Splenopancreatic Disconnection

Improved Selectivity of Distal Splenorenal Shunt

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Distal splenorenal shunt (DSRS) improves survival from variceal bleeding in nonalcoholic cirrhotics but not in alcoholic subjects. The metabolic response after DSRS is also different in alcoholic and nonalcoholic cirrhotics. Portal perfusion, quality of blood perfusing the liver, cardiac output, and liver blood flow do not change in nonalcoholics. In alcoholics, portal perfusion is frequently lost (60%), quality of blood perfusing the liver decreases, and cardiac output and liver blood flow increase. It is proposed that portal flow is lost in alcoholics *via* pancreatic and colonic collaterals after surgery. Elimination of this sump by adding complete dissection of the splenic vein and division of the splenocolic ligament to DSRS (splenopancreatic disconnection, SPD) could preserve portal perfusion, decrease shunt loss of hepatotrophic factor, and improve survival in alcoholic cirrhotics. This report compares data 1 year after surgery in two groups of cirrhotics: group I (8 nonalcoholic; 16 alcoholic) had DSRS without SPD; group II (17 nonalcoholic; 11 alcoholic) received DSRS + SPD. Methods: Portal perfusion grade, cardiac output (CO), liver blood flow (f), hepatic function (GEC), and hepatic volume (vol) were measured before and 1 year after surgery. Shunt loss of hepatotrophic factor was estimated by insulin response (change in plasma concentration over 10 minutes: AUC) after arginine stimulation. Results: Groups I and II were similar before surgery. Metabolically, nonalcoholics remained stable after both DSRS and DSRS + SPD. After standard DSRS, alcoholics lost portal perfusion (75%, $p < 0.05$), CO, and f increased ($p < 0.05$), and quality of blood perfusing the liver was decreased (GEC/f: $p < 0.05$). DSRS + SPD preserved portal perfusion better ($p < 0.05$) in alcoholic cirrhotics than did DSRS alone. After DSRS + SPD, the metabolic response in alcoholics resembled that of nonalcoholics. CO, f, and GEC/f remained stable. These data show: (1) DSRS + SPD preserves postoperative portal perfusion in alcoholic cirrhotics better than DSRS alone. (2) Metabolic response to DSRS + SPD is similar in alcoholic and nonalcoholic cirrhotics. (3) Because portal perfusion and metabolic integrity are preserved after DSRS + SPD, its use in alcoholic cirrhotics should improve survival.

DURING THE LAST 5 YEARS, endoscopic sclerotherapy has re-emerged as the safest method to treat acute bleeding from gastroesophageal varices.[1-4] However, there is a significant failure rate after sclerotherapy,[1,5-11] and surgery remains the only definitive way to prevent rebleeding. In nonalcoholic cirrhotics and patients with extrahepatic portal vein thrombosis or primary hepatic fibrosis, selective variceal decompression by distal splenorenal shunt (DSRS) represents optimal surgical therapy because postoperative portal perfusion is maintained for years and survival is improved.[12-16] In alcoholic cirrhotics, however, portal perfusion is lost in 60% of patients 1 year after DSRS, and survival is no better than that achieved by total portasystemic shunt.[15,16]

In 1984, Warren proposed that loss of portal perfusion after selective shunt occurred *via* the system of transpancreatic and colonic collaterals that developed after selective shunt between the high pressure portal circulation and the low pressure splenorenal anastomosis.[14,17] Loss of hepatotrophic factors (insulin) from the liver through this "pancreatic siphon" (Fig. 1)[18] was offered as one reason why selective shunt did not prolong survival in alcoholics who lost portal perfusion. Warren projected that, if the splenic vein were completely separated from the pancreas during selective shunt (splenopancreatic disconnection, SPD) (Fig. 2), the pancreatic siphon would not develop and postoperative portal perfusion and the selectivity of the operation could be preserved. Shortly after Warren's observations were published, Inokuchi described similar

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collateralization after selective shunt ("portal malcirculation") and corroborated its physiologic significance to the loss of portal perfusion.[19]

This report presents metabolic and hemodynamic data 1 year after surgery in a group of cirrhotics operated with DSRS plus SPD. For purposes of comparison, data from an unmatched but similar group of patients who received selective shunt without splenopancreatic disconnection are also presented.[17]

Material and Methods

Technical Aspects of DSRS + SPD

The patient is positioned with the left side elevated 20 degrees and the left arm adducted across the chest. A left subcostal incision is extended across the right rectus muscle. After dividing the gastrocolic ligament from the pylorus to the short gastric vessels, the splenocolic ligament is taken down, which exposes the hilum of the spleen, provides access to the lesser sac, splenic artery, and tail of the pancreas, and interrupts the major collateral pathway between the splenic vein and mesentery of the splenic flexure.

SPD is complete dissection of the splenic vein from the pancreas to its bifurcation at the splenic hilum. Dissection of the splenic vein should proceed on the vein with the posterior, inferior surface isolated before anterior and superior surfaces are approached.[20] The junction of the splenic and superior mesenteric vein should be controlled early in the dissection. Small splenopancreatic perforating veins are ligated and divided as they are encountered. Division of the splenic vein at its junction with the superior mesenteric vein will facilitate dissection of the splenic vein from the pancreas, as does intermittent clamping of the splenic artery to decrease blood loss. Superiorly rotating the pancreas with the surgeon's left hand behind the gland aids exposure as the splenic vein passes through the upper border of the pancreas (the pancreatic groove). In most cases, the splenic vein courses above the pancreas for several centimeters before entering the hilum of the spleen. Controlling the vein above the pancreas requires special care, since injury to the vein at the hilum may cause irreversible harm. If dissection of the splenic vein from the pancreas is difficult, complete mobilization of the spleen from its bed with rotation of both spleen and pancreas medially will allow dissection of the splenic vein from a posterior approach (Inokuchi maneuver).[19]

The splenorenal anastomosis is performed in the usual manner, as is interruption of the left and right gastric veins.[20]

Patient Populations

Selective decompression was performed in 111 patients between August 1983 and January 1986. Sixty-five patients had DSRS + SPD, and the remaining 46 patients received standard DSRS. The latter was usually performed in the emergent or urgent setting. Twenty-eight of the DSRS + SPD patients (17 nonalcoholic; 11 alcoholic) have been followed at least 1 year and define the study population.

An additional 10 patients with DSRS + SPD and 13 others who had standard DSRS had stimulated levels of insulin measured in their shunts at a median follow-up of 5 months (range: 3–15 months; $\bar{X}$ = 6.7 months) and 31 months (range: 6–60 months; $\bar{X}$ = 33.8 months), respectively.

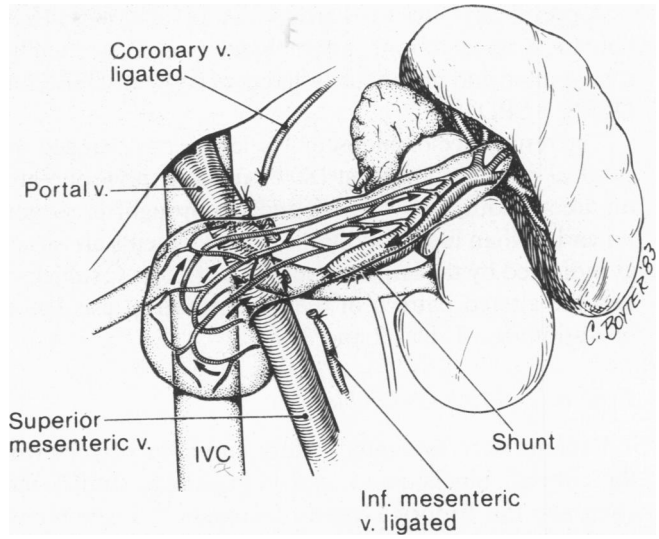

FIG. 1. Schematic of the "pancreatic siphon" that develops after DSRS, resulting in the loss of hepatotrophic factors from the liver. Arrows denote route of blood flow from the high pressure portal circulation to the low pressure splenorenal anastomosis.

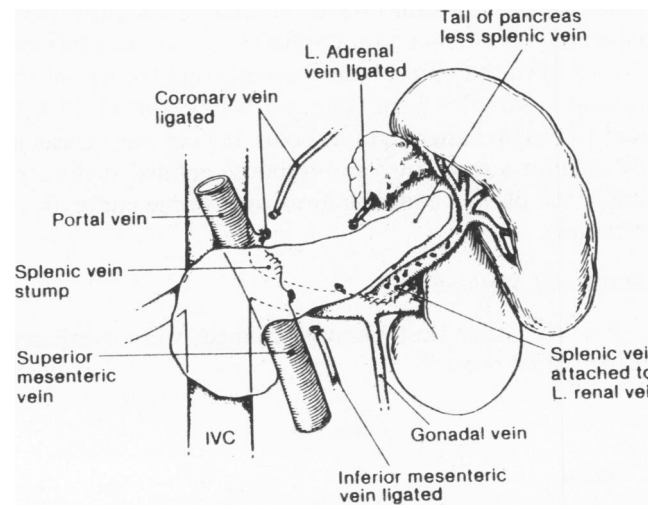

FIG. 2. DSRS + SPD. Note the complete separation of the splenic vein from the pancreas.

A previously published group[17] of 24 cirrhotics (16 alcoholic; 8 nonalcoholic) are included to compare the hemodynamic and metabolic differences between DSRS and DSRS + SPD.

The resurgence of endoscopic sclerosis has changed our surgical population. Most (80%) patients now operated on are sclerotherapy failures and are brought to surgery under less than ideal conditions. These patients are sicker, as indicated by the larger population of Class C subjects[11] and the altered pattern of their quantitative tests before operation (see Tables 2 and 3).

Evaluation Methods (Table 1)

Patients were evaluated before and after surgery with the clinical, biochemical, and hematologic studies that comprise the Emory Hepatic Database.[21] Liver biopsy confirmed the etiology of the cirrhosis.

Hepatic function was quantitated by galactose elimination capacity (GEC).[22] Liver blood flow was estimated by low dose galactose clearance (flow).[23] Cardiac output was measured by echocardiography or first pass nuclear cardiography (CO).[25] Portal perfusion was graded on the venous phase of the superior mesenteric angiogram: 1 (normal) → 4 (absent or reversal of flow).[26] Liver volume was computed from abdominal computed tomographic scan (vol).[24]

Quality of blood perfusing the liver was indexed by liver function divided by liver blood flow (GEC/flow).[17]

Insulin Studies

Loss of pancreatic hepatotrophic factor (insulin) through the shunt was estimated by measuring plasma insulin concentrations after intravenous arginine stimulation (50 ml of 10% solution/90 sec). Blood samples were drawn through a catheter placed *via* the femoral and left renal veins 2 cm into the splenorenal shunt during angiography. Simultaneous peripheral venous samples were drawn from the antecubital vein opposite the site of the infusion. Samples were collected before and at 2, 4, 6, and 10 minutes after arginine bolus. Insulin was measured by radioimmunoassay[27] and response defined as the area under the plasma concentration *versus* time curve (0–10 minutes).

Statistical Methods

Pre- to 1 year postoperative changes were compared using paired t-test.[28]

Results

Clinical

Splenopancreatic disconnection was performed in more than 90% of patients in whom it was attempted. The extra dissection extended surgery 30–60 minutes and increased transfusion requirements in some patients. Separating the entire length of the splenic vein from the pancreas was more difficult in alcoholic patients and subjects where chronic sclerotherapy had produced perivenular thickening and peripancreatic inflammation.

Operative mortality (in hospital or 30 days) after DSRS + SPD was 8% (5 of 65 patients). Four patients died of hepatic failure, which was linked to sepsis in three. The fifth patient died of complications related to intra-abdominal bleeding.

Two patients (3%) bled from recurrent varices in the early postoperative period and required portacaval shunt. Two others (3%) had intra-abdominal bleeding which required re-exploration. This incidence of rebleeding is not higher than after standard DSRS.[15,16] It had been anticipated that the extra dissection and subjection of the pancreas to undecompressed portal hypertensive pressures would increase postoperative bleeding.

Quantitative

The pre- to 1 year postoperative data for the 28 patients operated with DSRS + SPD is presented in Table 1. Comparison of data from the DSRS + SPD group with patients receiving DSRS without SPD is listed in Tables 2 and 3 and portrayed in Figures 3–8.

Portal perfusion. Portal perfusion was preserved in nonalcoholics and lost in alcoholics 1 year after DSRS without SPD.[17] Portal perfusion was preserved in both alcoholics and nonalcoholics 1 year after DSRS + SPD (Table 1). DSRS + SPD preserved portal perfusion better ($p < 0.05$) than DSRS without SPD in the alcoholic subset (Fig. 3).

Cardiac output. Cardiac output increased ($p < 0.05$) 1 year after DSRS without SPD when portal perfusion was lost in the alcoholic subset (Fig. 4). Cardiac output did not increase in nonalcoholics after standard DSRS when portal perfusion was preserved (Table 3). Cardiac output did not increase in alcoholics or nonalcoholics operated on with DSRS + SPD. These changes in cardiac output were significantly different ($p < 0.05$) in alcoholics operated on with DSRS + SPD compared to alcoholics receiving DSRS without SPD (Fig. 4).

Liver blood flow. Liver blood flow increased ($p < 0.05$) in alcoholic cirrhotics after standard DSRS 1 year after surgery (Fig. 5, Table 3).[17] Liver blood flow decreased ($p < 0.05$) in alcoholic cirrhotics 1 year after DSRS + SPD. Liver blood flow was higher ($p < 0.05$) before surgery in alcoholics who received DSRS + SPD. The difference between the change in liver blood flow (LBF, DSRS + SPD: postoperative–preoperative *vs.* LBF, DSRS without SPD: postoperative–preoperative) was also significant ($p < 0.05$) (Fig. 5). Liver blood flow did not increase after

TABLE 1. *Primary Splenopancreatic Disconnections—Preoperative and 1 Year*

				Bilirubin (0.1–1.1 mg/dl)		Albumin (3.2–5.6 g/dl)		P. T. (sec. prolonged)		Portal Perfusion		GEC (500 ± 50 mg/min)		Liver Volume (1493 ± 230 cm^3)		LBF (1378 ± 218 ml/min)		Cardiac Output (3.5–5.5 L/min)	
Patient	Age	Sex	Etiology	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Alcoholic																			
1	65	M	Alc	1.9	0.5	3.3	3.2	0.4	0.0	II	II	281	310	1842	1459	2117	1975	5.3	6.7
2	62	F	Alc	2.4	0.8	3.2	4.2	0.9	0.9	II	II	387	417	992	1107	924	1221	7.3	6.5
3	60	M	Alc	0.4	0.5	3.1	4.2	1.7	0.5	II	I	—	354	1850	1835	—	1691	8.1	9.1
4	70	M	Alc	1.1	1.1	3.8	3.3	1.1	0.5	II	I	—	334	1409	1141	865	726	—	—
5	51	M	Alc	2.0	2.6	2.7	3.0	2.7	2.7	II	IV	255	198	2160	2285	1922	1543	7.7	12.1
6	64	M	Alc	0.9	1.1	3.8	3.7	1.7	0.9	III	I	321	333	2349	2074	1987	1480	—	10.9
7	49	M	Alc	0.8	1.9	2.5	4.1	1.3	0.7	II	II	401	312	1626	1403	2037	1466	7.6	8.8
8	64	M	Alc	1.6	0.5	3.9	4.5	1.5	1.5	I	I	266	300	2323	1723	2016	1186	7.7	5.1
9	68	M	Alc	0.5	0.7	3.8	3.8	0.0	0.9	I	I	255	225	1056	944	1142	784	6.9	6.3
10	28	M	Alc	1.4	2.1	3.5	4.4	0.4	0.0	I	I	356	353	—	1399	1798	1349	8.6	—
11	30	M	Alc	2.2	2.8	3.1	3.2	3.0	2.0	II	III	303	297	1910	1470	1453	1233	15.9	11.7
Nonalcoholic																			
1	32	F	CAH	2.8	1.3	3.6	4.0	2.1	0.6	I	I	419	353	765	767	1221	918	3.0	—
2	59	M	BC	0.2	1.1	3.6	3.3	1.2	1.0	II	I	289	295	1106	995	1070	—	9.6	6.2
3	27	F	CAH	0.5	0.3	4.2	4.1	1.0	1.0	I	I	327	363	1128	355	1431	1167	6.1	5.8
4	61	F	CAH	0.9	2.4	2.9	2.3	1.6	2.2	I	I	267	179	753	618	766	700	7.8	7.7
5	57	F	CAH	0.9	1.4	3.2	3.5	1.2	0.9	II	I	269	296	1301	1418	922	916	5.9	5.6
6	32	M	CAH	1.1	2.1	3.4	3.2	0.1	0.5	I	II	376	273	1902	1002	1466	1783	5.3	7.6
7	44	F	CAH	0.7	1.2	3.8	4.1	0.0	0.0	I	I	364	368	977	918	943	719	—	8.2
8	62	F	CAH	0.7	1.2	4.4	4.2	0.5	0.3	II	II	461	378	1633	1270	1053	847	6.8	7.4
9	39	M	CAH	2.7	17.7	2.5	2.5	1.2	3.1	II	IV	281	306	1566	2471	765	1729	—	6.8
10	32	M	SCH CHOL	1.0	5.2	3.6	3.2	0.3	0.1	II	I	410	354	1677	1504	1601	1411	4.5	8.4
11	66	F	BC	0.9	1.1	3.4	3.3	0.0	0.2	I	I	466	353	1333	1163	933	1211	13.7	5.8
12	61	F	CRYPT	0.9	0.7	3.0	3.8	1.4	0.9	I	I	269	323	1000	1093	1116	692	5.6	—
13	59	M	CRYPT	2.0	1.6	3.0	3.6	1.1	0.1	I	I	314	485	2180	2510	1596	1257	10.2	7.2
14	58	M	CAH	1.8	4.0	3.4	3.2	1.9	0.7	I	I	354	362	1644	1717	1375	1221	8.7	6.2
15	61	M	CAH	1.7	1.1	2.8	3.3	3.2	1.5	II	I	289	286	1049	1232	787	1149	9.8	6.3
16	62	F	CRYPT	4.2	1.8	4.6	1.2	1.3	1.2	III	IV	160	181	801	823	988	—	9.7	9.1
17	47	M	SCH CHOL	0.9	1.5	3.1	3.0	0.8	1.7	I	I	351	286	1647	1576	1560	1439	9.7	7.7

CAH = chronic active hepatitis; BC = biliary cirrhosis; SCH CHOL = sclerosing cholangitis; CRYPT = cryptogenic cirrhosis.

TABLE 2. *Biochemical Changes before and at 1 Year after DSRS and DSRS+SPD*

	Total		Alcoholic		Nonalcoholic	
	Preop	1 Year	Preop	1 Year	Preop	1 Year
Bilirubin (0.1–1.1 mg/dl)						
DSRS+SPD	1.4 ± 0.9	2.3 ± 3.2	1.5 ± 0.8	1.4 ± 0.8	1.4 ± 1.0	2.7 ± 4.0
DSRS	1.1 ± 0.5	2.0 ± 0.9	1.0 ± 0.4	2.1 ± 1.0	1.1 ± 0.5	1.8 ± 0.7
Albumin (3.2–5.6 mg/dl)						
DSRS+SPD	3.4 ± 0.5	3.5 ± 0.7	3.4 ± 0.5	3.8 ± 0.5	3.4 ± 0.6	3.3 ± 0.8
DSRS	3.8 ± 0.5	3.6 ± 0.4	3.6 ± 0.5	3.6 ± 0.4	3.9 ± 0.4	3.5 ± 0.5
Prothrombin time (sec. prolonged)						
DSRS+SPD	1.2 ± 0.9	1.0 ± 0.8	1.4 ± 0.9	1.1 ± 0.8	1.1 ± 0.8	0.9 ± 0.8
DSRS	2.2 ± 1.0	1.6 ± 1.0	2.6 ± 0.9	1.7 ± 0.9	1.8 ± 1.0	1.5 ± 1.3

either operation in patients with nonalcoholic cirrhosis (Table 3).

Quantitative liver function (Tables 1 and 3). Galactose elimination capacity did not decrease significantly in any of the patient populations after DSRS with or without SPD.

TABLE 3. *Function and Hemodynamic Changes Preoperative and at 1 yr*

	DSRS (Mean ± S.D.)		DSRS + SPD (Mean ± S.D.)	
	Preop	1 Year	Preop	1 Year
GEC (500 ± 50 mg/min)				
Total	350 ± 99	315 ± 81	323 ± 66	316 ± 66
Alcoholics	337 ± 99	305 ± 69	313 ± 56	305 ± 65
Nonalcoholics	326 ± 98	324 ± 93	327 ± 72	320 ± 73
Flow (ml/min)				
Total	1089 ± 267	1152 ± 288	1399 ± 483	1207 ± 347
Alcoholics	1133 ± 265	1339 ± 406	1717 ± 545	1297 ± 346
Nonalcoholics	1045 ± 269	964 ± 169	1203 ± 300	1102 ± 321
GEC/flow × 100				
Total	32.1 ± 7.9	27.8 ± 8.1	25.8 ± 9.0	28.9 ± 1.0
Alcoholics	30.0 ± 7.3	24.7 ± 9.2	20.0 ± 9.0	23.4 ± 6.4
Nonalcoholics	35.2 ± 7.8	33.5 ± 6.6	30.1 ± 7.6	32.3 ± 10.4
Volume (1493 ± 2309 cm^3)				
Total	1801 ± 517	1574 ± 562	1518 ± 523	1403 ± 52
Alcoholics	2113 ± 600	1836 ± 637	1827 ± 520	1677 ± 602
Nonalcoholics	1489 ± 433	1311 ± 487	1306 ± 431	1216 ± 464
GEC/volume × 100				
Total	21.0 ± 6.0	21.2 ± 5.5	24.6 ± 9.9	25.1 ± 8.8
Alcoholics	17.3 ± 7.0	18.5 ± 6.6	19.5 ± 9.4	20.9 ± 8.3
Nonalcoholics	24.6 ± 4.1	25.6 ± 4.9	27.2 ± 9.4	28.3 ± 8.3
Cardiac output (3.5–5.5 L/min)				
Total	6.9 ± 1.5	9.3 ± 2.9	8.3 ± 2.8	7.9 ± 1.9
Alcoholics	6.8 ± 2.0	10.7 ± 3.0	8.3 ± 3.2	8.3 ± 2.6
Nonalcoholics	6.9 ± 1.0	7.9 ± 2.9	8.3 ± 2.5	7.2 ± 1.1

Quantitative liver function/liver blood flow. GEC/unit flow indexes hepatic function for a given volume of flow and is a measure of the quality of blood perfusing the liver (Fig. 6, Tables 1 and 3).[17] GEC/unit flow decreased ($p < 0.05$) in alcoholics who lost portal perfusion after DSRS without SPD. This resulted from a decrease in GEC and increase in liver blood flow ($p < 0.05$). When portal perfusion was preserved in alcoholics after DSRS + SPD, GEC decrease was less and liver blood flow also decreased. The difference in change in GEC/flow (GEC/f: DSRS + SPD, postoperative–preoperative *vs.* DSRS without SPD, postoperative–preoperative) was significantly different ($p < 0.05$) (Fig. 6, Table 3). GEC/f did not change in nonalcoholic cirrhotics after either operation when portal perfusion was preserved.

Liver volume. Liver volume decreased ($p < 0.05$) in all patients after both operations (Fig. 7, Tables 1 and 3).[17] Similar changes in liver volume have been observed in unoperated cirrhotics treated with chronic sclerotherapy.[11,30] Liver function/volume (GEC/vol × 100) was lower ($p < 0.05$) before surgery in alcoholic cirrhotics;

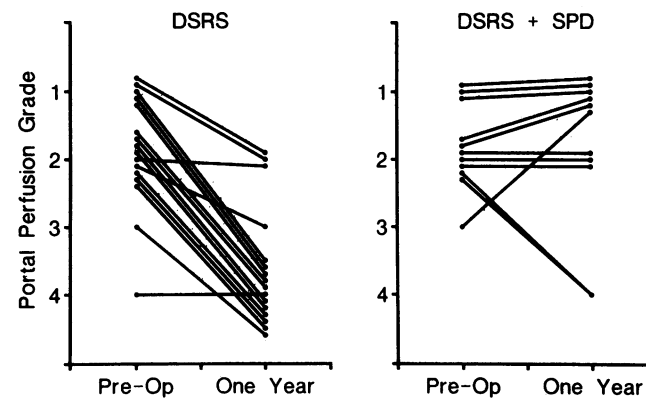

FIG. 3. Portal perfusion before and after operation in alcoholic patients. One represents normal perfusion; 2 and 3 are intermediate grades of perfusion; 4 is absent perfusion. Perfusion is better maintained at 1 year in the DSRS + SPD group.

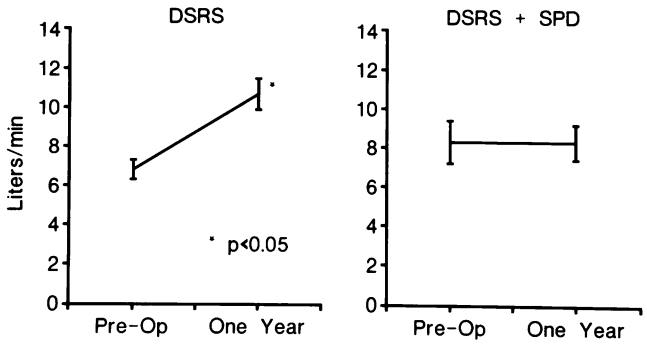

FIG. 4. Pre- and postoperative cardiac outputs in alcoholic patients ($\bar{X}$ ± S.E.). Cardiac output is not increased at 1 year following DSRS + SPD.

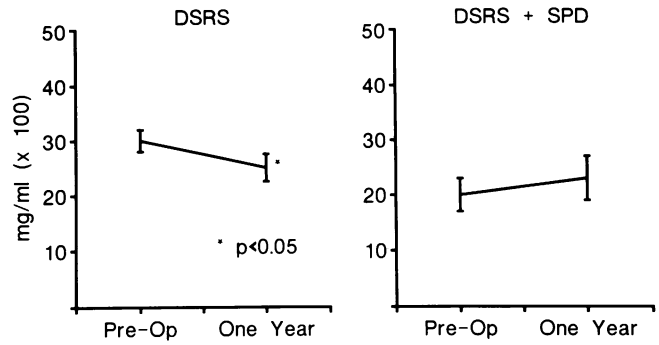

FIG. 6. Liver function per unit flow (GEC/flow × 100) before and after operation in alcoholic patients ($\bar{X}$ ± S.E.). Lower values indicate that an increased blood flow is needed to maintain a specific level of hepatocyte function. The quality of blood perfusing the liver decreased following DSRS while remaining unchanged at 1 year following DSRS + SPD.

patients with alcoholic cirrhosis having selective shunt had larger livers. This index (GEC/vol × 100) did not change significantly in alcoholics or nonalcoholics after either operation.

Insulin studies (Fig. 8). Peripheral and shunt insulin levels rose ($p < 0.05$) after arginine stimulation. Shunt levels of insulin were greater ($p < 0.05$) in patients with a pancreatic siphon after DSRS without SPD at 2 and 4 minutes after arginine stimulation than in patients without a pancreatic siphon after DSRS + SPD. Total loss of insulin over 10 minutes was also greater ($p < 0.05$) (Fig. 8) in patients who had a pancreatic siphon after DSRS without SPD compared to patients without a pancreatic siphon (AUC).

Discussion

SPD was conceived and implemented as a technical addition to DSRS to preserve portal perfusion and maintain the selectivity of the operation. Rikkers was first to document that nonalcoholics maintained portal perfusion better than alcoholics after selective shunt.[14] Zeppa made the crucial observation that survival was better in nonalcoholics than alcoholic cirrhotics after selective shunt.[31] Henderson showed that when portal perfusion was lost after selective shunt in alcoholics, the hyperdynamic state characterized by an increase in cardiac output and liver blood flow also developed. Henderson postulated that the loss of portal perfusion and the hyperdynamic state were linked to decreased survival after selective shunt in the alcoholic.[17] Other data reinforced this concept. Although quantitative liver function was maintained 1 year after selective shunt when portal perfusion was lost,[17] longer follow-up showed that liver function decreased and the incidence of hepatic encephalopathy increased in patients without prograde portal flow.[14] The 10-year follow-up of Emory's controlled trial reinforced the importance of maintaining portal perfusion over time.[15] The only patients with patent shunts who maintained hepatic function and encephalopathy-free existence were those in whom portal perfusion was preserved.[15]

These observations led to the concept of the pancreatic siphon (Fig. 1) and the addition of the SPD to selective shunt. DSRS + SPD eliminates the pancreatic siphon and preserves postoperative portal perfusion in the alcoholic cirrhotic better ($p < 0.05$) than DSRS without SPD. These

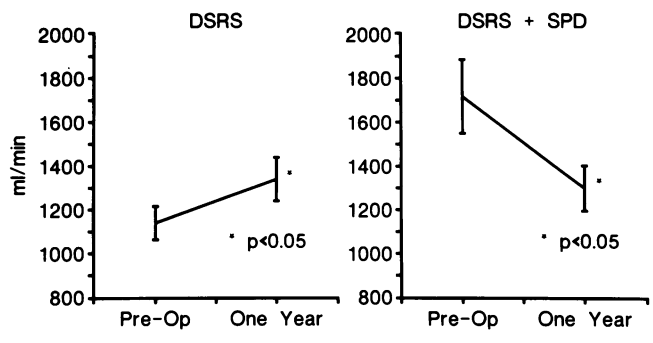

FIG. 5. Liver blood flow before and after operation in alcoholic patients ($\bar{X}$ ± S.E.). Liver blood flow significantly increased at 1 year after DSRS while falling after DSRS + SPD.

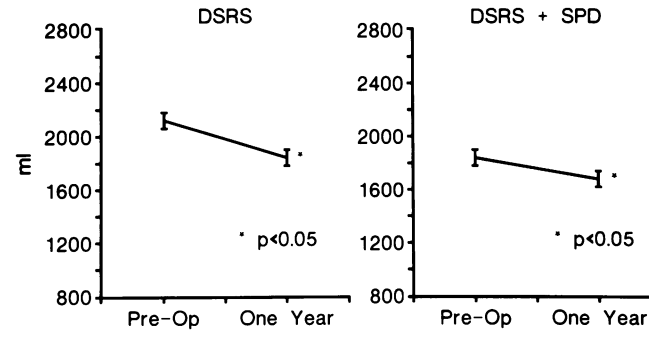

FIG. 7. Liver volume before and after operation in alcoholic patients ($\bar{X}$ ± S.E.). Both DSRS and DSRS + SPD show a decrease in liver volume at 1 year, and there is no difference in the rate of decrease between the two operations.

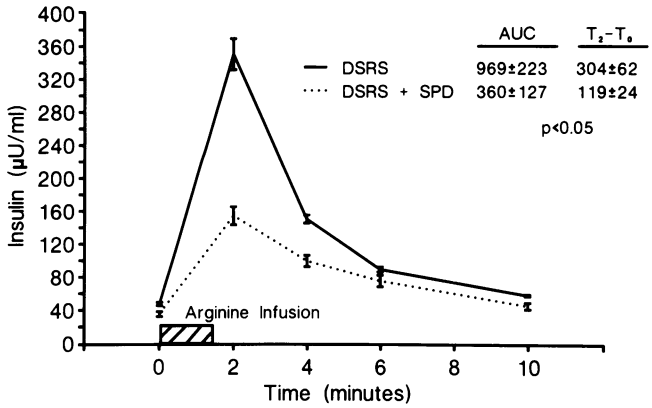

FIG. 8. Shunt insulin levels following stimulation of the pancreas by arginine infusion ($\bar{X}$ ± S.E.). Loss of insulin through the splenorenal shunt is greater after DSRS, as denoted by the large area under the insulin concentration curve (AUC).

data are supported by clinical experiments published by Inokuchi.[19] In his group of nonalcoholic subjects, Inokuchi emphasized the importance of minimizing the length of splenic vein left in the pancreas in communication with the portal vein. Even a short segment of splenic vein provides the origin for the development of an extensive network of collaterals that can—over time—enlarge and siphon flow from the high pressure portal circulation to the low pressure shunt. We concur with Inokuchi's concept and feel that the most efficacious approach is to harvest the entire splenic vein from its junction with the superior mesenteric vein to the splenic hilum. The Italian group[32] has also presented experience with a modified SPD that reinforces data presented in this report.

The data in this study also show that concurrent with preservation of portal perfusion after DSRS + SPD, the hyperdynamic state does not evolve in the alcoholic cirrhotic. The absence of the pancreatic pathway appears to protect against the development of a chronic hyperdynamic state. All patients after both DSRS and DSRS + SPD show a mild hyperdynamic response early in the postoperative period, which disappears after several months in patients who maintain portal flow (unpublished data). The early development of a low resistance collateral pathway (through the pancreas) during this phase may be critical to both loss of portal flow and the persistence of the hyperdynamic state. These possibilities remain conjectural. The data from this study show good maintenance of portal flow, no hyperdynamic response, and a favorable function/unit blood flow ratio in the alcoholic population. The hemodynamic and metabolic response to DSRS + SPD in alcoholics is similar to that seen after DSRS in nonalcoholics and unoperated patients treated with chronic sclerotherapy. Further work is required to define the mechanisms by which this has been achieved.

These data also show that when portal perfusion is preserved after DSRS + SPD, less insulin is lost through the shunt. This study does not show that more insulin was delivered to the liver. Further studies must be done to show if this occurs and to determine if hepatocyte integrity is enhanced.[33] This will require direct sampling of portal blood.

This report has focused on the alcoholic subset because this group loses portal perfusion after standard selective shunt. Should selective shunt plus disconnection be performed in nonalcoholics whose portal perfusion and liver function is already preserved long-term by standard shunt? We believe so because the pancreatic siphon also develops in nonalcoholics. Indeed, it was the loss of liver volume in the nonalcoholic (whose change in liver size is not subject to bouts of alcoholic hepatitis) that directed attention to the pancreatic siphon. We now know that loss of liver volume occurs in unoperated patients[11,30] and probably represents the natural history of disease. The very important work of Professor Inokuchi proves the value of separating splenic vein collaterals from the portal circulation in the nonalcoholic cirrhotic.[19]

The selective shunt plus splenopancreatic disconnection controls rebleeding as well as standard distal splenorenal shunt. The results of this initial experience show that only two patients with patent shunts rebled from varices (3%). One additional patient—one of the hospital mortalities—also bled from varices after disconnection, but bleeding was associated with terminal liver failure. We believe, however, that variceal bleeding after disconnection is hemodynamically different from variceal bleeding after standard selective shunt. After standard selective shunt, renal vein hypertension is common (10%) and constitutes the major cause of early rebleeding.[16] Complete dissection of the splenic vein from the pancreas eliminates the splenopancreatic tributaries and collaterals between the retroperitoneum and splenic vein that may aid variceal decompression. Hence the full dissection, coupled with gastric devascularization and coronary azygous disconnection, leaves the short gastric veins as the only pathway to decompress gastric and esophageal varices. In the immediate postoperative period, this may not provide an adequate outflow tract. All three patients who bled after DSRS + SPD did so from gastric varices near the short gastric vessels. The overall incidence of postshunt variceal bleeding has not been increased to date, and, while the pathophysiology may be different, we project that short gastric hypertension should be no more of a temporary clinical problem than renal vein hypertension.[16]

In summary, these data show that addition of complete dissection of the splenic vein plus interruption of the splenocolic ligament can preclude the development of pancreatic siphon and result in better preservation of portal perfusion after selective shunt. This allows the following conclusions: (1) DSRS + SPD preserves postoperative portal perfusion in the alcoholic and nonalcoholic cirrhotics. (2) SPD maintains portal perfusion in alcoholics

($p < 0.05$) better than the standard DSRS. (3) The hemodynamic and metabolic responses to DSRS + SPD are similar in the alcoholic and nonalcoholic population. (4) Hemodynamic stability and maintenance of the quantitative functions may be explained by better perfusion of the hepatocytes with portal blood and its hepatotrophic factors. (5) Five-year survival of alcoholics and even nonalcoholics may be improved after splenopancreatic disconnection.

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Discussion

DR. KIYOSHI INOKUCHI (Fukuoka, Japan): Many thanks for the privilege of the floor. I am very pleased to have listened to Dr. Millikan's fine presentation and to have looked at his beautiful operation of splenopancreatic disconnection at Emory University Hospital the day before yesterday. I feel that you have overcome problems inherent to the distal splenorenal shunt, such as loss of hepatic portal perfusion occurring after