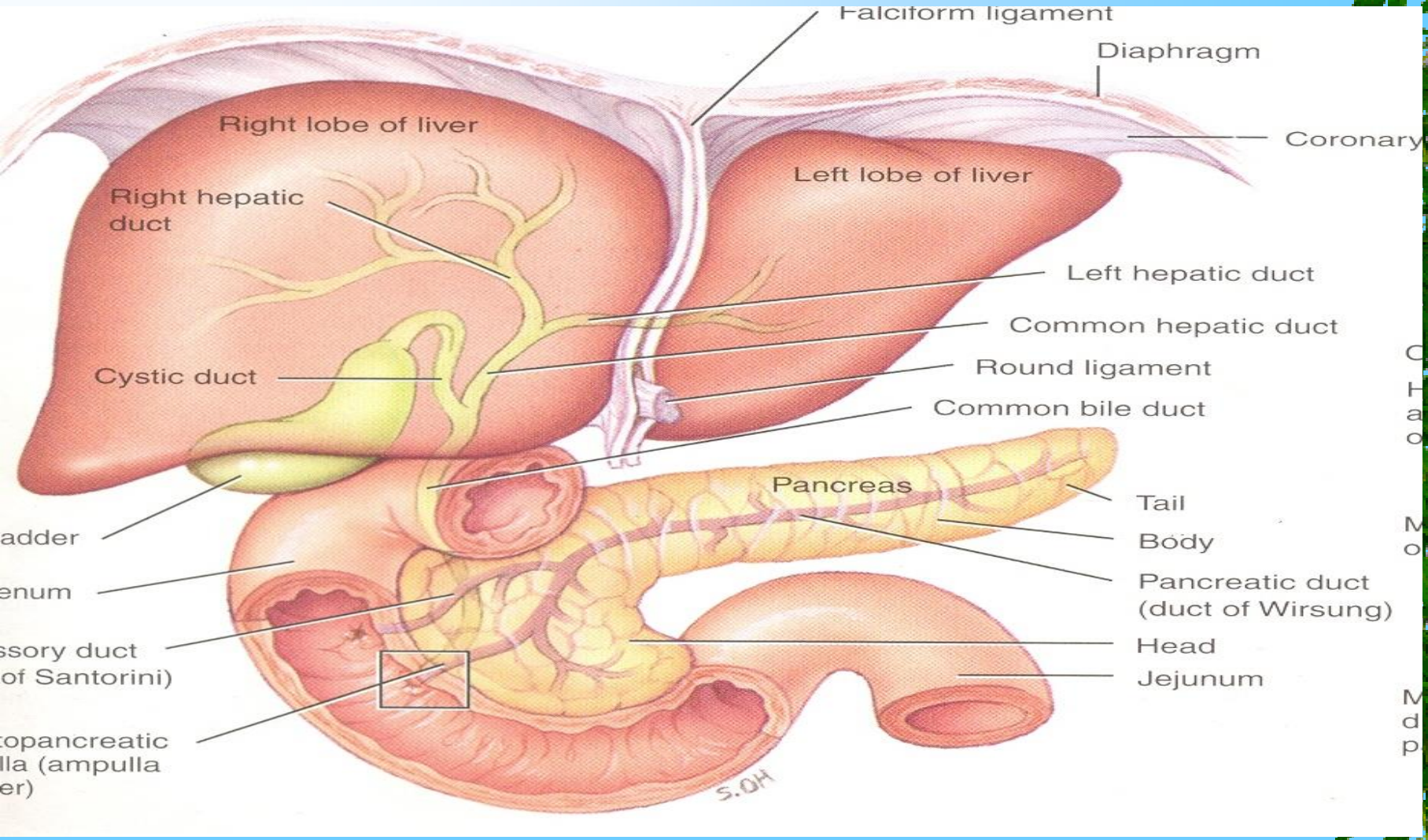
- LIVER CIRRHOSIS
- HEPATITIS

- MELBA DSOUZA

Liver Functions

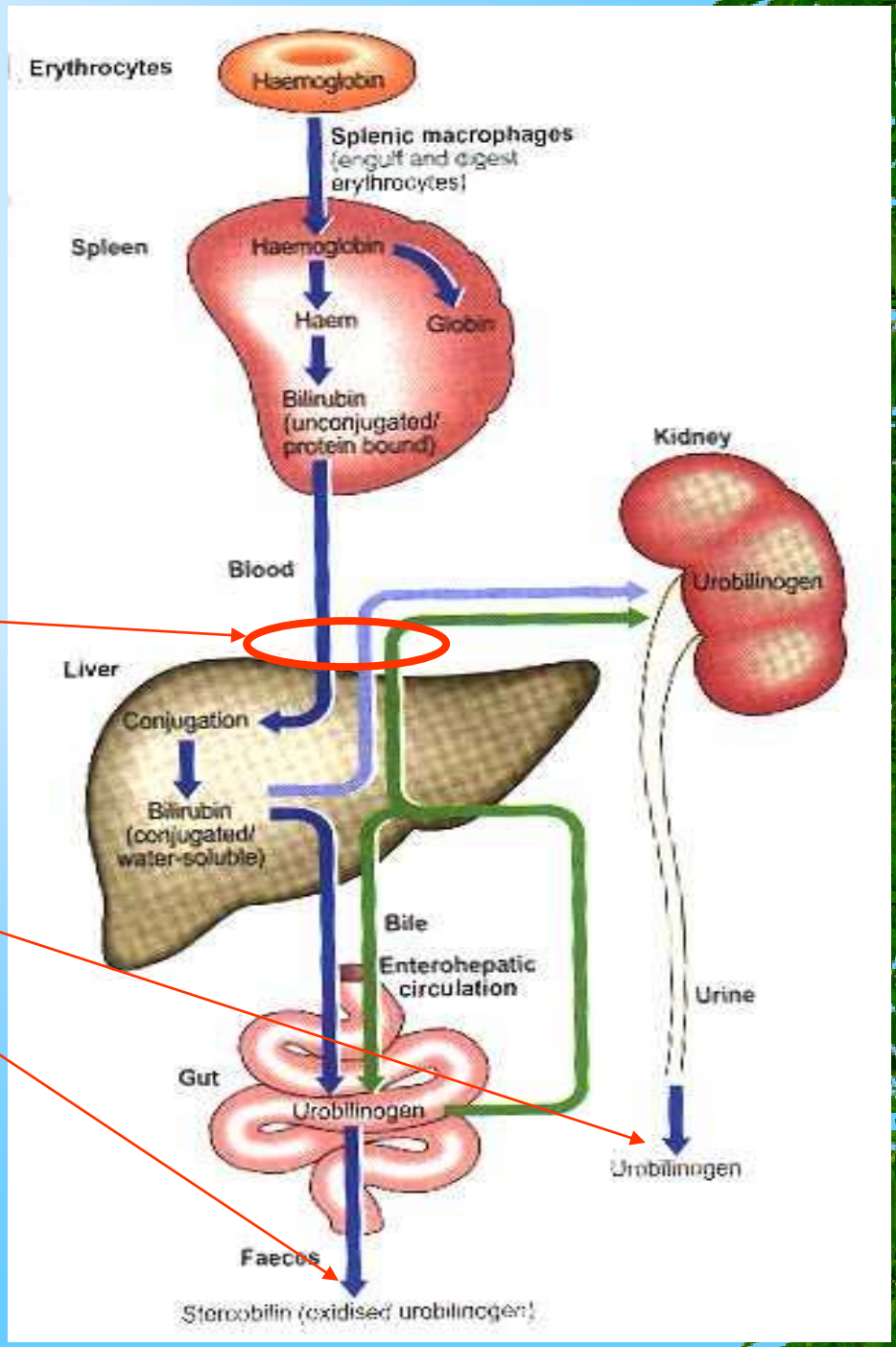
- **Metabolism** – Carbohydrate, Fat & Protein
- **Secretory** – bile, Bile acids, salts & pigments
- **Excretory** – Bilirubin, drugs, toxins
- **Synthesis** – Albumin, coagulation factors
- **Storage** – Vitamins, carbohydrates etc.
- **Detoxification** – toxins, ammonia, etc.

ANATOMY OF LIVER



Bilirubin Metabolism

- Blood
  - Conjugated & Conjugated
- Urine – Urobilinogen
- Stool – Stercobilin



Jaundice

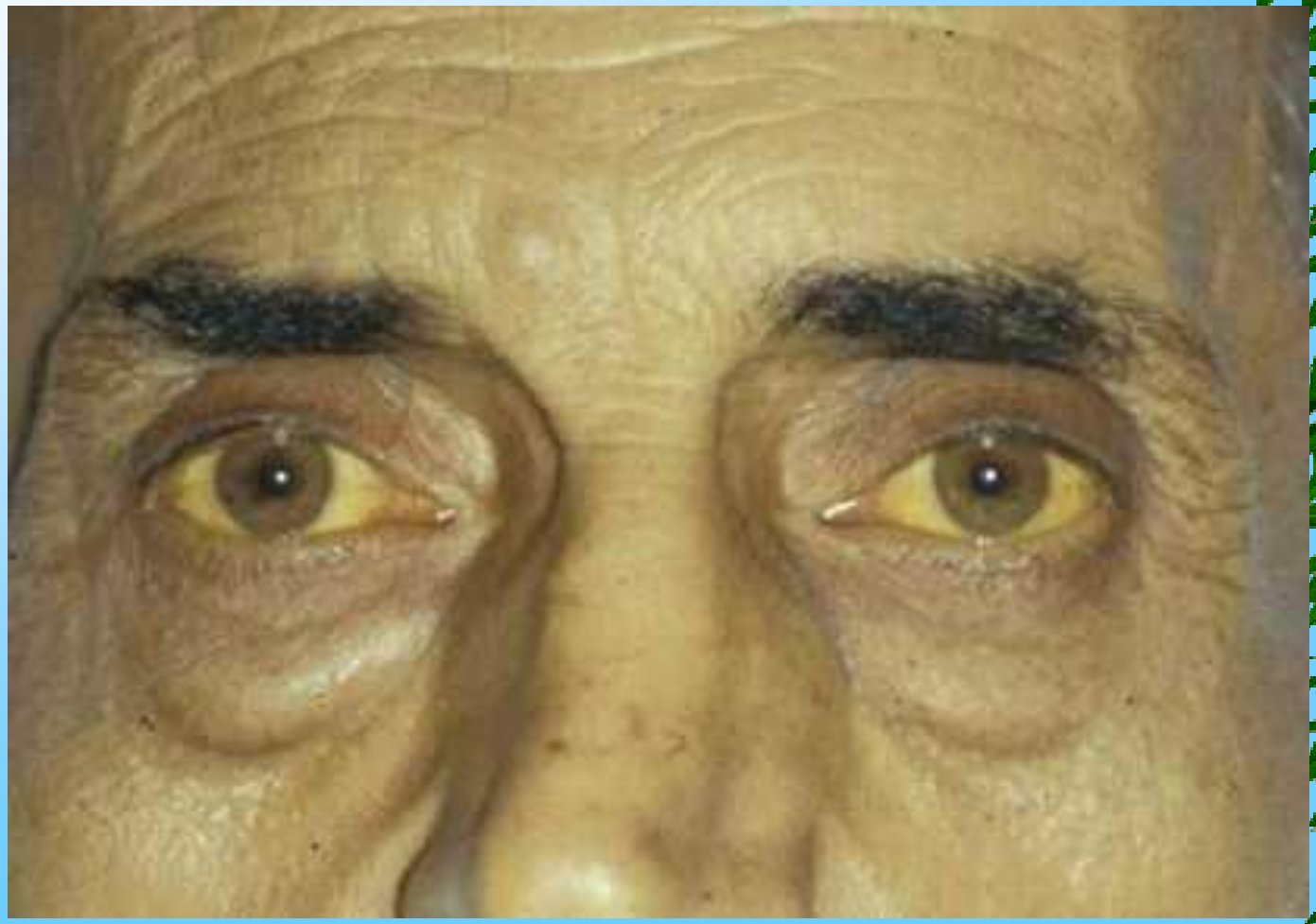
- Yellow discoloration of skin & sclera due to **excess serum bilirubin**. >40umol/l, (3mg/dl)
- Conjugated & Unconjugated types
- Obstructive & Non Obstructive (clinical)
- Pre-Hepatic, Hepatic & Post Hepatic types
- Jaundice - Not necessarily liver

Common Causes of Jaundice

- Pre Hepatic (Acholuric) - Hemolytic
  - Unconjugated/Indirect Bil, pale urine
- Hepatic – Viral, alcohol, toxins, drugs
  - Liver damage - unconjugated
  - Swelling, canalicular obstruction - Conjugated
- Post Hepatic (Obstructive) – Stone, tumor
  - Conjugated/Direct Bil, High colored urine,

Jaundice

Jaundice



Hepatic Cirrhosis

Diffuse destruction & regeneration of hepatic cells.

- Inflammation – Fibrosis - Regeneration of remaining hepatocytes form regenerating nodules.
- Diffuse scarring of liver (hepatocellular necrosis)

As necrotic tissue yields to fibrosis and cirrhosis

Damage liver tissues & normal vasculature

- Loss of normal architecture & function.

Impairs blood & lymph flow

Ultimately causes hepatic insufficiency.

CAUSES

- Excessive alcohol intake.
- Nutritional deficiency/ reduced protein intake
- Viral infection .
- Cholangitis.
- Right heart failure.
- Exposure to certain chemicals such as;
- CCl_4 ,Chlorinated naphthalene, Ar, P
- Infectious Schistosomiasis

Alcoholic Liver Injury (ALD)

- Ethyl alcohol : Common cause
- Alcoholic Liver disease - Patterns
  - Fatty change,
  - Acute hepatitis (Mallory Hyalin)
  - Chronic hepatitis with Portal fibrosis
  - Cirrhosis, Chronic Liver failure
- All reversible except cirrhosis stage.

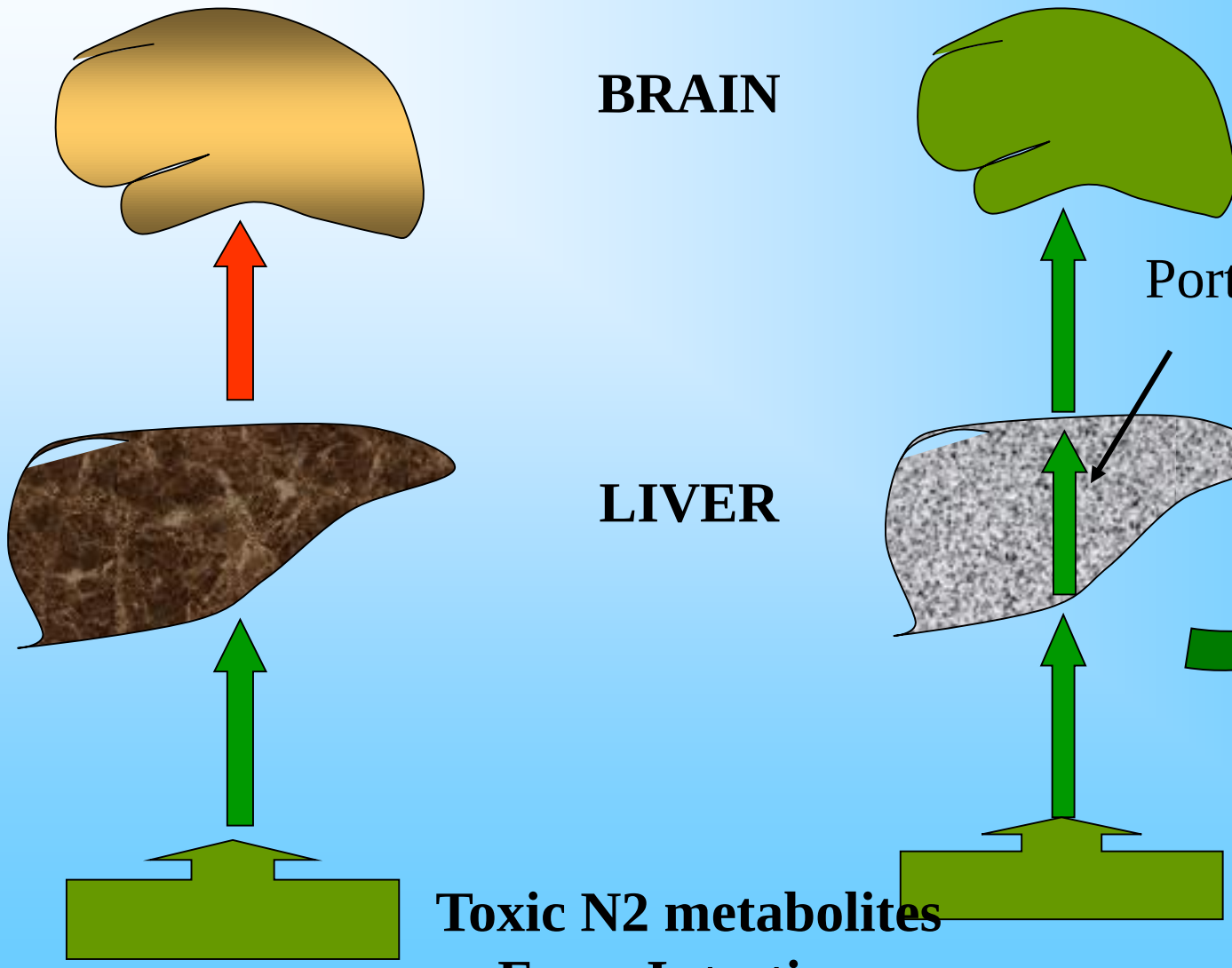
Alcoholic Liver Disease

1. Diffuse disorder of liver characterised by;
2. Complete loss of normal architecture,
3. Replaced by extensive fibrosis with,
4. Regenerating parenchymal nodules.

Complications

- Congestive splenomegaly.
- Esophageal varices
- Coagulopathy
- Hepatocellular/ Liver failure
- Hepatic encephalitis / coma.
- Hepatocellular carcinoma.
- Respiratory problems
- Portal hypertension
- Acute GI bleeding

Pathogenesis of Hepatic Encephalopathy



TYPES

- LANNEC'S CIRRHOSIS
- Macro nodular
- Alcohol abuse, malnutrition
- Accumulation of fat in the liver cells, progressing to scar tissue formation.

- POST NECROTIC CIRRHOSIS
- Micro nodular
- Previous viral hepatitis
- Severe inflammation with massive necrosis.

- CARDIAC CIRRHOSIS
- Right side heart failure
- Hepatomegaly with some fibrosis.
- BILIARY CIRRHOSIS
- Associated with biliary obstruction, usually in the common bile duct
- Chronic impairment of bile excretion

- Liver destruction
- Laennec's cirrhosis: episodes of necrosis involving the liver cells.

↡

Destroyed liver cells are gradually replaced by scar tissue exceeds that of the functioning liver tissue

↡

Eventually the amount of scar tissue exceeds that of the functioning liver tissue.

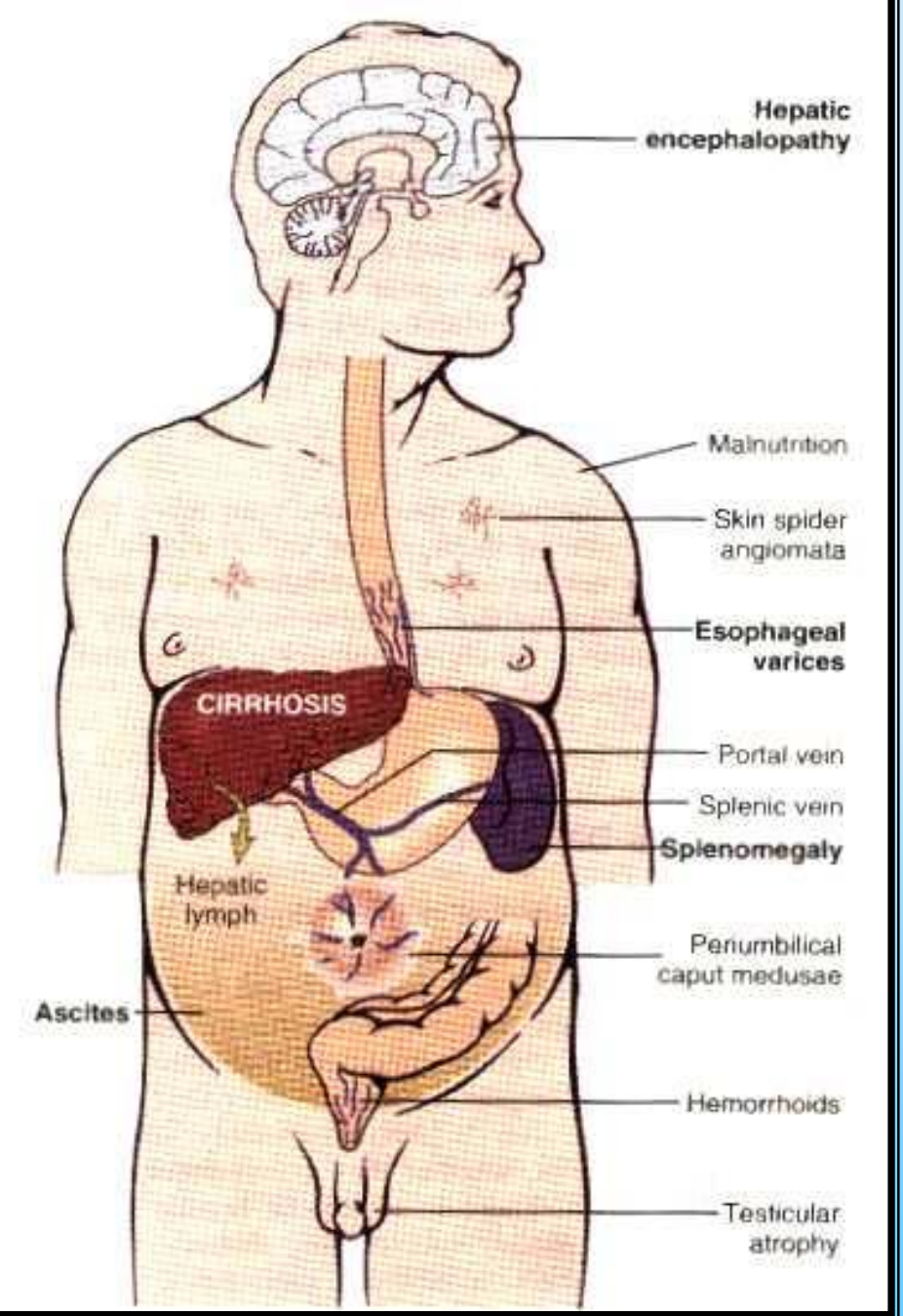
↡

Islands of residual normal tissue & regenerating liver tissue may project from the constricted areas.

↡

HOBNAIL appearance.

(Insidious onset & proceeds over a period of 30/more yrs)



Features

Hepatocellular failure.
- Malnutrition,
- low albumin, clotting factors,
- bleeding.
- Hepatic encephalopathy.
- Portal hypertension.

Ascites
- Porta systemic shunts,
- varices,
- Splenomegaly

Parenchymal regeneration

Portal obstruction

Jaundice

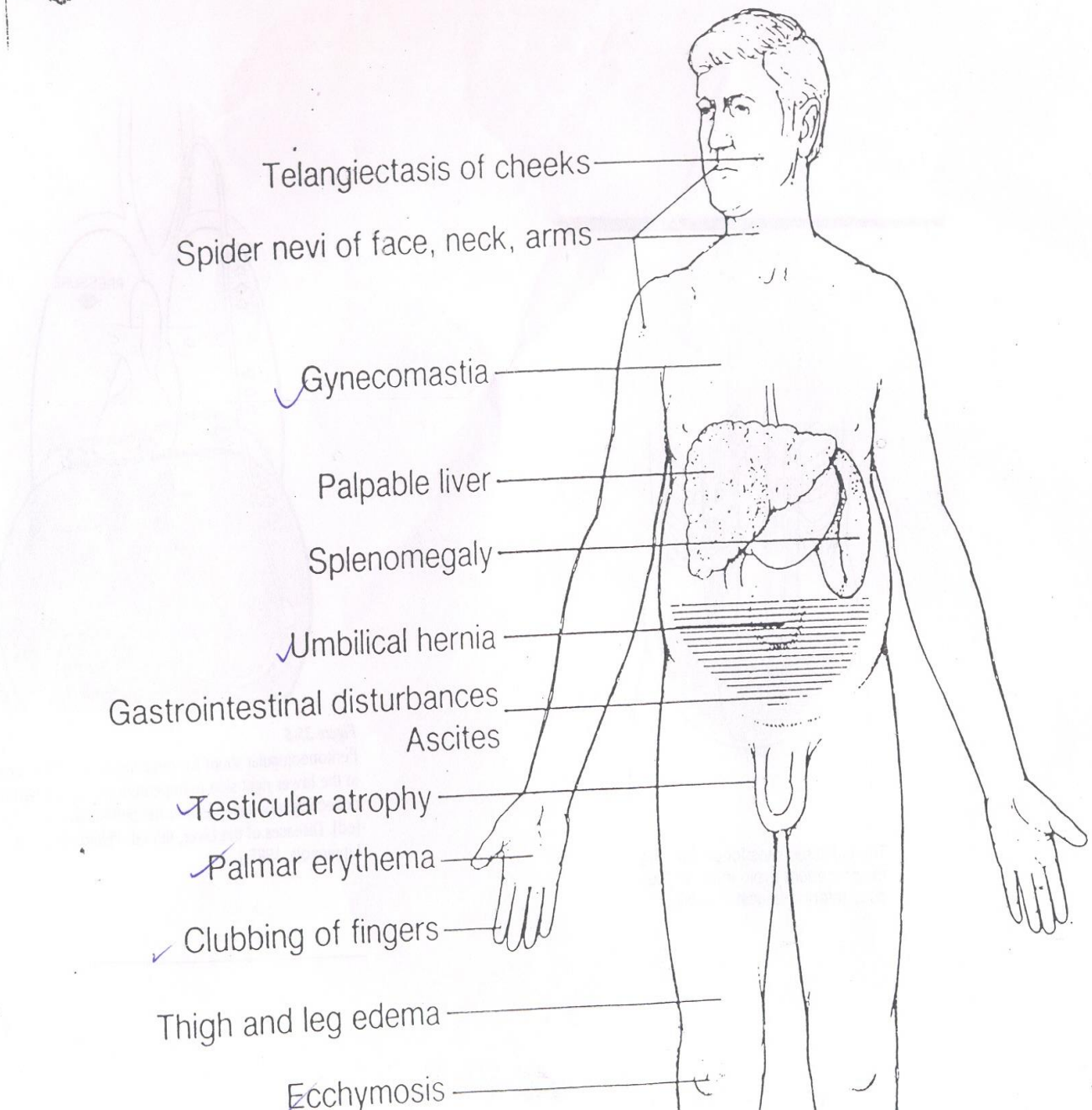
Coagulopathy

Hypoproteinemia

Toxemia

CLINICAL MANIFESTATIONS

- **COMPENSATED**
  - *Intermittent mild fever.*
  - *vascular spiders*
  - *palmar erythema*
  - *unexplained epistaxis*
  - *Ankle edema*
  - *Vague morning indigestion*
  - *Flatulent dyspepsia*
  - *Abdominal pain*
  - *Enlarged liver.*
  - *Splenomegaly*
- **DECOMPENSATED**
  - *Portal obstruction & ascites*
  - *Jaundice*
  - *Weakness*
  - *Muscle wasting*
  - *Weight loss*
  - *Continuous mild fever*
  - *Clubbing of fingers*
  - *Purpura*
  - *Epistaxis*
  - *Hypotension*
  - *White nails*
  - *Gonadal atrophy*


Telangiectasis of cheeks
Spider nevi of face, neck, arms
Gynecomastia
Palpable liver
Splenomegaly
Umbilical hernia
Gastrointestinal disturbances
Ascites
Testicular atrophy
Palmar erythema
Clubbing of fingers
Thigh and leg edema
Ecchymosis

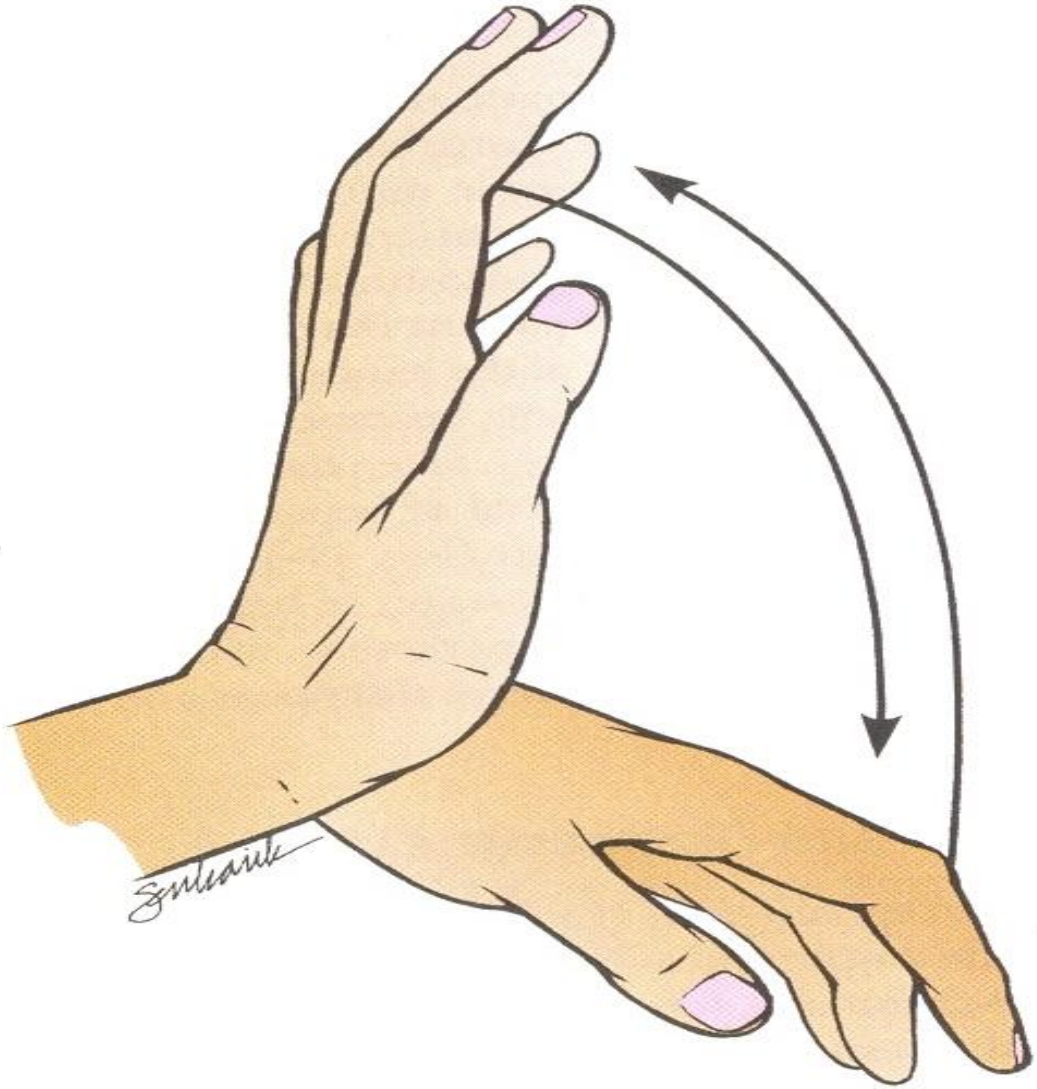
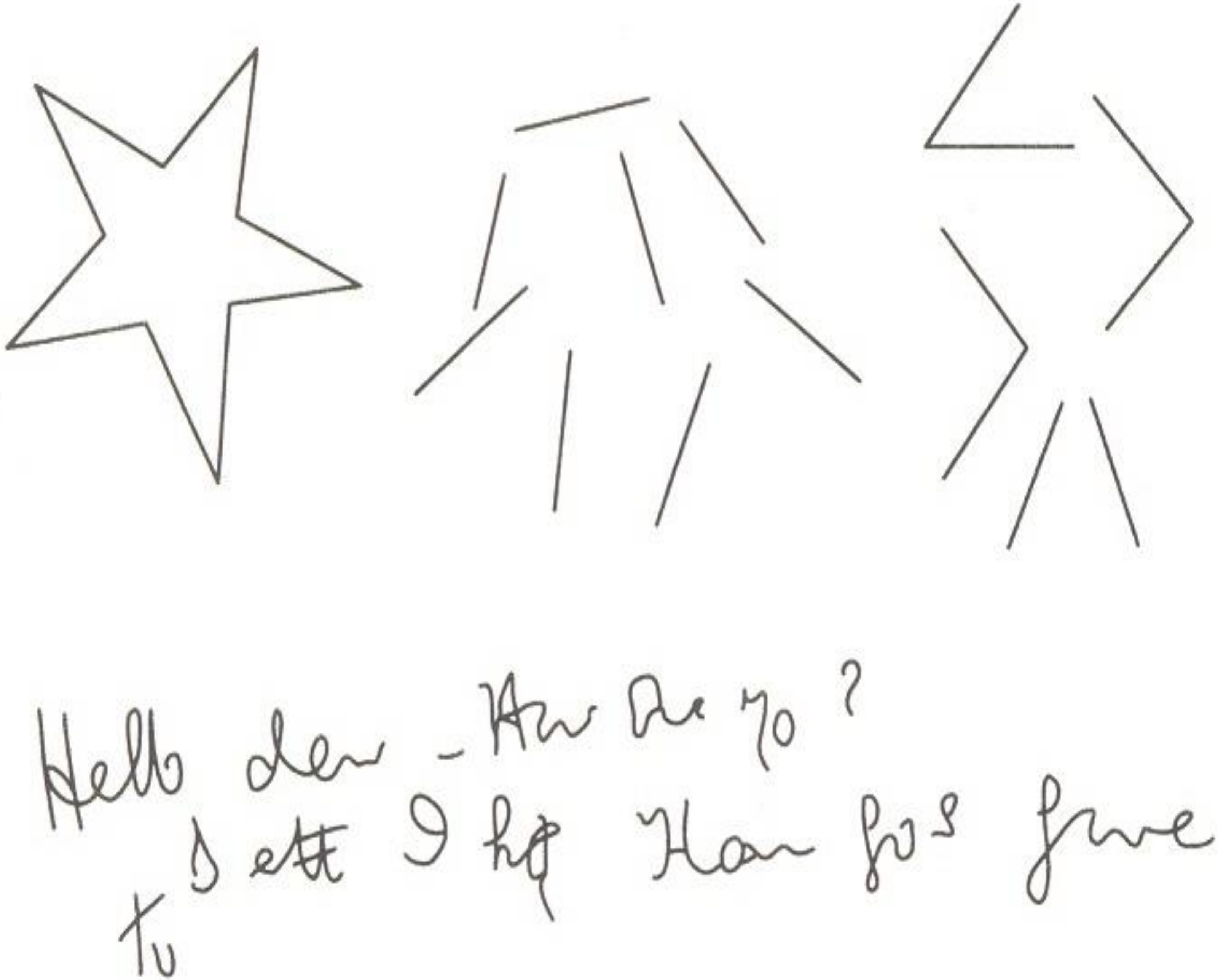


FIGURE 39-12 Asterixis or "liver flap" may occur in hepatic encephalopathy. The patient is asked to hold the arm out with the hand held upward (dorsiflexed). Within a few seconds, the hand falls forward involuntarily and then quickly returns to the dorsiflexed position.


Hello dear - How are yo ?

DIAGNOSTIC TEST

- **ENZYME TESTS**

Increase Liver enzymes

Increase S. Alkaline phosphatase, SGOT &SGPT.

S Cholinesterase decrease.

Blood tests (severe parenchymal liver dysfunction)

Increase total S. bilirubin, indirect bilirubin,

decreased total S. albumin **& S. Globulin** proteins

prolonged prothrombin time.

CT & MRI

Abnormal thickening & liver mass,liver size ,hepatic blood flow& obstruction

ESOPHAGO GASTRO DUODENOSCOPY

Bleeding esophageal varices, stomach irritation or ulceration

USG. Measure the difference in density of parenchymal cells & scar tissue.

URINE STUDIES; Increase bilirubin & urobilinogen

FECAL STUDIES; Decreased fecal urobilinogen level

MEDICAL MgX

- Antacids;to decrease gastric distress & minimize the possibility of GI bleeding.
- Potassium sparing diuretics;spironolactone,to decrease the ascites
- Inj:Vit B_{12};correct anemia.
- Multivitamin preparations are prescribed.
- Parenteral vit K: if the PT below normal & risk of bleeding.
- Toxic drugs are avoided (inactivated by liver);e.g.:diazapam,oral contraceptives &opiates.

NON-PHARMACOLOGICAL

Vitamin &nutritional supplements; promote healing of damaged liver cells&improve the general nutritional status.

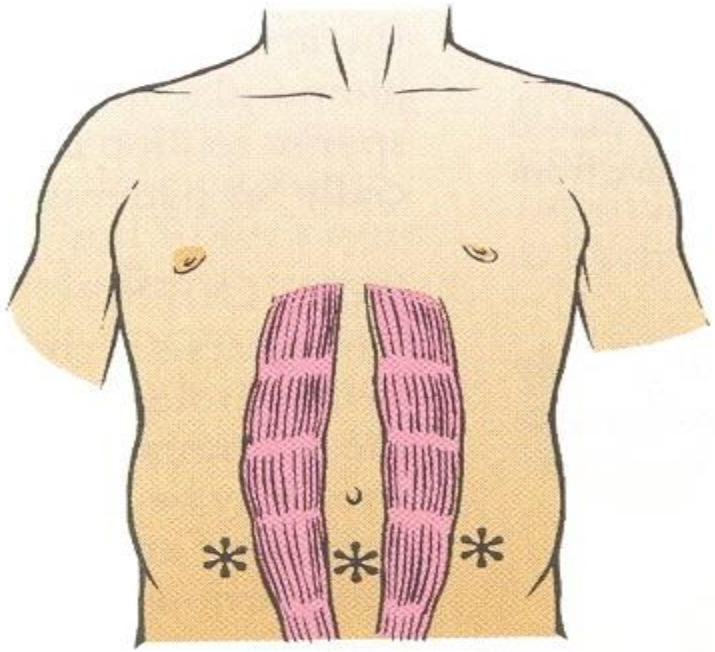
Adequate intake of proteins & calories is an essential part of protein

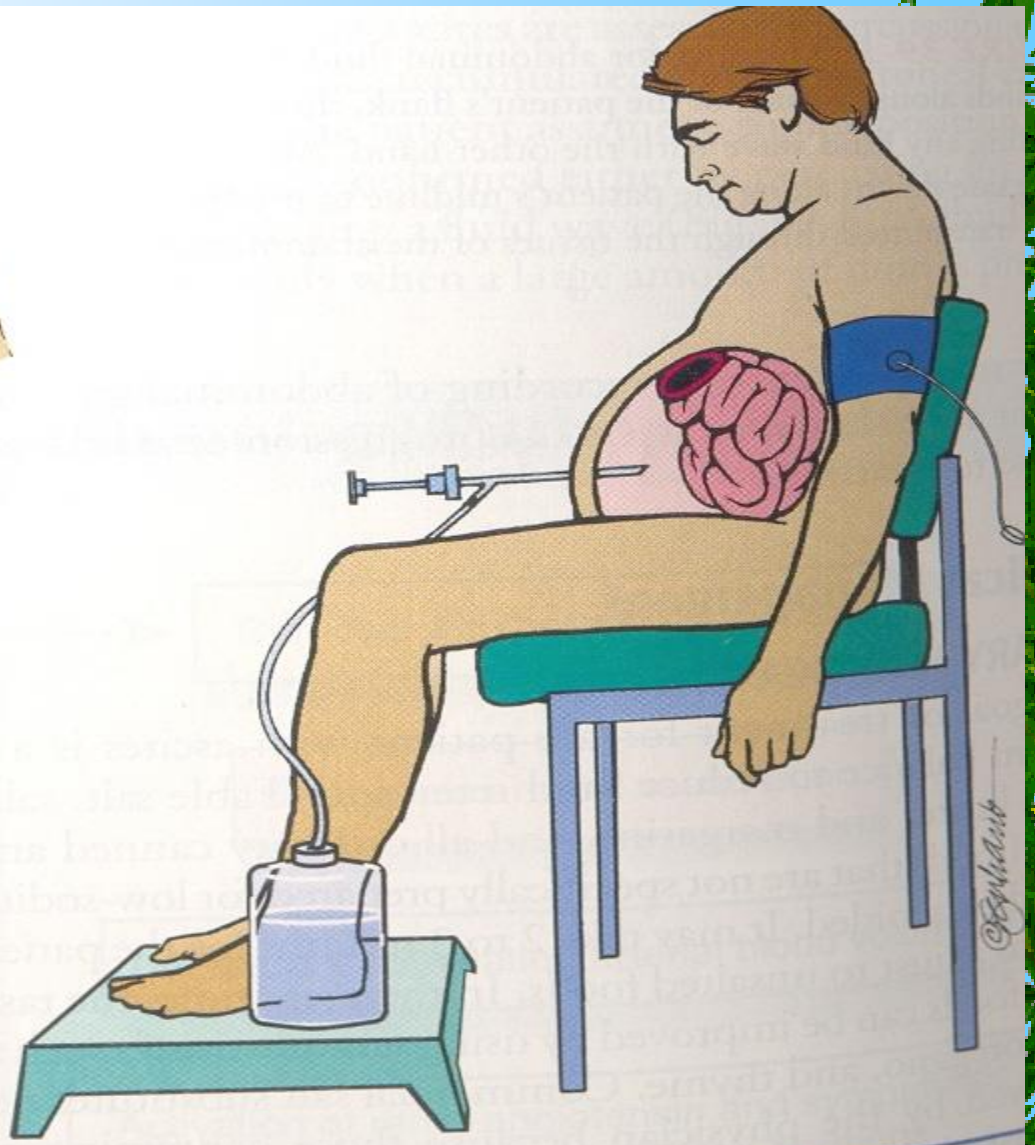
Avoidance of alcohol.

Sodium intake is carefully regulated and often restricted because of the potential for water retention,which can lead to edema,circulatory congestion &heart failure.

Fluid intake also may be restricted.

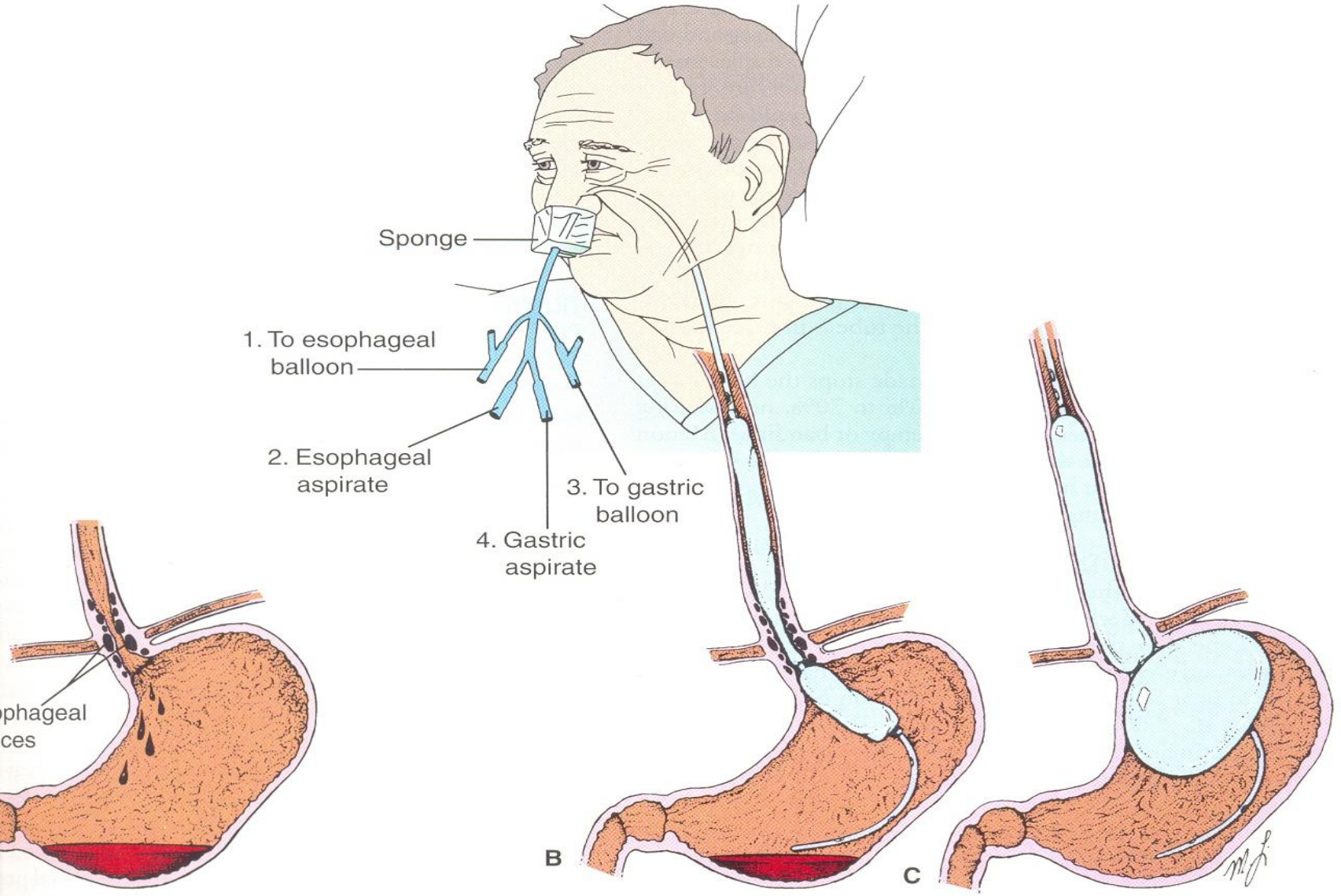
PARACENTESIS





Bleeding in Liver disease:

- vitamin K – in liver →gamma-carboxyglutamic acid – for coagulation factors II, VII, IX, and X.
- Liver disease → factor VII is the first to go → so the defect will appear initially in the extrinsic pathway, i.e., abnormal PT. When severe it affects both pathways.



39-7 Esophageal balloon tamponade to treat esophageal varices. (**A**) Dilated, bleeding esophageal veins f the lower esophagus. (**B**) A four-lumen esophageal tamponade tube with balloons (uninflated) in place. pression of bleeding esophageal varices by inflated esophageal and gastric balloons. The gastric and esophageal mit the nurse to aspirate secretions.

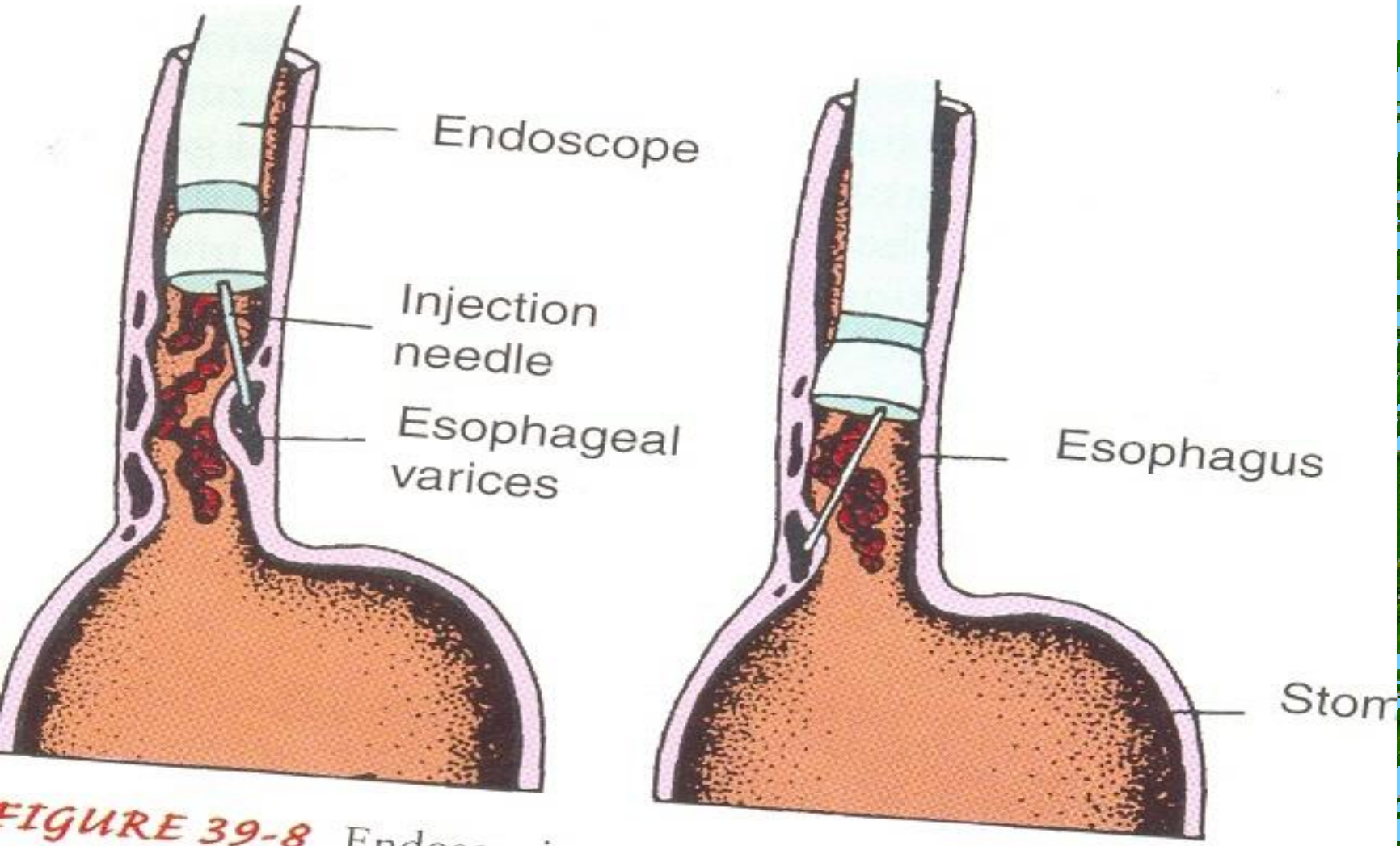


FIGURE 39-8 Endoscopic or injection sclerotherapy. Injection of s
osing agent into esophageal varices through an endoscope promotes thro
osis and eventual sclerosis, thereby obliterating the varices.

PORTAL HYPERTENSION

- Obstructed blood flow through the damaged liver results in blood pressure through out the portal venous system.
- Common Sym is spleenomegaly
- Major complications are ascites and varices.

PORTAL HYPERTENSION

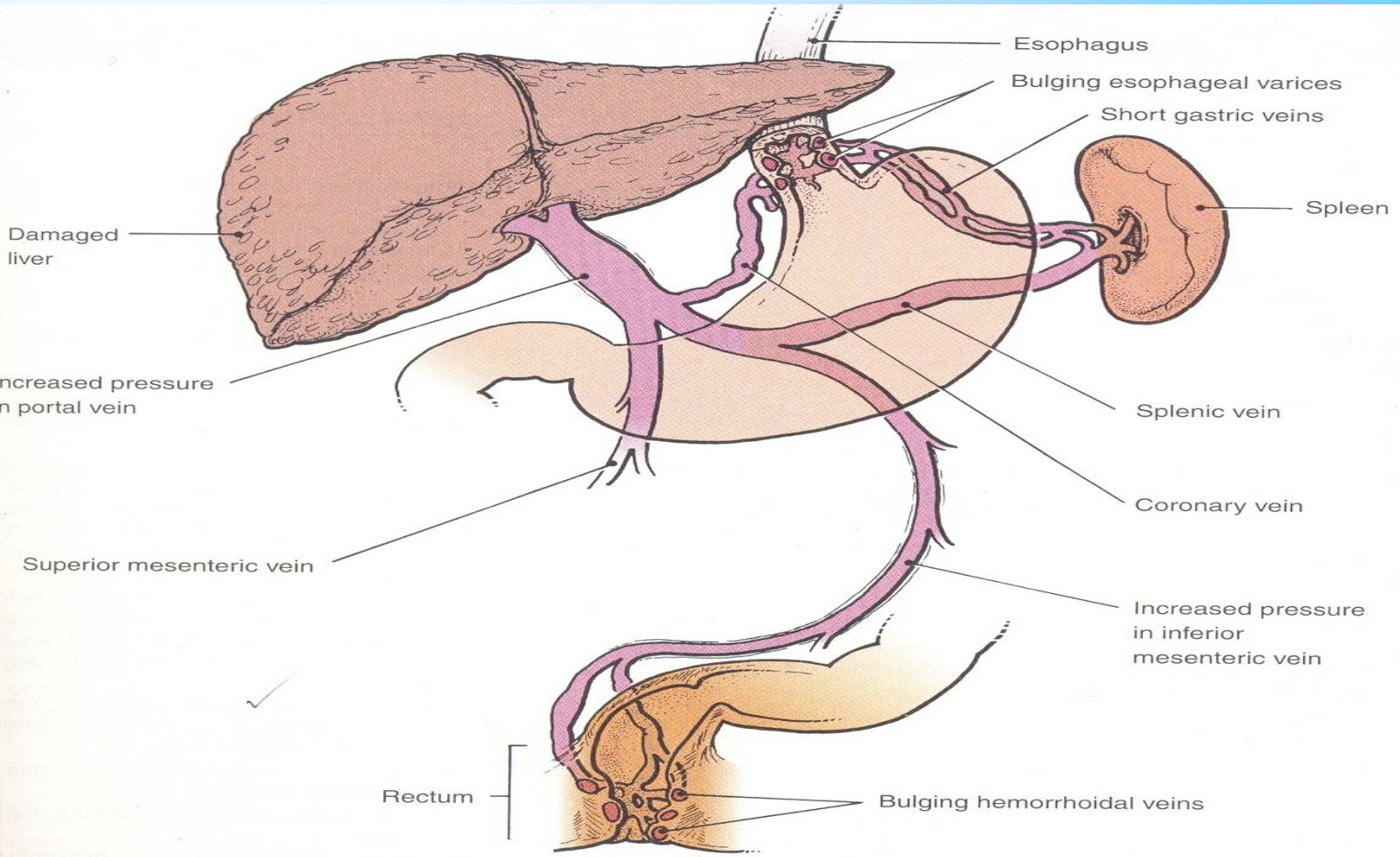


FIGURE 55-5 In portal hypertension, collateral vessels become dilated.

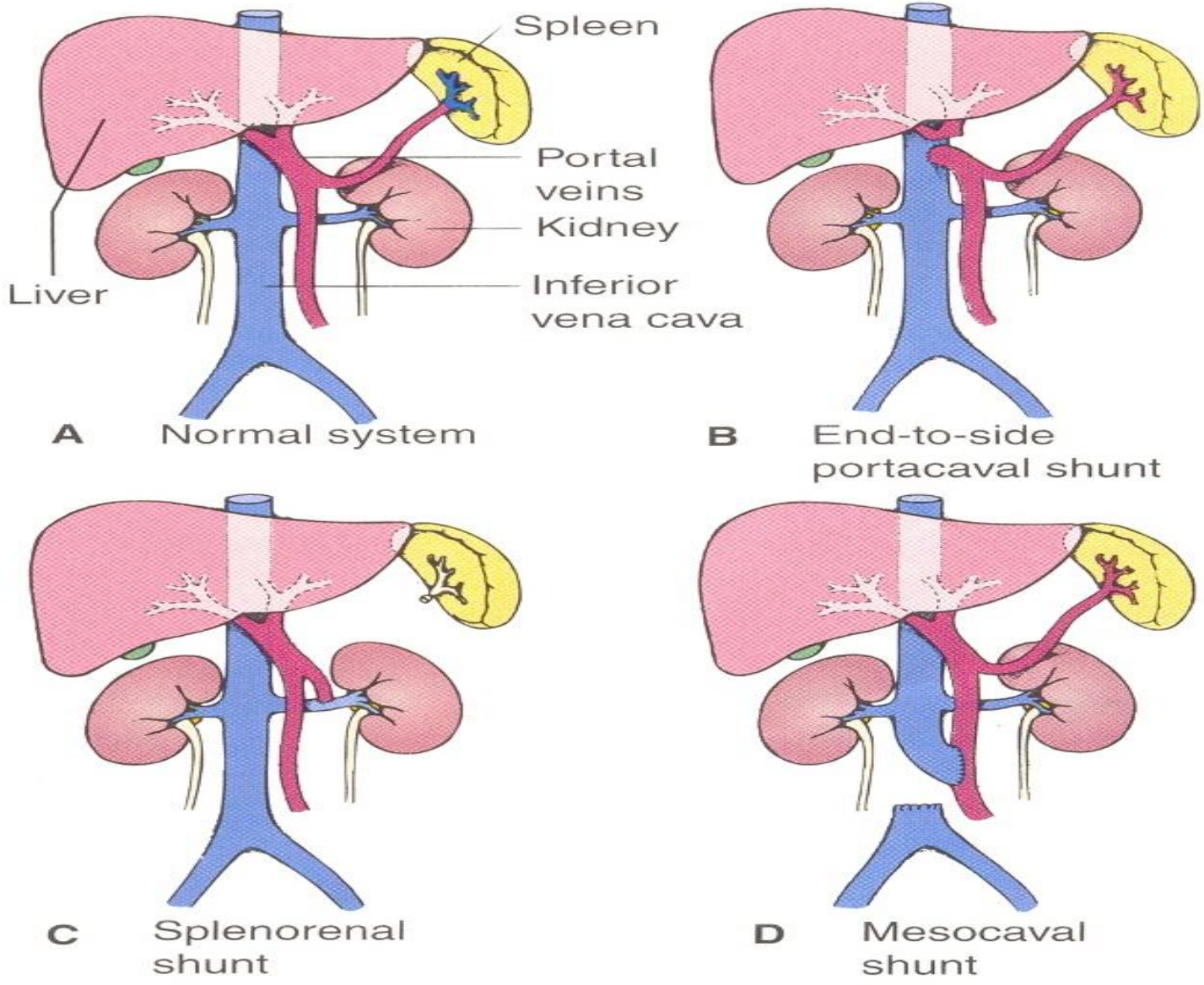
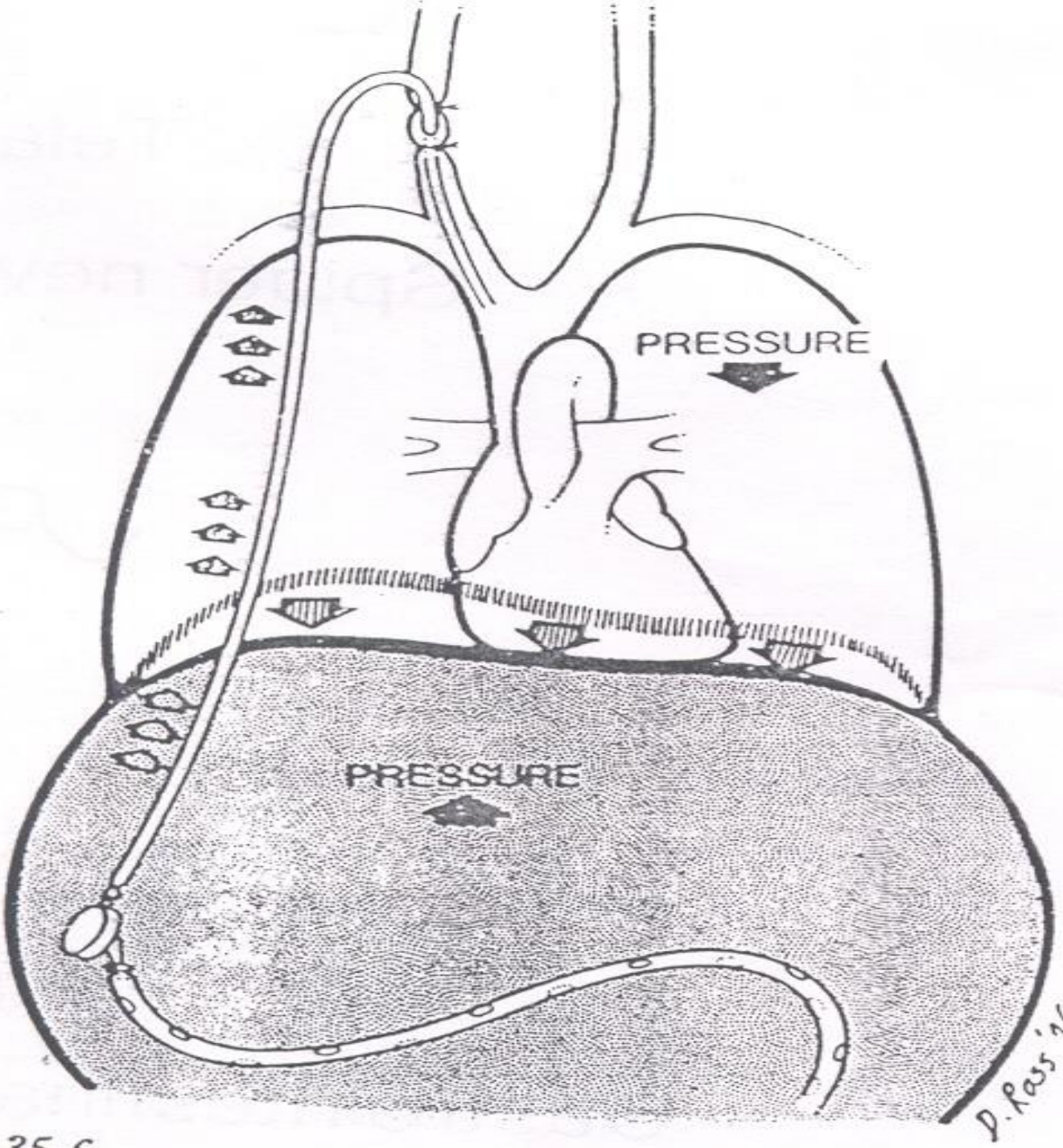


FIGURE 39-11 Portal systemic shunts. Normal portal system is shown in (**A**); examples of portal shunts to reduce portal pressure are shown in (**B**) to (**D**).



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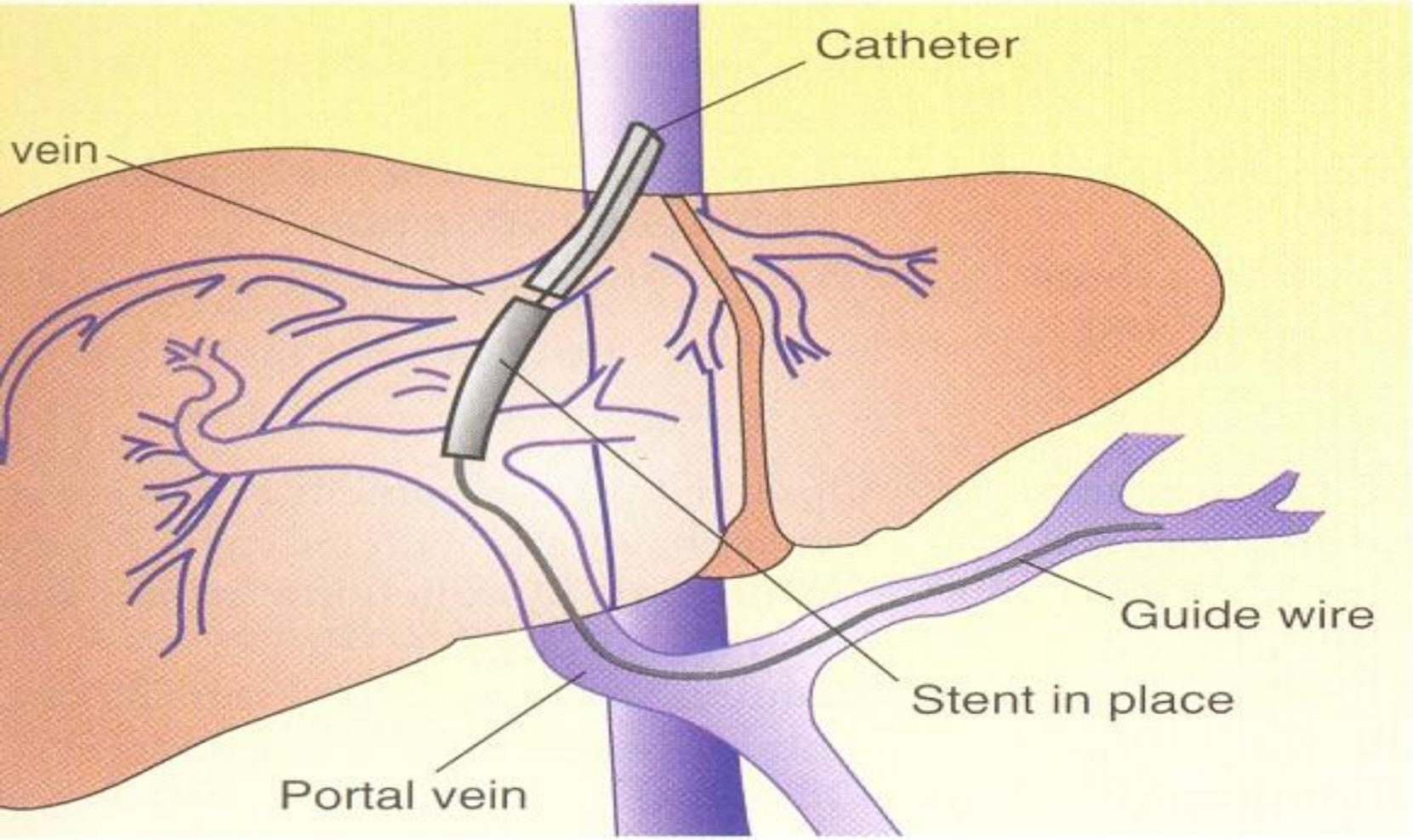

39-10 Transjugular intrahepatic portosystemic shunt (TIPS). inserted via catheter to the portal vein to divert blood flow and rtal hypertension.

Complications:

- Congestive splenomegaly.
- Bleeding varices.
- Hepatocellular failure.
  - Hepatic encephalitis / hepatic coma.
- Hepatocellular carcinoma.

ASSESMENT

- Assess for onset of symptoms
- History of precipitating factors, long term alcohol abuse as well as dietary intake &changes in the physical & mental status.
- Exposure to any hepatotoxic substances (medications, illicit IV /inj drugs ,inhalants)
- Assess for abdominal distension,GI bleeding ,& weight changes.

NURSING MgX

- Ineffective breathing pattern r/t ascites & restriction of thoracic excursion secondary to ascites, abdominal. distention,&fluid in the thoracic cavity.

- Chronic pain & discomfort r/t enlarged tender liver and ascites .

- Fluid volume excess r/t ascites & edema formation .

- Imbalanced nutrition less than body requirement r/t Abdominal distention, discomfort, anorexia
- Impaired skin integrity r/t pruritus from jaundice & edema.
- Risk for imbalanced body temperature hyperthermia r/t inflammatory process of cirrhosis or hepatitis.

- Disturbed thought process r/t deterioration of liver function &increased serum ammonia level.

- High risk for injury r/t altered clotting mechanisms &altered level of consciousness

Nursing care

- Position the patient comfortably;for maximal respiratory efficiency.

- Oxygen therapy required in liver failure to oxygenate the damaged cells & prevent further cell destruction.

- Nutrition
- Ascites /edema & no s/s of coma: nutritious, high protein diet
- Supplemented by vitamins of the A,B,C& K & Folic acid.

- Small frequent meals are tolerated better than 3 large meals, because of the abdominal pressure exerted by acsites.
- Pt with fatty stools should receive H_2O Soluble forms of fat soluble vitamins A,D&E.
- Folic acid & iron are prescribed to prevent anemia.
- If the pt develop coma;the amount of protein in the diet is decreased temporarily(1-1.5g/kg)

- Careful skincare is provided because of presence of subcutaneous edema.
- Protect from falls by using side rails.
- Use Of soft bristled tooth brush.

- **POTENTIAL COMPLICATIONS**

Use stool softeners ;prevents straining during defecation.

Hepatic encephalopathy is mainly caused by the accumulation of ammonia in the blood & its effects on cerebral metabolism

Use of lactulose & non absorbable intestinal tract antibiotics (neomycin 1-4gm)to decrease ammonia level.

SURGICAL MANAGEMENT

- **LIVER TRANSPLANTATION**

  Is an option for treating liver failure as well as chronic liver disease.

Conclusions:

- Common end result of diffuse liver damage. (Viral hepatitis, Alcohol, congenital, drugs, toxins & Idiopathic)
- Characterised by diffuse loss of architecture.
- Fibrous bands & regenerating nodules distort and abstruct blood flow. (inefficient function)
- Hepatocellular insufficiency & portal hypertension.
- Shrunken, scarred liver, ascitis

Hepatitis

- **Acute inflammatory disease of the liver caused by virus (most common), bacteria, toxic or chemical injury.**

Pathology of Hepatitis:

- Hepatitis: Inflammation of Liver
- Viral, Alcohol, immune, Drugs & Toxins
- Biliary obstruction – gall stones.
- Acute, Chronic & Fulminant - types
- Viral Hepatitis –
  - Specific – Heptitis **A**, **B, C, D**, **E**, & other
  - Systemic - CMV, EBV, other.

Hepatitis:

- Hepatitis: Inflammation of Liver
- Viral, Alcohol, immune, Drugs & Toxins
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  - Specific – Heptitis **A**, **B, C, D**, **E**, & other
  - Systemic - CMV, EBV, other.

Pattern of Viral Hepatitis:

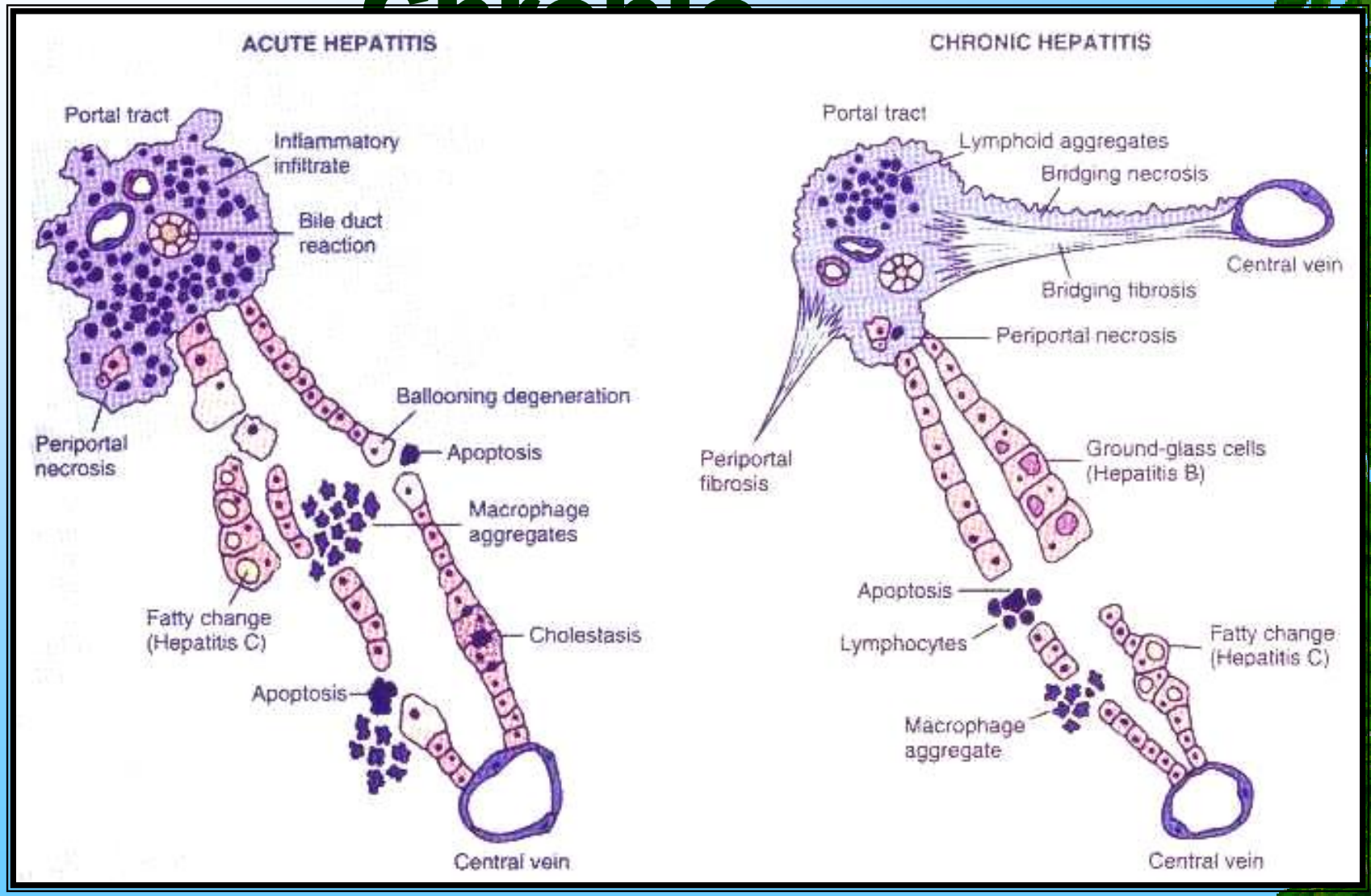
- Carrier state / Asymptomatic phase
- Acute hepatitis
- Chronic Hepatitis
  - Chronic Persistent Hepatitis (CPH)
  - Chronic Active Hepatitis (CAH)
- Fulminant hepatitis
- Cirrhosis
- Hepatocellular Carcinoma

Viral Hepatitis:

Microbiology

Virus	Hep-A	Hep-B	Hep-C
agent	ssRNA	dsDNA	ssRNA
Transm.	Feco-oral	Parenteral	Parenteral
Carrier state	None	0.1-1.0%	0.2-1.0%
Chronic Hepatitis	None	5-10%	**>50%**

Acute - Hepatitis - Chronic



Acute Hepatitis:

- Swelling and Apoptosis
- Piecemeal or Bridging, panacinar necrosis
- Inflammation – lymphocytes, Macrophages
- Ground glass hepatocytes – HBV
- Mild fatty change – HCV
- Portal inflammation and Cholestasis

Fulminant Hepatitis:

- Hepatic failure with in 2-3 weeks.
- Reactivation of chronic or acute hepatitis
- Massive necrosis, shrinkage, wrinkled
- Collapsed reticulin network
- Only portal tracts visible
- Little or massive inflammation – time
- More than a week – regenerative activity

Chronic Hepatitis:

- Persistent & Active types. CPH/CAH
- Lymphoid aggregates
- Periportal fibrosis
- Necrosis with fibrosis – bridging fibrosis.
- Cirrhosis – regenerating nodules.

PATHOPHYSIOLOGY

- Liver inflamed shortly after exposure to hepatotoxic chemicals or drugs,after lengthy alcohol abuse,or by invasion with an infectious organisms.
- Once the virus invades the hepatocytes,it alters their structure.
- Immune reaction ensues,in which the infected cells become inflamed & dysfunctional.
- Active disease process after the uptake,conjugation& excretion of bilirubin.
- Chronic persistent hepatitis (hep B,C&D): liver damage does not worsen,but it does not improve &the liver remains enlarged
- Some clients may develop cirrhosis.

STAGES

- Hepatic – Viral, alcohol, toxins, drugs
  - Liver damage - unconjugated
  - Swelling, canalicular obstruction - Conjugated

- Post Hepatic (Obstructive) – Stone, tumor
  - Conjugated/Direct Bil, High colored urine,

PREICTERIC/ Pre Hepatic (Acholuric) - Hemolytic

- Unconjugated/Indirect Bil, pale urine

Preceding the appearance of jaundice (s/s) nausea,vomiting,anorexia,fever,headache,enlargement of liver, spleen & lymph nodes, wt.loss.

ICTERIC/ Hepatic – Viral, alcohol, toxins, drugs

Jaundice and associated symptoms such as elevated bilirubin level,dark or tea color urine and clay colored stool

POSTICTERIC STAGE

Convalescent stage: jaundice decreases and the color of urine and stool returns to normal.

MANAGEMENT

- Rest,symptomatic support.
- Supporting nutritional intake.
- Intervention to minimize transmission.
- Pharmacological therapy-chronic hepatitisB,C&D (recombinant interferon Alfa-2b)
- Chronic stage liver transplantation performed(who do not respond to medical treatment).
- Immunosuppression (eg.cyclosporin,corticosteroid&azathioprine) must be done for transplantation to succeed.s

Conclusions: Hepatitis.

- Hepatitis – Alcohol, Virus (ABCD), Drugs…
- Hepatocyte damage – inflammation
- Acute / Chronic (Active / Persistent)
- Fever, Jaundice, Malaise, Fat intolerance.
- Complications.
- Alcohol – NAD, Acetaldehyde – metabolism