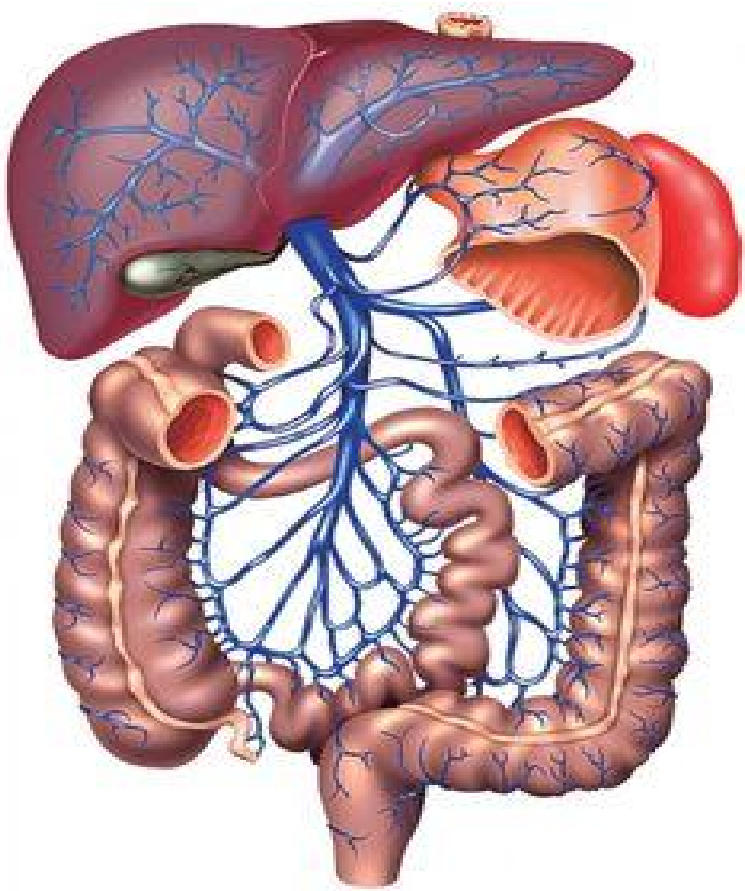

Portal Hypertension

Done By:
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Objective:
Not given by the doctor

Color Index:
-Doctor's Notes -Surgery Recall -Doctor's Slides -Important -Extra
-Davidson's

Correction File

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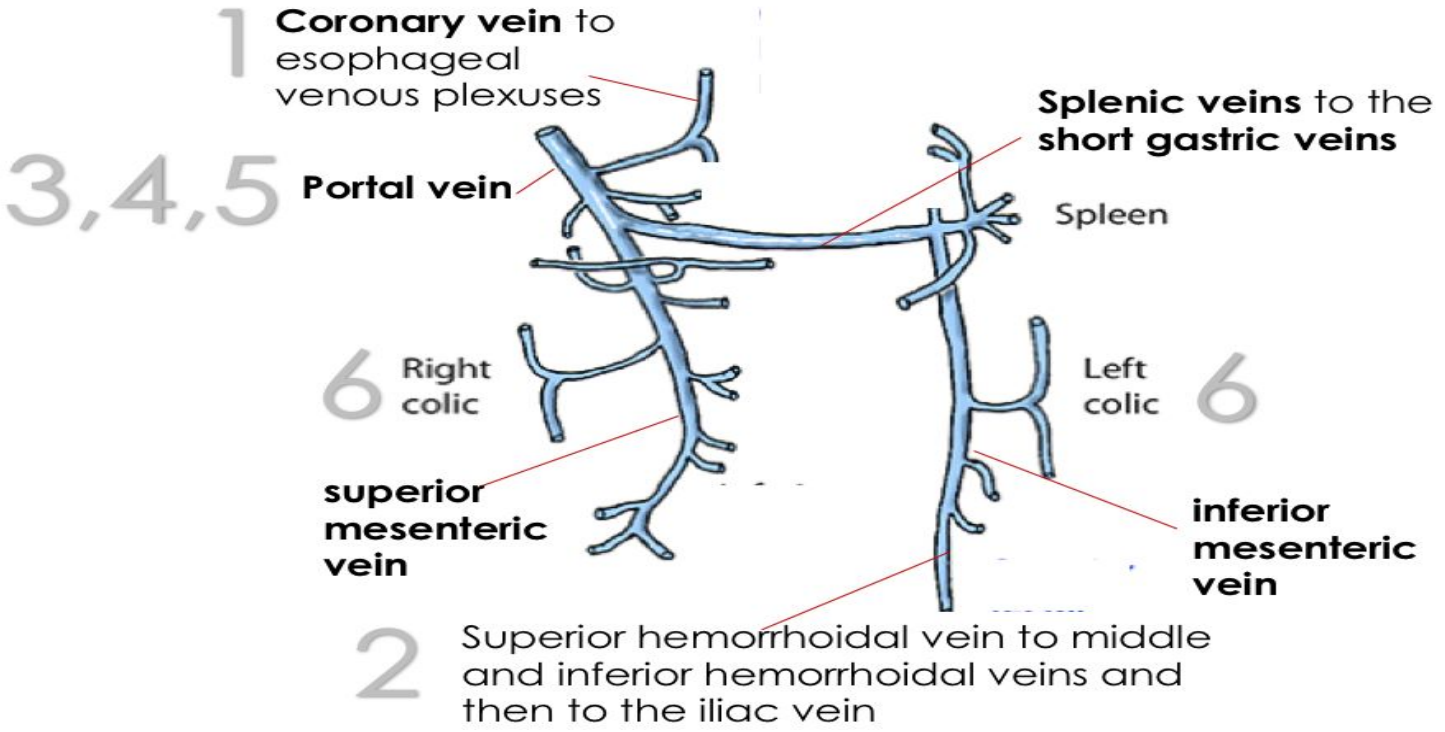
The **portal venous system** is responsible for directing blood from parts of the gastrointestinal tract to the liver. Substances absorbed in the small intestine travel first to the liver for processing before continuing to the heart. Not all of the gastrointestinal tract is part of this system. The system extends from about the lower portion of the esophagus to the upper part of the anal canal. It also includes venous drainage from the spleen and pancreas.

Portal venous system باختصار الدم بعد ما يطلع من الجهاز الهضمي يحتاج يروح محطة تصفية (الكبد) والناقل له هو

Amazing video that explains Portal circulation and portosystemic shunt (7:44 min)

portosystemic anastomosis



	PLACES OF PORTOCAVAL ANASTOMOSES	portal	systemic	complication
1	**LOWER END OF OESOPHAGUS**	Left gastric vein	accessory hemiazygos vein	**Esophageal varices**
2	**ANAL CANAL (upper part)**	Superior rectal vein	middle and inferior rectal vein	**Hemorrhoids**
3	**UMBLICUS**	Left branch of portal vein (through paraumblical vein)	veins of anterior abdominal wall (superior /inferior epigastric veins)	**Caput medusae**
4	**BARE AREA OF LIVER**	Hepatic venules	phrenic and intercostal veins	
5	**LIVER**	Patent ductus venosus	portal vein directly into inferior vena cava	
6	**POSTERIOR ABDOMINAL WALL**	Veins of retroperitoneal organ	renal vein	
Mnemonic (ALL in PUB) *				

IMAGINE=> Portal vein is the "main road", while Lower esophageal, Anal and umbilical veins are the "branches or collaterals"..
Normally (Portal pressure=systemic(Caval) pressure, so there is no backflow from the portal vein to the systemic circulation.
Normally:GIT+spleen⇒ portal vein⇒ Liver ⇒ Hepatic vein ⇒ IVC ⇒ Heart
Portal HTN: GIT⇒ Portal vein⇒ Liver (the pressure is too high) ⇒ Collaterals
Whenever there is crowded meen road people will run to the branches "collateral" (portosystemic shunt).

Portal Hypertension

The normal pressure of 5–15 cmH2O in the portal vein is consistently exceeded (above 25 cmH2O).
Portal hypertension is caused by increased resistance to portal venous blood flow, the obstruction being prehepatic, hepatic or posthepatic

Etiology :

1. **Prehepatic:**
   - •Thrombosis of portal or splenic vein⇒ Rare and it's most commonly due to neonatal umbilical sepsis.
   - •atresia of portal vein
2. **Hepatic:**
   - **Cirrhosis (>90% of cases)**

The most common cause of portal hypertension is cirrhosis resulting from chronic liver disease and is characterized by:
liver cell damage⇒ fibrosis and nodular regeneration⇒ The fibrosis obstructs portal venous return⇒ portal hypertension develops.
Alcohol is the most common aetiological factor in developed countries,
whereas in North Africa, the Middle East and China, schistosomiasis due to *Schistosoma mansonii* is a common cause.
Chronic active hepatitis and primary and secondary biliary cirrhosis may result in portal hypertension,
but in a large number of patients the cause remains obscure (cryptogenic cirrhosis).

   - Schistosomiasis: (most common cause outside North America)
   - Hepatitis, Hepatocellular carcinoma, Fibrosis
   - Metabolic : hemochromatosis, Wilson's disease thrombosis
3. **Posthepatic**
   - •Budd-Chiari syndrome :thrombosis of hepatic veins.

is a very rare condition, affecting 1 in a million adults. The condition is caused by occlusion of the hepatic veins that drain the liver. It presents with the classical triad of abdominal pain, ascites, and liver enlargement. The formation of a blood clot within the hepatic veins can lead to Budd–Chiari syndrome.

Physical finding :

★ **Asymptomatic** : symptoms are usually absent until they develop the complication.
★ **Complications:**

- **Splenomegaly** (most common)

Progressive enlargement of the spleen occurs as a result of vascular engorgement and associated hypertrophy resulting in Hypersplenism*.
Haematological consequences are anaemia, thrombocytopenia and leucopenia (with the resulting syndrome of hypersplenism).

*(is a condition in which the spleen becomes increasingly active and then rapidly removes the blood cells. It can result from any splenomegaly. It is most common with splenomegaly secondary to portal hypertension and haematological disorders.

- **Esophageal varices** (see no.1 in the pic)
- **Caput medusae** (engorgement of periumbilical veins) (see no.2 in the pic)
- **Cruveilhier-Baumgartenbruit** (periumbilical bruit heard with caput medusae)
- **Hemorrhoids** (see no.3 in the pic)
- **Others include :** Spider angioma ,palmar erythema , **ascites** .

truncal obesity and peripheral wasting, encephalopathy ,asterixis (flapping tremor or liver flap) ,gynecomastia, ,jaundice

- Underlying disease

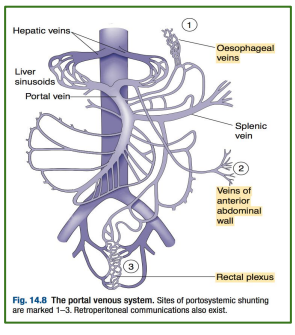


Fig. 14.8 The portal venous system. Sites of portosystemic shunting are marked 1–3. Retroperitoneal communications also exist.

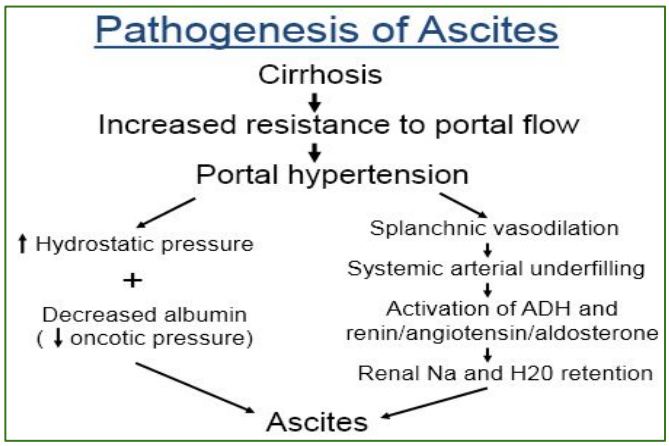
-Esophageal varices :

- Engorgement of the esophageal venous plexuses secondary to increased collateral blood flow from the portal system as a result of portal hypertension
- ★ **The most important and feared complication of portal hypertension is Bleeding from esophageal varices**
- the bleeding presents as **Hematemesis**, melena, hematochezia
- **Ruleo 2/3 :** 2/3 of patients with cirrhosis develop portal hypertension > 2/3 of them will develop esophageal varices>2/3 of those with varices will develop variceal hemorrhage
- mortality rate from an acute esophageal variceal bleed is 50%

-Ascites :

- Ascites may develop and is due to increased formation of hepatic and splanchnic lymph*, hypoalbuminaemia, and retention of salt and water. Increased aldosterone and antidiuretic hormone levels may contribute. Porto-systemic encephalopathy is due to an increased level of toxins such as ammonia in the systemic circulation.
- Ascites can be controlled by bed rest, salt and water restriction, and the aldosterone inhibitor spironolactone. If refractory, ascites can be treated by inserting a peritoneo-jugular (LeVeen) shunt, which allows one-way flow between the peritoneum and the jugular vein

*Lymphatic flow is increased proximal to the point of vascular obstruction and, when the capacity of the lymphatic system is surpassed, the transudate moves across the surfaces of the liver, mesentery and intestine into the peritoneal cavity.



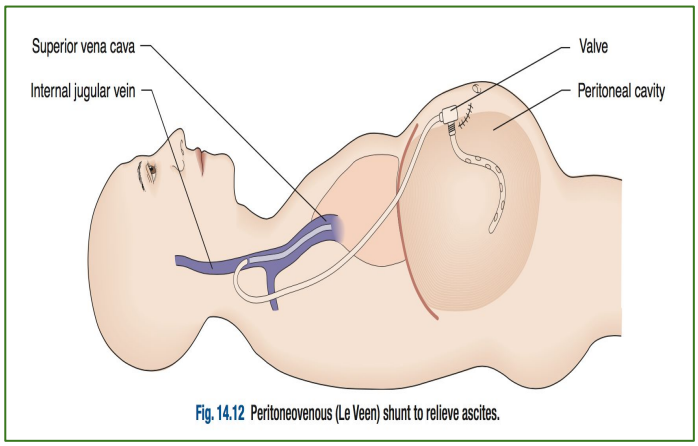


Fig. 14.12 Peritoneovenous (Le Veen) shunt to relieve ascites.

-Hepatic encephalopathy:

- Portosystemic encephalopathy is due to an increased level of toxins such as ammonia in the systemic circulation (Remember no hepatic filtration).
- This is particularly likely to develop where there are large spontaneous or surgically created portosystemic shunts*.
- In patients with cirrhosis, acute encephalopathy is most commonly associated with a precipitating factor, such as electrolyte disturbance, medications, gastrointestinal hemorrhage, or infection.

*The surgical portosystemic shunts (PSS) are a time-proven modality for treating portal hypertension. Recently, in the era of liver transplantation and the transjugular intrahepatic portosystemic shunts (TIPS), use of the PSS has declined (will be further explained in the coming pages).

*Signs/symptoms of liver disease:

Hepatomegaly, splenomegaly, icterus, pruritus (from bile salts in skin), blanching spider telangiectasia, gynecomastia, testicular atrophy, caput medusae, dark urine, clay-colored stools, bradycardia, edema, ascites, fever, fetor hepaticus
(sweet musty smell), hemorrhoids, variceal bleeding, anemia, body hair loss, liver tenderness, palmar erythema

★ **The most common non cirrhotic causes that we see specially in young females is venous thrombosis due to congenital hypercoagulable states.**

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)

★ **Very, very, very important MCQ question**

Table 14.2 Assessment of patients with portal hypertension using a modification of Child's grading system

Points scored			
Criterion	**1**	**2**	**3**
Encephalopathy	None	Minimal	Marked
Ascites	None	Slight	Moderate
Bilirubin (μmol/l)	< 35	35–50	> 50
Albumin (g/l)	> 35	28–35	< 28
Prothrombin ratio	< 1.4	1.4–2.0	> 2.0

Grade A = 5–6 points; grade B = 7–8 points; grade C = 10–15 points.

Group A: Good Prognosis, Group C: the worst

FIRST LINE OF TREATMENT :

A.Empiric treatment:

(the initial treatment of variceal bleeding is as with all upper GI bleeding) :

- Start treatment while you prepare the patient for endoscopy.
- **General approach of any upper GI bleeding is ABCs!!**
  - ★ **AIRWAYS** : intubation to protect from aspiration if needed.
  - ★ **BREATHING** : check the ventilation and oxygenation.
  - ★ **CIRCULATION** :check BP and end organ perfusion.
    - start resuscitation with 2 large peripheral IV and start 2 L normal saline or ringer lactate (what is the rate ? **As fast as you can !)**
    - **in sever bleeding often blood is given (**o negative blood until you get crossmatched blood.
    - correct coagulopathy (vitamin K, fresh frozen plasma)

B.Specific treatment :

1)Upper GI endoscopy

Diagnostic test of choice : allows the detection of varices and defines the site of bleeding to to rule out ulcers, gastritis.

This allows the detection of varices and defines whether they are or have been the site of bleeding. It is important to remember that peptic ulcer and gastritis are common complaints that occur in 20% of patients with varices.

Therapeutic options :

- **Emergent endoscopic sclerotherapy:** a sclerosing substance is injected into the esophageal varices under direct endoscopic vision. Repeated at weekly or fortnightly intervals
- **Endoscopic band ligation:** elastic band ligation of varices. **The method of choice for control of variceal haemorrhage,**

If haemorrhage is torrential and prevents direct injection, balloon tamponade, TIPS or other surgical treatment may be used to stop the bleeding.

There are many different types of balloons manufactured for the purpose of tamponading upper gastrointestinal bleeds, each with different volume capacities and aspiration ports tailored for the specific application:
The four-lumen Minnesota tube has largely replaced the three- lumen Sengstaken–Blakemore tube. The four lumina allow:

- aspiration of gastric contents
- compression of the oesophagogastric varices by the inflated gastric balloon
- compression of the oesophageal varices by the inflated oesophageal balloon
- aspiration of the oesophagus and pharynx to reduce pneumonic aspiration.

Balloon tamponade arrests bleeding from varices in over 90% of patients, but the tube is not left in place for more than 24–36 hours for fear of causing esophageal necrosis.

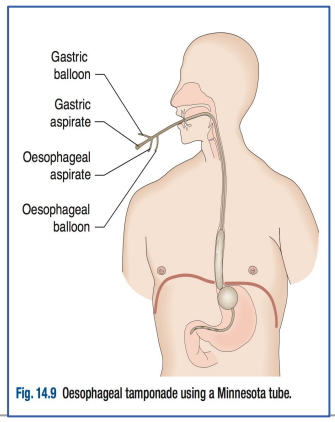


Fig. 14.9 Oesophageal tamponade using a Minnesota tube.

2)Pharmacologic options.

Somatostatin (Octreotide) If terlipressin is unavailable

- lowers portal venous pressure and arrest bleeding
- 50 mcg bolus followed by 50 mcg/hour by intravenous infusion)

IV vasopressin : to achieve vasoconstriction of the mesenteric vessels

•nitroglycerin :to avoid MI

•Prophylactic antibiotics :

- preferably before endoscopy although effectiveness has also been demonstrated when given after.
- Suggest intravenous ceftriaxone (1 g IV) or Cipro (400 mg IV BID)

★ **Once the bleeding is controlled**

1. Repeat endoscopic sclerotherapy/banding for ***Prevention of further bleeding*⇒** until the varices are completely sclerosed but excessive intervention may cause ulceration and necrosis.

2. Or do Surgical disconnection of the esophagus.

Second Line of Treatment :

★ **if sclerotherapy and conservative methods fail to stop the variceal bleeding or bleeding recurs then consider the following :**

1.Repeat sclerotherapy/banding and treat conservatively

2. TIPS(Transjugular Intrahepatic Portosystemic Shunt)

Angiographic radiologist places a small tube stent intrahepatically between the hepatic vein and a branch of the portal vein via a percutaneous jugular vein route

- a metal stent is inserted via the transjugular route using a guidewire passed through the hepatic vein to the intrahepatic branches of the portal vein.
- used occasionally to decompress the portal system and reduce the risk of further variceal haemorrhage in patients with well preserved liver function who fails emergent endoscopic treatment and has no complications from the bleeding or endoscopy.
- The choice of surgery usually depends upon the availability, training, and expertise of the surgeon. Although a selective shunt has some physiologic advantages, it may significantly exacerbate marked ascites.
- portosystemic encephalopathy is a major complication so in severe liver disease, transplantation is more likely to be considered if there is no contraindication.

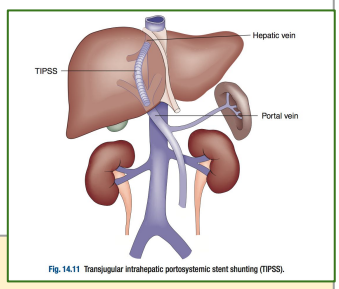


Fig. 14.11 Transjugular intrahepatic portosystemic stent shunting (TIPSS).

3. Surgical shunt (selective or partial)

Focus on the 1st two "mentioned in our lecture"

See P9 for further understanding

1-Selective shunt: Shunt that selectively decompresses the varices without decompressing the portal vein

2-partial shunt: Shunt that directly decompresses the portal vein, but only partially

3-Warren shunt (selective shunt)

•Distal splenorenal shunt with ligation of the coronary vein

•elective shunt procedure associated with flow incidence of encephalopathy in patients postoperatively because only the splenic flow is diverted to decompress the varices

•**contraindicated in** Ascites

4-End-to-side portocaval shunt:" total shunt"—portal vein (end) to IVC (side)

-**Side-to-side portocaval shunt** Side of portal vein anastomosed to side of IVC—partially preserves portal ow ("partial shunt")

5-**Synthetic portocaval H-graft** : "Partial shunt"—synthetic tube gra from the portal vein to the IVC (good option for patients with alcoholism; associated with lower incidence of encephalopathy and easier transplantation later)

6-**Synthetic mesocaval H-graft** :Synthetic graft from the SMV to the IVC

Most common perioperative cause of death following shunt procedure :Hepatic failure, secondary to decreased blood flow (accounts for two thirds of deaths)

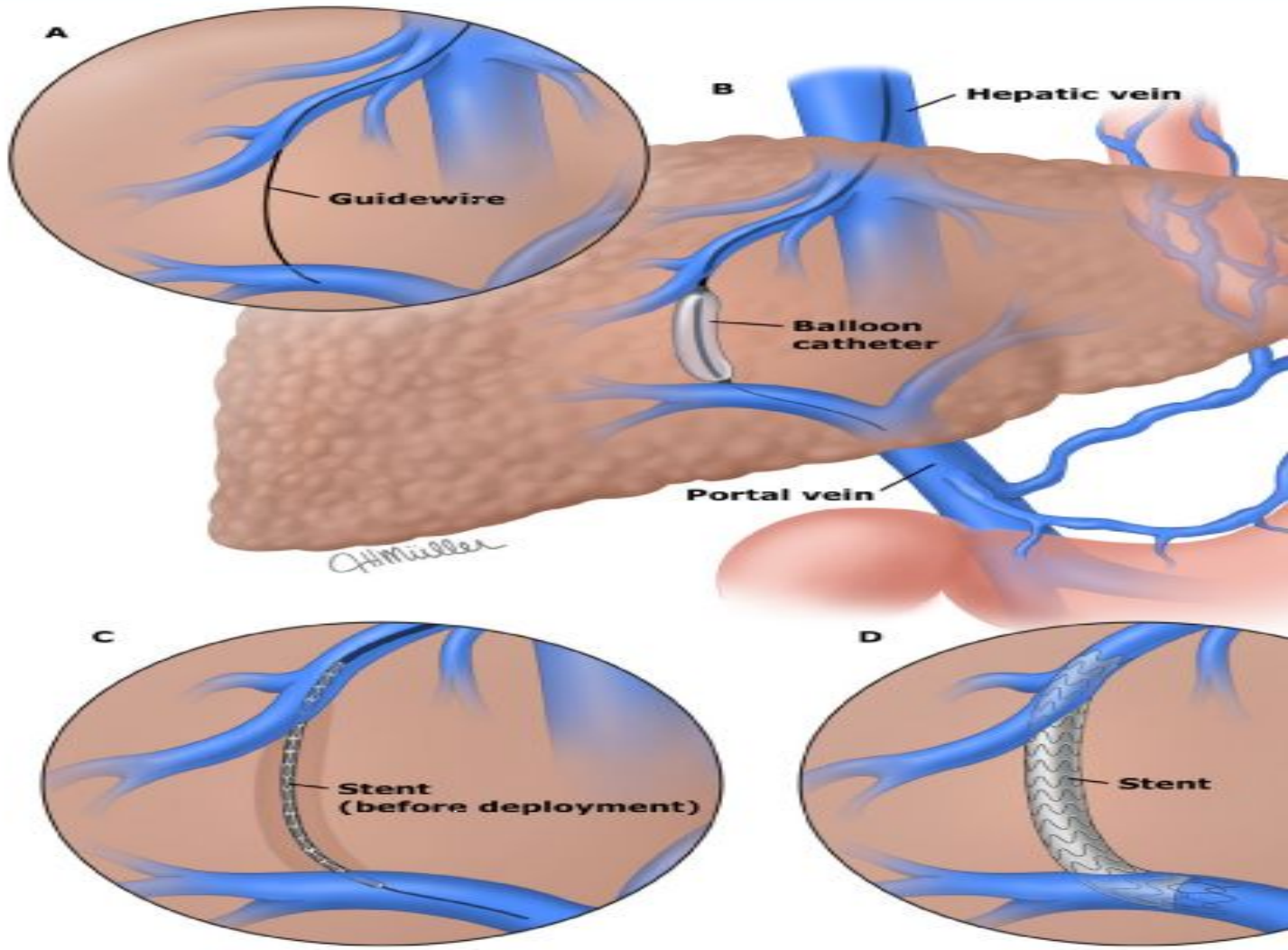
major postoperative morbidity a er a shunt procedure? Increased incidence of hepatic encephalopathy because of decreased portal blood flow to the liver and decreased clearance of toxins/metabolites from the blood

4.Liver transplantation

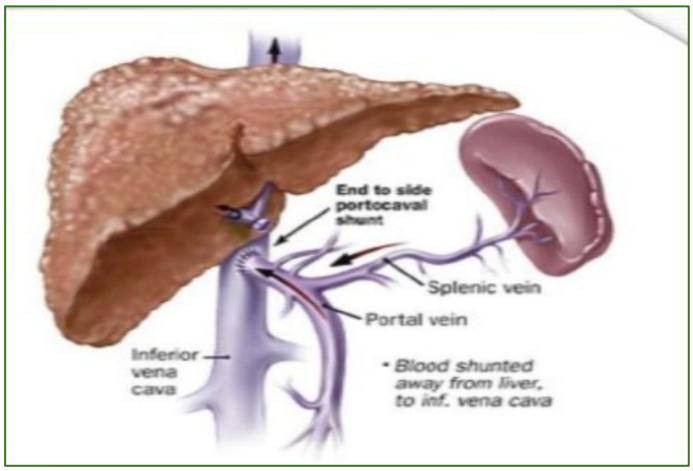
Table 14.3 Priorities in the management of bleeding oesophageal varices

Active resuscitation • Group and cross-match blood • Establish i.v. infusion line(s) • Monitor Pulse Blood pressure Hourly urine output Central venous pressure
Assessment of coagulation status • Prothrombin time • Platelet count
Urgent endoscopy **Control of bleeding** • Endoscopic banding or injection sclerotherapy • Tamponade (Minnesota tube) if bleeding uncontrolled • Pharmacological measures (e.g. vasopressin/octreotide)
Treatment of hepatocellular decompensation **Treatment/prevention of portosystemic encephalopathy** **Prevention of further bleeding from varices** • Endoscopic banding or injection sclerotherapy • Stapled oesophagogastric junction • Portosystemic shunting/transjugular intrahepatic portosystemic stent shunting (TIPSS) • Liver transplantation

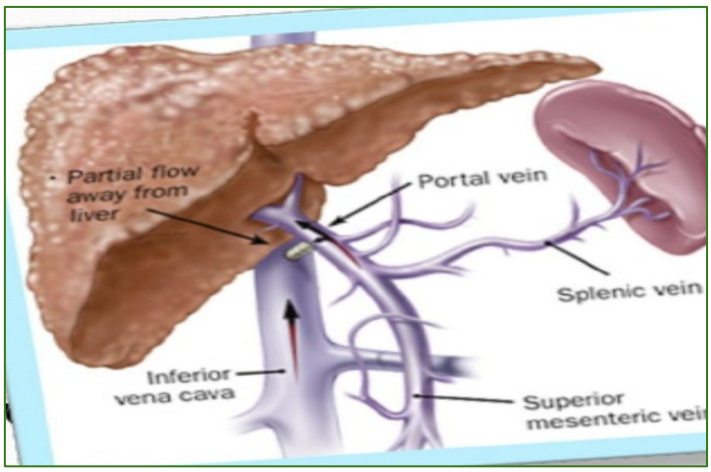
Transjugular intrahepatic portosystemic shunt



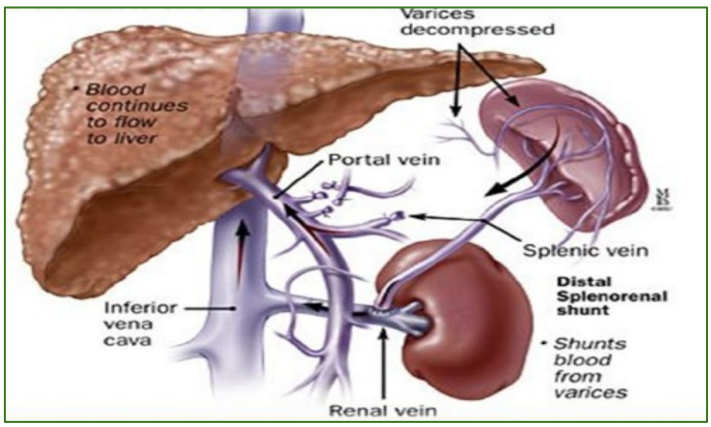
A transjugular intrahepatic portosystemic shunt (TIPS) is cre
passing a needle catheter via the transjugular route into the
and wedging it there. The needle is then extruded and advan
the liver parenchyma to the intrahepatic portion of the porta
stent is placed between the portal and hepatic veins. A TIPS
side-to-side surgical portacaval shunt, but does not require
anesthesia or major surgery for placement. (A) Passage of a
between the hepatic vein and the portal vein. (B) Inflation of
catheter within the liver to dilate the tract between the hepat
portal vein. (C) Deployment of the stent. (D) Stent in its fina



1.Non selective(end to side)



2. Side to side



3. Selective shunt

INDICATIONS OF LIVER RESECTION

Benign	Malignant
Adenoma: The most common benign liver lesion that can be resected. Has an **Estrogen receptors**, so **it gets bigger** in pregnancy and with oral contraceptives.The average age of occurrence is between 30–35 years of age. Diagnosis made is made by CT scan, U/S, biopsy (rule out hemangioma with RBC-tagged scan!) Should be resected because: **1. Could transfer to HCC. "malignant potential"** **2. High risk of rupture and bleeding.** **When we do surgery for adenomas?** • if it was tiny and small → follow up especially in boys. • If it was **medium or large "5cm<"** in female → tend to be more aggressive **and we remove it.** • If patient is on oral contraceptive → stop the oral contraceptive and see if it go away completely. **If did not go away completely → resect the tumor.** • If the patient want to get pregnant we should resect the tumor **before she get pregnant** "b/c high risk to rupture, which is both fetal to the mother and the baby" **Other benign tumors:** **observe the patient until he/she become symptomatic only then we do surgery.** The pain from the liver is very specific and localized, Not vague abdominal pain.	**Primary:** HCC: CC: cancer of the epithelial cells of the biliary ducts. Both are adenocarcinoma **Metastatic:** the most common liver tumors are secondary (metastatic) tumors, more commonly from colon. One of the indications for surgery in liver malignancies is **colorectal cancer metastasis**. It will increase 5 years survival in 30-40% of the patients. You resect even if it come back after resection.

❖ **Outcomes of liver surgery:**

- Mortality rate < 2%
- Complication rate 20-30%
- The only potential for cure in case of malignant tumor. Even in colon metastatic. Chemotherapy is used to shrink the tumor not cure it.

❖ **How much can we resect from the liver?**

In normal healthy individual we can resect 70% i.e. keeping 30% ≈ 2 segments out of 8.

❖ **Can we do anything else?** Not important.

Make the normal liver bigger, and the malignant tumor smaller.

Primary liver disease "HCC "	Metastatic liver disease "CRCLM"
What is the background of the liver?	
Diseased liver, usually cirrhotic, fatty or inflammation "Hepatitis B virus" or toxins. "aflatoxin". So, there is problem that generated the cancer.	Healthy and normal liver
How much resection can be tolerated?	
Limited, because a diseased liver will not tolerate 70% resection. Usually you can resect only 30%.	70% can be resected in normal liver.
The age of the patient	
Usually older, the patient himself cannot tolerate the surgery.	Usually younger. Colon cancer affect younger group then primary liver cancer,
Liver transplant?	
Transplant option **Only exesist in patient with severe cirrhosis and very small, primary adenocarcinoma liver cancer.** **NOT an option for all other liver cancers including all metastatic liver disease. Because all these cancers will come back.** **Indications for liver transplantation:** Cirrhosis and NO resection candidacy as well as no distant or lymph node metastases and no vascular invasion; the tumor must be **single**, 5-cm tumor or have three nodules, with none 3 cm.	**NEVER**

❖ Complications of liver resection:

Liver specific complication:

- **Liver Failure** very high chance to die.
- **Bleeding,** 25% of CO and 2 venous systems and arterial system = 3 holes.
- **Bile leaks.** don't respond to coagulation. Because of the leak you have high risk for developing Infections (wound, deep abscess).

General complications:

- UTI, Pneumonia, headache, fever …etc

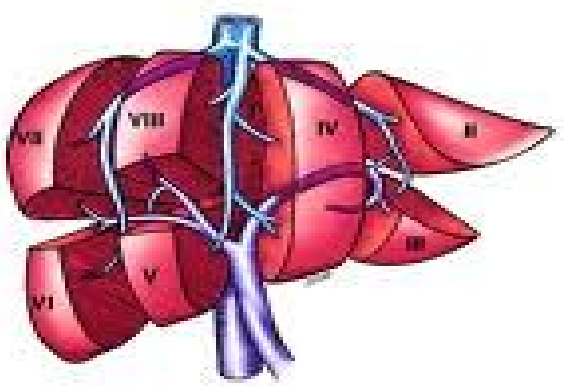
❖ Types of resections:

8 segments:

- ❖ Right side: segment 5,6,7,8. "50% of the liver"
- ❖ Left side: segment 2,3,4. "30-40% of the liver"
- ❖ Posterior: segment 1.

So, right hepatectomy= segment 5,6,7,8

Left hepatectomy= segment 2,3,4



What is the overall arrangement of the segments in the liver?

Clockwise, starting at segment 1

Liver Resection, From Davidson.

Resection involves mobilization of the liver from its peritoneal attachments. Following isolation, ligature and division of the appropriate vessels, the devascularized lobe or segment is separated by careful dissection, which may be facilitated by the use of an ultrasonic dissector. The hepatic veins or tributaries are controlled by suture ligation following removal of the resected specimen. Postoperative monitoring is undertaken in a high dependency environment and should include early blood gas, glucose and lactate measurement. Hepatic dysfunction may be evident from prolongation of the prothrombin time in major liver resectional surgery.

Tumors of the Liver.
From Davidson.

Tumors of the liver	
1-Benign Tumors.	
Cavernous Heamangioma	most common benign liver tumors. These lesions rarely reach a sufficient size to produce pain, abdominal swelling or haemorrhage. Heart failure rarely develops, if there is a large arteriovenous communication. Lesions discovered incidentally at laparotomy should be left alone; needle biopsy can be hazardous. Large symptomatic lesions should normally be resected only by an experienced surgeon.
Biliary Hamartoma	These are small fibrous lesions situated beneath the capsule of the liver. They can be mistaken for a small metastatic tumour unless a biopsy is obtained.
Focal Nodular Hyperplasia (FNH)	more common in females. The lesion is generally asymptomatic and may regress with time or on withdrawal of the contraceptive pill. Hyperplasia can be differentiated from adenoma by the central fibrous scar, which is often visible on ultrasound or CT. Such lesions do not undergo malignant transformation and do not require excision unless symptomatic.
Liver Cell Adenoma	uncommon and is found almost exclusively in women. The **use of contraceptives** containing high levels of oestrogen have been implicated causally. The majority present as solitary, well-encapsulated lesions, but malignant transformation has been reported. Generally present with right hypochondrial pain as a result of haemorrhage within the tumour.Superficial tumours may bleed spontaneously and present with symptoms of hemoperitoneum. Adenomas may be detected by ultrasonography or CT. LFTs and serum α-fetoprotein levels are usually normal. Treatment consists of formal hepatic resection because of the difficulties of distinguishing adenoma from a well-differentiated hepatoma, concerns that lesions may undergo malignant transformation, and the known risk of spontaneous haemorrhage. There is recent evidence to suggest that cytokeratin 7 and 19 immunostains along with neuronal adhesion molecule taken from liver biopsy can help in differentiating hepatic adenoma from FNH when radiological imaging is inconclusive.

2-Malignant Primary Tumors	
Hepatocellular Carcinoma (HCC)	It is common in Africa and the Far East and is more common in males.In the West, about two-thirds of patients have pre-existing cirrhosis and many others have evidence of hepatitis B or C infection. In Africa and the East, '**aflatoxin**' (derived from the fungus, Aspergillus flavus, which contaminates maize and nuts) is an important hepatocarcinogen. **Clinical features.** In non-cirrhotic patients, the tumour may have grown to a considerable size before giving rise to abdominal pain or swelling. In cirrhotic patients, HCC may become manifest as sudden deterioration in liver function,often associated with extension of the tumour into the portal venous system. Common presenting features are abdominal pain, weight loss, abdominal distension, fever and spontaneous intraperitoneal haemorrhage.Examination may reveal hepatomegaly. **Investigations.** Elevated α-fetoprotein (an oncofetal antigen) and ultrasound scanning. Percutaneous needle aspiration cytology and needle biopsy for histological confirmation should be reserved for patients who are not being considered for hepatic resection, as these investigations carry a small but significant risk of tumour dissemination and haemorrhage. Hepatocellular carcinoma is seen as an extremely vascular lesion on common and satellite lesions often surround the primary tumour, so that cure is uncommon.For advanced tumours, systemic chemotherapy. by selective arteriography (transarterial chemoembolization– TACE) and percutaneous ablation using radiofrequency and microwave energy have useful effect for small lesions not amenable to surgery. Liver transplantation has been used also in the treatment of this tumour, but the best results have been reported in cirrhotic patients in whom an incidental HCC has been found.
Cholangiocarcinoma	Rare, adenocarcinoma may arise anywhere in the biliary Tree. Include chronic parasitic infestation of the biliary tree in the Far East, and choledochal cysts. Jaundice, pain and an enlarged liver are the common presenting features, although there may be co-existing biliary infection causing the tumour to masquerade as a hepatic abscess. Resection offers the only prospect of cure.
Angiosarcoma	This rare tumour of the liver may arise after industrial exposure to vinyl chloride or exposure to the previously used radiological contrast medium, Thorotrast.
Hemangioendothelioma	This presents as a diffuse multifocal tumour and is rarely resectable at presentation.
Biliary Cystadenoma	This rare condition of the liver, with a marked female predominance, High risk of malignant transformation and should be resected.

The liver is a common site for metastatic disease; secondary liver tumours are 20 times more common than primary ones.Almost 90% of patients with hepatic metastases have tumour deposits in other sites.**Hepatomegaly and tenderness** are distinctive features, The patient may be cachectic, and ascites or jaundice may be present. Pyrexia occurs in 10%.The alkaline phosphatase and γ-glutamyl transpeptidase are often raised. Ultrasound and CT may demonstrate multiple filling defects. The diagnosis can be confirmed by aspiration cytology or needle biopsy undertaken under ultrasound control.There is no effective treatment for most patients with hepatic metastases due to the extent of liver involvement and the presence of extrahepatic disease. Nonetheless, for some tumours, notably those arising from the colon and rectum, disease may be confined to the liver and there is strong evidence of survival benefit if these are resected. Assessment of resectability will require a careful search to exclude or assess extrahepatic disease. This may necessitate colonoscopy and CT of chest, abdomen and pelvis. A more radical approach to resection of liver metastases has resulted from advances in chemotherapy and has been combined with staged resection of liver disease and preoperative portal embolization to induce hypertrophy of the intended residual liver. In well-selected patients, 5-year survival rates of 30–40% have been reported following resection. Non-curative resection may be considered exceptionally as a means of palliation in patients with symptomatic hepatic metastases such as a carcinoid or other neuroendocrine tumours.