

Liver cirrhosis

By

Dr/ zeinab hussien ali

Professor of medical surgical nursing



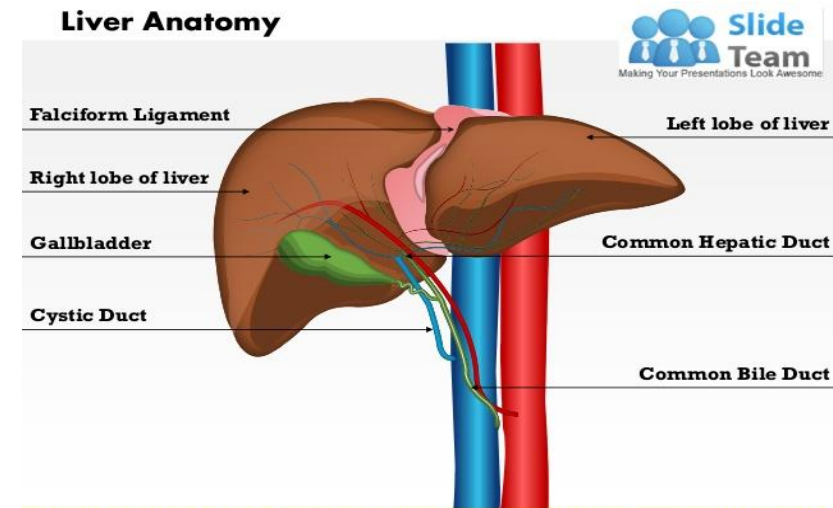


Introduction

- The liver is the largest gland of the body, and considered a chemical factory that manufactures, stores, alters, and excretes a large number of substances involved in metabolism. The location of the liver is essential in this function because it receives nutrient-rich blood directly from the gastrointestinal (GI) tract and then either stores or transforms these nutrients into chemicals that are used elsewhere in the body for metabolic needs.

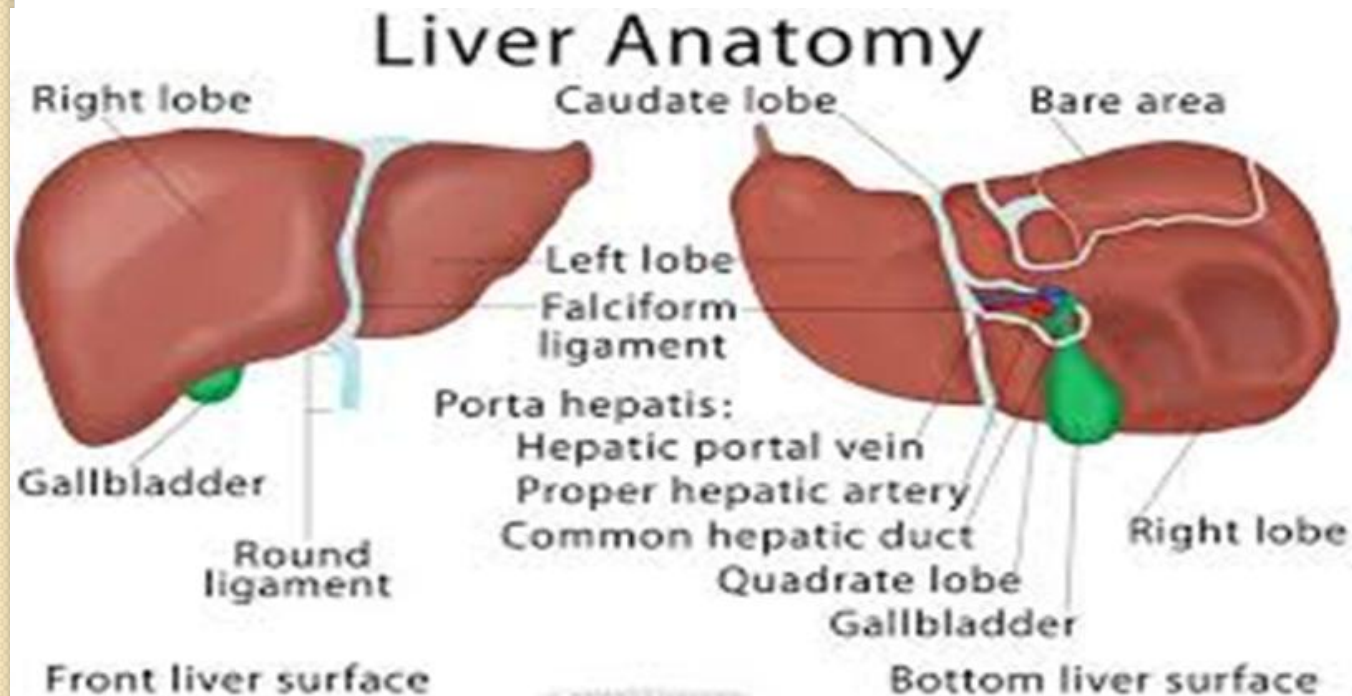
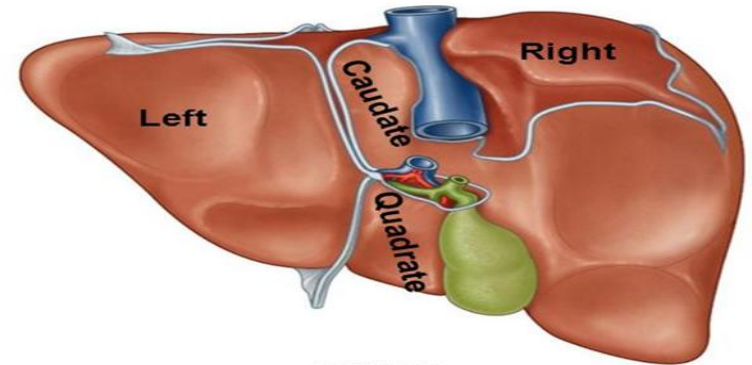
Anatomy of the Liver

- The liver is a large, highly vascular organ located behind the ribs in the upper right portion of abdominal cavity. It weighs between 1200 to 1500 g and is divided into four lobes. A thin layer of connective tissue surrounds each lobe, extending into the lobe itself and dividing the liver into small, functional units called lobules.



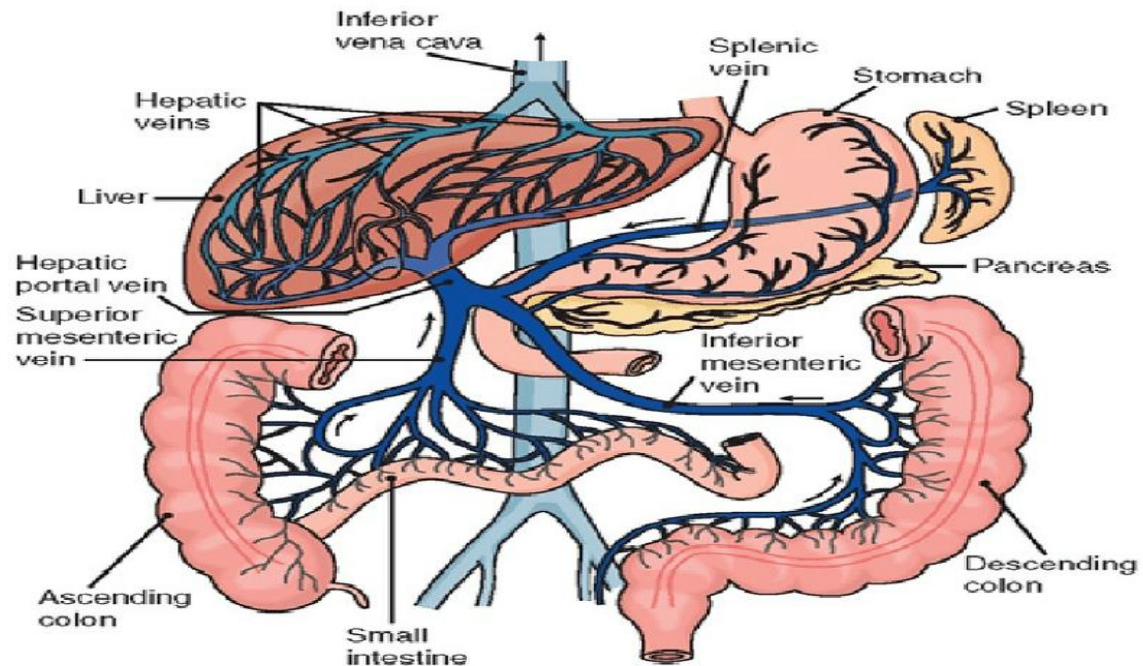
Name the 4 anatomical lobes of the liver

- Left
- Right
- Caudate
- Quadrate



Hepatic portal circulation:

Portal venous system drains blood to the liver from the abdominal part of the gastrointestinal canal, spleen, pancreas and gall bladder. Superior and inferior mesenteric veins join splenic vein to form portal vein which carries blood to liver, which in turn is drained by hepatic veins which pass into the IVC.



Inferior
vena cava

Capillary
bed in
liver

Hepatic
veins

Liver

Stomach

Large
intestine

Capillary
bed in
intestine

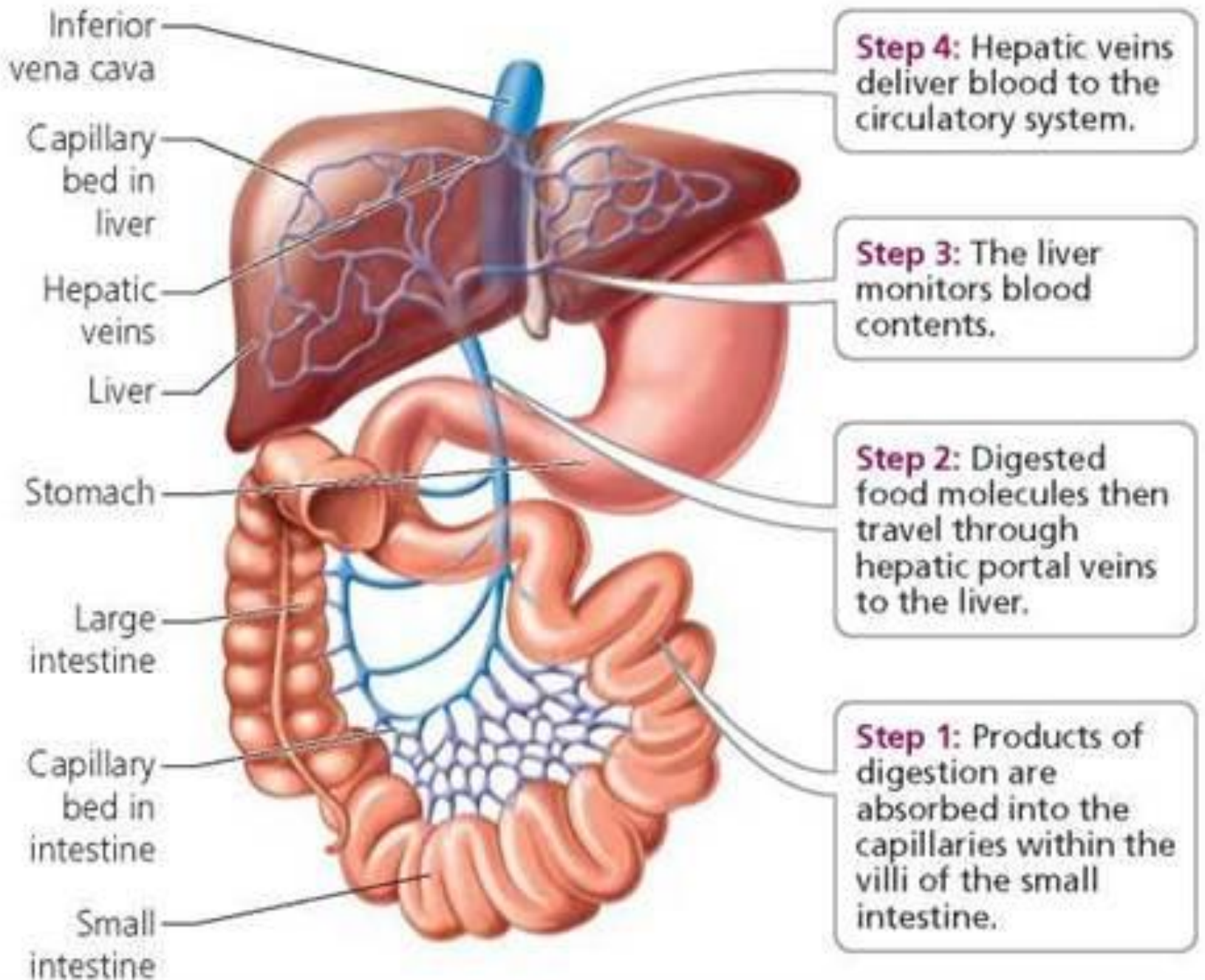
Small
intestine

Step 4: Hepatic veins
deliver blood to the
circulatory system.

Step 3: The liver
monitors blood
contents.

Step 2: Digested
food molecules then
travel through
hepatic portal veins
to the liver.

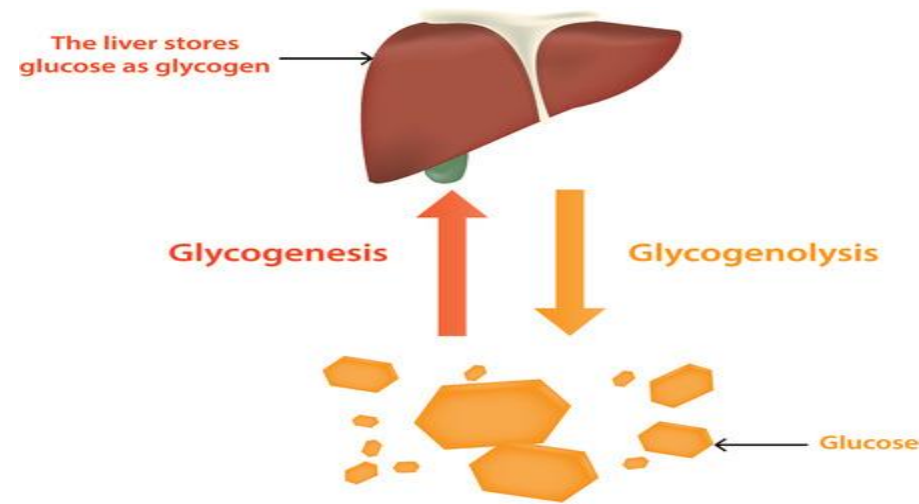
Step 1: Products of
digestion are
absorbed into the
capillaries within the
villi of the small
intestine.

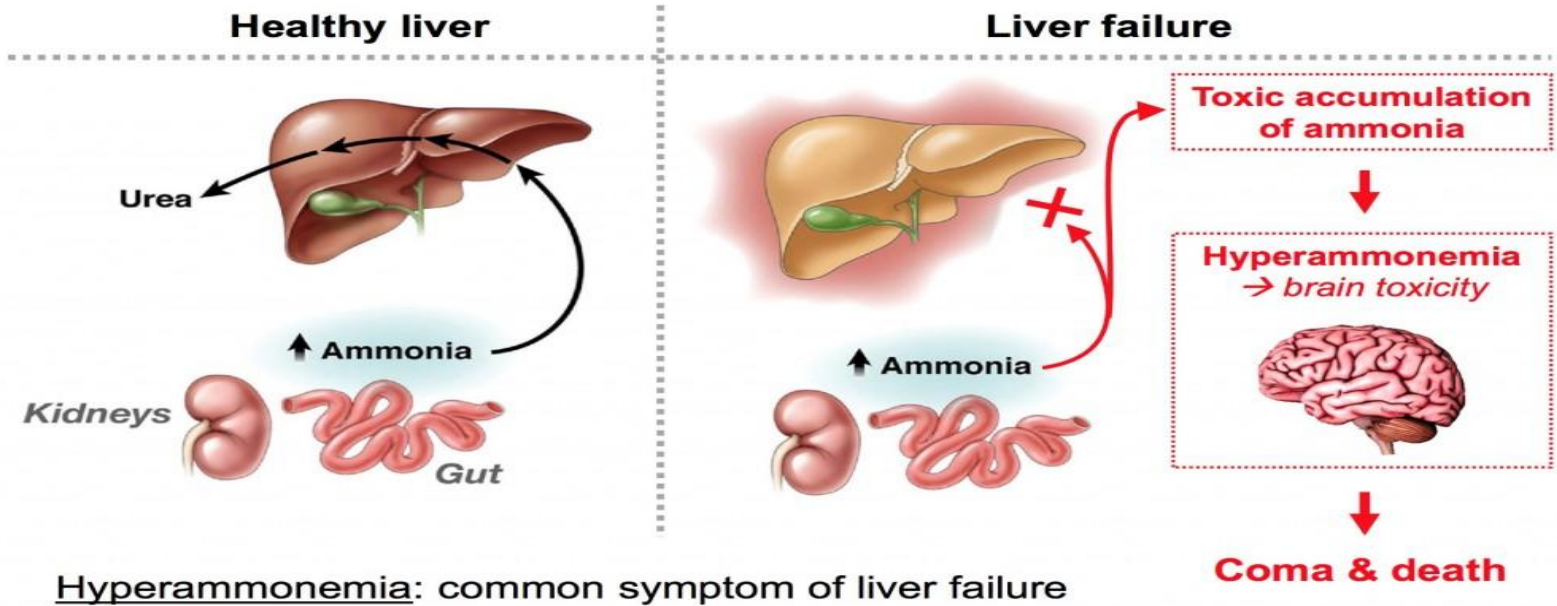


Functions of the Liver

1. Glucose Metabolism

- Glucose is taken up from the portal venous blood by the liver and converted into glycogen, which is stored in the hepatocytes. glycogen is converted back to glucose (**glycogenolysis**) and released as needed into bloodstream to maintain normal levels of blood glucose.





2. Ammonia Conversion

3. Protein Metabolism

- It synthesizes almost all of plasma proteins (except gamma-globulin), including albumin, alpha-globulins and beta-globulins, blood clotting factors, specific transport proteins, and most of the plasma lipoproteins

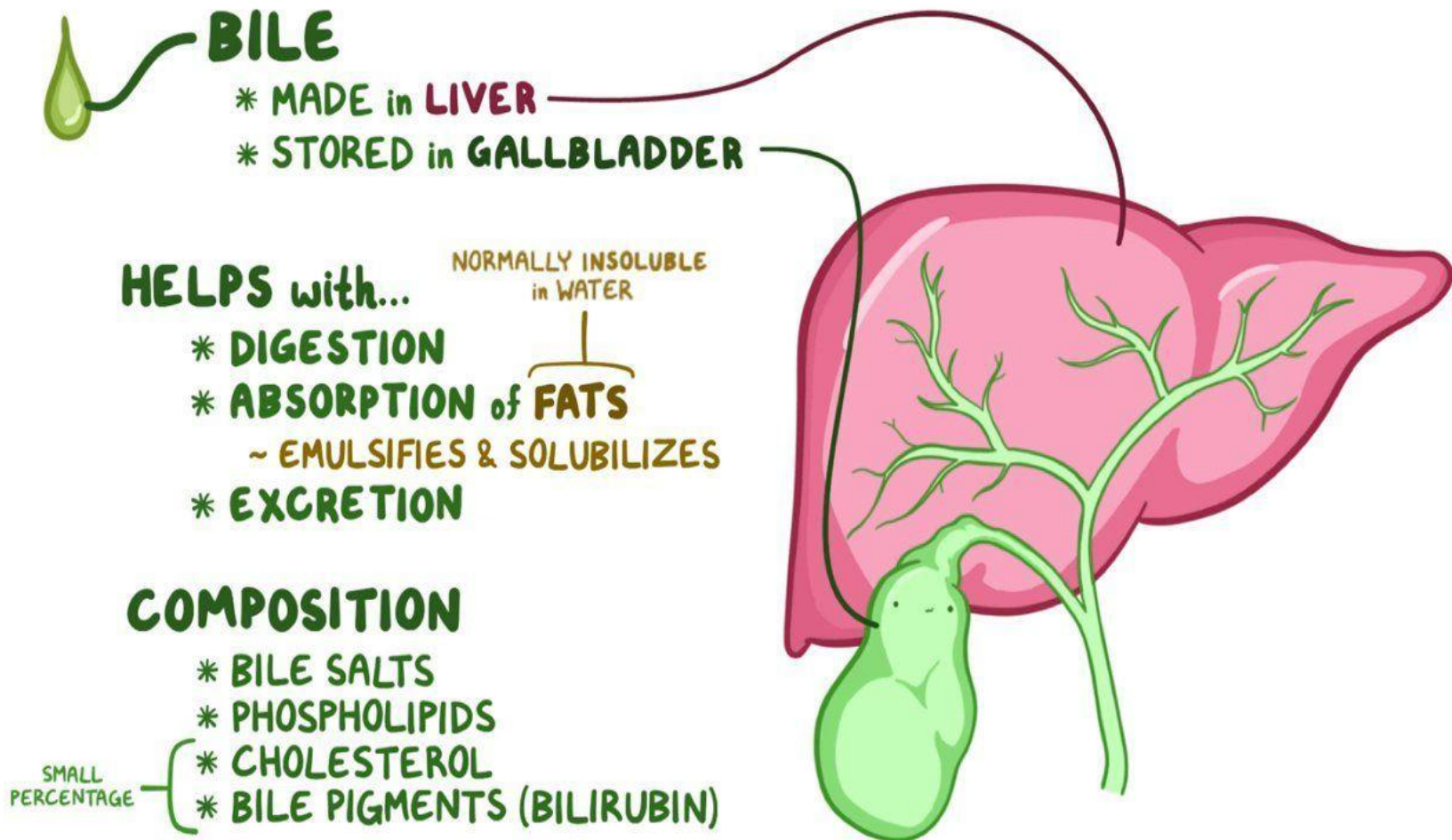
4. Fat Metabolism

- Fatty acids can be broken down for the production of energy and ketone bodies (acetoacetic acid, and acetone). Ketone bodies are small compounds that provide a source of energy for muscles and other tissues.

5. Vitamin and Iron Storage

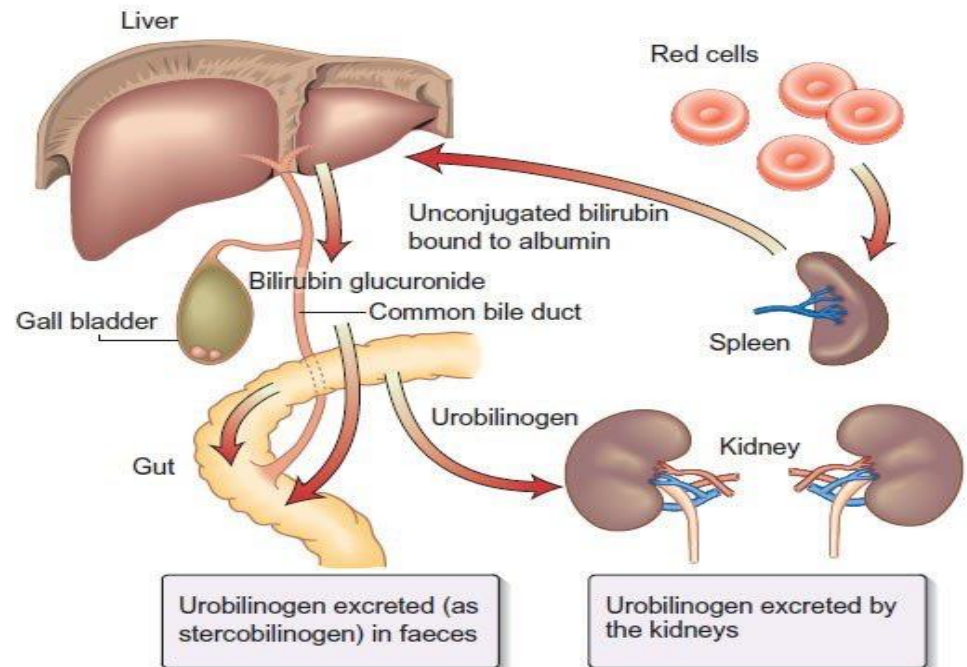
- Vitamins A, B, and D and several of the B-complex vitamins are stored in large amounts in the liver. Certain substances, such as iron and copper, are also stored.

6. Bile Formation

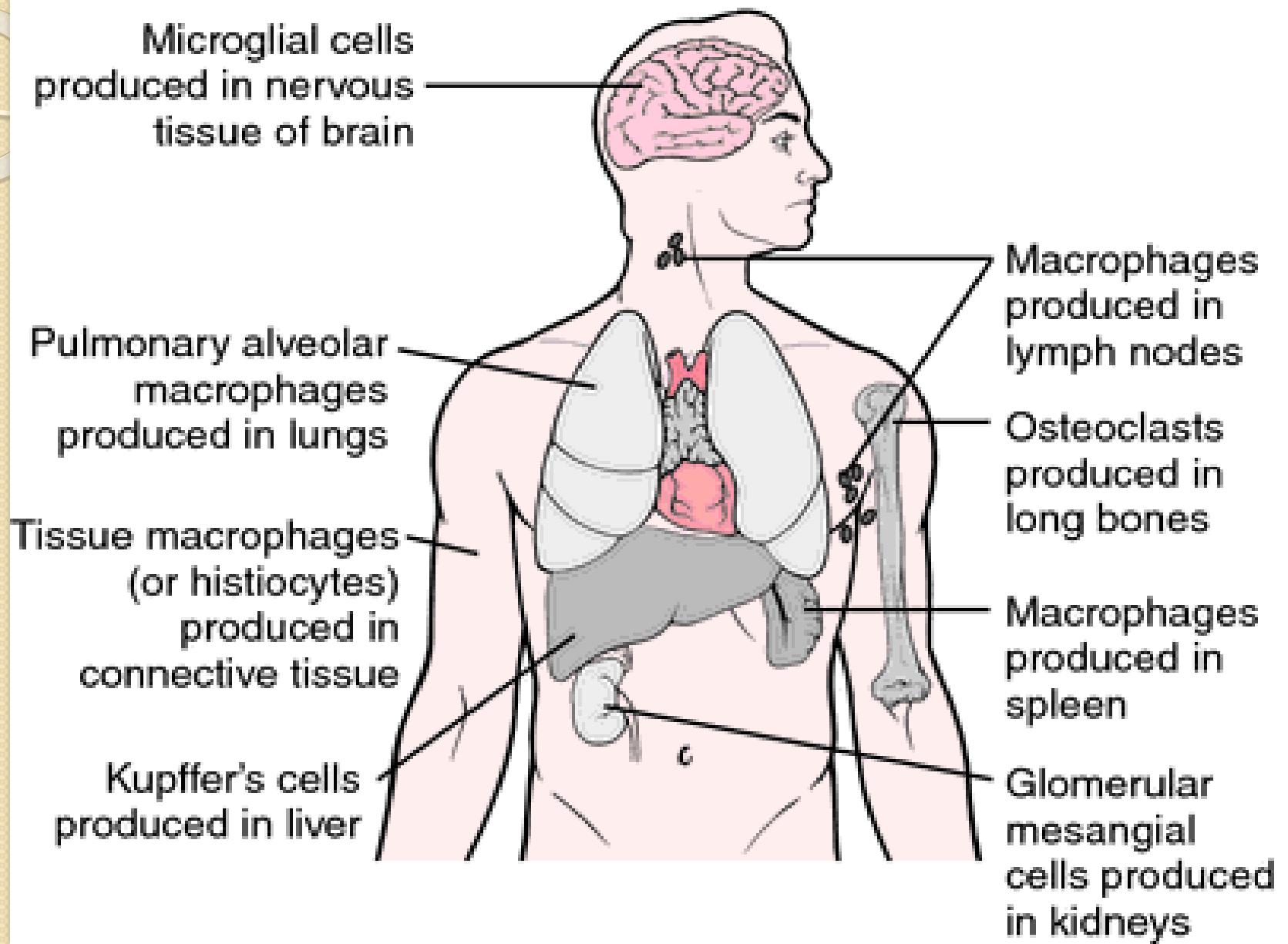


7. Bilirubin Excretion

- Bilirubin is a pigment derived from breakdown of hemoglobin by cells of reticuloendothelial system, including Kupffer cells of the liver. Hepatocytes remove bilirubin from blood and chemically modify it through conjugation to glucuronic acid, which make bilirubin more soluble in aqueous solutions.



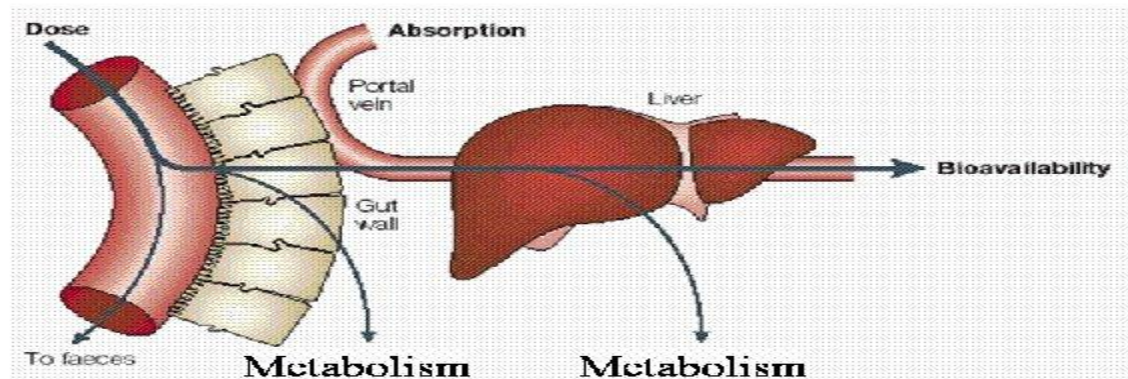
Reticuloendothelial system



8. Drug Metabolism

- The liver metabolizes many medications, such as barbiturates, opioids, sedatives, and anesthetics. Metabolism generally results in drug inactivation, although activation may also occur.

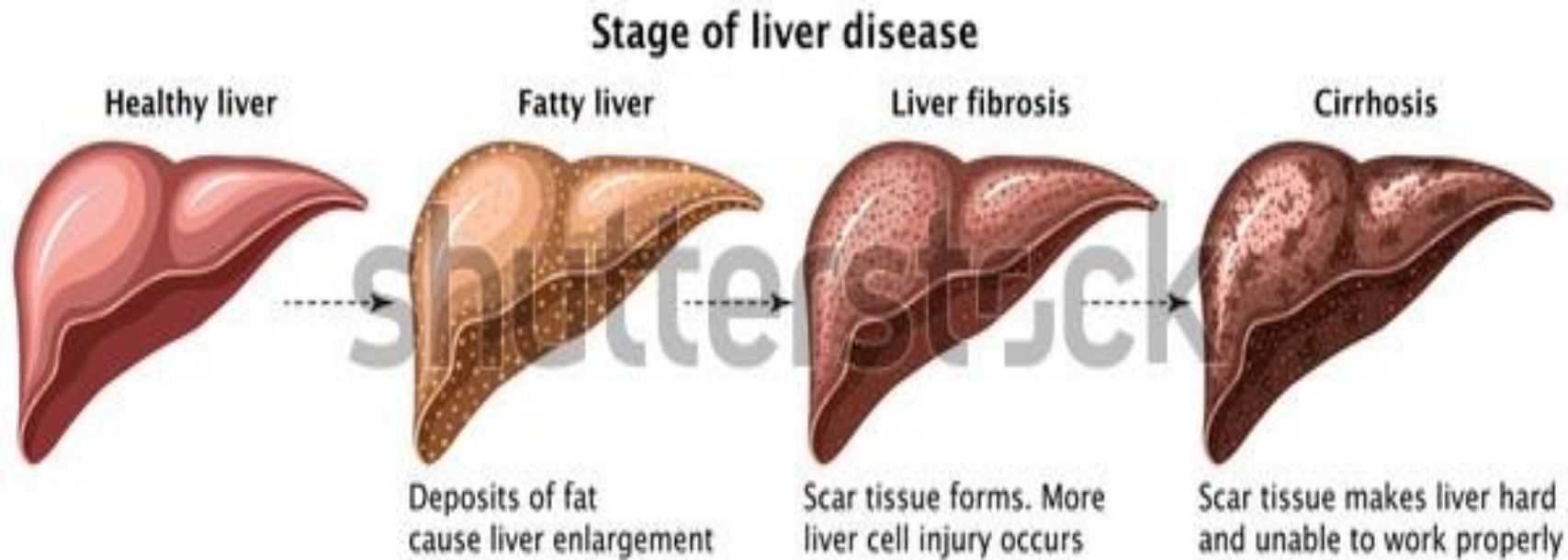
Drug Metabolism



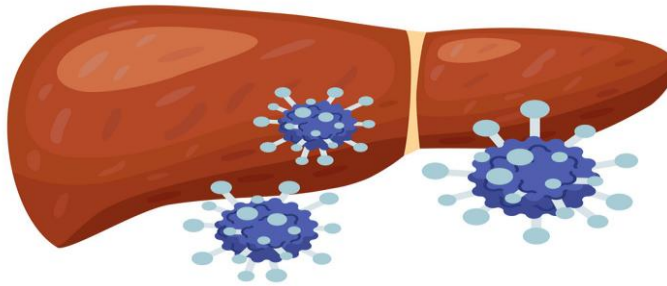
First pass metabolism

Hepatic (liver) cirrhosis

- **Cirrhosis** is a chronic disease characterized by replacement of normal liver tissue with diffuse fibrosis that disrupts structure and function of the liver.



Viral Hepatitis



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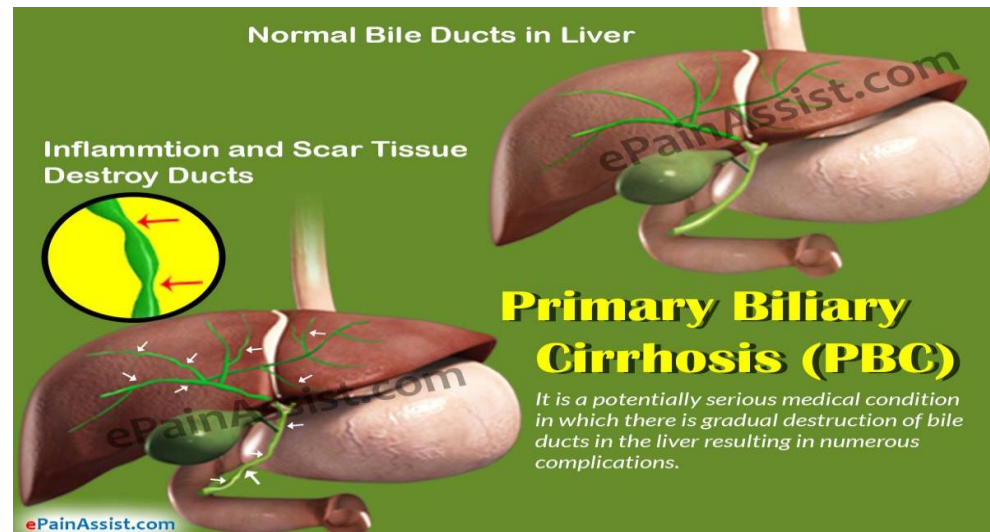
- 1) **Alcoholic cirrhosis**, in which the scar tissue surrounds the portal areas. This is most frequently caused by chronic alcoholism and is the most common type of cirrhosis.
- 2) **Post necrotic cirrhosis**, in which there are broad bands of scar tissues. This is a late result of a previous attack of acute viral hepatitis.



Scar tissue



Healthy tissue



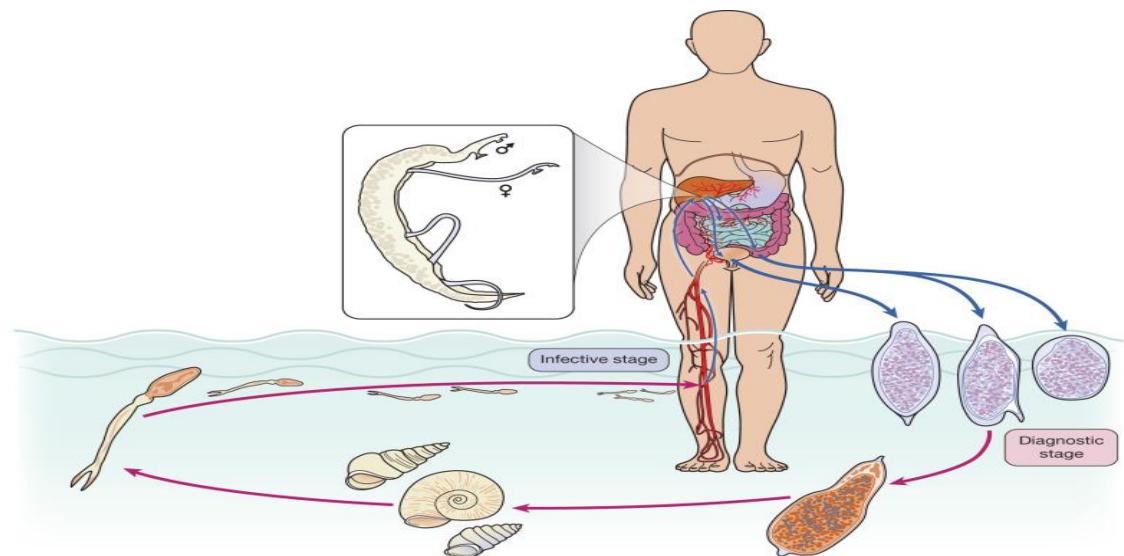
3) **Biliary cirrhosis:**

in which scarring occurs in the liver around bile ducts. This type of cirrhosis usually results from chronic biliary obstruction and infection; it is much less common than the other two types.

3) **Cardiac Cirrhosis:** results from long standing severe right-sided heart failure

Causes of cirrhosis

- ➡ Alcohol consumption is considered the major causative factor.
- ➡ Exposure to certain chemicals (carbon tetrachloride, arsenic, or phosphorus)
- ➡ Infectious schistosomiasis.



Clinical Manifestations

Signs and symptoms of cirrhosis increase in severity as disease progresses. It can be categorized as **compensated or decompensated cirrhosis**.

- **Compensated cirrhosis**

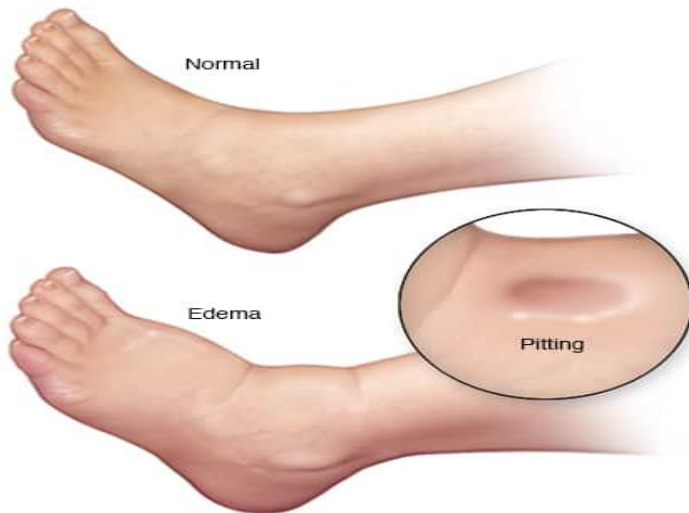
- Intermittent mild fever
- Vague morning indigestion
- Flatulent dyspepsia
- Abdominal pain
- Ankle edema
- Vascular spiders
- Palmar erythema (reddened palms in alcohol abuse)
- Unexplained epistaxis
- Firm, enlarged liver
- Splenomegaly.



Vascular spiders



Palmar erythema

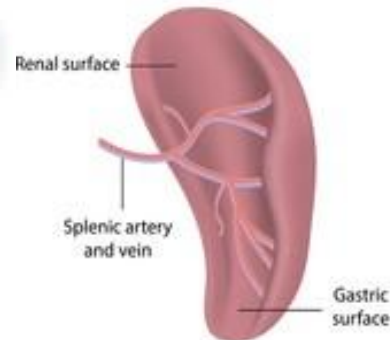


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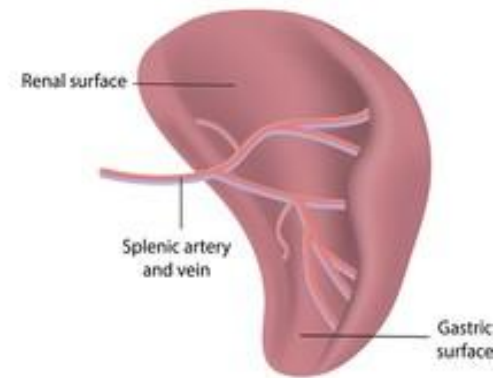
Ankle edema

Splenomegaly

Healthy Spleen



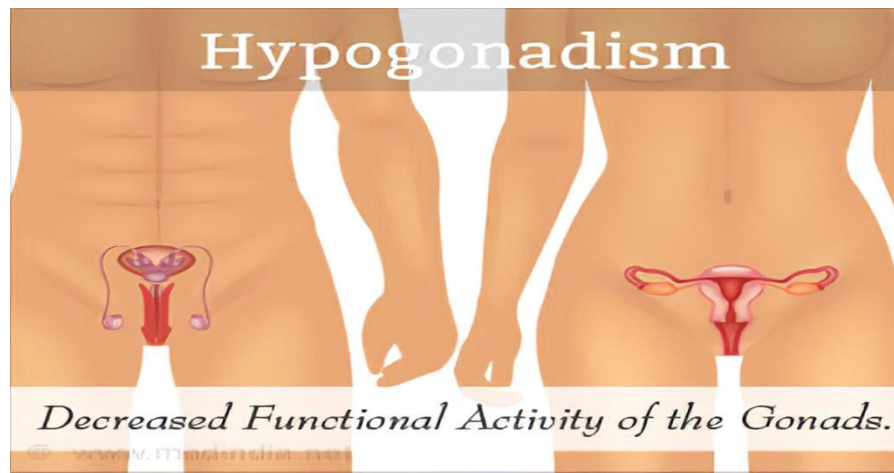
Enlarged Spleen

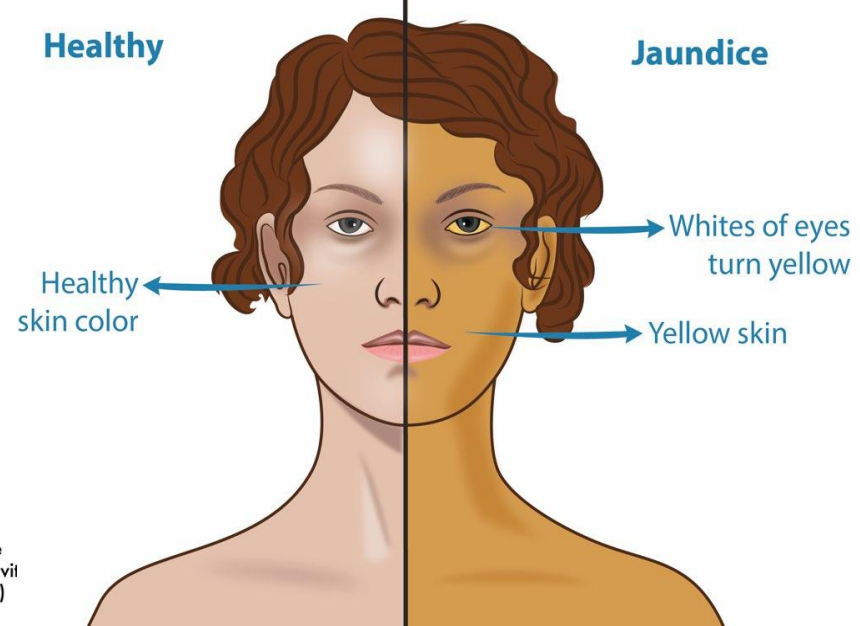
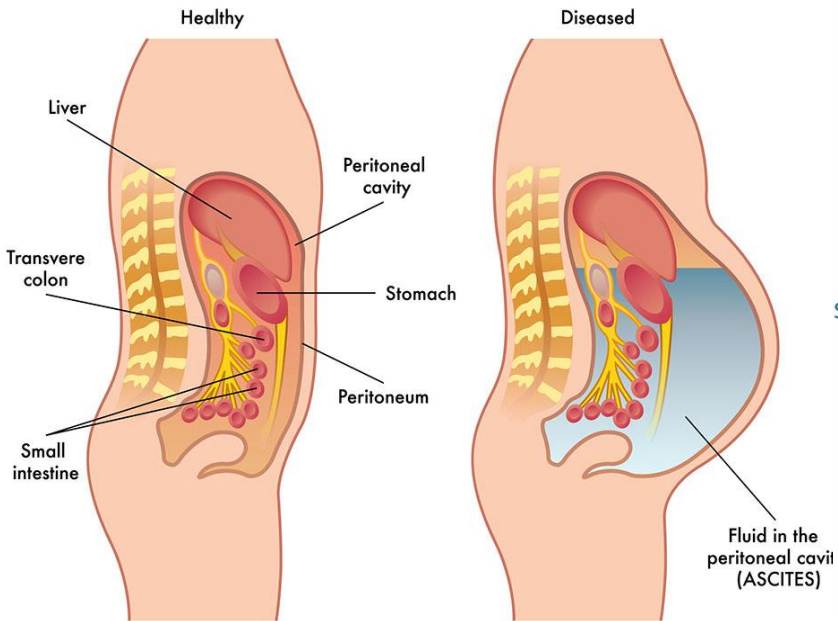


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- **Decompensated cirrhosis**

- Weakness
- Muscle wasting
- Weight loss
- Continuous mild fever
- Hypotension
- Clubbing of fingers
- Purpura (due to decreased platelet count)
- Spontaneous bruising
- Epistaxis
- Sparse body hair
- White nails
- Gonadal atrophy
- Ascites
- Jaundice





Clubbing of fingers

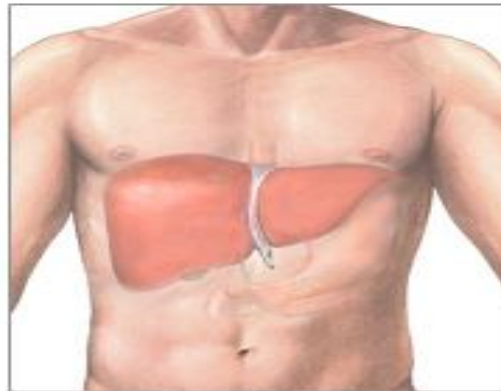


Purpura

Liver Enlargement

• Early in the course of cirrhosis, the liver tends to be large, and the cells are loaded with fat. Abdominal pain may be present because of recent, rapid enlargement of the liver. Later in the disease, the liver decreases in size as scar tissue contracts the liver tissue.

Normal liver



Enlarged liver due to hepatomegaly

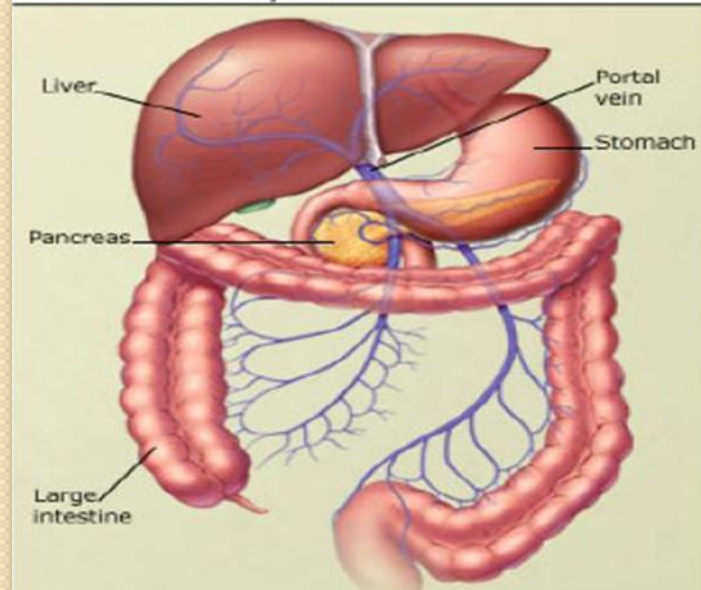


Portal hypertension

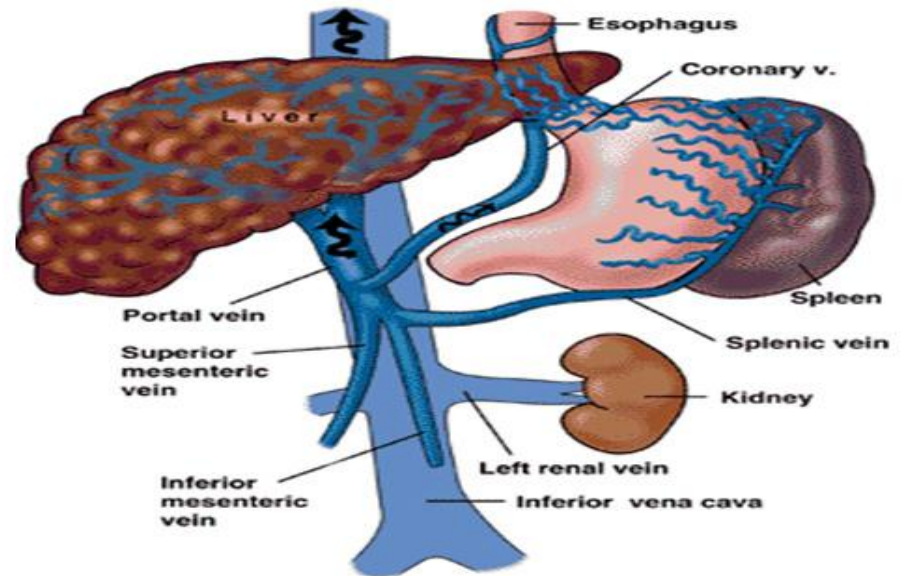
High blood pressure in the veins that supply the liver.

Cirrhosis slows the normal flow of blood through the liver, thus increasing pressure in the vein that brings blood to the liver from the intestines and spleen.

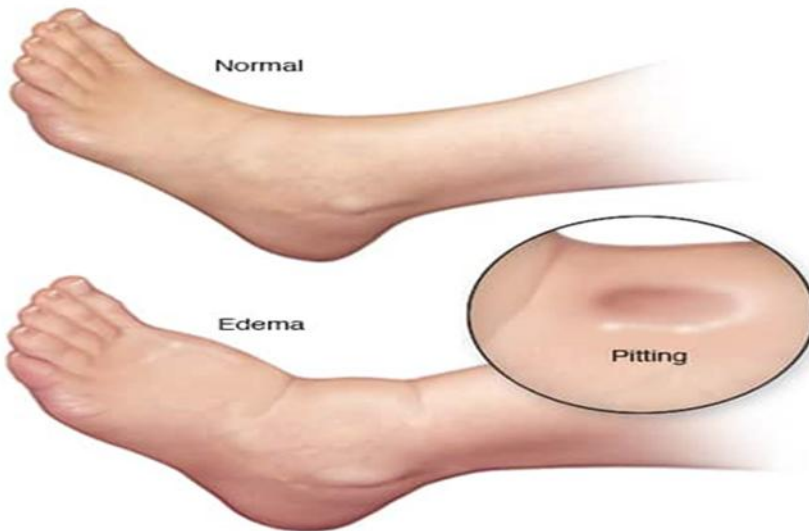
Portal Venous System



Portal Hypertension



⇒ ***Swelling in the legs and abdomen.*** The increased pressure in the portal vein can cause fluid to accumulate in the legs (edema) and in the abdomen (ascites). Edema and ascites also may result from the inability of the liver to make enough of certain blood proteins, such as albumin.

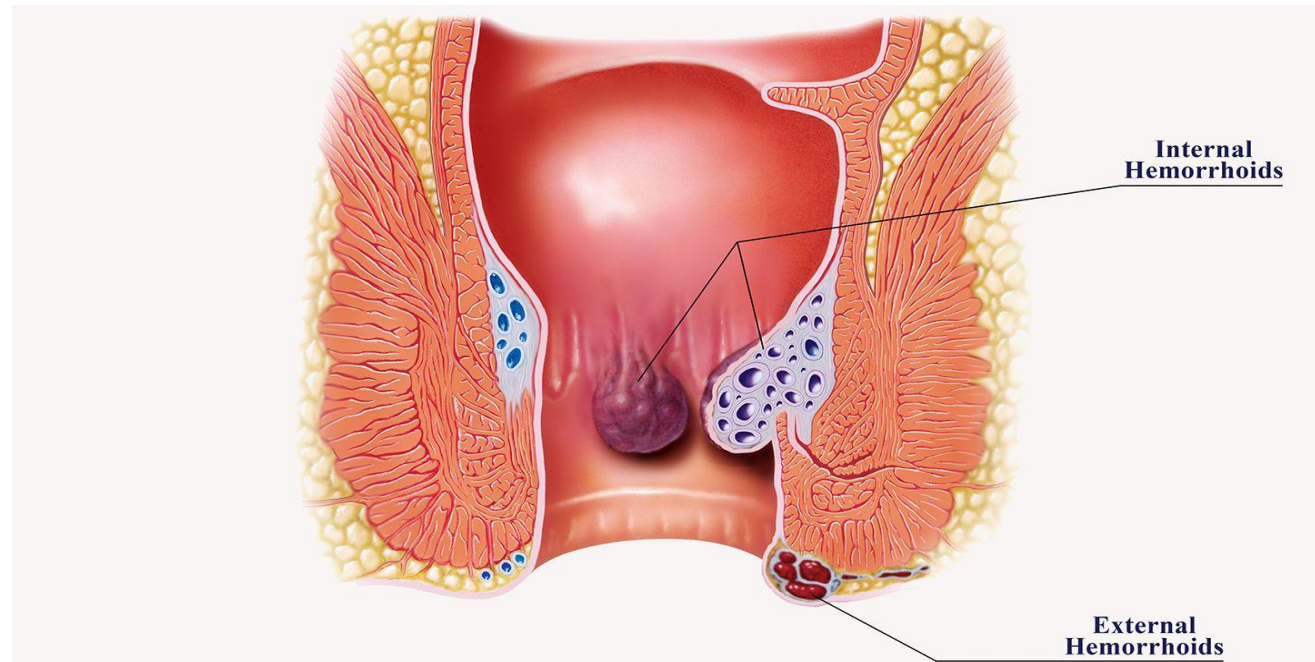
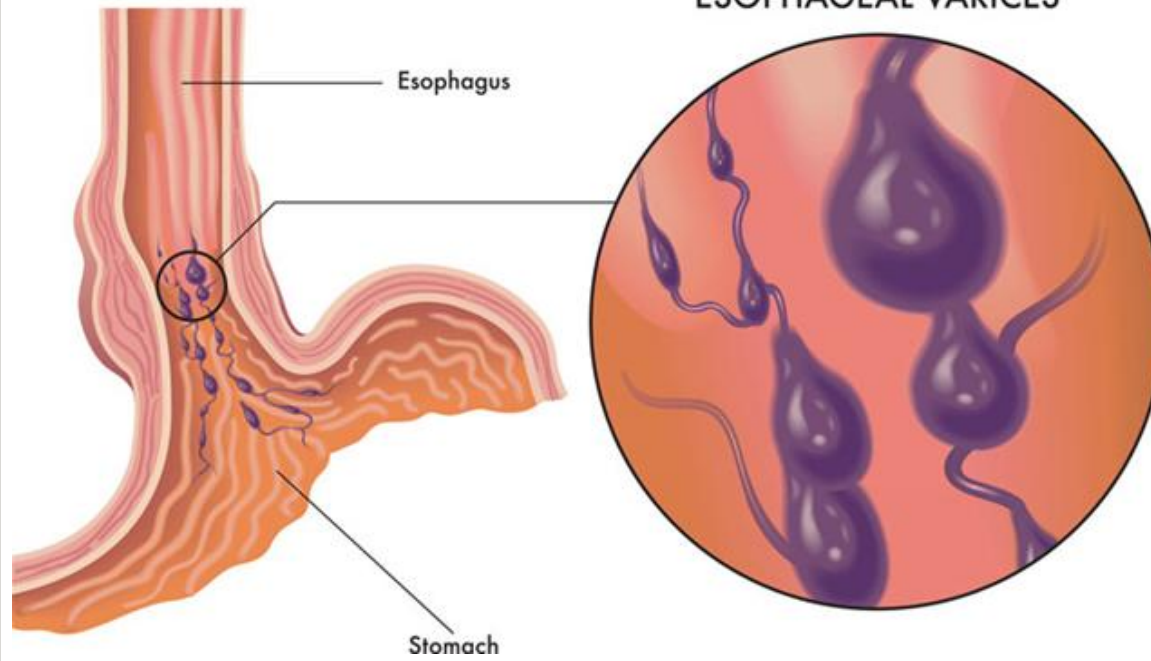


Gastrointestinal Varices

- Portal hypertension cause prominent, distended abdominal blood vessels, which are visible on abdominal inspection (caput medusae) and distended blood vessels throughout the GI tract in the esophagus (esophageal varices) or the stomach (gastric varices), and lower rectum (hemorrhoids). These are common sites of collateral blood vessels.



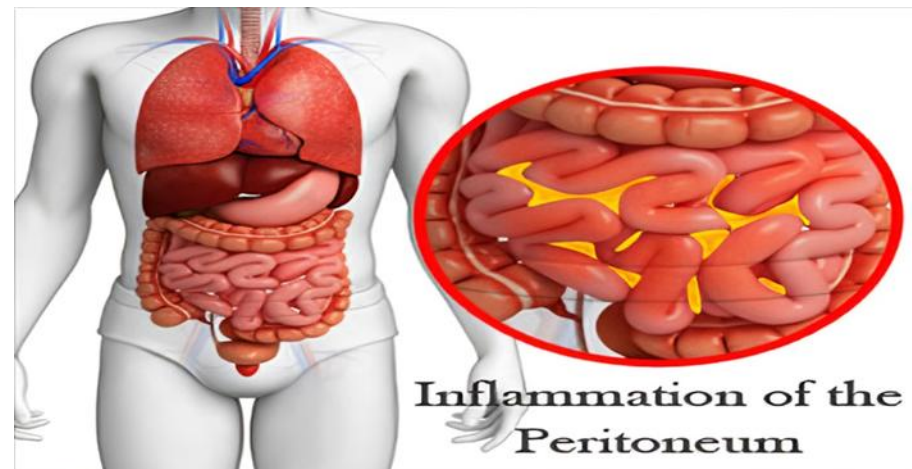
ESOPHAGEAL VARICES



Bleeding

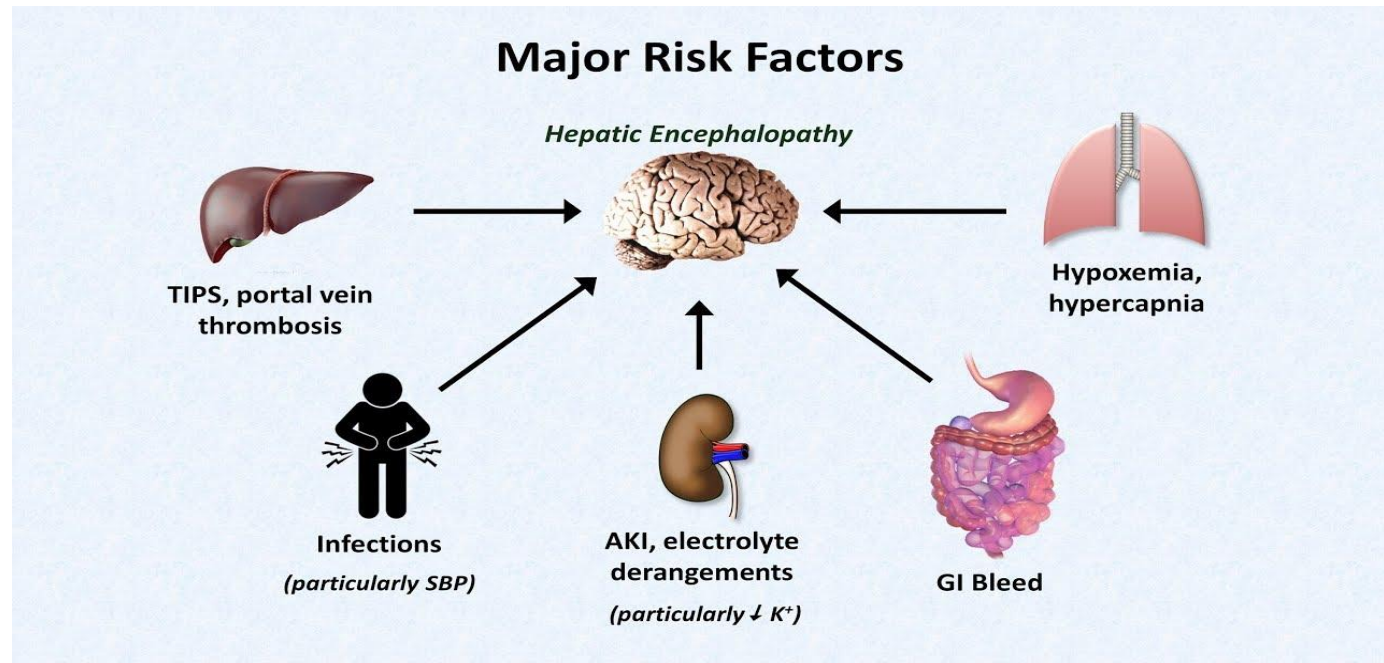
- ⇒ Portal hypertension causes blood to be redirected to smaller veins that are strained by extra pressure and can burst, causing serious bleeding. If liver can't make enough clotting factors, this also can contribute to continued bleeding.

Infections. In cirrhosis, the body may have difficulty fighting infections. Ascites can lead to spontaneous bacterial peritonitis (SBP).



Hepatic Encephalopathy

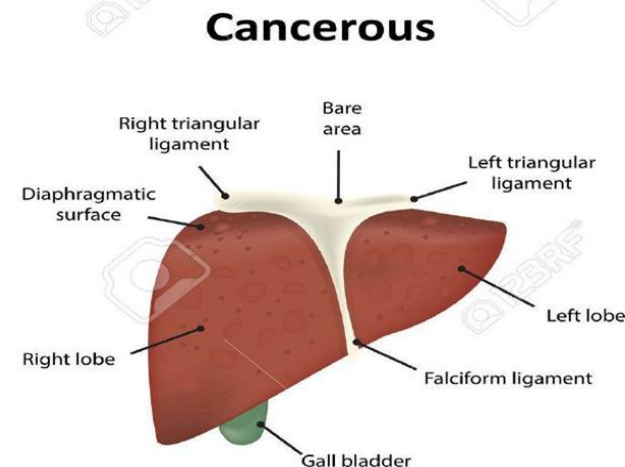
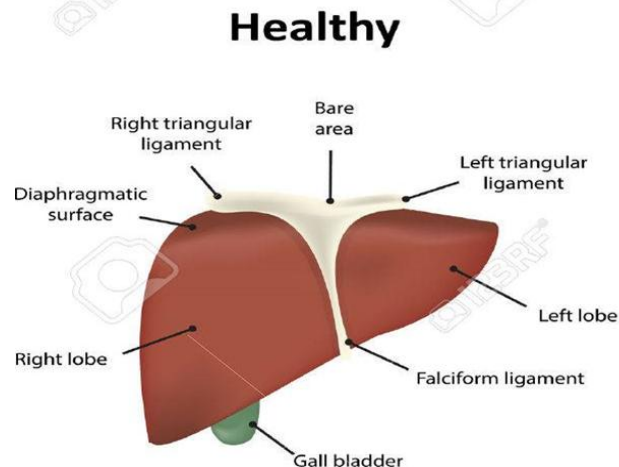
➤ **Buildup of toxins in the brain.** A liver damaged by cirrhosis isn't able to clear toxins from the blood. These toxins can then build up in the brain and cause mental confusion and difficulty concentrating. With time, hepatic encephalopathy can progress to coma.



⇒ **Jaundice** occurs when the diseased liver doesn't remove enough bilirubin from the blood. Jaundice causes yellowing of the skin and whites of the eyes and darkening of urine.

⇒ **Hepatocellular carcinoma.** A large proportion of people who develop liver cancer have pre-existing cirrhosis.

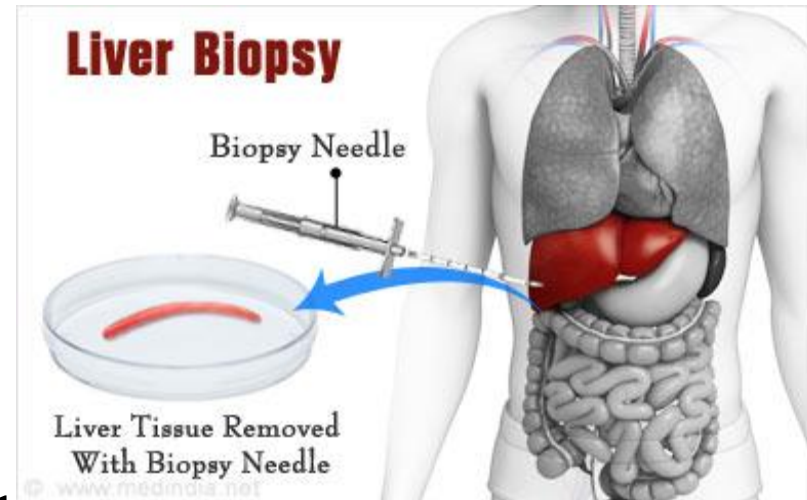
Hepatocellular Carcinoma



Assessment and Diagnostic Findings

- Liver function test: the serum albumin level tends to decrease. Enzyme tests indicate liver cell damage: serum alkaline phosphatase, and levels increase.

- Bilirubin tests are performed to measure bile excretion or retention.
- Prothrombin time
- Ultrasound scanning
- CT, MRI, and radioisotope liver scans.
- Liver biopsy.
- Testing of stool for occult blood



Medical Management

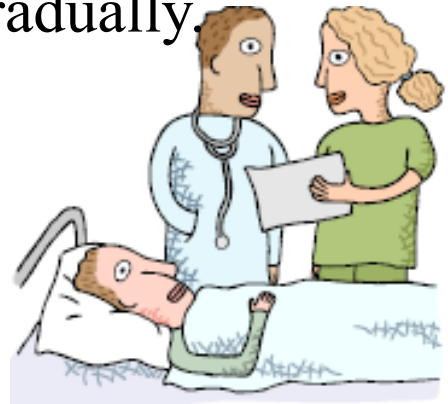
1. Antacids or histamine-2 (H₂) antagonists to decrease gastric distress and minimize the possibility of GI bleeding.
2. Vitamins and nutritional supplements promote healing of damaged liver cells and improve nutritional status.
3. Potassium-sparing diuretics may be indicated to decrease ascites.
4. An adequate diet and avoidance of alcohol are essential.
5. Herb milk thistle (*Silybum marianum*) to treat jaundice.
6. Silymarin has anti-inflammatory and antioxidant properties that may have beneficial effects, especially in hepatitis.

Nursing Management

Promoting Rest

To permit the liver to reestablish its function, it includes:

- Measure and record weight and fluid intake and output daily.
- Adjust the patient's position in bed for maximal respiratory efficiency.
- Oxygen therapy may be required in liver failure to oxygenate the damaged cells and prevent further cell destruction.
- Encourages the patient to increase activity gradually.



Improving Nutritional Status

- Provide a high-protein diet, for patient without ascites, edema, or signs of hepatic coma , supplemented by vitamins of B complex, as well as A, C, and K.
- Encourages the patient to eat small, frequent meals.
- Consider patient preferences
- Folic acid and iron are prescribed to prevent anemia.
- Protein is restricted if encephalopathy develops.
- Incorporating vegetable protein to meet protein needs may decrease the risk for encephalopathy.
- Sodium restriction to prevent ascites.



Providing Skin Care

- Providing careful skin care because of subcutaneous edema, the patient's immobility, jaundice, and increased susceptibility to skin breakdown and infection.
- Frequent changes in position are necessary to prevent pressure ulcers.
- Avoid irritating soaps and the use of adhesive tape prevent trauma to the skin.

Reducing Risk of Injury

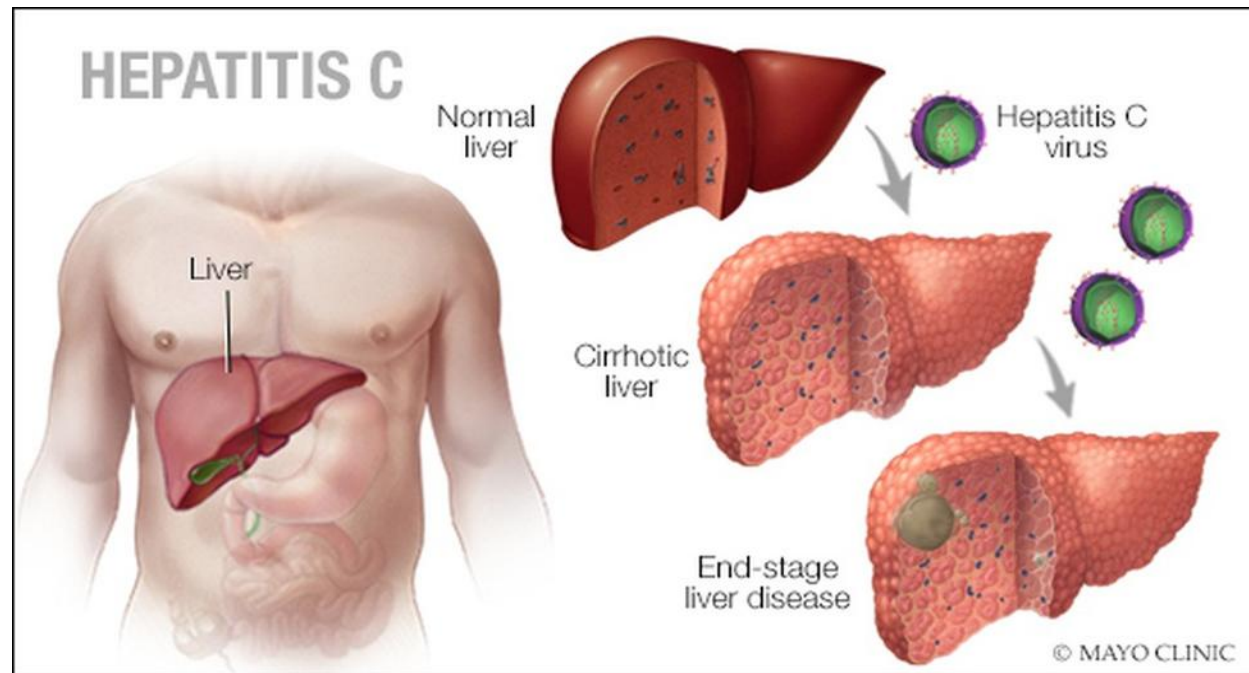
- Side rails should be in place and padded with blankets or other materials in case patient becomes agitated.
- Orients patient to time and place and explains all procedures to minimize agitation.
- Instruct patient to ask for assistance to get out of bed.
- Patient should use electric razor rather than safety razor.
- Soft-bristled toothbrush helps minimize bleeding gums,
- Apply pressure to vein puncture sites to minimize bleeding.

Viral hepatitis

- Viral hepatitis is a systemic, viral infection in which necrosis and inflammation of liver cells produce a characteristic cluster of clinical, biochemical, and cellular changes.
- Five definitive types of viral hepatitis have been identified: hepatitis A, B, C, D, and E. Hepatitis A and E are similar in mode of transmission (fecal–oral route), whereas hepatitis B, C, and D share many other characteristics.

Hepatitis C virus

- **Hepatitis C** is a liver disease caused by the hepatitis C virus: the virus can cause both acute and chronic hepatitis, ranging in severity from a mild illness lasting a few weeks to a serious, lifelong illness.



Risk factors for Hepatitis C

- Injecting drug through sharing of injection equipment.
- Reuse or inadequate sterilization of medical equipment.
- Transfusion of unscreened blood and blood products or organ transplant.
- Multiple sex partners, sexually transmitted disease.
- Children born to women infected with hepatitis C virus.
- Past treatment with hemodialysis.



Symptoms

- Incubation period for hepatitis C is **2 weeks to 6 months**. Following initial infection, approximately 80% of people do not exhibit any symptoms. Patients who are symptomatic **may exhibit:**
- Fever and fatigue.
- Decreased appetite, nausea, and vomiting.
- Abdominal pain and Joint pain.
- Dark urine and grey-coloured feces.
- Jaundice (yellowing of skin and the whites of the eyes).

Screening and diagnosis

- Hepatitis C is diagnosed by blood tests. There are two types of tests to confirm hepatitis C infection
 1. Antibody test : this detects antibodies against hepatitis C virus (anti-HCV) and is the most commonly used test.
 2. Hepatitis C PCR test: Also known as **Hepatitis C RNA test or viral RNA test**: A PCR (polymerase chain reaction) test in a pathology laboratory is necessary to see if the virus is still present and whether the person is still likely to be infectious. Hepatitis C viral RNA can be detected within 1 to 2 weeks of exposure.

Primary prevention interventions recommended by WHO:

- Hand hygiene: surgical hand preparation, hand washing and gloving.
- Safe and appropriate use of health care injections.
- Safe handling and disposal of sharps and waste.
- Testing of donated blood for hepatitis B and C.
- Training of health care personnel.
- Promotion of correct and consistent use of condoms.

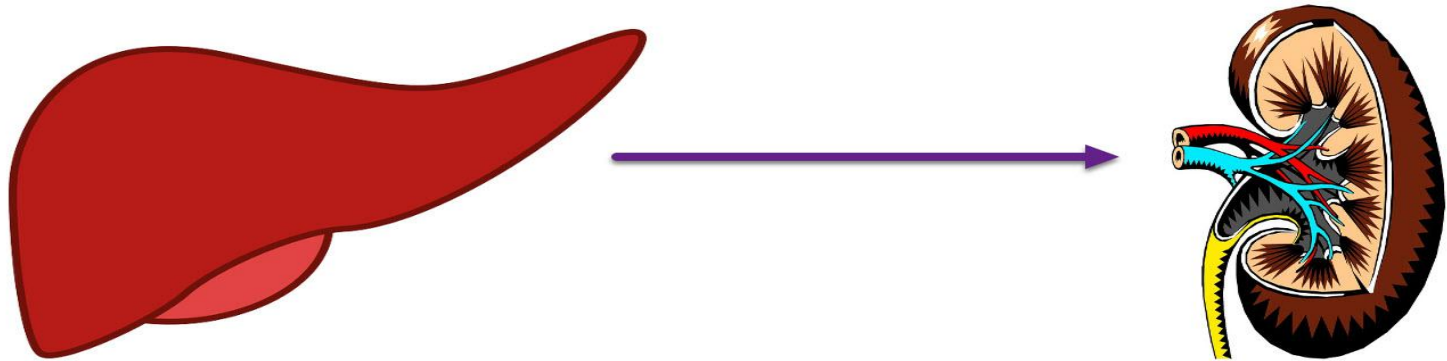
Secondary and tertiary prevention

- Education and counseling on options for care and treatment.
- Immunization with the hepatitis A and B vaccines to prevent coinfection from these hepatitis viruses and to protect their liver.
- Early and appropriate medical management including antiviral therapy if appropriate.
- Regular monitoring for early diagnosis of chronic liver disease.



Thank
you

Hepatorenal Syndrome

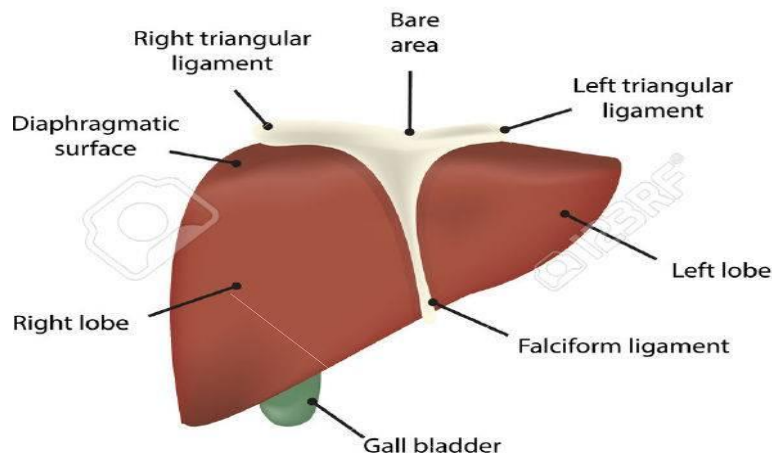


Hepatorenal syndrome refers to the development of renal failure in patients with severe liver disease. It is a life-threatening condition with poor prognosis.

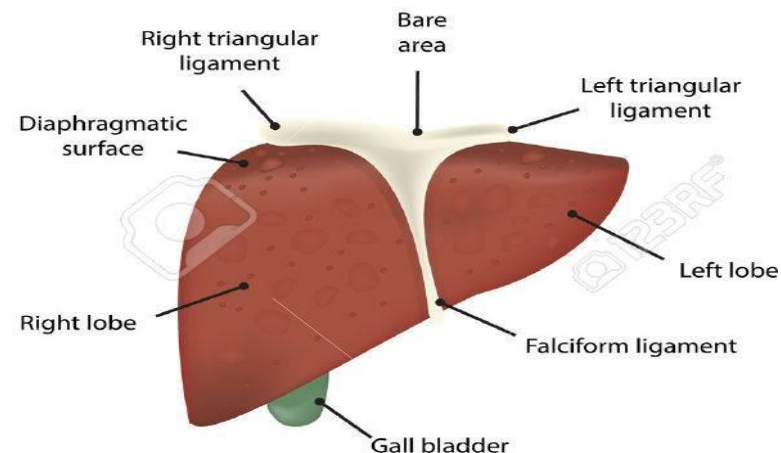
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Hepatocellular Carcinoma

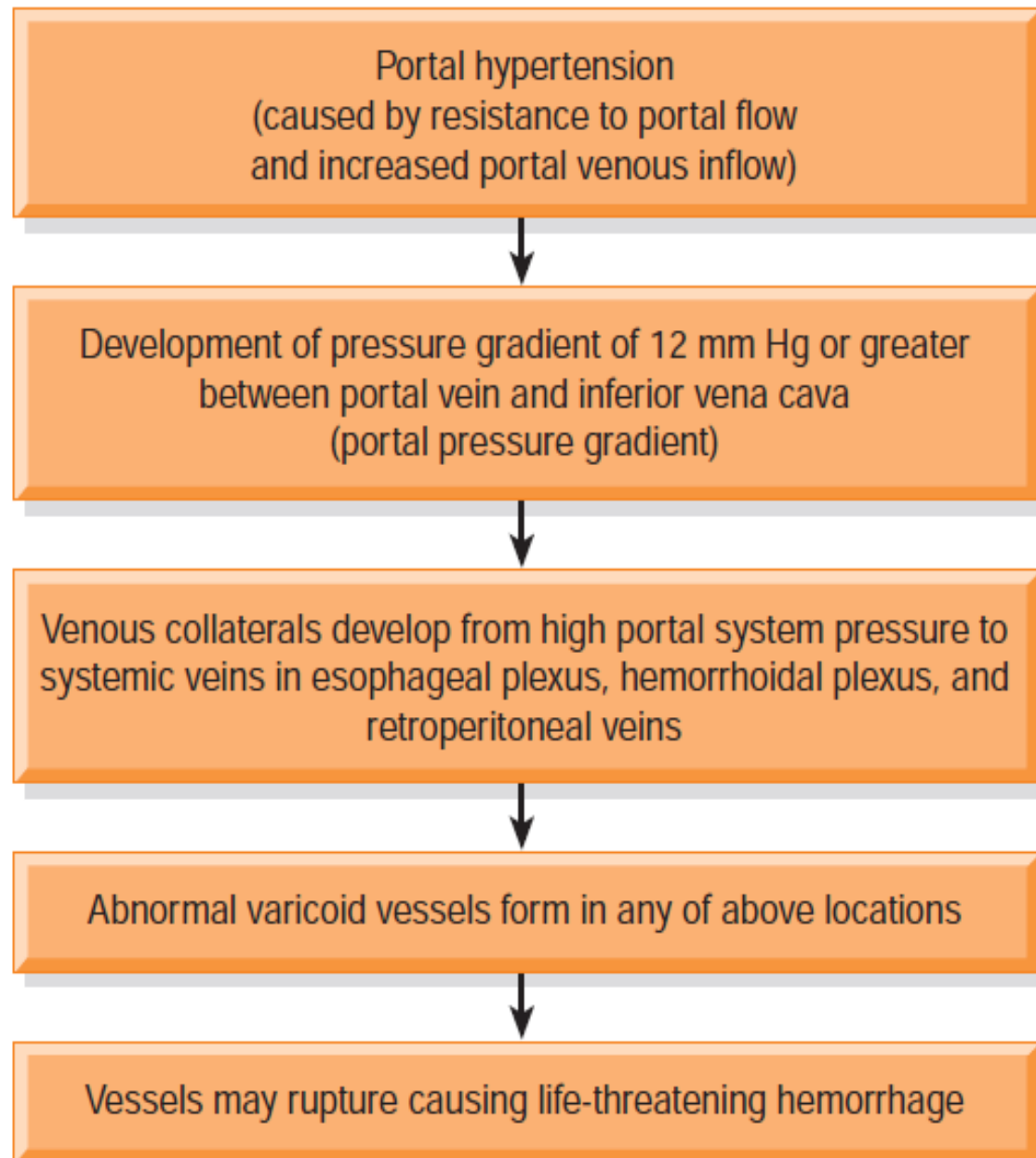
Healthy



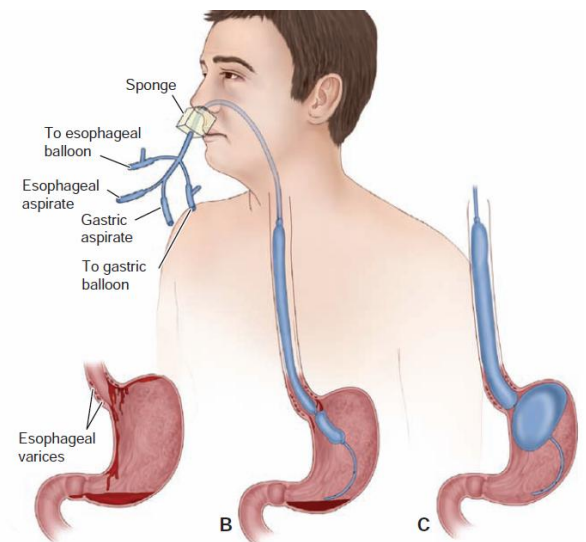
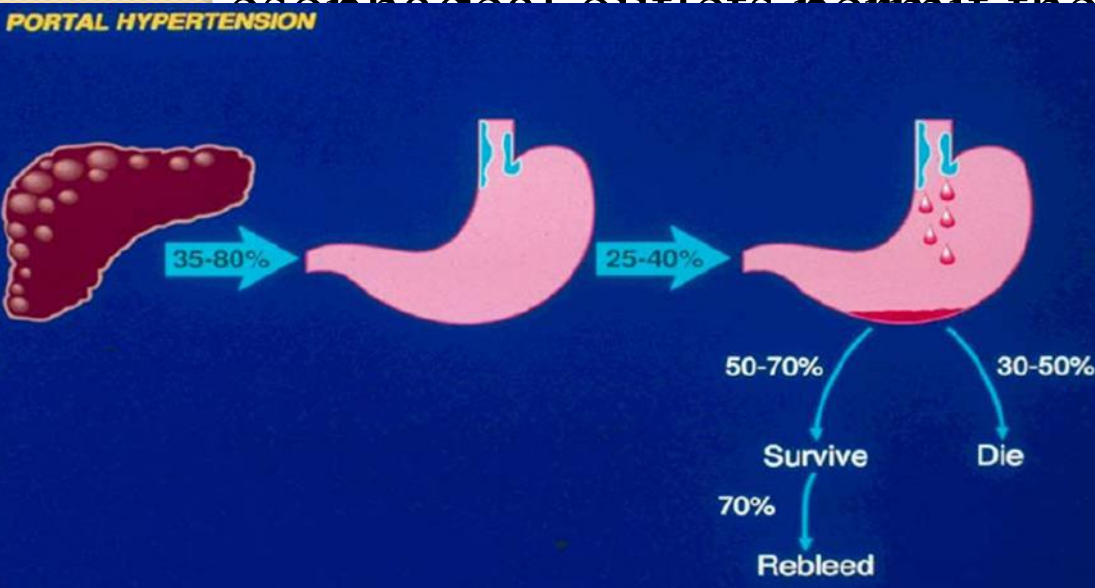
Cancerous



Pathogenesis of bleeding esophageal varices



Balloon tamponade to treat esophageal varices. A, Dilated, bleeding esophageal veins (varices) of the lower esophagus. B, A four-lumen esophageal tamponade tube with balloons (uninflated) in place. C, Compression of bleeding esophageal varices by inflated esophageal and gastric balloons. The gastric and esophageal outlets permit the nurse to



- Endoscopic or injection sclerotherapy. Injection of sclerosing agent into esophageal varices through an endoscope promotes thrombosis and eventual sclerosis, thereby obliterating the varices.

