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What's so bad about ascites?



- Painful
- Anorexia & malnutrition
- Reduced mobility with deconditioning
- Hernias
- Impaired ventilation with atelectasis & pneumonia
- Increased variceal pressure
- May become infected (SBP)

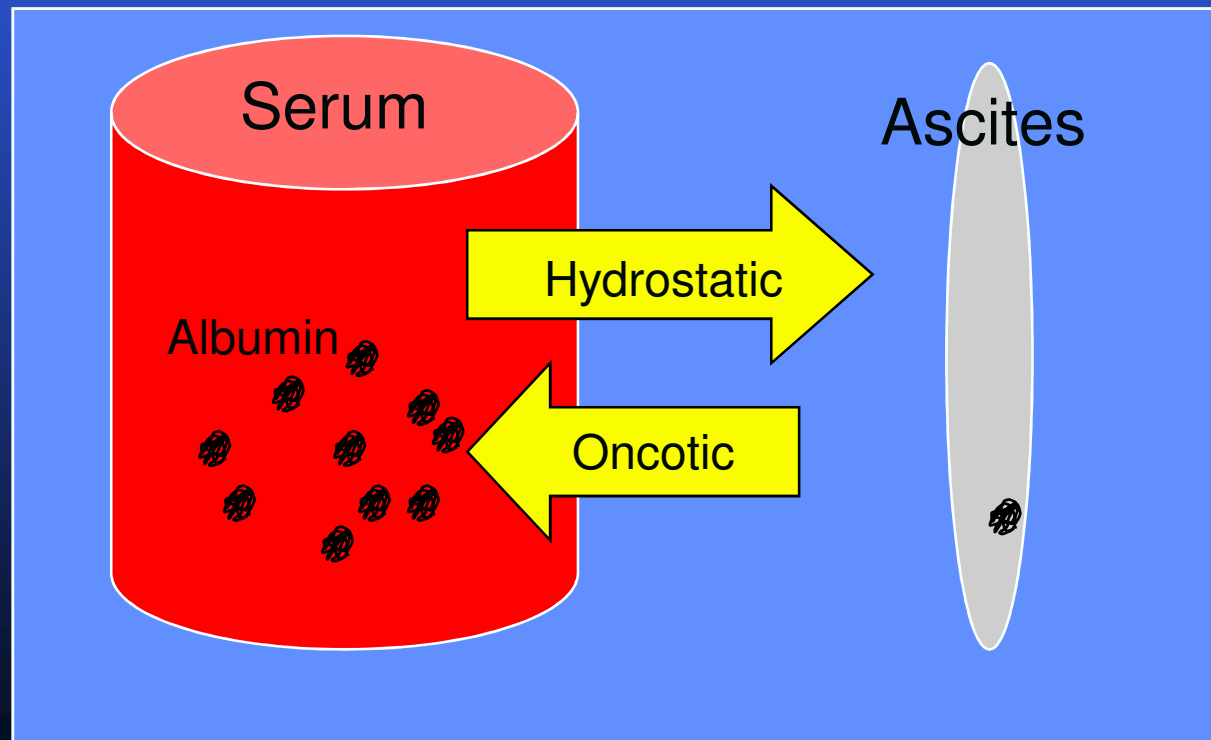
Causes of ascites

- Cirrhosis
- Hepatic congestion (CHF)
- Renal disease
- Pancreatic
- Malignancy
- Infections (TB)
- Inflammatory disease
- Hypothyroidism



Why do cirrhotics retain salt and water?

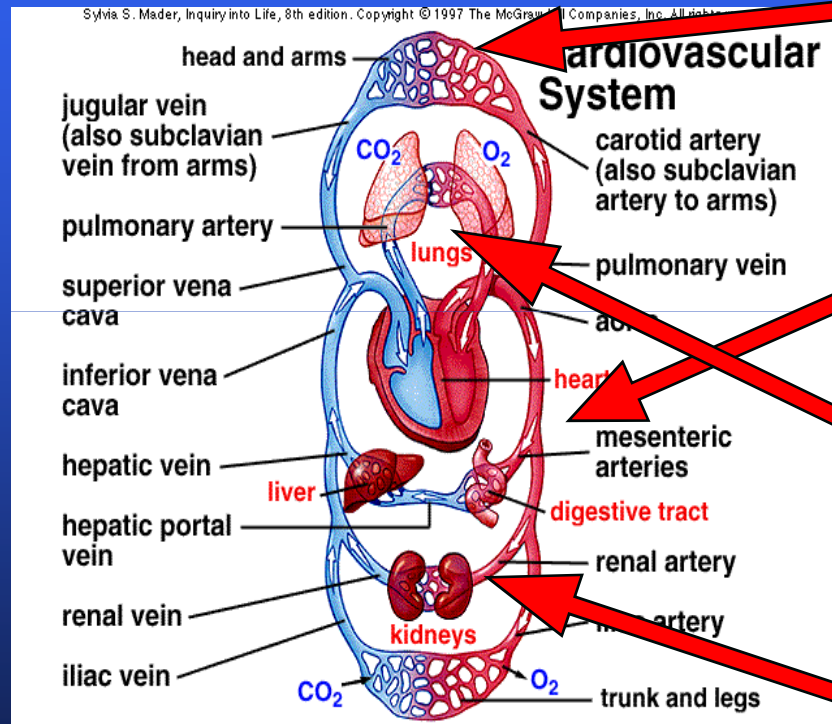
- Underfill
 - ◆ Low albumin & portal HTN
 - ◆ Transudation of fluid
 - ◆ Reduced renal perfusion
 - ◆ Renin release
 - ◆ Salt retention



Why do cirrhotics retain salt and water?

- Overflow
 - ◆ Systemic vasodilatation
 - ◆ Reduced renal perfusion
 - ◆ Renin, angiotensin system activation
 - ◆ Salt retention
 - ◆ Increased venous pressure
 - Portal
 - Systemic
 - ◆ Transudation of fluid

Features of the systemic hemodynamic derangement of cirrhosis



Systemic vasodilatation

- Low blood pressure
- High cardiac output

Mesenteric vasodilatation

- Portal hypertension

Pulmonary vasodilatation

- Hepatopulmonary syndrome

Renal vasodilatation

- Reduced GFR

Stages of ascites

- Salt avidity without ascites
- Overt edema/ascites
 - ◆ Responsive to diuretics/salt restriction
 - ◆ Refractory
- Hepatorenal syndrome
 - ◆ Type II
 - ◆ Type I

Medical Rx

- Salt restriction
- Distal tubular diuretics
 - ◆ Spironolactone
 - ◆ Amiloride
- Loop and proximal diuretics
 - ◆ Furosemide



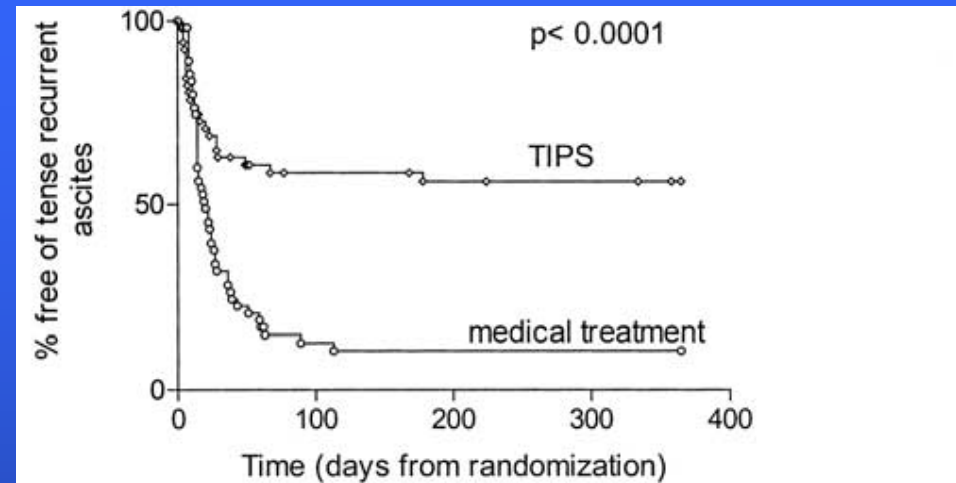
Resistant ascites

- Inadequate treatment
 - ◆ Patient noncompliance
 - ◆ Physician reluctance
- Refractory ascites
 - ◆ Failure to resolve despite maximal diuretics
 - ◆ Intolerance to treatment
 - Diuretic side effects (cramps, etc.)
 - Hyponatremia
 - Prerenal azotemia
- Hepatorenal syndrome, type II
 - ◆ Refractory ascites with persistent $\text{Cr} > 1.5$

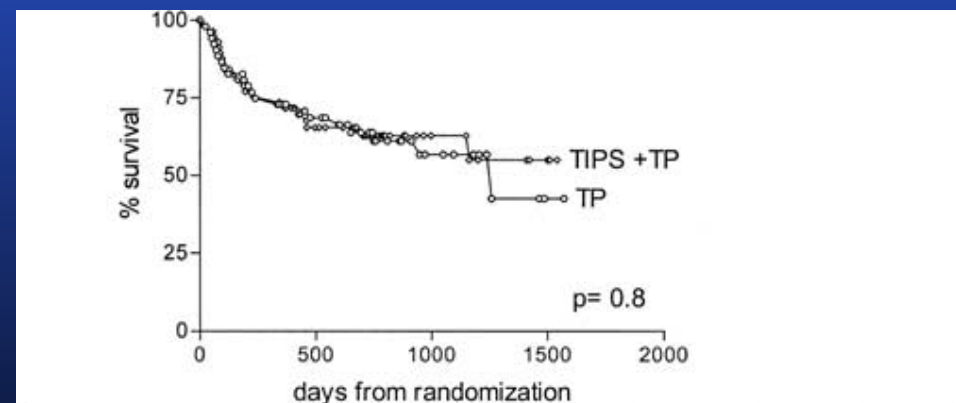


Refractory ascites: the role of TIPS

- TIPS lowers portal pressure and may reduce or eliminate need for therapeutic paracentesis
- However overall TIPS does not improve survival



	time (days)					
	0	30	60	180	360	
TP	57	23	10	6	5	
TP + TIPS	52	36	29	26	20	

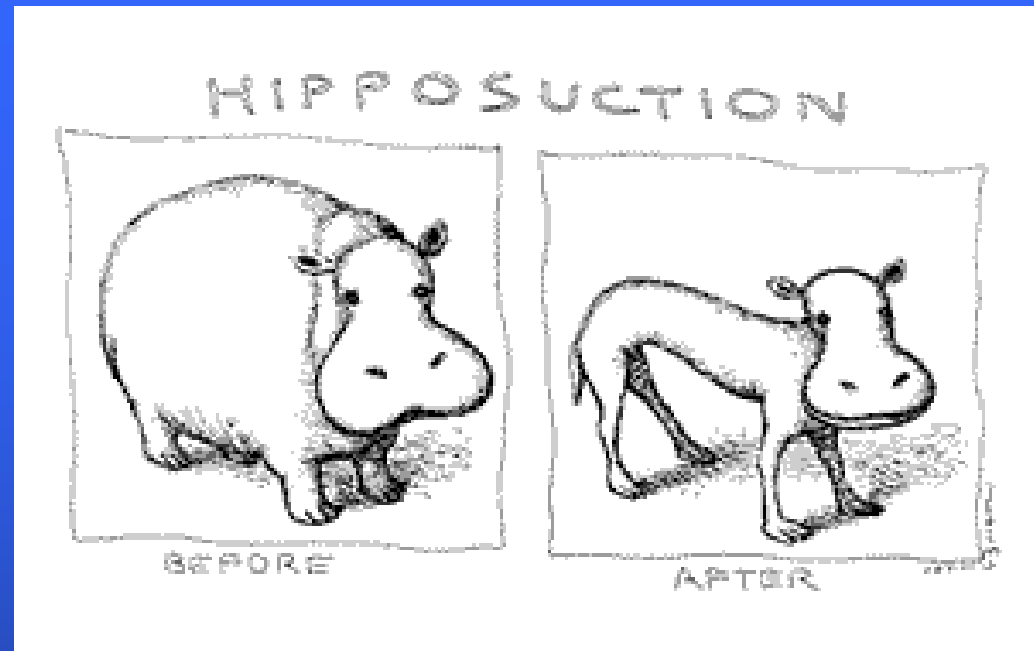


	days from randomization						
	0	50	100	250	500	750	1000
TIPS + TP	52	50	45	39	32	22	12
TP	57	56	42	30	28	20	10

When to consider TIPS for refractory ascites

- Treatment compliant patient
- Low MELD score
- Absence of encephalopathy
- Transplantation not imminent

Large volume (total) paracentesis



- Can be done as needed to relieve symptoms
- Benefits: comfort, nutrition, mobility, respiratory function, ?renal perfusion
- Risks:
 - ◆ Post paracentesis circulatory dysfunction: prevented with 50 g albumin (transudates only)
 - ◆ Hemorrhage, infection, perforation

Ascites



Paracentesis Tray



Ultrasound guidance



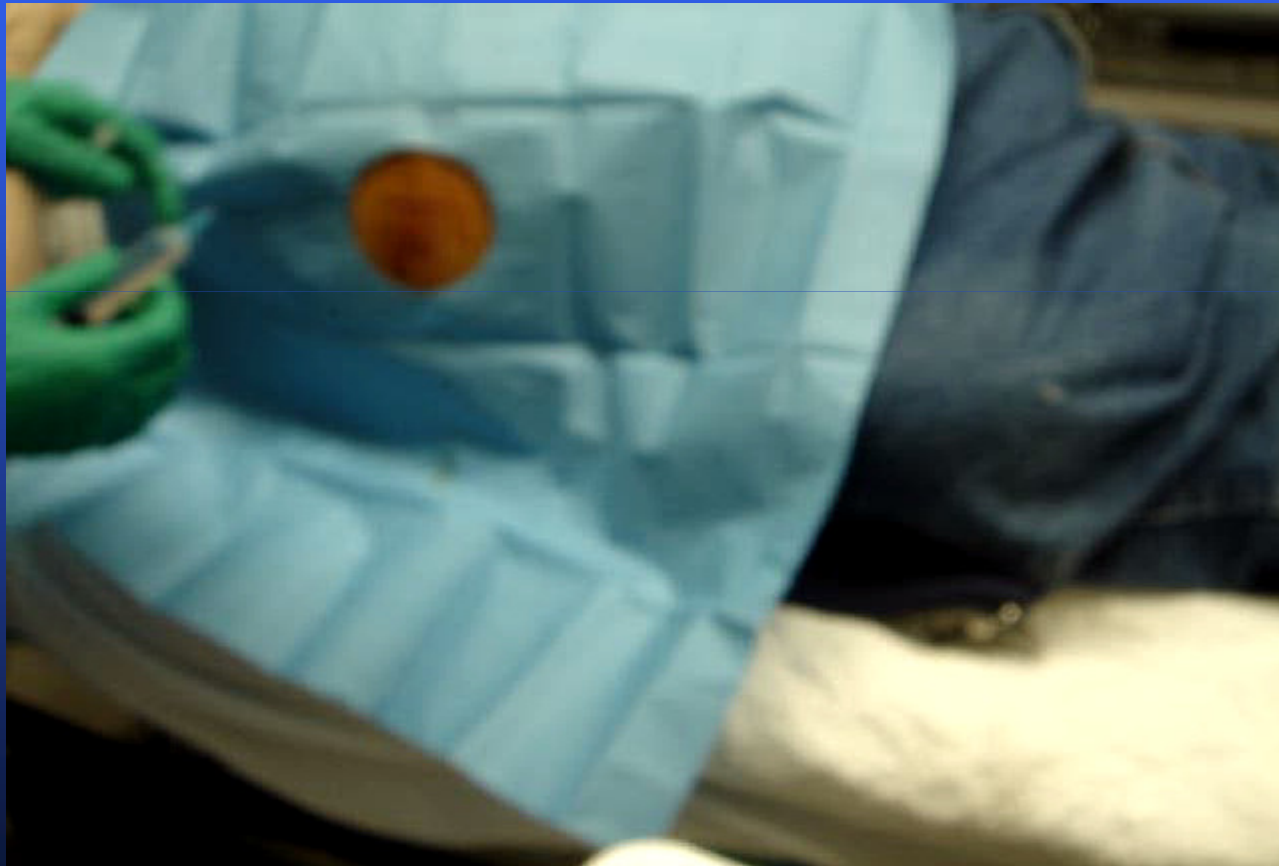
Landmarks



Site prep



Local anesthesia



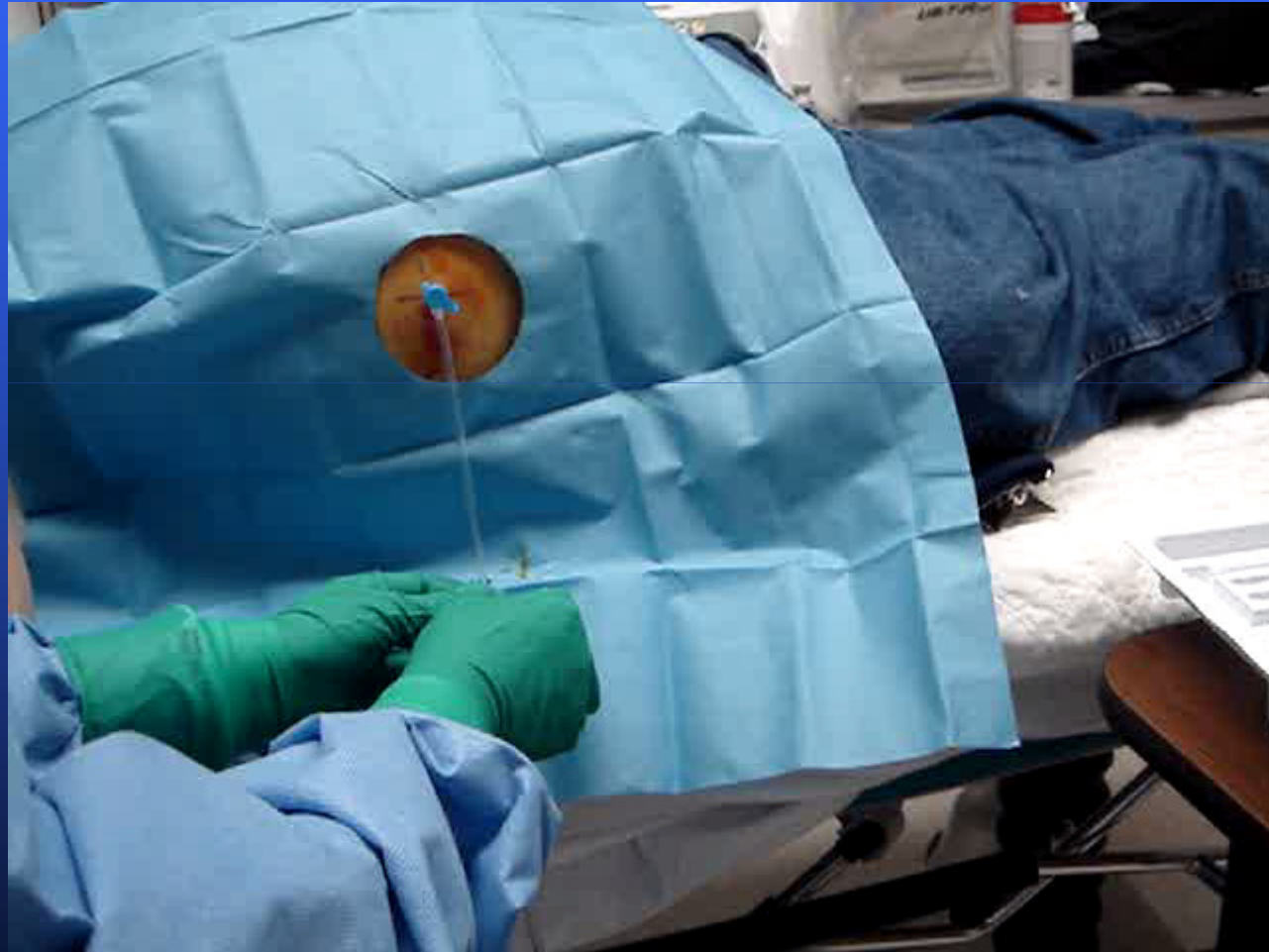
Insertion of Needle



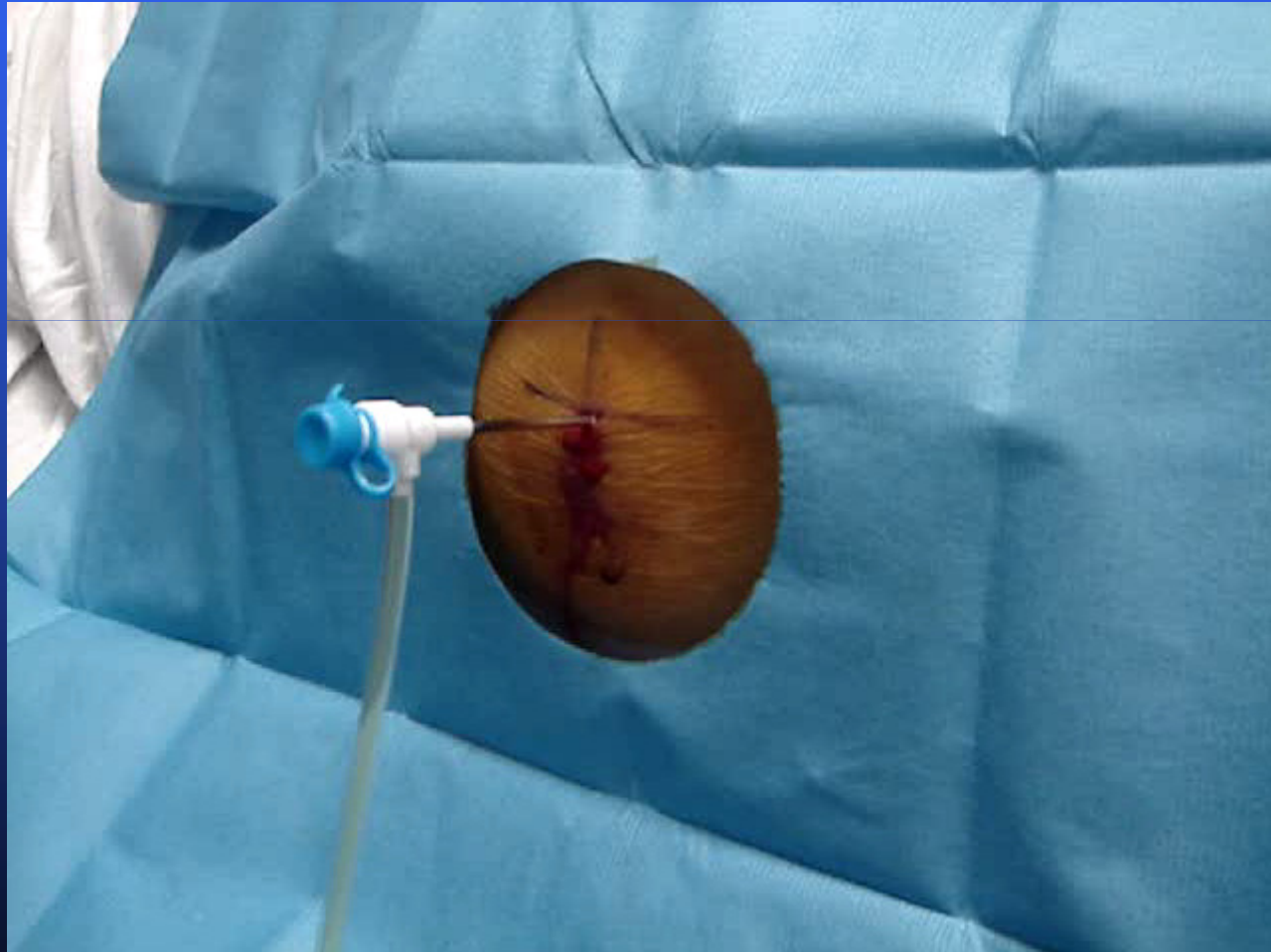
Suction vs gravity



Suction



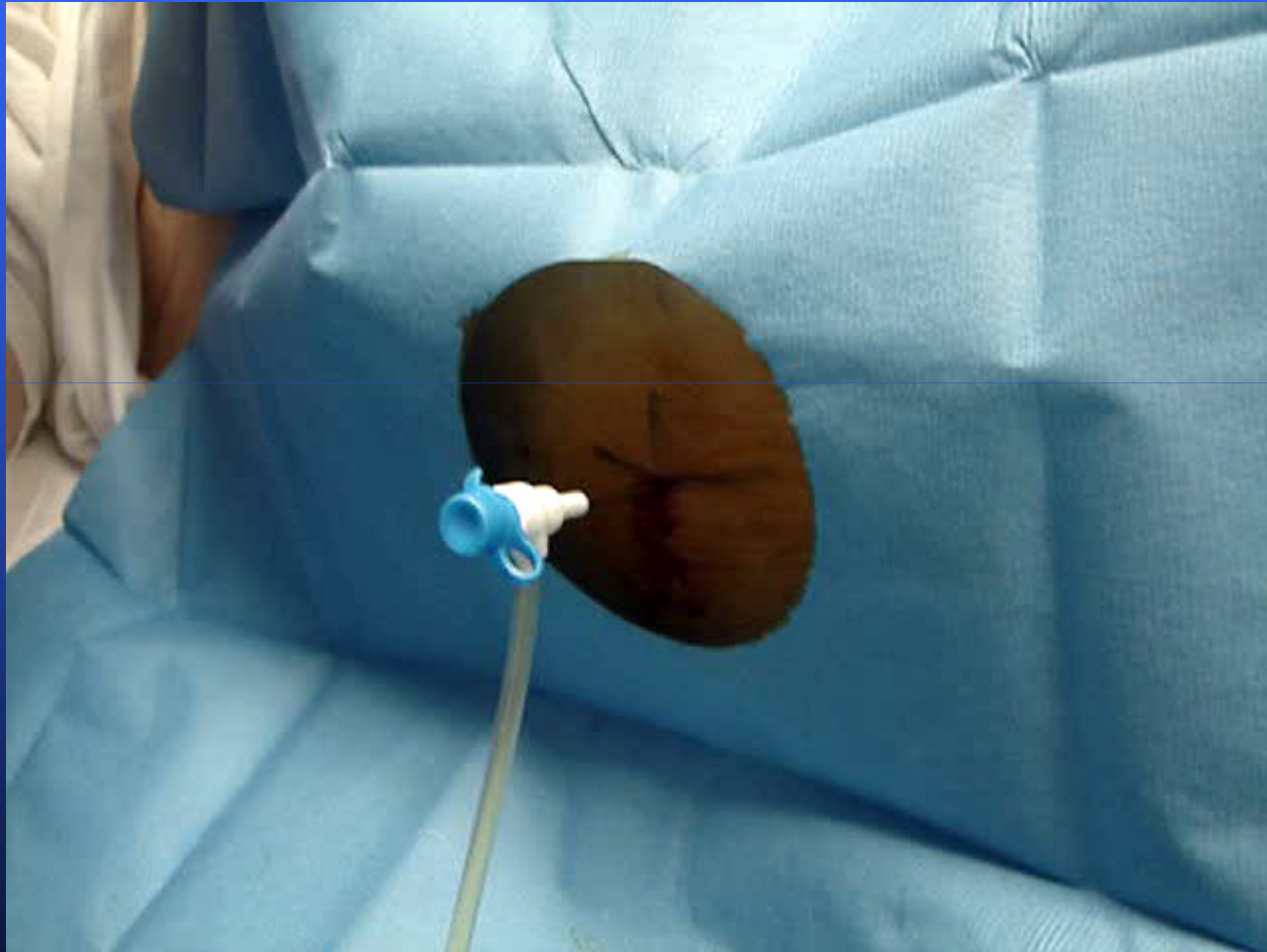
Skin changes



Self-contained system



Slowing down...repositioning



When to stop...

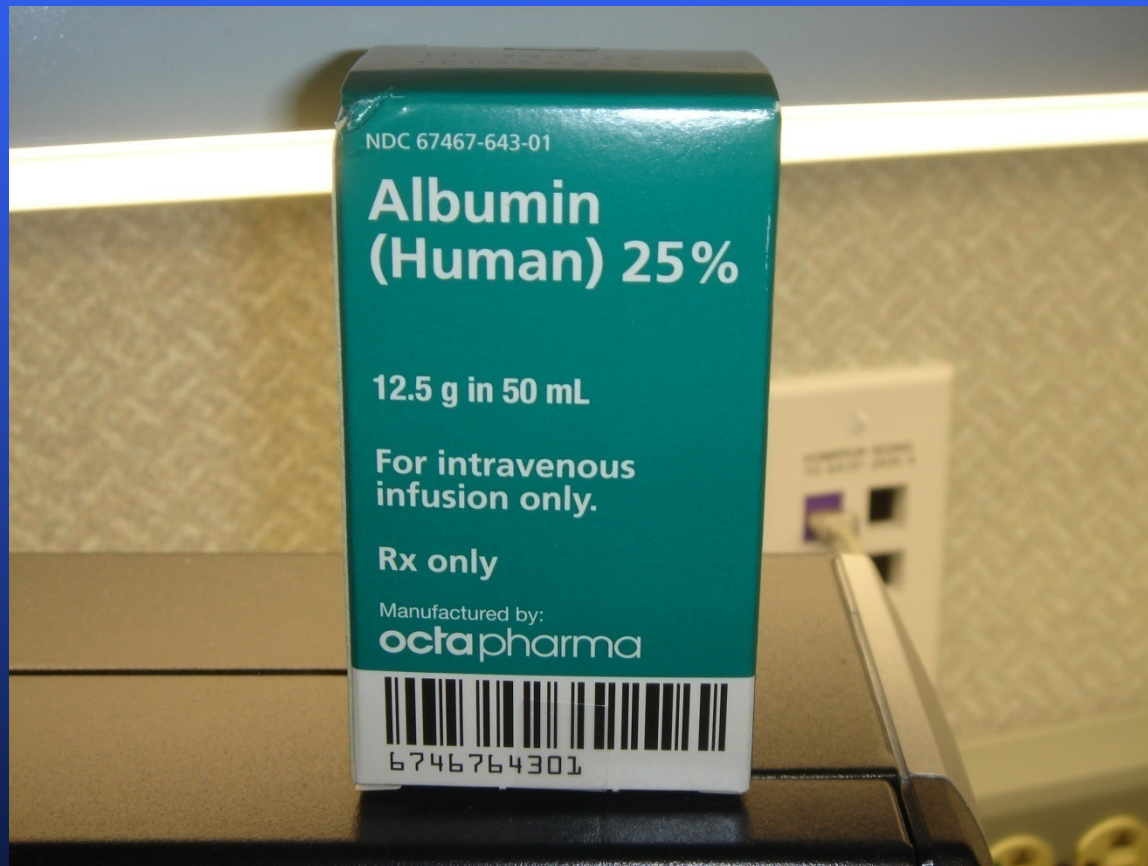
Procedure completion



Sample to Lab



Prevent PCD



Diagnostic paracentesis:

AASLD Practice Guidelines

BA Runyon, 2004. Hepatology 39:841

- Abdominal paracentesis should be performed and ascitic fluid should be obtained from patients with clinically apparent new onset ascites
- Initial lab investigation should include:
 - ◆ Cell count and diff
 - ◆ Total protein, albumin -> calculate SAAG
- Other studies can be ordered based on pretest probability of disease, including:
 - ◆ Culture: routine, AFB, fungal
 - ◆ Chemistry: glucose, LDH, Amylase, TG
 - ◆ Cytology

Paracentesis as a guide to diagnosis

- Low protein, high SAAG
 - **cirrhotic**
- High protein, high SAAG
 - **congestive**
 - R sided CHF
 - Constrictive pericarditis
 - Budd-Chiari
- Low protein, low SAAG
 - **hypoalbuminemic**
 - Nephrotic
 - Enteropathic
- High protein, low SAAG
 - **exudative**
 - Cancer
 - TB
 - Hypothyroid
 - Pancreatic

Low protein: < 2.5 g/dl

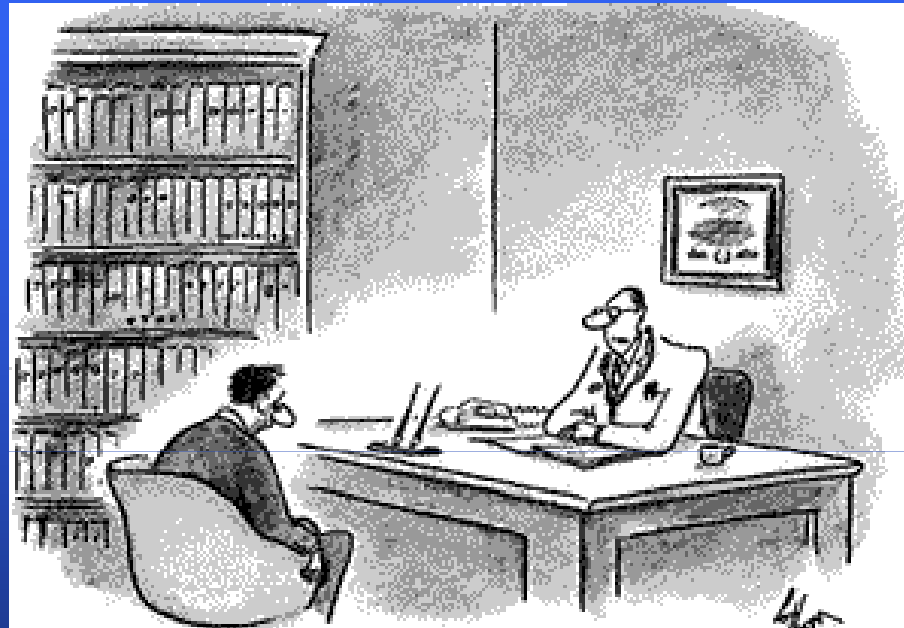
High SAAG: > 1.1 g/dl

Fluid Analysis

- Cell Count:
 - ◆ PMNs
 - ◆ Hemorrhagic ascites- corrected PMN
- Culture
 - ◆ Usually monomicrobial in SBP
- Protein and Albumin

Fluid Analysis

- Glucose
 - ◆ Usually falls below in secondary bacterial peritonitis
- LDH- releases from PMN lysis
 - ◆ Increased in SBP; further elevated in secondary bacterial peritonitis
- Amylase
 - ◆ Increased in pancreatitis and gut perforation



*Unfortunately, there's no cure—
there's not even a race for a cure*