Portal Hypertension: Introduction

As early as the 17th century, it was realized that structural changes in the portal circulation could cause gastrointestinal bleeding. In 1902, Gilbert and Carnot introduced the term "portal hypertension" to describe this condition. Portal hypertension is a pressure in the portal venous system that is at least 5 mm Hg higher than the pressure in the inferior vena cava. This increased pressure results from a functional obstruction to blood flow from any point in the portal system's origin (in the splanchnic bed) through the hepatic veins (exit into the systemic circulation) or from an increase in blood flow in the system.

Substantial progress has been made in understanding the pathophysiology of portal hypertension. This knowledge has led to the development of new therapeutic management approaches such as pharmacological therapies, endoscopic therapies, and surgical and radiological shunting procedures. Although many advances have been made in this field, the complications of portal hypertension (gastrointestinal hemorrhage, hepatic encephalopathy, hepatorenal syndrome, and ascites) continue to be the cause of significant morbidity and mortality. Portal hypertension remains one of the most serious sequelae of chronic liver disease.
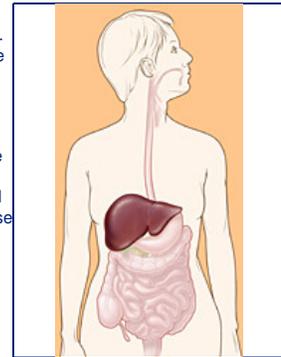

Figure 1. Location of liver in the body.

What is Portal Hypertension?
Portal hypertension is a term used to describe elevated pressures in the portal venous system (a major vein that leads to the liver). Portal hypertension may be caused by intrinsic liver disease, obstruction, or structural changes that result in increased portal venous flow or increased hepatic resistance. Normally, vascular channels are smooth, but liver cirrhosis can cause them to become irregular and tortuous with accompanying increased resistance to flow. This resistance causes increased pressure, resulting in varices or dilations of the veins and tributaries. Pressure within the portal system is dependent upon both input from blood flow in the portal vein, and hepatic resistance to outflow. Normally, portal vein pressure ranges between 1–4 mm Hg higher than the hepatic vein free pressure, and not more than 6 mm Hg higher than right atrial pressure. Pressures that exceed these limits define portal hypertension.

Symptoms
Gastrointestinal hemorrhage may be the initial presenting symptom of patients with portal hypertension. Those patients with more advanced liver disease often present with ascites, hepatic encephalopathy , jaundice, coagulopathy, or spider angiomata. Patients who are hemodynamically stable may have warm skin, hyperdynamic pulses, and low systolic blood pressures in the range of 100–110 mm Hg. Additionally, splenomegaly and dilated abdominal wall veins are also indicative of portal hypertension. Splenomegaly can result in sequestration of platelets from the systemic circulation, and low platelet counts may be the earliest abnormal laboratory finding. Hepatomegaly is variable and dependent upon the cause and stage of liver disease. Portal vein thrombosis may occur as a complication of portal hypertension but may also occur in cases of myeloproliferative or hypercoagulable disorders.

The clinical manifestations of portal hypertension may include caput medusae, splenomegaly, edema of the legs, and gynecomastia (less commonly) (Figure 2).
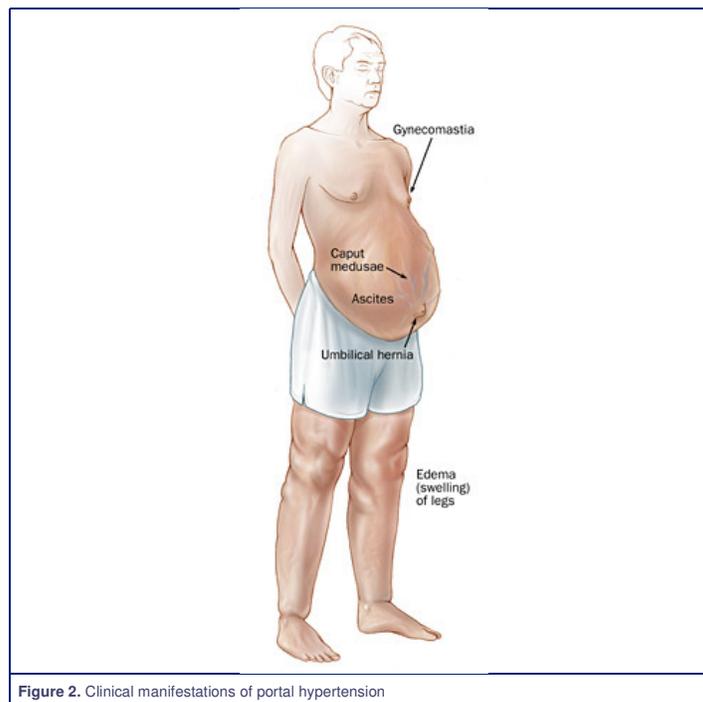

Figure 2. Clinical manifestations of portal hypertension

Caput medusae is a network of dilated veins surrounding the umbilicus. It is caused by increased blood flow in the umbilical and periumbilical veins and is often accompanied by an audible venous hum over the umbilical vein (Cruveilhier-Baumgarten murmur). Gynecomastia refers to the unilateral or bilateral abnormal enlargement of breast tissue behind the areola in males. It may be precipitated by hormonal imbalance or hormone-secreting tumors, testicular or pituitary tumors, liver failure, and antihypertensive medications or medications containing estrogen or steroids. Edema, or swelling of the legs, is seen in portal hypertensive patients because of alterations in systemic hemodynamics.

Portal Hypertension: Anatomy

The liver is located in the right upper quadrant, from the fifth intercostal space in the midclavicular line down to the right costal margin. The liver weighs approximately 1800 g in men and 1400 g in women. The surfaces of the liver are smooth and convex in the superior, anterior, and right lateral regions. Indentations from the colon, right kidney, duodenum , and stomach are apparent on the posterior surface.

The line between the vena cava and gallbladder divides the liver into right and left lobes. Each lobe has an independent vascular and duct supply. The liver is further divided into eight segments, each containing a pedicle of portal vessels, ducts, and hepatic veins.

The portal venous system extends from the intestinal capillaries to the hepatic sinusoids (Figure 3). This venous system carries the blood from the abdominal gastrointestinal tract, the pancreas, gallbladder, and spleen back to the heart (coursing through the liver). The largest vessel in this system is the portal vein, which is formed by the union of the splenic vein and superior mesenteric veins. The left gastric and right gastric veins and the posterior superior pancreaticoduodenal vein drain directly into the portal vein. The portal vein runs posterior to the pancreas, and its extrahepatic length may be anywhere from 5–9 cm. At the porta hepatis, it divides into the right and left portal veins within the liver, and the cystic vein typically drains into the right hepatic branch.
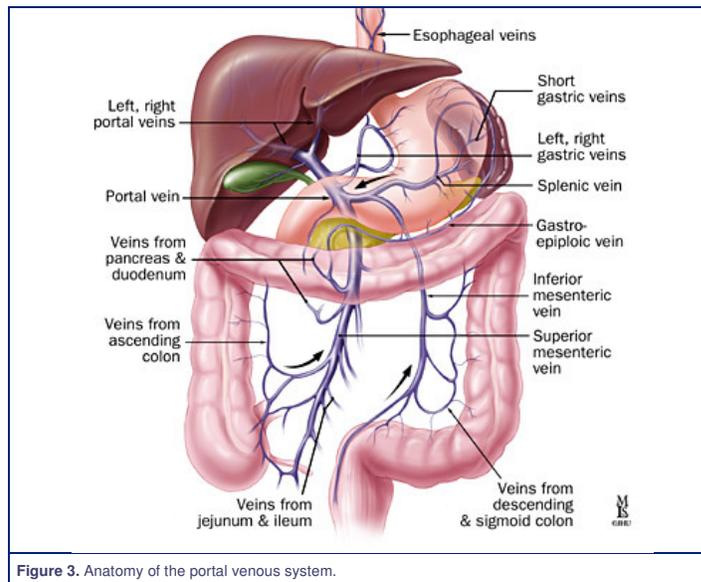

Figure 3. Anatomy of the portal venous system.

The portal vein supplies 70% of the blood flow to the liver, but only 40% of the liver oxygen supply. The remainder of the blood comes from the hepatic artery, and blood from both of these vessels mixes in the sinusoids.

The liver receives a tremendous volume of blood, on the order of 1.5 liters per minute. The dual blood supply allows the liver to remain relatively resistant to hypoxemia. Unlike the systemic vasculature, the hepatic vascular system is less influenced by vasodilation and vasoconstriction. This is because the sinusoidal pressures remain relatively constant despite changes in blood flow. A classic example is hepatic vein occlusion resulting in high sinusoidal pressure and extracellular extravasation of fluid. To maintain a constant inflow of blood to the liver, hepatic artery blood flow is inversely related to portal vein flow. This appears to be hormonally mediated rather than neurally mediated, since it persists in the transplanted liver.

Portal Hypertension: Causes

Overview

Normal portal vein pressures range from 5–10 mm Hg. The term portal hypertension refers to elevated pressures in the portal venous system. Venous pressure more than 5 mm Hg greater than the inferior vena cava pressure is defined as portal hypertension. Clinically it may be difficult to detect portal hypertension until pressures are much higher. There are many causes of portal hypertension including etiologies above the liver, within the liver, and below the liver.

Suprahepatic Causes

Suprahepatic abnormalities leading to portal hypertension include cardiac disease, hepatic vein etiology, and inferior vena cava thrombosis or webs. Hepatic vein thrombosis, or Budd-Chiari syndrome, has multiple etiologies but is generally related to a hypercoagulable state and often treatable with anticoagulation. Liver fibrosis can result from suprahepatic disease, and cirrhosis can also develop late in the disease course (Figure 4).
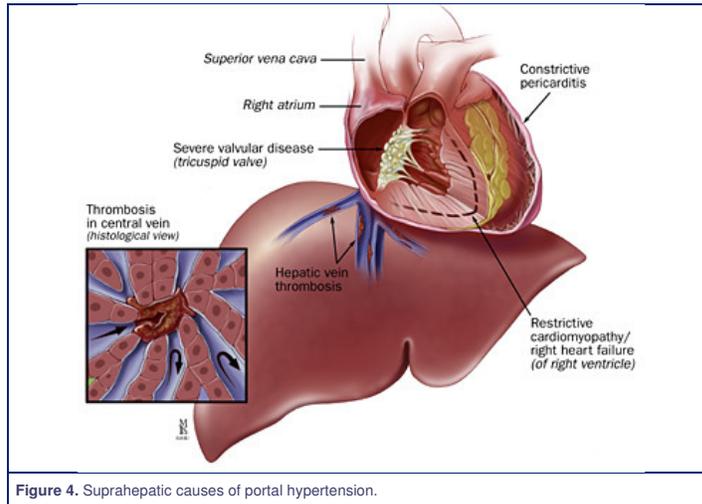
Figure 4. Suprahepatic causes of portal hypertension.

Hepatic Causes

Cirrhosis is the most common cause of portal hypertension, and chronic viral hepatitis C is the most common cause of cirrhosis in the United States. Alcohol-induced liver disease and cholestatic liver diseases are other common causes of cirrhosis. Less common causes include hemochromatosis, alpha 1-antitrypsin deficiency, drug-induced liver disease, and (in Eastern countries) hepatitis B. Portal hypertension is considered an advanced complication of cirrhosis. Once it has developed, the term "decompensated cirrhosis" is used (Figure 5).
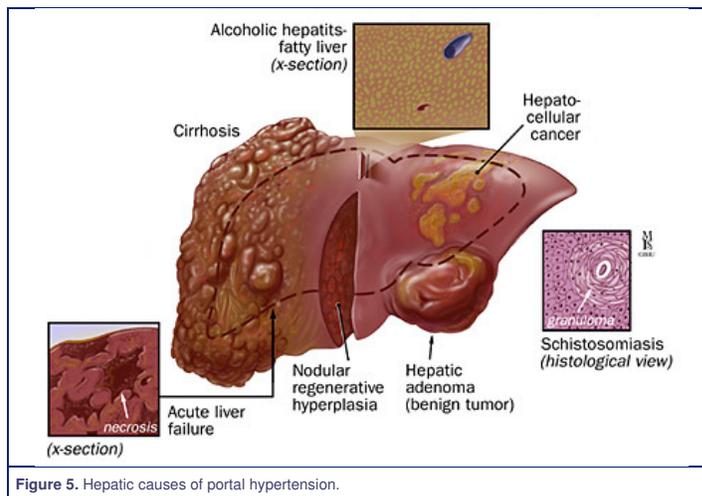
Figure 5. Hepatic causes of portal hypertension.

Infrahepatic Causes

Alterations of portal venous blood flow can also lead to portal hypertension. Arteriovenous malformation of the splenic vasculature, splenomegaly and portal vein thrombosis are examples of infrahepatic causes of portal hypertension. Overall, these are not common conditions (Figure 6).
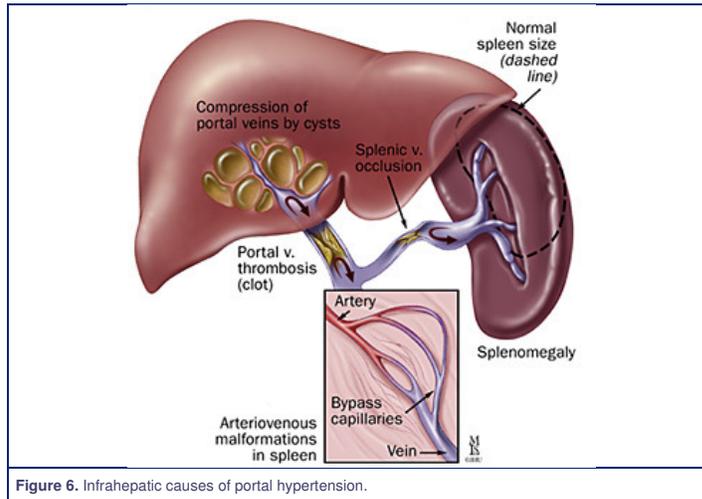
Figure 6. Infrahepatic causes of portal hypertension.

Cytokines

The mechanism of portal hypertension has been the subject of extensive research. The pathophysiology is thought to involve vasodilators produced by the body. Namely, cytokines such as tumor necrosis factor-alpha (TNF-alpha) and others may play a role in stimulating endothelial vasodilators such as nitric oxide and prostacyclin as well as non-endothelial vasodilators like glucagon. These molecules may affect pressure and flow in the splanchnic vasculature, leading to hypertension.

Portal Hypertension: Diagnosis

Overview

Portal hypertension can be diagnosed in several ways. Clinical diagnosis can be made in the setting of end-stage liver disease and in the presence of ascites and/or varices. Subclinical portal hypertension is much more difficult to diagnose, but low platelet levels, a large portal vein, and splenic enlargement on imaging studies are suggestive. Direct or indirect measurements of the portal vein may be accomplished using wedged hepatic vein pressure or splenic pulp pressure, but these methods are relatively invasive.

Imaging Studies

Imaging studies of patients with portal hypertension are helpful to make a diagnosis and to define portal venous anatomy. Duplex doppler ultrasonography is a noninvasive, low-cost method of diagnosis that provides sophisticated information. It is often the initial procedure performed and provides specifics regarding the direction and velocity of portal flow. Findings of increased hepatic echogenicity , splenomegaly, portal vein dilation, thrombotic occlusion, collaterals, and gallbladder wall thickening are indicative of portal hypertension. MRI (magnetic resonance imaging) and computed tomography (CT) are not particularly useful in making a diagnosis, but are capable of providing some of the same information.

Pressure Measurement Studies

Portal pressure measurement is not generally indicated. It is most often performed in setting of therapeutic or hemodynamic research studies. Clinically it is used to assess the efficacy of pharmacological agents or shunting procedures. Most approaches to portal pressure measurement are relatively invasive, with the exception of newer endoscopic techniques.   The most commonly used and preferred method for measuring the portal pressure is by indirectly calculating this pressure after occlusion of the hepatic vein. This is an invasive procedure, typically performed by interventional radiologists.

Endoscopic Diagnosis

Endoscopy is the standard diagnostic approach in patients with acute gastrointestinal hemorrhage after initial resuscitation. In most patients with cirrhosis (60–80%) bleeding is related to esophageal varices. In addition to making a definitive diagnosis, endoscopic therapy may be indicated for bleeding. Endoscopic examination may require endotracheal intubation in patients who have significant alteration in mental status as a result of severe hepatic decompensation.

Gastrointestinal endoscopy allows the physician to visualize and biopsy the mucosa of the upper gastrointestinal tract including the esophagus, stomach, and duodenum. The enteroscope allows visualization of at least 50% of the small intestine, including most of the jejunum and different degrees of the ileum. During endoscopic procedures, a pharyngeal topical anesthetic may be administered to help prevent gagging. Pain medication and a sedative may also be given prior to the procedure. The patient is placed in the left lateral position (Figure 7).
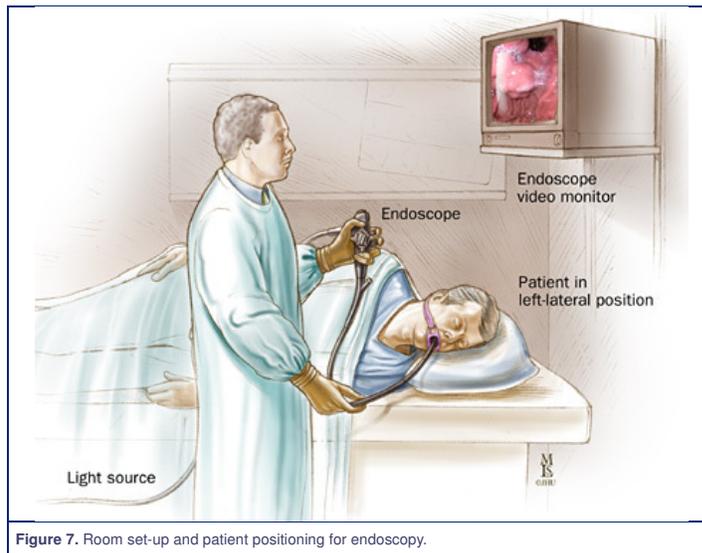

Figure 7. Room set-up and patient positioning for endoscopy.

The endoscope — a thin, flexible, lighted tube — is passed through the mouth and pharynx, and into the esophagus. The endoscope transmits images of the esophagus, stomach, and duodenum to a monitor, visible to the physician (Figure 8). Air may be introduced into the stomach to expand the folds of tissue and enhance the examination.
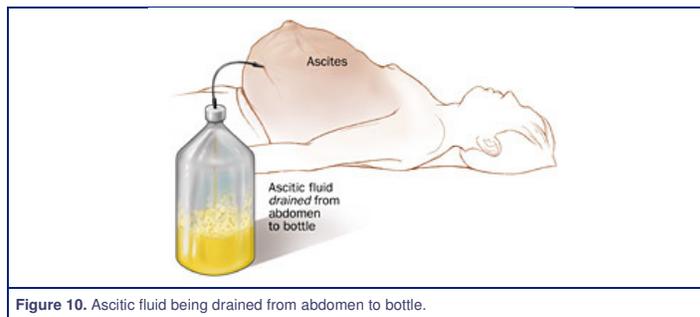
Portal Hypertension: Therapy

Overview

Treatment of portal hypertension is aimed at prevention of complications. The main goal of therapy is to decrease portal pressures. This is generally difficult to achieve and adequately maintain.

Ascites

Ascites is the presence of excess fluid in the peritoneal cavity. Ascites frequently develops in patients with chronic liver disease, but may be due to a wide range of causes. Clinically, patients may be asymptomatic or may have a variety of complaints including early satiety, increase in abdominal girth, or respiratory distress (depending upon the amount of fluid accumulation in the abdomen) (Figure 9). Patient with ascites often have abdominal distention, tympany of the top, bulging flanks, puddle sign, fluid wave, or shifting dullness on physical examination. The most important aspect in treating ascites is to restrict sodium to less than 2 g per day. More restrictive regimens are difficult to accomplish in the outpatient setting. Water restriction is generally not necessary unless patients develop hyponatremia. In this setting, fluid restriction to less than 1.5 liters per day is generally adequate. Diuretic therapy, to reduce sodium retention by the kidneys, is generally required. This is achieved through blocking the effects of the salt-regulatory hormone, aldosterone. Loop diuretics function at the ascending limb of the loop of Henle. Generally, a combination of spironolactone or other potassium-sparing diuretic along with a loop diuretic is required for complete diuresis. Patients need to be monitored closely for side effects, which include hyponatremia, hyperkalemia, hypokalemia , dehydration, hypotension, and azotemia.

Figure 9. Mechanism of ascites in portal hypertension; S=stomach; C=colon; I=intestine.
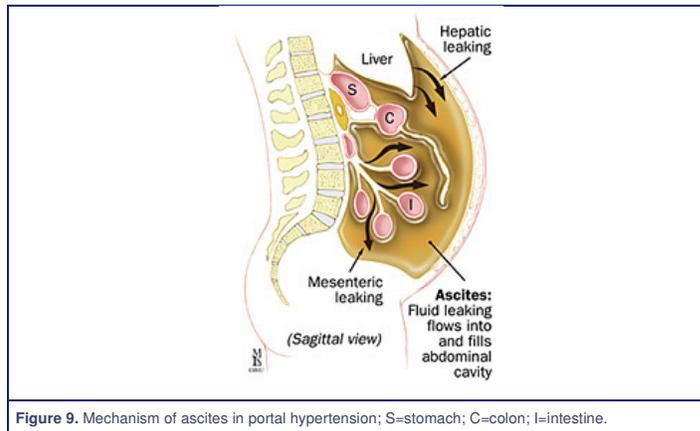
Large-volume paracentesis may still be required in patients with difficult-to-control ascites, or in patients who do not tolerate diuretic therapy. Abdominal paracentesis may be used to therapeutically remove ascites and is useful in relieving respiratory difficulties, acute oliguria or pain. Paracentesis is performed at the bedside. After sterilization of the abdominal wall, a local anesthetic is administered. A sterile needed is inserted by the physician into the abdomen and the ascitic fluid aspirated (Figure 10). Infusion of intravenous albumin after large-volume (greater than 5 liters) paracentesis is preferred.

Figure 10. Ascitic fluid being drained from abdomen to bottle.

Varices

Varices are varicose veins, visible on endoscopy, an upper GI series or other imaging studies, that occur in the esophagus or stomach as a result of portal hypertension (Figure 11). Cirrhosis causes severe scarring of the liver and impedes the normal circulation of blood. Varices develop when portal blood is rerouted to the systemic circulation , through collateral vessels, because of increased resistance to blood flow to or through the liver. Obstructions may occur in the hepatic veins, sinusoids, or portal veins. The pressure within these irregular vessels is great and they have the potential to rupture.

Figure 11. A-C, Esophageal and gastric varices with corresponding endoscopic views.
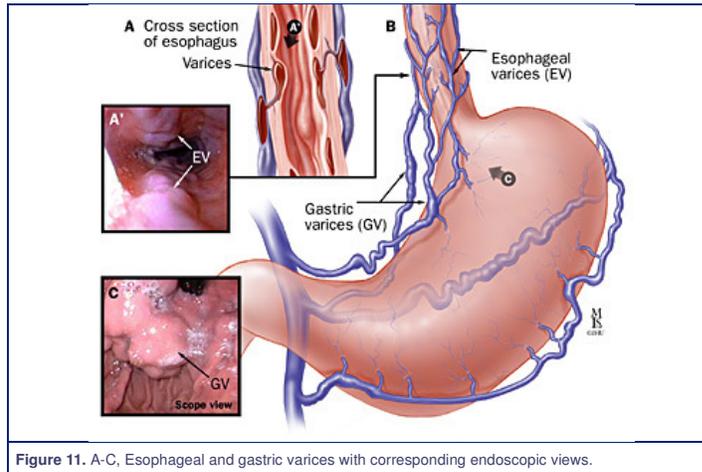
Acute bleeding from varices or nonvariceal sites in patients with portal hypertension requires prompt and appropriate measures to control bleeding and prevent recurrent episodes. Therapy is aimed at the prevention of recurrent episodes of variceal bleeding by lowering portal pressure and eliminating varices.

Medical Therapy

Medical management of bleeding esophageal or gastric varices may be instituted once the cause of the hemorrhage is documented to be variceal in origin. Drug treatment is aimed at reducing portal inflow, or collateral or intrahepatic resistance. When the hepatic venous pressure gradient is below 12 mm Hg, the danger of variceal bleeding is relatively low. Use of beta-blockers has been shown to decrease portal pressures, but side effects of the drugs are sometimes prohibitive. Propranolol is a nonselective beta-blocker that has been extensively studied and is effective in decreasing portal pressures. It decreases the risk of variceal bleeding both as a primary prophylaxis and after an initial bleeding episode. The dose needs to be titrated to decrease resting heart rate by 25%. Unfortunately, in patients with cirrhosis and no varices, the side-effects outweigh the benefits of beta-blockers therefore these medications should not be used for the prevention of variceal development. There are no other medical therapies that can be recommended to prevent variceal bleeding.

Use of vasopressin in the acutely bleeding patient is effective and works by decreasing splanchnic blood flow. Vasopressin therapy should be instituted in an intensive care unit through a central venous access line. The use of this drug is associated with side effects of vasoconstriction in other vascular beds, including cardiac vessels. Vasopressin should be administered with sublingual nitroglycerin. Somatostatin is currently the preferred drug for acute variceal bleeding. It also acts as a vasoconstrictor, but works only on the splanchnic bed, and therefore has fewer side effects. It is given as an intravenous bolus at 50 micrograms, followed by a constant infusion of 50 micrograms per hour.

The short-term (7 days) administration of antibiotics to patients with cirrhosis and acute variceal hemorrhage is the standard of practice. In these patients, antibiotics have been shown to reduce the rate of re-bleeding and increase overall survival. Antibiotics should be administered irrespective of the presence of ascites. It is preferred to select an antibiotic to target gram negative bacteria.

Endoscopic Therapy

Endoscopy plays a critical role in the diagnosis and treatment of gastrointestinal hemorrhage (Figure 12). Treatment options include sclerotherapy, banding of esophageal varices and balloon tamponade to control bleeding.


Figure 12. Room set-up and positioning for endoscopy.
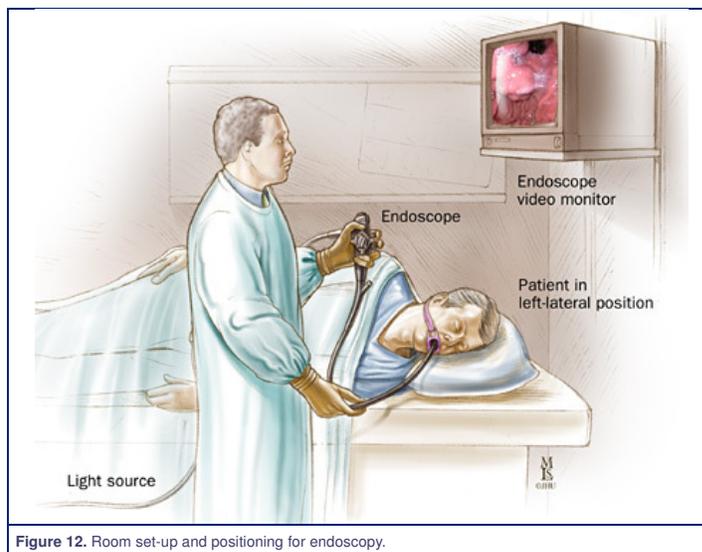
Banding

Acute variceal hemorrhage is ideally managed by variceal ligation with elastic rings, commonly called banding (Figure 13).

Figure 13. A, Endoscope with band ligators on tip; B, banded esophageal varices; C, corresponding endoscopic image.
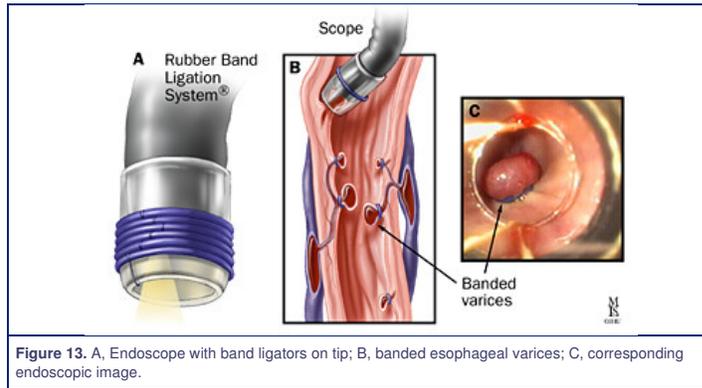
This method is performed endoscopically, and is safe and effective. This technique employs the use of small elastic rings that are placed over a suctioned varix. When a patient with suspected acute variceal hemorrhage is admitted to the hospital, treatment should be immediately initiated with the aforementioned pharmacologic therapies (somatostatin analogue). Then, upper endoscopy with variceal ligation should be attempted within twelve hours. Banding has fewer side effects and complications than sclerotherapy (see below) and is equally effective. After the initial banding session, subsequent sessions are scheduled with the intent to completely obliterate the varices.

Sclerotherapy
The use of sclerotherapy, or injection of a sclerosing agent directly into and around the varices, has been well studied. The technique consists of injecting 1–10 mL of sclerosing agent (sodium morrhuate, sodium tetradecyl sulfate, ethanolamine oleate, or absolute alcohol) into the varix beginning at the gastroesophageal junction and circumferentially into all columns. There is considerable variation in the type and volume of the agent used, as well as the site of injection. Comparison studies of various techniques and solutions have not shown significant advantages of any one method. In the setting of acute variceal hemorrhage, sclerotherapy should be reserved for patients in whom band ligation is not technically feasible. After the initial sclerotherapy session, subsequent sessions are scheduled with the intent to completely obliterate the varices. Common side effects include tachycardia, chest pain, fever, and ulceration at the injection site.

Figure 14. Sclerotherapy for esophageal varices.
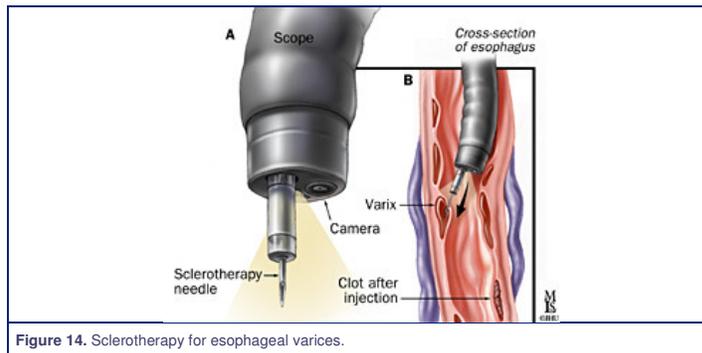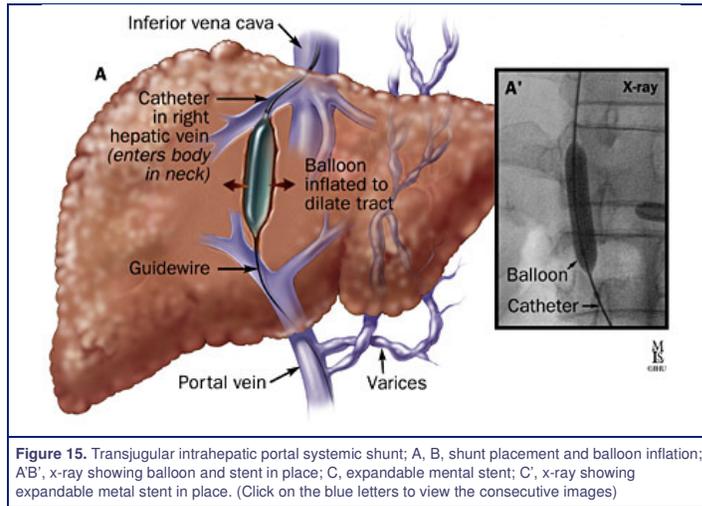
Balloon Tamponade
Balloon tamponade is useful to control variceal bleeding through compression. Use of one of three commercially available balloons to tamponade bleeding esophageal or gastric varices can be employed when medical management has not been successful, and endoscopic management has failed or is unavailable. Although quite effective as a temporary measure, there is a high risk of complications, especially aspiration. Only experienced physicians should perform the tube placement, and the patient should be carefully and continuously monitored.

Shunting Procedures
Nonsurgical Transjugular Intrahepatic Portal-Systemic Shunt (TIPSS)
Transjugular intrahepatic portal-systemic shunting is a radiologic procedure that has become very popular as an alternative method of controlling acute bleeding, especially if gastric varices are present. It is also indicated in patients who have had recurrent bleeding despite medical or endoscopic management. Contraindications to TIPSS placement include severe liver dysfunction, renal failure, and heart failure.

The procedure itself requires a high level of expertise, and is performed under fluoroscopic guidance using intravenous sedation. First, access to the hepatic vein is obtained through the right internal jugular vein. A needle is passed through the liver parenchyma into the portal vein, followed by dilation of the tract (Figure 15, A and B), and subsequent placement of a metal stent. The stent is then dilated to achieve a portal to hepatic vein gradient of less than 10 mm Hg (Figure 15C).

Figure 15. Transjugular intrahepatic portal systemic shunt; A, B, shunt placement and balloon inflation; A'B', x-ray showing balloon and stent in place; C, expandable mental stent; C', x-ray showing expandable metal stent in place. (Click on the blue letters to view the consecutive images)

Success is over 90% in experienced hands. The long-term utility of the stent is limited by a high occlusion rate from thrombosis or stenosis. The main side effect is worsening hepatic encephalopathy, which can be severe in a minority of patients. The patency of the stent can be checked by Doppler ultrasound . Stenosed stents can generally be revised.
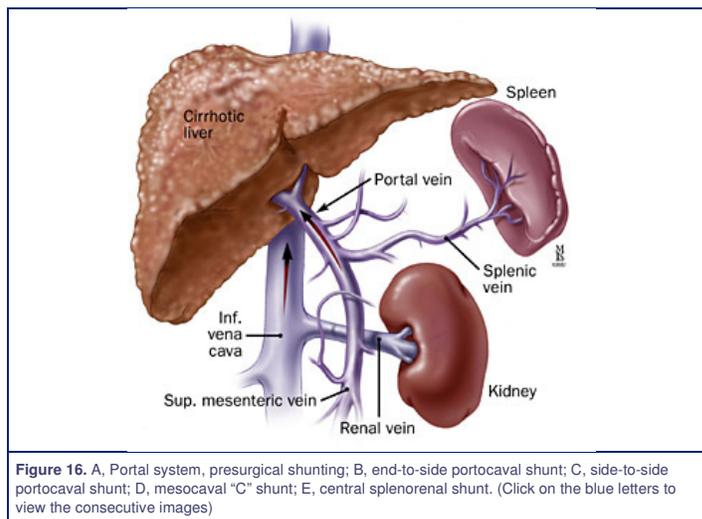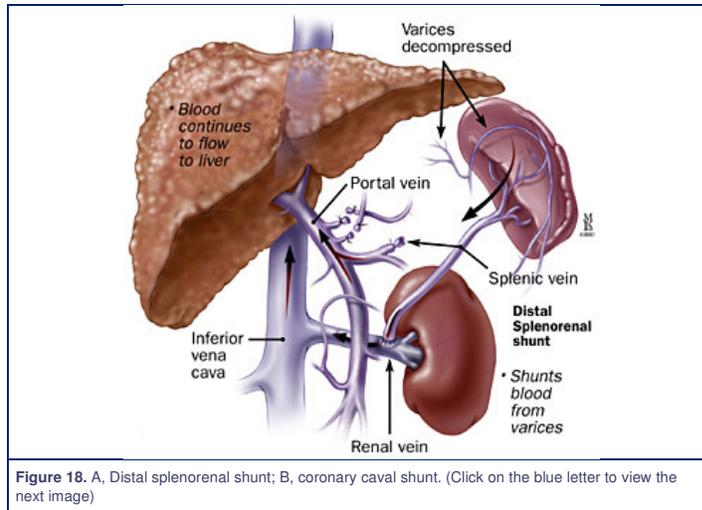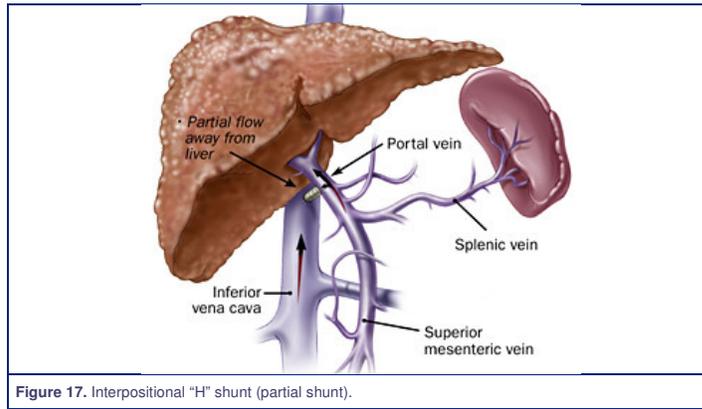
Surgical Shunts
The aim of surgical shunting in portal hypertension is threefold: 1) to reduce portal venous pressure, 2) to maintain hepatic and portal blood flow, and 3) to try to reduce or not complicate hepatic encephalopathy (Figure 16). Currently, there is no procedure that reliably and consistently fulfills all of these criteria.

The operative mortality in shunting procedures is about 5% in patients who are good surgical risks, and about 50% in those who are poor surgical risks. Surgical shunts are often very effective in patients with mild liver disease but have severe portal hypertension, such as in the case of acute hepatic vein occlusion (Budd-Chiari syndrome).

Shunts	Effectiveness	Complications

Total portal-systemic
Portocaval shunt
    (Figure 16 B and C)
Mesocaval shunt
    (Figure 16 D)
Central spleno-renal shunt
    (16 E)
	Control of variceal bleeding; Side-to-side has advantage over end-to-side anastomosis for control of ascites	Can worsen liver function; encephalopathy; high incidence of thrombosis in meocaval and central spleno-renal; 10% operative mortality

Partial portal-systemic
Interpositional shunt,
    8 mm diameter
    (Figure 17)
	Good if shunt size remains constant	Difficult keeping shunt systemic: constant size

Selective shunt
Distal spleno-renal shunt
    (Figure 18A)
Coronary caval shunt
    (Figure 18B)
	Control of varices, does not relieve portal hypertension centrally	6% operative mortality; may worsen encephalopathy

Table 1. Surgical Shunts

Figure 16. A, Portal system, presurgical shunting; B, end-to-side portocaval shunt; C, side-to-side portocaval shunt; D, mesocaval "C" shunt; E, central splenorenal shunt. (Click on the blue letters to view the consecutive images)

Figure 17. Interpositional "H" shunt (partial shunt).

Figure 18. A, Distal splenorenal shunt; B, coronary caval shunt. (Click on the blue letter to view the next image)
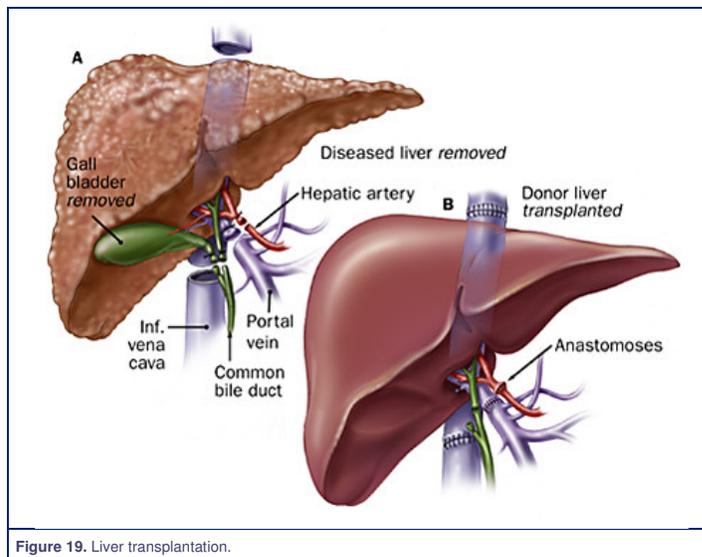
Liver Transplantation

Liver transplantation is the only effective treatment for end-stage liver disease. This option offers excellent patient survival and rehabilitation. Challenges of liver transplantation include a scarcity of human cadaver donors, rejection, and the limited financial resources of most patients. Liver transplantation is a long and complex surgery that involves the removal and the replacement of the body's largest solid organ. It requires surgical expertise in biliary and vascular reconstruction.

Variceal bleeding alone is not an indication for transplantation; refractory bleeding can elevate the listing status of patients awaiting transplant (Figure 19, A and B).

Figure 19. Liver transplantation.

For more information about Liver Transplantation

Overview

Complications secondary to portal venous hypertension can be life threatening and are often the main indication for transplantation in patients with advanced liver disease. Although there are several collaterals between portal and systemic venous circulation, those at the junction of the stomach and esophagus are particularly important. The dilated portosystemic collaterals at the junction of the stomach and esophagus are termed varices (Figure 20).

Figure 20. A, B, Esophageal and gastric varices; A', C, corresponding endoscopic images.
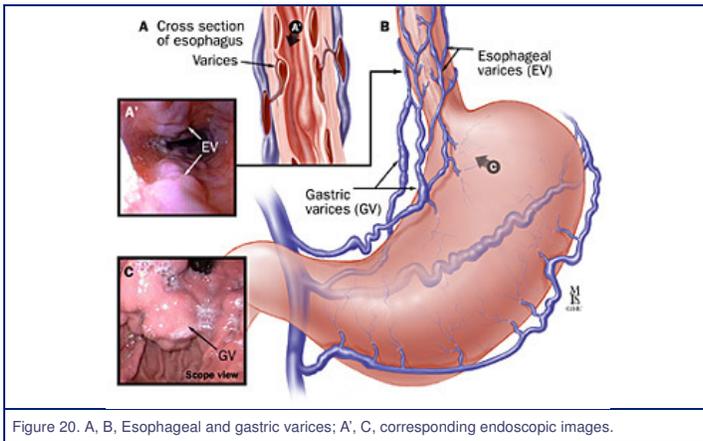
The risk of rupture and gastrointestinal bleeding from varices is related to the size of the varices and the severity of the portal hypertension. Bleeding from varices carries a risk of mortality as high as 40%. Prophylaxis with beta blockers has been shown to be effective. For this reason, all patients suspected of having portal hypertension should undergo upper endoscopy to evaluate the esophagus for varices. During an acute bleeding episode, upper endoscopy should be performed in the hemodynamically stable patient for both confirmation of bleeding source and therapy. Both banding and sclerotherapy are effective means of obliterating varices, although banding is preferred. Octreotide, administered intravenously, is a highly effective means of controlling acute bleeding.

Gastropathy
Bleeding can also occur from the gastric mucosa. Due to blood flow changes in mucosa, the integrity is often compromised and very friable. This is called portal hypertensive gastropathy. There is no effective management except relief of portal hypertension.

Splenomegaly
Enlargement of the spleen, or splenomegaly, is a common occurrence with portal hypertension (Figure 21). There is little correlation between the size of the spleen and the severity of portal hypertension. Hypersplenism with sequestration of platelets and leukocytes is a common phenomenon. Generally, there is no indication for platelet transfusions unless an invasive procedure is planned, and splenectomy is not indicated. Both hypersplenism and splenomegaly resolve (though not always completely) with decompression of portal hypertension.

Figure 21. Splenomegaly.
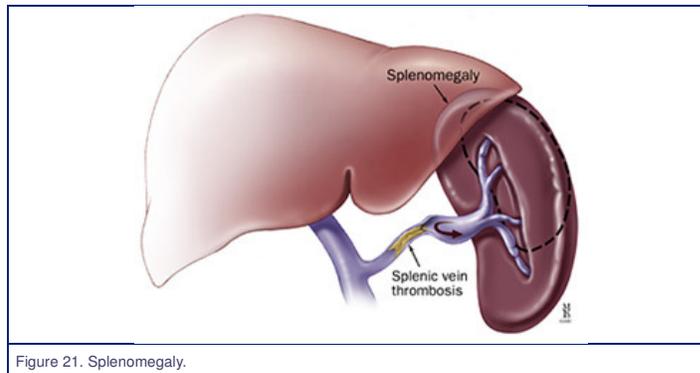
Ascites
Another complication of portal hypertension is the development of free peritoneal fluid or ascites. Ascites is lymphatic fluid that leaks across hepatic sinusoidal endothelium due to high hepatic sinusoidal pressure (Figure 22).
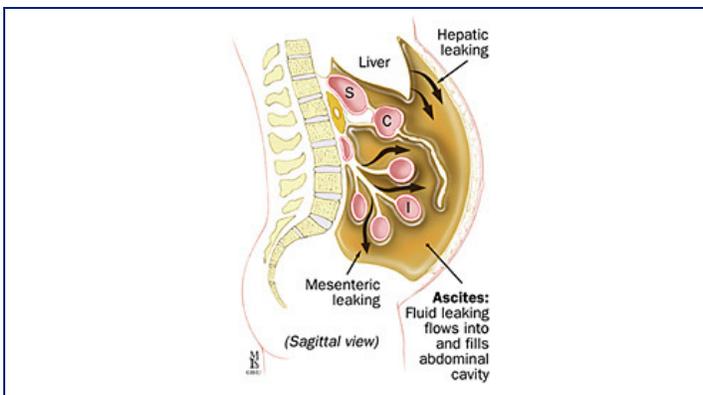
Figure 22. Mechanism of ascites in portal hypertension; S=stomach; C=colon; I=intestine.

Flow across this endothelium is normally controlled by an oncotic pressure gradient. However, the increase in lymphatic flow results in a loss of this oncotic gradient and formation of ascitic fluid. In addition to this, splanchnic lymph formation also contributes to ascites. The relative contribution of splanchnic lymph is not known. Intra-abdominal fluid is normally absorbed by the peritoneum. The exact mechanism of this fluid resorption is not known, but high intraperitoneal pressure results in net increase in absorption.

Abdominal paracentesis is the technique by which ascites is removed from the abdominal cavity. Paracentesis is performed at the bedside. After sterilization of the abdomen, local anesthetic is administered, a sterile needed is inserted by the physician into the abdomen, and the ascitic fluid aspirated. After large-volume paracentesis, intraperitoneal pressures drop and there is rapid reaccumulation of ascites. Ascitic fluid may be sent for laboratory analysis, which includes protein content, cytological analysis, and cultures for bacterial infections. Ascitic fluid was previously classified based on protein content. Low protein ascites was termed transudative and implied hepatic congestion, typically due to chronic liver disease. Fluid transfer occurs across hepatic sinusoids into interstitial tissues and the liver capsule into the peritoneal space. Exudative ascites, on the other hand, had higher protein content and implied a different pathogenesis. Namely, production was believed to be from the peritoneum.

Ascites	
Portal Hypertension	Non-portal hypertension
Cirrhosis	Tuberculosis
Cardiac disease	Pancreatic ascites
Liver tumors	Carcinomatosis
Hepatic failure	Nephrotic syndrome
Hepatic vein thrombosis	Lymphatic obstruction
Portal vein thrombosis	

Table 2. Ascites

A more important distinction to make when assessing ascitic fluid is whether the fluid is portal hypertensive in origin. An albumin gradient greater than 1.1 g/dL between the serum and ascitic fluid is highly suggestive of fluid that is portal hypertensive in origin.

The most important sequelae of ascites is the risk for development of spontaneous bacterial peritonitis (SBP). Due to the low protein content and oncotic pressure of portal hypertensive ascitic fluid, the risk of infection is very high. SBP can be difficult to diagnose due to an inconsistent clinical presentation. Pain is often absent and the only reliable way to diagnosis this condition is by paracentesis. Most cases are due to a single bacterial organism, with over 70% of cases secondary to Gram-negative bacilli. Streptococcal species and Staphylococcus make up the majority of other cases.

Diagnosis of spontaneous bacterial paracentesis is made based on an ascitic fluid (PMN) cell count. PMN counts that exceed 250/ml are suggestive of SBP. Cultures of ascitic fluid, taken at the time of paracentesis, are often positive if done properly. The preferred method is to inoculate two blood culture bottles with 10 to 20 mls of ascitic fluid. The treatment of choice for documented SBP is cefotaxime, 1 gram every 8 hours for a minimum of 5 days. Repeat paracentesis should be perfomed to check response to therapy. Patients should be prophylaxed against recurrent SBP by using ciprofloxacin, norfloxacin or trimethoprim/sulfamethoxazole.