Portal Hypertension

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Definition

- Portal pressure gradient (the difference in pressure between the portal vein and the hepatic veins) of 10 mmHg or greater.
- First step toward fluid retention in the setting of cirrhosis.
- Patients with cirrhosis but without PHT do not develop ascites or edema.
- A portal pressure >12 mmHg appears to be required for fluid retention.

Causes

- Cirrhosis is the most common cause of portal hypertension but it can also be present in the absence of cirrhosis, a condition referred to as "noncirrhotic portal hypertension".
- The causes of noncirrhotic portal hypertension can be divided into prehepatic, intrahepatic (presinusoidal, sinusoidal, and postsinusoidal), and posthepatic causes.

Classification of noncirrhotic portal hypertension

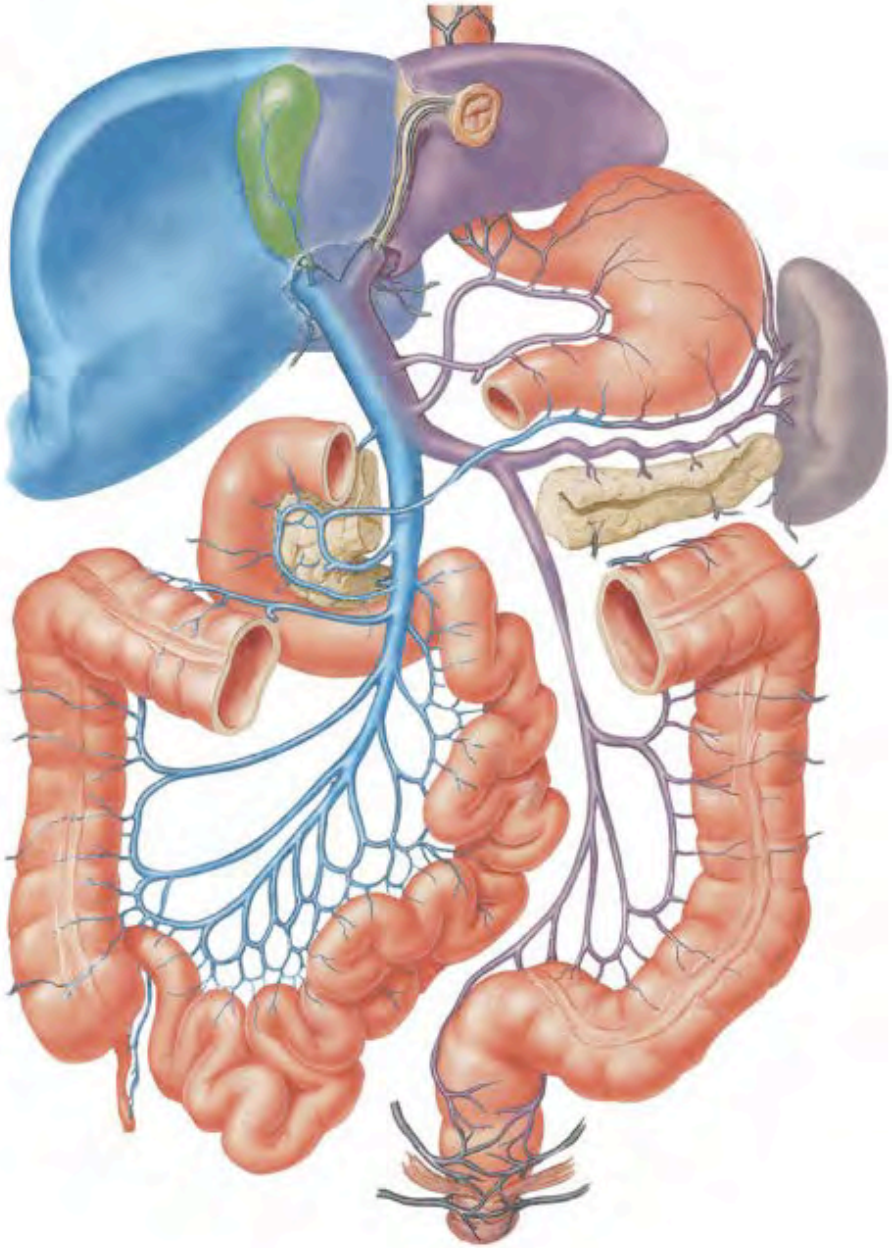
Prehepatic
Portal vein thrombosis
Splenic vein thombosis
Splanchnic arteriovenous fistula
Splenomegaly (lymphoma, Gaucher's disease)
Intrahepatic
Presinusoidal
Schistosomiasis
Idiopathic portal hypertension/Noncirrhotic portal fibrosis/Hepatoportal sclerosis
Primary biliary cirrhosis
Sarcoidosis
Congenital hepatic fibrosis
Sclerosing cholangitis
Hepatic arteriopetal fistula
Sinusoidal
Arsenic poisoning
Vinyl chloride toxicity
Vitamin A toxicity
Nodular regenerative hyperplasia
Postsinusoidal
Sinusoidal obstruction syndrome (Veno-occlusive disease)
Budd-Chiari syndrome
Posthepatic
IVC obstruction
Cardiac disease (constrictive pericarditis, restrictive cardiomyopathy)

Consequences of portal hypertension :

- Ascites (free fluid in the peritoneal cavity).
- Hepatic encephalopathy.
- Increased risk of spontaneous bacterial peritonitis.
- Increased risk of hepatorenal syndrome.
- Splenomegaly (enlargement of the spleen) with risk for pancytopenia.

Consequences of portal hypertension :

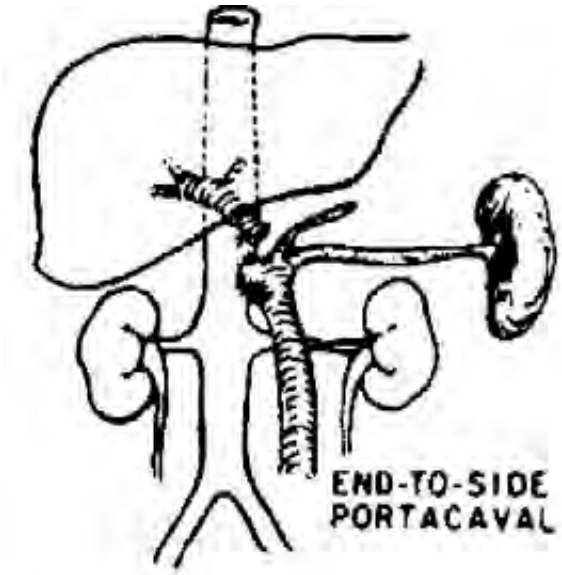
- Portacaval anastomoses:
  - Esophageal varices, gastric varices, anorectal varices (not to be confused with hemorrhoids), and caput medusae.
  - Esophageal and gastric varices pose an ongoing risk of life-threatening hemorrhage, with hematemesis or melena.

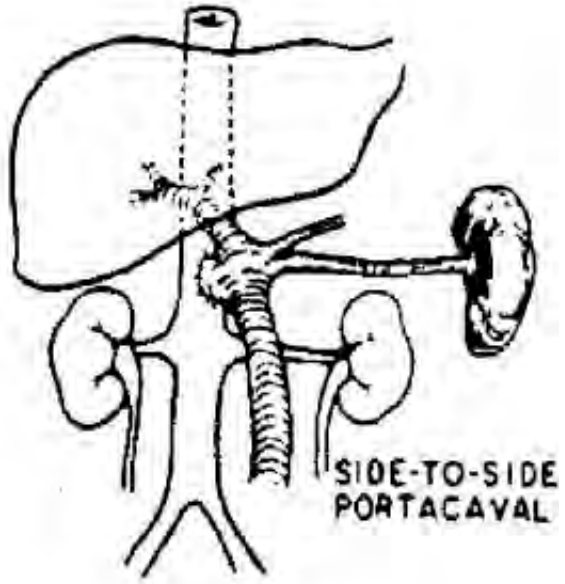


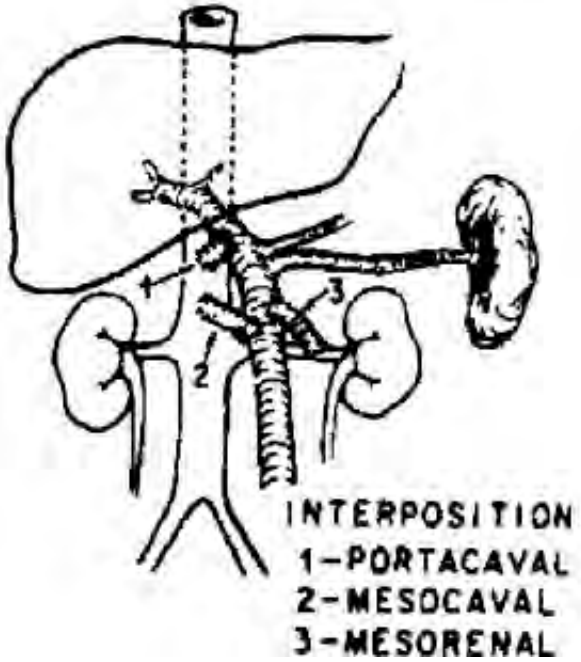
Portal Hypertension: The Role of Shunting Procedures

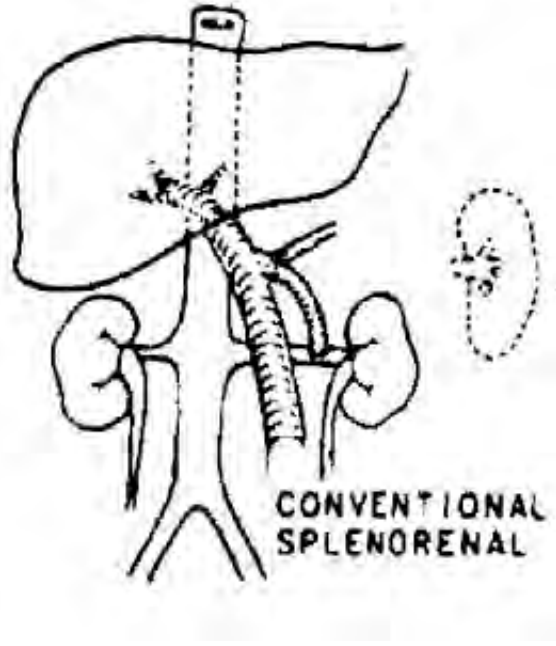
Non-selective:

- Two basic types of nonselective shunts
  - (1) the end-to-side portacaval shunt, which directly diverts all portal flow to the inferior vena cava.
  - (2) side-to-side portosystemic shunts, which leave the portal vein intact and decompress the liver, as well as the entire portal venous system.



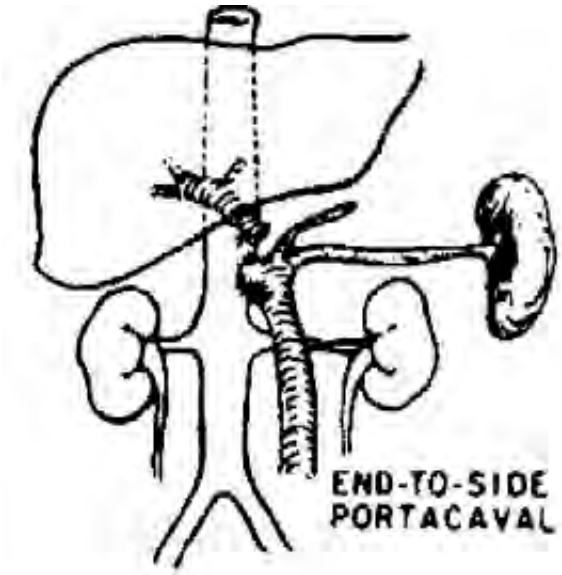


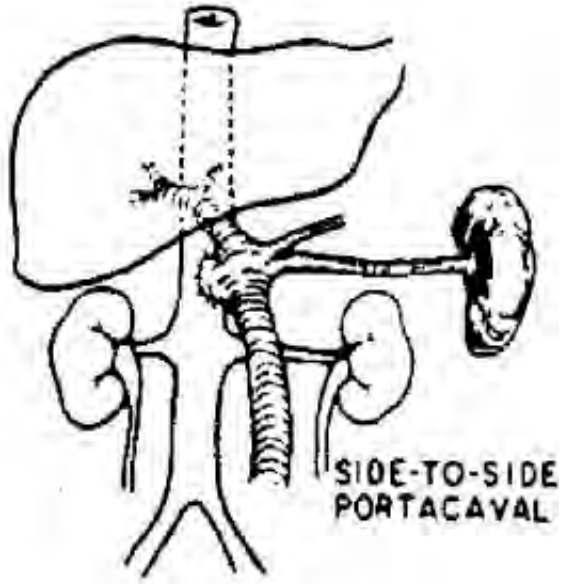


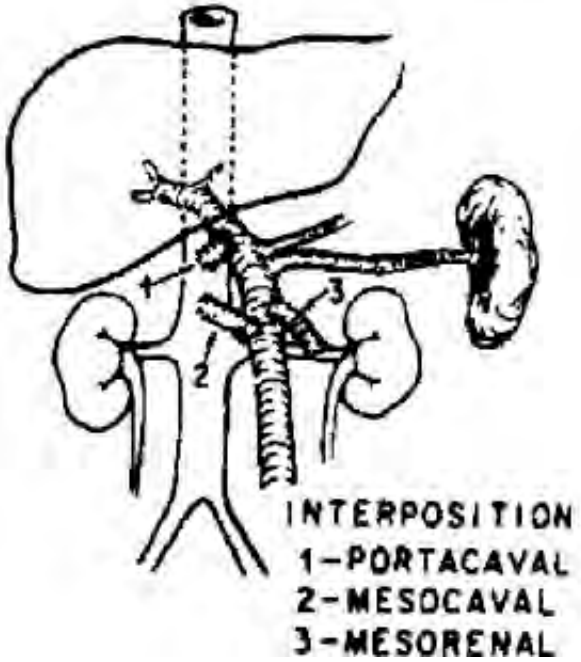


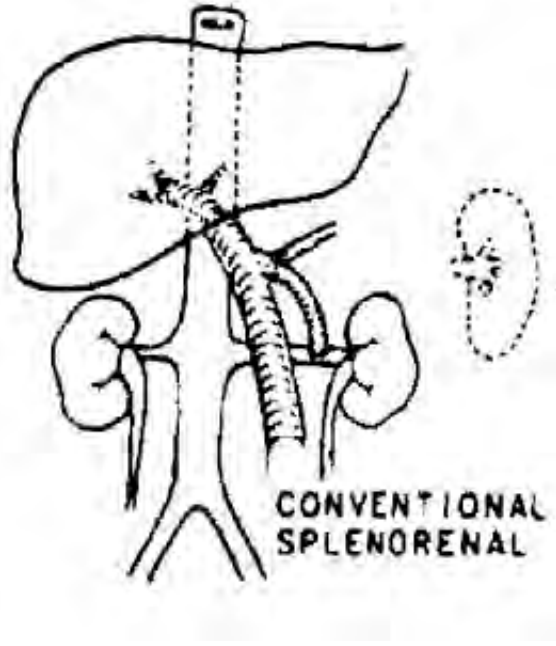
End-to-side portacaval shunt

- The end-to-side portacaval shunt (Eck fistula) was the first shunt introduced into clinical practice
- Because portal blood contains toxins, complete diversion of portal flow into the systemic circulation can result in accelerated hepatic failure and the development of portosystemic encephalopathy (PSE) in many patients.
- Little survival advantage over medical management because of an accelerated onset of hepatic failure.



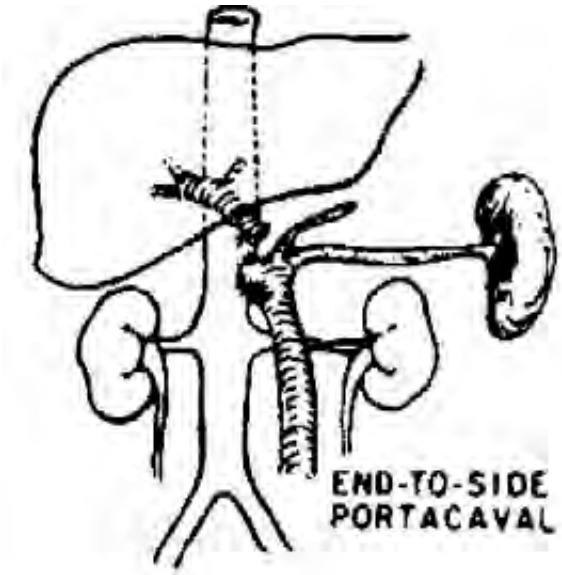


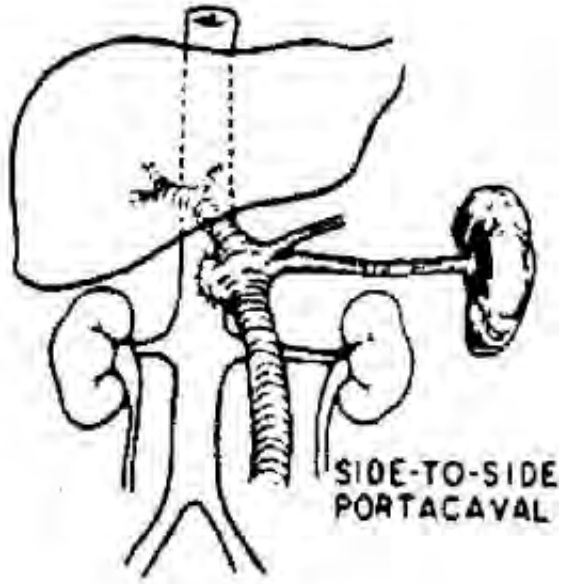


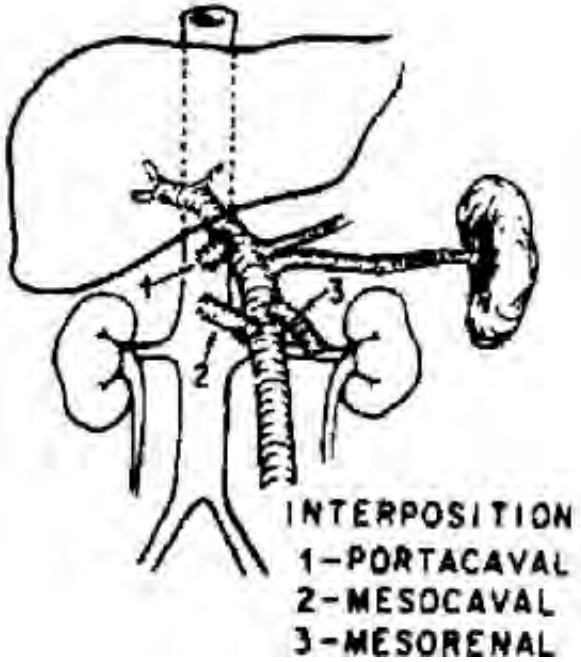


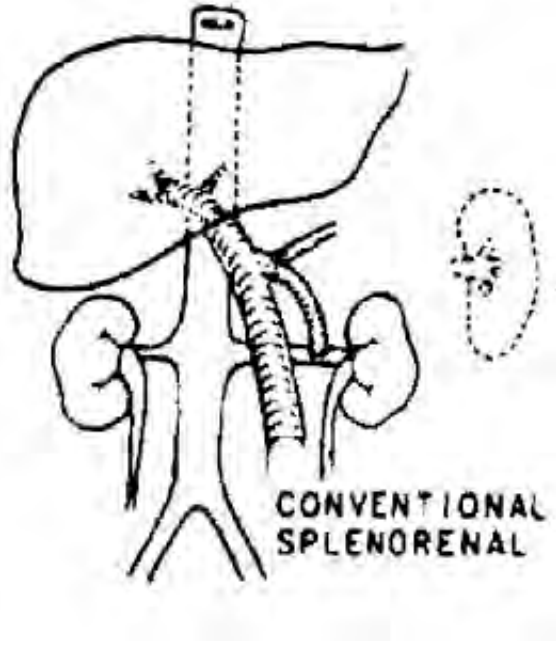
Side-to-side portosystemic shunts

- Were developed with the objective of preserving hepatic portal perfusion in addition to decompressing varices.
- They also completely divert portal flow away from the liver.
- These effectively decompress the liver, as well as the splanchnic venous circulation, making them effective in resolving medically intractable ascites and in preventing recurrent variceal bleeding.
- The resultant complete diversion of portal flow again results in a high frequency of postshunt encephalopathy and an acceleration of hepatic failure similar to that observed following the end-to-side portacaval shunt.



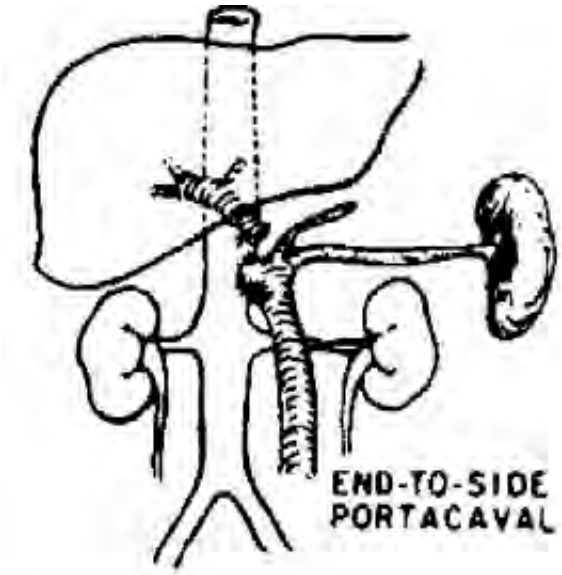


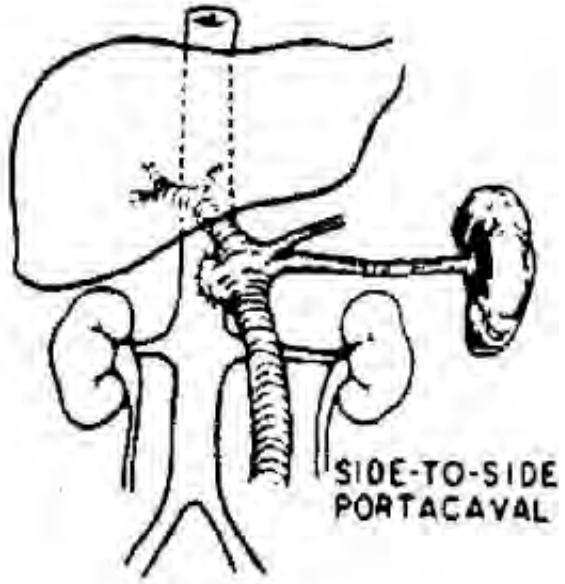


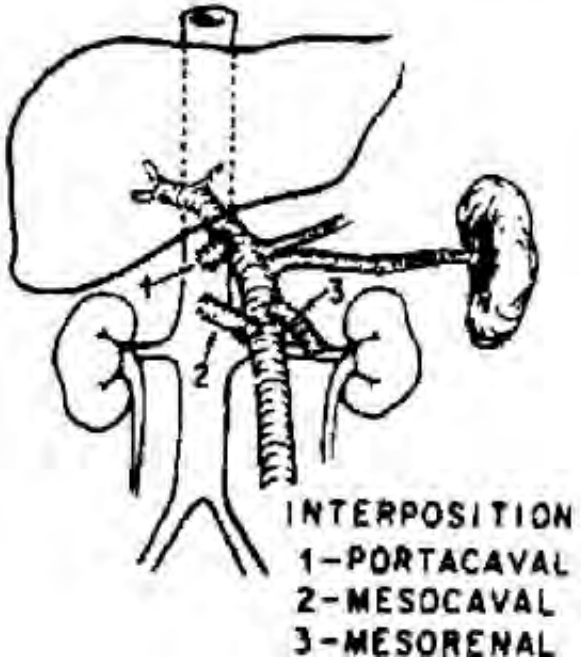


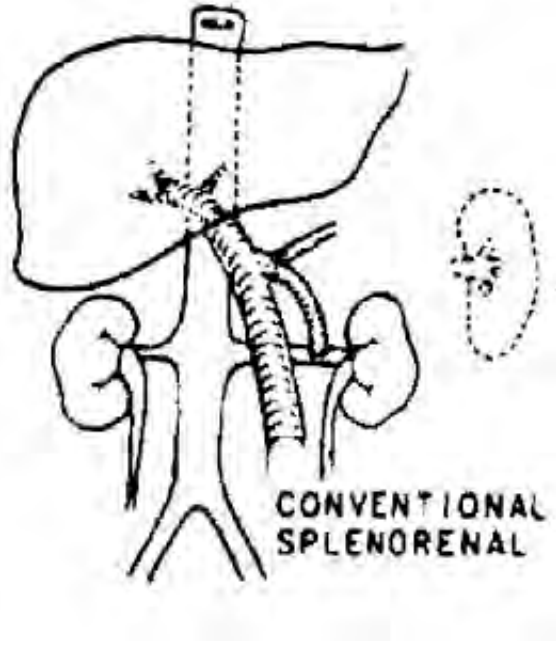
Other side-to-side portosystemic shunt

- Interposition graft placed in the portacaval, mesocaval, mesorenal, and splenorenal positions, all with the same effect of decompressing both the liver and the portal venous system.
- High late occlusion rate secondary to thrombus forming in the synthetic graft.



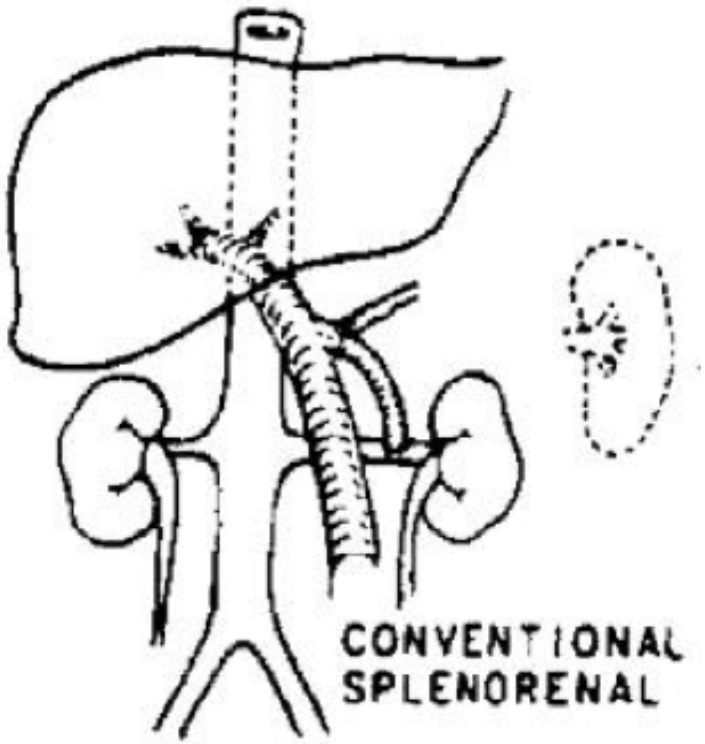






Conventional Splenorenal Shunt

- Consists of splenectomy and anastomosis of the proximal end of the splenic vein to the left renal vein.
- Risk of thrombosis
- Useful for severe hypersplenism

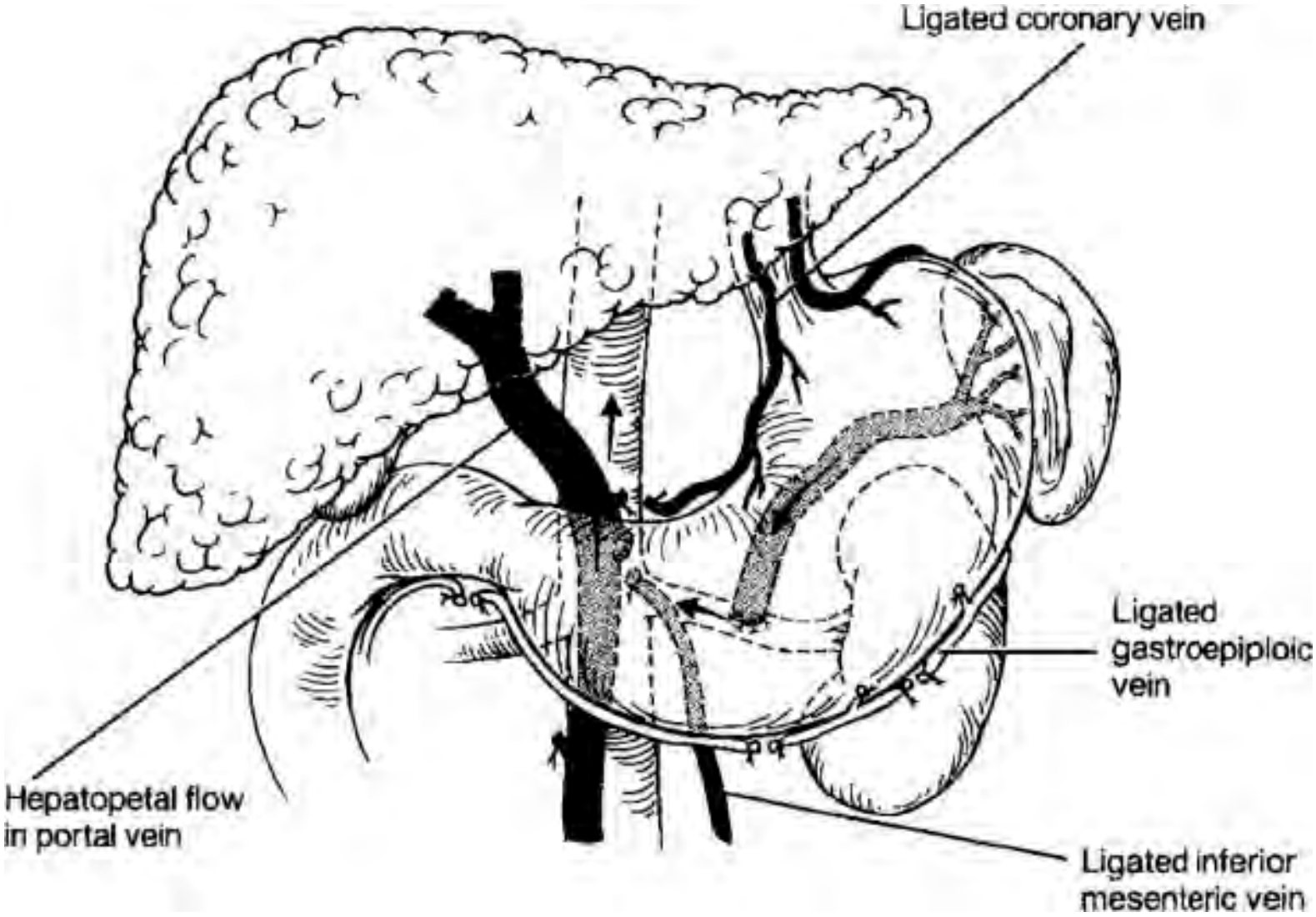


Indications

- Because the TIPS procedure also serves as a side-to-side nonselective shunt but can be achieved without an open operation, it has generally replaced the various types of nonselective shunts.
- These operations may rarely be indicated in patients with both medically intractable ascites and recurrent variceal bleeding after TIPS failure.
- Occasionally used in the desperate emergency setting when TIPS technology is not available.

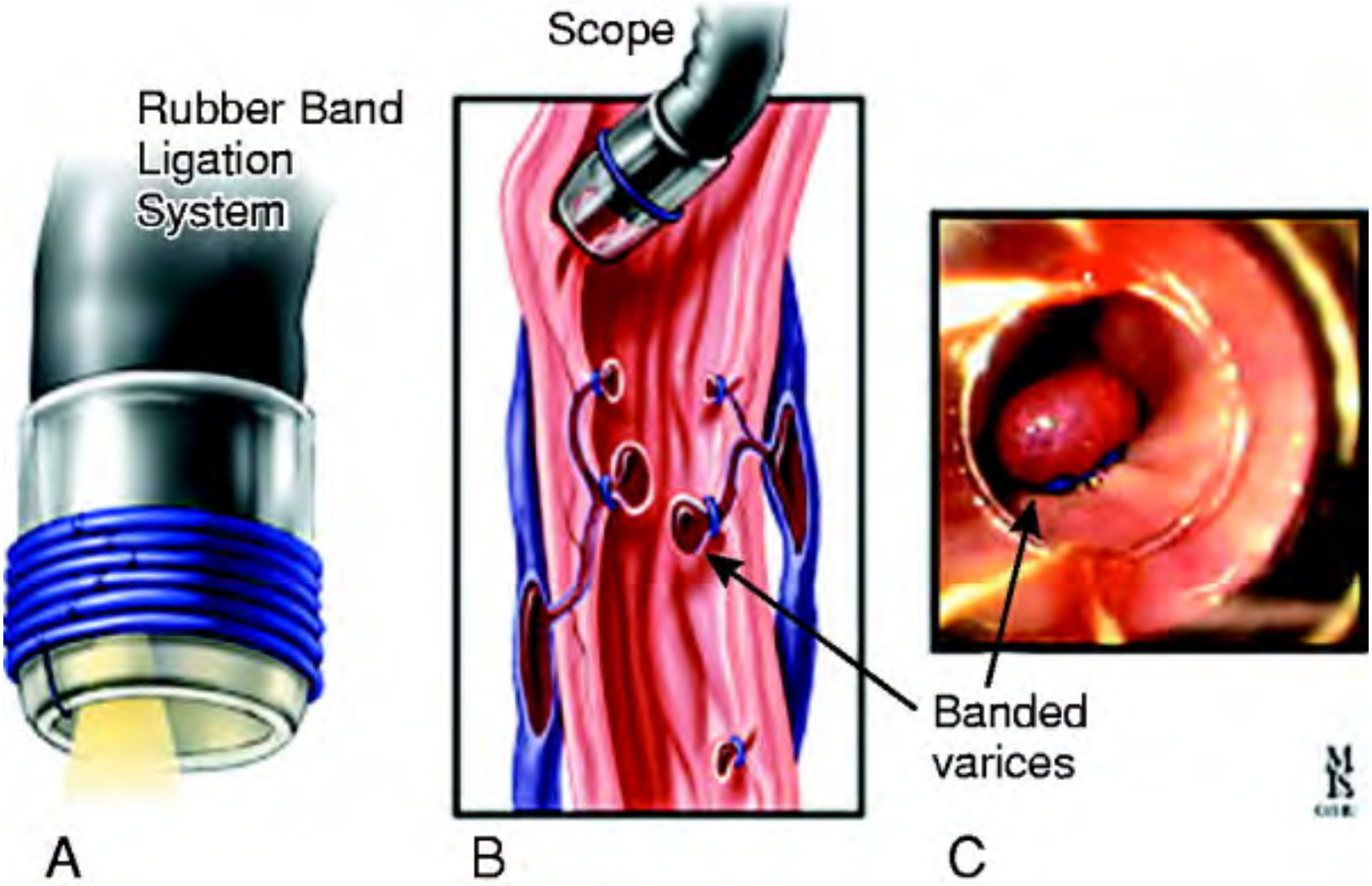
Selective Shunts

- The only selective shunt presently in use is the distal splenorenal shunt, conceived by Dr. W. Dean Warren in the late 1960s
- Because of its lower frequency of postshunt encephalopathy, the distal splenorenal shunt is the most commonly used portosystemic shunting procedure in most areas of the world.
- Confined to patients with preserved hepatic functional reserve (Child-Pugh classes A and B) and to the elective setting.



Treatment of Acute Variceal Bleeding

- Emergency treatment should be nonoperative whenever feasible.
- Endoscopic treatment (preferably variceal ligation) and pharmacotherapy (intravenous octreotide infusion) effectively control bleeding in more than 85% of patients.
- When these treatment modalities fail in the acute setting, TIPS rather than an operative shunt is the means by which portal decompression is achieved in most institutions.
- When TIPS is not indicated or the expertise is not available, an emergency portosystemic shunt operation, can be life saving.

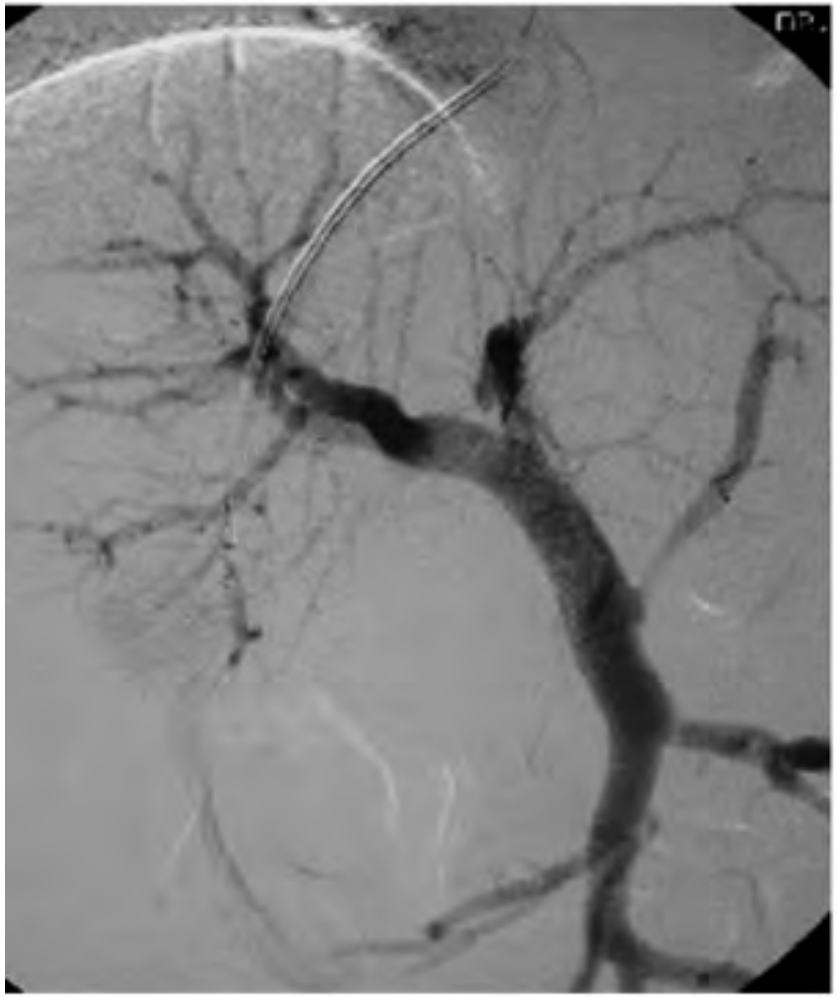


Transjugular intrahepatic portosystemic shunts (TIPS)

- Offers the only minimally invasive alternative to provide relief for the symptoms.
- With a primary variceal bleed, the risk of rebleeding is 50%.
- In patients with rebleeding that cannot be controlled with endoscopic management (refractory variceal bleeding) or who continue to rebleed (recurrent variceal bleeding), TIPS is an excellent option for portal decompression.
- Useful for short-term management of patients before liver transplantation because it does not interrupt the normal anatomic structure of the liver and portal system and make it an easier technically due to lower portal pressure.

Technique for TIPS

1. Accessing the Hepatic Veins
2. Carbon Dioxide Portography
3. Portal Vein Cannulation
4. Stent Placement



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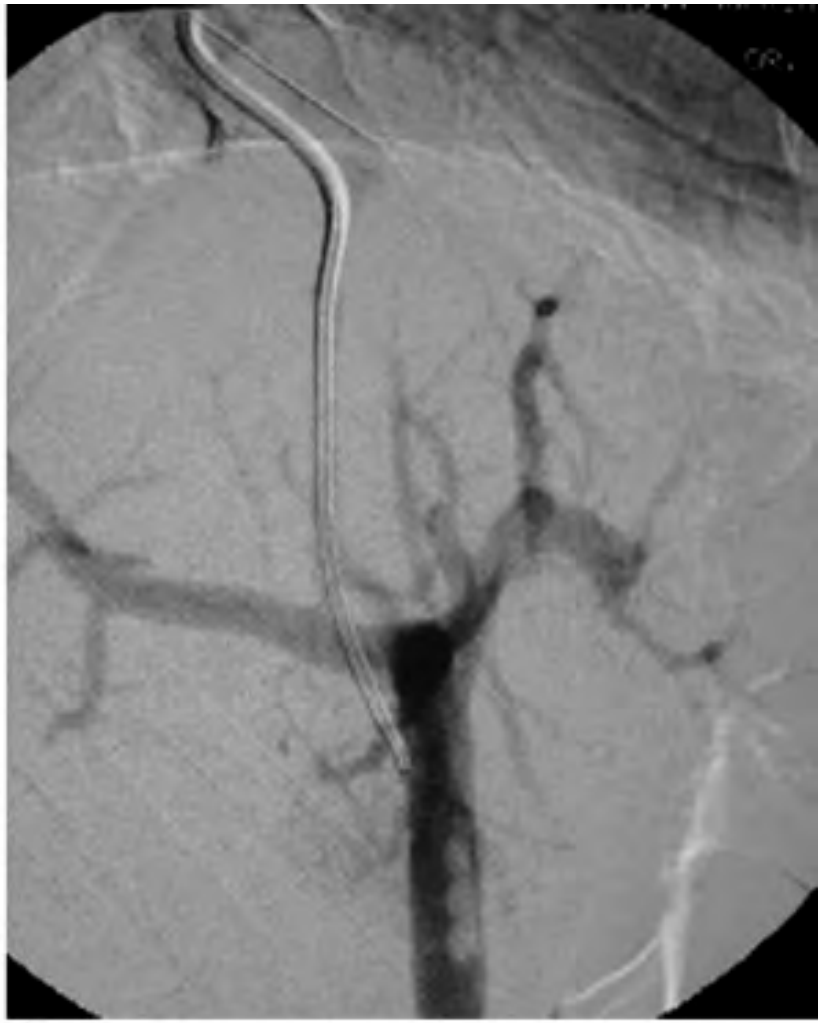

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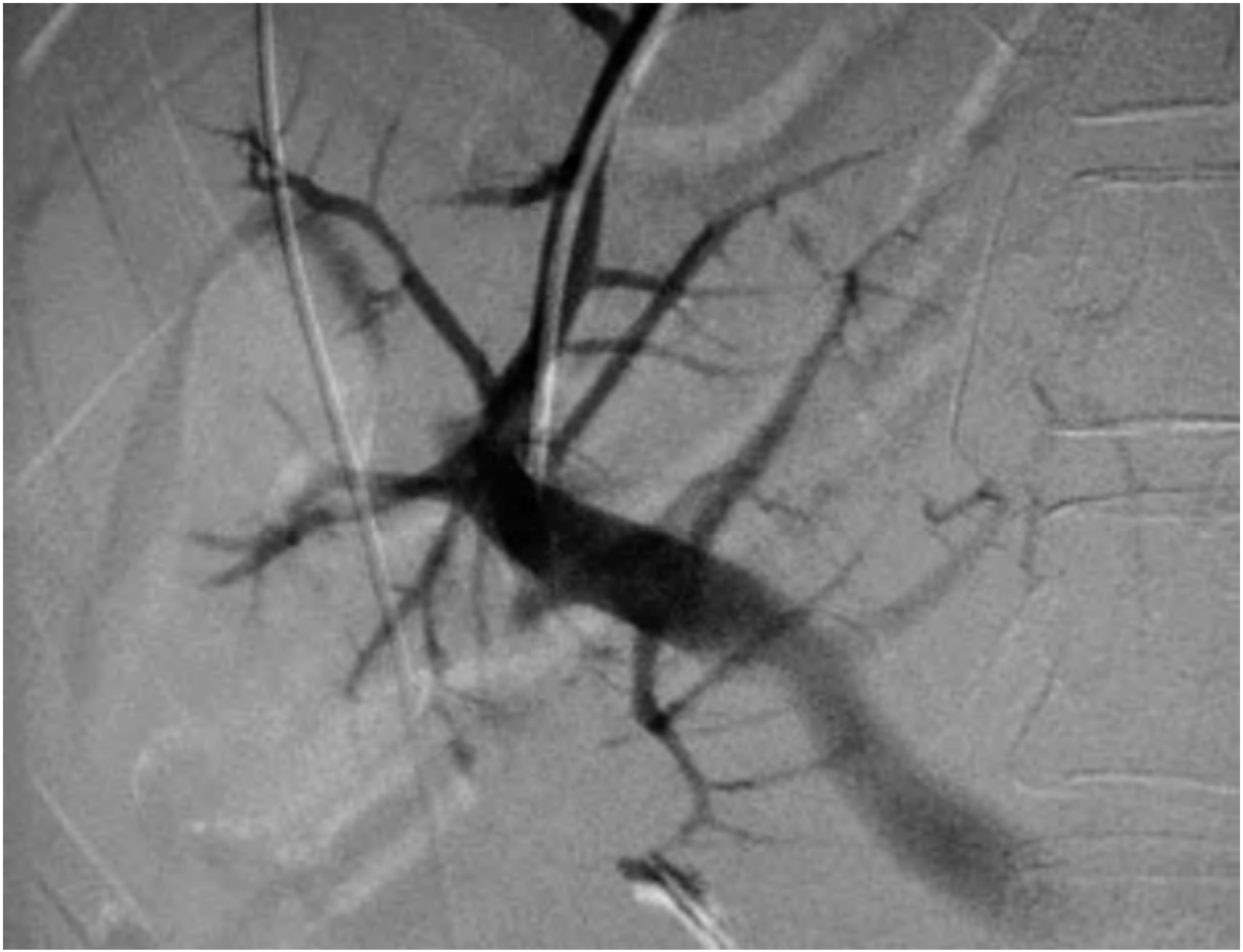

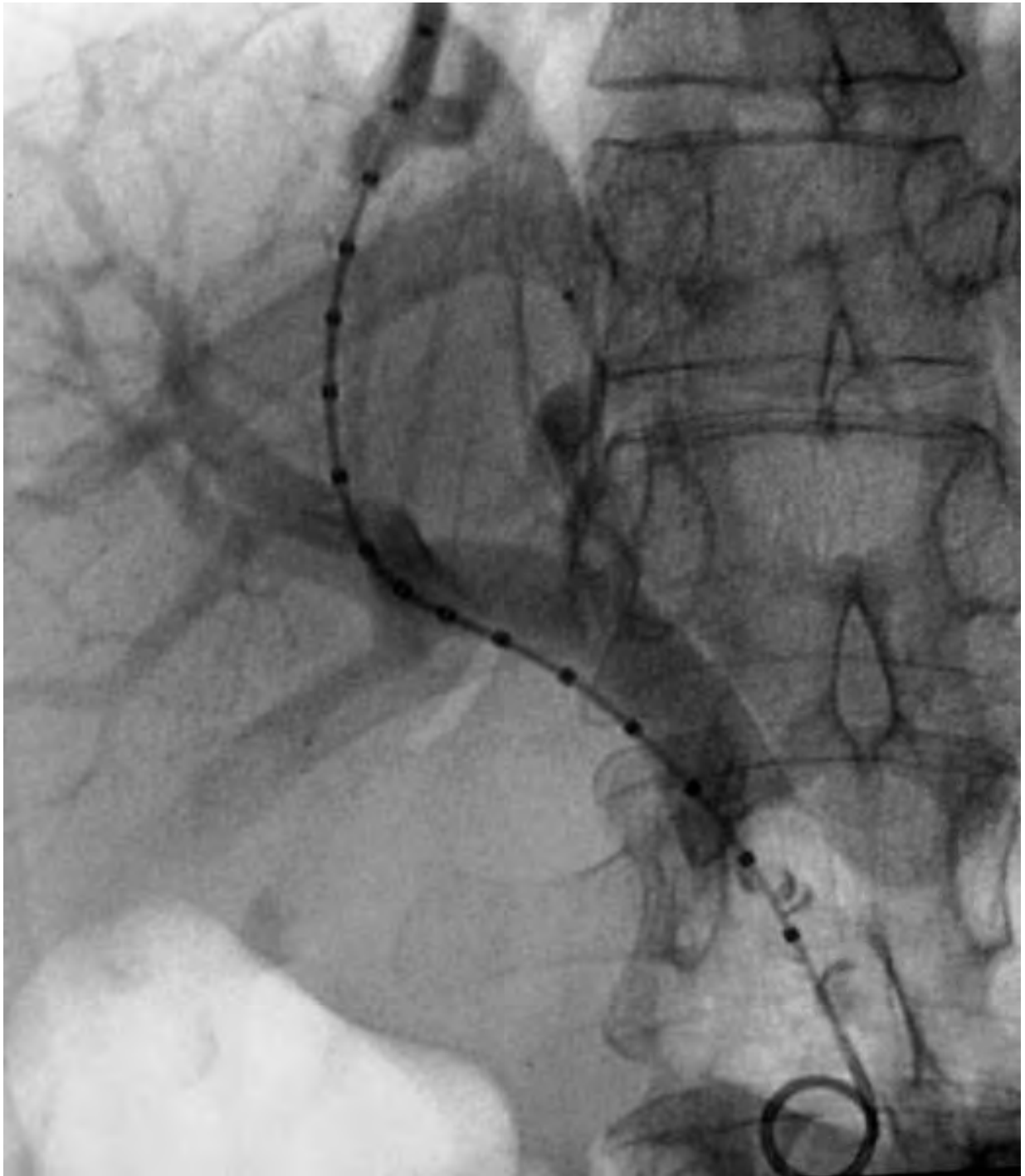

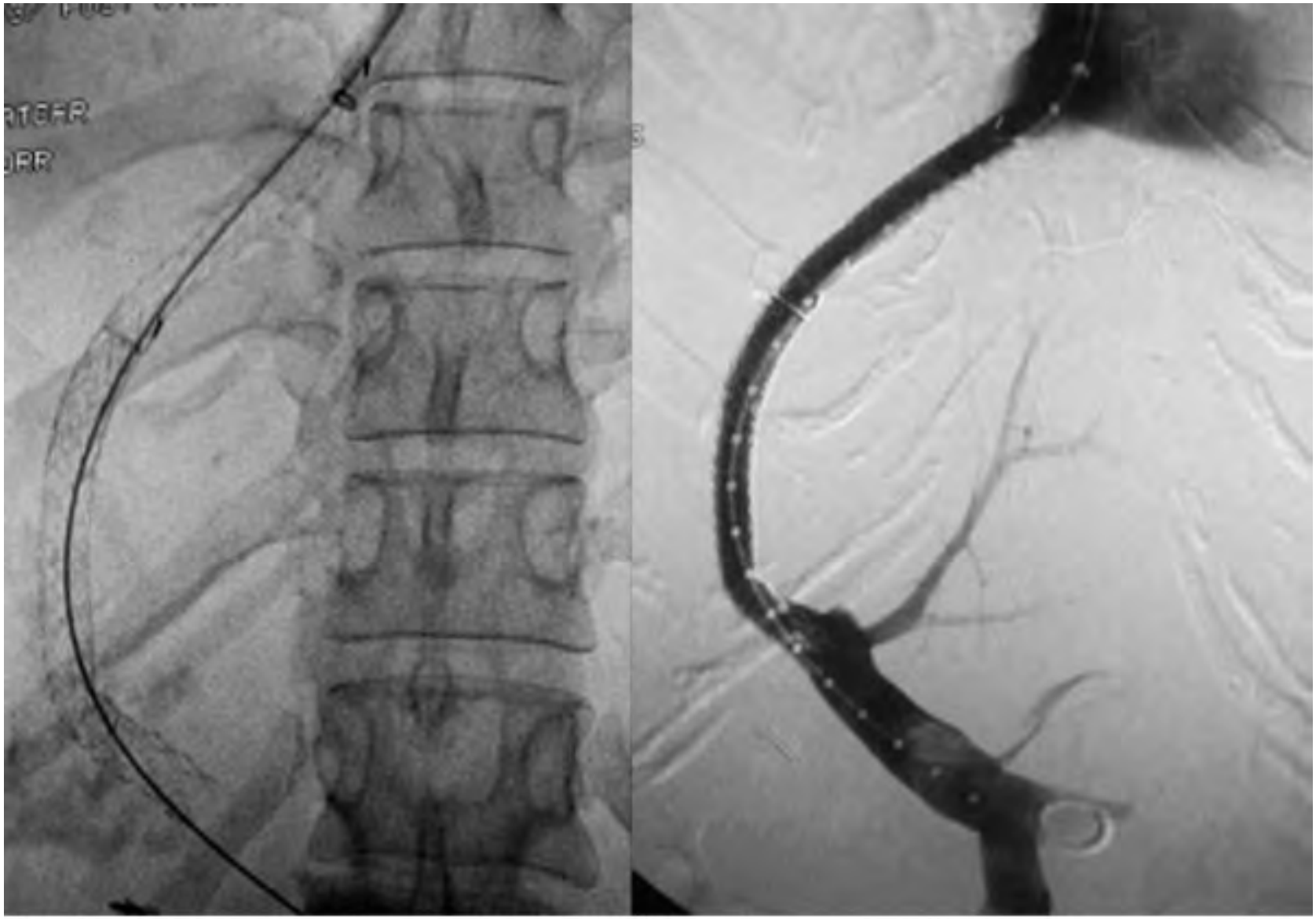

A B

Patient Selection

- Model for End-Stage Liver Disease
  - Increasing MELD score is associated with increasing severity of hepatic dysfunction and three-month mortality risk
  - Uses a patient's laboratory values for serum bilirubin, serum creatinine, and the international normalized ratio for prothrombin time (INR) to predict survival.

MELD

- MELD = 3.8[Ln serum bilirubin (mg/dL)] + 11.2[Ln INR] + 9.6[Ln serum creatinine (mg/dL)] + 6.4
- In interpreting the MELD Score in hospitalized patients, the 3 month mortality is:

  <9 — 1.9% mortality

  40 or more — 71.3% mortality

  30–39 — 52.6% mortality

  20–29 — 19.6% mortality

  10–19 — 6.0% mortality
- 3.78 (ln 1.29) + 11.2 (ln 1.3) + 9.57 (ln 1) + 6.43 = 10.33

Child-Pugh classification of severity of liver disease

Parameter	Points assigned		
	1	2	3
Ascites	Absent	Slight	Moderate
Bilirubin	<2 mg/dL (<34.2 micromol/liter)	2-3 mg/dL (34.2 to 51.3 micromol/liter)	>3 mg/dL (>51.3 micromol/liter)
Albumin	>3.5 g/dL (35 g/liter)	2.8-3.5 g/dL (28 to 35 g/liter)	<2.8 g/dL (<28 g/liter)
Prothrombin time			
Seconds over control	<4	4-6	>6
INR	<1.7	1.7-2.3	>2.3
Encephalopathy	None	Grade 1-2	Grade 3-4

Modified Child-Pugh classification of the severity of liver disease according to the degree of ascites, the plasma concentrations of bilirubin and albumin, the prothrombin time, and the degree of encephalopathy. A total score of 5-6 is considered grade A (well-compensated disease); 7-9 is grade B (significant functional compromise); and 10-15 is grade C (decompensated disease). These grades correlate with one- and two-year patient survival: grade A - 100 and 85 percent; grade B - 80 and 60 percent; and grade C - 45 and 35 percent.

I.R.R.

- CHILD SCORE:
  - Bil 1.29 = + 1, Alb: 4 = +1, INR 1.3 = +1, Ascites poorly controlled = +3, Encephalopathy none = +1 = 7
  - Significant Functional Compromise

Patient Selection

- Favorable results are usually seen in patients with a MELD score of 17 or less.
- Excellent survival in those with MELD scores of 10 or below.
- Patients with MELD scores over 24 are at high risk for early mortality and should not undergo elective TIPS.

Treatment of Ascites Caused by Cirrhosis

- International Ascites Club established three grades of ascites on the basis of severity:
  - Grade 1 ascites is detectable only by radiographic imaging with an ultrasound or computed tomography (CT) scan
  - Grade 2 ascites causes moderated symmetrical distention of the abdomen on clinical examination
  - Grade 3 causes exhibit abdominal distention, often with additional symptoms such as respiratory compromise, anorexia, and significant discomfort.

Medical Management

- Grade 1 ascites often can be treated with dietary salt restriction alone (initially started on a 2 g (88 mmol)/day sodium diet).
- Patients with grade 2 or 3 ascites require the addition of diuretic therapy to dietary salt restriction.
- Spironolactone, an aldosterone antagonist, is the most effective diuretic in the treatment of cirrhotic ascites.
- Patients with grade 3 ascites may require an initial large-volume paracentesis in addition to medical therapy.
- When more than 5 L of ascitic fluid is removed, patients should receive 8 gm/L of fluid removed of albumin during the procedure to reduce the likelihood of postparacentesis hypotension and azotemia.

Hepatorenal Syndrome

- Development of acute renal failure in a patient who has advanced liver disease
- Diagnostic criteria:
  - A plasma creatinine concentration above 1.5 mg/dL that progresses over days to weeks.
  - Urine red cell excretion of less than 50 cells per high power field (when no urinary catheter is in place) and protein excretion less than 500 mg/day.
  - Lack of improvement in renal function after volume expansion with intravenous albumin
  - The diagnosis of the hepatorenal syndrome is one of **exclusion**

Hepatorenal Syndrome

- Type I
  - More serious type
  - 50 percent lowering of the creatinine clearance to a value below 20 mL/min in less than a two week period
  - Twofold increase in serum creatinine to a level greater than 2.5 mg/dL
- Type II
  - Less severe renal insufficiency
  - Characterized by ascites that is resistant to diuretics.

Umbilical hernias with cirrhosis

- In general the following approach is recommended:
  - Patients with ruptured or incarcerated hernias are referred for immediate repair.
  - Patients with symptomatic hernias or those with marked thinning of the skin overlying the hernia sac (a sign of impending rupture), are referred for elective repair.
  - Patients with asymptomatic hernias are managed conservatively, with surgical correction of the hernia performed at the time of liver transplantation.

Liver Transplant

- The presence of cirrhosis alone is not sufficient to warrant transplantation.
- Considered when a patient has suffered from either a complication of portal hypertension or a manifestation of compromised hepatic synthetic function.
- Variceal hemorrhage, ascites, and encephalopathy are the primary manifestations of end-stage liver disease

Liver Transplantation

- Others:
  - Recurrent cholangitis in patients with primary sclerosing cholangitis (PSC)
  - Intractable pruritus in patients with primary biliary cirrhosis (PBC).

References

- Current Surgical Therapy 9th edition
- ACS Surgery, 2007 edition
- Up To Date
- Netter's Flash Cards