Hepatopedal Flow Restoration in Patients Intolerant of Total Portal Diversion

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This report describes an experience with operative restoration of hepatopedal portal blood flow in five patients intolerant of total splanchnic shunting. Portal flow was reestablished by takedown of the total shunt and construction of a selective, distal splenorenal shunt, or by isolation and arterialization of the hepatic limb of the shunted portal vein. In two patients, shunt revision was undertaken electively for chronic encephalopathy, which had been unresponsive to low-protein diet, intestinal antibiosis and oral lactulose. Eighteen and 48 months after operation, both patients have had no encephalopathy on an unrestricted protein intake, and work actively as homemakers. Needle liver biopsies showed enhanced mitotic activity in the early postoperative period, suggesting hepatocyte regeneration. In three patients, shunt conversion or arterialization was undertaken in desperate circumstances, characterized by liver failure (bilirubin > 10 mg/dl, albumin < 2.5 g/dl, prothrombin time > 16 sec), coma, and respirator dependency. Although the patients showed immediate, marked improvement in mentation, all three died of intraabdominal hemorrhage in the first few postoperative days, in spite of maximum blood product support. Two conclusions can be drawn from this limited experience: (1) at a time of election, restoration of hepatopedal portal flow can be accomplished with considerable benefit in patients with side-to-side portacaval or hemodynamically equivalent shunts, and (2) similar procedures in patients with fulminant liver failure are unlikely to succeed.

OPERATIVE DECOMPRESSION of splanchnic venous hypertension is remarkably effective in abolishing the threat of recurrent variceal hemorrhage,[1,2] but the procedure is haunted by the specter of encephalopathy and associated hepatic failure. Portal bypass does not significantly increase the incidence of encephalopathy over that seen in unshunted cirrhotics with comparable liver disease, but in shunted patients, encephalopathy is more likely to be incapacitating and to be allied with a diminished probability of survival.[1,3] Nevertheless, most postshunt encephalopathy is clearly episodic and easily treated by some combination of dietary protein restriction, catharsis, and antibiotic suppression of intestinal bacteria.[4]

In a very small number of patients, the episodes of postshunt encephalopathy become more frequent and less amenable to treatment, culminating in a state of chronic mental and neurologic impairment, whereby the patients become totally dependent on other family members and the hospital. This rare form of true chronic encephalopathy is a harbinger of early demise, abetted perhaps by a continual need to limit protein intake severely. Occasionally, this entire scenario is compressed into the first postoperative month. These unfortunate patients appear to tolerate total portal bypass for 3 to 7 days, and then rapidly become profoundly encephalopathic, with deteriorating liver function, despite vigorous medical treatment and the absence of any substantial gastrointestinal hemorrhage. This report describes our attempts to reverse this lethal progression, in both acute and chronic circumstances, by intervening operatively to restore hepatopedal portal blood flow.

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Methods

From 1973 to 1981, 114 individuals were entered into an ongoing, single-institution, prospective study of adult cirrhotic patients requiring operative splanchnic decompression for variceal bleeding. Selective distal splenorenal shunting[5] was introduced into this experience in 1976. The following year, the intuitive attractiveness of selective shunting and the impact of several reports documenting virtues of portal blood flow, almost regardless of source,[6-9] prompted us to develop criteria for offering patients, intolerant of total portal diversion, a second operation designed to restore their portal flow.

In the setting of chronic deterioration, patients were to have had at least 30 days of encephalopathy-related hospitalization in the year prior to their consideration, and to be heavily dependent on the care of others when

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TABLE 1. *Patients Selected for Operative Reestablishment of Hepatopedal Portal Flow*

Patient Number	Age (At Flow Restoration)	Sex	Cause of Cirrhosis	Original Shunt	Interval Between Procedures	Restoration by:
1	65	F	Alcohol	Side-to-side portacaval	7 Years	Conversion to SDSR*
2	72	F	Chronic active hepatitis	Side-to-side portacaval	8 Years	Conversion to SDSR
3	64	M	Alcohol	Side-to-side portacaval	3 Years	Arterialization of hepatic limb
4	51	M	Alcohol	Side-to-side portacaval	28 Days	Conversion to SDSR
5	48	F	Wilson's disease	End-to-side renosplenic	30 Days	Ligation of central splenic limb

* Selective distal splenorenal shunt.

they were outside of the hospital. These conditions had to be met at a time when the patients were receiving, as outpatients, a dietary protein intake of 40 g or less per day, and were being continually treated with a daily dose of at least 4 g of neomycin or 120 ml of lactulose. No level of abnormality was specified for plasma ammonia or any other liver function test.

In the acute setting, patients were to be free of substantive gastrointestinal bleeding; "coffee grounds" in the nasogastric aspirate and an occasional tinge of blood were accepted, but frank hemorrhage or the need for repetitive transfusion excluded the patient from consideration. Grade 3 encephalopathy[10] or coma was required in the face of vigorous catharsis, enemas, and intestinal antibiosis. Again, no aberrations of liver function tests were specified, but the serum Na^+ was to be 120 mEq/L or more, the K^+ in excess of 3.5 mEq/L, and the arterial pH less than 7.48. Since 1978, all patients have also had to have a negative computerized tomographic head scan.

Thus far, three individuals have met the parameters set forth for chronic deterioration, and two have fit the criteria delineating acute, refractory, postoperative encephalopathy and liver failure (Table 1). Each patient had had flexible endoscopy, a complete battery of liver function tests, and selective, celiac, and superior mesenteric angiography as part of the ongoing prospective study before the original shunt was done. Those in the chronic category had been followed with complete liver function tests at 3-month intervals for the first postoperative year, and then at least at 6-month intervals thereafter. Selective angiography was repeated before some of the flow-restoration operations in order to be certain that the splenic vein was patent. The patients in the acute category were hospitalized continuously between the two operations. These data were analyzed retrospectively to determine if there were predictors of intolerance to portal privation at the time that the original shunt was constructed, and to discern the characteristic features of acute and chronic deterioration.

Both acutely intolerant postoperative patients and one patient in the chronic category underwent their second operations under emergency circumstances; the other two patients, both with chronic encephalopathy, were operated on at a time of election. In three instances, hepatopedal portal flow was reestablished by constructing a distal splenorenal shunt and performing a meticulous portomesenteric-gastrosplenic disconnection,[11] including ligation of the coronary vein at its junction with the portal vein. As a final step, the original side-to-side portacaval shunt was taken down, favoring maintenance of the portal lumen at the expense of a modest divot in the inferior vena cava (Fig. 1). In one patient with a side-to-side portacaval shunt and incorrectable clotting abnormalities, arterialization of the hepatic limb of the portal vein was used as an alternative to shunt conversion, based on the premise that it would be advantageous to limit all dissection to a single area in a situation of tenuous hemostasis. The hepatic-portal limb of the side-to-side shunt was isolated with a single heavy ligature (Fig. 2), and then, with 4× magnification loupes, a 3-mm arteriovenous fistula was constructed between the proper hepatic artery and the isolated portal vein limb.

The remaining patient had Wilson's disease with massive ascites, variceal bleeding, and a 3-year history of recurrent difficulties with multiple peritoneovenous plastic shunts. The patient's agonizing experience with peritoneovenous shunting and the reputed poor tolerance of total portal bypass in advanced Wilson's disease[12] posed a dilemma which was addressed at the time of her first operation, by electing a readily convertible type of total shunt. The left renal vein was divided, and its central end sewn to the side of the splenic vein. The renal end of the transected renal vein was oversewn. All of the recognized portomesenteric and gastrosplenic venous connections were severed, although the coronary-

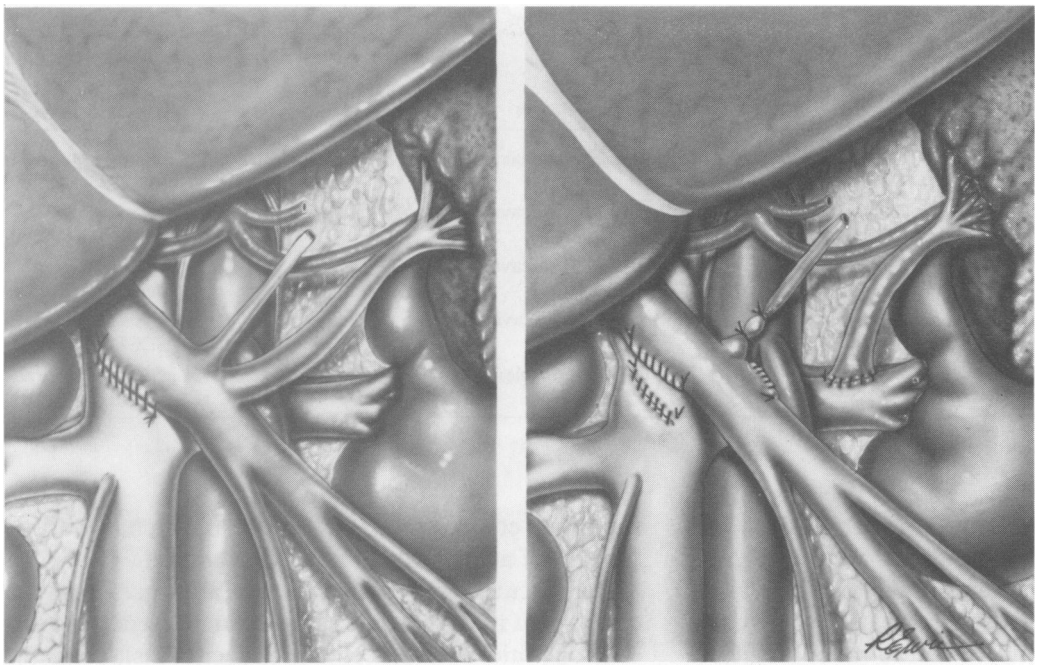

FIG. 1. Conversion from a total side-to-side portacaval to a selective distal splenorenal shunt.

portal vein juncture itself was never identified. At the second operation, the shunt was converted to a selective configuration by simply ligating the splenic vein between the shunt and the confluence of the splenic and superior mesenteric veins.

FIG. 2. Restoration of portal flow by isolation and arterialization of the hepatic portion of the portal vein (original shunt: side-to-side portacaval).

All patients had had liver biopsies at the time of their original shunt procedures. Repeat biopsies were done as soon as the abdomen was opened in the operations to restore potential flow, and serial liver biopsies were obtained after operation at one and two weeks and at one month in the survivors. Hematoxylin and eosin-, Masson's trichrome-, and Wilder's reticulin-stained sections were examined for histologic correlatives of portal privation, interoperative disease progression, and the trophic influence of restored portal blood flow.

The two patients who had their flow reestablished electively and their families were subjected to preoperative and repeated, semiannual, postoperative psychiatric social worker interviews to assess functional competence. Quantitation was achieved using a well-recognized scale which encompasses ratings for spontaneity and efficiency for 44 separate items in five major categories.[13] The patients who were operated on under emergent circumstances were too ill for this type of evaluation; changes in their functional status had to be assessed according to the rating system commonly used to score severity of head trauma.[14] These, and all other interval variables, were compared using student's t-test. The significance of categorical variables was tested by chi square analysis, using the Yates correction factor.

Results

At the time of their original shunt procedures, the five patients destined to be intolerant of total portal bypass were indistinguishable from the remaining shunt population in terms of age, liver function tests, potassium repletion, and renal function (Table 2). One was Child's class A, three were class B, and one was class C, a dis-

tribution similar to that of the larger group. Only one of the five had had preshunt encephalopathy, and that had been associated with an earlier, specific episode of bleeding. None of the five patients was diabetic. The only feature which tended to set these patients apart from the others was that four of the five shunts were done as emergency procedures, whereas only 35% of the rest of the shunt population required emergent variceal decompression. In part, because of the small number in the intolerant group, the difference was not statistically significant ($p > 0.1$).

The three patients who developed chronic encephalopathy all had at least one episode of confusion or semistupor that was severe enough to necessitate hospitalization during the first postshunt year (Fig. 3). Gross dietary indiscretion was the precipitating factor in two instances, and in the other, the episode was temporally related to bleeding from a duodenal ulcer. Because the causal events were so obvious, all three patients were discharged on a 70-g protein diet without specific medication other than multivitamins and antacids. Two patients, both women, enjoyed another 2 years without protein restriction, but then developed increasingly frequent episodes of encephalopathy, which were successively harder to abort. Each woman was admitted several times for intense treatment, including parenteral alimentation, and then discharged on a 40-g protein diet and 120 ml per day of lactulose. An oral branched-chain amino acid supplement was added for 6 weeks in one individual, without noticeable effect. Minor precipitating events, such as upper respiratory tract infections, became prominent, and by the sixth or seventh postoperative year, neither patient could exist at home without constant care and supervision. The reasons for hospitalization now included minor burns and large soft tissue ecchymoses from frequent falls.

During this course of clinical decay, there was no notable change in liver function tests. Bilirubin levels fluctuated between 1.5 and 2.0 mg/dl, the prothrombin time was prolonged by 2 or 3 seconds, albumin values hovered at the lower limit or normal, and plasma ammonia levels varied from 54 to 217 μmol/L (normal < 35 μmol/L), but there was no consistent trend.

TABLE 2. *Status at Time of Original Shunt (Mean Values ± SEM)*

Parameter	Conversion Patients (n = 5)	Remaining Shunt Population (n = 109)	Null Probability
Age (years)	56 ± 3	50 ± 1	>0.3
Albumin (g/dl)	3.5 ± 0.3	3.1 ± 0.1	>0.1
Bilirubin (mg/dl)	1.6 ± 0.7	2.6 ± 0.2	>0.3
SGOT (IU/L)	62.6 ± 22	80.5 ± 14	>0.7
PT (sec)	16.1 ± 1.6	14.5 ± 0.22	>0.1
PTT (sec)	42.4 ± 5.8	36.0 ± 0.69	>0.05
K^+ (mEq/L)	3.9 ± 0.3	4.0 ± 0.1	>0.7
BUN (mg/dl)	17.2 ± 4.1	18.4 ± 1.2	>0.8
Creatinine (mg/dl)	1.06 ± 0.19	1.10 ± 0.07	>0.9

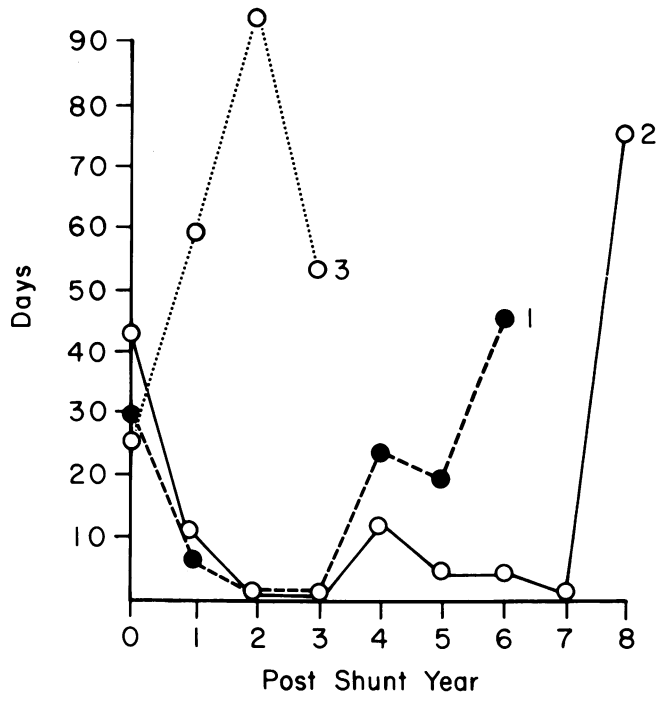

FIG. 3. Days hospitalized each year following total portal bypass for the patients exhibiting chronic deterioration.

These two patients had their total shunts converted electively to a selective configuration, and both underwent an immediate and lasting renaissance. Table 3 presents the data from their psychosocial evaluations before conversion and 3 months after operation. The significant increments in functional status have now persisted for 3 years in one individual and for 18 months in the other. Neither patient has any restriction on protein intake, nor do they take any medication to prevent encephalopathy. Both now live independently in their own apartments, whereas before, at best, they were invalids in their daughters' homes.

Liver biopsy specimens, taken at the time of shunt conversion, were essentially identical to the specimens obtained 7 and 8 years earlier. Even the liver of the patient with micronodular cirrhosis due to chronic active hepatitis showed no histological evidence of disease

TABLE 3. *Functional Life Scale Assessment Before and After Elective Conversion from Total to Selective Shunt*

	Patient 1		Patient 2	
Category	Pre	Post	Pre	Post
Cognition	7	73	4	87
Activities of daily living	3	82	8	100
Home activities	9	70	9	98
Outside activities	3	52	7	96
Social interaction	5	25	5	90
Mean	5	60*	7	94*

All scores are expressed as a percentage of applicable maximum.
* $p < 0.001$ pre *vs.* post rating.

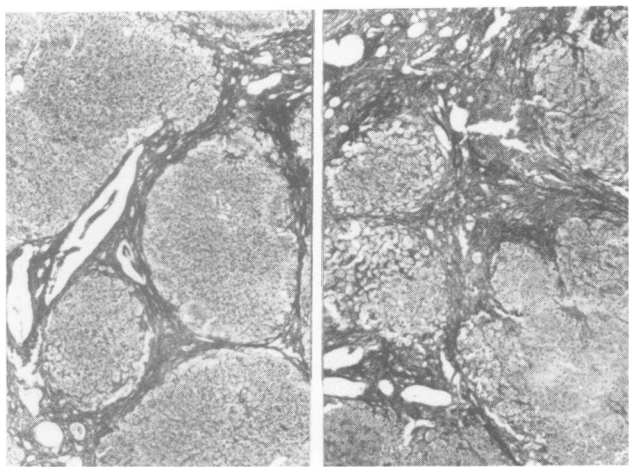

FIG. 4. The biopsy specimen from patient 2 taken at the time of original shunt (left) shows a comparable degree of cirrhosis 8 years later (right) (Wilder's reticulin stain, ×25).

progression (Fig. 4). Comparison of the postoperative liver biopsy specimens with the wedge specimens taken at the time of shunt conversion was hampered by the limited amount of tissue in the needle cores, but at one week, there was obvious hepatocyte proliferative activity (Fig. 5) which was not present in the operative specimens. Biopsies beyond the first postoperative week showed a few mitoses, but variability and sample size obscured any definition of the temporal span of replicative activity. Following conversion, there was no change in liver function profiles, except that after operation, plasma ammonia levels have never been measured above 50 μmol/L, even after an evening meal.

The third patient in the chronic category underwent a vagotomy and antrectomy for recurrent duodenal ulcer bleeding 2 years after his original shunt. The next year, he required frequent hospitalizations for grades 3 and 4 encephalopathy. His progressive encephalopathy was linked to an ominous trend in increasingly abnormal liver function tests, despite parenteral nutrition, oral branched-chain amino acid supplements, and chronic treatment with neomycin.

The two individuals in whom total portal bypass provoked acute postoperative encephalopathy and liver failure initially followed a salubrious course for 7 to 10

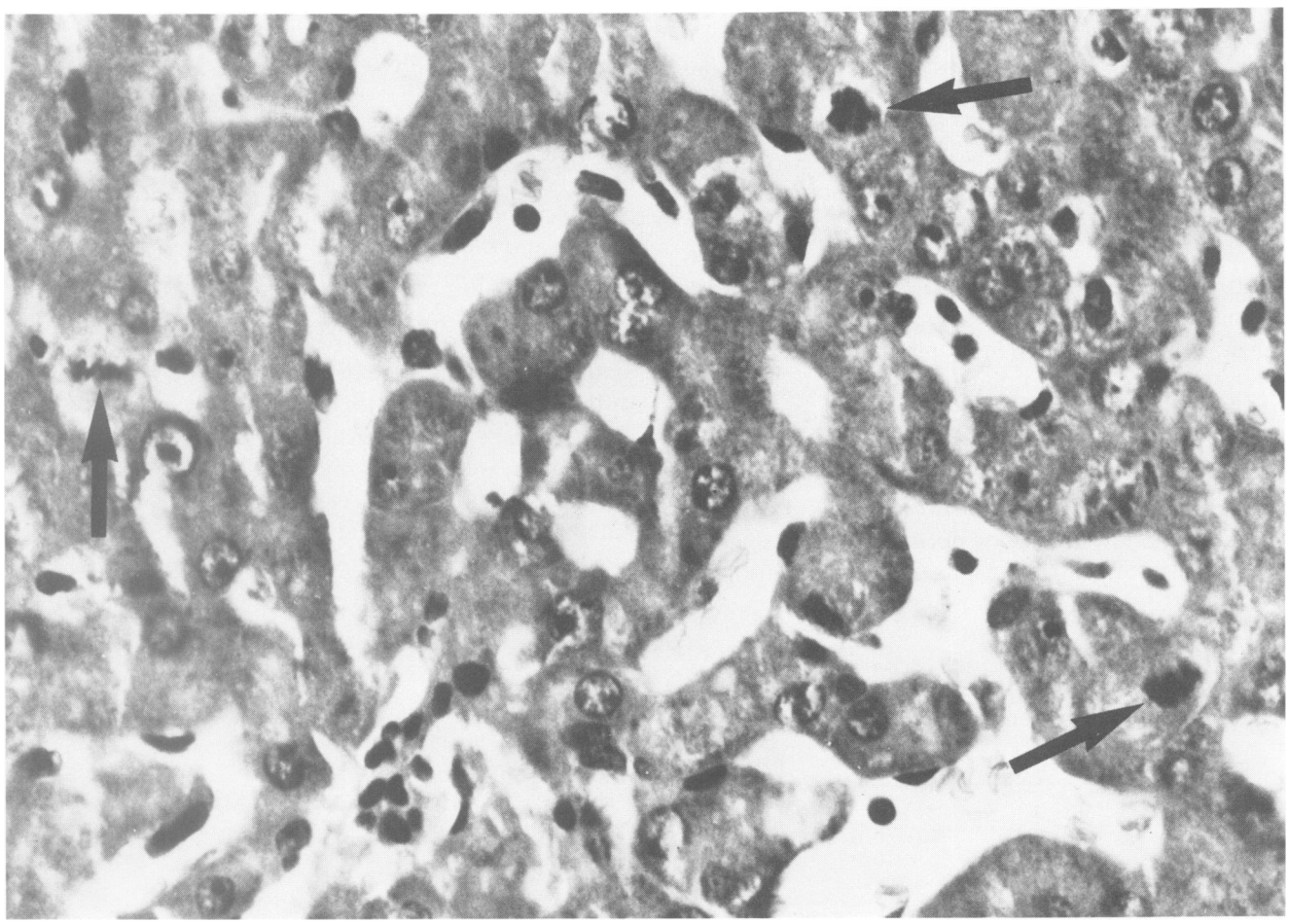

FIG. 5. Markedly increased mitotic activity is manifest by three mitoses in one high-power field (arrows). Biopsy specimen is from patient 1, 1 week after shunt conversion (H&E ×180).

TABLE 4. *Status at Time of Elective and Emergent Hepatopedal Flow Restoration (Mean ± SEM)*

Parameter	Elective	Emergent	Null Probability
Coma	0/2	3/3	—
Respirator dependent	0/2	3/3	—
Albumin (g/dl)	3.5 ± 0.2	2.3 ± 0.1	<0.01
Bilirubin	1.0 ± 0.05	30.1 ± 6.3	<0.05
SGOT (IU/L)	20 ± 4	130 ± 56	>0.2
PT (sec)	12 ± 0.5	23 ± 4	>0.1
PTT (sec)	36 ± 8	51 ± 5	>0.1

TABLE 5. *Results of Emergency Flow-Restorative Operations*

Clinical Summary	Patient 3	Patient 4	Patient 5
Blood transfused (liters)			
During operation	16.5	5.5	2.5
After operation	11.0	19.0	7.5
Glasgow Coma Scale*			
Before operation	7	5	5
After operation	12	12	13
Survival (days)	1	1	11

* $p < 0.001$ before *vs.* after operation.

days. They were ambulatory and on 20- to 40-g protein diets when their liver function tests began to deteriorate, coincident with the onset of encephalopathy. Progressive decay followed, despite all usual medical measures. These patients, and the man mentioned previously, came to their flow-restorative procedures with a clinical profile markedly different from that of the elective cases (Table 4).

Their operations were undertaken as emergency procedures, but with sufficient advance planning to assure availability of massive amounts of fresh-frozen plasma and platelet concentrates. In two instances, the procedures involved only a limited area of dissection. Nevertheless, control of intraoperative hemorrhage proved to be extraordinarily difficult, and the need for blood transfusion did not abate with the conclusion of the operations (Table 5). The outcome was dismal, with the longest survival being 11 days, but each patient awoke—at least transiently—and became communicative. The aggregate improvement in coma scale rating was significant and apparently related to restoration of portal blood flow.

The livers of the two patients, acutely intolerant of total diversion, were thought to be smaller than they were 4 weeks earlier. Histologically, there were extensive areas of necrosis and loss of hepatocyte mass to account for the gross impression of shrinkage (Fig. 6).

Discussion

Total portal diversion predisposes a patient to portalsystemic encephalopathy through three major mechanisms. First, lowering pressure in the mesenteric veins augments absorption of ammonia from the lumen of the colon[16] and may affect the absorption of other noxious agents as well.[5] Secondly, the shunt provides these agents with free access to the systemic circulation, without any opportunity for prior modification by the liver.[15] Thirdly, loss of the trophic influence of portal blood flow results in diminished liver cell mass and DNA content.[7,17] Despite these three avenues of adversity, postshunt encephalopathy is no more common than preshunt encephalopathy when appropriate, unshunted, control patients are used for comparison.[1,3] The same studies point out that encephalopathy in shunted patients is more likely to be severe, but of greater importance is the linkage of postshunt encephalopathy to a lesser probability of survival.[1,3,4] This lethal alliance is reflected in the anecdotal experience described in the current report.

The reported incidence of postshunt encephalopathy has varied from 16% to 77%,[4] reflecting variables in patient selection, thoroughness of follow-up, encephalopathy definition, and investigator enthusiasm,[18] but a reasonable figure from a well-followed series places the overall incidence at 32%.[4] Although it is stated often that the incidence of postshunt encephalopathy increases with time,[18] almost all patients who are going to have troublesome encephalopathy experience at least one episode during the first postshunt year,[3,4,15] as was the case in the present series. Fortunately, most postshunt encephalopathy is episodic[4] and related to dietary

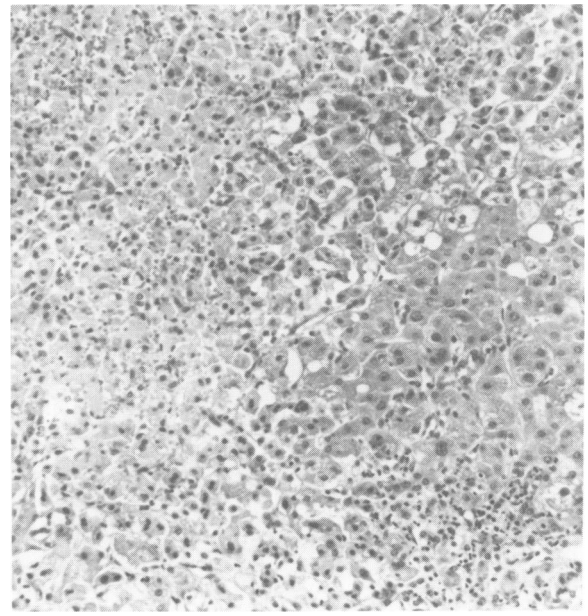

FIG. 6. Islands of intact parenchyma are evident (right), but there is extensive necrosis of liver. (H&E ×180).

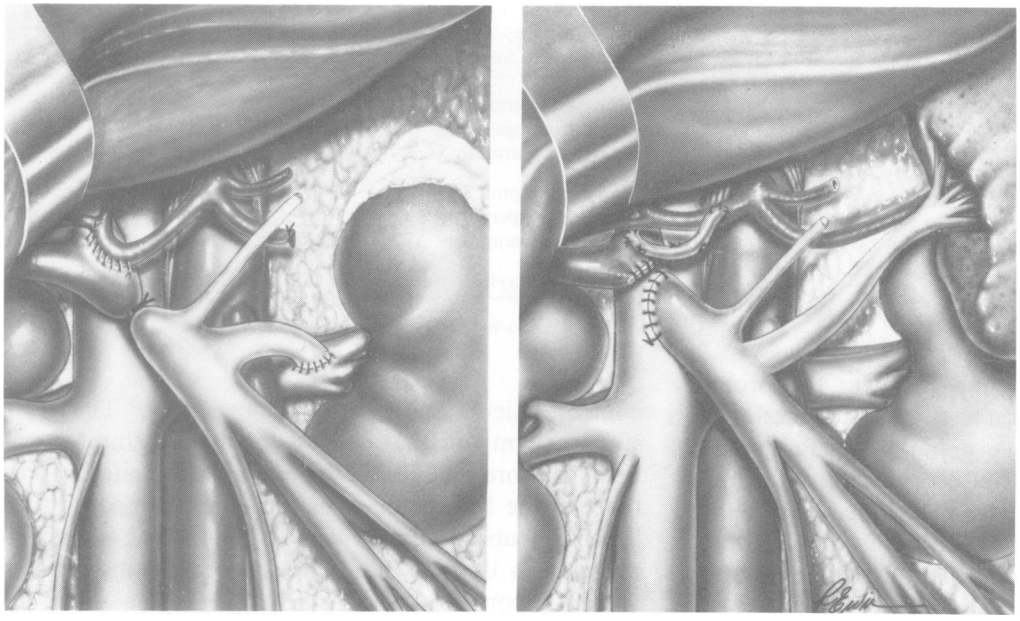

FIG. 7. Arterialization as a means of restoring hepatopedal portal flow in patients with central splenorenal (left) and end-to-side portacaval (right) shunts.

indiscretion, and thus can be controlled by relatively simple medical measures[10] combined with close supervision and education.[4]

Intuitively, patients with historic encephalopathy prior to shunting ought to have particular liability to incapacitating postshunt encephalopathy, but such is not the case.[3,4] Diabetes,[1] old age,[15] and Child's class[4] are the other time-honored risk factors, but severe postshunt encephalopathy is too uncommon and strikes too capriciously to permit prediction of risk for a given patient. Even retrospectively, nothing could be uncovered that would have distinguished the five principal subjects of this report at the time their original total shunts were done except for the ominous implications of advanced Wilson's disease in one instance.[12]

During the 1960s, there was enthusiasm for removing the colon from the mainstream of the gastrointestinal tract in patients with severe encephalopathy—either by excision, bypass, or simply constructing an end-ileostomy. The underlying principle was sound because it was known that the colon was the predominant site of ammonia absorption, but these procedures waned in popularity after a reported mortality rate of 26% in a controlled trial.[19] This experience was, of course, before modern methods of nutritional support were in use. The branched-chain enriched amino acid solutions offer particular promise in this regard,[20] and our small, discouraging experience with the oral preparation should not be overinterpreted.

Today, there are two operative approaches to the treatment of postshunt encephalopathy; both are directed at either maintaining or restoring hepatopedal portal flow, while simultaneously providing for decompression of esophageal and gastric varices. Selective shunting, the more popular method in North America,[5,18] has one practical and two theoretical advantages over arterialization of the portal vein in conjunction with an end-to-side shunt, a procedure more enthusiastically supported in Europe.[8,21] Pragmatically, the patency record for distal splenorenal shunts has been remarkably good,[11,18] whereas that for arterial-portal fistulae has not.[8] Theoretically, portal vein arterialization should be inferior to any technique providing for splanchnic venous flow through the portal vein towards the liver, because splanchnic venous blood has special properties,[7] present at best in diluted form in systemic blood.[22] In addition, the end-to-side shunt part of the arterialization scheme decompresses the mesenteric veins, facilitating ammonia absorption.[16] The distal splenorenal shunt does become less selective with the passage of time,[11] but it never completely decompresses the mesenteric veins or the liver sinusoids.[18]

Conversion from a side-to-side portacaval shunt to a selective distal splenorenal shunt was easier than anticipated because there was minimal tissue reaction around the patent shunt, and because dissection of the splenic vein could be done in the absence of portal hypertension. The field was draped so as to include a groin area if a vein graft were needed, but apparently a side-to-side portacaval shunt can often be taken down with preservation of the portal lumen and only a modest diminution in the calibre of the inferior vena cava. This

TABLE 6. *Documented Instances of Shunt Ligation for Encephalopathy*

Author (Year)	Elective/Emergency	Post-shunt Interval	Replacement Selective Shunt	Follow-up
Kakos GS, et al.[24] (1973)	Elective	10 Years	No	3 Months
Warren WD, et al.[25] (1980)	Elective	22 Years	No	6 Months
Hanna SS, et al.[26] (1981)	Emergency	17 days	Yes	5 Days*
	Emergency	1 Year	Yes	7 Days*
	Elective	6 Months	Yes	6 Months*
	Elective	2 Months	No	13 Months*
Warren WD, et al.[18] (1982)	Elective	6 Months	Yes	1 Year

* Known to be dead.

operation was successful, when done at a time of election, based on predetermined criteria of suitability. Both patients treated in this way had a complete turnabout in their clinical course. Increased hepatocyte mitotic activity was documented, as evidence of the hepatotrophic effect of restoring portal blood flow. On the other hand, in the setting of acute, refractory postoperative encephalopathy and liver failure, shunt conversion was of no avail, even when it could be accomplished with a single ligature on the central portion of the splenic vein. Here too, there was histologic correlation: the extensive atrophy and hepatocyte necrosis characterizing these livers documented the destructive effect on a vulnerable liver of sudden portal privation. Flow restoration was too little, and too late, to reverse this process.

Isolation and arterialization of the hepatic portion of the portal vein was chosen as a technique to avoid multiple dissection areas in an emergency operation to restore portal flow in the presence of a side-to-side portacaval shunt. This singular experience offers no evidence that total hepatic blood flow was indeed augmented by the procedure, as the technique selected left no space beyond the fistula for direct flow measurement (Figure 2). Ideally, flow in the arterialized portal vein should approximate, or be less than, normal portal vein flow;[6,8,21,23] hence—the small 3-mm fistula. Confining all dissection to a single area was not the answer to the futility of attempting to restore portal flow in the milieu of a failing liver, but if, under elective circumstances, arterialization would be as beneficial as selective shunt conversion, then portal flow could be restored after any type of total shunt. End-to-side shunts and central splenorenal shunts cannot be changed into selective shunts, but both could be isolated and the hepatic portion arterialized (Fig. 7).

The published experience with operative portal flow restoration encompasses only seven cases (Table 6).[18,24–26] The general experience parallels what has been reported here: elective reestablishment of flow induces dramatic improvement in the functional status of carefully selected individuals, but the pivotal role of the liver in hemostasis precludes successful outcome for emergency attempts to rescue patients in hepatic failure.[26] Three of the seven flow-restoration operations involved merely occluding the total shunt, raising the question of the need for a replacement selective shunt.[24–26] Recurrent variceal bleeding has not been reported (longest follow-up, 13 months[26]), and patients without a shunt face no theoretical hazard of recurrent encephalopathy consequent to gradual selectivity decay. In the absence of any pertinent data, the excellent clinical results of elective shunt conversions reported here, 18 months and 4 years after the fact, offer at least limited testimony in favor of replacement selective shunting.

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Discussion

Dr. Robert Zeppa (Miami, Florida): Five-therapeutic prospective trials involve the comparison of portosystemic shunting with distal splenorenal shunting—the box score at the moment with respect to portosystemic encephalopathy is 3 to 2, 3 winning in terms of a lowered incidence. What does this mean?

I think it points to an enormous subjective error in the evaluation of portosystemic encephalopathy, which was not corrected by creating a prospective randomized trial. There is only one of the trials which has demonstrated in a meaningful way, by measuring a biochemical function for which there can be no argument; that is the trial that was conducted in Atlanta, where maximum rate of urea synthesis was an end point, one that can be reproduced over and over again.

Therefore, I have exposed my biases clearly as to which is the better shunt under those circumstances.

In this paper Dr. Chandler points out that a meticulous portal-azygos disconnection operation was conducted at the same time they did this. This is important. Sufficient data now are available to point out that one can retard the development of major channel collaterals to a distal splenorenal shunt by doing a careful portalazygos in the area that has been described so well.

The authors have also mentioned in their paper "that post-shunt encephalopathy is no more common than pre-shunt encephalopathy," and I would like to take exception to that, with Dr. Chandler's indulgence. I think that the only way that one can accept that statement is if it is made applicable to a single etiology of portal hypertension and liver disease.

For example, in patients with schistosomiasis there is no pre-shunt encephalopathy. It is totally unknown. It only occurs at the rate of 60% when one does a portosystemic procedure. So the statement may well be true, but it would be true only when applied to those patients who suffer from alcoholic liver disease and not to the others.

A paper from Ryer's group describes taking down seven splenorenal shunts in patients with schistosomiasis, and measuring two important features: (1) the change in serum albumin, which increased in all patients, and (2) liver size, which increased in all patients.

Our own experience with this kind of problem has nothing to do with taking down shunts, but we were faced with a 42-year-old man with chronic, active liver disease, who was on about 50 mg of prednisone daily, and who had bled four times; the hepatologist requested that we stop the bleeding. The patient had a double coronary. I missed it—ligated one. By the twelfth postoperative day he was in stage 4 coma.

Angiography at that point revealed a fairly large channel, well opacified on the venous phase films of the superior mesenteric artery injection, and no opacification of the liver. He was taken to the operating room and this channel was ligated; the man was awake and out of the intensive care unit in three days.

Patients who have chronic active liver disease are notoriously sensitive, and that is why in the paper that preceded this one, describing hepatitis following a coronary artery bypass, it is no surprise to me that that patient went into coma very soon after a portosystemic shunt.

Dr. Chandler, in the entire series that you cited in the manuscript, what was the percentage with respect to alcoholism *vs.* nonalcoholism? Were there any on steroids, and was this a harbinger of the development of portosystemic encephalopathy? And did you attempt to assess liver size before and after the conversion?

Dr. Gardner W. Smith (Baltimore, Maryland): If you are going to get into this kind of trouble, you could make it easier for yourself to start with by doing something different in the way of a shunt. I can think of nothing more difficult than trying to reverse a side-to-side portacaval shunt, and it occurs to me that perhaps Dr. Talman's preference for end-to-side shunts could be defended in this regard.

I might remind you that many years ago Dr. Rousselot strongly urged that the umbilical vein be preserved whenever you perform a portacaval shunt—specifically an end-to-side one—and he made this recommendation on the grounds that, in the event of a shunt thrombosis, a potential collateral pathway would still be available.

Dr. Chandler, if he is going to do end-to-side shunts, would give us another reason to heed Dr. Rousselot's advice. As Dr. Adamson has pointed out, if you have cause to restore liver blood flow at some subsequent time by arterializing the portal vein, it is a lot easier to arterialize the reopened umbilical vein than it is to try to arterialize the hepatic stump of the portal vein as a secondary procedure.

Dr. W. Dean Warren (Atlanta, Georgia): I agree with almost everything the author has said.

One of the worst mistakes made in this field is to do a splenectomy for the hypersplenism of portal hypertension. This is almost never needed from a standpoint of the coagulopathy, and frequently leads