Cirrhosis and Portal Hypertension Management and Treatment Options

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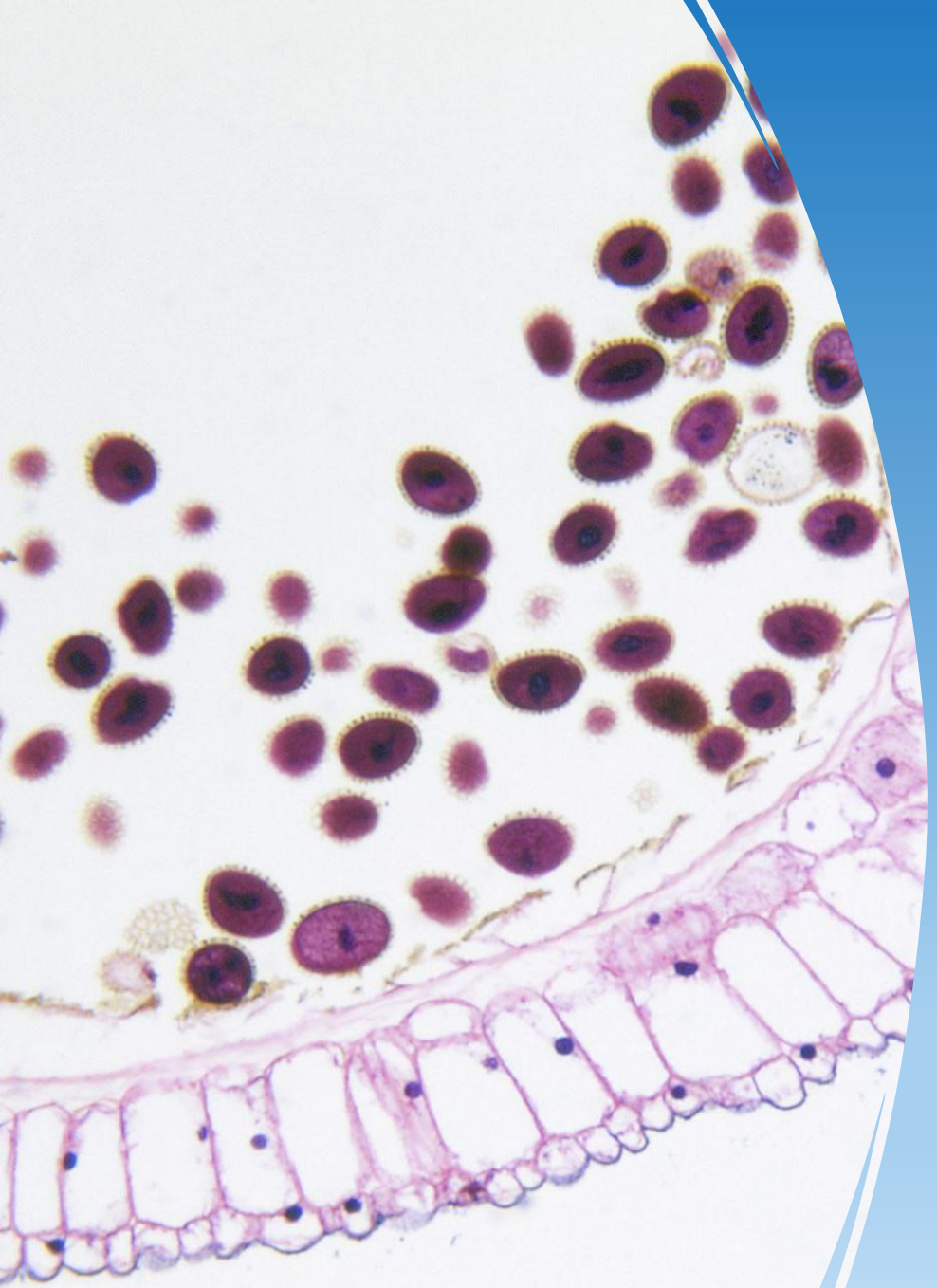
Disclosures

- None

Learning Objectives

- Recognize signs of cirrhosis
- Management of complications of cirrhosis
- Scoring systems for cirrhosis
- Treatment options for complications of cirrhosis



Cirrhosis

- The most common cause of portal hypertension is cirrhosis, or scarring of the liver. Cirrhosis results from the healing of a liver injury caused by hepatitis, alcohol abuse or other causes of liver damage. In cirrhosis, the scar tissue blocks the flow of blood through the liver and slows its processing functions.
- Portal hypertension may also be caused by thrombosis, or a blood clot that develops in the portal vein.

What does the Child-Pugh score tell you?

- The Child-Pugh score is a system for assessing the prognosis — including the required strength of treatment and necessity of liver transplant — of chronic liver disease, primarily cirrhosis.
- It provides a forecast of the increasing severity of your liver disease and your expected survival rate.

2 Minute Medicine®	Child-Pugh Score		2minutemedicine.com
Factor	**1 point**	**2 points**	**3 points**
Total bilirubin (µmol/L)	<34	34-50	>50
Serum albumin (g/L)	>35	28-35	<28
PT INR	<1.7	1.71-2.30	>2.30
Ascites	None	Mild	Moderate to Severe
Hepatic encephalopathy	None	Grade I-II (or suppressed with medication)	Grade III-IV (or refractory)

	Class A	**Class B**	**Class C**
Total points	5-6	7-9	10-15
1-year survival	100%	80%	45%

Table I. Child-Pugh score.

Why Do I Not Want to Operate on Cirrhotic Patients ?

- In their 1997 study, Mansour et al found the mortality in Child's
- class A was 10%,
- Class B 30%
- Class C 82%

Complications of Cirrhosis

- Encephalopathy
- Bleeding esophageal varices
- Ascites
- Coagulopathy

Portal Hypertension

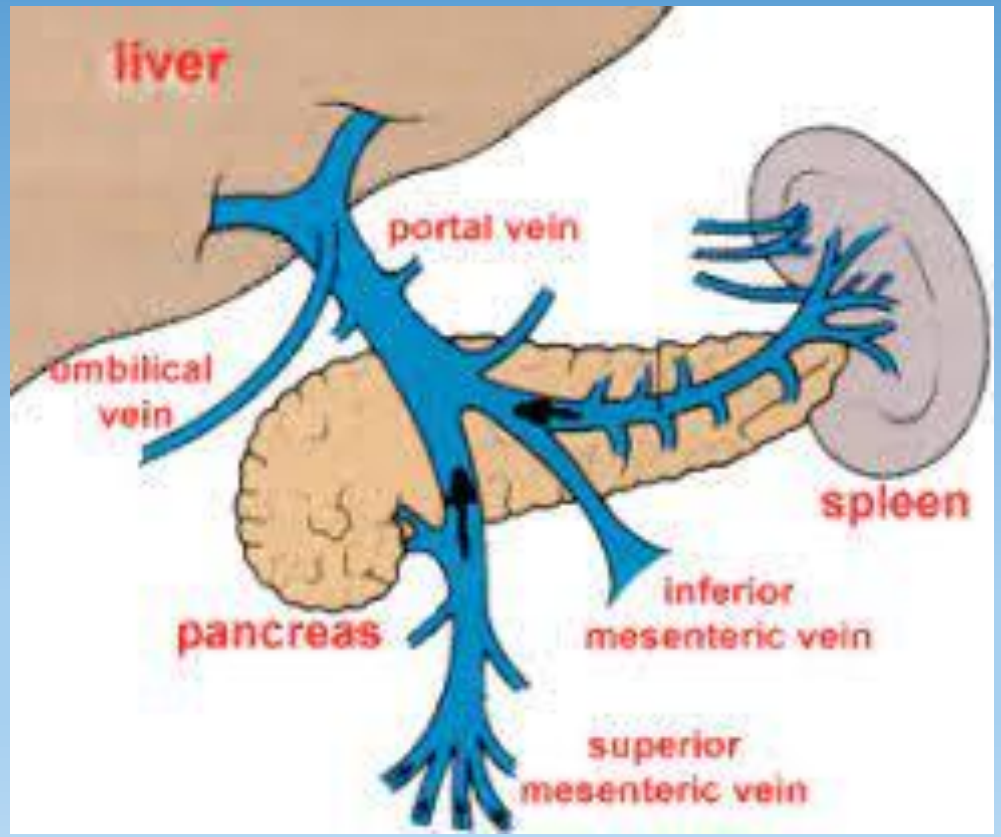
Portal hypertension is high blood pressure in the portal vein.

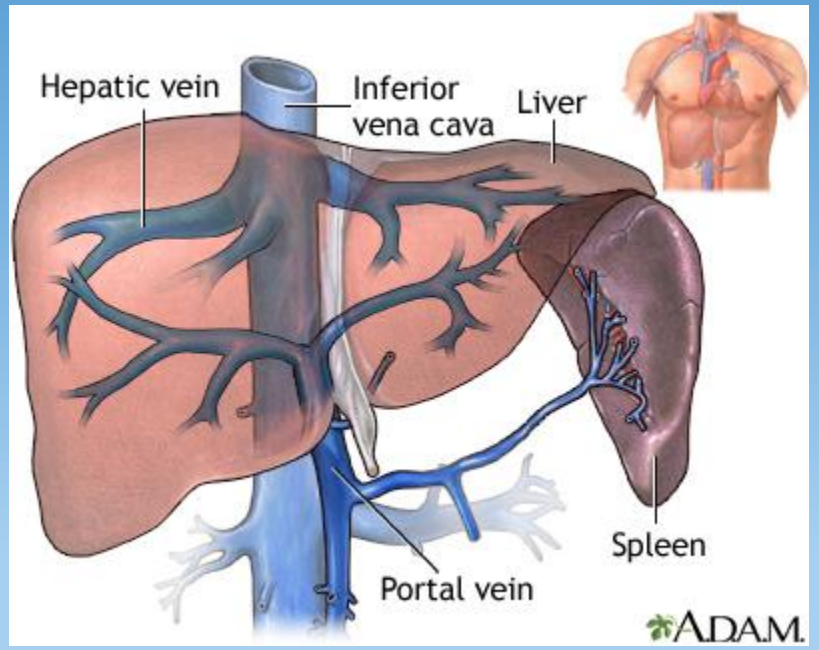
The portal vein is located in the abdomen. It gets blood from large and small intestines, stomach, pancreas, spleen and carries it to the liver.

Locations

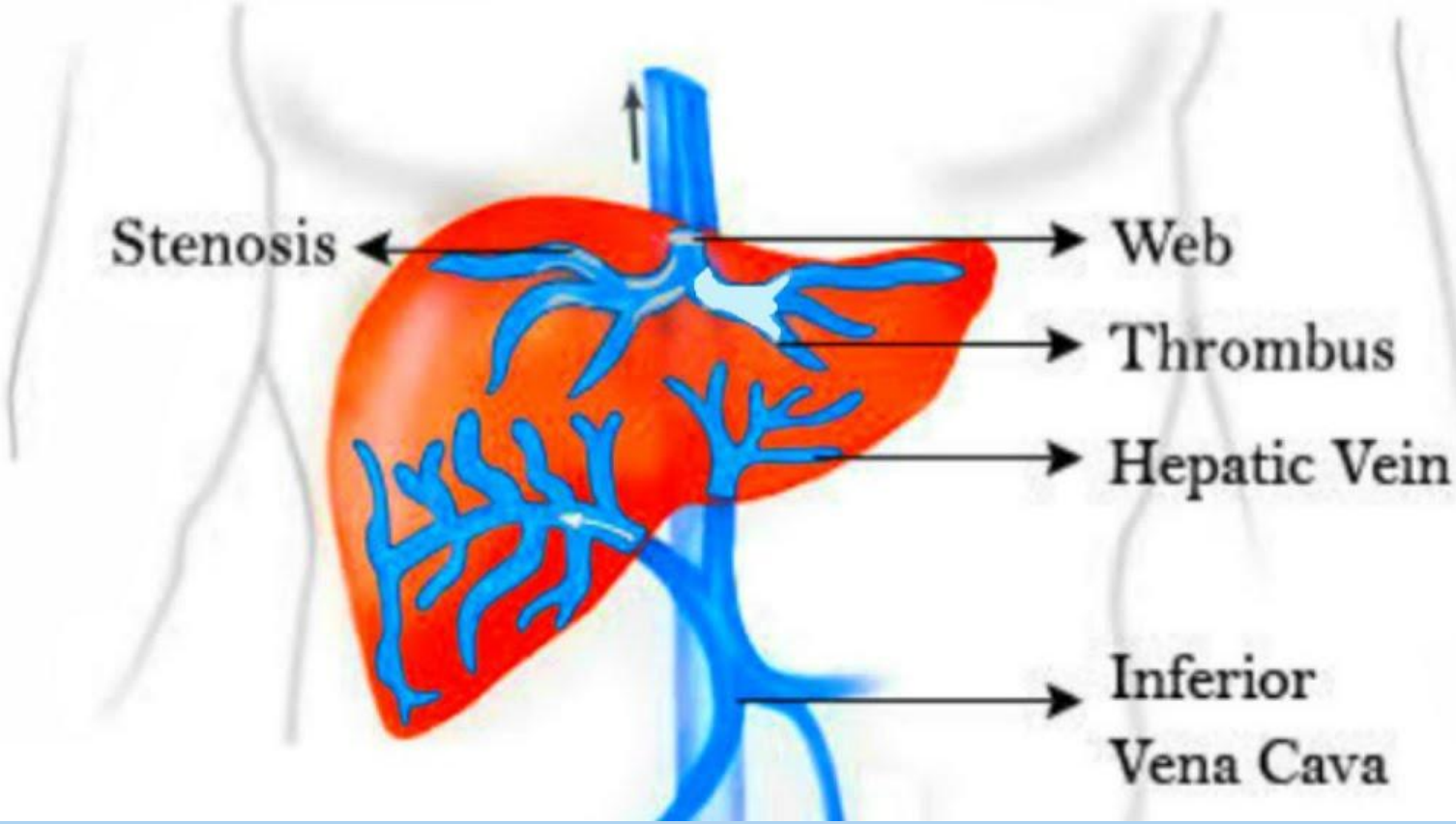
Pre-sinusoidal	Sinusoidal	Post- sinusoidal
• Portal Vein thrombus	• cirrhosis	• Hepatic vein thrombosis • Bud-chiari syndrome

Venous Blood Flow





BUDD - CHIARI SYNDROME



Encephalopathy treatment

Decrease protein intake

Lactulose

Manage cerebral edema in acute failure

- Cause of death in liver failure

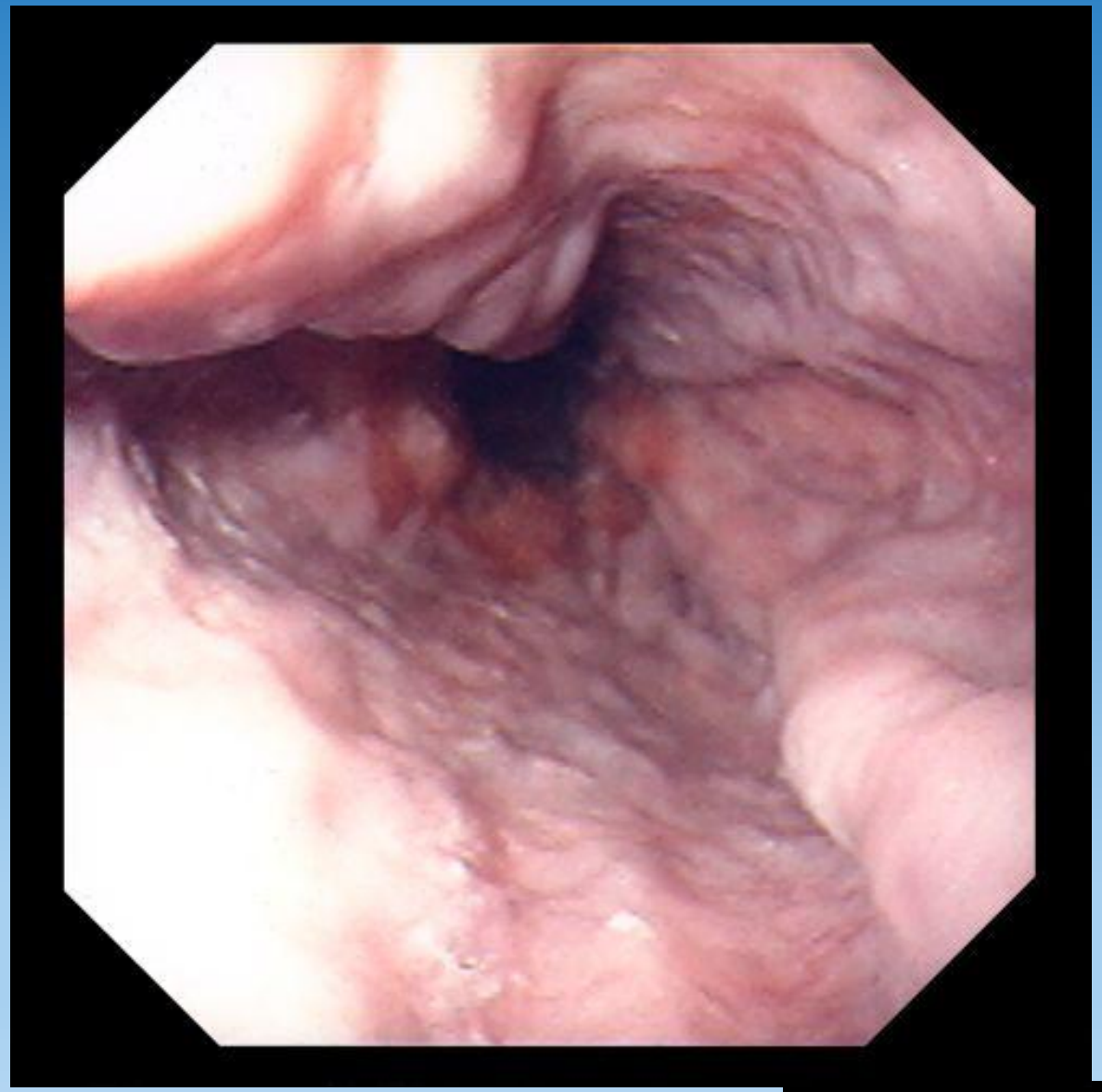
Encephalopathy

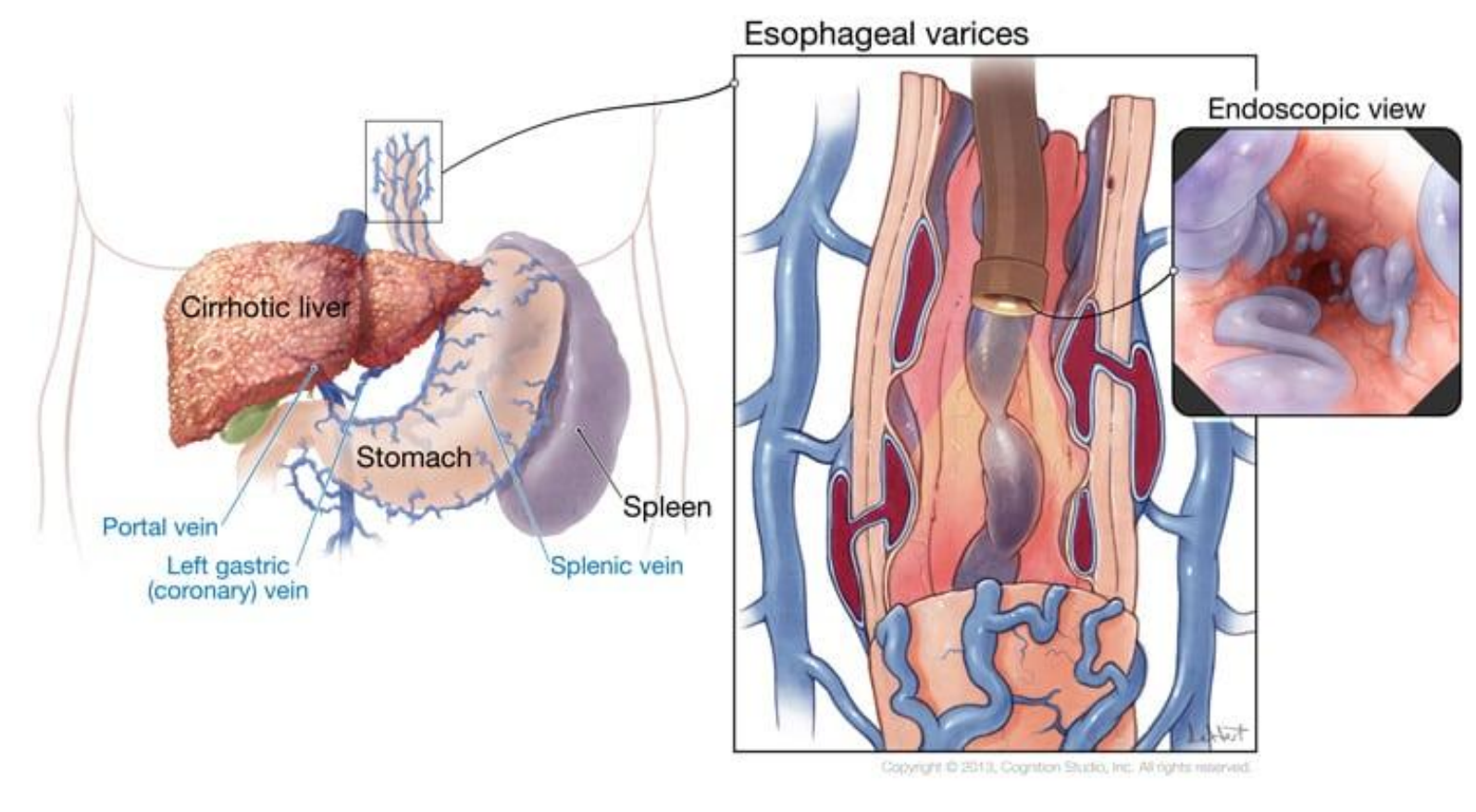
Lactulose is also used to reduce the amount of ammonia in the blood of patients with liver disease.

Its chief mechanism of action is by decreasing the intestinal production and absorption of ammonia.

Portal Hypertension

Esophageal Varices





Esophogeal Varices Bleeding

Left Coronary Vein (Left gastric vein)

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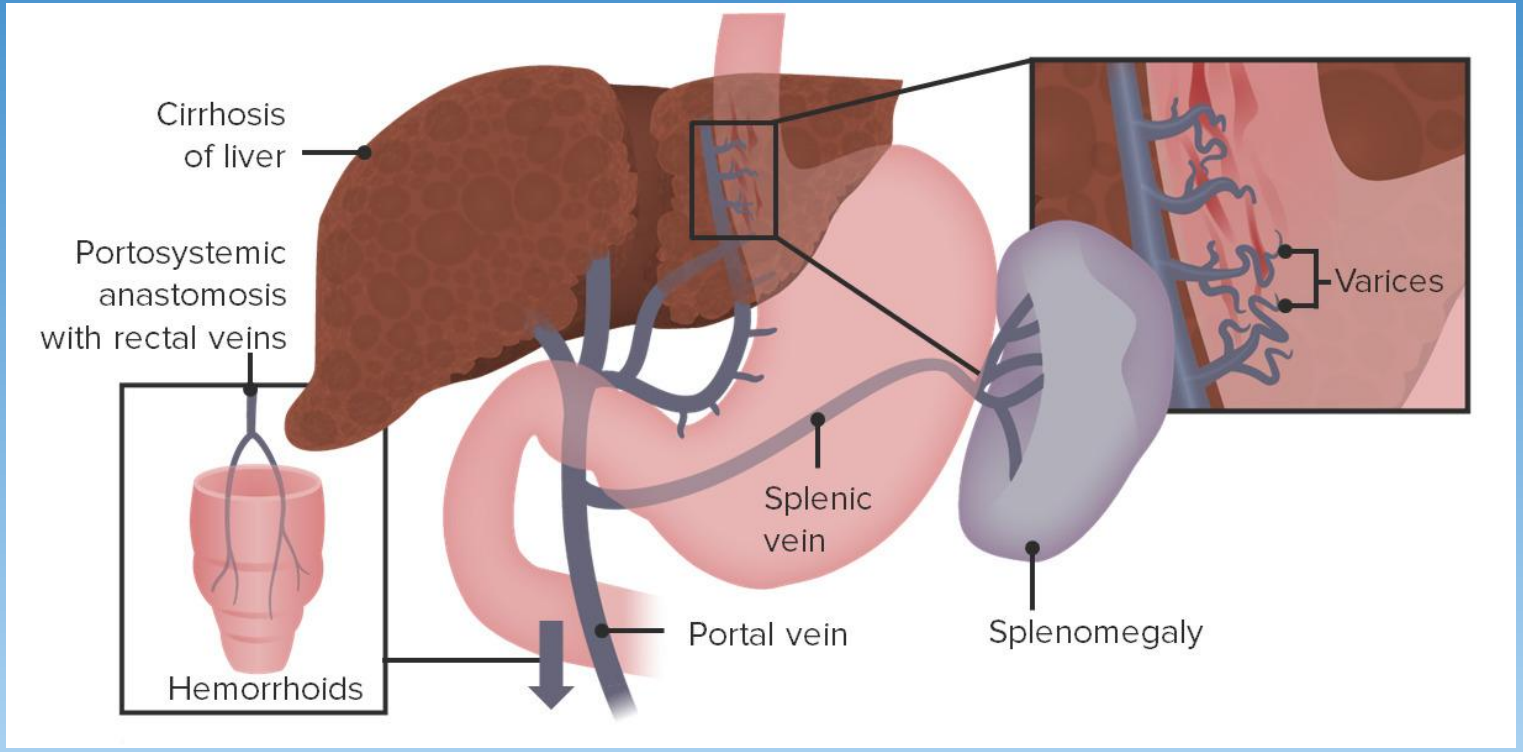
Portal Hypertension Hemorrhoids

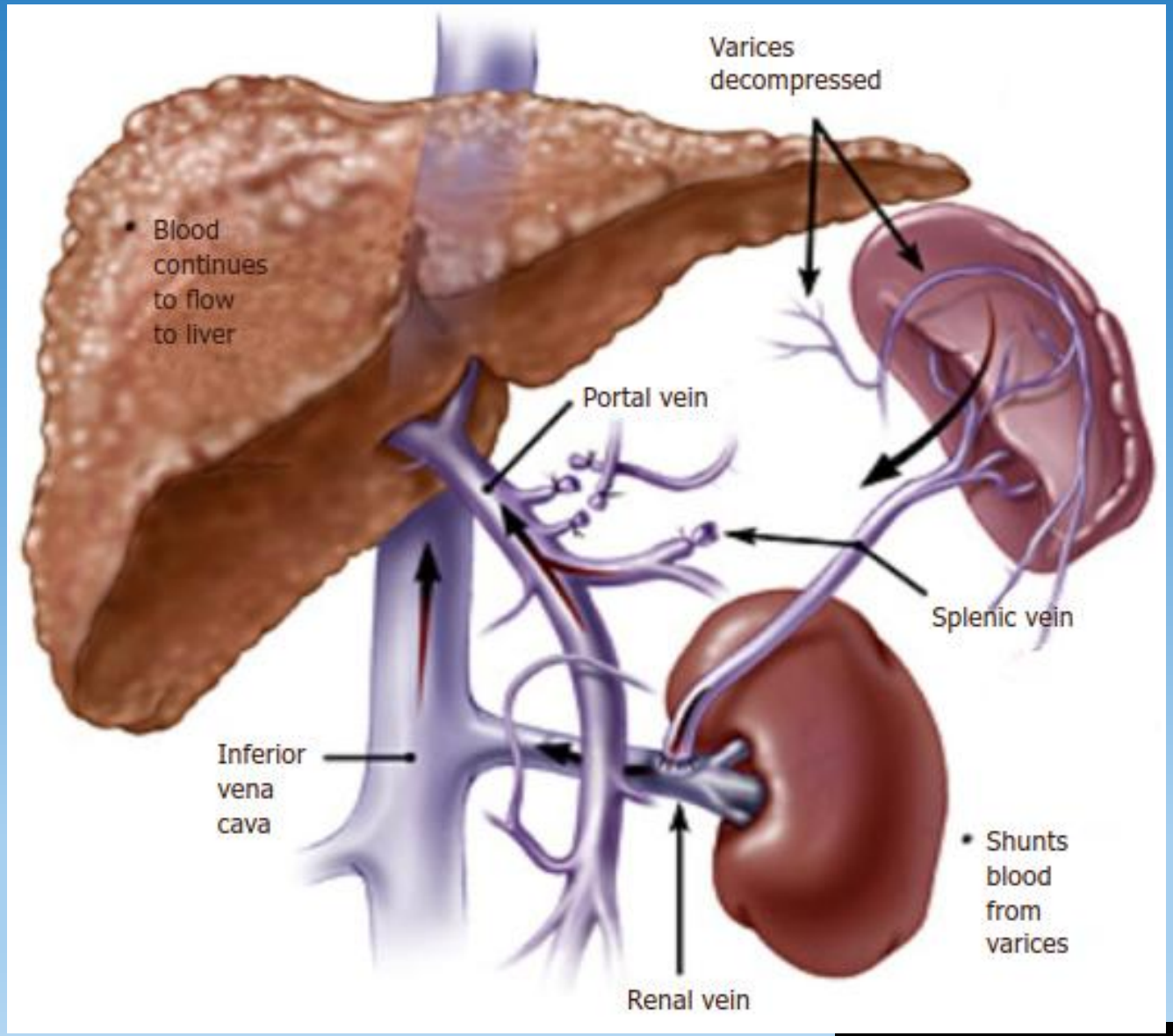


Balloon Occlusion

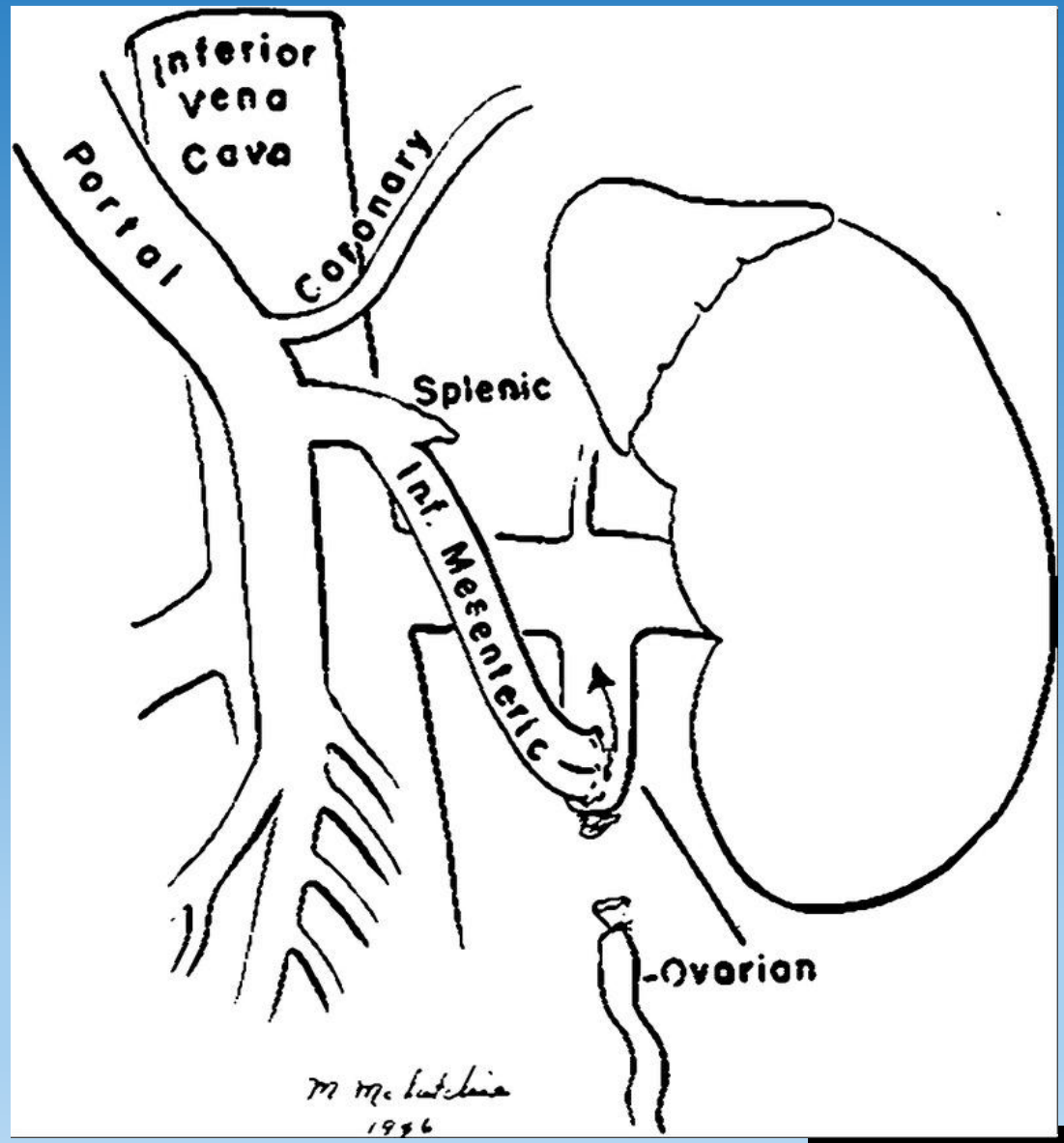
Shunts

- Hepatorenal
- Splenorenal
- Portal Caval
- TIPS

- Spleno-renal Shunt

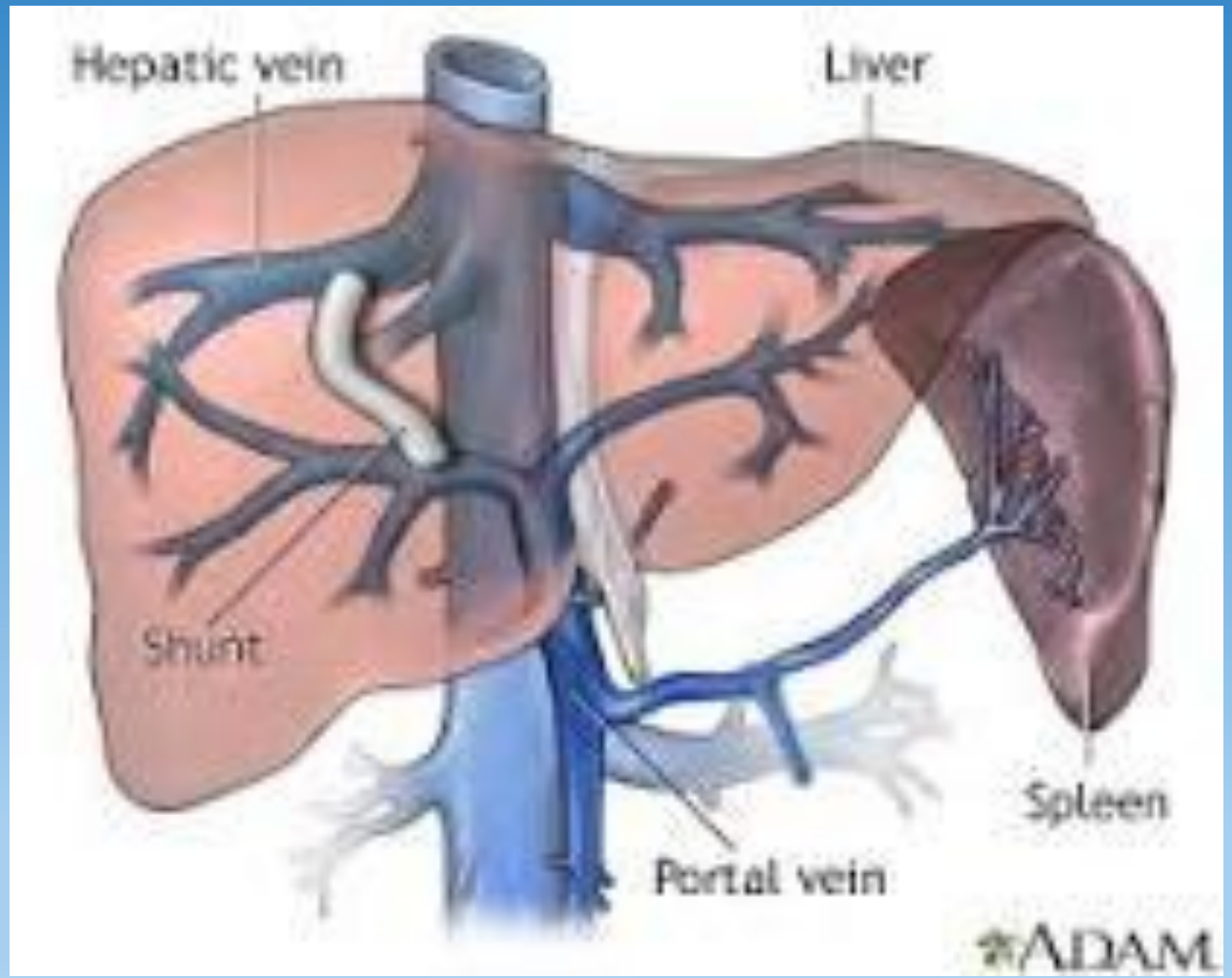


- Meso-renal shunt



Portosystemic Shunts

- A common method for reducing portal hypertension is to divert blood from the portal venous system to the systemic venous system by creating a communication between the hepatic portal vein and the IVC.
- Another way of reducing portal pressure is to join the splenic vein to the left renal vein, after splenectomy. (*splenorenal anastomosis or shunt*)

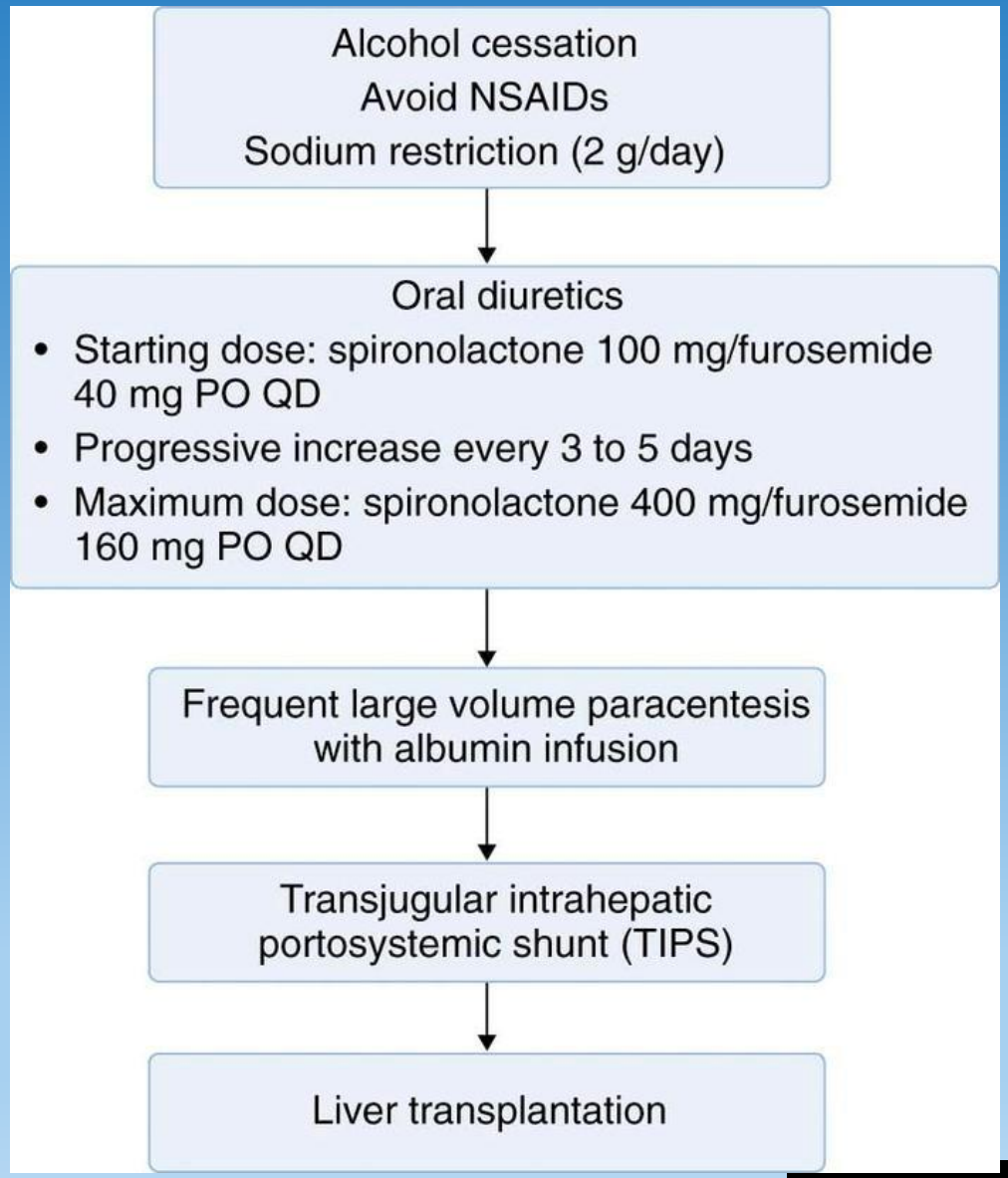


TIPS

- Transjugular Intrahepatic Portosystemic Shunt
- Shunt is placed to off load portal pressure

Ascites

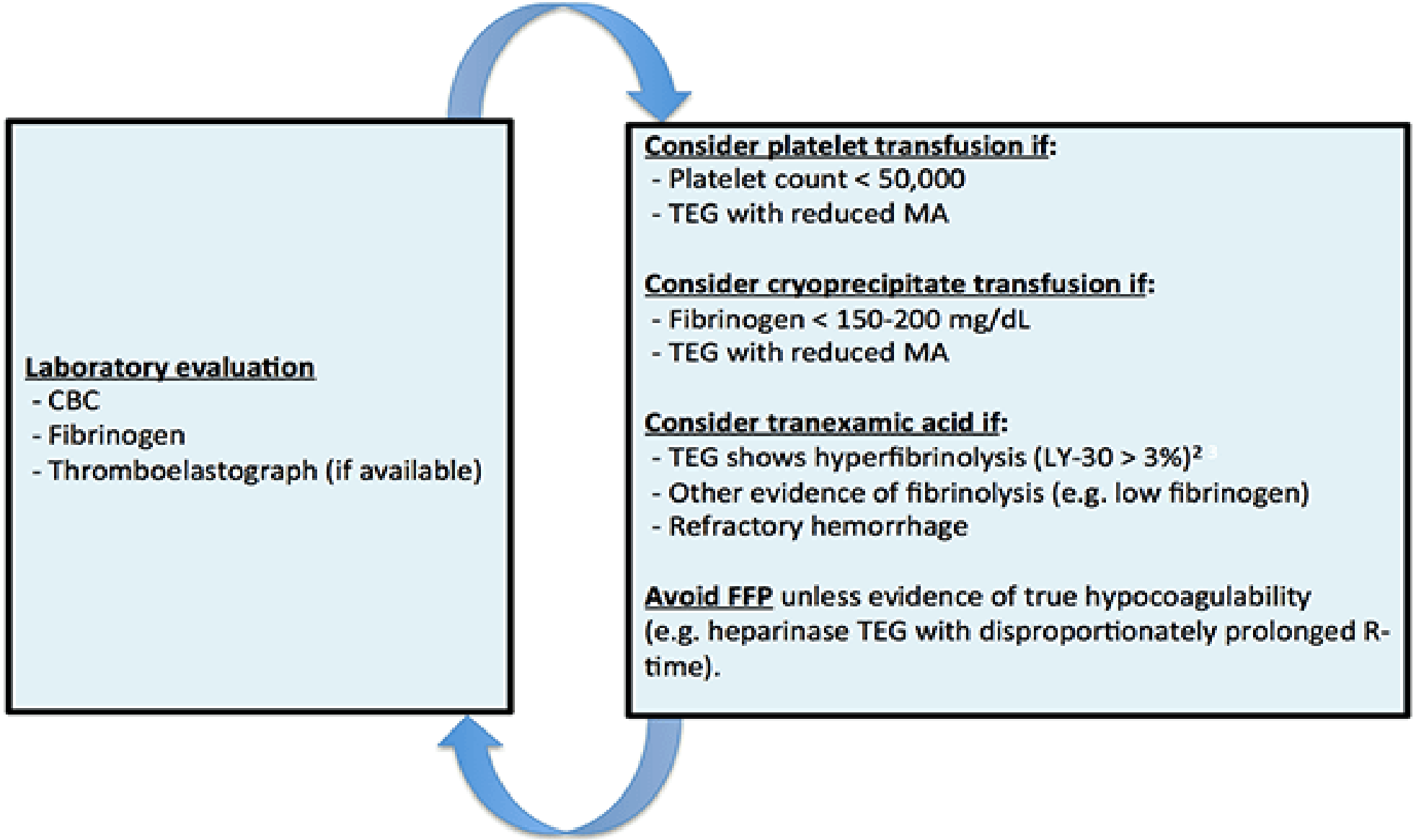
- How long can someone live with ascites from cirrhosis of the liver?
- Ascites is often the earliest complication of ESLD
- when present it indicates 50% 2-year mortality.
- Median survival is 6 months when ascites becomes refractory.
- Encephalopathy that is severe or refractory has a 12-month average survival.

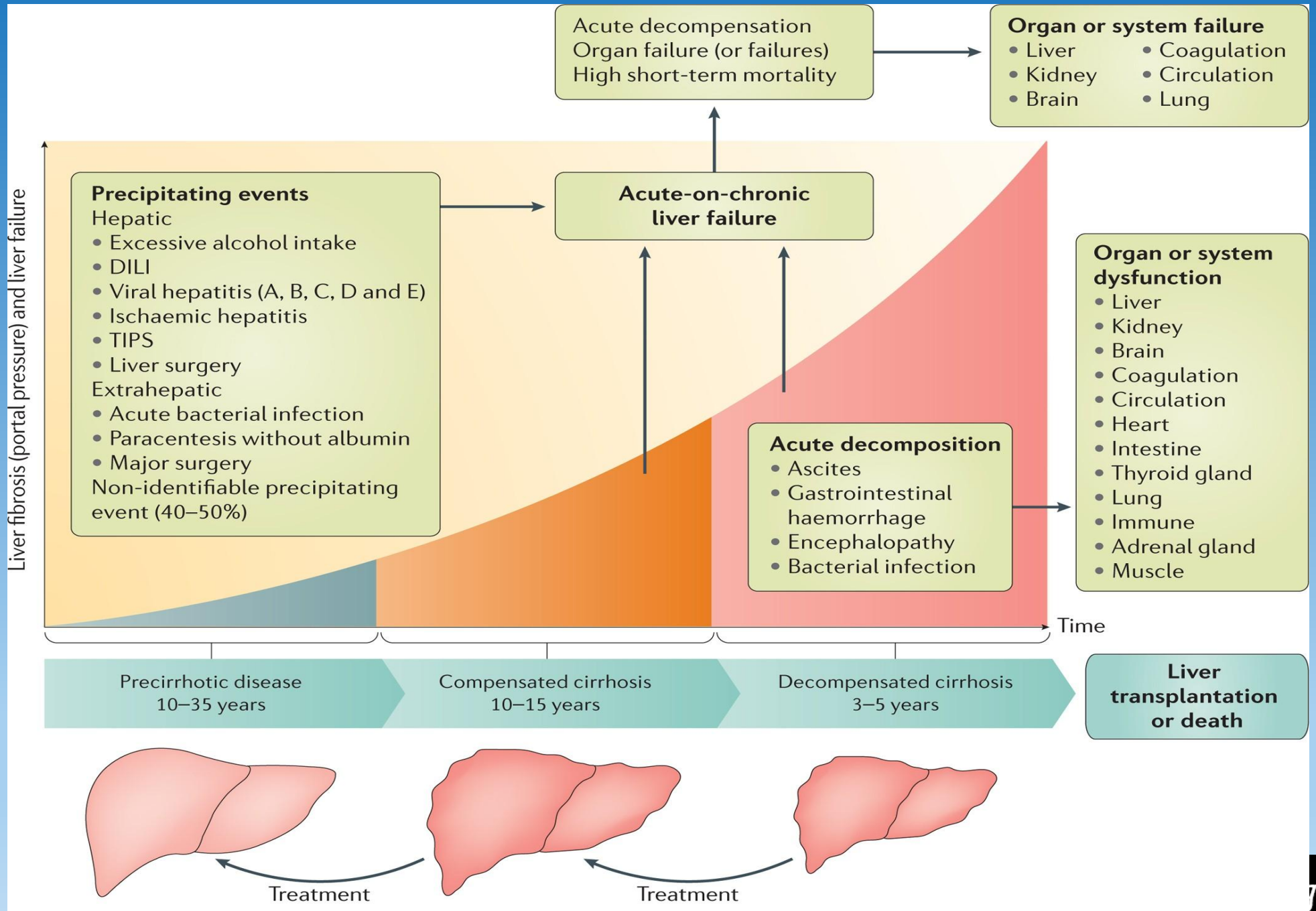


Coagulopathy

Changes that Impair Hemostatis	Changes that Impair Hemostatis
Primary Hemostasis	
• Thrombocytopenia • Functional platelet defects • Increased production of nitric oxide and prostacyclin	• Elevated levels of vWF • Decreased levels of ADAMTS-13
Secondary Hemostasis	
• Low levels of Factors II, V, VII, IX, X, XI • Vitamin K deficiency • Dysfibrinogenemia	• Elevated levels of Factor VIII • Decrease levels of Protein C & S, AT-III, α2-macroglobulin, heparin cofactor II
Fibrinolysis	
• Low levels of α2-antiplasmin, factor XIII, TAFI • Elevated levels of t-PA	• Low levels of plasminogen

Coagulation management in patient with cirrhosis and hemorrhage




Acute decompensation
Organ failure (or failures)
High short-term mortality
Organ or system failure
• Liver
• Kidney
• Brain
• Coagulation
• Circulation
• Lung
Liver fibrosis (portal pressure) and liver failure
Precipitating events
Hepatic
• Excessive alcohol intake
• DILI
• Viral hepatitis (A, B, C, D and E)
• Ischaemic hepatitis
• TIPS
• Liver surgery
Extrahepatic
• Acute bacterial infection
• Paracentesis without albumin
• Major surgery
Non-identifiable precipitating event (40–50%)
Acute-on-chronic liver failure
Acute decomposition
• Ascites
• Gastrointestinal haemorrhage
• Encephalopathy
• Bacterial infection
Organ or system dysfunction
• Liver
• Kidney
• Brain
• Coagulation
• Circulation
• Heart
• Intestine
• Thyroid gland
• Lung
• Immune
• Adrenal gland
• Muscle
Time
Precirrhotic disease
10–35 years
Compensated cirrhosis
10–15 years
Decompensated cirrhosis
3–5 years
Liver transplantation or death
Treatment
Treatment

Work Cited

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- portal vein - Google Search
- managing ascites in cirrhosis - Google Search