

Cirrhosis and Its Complications

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Disclosures

None

Outline

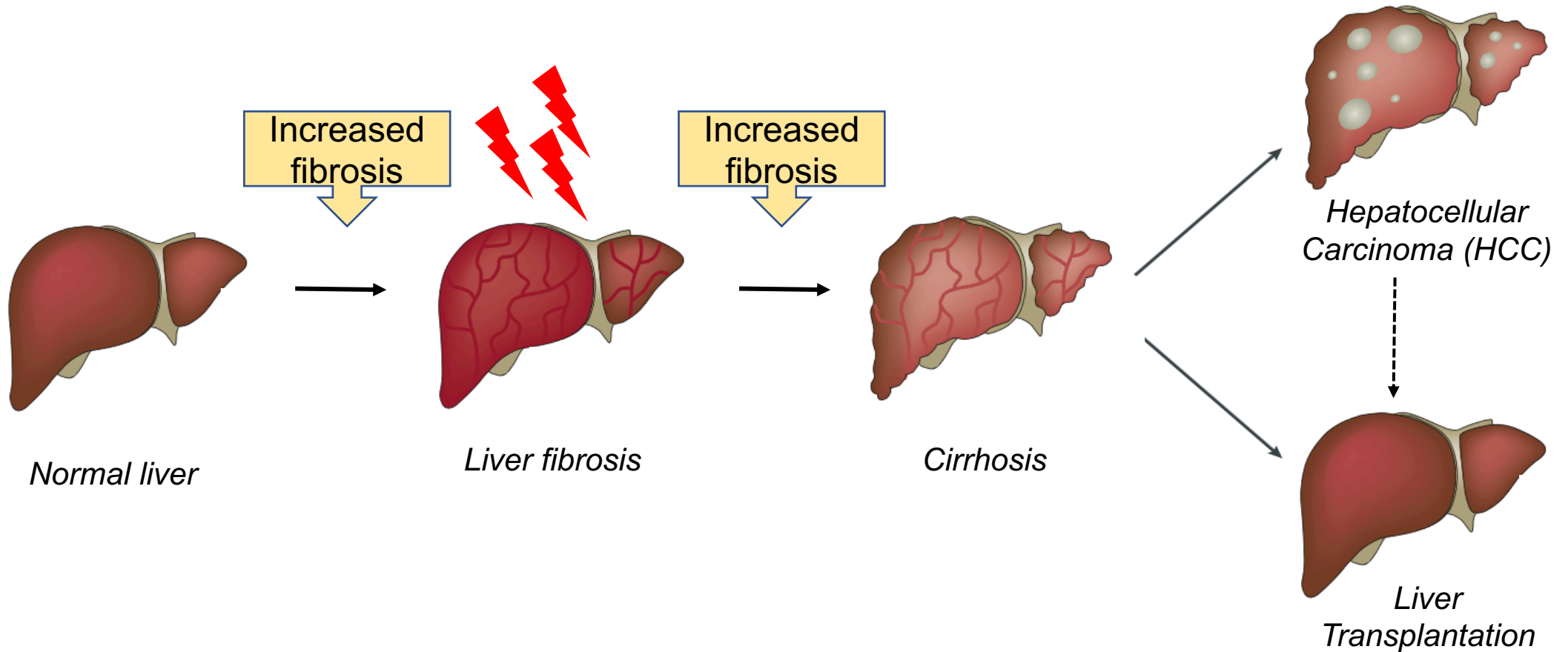
Anatomy, histology and pathophysiology

Definition and etiologies

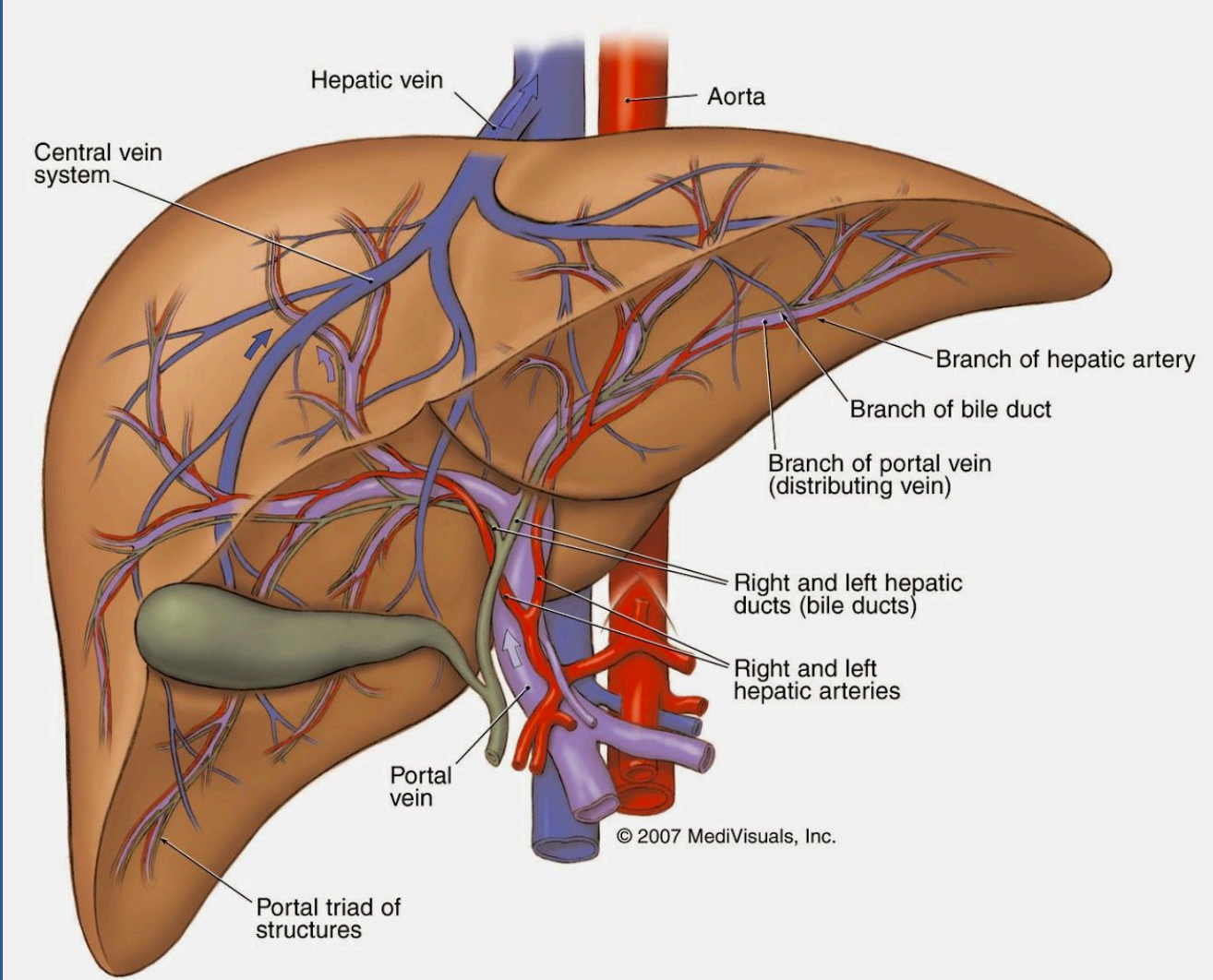
Diagnosis

Complications

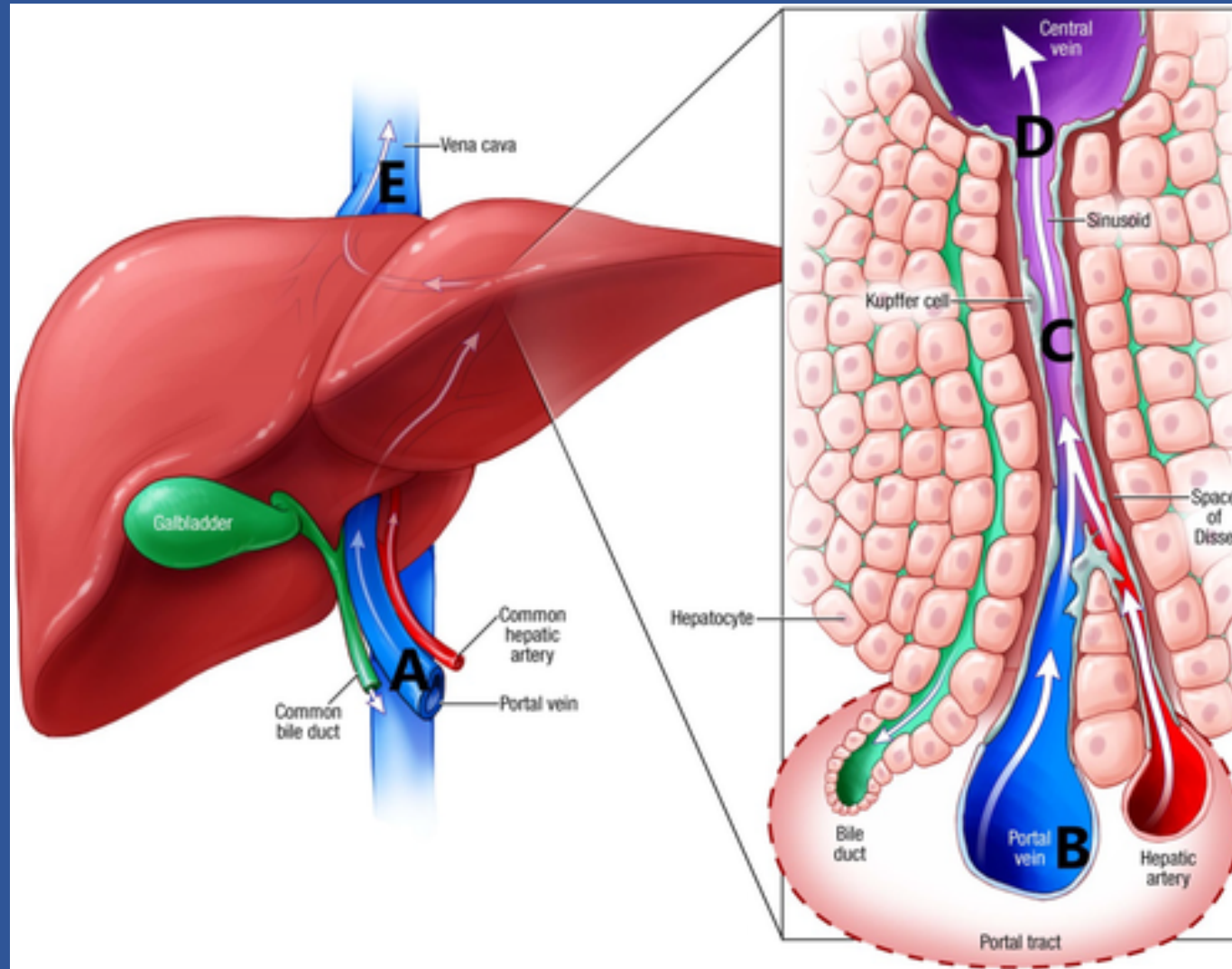
Cirrhosis is the “end stage” or final pathway in hepatic fibrosis



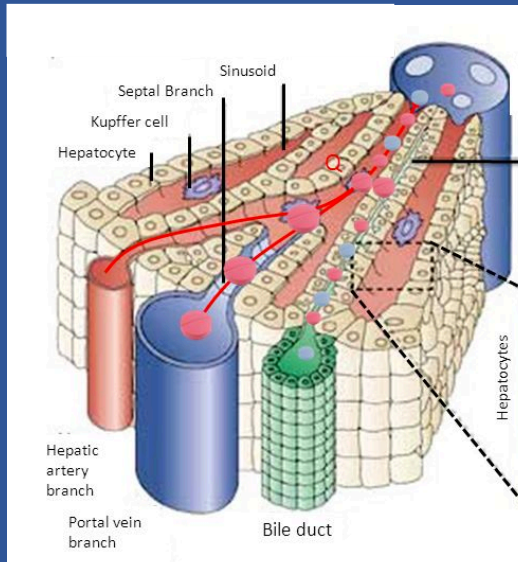
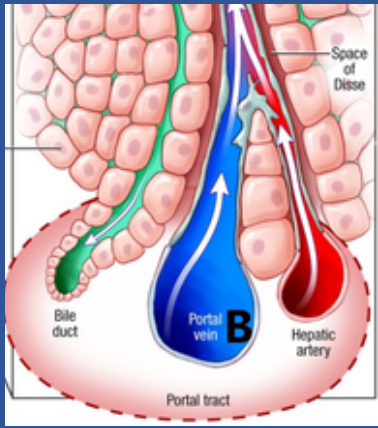
Anatomy of the Liver



Anatomy of the Hepatic Lobule

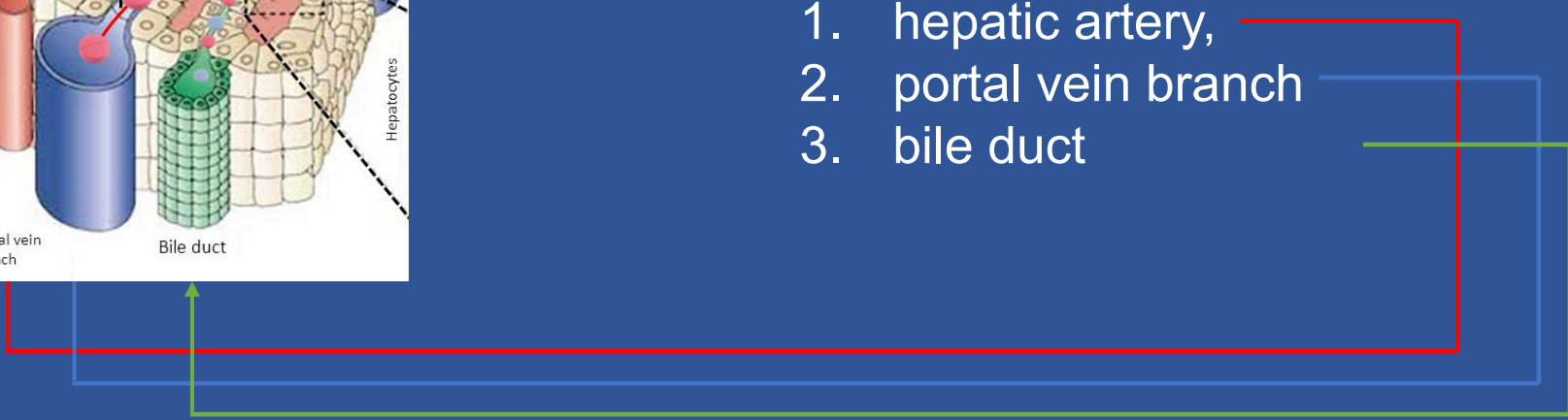


Stages of Fibrosis to Cirrhosis

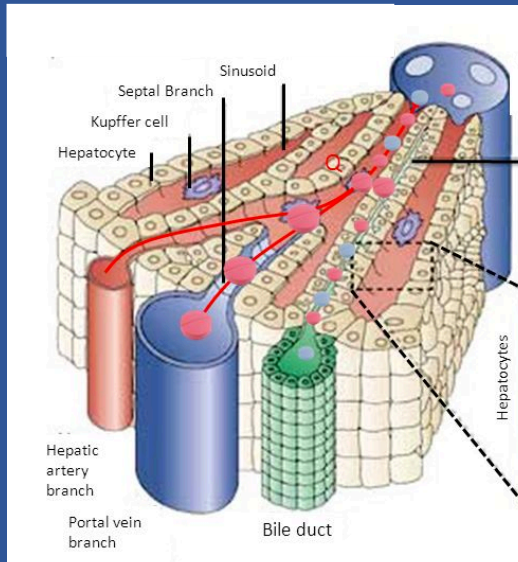
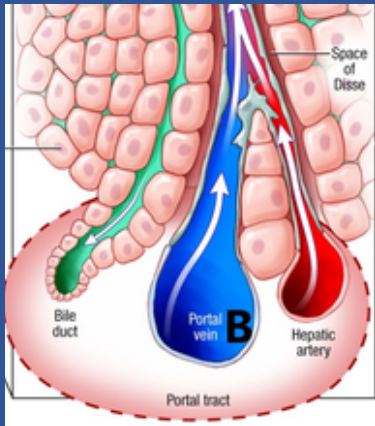


Portal triad:

1. hepatic artery,
2. portal vein branch
3. bile duct



Stages of Fibrosis to Cirrhosis

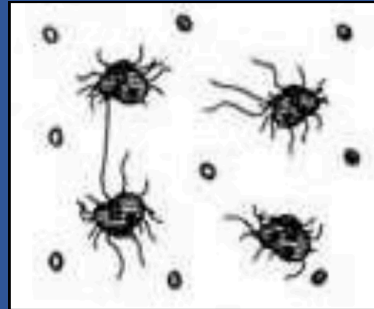


F1



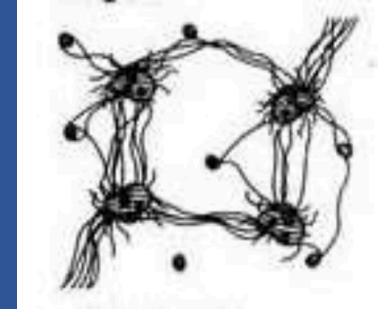
Portal fibrosis

F2



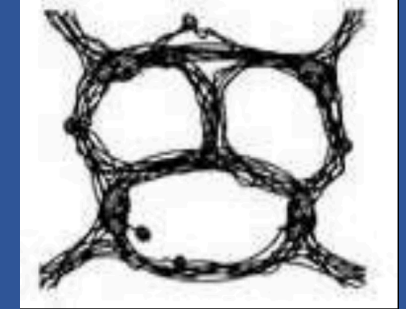
Peri-portal fibrosis

F3



Septal fibrosis
Portal-portal fibrosis, "bridging"

F4



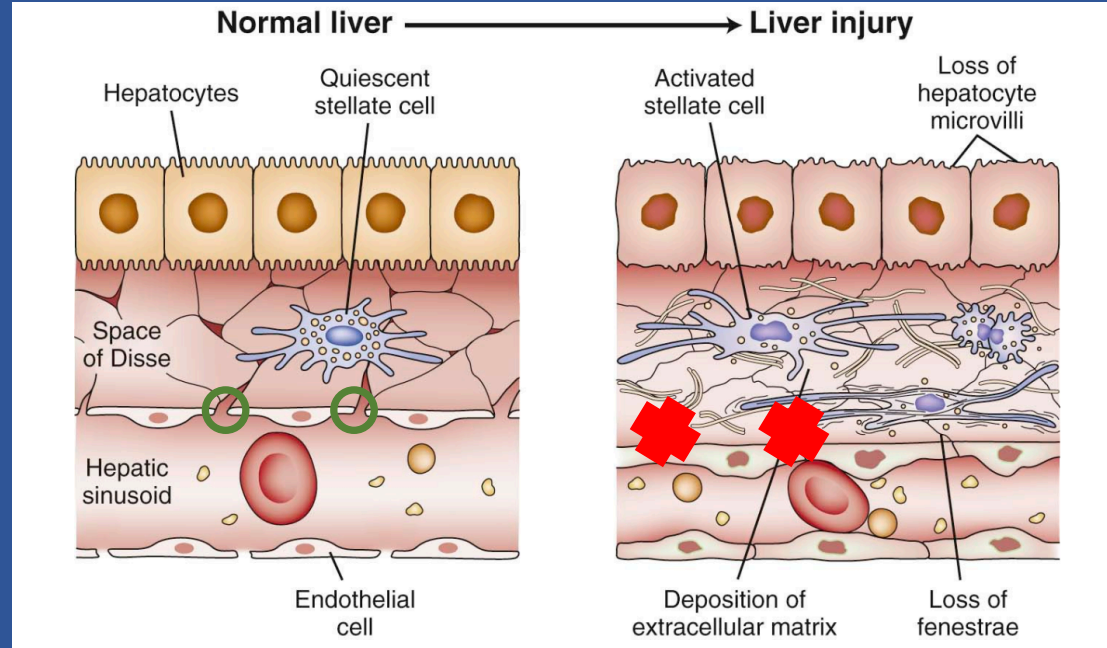
Cirrhosis

Normal Portal Pressure

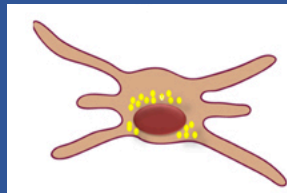
Increased Portal Pressure



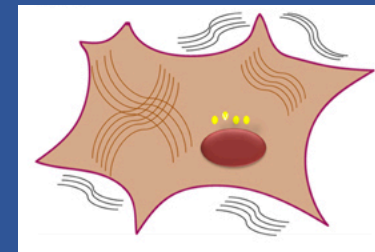
The Cell Responsible for Fibrosis: Stellate Cell



Normal cells



Fibrosis



Normal Portal Pressure

Increased Portal Pressure



Etiologies of Cirrhosis

Viral

- HBV/HDV
- HCV
- HEV (transplant)

Toxic

- Alcohol
- Arsenic

Autoimmune

- Autoimmune hepatitis
- Primary biliary cholangitis
- Primary sclerosing cholangitis

Metabolic

- NAFLD/NASH
- Alpha-1 antitrypsin
- Glycogen storage disease
- Hemochromatosis
- Wilson

Biliary

- Atresia
- Biliary stone
- Tumor

Vascular

- Budd-Chiari
- Cardiac

Genetic

- Cystic fibrosis
- Lysosomal storage

Drugs

- Prescribed or not

Portal Hypertension

Causes by:

1. Increased intrahepatic resistance of blood flow through the liver due to the nodules and fibrosis due to fibrosis
2. Increased blood flow to due to vasodilation of the splanchnic(abdominal) vascular bed

Cirrhosis Consists of Clinical Stages

Normal Portal Pressure

Increased Portal Pressure



Chronic liver
disease



Compensated



Decompensated



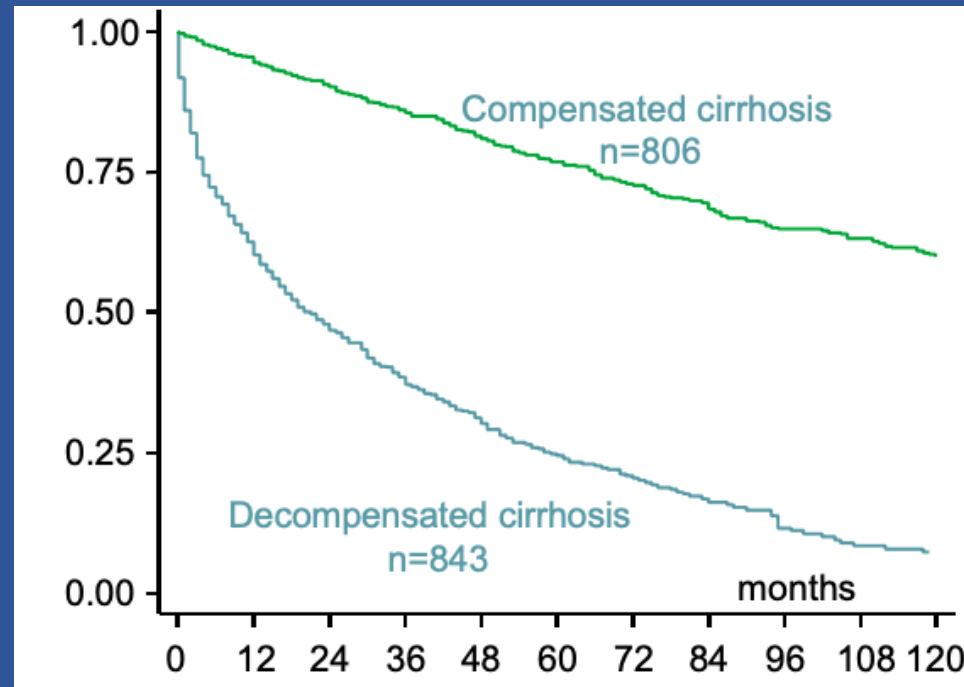
Death

40% cirrhosis
patients have NO
symptoms



- Variceal hemorrhage
- Ascites
- Encephalopathy
- Jaundice

Patients with Decompensated Cirrhosis Have Decreased Survival Compared to Compensated



**Median survival
>12 years**

**Median survival
~2 years**

How to Diagnose Cirrhosis?

Liver biopsy!

BUT, many clinical clues to help you....

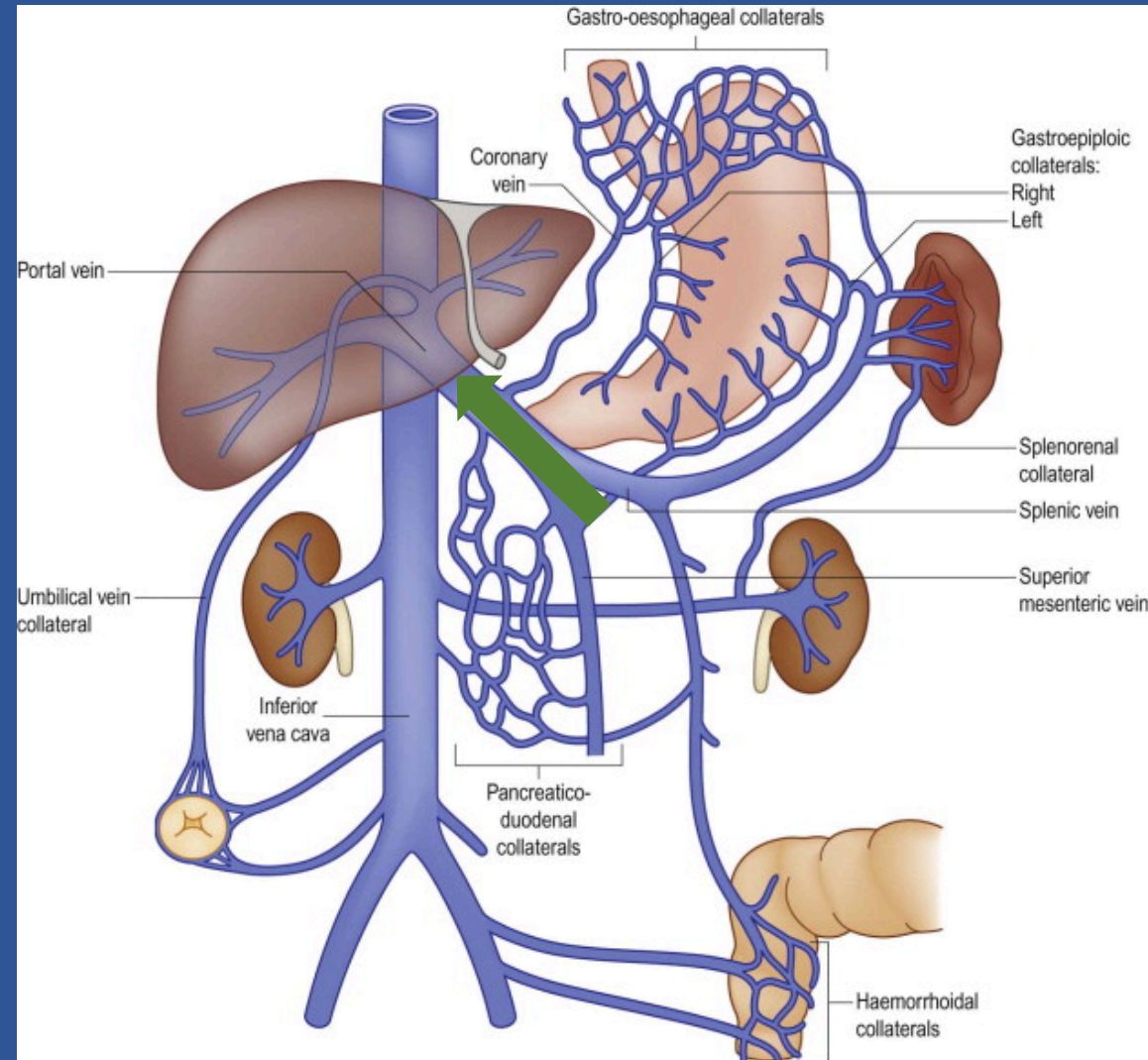
Estrogen Metabolism: Palmar erythema



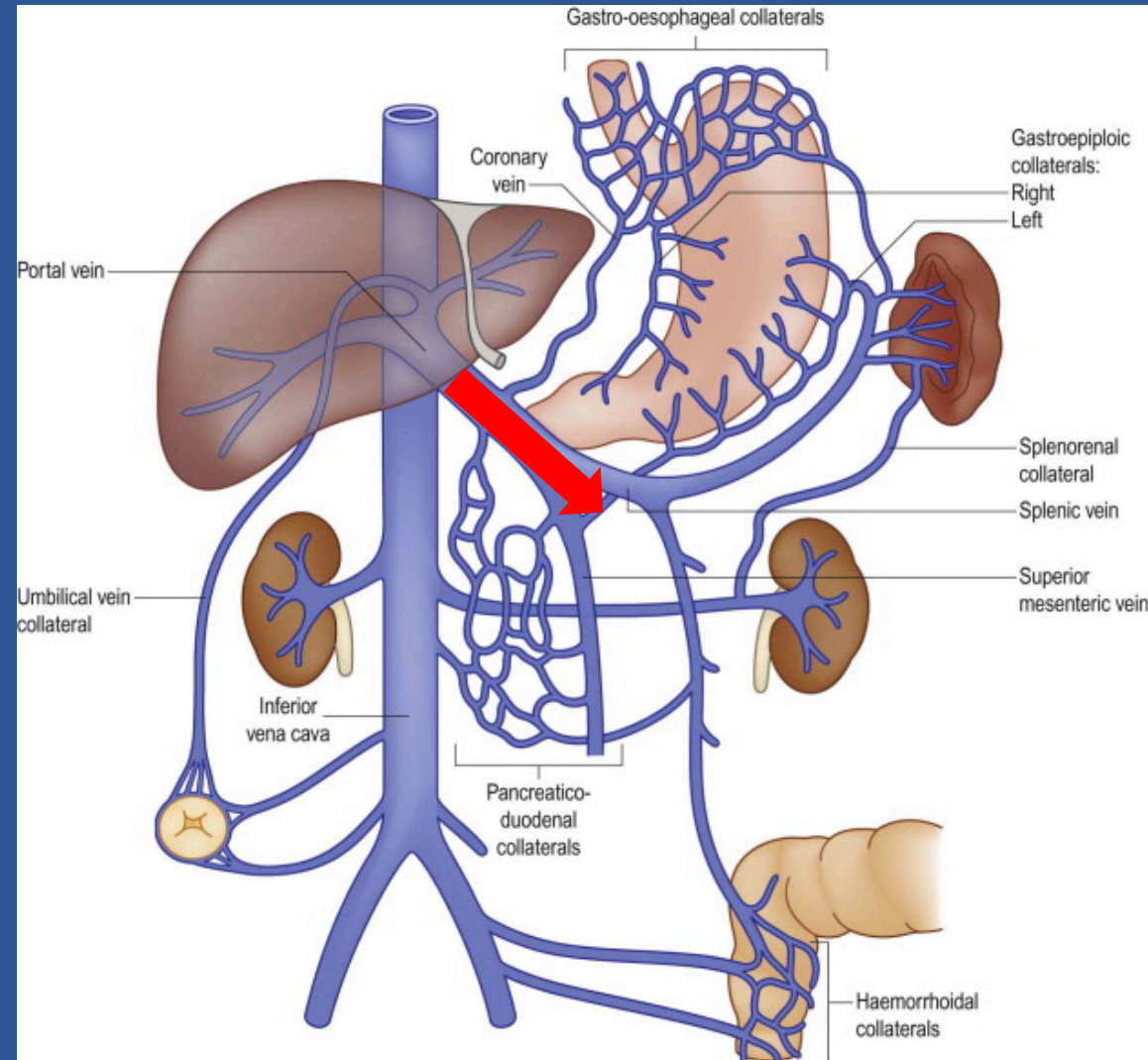
Estrogen Metabolism: Spider Telegiectasias



Collateral Vessels: GI Bleeding



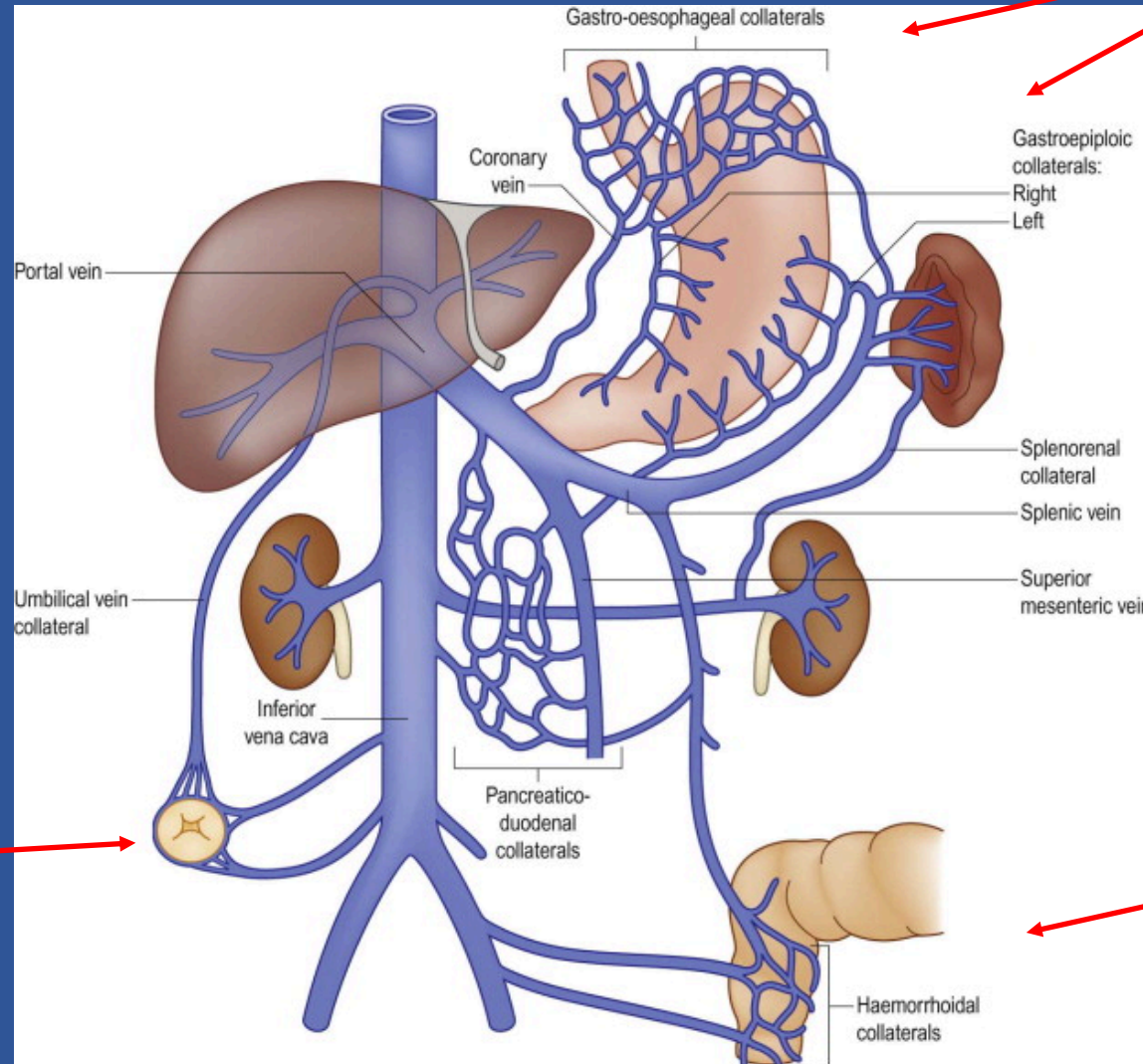
Collateral Vessels: GI Bleeding



Collateral Vessels: GI Bleeding

Sites of porto-caval anastomosis:

1. Lower esophagus
2. Paraumbilical vein
3. Upper end of anal canal
4. Retroperitoneal



Esophageal and gastric varices

Portocaval shunts

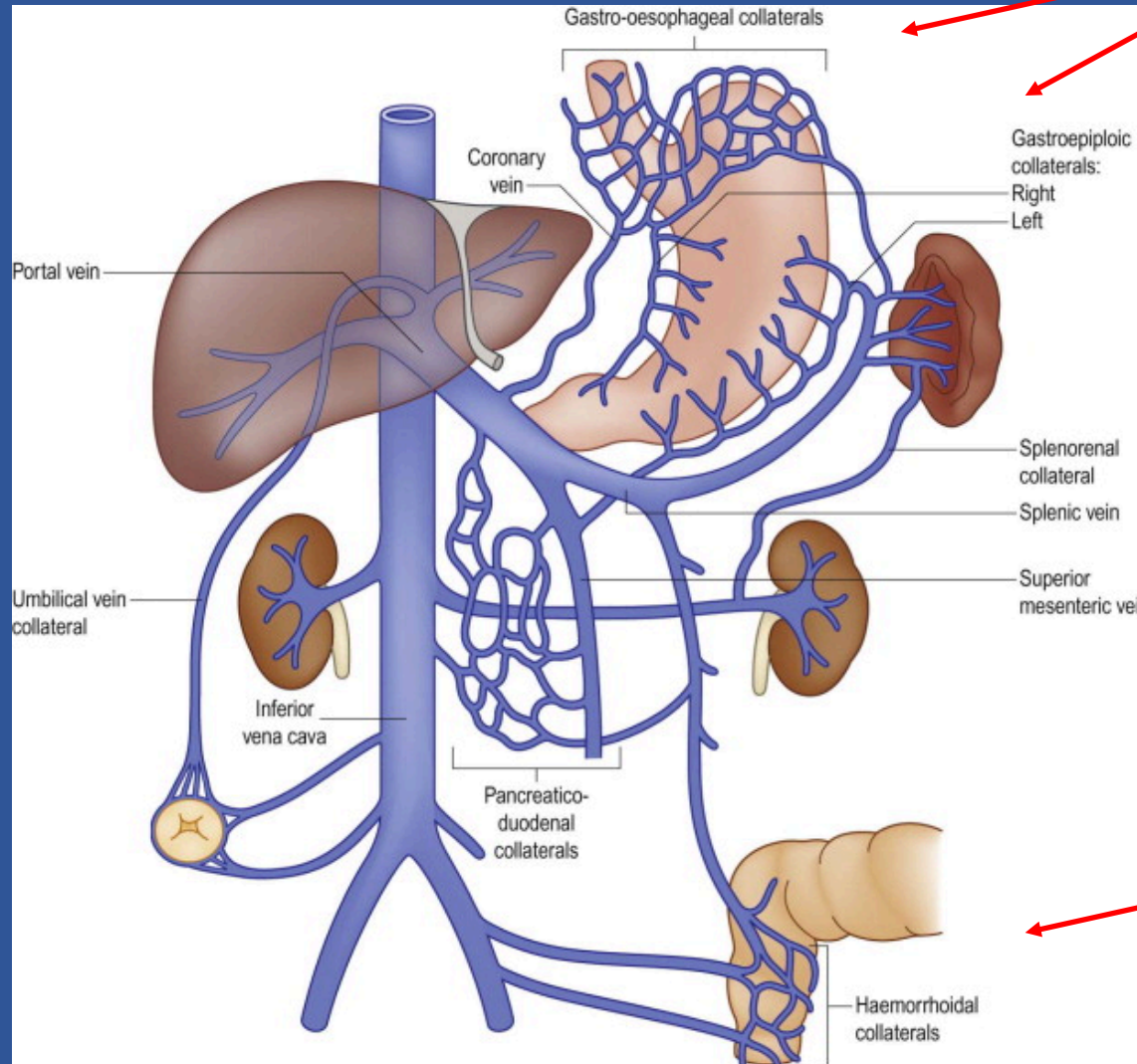
Rectal varices

Umbilical veins

Collateral Vessels: GI Bleeding

Treatment of varices/portal hypertension causing GI bleeding:

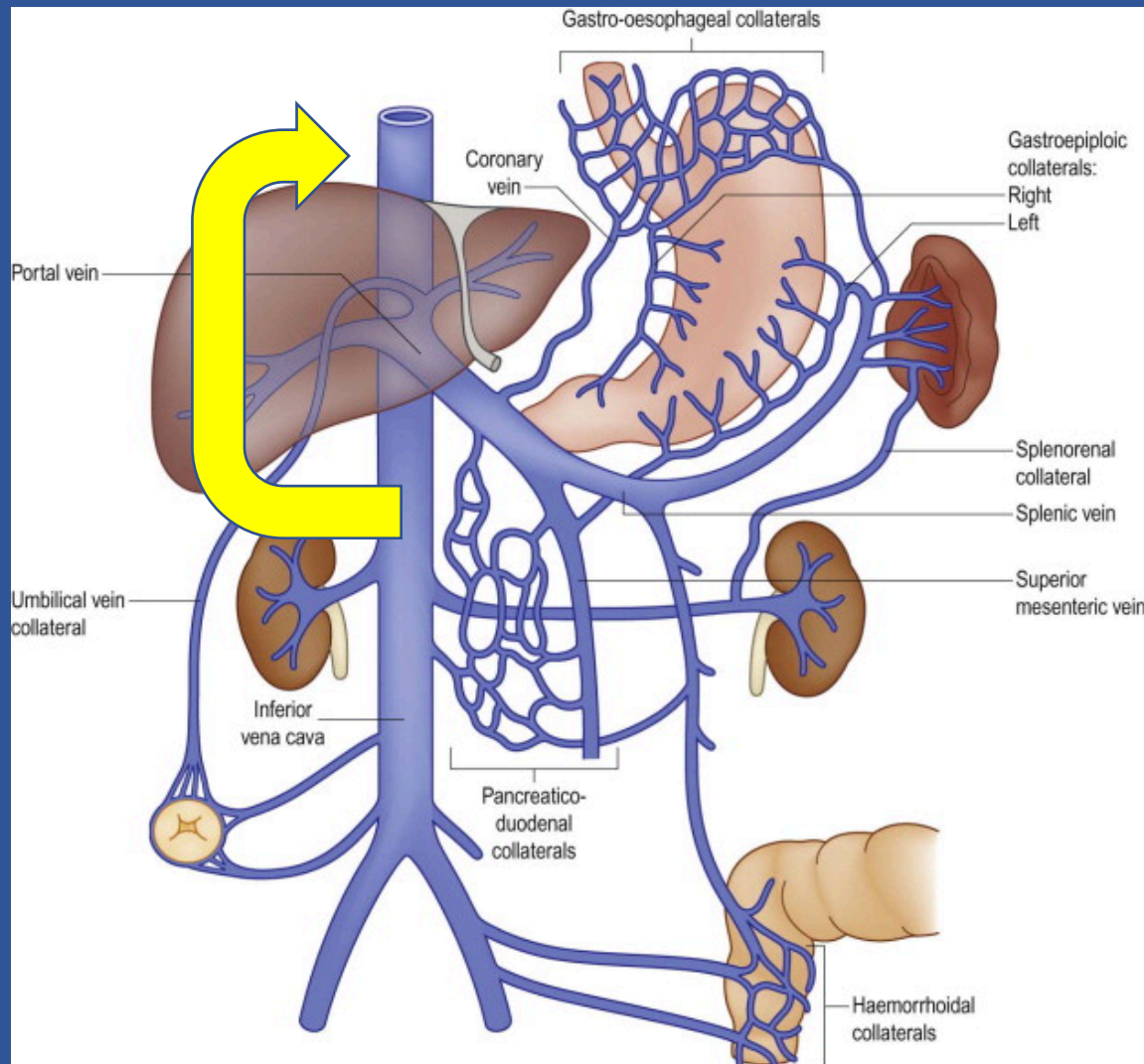
1. Band ligation
2. Sclerotherapy
3. Non-selective beta blockers
4. TIPSS (Transjugular Intrahepatic PortoSystemic Shunt)



Esophageal and gastric varices

Rectal varices

Collateral Vessels: Encephalopathy



Confusion and change in mental status/cognition associated with ammonia levels and swelling of brain cells (astrocytes)

Treat with:

1. Lactulose
2. Rifaximin
3. Other antibiotics
4. Zinc
5. Change in microbiome?

Ascites

Askites → Greek work “bag”



Accumulation of fluid in the peritoneal cavity

Pearls:

1. Always do paracentesis for new ascites
2. Always do paracentesis for patients admitted with ascites (infection!!!)

Treatment:

1. Low salt diet (<2 grams/day)
2. Water pills
3. Paracenteses

Spontaneous Bacterial Peritonitis (SBP)

Patients with ascites are at risk of SBP

Occurs due to translocation of bacteria from the gut to the ascitic fluid (has low immune cells due to cirrhosis)

Not always symptomatic (diagnosed with paracentesis)

Primary prophylaxis in patients with GI bleeding important

Antihemostatic Changes

↓ Peripheral blood platelet count
↑ Nitric oxide and prostacyclin

↓ Factors II, V, VII, IX, X, XI
↓ Fibrinogen

Dysfibrinogenemia

↓ Alpha-2 antiplasmin
↓ TAFI
↓ Factor XIII

↑tPA

Primary Hemostasis

Coagulation

Fibrinolysis

Prohemostatic Changes

↑ vWF
↓ ADAMTS-13

↓ Protein C and protein S
↓ Antithrombin
↓ TFPI

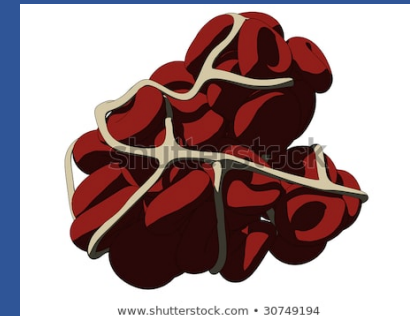
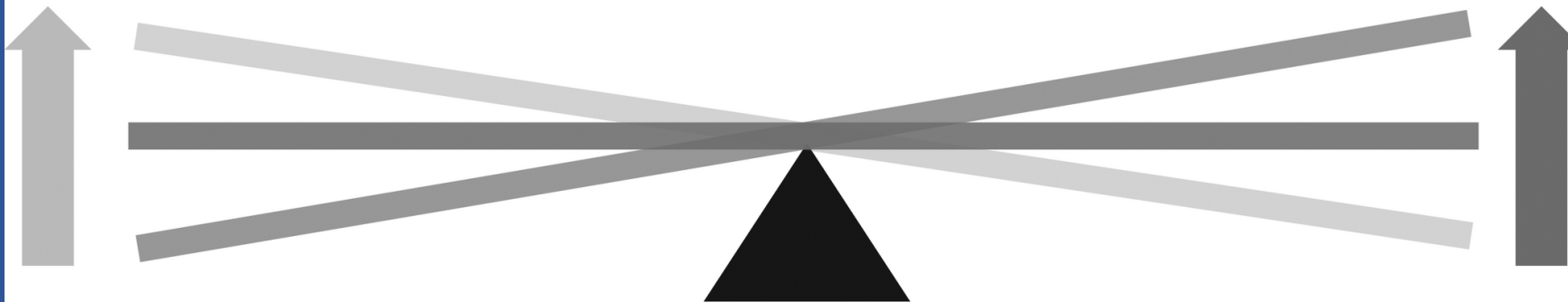
↑ Factor VIII

↓ Plasminogen

↑ PAI-1

Bleeding

Thrombosis



INR Is High and Platelets Are Low: Do I Transfuse?

INR and platelets are low BUT generally not increased risk of bleeding

Vitamin K

One trial at the VA showed benefit

AASLD and EASL do not recommend transfusing for paracentesis

Heart catheterization, liver biopsies...not increased risk

Summary

Cirrhosis is a complex disease

Affects many organs and has many manifestations

Understanding these differences change management and improve care