Peritoneo-Venous Shunting for Ascites

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A new minor surgical procedure for ascites has been devised wherein a specially designated one way pressure activated valve is implanted to create a permanent peritoneo-venous shunt. The normally closed valves opens only when the peritoneal pressure rises 3–5 cm higher than the intrathoracic venous pressure thus preventing backflow of blood and closing the valve should the venous pressure rise from the over-infusion of ascitic fluid. The procedure has been performed on 45 patients but nine were terminal at the time of surgery. Prolonged relief of ascites occurred in 28 of 37 cases.

ASCITES is a disease which is caused by obstruction to the hepatic venous outflow tract with resultant increased filtration of fluid from the vascular compartment. This is most certainly a mechanical problem; and logic tells us that diseases which have a mechanical basis are best treated surgically while diseases which are biochemical in nature respond more favorably to medicine. Since surgeons are preoccupied with the operation of portacaval shunt, internists are inclined to treat this disease medically and withhold surgery as a last resort in end stage disease.

The salient pathophysiology of ascites due to cirrhosis is the increased formation of fluid in the peritoneal cavity. This fluid formation obeys the principles set down by Starling. Equilibrium is reached with a tense intra-abdominal hydrostatic fluid pressure. At equilibrium, the colloid osmotic pressure of the serum minus the colloid osmotic pressure of the ascitic fluid is equal to the portal capillary pressure minus the intra-abdominal hydrostatic pressure. Most of the fluid is filtered from the surface of the liver, but the visceral peritoneum makes a significant contribution. Liver capillaries are permeable to protein otherwise plasmsa proteins synthesized by the liver could not gain access to the circulation. This circumstance accounts for the high concentration of protein found in ascitic fluid, and impedes the reabsorption of fluid from the peritoneal cavity. Sherlock[1] has found that serum osmotic pressure is considerably reduced in cirrhotics with ascites.

Internists have concentrated their attack on salt metabolism, although there is some doubt whether renal handling of sodium in cirrhosis is abnormal. When the interstitial fluid requirement is satisfied, the injection of sodium chloride into patients with ascites is followed by naturesis.[7]

Salt intake does play an important role in the formation of ascites just as it does in the formation of peripheral edema in the hypoproteinemia of malnutrition. Salt loading not only favors retention of extracellular fluid in normals but accentuates the collection of extracellular fluid in areas of predilection secondary to venous obstruction such as the lung in left heart failure and the extremities in right heart failure, or the abdomen in ascites. Sodium excretion in the urine is sharply diminished during the formation of edema or ascites. Medical therapy therefore must decrease the effective, extracellular fluid volume before it influences the removal of extracellular fluid in areas of predilection. Iatrogenic reduction in the extracellular fluid volume may be the stimulus for the increased absorption of sodium by the renal tubules.

The development of potent diuretics has given the internists an opportunity to intensify the treatment of cirrhotics. As a consequence, most medically treated patients had become hyponatremic, oliguric, hypovolemic and axotemic before they were referred to surgery. Some patients were so depleted that they had

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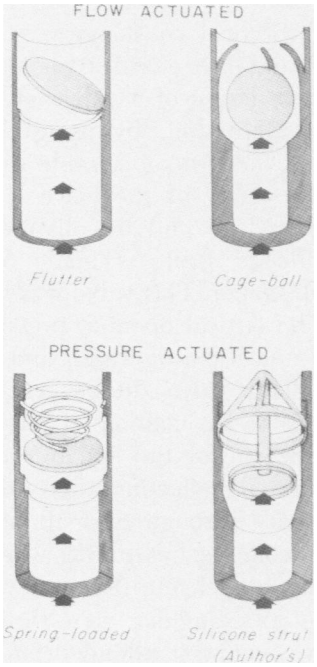

FIG. 1. Schematic depicting the operating principles and types of one way valves with respect to their mechanisms of closure.

become hypotensive. In a recent study,[2] 34 of 48 cirrhotics with ascites had significant renal failure at death with a strong iatrogenic etiology.

Not only is diuretic therapy and salt restriction unphysiologic, it makes health an unattainable goal for cirrhotics with ascites.

Why not restore the filtered plasma which has become ascitic fluid back to the circulatory system from whence it arose? There is a good deal of accumulated literature which shows that infused ascitic fluid causes diuresis and naturesis,[9] restores patients vigor,[3] and is generally favorable to renal function.[5]

It is little wonder, therefore, that surgeons have attempted the continuous reinfused of ascites to the venous system by a peritoneo-venous shunt.[4,6,8] Still, the published results are few and inconclusive. Only one long term followup is available. Surgeons have invested too little time and effort into defining and solving the problems of peritoneo-venous shunt which might transform the procedure from an idea of promise to a surgical practicality. "Off the shelf" valves, available for the treatment of hydrocephalus have been selected without basic consideration for the hydrodynamic requirements. The technique of surgery and the hydrodynamic requirements involved in the successful transport of this

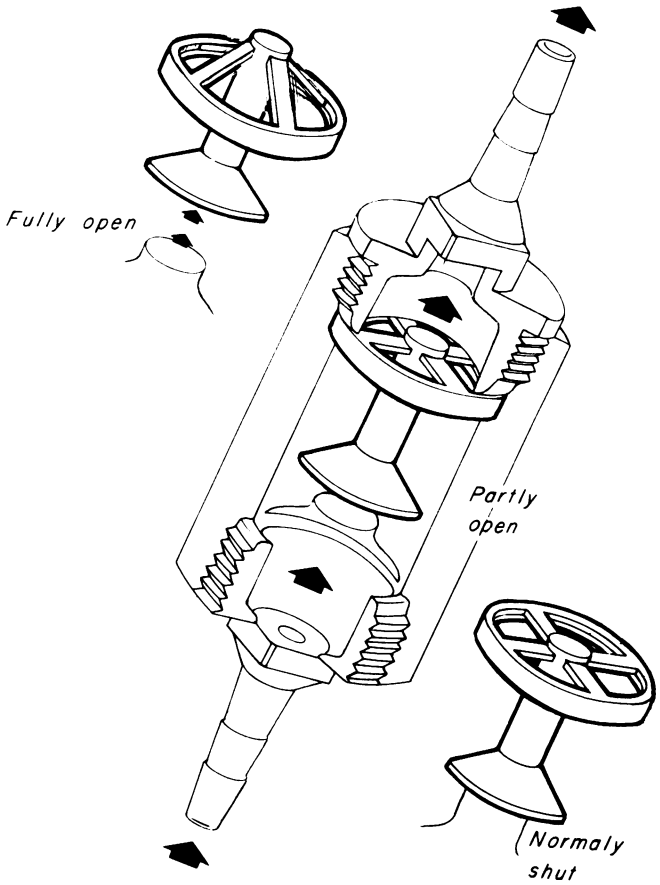

FIG. 2. Construction of the valve used by the authors. The valve is held in the normally closed position by tension on the silicone rubber struts.

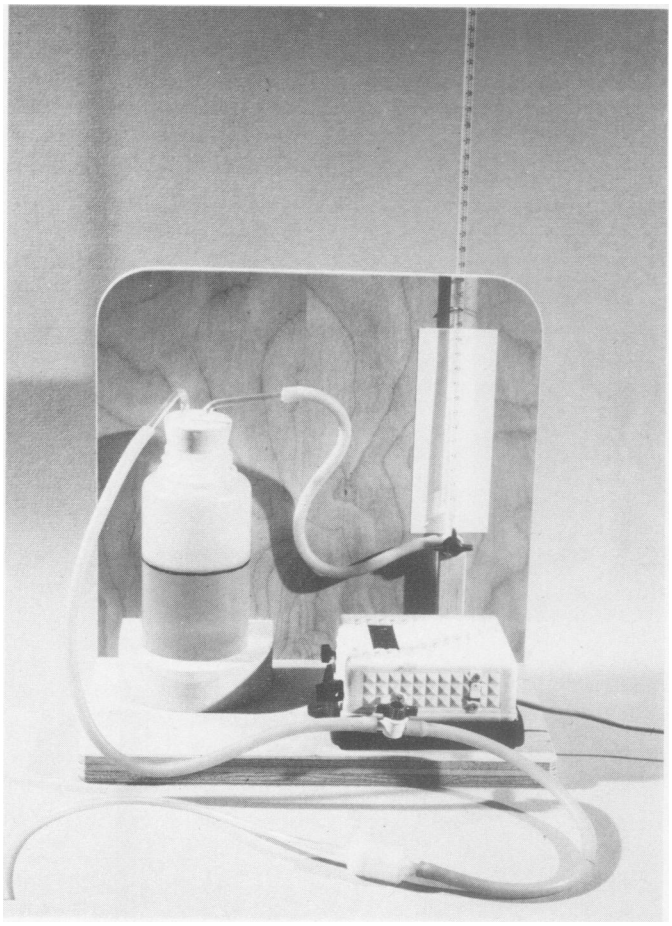

FIG. 3. Valve function is examined manometrically. Opening pressure and competence at low pressures are important characteristics.

fluid both require study. These aspects of the problem prompted our investigations in animals and humans.

Experimental

The exact details of valve requirements were worked out by *in vitro* hydrodynamic studies and dog experiments which will be merely summarized here and published elsewhere.

Valves may be considered to be of two types: flow actuated and pressure actuated (Fig. 1). Flow actuated valves require a slight reversal of flow to seat a ball or a leaflet over a restricting orifice. Valve closure requires a minimum rate of flow to mechanically move the leaflet or ball. At very low flow rates, such flow activated valves become incompetent. Reverse flow even occurs in the saphenous veins at low pressure differentials. This slight regurgitation of blood at low pressures may cause the displacement of only 2 or 3 cm^3 of blood, hardly sufficient to be called serious back bleeding; but, the blood which enters the tube is now contacting a thrombogenic surface and clotting promptly ensues, thus occluding the lumen.

Varying types of valves were tested in dogs rendered ascitic by caval ligation. Flow sensitive valves failed when most of the ascitic fluid had been drained and the venous and peritoneal pressure differences approached one another.

Since the inside surface of the silicone rubber tubing must always look at ascitic fluid rather than blood, reversed flow is not tolerable. This can be prevented with a pressure sensitive valve. Since no satisfactory valve was available, a new design was undertaken. For theoretical considerations, the pressure activated valve should have a critical opening pressure of 2-5 cm of water. The design of a valve with such sensitivity seemed impossible; but, by using the tension created by small silicone struts a suitable valve was developed (Fig. 2).* The body of the valve is made of non reactive polypropylene and the diaphragm valve with its attached silicone struts is made of a low durometer silicone elastomer. This valve was bench tested *in vitro* (Fig. 3). The critical opening pressure was consistantly between 3 and 5 cm of water, and leakage did not occur.

This valve design was tested in 6 dogs rendered ascitic by ligation of the vena cava cephalad to the entrance of the hepatic veins. The valve and the perforated collecting tube were placed in the peritoneal cavity through a small abdominal incision. The solid silicone venous tubing was drawn through a subcutaneous tunnel into the neck where it was inserted into the internal jugular vein through a venotomy. The tube was cut so it extended into the superior vena cava. The appearance of a preoperative and two month postoperative dog is shown in Fig. 4 together with a schematic diagram of this shunt. The valve remained patent in experimental dogs for over three months at which time the animals were destroyed.

The critical opening pressure of 3-5 cm of water was chosen to prevent blood from regurgitating into the silicone rubber tubing and at the same time to take

* Valve available on experimental basis from Dr. LeVeen.

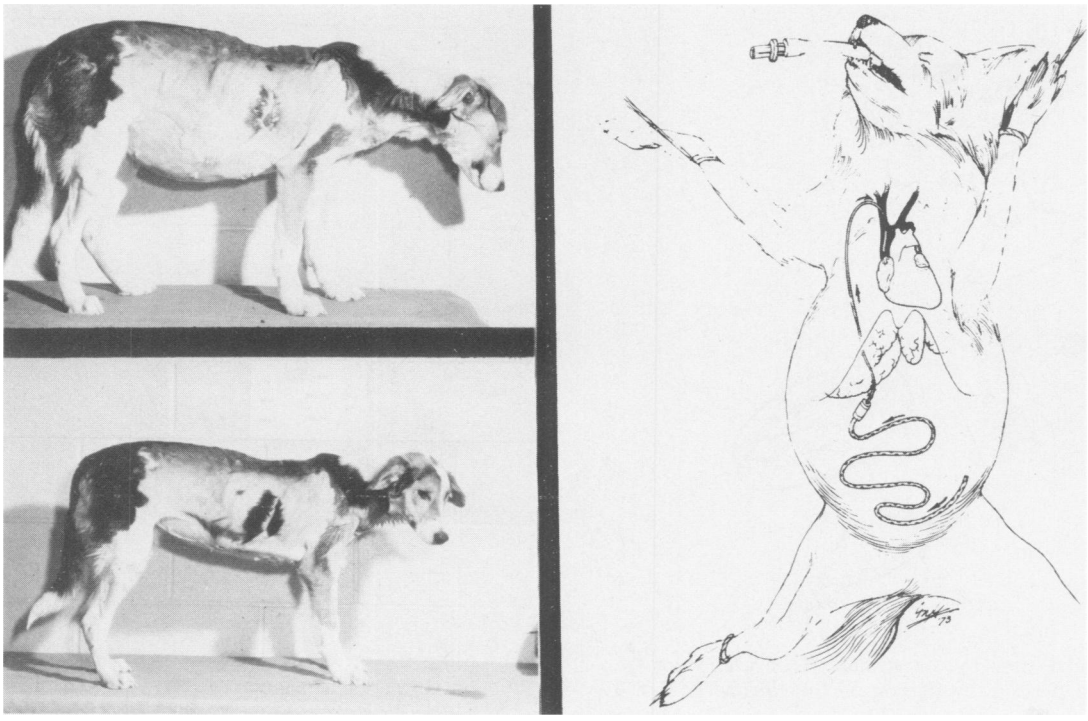

FIG. 4. An ascitic dog is shown prior to peritoneo venous shunt and two months after surgery. The diagram illustrates the placement of the valve system. Alleviation of ascites and gain in muscle mass attests the success of the surgery.

advantage of the pumping action of respiration. As the diaphragm descends during respiration, the intra-thoracic pressure falls and the intra-abdominal pressure rises. This produces a pressure differential sufficient to empty the ascitic fluid into the vena cava in ascitic animals and humans. In the reclining position the differential pressure changes between the peritoneum and the superior vena cava are shown in Fig. 5 during normal respiration, forced deep respirations, inspiring against resistance and expiring against resistance. Notice that at the peak of inspiration the peritoneal pressure exceeds 5 cm of water allowing ascitic fluid to open the valve. Forced deep respirations exaggerate the normal excursions and bring the differential pressure between the peritoneum and the vena cava far above 5 cm with each respiration. Inspiration against resistance of 5 cm of water is very effective in widening the differential between the caval and peritoneal pressure and can be used clinically to hasten the emptying of ascitic fluid into the venous system. On the other hand, expiration against resistance or intermittent positive pressure breathing leads to closure of the valve. Insertion of a pressure sensitive valve with a critical opening pressure introduces a new homeostatic mechanism which prevents overinfusion. As more fluid is pumped into the venous system, the venous pressure rises. Overinfusion is not possible since a significant rise in venous pressure closes the valve and protects against heart failure and pulmonary edema. Therefore, it is unnecessary to monitor central venous pressure after introduction of the valve.

Clinical Experience

Techniques—Management

Most procedures were done under local anesthesia. A transverse incision 2–3 inches in length is made in the abdomen medial to the anterior axillar line lateral to the rectus sheath and below the liver edge. The abdominal muscles are split. If the ascites is tense, a small amount of fluid may be allowed to escape; but the loss is kept to a minimum by traction on sutures placed prior to opening peritoneum. These sutures are used to pull the peritoneal edges together. After the peritoneum is opened, the valve and its perforated tubing are quickly inserted into the abdominal cavity, and the peritoneum closed around the stem of the valve or the tubing. Closure of the muscles and anterior rectus sheath prevents leakage of peritoneal fluid from the wound. Gentamicin solution (40 mg in 100 cc saline) is instilled in the wound during closure. A long tunneling instrument is pushed subcutaneously from the abdominal wound toward the neck. This procedure may not require local anesthesia in a well-sedated patient. A transverse incision 1½ fingers above the clavicle exposes either the external or internal jugular veins. Local anesthesia and incision of fascia over the clavicle is almost always necessary to complete the subcutaneous tunnel without pain. A long heavy silk tie is drawn through the tunnel to the abdominal wound.

The venous tubing is doubled at its end and tied to form a loop. The silk suture from the neck is tied into this loop. The tubing is then pulled into the neck wound. A venotomy site is chosen either in the internal or external jugular. The distance from the venotomy site to the 2nd interspace is measured with the venous tubing and the excess is trimmed. A longitudinal venotomy is made between two 5–0 traction sutures. The vein is tied above with a ligature and a ligature below is tied after insertion of the tubing. If any obstruction to the passage of the full length of tube is encountered in the external jugular, the tube should be appropriately shortened or

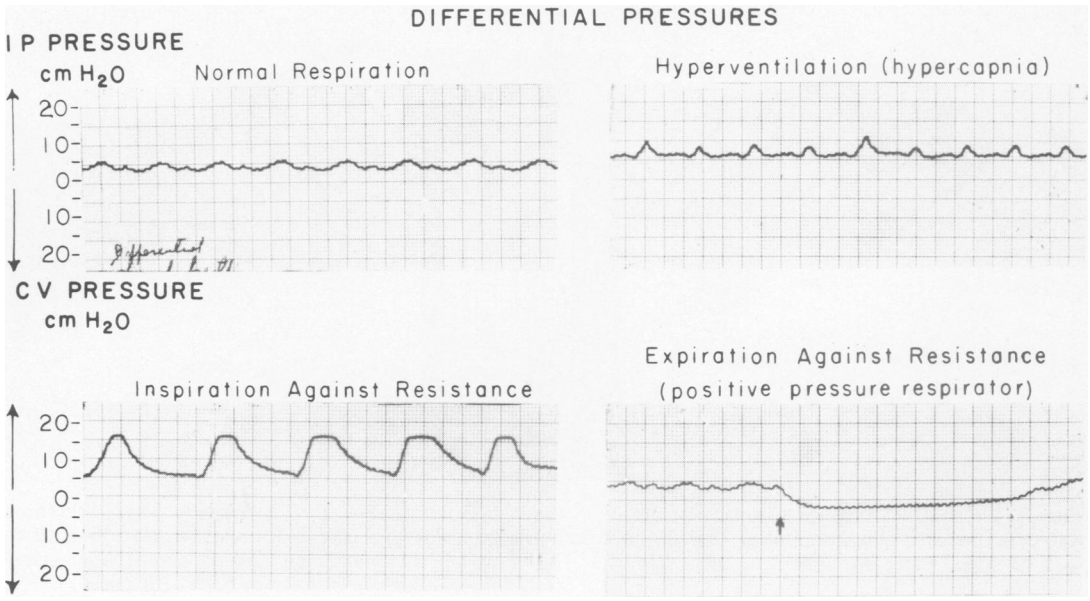

FIG. 5. Recordings demonstrating pressure changes between peritoneal cavity and superior vena cava during normal respiration, hyperventilation, inspiration and expiration against resistance. Note the large pressure gradient which develops with inspiratory resistance.

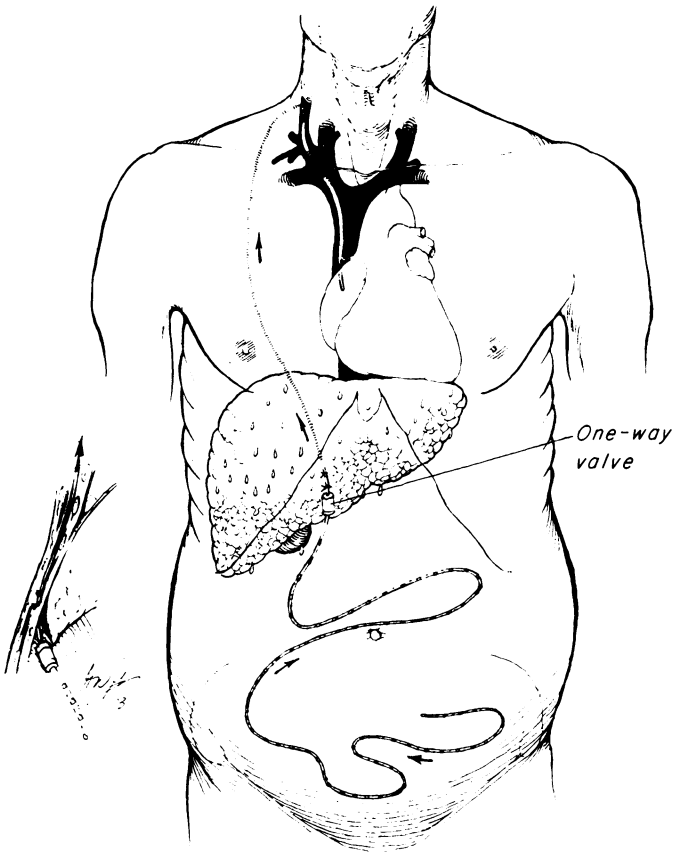

FIG. 6. The phantom view illustrates the placement of the valve and collecting tube in the peritoneal cavity and the venous tube in the superior vena cava in the completed operation.

placed in the internal jugular. The completed operation is shown in Fig. 6.

If the abdomen in tense, external compression is avoided for the first 12–24 hours. Thereafter, an external

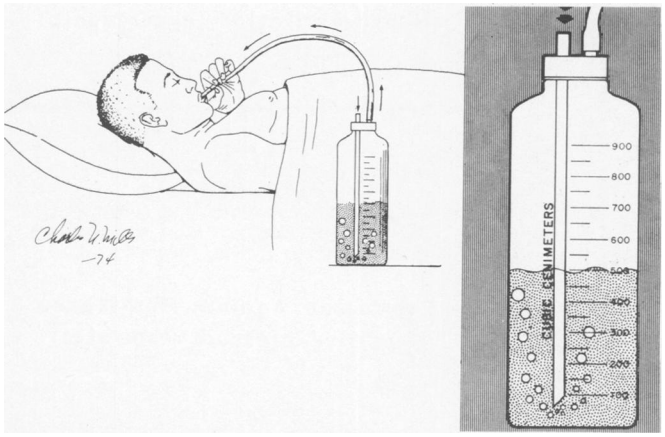

FIG. 7. The patient inspires against resistance of about 5 cm of water to increase the differential pressure between the superior vena cava and the peritoneum.

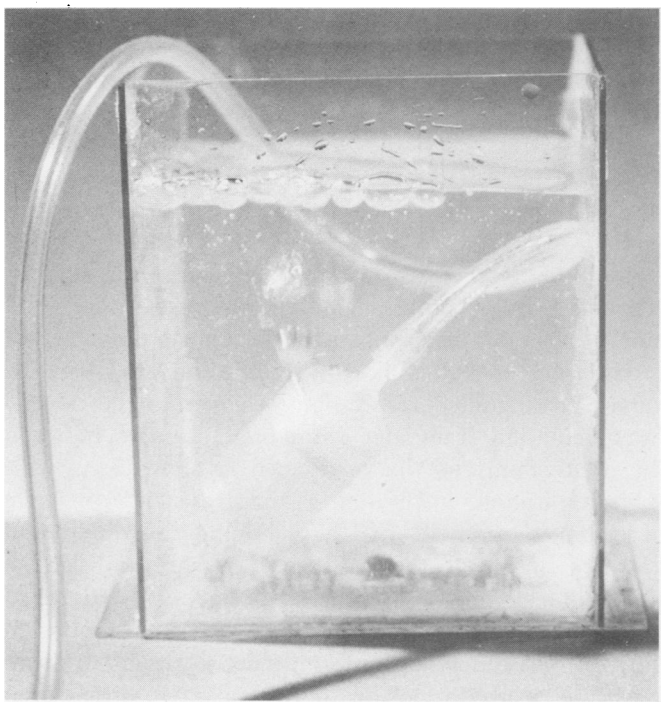

FIG. 8. The leak along the threads developed after sterilization and was not detected until valve failure had occurred.

elastic binder is affixed. After 24–48 hours, the patient is started on respiratory exercises which consist of inspiring through a tube against resistance of 5 cm of water (Fig. 7). This breathing exercise is done in the reclining position. It is continued postoperatively for 15 minutes 4 times a day, to take advantage of the pumping action created by the negative intrathoracic pressure. Patients with thinned and stretched abdominal walls will not mobilize all of the ascitic fluid without respiratory exercises. They will merely overflow from a large residual 3rd space. Eliminating all of the ascites takes the

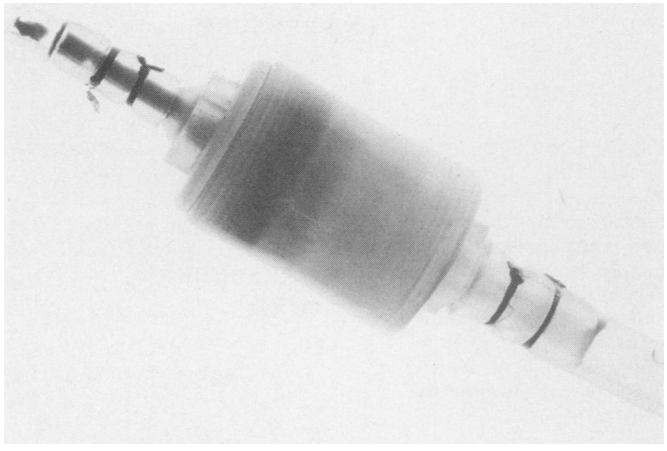

FIG. 9. The leakage along the threads allowed blood to displace ascitic fluid in clot forms down to the valve but the valve is competent and blood is kept from extending below the valve.

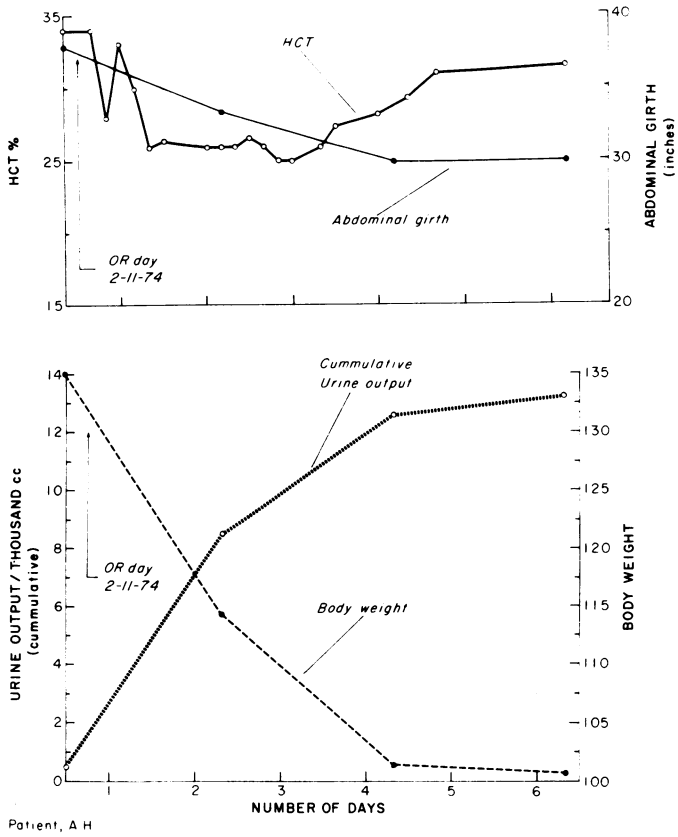

FIG. 10. The hematocrit drops precipitously as the girth circumference decreases. It remains low until the third day when the body weight stabilizes and urinary output tapers off after a weight loss of 30 pounds. The failure of the hematocrit to reach normal indicates a deficiency in the red cell mass. The appearance of the patient is shown in Fig. 16.

cooperation of the patient in performing the respiratory exercises three or four times daily for 10–15 minutes.

Results

The patient series consists of 19 cases personally operated upon and studied and 26 patients operated upon by other surgeons who generously supplied their patient data. Some of these patients were jaundiced and were at the end stage of liver disease and expired within the first week after valve insertion, although one patient survived a month, this accounted for five deaths. Jaundiced patients in liver failure cannot be helped sufficiently by valve insertion. Six patients were anuric or oliguric when transferred to surgery. Two of the six were hypotensive, four of these six patients failed to respond with an increased urinary output and died in renal failure. One of these patients had cardiomyopathy and the venous pressure rose without augumenting the cardiac output or the urinary excretion. Two patients responded with increased cardiac output increased urinary output and survived to lose their ascites. One of these desperately ill patients has lost his ascites, improved his renal function and is now enjoying restored health. This experience has led us to reject patients for surgery if volume expansion with plasma or blood combined with furosamide does not bring about an increase in urinary output. This has left 13 patients in our series and twenty one patients from the outside series for extended observation. Two patients succumbed from bleeding varices after successful treatment for ascites, both patients are considered successes with respect to the ascites treatment.

Failures

Two patients, one in our series and one in the outside group, developed infections and required removal of their shunts 2½ weeks postoperatively. Both patients had recovered from their ascites in the interim and in spite of the removal of the tube were physically improved and more easily managed. In one patient, the ascites did not reappear. In our patient, prophylactic antibiotics had

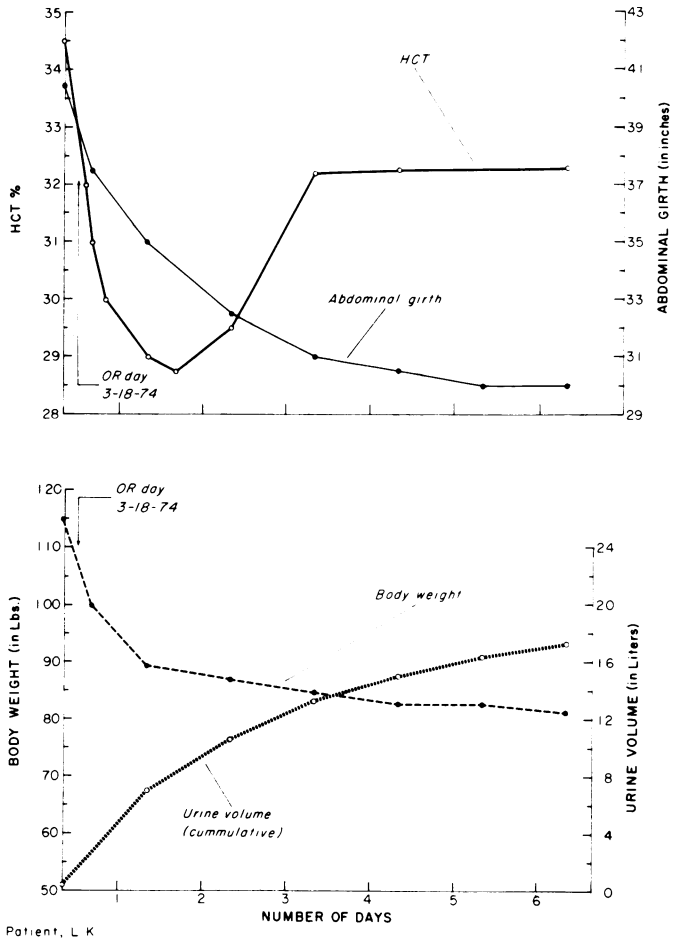

FIG. 11. The patient is a double amputee with ascites. Placement of the valve produces a hemodilution lasting for four days. Notice that the hematocrit never returns to normal indicating a true red cell deficit. The patients appearance is shown in Fig. 15.

been inadvertently omitted. One third of the shunts became occluded usually 1–2 weeks postoperatively after the ascites had been completely disappeared. The sudden reappearance of ascites heralds an occlusion of the shunt. All removed valves were examined to ascertain the cause for failure. Ninety per cent of the failures were produced by leakage of ascitic fluid around the threads of the valve allowing blood to replace the ascitic fluid in the venous tubing (Figs. 8, 9). This defect has now been corrected by sealing the threads. This blockage can easily be flushed from the venous tube and the valve replaced. It was unnecessary to replace the venous tube in any. Two patients required two replacements of the valve. In one case failure occurred after three months. Nevertheless, shunts which are patent longer than one month usually remain patent.

Correction of the defects in the valve should reduce occlusion rate to about 5%. Occlusion of the shunt was due to thrombosis in the venous tube due to a faulty valve system in all but one instance. Occlusion of the shunt produces only temporary failure and requires prompt valve replacement. Permanent failure was encountered in one patient with tricuspid insufficiency with such a high venous pressure that the valve would not open. One patient with a huge hepatoma and ascites responded favorably for two months but seemed to do less well during the last two months of his life, although no definite evidence of either tube blockage or excessive ascitic fluid was confirmed to us. The result was considered satisfactory by his surgeon.

Clinical Success

Thirteen patients in our series after one or more valve insertions have been followed for one week to 1½ years with relief of ascites. Five are more than six months with ascites relieved although one required a valve change in this interval. Twenty-one outside cases on whom information is available have lost their ascites from two weeks to six months. Six have occluded and have not been replaced with the new sealed valves, although three patients have had one or more valve replacements.

The postoperative weight loss averaged about 14 pounds per day with the loss accountable in the excretion of urine. Most patients lose at least 50 pounds of liquid. The hematocrit falls immediately after surgery due to hemodilution. The reduction in abdominal girth indicates that hemodilution is occurring (Figs. 10, 11). As urinary loss continues and ascites diminishes, the hematocrit returns toward normal but never reaches the preoperative level indicating that these patients are depleted of red cell mass. The red cell deficit is always more severe than realized and transfusions of packed cells may be necessary lest the hematocrit fall too low during the period of hemodilution (Fig. 12). The largest urinary loss in a single day has been 13 L. Such a diuresis

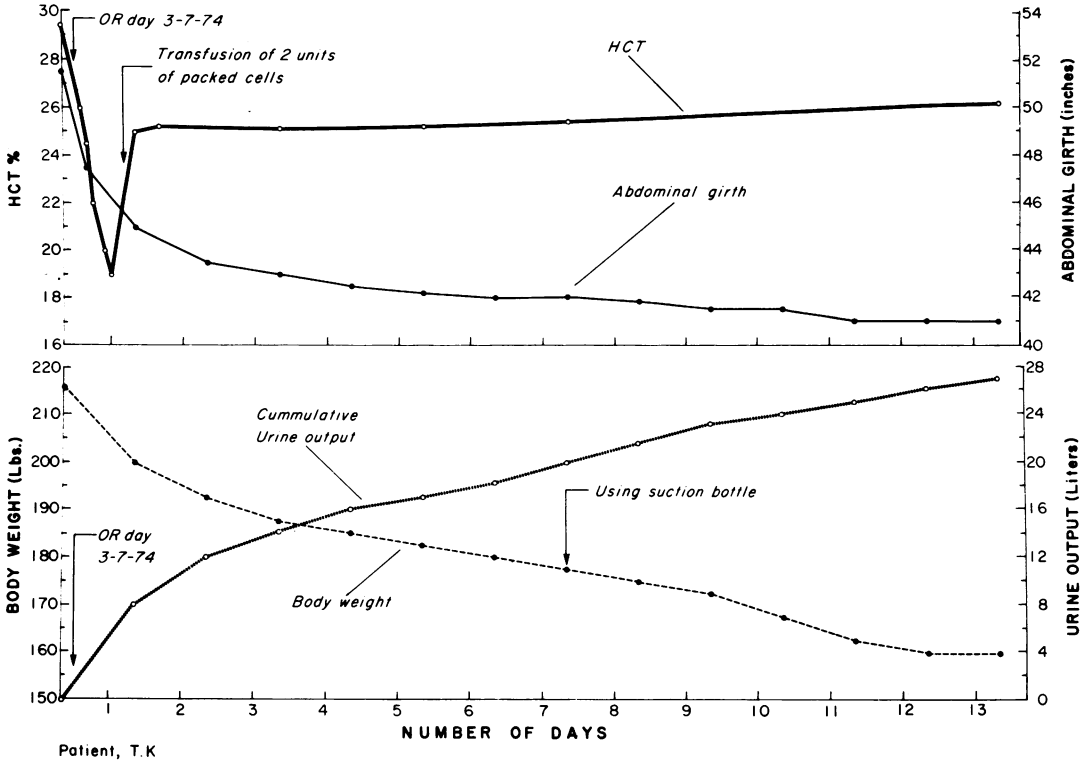

FIG. 12. The drop in the hematocrit was serious enough in this patient to require transfusion of packed red cells. In spite of these transfusions there is still a deficiency in the red cell mass. A weight loss of 58 pounds of ascitic fluid occurred. The patients physical appearance is shown in Fig. 14.

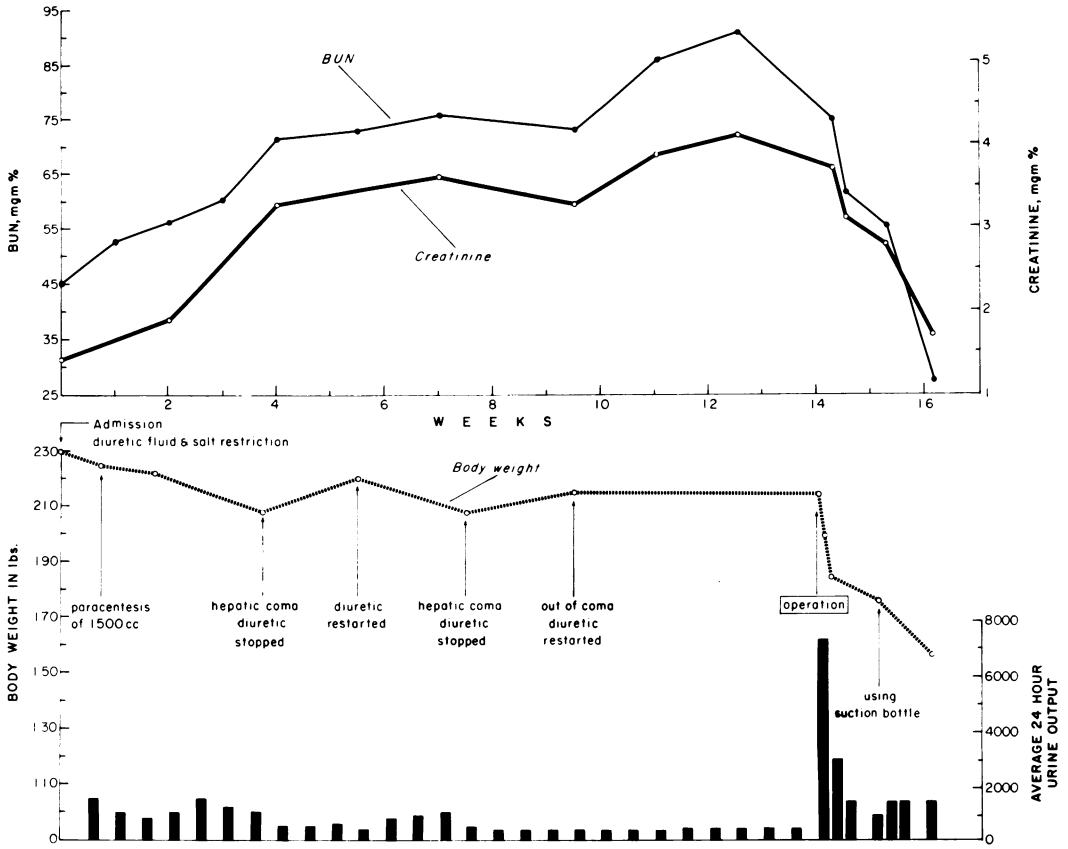

FIG. 13. The hospital course of a patient is charted. Notice that strict medical therapy and paracentesis produced only a 10 pound weight loss. Each time diuretics were tried the patient lapsed into coma with persistent oliguria. Finally, a peritoneo-venous shunt resulted in the loss of 58 pounds of fluid and a drop in the blood urea nitrogen and restoration of health and comfort. The course is that of patient shown in Fig. 14.

may be accompanied by a fall in serum potassium sufficient to require I.V. replacement therapy. Parenteral furosamide is given in 40 mg doses around the clock for the first 48 hours to maintain a maximal urinary volume. Later, the dosage is given orally. By the end of the first 10 days, the requirements for furosamide may decrease to once or twice a week or none at all. The excessive use of salt in food is avoided, but salt intake is not restricted.

The usual hospital course of a patient with severe ascites is charted in Fig. 13. The admission weight was 230 pounds. Rigorous therapy consisting of paracentesis (1500 cc) salt restriction, and diuretics brought about a loss of 20 pounds with an associated rise in the B.U.N. and the induction of hepatic coma requiring the abandonment of therapy. The patient emerged from coma and restored 10 pounds to his body weight. Diuretics

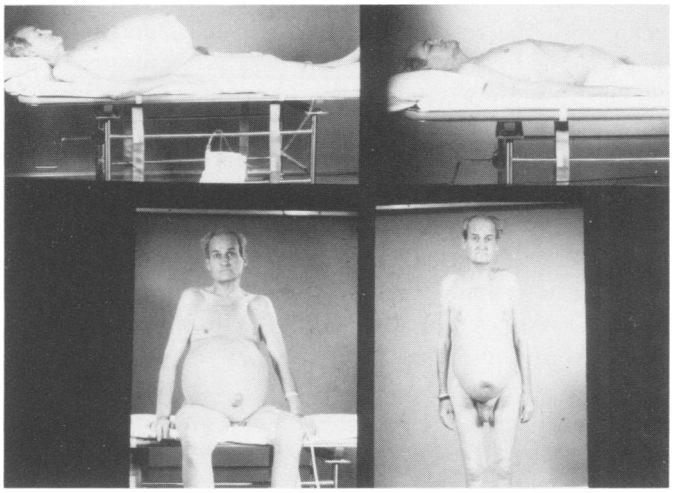

FIG. 14. The before and after appearance of the patient whose course is charted in Figs. 12 and 13. The patient was unable to stand preoperatively. Probably because the high B.U.N. raised his blood ammonia levels sufficiently to cause ataxia.

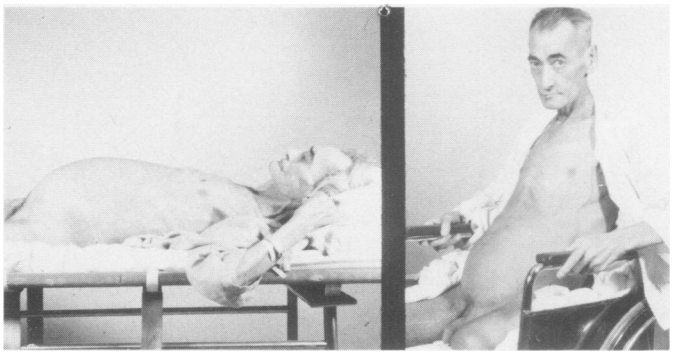

FIG. 15. The patient is a double amputee with cirrhosis. His first valve clotted after three months and was replaced with a valve which has remained patent.

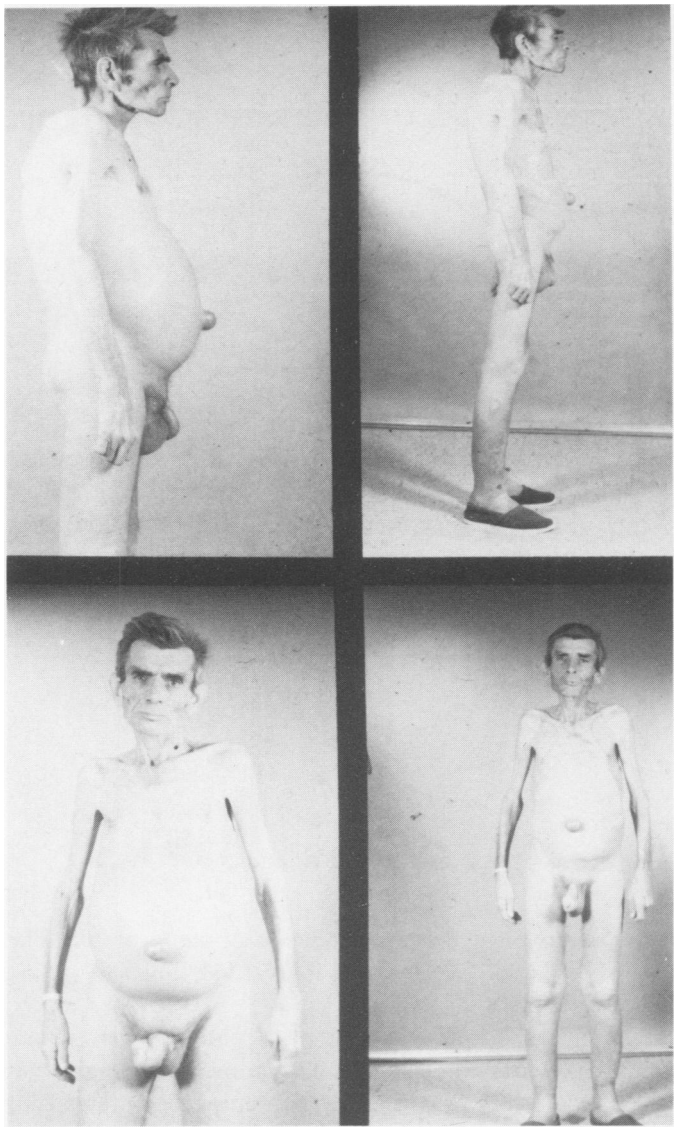

FIG. 16. The tense abdominal ascites and the usual thin walled umbilical hernia is shown. One must guard against the entrance of air into the peritoneal cavity on the subsequent repair.

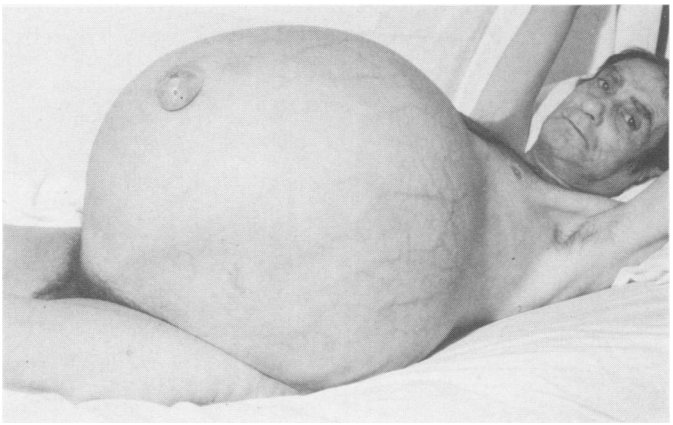

FIG. 17a. This patient had such tense ascites that he could not stand and could only breath if lying on his side. The valve has been functional for 1½ years, but the patient is uncooperative about his breathing exercises and always carried residual fluid until a second valve was inserted.

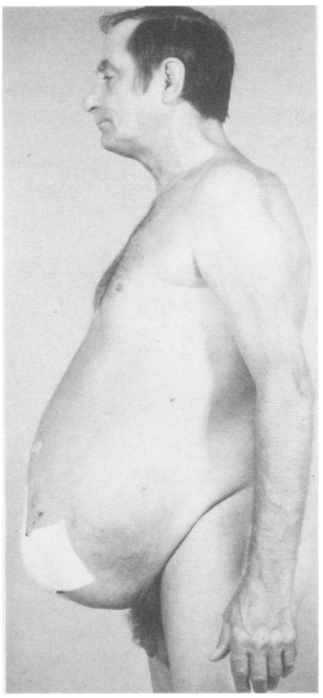

FIG. 17b. Appearance of patient in Fig. 17a, 1½ years later shows the residual ascitic fluid. The valve has been inspected to verify its patency and function. Since a second valve insertion the abdomen has become and remained flat.

were reinstated which again thrust the patient into hepatic coma. Thereafter, the patient maintained his weight at 217 pounds but after 14 weeks of therapy his renal function was failing. He became hypotensive and the average daily urinary output dropped to 20 cc per hour. The placement of a peritoneo venous shunt resulted in a weight loss of 55 pounds within the week. The B.U.N. has returned to normal and remained there. The patient was discharged two weeks post surgery without ascites and without dietary restriction or diuretics.

The typical appearance of patients before and after operations are shown in Figs. 14–18. Notice not only the loss of ascitic fluid, but the rapid loss of the cirrhotic facies with the restoration of muscle mass and fat. The facies begin to change within weeks after surgery Figs. 19, 20. Most impressive is the immediate sense of well being and return of appetite and strength. This is accompanied by a weight gain without the recurrence of ascites.

Excluding immediate deaths from end stage disease, and infections in whom the valves were removed, a total of 34 cases remain for analysis. Seven cases originally lost their ascites but the shunts have clotted and the valves were not replaced. These patients could be converted to successes. All but the patient with tricuspid insufficiency of the remaining 27 cases were successful.

Nevertheless, the procedure was successful in 26 of 34 cases.

Discussion

Plasma proteins have risen to normal following shunting with a return of the albumin globulin ratio toward

normal. Of special interest has been the improvement in kidney function following peritoneo-venous shunts. This has not only been accompanied by an increased urinary output but by a higher concentration of urea in the excreted urine. Elevated blood urea nitrogen falls rapidly to normal and remains in the normal range. This is of special interest since medical therapy tends to precipitate renal failure.

It is essential that the surgeon not try to combine medical therapy with peritoneo-venous shunt. All that could possibly be accomplished with diuretics then would be a dimunition in the rate at which ascitic fluid is formed by filtration. A reduction in this rate may not be desirable since it would reduce the amount of fluid flow through the shunt. A reduced fluid flow might precipitate clotting in the silicone tubing. Diuretics should be reserved for the treatment of peripheral or pulmonary edema and not be used to compete with the shunt in the reduction of ascitic fluid.

The same reasoning applied to the use of salt. Our inclination is not to restrict salt.

Obviously, renal function and the handling of salt and water require future studies in shunted patients.

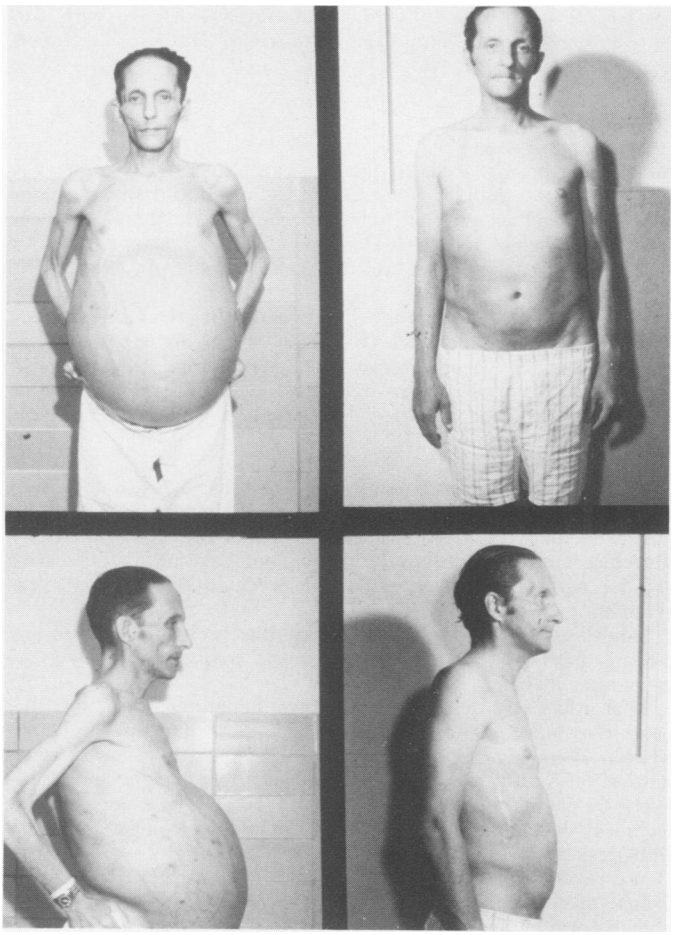

FIG. 18. A patient approaching terminal wasting ascites is shown before valve placement and 8 months later.

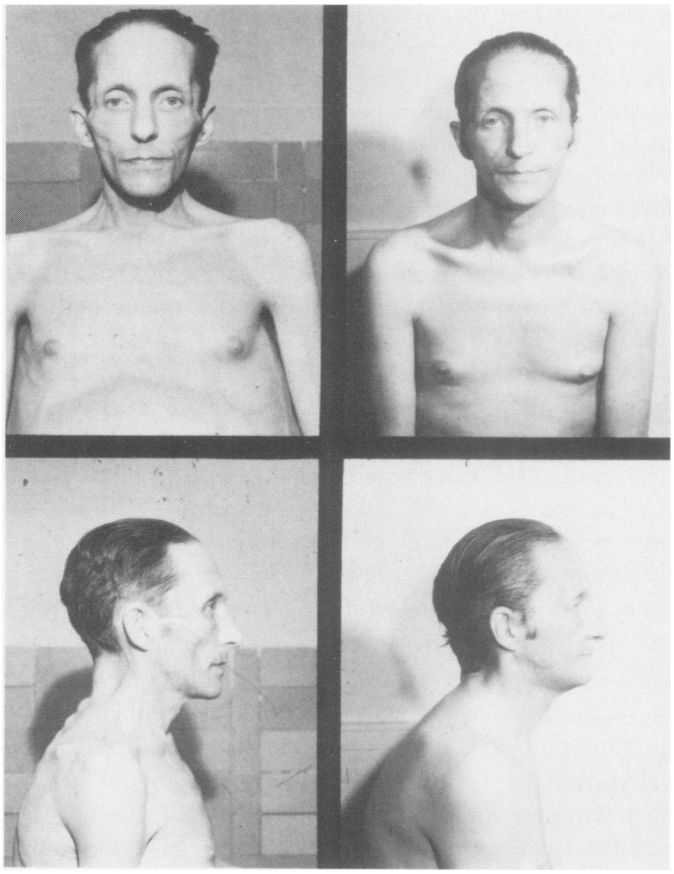

FIG. 19. The postoperative facies of the patient in Fig. 18 shows the loss of the so-called cirrhotic facies.

The operation is simple and can be performed with little or no trauma or blood loss. The results are gratifying and long-lasting. Most important, the patient no longer thinks of health as a non-attainable goal. Many of the patients suffered sufficiently to condition themselves negatively to the use of alcohol, but a few have gone back to heavy drinking and have returned with jaundice and hepatic decompensation.

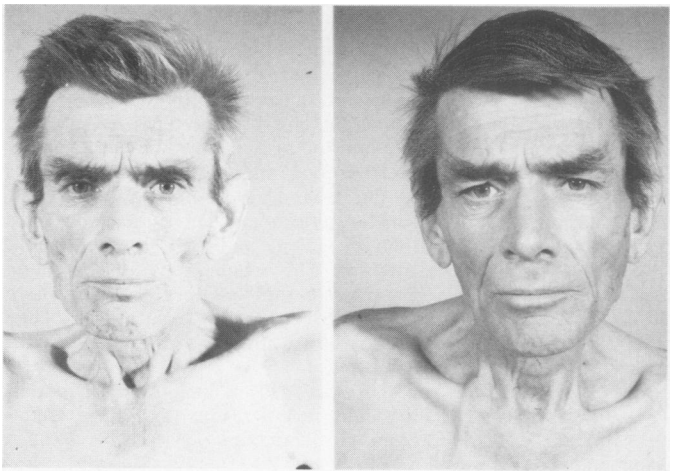

FIG. 20. The rapid change in facies is shown in photographs taken 3½ weeks following surgery.

One patient hemorrhaged from esophageal varices and developed ascites after a disconnection operation with sclerosis of the varices. This patient then was successfully treated with a peritoneo venous shunt and has been well for six months. However, patients who develop bleeding after a peritoneo venous shunt are not so fortunate. They cannot have a laparotomy without interrupting the peritoneo venous shunt, otherwise the air which enters the abdomen at laparotomy would be transported to the venous system and produce fatal air emboli.

One patient who was being dialyzed for chronic renal failure developed ascites. The ascitic fluid developed a uriniferous odor and the peritoneum became thickened. The ascites impeded the effectiveness of the dialysis. This patient has had a peritoneo venous shunt and is now being successfully managed by dialysis.

Thrombosis of the shunt has been a problem, but each time a correctable defect has been encountered. With the new modifications in the valve system, the thrombosis rate should decrease to about 5%. If this should not be realized it may be wise to consider the subcutaneous implantation of a flushing system which will clear the valve and the venous tubing. Only subsequent experience will determine whether modifications are necessary.

Although thrombosis of the tubing is sometimes difficult to detect, the use of radioactive isotopes has been of value to show tubal patency.

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Discussion

Dr. William Rhoads Waddell (Denver, Colorado): I'm speaking for a small group of surgeons in Denver and surroundings who have been evaluating a similar type of approach with an adaptation of a valve designed by an engineer at Denver University named John Newkirk. He designed this valve for the neurosurgeons, and it has had extensive use throughout the world for the treatment of internal hydrocephalus.

Originally, one of the attractive features of it was that the whole thing costs less than $10, but now it's up to $65, and the valve I'm speaking about is even worse. It costs $150.

The concept of the valve mechanism is somewhat different from that described by Dr. LeVeen, and I will show it to you now. (Slide) This is the whole apparatus. This is a chamber which is placed over the iliac crest. The length of both arms can be adjusted. This end goes into the peritoneal cavity, and this is introduced through the saphenous vein, up through the vena cava, to about the level of the diaphragm, or to the intrathoracic level.

(Slide) This shows a cross section and how it works. This is the chamber, and this flange on it has Dacron incorporated into it, so that it can be sutured to the periosteum. There are two valves that allow flow in only one direction, and they are both split valves of the same construction. The whole apparatus is made of silicone rubber. These are pressure-activated valves also. There is no critical opening pressure for either of these valves. They open with the slightest positive pressure, and they close with positive pressure on the distal end.

Compression of the chamber—and this is one of the essentials we believe—allows discharge of the fluid from within the chamber. It can be made of any size, but the ones we have been using are about 5 cc.

(Slide) This shows the manner in which all but two of these valves have been placed. In the two exceptions the chamber was placed over the pubis. I think this procedure does require general anesthesia, and in addition to what's shown here there has to be an incision below the groin to expose the saphenous vein. We haven't had any infection, and we have not had any clotting in eight patients.

Now, one of the differences between what Dr. LeVeen has reported and our experience is that six of our eight patients had ascites on the basis of extensive malignancy, and this particular patient had chylous ascites arising secondary to extensive reticulum cell sarcoma. She had been treated with radiation and extensive chemotherapy.

Dr. L. R. Eidemiller (Portland, Oregon): This august body hardly needs to be reminded of the unsatisfactory nature of the medical therapy of ascites, consisting of fluid restriction, bed rest, diuretics, and thus further progression of catabolism, decreasing visceral perfusion, and the frequent precipitation of hepatorenal syndrome.

Side-to-side portacaval shunting for this problem carries a significant morbidity and mortality, with, frequently, further deterioration in hepatic function.

That relief of ascites may improve hepatorenal function has been shown by clinical observation, and documented by many writers. Retransfusion of ascitic fluid has been done on an intermittent basis, and shown to be of at least transient benefit. However, the general application of this principle has awaited the technical development of a suitable technique to make it logistically feasible over a long term.

We recently encountered an unusual case of Budd Chiari syndrome of undetermined etiology which demonstrated to us the effective use of the LeVeen peritoneal-venous shunt. A 34-year-old man was explored at another hospital for fatigue, weight loss and