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Discussion

Dr. Robert Zeppa (Miami, Florida): Five-therapeutic prospective trials involve the comparison of portosystemic shunting with distal splenorenal shunting—the box score at the moment with respect to portosystemic encephalopathy is 3 to 2, 3 winning in terms of a lowered incidence. What does this mean?

I think it points to an enormous subjective error in the evaluation of portosystemic encephalopathy, which was not corrected by creating a prospective randomized trial. There is only one of the trials which has demonstrated in a meaningful way, by measuring a biochemical function for which there can be no argument; that is the trial that was conducted in Atlanta, where maximum rate of urea synthesis was an end point, one that can be reproduced over and over again.

Therefore, I have exposed my biases clearly as to which is the better shunt under those circumstances.

In this paper Dr. Chandler points out that a meticulous portal-azygos disconnection operation was conducted at the same time they did this. This is important. Sufficient data now are available to point out that one can retard the development of major channel collaterals to a distal splenorenal shunt by doing a careful portalazygos in the area that has been described so well.

The authors have also mentioned in their paper "that post-shunt encephalopathy is no more common than pre-shunt encephalopathy," and I would like to take exception to that, with Dr. Chandler's indulgence. I think that the only way that one can accept that statement is if it is made applicable to a single etiology of portal hypertension and liver disease.

For example, in patients with schistosomiasis there is no pre-shunt encephalopathy. It is totally unknown. It only occurs at the rate of 60% when one does a portosystemic procedure. So the statement may well be true, but it would be true only when applied to those patients who suffer from alcoholic liver disease and not to the others.

A paper from Ryer's group describes taking down seven splenorenal shunts in patients with schistosomiasis, and measuring two important features: (1) the change in serum albumin, which increased in all patients, and (2) liver size, which increased in all patients.

Our own experience with this kind of problem has nothing to do with taking down shunts, but we were faced with a 42-year-old man with chronic, active liver disease, who was on about 50 mg of prednisone daily, and who had bled four times; the hepatologist requested that we stop the bleeding. The patient had a double coronary. I missed it—ligated one. By the twelfth postoperative day he was in stage 4 coma.

Angiography at that point revealed a fairly large channel, well opacified on the venous phase films of the superior mesenteric artery injection, and no opacification of the liver. He was taken to the operating room and this channel was ligated; the man was awake and out of the intensive care unit in three days.

Patients who have chronic active liver disease are notoriously sensitive, and that is why in the paper that preceded this one, describing hepatitis following a coronary artery bypass, it is no surprise to me that that patient went into coma very soon after a portosystemic shunt.

Dr. Chandler, in the entire series that you cited in the manuscript, what was the percentage with respect to alcoholism *vs.* nonalcoholism? Were there any on steroids, and was this a harbinger of the development of portosystemic encephalopathy? And did you attempt to assess liver size before and after the conversion?

Dr. Gardner W. Smith (Baltimore, Maryland): If you are going to get into this kind of trouble, you could make it easier for yourself to start with by doing something different in the way of a shunt. I can think of nothing more difficult than trying to reverse a side-to-side portacaval shunt, and it occurs to me that perhaps Dr. Talman's preference for end-to-side shunts could be defended in this regard.

I might remind you that many years ago Dr. Rousselot strongly urged that the umbilical vein be preserved whenever you perform a portacaval shunt—specifically an end-to-side one—and he made this recommendation on the grounds that, in the event of a shunt thrombosis, a potential collateral pathway would still be available.

Dr. Chandler, if he is going to do end-to-side shunts, would give us another reason to heed Dr. Rousselot's advice. As Dr. Adamson has pointed out, if you have cause to restore liver blood flow at some subsequent time by arterializing the portal vein, it is a lot easier to arterialize the reopened umbilical vein than it is to try to arterialize the hepatic stump of the portal vein as a secondary procedure.

Dr. W. Dean Warren (Atlanta, Georgia): I agree with almost everything the author has said.

One of the worst mistakes made in this field is to do a splenectomy for the hypersplenism of portal hypertension. This is almost never needed from a standpoint of the coagulopathy, and frequently leads

to a post-splenectomy thrombosis of other radicals that precipitates bleeding from varices. The chance for optimal therapy usually has been lost at this point, and the performance of a splenectomy for a platelet count of 50,000 is a very bad mistake.

In an operation which we were forced to perform upon her in order to control persistent bleeding through a "gastritis," the patient had a patent superior mesenteric vein and we took one branch of that vein and made a small anastomosis in a retrograde fashion to the inferior vena cava.

There was a great enlargement in the branch of the mesenteric vein and the mesocaval shunt. The patient had become severely encephalopathic at this point, and this procedure was the ligation of the mesenteric branch, closing the mesocaval shunt and the creation of a coronary left renal shunt similar to the procedure utilized by Inokuchi in Japan.

The metabolic data on this woman in our Clinical Research Center unit while on a 40-g protein diet, shows that the fraction of total hepatic blood flow comprised of portal venous blood has jumped from 0 to 40%. The actual total hepatic blood flow has doubled from 646 to 1200 cc/min. Fasting blood ammonia has decreased from 130 μg/dl to a normal of 27 on a 40-g protein diet. That is real hypoammonemia prior to the closure of the shunt which is instituted again in the ammonia tolerance score. Her preoperative value calculated under the curve was 14,000, and this dropped to 1000 after operation, a difference of 14 magnitudes. As I have mentioned previously, this nonprotein nitrogen metabolic test was mentioned by Pavlov in his classic paper in 1893.

In summary, this is a classically studied patient to demonstrate that closure of a total shunt will reverse encephalopathy in the cirrhotic, just as it did in the patient we presented two or three years ago who had severe encephalopathy following a total shunt for a portal vein thrombosis. We have now closed shunts in a variety of ways, including surgically, by embolization, and by the placement of balloons. This inevitably results in hepatic function improvement if the patient withstands the impact of the initial procedure. Dr. Chandler correctly points out that one must avoid the very far-advanced, chronically ill patients, because they simply will not tolerate this type of open surgery. When the shunt is closed, if the patient already has a coagulopathy, the chance of death from bleeding into the abdomen is very great indeed. However, closure of the shunt by nonoperative means and later prevention of hemorrhage with variceal sclerosis is another approach to this problem in these very far-advanced cirrhotics.

DR. JAMES G. CHANDLER (Closing discussion): Bob Zeppa is certainly correct. The statement in the manuscript refers to the incidence of pre-shunt and post-shunt encephalopathy as being the same, if appropriate controls are considered, but that statement should only apply to patients with alcoholic cirrhosis.

In answer to the specific questions that Dr. Zeppa posed, 72% of the patients at the University of Virginia that I have shunted have been alcoholics. Two patients in the overall shunt group received steroids at some time during their post-shunt course for treatment of chronic arthritis. None of the five study patients have received steroids.

We did try to evaluate liver size. Although I was present at all 114 operations, retrospectively, when I looked over my notes, I was unimpressed with my accuracy in describing liver size.