic hepatitis**

? 2. LDH

- LDH-5 (liver component) increases in hepatic injury.
-

? B. Tests for Cholestasis (Obstruction)

? 1. Alkaline Phosphatase (ALP)

- Raised in **biliary obstruction**
- Also high in bone disease ? check GGT to confirm liver cause

? 2. ?-Glutamyl Transferase (GGT)

- High in alcohol intake and cholestasis
 - Helps differentiate **hepatic ALP** from **bone ALP**
-

? C. Tests for Synthetic (Metabolic) Function

? 1. Serum Albumin

- Low in chronic liver disease
- Normal in acute hepatitis (long half-life ~21 days)

? 2. Prothrombin Time (PT/INR)

- Prolonged in:
 - Acute liver failure
 - Vitamin K deficiency
 - Cholestasis (poor vitamin K absorption)

? 3. Serum Cholesterol

- Low in severe liver disease
 - High in obstructive jaundice (due to impaired excretion)
-

? D. Tests for Excretory Function

? 1. Serum bilirubin (total, direct, indirect)

Assesses conjugation + excretion.

? 2. Urine bilirubin & urobilinogen

- **Urine bilirubin positive** ? conjugated hyperbilirubinemia
 - **Urine urobilinogen high** ? hemolysis
 - **Urine urobilinogen absent** ? obstructive jaundice
-

? E. Test for Detoxification Function

? Ammonia

- Increased in **hepatic failure, hepatic encephalopathy**
-

? 2. SERUM BILIRUBIN

Bilirubin is produced from heme breakdown.

? Types

? 1. Unconjugated (Indirect) bilirubin

- Water-insoluble
- Albumin-bound
- Formed in spleen
- Increased in:
 - Hemolysis
 - Gilbert syndrome
 - Crigler-Najjar
 - Neonatal jaundice

? 2. Conjugated (Direct) bilirubin

- Water-soluble
- Conjugated by **UDP-glucuronyl transferase**
- Increased in:
 - Hepatocellular disease
 - Obstructive jaundice
 - Dubin-Johnson

- Rotor syndrome

? Normal Bilirubin Values

- **Total:** 0.3–1.2 mg/dL
- **Direct:** 0.1–0.3 mg/dL
- **Indirect:** subtract direct from total

? 3. CLASSIFICATION OF JAUNDICE

(Based on site of defect)

? A. Pre-Hepatic (Hemolytic Jaundice)

? Cause

- Hemolysis (G6PD deficiency, thalassemia, malaria)

? Biochemical Features

- ? Unconjugated bilirubin
- Normal direct bilirubin
- ? Urobilinogen
- No urine bilirubin

- Normal ALP/ALT

? Urine

- Urine bilirubin: **absent**
 - Urobilinogen: **increased**
-

? B. Hepatic (Hepatocellular Jaundice)

? Cause

- Viral hepatitis
- Alcoholic hepatitis
- Drug-induced liver injury
- Cirrhosis

? Biochemical Features

- ? Both unconjugated + conjugated bilirubin
- ? ALT >> AST or AST >> ALT
- Moderate ALP elevation
- ? Albumin (chronic)
- Prolonged PT (vitamin K deficiency)

? Urine

- Urine bilirubin: **present**
 - Urobilinogen: **variable**
-

? C. Post-Hepatic (Obstructive Jaundice)

? Causes

- Gallstones
- Carcinoma head of pancreas
- Biliary stricture
- Primary sclerosing cholangitis

? Biochemical Features

- ? Conjugated bilirubin
- **Very high ALP**
- High GGT
- Cholesterol ?
- Pale stools (no stercobilin)
- Dark urine (bilirubin present)

? Urine

- Urine bilirubin: **present**
- Urobilinogen: **absent**

? 4. BILE ACIDS AND BILE SALTS

? Bile Acids

Produced in liver from cholesterol.

Primary bile acids

- Cholic acid
- Chenodeoxycholic acid

Secondary bile acids

(Produced by gut bacteria)

- Deoxycholic acid
- Lithocholic acid

? Bile Salts

Bile acids conjugated with:

- **Glycine**

- **Taurine**

? form **bile salts** (better solubility, detergent action)

Examples:

- Glycocholate
 - Taurocholate
-

? Functions of Bile Acids & Bile Salts

? 1. Fat Digestion

- Emulsify dietary fats
- Increase surface area ? pancreatic lipase action

? 2. Micelle Formation

- Solubilize monoglycerides + fatty acids + fat-soluble vitamins

? 3. Elimination

- Route for excretion of **bilirubin, cholesterol, drugs**

? 4. Regulation

- Inhibit HMG-CoA reductase
 - Control bile acid synthesis by feedback
-

? High-Yield Summary

- ALT is most specific for liver injury.
- ALP + GGT rise sharply in obstruction.
- Conjugated bilirubin in urine = hepatocellular or obstructive jaundice.
- Hemolytic jaundice = ? indirect bilirubin + ? urobilinogen.
- Obstructive jaundice = ? direct bilirubin + no urobilinogen + pale stool.
- Bile salts are conjugated bile acids ? essential for fat digestion.

? TESTS BASED ON THE METABOLIC CAPACITY OF THE LIVER

These tests assess how well the liver performs **biotransformation, detoxification, and metabolic reactions**.

? 1. Galactose Tolerance Test

Principle

Liver converts **galactose ? glucose**.

If hepatic metabolism is impaired, conversion is delayed.

Interpretation

- **High blood galactose after oral load** ? impaired hepatic metabolism
- Seen in:

- Hepatocellular dysfunction
 - Cirrhosis
 - Acute hepatitis
-

? 2. Fructose Tolerance Test

Similar to galactose test.

Interpretation

- Delayed clearance of fructose ? reduced hepatic metabolic ability.

(Rarely used today.)

? 3. Bromosulphthalein (BSP) Excretion Test

A classic test (historical but high-yield).

Principle

- BSP dye is taken up by hepatocytes, conjugated, and excreted into bile.
- Measures **uptake + conjugation + excretion**.

Interpretation

- **High retention of BSP** at 45 minutes ? indicates hepatocellular dysfunction.

Seen in:

- Cirrhosis
- Chronic hepatitis
- Drug-induced liver injury

(Not used in modern practice but remains viva favorite.)

? 4. Serum Ammonia Levels

Principle

Liver converts ammonia → urea.

Failure of metabolism → → ammonia.

Increased In:

- Acute liver failure
 - Hepatic encephalopathy
 - Severe hepatocellular dysfunction
 - Portosystemic shunts
-

? 5. Aminopyrine Breath Test

Principle

Evaluates microsomal enzyme — **cytochrome P450** activity.

Interpretation

Low CO₂ excretion after aminopyrine load = impaired P450 activity.

(Advanced test, rarely used but conceptually important.)

? TESTS BASED ON SYNTHETIC FUNCTION OF THE LIVER

These tests measure **how well the liver synthesizes essential proteins, coagulation factors, and lipids.**

? 1. Serum Albumin

Significance

- Reflects chronic liver disease (long half-life ~21 days)
- Unchanged in acute hepatitis

Low Albumin Seen In:

- Cirrhosis
 - Chronic hepatitis
 - Nephrotic syndrome
 - Malnutrition
-

? 2. Prothrombin Time (PT/INR)

Principle

Liver synthesizes clotting factors II, VII, IX, X.

Prolonged PT Indicates:

- Acute liver failure
- Cholestasis (vitamin K malabsorption)
- Severe hepatocellular injury
- Vitamin K deficiency
- Warfarin therapy

Most Important Point

PT is the best indicator of acute liver synthetic function.

? 3. Serum Cholesterol

Interpretation

- **Low cholesterol:** chronic liver disease, advanced cirrhosis
 - **High cholesterol:** obstructive jaundice (impaired excretion)
-

? 4. Serum Pseudocholinesterase (PChE)

Significance

Synthesized in liver; levels correlate with synthetic capacity.

Decreased In:

- Chronic liver disease
- Cirrhosis
- Hepatocellular failure
- Malnutrition

Used clinically in:

- Organophosphate poisoning (to assess recovery)

? 5. Coagulation Factors

Liver produces:

- Fibrinogen
- Prothrombin
- Factors V, VII, IX, X

Factor V

Very specific marker:

? **Reduced only in liver disease**, NOT in vitamin K deficiency.

? High-Yield Summary (Perfect for Last-Minute Revision)

- **Galactose tolerance test, fructose clearance, BSP test** ? test metabolic detoxification.

- **Serum ammonia** rises in hepatic encephalopathy.
- **Albumin** reflects chronic liver disease.
- **PT/INR** is the most sensitive marker of **acute** liver synthetic failure.
- **Pseudocholinesterase** falls in chronic liver disease.
- **Low cholesterol** ? end-stage liver disease; **high cholesterol** ? obstructive jaundice.

? SERUM ENZYMES AS MARKERS OF HEPATOBILIARY DISEASES

Liver diseases are best interpreted by knowing which enzyme pattern corresponds to **hepatocellular damage**, **cholestasis**, or **infiltrative disease**.

? 1. Aminotransferases (ALT & AST)

? ALT (Alanine transaminase)

- More **specific for liver**.
- Very high in **acute viral hepatitis**.
- Moderately elevated in alcoholic liver disease, fatty liver.

? AST (Aspartate transaminase)

- Found in liver, muscle, heart, kidney.
- High in **alcoholic hepatitis** (AST:ALT > 2).

- Also high in muscle injury (important for differential).

? AST/ALT Ratio

- 2 ? **Alcoholic hepatitis**
 - <1 ? **Viral hepatitis**
-

? 2. Alkaline Phosphatase (ALP)

? Marker of cholestasis

- Very high in **obstructive jaundice**.
 - Also high in bone disease ? confirm with GGT.
-

? 3. ?-Glutamyl Transferase (GGT)

? Marker of biliary obstruction & alcohol use

- High in:
 - Obstruction
 - Alcoholic liver disease
 - Enzyme induction (drugs: phenytoin)

Used to differentiate **hepatic ALP** from **bone ALP**.

? 4. 5'-Nucleotidase

- Specific for **cholestasis**
- Increases parallel to ALP but not elevated in bone disorders.

? 5. Lactate Dehydrogenase (LDH)

- **LDH-5** rises in hepatic necrosis.
- LDH-1 > LDH-2 is seen in MI (not liver).

? 6. Glutamate Dehydrogenase (GLDH)

- Mitochondrial enzyme
- Elevated in **severe hepatocellular injury**, especially alcoholic hepatitis.

? 7. Pseudocholinesterase (PChE)

- Synthesized in liver
- Low in **chronic liver disease**, malnutrition, cirrhosis.

? Interpretation Pattern (Very High Yield)

PATTERN	LIKELY DIAGNOSIS
ALT, AST very high (>1000)	Acute viral hepatitis, ischemic injury
ALP + GGT very high	Obstructive jaundice
AST > ALT (ratio >2)	Alcoholic hepatitis
Low albumin + prolonged PT	Chronic liver failure
High bilirubin + high ALP	Cholestasis
High unconjugated bilirubin	Hemolysis

? GASTRIC FUNCTION TESTS

These tests evaluate **acid secretion**, **parietal cell function**, and **gastrin levels**.

? 1. Basal Acid Output (BAO)

? Method

- Measure gastric acid secretion in fasting state
- Aspirate gastric contents over **1 hour** using Ryle's tube
- Expressed as mEq/hour

? Normal

- 1–5 mEq/hour

? Increased In:

- Zollinger–Ellison syndrome
- Gastrinoma
- Hyperparathyroidism

? Decreased/Absent In:

- Achlorhydria
- Pernicious anemia
- Chronic atrophic gastritis

? 2. Maximal Acid Output (MAO)

? Method

- Measure acid output after **pentagastrin** stimulation.
- Represents maximal parietal cell capacity.

? Interpretation

- **MAO very high** ? gastrinoma
- **MAO low** ? pernicious anemia, gastric atrophy

? 3. BAO/MAO Ratio

? High ratio (>0.6) indicates:

- Gastrinoma

? Low ratio in:

- Normal individuals
- Pernicious anemia

? 4. Tubeless Gastric Analysis (Sensitive marker)

? Principle

Use **Hollander test**, **diagnostic caffeine test**, or **dye recovery test** to determine presence of acid **without gastric intubation**.

? Uses

- Screening for **achlorhydria**
- Monitoring **pernicious anemia** patients

(Less commonly used today.)

? 5. Serum Gastrin Levels

? High Gastrin Seen In:

- Zollinger–Ellison syndrome (gastrinoma)
- Atrophic gastritis
- Pernicious anemia

- PPI therapy

? Low Gastrin:

- Acid hypersecretion due to feedback inhibition

? 6. Pentagastrin Stimulation Test

? Purpose

Tests **parietal cell function** and **HCl secretion**.

? Uses

- Diagnosis of **achlorhydria** vs **hypochlorhydria**
- Distinguish **pernicious anemia** (no response) from **gastritis** (partial response)

? HYDROCHLORIC ACID SECRETION

HCl secretion is tested to evaluate **parietal cell activity** and conditions altering acid levels.

? 1. Tests for HCl Secretion

? 1. Basal Acid Output (BAO)

Measures fasting acid secretion.

? 2. Maximal Acid Output (MAO)

After pentagastrin stimulation.

? 3. Peak Acid Output (PAO)

Highest amount secreted after stimulation.

? 4. Titration of Gastric Juice

Measures pH using titration against NaOH.

? 2. Causes of Increased HCl Secretion

- Zollinger–Ellison syndrome (gastrinoma)
 - Duodenal ulcers
 - Hyperparathyroidism
 - Massive vagal stimulation
 - G cell hyperfunction
-

? 3. Causes of Decreased/Absent HCl Secretion

- **Achlorhydria**
 - Pernicious anemia
 - Chronic atrophic gastritis
 - Gastric carcinoma
 - Vagotomy
 - PPI therapy (omeprazole)
-

? 4. Pernicious Anemia Acid Pattern

- **BAO = 0**
- **MAO = 0**
- Serum gastrin **very high**
- Parietal cell antibodies present

? SUPER-HIGH-YIELD PEARLS

- ALP and GGT together = **best markers of cholestasis**.
- AST:ALT >2 ? **alcoholic liver disease**.
- ALT > AST ? **viral hepatitis**.
- Absence of HCl = **achlorhydria** (pernicious anemia).
- High BAO + high MAO + very high gastrin = **gastrinoma**.
- Normal BAO + low MAO = **parietal cell loss** (atrophic gastritis).

? ASSESSMENT OF FREE & TOTAL ACIDITY

Gastric acidity is measured from **gastric juice obtained via Ryle's tube** after fasting.

? 1. FREE ACIDITY

? Definition

Amount of **free HCl** present in gastric juice (not bound to proteins).

? Method

- Gastric juice is titrated with **0.1 N NaOH**
- Indicator: **Topfer's reagent**
 - Changes color when free HCl is neutralized

? Normal Values

- **Free acidity:** 20–40 mEq/L

? Clinical Importance

- **Increased in:**
 - Zollinger–Ellison syndrome
 - Duodenal ulcer
 - G-cell hyperfunction
 - Hyperparathyroidism
 - **Decreased/Absent in:**
 - Pernicious anemia (achlorhydria)
 - Chronic atrophic gastritis
 - Gastric carcinoma
 - Long-term PPI therapy
-

? 2. TOTAL ACIDITY

? Definition

Sum of:

- Free HCl
- Combined HCl
- Other acids (organic acids)

? Method

- Continue titration beyond Topfer endpoint
- Use **phenolphthalein indicator**
- Neutralizes total acid content

? Normal Values

- **Total acidity:** 40–70 mEq/L

? Clinical Importance

- Increased in acid hypersecretion states
- Decreased in achlorhydria

? 3. Achlorhydria vs Hypochlorhydria

CONDITION	BAO	MAO	INTERPRETATION
Achlorhydria	0	0	No acid even after stimulation
Hypochlorhydria	Low	Moderate	Reduced, but responds to stimulation

Seen in:

- Pernicious anemia (complete achlorhydria)
- Gastric atrophy
- Gastric carcinoma

? 4. Clinical Applications of Gastric Acidity Tests

- Diagnosis of **achlorhydria**
- Evaluation of **gastrinoma** (high BAO/MAO)
- Differentiating pernicious anemia vs chronic gastritis
- Monitoring post-vagotomy or PPI therapy

? PANCREATIC FUNCTION TESTS

Pancreatic tests assess **enzyme secretion**, **bicarbonate secretion**, and **exocrine function**.

? 1. DIRECT PANCREATIC FUNCTION TESTS

(Most accurate but invasive)

? A. Secretin–Pancreozymin Test

Gold standard for exocrine pancreatic function.

? Procedure

- Secretin ? stimulates **bicarbonate** secretion
- CCK/pancreozymin ? stimulates **enzyme** secretion
- Duodenal contents aspirated & measured

? Interpretation

- **Low bicarbonate + low enzymes** ? chronic pancreatitis
 - **Normal** values ? excludes major pancreatic disease
-

? B. Lundh Meal Test

Stimulates pancreas by giving a standard meal.

? Interpretation

- Low trypsin/chymotrypsin in aspirate ? pancreatic insufficiency
-

? 2. INDIRECT PANCREATIC FUNCTION TESTS

(Non-invasive, used clinically)

? A. Serum Amylase

? Increased In:

- Acute pancreatitis
- Pancreatic duct obstruction
- Perforated ulcer
- Salivary gland disease

? Limitations

- Returns to normal within 2–3 days
 - Not specific for pancreas
-

? B. Serum Lipase

? Better than amylase

- More specific for pancreas
- Remains elevated longer (7–10 days)

? Markedly elevated in:

- Acute pancreatitis
- Pancreatic necrosis

? C. Fecal Fat Estimation (Stool Fat Test)

Detects **fat malabsorption** due to lack of pancreatic enzymes.

? Features

- 7 g/day of fat in stool indicates steatorrhea
- Seen in:
 - Chronic pancreatitis
 - Cystic fibrosis
 - Pancreatic carcinoma

? D. Fecal Elastase-1 (Most useful modern test)

? Interpretation

- 200 µg/g stool ? normal
- 100–200 ? mild insufficiency
- <100 ? severe exocrine insufficiency

Used widely to diagnose:

- Chronic pancreatitis

- Cystic fibrosis
-

? E. Serum Trypsinogen

? Low in:

- Severe chronic pancreatitis
 - Cystic fibrosis
-

? F. 72-Hour Fecal Fat Quantitative Test

? Gold standard for steatorrhea

- Patient consumes 100 g fat/day
 - 7 g/day fat in stool ? steatorrhea
 - Seen in severe pancreatic insufficiency
-

? 3. Functional Imaging Tests

? A. CT Abdomen

Detects:

- Pancreatic calcifications (chronic pancreatitis)
 - Necrosis
-

- Mass lesions

? B. MRCP

Evaluates pancreatic ducts.

? C. Endoscopic Ultrasound (EUS)

Most sensitive for early chronic pancreatitis.

? 4. Summary Table (Ultra-High Yield)

TEST	MEASURES	INCREASED IN	DECREASED IN
Serum amylase	Amylase	Acute pancreatitis	—
Serum lipase	Lipase	Acute pancreatitis	—
Fecal elastase	Pancreatic enzymes	—	Chronic pancreatitis
Serum trypsinogen	Exocrine function	Acute pancreatitis	Chronic pancreatitis
Secretin test	Bicarbonate output	—	Pancreatic insufficiency
Stool fat test	Fat malabsorption	Steatorrhea	—

? High-Yield PEARLS

- Lipase > amylase for diagnosing acute pancreatitis.
- Fecal elastase-1 is the **best non-invasive** test for chronic pancreatitis.
- Secretin-Pancreozymin test is the **gold standard** for exocrine pancreatic function.

- Achlorhydria shows **zero free and total acidity**.
- Gastrinoma ? **very high BAO & MAO** + high gastrin.

? IMPORTANT POINTS TO REMEMBER (Whole Chapter)

? LIVER FUNCTION TESTS (LFTs)

? 1. ALT is more specific for liver injury than AST.

ALT predominantly comes from hepatocytes; AST is also from muscle and heart.

? 2. AST:ALT > 2 strongly suggests alcoholic hepatitis.

? 3. ALP and GGT are the best indicators of cholestasis.

- ALP ? (also in bone disease)
- GGT ? (confirm hepatic origin; also ? in alcohol intake)

? 4. Prothrombin Time (PT/INR) is the most sensitive indicator of acute liver failure.

? 5. Albumin reflects chronic liver disease, not acute hepatitis.

? 6. Unconjugated bilirubin ? in hemolysis; conjugated bilirubin ? in cholestasis.

? 7. Absence of urine urobilinogen ? obstructive jaundice.

? 8. Urine bilirubin is always conjugated bilirubin.

? 9. Serum ammonia increases in hepatic encephalopathy.

? JAUNDICE CLASSIFICATION

? 10. Hemolytic (pre-hepatic) jaundice — ? indirect bilirubin, ? urobilinogen, no urine bilirubin.

? 11. Hepatocellular jaundice — mixed direct and indirect bilirubin elevation.

? 12. Obstructive jaundice — very high direct bilirubin, very high ALP/GGT, pale stools, dark urine.

? 13. In Dubin–Johnson and Rotor syndrome:

- Direct bilirubin ?
- Liver function otherwise normal

? BILE ACIDS & BILE SALTS

? 14. Bile acids are conjugated with glycine/taurine ? bile salts.

? 15. Bile salts are essential for fat digestion & micelle formation.

? 16. Primary bile acids ? cholic & chenodeoxycholic acid.

? 17. Secondary bile acids are formed by intestinal bacteria.

? GASTRIC FUNCTION & ACIDITY

? 18. Gastrinoma shows very high BAO and MAO with high serum gastrin.

? 19. Pernicious anemia shows complete achlorhydria (BAO = 0, MAO = 0).

? 20. Free acidity measures only free HCl; total acidity measures free + bound acid.

? 21. Pentagastrin stimulation test assesses parietal cell function.

? 22. Achlorhydria is strongly associated with autoimmune atrophic gastritis.

? PANCREATIC FUNCTION TESTS

? 23. Serum lipase is more specific and remains elevated longer than amylase.

? 24. Acute pancreatitis: amylase & lipase increase sharply.

? 25. Chronic pancreatitis:

- Amylase often normal
- Fecal elastase ?
- Fat malabsorption ?

? 26. Fecal elastase-1 is the best non-invasive test for pancreatic insufficiency.

? 27. Secretin–pancreozymin test is the gold standard for exocrine pancreatic function.

? 28. Stool fat > 7 g/day indicates steatorrhea.

? 29. Low serum trypsinogen indicates severe chronic pancreatitis or cystic fibrosis.

? ILLUSTRATIVE PATTERNS (Ultra High Yield)

? 30. Viral hepatitis pattern

- ALT ??
- AST ??
- Bilirubin ?
- ALP mild ?

? 31. Alcoholic hepatitis

- AST > ALT (ratio > 2)

- GGT ?

? 32. Obstructive jaundice

- ALP ??
- GGT ??
- Direct bilirubin ?
- Pale stool

? 33. Hemolysis

- Indirect bilirubin ?
- Urine bilirubin absent
- Urobilinogen ?

? 34. Chronic liver disease

- Albumin ?
- PT ?
- Cholesterol ?

? MOST EXAM-FAVORITE CONCEPTS

? 35. PT/INR is the earliest deranged test in acute liver failure.

? 36. Only conjugated bilirubin appears in urine.

? 37. Pale stools = no stercobilin ? obstruction.

? 38. Gastrin very high + high BAO = gastrinoma.

? 39. Lipase is more specific than amylase for pancreatitis.

? 40. Fecal elastase < 100 µg/g = severe exocrine pancreatic insufficiency.

? MCQs — Whole Chapter

1. Which enzyme is MOST specific for liver injury?

- A. AST
- B. ALT
- C. ALP
- D. GGT

Answer: B. ALT

2. AST:ALT ratio > 2 suggests:

- A. Viral hepatitis
- B. Alcoholic hepatitis
- C. Obstructive jaundice
- D. Cirrhosis only

Answer: B. Alcoholic hepatitis

3. The BEST marker of cholestasis is:

- A. AST
- B. ALT
- C. ALP + GGT
- D. LDH

Answer: C. ALP + GGT

4. Which LFT is most sensitive for acute liver failure?

- A. Albumin
- B. Serum bilirubin
- C. PT/INR
- D. ALP

Answer: C. PT/INR

5. Serum albumin reflects:

- A. Acute hepatocellular injury
- B. Chronic liver disease
- C. Degree of cholestasis
- D. Gallstone obstruction

Answer: B. Chronic liver disease

6. Unconjugated hyperbilirubinemia is characteristic of:

- A. Obstruction
- B. Hemolysis
- C. Dubin–Johnson syndrome
- D. Rotor syndrome

Answer: B. Hemolysis

7. Conjugated bilirubin is increased in:

- A. Hemolysis
- B. Pernicious anemia
- C. Obstructive jaundice
- D. Gilbert syndrome

Answer: C. Obstructive jaundice

8. Urine bilirubin is present in:

- A. Hemolysis
- B. Gilbert syndrome
- C. Obstructive jaundice
- D. No liver disease

Answer: C. Obstructive jaundice

9. Urobilinogen is absent in:

- A. Viral hepatitis
- B. Hemolysis
- C. Obstructive jaundice
- D. Gilbert syndrome

Answer: C. Obstructive jaundice

10. Pale clay-colored stools indicate:

- A. Hemolysis
- B. Hepatic jaundice
- C. Obstruction of bile ducts
- D. Dubin–Johnson syndrome

Answer: C. Obstruction

11. Primary bile acids include:

- A. Cholic acid and chenodeoxycholic acid
- B. Lithocholic and deoxycholic acid
- C. Taurocholate only
- D. Glycocholate only

Answer: A

12. Bile salts are formed by conjugation with:

- A. Serine
- B. Taurine and glycine
- C. Tryptophan
- D. Alanine

Answer: B

13. A hallmark of Dubin–Johnson syndrome is:

- A. Increased unconjugated bilirubin
- B. Black liver pigmentation
- C. Absent bile salts
- D. Increased urobilinogen

Answer: B

14. Best test for exocrine pancreatic insufficiency:

- A. Serum lipase
- B. Serum amylase
- C. Fecal elastase-1
- D. Serum trypsinogen

Answer: C. Fecal elastase-1

15. Which enzyme remains elevated longest in acute pancreatitis?

- A. AST
- B. Amylase
- C. Lipase
- D. LDH

Answer: C. Lipase

16. 72-hour stool fat estimation is used to diagnose:

- A. Gastrinoma
- B. Steatorrhea
- C. Achlorhydria
- D. Bile acid malabsorption

Answer: B. Steatorrhea

17. The gold standard for pancreatic function is:

- A. Secretin–pancreozymin test
- B. Serum lipase
- C. Fecal fat test
- D. Serum amylase

Answer: A

18. Achlorhydria is seen in:

- A. Zollinger–Ellison syndrome
- B. Pernicious anemia
- C. Duodenal ulcer
- D. Hyperparathyroidism

Answer: B. Pernicious anemia

19. Zollinger–Ellison syndrome is associated with:

- A. Low BAO
- B. High BAO and MAO
- C. Absent BAO
- D. No change in acid output

Answer: B

20. Free acidity is measured using:

- A. Phenolphthalein
- B. Topfer's reagent
- C. Methyl red
- D. Congo red

Answer: B

21. Total acidity is measured using:

- A. Topfer's reagent
- B. Bromothymol blue
- C. Phenolphthalein
- D. Ninhydrin

Answer: C

22. Which test evaluates parietal cell function?

- A. Hollander test
- B. Secretin test
- C. Pentagastrin stimulation test
- D. Glucose tolerance test

Answer: C

23. A normal adult stores bile acids primarily in:

- A. Liver
- B. Stomach
- C. Gallbladder
- D. Colon

Answer: C

24. Which enzyme is decreased in chronic liver disease?

- A. GGT
- B. Lipase
- C. Pseudocholinesterase
- D. ALP

Answer: C

25. Serum trypsinogen is LOW in:

- A. Acute pancreatitis
- B. Chronic pancreatitis
- C. Obstructive jaundice
- D. Hemolysis

Answer: B

26. Which parameter differentiates bone ALP from liver ALP?

- A. ALT
- B. AST
- C. GGT
- D. LDH

Answer: C

27. Hypergastrinemia with low acid output indicates:

- A. Gastrinoma
- B. Pernicious anemia
- C. Duodenal ulcer
- D. Chronic pancreatitis

Answer: B

28. A patient with high indirect bilirubin, high reticulocyte count, and no urine bilirubin likely has:

- A. Hepatic jaundice
- B. Obstructive jaundice
- C. Hemolytic jaundice
- D. Dubin–Johnson syndrome

Answer: C

29. Secretin stimulates secretion of:

- A. Pancreatic enzymes
- B. Bicarbonate-rich pancreatic juice
- C. Bile salts
- D. Gastric HCl

Answer: B

30. Which test is MOST useful for early chronic pancreatitis?

- A. Serum amylase
- B. Abdominal X-ray
- C. Endoscopic ultrasound (EUS)
- D. Serum bilirubin

Answer: C

? CLINICAL CASE–BASED QUESTIONS (Whole Chapter)

? 1. A 24-year-old male presents with yellow eyes and dark urine. Labs show:

- Total bilirubin: 9 mg/dL

- Direct bilirubin: 6 mg/dL
- ALP: ??
- GGT: ??
- Urine bilirubin: Present
- Stool: Pale

Most likely diagnosis?

Answer: Obstructive jaundice

(Conjugated hyperbilirubinemia + high ALP/GGT + dark urine + pale stools)

? 2. A patient with chronic alcoholism shows AST 180, ALT 70. AST/ALT ratio > 2.

What is the diagnosis?

Answer: Alcoholic hepatitis

(AST > ALT, ratio > 2 with GGT elevation)

? 3. A neonate has persistent jaundice. Labs show:

- Total bilirubin: 15 mg/dL
- Indirect bilirubin: 14 mg/dL
- No bilirubin in urine

What type of jaundice?

Answer: Unconjugated hyperbilirubinemia (likely physiologic or hemolysis)

? 4. A 45-year-old woman has pruritus, pale stools, high ALP, high GGT, and high cholesterol.

What is the most probable cause?

Answer: Extrahepatic biliary obstruction

(Cholestasis + fat malabsorption + pruritus + high ALP/GGT)

? 5. A patient has dark brown/black liver on biopsy, direct hyperbilirubinemia, normal enzymes.

Diagnosis?

Answer: Dubin–Johnson syndrome

? 6. A man presents with fatigue. Labs:

- Albumin ?
- PT prolonged
- AST/ALT mildly raised
- Cholesterol ?

What does this indicate?

Answer: Chronic liver disease (cirrhosis)

(Low synthetic function + low cholesterol)

? 7. A 35-year-old woman has severe epigastric pain radiating to the back. Labs show:

- Serum amylase: 1200 IU/L
- Serum lipase: 2600 IU/L
- Hypocalcemia

Diagnosis?

Answer: Acute pancreatitis

(Lipase > amylase + typical clinical picture)

? 8. A patient with chronic diarrhea has:

- Fecal fat: 20 g/day
- Fecal elastase-1: 70 µg/g stool
- Serum trypsinogen: Low

Cause?

Answer: Severe exocrine pancreatic insufficiency (chronic pancreatitis)

? 9. A 52-year-old man has fasting gastric juice with pH < 2 and extremely high BAO and MAO.

Serum gastrin is markedly elevated.

Diagnosis?

Answer: Zollinger–Ellison syndrome (gastrinoma)

? 10. A woman has anemia, glossitis, neuropathy. Gastric analysis: BAO = 0, MAO = 0.

Serum gastrin is very high.

Diagnosis?

Answer: Pernicious anemia (achlorhydria + high gastrin)

? 11. A patient with gallstones develops:

- Direct bilirubin ?
- ALP ??
- GGT ??
- Urobilinogen absent

What is the biochemical explanation?

Answer: Conjugated bilirubin cannot reach the intestine ? urobilinogen not formed.

? 12. A young man with hemolytic anemia shows:

- Indirect bilirubin ?
- Urine bilirubin absent
- Urine urobilinogen ??

Type of jaundice?

Answer: Pre-hepatic (hemolytic) jaundice

? 13. A 10-year-old child with cystic fibrosis has foul-smelling stools floating on water.

Stool fat: 15 g/day.

Fecal elastase: 80 µg/g.

Diagnosis?

Answer: Severe exocrine pancreatic insufficiency due to cystic fibrosis

? 14. After starting high-dose PPIs, a patient shows:

- Gastric pH > 4
- BAO ?
- Serum gastrin ?

What does this indicate?

Answer: PPI-induced hypochlorhydria ? compensatory hypergastrinemia

? 15. A patient presents with jaundice. Labs:

- ALT ??
- AST ??
- Direct bilirubin moderately ?
- Indirect bilirubin ?
- ALP normal

Diagnosis?

Answer: Hepatocellular jaundice (likely viral hepatitis)

? 16. A patient with suspected liver failure shows:

- PT prolonged
- Albumin low
- Bilirubin high
- ALT moderately raised

Which is the best indicator of severity?

Answer: PT prolongation

? 17. A 30-year-old presents with pruritus, high ALP, but normal AST/ALT.

Serum bilirubin is slightly increased.

What is the next best test?

Answer: GGT (to confirm cholestatic origin of raised ALP)

? 18. A patient has the following gastric findings:

- Free acidity: 0
- Total acidity: 0
- After pentagastrin: still 0
- Serum intrinsic factor antibodies present

Diagnosis?

Answer: Autoimmune atrophic gastritis ? pernicious anemia

? 19. A man with chronic alcoholism shows:

- Low serum amylase
- Low serum lipase
- Fatty stools

- Fecal elastase 90 µg/g

Diagnosis?

Answer: Chronic pancreatitis (burnt-out pancreas)

? 20. A patient with cholestasis has fat malabsorption. Which bile component is responsible?

Answer: Loss of bile salts ? impaired micelle formation ? fat malabsorption

? VIVA VOCE — Whole Chapter (LFT + Gastric + Pancreatic Tests)

? LIVER FUNCTION TESTS

1. Which enzyme is most specific for liver injury?

ALT

2. What does AST:ALT > 2 indicate?

Alcoholic hepatitis

3. What does a very high ALT (>1000 IU/L) indicate?

Acute viral hepatitis or ischemic injury

4. Which enzymes indicate cholestasis?

ALP and GGT

5. Why does GGT help interpret ALP rise?

Because GGT rises only in liver disease, not in bone disorders.

6. Which LFT reflects chronic liver disease?

Serum albumin (long half-life)

7. Which test reflects acute synthetic liver failure?

PT/INR

8. Which bilirubin appears in urine?

Conjugated bilirubin only

9. What causes unconjugated hyperbilirubinemia?

Hemolysis, Gilbert syndrome, Crigler–Najjar

10. What causes conjugated hyperbilirubinemia?

Obstruction, hepatocellular disease, Dubin–Johnson

? JAUNDICE

11. What is the hallmark of hemolytic jaundice?

High indirect bilirubin + high urobilinogen + no urine bilirubin

12. What is the hallmark of obstructive jaundice?

High direct bilirubin + high ALP/GGT + pale stool + dark urine

13. Why is stool pale in obstruction?

Lack of stercobilinogen.

14. Why is urine urobilinogen absent in obstruction?

Conjugated bilirubin cannot reach the intestine.

15. What is unique about Dubin–Johnson syndrome?

Black liver due to pigment accumulation.

16. What is Rotor syndrome?

Conjugated hyperbilirubinemia with normal liver histology

? BILE ACIDS & BILE SALTS

17. Primary bile acids?

Cholic and chenodeoxycholic acid

18. Secondary bile acids?

Deoxycholic and lithocholic acid

19. Bile salts are conjugated with?

Glycine or taurine

20. Main function of bile salts?

Fat emulsification and micelle formation

? GASTRIC FUNCTION TESTS

21. What does BAO measure?

Basal acid output (fasting HCl secretion)

22. When is BAO increased?

Zollinger–Ellison syndrome

23. What does MAO measure?

Maximum acid output after stimulation (usually pentagastrin)

24. What is achlorhydria?

Absence of free HCl.

25. Achlorhydria is seen in?

Pernicious anemia, atrophic gastritis

26. What is the BAO/MAO ratio in gastrinoma?

Usually >0.6

27. What does the pentagastrin test evaluate?

Parietal cell function and HCl secretion

? HYDROCHLORIC ACID SECRETION

28. Indicator used to detect free acidity?

Topfer's reagent

29. Indicator for total acidity?

Phenolphthalein

30. Normal free acidity range?

20–40 mEq/L

31. Normal total acidity range?

40–70 mEq/L

32. Conditions with increased HCl secretion?

Peptic ulcer disease, gastrinoma

? PANCREATIC FUNCTION TESTS

33. Which enzyme is most specific for pancreatitis?

Lipase

34. Which enzyme rises earlier in pancreatitis?

Amylase

35. Which enzyme stays elevated longer?

Lipase (7–10 days)

36. What is the best non-invasive test for exocrine pancreatic insufficiency?

Fecal elastase-1

37. What is the gold standard test for pancreatic exocrine function?

Secretin–pancreozymin test

38. Stool fat >7 g/day indicates?

Steatorrhea

39. Causes of pancreatic steatorrhea?

Chronic pancreatitis, cystic fibrosis, pancreatic cancer

40. Low serum trypsinogen occurs in?

Chronic pancreatitis, cystic fibrosis

? INTEGRATED CLINICAL VIVA QUESTIONS

41. Why is PT prolonged in cholestasis?

Vitamin K absorption ? ? ? clotting factors.

42. Why is ALP high in obstructive jaundice?

Backpressure in bile canaliculi ? increased ALP synthesis.

43. Why does unconjugated bilirubin not appear in urine?

It is bound to albumin and water-insoluble.

44. What causes dark urine in obstructive jaundice?

Excess conjugated bilirubin excreted in urine.

45. Why do patients with obstruction develop pruritus?

Retention of bile salts in blood.

46. Why is gastrin high in pernicious anemia?

Loss of acid ? **loss of negative feedback** ? gastrin ?.

47. What does high BAO + low MAO indicate?

Gastric outlet obstruction or retained acid.

48. Why do patients with chronic pancreatitis have normal amylase?

Pancreatic tissue is **burnt out**, unable to release enzymes.

? SUPER HIGH-YIELD LAST-MINUTE PEARLS

49. PT/INR is the best marker of acute liver synthetic function.

50. Fecal elastase is the best screening test for pancreatic insufficiency.

51. Only conjugated bilirubin appears in urine.

52. Pale stool = obstructive jaundice until proven otherwise.

53. Gastrinoma = high BAO + high MAO + high gastrin.

54. Lipase > amylase for diagnosis of pancreatitis.