Results of a Modified Distal Spleno-renal Shunt for Portal Hypertension

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Twenty-five patients were treated with a distal spleno-renal shunt modified after that of Warren. The operative mortality was 4/25. One patient had an early thrombosis. All postoperative angiography otherwise showed patent shunts. After a median observation time of 43 months, 10/20 patients included in the followup were dead. The chief cause of death was liver failure. Encephalopathy has been common although generally of minor degree. Hypersplenism, judged by thrombocyte count, was not significantly affected by the operation. Six of 21 patients have had gastrointestinal hemorrhage after the operation but no hemorrhage proved fatal. Postoperatively esophageal varices size was considerably diminished in most cases as judged by contrast x-ray. Ascites has not been a problem in this series. Postoperative angiography showed a marked and rapid reduction of portal blood flow to the liver with progressively more blood deviated through the coronary vein towards the shunt. This reduction in portal flow is a possible explanation of the high frequency of postoperative liver failure. This version of the distal spleno-renal shunt has probably no advantages over the portacaval shunt.

CONSTANT DEBATE has been going on for the last ${f A}$ decade about the suitability of porta-caval shunts in portal hypertension. Although they effectively prevent gastroesophageal hemorrhages it is difficult to prove that they increase survival. They definitely do not seem to be of any use when done prophylactically, that is in patients with no previous bleeding episodes.^{4,10} With regard to therapeutic shunts no final answer has been given but it is remarkable that controlled prospective investigations^{7,11} do not show a statistically significant increased survival. This is probably partly due to the fact that the decreased number of deaths from hemorrhages is balanced by an increase in deaths from hepatic failure. It may be that this increase is due to the reduced liver perfusion after the shunt. This theory was rational for the concept of the distal spleno-renal shunt as a means of From the Department of Surgery and the Department of Diagnostic Radiology, University of Lund, Lund, Sweden

preserving the hepatic portal perfusion while still selectively draining the esophageal varices. This operation was described in the late sixties by Dean Warren¹⁶ and Texeira.¹⁴ The first reports showed a very high operative mortality¹⁷ and we therefore modified Warren's operation to overcome what we thought to be the cause of this mortality. Our first operation was performed in February 1970 and this is a report of our experience with the first 25 shunts.

Material and Method

From February 1970 to February 1973 the distal spleno-renal shunt was considered the operation of choice for gastrointestinal bleeding from portal hypertension and was performed whenever possible. These shunts were made both as acute and elective operations and no hemodynamic or other selections were made. Thus, ascites was not regarded as a contraindication. However, as not all patients with bleeding varices came to operation a certain selection was unavoidable. All patients had esophageal varices noted on barium examinations and/or endoscopy. Acute endoscopy during active bleeding was not performed routinely. All patients were subjected to liver biopsy either preoperatively or during the operation. Angiography of the celiac and the superior mesenteric arteries with studies of the venous phase was carried out before the operation in most cases to confirm the patency of the splenic and portal veins.

Twenty-five patients were included in the series, 14 men and 11 women. The mean age was 52 years and the age

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FIG. 1. The patients grouped according to age. Mean age 52 years.



The operation performed was a modification of Warren's distal spleno-renal shunt (Fig. 3). In the first reports from Warren's group^{16,17} the initial mortality was very high and postoperative ascites very common. We thought that an important factor leading to this result was the ligation of all small portal communicating veins. This could, in some cases with high liver-resistance, lead to almost complete portal outflow block. We therefore ligated the coronary vein close to the esophagogastric junction or ligated only the paraesophageal veins where they leave the coronary vein. No attempt at "gastric devascularization" was done. We thought that this would make the blood run through the small gastric veins to the shunt and thus the varices had been moved to a "safer" place. As the communicating veins between the portal vein and the shunts were small, we considered that the shunt would not deprive the portal circulation of any undue amount of blood and that our modification also was a true "selective" shunt like the Warren shunt.

Results

Hospital mortality. Four of the 25 patients died within one month of the operation while still in hospital. One died due to a technical error in the shunt procedure with hemoperitoneum, and continuous gastrointestinal

FIG. 2. The patients grouped according to liver disease etiology and liver function.



bleeding. Thrombosis of the shunt was also found at autopsy. The others died for various reasons not related to the type of shunt performed.

Shunt thrombosis. Apart from the above-mentioned patient, one other shunt seems to have occluded. The patient with this shunt rebled one month after the operation and angiography showed a thrombosed shunt. A porta-caval shunt was performed and he was withdrawn from this series. All postoperative angiographies otherwise showed a patent shunt. At autopsy 6 more shunts were inspected, all of good size and with no thrombosis.

Re-bleedings. Six of the 21 patients who survived the operation have since had upper gastrointestinal bleeding. One was the patient with an occluded shunt. One patient has had two minor re-bleedings but no esophaeal or gastric varices are left and no other cause of



FIG. 3. Schematic drawing of our version of the distal spleno-renal shunt.

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FIGS. 4a and b. A typical picture from the venous phase of sup. mes. art. angiography. (top) Preoperative, (bottom) one month after the operation, showing the rapid reduction in portal diameter and hepatic perfusion after the shunt.

the bleeding has been detected. One patient bled from an erosive gastritis. In three cases bleeding from esophageal varices could not be excluded. No patient has died from the hemorrhages.

Variceal decompression. Eighteen patients were subjected to a followup examination three months postoperatively. On that occasion the size of the esophageal varices compared to preoperative data as judged from barium examinations was unchanged in 5 cases, reduced in 6, and the varices had completely disappeared in 6 cases. In one patient, esophageal varices were never demonstrated by this method.

Survival. Of the 20 patients from whom we obtained followup data, 10 died within the observation time. Five of these 10 had liver failure as the primary cause of death and in yet another 3 liver failure was a contributing cause of death. Only two deaths were unrelated to liver failure (hepatoma, cerebral hemorrhage).

Encephalopathy. Nine patients are still studied regularly. None of these has had any coma episodes, but cerebral dysfunction is a common feature. Two of these 9 patients showed a moderate clinical encephalopathy prior to operation. No distinct progression of their encephalopathy has been noticed. Three of the other 7 patients have shown a progression with clinical signs of moderate encephalopathy and slowing of the dominant waves in EEG. Four patients have no clinical signs of encephalopathy but in two of these the EEG is distinctly pathological. Only two are without any detectable sign of brain damage as a result of their liver disease.

The psychometric test results in the 12 consecutive tested patients were compared to the age-adjusted test results of 13 patients with consecutive porta-caval shunts operated in the period immediately prior to the patients with spleno-renal shunts. There was a tendency for the patients with spleno-renal shunts to have poorer results in all tests but no differences were statistically significant.

Hemodynamic changes. Portal pressure in 11 patients was measured during the operation, prior to and after completion of the shunt. Median portal pressure before the shunt was 33.5 cm H₂O. The changes in pressure after creation of the shunt range from +5 to -6 cm H₂O. The mean of these changes did not differ significantly from zero (P > 0.1 Wilcoxon's test).

Pre- and postoperative angiography was performed in 16 patients. Hepatopetal flow was evident in all patients prior to the operation. Twelve patients were re-examined within one week after the operation. Reversal of portal vein flow had occurred in two patients. Reversal of the flow was noted later in another 4 patients.

Assuming unchanged liver resistance, an estimate of the portal flow could be obtained by measuring the portal vein diameter on the radiograms (Fig. 4). Preoperatively, the mean portal diameter was 19 mm. The mean decrease within one month after the operation was 5 mm (range -1 to -20 mm). One year after the operation the mean decrease was 9 mm (range -4 to -20 mm). Preoperatively the collateral flow was mainly through the coronary vein to esophageal varices. Postoperatively the flow through the coronary vein was progressively deviated through tortuous veins in the gastric fundus to the splenic vein and from there to the patent shunt. This new route of collateralization increased with diminished portal vein flow.

Hypersplenism. The effect of the operation on the thrombocyte count was insignificant. Mean preoperative values were compared to postoperative values studied three months and later after the operation. The differences varied from -118.000 to +157.000 which did not differ significantly from zero (P > 0.10 Wilcoxon's test). To test if there was still a positive effect in patients with thrombocytopenia which was masked by the random changes in patients with normal preoperative counts, the negative correlation between the degree of increase and the preoperative count was tested with Spearman's R-test. No correlation was, however, found (P > 0.10).

The size of the spleen as measured on the angiographic films showed an obvious decrease. Nine patients underwent angiography at the one-year followup. Two of these had normal-sized spleens preoperatively (length 16-18 cm). Seven spleens were enlarged. All of them showed considerably reduced size at followup. One of the reductions was, however, due to an infarction of the spleen. Five of the patients were only angiographed 3-6 months after the operation. Four of these had enlarged spleens. Reduction in size was noted in two while two were unchanged.

Ascites. One of the patients had moderate ascites postoperatively. However, it disappeared after diuretic therapy within a few months. One patient had severe ascites preoperatively. It disappeared postoperatively but soon returned. In the other patients no significant changes in ascites or therapy against it has been evoked by the operation. Thus postoperative ascites has been no problem in this series.

Discussion

The distal spleno-renal shunt has been subjected to some enthusiastic reports.^{5,8,13,15,17,18} However, no controlled long-term followup study seems to have been made, though one is in progress.¹⁸ These reports are all concerned with Warren's original type of distal splenorenal shunt, with a postulated complete division of the portal system into two venous systems. A comparison between Warren's operation and ours reveals no large differences. It is possible that certain hemodynamic differences between the two operations exist. However, portal pressure changes after completion of the shunt are insignificant both in our method and in that of Warren.¹²

The amount of decompressions of esophageal varices indicates the effectiveness of the shunt in preventing major varix bleeding. If only patients with patent shunts are included, 5/20 (95% confidence limits 9-49%) have had upper gastrointestinal bleedings, and 0/20 (95% confidence limits 0-17%) have died as a result of bleeding following the operation. If no operation had been performed, more frequent rebleeding causing several deaths could have been expected.

We can compare our data with patients in other reports³ who had esophageal bleeding and fulfilled the criteria of selection to porta-caval shunt but were not operated. If we take those patients with an observation time of 43 months and longer, we find that 18/23 had gastrointestinal rebleeding within those 43 months in Cohn and Blaisdell's³ material, as compared with 5/20 in ours. This difference is statistically significant (P < 0.01 Fisher's exact test). Of 23 patients in Cohn and Blaisdell's material 9 died from gastrointestinal hemorrhages as compared with 0/21 in our material. This difference is also statistically significant (P < 0.01 Fisher's exact test).

It is worth mentioning that even portocaval shunts have a rebleeding frequency of about 10%. For instance in Brick's² investigation 8/80 patients rebled (95% confidence limits 4.4–18.8%). In a survey by Bengmark¹ listing 12 different studies, but not including the above mentioned one, the median rebleeding frequency was 12% (range 2.0–21.2%).

The important claims made by Dr. Warren that his shunt prevents acceleration of hepatic degeneration and hepatic encephalopathy could not be supported in our modification. Late deaths showed the same pattern as after porta-caval shunt with a high percentage due to liver failure. The frequency of hepatic encephalopathy is often difficult to evaluate but encephalopathy problems do in fact exist to an important degree. This is in contrast with other reports.¹⁸ As indicated by our investigation the degree of encephalopathy was about the same as in a group with porta-caval shunts, but as the comparability of the groups is difficult to evaluate, these data have to be interpreted with care. However, nothing indicates that the distal spleno-renal shunt is superior to porta-caval shunt in this respect.

The rapid deterioration of liver function corresponds well to the rapidly diminishing flow through the portal vein as seen by angiography. Reversal of flow to hepatofugal flow was rather common and reduction of portal diameters was seen in all cases. A likely explanation of the striking change in some cases within a week after the operation is that a diversion of the splenic vein flow could almost instantly cause a dramatic reduction of the portal flow and even make it reverse to hepato-fugal flow. A rapid further deterioration of portal flow was also noted, due to increasing amount of blood escaping through successively dilated small veins towards the shunt. These results also cast some doubts on the claims of long-time hepatic portal perfusion after the original Warren operation¹⁸ as the effect of splenic vein flow diversion must be similar in both operations. Also, it is hardly possible to break every communication from the major portal compartment to the portal compartment drained by the shunt even if rather extensive vein ligation is made. The small remaining vessels and vessels in adhesions made by the operation may be able to dilate and lead progressively more blood to the shunt as happened in our operation. To evaluate this problem further, we have to wait for a thorough long-term angiographic followup of patients with the Warren operation, a study that hitherto does not seem to exist.

In a very recent report by Nabseth⁹ the shunt used was a modification very similar to ours. The flow pattern after the operation indicates that their late results will be the same as ours, but their followup time is still very short.

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