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## Liver transplantation in patients with patent splenorenal shunts

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### Abstract

Patent distal splenorenal shunts (Warren shunt) have been reported to cause decreases in the portal perfusion pressure and the total hepatic blood flow. Such hemodynamic alterations could have adverse effects on the transplanted liver. The experience with hepatic replacement in four patients with patent Warren shunts is reported. Operative findings were phlebosclerotic portal veins of small size and diminished portal blood flows. Hepatofugal collateral channels created by the construction of the Warren shunt were eliminated by division of the shunt and splenectomy in three patients and splenectomy alone in the other. All patients recovered; thus the presence of a patent Warren shunt should not be a contraindication for hepatic transplantation.

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Distal splenorenal shunts introduced by Warren, Zeppa, and Fomon in 1967 have become a popular method of therapy in the prevention of recurrent gastroesophageal hemorrhage in patients with portal hypertension.<sup>1</sup> The objective of the Warren shunt was to selectively decompress the gastroesophageal varices while maintaining portal perfusion.<sup>1</sup> The advantage of maintaining the portal perfusion is to prevent complications such as encephalopathy and hepatic atrophy, which have been observed with complete diversion of the portal blood flow.<sup>2,3</sup>

Numerous trials have proved the effectiveness of the distal splenorenal shunt in preventing hemorrhage without an exorbitant risk of encephalopathy, but the progression of liver disease often leads to hepatic failure.<sup>4,5</sup> The only recourse in this situation is liver transplantation. To date there have been no reports of liver replacement after placement of the Warren shunts, and the expected difficulties have not been delineated. We report our experience with liver transplantation in four patients with patent splenorenal shunts and end-stage liver disease.

### PATIENTS AND METHODS

Five patients with distal splenorenal shunts were among our first 500 consecutive liver transplant recipients. Selective angiography was performed in four patients to determine patency of the shunts and quality of perfusion of the portal vein. One of these patients was not included in this report since thrombosis of the shunt with a patent portal vein was demonstrated by angiography. Liver transplantation on this particular patient was not any different from that of other recipients without portosystemic shunts. The patient who had no preoperative angiography was found to have a patent splenorenal shunt intraoperatively.

The following parameters were reviewed: age and diagnosis, time interval from the creation of the Warren shunt to the liver transplant, size and characteristics of the portal vein, blood flow in the portal vein, management of the splenorenal shunt, and survival.

Liver transplantation was performed following techniques previously described.<sup>6</sup> The liver was replaced first followed by the splenectomy. The shunt was then ligated in three patients. The venovenous bypass was used in all patients.<sup>7</sup> A cannula was fed into the inferior vena cava via the femoral vein, and another cannula was inserted into the portal vein, decompressing both the systemic and splanchnic systems, respectively. With the use of a rollerhead pump, the blood was then delivered into the axillary vein via a single cannula. A flowmeter placed in this cannula recorded the total blood flow in the bypass (sum of portal vein and vena cava blood flows). The blood flow in the portal vein was determined in all four patients by subtracting the flow of the vena cava from the total blood flow that was performed when the portal vein cannula was removed for the portal vein reconstruction.

## RESULTS

Tables I and II summarize the results. In all patients, the portal vein was small and thickened by what appear to be fibrous plaques. The dissection of the hilum was easy since this area was undisturbed during previous operations. The flow in the portal vein was undetectable in cases 2 through 4 because no change was noted in the total blood flow when the portal vein cannula was removed. Splenectomy was performed in all patients without any difficulty; however, ligation of the shunt in patient 2, approached through the lesser sac, was difficult because of the presence of large collateral vessels. This patient developed pancreatitis and formation of a pancreatic fistula that resolved with nonoperative management. In cases 1 and 4, the Warren shunt was approached through the mesocolon without any problems, but in case 3, no attempt was made to identify the Warren shunt because of the presence of dense adhesions. All patients are alive and well with normal hepatic function.

## DISCUSSION

The objective of the Warren shunt is to decompress the esophageal varices through the short gastric and splenic veins via the splenorenal shunt.<sup>1, 5</sup> Hemodynamic studies have shown a decrease in portal perfusion pressure after the construction of a distal splenorenal shunt.<sup>8</sup> This is partly explained by the elimination of inflow from the splenic vein that is diverted into the vena cava and partly by a "leak" from the high-pressure mesenteric venous bed into the decompressed gastrosplenic bed.

Consequently, a decrease in the total hepatic blood flow has been observed in patients with splenorenal shunts.<sup>8</sup> At the same time, it is known that the Warren shunt is a high-flow shunt. Twofold increases in the blood flow of the splenic vein have been noted after the construction of distal splenorenal shunts.<sup>8</sup> Such hemodynamic alterations could have adverse effects on the transplanted liver and were the basis for initial concern when liver transplants were performed in these patients. It was feared that portal blood flow would be shunted away from the new liver by the old collaterals. The flow determinations of the venovenous bypass of our four patients demonstrated the insignificance of the contribution by the portal vein to portal perfusion. Little or no change in the blood flow was noticed when the cannula from the portal vein was removed for the portal vein reconstruction. The portal limb of the venous bypass was probably unnecessary since the splanchnic circulation appeared to be well decompressed via the vena cava. There were consistent anatomic changes; the diameter of the portal veins measured from 1.2 to 1.5 cm, which is smaller than that of other adult liver transplant recipients whose portal vein diameter average 2.5 cm. The decrease in the portal vein size has been demonstrated angiographically in patients with splenorenal shunts, and it is probably caused

by a “steal” that occurs with the gradual development of collateral hepatofugal channels.<sup>8</sup> However, the small size of the portal vein and the phleboscrosis observed in all four patients did not prevent the reconstruction of good anastomoses of the portal vein.

Because of concerns about the collateral circulation, plans were made to remove the venous collaterals surgically created by the Warren shunt or caused by its construction. Although replacement of the cirrhotic liver eliminates portal hypertension, there are conditions after hepatic transplantation that may be associated with an increased outflow resistance such as primary graft nonfunction, rejection, and fluid overload. In these situations, the blood flow will probably be drained via the shunt and away from the liver. The transplantations were relatively easy since no dissections had been performed in the hilum. Liver transplantation was performed first and then followed by the splenectomy when the patient was stable and the preoperative coagulopathy was corrected. Furthermore, the splanchnic circulation was decompressed after hepatic replacement, which facilitated the splenectomy and dissection of the shunt. All patients recovered and none had any evidence of complications from the portal vein. At the time this article was written, all patients were alive and well with normal liver function tests. Patency of the portal vein has been demonstrated in all patients after surgery by Doppler ultrasound.

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**Table 1**

Liver transplantation in patients with Warren shunts

Pt.	Age (yr)	Sex	Diagnosis	Interval from SRS to OLT (yr)	Shunt management	Survival after OLT (mo)
1	48	M	$\alpha_1$ -Antitrypsin deficiency	3	Splenectomy and division	13
2	37	F	Primary biliary cirrhosis	5	Splenectomy and division	14
3	32	M	Sclerosing cholangitis	1	Splenectomy	9
4	29	F	Cryptogenic cirrhosis	4	Splenectomy and ligation	7

*Legend:* SRS, Distal splenorenal shunt; OLT, Orthotopic liver transplantation.

**Table II**

Findings in patients with splenorenal shunts and end-stage liver disease

Pt.	Preoperative angiography			Intraoperative characteristics of portal vein		
	Portal perfusion	Warren shunt	Total bypass flow ml/min	Size (cm)	Phleboscrosis	Flow ml/min
1	Fair	Patent	3500	1.5	Present	500
2	Poor	Patent	2000	1.2	Present	ND
3	Poor	Patent	2400	1.2	Present	ND
4	—	—	2400	1.2	Present	ND

*Legend:* ND, Nondetectable.