ANNALS OF SURGERY

Vol. 173 April 1971 No. 4

Hepatic Venous Angiography in the Evaluation of Cirrhosis of the Liver

GARDNER W. SMITH, M.D., TORBJØRN WESTGAARD, M.D., RAGNAR BJÖRN-HANSEN, M.D.

From the Departments of Surgery and Radiology, University of Virginia School of Medicine, Charlottesville, Virginia 22901

CIRRHOTIC alterations in the hepatic venous system were recognized over a century ago.[7] The anatomic significance of these changes has been repeatedly emphasized,[10, 11, 13, 19, 20] and the marked contraction of the hepatic venous bed in cirrhosis has been well documented.[4, 10, 13] The demonstrated physiologic importance of sinusoidal hypertension [32] has led to increasing acceptance of the concept of "outflow obstruction" as the basic pathophysiologic mechanism of portal hypertension in cirrhosis of the liver.

Hepatic venous angiography was first proposed by Rappaport in 1951,[22] based upon the technic of hepatic vein catheterization previously described by Warren and Brannon.[31] The clinical diagnostic possibilities of this procedure were recognized early by Tori [28] and by Celis and his associates,[5] although neither of these authors studied cirrhotic patients. Interest in the radiologic visualization of the hepatic veins has spawned a great variety of technics including the use of single and double balloon occlusion of the inferior vena cava,[15, 18, 23, 29] increased intrabronchial pressure to force contrast material into the hepatic veins,[17, 18] intravenous carbon dioxide injection,[6] and the direct percutaneous transhepatic introduction of contrast material.[16, 21] The wedged hepatic venous angiogram has also been described as a means of demonstrating changes in the hepatic veins [12, 24] and also as a method of visualizing the portal venous system.[9, 35, 37] However, despite the recognized importance of alterations in the hepatic veins in cirrhosis and the relatively widespread interest in the technics of hepatic venogra-

Submitted for publication August 13, 1970.

Supported by U. S. Public Health Service Grant HE-04644.

Presented at the 43rd Annual Meeting of the Halsted Society, Nashville, Tenn., September 11–13, 1969.

Reprint Requests: Department of Surgery, Baltimore City Hospitals, 4940 Eastern Avenue, Baltimore, Maryland 21224.

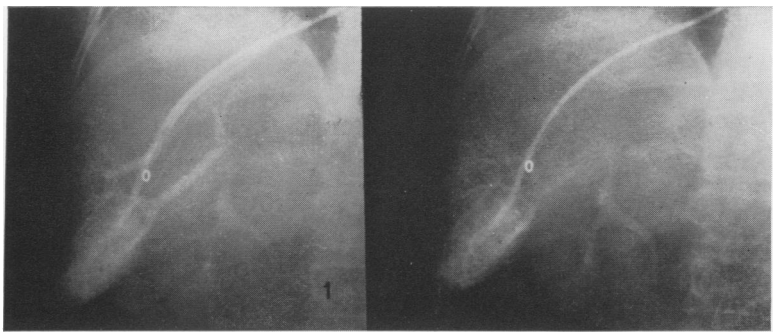

FIG. 1. Wedged hepatic venous angiogram. Early cirrhosis.

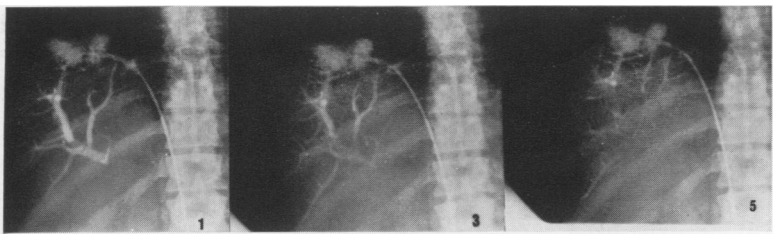

FIG. 2. Wedged hepatic venous angiogram showing marked portal vein filling.

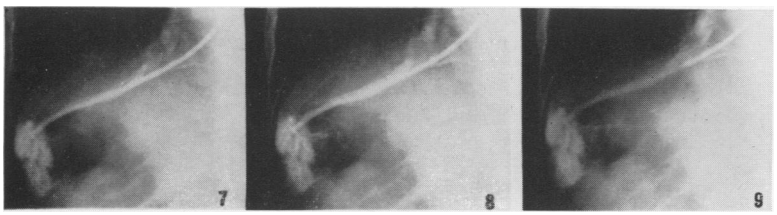

FIG. 3. Wedged hepatic venous angiogram showing reversed flow in the portal vein.

phy, only Britton and his colleagues have systematically evaluated the *in vivo* changes in the hepatic venous system associated with cirrhosis of the liver.[1, 2, 3]

During the hemodynamic assessment of patients with portal hypertension hepatic vein catheterization has been a routine procedure, and it soon became apparent that hepatic venous angiography was a useful additional study. We have performed 84 such angiograms in patients with portal hypertension secondary to cirrhosis of the liver. A large amount of additional information has been accumulated on these patients and programmed for computer analysis. It is proposed to describe the progressive alterations of the hepatic veins in cirrhosis, and then to correlate these anatomic changes with other clinical, physiological and biochemical parameters of the disease.

Methods

The technic of hepatic vein catheterization is essentially that of Taylor and Myers,[26] and the free hepatic venous angiograms are obtained in the fashion described by Tori.[28] A 100 cm. end-hole cardiac catheter, size 6F to 8F, is introduced into a suitable median basilic or brachial vein through a cut-down in the right antecubital fossa. This catheter is passed under fluoroscopic control through the right atrium to the inferior vena cava and thence into as many different sites in the right and left hepatic veins as can be entered. After obtaining appropriate pressures and blood samples, the catheter is placed in a wedged position, usually in a right hepatic vein, and this position is checked by pressure tracings and by injection of contrast medium. A wedged hepatic venous angiogram is then performed by injecting 10 ml. of 35 per cent Renovist by hand injection. Serial anteroposterior films are made with an Elema-Schonander rapid cassette changer at two per second for 5 seconds, starting when the injection is almost complete. The end-hole catheter is then replaced with an angiographic catheter with terminal side holes. This catheter is placed in a free posi-

tion, usually in a right hepatic vein, and 25 ml. of the same contrast material is injected at 650 p.s.i. with a pressure injector. Serial anteroposterior films are again obtained at two per second for 5 seconds, commencing at the start of the injection. In patients with severely truncated hepatic veins pressure injection causes dislodgement of the catheter, and in these cases the pressure is either reduced or the contrast material is injected by hand.

Results of Wedged Angiograms

Wedged hepatic venous angiograms were performed in 83 of these 84 studies. They were assessed primarily as to the presence and degree of portal vein filling, although it was possible to evaluate the extent and normality of sinusoidal filling and the occurrence of fine nodularity of the small hepatic veins. Figure 1 is an example of an early cirrhotic wedged angiogram, nearly normal, with a lobular pattern of sinusoidal filling, evidence of moderate opacification of the portal vein, and some fine nodularity of the hepatic venous radicles.

Portal vein filling was demonstrated in 20 instances, and was occasionally quite marked (Fig. 2). An attempt was made to correlate the extent of portal vein filling with the severity of the cirrhotic process. Among six phlebograms in patients with only fatty metamorphosis, the portal vein was visualized in four and was extensively opacified in one. On the other hand, whereas the portal vein was demonstrated nine times among 31 angiograms with moderately advanced disease, in six cases of far-advanced cirrhosis the portal vein was never visualized. Extensive portal vein filling occurred in three early cases and in no advanced cases. Reversal of flow in the portal vein was noted in only one patient in the entire group (Fig. 3), and this patient had moderately early cirrhosis.

Results of Free Angiograms

The major thrust of this investigation is the anatomic classification of the stages of cirrhosis of the liver on the basis of changes in the free hepatic venous angiogram. The normal hepatic venogram (Fig. 4) shows smoothly tapered hepatic veins with regular branches such that at least fifth order branching is demonstrated. The major trunks subdivide in an orderly progressive fashion peripherally, and there are additional small branches arising directly from the main veins. Under the conditions of pressure injection, sinusoidal filling is regularly observed as a delicate lacework surrounding the venules. No irregularities of the vein walls are noted. Such a normal free hepatic venous angiogram is classified as Stage I, and four fell in this category.

In the presence of cirrhosis the hepatic veins undergo progressive alterations commensurate with the degree of scarring and the extent of regeneration. In our interpretation of the venous angiogram four features of this process have been graded qualitatively on a 0 to 3+ basis: loss of

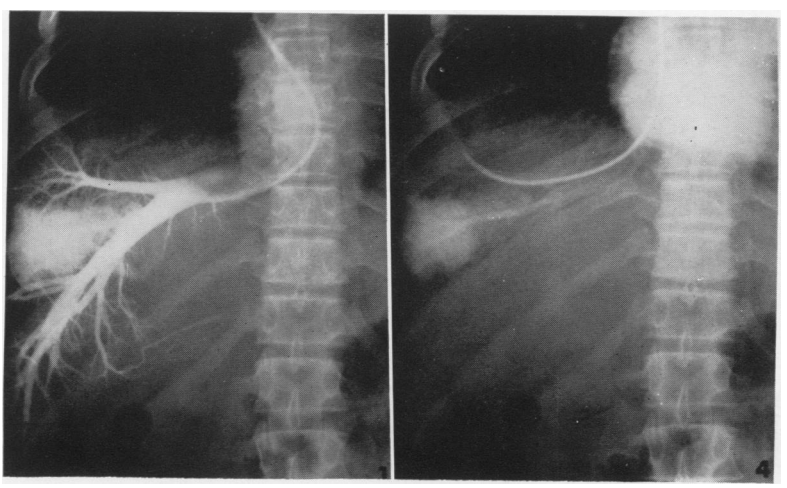

FIG. 4. Free hepatic venous angiogram. Normal. Stage I.

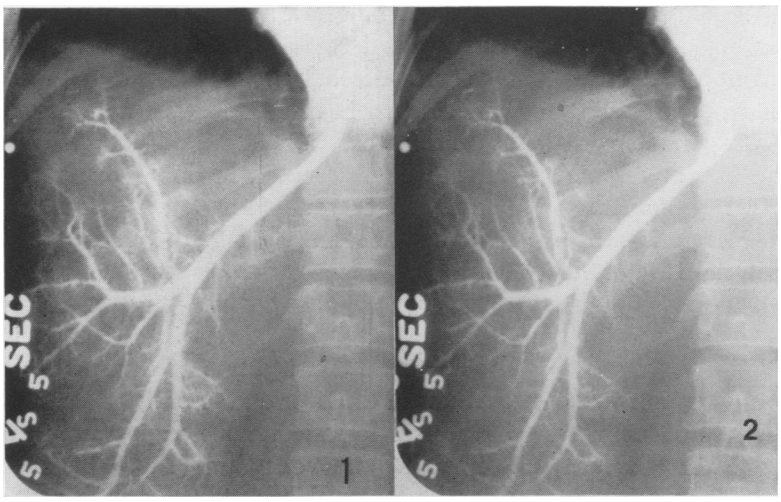

FIG. 5. Free hepatic venous angiogram. Stage II.

branching, loss of tapering, loss of sinusoidal filling and fine nodularity or scalloping of the small venous radicles. The loss of branching, tapering and sinusoidal filling is taken as evidence of progressive scarring, and the nodularity is indicative of hepatic regeneration. For every angiogram each of these characteristics has been graded, key-punched and transferred to magnetic tape for computer analysis. On the basis of increasing evidence of scarring and decreasing evidence of regeneration, it has been possible to distinguish four progressive stages of hepatic venous pathologic changes in cirrhosis.

Stage II angiograms (Fig. 5) show minimal loss of tapering and branching, sinusoidal filling is often normal, and the presence of fine nodularity varies from none to extensive. There were 33 venograms in this stage. In Stage III (Fig. 6) there is clear-cut loss of branching, tapering and sinusoidal filling with, again, variable degrees of nodularity. At Stage IV (Fig. 7) branching and tapering are severely curtailed, sinusoidal filling may still be present and

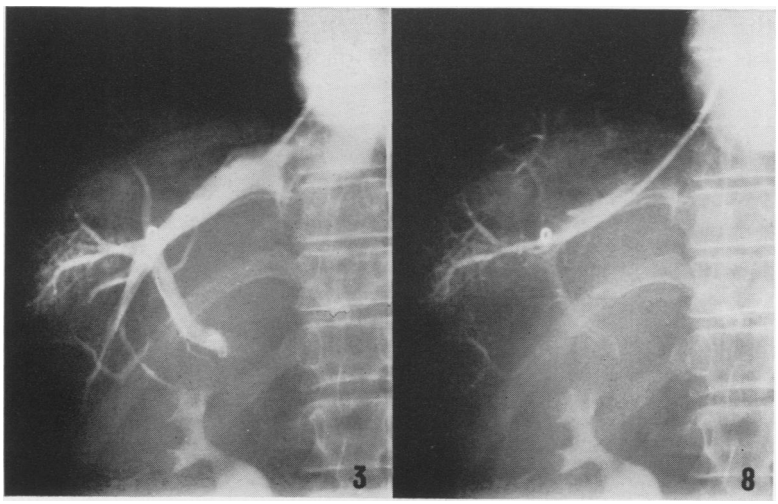

FIG. 6. Free hepatic venous angiogram. Stage III.

nodularity, if demonstrated, is minimal. Twenty angiograms fell into each of these two stages. Stage V (Fig. 8) is the end-stage with almost complete obliteration of the hepatic venous system, no sinusoidal filling and no nodular regeneration. This stage can be so far advanced that one wonders how hepatic venous flow is maintained at all (Fig. 9). There were seven phlebograms in this group.

In addition to the basic staging, several other features of the free hepatic venous angiogram were noted. Large, coarse indentations of the major venous trunks were occasionally observed and, when present, were associated with postnecrotic cirrhosis. On rare occasions hepatic venous collateral circulation was seen (Fig. 10). This usually occurs in relatively normal angiograms and is important primarily because it must be distinguished from portal vein filling. Even very early venous changes can be detected, since among six patients with fatty metamorphosis alone four of the angiograms were classified as Stage II and only two were normal. Finally, in eleven patients the creation of a side-to-side portocaval shunt made it possible to obtain simultaneous hepatic and portal venous angiograms by direct catheterization. The contrast between the two systems may be quite striking (Fig. 11), and confirms the widespread observation that cirrhosis causes severe alterations in the hepatic veins before significantly affecting the portal veins.

Angiographic Correlations

The anatomic changes in these angiograms are quite convincing, but it remains to correlate these changes with other features of the cirrhotic process. Because of a ready analogy to the Budd-Chiari syndrome, one might predict an association with the presence and severity of clinical ascites. In this series (Table 1) ascites was present in 51 per cent of Stage I and Stage II angiograms and only 30 per cent of those

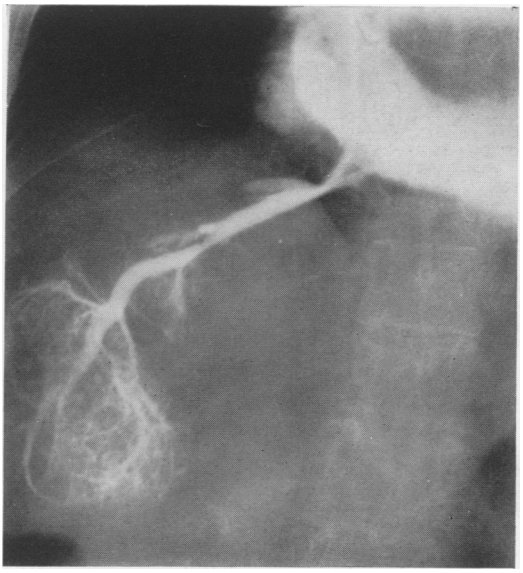

FIG. 7. Free hepatic venous angiogram. Stage IV.

classified as Stage IV and Stage V. Furthermore, it was severe in 24 per cent of the former and only 4 per cent of the latter. These differences are statistically significant. However, hepatic venous obstruction by regenerating nodules is a feature of this problem since among 39 patients with ascites 13 had 2+ or 3+ nodularity and only four had none, whereas in 45 patients with no ascites only eight had greater than 1+ nodularity and 16 had none.

The other commonly accepted clinical manifestations of severe cirrhosis are esophageal varices, encephalopathy, a shrunken scarred liver and jaundice. The presence of varices was assessed by x-ray of barium swallow, splenoportogram and esophagoscopy (Table 2) and their severity was determined by the splenoportogram. It appears that there is no difference among the stages in the severity of the varices, but that they are perhaps more frequently absent in the early stages. Encephalopathy was gauged by impaired mentation, asterixis and coma (Table 3). In late-stage patients it was present in 60 per cent and severe in 19 per cent, whereas in the early

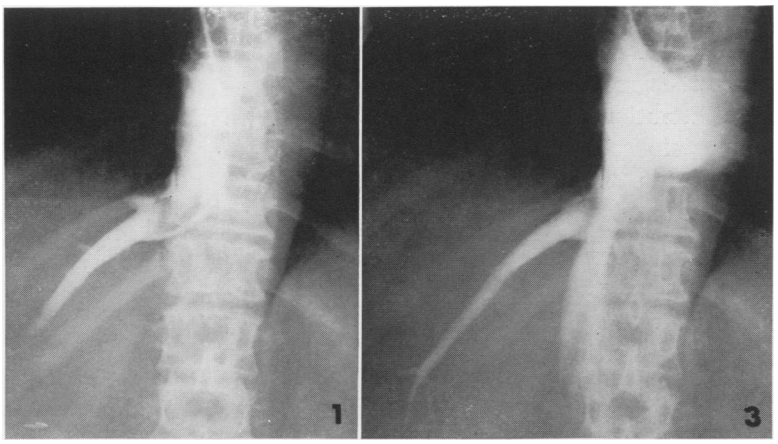

FIG. 8. Free hepatic venous angiogram. Stage V.

stages it was present in 38 per cent and severe in only eight per cent. These differences are statistically significant. Size of the liver was estimated by physical examination and by hepatic scan (Table 4). While there is overlap between the mildly enlarged, normal and mildly shrunken livers, marked enlargement is considerably more common in the early stages and severe scarring is confined to Stages IV and V. Jaundice was estimated clinically and measured as total serum bilirubin (Table 5). On clinical grounds there are no real differences, and indeed two severe cases occurred in early stages in patients with acute alcoholic hepatitis. Total serum bilirubin appears to be more frequently and markedly elevated in the late stages, but even these differences are not statistically significant.

Among biochemical parameters, the prothrombin time, alkaline phosphatase and serum albumin have been correlated with the angiographic stages. Prothrombin time was normal in 59 per cent of the early stages and in only 30 per cent of the late stages, with a mean prothrombin time of 12.9 ± 0.4 seconds in Stage I and 15.6 ± 0.7 seconds in Stage V. Alkaline phosphatase showed less marked differences, being normal in 41 per cent of Stages I and II and in 22 per cent of Stages IV and V. The mean values were essentially the same in both groups. Serum albumin was less than 3.0 Gm./100 ml. in 36 per cent of the early cases and in 48 per cent of the late ones. The mean serum albumin was 3.3 ± 0.5 Gm./100 ml. in Stage I as compared with 2.8 ± 0.2 Gm./100 ml. in Stage V.

Hemodynamic parameters were evaluated, including splenic pulp pressure, wedged hepatic vein pressure, free portal

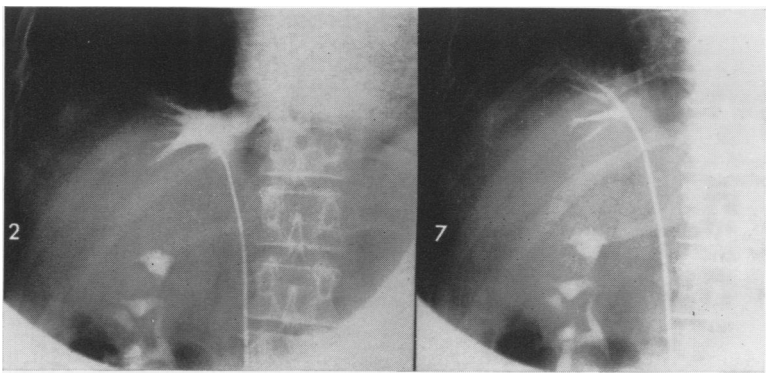

FIG. 9. Free hepatic venous angiogram. Stage V. Very far advanced.

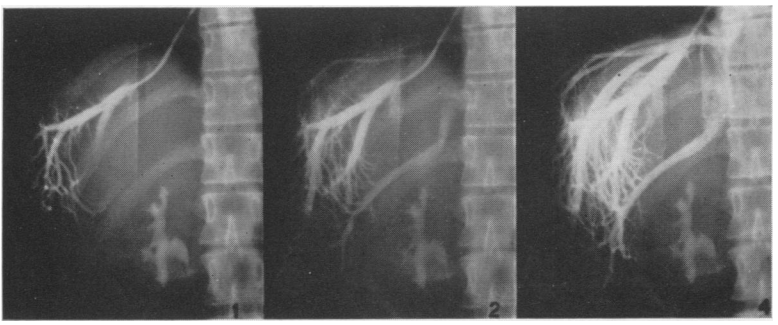

FIG. 10. Free hepatic venous angiogram showing hepatic venous collateral circulation.

pressure, maximum perfusion pressure, measured portal blood flow and estimated total liver blood flow. No correlation was found between the angiographic stages and splenic pulp pressure, wedged hepatic vein pressure or free portal pressure. Maximum perfusion pressure fell from a mean of 294 mm. saline in Stages I and II to 169 mm. saline in Stages IV and V. There were no differences among the stages in measured portal blood flow. Total hepatic blood flow, as estimated by the radioactive colloidal gold technic, was significantly depressed in the late stages (Table 6). The mean estimated hepatic blood flow in Stage I was 17.5 ± 2.8 per cent as compared with a mean of 9.7 ± 1.7 per cent in Stage V.

Finally the prognosis in early and mild cirrhosis should certainly be better than it is in the later stages, both as regards surgical survival and as regards the appearance of late complications of the cirrhotic process. The survival among 24 patients undergoing either portacaval shunt or splenectomy and coronary vein ligation has been compared in Table 7, and it appears that the operative mortality is greater in the late stages with, in fact, a 50 per cent mortality in Stage IV. No patients in Stage V were considered suitable surgical candidates. Fourteen of the 16 surgical survivors have been re-evaluated and their follow-up (Table 8) reveals a greater incidence of encephalopathy, jaundice and late death in the advanced stages, although the incidence of hemorrhage and ascites was the same in all groups. Twenty-four patients who were managed medically had subsequent re-evaluation (Table 9). Among them the late mortality was greater in the late stages, but the occurrence of encephalopathy, hemorrhage, jaundice and ascites was roughly equal at all stages. Asterixis and coma, however, occurred exclusively in Stages IV and V.

Discussion

Our results with the wedged hepatic venous angiograms are somewhat at variance with those reported by others. There is disagreement as to whether portal vein filling

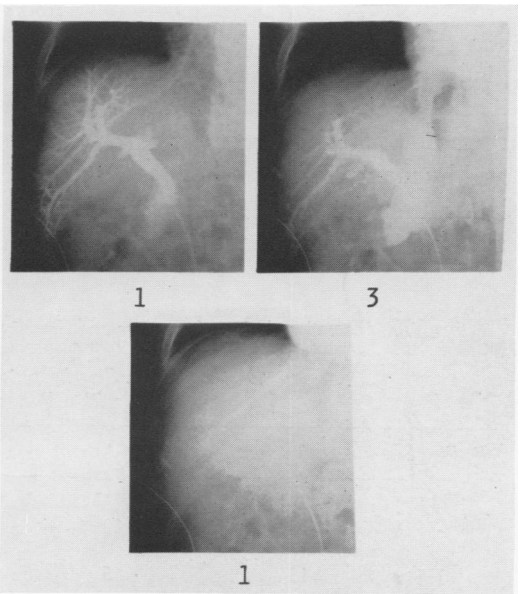

FIG. 11. Portal venous angiogram (Top) and hepatic venous angiogram (Bottom) showing contrast between these two venous systems in cirrhosis.

TABLE 1. *Correlation of Presence and Severity of Clinical Ascites with the Angiographic Stages*

Stage	None	Mild	Moderate	Severe
I	4	0	0	0
II	14 } 49%	6 } 16%	4 } 11%	9 } 24%
III	8	1	6	5
IV	12	1	6	1
V	7 } 70%	0 } 4%	0 } 22%	0 } 4%

is more prominent in patients with normal livers or in the presence of severe cirrhosis.[2, 5, 9, 12, 23, 24, 25, 34, 35] Since portal to hepatic venous shunts have been demonstrated in this disease,[10, 14, 20] it seems reasonable to predict that portal venous filling will be more marked as the cirrhotic process progresses. Indeed Warren and his associates have staged lesions on this basis.[34] However, in our experience the portal vein was visualized most frequently among patients with early disease, and it was never seen either in patients with normal livers or in those with far-advanced cirrhosis.

It has further been stated that reversal of flow in the portal vein can be demonstrated in the wedged hepatic venous angiogram.[34, 35] If reversal of flow is present, it should be demonstrable at the time of operation both by direct flow measurement and by maximum perfusion pressure.[33] This latter pressure is the difference between mesenteric occluded and hepatic occluded portal pressure, and represents the pressure head driving portal blood into the liver. Maximum perfusion pressure should be negative if portal blood flow is reversed. Yet in our one patient with reversal of flow in the angiogram a hepatopetal portal blood flow of 480 ml./min. was measured at operation, and the maximum perfusion pressure was 240 mm. of saline. Two other patients did have a negative maximum perfusion pressure, and one of these had reversed portal flow by direct measurement as well, yet neither had reversal of flow demonstrated in the wedged venogram, and one of them had no portal vein filling whatsoever in two preoperative angiograms. Finally eleven patients had wedged hepatic venograms after side-to-side portacaval shunts. Such a procedure probably invariably results in reversal of flow in the hepatic limb of the portal vein,[36] yet none of these patients demonstrated such reversal in postoperative phlebograms. From this experience we must conclude that the demonstration of reversed portal vein flow in the wedged hepatic venous angiogram is a rare phenomenon of dubious significance.

The anatomic features of the free hepatic venous angiogram have been well described in the past. Others have also identified loss of branching, tapering and sinusoidal filling with progressive scarring,[1, 2, 8, 23, 24, 30] and nodularity with hepatic regeneration.[1, 11] Although the details vary, both

TABLE 2. *Correlation of presence and Severity of Esophageal Varices with the Angiographic Stages, Presence Determined by Any Combination of Barium Swallow, Splenoportogram and Esophagoscopy. Severity Determined by Splenoportogram. Patients with a Portacaval Shunt Excluded*

Stage	Present	Absent	Mild	Moderate	Severe
I	2	1	0	1	0
II	16 } 58%	12 } 42%	1 } 7%	7 } 57%	5 } 36%
III	13	5	5	4	4
IV	11	5	1	5	3
V	1 } 67%	1 } 33%	— } 11%	— } 56%	— } 33%

Britton [2] and Doehner [8] staged the changes in the hepatic venogram and found, as we did, that these stages correlate with the severity of the cirrhotic process. There is some disagreement as to the early phases, since we found angiographic changes in the presence of fatty metamorphosis alone, as did Hales,[10] but others could demonstrate no venographic changes in this early stage of the disease.[8, 14] We concur that a definitive venographic distinction cannot be made between nutritional and postnecrotic cirrhosis,[10, 19] but large course nodules, when present, signify the postnecrotic type.[12, 14] There is general agreement that major anatomic aberrations occur in the hepatic veins before there are significant changes in the portal veins.[8, 10, 19, 24]

Clinical, biochemical and hemodynamic correlations with these angiographic changes have been attempted in the past with variable results. Classically the anatomic aberrations have been compared to the Budd-Chiari syndrome and thus associated with clinical ascites.[2, 3, 8, 13] On the other hand Teague *et al.* have shown that ascites is actually characteristic of early cirrhosis with mild vascular alterations,[27] and Britton found that this clinical feature of cirrhosis is associated either with early or with far-advanced phlebographic changes.[2, 3] Others have stated that changes in the hepatic venous angiogram do not correlate with ascites at all.[10, 19] In our experience both the occurrence and severity of ascites showed a statistically positive correlation with the early angiographic stages and with the presence of regenerating nodules.

TABLE 3. *Correlation of Presence and Severity of Encephalopathy with the Angiographic Stages*

Stage	Absent	Mild	Severe
I	4	0	0
II	19 } 62%	11 } 30%	3 } 8%
III	10	7	3
IV	8	8	4
V	3 } 40%	3 } 41%	1 } 19%

There have been previous attempts to compare the clinical and biochemical parameters of cirrhosis with changes in the hepatic venous angiogram, with sometimes conflicting results. Piper [19] found no correlation between the radiologic changes and esophageal varices or serum albumin. Britton *et al.*[2] and Doehner [8] noted low serum albumin in the late stages, and the latter also found decreased liver size with progressive angiographic phases. He concurred that the presence and severity of esophageal varices could not be correlated with these phases, nor could he find any relationship to serum bilirubin or alkaline phosphatase. We agree that there is no correlation between the angiographic stages and varices, clinical jaundice, serum bilirubin or alkaline phosphatase, and that there is a positive correlation with hypoalbuminemia and with a small liver. We have also found that the progressive phle-

TABLE 4. *Correlation of Liver Size with the Angiographic Stages. Size Estimated by Physical Examination and by Liver Scan*

Stage	Markedly Enlarged	Enlarged	Normal	Shrunken	Markedly Shrunken
I	1	2	0	1	0
II	7 } 22%	13 } 40%	10 } 27%	3 } 11%	0 } 0%
III	4	10	6	0	0
IV	2	11	3	3	1
V	0 } 7%	2 } 48%	2 } 18.5%	2 } 18.5%	1 } 7%

TABLE 5. *Correlation of Clinical Jaundice and of Total Serum Bilirubin with the Angiographic Stages*

Stage	None	Clinical: Mild	Clinical: Moderate	Clinical: Severe
I	3	0	0	1
II	23 } 70%	7 } 19%	2 } 6%	1 } 5%
III	12	7	0	1
IV	13	4	3	0
V	3 } 59%	2 } 22%	2 } 19%	0 } 0%
		Total Bilirubin (mg./100 ml.): <1.5	1.5–3.0	>3.0
I		3	0	1
II		20 } 62%	7 } 19%	6 } 19%
III		14	3	3
IV		10	6	4
V		2 } 44%	1 } 26%	4 } 30%

bographic stages bear a positive relationship to the incidence of encephalopathy and to an elevated prothrombin time.

Britton made some hemodynamic comparisons,[2, 3] noting that free portal pressure does not correlate with the stage of hepatic venous disease, and that hepatic occluded portal pressure does. Since with progressive outflow block there is also progressive development of portal collateral circulation, no correlation would be expected between the angiographic stages and portal pressure, and none was found. This was the case whether portal pressure was measured as free portal pressure, as splenic pulp pressure or as wedged hepatic vein pressure. However, deterioration in the hepatic venous outflow system should result in diminished portal blood flow. Britton's results with hepatic occluded portal pressure and ours with maximum perfusion pressure tend to confirm this. We could not prove it, however, by measured portal blood flow at operation, perhaps because so few values were available. On the other hand, there is a definite correlation between diminished estimated total liver blood flow and progressive hepatic venous obstruction.

No previous attempts have been made to assess prognosis relative to the hepatic venous angiogram. The findings of this investigation suggest that the angiographic

TABLE 6. *Correlation of Estimated Total Liver Blood Flow with the Angiographic Stages. Figures Represent Per Cent of Total Blood Volume Perfusing the Liver per Minute*

Stage	<12%	12%–20%	>20%
I	1	2	1
II	8 } 24%	17 } 52%	8 } 24%
III	6	9	5
IV	5	13	2
V	5 } 37%	2 } 56%	0 } 7%

TABLE 7. *Correlation of Surgical Survival with the Angiographic Stages*

Stage	Survived	Operative Mortality
I	1	0
II	5	2 (29%)
III	7	3 (30%)
IV	3	3 (50%)
V	0	0

changes can be correlated with operative mortality and with long-term surgical survival, as well as with the late survival of medically treated patients. Progressive phlebographic changes also show a relationship to the incidence of severe encephalopathy in both medical and surgical patients, and to jaundice in the latter group.

Summary and Conclusions

The technic of hepatic venous angiography, both wedged and free, is a relatively simple one which can be accomplished easily at the time of hepatic vein catheterization. In this series of 84 cirrhotic patients there were no complications from the procedure. The changes noted in the wedged angiograms assist in the over-all interpretation of the anatomic alterations in the hepatic veins, and portal vein filling is frequently demonstrated. However, the presence and extent of portal vein filling does not reflect the severity of the cirrhosis, and the demonstration of reversed flow in the portal vein by this technic is a rare phenomenon of dubious significance. The staged progression of hepatic venous pathologic changes observed in the free angiograms is anatomically quite convincing. These anatomic changes show a positive correlation with progressive cirrhosis as assessed by the incidence of encephalopathy, a shrunken liver, elevated prothrombin time, hypoalbuminemia, a diminished maximum perfusion pressure, estimated total hepatic blood flow, and both surgical and medical prognosis. The pressure and severity of ascites is positively correlated with early anatomic changes. There is no apparent correlation of the angiographic stages with the presence of esophageal varices or clinical jaundice or with serum bilirubin, alkaline phosphatase, portal pressure and measured portal blood flow. It is concluded that evaluation of the hepatic venous angiogram does assist in assessing the stage of the cirrhotic process and is of value in predicting both surgical risk and long-term prognosis.

TABLE 8. *Correlation of Surgical Follow-up Results with the Angiographic Stages*

Stage	No. of Patients	Average Duration (mos.)	Encephalopathy	Hemorrhage	Jaundice	Ascites	Death
I	1	1	0 } 20%	0 } 40%	0 } 20%	0 } 20%	0 } 20%
II	4	10	1 }	2 }	1 }	1 }	1 }
III	6	15	2	0	2	0	0
IV	3	19	2 (67%)	1 (33%)	1 (33%)	1 (33%)	2 (67%)
V	0	0	0	0	0	0	0

TABLE 9. *Correlation of Medical Follow-up Results with the Angiographic Stages*

Stage	No. of Patients	Average Duration (mos.)	Encephalopathy	Hemorrhage	Jaundice	Ascites	Death
I	2	24	1 } 60%	0 } 30%	0 } 20%	1 } 50%	0 } 30%
II	8	17	5 }	3 }	2 }	4 }	3 }
III	7	17	2	4	2	4	3
IV	4	17	2 } 43%	2 } 29%	1 } 29%	3 } 57%	3 } 57%
V	3	26	1 }	0 }	1 }	1 }	1 }

References

1. Britton, R. C. and Shirey, E. K.: Cineportography and Dynamics of Portal Flow Following Shunt Procedures. Arch. Surg., **84**:25, 1962.
2. Britton, R. C., Brown, C. H. and Shirey, E. K.: Intrahepatic Veno-Occlusive Disease in Cirrhosis with Chronic Ascites. Diagnosis by Hepatic Phlebography and Results of Surgical Treatment. Ann. Surg., **158**:370, 1963.
3. Britton, R. C.: The Hepatic Outflow Tract and Ascites: Angiographic and Cinefluorographic Studies. Amer. J. Gastroent., **44**:443, 1965.
4. Carter, J. H., Welch, C. S. and Barron, R. E.: Changes in the Hepatic Blood Vessels in Cirrhosis of the Liver. Surg. Gynec. Obstet., **113**:133, 1961.
5. Celis, A., Villalobos, M. E., Del Castillo, H. and Espinosa, J. F.: Roentgenographic Opacity of the Hepatic Circulation. Amer. J. Roentgen., **74**:1089, 1955.
6. Collins, J. R. and Vix, V. A.: Hepatic Vein Visualization by Intravenous Carbon Dioxide Injection. Radiology, **89**:864, 1967.
7. Frerichs, F. T.: A Clinical Treatise on Diseases of the Liver. Murchison, C. (Trans.), The New Sydenham Society, London, **2**:28, 1861.
8. Doehner, G. A.: The Hepatic Venous System. Its Normal Roentgen Anatomy. Its Pathologic Roentgen Anatomy. Radiology, **90**: 1119, 1968.
9. Greenspan, R. H., Capps, J. H., Widmann, W. D. and Hales, M. R.: Transhepatic Portal Venography: A New Method of Portal Vein Visualization. Radiology, **78**:248, 1962.
10. Hales, M. R., Allan, J. S. and Hall, E. M.: Injection-Corrosion Studies of Normal and Cirrhotic Livers. Amer. J. Path., **35**:909, 1959.
11. Ketty, R. H., Baggenstoss, A. H. and Butt, H. R.: The Relation of the Regenerated Liver Nodule to the Vascular Bed in Cirrhosis. Gastroenterology, **15**:285, 1950.
12. Kreel, L., Freston, J. W. and Clain, D.: Vascular Radiology in the Budd-Chiari Syndrome. Brit. J. Radiol., **40**:755, 1967.
13. Madden, J. L., Lóre, J. M., Jr., Gerold, F. P. and Ravid, J. M.: The Pathogenesis of Ascites and a Consideration of Its Treatment. Surg. Gynec. Obstet., **99**:385, 1954.
14. Mann, J. D., Wakim, K. G. and Baggenstoss, A. H.: Alterations in the Vasculature of the Diseased Liver. Gastroenterology, **25**:540, 1953.
15. McCaughan, J. S.: Retrograde Visualization of the Hepatic Veins in Dogs. Surgery, **53**:352, 1963.
16. Moreno, A. H., Rousselot, L. M., Burchell, A. R., Bono, R. F. and Burke, J. H.: Studies on the Outflow Tracts of the Liver. I. On a Method For the Functional Demonstration of the Outflow Tracts of the Liver and Its Application to the Study of Hepatic Hemodynamics in Normal and Cirrhotic Rats. Ann. Surg., **155**:412, 1962.
17. Nogueira, C. E. D.: Panhepatography: Simultaneous Radiographic Visualization of All Hepatic Veins. Amer. J. Digest. Dis., **6**:772, 1961.
18. Norhagen, A.: Selective Angiography of the Hepatic Veins. Acta Radiol. Suppl., 221, 1963.
19. Piper, D. W.: A Radiographic Study of the Portal and Hepatic Venous Systems in Cirrhosis of the Liver. Amer. J. Digest. Dis., **6**:499, 1961.
20. Popper, H., Elias, H. and Petty, D. E.: Vascular Pattern of the Cirrhotic Liver. Amer J. Clin. Path., **22**:717, 1952.
21. Ramsey, G. C. and Britton, R. C.: Intraparenchymal Angiography in the Diagnosis of Hepatic Veno-Occlusive Diseases. Radiology, **90**:716, 1968.
22. Rappaport, A. M.: Hepatic Venography. Acta Radiol., **36**:165, 1951.
23. Rappaport, A. M., Holmes, B., Stolberg, H. O., McIntyre, J. L. and Baird, R. J.: Hepatic Venography. Gastroenterology, **46**:115, 1964.
24. Schlant, R. C., Galambos, J. T., Shuford, W. H., Rawls, W. J. Winter, T. S. and Edwards, F. K.: The Clinical Usefulness of Wedged Hepatic Venography. Amer. J. Med., **35**:343, 1963.
25. Sherlock, S. and Sheldon, S.: In Vascular Roentgenology by Schobinger, R. A. and Ruzicka, F. F., pps. 567–571, New York, MacMillan, 1964.
26. Taylor, W. J. and Myers, J. D.: Occlusive Hepatic Venous Catheterization in the Study of the Normal Liver, Cirrhosis of the Liver and Noncirrhotic Portal Hypertension. Circulation, **13**:368, 1956.
27. Teague, F. B., Jr., Warren, W. D. and Respess, J. C.: Vascular Physiology in Portal Hypertension with Ascites. Clinical and Experimental Studies and Role of Portacaval Shunt. Ann. Surg., **163**:112, 1966.
28. Tori, G.: Hepatic Venography in Man. Acta Radiol., **39**:89, 1953.
29. Tori, G. and Scott, W. G.: Experimental Method for Visualization of the Hepatic Vein—Venous Hepatography. Amer. J. Roentgen., **70**:242, 1953.
30. Tori, G.: In Vascular Roentgenology by Schobinger, R. A. and Ruzicka, F. F., pps. 564–566, New York, MacMillan, 1964.
31. Warren, J. V. and Brannon, E. S.: A Method for Obtaining Blood Samples Directly From the Hepatic Vein in Man. Proc. Soc. Exper. Biol. & Med., **55**:144, 1944.
32. Warren, W. D. and Muller, W. H., Jr.: A Clarification of Some Hemodynamic Changes in Cirrhosis and Their Surgical Significance. Ann. Surg., **150**:413, 1959.
33. Warren, W. D. and Restrepo, J. E., Respess, J. C. and Muller, W. H., Jr.: The Importance of Hemodynamic Studies in Management of Portal Hypertension. Ann. Surg., **158**:387, 1963.
34. Warren, W. D., Fomon, J. J., Viamonte, M. and Zeppa, R.: The Preoperative Assessment of Portal Hypertension. Ann. Surg., **165**:999, 1967.
35. Warren, W. D., Fomon, J. J., Viamonte, M., Martinez, L. O. and Kalser, M.: Spontaneous Reversal of Portal Venous Blood Flow in Cirrhosis. Surg. Gynec. Obstet., **126**:315, 1968.
36. Warren, W. D. and Fomon, J. J.: Reflections on Post-Portacaval Shunt Morbidity. J. Cardiovasc. Surg., **9**:453, 1968.
37. Widmann, W. D., Greenspan, R. H., Hales, M. R. and Capps, J. H.: A New Method for Portal Venography: Retrograde Hepatic Flushing. Proc. Soc. Exper. Biol. Med., **106**:540, 1961.