The LeVeen Shunt in the Elective Treatment of Intractable Ascites in Cirrhosis

A Prospective Study on 140 Patients

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One hundred and forty patients with an intractable ascites complicating a chronic liver disease received a peritoneovenous shunt (PVS) using the LeVeen valve. Operative mortality was ten per cent but was 25% in patients with severe liver failure. Intraoperative drainage of ascites sharply decreased postoperative complications and mortality. One-year actuarial survival rate was 81.4%, respectively 77.7%, 61.3%, and 24.7% in patients with good liver function and moderate or severe liver failure. Variceal hemorrhage occurred in 11 patients and late infection in another 11 patients. Thirty-eight patients (30.5%) had recurrence of ascites. This was mostly due to an obstruction on the venous side of the shunt. An elective portacaval shunt had to be done in 23 patients for recurrence of ascites or variceal bleeding. Among the 57 patients still alive at time of writing, 51 were free of ascites. These results suggest that PVS is an efficient operation. This procedure may be largely indicated in the selected and small group of cirrhotic patients with true intractable ascites and moderate or no liver insufficiency.

THE PERITONEOVENOUS LeVeen shunt has been available for the treatment of intractable ascites for 10 years.[1] Initial reports of its results were rather enthusiastic and prompted surgeons to operate on many cirrhotic patients with ascites. Further experience was followed by the reports of a rising number of complications, including disseminated intravascular coagulation,[2] cardiac failure,[3] infection,[4] variceal hemorrhage,[5,6] and venous thrombosis.[7,8] These complications were responsible for a high operative mortality, shunt failure, and a low incidence of late survival. Peritoneovenous shunt (PVS) cannot be considered the safe, fast, simple surgical act that it was supposed to be. Serious doubts were recently expressed on the usefulness of this operation in patients with chronic liver disease and intractable ascites.[9] Since 1976, 140 consecutive patients with chronic liver disease referred to our surgical unit for an intractable ascites received a LeVeen shunt. The purposes of this study was to analyze the clinical results of PVS in order to determine the advantages and risks of this procedure. Such a prospective study ought to be done before undergoing a controlled study comparing PVS to another approach of the treatment of intractable ascites.

Patients and Methods

From October 1976 through February 1983, 140 consecutive patients with intractable ascites complicating a chronic liver disease were treated by PVS. During the same period of time, ten patients did not receive any surgical treatment because of end-stage liver disease and died shortly thereafter. Eight other patients were treated by a side-to-side portacaval shunt because of the presence of a cardiac disease (1 case) or of previous variceal bleeding (7 cases).

One hundred and twenty patients had alcoholic cirrhosis, 18 had nonalcoholic cirrhosis (4 of which were associated with chronic active hepatitis), one had chronic Budd-Chiari syndrome, and one had systemic mastocytosis. They were referred from 15 units specialized in hepatogastroenterology (mean number of patients referred per year for each unit: 1.6) because of the failure to control ascites by medical means, including at least bed rest, suppression of alcoholic intake, salt restricted diet (<20 mmol/day), water restriction (<1 l/day), spironolactone (at least 400 mg/day), and/or furosemide (at least 80 mg/day). The medical management had been pursued 1 to 15 months (mean: 4 months) before referral. In 27 patients, concentration-reinfusion of ascites

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TABLE 1. *Causes of Operative Mortality in 14 Patients Treated by PVS for Intractable Ascites and Chronic Liver Disease*

Sex	Age	Degree of Liver Failure	Date of Death (Postoperative Day)	Etiology
F	65	Moderate	12	Upper G.I. bleeding, site not found at endoscopy
M	58	No	10	Upper G.I. bleeding, acute gastric ulcer
M	70	Moderate	7	Upper G.I. bleeding, site not found at endoscopy
F	42	Severe	12	Hepatic coma
M	37	Severe	7	Hepatic coma
F	42	Severe	20	Septicemia and peritonitis (*Enterobacter cloacae*)
M	52	Severe	5	Intracerebral hemorrhage
M	40	Moderate	10	Unknown, sudden death 2 days after being discharged at home
M	58	No	11	Intracerebral hemorrhage
F	57	Severe	3	Cardiac failure
M	47	No	40	Upper G.I. bleeding, acute gastric ulcer, peritonitis (*Pneumococcus*)
M	39	Moderate	15	Septicemia, *Escherichia coli*
M	69	Severe	12	Septicemia, *Staphylococcus aureus*
M	45	No	30	Septicemia, *Staphylococcus aureus*

had been performed one or several times. Child Classification was not used since all patients would have been included in groups B or C because of ascites. Therefore, the degree of liver failure was estimated according to the following three criteria:[10] serum bilirubin > 3.5 μmol/100 ml, coagulation factors II and V < 45%, and chronic encephalopathy (mental confusion).

Sixty-six patients had good liver function (no criterion), 50 had moderate liver failure (1 criterion), and 24 had severe liver failure (2 or 3 criteria). At the time of operation, renal failure (BUN > 10 mmol/l and/or serum creatinin > 100 μmol/l) was present in 52 patients, and 105 patients had a natremia below 125 mmol/l. The nutritional status of these patients was poor and has been previously reported.[11] None of the alcoholic patients had clinical or biological evidence of alcoholic hepatitis at the time of surgery. The search for liver carcinoma was systematically done by ultrasonography and serum α-fetoprotein measurement: eight patients had a hepatocellular carcinoma. A sample of ascitic fluid was taken just before operation to check the leucocyte count and to test the absence of microorganisms.

PVS was done using the LeVeen shunt. The venous catheter was inserted in the right jugular vein in all but two cases. The tip of the venous tubing was placed at the junction of the superior vena cava and of the right atrium under x-ray control. The functioning of the shunt was always tested by injecting methylen blue in the peritoneal cavity at the end of operation and checking its appearance in the cervical portion of the venous tubing. From the forty-second patient on, approximately 50% of ascitic volume was systematically discarded during operation. Postoperative treatment included chest physiotherapy, abdominal binding, furosemide (80 mg per day p.o.) for 2 to 6 months, potassium, and antacids. Antibiotics were given in case of postoperative infection or unexplained fever. For the last 54 patients, oxacillin was systematically given p.o. for the first 10 postoperative days.

All but two patients were seen within 6 months of preparation of this manuscript. Two patients were lost to follow-up 1 year and 17 months, respectively, after PVS.

TABLE 2. *Etiologies of the Recurrence of Ascites in 38 Patients Receiving PVS*

Obstruction at the tip of the venous catheter		24
Fibrin sheath around the catheter	7	
Thrombosis of the superior vena cava	17	
Obstruction of the valve		10
Displacement of the venous catheter		1
Unknown		3

Results

Operative Mortality

Fourteen patients died within 2 months after surgery (10%). Data on those patients are detailed in Table 1. Operative mortality was significantly more frequent in patients with severe liver failure (25%) than in those with good liver function (6%, $p < 0.001$) or moderate liver failure (8%, $p < 0.01$). It was significantly lower in patients undergoing ascites drainage at operation (5%) than in the 42 former patients (21%, $p < 0.001$).

Operative Complications

Aside from the complications enumerated in Table 1, two other patients had staphylococcal infection of ascites which prompted removal of the material. Two patients experienced severe pulmonary edema. Most of

the patients had ecchymosis along the subcutaneous tunnel of the venous tubing. None of the patients developed diffuse bleeding. Thorough coagulation studies were performed in 21 patients. These data have been previously published.[12] Ascites evacuation considerably decreased the extent of postoperative coagulation disorders.

Recurrences

Among the 126 patients who survived operation, 38 (30.5%) had recurrence of ascites 15 days to 24 months following surgery. No recurrence occurred after 2 years. The rate of recurrence was the same irrespective of the severity of liver failure. The causes of recurrences are listed in Table 2.

Obstruction at the level of the venous tubing was the most frequent (24 patients, 19% of survivors, 63% of recurrences). The rate of obstruction of the venous tubing remained constant despite minor modifications of the surgical technique and the evacuation of ascites during the procedure. Obstruction was due to a sheath of fibrin around the catheter (Fig. 1), or to a large clot obstructing the vena cava (Fig. 2), as demonstrated by percutaneous opacification of the catheter and venous angiography. In none of the patients of this series was there are any clinical evidence of pulmonary embolism. Clearance of ascites was ultimately obtained in 18 of these patients by placement of the catheter in the left jugular vein (4 cases), a peritoneosaphenous shunt (1 case), or a side-to-side portacaval shunt (13 cases). Four other patients died and two remained ascitic.

The second most frequent cause of recurrences was the obstruction of the valve by fibrin deposits (10 patients, 8% of survivors, 26% of recurrences). Replacement of the clotted valve was followed by the disappearance of ascites in nine cases. In the remaining patient, ascites remained despite replacement of the valve and a patent venous tubing. He was successfully treated by a portacaval shunt.

Other causes of recurrences were rare: in one patient, the end of the tubing was situated below the diaphragm and had to be slightly pulled back. In three, no definite cause was found leading to the successful performance of a second peritoneojugular shunt in two and a portacaval shunt in one.

Upper Gastrointestinal Hemorrhage

Sixteen patients (12.7% of survivors) experienced upper GI bleeding 2 to 59 months after PVS. None of them had bled before PVS. The causes of bleeding were ruptured esophageal varices in 11, duodenal ulcer in two, mucosal ulceration in one, and unknown in two. The prevalence of bleeding was not significantly different

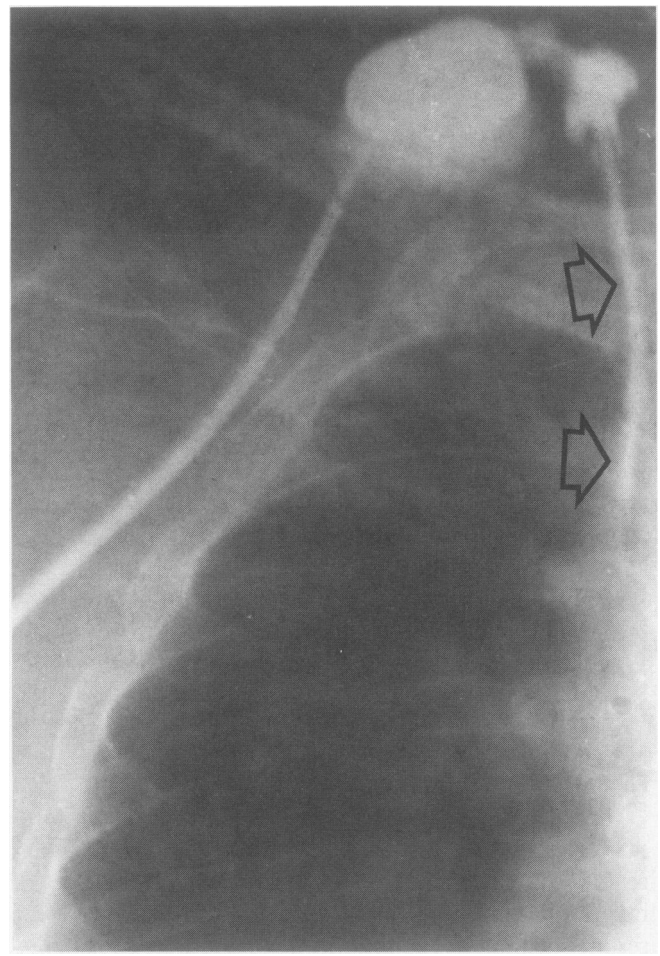


FIG. 1. Percutaneous injection of contrast medium in the venous catheter of a patient with recurrent ascites 6 months after surgery: obstruction of the venous catheter of the LeVeen shunt by a sheath of fibrin (arrows).

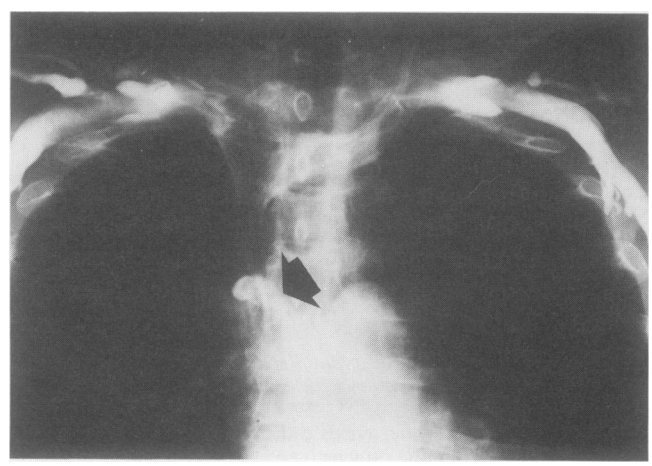


FIG. 2. Superior vena caval angiography of a patient with a recurrent ascites 9 months after surgery: complete obstruction of the superior vena cava. Contrast medium is flowing downhill through the azygos vein (arrow).

TABLE 3. *Late Infection in 11 Patients Treated by LeVeen Shunt for an Intractable Ascites Complicating a Chronic Liver Disease*

Sex	Age	Degree of Liver Failure	Delay Between Operation and Infection (Months)	Site of Infection	Micro-organism	Treatment	Outcome
F	54	No	5	Subcutaneous	*Escherichia coli*	Removal of the shunt	Ascites, liver failure, and death
F	70	No	4	Septicemia	*Staphylococcus aureus*	Removal of the shunt	Recovery, portacaval shunt
F	48	No	5	Ascites	*Staphylococcus aureus*	Removal of the shunt	Septic shock and death
F	74	Moderate	12	Ascites	*Escherichia coli*	*	Septic shock and death
M	71	Moderate	9	Ascites	*Klebsiella pneumoniae*	*	Septic shock and death
F	67	Moderate	8	Septicemia	*Streptococcus*	—	Recovery
F	59	Moderate	12	Septicemia	*Staphylococcus aureus*	—	Recovery from infection, liver failure, and death
F	52	Moderate	1	Ascites	*Streptococcus foecalis*	Removal of the shunt	Recovery
F	56	Moderate	5	Septicemia	*Escherichia coli*	—	Recovery from infection, liver failure, and death
F	44	Severe	3	Ascites	*Escherichia coli*	*	Septic shock and death
F	55	Severe	4	Septicemia	*Escherichia coli*	—	Recovery

* Three patients died of septic shock before removal of the shunt could be undertaken.

in patients with good liver function (9.7%) and moderate liver failure (13%). It was significantly ($p < 0.01$) higher in patients with severe liver failure (22%). Two of the 11 patients with variceal hemorrhage had an emergency portacaval shunt and died. The nine other patients with variceal bleeding were electively treated by portal systemic shunts (6 patients, no mortality), and more recently, by endoscopic sclerotherapy (2 patients) or propranolol (1 patient). The overall mortality in patients with bleeding was 31%. Bleeding accounted for 3.9% of the late deaths.

TABLE 4. *Etiologies of Late Deaths in 69 Patients Following PVS*

		(%)
Progressive liver failure	13	19
Hepatocellular carcinoma	12	17.5
Infection	7	10
Upper G.I. bleeding	6	8.5
Alcoholic hepatitis	7	7.5
Encephalopathy	4	6
Miscellaneous	8	11.5
Cerebral hemorrhage: 2		
Myocardial infarction: 2		
B virus hepatitis: 1		
Intestinal obstruction: 1		
Breast carcinoma: 1		
Tuberculosis: 1		
Unknown	14	20

Infection

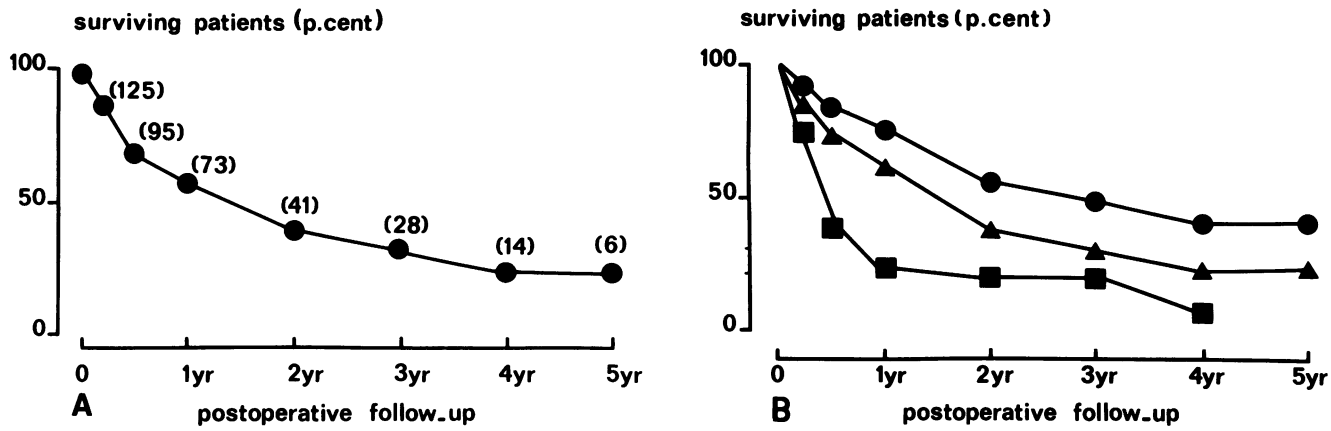
Eleven patients (8.7% of survivors) had infection. It always occurred during the first postoperative year. The prevalence of infection was significantly greater in patients with severe (15.7%) or moderate liver failure (13%) than in patients with good liver function (4.8%) ($p < 0.01$). Data on patients with infection are given in Table 3. The overall mortality of infection was 63% (7 patients out of 11). Infection was responsible for late deaths in 5.6% of the patients (3.2% in patients with good liver function, 8.7% in patients with moderate liver failure, and 5.6% in patients with severe liver failure).

Peritoneal Fibrosis

Three patients developed peritoneal fibrosis. This was discovered at time of surgery for a portacaval shunt in two and made its performance difficult. One patient experienced several bouts of intestinal obstruction due to a peritoneal cocoon. It was not possible to free the bowel at reoperation and the patient died from another attack of intestinal obstruction.

Late Mortality

Sixty-nine patients died 2 to 45 months following operation. The causes of late deaths are listed in Table



FIGS. 3A and B. *A.* Postoperative actuarial survival curve of 140 cirrhotic patients with intractable ascites treated by PVS. *B.* Postoperative actuarial survival curve of cirrhotic patients with good liver function (●), moderate liver failure (▲), and severe liver failure (■), following PVS for the treatment of intractable ascites.

4. Most of them were related to a complication of the liver disease. Eight patients (6.3%) died from a complication related to the procedure, seven from an infection and one from peritoneal fibrosis.

Long-term Survival

Fifty-seven patients were alive at time of writing (40.7% of the whole group). Actuarial curves are indicated in Figure 3. One year survival was 61.4% for the total group of patients. It was 77% in patients with good liver function, 61.3% in patients with moderate liver failure, and 24.7% in those with severe liver failure. In patients with good liver function, 3-year survival was 49%. Among the 57 patients still alive, 46 have been treated by a PVS alone and 11 had an additional portacaval shunt. Fifty-two patients did not have ascites. Five patients had ascites due to failure of the PVS or failure of a portacaval shunt (PCS). All the surviving patients had a normal renal function and nutritional status. This late finding has already been reported elsewhere.[11] Among the 57 late survivors, 22 (39%) had no recurrences nor complications throughout the whole follow-up.

Discussion

Several points of our results deserve emphasis: (1) the operative mortality was low, (2) the rate of late complications was small, and (3) long-term results were hindered by a high rate of recurrences.

The operative mortality was lower than that reported in other large series.[13–16] This seemed mainly related to improvement in the perioperative management of those patients and particularly to (1) the external drainage of ascites at operation and (2) the prophylactic use of oxacillin. Massive transfusion of ascitic fluid is responsible for an increase in cardiopulmonary pressures and cardiac output,[3,17] and for coagulation disorders leading to disseminated intravascular coagulation (DIC).[2,18–21] These changes may have favored bleeding and cardiac failure observed in six of early postoperative deaths. Perioperative removal of ascites decreases the intensity of the postoperative hemodynamic changes (personal data, not shown) and of the DIC.[12] Since we introduced this modification in our technique, the operative mortality dropped dramatically from 21% to five per cent. On the other hand, *Staphylococcus aureus* is the most frequent organism found in postoperative infection in patients treated by a PVS.[4,22] *S. aureus* infection was responsible for early death in two patients of this series. This prompted us to give an antistaphylococcal agent during and after the procedure. Since we prophylactically give oxacillin, no staphylococcal infection has been observed. Despite these measures, patients with severe liver failure remained at risk. All patients with a serum bilirubin level above 6 μmol/100 ml and coagulation factors below 30% died. We no longer consider such patients as candidates for surgical treatment of intractable ascites.

There were few late complications. Variceal hemorrhage occurred in less than ten per cent of the patients. This suggests that PVS does not favor variceal bleeding, as it has been claimed by LeVeen et al.[23] and Markey et al.[5] Variceal hemorrhage was usually controlled by nonoperative means. Prevention of recurrent bleeding was obtained in most of the patients by portal diversion with removal of the LeVeen shunt. Recently we have treated patients with variceal bleeding by endoscopic sclerotherapy rather than by a portal systemic shunt. Variceal bleeding was a rare cause of late deaths in patients treated by PVS.

The rate of late infectious complications (8.7%) was not much different from the incidence of eight per cent spontaneous bacterial peritonitis in cirrhosis with ascites reported by Conn et al.[24] *S. aureus* and *Escherichia coli* were the microorganisms most frequently found. No infection was observed after the first postoperative year.

This may be related to the dramatic improvement in the nutritional and immunological status observed lately in cirrhotic patients receiving a PVS.[11] Mortality was high in infected patients and reached 80% in those with peritonitis. The only survival in the latter occurred after removal of the shunt which has been recently advocated by Wormser and Hubbard.[22] In patients with septicemia, without proven peritonitis, antibiotics alone cured the infection in all of our patients.

The main hinderance of PVS in the present series was the high rate of recurrences by thrombosis at the tip of the venous catheter. The rate of this complication was twice as frequent in our experience than in the LeVeen experience.[25] Removal of ascites at operation, while decreasing the postoperative DIC, did not affect the incidence of venous thrombosis. Venous thrombosis mostly occurred during the first 6 postoperative months. It often occluded the superior vena cava, precluding the performance of another shunt in this area. Therefore, portal systemic shunt was the only alternative in those patients, although peritoneosaphenous shunt has been successfully performed in one other patient. This high rate of thrombosis seems related to the prolonged catheterization of the vena cava with a large silastic catheter. Improvement in the material used, in order to decrease the thrombogenicity of the venous catheter, is mandatory to increase the long-term success rate of PVS.

In patients with a functioning shunt, long-term results were excellent. PVS permitted the patients to resume a normal life. The comparison of actuarial survival of our patients to that of patients not treated by PVS[26–29] suggests that this procedure prolongs life expectancy of cirrhotic patients with intractable ascites.

PVS is a simple operation. Provided caution in the operative technique and the perioperative management, it carries out a low mortality rate. The risk of late complications is small. The long-term survival is similar to what has been reported following portal diversion in similar patients, in our experience.[30] However, PVS does not carry along the risk of chronic encephalopathy. For these reasons, our results suggest that PVS should be indicated early in cirrhotic patients with truly intractable ascites. A portal systemic shunt should be indicated only in patients with previous variceal bleeding and in those with ascitic recurrence related to thrombosis of the superior vena cava.

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