50. Rudman D, Difulco TH, Galambos JT, et al. Maximal rates of excretion and synthesis of urea in normal and cirrhotic subjects. J Clin Invest 1973; 52:2241–2249.
51. Rypins EB, Henderson JM, Fulenwider JT, et al. A tracer method of measuring rate of urea synthesis in normal and cirrhotic subjects. Gastroenterology 1980; 78:1419–1424.
52. Ansley JD, Isaacs JW, Rikkers LF, et al. Quantitative tests of nitrogen metabolism in cirrhosis: relation to other manifestations of liver disease. Gastroenterology 1978; 75:570–579.
53. Tygstrup N. Determination of the hepatic elimination capacity (Lm) of galactose by single injection. Scand J Clin Lab Invest Suppl 1966; 92:118–125.
54. Henderson JM, Heymsfield SB, Horowitz J, Kutner MH. Measurement of liver and spleen volume by computed tomography. Radiology 1981; 141:525–527.
55. Parsons-Smith BG, Summerskill WHJ, Dawson AM, et al. The electroencephalograph in liver disease. Lancet 1957; 2:867–871.
56. Rikkers LF, Jenko P, Rudman D, Freides D. Subclinical encephalopathy: detection, prevalence, and relationship to nitrogen metabolism. Gastroenterology 1978; 75:462–469.
57. Henderson JM, Kutner MH, Bain RP. First-order clearance of plasma galactose: the effect of liver disease. Gastroenterology 1982; 83:1090–1096.
58. Warren WD, Millikan WJ. Selective transsplenic decompression procedure: changes in technique after 300 cases. Contemp Surg 1981; 18:11–32.
59. Smith RB, Warren WD, Salam AA, et al. DACRON interposition shunts for portal hypertension: an analysis of morbidity correlates. Ann Surg 1980; 192:9–17.
60. Stipa S, Ziparo V. Mesenterico caval shunt with autologous jugular vein. World J Surg 1984; 8:702–705.
61. Jackson FC, Perrin EB, Smith AG, et al. A clinical investigation of the portacaval shunt: II, survival analysis of the prophylactic operation. Am J Surg 1968; 115:22.
62. Conn HO, Lindenmuth WW, May CJ, Ramsby GR. Prophylactic portacaval anastomosis. A tale of two studies. Medicine 1972; 51:27.
63. Conn HO. Editorial: therapeutic portacaval anastomosis: to shunt or not to shunt. Gastroenterology 1974; 67:1065–1071.
64. Malt RB, Malt RA. Tests and management affecting survival after portacaval and splenorenal shunts. Surg Gynecol Obstet 1979; 149:220–224.
65. Mikkelsen WP. Therapeutic portocaval shunt. Preliminary data on controlled trial and morbid effects of acute hyaline necrosis. Arch Surg 1974; 108:302–305.
66. Maillard JN, Flamant YM, Hay JM, et al. Selectivity of the distal splenorenal shunt. Surgery 1979; 86:663–671.
67. Belghiti J, Grenier P, Nouel O, et al. Long-term loss of Warren's shunt selectivity. Angiographic demonstration. Arch Surg 1981; 116:1121–1124.
68. Malt RA, Nabseth DC, Orloff MS, Stipa S. Portal hypertension. N Engl J Med 1979: 301:617–618.
69. Nabseth D. The distal splenorenal shunt: an enigma. Am J Surg 1981; 141:579–581.
70. Warren WD, Millikan WJ, Henderson JM, et al. Selective variceal decompression after splenectomy or splenic vein thrombosis—with a note on splenopancreatic disconnection. Ann Surg 1984; 199:694–702.
71. Henderson JM, Millikan WJ, Wright-Bacon L, et al. Hemodynamic differences between alcoholic and nonalcoholic cirrhotics following distal splenorenal shunt—effect on survival? Ann Surg 1983; 198:325–334.
72. Smith-Liang G, Scott J, Long RG, et al.Role of percutaneous transhepatic obliteration of varices in the management of hemorrhage from gastro-esophageal varices. Gastroenterology 1981; 80:1031–1037.
73. Henderson JM, Galloway J, Gong-Liang J, et al. Portaprival collaterals following distal splenorenal shunt: incidence, magnitude and associated portal perfusion changes. Radiology, in press.
74. Zeppa R, Hutson DG, Levi JU, Livingstone AS. Factors influencing survival after distal splenorenal shunt. World J Surg 1984; 5: 733–738.
75. Zeppa R, Hensley GT, Levi JU, et al. The comparative survival of alcoholics *versus* nonalcoholics after distal splenorenal shunt. Ann Surg 1978; 187:510–514.
76. Warren WD, Millikan WJ, Henderson JM, et al. Ten years portal hypertensive surgery at Emory: results and new perspectives. Ann Surg 1982; 195:530–542.
77. Starzl TE, Francavilla A, Halgrimson CG, et al. The origin, hormonal nature and action of hepatotrophic substances in portal venous blood. Surg Gynecol Obstet 1973; 137:179–199.
78. Warren WD. Control of variceal bleeding: reassessment of rational. Am J Surg 1983; 145:8–16.

Discussion

Dr. Robert Zeppa (Miami, Florida): We are indebted to Dr. Millikan and his colleagues for this elegant work. In fact, when you have an opportunity to review the manuscript in the *Annals of Surgery*, you will find that it is more than elegant and extraordinarily informative.

This is a trial that has been carried out longer than any other prospective randomized trial concerning efficacy of shunts, short of the first one that was done in Boston. None of the others have been carried out this far, and that one in Boston stopped at 7 years.

This well-written paper carries the meat of the argument, summarized by Dr. Millikan, clearly showing the therapeutic advantage of distal splenorenal shunt.

I would like to corroborate Dr. Millikan's findings (slide) concerning survival of nonalcoholics. My colleagues and I, Drs. Hutson, Livingston, and Levi, updated this information as of July 1983. The middle bar is an actuarial analysis of survival of our entire group at that time, with the numbers listed on the slide. The upper graph depicts the survival of nonalcoholics out to 8 years. The standard errors are still too large at this point. It will be 15 years from the time we started this until we will be able to tell you about the 10-year survival with a degree of confidence.

Note the standard errors. We begin to see, as Dr. Millikan point out, a separation in survival in those groups at about 2 years. The lower curve depicts the survival of the alcoholics, but within this cohort of nonalcoholic patients, uniformity is not the order of the day.

(Slide) Examine this particular curve, actuarial analyses. This depicts the survival of our patients who were on steroids, 20% of the population of nonalcoholics. Note the improved survival of those not on steroids when you pull these patients out.

I would like to ask Dr. Millikan three questions. Does age play a role in the encephalopathy after distal splenorenal shunt in your series? In ours it has not—which in entirely different from the experience reported for total portal diversion.

Second, are there later measurements of the maximum rate of urea synthesis? From the manuscript, it seemed to have improved in 4 years, and I wonder if that is just a reflection of those patients with rags in the venous system, where clots develop.

Third, do you have any data at all concerning survival in the alcoholic cohort where continued alcohol abuse is a problem? Dr. Hutson, your Local Arrangements Chairman, has been looking at our population of patients who have, we think, stopped drinking, and we have gone through a long rigamarole. We have no statistically significant information now, but the trend seems to be that these people will have, following distal splenorenal shunt, an equal survival to the nonalcoholics.

It was a pleasure to listen to this very important, scholarly work. I believe that this presentation represents the final effective nail in the coffin of the Philistines, who have doubted the efficacy of distal splenorenal shunt in the therapy of variceal hemorrhage.

Dr. Jere W. Lord, Jr. (New York, New York): I did not realize I would be among the Philistines. In fact, I agree with Dr. Zeppa that this excellent paper by Dr. Millikan and his associates has relegated central venous shunts, such as splenorenal and interposition mesocaval,

to a virtually moribund state—and Dr. Warren is prepared to carry out the last rites.

Before that, however, I think there are four suggestions that possibly central venous shunts are not moribund.

First of all, if a miracle could take place and Dr. Robert Linton could come to this podium, those of you who have ever heard him discuss this subject would find him very effective. I refer to his 1965 editorial in *Surgery, Gynecology, and Obstetrics,* where he wrote that the splenorenal shunt was far superior to the portacaval end-to-side or side-to-side shunt for the important reason that the splenic vein is a small vein and permits only a certain amount of flow, compared to the large portacaval flow, and therefore lowers the preshunt pressure only slightly. The minimal fall was sufficient to control the variceal bleeding, but did not lead to encephalopathy. His follow-up for 5 years or longer was 57% alive and well.

A second miracle might bring us Dr. Ted Drapanas. Those of us who had the privilege of enjoying the banter between Drs. Drapanas and Warren from 1970 to 1975 would realize that Dr. Drapanas could still make a pretty good case for the interposition shunt. The principle again is that the mesocaval shunt, no matter how large the prosthesis, Dacron®, Teflon®, or GoreTex®, and the large size of the vena cava, the superior mesenteric vein is a small vein, about 1 cm in diameter, varying in our measurements between 8 to 11 mm.

Third, I would like to mention our own experience over 27 years. I have personally operated on 20 private patients; 10 were splenorenal and 10 were interposition mesocaval shunts. During the same time at Bellevue and Cabrini Hospitals, there were many on the ward services, but we were unable to follow them after surgery after they left the hospital. Of the 20 patients, one died 3 weeks after surgery following mesocaval shunt, due to congestive heart failure and acute hemorrhagic pancreatitis; at autopsy the shunt was patent. Nineteen patients lived. The mean fall in portal pressure in the 20 patients was 165 mm of saline, from 415 to 250.

One difference between our population and Dr. Millikan's is that, out of six of the patients who survived and did well (that is, no further hemorrhages or clinical encephalopathy requiring hospital admission), three died of malignancies at 2, 5, and 7 years; two died of cerebrovascular accidents at 2 and 5 years, and one died from a coronary occlusion at 3½ years. I do not think a distal splenorenal shunt would have altered that group's survival. Two patients were lost to follow-up after 8 and 13 years.

If we include the six patients who died of conditions unrelated to the liver and bleeding, the mean follow-up is 9 years without hemorrhage, without clinical encephalopathy, except for one patient. Five of the patients are still alive and well at 6, 6, 7, 13, and 16 years.

Shunt closure occurred in three of my 10 patients. Two were technical and pathologic problems, and closed very quickly while they were still in the hospital. One patient closed 14 months later and came back with massive hemorrhage, which we could not control. An autopsy showed a fresh thrombus in the GoreTex graft at the junction of the superior mesenteric vein and the prosthesis.

If we exclude the six patients who died of cardiac and malignant disease, we are left with 14 patients with a mean survival of 13 years, including one postoperative death and one death from hemorrhage. Five of those 14 are alive and well as of August 1984.

Lastly, Dr. James Sarfeh from the University of California at Irvine is studying the small caliber shunt between the portal vein and the superior vena cava, using a 10-mm GoreTex® prosthesis. He advised me in August that he is trying smaller, 8-mm shunts. They remain angiographically patent and prograde flow to the liver is maintained in 50% of them.

In conclusion, I suggest that central venous shunts are alive and well.

DR. PAUL T. DECAMP (New Orleans, Louisiana): I cannot express my indebtedness to the Emory and the Miami groups for originally making a convert out of me, and I arise just to reemphasize something I think is becoming more and more apparent. From the very beginning, the problem in the surgical treatment of cirrhosis of the liver and portal hypertension has been distinguishing between the possible beneficial or the possible deleterious effects of the surgical procedure performed, as against the presumed or real progression of the liver disease. Our own experience with some 30-odd distal splenorenal shunts has been that it works very well and, if the liver is not continually insulted (either by a virus or by alcohol), the patients will do very well and just about equally well in the two groups.

I think the reason (if you lump all the postnecrotic cirrhotics) is that, in the patient cohorts seen in many institutions, fewer of the posthepatics will continue to have active viral infection, as against those who keep drinking in the alcoholic group.

My point is that I think it is very, very important to try to separate out what is happening to the patients whose livers are no longer being insulted, as against those who are. I think we will see increasingly the obvious difference.

DR. J. M. HENDERSON (Closing discussion): It is my privilege to close this paper, and I would like to take each of the discussants in turn.

In response to Dr. Zeppa, our nonalcoholic data and yours agree entirely: their survival is improved. Unlike you, we have not broken down survival by the variable of steroid therapy, but clinical impression would concur with your findings.

We have not demonstrated conclusively that age is a factor in incidence of encephalopathy. The real question is, what is the incidence of encephalopathy in these patients over 10 years, shunted or not shunted? Review of the total shunt trials against medical therapy showed a 20 to 25% incidence of encephalopathy in the medical therapy groups. The 55 patients in this study were good-risk patients; all were Child's A or B at randomization. The 25% incidence of encephalopathy in the selectively shunted patients is a reflection of progress of disease.

The maximum rate of urea synthesis showed early decrease in that quantitative function test in the total shunt patients, and maintenance in the selectively shunted patients. Late follow-up looks at survivors, and that difference between groups is lost as you look at the later survivors with that specific test. Quantitative function, measured by galactose elimination capacity at 10 years showed significantly better hepatocyte function in those with selective shunting who have maintained portal perfusion. There is no doubt in my mind that portal perfusion and hepatic function go hand in hand. If you can maintain portal perfusion, you can maintain hepatocyte function.

To Dr. Lord, a pioneer in this field, I would like to make the following points:

In respect to central splenorenal shunt, there are two areas of data that do not support its advantage over other total portal systemic shunts. First, Dr. Malt recently presented the MGH series of central splenorenal shunts and stated categorically that he could find no difference between those and end-to-side portacaval shunts. Both had similar encephalophathy and other morbidity. In addition, Silvano Raia, looking at the schistosomiasis population in Brazil, has documented a devastating encephalopathy rate in patients with central splenorenal shunts to be the same for other total shunts. He cut short his prospective randomized trial on that basis.

Regarding interposition shunts, Dr. Drapanas' data are not supported. The occlusion rate is high, as is the encephalophathy rate in those patients. Dr. Sarfeh's series of small interposition shunts is early in his experience. We need to wait and see what happens. I have great difficulty in believing you can provide just enough decompression through a small shunt to prevent the varices from bleeding, without decompressing the portal perfusion. To me that is untenable hemodynamically. I believe we will see one of two things, either shunt occlusion or encephalopathy will occur in those patients over time.

Your own series is an excellent small series, but relates to highly selected patients. With good-risk patients you have achieved good results.

To Dr. deCamp I would say, we welcome a convert. The balance of the deleterious effects of shunt *versus* the hepatocyte damage associated with natural history and progress of the underlying disease is important. Selective shunting provides excellent bleeding control. Maintenance of portal perfusion maintains hepatocyte function. The data from this trial shows that selective shunting does hold the advantage over the total portal decompression.