# Hepatobiliary disease

# Hepatology Davidson's principles and practice of medicine

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### Learning outcomes

LO1:Anatomy of Hepatobiliary system

LO2:Metabolism of bilirubin

LO3:Pathophysiology of jaundice

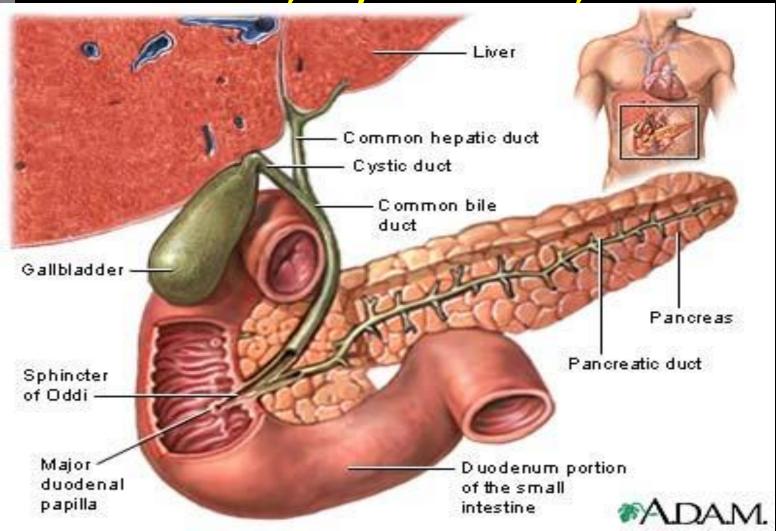
LO4: Mechanism of jaundice

LO5: Aetiology of jaundice

LO6:Laboratory investigation of hepatobiliary

diseases

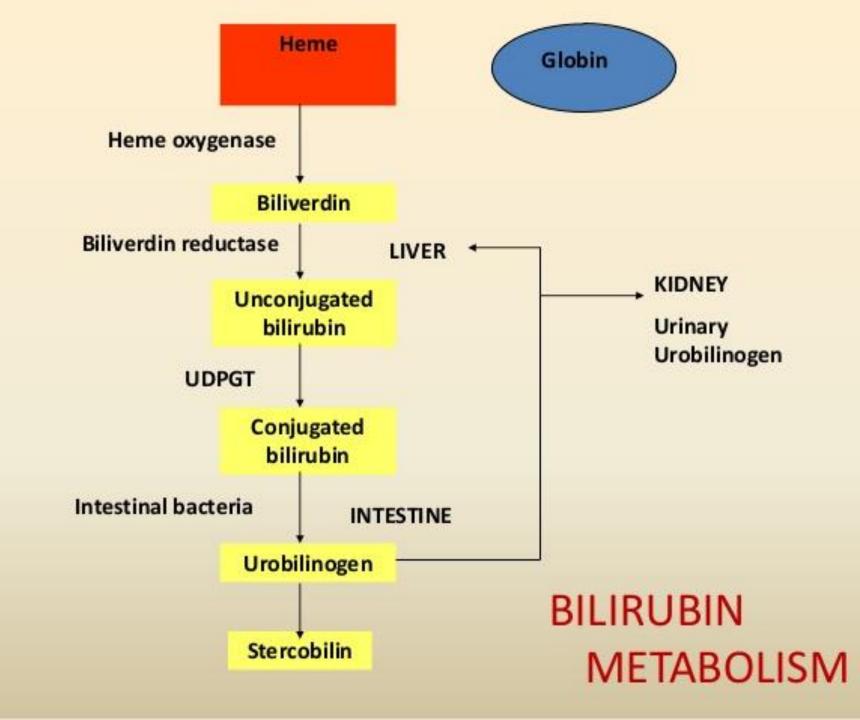
# The anatomy of Biliary tract



#### *L02*

### Bilirubin metabolism

- Major metabolite of heme
- Heme is found in hemoglobin, myoglobin and cytochrome.
- Most of daily production (0.2 to 0.3g/dL) is derived from breakdown of senescent erythrocytes
- Rate of systemic bilirubin production is equal to the rates of hepatic uptakes, conjugation, and biliary excretion.



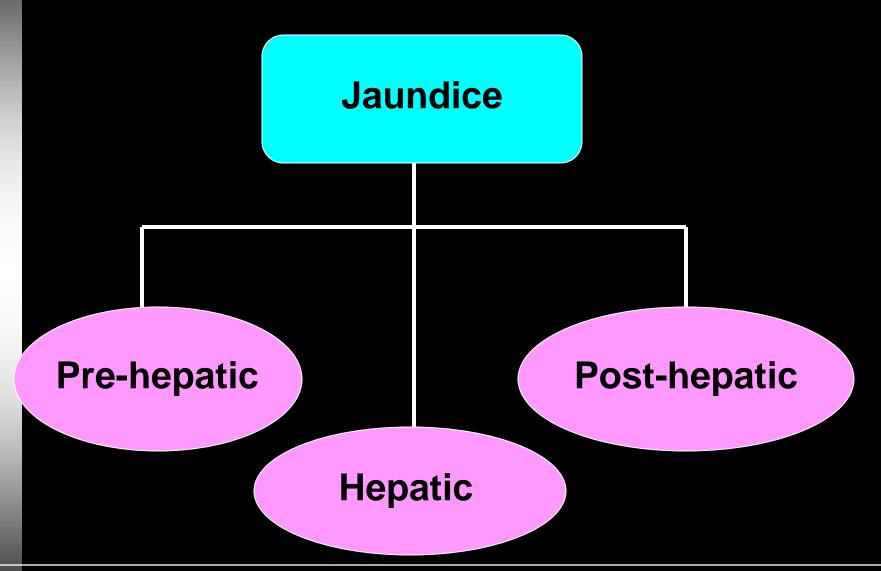
### LO3:Pathophysiology of jaundice

- Disturbance in bilirubin production or clearance.
- It is characterized by yellow color of white of the eyes (sclera) and skin
- Serum bilirubin levels rise above 2.0 to 2.5 mg/dL; level as high as 30-40mg/dL can occur with severe disease
- † also called as hyperbilrubinaemia.

## LO4: Mechanism of jaundice

- Excessive production of bilirubin
- Reduced hepatic uptake
- Impaired conjugation
- Decreased hepato-cellular excretion
- Impaired bile flow (both intrahepatic and extrahepatic)

# LO5: Aetiology of jaundice



### LO5:Pre-hepatic jaundice

- Excessive production of bilirubin due to excessive destruction of red blood cells.
- This results in overproduction of bilirubin beyond the capacity of the liver to conjugate and excrete bilirubin.

# LO5:Hepatic jaundice

- Defective hepatic uptake
- Abnormal conjugation
- Hepatocellular damage

### LO5:Defective hepatic uptake

- Unconjugated bilirubin in the plasma is carried into the liver by intracellular transport proteins.
- Absences of these proteins result in failure of bilirubin uptake, leading to unconjugated hyperbilrubinaemia (e.g. Gilbert Syndrome).
- Defective of blood supply to the liver also can cause abnormality of bilirubin metabolism
- These problems happen in congestive heart failure, pathway shunt due to surgery or congenital and adverse effect from drug intake.

- Abnormal conjugation
  - Partial deficiency of glucoronyl transferase
  - drugs may interfere with this enzyme system e.g. Novobiocin

- Hepatocellular damage
  - Acute or chronic hepatocellular injury

# LO5:Post hepatic jaundice

- Obstruction or impaired excretion of bilirubin Failure of transfer of bilirubin glucuronide from the liver cell into the canaliculus (e.g Dubin-Johnson syndrome and Rotor's syndrome).
- Obstruction at the intra-hepatic level (cholestasis)
- □ Obstruction to the flow of bile in the intralobular biliary canaliculli

### LO5:Post hepatic jaundice:

- ☐ Intra-hepatic cholestasis occurs in:
  - in viral hepatitis
  - alcoholic liver disease
  - as a toxic reaction to drugs, including androgens (methyltestosterone), anabolic steroids, oral contraceptives, and phenothiazines
  - in benign familial cholestatic jaundice, a rare familial disease in which recurrent attacks of cholestatic jaundice represent the only abnormality

☐ Extra-hepatic obstruction

Obstruction involve main hepatic ducts, the common hepatic duct, or common bile duct.

Complete obstructive jaundice prevents entry of bilirubin into the intestine, producing pale clay-colored or chalky stools

Absence of fecal and urinary urobilinogen dark brown (tea colored) urine containing bilirubin.

### LO6:Laboratory investigation

- > FBC (hemolysis)
- **Liver function test** 
  - -Serum aminotransferase (AST,ALT)
  - Serology for hepatitis including HCAb, HBsAg, HBcAb
  - ALP: if elevated or if an obstruction is suspected, images of the bile ducts should be obtained.
  - GGT(gamma-glutamyl transferase)
  - Fractionated bilirubin
  - Prothrombin time (PT/INR), aPTT.
  - Albumin and Globulin

**LO6:** 

- U/S
- MRCP