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Discussion

Dr. Robert R. Linton (Boston, Massachusetts): I greatly admire Dr. Malt's skill as a surgeon since he saved my life two years ago after an automobile accident.

In my opinion too much emphasis has been placed on the necessity of producing a large caliber portal systemic venous shunt to prevent further esophageal bleeding, completely ignoring the fact that they result in a higher incidence of postshunt encephalopathy and liver failure than smaller caliber shunts. If they are "done right" they control esophageal bleeding and prevent postshunt encephalopathy and liver failure.

(Slide). This first slide shows that in 109 consecutive cirrhotic patients followed for 5 years after splenectomies and splenorenal shunts, the over-all mortality rate was approximately 43%, whereas in 40 patients with direct portacaval end-to-side shunts, the mortality rate was 64%, so that it would seem to me, more patients live longer, more useful and happier lives with the smaller caliber splenorenal shunts.

(Slide). Next I will show you a few examples of what can be accomplished with even smaller shunts than the splenorenal end-to-side type. This shows you a man of 64 years of age with massive ascites for three years secondary to portal cirrhosis. He had been under the best of medical care until the time he was referred to me for a splenectomy and an end-to-side splenorenal shunt. He had required numerous abdominal paracenteses about once a week before he came to my service.

(Slide). This shows the splenorenal shunt that I performed. Note the shortness of the segment of the splenic vein used to make the anastomosis to the renal vein. It is my opinion that it is important to use as short a segment as possible in order to prevent thrombosis of the shunt. His ascites has disappeared and he is living a normal life.

(Slide). This is a venous portogram that shows no evidence of a splenic, superior mesenteric, or portal vein. It was performed on a young man who had an extrahepatic portal bed block. (Slide). This demonstrates very clea ly that you can accomplish a very small caliber shunt, using a segment of his saphenous vein, only 8 mm in diameter. It was anastomosed to a varix in the mesentery of the small bowel and to the inferior vena cava, crossing over the third portion of the duodenum, thus establishing a mesocaval H shunt.

This patient had had many, many hemorrhages when I saw him first at the age of 15. He had had a previous splenectomy and a splenorenal shunt that had failed to control his bleeding. I first treated him by transthoracoesophageal ligation of his varices, but this did not control his bleeding so I finally constructed this H type of mesocaval shunt in 1964.

At the present time he is now married, has a job and has even been able to take out a satisfactory life insurance policy. Up to 1964 he had received approximately 500 blood transfusions, and since 1964 he has received none, a real triumph for a small caliber shunt.

(Slide). The patient on whom I performed this type of shunt had had a previous splenectomy without a shunt but continued to bleed many times. He came to me in 1954 and I was able to construct what I have called a coronocaval shunt by anastomosing the distal end of the left gastric or coronary vein, 8 mm in diameter, to the inferior vena cava. This is a very selective type of shunt as it drains the venous esophageal blood directly into the low pressure caval system.

This man has been employed ever since the operation in 1954. He is still leading an active normal life now 22 years since his coronocaval anastomosis was performed, another beautiful example of the therapeutic success of a small caliber portal systemic venous shunt.

(Slide). This shows a splenorenal shunt performed in a patient who was a Class C risk according to Dr. Child's classification, and I had to first suture his esophageal varices transthoracoesophageally to save his life. Then 6 weeks later I constructed a splenorenal shunt using a three piece panelled saphenous vein graft because the splenic vein was not sufficiently long. It was not more than 7 or 8 mm in diameter. It has been 10 years since the shunt was constructed and he is without any evidence of encephalopathy or esophageal bleeding.

In conclusion it is my opinion that what we should be interested in is how long and well are the patients going to live as well as the control of their esophageal varices after shunt surgery. It seems to me, if the younger surgeons interested in this symptom complex will learn how to construct these small caliber shunts that I have described, the results will be even better than my splenorenal ones. Finally I would like to emphasize that it is of vital importance in order to obtain the best results in shunt surgery that, in addition to having a skillful surgeon, one must have an internist who is extremely interested in these sick liver patients and an anesthetist who is cooperative and particularly interested in giving one the type of anesthesia that you think is the most desirable.

DR. DONALD CLARK NABSETH (Boston, Massachusetts): I would like to emphasize the importance of an intact clotting mechanism in determining immediate mortality in cirrhotic patients undergoing shunting. This is especially true if one chooses to do the distal splenorenal shunt, where one does not have the advantage of a marked decrease in portal pressure in the immediate postoperative period.

In our series of 27 splenorenal shunts, we have had one operative death and several close calls from persistent intra-abdominal hemorrhage. All patients were in Child Category B or C, and seemed to have little postoperative difficulty with other hepatic metabolic defects.

What I wish to suggest is that the clotting defects, whether existing preoperatively or arising intraoperatively, may be the most important determinant of operative mortality, at least in cirrhotic patients undergoing the distal splenorenal shunt.

DR. W. DEAN WARREN (Atlanta, Georgia): I actually agree with most of what Dr. Malt says, and what I would like to do is, simply, to show you some nitrogen metabolism studies, as they reflect encephalopathy. One of Dr. Malt's questions is: How can certain groups quote such a low encephalopathy level, when in their control study 38% of the patients already were classified as being encephalopathic?

The reason is, we do not consider a single episode, such as a patient having a massive hemorrhage, and then becoming confused during that episode, as having encephalopathy. We were referring to chronic portal systemic encephalopathy, in which a patient is chronically unable to tolerate a 60 gm protein diet, or has an abnormal EEG on a 40 cm protein diet.

This was our original data on a test which was developed in Atlanta, called the maximum rate of urea synthesis. I will not go into all the details of it, but, as you know, ammonia, and many other nitrogen-containing compounds are metabolized through the Krebs-Henseleit cycle. We have identified, and it has now been confirmed in several other centers, that there are other major deficiencies in the cirrhotic liver at the two major pathways of entry; namely, ornithine transcarbamylase and argininosuccinylsuccinase.

In addition to that, a total shunt changes the portal flow to the liver. In this slide you can see that the selective postoperative shunt and the total, which are mesocaval shunts, located below, are both equal preoperatively, but there is a significant drop in the postoperative level, and the number of encephalopathy patients in the yellow group, or the total group, is now 8, and we have one in the green group by our criteria, which, I stress, is chronic portal systemic encephalopathy, not episodic.

The way that we can cut it below control levels is that we simply stop the number of hemorrhages that these patients have.

In order to develop a procedure that can be carried out in an ordinary hospital, Dr. Rudman has recently restudied the whole problem of the technique of ammonia, and this will be published in the *Journal of Laboratory Chemistry* in May, and he has shown that you can construct a curve that can be used very much as a glucose tolerance curve. If your values are not at least 40 mcg/%, or less, then you are not doing the test correctly. Below are the normals; above, the cirrhotics.

The next slide shows you again our randomized—and I stress this—our randomized series of the selective shunt versus the total shunt. And here again the results are highly significant, the value being less than 0.001.

In this test, immediate flow is one of the factors to the liver, and you can see the tremendous change. This may drop by a seven or eight times in this particular test, because portal flow is lost to the liver, and, again, the very low values correlate with encephalopathy.

Finally, I want to point out that there are going to be a number of studies of nitrogen compounds which are going to be implicated in encephalopathy. Dr. Ferrah, of our metabolic study group, has recently devised the radioimmunoassay of tyramine, and these are his preliminary data, which are being published shortly, showing that the controls and the nonhepatitic patients have very low levels of tyramine in the blood. The nonencephalopathic cirrhotic is significantly higher, and the encephalopathic cirrhotics, which are the total shunts, are far higher than any of the other groups studied.

Thus we believe that these data continue to support the superiority of portal perfusion of the liver, as it relates to chronic systemic encephalopathy and nitrogen metabolism.

DR. WILLIAM V. MCDERMOTT, JR. (Boston, Massachusetts): Our total experience over a number of years differs in several ways

from Dr. Malt's data which have been presented. Interestingly, however, it also differs within itself in different segments of time in which these data have been analyzed.

This amounts to about 500 shunts from 1945 up to 1974. The proportion of different types of shunts varies from time to time as you see, with the popularity.

The incidence of encephalopathy differs quite significantly from Dr. Malt's data. I don't propose to try to analyze the difference, or the reasons for these differences in any detail. Obviously these involve selectivity at various points in history. Our data would tend to agree with Dr. Linton, in that the incidence of encephalopathy is considerably lower, with the splenorenal shunt than it has been with the portacaval shunt, although this perhaps is counterbalanced by the fact that the rebleeding after construction of a splenorenal shunt has been considerably higher, perhaps roughly twice that as seen in the portacaval.

I also would like to emphasize that we use the criterion of encephalopathy as mentioned by Dr. Warren; that is, a chronic, postshunt encephalopathy, not the hepatocellular failure of the liver which occurs with and without portal-systemic shunting, and which is related more to a disorder of the hepatic cell than it is to the bypass of the intrinsic mechanisms of the liver by portal blood.

In terms of the long-term survival by cumulative tables, this would suggest the splenorenal shunt does have, in fact, a preferential survival value over the portacaval.

On the other hand, as Dr. Malt has pointed out, one must analyze the differences in the groups, and in this particular segment of our studies over the years you can see that, by liver index, calculated as a composite group of a number of liver function tests, and by the operative risk, as assessed by the anesthesiologists in their physical status scale, the splenorenal group, by chance or by selection, was actually better by these standards than the portacaval; and that probably accounts for the better survival, rather than any intrinsic difference in the shunts themselves.

I would agree with his comments on prospective studies. I'm sure you are familiar with many of the prospective studies in this area which have been done by the Boston In-Hospital Liver Group, and by others, on the prophylactic and the therapeutic shunts. We're in the process now of randomizing the distal splenorenal shunt, which has been popularized by Dr. Warren, and which may well prove to have a lower incidence of encephalopathy.

DR. MARSHALL J. ORLOFF (San Diego, California): Our prospective studies of unselected bleeding cirrhotic patients who underwent emergency portacaval shunt within 8 hours of admission, and involved all comers, generally support the conclusions that Dr. Malt presented to us today regarding the predictors of survival. In our most recent analysis, we found only two factors preoperatively that had a significant adverse effect on the outcome.

The first was the presence of ascites, which Dr. Malt emphasized, and the second was a SGOT level of 100 or more units, which was a reflection of acute alcoholic hepatitis and active hepatic necrosis superimposed on chronic cirrhosis.

We analyzed over seventy other factors, alone or in combination, and none of them had a statistically significant influence on survival at the 95% level of confidence.

However, the crucial question is: Do the large number of so-called poor-risk bleeding cirrhotic patients, including those with ascites or any other predictors of a high mortality rate, have a better chance of survival with portacaval shunt than with any other available form of therapy? Dr. Malt's analysis did not deal with this question.

In most fields of surgery, including the surgery of portal hypertension, surgeons have had almost a reflex abhorrence for an operative mortality rate of 10 to 20% or higher, or a long-term survival rate of less than 60 or 70%. The main exception to this reflex is in the field of cancer, in which the recognized high lethality of the disease makes an operative mortality rate of 20% and a 5-year survival rate of 30% quite acceptable, and, in some cancers, really quite excellent.

What is often ignored is the fact that cirrhosis with varix bleeding is every bit as lethal as most cancers. Unless the portal hypertension is decompressed, 5-year survival of an unselected population of bleeding cirrhotics is not much more than 5 to 10%.

Our studies in 233 unselected patients have shown a 10-year survival with medical treatment of zero, with emergency transesophageal ligation, followed by elective shunt (11%), and emergency portacaval shunt (29%).

When you consider that, in a 10-year period of time, in the United States 350,000 people die of cirrhosis, and about 117,000 of these die of bleeding varices, the salvage of life with emergency portacaval shunt is really quite large, amounting to some 35,000 patients.

It is possible by restricting portacaval shunt to only the good-risk patients to obtain a 4 or 5% operative mortality rate and a high long-term survival. I'm thankful that Dr. Malt and his colleagues did not adopt such a restrictive policy, since so doing is appropriate only if there is an alternative form of effective therapy. For cirrhosis with bleeding varices there is no alternative form of effective treatment; and, therefore, restriction of the operation to good-risk patients will have a negligible impact on the over-all survival of the disease.

For example, in the statistics reported by the Boston Interhospital Liver Group in 1974, only 9% of some 832 patients with bleeding varices were subjected to portacaval shunt. It is simply not possible for an operation to significantly affect the survival of a lethal disease when only 9% of the patients have the operation.

The important objective in the therapy of cirrhosis must be improving the survival of the over-all population with the disease, not selecting a few patients who will have a low operative mortality rate.

As of this date, the results of our studies indicate that this objective is possible only by the broad application of the portacaval shunt to many patients.

DR. RONALD A. MALT (closing discussion):

Dr. Linton is the doyen of portal-hypertension surgery and continues to set the standard to which we aspire.

In our original scheme for weighing the figures, we used clotting defects, but it turned out that, although it was a very good predictive factor, it was not so good as some of the others. Therefore we discarded it to keep things simple. I agree with Dr. Nabseth entirely. If the patient isn't going to clot, he is not going to survive. The important point that both Drs. McDermott and Warren brought up is the definition of encephalopathy. Unless we agree on what we are talking about, we are never going to come to the same conclusions. I propose that all of us who are interested in this problem should decide on a common definition of encephalopathy, whether clinical or chemical and that we should all adopt this in reporting our data.

Dr. Orloff's comments are germane. We had SGOT in our original list of predictors, but discarded it for the same reason we chose not to use clotting factors.

I am glad that in our hospital the survival rate after medical treatment or ligation of varices is a bit better than it is in California, and that our shunt comparisons don't have to be made with such ominous data. But, certainly, it is true that about 20% 5-year survival rate in untreated patients is the best one can hope for. One could even turn the issue and ask: If the surgical survival rate is only 30 or 40% with all this effort, is that appropriate, or should we be devoting ourselves to some other line of endeavor?