الجامعة السورية الخاصة
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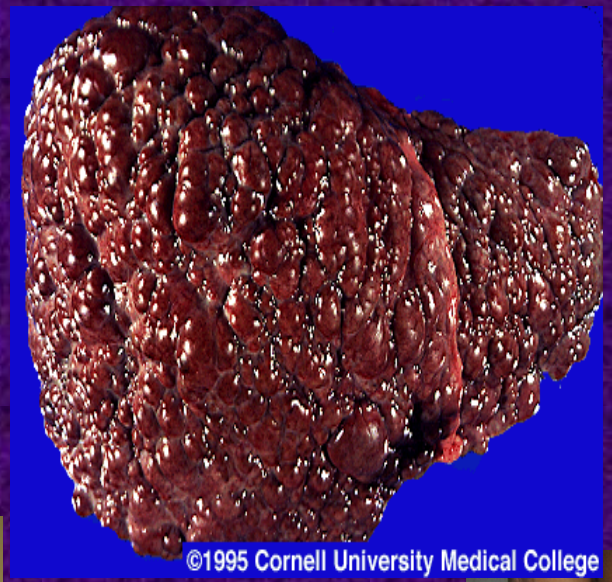
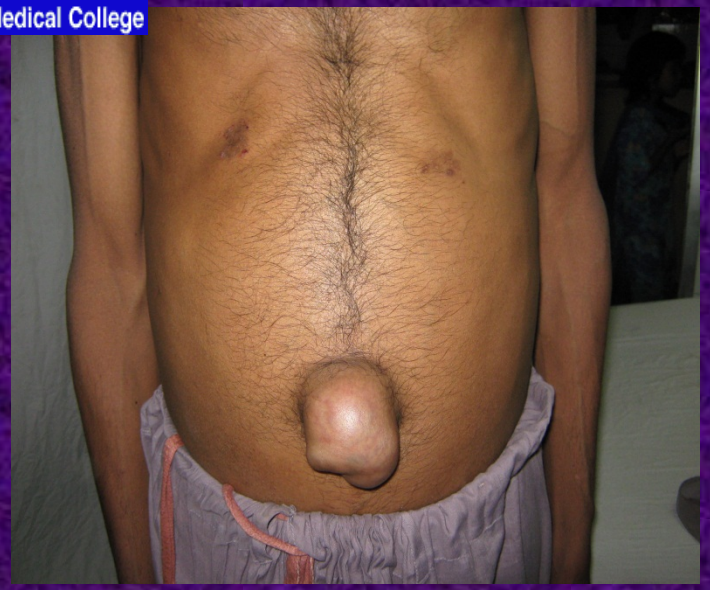
Third lecture 2019 -2020
complication of cirrhosis

Complication of cirrhosis

- Ascites
- Varices
- Encephalopathy
- Hepatocellular carcinoma
- HRS

Outline





- Ascites is the most common of the major complications of liver cirrhosis

Etymology

- Ascites
- Greek askiEs : “Dropsy”
- Meaning leather bag , sack

- 80% due to Cirrhosis (USA)

- **Although Ascites is not the root of all evil, many of the catastrophic complications of cirrhosis are rooted in ascites**

Harold Conn

Statistics

- 50% of cirrhotics will develop Ascites within 10 years of diagnosis.
- -
- 50% die within 5 years of onset

Ascites

occurs when there is a disruption in the pressure forces between intravascular and extravascular fluid spaces, which allows extravascular fluid to accumulate in the anterior peritoneal cavity.

Decreased osmotic pressure :

A- Hypoalbuminemia:

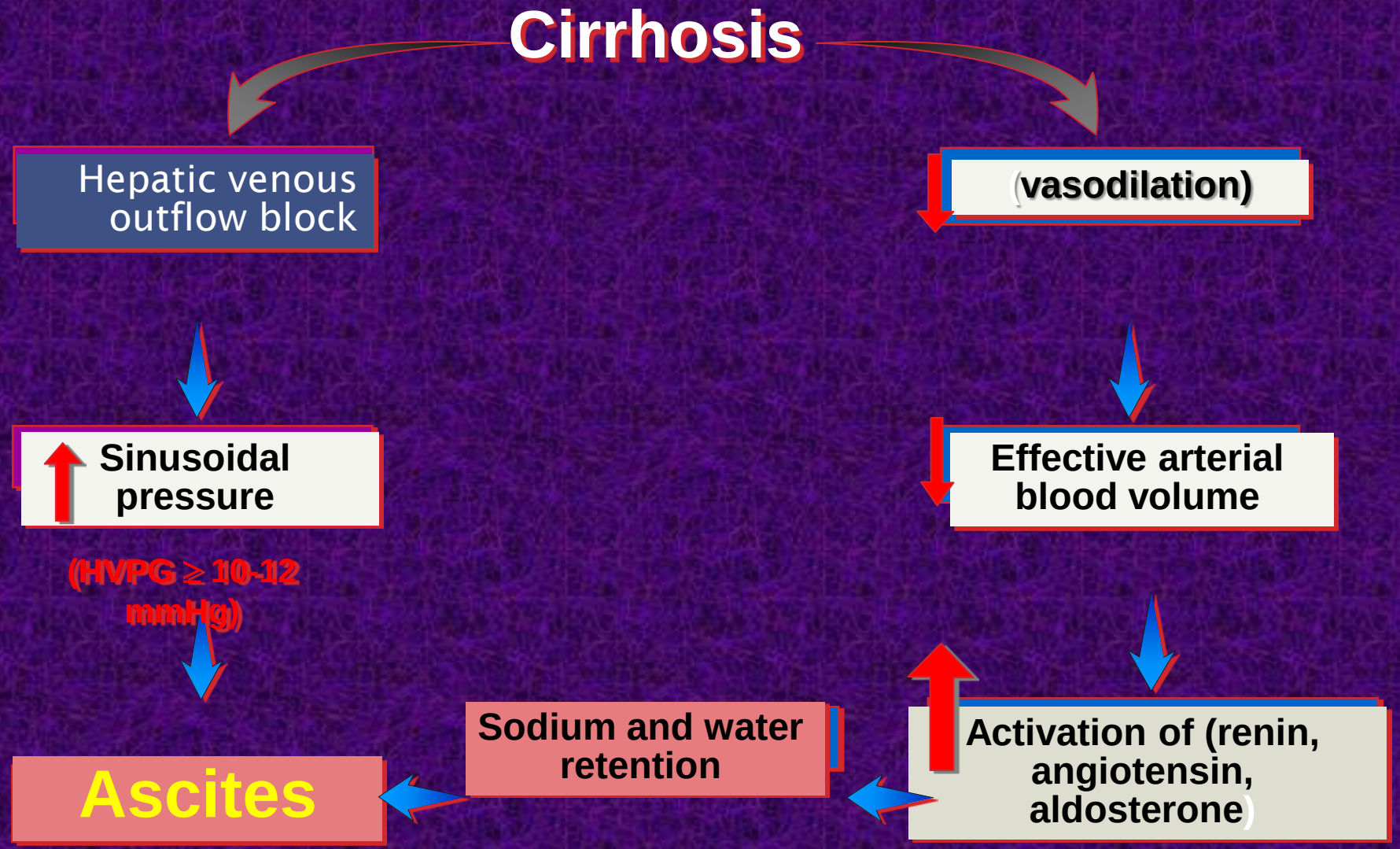
++ Nephrotic syndrome

++Protein-losing enteropathy

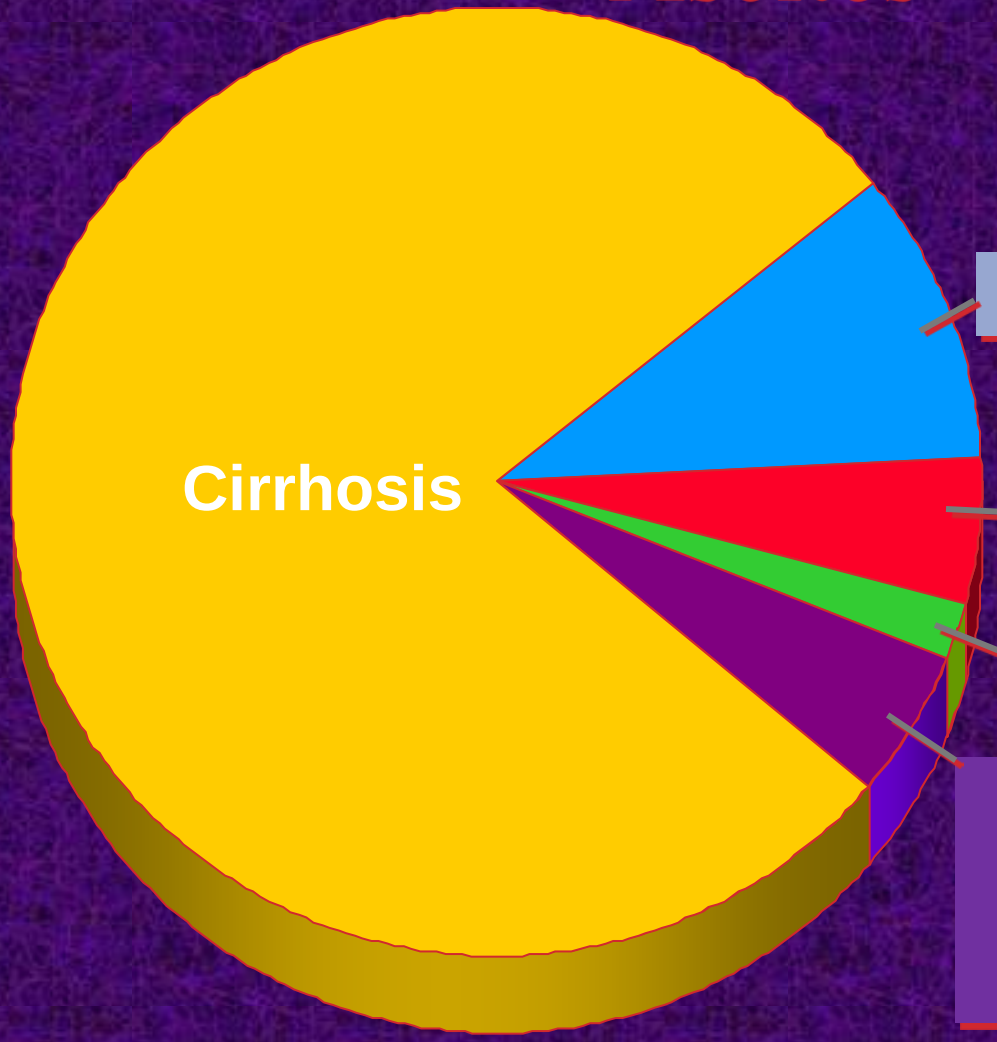
++Malnutrition

B- Cirrhosis or hepatic insufficiency

PATHOGENESIS OF ASCITES



Cirrhosis is the Most Common Cause of Ascites



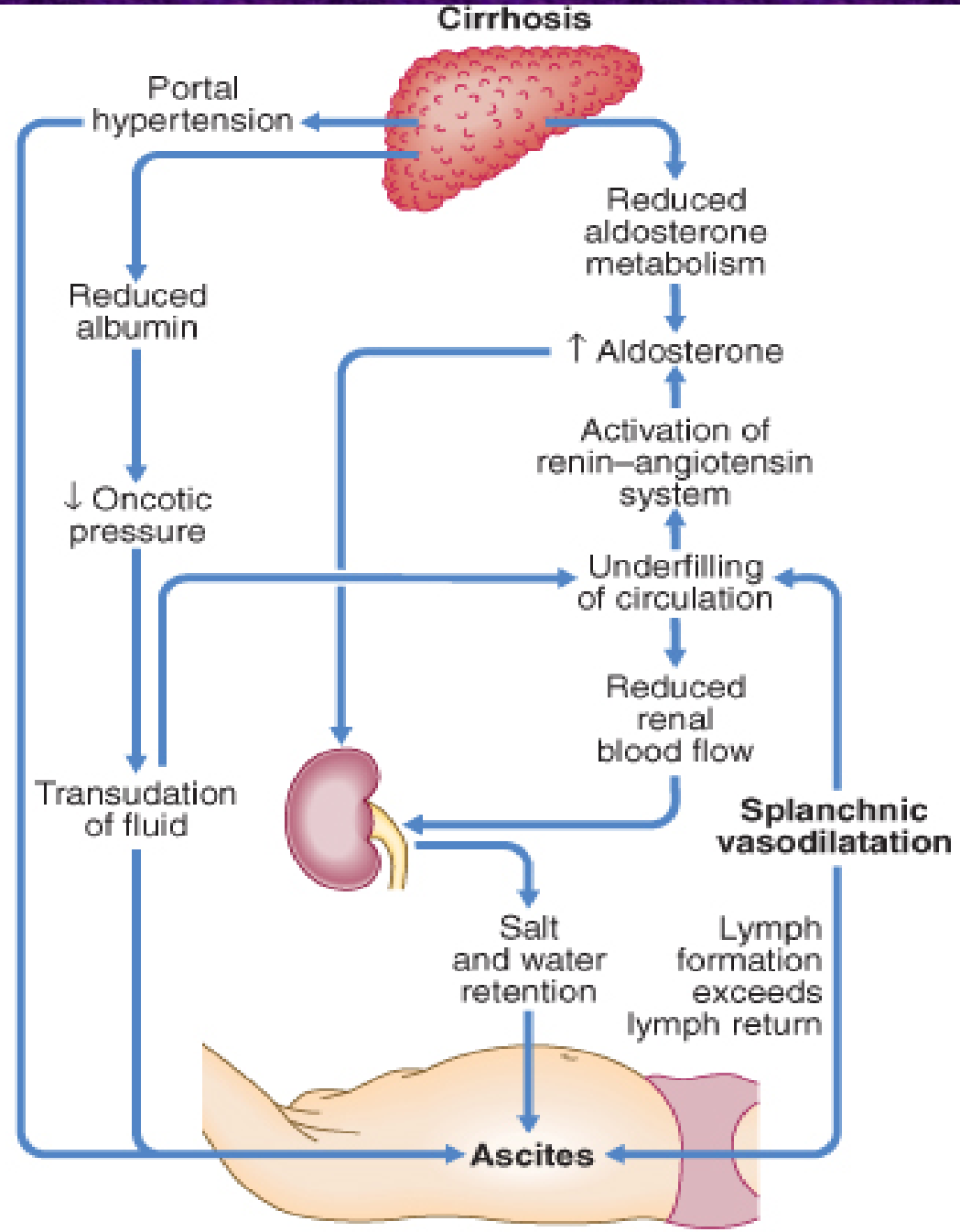
Peritoneal malignancy

Heart failure

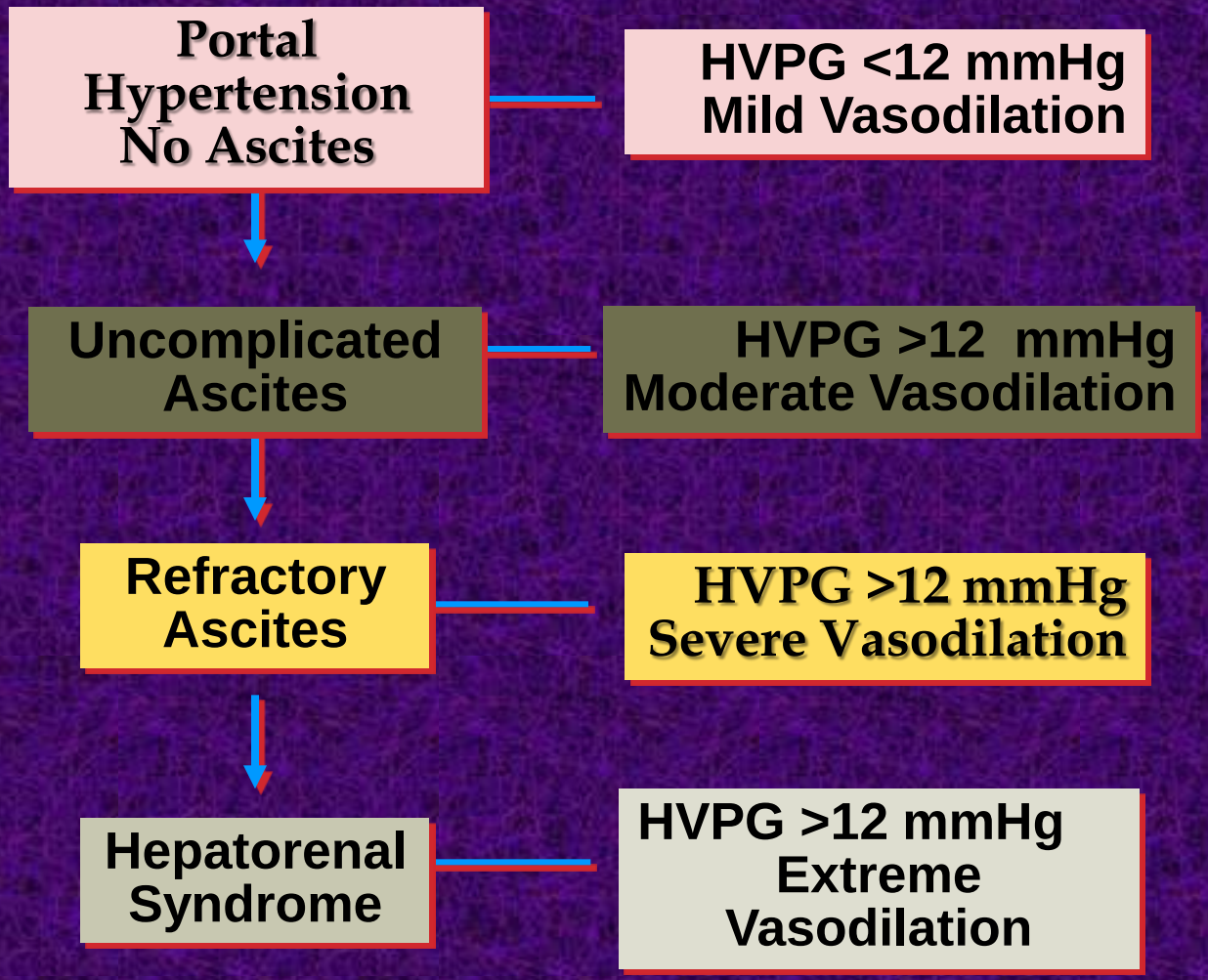
Peritoneal tuberculosis

Others
- Pancreatic
- Budd-Chiari syndrome
- Nephrogenic ascites

Portal Hypertensive SAAG≥1.1	Nonportal Hypertensive SAAG<1.1
Cirrhosis	Peritoneal Carcinomatosis
Heart Failure	Heart Failure
Cancer (nonperitoneal)	Pancreatic Ascites
Budd-Chiari,	Nephrotic Syndrome
Portal Vein thrombosis	Chylous
Alcoholic hepatitis	Serositis (CVD)
Acute liver failure	TB
Sinusoidal obstructive syndrome (VOD)	Myxedema
	Biliary ascites



Natural History of Ascites



hepatic venous pressure gradient (HVPG)

Complications of ascites Cirrhosis

- SBP
- Abd. Hernia
- Impairment of respiratory function
- HRS **rare without ascites**

IN LIVER CIRRHOSIS

- Presence of ascites needs 2 things

  1-portal hypertention

  2- retention of salt and water due to liver failure

Tests of Ascites/Typical
initial tests

- Cell count
- Total protein
- Albumin

Diagnosis of Ascites/optional

- Culture Glucose
- LDH
- Amylase
  Gram stain

SAAG	Testing	Diagnosis	Confirm
≥1.1	TP<2.5	cirrhosis	Imaging, liver biopsy
≥1.1	Glucose<50, LDH<225 +culture,GS	SBP	
≥1.1	+cytology	Carcinomatosis+ portal hin	Cancer workup
<1.1	TP≥2.5	Cardiac ascites	ECHO
<1.1	TP>1.1, glucose<60 Polymicrobial LDH<225	2ndary peritonitis	Abd imaging/CT abd
<1.1	Amylase>100	Pancreatic ascites	Imaging, ERCP

Is it common?

- 1/3 of admitted cirrhotic pts will have ascites
- either
- On admission
- Developed during Hospital stay

Treatment of Ascities

Patience is the key to ascities therapy

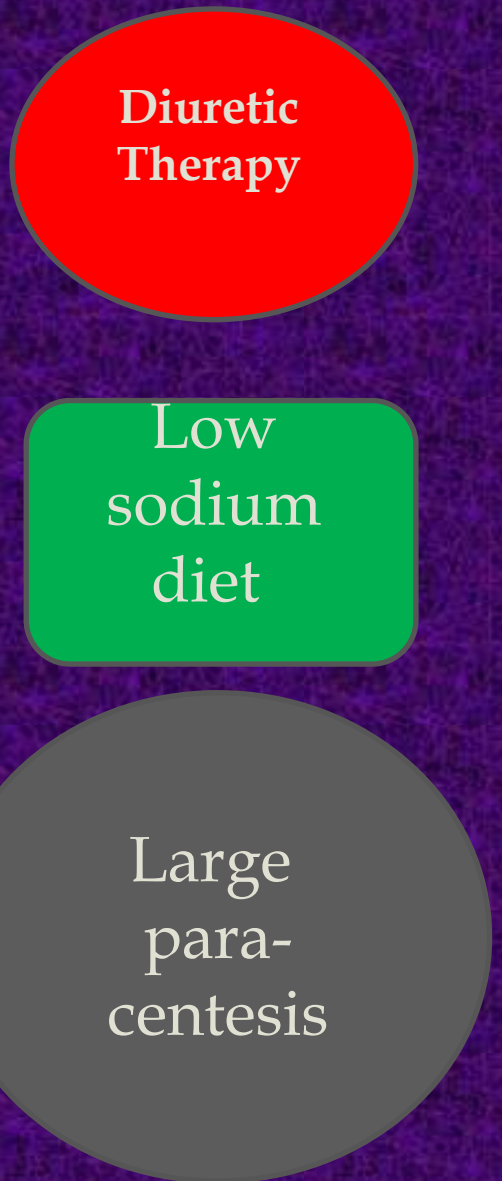
Management of Ascites

Diuretic Therapy

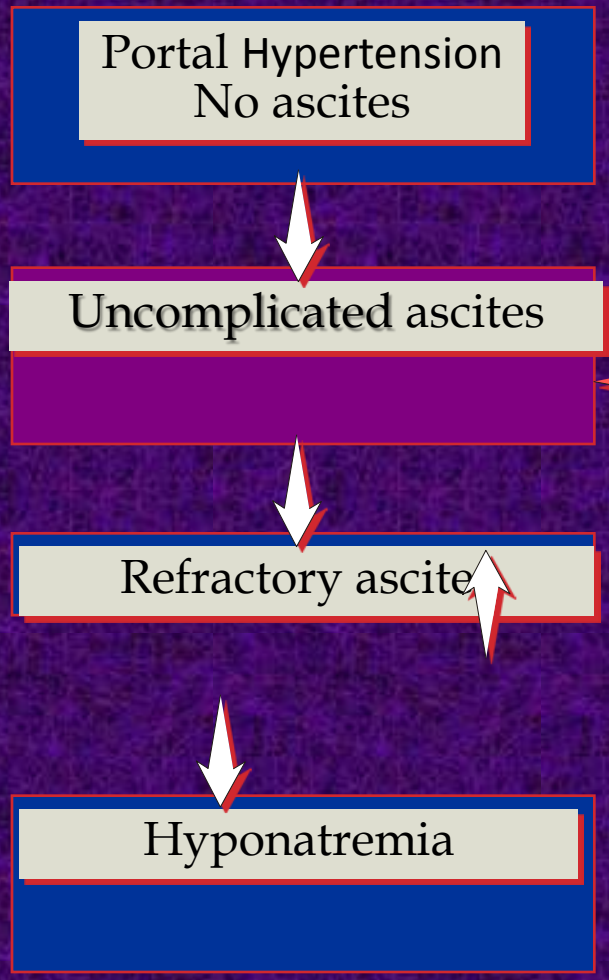
Dosage

- Spironolactone or /with
- Furosemide

- Low sodium diet
- TIPPS
- Large paracentesis
- Fluid restriction if Na is low



Treatment of Ascites



1) Salt restriction + diuretics

2) - Large volume paracentesis (LVP) plus iv. Albumin → diuretics

Key concepts

Spironolactone is the mainstay of ascites therapy.

Loop and Thiazide diuretics should not be used as either primary or monotherapy, they are adjuncts to sodium restriction and Spironolactone.

Bacterial infection in cirrhotic

- **20% SBP**
- 20% UTI
- 15% pneumonia
- 12% Bacteremia

Fernandez et al Hepatology 2002

?factors
Developing Bacterial infection

2 main factors :

1-Severity of liver disease

2-GI bleed: 50% cirrhotic admitted with bleed will develop SBP

SBP

- 20% of cirrhotic with ascites
- 15% mortality
- 50% are bacteremic

BP Spontaneous?

Absence of:

- Intra abdominal inflammatory Focus

  pancreatitis ,cholicystitis, abscess

- Hollow viscus perforation

SBP

- Diagnosis >250 PMN
- >500 WBC or gram stain

peritonitis

- Infected ascites

>250 PMN

Early recognition of SBP

- Fever
- Abdominal Pain
- **Worsening of renal failure**

SBP

- 70% of patients present with fever
-60% with abdominal pain,
- 50% of with changes in mental status
- 40% of with abdominal tenderness

Spontaneous Bacterial Peritonitis (SBP)

- **Initial Assessment and Diagnosis**
- History
- Physical examination
- Abdominal ultrasound
- Abdominal paracentesis
- Ascetic fluid analysis including a wcc and differential, ascetic fluid total protein,
- serum-ascites albumin gradient
- Culture of ascetic fluid at bedside in blood culture bottles

Deterioration of cirrhotic patient

- A frequent cause of sudden deterioration is the development of (SBP).

Bacterial

- E coli is main bacteria in SBP

  Not the only one

-

Treatment SBP

- **Antibiotics : cefotaxime , amoxicillin/clavulanic acid**
- **IV albumin**

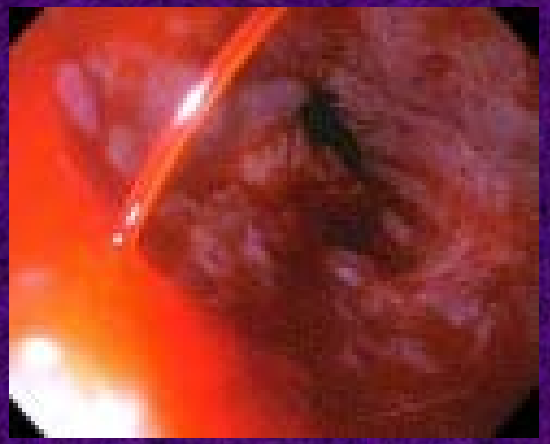
- **Avoid** therapeutic paracentesis

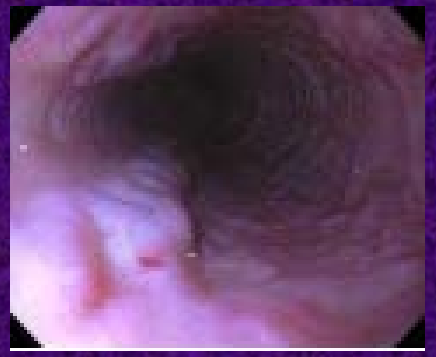
THE KIDNEY IN CIRRHOSIS

Abnormality	**Clinical consequence**
Sodium retention	Ascites and edema
Water retention	Dilutional hyponatremia
Renal vasoconstriction	Hepatorenal syndrome

Esophageal varices

- While most patients with cirrhosis form varices
- only 1/3 will bleed from them

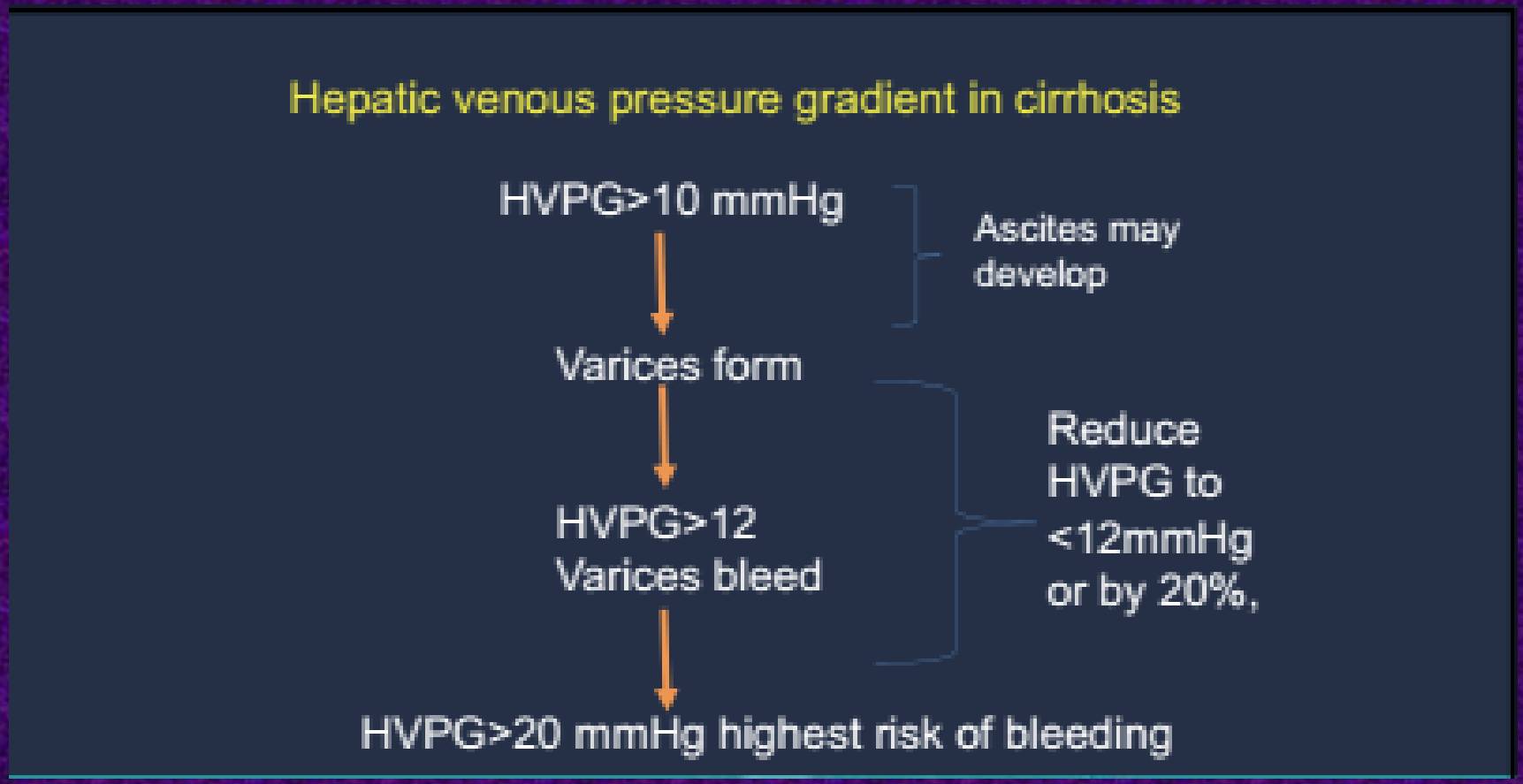




Esophageal varices
predicted first bleed

- 1-Child-Pugh class
  2-the size of
- -presence of red color signs on, EV at endoscopy

Hepatic venous pressure gradient in cirrhosis



Management of acute variceal bleeding

- General supportive measures
- -RBC transfusions if HG <8
  -IV **octreotide** infusion is recommended on first suspicion of variceal bleeding
- -Prophylactic antibiotic
- -endotracheal intubation
- Ligation or Sclerotherapy
- Management after the acute bleed from EV.

(EVL) or (EVS)

- Both are equally effective in stopping bleeding
- complications after EVL (eg., bleeding from esophageal ulcers) are fewer than after EVS

Endoscopic Variceal Band Ligation

Technique and follow-up

Technique

Start at GEJ

↓

Apply suction
1-2 seconds

↓

Deploy band

↓

Continue suction
1-2 seconds

↓

Progress upward:
-helically
-4-6 bands
-lower 5 cm

Follow-up

Re-band
Q 2 weeks

↓

Obliteration

↓

Re-screen
Q 3 months

TIPS for variceal bleeding

- (1)failure of medical and endoscopic management in an acute bleed setting

- (2) recurrent EV bleeding despite NSBB and EVL with intent to obliterate.

General Management of Acute Variceal Bleeding

Recommended	Consider
Early endoscopy (within 12 hours)	Platelets
Transfuse to Hb 7-8 gm/dL	Early TIPS
Prophylactic antibiotics (ceftriaxone)	FFP; *NOT* rFVIIa
Octreotide IV 72 hours	Lactulose
	Endotracheal intubation

Adapted from: Bosch, et al. Semin Liver Dis. 2008; 28: 3.

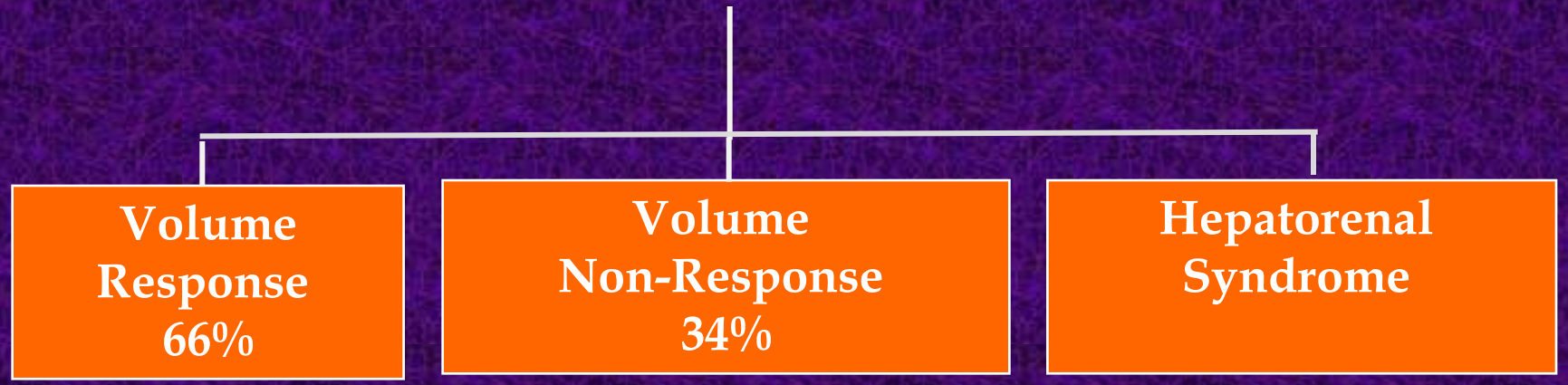
Garcia-Tsao, et al. AASLD Practice Guidelines. Hepatology. 2007; 46: 922.

Gastric varices.

- GV are found less commonly than EV in patients with cirrhosis (20%).

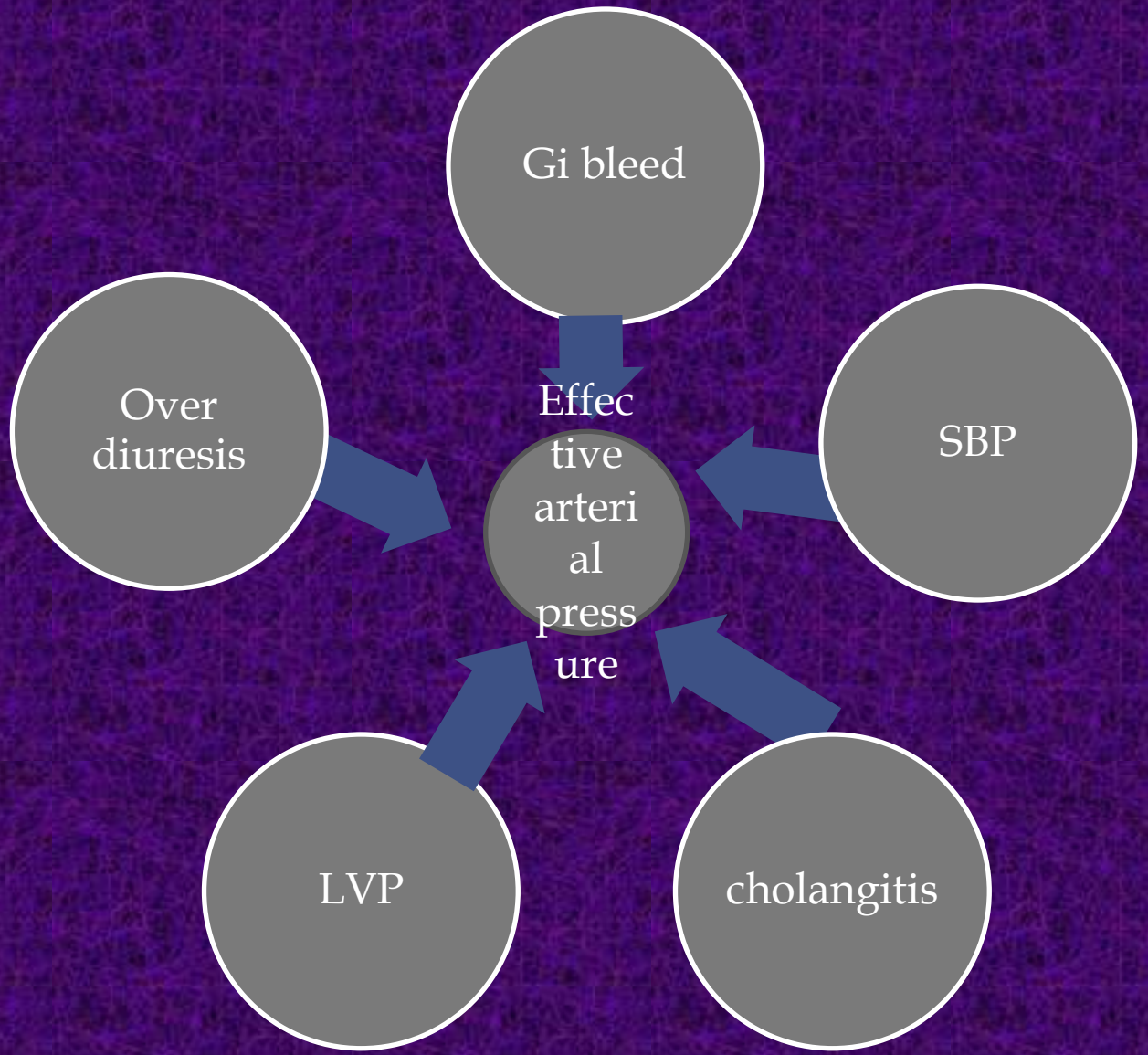
  Such bleeds are clinically more dramatic than from EV (higher transfusion requirements, higher mortality).

RENAL INJURY IN CIRRHOSIS



Most common causes

1) Sepsis	1) NSAIDS
2) GI hemorrhage	2) Contrast dye
3) Diarrhea	3) Intrinsic renal disease
4) Aggressive use of diuretics	

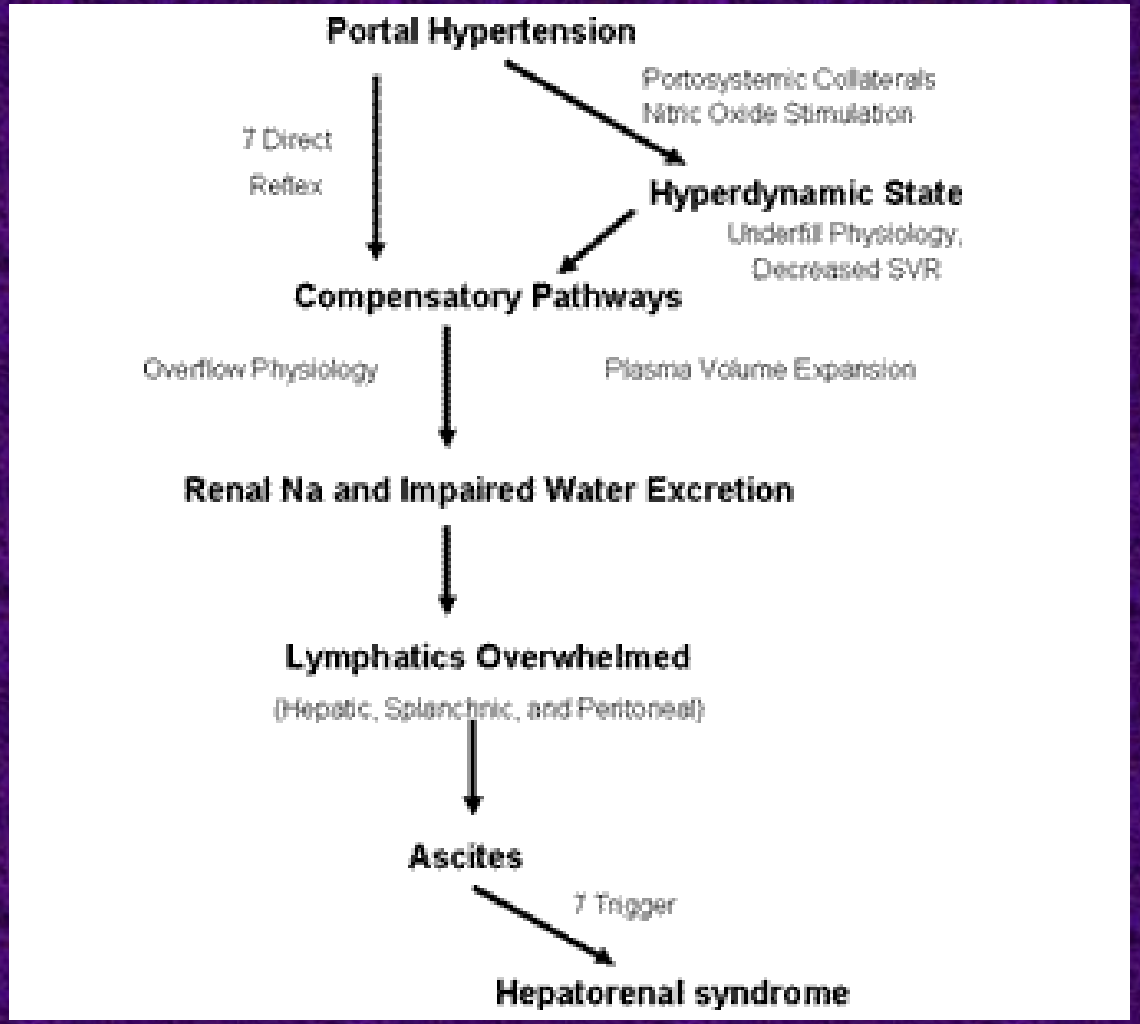

Gi bleed
Over diuresis
Effective arterial pressure
SBP
LVP
cholangitis

Hepatorenal syndrome

- Low urine sodium in the absence of renal pathology
- Type 1 doubling of serum creatinine >2.5 mg/dl <2 weeks
- Median survival 2 weeks

Hepatorenal syndrome

- Low urine sodium in the absence of renal pathology
- Type 2 doubling of serum creatinine >1.5 mg/dl over time
- Median survival 6 months


Portal Hypertension
Portosystemic Collaterals
Nitric Oxide Stimulation
↑ Direct
Reflex
Hyperdynamic State
Underfill Physiology,
Decreased SVR
Compensatory Pathways
Overflow Physiology
Plasma Volume Expansion
Renal Na and Impaired Water Excretion
Lymphatics Overwhelmed
(Hepatic, Splanchnic, and Peritoneal)
Ascites
↑ Trigger
Hepatorenal syndrome

Hepatic Encephalopathy

Precipitating Events

Event	Mechanism
	↑ gut ammonia production
Constipation	-
GI bleed	-
Porto-systemic shunting	↓ neurotoxin clearance
Fever, infection	-
Dehydration, azotemia	↓ renal excretion of ammonium
Hypokalemia	-
Sedatives (BDZ)	↑ inhibitory neurotransmission (GABA)

Management of Hepatic Encephalopathy

1º Therapy	2º Considerations
Remove Precipitant	**Correct hyponatremia**
↓	↓
Lactulose (PO dose to achieve 2-3 soft stools/d; enema in comatose patients)	Dietary Modification -fiber -dairy/vegetable protein *-(protein restrict-no)*
↓	↓
Antibiotics -rifaximin *-(neomycin-no)* *-(metronidazole-no)*	Raise serum K^+ to 5-5.5
↓	↓
Search for spontaneous porto-systemic shunts	Supplement Zinc Probiotics?

THANK YOU FOR YOUR TIME .

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