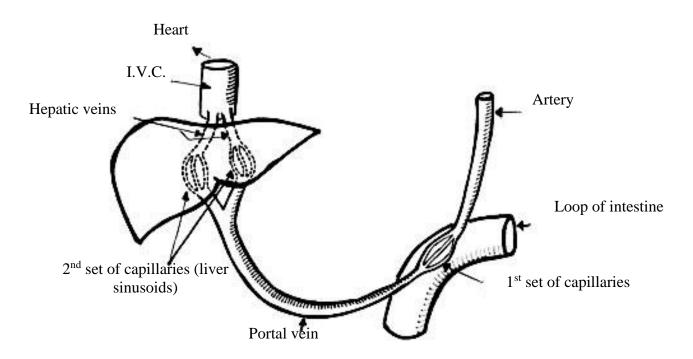
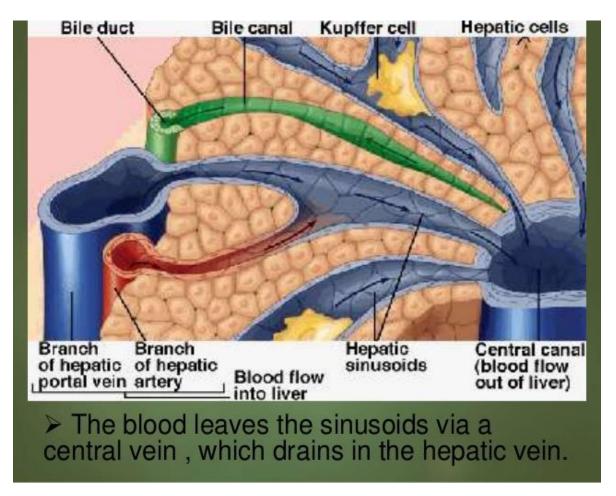
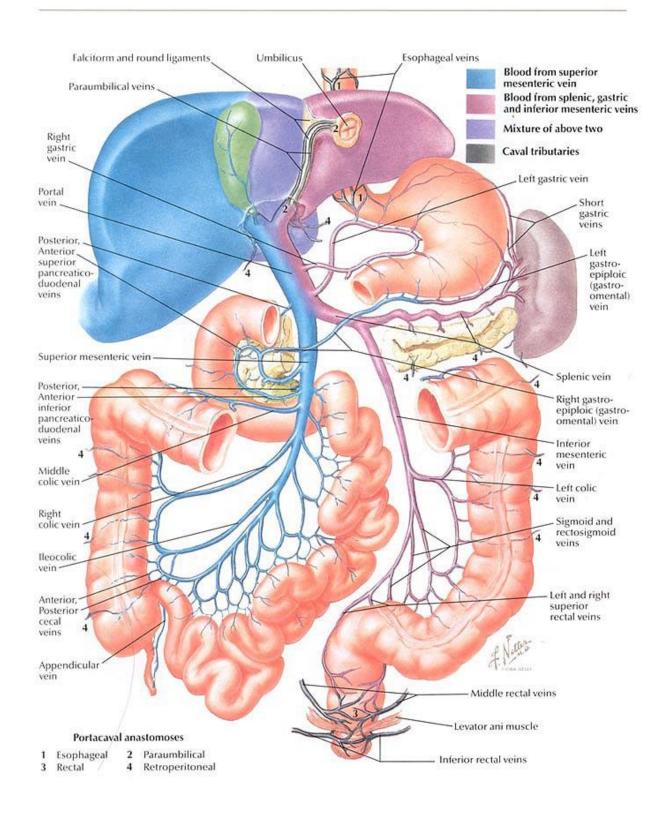
* Portal Circulation*





Portal Vein Tributaries: Portacaval Anastomoses



PORTAL HYPERTENSION

* **Definition:** Increase of the portal venous pressure above 10 mm Hg (N:5-10 mm Hg.)

* Aetiology:

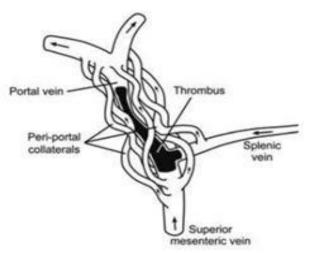
- Portal hypertension occurs mainly due to increase resistance to portal blood flow .
- Causes of portal hypertension are classified into :

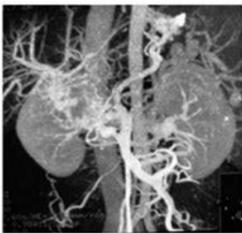
I) Pre-hepatic (Infra-hepatic) causes:

- 1) **Portal vein thrombosis** due to neonatal umbilical sepsis or abdominal infection eg. Complication of appendicitis , pancreatitis or inflamed pile .
- 2) *Congenital* cavernomatous transformation, atresia or stenosis of portal vein .

Cavernous transformation

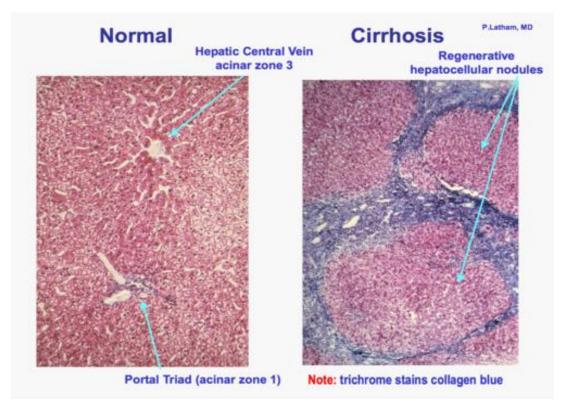
Cavernoma may be identified as soon as 15-30 days after the onset with abdominal symptoms



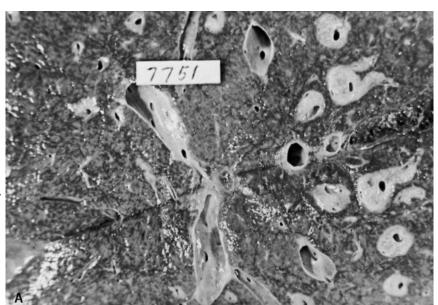


Zhang, World J Gastroenterol, 2011

- 3) Compression by neighboring tumor e.g. *carcinoma* of pancreas or malignant lymph nodes in the porta hepatis.
- 4) Increase blood viscosity as polycythemia
- *II) Intra-hepatic causes:* (90% of cases)
 - 1) Cirrhosis: (the commonest cause) It is sinusoidal & post-sinusoidal obstruction due to:
 - a. Compression of sinusoids.
 - b. Compression of hepatic veins & venules.
 - c. Development of A-V shunts between branches of hepatic artery & portal vein .



- 2) Bilharzial periportal fibrosis which is presinusoidal obstruction.
- *3) Veno-occlusive disease* (intra-hepatic obstruction of hepatic veins).
- 4) Lymphoma , leukemia and sarcoidosis : Due to infilteration



Bilharzial
periportal fibrosis

III) Supra-hepatic causes: Rare, due to

- 1) Repeated R.H.F, Constrictive pericarditis, pericardial effusion & Tricuspid valve disease.
- 2) **Budd-Chiari syndrome:** thrombosis of hepatic veins at their opening in I.V.C. It may be spontaneous, due to tumour invastion, oral contraceptives, polycythaemia or certain plants.

* Pathology, Complication & C/P:

1. The Liver:

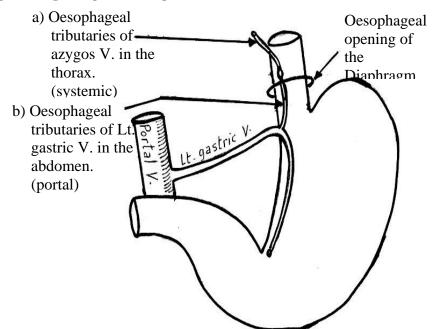
- a. In supra-hepatic causes:
 - **In early** cases → enlarged, soft, and tender.
 - In late cases → cardiac cirrhosis occur → enlarged, tender, firm & sharp edges.

b. In hepatic causes:

- Usually cirrhosis \rightarrow first enlarged then shrunken, firm and sharp edge with nodular surface .
- In periportal fibrosis (according to the stage)
- **c.** *In infra-hepatic causes*: The liver is usually **normal**.
- **2. Splenomegaly** due to congestion , RES hyperplasia & 2ry hypersplenism.

- **Splenic swelling** is felt firm, smooth with rounded edge in the left hypocondrium and it has a notch on its superior border.
- Pain in the left hypochondrium which may be:
 - a. **Dragging** pain & due to traction on the peritoneal ligaments of the spleen.
 - b. **Heavness**: The patient feel his enlarged spleen.
 - c. **Dull** pain due to stretch of splenic capsule.
 - d. **Stitching or stabbing** pain due to stasis → splenic infarction → perisplenitis.
- Hypersplenism → anaemia, repeated infections and bleeding tendency.
- **3.** *Anorexia, dyspepsia, distenion* and abdominal discomfort due to G.I.T congestion, pressure by splenomegaly, associated chronic peptic ulcer or reflex from liver disease.
- **4.** There may *be haematemesis and melaena* usually due to rupture oesophageal varices or less commonly due to bleeding chronic peptic ulcer which is very common in patient with liver disease (due to lack of inactivation of gastrin and histamine) .

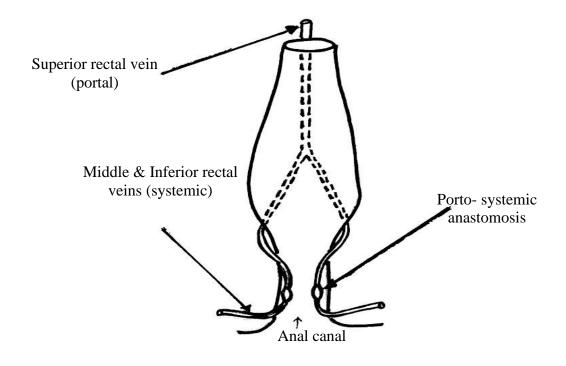
5. Opening of porto-systemic anastomosis:

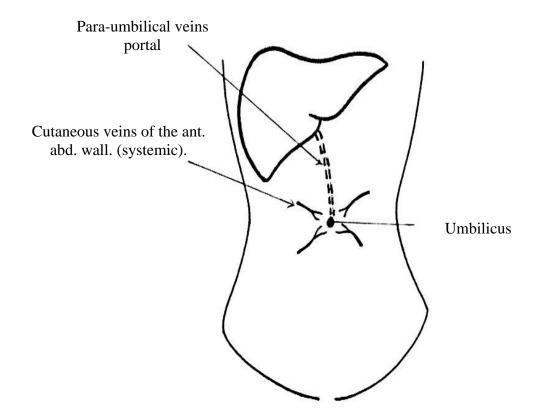


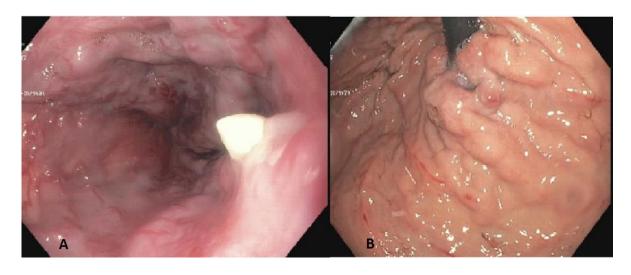
	* Bo	etween			
* Site	* Portal	* Systemic	* Clinical importance		
1. S.m & outer	Oesophageal	Oesophageal	Oesophageal & gastric		
surface of	Veins of left	veins of azygos &	varices which may be:		
lower part of	gastric & short	hemiazygos veins.	Silent: discovered by		
oesophagus.	gastric veins .		investigations.		
& upper part			■ Active i.e bleeding →		
of stomach.			melaena, haematemesis		
			or fresh bleeding per		
			rectum in severe cases.		
2. S.m of	Tributaries of	Tributaries of	■ Ano-rectal varices →		
lower part of	superior	middle & inferior	not dangerous as it is silent		
rectum &	rectal Vein.	rectal Veins.	or minimal fresh bleeding		
upper 1/2 of			per rectum (surrounded by		
anal canal.			sphincter), present early.		
3. Around	Unobliterated	■ Superior &	- Caput medusae (dilated		
umbilicus.	para-umbilical	inferior epigastric	veins around the		
	$\text{veins} \rightarrow \text{left}$	Veins	umbilicus) with blood flow		
	branch of		away from umbilicus with a		
	portal.		continuous thrill on		
			palpation & venous hum on		
			auscultation.		
4. Reto-	Tributaries of	Systemic veins	Silent detected by		
peritoneal.	portal veins	draining posterior	ultrasound or during		
	in the liver,	abdominal wall ,	operation.		
	spleen ,	diaphragmatic &			
	duodenum &	subdiaphragmatic			
	colon .	veins.			

★ N.B.:

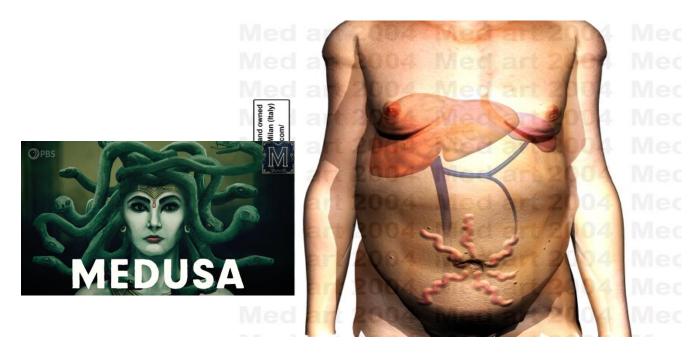
- Oesophageal & gastric varices are dilated, elongated, tortuous veins in the submucosa and peri-oesophageal & perigastric layers.
- These varices are the most serious complication as these may cause fatal bleeding (mortality rate is over 40% with any line of treatment)
- Oesophageal varices are dangerous as -ve intra-thoracic pressure keeps the veins patent.
- Bleeding usually occurs from the submucosa of *lower 5 cm* of the oesophagus (because veins are **not supported**).
- Factors predispose to bleeding varices :
 - 1. High portal hypertension.
 - 2. Large varices with signs of impending rupture.
 - 3. Patient in group C Child's classification.
- Varices are usually silent, it bleed only in 40% of cases due to:
 - 1. **Burst by** sudden increase in portal venous pressure as during coughing.
 - 2. **Reflux oesophagitis** → erosion of the overlying mucosa.
 - 3. Trauma by food.
 - 4. Bleeding tendency.
- The cause of death may be due to:
 - 1. **Shock** \rightarrow **hepato-renal failure** due to hypotension.
 - Amoniacal encephalopathy due to passage of blood to the intestine where it is acted upon by the intestinal bacteria
 → excess ammonia formation.
 - 3. Respiratory complication.







Endoscopic view of oesophageal (A) & gastric (B) varices

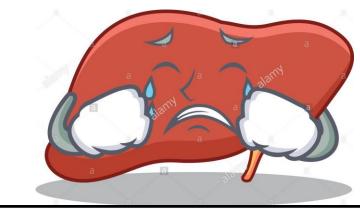


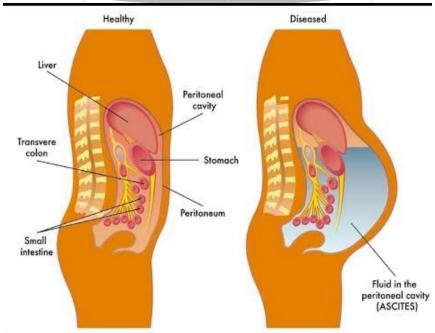
Caput medusae



Proctoscopic view of anorectal varices

- **6.** *Ascites:* It indicates a marked degree of **liver dysfunction**. It is due to:
 - a. *Hypoalbuminaemia* (below 3 gm/100ml) *is the most important cause* due to liver dysfunction.
 - b. *Portal hypertension:* alone **never cause ascites** but it is a localizing factor to the filteration of fluid into the peritoneal cavity .
 - c. Failure of the liver to *inactivate* aldosterone and A.D.H.
 - d. **Lymphorrhea:** Intrahepatic congestion & lymphatic obstruction \rightarrow transudation of fluid on the surface of the liver .





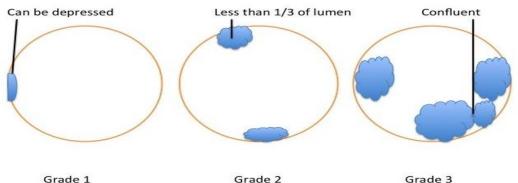


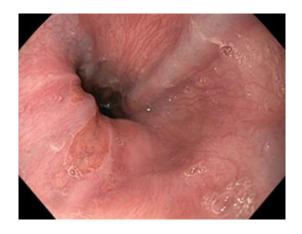
7. Manifestations of *hepatocellular insufficiency in late cases :* (See medicine)

- Spider naevi , palmar erythema , testicular atrophy ,gynaecomastia , bleeding tendencyetc .
- jaundice in late cases and it is a bad prognostic sign .
- Ascites in late cases .
- * **Investigations:** The **aim** of investigations are:
 - I) Assessment of Liver functions by liver function tests. (Mention)
 - Prothrombin time & concentration are the most sensitive liver function.

II) Detection of oesophageal varices by:

- 1) Upper GIT endoscopy:
 - It is the most important investigation to diagnose esophageal
 and gastric varices .
 - Endoscopic grading of esophageal varices:
 - Grade 1: Small, straight esophageal varices.
 - Grade 2: moderate, dilated, tortuous esophageal varices occupying less than one third of the lumen.
 - Grade 3: Large, coil-shaped esophageal varices occupying more than one third of the lumen.
 - Grade 4: esophageal varices with high risk of bleeding as indicated by red sign (red patches on the varices) or varices over varices (Small varices lying on larger ones)







Esophageal varices

Grade 1

Esophageal varices

Grade 2

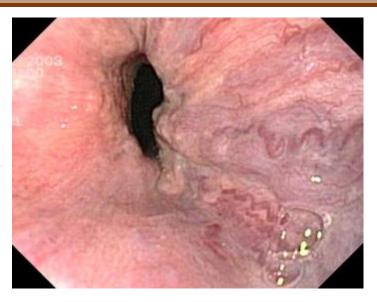
Esophageal varices
Grade 3



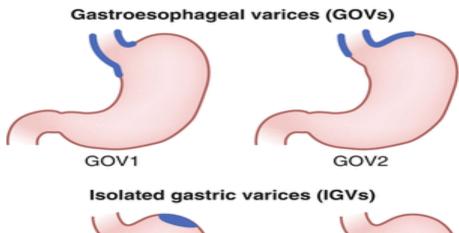
Esophageal varices
With red sign

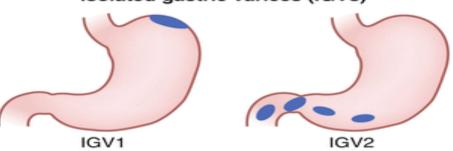


Esophageal varices with varices over varices



- Endoscopic classification of gastro-esphageal varices :
 - **Gastro-esphageal varices type I**: (75%) esphageal varices extending to the lesser curvature.
 - **Gastro-esphageal varices type II**: (the largest) esphageal varices extending to the fundus of stomach.
- Endoscopic classification of isolated gastric varices :(rare)
 - Isolated gastric varices type I : gastric varices in the fundus
 - **Isolated gastric varices type II** : (most rare)gastric varices in the body , antrum or prepyloric .









3)Duplex scanning: Show dilated portal vein & dilated venous collaterals.

III) Detection of hypersplenism:

- 1)Blood picture will reveal anaemia, leucopenia, thrombocytopenia or pancytopenia.
- **2)Bone marrow examination** may reveal hypercellularity & exclude leukaemia as a cause of hepatosplenomegally.
- **3)Radioactive chromium tagged patient own RBCs** will show diminish half life of RBCs & increased radioactivity over the spleen.

IV) Diagnosis of the etiology of liver disease by:

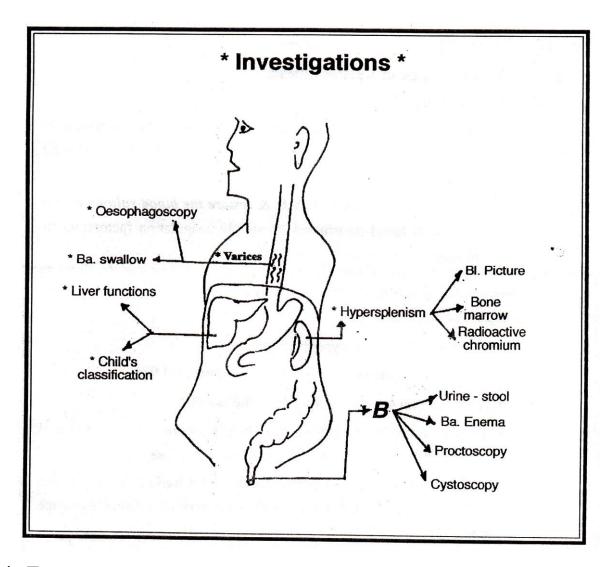
- 1) Immunological tests for hepatitis markers.
- **2) Liver biopsy** which may be preoperative needle biopsy of operative wedge biopsy (after assessment of prothrombin time & concentration).

3) Ultrasound.

- V) Other important investigations in Egypt :
 - 1) Urine and stool analysis: for Bilharzial ova.
 - 2) Ba. enema: may show Bilharzial polypi as multiple smooth filling defects.
 - 3) **Sigmoidoscopy:** May show anorectal varices, Bilharzial polypi & to obtain rectal biopsy (sure diagnosis of Bilharziasis).
 - **5) Cystoscopy** to detect urinary Bilharziasis.
- **VI)** To have an idea about the parenchymatous functions of the liver, the parameters of *Child's classification* can provide a useful idea.

		Class A (1 point) 5-8	Class B (2 point) 7 - 9		Class C (3 point) 10 - 15
1. Serum bilirubin mg%	•	Blelow 2	■ 2 – 3	•	above 3
2. Serum albumin gm %	•	above 3.5	■ 2.8 – 3.5	•	below 2.8
3. Ascites	•	No	Mild (respond to diuretics)	•	Severe ot respond to uretics)
4. Neurological signs	•	No	minimal	•	coma
5.Prothrombin time (sec prolonged)	•	Less than 4	4 -6	•	More than 6

- Patient in class A (mild liver disease) is fit for major surgery
- Patient in class B (moderate liver disease) .
- Patient in class C (severe liver disease) is unfit for major surgery.



* Treatment:

 Endoscopic screening for varices should be done for all cirrhotic patients.

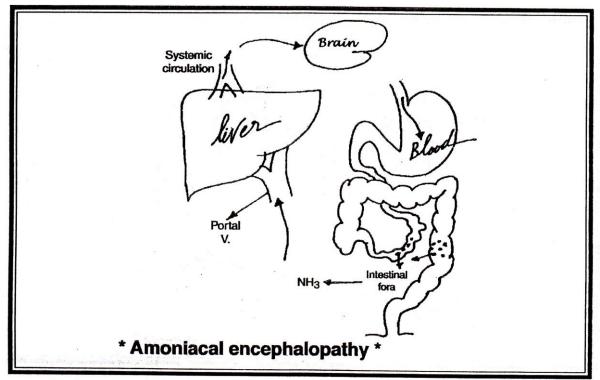
A. If there are no varices: Only medical treatment

- a) Avoid alcohol or hepatotoxic drugs.
- b) **Liver tonics:** Vitamin K and B complex, excess carbohydrate diet.
- c) Omeprazol to avoid hyperacidity & reflux esophagitis .
- c) Follow up by upper GIT endoscopy after 3 years .

- **B.** If there are varices: Depends on silent varices or active varices.
 - 1) Silent varices: (never bleeds before).
 - a) Medical treatment: (as before) +
 - b) Beta blockers eg. Propranolol or nadolol
 - c) If small silent varices, follow up by upper GIT endoscopy after2 year.
 - d) **Prophylactic banding:** To avoid haematemesis, only recommended for **large** varices or **impeding** rupture (red sign or varices over varices) i.e. Grade III or IV.
- **2)** *Active varices* : (bleeding varices)
 - I) During the attack of haematemesis: (surgical emergency)
 - 4. Antishock measures:
 - Admission, rest in bed, warmth, clear airway, O₂ inhalation, and sedation by valium (never morphia or pethidine as it is metabolised in the liver), insert folley's catheter.
 - Insert a wide bore canula or C.V.P. & blood sample is taken for complete blood picture, liver function tests, coagulation study and cross matching for at least 4 unites of fresh blood.
 - **Restore the blood volume** by I.V fluid & fresh blood transfusion (to supply coagulation factors and to avoid high amonia content in stored blood).

5. Prevention of amoniacal encephalopathy:

- Ryle tube: To aspirate gastric contents hourly → prevent propagation of blood to the gut & to observe the progress of the case.
- Repeated **enema** to remove blood from the colon.

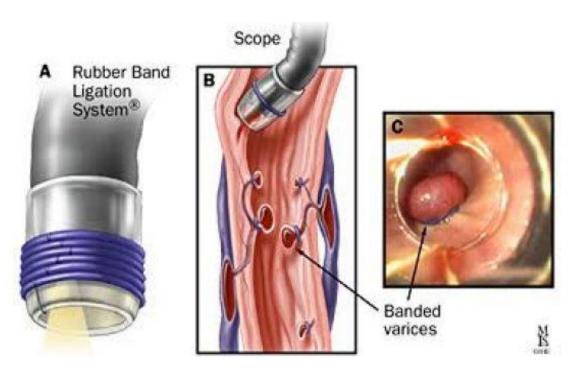


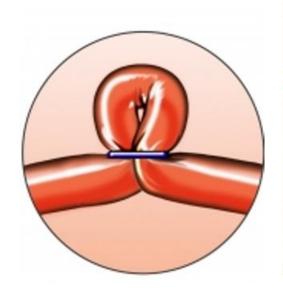
- Lactulose: split into lactose (cause diarrhea) and lactic acid (acidify the colon → inhibit bacterial flora and combines with ammonia).
- **Intestinal antiseptic** as neormycin 500mg/4 hours to reduce intestinal flora.
- **6.** Correct coagulopathy by **Vitamin K I.V and fresh frozen plasma**.
- 7. *I.V. omeprazol* to reduce gastric acidity.
- 8. Observation: Vital signs, urine output, C.V.P. and amount of bleeding.
- 9. Measures to stop bleeding:
 - a) Once the patient is resuscitated Urgent upper GITEndoscopy under diazepam :
 - Diagnostic: to confirm the diagnosis and to exclude other causes of haematemesis.

• Therapeutic :

1) Endoscopic rubber band ligation:

- > This method is **now the 1**st. **choice** for control of bleeding varices .
- The **best, simplest and safest** way for emergency control of bleeding varices (90%).
- **▶ Encircle** each varix by tight band → thrombosis.
- > It is as effective **as sclerotherapy** in the control of bleeding.





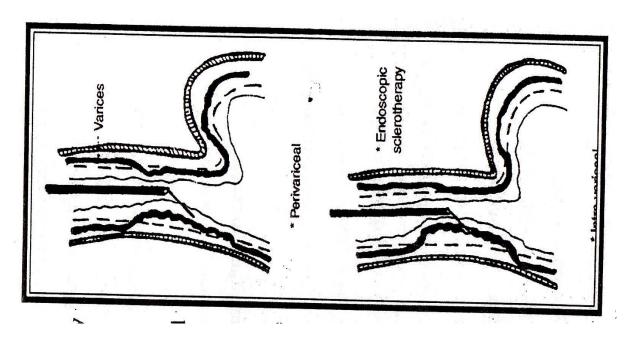


2) Endoscopic sclerotherapy:

➤ It is a **simple and safe** way for emergency control of bleeding varices (90 %).

> Methods:

- ♣ Oesophago-gastric **endoscopy** is performed under diazepam.
- ♣ There are **3 methods** for injection of injection of varices:
 - a. **Inravariceal** injection of 5 ml ethanolamine oleate into each esophageal varices , near the gastro-esophageal junction , to induce thrombosis inside the varices.
 - ◆ In gastric varices , histoacryl (solidifies after contact with blood) is injected .
 - **b.Perivariceal** injection of 0.5 ml aethoxysclerol , at multiple sites alongside of each varix , to induce fibrosis around varices.
 - c. Both methods can be combined.
- ♣ Injection must be **repeated at 2 weekly** sessions until the varices are obliterated .
- > **Comlications:** (rare) retnoternal discomfort , oesophageal ulceration, stricture , perforation, mediastinitis & fever.



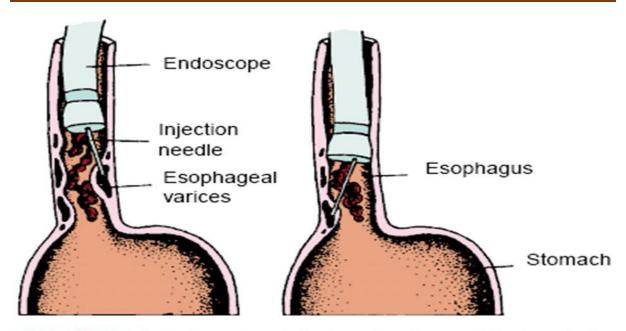


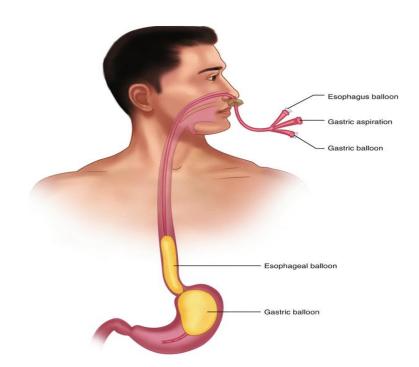
FIGURE 39-8 Endoscopic or injection sclerotherapy. Injection of sclerosing agent into esophageal varices through an endoscope promotes thrombosis and eventual sclerosis, thereby obliterating the varices.

- 3) **Drugs:** Vasoconstriction of visceral arterioles →Reduce splanchnic blood flow → reduce portal venous pressure → reduce variceal blood flow → reduce intravariceal pressure
 - Indication: It is only a temporary measure until endoscopy is performed.
 - Method:
 - **a) Vasopressin:** 0.2 unit/kg in 200 c.c of 5% dextrose over 20 minutes.
 - > Recently, **glypressin** is used as it is long acting and has fewer side effects.
 - ➤ It is **contraindicated** in elderly ,hypertension , ischaemic heart disease and pregnancy .
 - **b) Somatostatin:** (expensive)

4)Ballon tamponade by Sengestaken-Blakemore Tube:

- Indication: If bleeding is sever and interfere with endoscopic treatment, balloon tamponade or drugs are only used as a temporary measure while arranging for endoscopic treatment or emergency TIPSS.
- Composition: It is 4 lumenal tube:
 - One channel for gastric aspiration or feeding.
 - ➤ One channel for inflation of gastric balloon first with air (200ml), and pulled upwards to press the gastric fundus.

 If bleeding continues, inflate the esophageal balloon.
 - One channel for inflation of oesophageal balloon with air (pressure should not exceed 40 mmHg).
 - > A 4th. channel for aspiration of saliva.



Action: Compression of the bleeding varices.

Duration of application: Maximum time 48 hours then
 deflate the balloons in the endoscopy unite.

Complications:

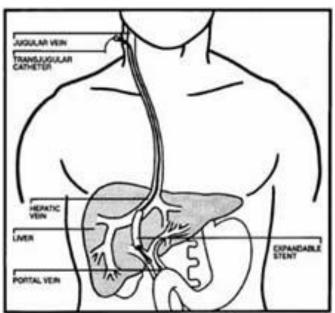
- > Discomfort to the patient .
- ➤ Ulceration in the oesophagus, pharynx, nostril.
- > Recurrence of bleeding after balloon deflation (60%).
- ➤ Aspiration pneumonia, lung bscess or asphyxia.
- **5)** Transjuglar intrahepatic **p**orto-**s**ystemic **s**hunt (TIPSS) .
 - Indication: if all previous measures fails to stop bleeding.

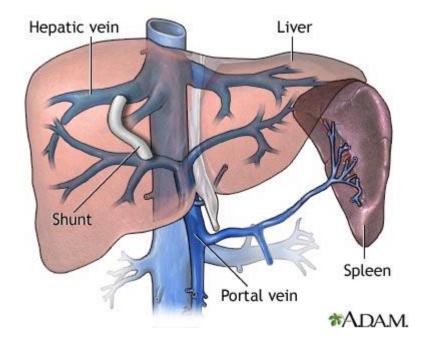
Method:

- > Under fluoroscopic control a guide wire is passed from internal jugular vein to small hepatic vein inside the liver .
- ➤ The wire is then passed through the liver to reach a branch of portal vein .
- > A stent is passed to creat a shunt between the portal branch and hepatic vein .

Complications :

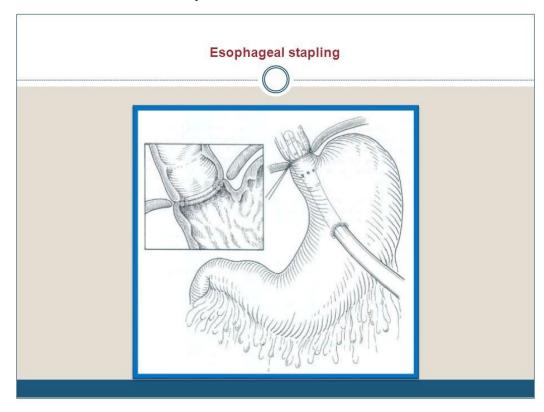
- > Perforation of liver capsule leading to fatal haemorrhage .
- > Encephalopathy in 40% of patients .
- > Shunt occlusion in 50% of patients after one year .





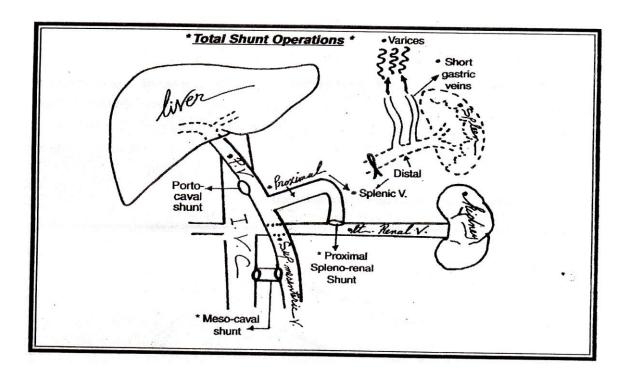
6) Surgical Treatment:

- Very very rarely, if all the previous measures fail to stop bleeding, surgery by oesphageal stapling may be performed with high mortality provided the patient is fit for surgery (actually this is rare).
 - Contraindications: Patients unfit surgery (class C child's classification).



- **II-** *In between the attacks of haematemesis:* (Measures to prevent recurrence)
 - 1) The most accepted treatment for oesophageal varices nowadays is repeated upper GIT endoscopy with band ligation or sclerotherapy until varices are obliterated.
 - 2) *Medical treatment by beta adrenergic blockers:* Decrease the incidence of recurrent bleeding.
 - 3) Surgery: (very rarely needed in modern surgery & nowadays portal hypertension is considered as a medical problem)
 - Indication: If band ligation & sclerotherapy fail to stop recurrent attacks of bleeding provided that patient is fit for surgery.
 - Methods: One of the followings may be done:
 - A) Porto-systemic shunt operations:
 - Idea: shunting portal blood to systemic circulation, away from the liver, to by pass the obstruction → lower portal venous pressure with collapse of varices and bleeding stop.
 - These may be:
 - 1. **Total shunt operations:** (not done in modern surgery).
 - a. Porto-caval shunt: Anastomosing the portal vein to the I.V.C either end to side or side to side.
 - Advantages: decrease the portal pressure markedly
 → prevent bleeding.
 - Disadvantages:
 - 1. Marked deterioration of liver.
 - 2. Encephalopathy is common.

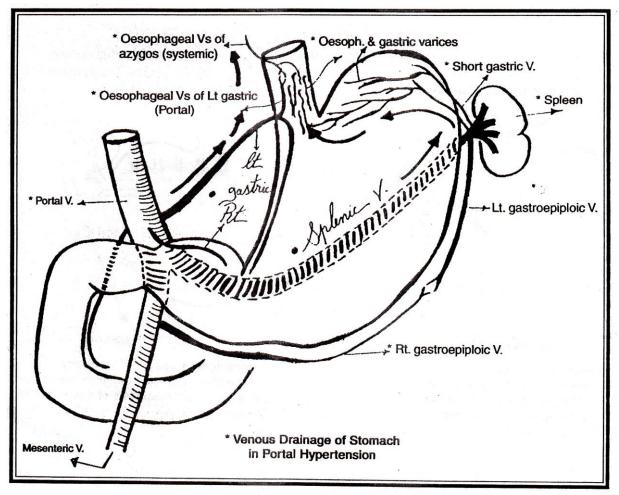
- **b. Proximal spleno-renal shunt:** Splenectomy is done then anastomosing the proximal part of splenic vein to the left renal vein.
 - •Advantage: Deterioration in the liver function and encephalopathy are rare.
 - Disadvantages: Decrease in the portal pressure is not marked → less effective to prevent bleeding from varices.
 - •Indication: Thrombosis of portal vein or hypersplenism.
 - •Contraindication: If the splenic vein is less than 1 cm in diameter.
- c. Meso-caval shunt: A graft is interposed between the superior mesenteric vein & I.V.C.
 - **Disadvantage:** The incidence of thrombosis is high.

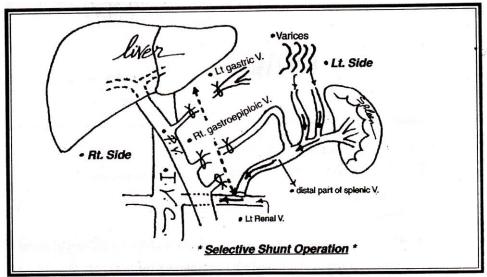


2. Selective shunt operation: *Includes a distal spleno-renal* shunt (Warren's operation), the best, anastomosing the

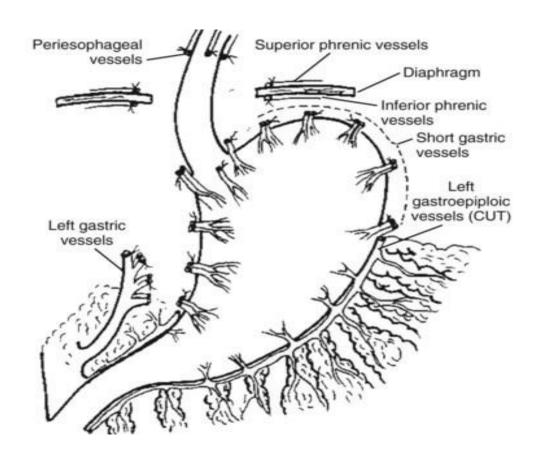
distal end of splenic vein, to the left renal vein without splenectomy with ligation of Lt. & Rt. gastric veins, right gastroepiploic vein and any vein crosses between right and left side of the portal circulation \rightarrow **Selective decompression of the varices.**

Advantage: liver functions are not affected.



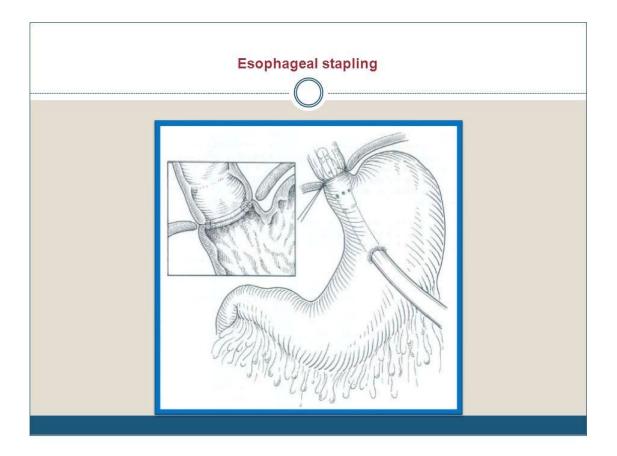


- **B)Porto-systemic disconnection operations:** (devascularization and transection operations).
 - **Idea**: These operations aim at disconnection the portal system in the upper part of the stomach with its high pressure from the systemic circulation at the lower end of oesophagus.
 - The **most popular** operations are:
 - **1.** *Hassab's operation:* Splenectomy with extensive devascularisation of the stomach leaving only the right gastroepiploic vessels + devasculariseation of the abdominal part of the aesophagus.



2. Oesophageal stapling:

- It is the most popular operation nowadays if surgery is performed
- A stapling instrument is introduced into the lower oesoph. through a small hole in the anterior wall of the stomach. The instrument simultaneously divides the oesoph. and joins it with staples which occlude the vessels.



C) Liver transplantation:

- Indication: end stage liver disease.
- **Idea**: treatment of the cause of portal hypertension by removal of the cause of the obstruction of portal blood flow.