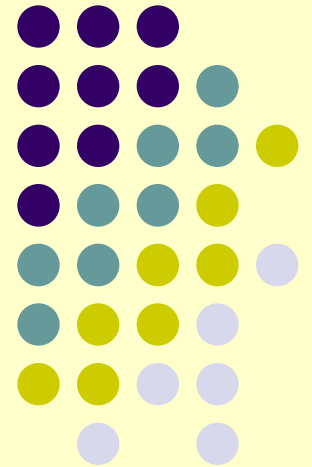
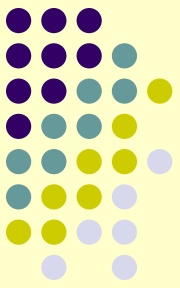


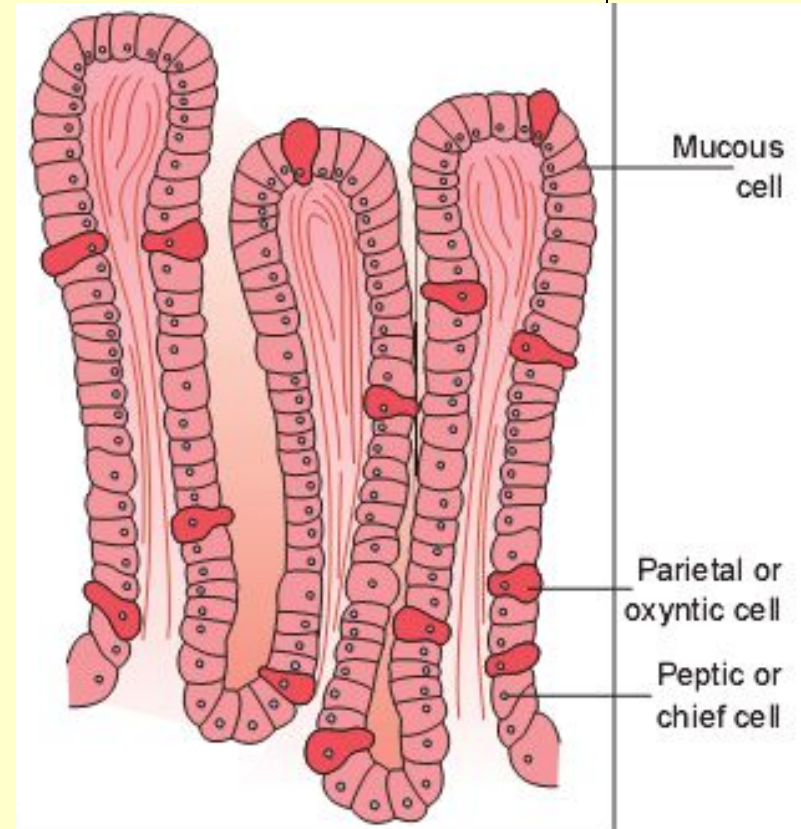
GIT disorders

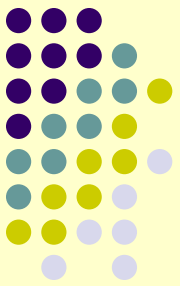


Gastric mucosal barrier



- tight cellular junctions
- presence of a protective mucus layer
- bicarbonate ions (HCO_3^-) secretion
- synthesis of prostaglandins

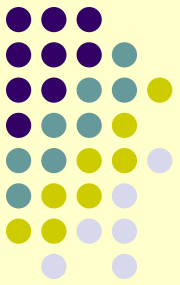




Acute gastritis

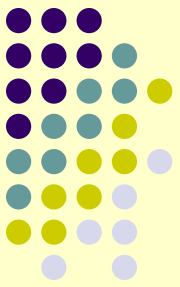
Causes

- Diet and personal habits (excessive alcohol, smoking, malnutrition).
- Infections:
 - bacterial - Helicobacter pylori, diphtheria, salmonellosis, staphylococcal food poisoning;
 - viral - viral hepatitis, influenza.
- Drugs (NSAIDs, cortisone).
- Chemical and physical agents.
- Severe stress.



Gastritis pathogenesis

- **Reduced blood flow** □ mucosal hypo-perfusion □ ischemia.
- **Increased acid secretion** (in H.pylori infection) □ damage to epithelial barrier.
- **Decreased production of bicarbonates.**



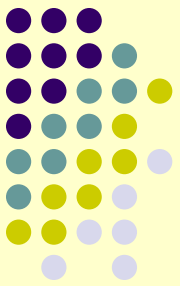
Types of chronic gastritis

Type A Gastritis (Autoimmune gastritis).

- antibodies against parietal cells and intrinsic factor.
- other autoimmune diseases .
- gastric atrophy
- hypo- or achlorhydria.

Type B Gastritis (Helicobacter pylori-related).

- excessive secretion of acid (hypersecretory gastritis)
- associated peptic ulcer



Types of chronic gastritis

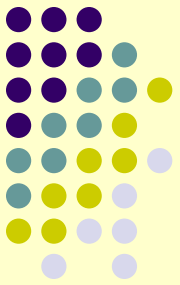
Type AB Gastritis (environmental)

- gastric atrophy
- caused by environmental factors.

Type C Gastritis (Chemical)

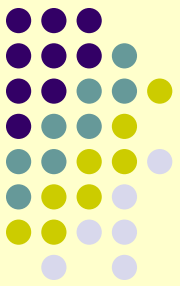
- due to reflux of alkaline duodenal contents, pancreatic secretions, and bile into the stomach.
- in persons after GIT surgery, with gastric ulcer, gallbladder diseases.

Peptic ulcer disease



Ethiology:

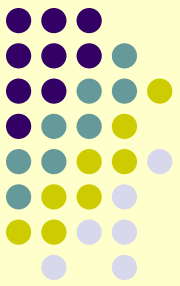
- H pylori infection
- NSAIDs (aspirin - the most ulcerogenic)
- Lifestyle factors
- Severe physiologic stress
- Genetic factors (hereditary predisposition)



Stress ulceration

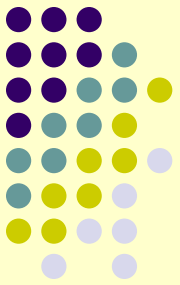
High level of glucocorticoids and adrenaline (stress hormones) causes:

- mucus secretion (glucocorticoids)
- regeneration of gastric epithelial cells (glucocorticoids)
- microcirculation and ischemia of mucosal tunic (glucocorticoids, adrenaline)
 - other reasons of ischemia (collapse, shock, acute blood loss, spasm of blood vessels)
- tonus of the vagal nerve HCl and pepsin secretion.



Clinical manifestations

- affection of one or all layers of stomach
- remissions and exacerbation
- healing with scar formation
- stomach discomfort and pain.
 - periodicity of pain (on empty stomach).
 - recurrence of pain.
 - pain is relieved by food or antacids.



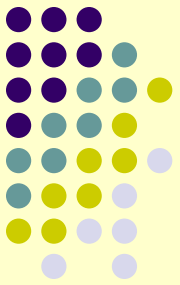
Complications

Hemorrhage

- bleeding from granulation tissue
- erosion of an ulcer into an artery or vein

Hematemesis or melena.

Acute hemorrhage – signs of circulatory shock depending on the amount of blood loss.



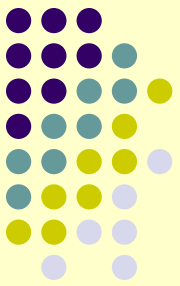
Complications

Obstruction

- edema, spasm or contraction of scar.
- epigastric fullness, vomiting of undigested food.

Perforation

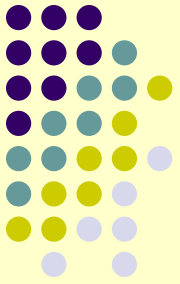
- GI contents enter the peritoneum (peritonitis),
- ulcer penetrate adjacent structures (pancreas),
- severe pain radiating into the back.



Therapy principles

- Eradication of *Helicobacter pylori* with antibiotics
- Inhibition of gastric secretion
 - H2 histamine receptor antagonists (cimetidine, ranitidine)
 - Prostaglandin E1 analogues (misoprostol)
- Surgical management
 - highly selective vagotomy in order to inhibit gastric secretion

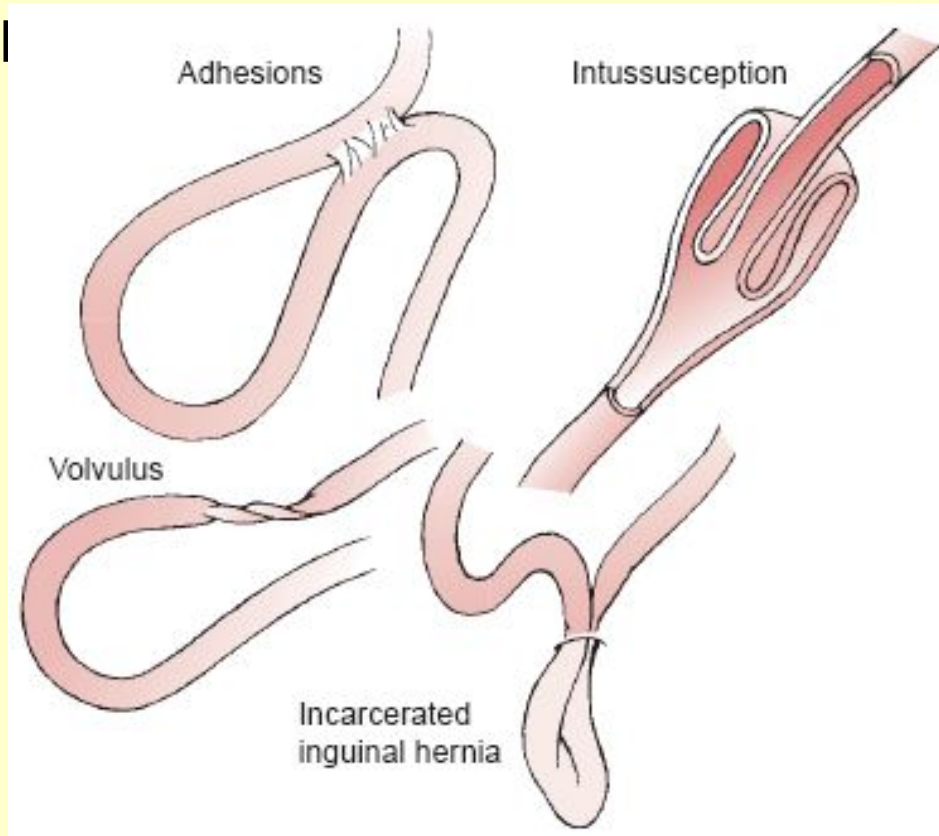
Intestinal obstruction

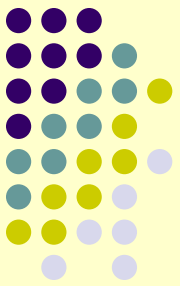


Mechanical obstruction

Causes:

- external hernia
- postoperative adhesions.
- strictures,
- tumor, foreign bodies



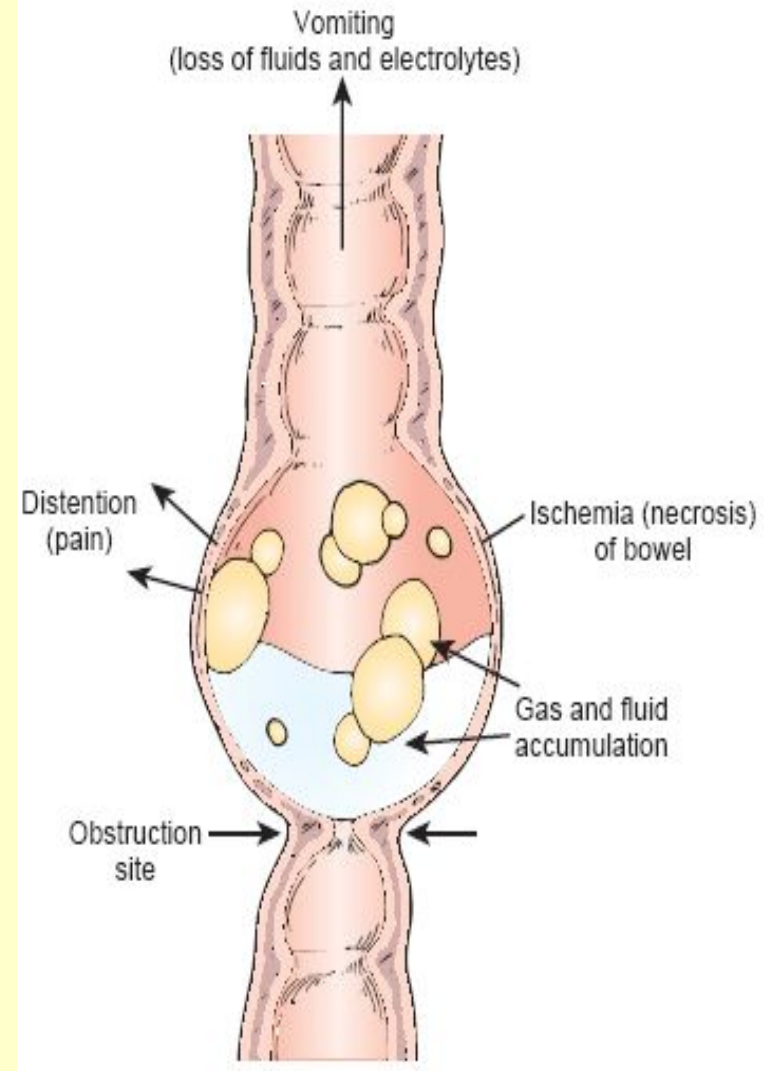
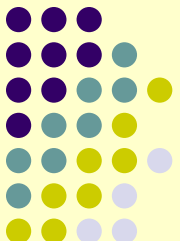


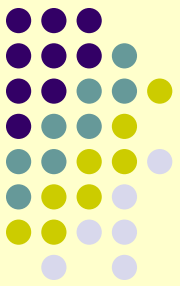
Intestinal obstruction

Paralytic, or adynamic, obstruction

- after abdominal surgery
- inflammatory conditions of the abdomen,
- pelvic and back injuries.
- chemical irritation (bile, bacterial toxins, electrolyte imbalances).

Intestinal obstruction pathogenesis



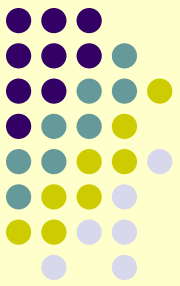


Intestinal autointoxication

poisoning of the organism by toxic substances from the bowels.

The causes and mechanisms:

- **formation of the toxic substances** - skatole, cresol, indole, phenol.
- **permeability of the intestinal wall** - inflammation and distension of bowels.
- **Hepatic failure** due to the decrease of the liver detoxication activity.

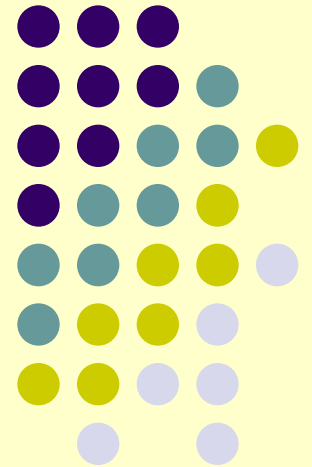


Intestinal autointoxication

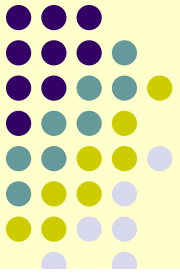
General symptoms

- ABP and pain sensitivity,
- glycogen amount in the liver, hyperglycaemia,
- myocardial dystrophy
- respiratory depression,
- headaches, brain activity inhibition up to coma
- appetite, violation of digestion, anemia.

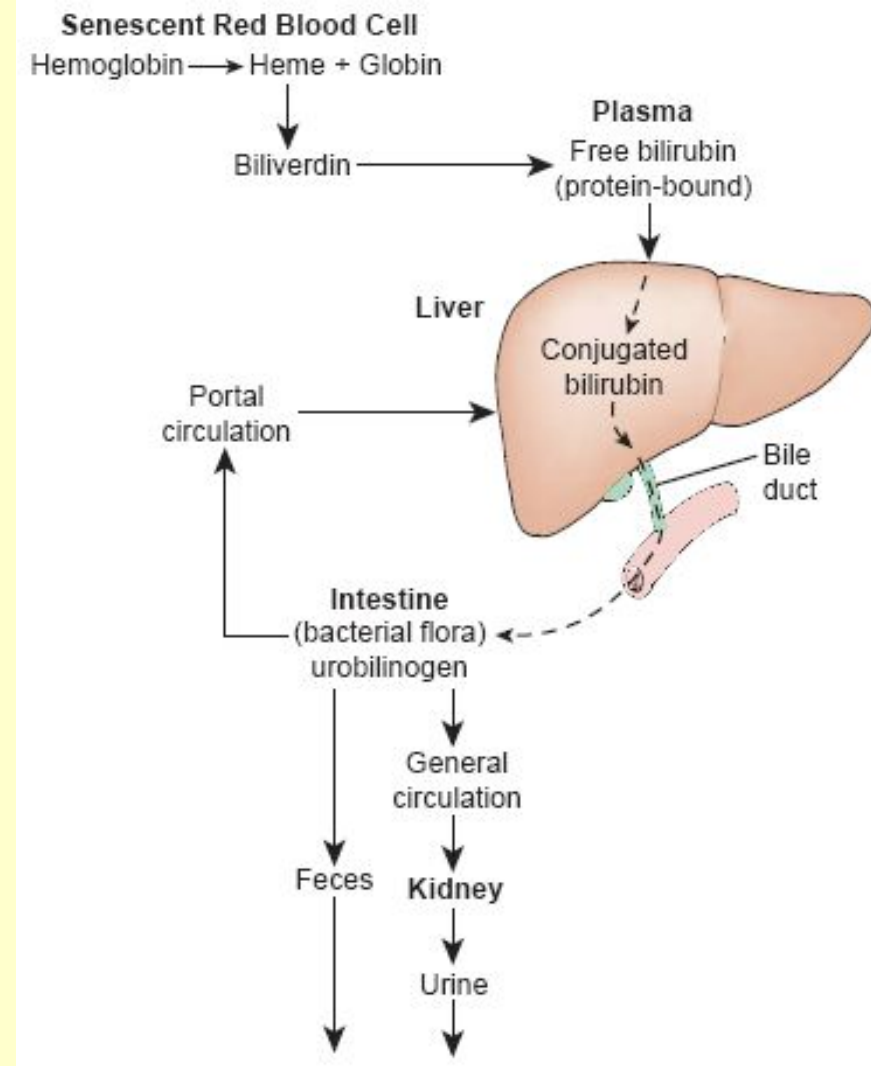
Liver pathology



Normal bilirubin metabolism



Unconjugated (free) bilirubin	Conjugated bilirubin
Undirect bilirubin	Direct bilirubin
Lipid-soluble	Water-soluble
Normally in plasma	Normally in bile
Not filtered by glomureli	Small percent filtered by kidney

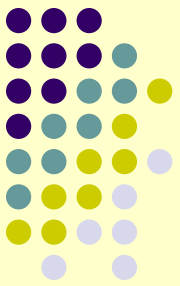


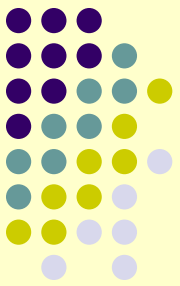
Jaundice

- Yellowish discoloration of the skin, mucosal surfaces and deep tissues

Reasons:

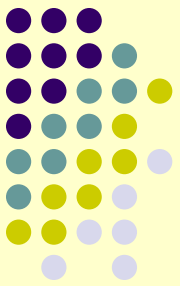
- destruction of RBC
- impaired uptake of bilirubin by liver cells
- conjugation
- bilirubin secretion





Prehepatic jaundice

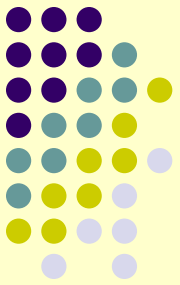
- Reason - hemolysis of red blood cells:
 - Hemolytic blood transfusion reaction
 - Hereditary and acquired hemolytic anemias
 - Neonatal jaundice (physiologic jaundice)
- Blood - unconjugated bilirubin
- Urine – urobiline normal or
- Faeces – stercobiline



Hepatic jaundice

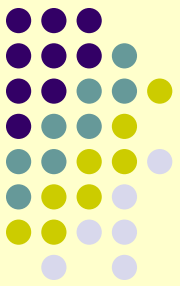
- Synonym – intrahepatic or hepatocellular jaundice
- Hepatitis, cirrhosis, cancer of the liver.
 - bilirubin uptake, conjugation, excretion
- Blood - unconjugated bilirubin , conjugated bilirubin
- Urine – urobilin normal or , bilirubin
- Faeces – stercobiline normal or

Hepatic jaundice



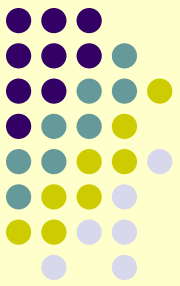
Hereditary disorders:

- bilirubin uptake (Gilbert's syndrome);
- of enzymes supporting conjugation (Crigler-Najjar syndrome);
- bilirubin excretion (Dubin-Johnson syndrome).



Posthepatic jaundice

- Synonym – mechanical, obstructive, cholestatic jaundice
- Reasons – obstruction of bile flow between the liver and the intestine
 - Structural disorders of the bile duct
 - Cholelithiasis
 - Tumors in the bile duct
- Blood - conjugated bilirubin \uparrow , bile salts, cholesterol
- Urine – urobilin absent, bilirubin \square
- Faeces – stercobiline absent

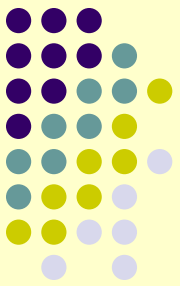


Cholemia - bile in blood

□ levels of cholesterol, bile acids and bilirubin

Clinical signs of cholemia:

- Urine dark color.
- Xanthomas formation (due to excess of cholesterol)
- Skin itching (pruritis)
- Arterial hypotension
- Bradycardia
- □ irritability and excitability of the patient
- Depression, insomnia, increased fatigueability
- Multiply subcutaneous hemorrhages

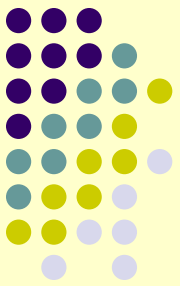


Acholia

- or absence of bile secretion into the intestines

Clinical signs of acholia:

- steatorrhea - fat, clay colored stools
- intestinal autointoxication and disbacteriosis development
- deficiency of fat soluble vitamins (A,D,E,K)



Hepatic failure

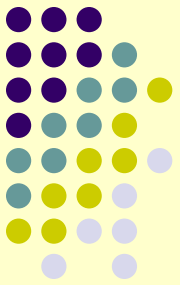
severe impairment of the liver functions

- Acute failure (fulminant hepatitis)
- Chronic failure (alcoholic liver cirrhosis)

Clinical signs

Fetor hepaticus - musty, sweetish odor of the breath in the patient with liver failure.

Hepatic failure



Hematologic Disorders.

- anemia due to
 - blood loss,
 - excessive destruction or impaired formation of RBC,
 - folic acid deficiency
- leukopenia, thrombocytopenia due to excessive destruction as the result of splenomegaly,
- coagulation defects due to □ protein synthesis by the liver, vitamin K deficiency

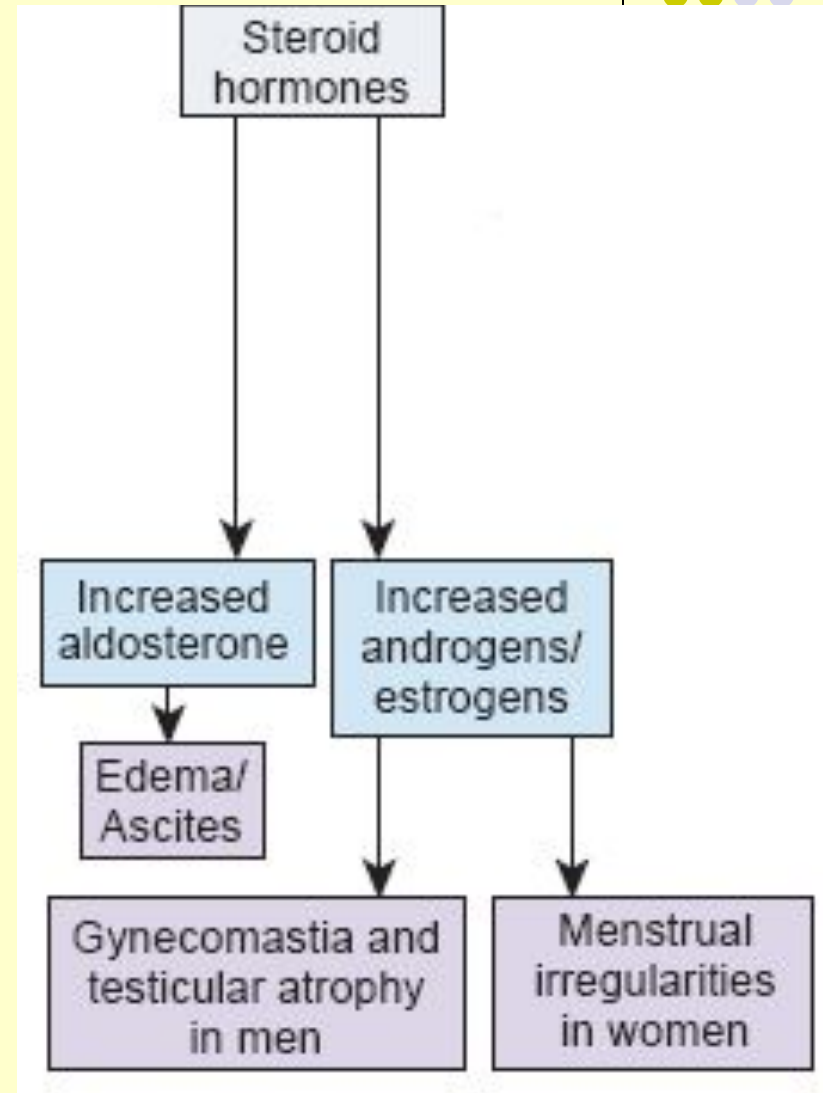
Hepatic failure

Endocrine Disorders

–impaired steroid hormones metabolism

Skin Disorders.

- telangiectases
- palmar erythema
- clubbing of the fingers
- jaundice



Hepatic failure

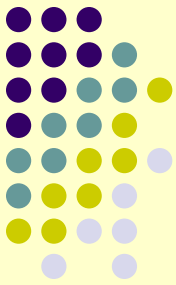
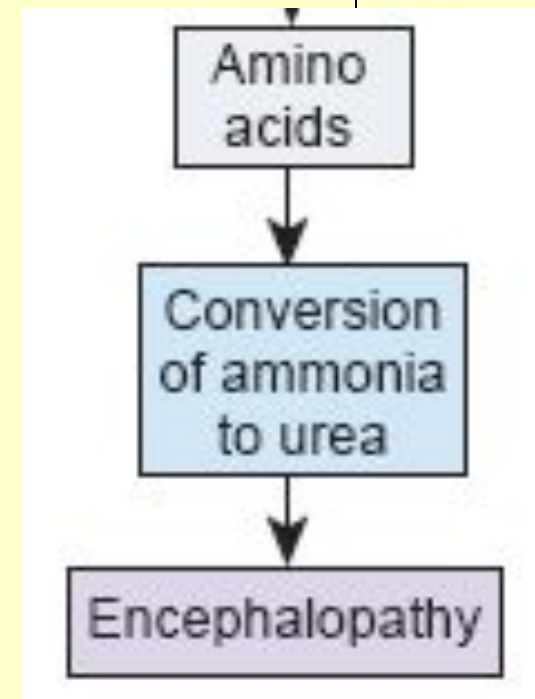
Hepatic Encephalopathy

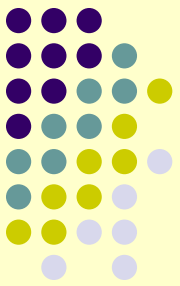
Stages I - IV (from irritability to coma)

- flapping tremor - asterixis;
- memory loss;
- personality changes;
- impaired speech and movements.

Pathogenesis - accumulation of neurotoxins.

- Ammonia enters general and cerebral circulation.
- Worsening after protein meals





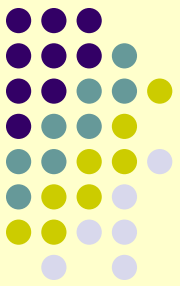
Hepatic failure

Hepatorenal Syndrome

- Acute liver failure kidneys hypoperfusion reduction in glomerular filtration rate kidney failure

Clinical signs:

- urine output (oliguria)
- blood urea, nitrogen and creatinine levels.
- renin secretion ABP

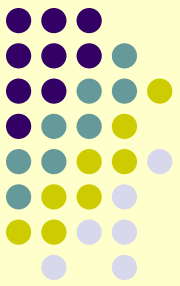


Portal hypertension

- □ resistance to flow in the portal venous system and □ portal vein pressure

Prehepatic portal hypertension:

- portal vein thrombosis
- external compression due to cancer or enlarged lymph nodes.



Portal hypertension

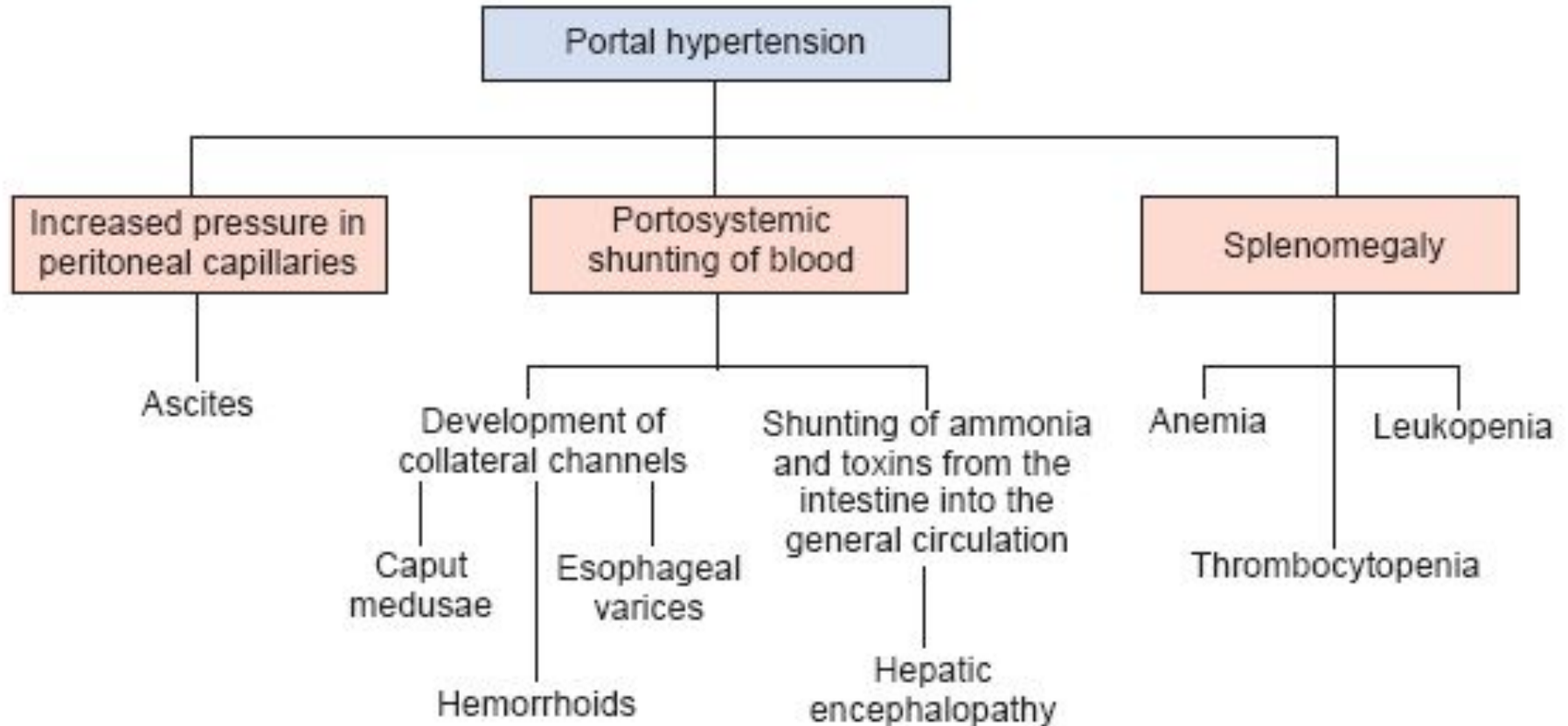
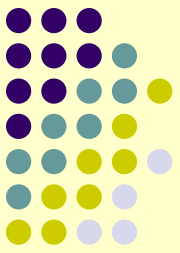
Intrahepatic portal hypertension:

- liver cirrhosis.
- infestation of the liver with schistosomes
- polycystic liver
- hepatic tumors.

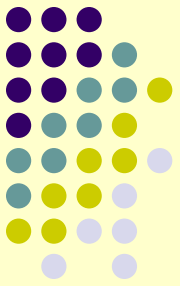
Post hepatic portal hypertension:

- thrombosis of the hepatic veins,
- severe right-sided heart failure
- Budd-Chiari syndrome
 - congestive disease of the liver caused by occlusion of the portal veins and their tributaries.

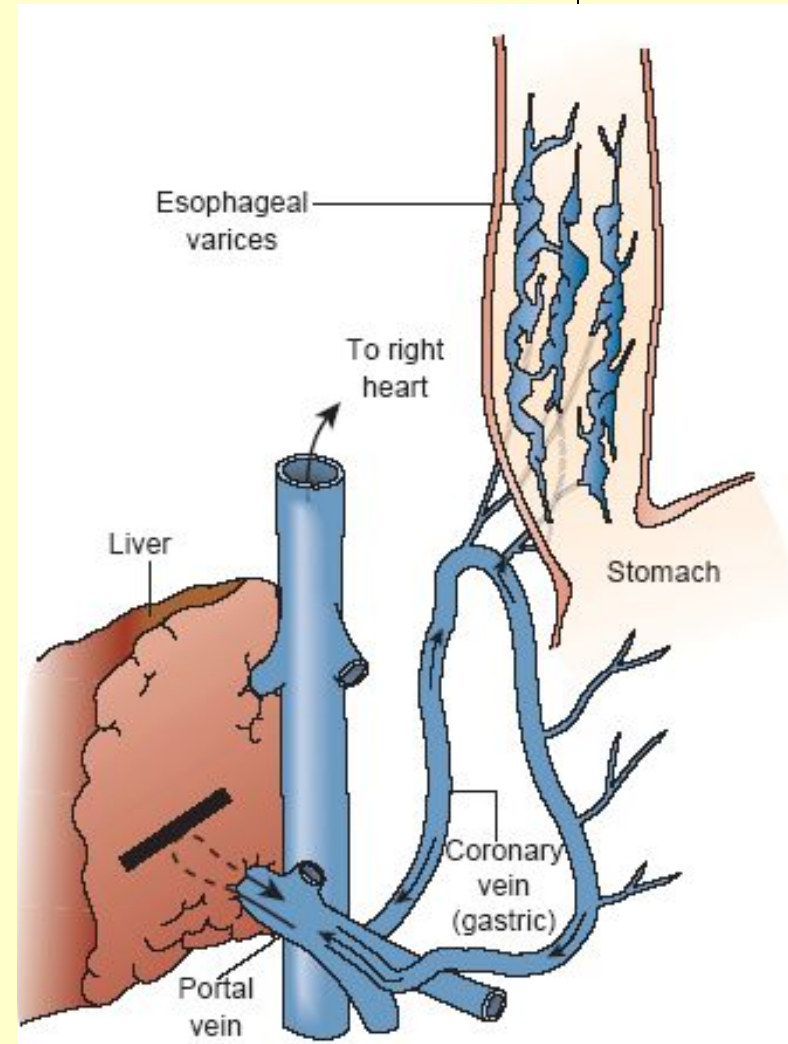
Complications of portal hypertension



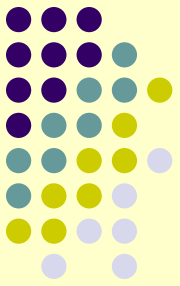
Complications of portal hypertension



- **Portosystemic Shunts.**
- **caput medusae** - dilated veins around the umbilicus
- portopulmonary shunts – results in cyanosis.
- *esophageal varices* - are subject to rupture, producing massive and sometimes fatal hemorrhage.



Hepatolienal syndrome



Enlargement of liver is usually combined with the enlargement of spleen due to:

- common vein system
- common innervation
- common lymphatic