NAUSEA
VOMITING
JAUNDICE

TUCOM
Internal Medicine
3rd year
Dr. Hasan.I.Sultan

NAUSEA & VOMITING

Learning objectives;

1. Make a definition of vomiting, nausea and regurgitation.
2. Clarify the mechanism of vomiting.
3. List the causes of vomiting.
4. Understand the examination points in patient with vomiting.
5. List the complications of vomiting
6. Outline the treatment of nausea and vomiting.

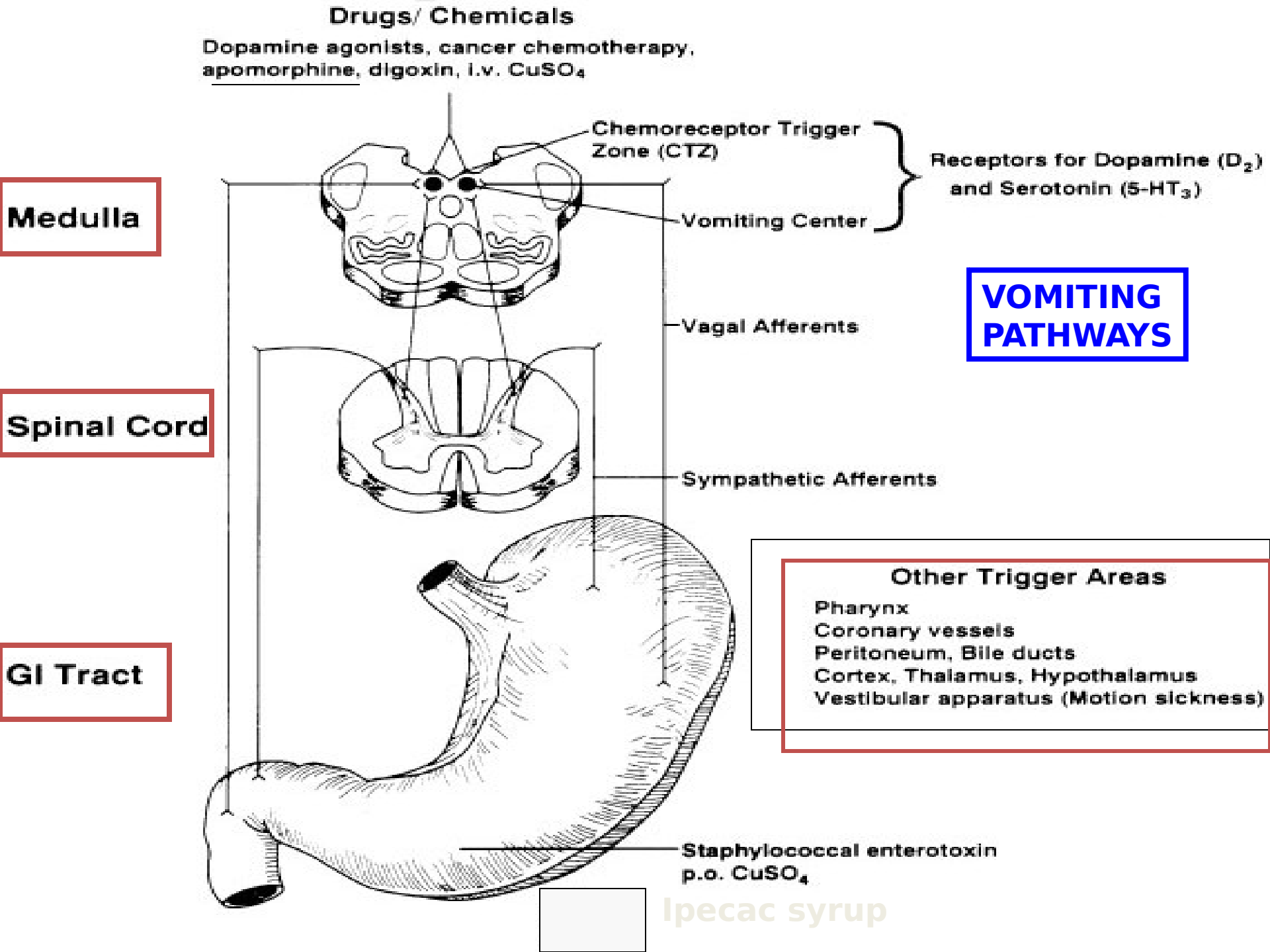
VOMITING

- **Vomiting;** Is a forcible ejection of gastric contents, due to synchronous contraction of the diaphragm, intercostal muscles and abdominal muscles, raises intra-abdominal pressure with relaxation of the lower oesophageal sphincter. Is a highly integrated and complex reflex involving both autonomic and somatic neural pathways.
- **Nausea;** Is the subjective feeling of a need to vomit.
- **Regurgitation;** the effortless passage of gastric contents into the mouth.
- **Projectile vomiting;** refers to vomiting that is sudden, usually without nausea, and so vigorous that the vomit is forcefully projected to a distance. Projectile vomiting is associated with increased intracranial pressure or pyloric obstruction.

Mechanisms; Vomiting is coordinated by the brain stem and is effected by neuromuscular responses in the gut, pharynx, and thoracoabdominal wall

Activators of Emesis; act at several sites.

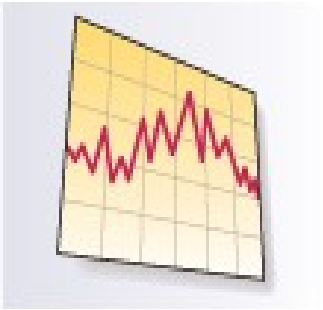
1. **Cerebral cortex;** by unpleasant thoughts or smells
2. **Cranial nerves;** after gag reflex activation
3. **Postrema;** a medullary nucleus, responds to blood borne emetic stimuli and is termed the ***chemoreceptor trigger zone***. Many emetogenic drugs act on the area postrema, as do bacterial toxins and metabolic factors produced during uremia, hypoxia, and ketoacidosis, by activation of 5-HT3, M1, H1, and dopamine D2 receptor subtypes
4. **Labyrinthine apparatus;** motion sickness and inner ear disorders, by activation of cholinergic muscarinic M1 and histaminergic H1 receptors.
5. **Gastric irritants;** such as cytotoxic agents stimulate gastroduodenal vagal afferent nerves


Drugs/ Chemicals
Dopamine agonists, cancer chemotherapy, apomorphine, digoxin, I.v. $CuSO_4$
Chemoreceptor Trigger Zone (CTZ)
Vomiting Center
Receptors for Dopamine (D_2) and Serotonin (5-HT_3)
Medulla
VOMITING PATHWAYS
Vagal Afferents
Spinal Cord
Sympathetic Afferents
Other Trigger Areas
Pharynx
Coronary vessels
Peritoneum, Bile ducts
Cortex, Thalamus, Hypothalamus
Vestibular apparatus (Motion sickness)
GI Tract
Staphylococcal enterotoxin
p.o. $CuSO_4$
Ipecac syrup

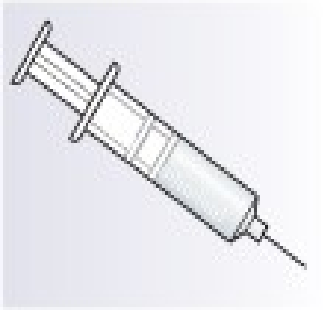
The major causes of vomiting

Alcoholism

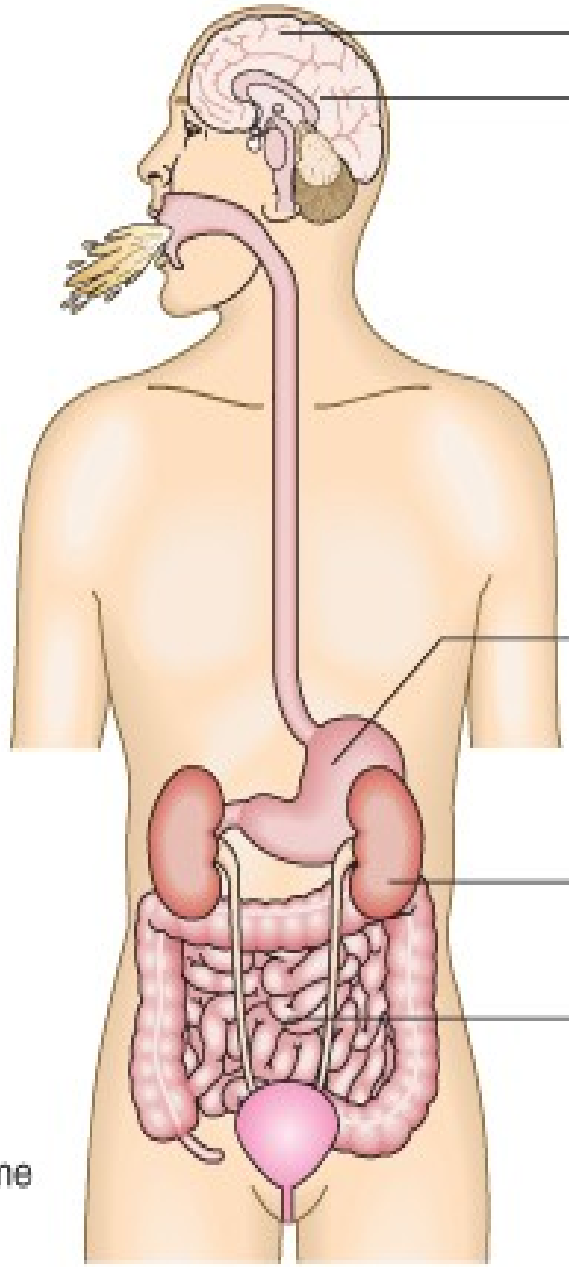
Drugs
- NSAIDs
- Opiates
- Digoxin
- Antibiotics
- Cytotoxins



Infections
- Hepatitis
- Gastroenteritis
- Urinary tract infection



Metabolic
- Diabetic ketoacidosis
- Addison's disease
- Cyclical vomiting syndrome



Psychogenic

CNS disorders
- Vestibular neuronitis
- Migraine
- Raised intracranial pressure
- Meningitis

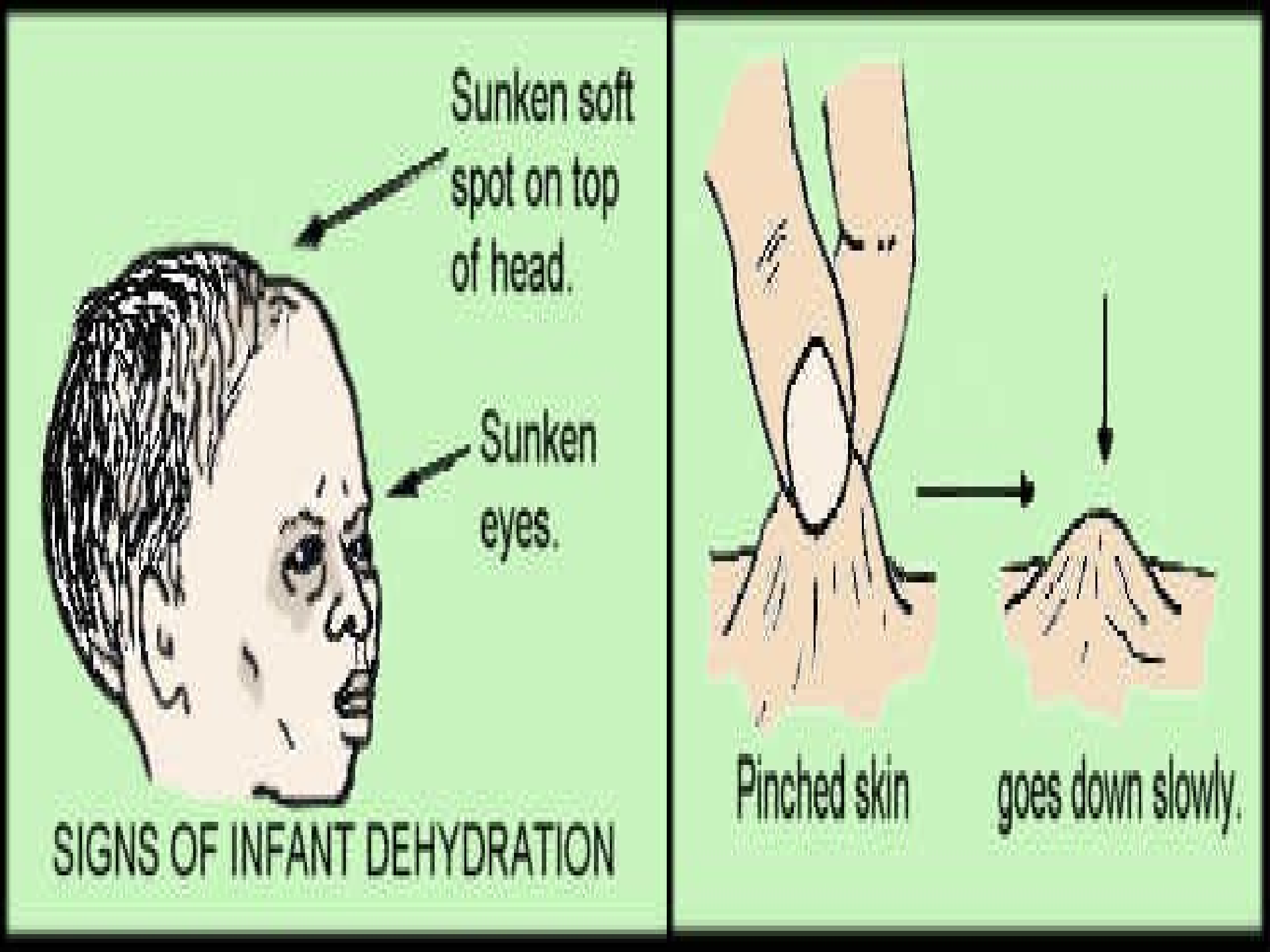
Gastroduodenal
- Peptic ulcer disease
- Gastric cancer
- Gastroparesis

Uraemia

The acute abdomen
- Appendicitis
- Cholecystitis
- Pancreatitis
- Intestinal obstruction

Examination;

- May reveal signs of dehydration, fever and infection.
- Evidence of abdominal masses, peritonitis or intestinal obstruction must be sought.
- Neurological signs including papilloedema, nystagmus, photophobia and neck stiffness.
- Other findings may suggest alcoholism, pregnancy or bulimia as the underlying diagnosis.
- The diagnostic approach will be dictated by the history and examination


Sunken soft spot on top of head.
Sunken eyes.
SIGNS OF INFANT DEHYDRATION
Pinched skin
goes down slowly.

Complications of vomiting

1. Fluid and electrolyte imbalances; Dehydration, metabolic alkalosis, hypokalemia and prerenal azotemia.
2. Nutritional deficiencies
3. Aspiration pneumonia
4. Mallory-Weiss tears
5. Esophageal rupture

Treatment of nausea and vomiting

1. Treat complications regardless of cause e.g., replace salt, water, potassium losses.
2. Identify and treat underlying cause, whenever possible.
3. Relief the symptoms (nausea and vomiting).
4. Use preventive measures when vomiting is likely to occur (e.g., cancer chemotherapy, parenteral opiate administration).

Drugs for treatment of nausea and vomiting

A- Antiemetic agents;

1. Antihistaminergic; Dimenhydrinate, meclizine --- esp. for motion sickness, inner ear disease.
2. Anticholinergic; Scopolamine --- esp. for motion sickness, inner ear disease.
3. Antidopaminergic; Prochlorperazine --- esp. for medication-, toxin-, or metabolic-induced emesis.
4. 5-HT3 antagonist; Ondansetron, granisetron ---- esp. for chemotherapy- and radiation-induced emesis, postoperative emesis
5. Tricyclic antidepressant; Amitriptyline, nortriptyline --- esp. for chronic idiopathic nausea, functional vomiting.

B- Prokinetic agents;

1. **Antidopaminergic; Metoclopramide, Domperidone --- for gastroparesis.**
2. **Motilin agonist; Erythromycin --- for gastroparesis.**

C- Special settings;

1- Benzodiazepines; Lorazepam --- Anticipatory nausea and vomiting with chemotherapy.

2- Glucocorticoids; Methylprednisolone, dexamethasone --- for chemotherapy-induced emesis

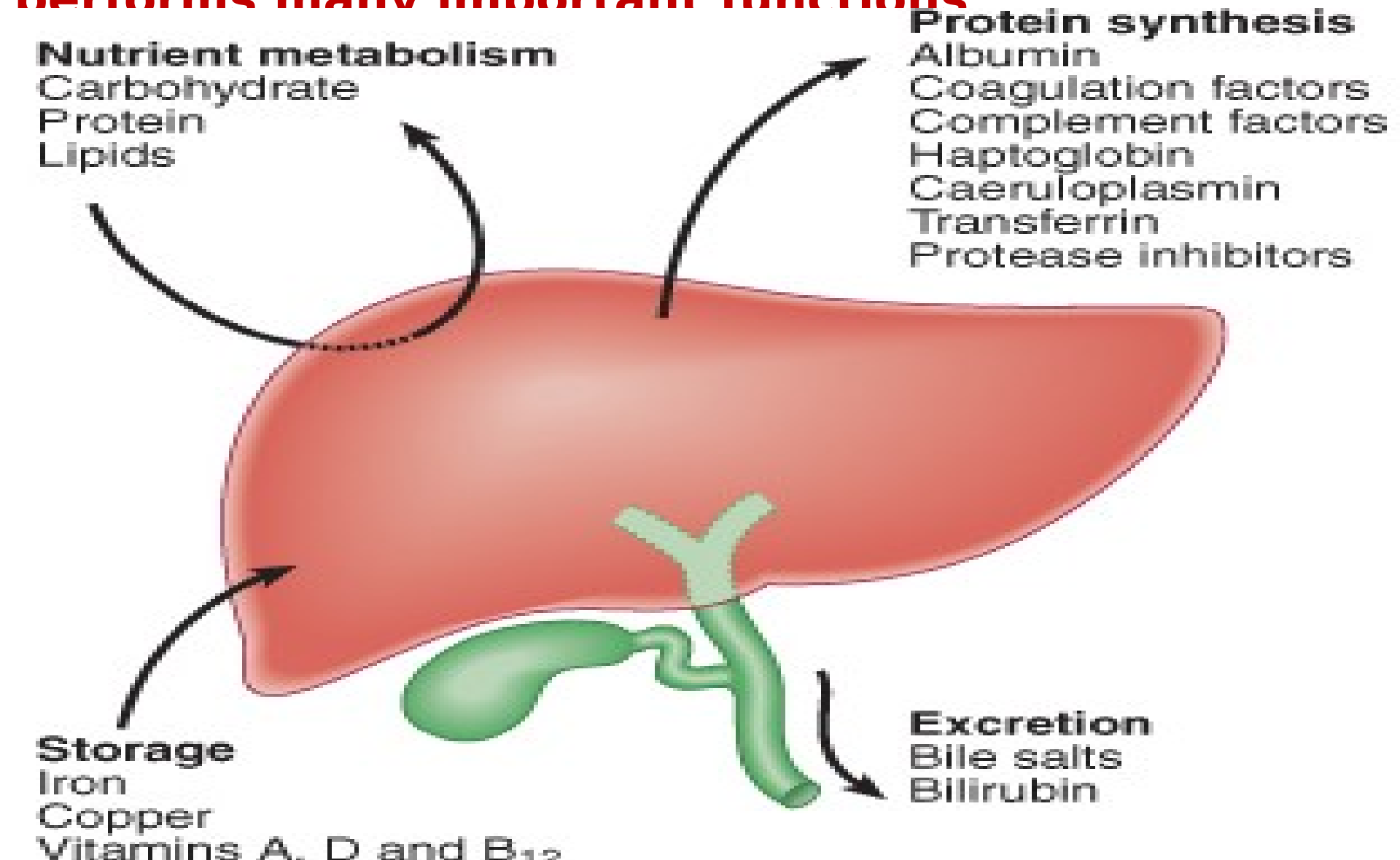
JAUNDICE

Learning objectives;

1. Define jaundice.
2. Clarify the normal function of the liver.
3. Understand the mechanism of bilirubin metabolism.
4. Understand the concept of haemolytic jaundice.
5. Recognize the causes of congenital non-haemolytic hyperbilirubinaemia.
6. Describe the concept of hepatocellular jaundice.
7. Recognize the concept of cholestatic jaundice.
8. List the important investigations of jaundiced patient.

JAUNDICE

The liver is the largest organ in the body and performs many important functions

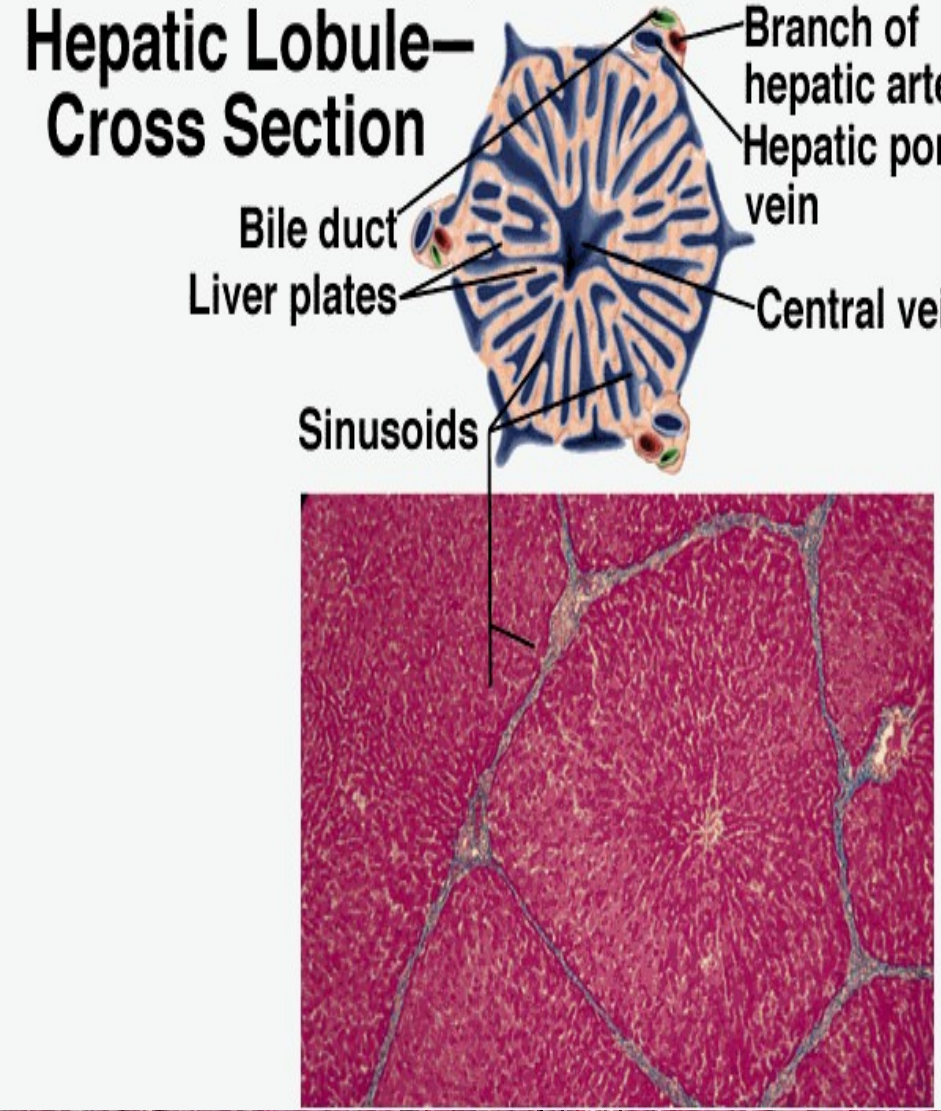


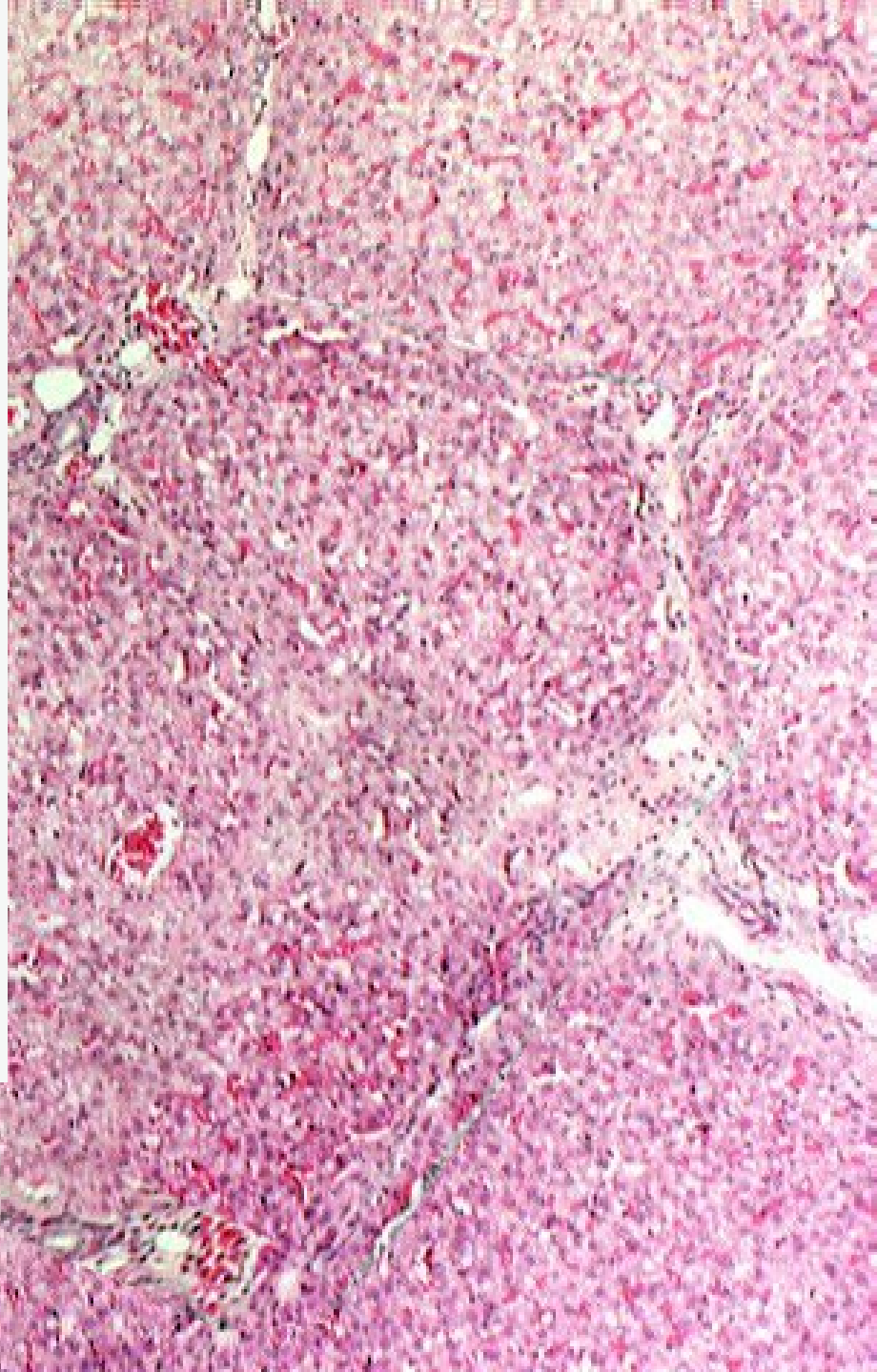
- weighing 1.2-1.5 kg.---divided into the left and right lobes --- divided into a total of eight segments --- lobules --- The functional unit of the liver is the hepatic acinus .

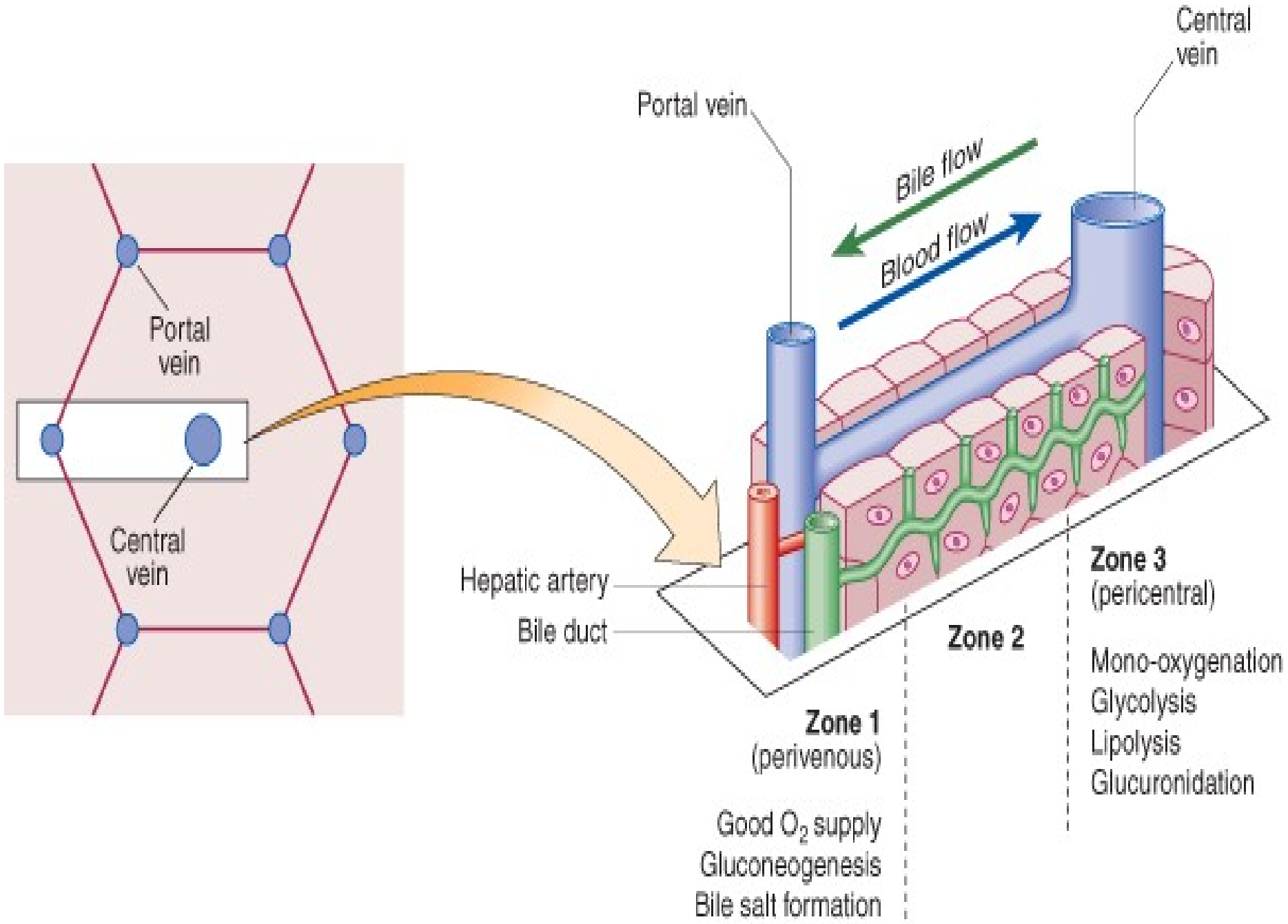
LIVER FUNCTION TESTS USED TO ASSESS LIVER DISEASE

1. Bilirubin
2. Aminotransferases
3. Alkaline phosphatase
4. Gamma-glutamyl transferase
5. Albumin

Hepatic Lobule– Cross Section

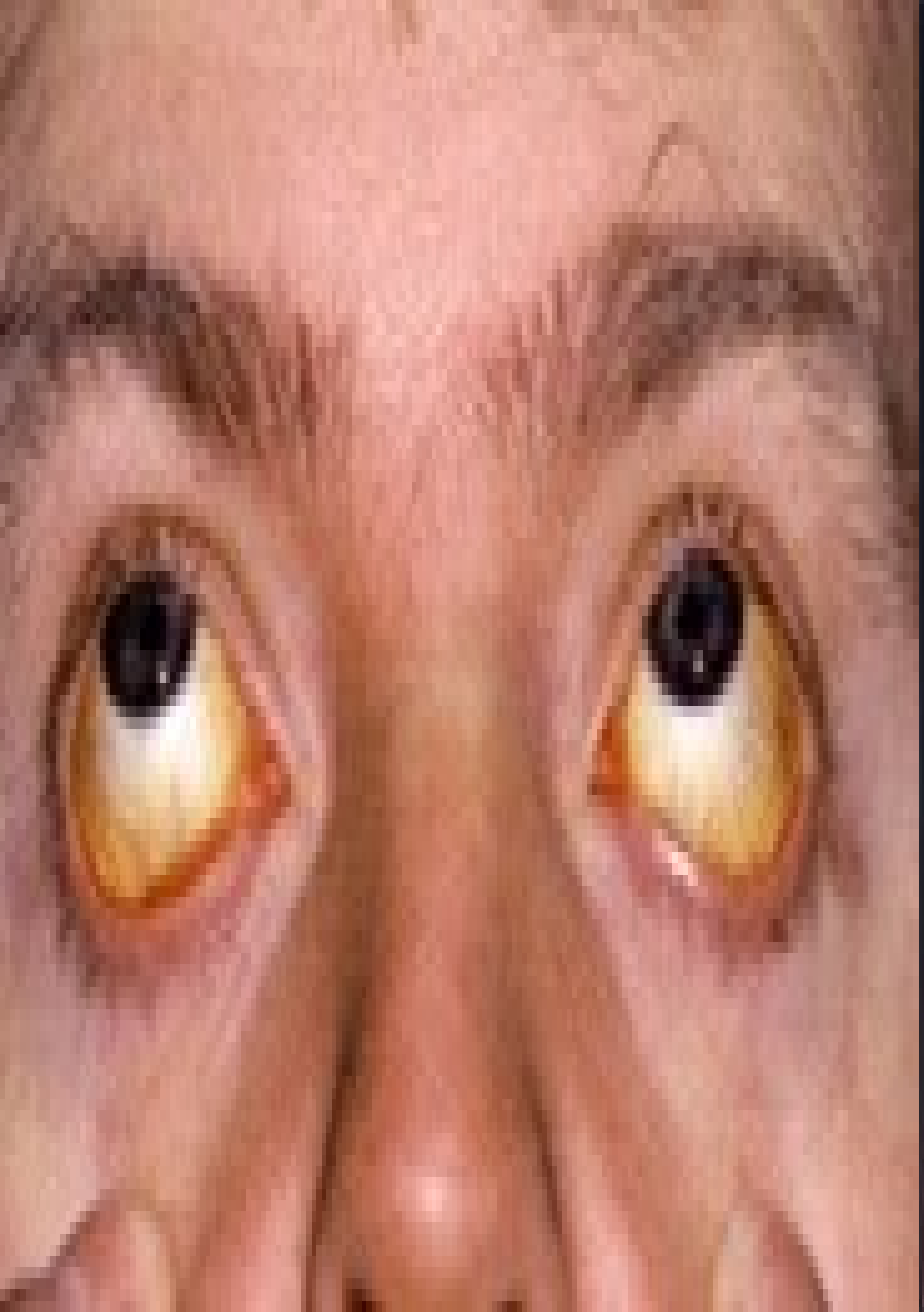


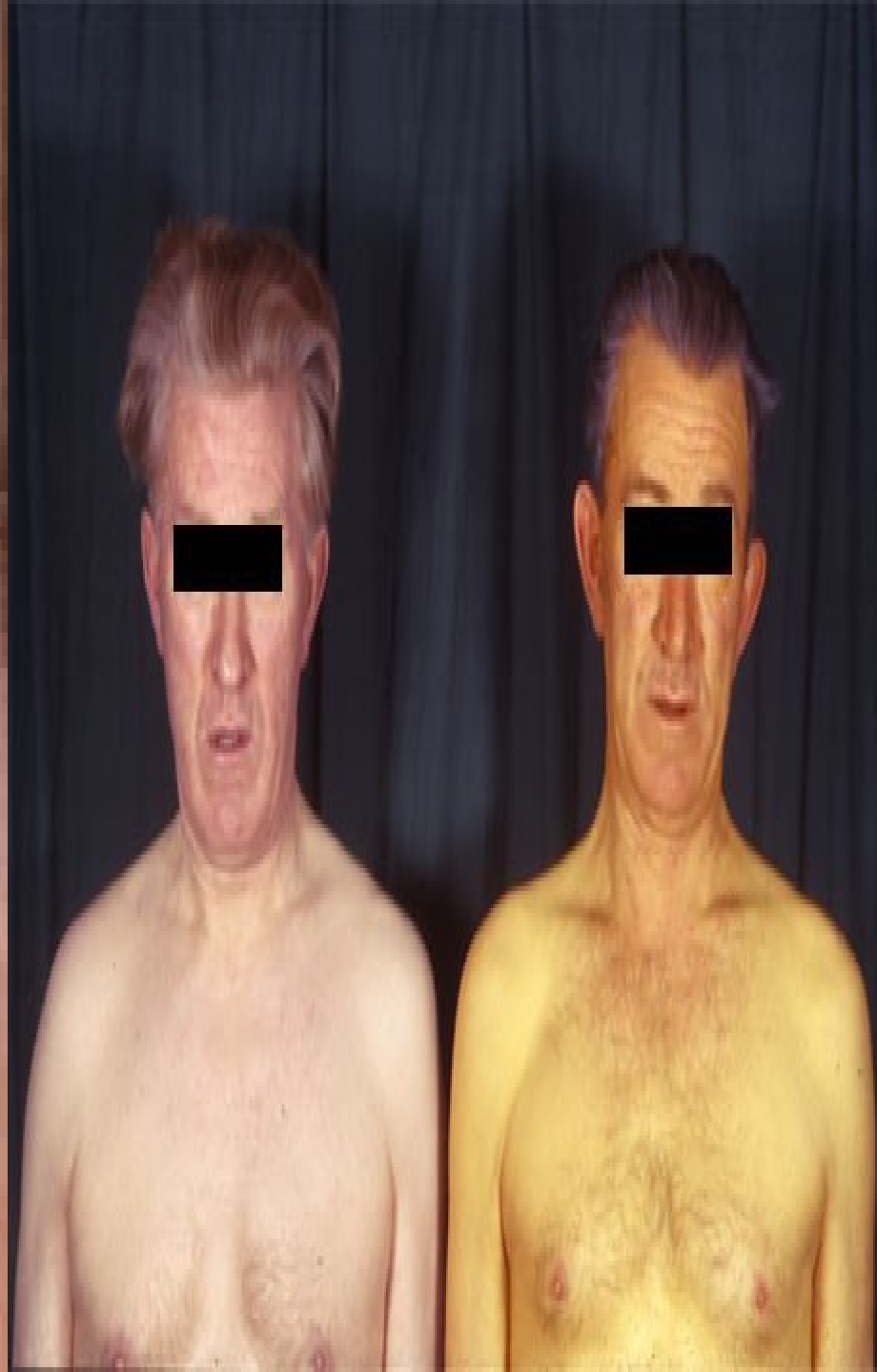




JAUNDICE

- **Jaundice refers to the yellow appearance of the skin, sclerae and mucous membranes resulting from an increased bilirubin concentration in the body fluids.**
- **It is usually detectable clinically when the plasma bilirubin exceeds 50 µmol/l (~3 mg/dl). Normal range (0.5 to 1.0 mg/dl) (5 mmol/l to 17 mmol/l).**
- **Serum bilirubin are best detected by examining the sclerae, which have a particular affinity for bilirubin due to their high elastin content.**
- **More difficult if the examining room has fluorescent lighting.**
- **A second place to examine is underneath the tongue.**



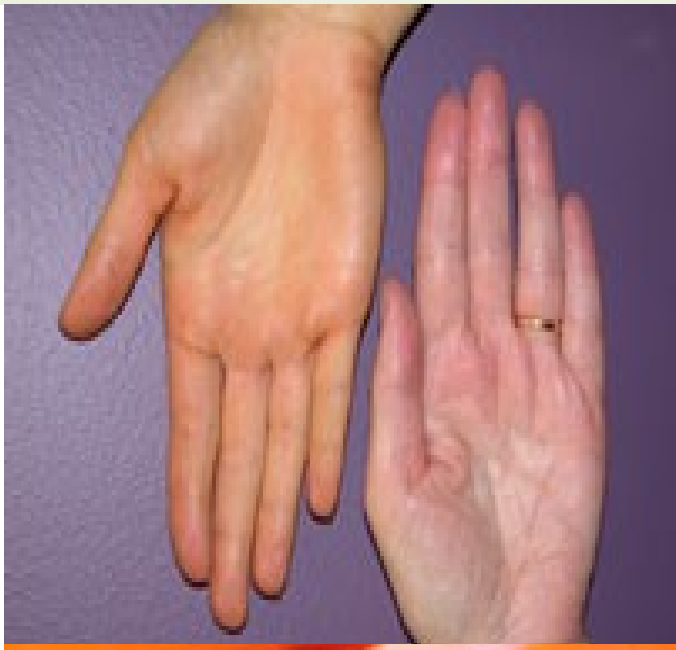


Differential diagnosis for yellowing of the skin

1- Carotenoderma; Is the yellow color of skin (but not sclerae) by the presence of carotene; it occurs in healthy individuals who ingest excessive amounts of vegetables and fruits that contain carotene, such as carrots, leafy vegetables, and oranges.

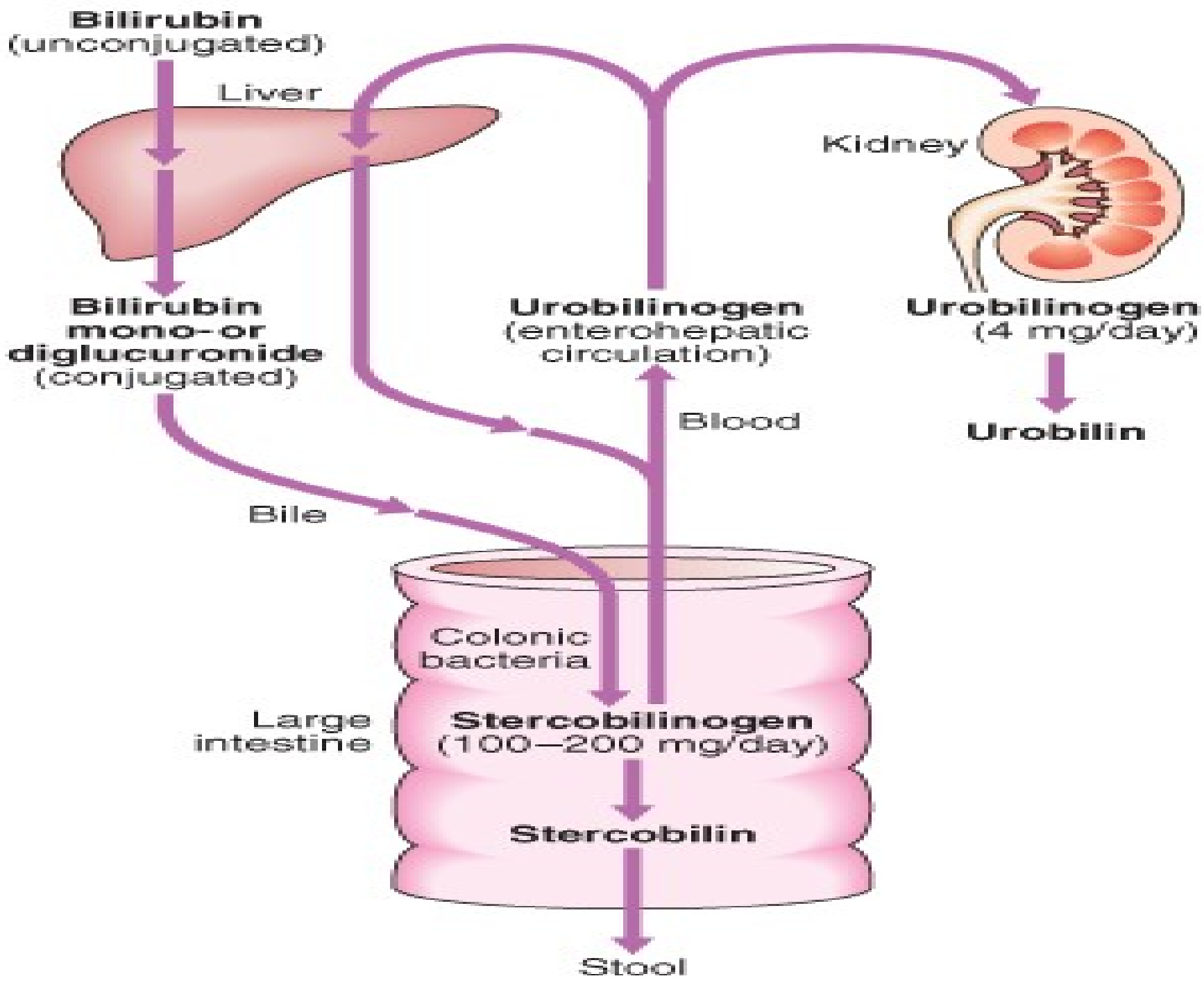
2- Drug; Quinacrine

3- Excessive exposure to phenols.



Bilirubin metabolism

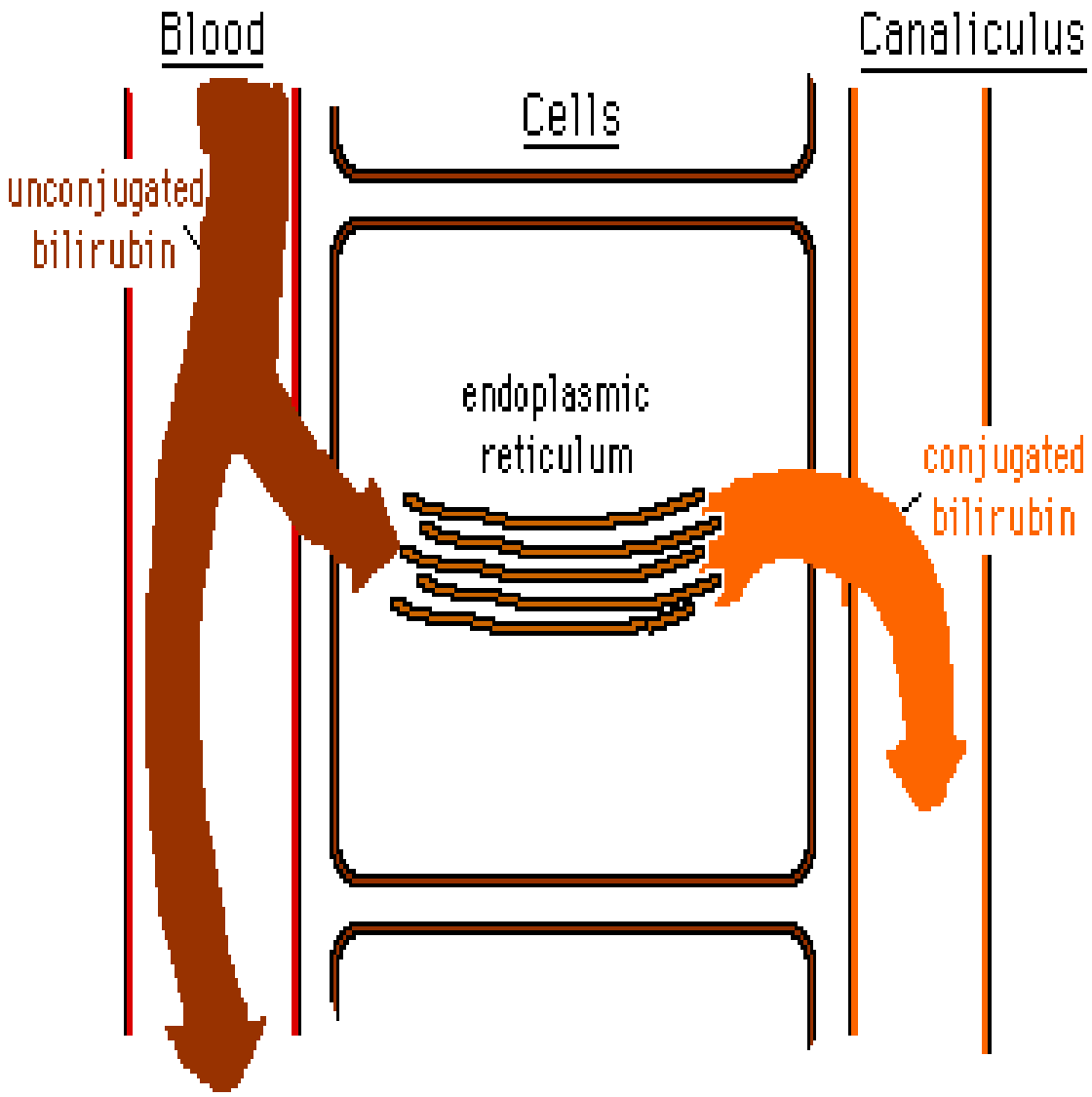
- **Between 425 and 510 mmol (250-300 mg) of unconjugated bilirubin is produced from the catabolism of haem every day**
- **Bilirubin in the blood is normally almost all unconjugated and, because it is not water-soluble, is bound to albumin and does not pass into the urine.**
- **Unconjugated bilirubin is conjugated by glucuronyl transferase, into bilirubin mono- and diglucuronide. Is**

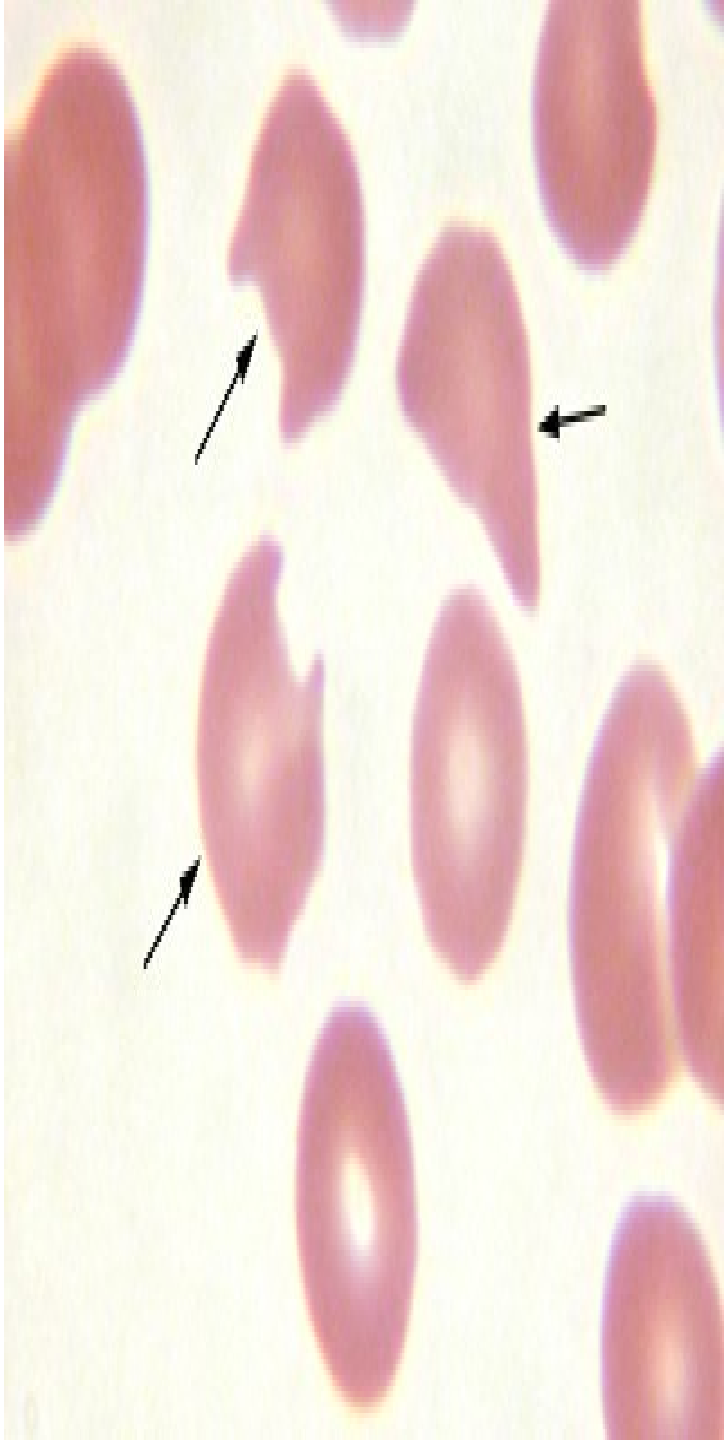

Bilirubin
(unconjugated)
Liver
Kidney
Bilirubin
mono- or
diglucuronide
(conjugated)
Urobilinogen
(enterohepatic
circulation)
Urobilinogen
(4 mg/day)
Blood
Urobilin
Bile
Colonic
bacteria
Large
intestine
Stercobilinogen
(100—200 mg/day)
Stercobilin
Stool

HAEMOLYTIC JAUNDICE

- Results from increased destruction of red blood cells or their precursors in the marrow.
- Jaundice is usually mild.
- No stigmata of chronic liver disease
- Normal-coloured stools, and urine, but urine to turn dark on standing as urobilin is formed.
- Pallor due to anaemia, and splenomegaly
- Plasma bilirubin less than 100 µmol/l (~6 mg/dl) and the LFTs are normal. Unconjugated hyperbilirubinaemia
- Blood film show = haemolytic anaemia

Increased Bilirubin Production Beyond the Liver's Capacity to Conjugate It



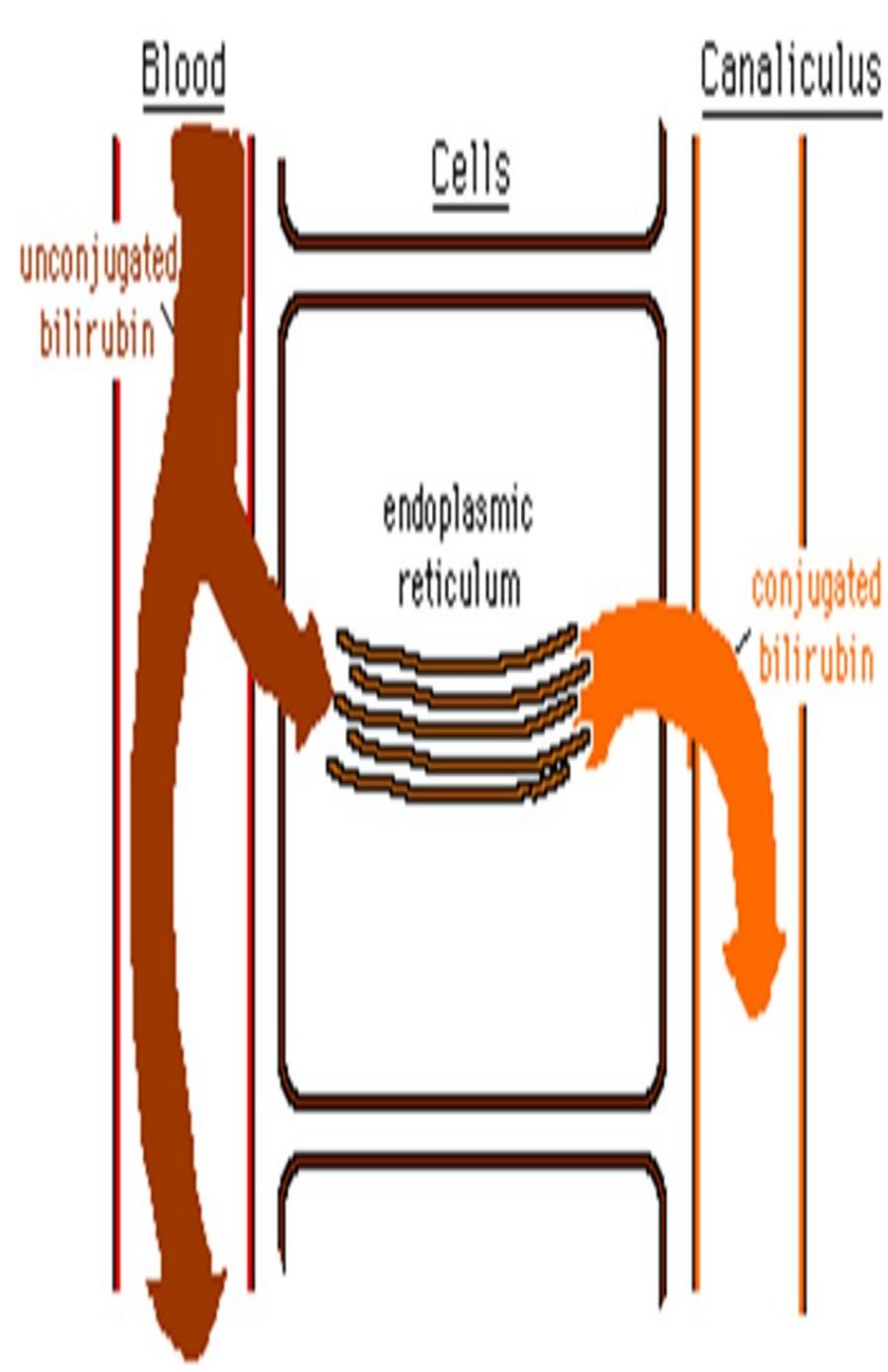


CONGENITAL NON-HAEMOLYTIC HYPERBILIRUBINAEMIA

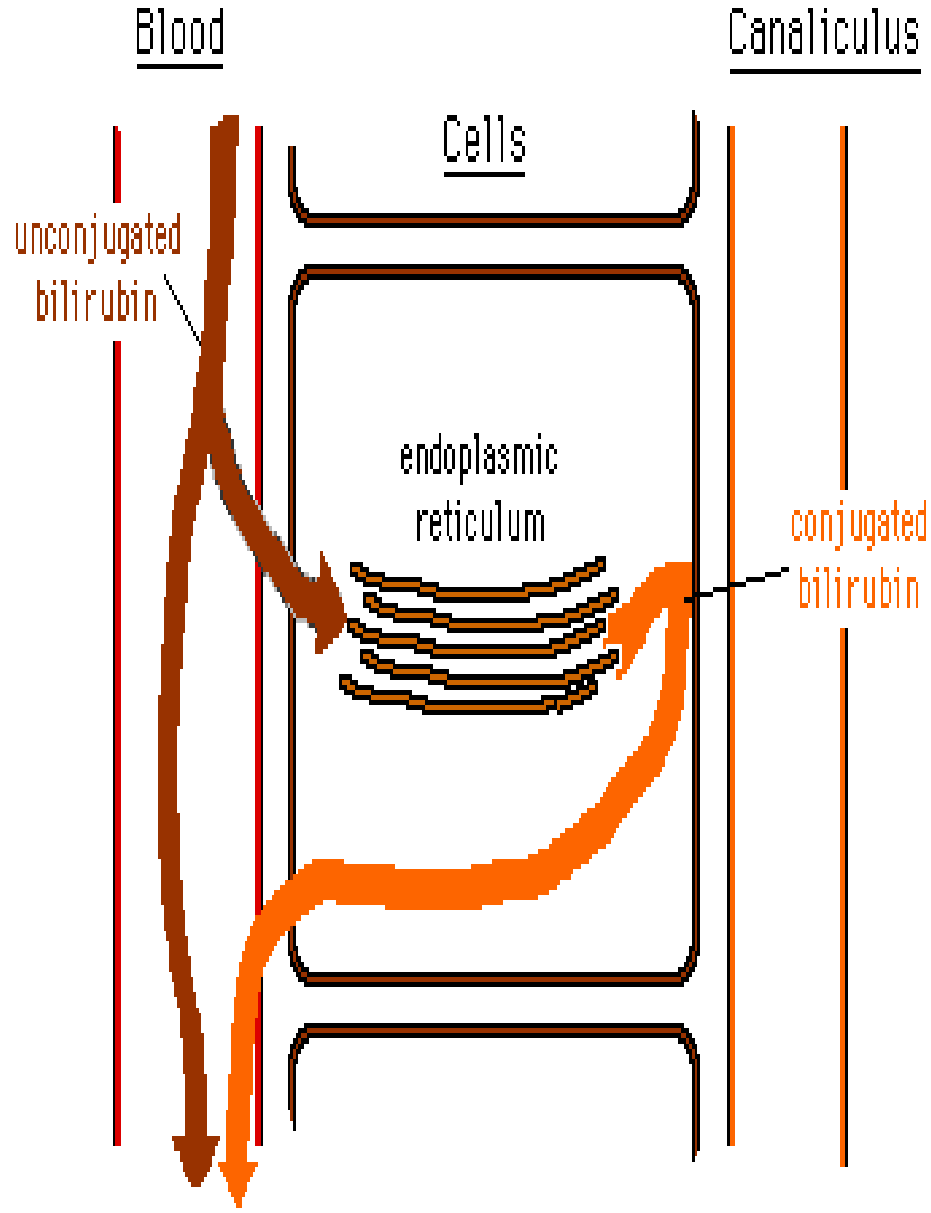
Syndrome	Inheritance	Abnormality	Clinical features/treatment
UNCONJUGATED HYPERBILIRUBINAEMIA			
Gilbert's	Autosomal dominant	↓ Glucuronyl transferase	Mild jaundice, especially with fasting
		↓ Bilirubin uptake	No treatment necessary
Crigler-Najjar			
Type I	Autosomal recessive	Absent glucuronyl transferase	Rapid death in neonate (kernicterus)
Type II	Autosomal dominant	↓↓ Glucuronyl transferase	Presents in neonate Phenobarbital, ultraviolet light or liver transplant as treatment
CONJUGATED HYPERBILIRUBINAEMIA			
Dubin-Johnson	Autosomal recessive	↓ Canalicular excretion of organic anions including bilirubin	Mild No treatment necessary
Rotor's	Autosomal	↓ Bilirubin uptake	Mild

HEPATOCELLULAR JAUNDICE

- **Results from an inability of the liver to transport bilirubin into the bile, occurring as a consequence of parenchymal liver disease**
- **The concentrations of both unconjugated and conjugated bilirubin in the blood increase.**
- **In addition, swelling of cells and oedema resulting from the disease itself may cause obstruction of the biliary canaliculi (sever disease).**



Defective Secretion of Conjugated Bilirubin from Liver Cells



CHOLESTATIC JAUNDICE

Conjugated bilirubin is unable to enter the bile canaliculi and passes back into the blood.

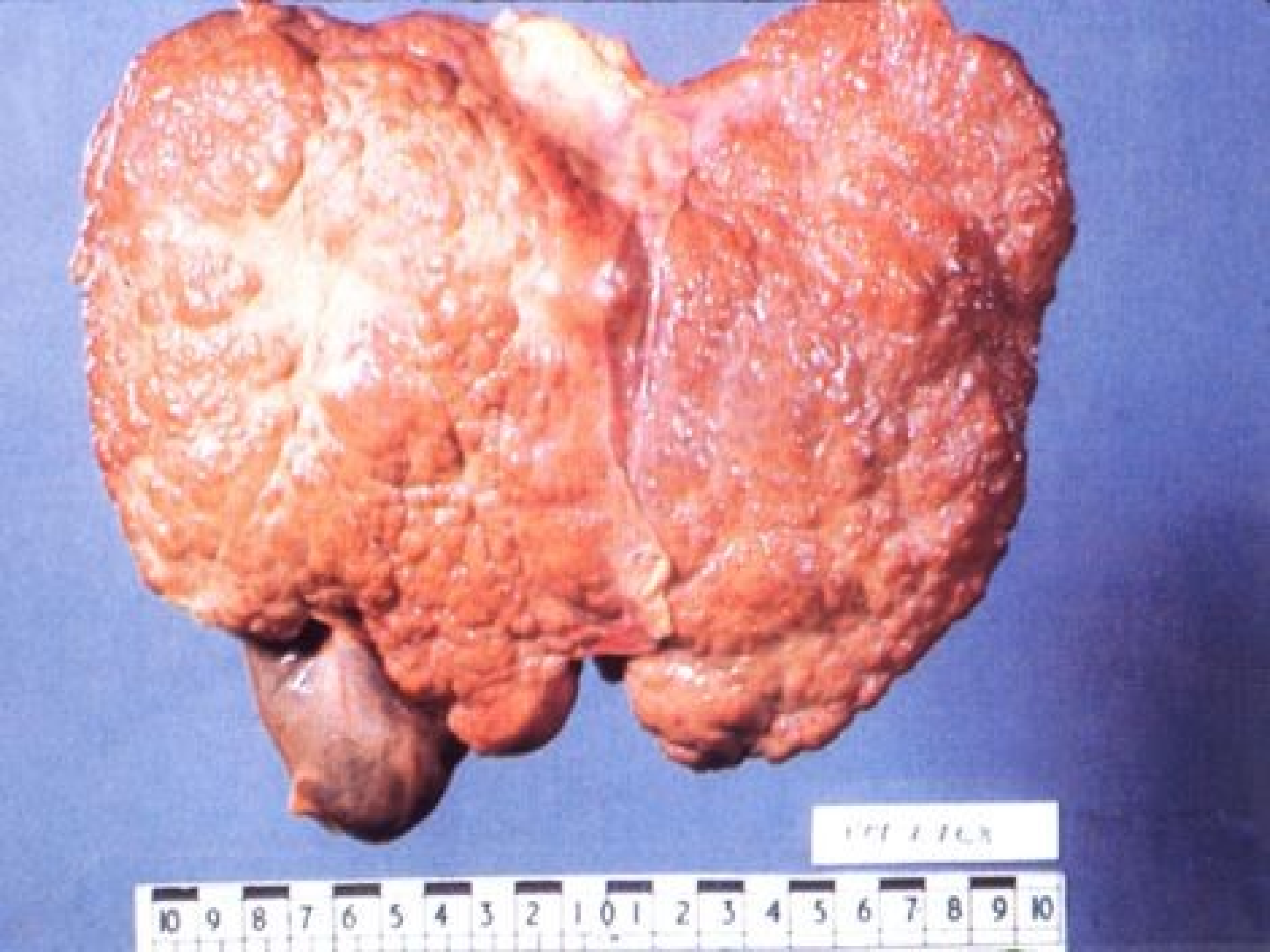
Aetiology;

1- Intrahepatic;

- Primary biliary cirrhosis
- Primary sclerosing cholangitis
- Alcohol
- Drugs
- Viral hepatitis
- Autoimmune hepatitis
- Cystic fibrosis
- Severe bacterial infections
- Post-operative

2- Extrahepatic;

- Choledocholithiasis
- Carcinoma
  - Ampullary
  - Pancreatic
  - Bile duct (cholangiocarcinoma)
  - Secondary
- Parasitic infection
- Traumatic biliary strictures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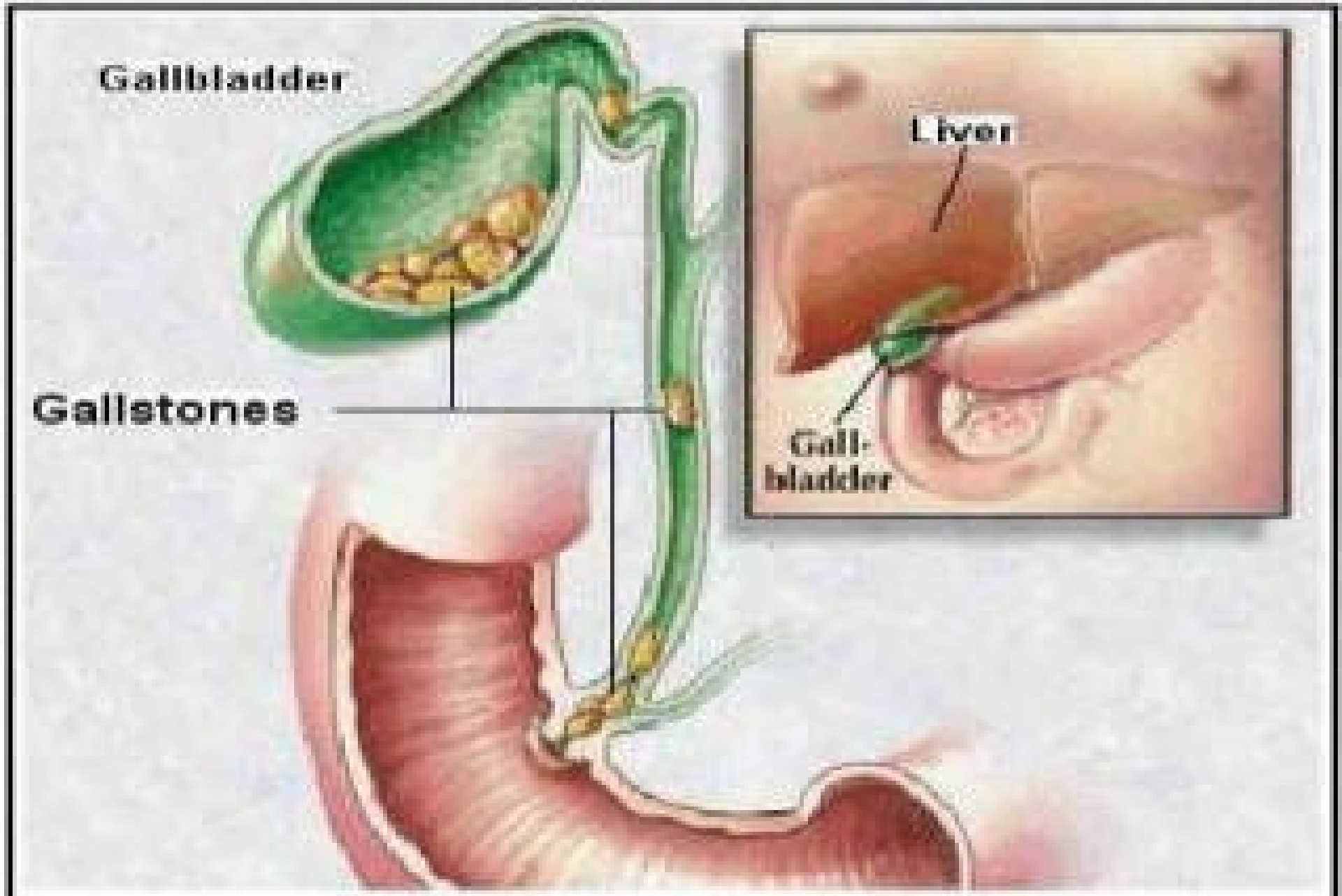
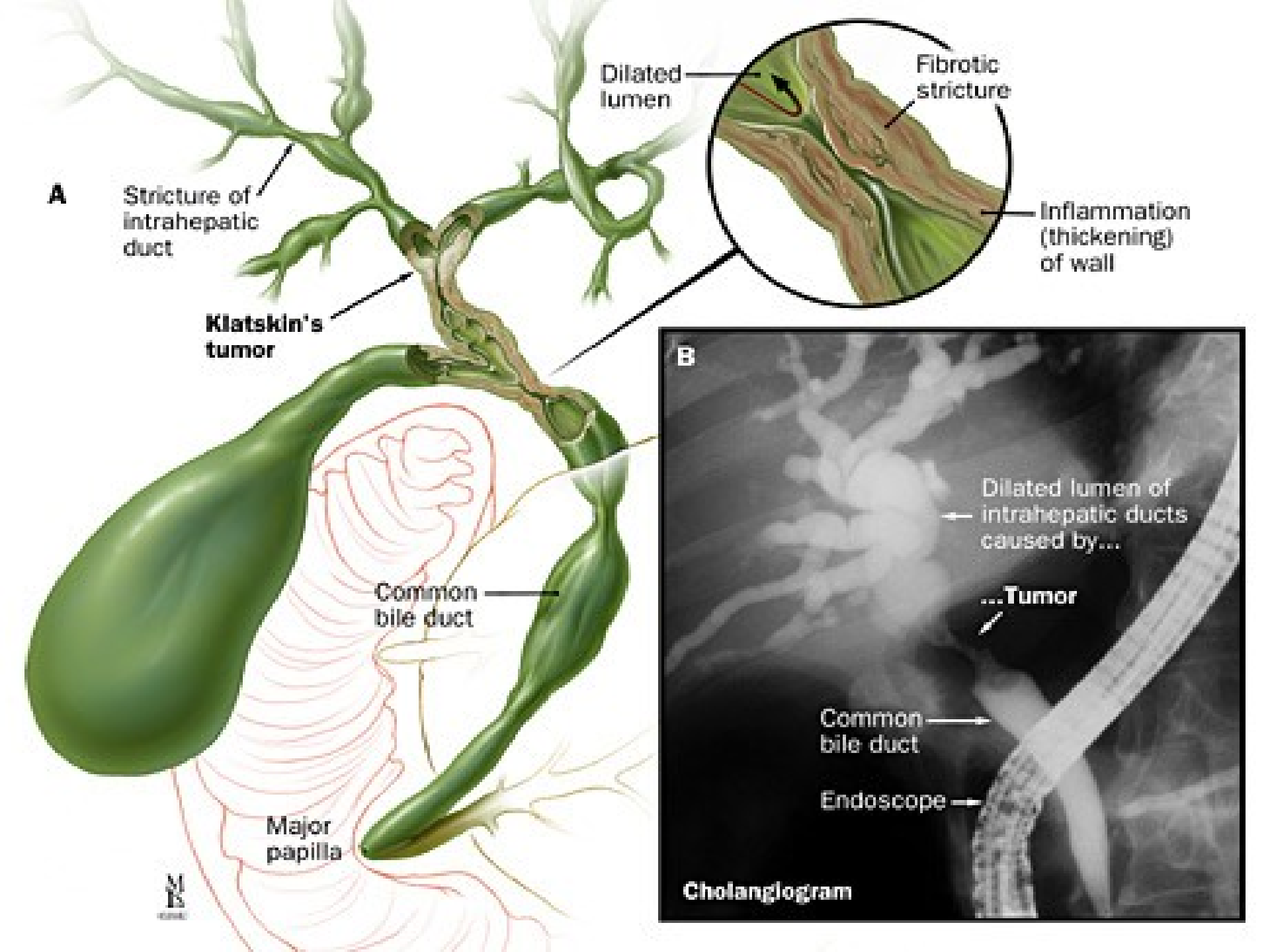


Diagram showing gallstones inside gallbladder and gall duct.


A
Stricture of intrahepatic duct
Klatskin's tumor
Common bile duct
Major papilla
Dilated lumen
Fibrotic stricture
Inflammation (thickening) of wall
B
Dilated lumen of intrahepatic ducts caused by...
...Tumor
Common bile duct
Endoscope
Cholanglogram

CLINICAL FEATURES IN CHOLESTATIC JAUNDICE

A- Cholestasis;

1- Early features

- Jaundice
- Dark urine
- Pale stools
- Pruritus

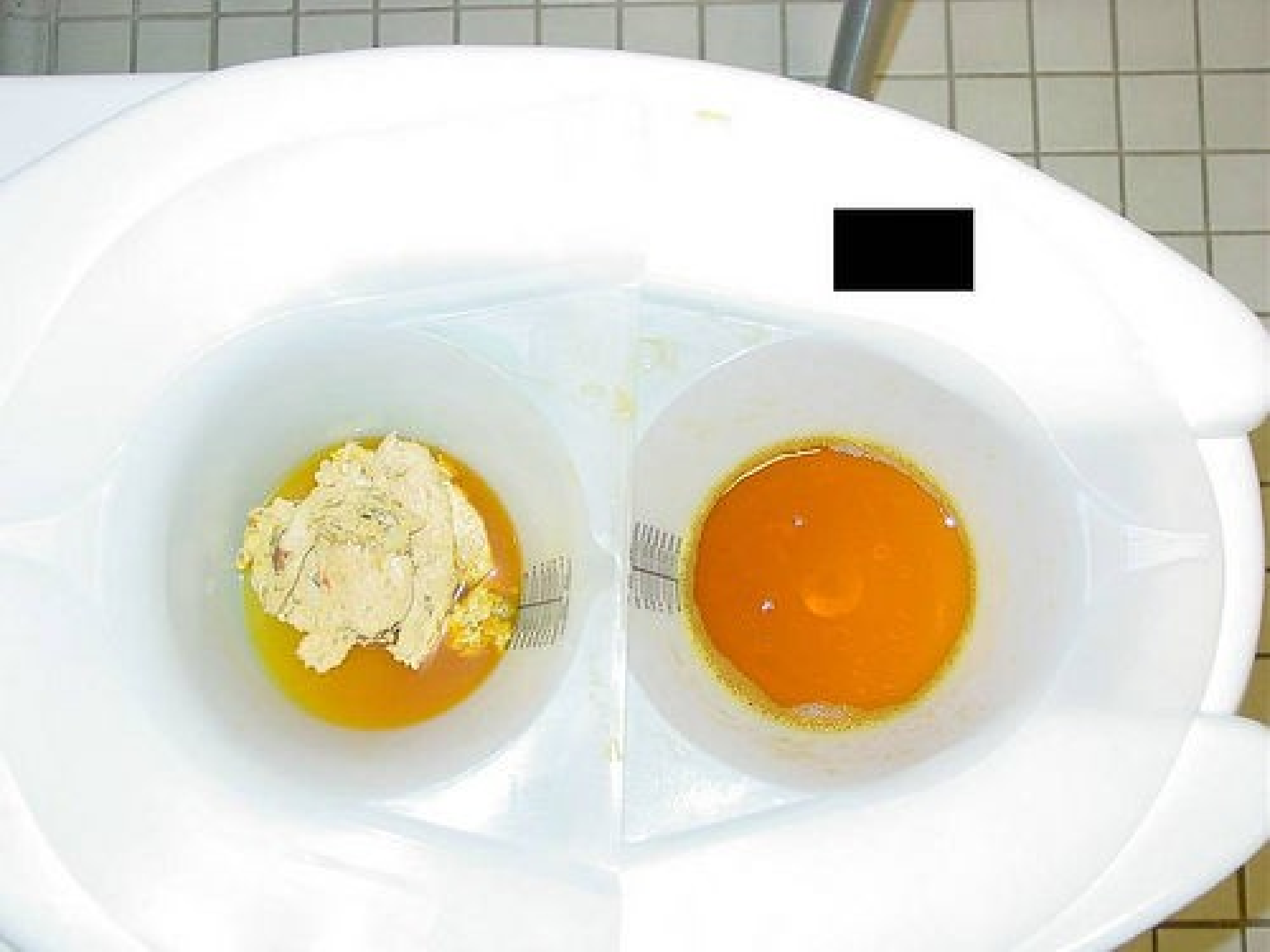
2- Late features

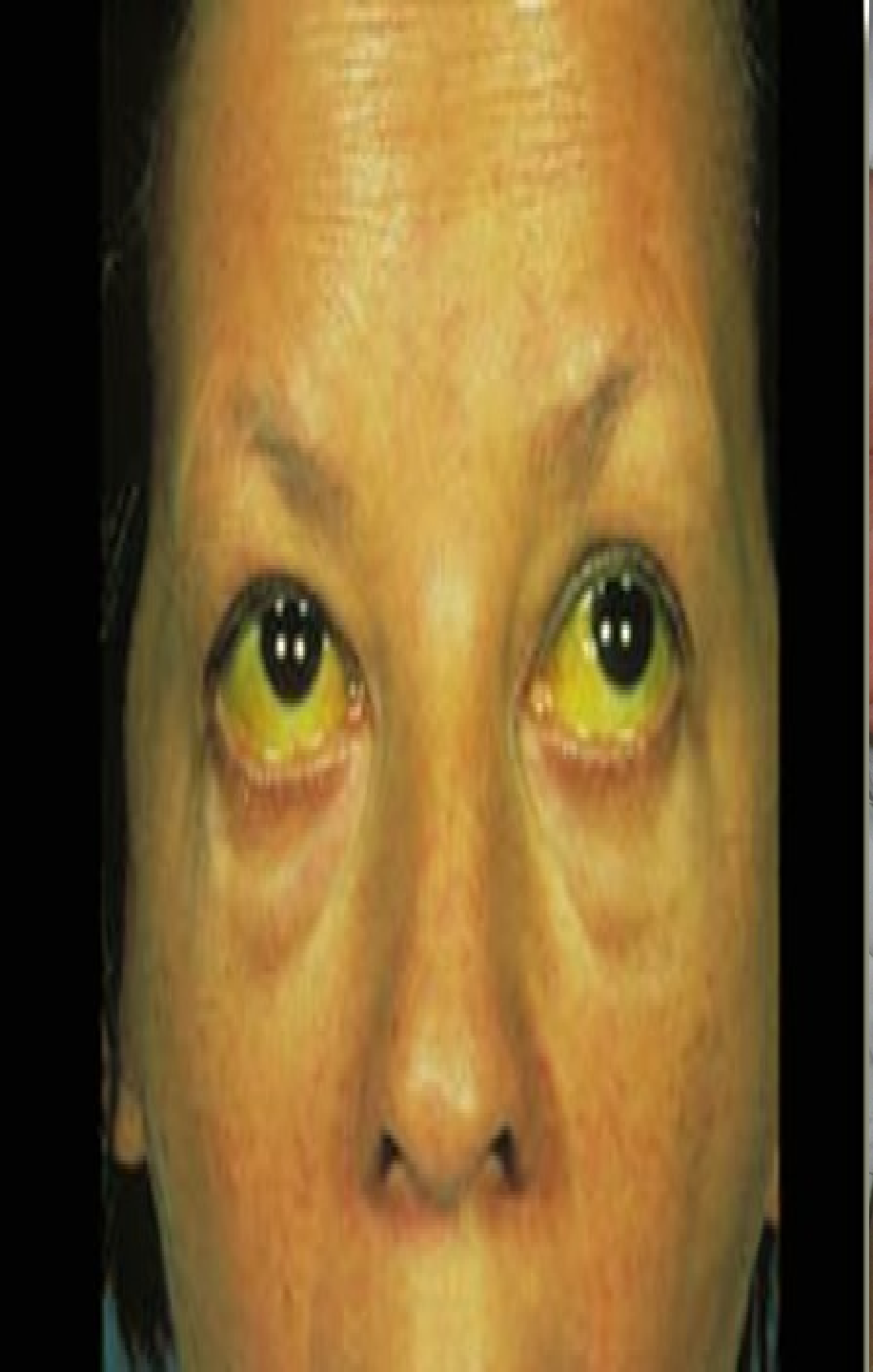
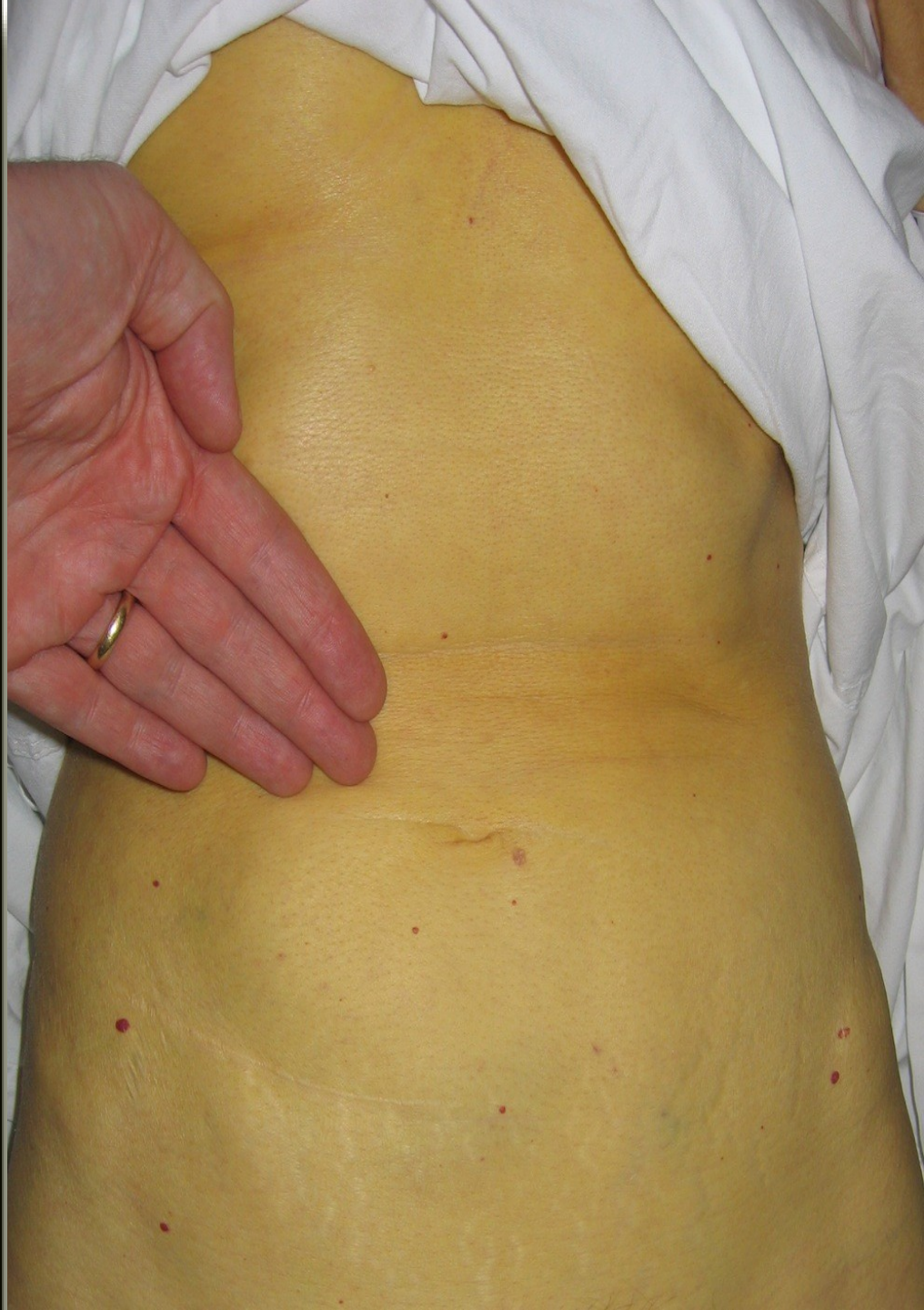
- Xanthelasma and xanthomas
- Malabsorption
  - Weight loss
  - Steatorrhoea
  - Osteomalacia
  - Bleeding tendency

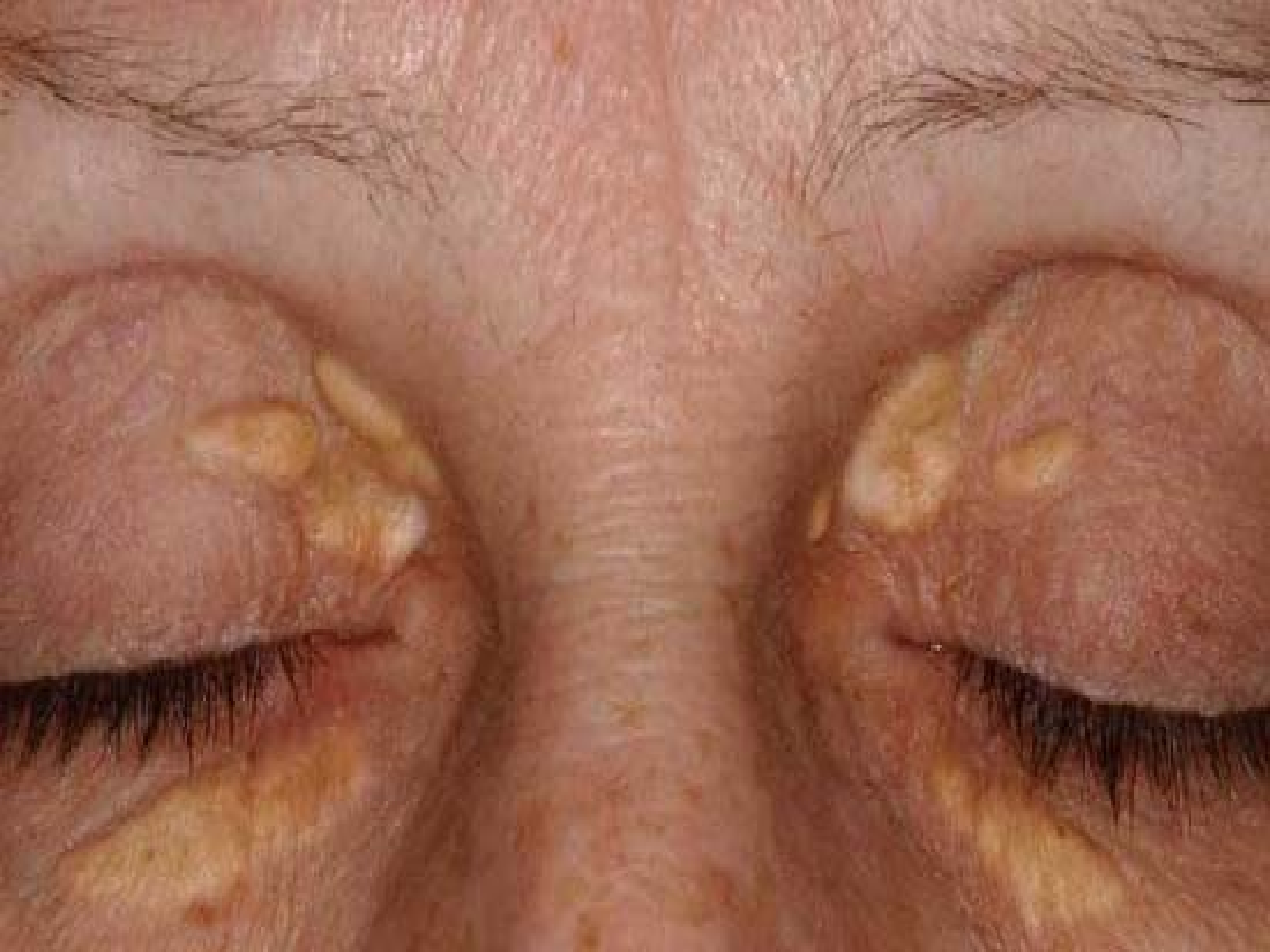
B- Cholangitis;

Charcot's triad

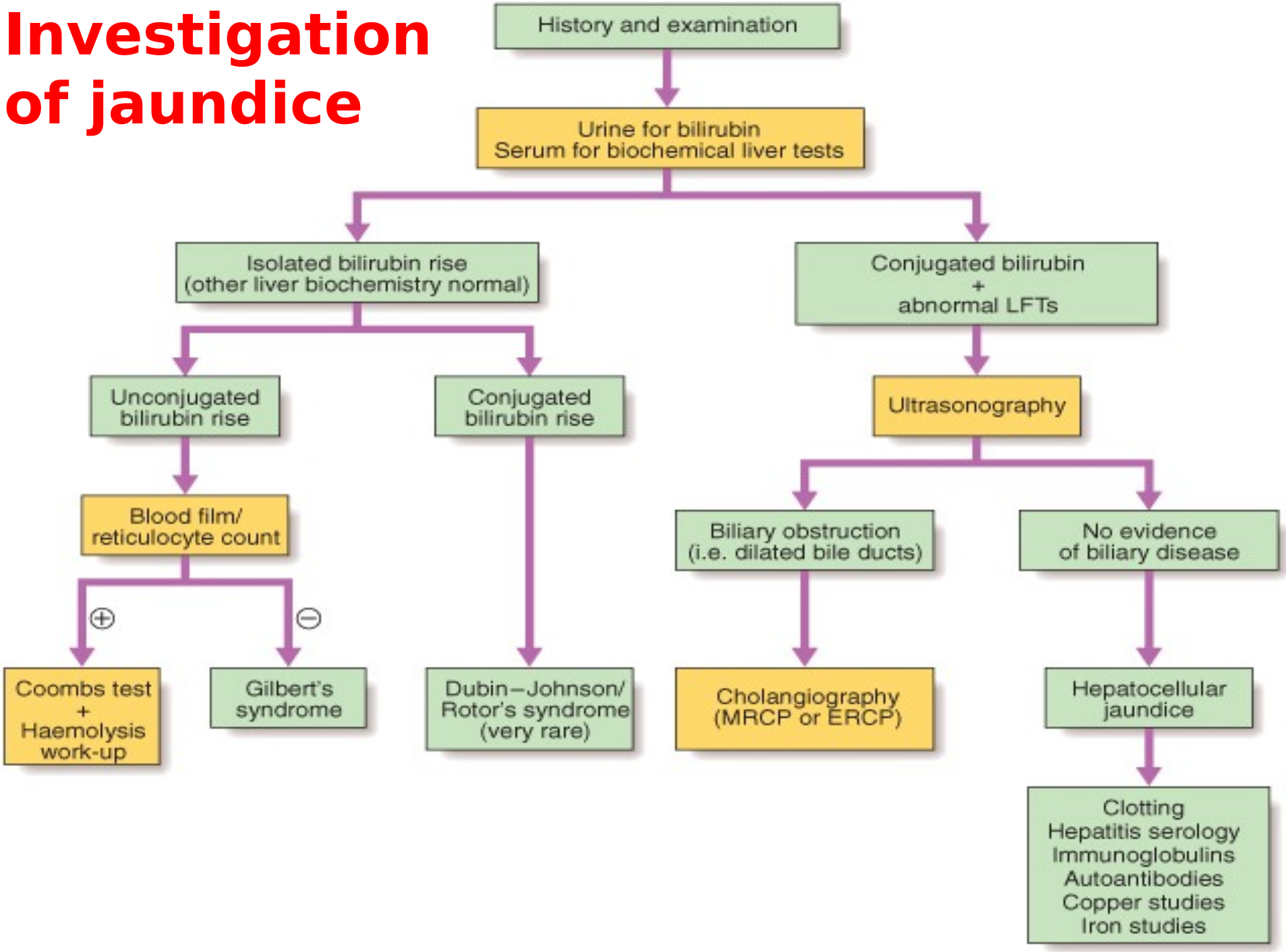
1. Fever
2. Rigors
3. Right upper quadrant abdominal pain.





Investigation of jaundice



'Hepatitic' and 'cholestatic'/'obstructive' LFTs

Pattern	AST/ALT	GGT	ALP
Biliary obstruction	↑	↑↑	↑↑↑
Hepatitis	↑↑↑	↑	↑

↑ mild elevation (< twice normal); ↑↑ moderate elevation (2-5 times normal); ↑↑↑ marked elevation (> 5 times normal).

Thanks