MEDICAL EDUCATION RESOURCE

# 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**

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## ? 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)

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## ? 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

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## ? E. Test for Detoxification Function

### ? Ammonia

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

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## ? 2. SERUM BILIRUBIN

Bilirubin is produced from heme breakdown.

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## ? 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

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### ? Normal Bilirubin Values

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

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## ? 3. CLASSIFICATION OF JAUNDICE

(Based on site of defect)

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### ? 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**

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### ? 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**

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## ? 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**

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## ? 4. BILE ACIDS AND BILE SALTS

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### ? 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

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### ? Bile Salts

Bile acids conjugated with:

- **Glycine**

- **Taurine**

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

Examples:

- Glycocholate
- Taurocholate

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## ? 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

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## ? 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**.

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### ? 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

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### ? 2. Fructose Tolerance Test

Similar to galactose test.

**Interpretation**

- Delayed clearance of fructose ? reduced hepatic metabolic ability.

*(Rarely used today.)*

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### ? 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.)*

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### ? 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

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### ? 5. Aminopyrine Breath Test

**Principle**

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

**Interpretation**

Low CO? excretion after aminopyrine load = impaired P450 activity.

*(Advanced test, rarely used but conceptually important.)*

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## ? TESTS BASED ON SYNTHETIC FUNCTION OF THE LIVER

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

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### ? 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

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### ? 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.**

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### ? 3. Serum Cholesterol

**Interpretation**

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

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### ? 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)

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### ? 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.

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## ? 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**.

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### ? 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**

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### ? 2. Alkaline Phosphatase (ALP)

**? Marker of cholestasis**

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

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### ? 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.

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### ? 5. Lactate Dehydrogenase (LDH)

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

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### ? 6. Glutamate Dehydrogenase (GLDH)

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

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### ? 7. Pseudocholinesterase (PChE)

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

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## ? 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

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## ? 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

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## ? 3. BAO/MAO Ratio

**? High ratio (>0.6) indicates:**

- Gastrinoma

**? Low ratio in:**

- Normal individuals
- Pernicious anemia

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## ? 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.)*

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## ? 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

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## ? 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)

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## ? HYDROCHLORIC ACID SECRETION

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

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## ? 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

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## ? 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

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### ? B. Lundh Meal Test

Stimulates pancreas by giving a standard meal.

**? Interpretation**

- Low trypsin/chymotrypsin in aspirate ? pancreatic insufficiency

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## ? 2. INDIRECT PANCREATIC FUNCTION TESTS

(Non-invasive, used clinically)

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## ? 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

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## ? 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

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## ? 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

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### ? E. Serum Trypsinogen

**? Low in:**

- Severe chronic pancreatitis
- Cystic fibrosis

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### ? 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

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## ? 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.**

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## ? 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

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**1. Which enzyme is MOST specific for liver injury?**

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

**Answer: B. ALT**

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**2. AST:ALT ratio > 2 suggests:**

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

**Answer: B. Alcoholic hepatitis**

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**3. The BEST marker of cholestasis is:**

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

**Answer: C. ALP + GGT**

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

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

**Answer: C. PT/INR**

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**5. Serum albumin reflects:**

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

**Answer: B. Chronic liver disease**

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**6. Unconjugated hyperbilirubinemia is characteristic of:**

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

**Answer: B. Hemolysis**

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**7. Conjugated bilirubin is increased in:**

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

**Answer: C. Obstructive jaundice**

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**8. Urine bilirubin is present in:**

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

**Answer: C. Obstructive jaundice**

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**9. Urobilinogen is absent in:**

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

**Answer: C. Obstructive jaundice**

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**10. Pale clay-colored stools indicate:**

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

**Answer: C. Obstruction**

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**11. Primary bile acids include:**

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

**Answer: A**

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**12. Bile salts are formed by conjugation with:**

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

**Answer: B**

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**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**

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**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**

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**15. Which enzyme remains elevated longest in acute pancreatitis?**

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

**Answer: C. Lipase**

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

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

**Answer: B. Steatorrhea**

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**17. The gold standard for pancreatic function is:**

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

**Answer: A**

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**18. Achlorhydria is seen in:**

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

**Answer: B. Pernicious anemia**

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**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**

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**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)

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### ? 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.**