A Systematic Appraisal of Portacaval H-Graft Diameters

Clinical and Hemodynamic Perspectives

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Over a period of 10 years, the authors have systematically reduced portacaval H-graft diameters. Their objective was to achieve partial shunting of portal flow without reversal of hepatic flow. This report summarizes their clinical and hemodynamic observations in 68 surviving patients with cirrhosis (mostly alcoholic) and variceal hemorrhage who underwent portacaval H-grafts ranging from 20 to 8 mm diameters. When shunt diameters were reduced to 10 and 8 mm and combined with aggressive portal collateral ablation, portal pressures increased significantly over larger H-grafts. Only 3% of patients with 20–12 mm H-grafts had prograde portal flow after operation, compared with 46 and 82% after 10 and 8 mm H-grafts, respectively ($p < 0.001$). The incidence of encephalopathy diminished from 39% in the 20–12 mm H-graft group to 19 and 9% after 10 and 8 mm grafts, respectively ($p < 0.04$). None of the patients with 10 or 8 mm PTFE grafts rebled from varices in the follow-up period (4–61 months). It is concluded that partial shunting of portal flow is hemodynamically feasible. It can be achieved in most patients using 8 mm polytetrafluoroethylene (PTFE) portacaval H-grafts combined with portal collateral ablation. Preserving prograde portal flow by partial shunting correlates with reduced encephalopathy rates after operation. Despite maintaining a relatively hypertensive portal system, partial shunts effectively prevent variceal hemorrhage.

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WE BEGAN PERFORMING portacaval H-grafts more than 10 years ago for two reasons. First, a direct side-to-side anastomosis could be a difficult and time-consuming procedure. Second, we and others experienced a high failure rate of the mesocaval shunt for controlling variceal hemorrhage.[1-5] Large diameter (16–20 mm) portacaval H-grafts effectively prevented variceal rebleeding. However, all patients lost prograde portal flow, and hepatic encephalopathy occurred frequently. To determine if maintaining hepatic portal perfusion is important for preventing hepatic encephalopathy, we systematically reduced portacaval H-graft diameters. We postulated that we would ultimately find an H-graft diameter small enough to preserve prograde portal flow in most patients.

Our studies were conducted in four phases, each phase examining the hemodynamic and clinical results of progressively smaller H-graft diameters. This report summarizes a decade of investigations leading to the concept of partial shunting for control of variceal hemorrhage in alcoholic cirrhosis.

Materials and Methods

Our investigations of portacaval H-grafts (PCHG) were initiated in 1975 and were conducted in four phases: (1) 20–16 mm diameter Dacron® H-grafts; (2) 14–12 mm Dacron H-grafts; (3) 10 mm polytetrafluoroethylene (PTFE) H-grafts with portal collateral ligation; (4) 8 mm PTFE H-grafts with collateral ligation.

A total of 88 patients were entered into the studies conducted first at Albany Medical College, then at University of California, Irvine, affiliate hospitals. The majority (93%) of patients had biopsy-proven alcoholic cirrhosis. All were documented to have bled from gastroesophageal varices. Patients with preoperative thromboses of the portal system undergoing portacaval H-grafts are not entered into the present studies, most having been reported on previously.[6]

Twenty patients died within 3 months of operation. Their operative circumstance, shunt status, and causes of death are shown in Table 1. These patients are excluded from further consideration in the remainder of our report.

After operation, all survivors were followed during frequent visits to private and institutional clinics. Eight patients were lost to follow-up from 3 to 31 months after

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TABLE 1. *Analysis of Operative Mortality after Portacaval H-Graft*

Patient #	H-Graft Diameter (mm)	Preoperative Child's Class	Operative Circumstance	Time Interval until Death	Shunt Status	Cause of Death
1	20	B	Emergency	2 weeks	Patent (autopsy)	Hepatic failure
2	20	C	Emergency	4 weeks	Patent (autopsy)	Hepatic failure
3	18	B	Emergency	24 hours	Undetermined	Intra- and postoperative coagulopathy
4	18	B	Elective	6 weeks	Patent (autopsy)	Ruptured hepatoma
5	18	C	Emergency	1 week	Undetermined	Sepsis and hepatic failure
6	16	C	Elective	2 weeks	Patent (autopsy)	Aspiration pneumonia
7	16	C	Emergency	11 weeks	Undetermined	Hepatic failure
8	16	C	Emergency	3 days	Patent (autopsy)	Multiple systems failure
9	14	B	Emergency	5 days	Undetermined	Infected ascites, liver failure
10	12	C	Emergency	10 days	Patent (angio)	Hepatorenal failure
11	12	C	Emergency	2 weeks	Patent (autopsy)	Cecal perforation
12	12	C	Emergency	<2 days	Undetermined	Intra- and postoperative coagulopathy
13	12	C	Emergency	2 weeks	Patent (angio)	Hepatic failure
14	10	C	Emergency	2 weeks	Patent (autopsy)	Hepatorenal failure
15	10	C	Emergency	3 days	Undetermined	Hepatic failure and sepsis
16	10	C	Emergency	2 weeks	Patent (autopsy)	Hepatic failure
17	10	B	Emergency	3 weeks	Patent (angio)	Renal failure
18	10	C	Emergency	5 weeks	Patent (angio)	Hepatic failure
19	8	B	Emergency	2 weeks	Graft infection (operation)	Sepsis
20	8	C	Emergency	5 weeks	Patent (angio)	Hepatic failure and sepsis

operation. The average follow-up in months (and range) for each phase is as follows: Phase 1: 24 (range: 4–51); Phase 2: 33 (range: 6–56); Phase 3: 31 (range: 7–61); Phase 4: 23 (range: 4–43).

All patients had postoperative angiographic studies. Eighteen patients had postoperative venous phase superior mesenteric arteriography to assess portal perfusion. Fifty underwent transfemoral shunt cannulation. Portal and vena caval pressures were obtained by direct manometry during these studies. Graft patency and the direction of portal flow were determined fluoroscopically.[7] Since 1980 we have added scintigraphy to fluoroscopy to confirm portal flow direction.[8] In addition, 32 of 37 patients with 8 or 10 mm PCHGs have had repeat angiography from 6 months to 5 years after their initial studies as an ongoing study of PTFE grafts in the portal system. Any patient with upper intestinal bleeding after operation underwent endoscopy followed by angiography to assess shunt patency.

Before operation, we assigned each patient's Child's category on the day of the operation. After operation, we carefully screened patients for evidence of hepatic encephalopathy once they had fully recovered from their operations. Hepatic encephalopathy was diagnosed if family members reported confusion, memory loss, or personality changes. Presence of asterixis or hospitalization for any neurological symptoms were also used as criteria for encephalopathy. All such patients were treated with lactulose and/or restriction of dietary protein.

Operative Technique

The operative technique has evolved over the course of the study. We are currently using 8 mm ringed PTFE grafts. Supported grafts develop a gentle bow after releasing traction on the liver, unlike unsupported PTFE grafts, which tend to kink or are compressed by adjacent viscera. The graft length usually required is between 3 and 5 cm.

Long bevels are fashioned at both ends of the graft and placed at a 90-degree rotation to each other. These bevels conform with the angles of the vena caval and portal vein anastomoses and double the cross-sectional area for the anastomosis. Each graft is placed in a closed 20 ml syringe filled with heparin, and air is removed from the interstices of the graft by alternatively compressing and releasing the plunger.[9]

An extended right subcostal incision is used. The lateral aspect of the portal vein is exposed first, then the anterior surface of the vena cava. The dissection is limited to accommodate the width of a small Satinsky clamp. The vena caval anastomosis is performed first. The pre-cut bevel causes the graft to angle approximately 30 degrees cephalad from the anterior surface of the vena cava. The bevel at the portal vein anastomosis produces a 30–45

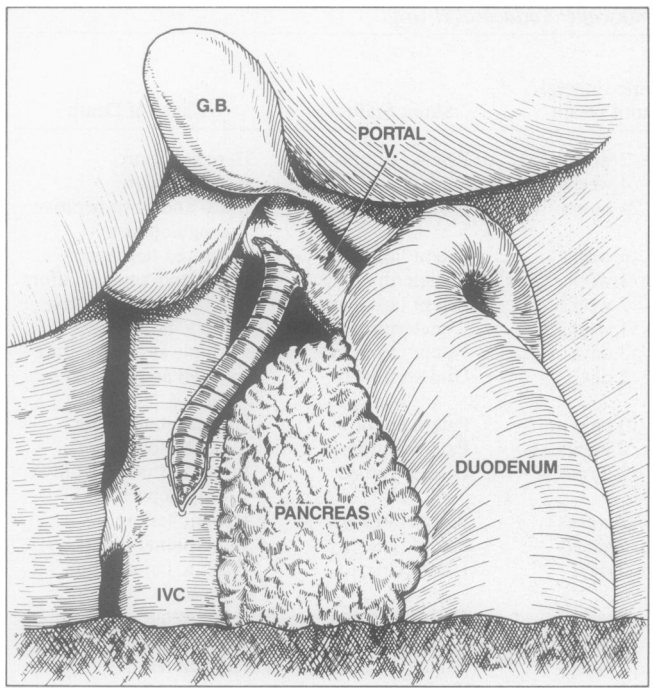

FIG. 1. Schematic diagram of portacaval H-graft.

degree caudad angle from the lateral surface of portal vein (Fig. 1).

A simple continuous suture can produce excessive inversion of the anastomosis. Therefore, a continuous everting horizontal mattress technique is used. The portal anastomosis is facilitated by pre-placing the entire posterior suture line before coapting the graft to the portal vein. Before the portal anastomosis is completed, the graft is flushed with heparinized saline to remove thrombi, fibrin, and other tissue debris.

After the clamps are removed, a thrill should be palpable in the vena cava near the anastomosis. Its absence should be investigated. If there is any doubt, a small opening (1–2 mm) is made in the anterior midportion of the graft. A 1 ml Fogarty balloon catheter is passed through both anastomoses and withdrawn with the balloon inflated. This maneuver removes small thrombi from the portal vein (accumulated during clamping) and ensures that the anastomoses are free of technical errors. Following construction of the shunt, portal collateral veins are interrupted. The umbilical vein is divided at the liver edge. The gastroepiploic veins are interrupted in continuity with surgical clips. The large periesophageal veins on the medial side of the esophagus are also suture-ligated. The coronary vein is suture-ligated in the lesser sac as it enters the gastrohepatic ligament. The inferior mesenteric vein is interrupted at the ligament of Trietz. The aim of collateral ligation is to direct more portal flow towards the liver and shunt. It also provides an additional protective measure against continued hemorrhage in actively bleeding patients.

Routine angiography is performed within 1 week of operation on all patients. The portal system is cannulated through the H-graft *via* a percutaneous transfemoral approach. A small amount of contrast is hand-injected into the portal vein cephalad to the anastomosis. Direction of portal flow is determined by observing the flow of contrast fluoroscopically. The angiographic catheter is then passed into a peripheral mesenteric or splenic tributary, and serial angiograms are obtained. Any large unligated portasystemic collateral veins are embolized using Gianturco coils. Before completing the procedure, portal and vena caval pressures are measured by direct manometry.

Early shunt thrombosis is managed by inflating a Gruntzig balloon catheter in the shunt. Once some flow is established, an angiography catheter is left with the tip in the portal vein adjacent to the anastomosis. Streptokinase is infused at 5000 U/h for 24 hours, and angiography is repeated.

Results

The investigations of PCHG were conducted in four phases. Of the 68 surviving patients, 19 had 20–16 mm grafts, 12 had 14–12 mm grafts, 26 had 10 mm grafts, and 11 had 8 mm grafts. The clinical and hemodynamic results of the four phases are summarized in Figures 2–4.

Hemodynamic Findings

Mean corrected portal pressures (portal minus caval pressure) significantly increased when shunt diameter was reduced to 10 mm and combined with aggressive portal collateral ligation. Reducing shunt diameter to 8 mm resulted in a relatively hypertensive portal system (17 ± 4 cm water). These corrected portal pressures were more than four times greater than those after 20–16 mm diameter H-grafts (Fig. 2).

No patients with 20–16 mm diameter PCHG had prograde portal flow after operation. Only one patient in the 14 or 12 mm PCHG group had prograde flow. When shunt diameter was reduced to 10 mm and combined with collateral ablation, prograde flow was achieved in 12 of 26 patients. With 8 mm shunts, nine of 11 patients had prograde portal flow after operation (Fig. 3).

Postoperative Encephalopathy

Postoperative encephalopathy rates decreased as the shunt diameters were reduced to 10 and 8 mm (Fig. 4). Combined, the incidence of encephalopathy was 39% in patients with 20–12 mm PCHG, in contrast to 16% incidence in patients with 10 and 8 mm PCHG ($p < 0.04$).

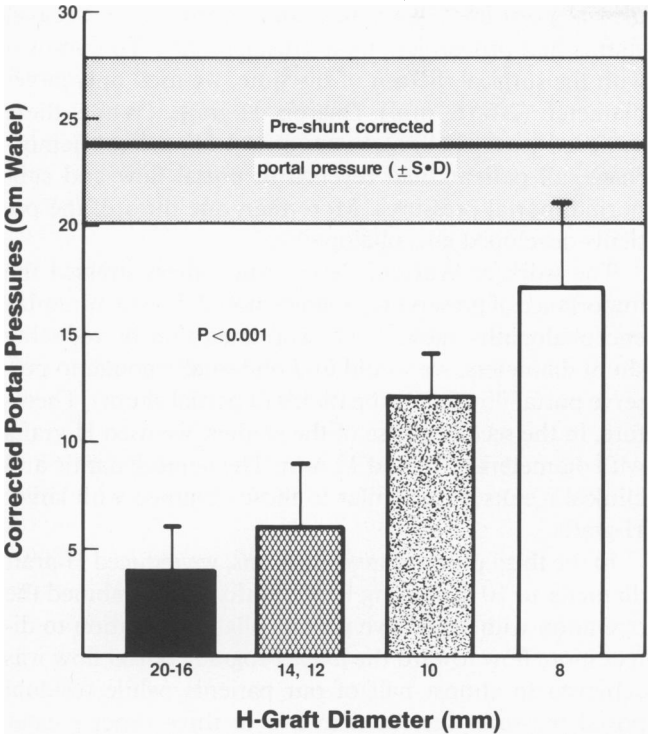

FIG. 2. Corrected (portal–caval) pressures (±SD) following portacaval H-grafts of diminishing diameters.

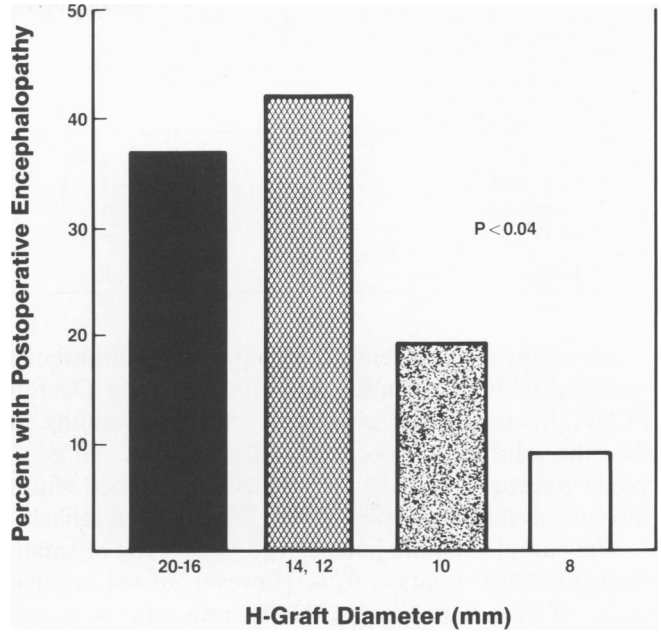

FIG. 4. Incidence of postoperative encephalopathy (%) with diminishing portacaval H-graft diameters.

This difference could not be explained by distribution of patients according to preoperative risk factors. There was no significant difference in distribution of Child's classes among the several shunt-diameter groups (Table 2). The proportion of emergency shunts was also not significantly dissimilar (35% for the 20–12 mm group and 30% for the 10–8 mm group).

Overall, the incidence of encephalopathy was 35% after operation in patients with reversed portal flow, compared with 9% in patients with prograde flow ($p = 0.02$).

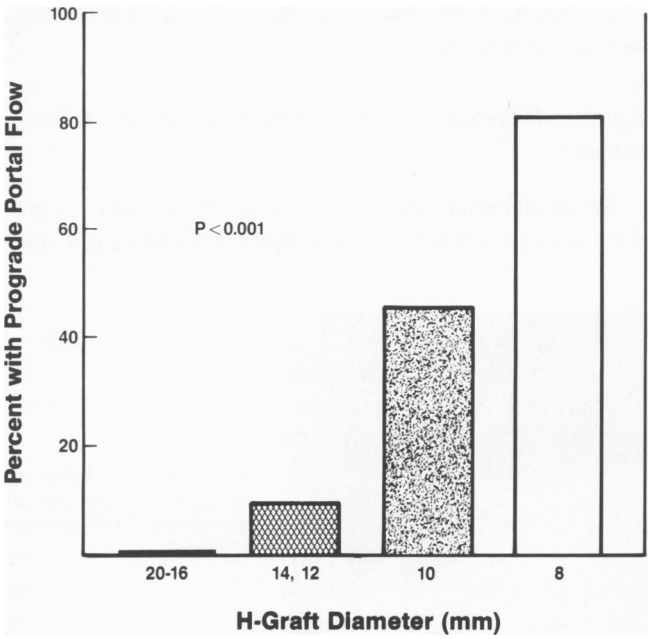

FIG. 3. Incidence of prograde portal flow (%) with diminishing portacaval H-graft diameters.

Rebleeding and Thrombosis Rates

Causes of upper gastrointestinal hemorrhage following PCHG are shown on Table 3. Except in three patients, the nonvariceal bleeds were minor and self-limited. In two patients with 8 and 10 mm shunts, alcohol-induced hemorrhagic gastritis 6 and 7 months after operation was severe enough to cause death from hepatic failure. In another patient with a 10 mm PCHG, bleeding from a gastric ulcer required partial gastrectomy for control. He recovered uneventfully.

TABLE 2. *Distribution of Patients According to Preoperative Child's Criteria*

H-graft Diameter (mm)	Child's Class			
	A	B	C	Total
20, 18, 16	10	8	1	19
14, 12	5	7	0	12
10	14	9	3	26
8	6	5	0	11
Total	35	29	4	68

TABLE 3. *Causes of Rebleeding after Portacaval H-Graft*

	H-Graft Diameter (mm)	
	20–12	10 or 8
Gastritis	2	4
Gastric Ulcer	—	1
Undetermined	—	1
Varices	2	—
Total	4	6

Recurrent variceal hemorrhage after shunt thrombosis occurred in two patients, both with a 14 mm Dacron PCHG. In one, thrombosis occurred within 6 months. In the other, thrombosis occurred within 4 years. An additional patient with a 10 mm PCHG developed shunt thrombosis following splenectomy.[7] He has not rebled.

The cumulative late patency (up to 5 years) of small-diameter PTFE grafts is 97%. However, of the original group of 44 patients having PTFE grafts (survivors and nonsurvivors), seven (16%) developed acute shunt thrombosis in the perioperative period. In one patient, intra-abdominal sepsis with graft infection resulted in death within 2 weeks of operation (Table 1). In the remaining six patients, perioperative graft thrombosis was manifested by increasing ascites and diminishing urinary output and sodium within 48 hours of operation. Angiography confirmed the diagnosis. The first three patients had successful operative revision of the shunts. The grafts of the last three patients were successfully declotted angiographically (see Methods) and have remained patent by follow-up angiography from 3 to 24 months later.

Figures 5 and 6 are angiograms at 4- and 5-year intervals in two patients with 10 mm PTFE grafts. Both have maintained prograde portal flow.

Discussion

Our systematic appraisal of PCHG diameters was conducted in four phases. In the first phase we investigated the feasibility and safety of prosthetic material in the portacaval position. Experience with prosthetic mesocaval shunts had proven less than satisfactory.[1-3] To conform with the surgical dictum of the time, we used only large-diameter (20–16 mm) Dacron H-grafts. While these achieved excellent long-term control of variceal hemorrhage, all patients had reversal of portal flow and subnormal portal pressures. More than one third of the patients developed encephalopathy.

The work of Warren, Zeppa, and others stressed the importance of preserving prograde portal flow to minimize encephalopathy rates.[10] We postulated that by reducing shunt diameters, we would find one small enough to preserve portal flow in most patients (a partial shunt). Therefore, in the second phase of the studies, we used H-grafts with diameters of 14 and 12 mm. The hemodynamic and clinical results were similar to those obtained with larger H-grafts.

In the third phase of investigations, we reduced H-graft diameter to 10 mm using PTFE grafts. We combined the operation with aggressive portal collateral ablation to direct more flow toward the liver. Prograde portal flow was achieved in almost half of our patients, while residual portal pressures increased to two or three times greater than those after larger H-grafts. Therefore, the critical graft diameter at which portal hemodynamics change is 10 mm.

In the fourth phase, we reduced PTFE graft diameter to 8 mm. Prograde portal flow was achieved in more than 80% of patients, and the portal system remained relatively hypertensive. Our objective of producing partial shunts in a majority of patients was attained.

In achieving the goal of partial shunting, our decade of investigations produced other findings with important implications in the field of portal hypertension surgery, as explained below.

Efficacy of Partial Shunts in Preventing Variceal Rehemorrhage

Small diameter (10 or 8 mm) portacaval H-grafts prevent variceal rehemorrhage despite maintaining a rela-

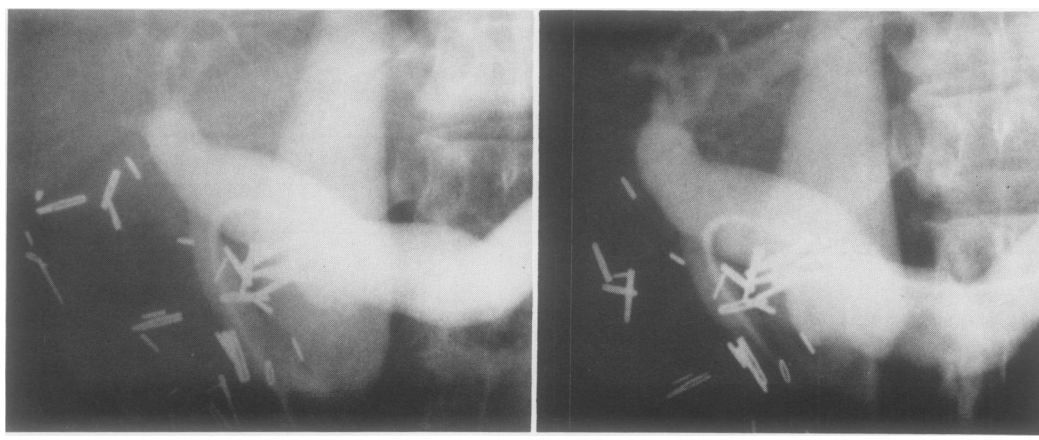

FIG. 5. Early and follow-up angiography (4-year interval) after 10 mm PTFE portacaval H-graft.

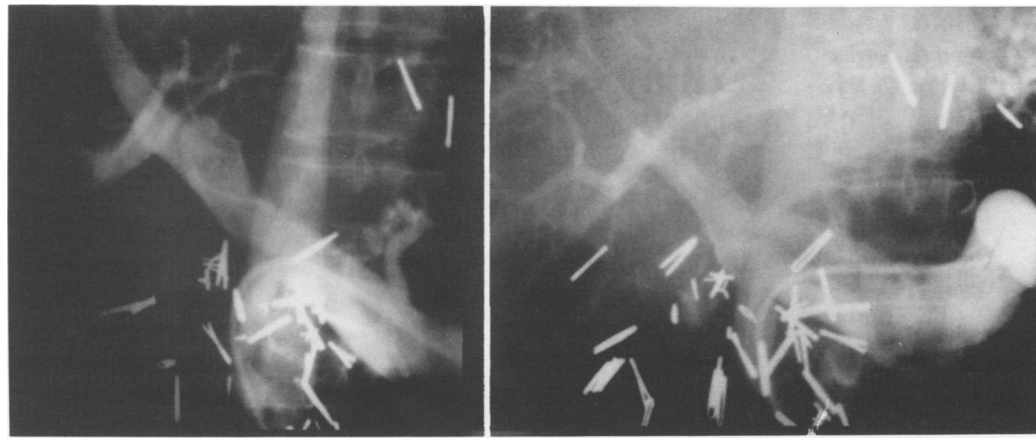

FIG. 6. Early and follow-up angiography (5-year interval) after 10 mm PTFE portacaval H-graft.

tively hypertensive portal system. Although aggressive collateral ablation may add further protection, several patients with unligated coronary veins have not rebled from varices. These observations suggest that each patient has a "threshold" pressure at which varices are at risk for bleeding. As long as pressures are maintained below the threshold, hemorrhage is prevented. Our results dispute the widely held belief that only total decompression of variceal pressure is therapeutically effective.

The Use of PTFE Grafts in the Portal System

Although we initially used Dacron grafts in the 20–12 mm diameter group, this may not be the best material for interposition shunts. Others have found a high late thrombosis rate.[5,6] We therefore elected to use PTFE grafts for 10 and 8 mm shunts. The 97% late patency rate (up to 5 years) with these grafts attests to their efficacy. Our major difficulty with PTFE has been an overall 16% rate of perioperative thrombosis, a problem not encountered with Dacron grafts. We are studying whether the use of ringed PTFE grafts, perioperative antiplatelet therapy, and forceful injection of heparin into the interstices of the graft will prevent perioperative thrombosis.

Angiography plays a critical role for identifying perioperative graft thrombosis and for embolizing collaterals missed at operation. We have successfully treated perioperative thromboses by infusing low dose streptokinase into the portal vein above the anastomotic site. The grafts have remained patent in the follow-up period. Easy access to the portal system *via* the H-graft is an advantage of our operation.

We prefer prosthetic grafts to direct anastomoses for several reasons. First, the grafts have a caudad angle from the portal vein and are implanted into the vena cava against the flow. This, as well as the length of the graft, should add considerable resistance to flow through the shunt compared with single stoma side-to-side anastomoses. Second, prosthetic grafts cannot expand while direct anastomoses can widen with time. Finally, grafts require less extensive portal vein and vena caval dissection. Therefore, the operation is technically easier than direct side-to-side shunts, with less blood loss.

Postoperative Encephalopathy Following Partial Shunts

Preoperative risk factors for developing encephalopathy were similar for each group. However, postoperative encephalopathy rates were significantly diminished with smaller grafts. This reduction correlated with maintenance of prograde flow. Our finding supports Warren's hypothesis that hepatic portal perfusion is important for preventing postoperative encephalopathy.[10] Additionally, maintaining high portal pressures may reduce absorption of potential neurotoxins from the gut, as suggested by Orloff and Warren.[11,12] We have previously observed that corrected portal pressure is inversely related to graft size. However, the correlation between portal pressure and encephalopathy was not as strong as the correlation between direction of portal flow and encephalopathy.[13]

Our results are similar to those of Marion, Balique, and others who have been investigating partial shunting in France.[14,15] The French investigators, using small stoma side-to-side portacaval shunts, observed prograde portal flow in more than 70% of their patients. In these patients, postoperative encephalopathy rates were significantly reduced. Further, their work supports our conclusion that total portal decompression is unnecessary to prevent variceal hemorrhage.

Alternatives for Managing Variceal Hemorrhage

Endoscopic sclerotherapy has become the first approach for control of variceal hemorrhage at many medical centers.[16] However, sclerotherapy is not a panacea. Long-term control of variceal hemorrhage is unproven and awaits further studies. Gastric varices or hemorrhagic gastritis in the congested stomach can supervene once esophageal varices have been sclerosed.[17] Esophageal ulcers and strictures may follow injection. Other potential limitations

of sclerotherapy include noncompliance of the patient for repeated injections and rebleeding before completing therapy.

The efficacy of other nonoperative therapies such as propranolol remains controversial and will also require extensive long-term studies.[18] Alcoholics are notoriously unreliable in adhering to treatment schedules.

Surgical devascularization procedures have not met with uniform success in the United States.[19,20] Rebleeding is a frequent problem. Some of the procedures have proven extremely difficult and have a high operative mortality, especially in high risk patients. Therefore, for those failing nonsurgical therapy, three major surgical options remain—total, selective, and partial shunts.

Total shunts produce unacceptably high rates of postoperative encephalopathy. Selective shunts reduce this complication, as shown in several series.[21,22] Unfortunately, in alcoholics, the standard distal splenorenal shunt can lose its selectivity over time.[23,24] This may explain the disappointingly high incidences of encephalopathy in prospective trials that study mostly alcoholics. Recently introduced modifications may prevent progressive loss of portal perfusion.[25] However, their success will require long-term follow-up. Patients failing sclerotherapy or other nonsurgical management will often be actively bleeding or have more advanced cirrhosis with ascites.[16] Selective shunts are not usually used in these patients. Partial shunts using 8 or 10 mm PTFE grafts can be expeditiously performed and have few contraindications. Further, postoperative portal flow patterns have not changed significantly in our follow-up studies.[26] Therefore, we feel that partial shunts have promise for managing variceal bleeding in alcoholics and could become more important in the future.

Our investigations have led us to an operation capable of maintaining elevated portal pressures and preserving prograde portal flow in most cirrhotics. Reduced encephalopathy rates correspond with smaller shunt diameters. Variceal rehemorrhage and late shunt thrombosis rates remain low. An overall 16% perioperative PTFE shunt thrombosis rate was the single drawback. However, we do not find this to be an insurmountable problem. Angiographic intervention has proven successful in restoring long-term graft patency without reoperation. Do the potential benefits of partial shunting outweigh the potential risks? Clearly, the answer will require prospective randomized trials, our fifth and final phase of these investigations.

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