Further Experience with Peritoneo-venous Shunt for Ascites

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A specially devised pressure-sensitive valve forms the basis for a new peritoneo-venous shunt operation which delivers ascitic fluid continuously into the venous system. It is effective in ascites attributed to different causes. The procedure is simple and brings a long lasting relief with recovery in strength and nutrition and improved kidney function. Hepatorenal syndrome in ascites is reversed.

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IN ASCITES due to cirrhosis, venous outflow block increases filtration from hepatic and visceral capillaries. Because liver capillaries are permeable to protein, more protein is contained in ascitic fluid than in ultrafiltrates elsewhere in the body. Until recently, portocaval shunt was the only form of surgery applicable to ascites, but portocaval shunts induce encephalopathy, and death from liver failure is common. A high operative mortality is an added deterrent. As a consequence, internists have been reluctant to refer these patients to surgeons. Yet drug therapy is not a viable alternative. It contracts the extra-cellular fluid space and brings on renal failure. The majority of ascitics die from renal failure even if given sporadic reinfusions of plasma or ascitic fluid.[1] The remarkable similarity in the pattern of events suggests that renal failure has a common pathogenesis in patients with ascites. The patients develop oliguria which is refractory to diuretics. Urine sodium concentrations fall and hyponatremia is frequent. Medical therapy involves a prolonged hospital stay and the adherence to a strict regime involving both diet and drugs, and often culminates in a mortality.

Presented at the conjoint meeting of the Philadelphia Academy of Surgery, the New York Surgical Society, the Boston Surgical Society, and the Baltimore Academy of Surgery, March 10, 1976, at the College of Surgeons, Philadelphia, Pennsylvania.

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Reason tells us that mechanical diseases should respond best to surgical correction while biochemical disturbances should logically yield to drugs.[5] Since ascites is a hydrodynamic defect, why not return the ascitic fluid to the venous system from whence it arose? Intermittent infusion of ascitic fluid brings relief, but the benefits are too transient to be of value in the continuing management of the disease.[3] In addition, repeated paracentesis is not without hazard.

Method

A simple surgical operation was devised which continuously returns ascitic fluid to the venous system.[5] The subsequent experience with this operation prompted this report. The peritoneum is drained through a one way valve into a silicone rubber tube which terminates in the superior vena cava (Fig. 1). As the diaphragm descends during inspiration, the intraperitoneal fluid pressure rises, whereas that in the intrathoracic superior vena cava falls. This results in a pressure differential above 5 cm H_2O. Respiration provides the force which opens the valve and propels the fluid into the superior vena cava.

The venous tube remains patent only when its interior contains ascitic fluid. The specially designed pressure-sensitive valve totally prevents entry of blood into the venous tubing. A blood-ascitic fluid interface is maintained at the tip of the tube and clotting has not been a major problem to date.

In September 1974, the valve casing was altered to

enable it to be placed extraperitoneally rather than intraperitoneally. The mechanism of action remains unchanged. Silicone rubber tension struts on the valve diaphragm act like a spring which maintains the valve in a closed position (Fig. 2). Yet, a force of only 4 cm H_2O is sufficient to open the valve. The fluid dynamic properties of other valves were found to be unsuitable. The present valve is leak proof at both low and high pressures.

Antibiotics are used preoperatively and intraoperatively to prevent infection. Anemia is corrected prior to surgery. The operation is done under local anesthesia. The disc valve is placed extraperitoneally deep to the abdominal musculature and slightly medial to the anterior axilliary line while the valve stem and collecting tube enter the peritoneal cavity. The venous tube extends from the valve through the subcutaneous tissue on the chest wall, into the neck where it enters the internal jugular vein, and the tip of the tube lies in the superior vena cava (Fig. 1). Loss of ascitic fluid is minimized during surgery. Meticulous closure of the abdominal wound will reduce the risk of leakage which can be responsible for infection.

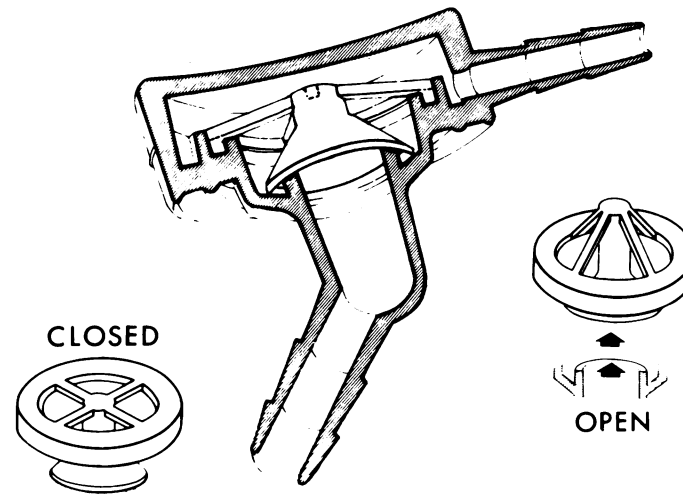

FIG. 2. A cross section of the valve. Notice the silicone rubber struts are attached to a ring which attaches to the valve casing. The valve is in the normally closed position and opens with 3 cm of water pressure.

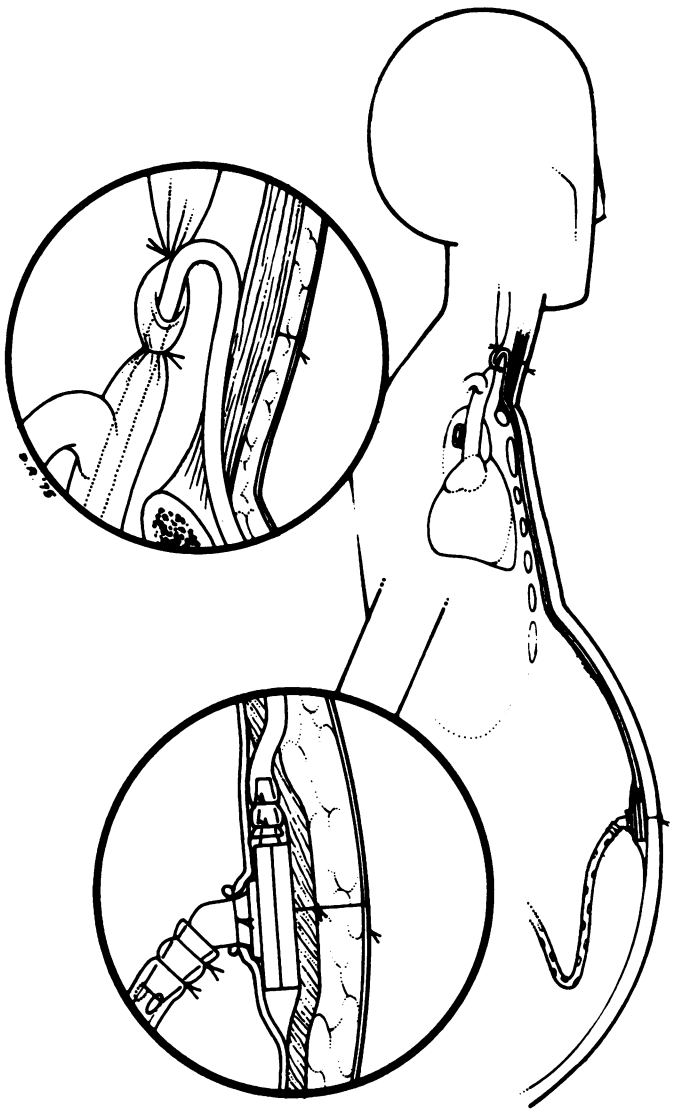

FIG. 1. The valve lies outside of the peritoneum and deep to the abdominal muscles. The venous collecting tube traverses the subcutaneous tissue of the chest wall into the neck where it enters the internal jugular vein. The tip of the tubing is pushed into the superior vena cava (3 in).

An elastic abdominal binder is worn after surgery to raise the intraperitoneal pressure. Respiratory exercise employing inspiration against a resistance augment the flow of ascitic fluid into the venous system. Furosemide is administered postoperatively in adequate dosage to keep the urinary output maximal. The hematocrit is continuously monitored as hemodilution occurs immediately following surgery. Any significant rise in venous pressure closes the valve, interrupts the flow, which protects against over-transfusion.

Material

Sixty-one patients with ascites were treated with a LeVeen continuous peritoneal jugular shunt at the Brooklyn VA Hospital between 1973 and March 1976. The age, cause of ascites, and whether or not the patient had become refractory to diuretics prior to surgery was noted. Weight, abdominal girth, urine flow, plasma, bilirubin, creatinine and blood urea nitrogen were obtained where possible both before and 7–10 days after surgery. Measurements confined exclusively to those patients who were refractory to diuretic therapy prior to shunting will be presented elsewhere.[8] The mean values were compared by the Students' *t* test for paired

TABLE 1. *Causes of Ascites in Patients Treated with a LeVeen Shunt*

	B.V.A.H.	Other	Combined
Cirrhosis			
Alcoholic	57	170	227
Post Necrotic	2	6	8
Malignant	3	15	18
Chylous	—	10	10
Budd Chiari	—	8	8
Renal	—	5	5
Amyloid	—	2	2
Total	62	216	278

data. Complications encountered were listed and when death occurred its underlying cause was evaluated. Two hundred sixteen of 620 patients treated outside and referred to the authors were analyzed as to the underlying cause of the ascites and the complications following surgery.

Results

All patients in the Brooklyn VA Hospital series were male. Their ages varied between 32 and 76 years with a median value of 58. In 55 patients, the underlying cause of ascites was attributed to cirrhosis secondary to alcoholism, whereas it was post-necrotic in two. In three patients the shunt was inserted in the terminal stages of malignant ascites.

In the patients treated outside the Brooklyn VA Hospital, the majority had underlying cirrhosis (Table 1). In 15 patients, the ascites was secondary to metastatic carcinoma, with peritoneal fluid positive for cancer cells in 9. These patients were relieved of the discomfort caused by ascites even though the disease may have been more widely disseminated. One patient died 4½ months after a peritoneo-jugular shunt. At autopsy, implants of carcinoma could be seen on the intima of the superior vena cava at the opening of the patent venous tube. These patients rarely survive longer than 6 months. One patient with a hepatoma survived almost two years. The palliation has often been marked.

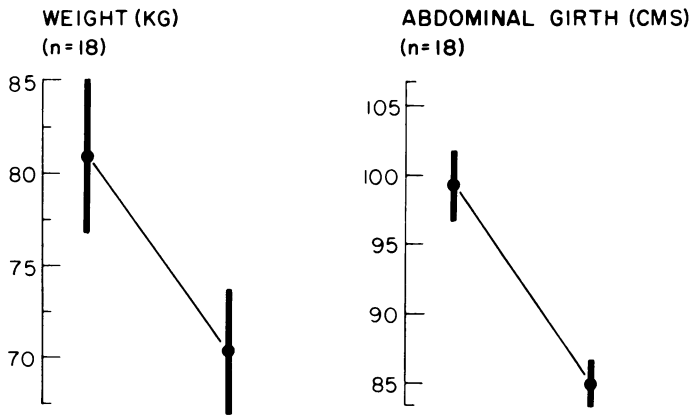

FIG. 3. Changes in weight and abdominal girth preoperatively and 10 days postoperatively.

There were 10 patients with chylous ascites. Two were malnourished children who made rapid dramatic recoveries and, at last information, 6 months following surgery, remained well. The valve was functional in 7 of 8 adult patients. The onset of chylous ascites was spontaneous in 3 adult patients. The shunt was functional in two. The third patient developed peritonitis following a replacement of the shunt, and the valve was removed because of infection. The infection cured the chylous ascites. Five adult patients developed chylous ascites after a distal spleno-renal shunt. Four showed complete relief whereas one who had developed peritonitis prior to the P-V shunt, succumbed. The inadvertent surgical opening of a large lymphatic channel is not catastrophic in normal individuals; but, in cirrhotics, the rapid flow of liver lymph may cause a persistent intraperitoneal leak of chylous fluid. This may explain the occurrence of chylous ascites after distal spleno-renal shunts.

The most striking results were achieved in 8 patients with Budd-Chiari syndrome. All were critically ill prior to surgery and 6 made a spectacular recovery. In one failure, transmigration of intestinal bacteria had occurred and acute peritonitis was present at the time of placement of the shunt. This patient died in septic shock. Another patient was relieved of ascites but developed persistent pleural effusion. The subsequent course of her hydrothorax is unknown to us. Recovery in the remaining cases is most gratifying since it occurs in young individuals with thrombosis of the vena cava at the level of the liver, thus excluding any form of portacaval shunt. Previously, mortality has been extremely high with only occasional survivors.

Five patients developed uremic or renal ascites while on hemodialysis for chronic renal failure. No cause for the ascites could be determined.[2] These patients were successfully managed with a peritoneo-venous shunt, and their ascites has remained well controlled.

Two patients had developed amyloidosis with ascites. The peritoneo-jugular shunt relieved the ascites. No further information was supplied as to whether this favorably influenced the course of the amyloidosis.

There was a significant reduction in weight and abdominal girth with an increase in urine flow after insertion of the shunt (Figs. 3 and 4). All patients showed a fall in hematocrit.

There were three patients who failed to respond. Two patients had a markedly elevated bilirubin and one had severe hypotension due to gastrointestinal bleeding. These three patients had developed acute tubular necrosis

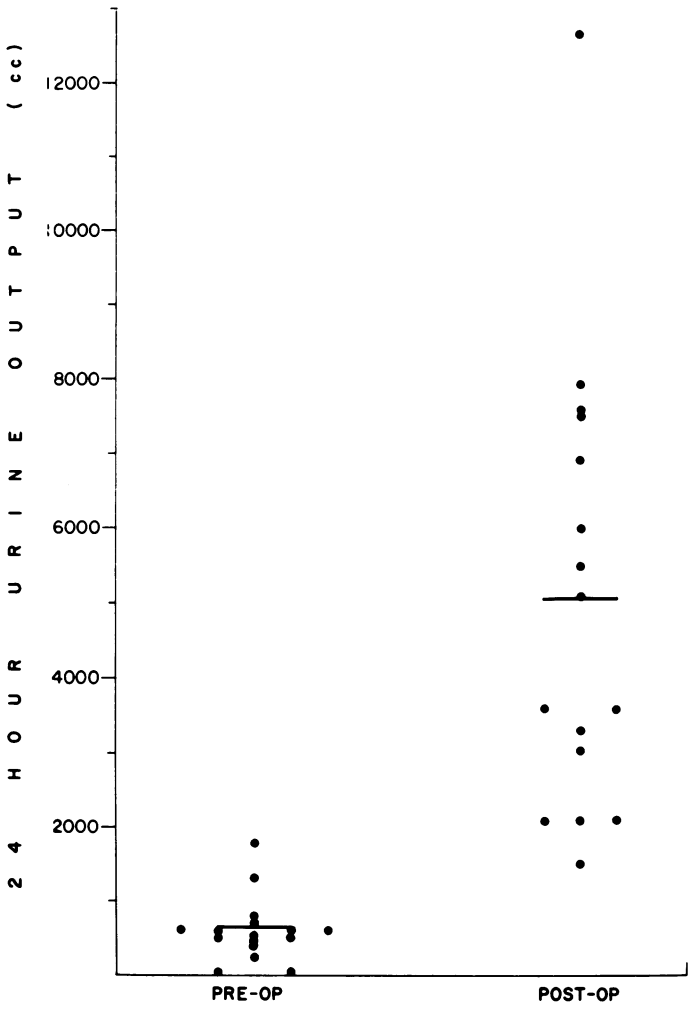

FIG. 4. The urinary output preoperatively is low. Diuresis occurs promptly postoperatively.

as manifested by the high urinary sodium content when urine flow had markedly diminished. Delay in inserting the shunt had led to irreversible renal failure.

The main complications encountered after surgery are listed in Table 2. Five of 6 patients who had gastrointestinal bleeding in the month prior to surgery, had recurrent massive bleeding indicating that surgery for variceal bleeding should precede a peritoneo-venous shunt for ascites. Diffuse subcutaneous bleeding occurred postoperatively in 3 patients in the Brooklyn VA Hospital and in 7 of the referred patients. In 4 patients bleeding occurred at the operative incision. The severity varied from very mild to diffuse bleeding with severe purpura. Five were successfully treated by epsilon amino caproic acid (EACA), small amounts of heparin, fibrinogen and platelets. In the milder cases, only heparin and EACA were administered for one week. If therapy was discontinued before a week, there was usually recurrence of bleeding. Five patients in the Brooklyn VA Hospital series underwent surgery during the early phase of acute alcoholic hepatitis. The serum bilirubin exceeded 10%. This group showed dramatic improvement as regards to their urinary output and correction of ascites. They, nevertheless, succumbed on the third or fourth day following surgery due to acute fulminating hepatic failure.

In the referred series, two patients had frank peritonitis with turbid ascitic fluid. They succumbed to sepsis after the shunt, one developing immediate septic shock. Excluding these cases, wound sepsis occurred in 9 cases and resulting in 7 deaths. Ascitic fluid leakage occurred in 8 patients in the Brooklyn VA Hospital series. Leakage was corrected by inserting interrupted sutures in the wound under local anesthesia.

Changes in weight, abdominal girth, urine flow and B.U.N.

Figures 3 and 4 show the significant improvement in mean weight abdominal girth ($P < 0.001$) and urine flow ($P < 0.001$) that occurred in the postoperative period. The preoperative mean B.U.N. fell significantly ($P < 0.05$) (39 vs 23 mg/dl) when repeated 10 days after surgery. There were 9 patients with hepatorenal syndrome. All showed initial improvement and 5 are alive with markedly reduced abdominal girth 3–26 months after shunting. The course of a patient with hepatorenal syndrome is charted in Fig. 5. The patient lapsed into coma as the B.U.N. rose. The patient's urinary output fell during medical therapy and no longer responded to diuretics but during the first postoperative day, the urinary output rose to 7000 cc. The hematocrit fell with the hemodilution which occurred immediately postoperatively and the abdominal girth diminished. The preoperative and postoperative photographs of this seriously ill patient[5] are shown in Fig. 6 to document his

TABLE 2. *Complications Encountered in 62 Patients of the B.V.A.H. Series*

Subcutaneous bleeding	
Moderate or severe	4
Mild	6
Gastrointestinal bleeding	
Recurrent*	5
Spontaneous	1
Wound infection	3
Ascitic fluid leakage	10
Septicemia	2
Early death in patients with high preoperative bilirubin	5

* Insertion of a LeVeen shunt is not recommended in patients with bleeding esophageal varices unless the portal system is first decompressed by porto-systemic shunting.

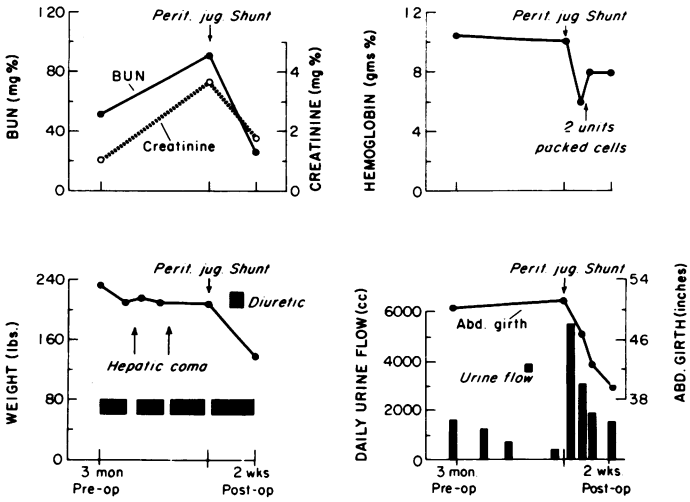

FIG. 5. The course of a patient who develops hepatorenal syndrome. The BUN rises to 100 and the urinary output practically ceases in spite of diuretic drugs. Hepatic coma develops as the BUN rises requiring the temporary withholding of diuretic drugs. No weight is lost until a P-V shunt is done. Notice the prompt fall in hematocrit. The urinary output rises as the weight falls. The patient has remained well with a flat abdomen and a normal BUN for 2 years.

appearance two years after surgery. His shunt is functioning and his health is good.

Discussion

Patients with ascites show progressive renal deterioration over a short or prolonged interval on diuretic therapy. The blood urea nitrogen rises and eventually the hepatorenal syndrome with severe oliguria develops. The acute tubular necrosis in nonshunted patients can be produced by diuretics if the plasma volume is reduced sufficiently to provoke hypovolemic shock. Tubular necrosis can be distinguished from the hepatorenal syndrome by the urinary sodium concentration which is high with tubular necrosis since the tubules do not absorb sodium. In the hepato-renal syndrome, the absorptive capacity of the renal tubules for sodium is so powerful that it resists large doses of diuretics.

Insertion of the peritoneal jugular shunt significantly reduces weight, abdominal girth and increases urine flow in ascites secondary to liver cirrhosis. Refractory ascites[8] as well as the hepato-renal syndrome[4] yields to this therapy. The abnormal renal physiology in patients with ascites is the subject of a special in depth study.[4] It shows the primary defect to be a greatly diminished renal plasma flow and a reduced sodium clearance. The extremely low urinary sodium concentration is only partly the result of changes in renal hemodynamics. Serum aldosterone was measured in 5 patients, 4 of which were markedly elevated. The high serum aldosterone in patients with ascites without shunts cannot be the result of poor liver metabolism as previously taught, since a return to normal concentrations occurs within 5 days after surgery.[8] Salt restriction and diuretics accentuate existing hyperaldosteronism, and fail to significantly improve urinary sodium excretion.[4] On the other hand, diuretics are indicated postoperatively as this significantly improves urine flow[8] and sodium excretion.

Hyperammonemia and encephalopathy are frequently encountered during the medical therapy of ascites due to cirrhosis. This improves following a peritoneo-venous shunt probably because of improved renal function. Ammonia intoxication is explained by the fact that 20% of total body urea diffuses into the colon each day[7] and is immediately converted to ammonia.[9] The higher the BUN, the more ammonia is formed in the colon. Some ammonia bypasses the liver, and the cirrhotic liver is less able to convert ammonia back to urea. The seemingly large amount of urea which diffuses into the colon is accounted for by the extremely high water turnover in the colon.[6] Patients with elevated BUN concentrations may turn out to be concealed cardiacs. This factor should be borne in mind as these patients respond less favorably. Cirrhosis induces functional cardiac defects which can lead to high output failure. Often, the functional extracellular fluid space has been so depleted that the cardiac problems become apparent only after the P-V shunt has expanded the extracellular fluid. The ascites fails to respond with a patent shunt when cardiac function is impaired and the venous pressure persistently elevated. The elevated venous pressure prevents opening of the valve and reduces the differential pressure between the peritoneum and the thorax. Such patients require treatment with diuretics, digoxin, salt restriction and bed rest. They must remain supine for longer periods during the day and repeatedly inspire through a water sealed bottle at a resistance of 7 or 8 cm H_2O. This requires added effort on their part, if the ascites is to be controlled. If the patient is a known cardiac, it is best to remove the greater part of ascitic fluid at the time of surgery rather than to depend on urinary excretion to remove it.

The portal vein pressure varies directly with the blood pressure. The expansion of the blood volume and the increased cardiac output which occurs after peritoneo-venous shunts does increase the portal vein pressure and, with it, the incidence of bleeding from eosphageal varices. The administration of diuretics will minimize the sudden circulatory overloading. The high incidence of postoperative bleeding in patients with previous bleeding would suggest that only after decompression of the portal system should a P-J shunt be inserted for ascites.

The presence of a high bilirubin secondary to acute alcoholic hepatitis constitutes a relative contraindication

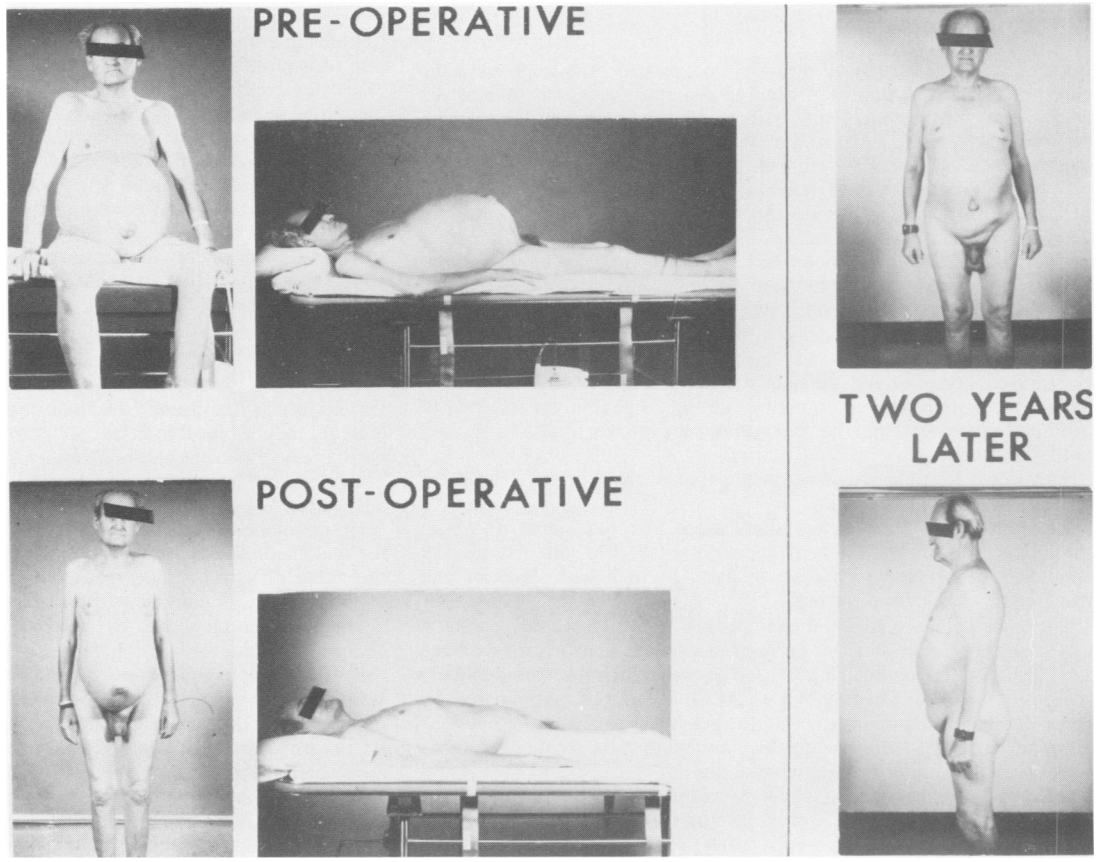

FIG. 6. The patient whose course is charted in Figure 6. His preoperative and postoperative photographs are contrasted to his appearance two years later. His health is good and his abdomen is flat. He has put on muscle mass and fat tissue.

to shunting. Surgery is not recommended for those who have bilirubin above 8–10 mg/100 ml.

Fever is a frequent postoperative occurrence and may alarm the physician. It is of no significance on its own. There is no leukocytosis. Blood cultures have been negative and leukocytosis is absent. The fever responds well to small doses of glucocorticoids. Every patient must be placed on prophylactic antibiotics with a penicillinase resistant penicillin. In addition, the wound is washed with 10% gentamicin solution. Most patients who become infected, had leakage of ascitic fluid which prevented sealing of the wound and established a portal of entry. Meticulous closure of the abdominal wound is essential. Closure of the skin with a continuous suture of polyglycolic acid may be desirable. Any leakage of fluid should be immediately treated by application of an iodophor germicide and insertion of additional skin sutures. It may be necessary to change the position of the valve.

The insertion of the shunt successfully overcomes refractory ascites. In the Brooklyn V.A. Hospital series, patients who had a bilirubin below 5 mg/100 ml, and had had no previous esophageal variceal bleeding were analyzed for survival: 70% lived more than 6 months and 63% more than 12 months following surgery.[8] This analysis included only those who had become refractory to diuretics and also those with the hepatorenal syndrome where the prognosis is uniformly fatal.

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