Exclusion of Nonisolated Splenic Vein in Distal Splenorenal Shunt for Prevention of Portal Malcirculation

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In an attempt to prevent portoprival malcirculation after distal splenorenal shunt (DSRS), a splenic hilar renal shunt (HRS) with proximal flush ligation of splenic vein was designed. To accomplish this procedure, two methods were compared: HRS alone (Group A) and HRS plus proximal flush ligation of the splenic vein (Group B). In Group A, which included 20 cirrhotic patients with esophageal varices, angiographic as well as pulsed Doppler flowmetric follow-up study revealed a portal thrombosis in two patients and severe narrowing of a portal vein in another two. Considerable stealing flow was observed in these four patients. In the Group B series, which included 33 cirrhotic patients, there were no gross changes in the portal hemodynamics. Normal prograde portal flow was confirmed by Doppler flowmeter in this series including 14 patients of more than 8 months after surgery. When the amount of nonisolated splenic vein embedded in the pancreas is minimized, portal malcirculation after distal splenorenal shunt can, to a great extent, be prevented.

ALTHOUGH THE DISTAL SPLENORENAL SHUNT by Warren[1] has shown considerable success in eliminating esophageal varices with a low incidence of Eck's syndrome, there are reports of thrombosis of the portal vein[2–4] or retrograde flow of portal circulation[5] after this shunt. Isomatsu[6] noted at autopsy that communicating veins of a distal splenic vein had become remarkably dilated in patients who had undergone the Warren shunt. This suggested that these dilated communicating veins would divert flow of the portal venous blood from the trunk into the renal vein, thereby converting the original distal splenorenal shunt to a central splenorenal one. Such a distal splenorenal stealing route was confirmed in reports[2–4,7,8] in which an increased number of collaterals was noted around the distal splenic venous area.

A splenic hilar renal shunt was designed so that the distal splenic vein could be isolated from the pancreas, as close as possible to the splenic hilum. Twenty patients underwent surgery and it was assumed that malcirculation would no longer present a problem. Two of the patients had a thrombosis, and angiography taken in one revealed abundant collaterals at the proximal splenic vein, and here the portal venous blood was diverted to the cephalad direction.

This experience clearly indicated that there were two features involved in stealing of portal venous blood; one is proximal splenic-gastric stealing and the other, distal splenic-renal stealing. The exclusion of both proximal and distal splenic veins is essential to prevent occurrence of malcirculation (Fig. 1). To devise a method that would meet the above requirements, a splenic hilar renal shunt with proximal flush ligation of a splenic vein, in other words, a modification of the Warren shunt, was proposed.

Materials and Methods

The subjects were two groups of patients with a histologically proven postnecrotic cirrhosis of the liver, and all were admitted to the 2nd Department of Surgery, Kyushu University Hospital. Group A included 20 patients who underwent splenic hilar renal shunt (HRS) alone. These patients were seen from April 1979 to November 1981, 14 receiving therapeutic and six prophylactic treatment. Group B included 33 patients who underwent HRS plus proximal flush ligation of a splenic vein, treated from December 1981 to November 1983, 25 being therapeutic and eight prophylactic. Each operation was elective, following tests to determine the preoperative liver function (Table 1). All the candidates selected according to the data on the liver function tests were surgically treated. There were 18 men and 2 women in Group A and 25 and eight in Group B, respectively. Follow-up period of Group A was 1 to 38 months (average 20 mos.), while that of Group B was 1 to 27 months (average 13 mos.).

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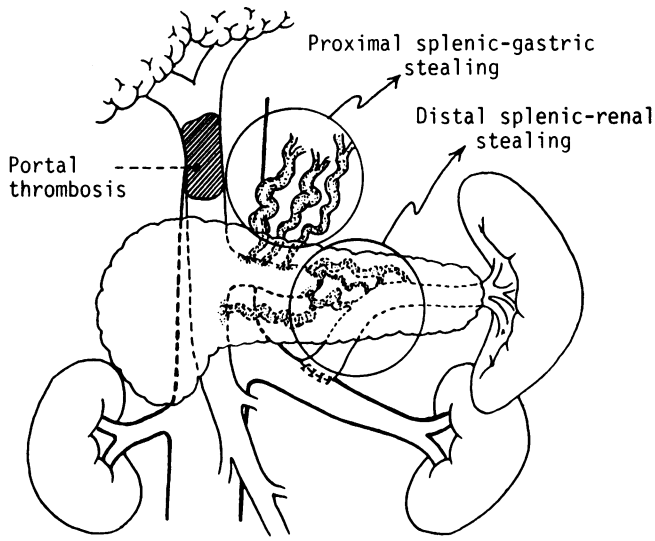


FIG. 1. Assumed mode of malcirculation after a distal splenorenal shunt.

To assess postoperative changes in the portal circulation, the venous phase of the celiac arteriogram as well as of the superior mesenteric arteriogram were studied. Angiographic assessment of portal perfusion of the liver was made according to the classification of Nordlinger et al.[9] Angiography was routinely done before discharge from the hospital and most patients refused arteriography thereafter. To avoid the potential hazard of arteriography, we used a pulsed Doppler flowmeter (Toshiba, SDL-01A) combined with an electronic linear scanner (Toshiba, SAL-50A).[10] This equipment made real-time measurement of the portal blood and the shunt flow feasible.

TABLE 1. *Data on Liver Function Immediately Before Operation*

	Group A (N = 20)	Group B (N = 33)
Albumin (g/dl)*	3.3 ± 0.4	3.7 ± 0.5
Bilirubin (mg/dl)*	1.4 ± 0.8	1.2 ± 0.5
Prothrombin time		
Not prolonged	5	5
Prolonged	15	28
(Prolongation, sec)*	(2.0 ± 1.2)	(1.6 ± 1.4)
ICG R_{15} (%)†	34.3 ± 9.3	28.1 ± 9.4
Ascites		
None	11	21
Moderate	9	12
Encephalopathy		
None	17	32
Minimal	3	1
Nutritional status		
Good	17	27
Moderate	3	6

* Figures are expressed as mean ± SD.
† Indocyanin green retention at 15 minutes, mean ± SD.

Operative Procedures

1. Transverse incision is made along the costal margin with a longer extension at the left flank.

2. To identify the splenic vein near the hilum of the spleen, the spleen was mobilized from a lateral approach and the colic ligament was severed. Along the outer margin of the spleen, the spleno-renal ligament was incised using an electrocoagulator (Fig. 2A). With gentle and blunt dissection, the spleen is thus mobilized and lifted up medially together with the tail of the pancreas. The splenic vein, which is readily detectable near the hilum of the spleen, is carefully mobilized as near to the bifurcation of that vein at the hilum (Fig. 2B).

3. Under the same operative field, a left renal vein is mobilized as long as necessary.

4. When the mobilization of the splenic vein can be done in such a manner as to facilitate anastomosis to the renal vein, this vein is directly anastomosed, in the usual fashion (Fig. 2C). When mobilization is difficult, an autovein graft is used to interpose the gaps between the splenic and renal veins. This autovein is taken from the internal jugular vein of a superficial femoral vein, depending on the size required.

5. Working the retroperitoneum medially and lifting the body of the pancreas, the proximal splenic vein is exposed and ligated flush to the juncture of the portal vein (Fig. 2D). When the mobilization of the splenic vein from a lateral approach can be proceeded deeply up to its portal juncture, separate procedure of flush ligation of the proximal splenic vein can be saved.

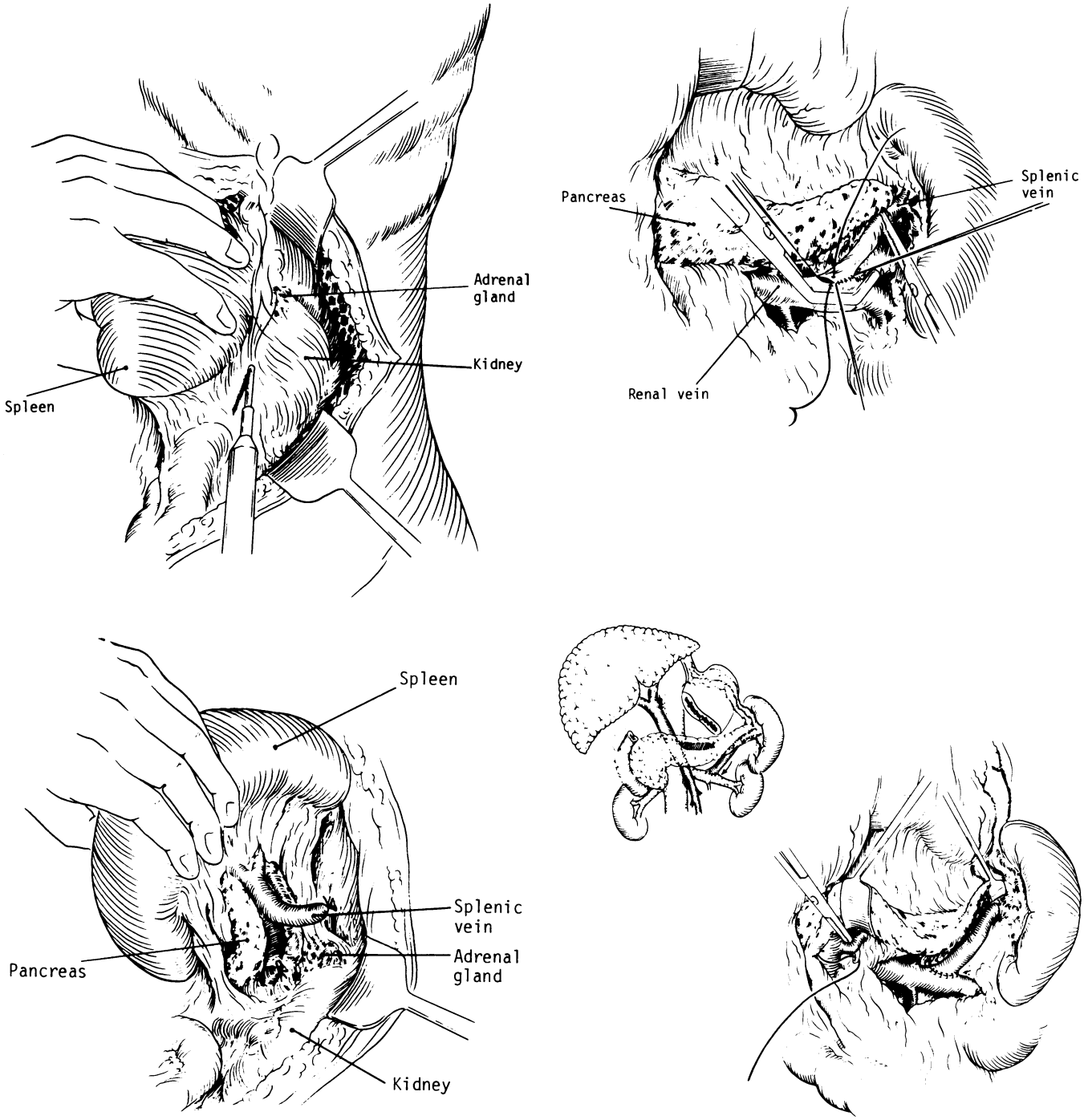
6. The left gastric vein is ligated as near as possible to the pancreatic bed. It is also mandatory to dissect the posterior gastric vessels to accomplish the pancreato-gastric disconnection. The right gastroepiploic arcade is not completely abolished.

Procedures 1 to 4 were used for the splenic hilar renal shunt (Group A) and procedures 1 to 5 were used in case of a proximal flush ligation of a splenic vein (Group B). Procedure 6 was used for both groups.

Results

Group A (HRS Alone)

Of 20 patients undergoing HRS alone, early death within the first 30 postoperative days occurred in one (5.0%) and late death in eight: three from hepatic failure, one from hepatoma, and one from variceal bleeding. In two there was postoperative bleeding (10%) and in three Eck's syndrome (15%) (Table 2). Patency of the shunt angiologically examined in 12 patients was confirmed in all. Marked regression of esophageal varices was noted in all 17 patients subjected to endoscopic examination after surgery.



FIGS. 2A–D. Operative procedures of splenic hilar renal shunt with proximal flush ligation of splenic vein. *A* (*top left*). Mobilization of the spleen by the lateral approach. *B* (*bottom left*). Mobilization of the splenic vein. *C* (*top right*). Distal splenorenal anastomosis. *D* (*bottom right*). Flush ligation of the proximal splenic vein.

Postoperative gross portal malcirculation was encountered in four patients: complete thrombosis in two and severe stenosis in two. Detailed data on examinations of postoperative hemodynamic changes are given in Table 3. Patient No. 3, a cirrhotic patient, had encephalopathy 2 months after operation. An angiogram taken at that time showed a slight narrowing of the portal vein with abundant collaterals around the stomach. The angiogram taken 9 months after surgery revealed complete obstruction of the portal vein and all the portal venous blood flowing into the splenic vein, thereby to the cephalad direction (Fig. 3). A similar postoperative

TABLE 2. *Clinical Results of Hilar Renal Shunt*

	Without Proximal Flush Ligation of Splenic Vein		With Proximal Flush Ligation of Splenic Vein	
No. of patients	20		33	
Therapeutic	14		25	
Prophylactic	6		8	
Follow-up	1–38 mos. (av. 20 mos.)		1–27 mos. (av. 13 mos.)	
Mortality				
Operative*	1 (5.0%)		1 (3.0%)	
Late	8 (40.0%)		4 (12.1%)	
	Hepatic failure	3	Hepatic failure	2
	Hepatoma	1	Hepatoma	1
	Variceal bleeding	1	Others	1
	Others	3		
Late survivors	11 (55.0%)		28 (84.8%)	
Postoperative bleeding	2 (10.0%)		2 (6.1%)	
Eck's syndrome	3 (15.0%)		0 (0.0%)	
Postoperative portal malcirculation	4 (20.0%)		0 (0.0%)	
	Thrombosis	2		
	Stenosis	2		

* Death within the first 30 postoperative days.

course was observed in patient No. 12. Collateralization around the left gastric vein, gastroepiploic vein, or the proximal splenic vein was seen in eight of 12 patients examined. There was no evidence of increased collateralization in the distal splenic venous area.

Of vital interest is the hemodynamic change measured by Doppler flowmeter. Blood at the proximal splenic vein, of which the distal end was closed at the time of operation, was seen to flow hepatofugally in seven patients (Fig. 4). The amount of stealing blood measured was considerably large, that is, 749, 1222, 825, 221, 981, and 841 ml/min in six respective cases. No quantitative measurement was performed in one patient. To these seven plus another patient with portal thrombosis, stealing flow from a proximal splenic vein occurred in more than one-half of the number of patients examined.

Group B (HRS with Proximal Flush Ligation of a Splenic Vein)

Thirty-three patients underwent this operation. Early death within the first 30 postoperative days occurred in one (3.0%) and late death in four (12.1%): two from hepatic failure and one from hepatoma. Rate of post-

TABLE 3. *Portal Circulation After Hilar Renal Shunt Alone (Group A)*

	Angiographic Change				Doppler Measurement				
	Portal Perfusion Grade* (Diameter of PV, mm)								
Patient	Preop.	Discharge	Follow-up (9–34 mos.)	Postop. Collateralization	Month Postop.	Portal Flow (Prograde, ml/min)	Shunt Flow (ml/min)	Stealing Flow via PSV (ml/min)	Portal Thrombosis or Severe Narrowing
1	I (16)	I (15)	—	PSV (+), LGV (+)	32	244	714	749	no
2	III (16)	I (17)	—	PSV (+), LGV (+)	—	—	—	—	no
3	I (16)	I (10)	IV (−)	PSV (++)	34	0	1161	1222	obstruction
4	I (17)	I (17)	II (6)	PSV (++)	26	159	937	825	narrowing
5	II (16)	I (14)	II (5)	PSV (++), GEV (+)	25	81	1102	221	narrowing
6	III (16)	I (18)	—	nil	—	—	—	—	no
7	I (19)	II (15)	—	PSV (+)	24	206	763	patent†	no
8	III (12)	I (12)	—	nil	—	—	—	—	no
9	I (20)	I (19)	—	nil	—	—	—	—	no
10	I (22)	I (22)	—	nil	27	260	1370	981	no
11	—	—	II (10)	PSV (++)	32	236	1091	841	no
12	I (16)	IV (−)	—	PSV (++)	—	—	—	—	obstruction

* According to the classification of Nordlinger et al.[9] PV–portal vein, GEV–gastroepiproic vein with hepatofugal flow, LGV–left gastric vein, PSV–proximal splenic vein, with minor (+) and major (++) collateralizaiton.

† Patency was ultrasonically confirmed but no quantitative measurement of blood flow was made.

operative variceal bleeding was 6.1%. Eck's syndrome was not evident (Table 2). Patent shunt was confirmed in 29 of 30 patients angiologically examined. Marked improvement of varices was endoscopically documented in 30 of 31 patients examined. There was no postoperative malcirculation. Venous phase of celiac and superior mesenteric arteriograms visualized the portal vein with a considerable sharp contrast. There was almost no change in pre- and postoperative angiograms (Fig. 5). No gross change of opacification in the portal vein, such as obliteration of narrowing, was noted except for a moderate occurrence of collateralization around the left gastric vein of gastroepiploic vein in four patients (Table 4). In all subjects examined, 14 seen more than 8 months after surgery, normal prograde portal flow was confirmed by Doppler flowmeter (Fig. 6).

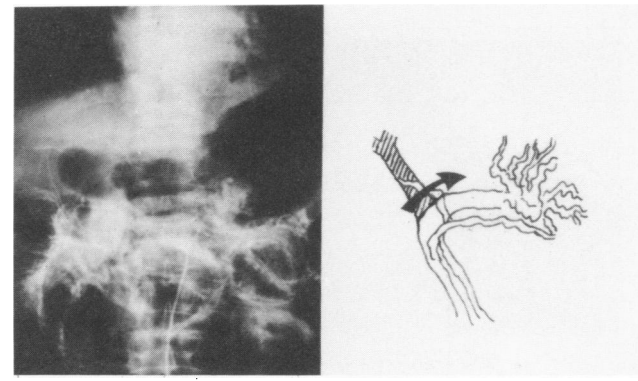


FIG. 3. Venous phase of the superior mesenteric arteriography in patient No. 3 (Table 3) 9 months after splenic hilar renal shunt alone (Group A). Portal blood steal through the proximal splenic vein with portal vein obstruction.

Discussion

The splenic hilar renal shunt, with or without proximal flush ligation of a splenic vein, was technically feasible in all 53 patients. The lateral approach we used made complete isolation of the distal splenic vein from the pancreas feasible, thus overcoming difficulties involved in conventional approaches. In cases when a long mobilization of a distal splenic vein from the pancreas was difficult due to pancreatitis, an autovein graft was used to achieve the splenic hilar renal anastomosis. Such technical advantages of the procedure contributed to the high operability.

Application of new armamentation, a combined system of an electronic linear scanner and a pulsed Doppler flowmeter, was most useful to pursue follow-up changes in portal hemodynamics after surgery. Though this system has methodological limitations, particularly with respect to assumptions on linearity of the flow, the direction of blood flow and gross alteration of flow rate could rationally be evaluated using our combined system. The hazard of repeated postoperative angiography was thus eliminated, and a noninvasive quantitative measurement of blood flow in the portal system was feasible.

In the Group B series, 29 patent shunts were visualized on 30 splenic arteriograms after surgery, and late patency was also confirmed in all of 19 patients who underwent ultrasonic examination in the follow-up period. Neither angiographic nor ultrasonic assessment was made in the remaining three patients. The overall patency rate of the shunt was considered to be fairly compatible with that of distal splenorenal shunt, reported by Warren et al.[11]

The current study revealed that the splenic hilar renal shunt with proximal flush ligation of a splenic vein caused no gross abnormal hemodynamic changes in the portal system. Since the occurrence of total or partial thrombosis after Warren shunt is reported several months after surgery,[2-4,12,13] the length of follow-up in the Group B series may be sufficient to evaluate the superiority of the procedure used.

Isomatsu[6] reported that the pancreatic veins developed to an enormous size at the area where the nonisolated distal splenic vein is embedded, thereby strongly suggesting that such dilated veins facilitate flow of the

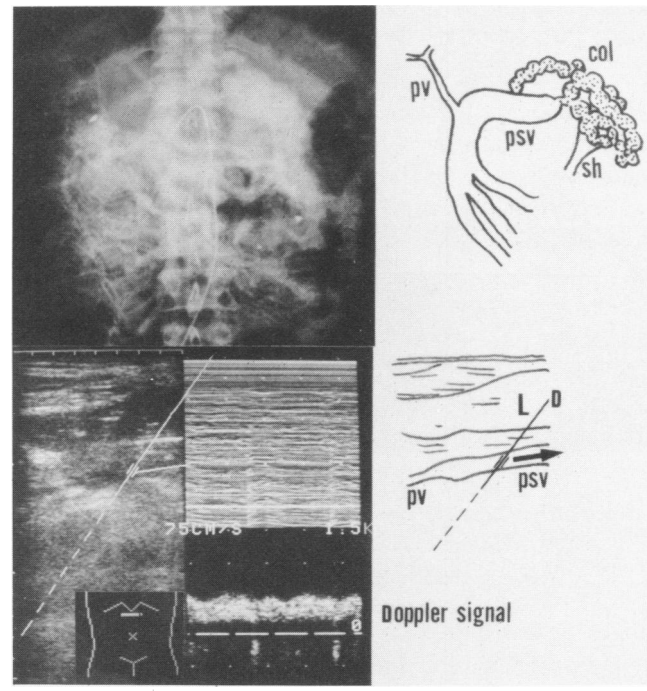


FIG. 4. Venous phase of the superior mesenteric arteriography in patient No. 4 (Table 3) 25 months after splenic hilar renal shunt alone (Group A) and measurement of blood flow by pulsed Doppler flowmeter combined with ultrasonic linear scanner. A marked narrowing of the portal vein was noted. The mesenteric blood flow was hepatofugal through the proximal splenic vein and prominently developed collaterals which led to the shunt (*above*). The proximal splenic vein was visualized on the B-mode scanning of the epigastrium. Doppler signals obtained from the vein indicated a hepatofugal flow in the leftward direction estimated as 825 ml/min (*below*) (pv–portal vein, psv–proximal splenic vein, col–collateral veins, sh–shunt, L–liver, D–Doppler beam with sampling site represented by double bars).

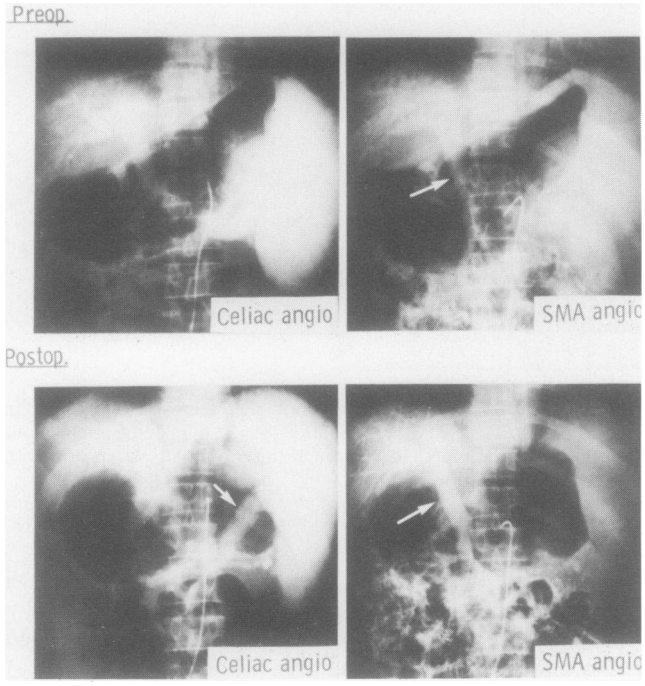
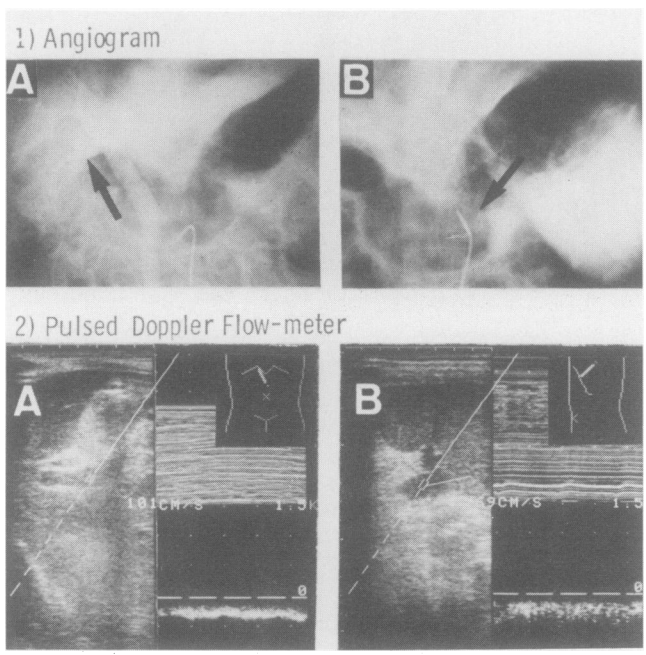


FIG. 5. Angiographic assessment of the portal perfusion after splenic hilar renal shunt with proximal flush ligation of a splenic vein in patient No. 2 in Table 4 (Group B). A hepatopedal portal flow was maintained 1 month after surgery. Collateralization is not evident. White arrows indicate the portal vein before surgery (*right upper*), after surgery (*right below*), and the shunt (*left below*).



FIGS. 6A and B. Angiograms and pulsed Doppler flowmetries in patient No. 20 in Table 4 (Group B) 1 month after surgery. *A.* venous phase of the superior mesenteric arteriography indicates a hepatopedal portal flow (black arrow). Pulsed Doppler flowmeter revealed a sufficient portal flow of 896 ml/min. *B.* venous phase of the splenic arteriography indicates a patent shunt (black arrow) with a blood flow of 384 ml/min.

portal venous blood into the distal splenic vein where the pressure is lowered by the shunt. In the Group A series of patients treated in our clinic, the postoperative angiogram revealed that the portal blood was stolen away via the proximal splenic vein toward the cephalad gastric area. Doppler flowmeter provided evidence that the proximal splenic vein enabled large amounts of portal venous blood to flow to the gastrosplenic area.

Rikkers et al.[12] and Zeppa[14] recommended ligation of the proximal splenic vein when possible, as thrombosis would occur secondary to portal vein trauma. Although propagation of the clot inside the cul-de-sac of the proximal splenic vein may sometimes be a cause of portal thrombosis,[3,12] in our experience, in no patient was portal thrombus caused by splenic vein thrombosis. We wish to stress that a blinded proximal splenic vein makes production of hepatofugal collaterals feasible, which sometimes abolish the portal flow.

Exceptional efforts have been directed to a portamesenteric gastrosplenic disconnection, in the hope of avoiding hemodynamic hazards and of securing shunt selectivity.[3,7,12] Others designed a method in which extended disconnection was not performed, on the premise that an untoward increase in hepatopedal flow would enhance the postoperative ascites.[5,15] However, in both modes of operation, abnormal collateralization, diminution, or occlusion of the portal vein did occur in a considerable number of patients. Whether or not portamesenteric gastrosplenic disconnection should be done seems to be an irrelevant matter, as related to hemodynamic hazards. We hold the view that a regional hyperdynamic circulation occurring in cirrhotic patients is responsible for the shunt selectivity.[16]

Henderson et al.[3] reported episodes of portal thrombosis in 28% in their series of Warren shunt, despite a flush ligation of the splenic vein. Since the portoprival collateralization described in the literature[2,4,7,8] usually involves entering the splenic hilum through a tortously developed channel around the proximal splenic vein, in addition to ligation of the proximal splenic vein, the exclusion of a distal splenic vein seems mandatory to avoid distal splenic venous stealing.

The most probable mode of portal malcirculation after the Warren shunt primarily involves proximal splenic-gastric stealing and distal splenic-renal stealing, as illustrated in Figure 1. A proposed method of operation to avoid such stealing is one involving exclusion of the nonisolated splenic vein, namely splenic hilar renal shunt with proximal flush ligation of a splenic vein.

An even longer follow-up of a larger number of patients is expected to confirm the superiority of isolation of both proximal and distal splenic veins. The distal splenorenal shunt is often looked upon with disfavor because of occasional occurrence of portal malcirculation. However, the ultimate evaluation of trans-splenic decompression of esophageal varices should be postponed

TABLE 4. *Portal Circulation After Hilar Renal Shunt with Proximal Flush Ligation of a Splenic Vein (Group B)*

	Angiographic Change				Doppler Measurement			
	Portal Perfusion Grade* (diameter of PV, mm)							
Patient	Preop.	Discharge	Follow-up (3–15 mos.)	Postop Collateralization	Month Postop.	Portal Flow (Prograde, ml/min)	Shunt Flow (ml/min)	Portal Thrombosis or Severe Narrowing
1	I (15)	I (13)	—	nil	15	444	217	no
2	I (15)	I (15)	—	nil	18	606	897	no
3	II (15)	I (15)	I (14)	nil	16	461	422	no
4	II (15)	I (16)	—	nil	13	598	patent†	no
5	I (18)	I (19)	—	nil	15	717	patent†	no
6	III (19)	I (20)	—	nil	14	668	525	no
7	II (17)	I (17)	—	LGV (+)	10	537	302	no
8	II (16)	II (15)	—	GEV (+)	19	406	522	no
9	II (12)	I (12)	I (13)	nil	9	patent†	496	no
10	I (12)	I (14)	—	nil	9	559	patent†	no
11	I (15)	I (15)	—	LGV (+)	9	876	202	no
12	—	I (16)	—	nil	8	972	340	no
13	I (15)	I (15)	I (15)	nil	11	756	412	no
14	I (17)	I (17)	—	nil	4	778	1145	no
15	I (15)	I (15)	—	nil	4	479	patent†	no
16	II (17)	II (17)	I (15)	LGV (+)	4	588	567	no
17	I (13)	I (12)	—	nil	11	718	288	no
18	II (14)	I (18)	—	nil	4	929	596	no
19	I (19)	I (18)	—	nil	3	1036	1256	no
20	I (17)	I (16)	—	nil	1	896	384	no
21	II (20)	I (21)	—	nil	1	548	737	no
22	I (15)	I (11)	—	nil	1	469	722	no
23	I (14)	I (12)	—	nil	—	—	—	no
24	III (12)	III (12)	—	nil	—	—	—	no
25	I (14)	I (13)	—	nil	—	—	—	no
26	I (11)	I (11)	—	nil	1	646	360	no
27	II (13)	II (12)	—	nil	1	396	374	no
28	I (14)	I (12)	—	nil	1	628	301	no
29	II (15)	II (14)	—	nil	1	574	638	no

* According to the classification of Nordlinger et al.[9] PV–portal vein, GEV–gastroepiproic vein with hepatofugal flow, LGV–left gastric vein.

† Patency was ultrasonically confirmed but no quantitative measurement of blood flow was made.

until the clinical results of such an approach, as specified in terms of isolation of splenic vein, are fully documented.

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