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

epigastric pain and jaundice. At laparotomy, he was found to have 200–300 cc of slightly bloody ascitic fluid, portal venous hypertension, and a swollen, turgid liver and spleen. No tumor could be identified. Liver biopsy showed canalicular biliary stasis and venous congestion, but no evidence of tumor.

Postoperatively, he did very poorly, and was transferred to our hospital with progressive ascites and hepatorenal failure. There an extensive workup failed to reveal the underlying cause of his illness, but vena cavography and attempted transjugular cholangiography revealed apparent thrombosis of the hepatic veins and the intrahepatic vena cava.

Visceral angiography confirmed the presence of portal hypertension, and also disclosed portal and splenic vein thrombosis. Again, no evidence of a tumor was seen.

By this time the patient was deteriorating rapidly, with massive ascites requiring daily paracentesis for increasing respiratory embarrassment. His hepatorenal failure and premoribund state made him a very poor candidate for any kind of surgery. However, the LeVeen shunt was placed under local anesthesia with a single injection of ketamine while the tunnel was made.

Certain modifications were required, because by this time he had also thrombosed his jugular venous system, and so we placed the venous end of the shunt through the cephalic vein, through our standard approach for axillary-femoral, or axillary-axillary bypass graft, and in so doing obviated the need to cross the clavicle.

(Slide) This displays the prompt recovery in hepatic and renal function after shunt placement, but it does not really demonstrate to you the dramatic clinical improvement which occurred in this patient. He was discharged at 14 days, ascites free, and now five months after operation has nearly normal liver function.

His underlying problem continues to defy diagnosis, but his clinical course has certainly been excellent.

Another instance where we feel that ascites plays a very critical role is the patient who has undergone portal systemic shunting, particularly in our current series at Oregon, of Warren distal splenorenal shunt. Many of these patients develop ascites in the postoperative period. I'd like to ask Dr. LeVeen, and also Dr. Warren, if they feel that the morbidity and mortality and perhaps slow recovery in some of these patients could be aided by placement of such a device.

Dr. Thomas C. Moore (Torrance, California): I'd like to comment upon the use of peritoneal-venous shunting and alternate means for the management of intractable ascites in early infancy.

Approximately ten years ago, in Muncie, Indiana, I managed a five-week-old infant with massive bile peritonitis, who had a spontaneous slough of the junction of the cystic duct, by means of a roux-Y anastomosis of the proximal jejunum to the small remnant of common hepatic duct.

The patient recovered, but developed massive and uncontrollable ascites four months following operation, associated with a roentgen demonstration of extrahepatic portal vein occlusion.

Dr. John Waldhausen, at the Indiana University Medical Center at the time, connected this infant's peritoneal cavity and the right atrium by means of a Holter valve. The ascites was relieved, and the patient did well during an approximate six months' period of followup, but was lost to followup when the family moved to Alabama shortly thereafter.

Due to a natural reluctance to plunge into medical publication, and not being sure quite what to call it, and feeling that no one would believe me anyway, this unusual experience has never been reported.

Recently at the Harbor General Hospital, Dr. Morris Asch, an associate, and I, had a patient with intractable chylous ascites—intractable to standard dietary means—that we were concerned about. Dr. Asch contacted Dr. LeVeen regarding the obtaining of one of his devices. However, in the interim we elected to employ hyperalimentation, and during a three-week period of hyperalimentation the chylous ascites disappeared and has not returned during a four-month followup.

Recent experiences with chronic peritoneal dialysis and implantable cannulas and chronic perivascular access conduits for hemodialysis and chronic parenteral ailmentation suggests the feasibility of the approach employed by Drs. LeVeen and Christoudias. Their method appears to be a safe and simple means of managing a difficult problem. It certainly is a major contribution.

Dr. Harry H. LeVeen (Closing discussion): In response to Dr. Waddell's comments, it is possible that other valves can be used, but they must be pressure activated. A slit valve is a flow-activated valve. Even the saphenous vein becomes incompetent at low pressures. One never knows whether those valve units employing a flushing pump thrombose and reestablish patency when a clot is flushed out of the tubing. Such a pump should not be necessary if thrombosis does not occur, and for that reason, one was not incorporated into our system. Such a device may be required and we can always add it; but, at this present moment, it doesn't seem necessary.

I want to thank Dr. Eidemiller for presenting his unusual case. Budd-Chari's syndrome carries such a grave mortality that it is remarkable that such a serious problem can be handled in such a simple manner and with such excellent results. I'd like to show you, if I may, some additional visual evidence of before and after therapy of some of these patients.

(Slide) This before and after picture shows a tense abdomen. The closeup shows how shiny his abdominal skin has become. He has edema of the lower half of his body as well due to the high abdominal fluid pressure. Postoperatively, his abdomen is flat and the edema is gone.

(Slide) This is another before and after picture of a man who lost 40 pounds of ascitic fluid after placement of the shunt.

(Slide) This man was sitting in another hospital awaiting attention for an ulcerating umbilical hernia, due to his tremendous ascites. They sent him to us and after a shunt we have repaired his hernia.

Most striking in all of these patients is the pinched facies with sunken cheeks and thin nose. Notice how the face fills out after this shunt has been done.