ANNALS OF SURGERY
Vol. 225, No. 5, 601–608

Differential Effects on Portal and Effective Hepatic Blood Flow

A Comparison Between Transjugular Intrahepatic Portasystemic Shunt and Small-Diameter H-Graft Portacaval Shunt

Alexander S. Rosemurgy, M.D.,* Emmanuel E. Zervos, M.D.,* Sarah E. Goode, R.N.,* Thomas J. Black, M.D.,† and Bruce R. Zwiebel, M.D.†

From the Departments of Surgery and Radiology,† College of Medicine, University of South Florida, Tampa, Florida*

Objective
This study was undertaken to determine the effects of transjugular intrahepatic portasystemic shunt (TIPS) and small-diameter prosthetic H-graft portacaval shunt (HGPCS) on portal and effective hepatic blood flow.

Summary Background Data
Mortality after TIPS is higher than after HGPCS for bleeding varices. This higher mortality is because of hepatic failure, possibly a result of excessive diminution of hepatic blood flow.

Methods
Forty patients randomized prospectively to undergo TIPS or HGPCS had effective hepatic blood flow determined 1 day preshunt and 5 days postshunt using low-dose galactose clearance. Portal blood flow was determined using color-flow Doppler ultrasound.

Results
Treatment groups were similar in age, gender, and Child's class. Each procedure significantly reduced portal pressures and portasystemic pressure gradients. Portal flow after TIPS increased (21 mL/second ± 11.9 to 31 mL/second ± 16.9, $p < 0.05$), whereas it remained unchanged after HGPCS (26 mL/second ± 27.7 to 14 mL/second ± 41.1, p = n.s.). Effective hepatic blood flow was diminished significantly after TIPS (1684 mL/minute ± 2161 to 676 mL/minute ± 451, $p < 0.05$) and was unaffected by HGPCS (1901 mL/minute ± 1818 to 1662 mL/minute ± 1035, p = n.s.).

Conclusions
Both TIPS and HGPCS achieved significant reductions in portal vein to inferior vena cava pressure gradients. Portal flow increased after TIPS, although most portal flow was diverted through the shunt. Effective hepatic flow is reduced significantly after TIPS but well preserved after HGPCS. Hepatic decompensation and mortality after TIPS may be because, at least in part, of reductions in nutrient hepatic flow.

Transjugular intrahepatic portasystemic shunt (TIPS) has been embraced by the medical community for its use in treating complicated portal hypertension.[1-4] Proponents cite TIPS' ability to reduce portal hypertension and, thereby, relieve variceal hemorrhage while maintaining low rates of rehemorrhage and survival comparable to the best-published results seen with surgical shunts.[5] Detractors of TIPS note high stenosis and occlusion rates and point out that follow-up generally is presented as rates of "assisted patency," inferring the effort necessary to maintain TIPS patency.[6-11] They also point out that recurrent bleeding after TIPS, although generally because of shunt occlusion, also can occur with patent TIPS. Lastly, many practitioners think that early and intermediate-term survival after TIPS does not support its routine use.[8-11] The debate over TIPS is a highly emotional one, lacking data from large clinical trials comparing it to surgical shunting.

Over the past several years, we have been comparing TIPS to small-diameter prosthetic H-graft portacaval shunts through a randomized, prospective clinical trial.[12,13] Early results indicate higher failure rates with TIPS, with late mortality after TIPS also higher. Although mortality, both perioperatively and by 1 to 2 years, nearly is always because of liver failure, no ready explanation for differences in mortality between shunts is available. Reflecting years of thought and research on shunting, most investigators would presume that the changes in nutrient hepatic flow that occur with shunting could explain differences in mortality and hepatic function after shunting.

Both small-diameter prosthetic H-graft portal caval shunts and TIPS achieve partial portal decompression.[1,12-17] The purpose of this study was to determine if, in achieving partial portal decompression, these shunts differentially impact portal blood flow and maintenance of nutrient hepatic blood flow. Our hypotheses in undertaking this study were that TIPS would increase and H-graft shunts would decrease portal vein blood flow into the liver, and that postshunt nutrient hepatic blood flow would be similar after TIPS and small-diameter prosthetic H-graft portacaval shunt.

METHODS

Randomized Trial

Forty patients were randomized prospectively to undergo TIPS or small-diameter prosthetic H-graft portacaval shunt as part of a larger prospective clinical trial. The 20 patients undergoing each shunt procedure were similar in gender, age, cause of cirrhoses, presence of ascites, Child's class, and timing of portal decompression (Table 1).

Table 1. PATIENTS UNDERGOING EITHER TIPS OR SMALL DIAMETER PROSTHETIC HGPCS

	TIPS	HGPCS
Number	20	20
Age (years ± SD)	55 ± 12.5	52 ± 13.1
Gender (% male)	70	65
Cirrhosis (EtOH)	80%	85%
Preoperative ascites	65%	80%
Child's class (A, B, C)	20%, 40%, 40%	15%, 65%, 20%
Timing of Shunting (elective, urgent, emergent)	13, 6, 1	15, 2 3

TIPS = transjugular intrahepatic portosystemic shunt; HGPCS = H-graft portacaval shunt; EtOH = ethyl alcohol.

Creation of H-Graft Shunt

Our technique of H-graft shunt has been described in detail.[18] In short, 8-mm externally reinforced polytetrafluroethylene (W. L. Gore, Flagstaff, AZ) is interposed between the portal vein bifurcation and the inferior vena cava. The graft measures 3 cm from toe to toe and 1 ½ cm from heel to heel with the bevels at each end oriented at 90° to one another. Portal vein and inferior vena cava pressures are transduced and blood flow determined using color-flow Doppler ultrasound intraoperatively both preshunt and postshunt. Aggressive attempts to ligate collaterals arising from the portal vein are not undertaken. Shunt patency is assessed by venography and manometry through transfemoral cannulation on approximately postoperative day 5. Large collaterals noted venographically are embolized with wire coils.

Creation of Transjugular Intrahepatic Portasystemic Shunt

Our technique for TIPS placement has been described in detail.[12] With the patient under general anesthesia, the shunt is placed using cannulation of right internal jugular vein. After cannulation of the right hepatic vein, a needle is advanced over a wire through the substance of the liver in the direction of the portal vein. Negative pressure is maintained in the needle as it is advanced. With return of blood, signifying entrance into the portal vein, portal

Presented at the 108th Annual Meeting of the Southern Surgical Association, December 1-4, 1996, Palm Beach, Florida.
Address reprint requests to Alexander S. Rosemurgy, M.D., Tampa General Hospital, P.O. Box 1289, Tampa, FL 33601.
Accepted for publication December 13, 1996.

venography confirms location of the needle. An 8-mm Ultrathin angioplasty balloon (Meditech, Boston Scientific, Watertown, MA) is inserted into the tract that connects the portal and hepatic veins and the tract is dilated. A metal Schneider Wallstent (Pfizer, New York, NY) is used as the shunt stent. The stents are 8 mm in diameter when placed, but they are dilated up to 10 mm as needed to achieve a gradient between the portal vein and the inferior vena cava of <12 mmHg. All patients undergo color-flow Doppler ultrasound study of their TIPS 2 to 4 days after shunting. Midstent flow rates of 100 cm/second are sought. Flow rates less than this often presage dilation or thrombectomy at subsequent venography. Transjugular shunt venography and manometry are always undertaken to confirm shunt patency and function before discharge.

Determination of Effective Hepatic Blood Flow

Effective hepatic blood flow was determined by low-dose galactose clearance using a modification of the techniques described by Henderson et al.[19] After an overnight fast, within 1 day preshunt and 4 to 6 days postshunt, the patient was given a 250-mg bolus of 5% galactose intravenously. This bolus was followed by a constant infusion of 75-mg galactose per minute for 1 hour. Peripheral blood samples were obtained before the bolus, 30 minutes, and 60 minutes after starting the constant infusion. The blood samples were collected in fluoride- and oxalate-coated evacuated tubes, placed on ice, and centrifuged immediately. Plasma was withdrawn from the tubes and stored at −70 C until assayed. Samples were assayed for galactose using the YSI model 2700 Select Biochemistry Analyzer (YSI, Yellow Springs, OH). Clearance (milliliter per minute) was calculated using infusion rate (milligram per minute) and steady-state concentration (milligram per milliliter) and is a reliable estimator of effective hepatic flow.[19–25] All assays were undertaken in a masked fashion by technicians.

Determination of Portal Blood Flow

Peak portal blood flow was calculated using color-flow Doppler ultrasound (Acuson Linear Array; Acuson, Mountain View, CA). Portal vein cross-sectional area was determined and portal vein mean flow velocity was measured extracorporeally before TIPS and within 2 to 4 days after TIPS. Similarly, portal blood flow was calculated intraoperatively intracorporeally before and after construction of the H-graft portacaval shunt. Postshunt, portal flow was determined cephalad to the H-graft shunt.

Table 2. BOTH TIPS AND SMALL DIAMETER PROSTHETIC HGPCS ACHIEVED PARTIAL PORTAL DECOMPRESSION

	TIPS	HGPCS
Preshunt PV pressure (mm Hg)	33 ± 8.0	31 ± 4.1
Postshunt PV pressure (mm Hg)	26 ± 8.2†	21 ± 4.7*†
Preshunt PV–IVC pressure gradients (mm Hg)	18 ± 6.3	16 ± 3.9
Postshunt PV–IVC pressure gradients (mm Hg)	9 ± 3.5†	6 ± 4.5*†

PV = portal vein; IVC = inferior vena cava.
* less than after TIPS $p < 0.03$, Student's *t*-test.
† less than preshunt $p < 0.01$, paired Student's *t*-test.
Data are expressed as mean ± SD.

Outcome and Data Analysis

Early deaths were defined as those occurring within 30 days of shunting with late deaths occurring more than 30 days from shunting. Presence or absence of ascites was determined by physical examination or ultrasound and noted. Data were entered into a personal computer. Statistical analysis was undertaken using TRUE EPISTAT (EPISTAT Services, Richardson, TX). Significance was accepted with 95% confidence. Unmatched sample means were compared using Student's t test, whereas paired data was compared using Student's t test for paired data.

RESULTS

Portal Decompression

Both shunts achieved partial portal decompression. For both TIPS and H-graft shunts, there was a significant decrease in portal vein pressures and portal vein–inferior vena cava pressure gradients with shunting. The H-graft shunts led to lower postshunt portal vein pressures and lower postshunt portal vein–inferior vena cava pressure gradients (Table 2).

Portal and Effective Hepatic Blood Flow

Portal blood flow increased by 48% after TIPS. Conversely, effective hepatic blood flow significantly *decreased* by 60% after TIPS. After H-graft shunt, portal blood flow decreased by 46%, although this was not significant ($p = 0.17$, Student's t test for paired data). Effective hepatic blood flow decreased little (13%) after H-graft shunt. Portal blood flow after TIPS was greater than after H-graft shunt, although preshunt flows were not dif-

Table 3. EFFECTS OF SHUNTING ON PORTAL AND EFFECTIVE HEPATIC BLOOD (EHBF) FLOW

		Portal Flow (ml/sec)		EHBF (ml/min)	
	N	preshunt	postshunt	preshunt	postshunt
TIPS	20	21 ± 11.9	31 ± 16.9*	1684 ± 2161.8	676 ± 451.3*
HGPCS	20	26 ± 27.7	14 ± 40.7†	1901 ± 1818.7	1662 ± 1035.4‡

TIPS = transjugular intrahepatic portosystemic shunt; HGPCS = H-graft portacaval shunt.
* significantly changed from preshunt value ($p < 0.02$, paired Student's t test).
† different than after TIPS ($p = 0.09$, Student's t test).
‡ different than after TIPS ($p < 0.001$, Student's t test).

ferent. Conversely, effective hepatic blood flow after TIPS was significantly less than after H-graft shunt, although preshunt flows were similar (Table 3).

Mortality

Three patients died within 30 days of TIPS placement, two because of liver failure and one because of liver failure and variceal hemorrhage despite a patent shunt. Two patients died within 30 days of undergoing H-graft shunt, both because of liver failure. The patients dying after each of the shunts were similar by demographic measures. Preshunt and postshunt portal vein pressures and portal vein–inferior vena cava pressure gradients were comparable for those suffering early deaths after TIPS and H-graft shunts. Effective hepatic blood flow did not decrease in patients dying within 30 days of shunting, although preshunt flow seemed lower (947 mL/minute ± 305.6 *vs.* 1913 mL/minute ± 2054.0, $p = 0.30$, Student's t test) (Table 4). Postshunt-effective hepatic blood flow was not different between those surviving or dying early after shunting (1199 mL/minute ± 976.3 *vs.* 957 mL/minute ± 303.5, $p = 0.59$, Student's t test). As well, specifically for each shunt, preshunt- and postshunt-effective hepatic flow were not different between those surviving *versus* dying after TIPS or H-graft shunt (Table 5).

Late deaths occurred in five patients after TIPS and in none after H-graft shunts ($p = 0.02$, Fisher's exact test). Four deaths were because of liver failure and one was because of a motor vehicle crash. Discounting the latter death, late mortality still was higher after TIPS compared to that of H-graft shunts (4 of 16 *vs.* 0 of 18, $p = 0.04$, Fisher's exact test, Table 6). Patients dying late were not different descriptively than were survivors. With TIPS, preshunt-effective hepatic blood flow seemed higher, although not significantly so, for long-term survivors than for those dying late deaths (2172 mL/minute ± 2575.0 (STD) *vs.* 632 mL/minute ± 270.5; $p = 0.26$ by Student's t test). After TIPS, postshunt-effective hepatic blood flow was similar in long-term survivors and in those dying late deaths (548 mL/minute ± 456 *vs.* 648 mL/minute ± 408; $p = 0.70$ by Student's t test).

Fifteen patients (75%) undergoing TIPS had preshunt-effective hepatic blood flow of <1500 mL/minute. Of those, three (20%) died within 30 days of TIPS and four (25%) died late. Fourteen patients (70%) undergoing H-graft shunts had preshunt-effective hepatic blood flow of <1500 mL/minute. Of those, two (14%) died within 30 days of shunting and none died late. Excluding the accidental death, mortality was nearly significantly greater after TIPS than after H-graft shunt (7 of 19 after TIPS

Table 4. EARLY DEATHS FOLLOWING TIPS AND SMALL DIAMETER PROSTHETIC HGPCS

	TIPS	HGPCS
Number	3	2
Gender	3 male	1 male, 1 female
Age (years ± SD)	49 ± 10.8	54 ± 25.4
Child's class	1B, 2C	1B, 1C
Shunt timing (#elective, urgent, emergent)	1, 1, 1	0, 1, 1
Cirrhosis	2 EtOH, 1 α-1AD	2 EtOH/hepatitis C
Ascites	3	2
Pre PV pressures (mmHg)	31 ± 4.8	31 ± 0
Post PV pressures (mmHg)	24 ± 5.1	12 ± 0.5
Preshunt PV-IVC gradient	16 ± 7.1	18 ± 0
Postshunt PV-IVC gradient	10 ± 4.4	6 ± 3.5
EHBF preshunt (ml/min)	1026 ± 394.7	828 ± 120.9
EHBF postshunt (ml/min)	1094 ± 6.0	751 ± 476.6

TIPS = transjugular intrahepatic portosystemic shunt; HGPCS = H-graft portacaval shunt; EtOH = ethylalcohol; EHBF = effective hepatic blood flow; PV = portal vein; IVC = inferior vena cava; AD = antitrypsin deficiency.
Data are expressed as mean ± SD when appropriate.

Table 5. EHBF PRESHUNT AND POSTSHUNT FOR THOSE PATIENTS SURVIVING OR DYING WITHIN 30 DAYS AFTER TIPS (HGPCS)

	TIPS	HGPCS
Preshunt EHBF-Survivors	1799 ± 2261.8	2020 ± 1883
Preshunt EHBF-Non-survivors	1026 ± 394.7*	828 ± 120.9†
Postshunt EHBF-Survivors	602 ± 437	1763 ± 1037
Postshunt EHBF-Non-survivors	1094 ± 6.0‡	751 ± 476.6§

EHBF = effective hepatic bloodflow; TIPS = transjugular intrahepatic portosystemic shunt; HGPCS = H-graft portacaval shunt.
Compared to survivors undergoing the same shunt by Student's *t*-test: * p = 0.57,
† p = 0.39, ‡ p = 0.07, § p = 0.20.
Data are presented as mean ml/min ± SD.

vs. 2 of 20 after H-graft shunt; p = 0.06, Fisher's exact test).

DISCUSSION

The role of TIPS in the treatment of bleeding varices and portal hypertension continues to evolve. Several prospective, randomized trials are in progress or are being initiated to define its role relative to surgical shunting. Early results from our prospective clinical trial, as well as growing experience from multiple investigators, suggest that hepatic failure after TIPS placement may be relatively high.[2,8,12,13,26] In patients of advanced Child's class with variceal hemorrhage, hepatic failure is not a totally unexpected event after shunting[27]; a possible explanation for postshunt liver failure is the loss of nutrient hepatic blood flow. This study was therefore undertaken to determine if TIPS and small-diameter prosthetic H-graft portacaval shunts lead to differences in portal and effective hepatic blood flow after shunting. Although both shunts achieve partial portal decompression, each leads to different postshunt values of portal and effective hepatic blood flow. The implications of these differences potentially are far reaching.

The patients undergoing either TIPS or H-graft shunt in this study collectively were similar. The cause and extent of their cirrhosis were similar, as was the timing of shunting. Both shunts achieved partial portal decompression, although the H-graft shunts led to lower portal pressures and lower portal vein–inferior vena cava pressure gradients. Rates of perioperative mortality after each shunt were similar, with death coming as a consequence of liver failure. Deaths more than 30 days after shunting occurred only after TIPS and primarily were a result of liver failure. Mortality after H-graft shunts was small (10%), whereas mortality due to liver failure after TIPS approached 40%.

Because the patients undergoing each shunt were similar, differences in postshunt liver failure and mortality may be because of differences in postshunt nutrient blood flow to the liver. Although the initial thought might be that TIPS reduces portal blood flow more than the surgical shunt, postshunt portal blood flow actually increases. The increase may reflect a steal phenomenon through the shunt, with TIPS acting as a low-resistance portal outflow tract or a high-volume conduit connecting the portal and the systemic venous system at the hepatic vein. Though portal flow increases and is greater after TIPS than after H-graft shunts, the nutrient quality of the increased flow is suspect.

To determine nutrient hepatic blood flow, we used low-dose galactose clearance. This established technique[19–25] documented that there is a significant (60%) decrease in effective hepatic blood flow that is measurable within days after TIPS, whereas effective hepatic blood flow after H-graft shunting decreased by only slightly >10%. For each shunt, could changes in effective hepatic blood flow with shunting be because of a loss of nutrient portal blood flow? After H-graft shunts, portal blood flow decreased by nearly 700 mL/minute, more than enough to account for the total decrease in effective hepatic blood flow. Arterialization of the liver must occur after H-graft shunting to maintain effective hepatic blood flow. Large decreases in effective hepatic blood flow after TIPS (if because of a loss of portal flow) only could be explained by a complete loss of portal flow. This would be consistent with the earlier description of portal flow after TIPS: increased portal flow diverted through the stent and away from the hepatic parenchyma. Because portal flow proxi-

Table 6. LATE DEATHS AFTER TIPS

	TIPS
Number	4
Age	58 years ± 14.2
Gender	2 male, 2 female
Child's class	2A, 2B
Shunt timing	3 elective, 1 urgent
Cirrhosis	3 alcohol, 1 idiopathic
Ascites	1 with, 3 without
Portal pressure preshunt	32 ± 7.0 mm Hg (SD)
Portal pressure postshunt	27 ± 8.1 mm Hg
Preshunt PV-IVC	16 ± 5.2 mm Hg
Postshunt PV-IVC	10 ± 3.7 mm Hg

TIPS = transjugular intrahepatic portosystemic shunt; PV = portal vein; IVC = inferior vena cava.

mal to the TIPS, as measured by color-flow Doppler, actually *increased* with shunting, the decrease in effective hepatic blood flow with TIPS supports the theory that essentially all portal flow after TIPS is non-nutrient, racing up the stent into the systemic venous system.

These explanations of changes in portal and effective hepatic flow after shunting, although plausible, would be most credible if they correlated with clinical outcomes after shunting. Those dying early after shunting seemed to have lower preshunt-effective hepatic blood flow than those surviving. In contrast, postshunt-effective hepatic blood flow in patients dying within 30 days of TIPS was not different from that in survivors, although early liver failure occurred in 15%. Effective hepatic blood flow changed negligibly with shunting in those dying early after either shunt. Although both shunts, particularly TIPS, decrease effective hepatic blood flow, the occurrence of postshunt hepatic failure correlates poorly with decreases in effective hepatic blood flow after TIPS, making implications of a decrease in effective hepatic blood flow with shunting unclear. Early mortality after either shunt may reflect poor preshunt hepatic nutrient flow and, thereby, poor hepatic reserve. A resultant decrease in that flow after shunting may not be prerequisite for postshunt hepatic failure. Low-dose galactose clearance may be a valuable preshunt screening measure to identify patients at high risk to experience early postshunt hepatic failure.

Preshunt low-dose galactose clearance as a prognostic measure is an attractive concept. Its use, however, is not supported by a closer review of the data in this study. Preshunt-effective hepatic blood flow was <1500 mL/minute in a similar number of patients undergoing TIPS and H-graft shunts. Early mortality in each group also was similar. Late mortality, however, was much higher (4 of 16) after TIPS than after H-graft shunts (0 of 18). Thus, the increased mortality after TIPS cannot be explained solely by preshunt-effective hepatic blood flow. Transjugular intrahepatic portasystemic shunt seems to impact postshunt liver function negatively, although in an as-yet undefined way.

Transjugular intrahepatic portasystemic shunt decreases effective hepatic blood flow more than do H-graft shunts. Those dying early after shunting, whether undergoing TIPS or H-graft, had poor preshunt-effective hepatic blood flow. In those patients, effective hepatic blood flow changed little with shunting. Late mortality occurred only after TIPS. As with early mortality after TIPS, those patients dying late had poor preshunt-effective hepatic blood flow. Postshunt-effective hepatic blood flow was a poor predictor of late outcome, as it was with early outcome. Those dying late after TIPS had, on average, only small changes in effective hepatic blood flow with shunting. Although TIPS decreased effective hepatic blood flow more so than H-graft shunts, it seems that only preshunt-effective hepatic blood flow is a marker of outcome in this small prospective, randomized study. The significance of the decrease in effective hepatic blood flow with shunting is, as yet, unknown in the context of this randomized trial.

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Discussion

DR. J. MICHAEL HENDERSON (Cleveland, Ohio): Dr. Cameron, Dr. Copeland, Members, and Guests. I appreciate the opportunity to discuss this paper and to add some of my thoughts as to the importance of these hemodynamic changes in the shunt population.

I would like to applaud Dr. Rosemurgy for undertaking this prospective randomized control trial in which he has continued to add patients. This is an important study to answer clinical questions about the efficacy of transjugular intrahepatic portosystemic shunt (TIPS) versus surgical shunts.

I would like to break my discussion down into a couple of major points. First, let's talk about the portal flow. I think it is misleading to say we have increased portal flow with TIPS. Yes, there is increased flow in the main trunk of the portal vein, but I do not think there is increased portal flow through the sinusoids, and that is where it counts. So my first question to you, Alex, is do you have data regarding right and left branch of the portal vein? TIPS usually takes off just above the bifurcation, and in a functioning TIPS, which is decompressing portal hypertension, there is usually reversal of flow in the right and left branches of the portal vein coming back down to the TIPS and going out through the shunt. So I think we need to be careful when talking about increased flow in the portal vein.

The second issue I would like to raise is effective liver blood flow. I, again, applaud you for using low-dose galactose clearance. It has been one of my hobby horses for the last fifteen years, but the more I have used it, the more confused I have become about exactly what we are measuring.

Effective flow is a clearance method that is going to measure any blood flow going past functioning hepatic cells. It does not matter if it comes from the portal vein or the hepatic artery. These are such low doses of galactose, that, providing blood is seen by the hepatocytes, it is totally extracted at these very low concentrations.

In some of my previously published work, we have shown as the liver gets sicker, it tries to compensate by increasing effective blood flow. I think the missing component to the equation here is overall hemodynamic changes. These patients, when they get their total shunts, increase cardiac output. They become systemically hyperdynamic in an attempt to compensate for loss of portal flow to the liver.

I have asked you this question before, and I ask again if you have any cardiac output data in this population. As we look at patients with TIPS in particular and, over the years, your group of patients with partial shunts, do you have any data as to whether there are any systemic hemodynamic changes that might tie in with these effective blood flow changes that you are showing us? The only way I can put this data together is to say that your group of patients with TIPS did not show the compensation with systemic hyperdynamic changes.

Finally, I would like to raise the issue of the time scale of your observations. Hemodynamics are dynamic. You measured flow before and one week after the shunts were placed in these two groups of patients. Do you have any data at a later time frame in either of these groups of patients?

I think the changes do continue to happen. Stenosis in TIPS is a phenomenon, and I suspect that with stenosis, the effective as well as the portal flow are restored up beyond the TIPS into the hepatic parenchyma.

I think this is an important study. The clinical study is very important. The hemodynamics are very difficult to sort through. I applaud your effort to try and bring some sense to the hemodynamic changes in these two groups of patients.

Thank you very much.

DR. WILLIAM C. MEYERS (Worcester, Massachusetts): Dr. Rosemurgy and colleagues have provided some hard data that show the TIPS procedure is not all that it is said to be. I was recently on an National Institutes of Health study section for TIPS versus traditional methods to treat portal hypertension, and I was impressed by two things. Number one, there is an impressive volume of data that show the patency rate of TIPS to be about 50% at one year. And, two, TIPS has become one of the more common procedures to be performed by community arteriographers. Often, the latter are done by inexperienced radiologists for questionable indications, or they are done in patients who might benefit by a different procedure.

I congratulate the authors for these data and ask one question. Besides the short-term relief of variceal bleeding, or as a last-ditch effort, to stop bleeding, what do you now believe are the indications for TIPS?

Thank you.

DR. ALEXANDER S. ROSEMURGY, II (Closing Discussion): I would like to thank Dr. Henderson and Dr. Meyers for their