Ministry of Higher Education and Scientific Research

AL Mustansiriyah University







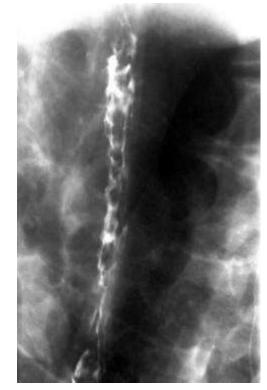
PORTAL HYPERTENSION



Portogram shows opacification of gastroesophageal varices.

http://clinicalgate.com/radiologic-hepatobiliary-interventions/

الدكتور ذو الفقار حسن بيعي

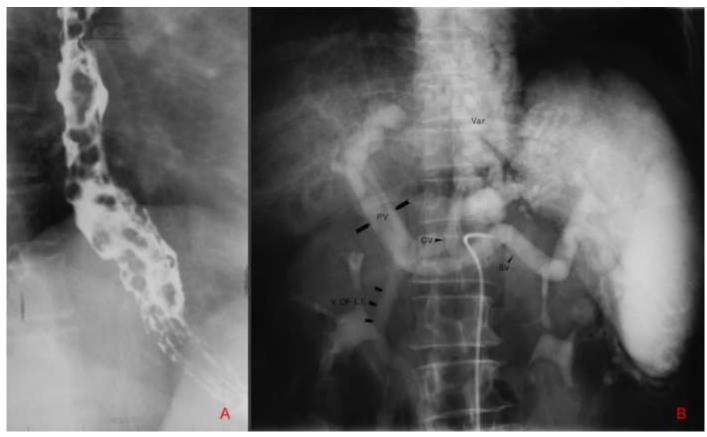


courtesyhttp://emedicine.medscape.com/article/372708-overview



PORTAL HYPERTENSION

الدكتور ذو الفقار حسن بيعي



Courtesy https://www.med-ed.virginia.edu/courses/rad/gi/esophagus/struct03.html



Objectives:

The causes of portal hypertension.

#The management of : Portal hypertension. Acute bleeding varices.(behavior)

#The prevention of the hepatic encephalopathy.(skill)

Liver blood supply

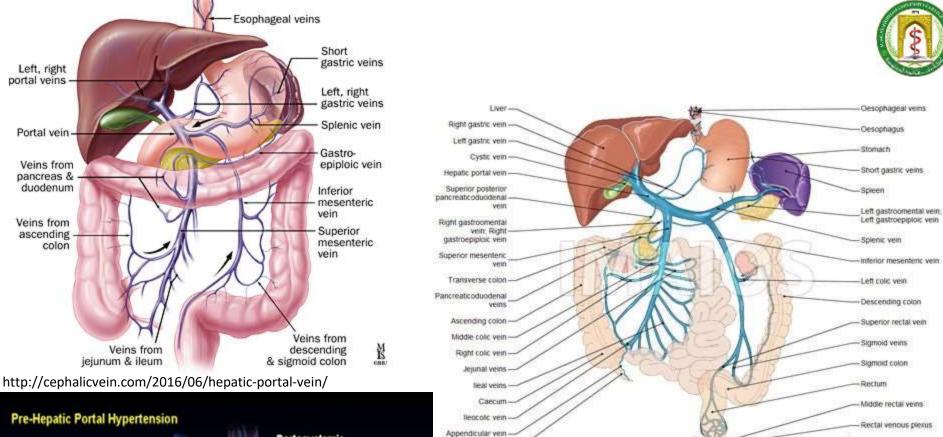


The blood supply to the liver is unique, 80 per cent being derived from the portal vein and 20 per cent from the hepatic artery.

Liver blood supply

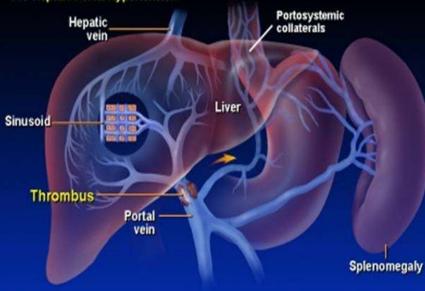


The portal vein arises from the confluence of the splenic vein and the superior mesenteric vein behind the neck of the pancreas. It has some important tributaries, including the left gastric vein which joins just above the pancreas.



Appendix

Russacons : A Moheau - ND



https://www.gastroslides.org/main/browse_deck.asp?tpc=14&mxpg=414&pg=5050

Interior rectal veins

Liver blood supply

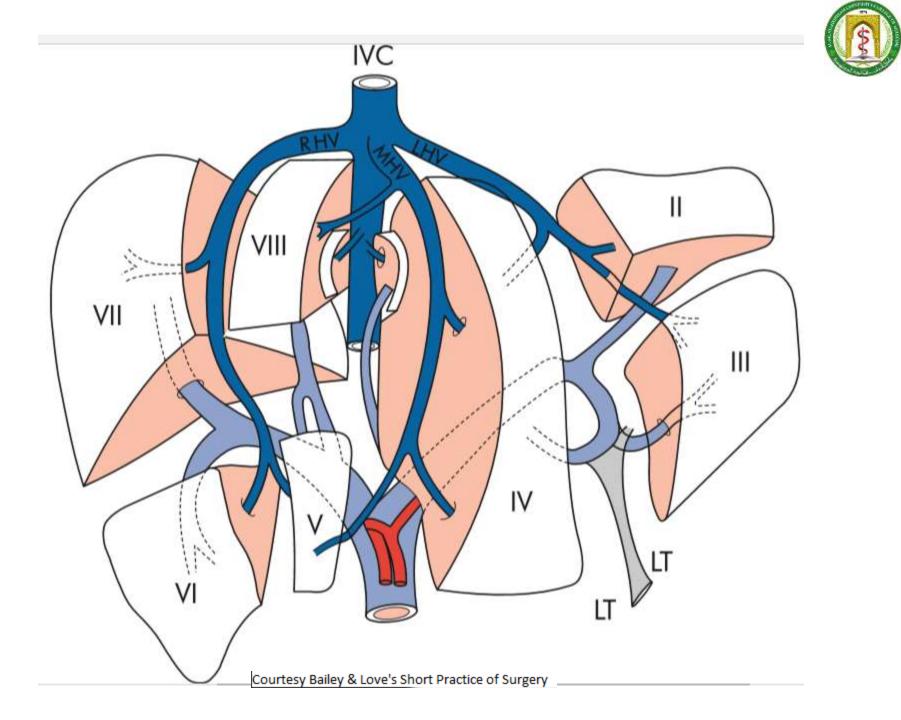


The portal vein often gives off two large branches to the right lobe, which are usually outside the liver for a short length, before giving a left portal vein branch that runs behind the left hepatic duct.

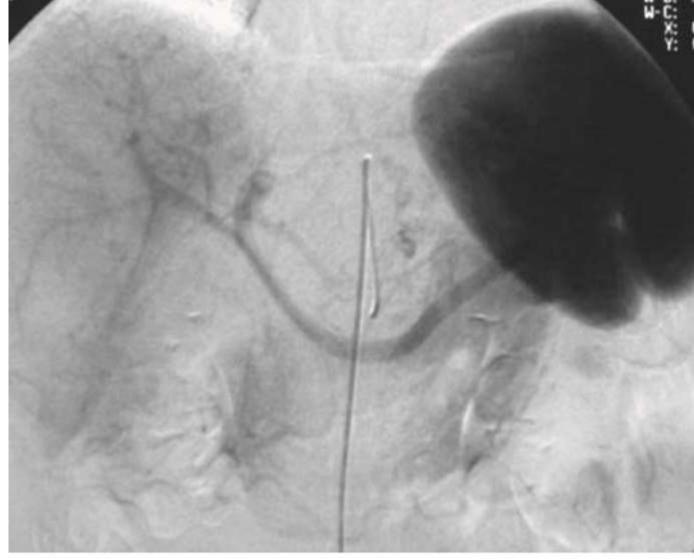
Liver blood supply



The portal vein forms from the superior mesenteric and splenic veins, and divides into right and left branches in the hilum of the liver.







Courtesy Bailey & Love's Short Practice of Surgery



Main functions of the liver

Maintaining core body temperature pH balance and correction of lactic acidosis Synthesis of clotting factors Glucose metabolism, glycolysis and gluconeogenesis Urea formation from protein catabolism Bilirubin formation from haemoglobin degradation Drug and hormone metabolism and excretion

Routinely available tests of liver function.

Test	Normal range
Bilirubin	5–17 µmol/L
Alkaline phosphatase (ALP)	35–130 IU/L
Aspartate transaminase (AST)	5–40 IU/L
Alanine transaminase (ALT) Gamma-glutamyl transpeptidase (GGT)	5–40 IU/L 10–48 IU/L
Albumin	35–50 g/L
Prothrombin time (PT)	12-16 s





Courtesy Bailey & Love's Short Practice of Surgery



A patient with end? stage liver cirrhosis disease, demonstrating muscle wasting and gross abdominal distension due to ascites.



Lethargy Fever Jaundice Protein catabolism (wasting) Coagulopathy (bruising) Cardiac (hyperdynamic circulation) Neurological (hepatic encephalopathy) Portal hypertension Ascites Oesophageal varices Splenomegaly Cutaneous Spider naevi Palmar erythema



Features of chronic liver disease.

Lethargy Fever Jaundice Protein catabolism (wasting) Coagulopathy (bruising) Cardiac (hyperdynamic circulation) Neurological (hepatic encephalopathy) Portal hypertension Ascites **Oesophageal** varices Splenomegaly Cutaneous Spider naevi Palmar erythema



Stigmata of liver disease

White nails Dupuytren's contractures Liver palms Liver flap Spider naevi Gynaecomastia Loss of axillary and pubic hair Foetor hepaticus Ascites Caput medusa



Two prognostic models used to assess the severity of chronic liver disease and perioperative risk.



The Child– Turcotte–Pugh (CTP) Classification

The Model for EndStage Liver Disease (MELD) score.



The Child– Turcotte–Pugh (CTP) classification has been modified from the original Child classification which was developed to predict mortality following shunt surgery in cirrhotic patients.



Points	1 point each	2 points each	3 points each
Bilirubin (µmol/L)	<34	34–50	>50
Albumin (g/L)	>35	25-35	<25
Ascites	None	Easily controlled	Poorly controlled
Encephalopathy	None	Grade I–II	Grade III–IV
INR	<1.7	1.7-2.2	>2.2

CTP A = 5–6 points; CTP B = 7–9 points; CTP C = 10-15 points. INR, international normalised ratio.



Child–Turcotte–Pugh (CTP) classification of hepatocellular function in cirrhosis.

Points	1 point each	2 points each	3 points each
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Albumin (g/L)	>35	25-35	<25
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The Model for EndStage Liver Disease (MELD) score was originally developed to predict short-term prognosis in patients undergoing transjugular intrahepatic portosystemic shunt insertion, but has now been adopted to prioritise patients awaiting liver transplantation.

The survival probability of patients with end-stage liver disease is computed based on the patient's international normalised ratio (INR), serum bilirubin and serum creatinine.

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IMAGING THE LIVER



Doppler ultrasound allows flow in the hepatic artery, portal vein and hepatic veins to be assessed.

The venous phase of the intravenous contrast (CT)maps the branches of the portal vein within the liver and the drainage via the hepatic veins.

A liver biopsy to confirm or exclude chronic liver disease can also be performed during laparoscopy.



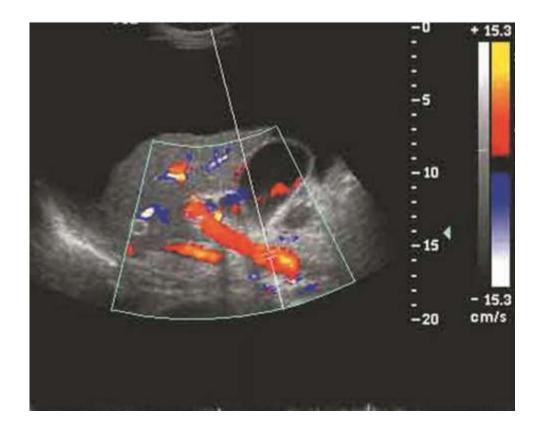




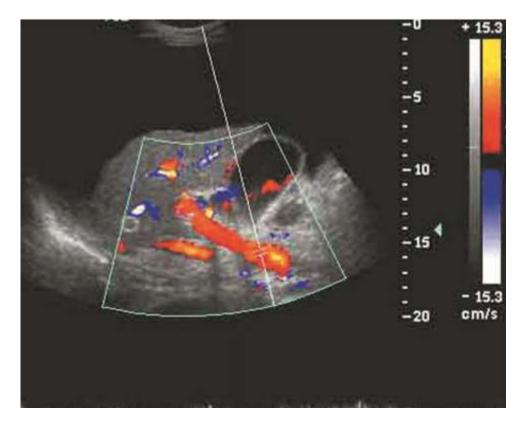


Sagittal ultrasound image of the liver in a patient with cirrhosis demonstrates nodularity of the liver surface and extensive ascites.









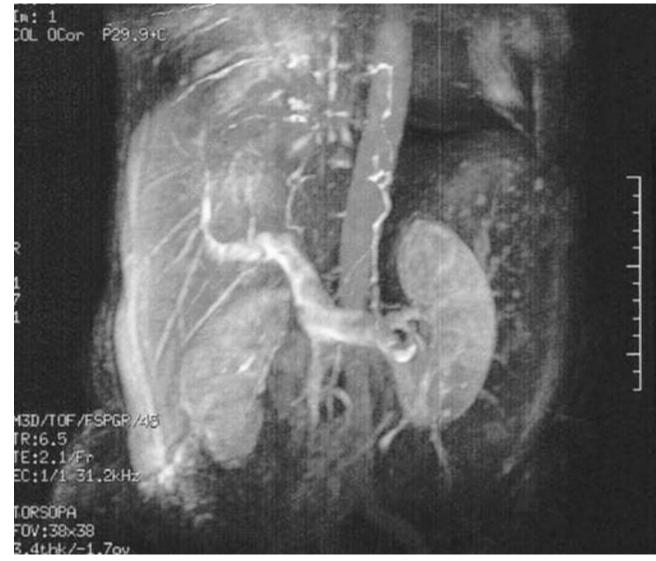
Doppler ultrasound illustrates portal vein flow with a normal direction.

IMAGING THE LIVER



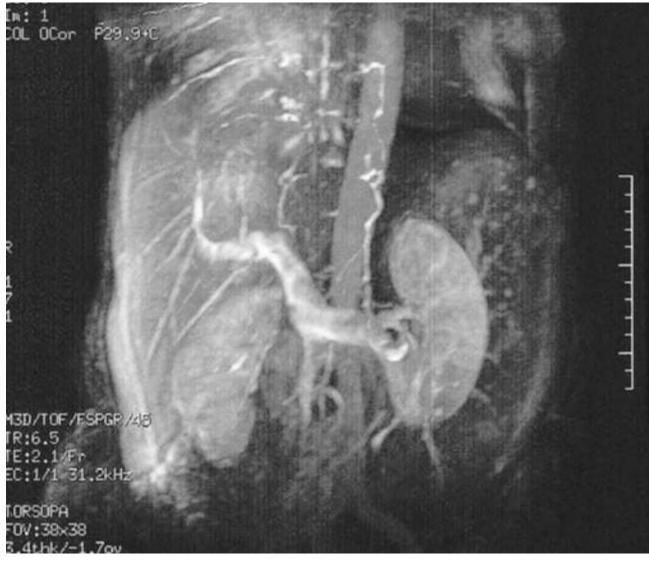
- Magnetic resonance angiography (MRA) provides high-quality images of the hepatic artery and portal vein, without the need for arterial cannulation.
- It is used as an alternative to selective hepatic angiography for diagnosis.
- It is particularly useful in patients with chronic liver disease and coagulopathy in whom the patency of the portal vein and its branches is in question.







Magnetic resonance imaging (MRI) scan showing massive hepatosplenomegaly. Note the prominent portal system and the left kidney, which is superimposed over the grossly enlarged spleen.





INTERNATIONAL STUDENTS' EDITION

Bailey & Love's

CHAPTER 6



PORTAL HYPERTENSION

- An elevation in portal pressure.
- It is most commonly found in the presence of liver cirrhosis. It may be present in patients with
- Extrahepatic portal vein occlusion,
- Intrahepatic veno-occlusive disease
- Occlusion of the main hepatic veins (Budd-Chiari syndrome (BCS)).

PORTAL HYPERTENSION



Another classification of causes:

Outside the portal vein. Inside the portal vein. In the wall of portal vein.



Causes of portal hypertension

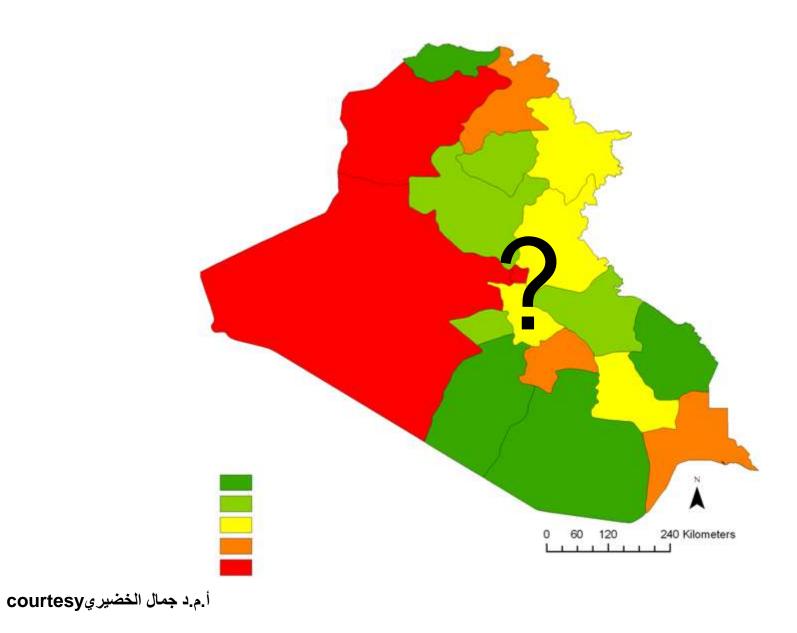
Presinuoidal

Extrahepatic Portal vein thrombosis or atresia Splenic vein thrombosis Portal arteriovenous fistula Pancreatitis Hypersplenism Intrahepatic Schistosomiasis Cirrhosis

Postsinusoidal

Budd–Chiari syndrome Congestive heart failure/pericarditis Veno-occlusive disease





PORTAL HYPERTENSION

Portal hypertension produces no symptoms, it is usually diagnosed following presentation with decompensated chronic liver disease and encephalopathy, ascites or variceal bleeding.

Varices usually present with the acute onset of a large-volume haematemesis.

The lower oesophagus is the most common site for variceal bleeding.

The diagnosis may be suspected if the patient is known to have liver cirrhosis, but it needs to be confirmed following initial resuscitation (upper gastrointestinal endoscopy)of the patient.

Management of bleeding varices General resuscitation

Variceal haemorrhage is an emergency.

Patients with massive haemorrhage should be admitted to the intensive treatment unit (ITU).

Venous access should be obtained through two large bore peripheral cannulae.

Colloids should be administered while adequate blood is obtained (initially ten units).

Blood volume should be replaced with colloids, plasma expanders and blood transfusions.

Liver function tests will reveal underlying liver disease, and a coagulation profile will reveal any underlying coagulopathy.

Hypervolaemia should be avoided since this may increase portal pressure and exacerbate the bleeding.

Vitamin K is administered (10 mg intravenously), but correction of a coagulopathy will require the administration of fresh-frozen plasma (FFP).

An associated thrombocytopenia is usually secondary to hypersplenism due to cirrhosis and is treated if the platelet count falls below $50 \times 109/L$.

Treatment with a splanchnic vasoconstrictor should be started.

Administration of a prophylactic antibiotic is recommended to prevent or treat associated bacterial infection.

As soon as the patient is haemodynamically stabilised an upper gastrointestinal endoscopy should be performed to establish the diagnosis because 50 % of patients with portal hypertension will have a nonvariceal source of bleeding.

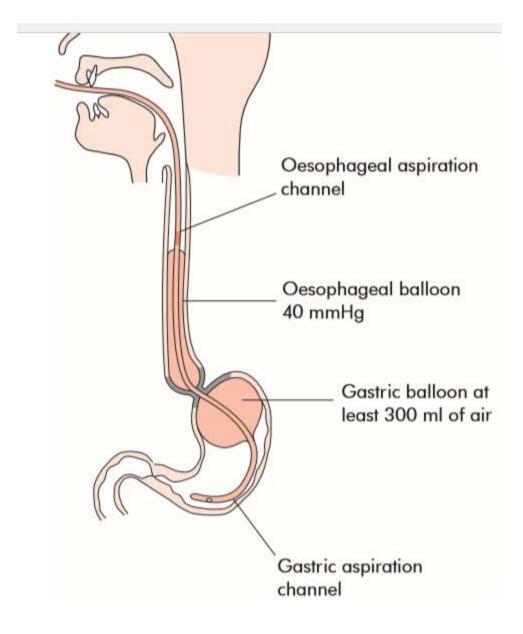
Variceal bleeding is often associated with hepatic encephalopathy and, in these circumstances and when bleeding is severe, endotracheal intubation will be required for endoscopy.

Bronchial aspiration is a frequent complication of variceal bleeding

If the rate of blood loss prohibits endoscopic evaluation, a Sengstaken–Blakemore tube may be inserted to provide temporary haemostasis.

Balloon tamponade is very effective in stopping bleeding and once the patient is stabilised, a more definitive treatment can be carried out.

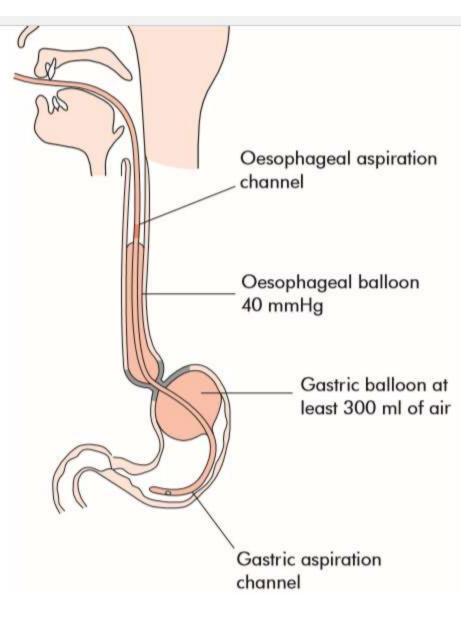




Courtesy Bailey & Love's Short Practice of Surgery

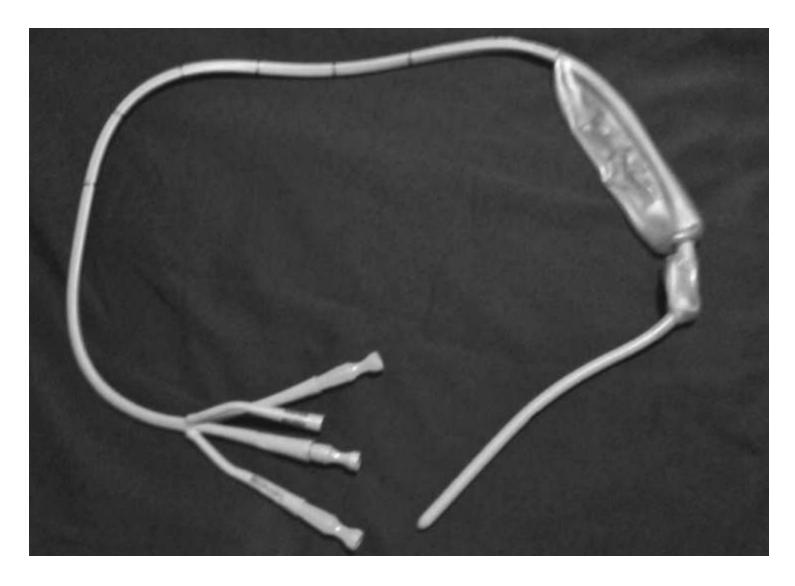


Oesophageal balloon tamponade.

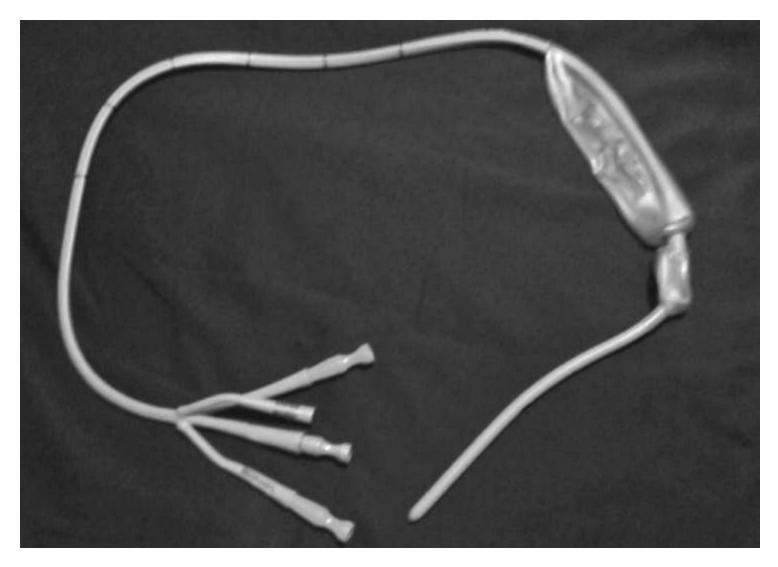


Courtesy Bailey & Love's Short Practice of Surgery



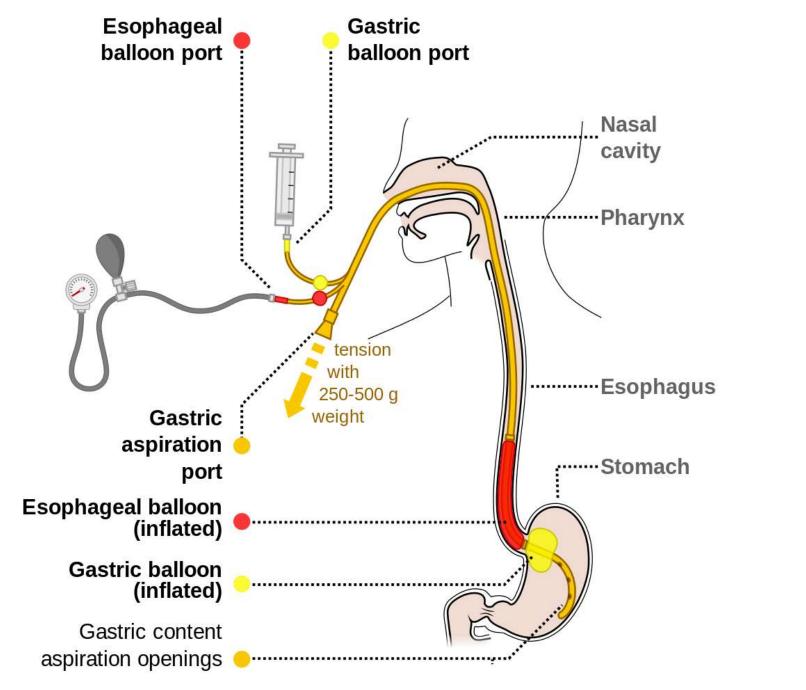






Sengstaken-Blakemore tube

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https://en.wikipedia.org/wiki/Sengstaken%E2%80%93Blakemore_tube

Once inserted, the gastric balloon is inflated with 300 mL of air and retracted to the gastric fundus, where the varices at the oesophagogastric junction are tamponaded by the subsequent inflation of the oesophageal balloon to a pressure of 40 mmHg.

The two remaining channels allow gastric and oesophageal aspiration.

An x-ray is used to confirm the position of the tube.

The balloons should be temporarily deflated after 12 hours to prevent pressure necrosis of the oesophagus.

Aspiration pneumonia and oesophageal ulceration are other complications.

Drug treatment

Splanchnic vasoconstrictors reduce portal flow and pressure and should be started early in the treatment of variceal haemorrhage.

Vasopressin is a potent vasocontrictor and has been the most extensively used drug for the initial control of variceal haemorrhage, but it can cause myocardial ischaemia, arrhythmias, mesenteric and limb ischaemia.

Terlipressin, somatostatin and octreotide are safer than vasopressin and equally effective.

Endoscopic treatment

Treatment with a vasoconstrictor combined with endoscopic therapy is the standard medical treatment for acute variceal bleeding.

The majority of variceal bleeds will respond to a single course.





Courtesy Bailey & Love's Short Practice of Surgery

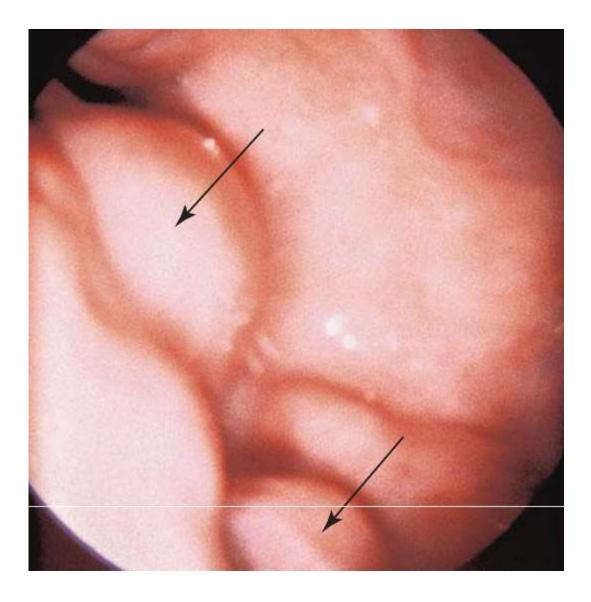




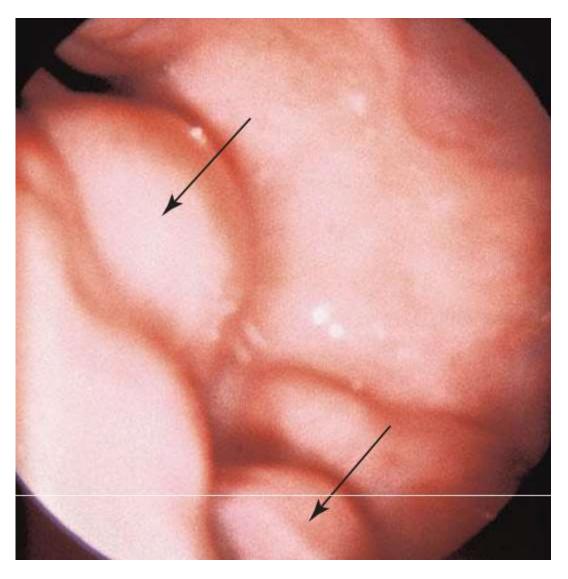
Grade 2 oesophageal varices

Courtesy Bailey & Love's Short Practice of Surgery









Endoscopy demonstrating Oesophageal varices

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Endoscopic treatment

The two most commonly used endoscopic techniques are

Endoscopic band ligation

which involves placing a constricting rubber band at the base of the varix, Banding is significantly better in preventing rebleeding and is the preferred option.

Endoscopic sclerotherapy

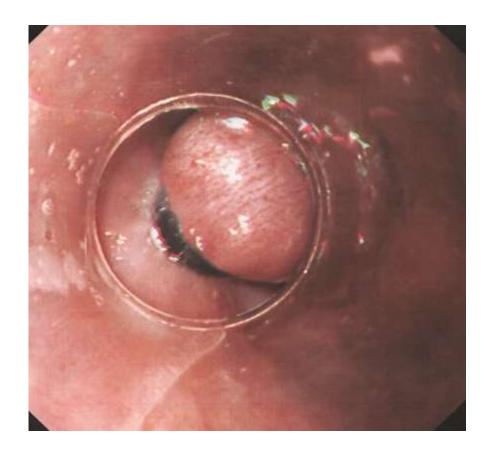
which involves injection of a sclerosant, such as polidocanol (1–3%) Or ethanolamine (5%), into or around the varix. Both are effective in controlling the bleed.





Courtesy Bailey & Love's Short Practice of Surgery



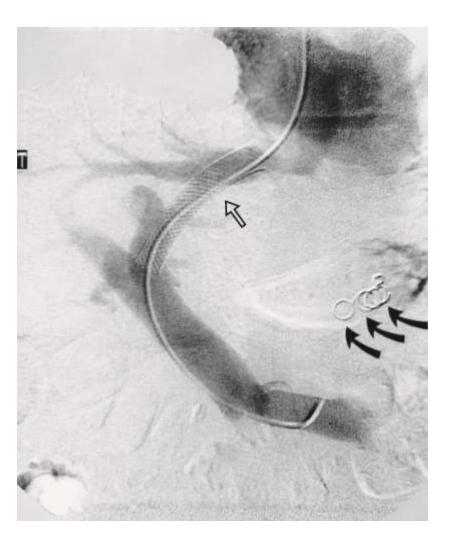


The application of bands to ligate the vessel and reduce blood flow

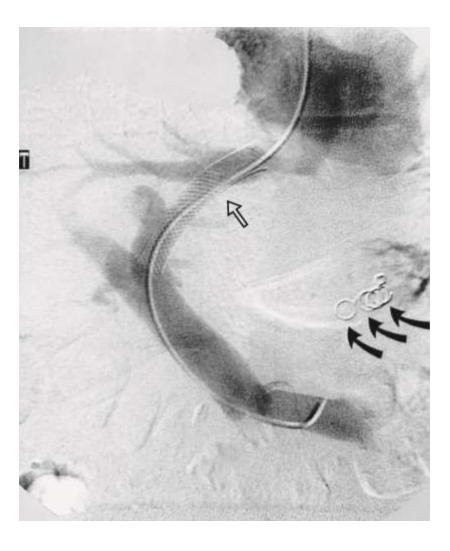
Transjugular intrahepatic portosystemic stent shunts **(TIPSS)**

The emergency management of variceal haemorrhage has been revolutionised by the introduction of transjugular intrahepatic portosystemic stent shunts (TIPSS) in 1988.









The angiogram following insertion of a transjugular intrahepatic portosystemic stent shunt (open arrow). Injection of contrast into the portal vein flows through the metallic stent and outlines the right hepatic vein. Pressure measurements are taken from within the portal vein before and after insertion.

It **(TIPSS)** has become the main treatment of variceal haemorrhage that has not responded to drug treatment and endoscopic therapy.

The shunts are inserted under local anaesthetic, analgesia and sedation using fluoroscopic guidance and ultrasonography.

Via the internal jugular vein and SVC, a guidewire is inserted into a hepatic vein and through the hepatic parenchyma into a branch of the portal vein.

Management of bleeding varices (TIPSS)

The track through the parenchyma is then dilated with a balloon catheter to allow insertion of a metallic stent, which is expanded once a satisfactory position is achieved to form a channel between systemic and portal venous systems.

A satisfactory drop in portal venous pressure is usually associated with good control of the variceal haemorrhage.

(TIPSS)

- The main early complication of this technique is perforation of the liver capsule, which can be associated with fatal intraperitoneal haemorrhage.
- Occlusion may result in further variceal haemorrhage and occurs more commonly in patients with wellcompensated liver disease and good synthetic function.
- Post-shunt encephalopathy is the confusional state caused by the portal blood bypassing the detoxification of the liver. It occurs in about 40 per cent of patients, a similar incidence to that found after surgical shunts. If severe, the lumen of the TIPSS can be reduced by insertion of a smaller stent.

The main long-term complication of (TIPSS) is stenosis of the shunt, which is common (approximately 50 per cent at one year) and may present as further variceal haemorrhage.

The main contraindication to (TIPSS) is portal vein occlusion.

The Surgical shunts for variceal haemorrhage

The increasing availability of liver transplantation and TIPSS has greatly reduced the indications for surgical shunts.

The Surgical shunts for variceal haemorrhage

It is rarely considered for the acute management of variceal haemorrhage, as the morbidity and mortality in these circumstances are high.

The main current indication for a surgical shunt is a patient with Child's grade A cirrhosis, in whom the initial bleed has been controlled by sclerotherapy. Longterm β -blocker therapy and chronic sclerotherapy or banding are the main alternatives.

The Surgical shunts for variceal haemorrhage

Surgical shunts are an effective method of preventing rebleeding from oesophageal or gastric varices, as they reduce the pressure in the portal circulation by diverting the blood into the low-pressure systemic circulation.

The Surgical shunts for variceal haemorrhage

- Shunts may be divided into
- Selective (e.g. splenorenal) and
- Non-selective (e.g. portocaval

Shunts may be divided into

Selective (e.g. splenorenal) is attempting to preserve blood flow to the liver while decompressing the left side of the portal circulation responsible for giving rise to the oesophageal and gastric varices.

Selective shunts may be associated with a lower incidence of portal systemic encephalopathy (PSE), a confusional state commonly found in patients with chronic liver disease who have undergone radiological or surgical portosystemic shunts.

Surgical shunts.

There is no evidence that prophylactic shunting is beneficial in patients with varices that have not bled.

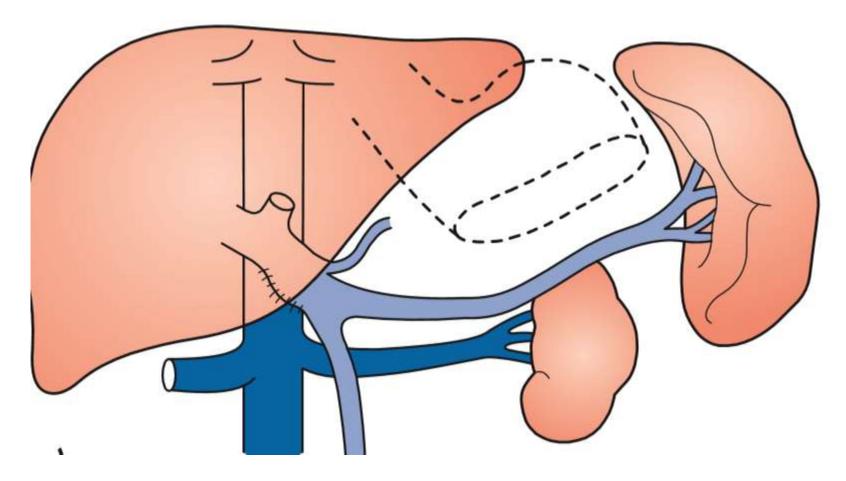
Surgical shunts.

Surgical shunts for portal hypertension involve shunting portal blood into the systemic veins.

This commonly involves :

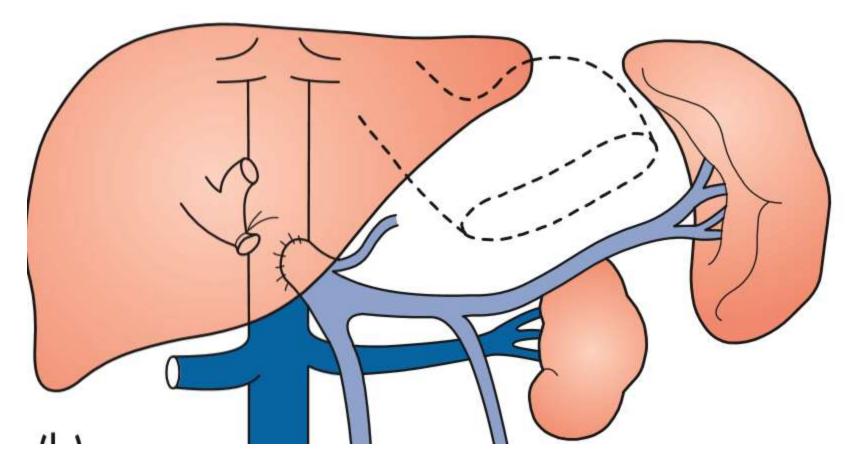
- Side-to-side portocaval anastomosis.
- End-to-side portocaval anastomosis.
- Mesocaval anastomosis.
- Splenorenal anastomosis.





Surgical shunts. Side-to-side portocaval anastomosis

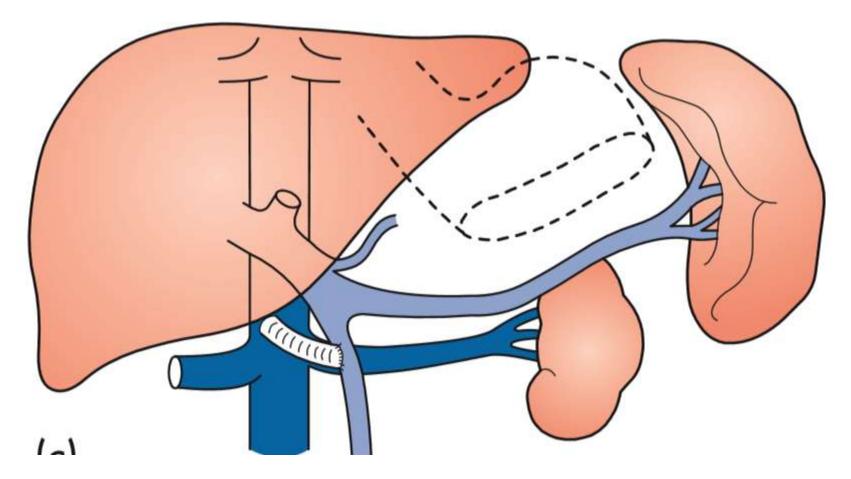




Surgical shunts. End-to-side portocaval anastomosis

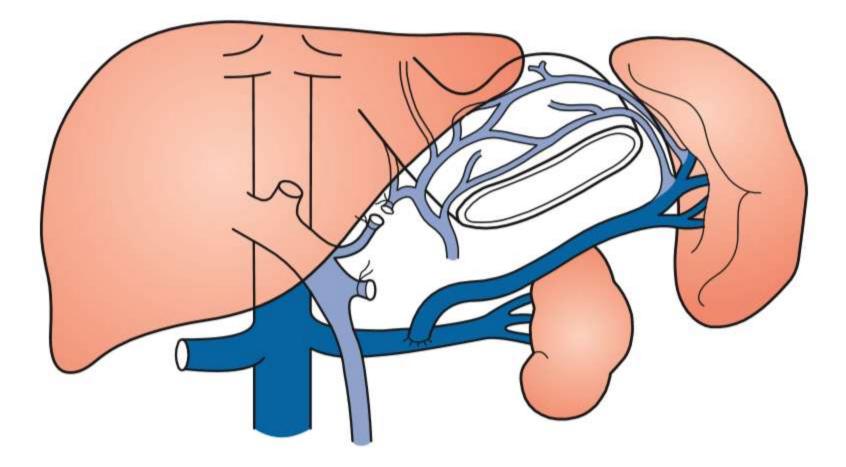
Courtesy Bailey & Love's Short Practice of Surgery





Surgical shunts. Mesocaval anastomosis





Surgical shunts. Splenorenal anastomosis

Oesophageal stapled transection

This technique for the management of bleeding oesophageal varices uses the circular stapling device for stapling and resecting a doughnut ring of the lower oesophagus.

As with surgical shunts in the acute situation, it was associated with a high perioperative mortality and has been largely abandoned in centres where TIPSS is available.

Management of recurrent variceal bleeds secondary to splenic or portal vein thrombosis

Treatment is by splenectomy and

gastro-oesophageal devascularisation,

in which the blood supply to the greater and lesser curve of the stomach and lower oesophagus is divided.

Management of recurrent variceal bleeds secondary to splenic or portal vein thrombosis

Splenic vein thrombosis may be seen secondary to chronic pancreatitis,

and **portal vein thrombosis** is a common late complication of **liver cirrhosis**.

Variceal bleeding and orthotopic liver transplantation

Liver transplantation is the only therapy which will treat portal hypertension and the underlying liver disease.

Variceal bleeding and orthotopic liver transplantation

The management of variceal bleeding should always take into account the possibility of liver transplantation when this is available.

Variceal bleeding and orthotopic liver transplantation

TIPSS would be the preferred management for bleeds resistant to sclerotherapy, as long as placement is optimal.

Previous surgical shunts greatly increase the morbidity associated with orthotopic liver transplantation.



Management of bleeding oesophageal varices

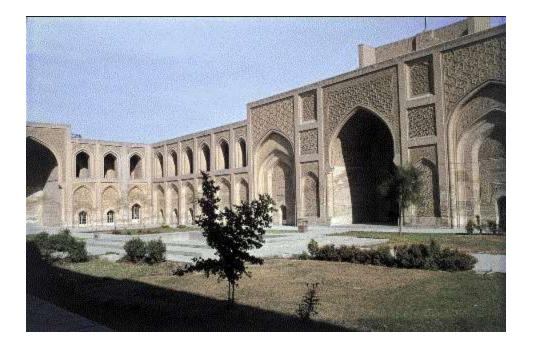
- Blood transfusion
- Correct coagulopathy
- Oesophageal balloon tamponade (Sengstaken–Blakemore tube)
- Drug therapy (vasopressin/octreotide)
- Endoscopic sclerotherapy or banding
- Assess portal vein patency (Doppler ultrasound or CT)
- Transjugular intrahepatic portosystemic stent shunts (TIPSS)
- Surgery
 - Portosystemic shunts
 - Oesophageal transection
 - Splenectomy and gastric devascularisation





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