Surgical Management of Portal Hypertension

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Portal hypertension is frequently complicated by upper gastrointestinal tract bleeding and ascites. Hemorrhage from esophageal varices is the most common cause of death from portal hypertension. Medical treatment, including resuscitation, vasoactive drugs, and endoscopic sclerosis, is the preferred initial therapy. Patients with refractory hemorrhage frequently are referred for immediate surgical intervention (usually emergency portacaval shunt). An additional cohort of patients with a history of at least 1 episode of variceal hemorrhage is likely to benefit from elective shunt operations. Shunt operations are classified as total, partial, or selective shunts based on their hemodynamic characteristics. Angiographically created shunts have been introduced recently as an alternative to operative shunts in certain circumstances. Devascularization of the esophagus or splenectomy is done for specific indications. Medically intractable ascites is a separate indication for surgical intervention. Liver transplantation has been advocated for patients whose portal hypertension is a consequence of end-stage liver disease. In the context of an increasingly complex set of treatment options, we present an overview of surgical therapy for complications of portal hypertension.

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The portal vein, which provides the principal venous drainage for the splanchnic circulation, arises behind the head of the pancreas where the superior mesenteric and splenic veins join. It courses to the porta hepatis with the proper hepatic artery and common bile duct. Anatomic connections between the portal and systemic circulations exist at the level of the hepatic sinusoids, the gastroesophageal junction, the hemorrhoidal plexus in the rectum, and the Retzius veins in the retroperitoneum. When portal pressure is elevated long term, a patent umbilical vein may direct portal blood into systemic veins within the abdominal wall, resulting in the physical findings of a caput medusae and, often, a venous hum (Cruveilhier-Baumgarten syndrome).

Portal pressure is expressed conveniently as corrected portal pressure (portal pressure minus central venous pressure). The relationship between pressure, flow, and resistance in a circuit is defined by Ohm's law—pressure equals flow times resistance. Normal corrected portal pressure (<10 mm of mercury) increases in proportion to the resistance to the flow of blood from the splanchnic to the systemic venous circulation. Pressure also increases when flow increases. Under conditions of extreme resistance, portal pressure can exceed 50 mm of mercury, but usually collateral channels limit portal pressures to no more than 30 mm of mercury.[1]

Increased splanchnic blood flow associated with a hyperdynamic systemic circulation is commonly seen in portal hypertension.[2] This increased flow is largely shunted through collateral vessels, bypassing the liver. Flow in the portal vein itself typically is reduced in portal hypertension, but may be increased due to neurohumoral influences, which are the subject of intense investigation.[3] Eventually flow may cease or reverse direction. Prograde flow is also called hepatopedal; reversed flow is hepatofugal.

Portal hypertension is classified as presinusoidal or postsinusoidal on the basis of the anatomic location of the resistance to portal flow.[4] Prehepatic obstruction of the portal vein results from congenital atresia, thrombosis, or extrinsic compression. Intrahepatic blockages occur in cirrhosis, inborn errors of metabolism, and schistosomiasis. Schistosomiasis and biliary cirrhosis produce a presinusoidal blockage, whereas alcoholic cirrhosis produces a resistance that is primarily intrasinusoidal or postsinusoidal. Posthepatic portal hypertension is associated with the rare Budd-Chiari syndrome (thrombosis of the hepatic veins or obstruction of the retrohepatic vena cava). The underlying cause of portal hypertension influences the options for therapy. In the United States, alcoholic cirrhosis is the most common cause of portal hypertension. Our discussion is directed principally to the management of patients with this disorder.*

The most urgent indication for the surgical treatment of portal hypertension is hemorrhage. Life-threatening hemorrhage results from the rupture of thin-walled submucosal venous channels in the distal esophagus, which dilate as a consequence of elevated portal pressure and flow. These varices develop to a variable degree in the

*See also the editorial by J. M. Henderson, MD, "Portal Hypertension—The Surgical Pendulum," on pages 554-555 of this issue.

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presence of portal hypertension. Neither the severity of varices nor the likelihood of rupture is directly related to the measured portal pressure. Rather, portal pressure must be elevated above a threshold—about 12 mm of mercury—after which wall tension and local structural factors probably interact to produce hemorrhage.[5] Each episode of hemorrhage carries a mortality of about 50%. After bleeding stops, the likelihood of recurrent bleeding without specific therapy to reduce portal pressure is about 75%.[4]

Another cause of upper gastrointestinal tract bleeding in a patient with cirrhosis, more recently characterized, is portal hypertensive gastropathy, previously thought to be a form of gastritis. Portal hypertensive gastropathy has been shown to be a distinct disorder that is abolished by the reduction of portal pressure.[6] Impaired gastric mucosal barrier function, submucosal edema, and microvasculopathy are characteristic. The endoscopic appearance varies from a diffuse "snakeskin" mosaic pattern to an angry-appearing, beefy-red mucosa with active bleeding. Lesions are most commonly present in the fundus, but may extend anywhere in the stomach. Bleeding is most often chronic or transient. Massive hemorrhage is a less frequent occurrence.

Other causes of upper gastrointestinal tract bleeding are common in alcohol-dependent patients, though not directly related to the presence of portal hypertension. These include gastritis, peptic ulcer, and Mallory-Weiss tears. A thorough evaluation including endoscopic examination of the esophagus, stomach, and duodenum will secure the diagnosis in most cases. This distinction is important because the surgical treatment is different.

Ascites is produced by the transudation of fluid from serosal surfaces of bowel and liver capsule resulting from the altered Starling forces within the hypertensive portal circulation. Impaired synthesis of serum proteins due to underlying hepatic insufficiency also contributes to the formation of ascites.[7] Ascites can become massive and disabling. It can be complicated further by primary or secondary bacterial peritonitis. Outward pressure on the abdominal wall can exacerbate an umbilical hernia and promote skin breakdown, with resultant life-threatening infection of the ascitic fluid.

Child described a useful classification for patients with cirrhosis,[8] comprising the largest cohort of North Americans with portal hypertension (Table 1). This was modified by Pugh and co-workers to include additional criteria.[9] Patients can be stratified according to the Child class to predict mortality for shunt and nonshunt operations. We prefer the original Child classification to its various modifications because it is simpler and sufficiently accurate for rational clinical decision making.

TABLE 1.—*Child's Classification of Cirrhosis**

Clinical Feature	*Class A*	*B*	*C*
Serum bilirubin, grams/liter†	<20	20-30	> 30
Serum albumin, grams/liter†	>35	30-35	< 30
Ascites	None	Controlled	Tense
Encephalopathy	None	Controlled	Severe
Nutritional state	Normal	Adequate	Cachectic
Operative mortality (elective shunt), %	5	10	50

*Modified from Child.[8]
†To convert to conventional units (milligrams/deciliter), multiply by a factor of 0.1.

Initial Therapy for Variceal Hemorrhage

The first priorities in treating acute upper gastrointestinal tract hemorrhage of any cause are protecting the airway and establishing adequate intravenous access. To us, "adequate" means the capability to pass at least two 16- to 18-gauge peripheral catheters and possibly a 9-French introducer sheath.

Fluid resuscitation must be aggressive. A rapid transfusion of warmed whole blood or packed erythrocytes usually is indicated. The hematocrit should be maintained at or above 0.25 to 0.30 (25% to 30%) and rechecked frequently without waiting for posttransfusion equilibration of the extracellular fluid compartment. Coagulation indices—prothrombin time and partial thromboplastin time—should be measured and corrected with the administration of fresh frozen plasma and, if necessary, cryoprecipitate. Bleeding time should be measured; platelet transfusion frequently is required. Timely communication with the blood bank is of utmost importance and may be lifesaving.

A Foley catheter and hourly measurements of urine output are mandatory. Invasive monitoring should be instituted in the event of any hemodynamic instability. Hypotension, which is nearly always due to profound hypovolemic shock, should be treated with a rapid infusion of volume rather than with the administration of dopamine or adrenergic agonists. Metabolic acidosis in this setting is usually due to anaerobic glycolysis with lactate production in hypoperfused tissues; treatment is by the immediate correction of hypovolemia. Hypothermia, hypomagnesemia, and hypocalcemia will occur unless specifically prevented.

Vasopressin or one of its derivatives, usually along with nitroglycerin, is infused intravenously. Vasopressin promotes splanchnic vasoconstriction, thereby decreasing portal flow and portal pressure. Vasopressin is a nonspecific systemic arterial vasoconstrictor that also causes coronary artery vasoconstriction. Nitroglycerin is given to vasodilate the coronary arteries and the portal vein.[10] The coinfusion of intravenous vasopressin and nitroglycerin has been shown to be associated with fewer complications and better control of bleeding than the use of vasopressin alone, although the risk of death is unaltered.[11] Propranolol, a β-adrenergic antagonist, may prevent the first episode of variceal hemorrhage, but its use is not recommended for the control of acute hemorrhage.[12]

After nasogastric lavage, esophagogastroduodenoscopy is done. The site of bleeding is found, and endo-

scopic control of hemorrhage is attempted. Standard endoscopic therapy consists of intravariceal or paravariceal administration of sclerosant agents.[13] Rubber-band ligation through the endoscope has shown promise in a multicenter, randomized trial as a safer and perhaps more effective alternative to sclerotherapy.[14] In a single randomized trial, the use of emergency portacaval shunt was compared with that of endoscopic sclerotherapy for acute variceal hemorrhage.[15] Hospital mortality was equivalent—about 50% for both groups—with a lower incidence of encephalopathy in those treated by sclerotherapy. On this basis, the investigators suggested that sclerotherapy be done first and surgical shunt performed for those patients whose bleeding cannot be controlled endoscopically.

Should bleeding varices be refractory to initial measures, endotracheal intubation and gastric balloon tamponade with the Sengstaken-Blakemore tube are the next steps.[13] At this point, emergency surgical intervention is indicated and should not be delayed by further diagnostic efforts. Active resuscitation should continue until an anesthesiologist can assume care. From a surgeon's perspective, a warm and well-perfused patient has a far better prognosis than one with hypothermia, acidosis, and hypovolemia.

Operations to Reduce Portal Pressure

The portal pressure can be reduced below the critical threshold by various operative techniques. All shunts aim to reduce variceal pressures sufficiently to arrest or prevent bleeding. Surgical shunts are classified as total, selective, or partial shunts based on their specific hemodynamic properties. An angiographically placed stent, or transjugular intrahepatic portacaval shunt (TIPS), is another option that has recently become available in some centers.

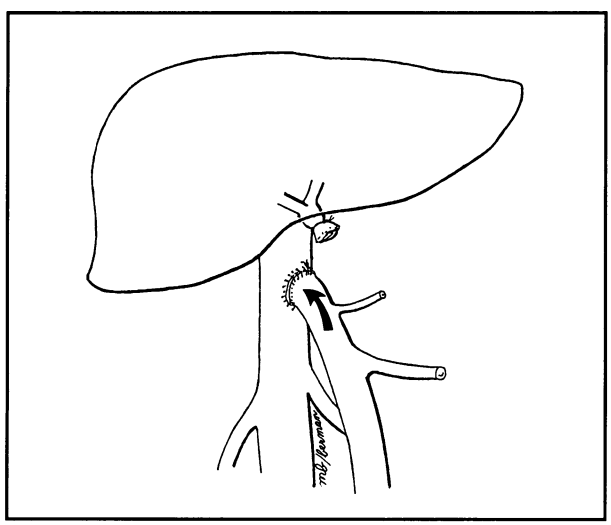

Figure 1.—*End-to-side portacaval shunt:* The portal vein is divided at the hilum of the liver, and its stump is suture ligated. The end of the portal vein is anastomosed to the side of the inferior vena cava. The direction of portal flow is indicated by the arrow. Perfusion of the liver is provided solely by the hepatic artery (not shown).

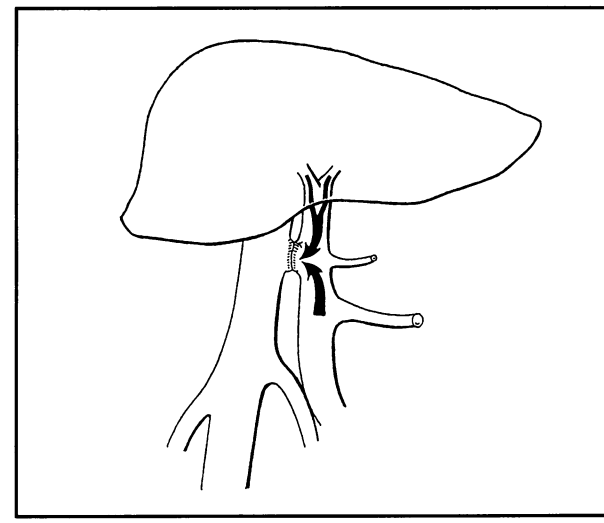

Figure 2.—*Side-to-side portacaval shunt:* The portal vein and inferior vena cava are mobilized and brought into proximity with one another. An anastomosis is created between the side of the portal vein and the side of the cava. Blood flow, indicated by the arrows, is hepatofugal.

Total Shunts

Portal blood flow can be totally diverted to the inferior vena cava by any of several techniques. Total shunts arrest hemorrhage in about 95% of cases but at the price of a 40% to 50% incidence of hepatic encephalopathy. In emergency situations, when bleeding cannot be controlled by less invasive means, total shunts may be lifesaving and encephalopathy becomes a secondary consideration.

The idea of a prophylactic shunt to protect an alcoholic patient with varices from the 50% mortality of the first variceal hemorrhage was once considered attractive. In four controlled trials in the United States that compared the use of total shunts with controls, three demonstrated poorer survival in shunted than in nonshunted patients,[16-18] and one showed no significant difference.[19] Whereas shunting decreased the risk of death from hemorrhage, deaths from liver failure overshadowed this advantage. For this reason, shunts are performed after (or sometimes during) the first episode of hemorrhage.

The end-to-side portacaval shunt (Figure 1) is performed by transecting the portal vein at its bifurcation within the porta hepatis and creating an anastomosis between the end of the portal vein and the side of the inferior vena cava. Thus, all portal flow is diverted around the liver, and the splanchnic system is totally decompressed.

Another total shunt is the side-to-side portacaval shunt (Figure 2). The difference from the end-to-side shunt is that the portal vein is not transected in the side-to-side shunt. The intact portal vein notwithstanding, pressure gradients favor hepatofugal blood flow (away from the liver) with a loss of portal perfusion. This has similar results to the end-to-side shunt—immediate cessation of

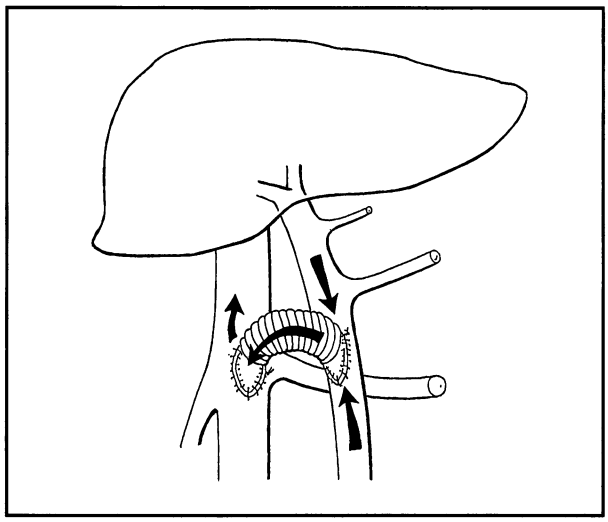

Figure 3.—*Interposition mesocaval shunt (mesocaval H graft):* The superior mesenteric vein and inferior vena cava are connected to one another by a large-diameter prosthetic conduit. The arrows indicate the direction of blood flow, which is hepatofugal.

bleeding and a high incidence of encephalopathy. A variation of this shunt is the large-diameter portacaval H graft (not shown) in which a prosthetic graft about 16 mm in diameter is used to shunt blood from the portal vein to the cava.

A third type of total shunt is the mesocaval shunt. The inferior vena cava is divided and anastomosed end-to-side to the superior mesenteric vein. Alternatively, a prosthetic graft is positioned between the superior mesenteric vein and the inferior vena cava (Figure 3). The technique is similar to the portacaval H graft. An advantage is that the shunt is far enough from the porta hepatis that the required dissection adds little difficulty to subsequent liver transplantation. A disadvantage of the mesocaval H graft is that the graft is relatively long, so there is a greater risk of graft occlusion by kinking or thrombosis. Results are similar to other total shunts.[20]

Selective Shunts

The distal splenorenal shunt (Figure 4) was developed to avoid the high rate of encephalopathy associated with the use of total shunts.[21] Anastomosing the distal splenic vein to the left renal vein selectively decompresses the gastric and splenic veins while maintaining relatively high pressures in the mesenteric and portal veins. Dividing the left gastric (coronary) vein and disconnecting the gastrosplenic and portomesenteric compartments by collateral ligation remain an important part of the procedure.[22,23]

Hepatopedal blood flow is preserved initially, with a low incidence of encephalopathy. In patients with alcoholism, collateral channels tend to dilate over time, eventually converting the selective shunt to a total one.[24] This shunt is rarely used in emergencies because portal decompression is selective, requiring time for bleeding to stop, and the procedure itself is time-consuming.

A meta-analysis of four randomized trials that compared the use of the distal splenorenal shunt with that of sclerotherapy found that the Warren shunt reduced the risk of rebleeding, did not worsen encephalopathy, and improved survival in nonalcoholic patients.[25] Patients with alcoholism did not have improved survival.

As an elective procedure in patients with portal hypertension from causes other than alcoholic cirrhosis, the Warren shunt is an effective and durable operation with extensive application worldwide. In large series, control of hemorrhage is nearly equivalent to that for total shunts (about 85%), and the incidence of encephalopathy is rare (<10%).[26] The Warren distal splenorenal shunt is particularly well suited for managing patients with extrahepatic portal vein thrombosis, of whom about 80% will have a patent splenic vein and thus be candidates for this procedure.[27]

Partial Shunts

Partial shunts were first proposed by Bismuth and associates.[28] Variceal bleeding occurs above a corrected portal pressure threshold of 12 mm of mercury.[5] Partial decompression of the portal vein to a pressure less than the critical threshold should stop variceal hemorrhage while preserving hepatopedal blood flow and preventing encephalopathy. Our investigations, based on Bismuth's concept, led to the clinical application of the small-diameter portacaval H graft.[29,30] A laboratory model predicted preserved hepatopedal flow in 50% of cases using a 10-mm conduit and in 80% using an 8-mm graft.[31] These predictions have been confirmed clinically in our center and

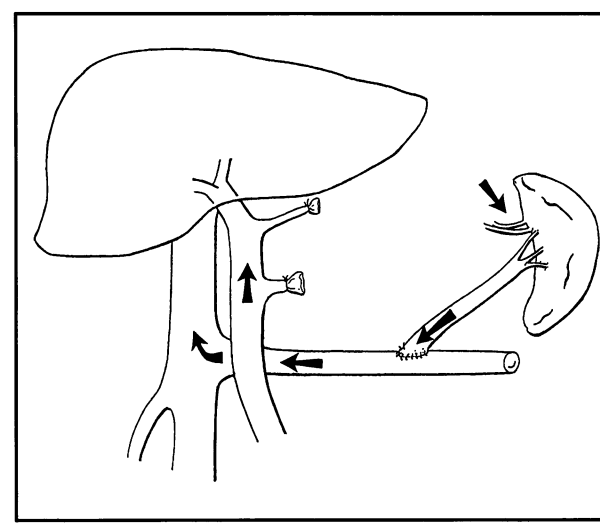

Figure 4.—*Distal splenorenal (Warren) shunt:* The splenic vein is divided near its junction with the superior mesenteric vein and dissected free of the pancreas (not shown). The end of the splenic vein is anastomosed to the distal left renal vein. Selective decompression of the gastric and splenic compartments of the splanchnic circulation is achieved. The portal and mesenteric compartments remain hypertensive. The arrows indicate the direction of blood flow. Portal flow to the liver is maintained, and gastroesophageal varices are decompressed through the spleen into the cava by the left renal vein.

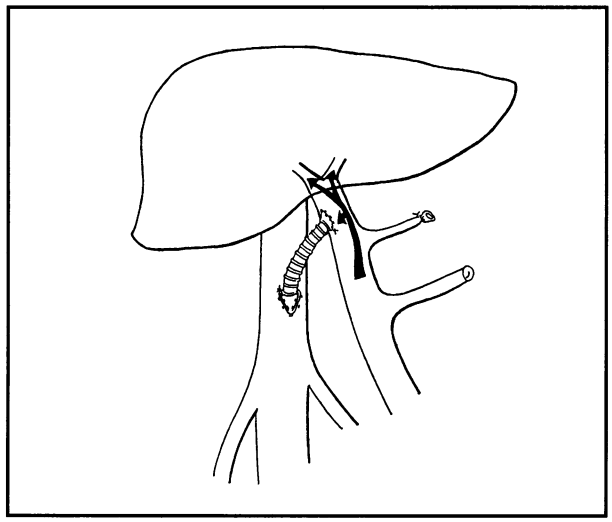

Figure 5.—*Small-diameter portacaval H graft:* The portal vein and inferior vena cava are connected by a small-diameter prosthetic graft. The portal vein is partially decompressed, and portal perfusion of the liver is preserved. The arrows indicate the typical pattern of blood flow, which is hepatopedal in 80% to 90% of patients.

others.[32-35] The small-diameter portacaval H graft consists of a short piece of ringed, expanded polytetrafluoroethylene (Gore-Tex) 8 mm in diameter, which is interposed between the portal vein and vena cava (Figure 5).

We recently reported a prospective, randomized trial comparing the use of large-diameter total shunts with that of small-diameter partial shunts.[36] Both shunts were 100% effective in controlling hemorrhage. Partial shunts preserved hepatopedal flow in 90% of patients and had a notably reduced incidence of encephalopathy. Long-term patency rates exceed 95%.

We recommend using small-diameter H grafts for patients with Child class A or B alcoholic cirrhosis and at least one previous episode of variceal hemorrhage.[37] Although this procedure has succeeded for the emergency control of bleeding and in patients with Child class C cirrhosis, the associated high mortality (around 50%) is unacceptable. In class C cirrhosis that cannot be improved by medical management, a rational alternative is TIPS followed by liver transplantation (see the next section). For acute life-threatening hemorrhage, the experience of the surgeon should determine the choice of shunt.

A New Procedure—Transjugular Intrahepatic Portacaval Shunt

Angiography has long been an important diagnostic method for planning elective surgical shunts. Recently, invasive angiographic techniques have been applied to the control of variceal hemorrhage. Transjugular intrahepatic portacaval shunt has been performed in many centers, and short-term data on uncontrolled, large series of patients are beginning to appear.[38,39] Initial enthusiasm has given way to cautious optimism and an effort to define more clearly the subset of patients most likely to benefit from the use of this technique.[40] The results of an uncontrolled series suggest that TIPS is particularly effective as a "bridge" to transplantation for patients with end-stage liver disease.[41]

The procedure is done by means of a percutaneous puncture of the right internal jugular vein. Structures are located by fluoroscopy and ultrasonography. Using a modification of the Seldinger technique, a guide wire is inserted into an intrahepatic branch of a hepatic vein. A needle is advanced over the guide wire through the substance of the liver into a nearby branch of the portal vein. The resulting tract is dilated with a balloon. An expandable stent of 8 to 10 mm in diameter is positioned to maintain patency of the communication between hepatic and portal veins (Figure 6). A patent portal vein is necessary for the performance of TIPS.

The advantages of TIPS include immediate portal decompression, the avoidance of general anesthesia, and a lack of intrusion into the portal hepatis.[42] Disadvantages include technical failure,[38,39,43] shunt stenosis or thrombosis in 30% to 50% of patients at one year,[39,43,44] with the possibility of rebleeding and other complications such as shunt migration or intra-abdominal hemorrhage. The rate of the latter complications is related to the experience of the interventionist, but stenoses and thromboses of the stents are thought to be due to neointimal hyperplasia. As the procedure continues to be refined, it is anticipated that the stenosis and thrombosis rates will improve.

Reported early mortality for emergency TIPS ranges from 30% to 56%.[39,43] The lower figure compares favorably with the approximately 50% operative mortality for emergency portacaval shunt, and the higher figure is not substantially worse. Early mortality for elective TIPS has been less than 10% in major series[39,43]; this is comparable to elective surgical shunts. Because a reporting bias is likely to exist with any new procedure (favorable results tend to be reported more often), prospective randomized trials are needed to determine the actual mortal-

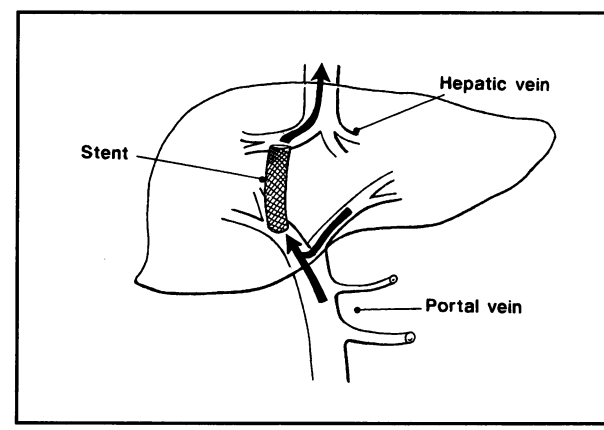

Figure 6.—*Transjugular intrahepatic portacaval shunt (TIPS):* By percutaneous access to the jugular vein, an expandable stent is positioned within the liver parenchyma to achieve a functional connection between a branch of the portal vein and a branch of the hepatic vein. The arrows indicate the direction of blood flow. Portal flow is directed through the stent to the superior vena cava. For clarity, the inferior vena cava is not shown.

ity. Mortality data stratified by Child class will be particularly valuable.

One group has cautioned against uncritical acceptance of TIPS for all patients with variceal hemorrhage.[43] These authors suggest that TIPS is most appropriate for patients with Child-Pugh class C cirrhosis and those awaiting liver transplantation. They emphasize stabilizing the patient and using standard modalities for the control of hemorrhage—endoscopic sclerotherapy, splanchnic vasoconstrictors and balloon tamponade—before TIPS is done. Proper resuscitation beforehand and participation by experienced anesthesiologists during emergency TIPS may improve results in patients with acute hemorrhage.

Other Operations for Complications of Portal Hypertension

Esophageal Transection and Devascularization

Sugiura and Futagawa described a complex procedure for the control of variceal bleeding, consisting of dividing and reanastomosing the gastroesophageal junction, followed by suture ligating the remaining collaterals on the surface of the stomach.[45] Sugiura's original operation requires doing both a laparotomy and a thoracotomy. The goal of the procedure is to directly obliterate varices and to occlude their inflow through dilated collateral vessels. Outcomes with this procedure in Japan, where nonalcoholic cirrhosis is the prevailing cause of portal hypertension, generally have been excellent.[46] Experience outside Japan has been less consistent.[47] Rebleeding rates vary widely. Hospital mortality, as with shunt procedures, is related to the severity of underlying liver disease and to the urgency of the procedure. Substantial complications can include anastomotic leaks and stenoses. The rates of encephalopathy are uniformly low, around 5% to 10% overall.[46,47]

A less complicated modification involves simultaneous transection and reanastomosis of the distal esophagus using a surgical circular stapler introduced into the esophageal lumen through an incision in the stomach.[48] This can be combined with suture ligation of the left gastric vein. The aim is to interrupt inflow to the varices without obliterating them directly. In four controlled trials comparing esophageal transection with sclerotherapy, hospital mortality was similar, and early rebleeding was higher after sclerotherapy in three of the trials.[49-52] Late rebleeding is not prevented by esophageal transection. One of these trials also compared the use of transection with that of total shunts[52]; mortality was no different, and control of bleeding was better in the group receiving the shunts.

For patients for whom medical therapy fails, but who cannot be shunted (because of portal vein thrombosis, for example), esophageal transection and reanastomosis is a satisfactory option. Those with better residual liver function will have better outcomes.[53] In addition, this potentially lifesaving procedure may be done in an acutely bleeding patient by surgeons who lack experience with emergency portacaval shunts.

Splenectomy

Thrombosis of the splenic vein may result in bleeding from gastric varices arising from the short gastric veins. In these uncommon cases, splenectomy is the definitive treatment.[54] Polyvalent pneumococcal vaccine and counseling about the possibility of overwhelming postsplenectomy sepsis syndrome should be given. Portal hypertension-associated hypersplenism with thrombocytopenia is not an indication for splenectomy.[55]

Surgical Treatment of Ascites

The medical control of ascites requires salt and water restriction, drug therapy, and maintaining serum protein levels adequate to exert normal intravascular oncotic forces.[56] Spironolactone antagonizes the elevated circulating aldosterone levels associated with portal hypertension. Loop diuretics such as furosemide enhance the excretion of excess extracellular fluid, but necessarily cause intravascular volume depletion at the same time. Therapeutic paracentesis is used as an adjunct to these measures.

Therapy may be limited by various competing factors. Salt and water restriction can be difficult to enforce. When it succeeds, the resulting hypovolemia may predispose to renal insufficiency, especially when diuretics are given during a state of prerenal azotemia.[56] Hepatic protein synthesis may be impaired by the underlying disease. Frequent therapeutic paracentesis may exacerbate the depletion of serum proteins. Adequate dietary protein intake may worsen encephalopathy.

In circumstances where medical measures cannot control ascites, portacaval shunts may do so for patients who otherwise meet the indications for shunting.[57] Total portacaval shunts will decrease ascites substantially over time, although at a cost of worsened encephalopathy in about 40% to 50% of patients. Transjugular intrahepatic portacaval shunt may act hemodynamically as a total or partial shunt[38,39] and appears to decrease ascites, but it also increases the incidence of encephalopathy. The ultimate role of TIPS in treating ascites remains to be defined. Selective and partial shunts are not reliable for controlling ascites, although usually they do not worsen it and may reduce it in some cases.[30,58] Portacaval shunts for ascites should be offered only to patients with a history of variceal bleeding in addition to refractory ascites. The choice of shunt procedure may be influenced by the added goal of controlling the ascites.

For the specific control of ascites, two types of peritoneovenous shunts are available in the United States.[59] The LeVeen shunt consists of a silicone (Silastic, Dow Corning Corp, Midland, Michigan) conduit with a passive, pressure-actuated, one-way valve. One end is placed in the peritoneal cavity by a minilaparotomy. The shunt is tunneled subcutaneously to the neck where the other end is secured in the internal jugular vein. The Denver shunt is similar except that its one-way valve is placed over a rib where it can be actively pumped by external compression. Both shunts aim to immediately recirculate ascites

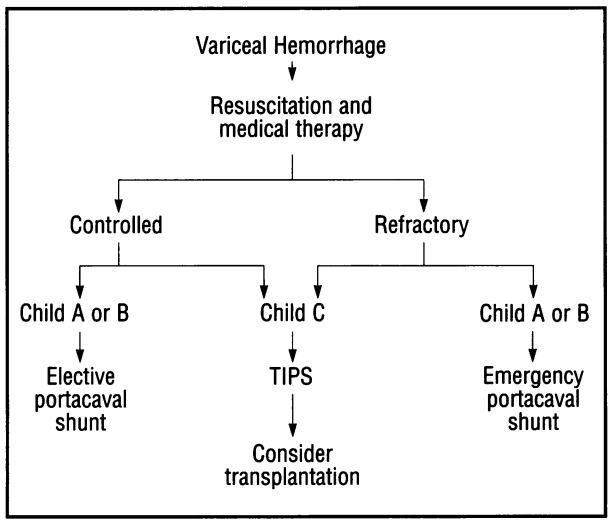

Figure 7.—An algorithm is given for the management of variceal hemorrhage. The urgency of a transjugular intrahepatic portacaval shunt (TIPS) will depend on whether or not hemorrhage can be controlled medically.

fluid into the vascular compartment. This is associated with a consumptive coagulopathy that may progress to disseminated intravascular coagulopathy.[60] Severe cases of coagulopathy are treated by ligation of the shunt. Other complications include congestive heart failure from volume overload, infection, venous thrombosis, and eventual occlusion.[61] In patients in whom the shunts work as designed, ascites is abolished, but the potential for variceal hemorrhage may be increased due to hypervolemia and coagulopathy. A large, prospective, randomized trial failed to show a survival benefit of the use of peritoneovenous shunt over medical therapy for massive ascites in alcoholic patients, although ascites was treated more effectively by surgical procedures.[62] As a last resort, peritoneovenous shunts may improve the quality of life for patients with refractory, disabling ascites but with considerable risk.

Liver Transplantation

Liver transplantation has become increasingly successful in recent years because of improvements in organ preservation, surgical technique, critical care, and immunosuppression.[63] Candidates for transplantation now may include alcoholic patients with end-stage cirrhosis. Transplantation for the treatment of alcohol-induced liver failure even in nonabstinent patients has been advocated by one prominent group because results are comparable to transplantation for other causes of liver failure, and their patients tend not to resume alcohol abuse after the transplantation.[64]

Indications for transplantation are not the same as those for portacaval shunt. Although the transplantation does correct portal hypertension, the goal of therapy is to restore hepatic function. Patients with variceal bleeding who have adequate hepatic reserve or reversible liver failure are shunted instead. Those with end-stage liver failure (manifested by persistent jaundice, encephalopathy, and inadequate synthetic capacity) are considered for transplantation.[65]

Once the need for transplantation is established, a patient undergoes an extensive medical and psychosocial evaluation to determine whether contraindications to transplantation exist. Absolute contraindications include sepsis, active substance abuse, a malignant neoplasm outside the liver, the failure of a second organ system, or the acquired immunodeficiency syndrome. Relative contraindications include noncompliance, intrahepatic carcinoma, advanced age, and multiple previous operations.[65] Portal vein thrombosis, until recently an important obstacle to transplantation, can now be dealt with using techniques to bypass or reconstruct the obstructed portal vein.[66] The requirement for immense financial resources and the serious nationwide shortage of organ donors may prove to be insurmountable barriers for many patients who might otherwise benefit from transplantation.

Patients who have undergone previous portacaval shunts may present technical challenges to a transplantation team. For this reason, it is important to weigh the likelihood of a future need for transplantation against the immediate need for controlling hemorrhage when choosing initial therapy for variceal bleeding. Mesocaval shunts, the distal splenorenal shunt, and TIPS are considered less likely to cause difficulty for a transplantation surgeon when compared with other shunt procedures.[67] Still, none of these is an ideal procedure in all circumstances, and decisions as to the preferred shunt operation must be made on an individual basis.[68]

Summary of Recommendations

Life-threatening variceal hemorrhage due to portal hypertension requires a rapid evaluation and aggressive resuscitation. Ideally, diagnostic measures and supportive treatment will be instituted simultaneously. Medical treatment will control acute bleeding in most patients. Decisions regarding definitive management then can be approached systematically. Factors such as the cause of portal hypertension, adequacy of hepatic reserve, possible candidacy for liver transplantation, and likely compliance with therapy can be assessed. Local skills will also influence management.

In the few patients whose hemorrhage proves refractory to initial therapy, emergency portacaval decompression should be done without delay. For patients with satisfactory hepatic reserve (Child class A and B), surgical portacaval shunt will arrest variceal hemorrhage with acceptable operative mortality. For those with end-stage liver disease (Child class C), TIPS followed by liver transplantation is indicated. Not all centers will offer these specialized procedures, and variations from this outline will often be justified by individual circumstances.

A simplified algorithm depicting our overall scheme for managing variceal hemorrhage is given in Figure 7. "Resuscitation and medical therapy" includes airway management, fluid resuscitation, the transfusion of blood products, pharmacotherapy, endoscopic control of bleed-

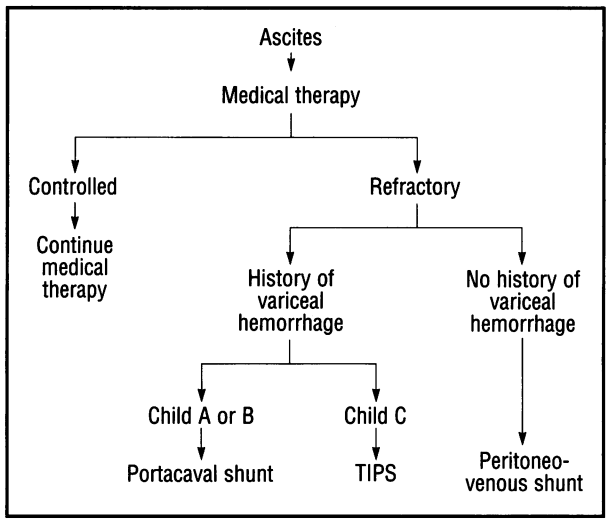

Figure 8.—An algorithm is shown for the management of ascites. Intractable ascites may be one of several factors suggesting the diagnosis of end-stage liver failure. For such patients, liver transplantation is the best therapy. TIPS = transjugular intrahepatic portacaval shunt

ing by sclerotherapy or ligation, and possible tamponade with the Sengstaken-Blakemore tube. It is reasonable to attempt endoscopic control of hemorrhage twice within a period of a few hours before declaring the failure of medical therapy and referring the patient for emergency surgical intervention. Resuscitative efforts must be vigorous and continuous, regardless of the therapy chosen. It is prudent to notify a surgeon (who may be asked to do an emergency procedure on short notice) early in the patient's hospital course.

Ascites is controlled with medical therapy—diuretics, salt and water restriction, and periodic paracentesis—in most patients. For those whose ascites proves refractory, surgical therapy may improve the quality of life. Patients with a concomitant history of variceal bleeding should be managed by portacaval shunt. This prevents future episodes of hemorrhage and controls ascites. Those who have never bled from varices may benefit from peritoneovenous shunting, so long as they are willing to accept the risks of bacterial peritonitis, congestive heart failure, intravascular coagulopathy, variceal bleeding, and shunt occlusion. Our simplified algorithm for the surgical treatment of ascites is shown in Figure 8.

End-stage liver failure is best treated by liver transplantation. For patients with sufficient resources who meet the indications and have no absolute contraindications, replacement of the liver is the definitive therapy for portal hypertension and its complications.

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