Hemodynamic Guidelines for Surgical Therapy of Portal Hypertension

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A prospective study of 98 patients with portal hypertension who hemorrhaged revealed that certain hemodynamic parameters were valuable in confirming the cause of cirrhosis, aiding in the selection of patients best suited for a selective distal splenorenal shunt, and in providing an estimate of prognosis. The presence of a pressure gradient of 4 mmHg or more between the right atrium and inferior vena cava was observed only in patients with alcoholic cirrhosis. The shape of the "pull-back" tracing between the wedge and free hepatic vein positions was "smooth" in postnecrotic disease and "lumpy" in alcoholic disease. The ratio of the aortic diastolic pressure divided by the hepatic (vein) wedge pressure segregated patients by cause and direction of portal blood flow.

TWO PREVIOUSLY REPORTED angiographic observations in patients with cirrhosis are an abnormal wedge hepatic venogram and an elevation of the wedge hepatic venous pressure (HWP).[11,12] In 100 liver panangiograms performed at the authors' institution, prior to the start of the present investigation, the wedge hepatic venogram categories, determined by the method of Viamonte.[12] were compared with the portal flow patterns demonstrated by indirect portography. They did not correlate with each other nor did the hepatic wedge venograms correlate with the clinical severity of each patient's cirrhosis. In some patients, the catheterized segments of liver were more severely cirrhotic than adjacent segments, and the resulting venogram did not correctly reflect the overall extent of disease throughout the liver.[4]

Similarly, higher HWP might be expected in patients with poorer clinical states; however, this was not supported by the authors' retrospective review of the cases before this study.

A rational choice for a surgical portosystemic shunt depends on an accurate evaluation of the predominant portal flow direction; therefore, one cannot rely on wedge hepatic venography, since it is easily perturbed by segmental influences, nor upon the HWP alone, since such a wide spectrum of portal hypertensive patients have nearly the same HWP. For these reasons, the authors sought another approach which might be more helpful.

One of the authors originally noticed that the aortic diastolic pressure (AoD) was usually approximately twice the HWP in most cirrhotic patients studied by panangiographic examination . This prospective study was designed with the hope that this relationship, as well as other hemodynamic parameters might be used to reliably and reproducibly gauge the direction of the portal flow. These could then provide a more dependable guide to the type of surgical therapy for each patient. Subsequently, an anatomic basis for the author's observations was sought.

Materials and Methods

Between September 1977 and April 1979, 98 patients with portal hypertension had complete prospective studies with surgical and pathologic proof of the cause of cirrhosis. The patients ranged in age from 21 to 80 years, equally divided between males and females. Fifty-five per cent of the patients were in the age range of 35–55 years. In order to perform a double blind assessment of the results, for each case, one of the authors reviewed the clinical records without knowledge of the angiographic–hemodynamic data, in order to obtain in-

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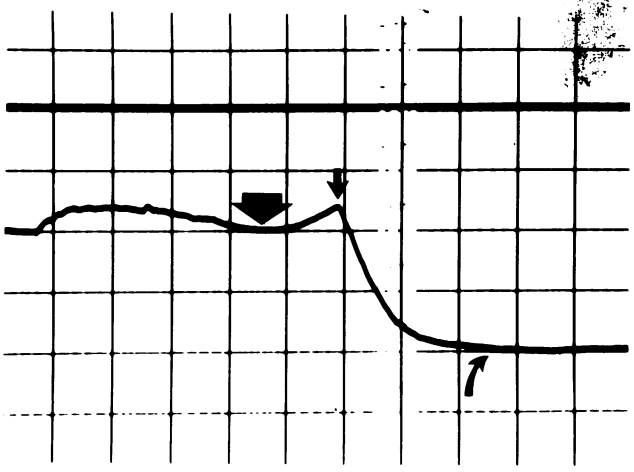

FIG. 1. In the wedge position, a pressure of 30 mmHg is recorded (wide arrow). The venous catheter is gently "pulled back" about 3 cm in a time interval of approximately 0.25 to 0.5 seconds, causing the dislodgement artifact (small arrow); there is then a smooth transition to the free hepatic vein pressure of 10 mmHg (curved arrow). The smoothness typifies postnecrotic cirrhosis pressure measurements. (Each horizontal line represents 10 mmHg with the wide line equal to 50 mmHg; each vertical is 1 second.)

formation concerning each patient's history of alcoholic use, exposure to persons having hepatitis or jaundice, and past medical history with regard to blood transfusions, documented hepatitis or other liver diseases. The chart reviews indicated that by standard clinical criteria, 63 patients had evidence of alcoholic cirrhosis, 28 had nonalcoholic cirrhosis (predominantly postnecrotic due to prior hepatitis in this series), and seven had presinusoidal causes for portal hypertension. When subsequent pathologic proof was obtained, 64 alcoholic patients and 26 nonalcoholic patients were identified, with eight having presinusoidal portal hypertension. Only one of the patients (with postnecrotic cirrhosis) had not yet hemorrhaged from endoscopically verified esophagogastric varices. Because the patient had been referred from a remote part of South America, it was felt clinically justified to evaluate his disease prior to his return home.

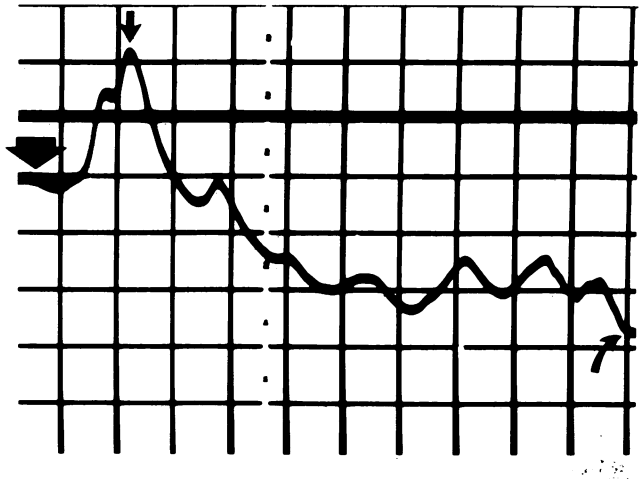

FIG. 2. In the wedge position, the tracing reveals a pressure of 40 mmHg (wide arrow). The venous catheter is gently "pulled back" about 3 cm in a time interval of approximately 0.25 to 0.5 seconds, causing the dislodgement artifact (small arrow); there are then several peaks and valleys recorded before the "free" hepatic vein pressure of 12 mm (curved arrow) is recorded. (It must be noticed that the catheter is not touched after dislodgement.) This shape is typical of the "alcoholic" pull-back tracing. (The scale is the same as in Figure 1, where the wide horizontal line is 50 mmHg and each vertical line is 1 second.)

All of these patients were referred for hepatic panangiographic examination, in order to determine their suitability for a selective distal splenorenal shunt. The referring surgical group[18] adhered to the preoperative protocol for patient management as described by Zeppa.[16,17]

A liver panangiographic examination was performed by the attending or house-staff who were unfamiliar with the particular patients' hospital chart data. This procedure involved measurement of pressures with the venous catheter (#7 French, end-hole) in the intrahepatic portion of the inferior vena cava (IVC), in the right atrium (RA), free within each of two right lobe segmental hepatic veins and wedged into each of two hepatic veins (HWP). After pressure measurement, in the wedged position, contrast medium was injected at a standard rate to evaluate retrograde filling of portal venules radiographically. The catheter was subsequently "pulled-back" from the wedge to the free position while a paper tracing of the pressure transition was made (Figs. 1 and 2). The venous catheter was finally moved to the left renal vein for pressure measurement and venography to document its position.

The arterial catheter was placed in the celiac (or splenic) and superior mesenteric artery, respectively, after systolic and diastolic pressure measurements in the abdominal aorta. A large volume of contrast material (75 ml) was injected into each vessel to opacify the portal venous system. This is the technique of indirect portography, by which the degree of filling of the portal vein from the splenic and superior mesenteric veins can be evaluated and the major portosystemic collaterals are shown. It is imperative to notice the anatomic positions of the splenic vein and left renal vein prior to consideration of a selective distal splenorenal shunt.[15]

All pressures were documented on an Electronics-for-Medicine® (DR-8) Monitor with a strip-chart recorder, from the output of a Statham P-231-D strain-gauge transducer connected to the angiographic catheters. The patient was in the supine position during the entire procedure. During the 32 months in which the patients were studied, two attending staff and ten house staff

physicians performed the panangiographic procedures.

Direction of portal blood flow was assessed as follows:

1) If portal venous radicles were opacified following contrast injection into both the celiac and superior mesenteric arteries, flow was considered hepatopetal.
2) If the portal vein was not opacified during the venous phase of either the celiac or superior mesenteric artery injections, flow was considered hepatofugal.
3) If the superior mesenteric artery injection opacified the portal venules, but the venous phase of the celiac artery injection did not, flow was considered easily reversible.

At some point during the hospitalization of the 98 patients in this study, 14 patients required transhepatic portographic examination for possible therapeutic embolization of the veins feeding acutely bleeding gastroesophageal varices. Most often, this was performed as an emergency procedure prior to elective panangiographic examination. During transhepatic portography, portal pressures were measured and the direction of the portal flow was assessed before and after obliteration of the varices.

The following three parameters were tabulated for each of the patients: 1) Shape of the "pull-back" tracing. 2) Measurements of RA and IVC pressures. 3) Calculation of the ratio between the aortic diastolic pressure (AoD) and the HWP $\left(\frac{\text{AoD}}{\text{HWP}}\right)$.

Results

Table 1 shows that the eight patients with presinusoidal causes of portal hypertension had a much lower HWP than did the cirrhotics.[8] The average hepatic (vein) wedge pressures were, however, virtually the same for alcoholic and nonalcoholic (postnecrotic) patients with cirrhosis.

Table 2 indicates that the lowest blood pressures were found in the nonalcoholic group of patients; the alcoholic patients and those with presinusoidal portal hypertension had comparable arterial pressures. Individual blood pressures did not segregate into ranges of values characteristic of a particular origin of portal hypertension.

The average of the ratio between the aortic diastolic pressure and the hepatic wedge pressure is shown in Table 3. All the patients with presinusoidal disease had $\frac{\text{AoD}}{\text{HWP}}$ ratios above 2.7; more importantly, the alcoholic patients' ratios averaged 2.3 if they had hepatopetal (prograde) portal flow; if hepatofugal (retrograde) flow had developed, the average ratio was 1.4. The postnecrotic patients with cirrhosis had a similar segregation, with average values of 1.6 for hepatopetal flow and 1.2 for hepatofugal flow. Seven of the postnecrotic patients had ratios above our expectations; of these, five had identifiable causes for the inapplicability of our criteria (Table 3). Therefore, two of the 26 postnecrotic cases are "errors" of the system; no exceptions were found for patients having presinusoidal portal hypertension nor for those with alcoholic cirrhosis.

After it was established that the mean right atrial pressures in all the patients studied were within normal limits (Table 4), measurements of pressure gradients between the right atrium and the intrahepatic portion of the inferior vena cava were tabulated; the average values are shown in Table 4. None of the alcoholic patients had less than a 4 mmHg gradient, while the other two groups all had less than 4 mmHg gradient, without exceptions. The final key result of this study was the recognition of a distinctly different shape of the "pull-back" tracing between the alcoholic and postnecrotic cirrhotics. Figure1 shows the smooth transition representative of all of the nonalcoholic patients with cirrhosis. Figure 2 illustrates the "peaks and valleys" that were representative of cases of alcoholic liver disease. Both tracings were made at the same amplitude and time scale settings and are, therefore, visually comparable. Incidental notice was made that the "pull-back" tracing was smooth, although from a lower wedge pressure in patients with presinusoidal portal hypertension.

Review of the patients' charts shows that 11 of the patients had clinically detectable ascites at the time of panangiographic examination. The hemoglobin level, drawn within 48 hours prior to each panangiographic examination, in patients who had not recently hemorrhaged nor received blood transfusions, was nearly always 10 g/100 ml (range: ±1 g). The average of the total protein level for the patients was 6.6 g/100 ml, which is in the normal range; the mean serum albumin values were 3.8 g/dl for the nonalcoholics (within normal limits), and 3.1 g/dl for the alcoholics, which is below normal. The alcoholic patients' mean alkaline phosphatase value was 127 units/L, while for the nonalcoholics it was 151 units/L (normal range: 30–80 units/L). The average SGOT level for the nonalcoholic patients, 92 units/L, was above the normal range (7–40), while the alcoholics' average was 27 units/L. Because the selection bias imposed on this study was toward stable, elective surgical candidates, patients having clinical criteria for alcoholic hepatitis were excluded from the series, as were those who were unstable due to active gastrointestinal hemorrhage or shock. The criteria necessary for application of the hemodynamic evaluation are: 1) At least one documented upper gas-

TABLE 1. *Average Hepatic Wedge Pressures, n = 98*

Cause	Portal Flow Pattern	Number	HWP Avg., mmHg	Standard Deviation, mmHg
Alcoholic	hepatopetal	35	29	±5.2
	stagnant	14	33	±3.4
	hepatofugal	15	40	±7.0
		64		
Postnecrotic	hepatopetal* (with high ratios)	7	30	±4.2
	hepatopetal	10	35	±3.8
	stagnant	6	40	±7.0
	hepatofugal	3	36	±4.7
		26		
Presinusoidal	hepatopetal	8	14	±3.6

* Patients with $\frac{AoD}{HWP}$ ratios inconsistent with our expectations.

No essential difference existed in the average hepatic wedge pressure between the two types of cirrhotics. The presinusoidal group averaged 14 mm Hg, which is significantly less than the average for cirrhotics. It is important to observe that in this series as well as in others,[8] presinusoidal causes for portal hypertension do elevate the HWP slightly above normal.

trointestinal hemorrhage from gastric or esophageal varices. 2) Absence of shock or congestive heart failure at the time when pressure measurements are made. 3) No use of drugs that alter arterial or portal pressure if possible (including vasopressin). 4) Essential hypertension absent prior to developing portal hypertension. 5) Veno-venous shunts not seen at wedge hepatic venography (the measured HWP is not a reliable guide to portal pressure in this situation).

Discussion

The purpose of this study has been to attempt to identify hemodynamic criteria which would be of value in the selection of patients with portal hypertension who are best suited for the distal splenorenal shunt. These criteria may be of greatest value in providing an objective parameter to assess the direction of portal flow, and may be helpful to confirm the cause of cirrhosis. Careful review of over 100 cases prior to beginning this study has confirmed that the hemodynamic parameters previously studied have not been adequate for these purposes. Measurement of an elevated HWP indicates only that cirrhosis is the most likely cause for portal hypertension, but does not help to distinguish patients with antegrade or retrograde portal flow. The calculation of a "corrected sinusoidal pressure" by subtracting the inferior vena caval pressure from the HWP has been similarly disappointing. These values have not been of

TABLE 2. *Average Abdominal Aortic Systolic and Diastolic Pressures, n = 98*

Cause	Portal Flow Pattern	Number	Systolic/Diastolic mmHg	Standard Deviation Systolic/Diastolic, mmHg
Alcoholic	hepatopetal	35	118/65	±21/10
	stagnant	14	116/59	±15/6
	hepatofugal	15	110/57	±25/9
		64		
Postnecrotic	hepatopetal* (with high ratios)	7	98/55	±19/9
	hepatopetal	10	115/57	±17/6
	stagnant	6	88/56	±23/22
	hepatofugal	3	72/43	±22/5
		26		
Presinusoidal	hepatopetal	8	104/63	±18/14

* The group of patients whose $\frac{AoD}{HWP}$ ratios are above the predicted range.

Although the aortic pressures are slightly lower in the postnecrotic groups compared with the other two groups, the differences are not significant. Individual postnecrotic cases frequently overlapped the alcoholic blood pressure ranges.

TABLE 3. *Average of Ratio Between the Aortic Diastolic Pressure (AoD) and the Hepatic Wedge Pressure (HWP)*

Cause	Portal Flow Pattern	Number	Ratio Range $\frac{AoD}{HWP}$	Average	Standard Deviation
Alcoholic	hepatopetal	35	2.6-2.0	2.3	±0.19
	stagnant	14	1.9-1.7	1.8	±0.07
	hepatofugal	15	1.6-1.1	1.4	±0.13
		64			
Postnecrotic	hepatopetal* (with high ratios)	7	1.9-1.7	1.9	±0.07
	hepatopetal	10	1.7-1.5	1.6	±0.08
	stagnant	6	1.4	1.4	±0
	hepatofugal	3	1.3-1.0	1.2	±0.17
		26			
Presinusoidal	hepatopetal	8	6.0-2.7	4.9	±1.2

* Patients whose ratios were above our expected range had the other hemodynamic criteria of postnecrotic disease, *i.e*, no pressure gradient between RA and IVC and a "smooth" pull-back pressure tracing. Likewise, alcoholic patients (by history) whose portal flow was stagnant or hepatofugal, with $\frac{AoD}{HWP}$ ratios that overlapped the postnecrotic range, retained an RA-IVC pressure gradient and a "lumpy" pull-back tracing, which characterized them hemodynamically as having alcoholic cirrhosis. It should be noticed that of the seven patients with ratios above our predictions, three had radiographically identifiable hepatic veno-venous shunts, leading to a falsely low HWP; two had been "essential hypertensives" before liver disease developed, thus having an abnormally elevated aortic pressure coexisting with end-stage cirrhosis.

any predictive value in anticipating the "results of operations for portal hypertension . . ."[5] Likewise, venography performed with a catheter in the hepatic wedge position has been unreliable in defining the predominant flow pattern in the portal vein, when compared with the results of both preoperative angiography and intraoperative flow direction determination.

Zeppa has emphasized that if "portal perfusion to the liver was demonstrable in the patient with cirrhosis,"[17] a distal splenorenal shunt is preferred. Preservation of antegrade portal perfusion has customarily been visually determined by examining the venous phase films of a superior mesenteric angiogram. Three variable factors limit the adequacy of this method. It has been observed that when portal flow is readily reversible, indirect portographic examination with the patient in the supine position may show hepatofugal flow; a repeat examination with elevation of the left side of the patient during filming ("the right posterior oblique" projection) or after injection of a vasodilator such as tolazoline into the superior mesenteric artery prior to angiographic examination may show hepatopetal portal flow. It becomes uncertain as to which flow pattern is the "correct" one. Observer error can be significant in precisely those cases where opacification of portal venules is weak. To obviate the conflicting results obtained when indirect portography is performed in different positions, and with or without vasodilator augmentation, our objective hemodynamic data were always obtained prior to injection of radiographic contrast material or other drugs, with the patient in the supine position. Therefore our approach to evaluation of direction of portal flow was consistent from case to case, as well as between the same patients studied before and after operation. Despite the fact that the 98 liver panangiographic examinations in this series were performed by two attending radiologists and ten radiology house staff, during the 32 months of the study, the results were consistent throughout. Therefore it is unlikely that the characterstic shapes of the "pull-back" tracings or the absolute values of the arterial or venous pressures were due to an artifact of technique of any individual.

Experience at this institution with 65 cases of percutaneous transhepatic portography has been for interventional purposes. Prior to therapeutic obliteration of the left gastric and short gastric veins in patients acutely bleeding from varices, each patient underwent indirect portographic examination prior to puncture of the liver. Those who were subsequently stabilized had complete panangiograms with hemodynamic data, and are included in this series. There was an excellent correlation of the direction of portal blood flow noticed at transhepatic portographic examination with the hemodynamic predictions made at panangiographic examination. As noticed earlier, there was very little correlation with wedge hepatic venogram categories. The absolute values of the hepatic wedge pressure and the aortic pressures by themselves had no relationship to portal blood flow direction. There are some[13] who feel the transhepatic approach to the portal system is suitable for purely diagnostic purposes in clinically stable patients, in order to obtain reliable information about portal blood flow, pressure, as well as the size and location of portosystemic collaterals. Since this method carries considerably higher risk for the patient than does

TABLE 4. *Average Mean Right Atrial Pressures 2nd Average Gradient Between Intrahepatic Inferior Vena Cava 2nd Right Atrium*

	Alcoholics	Postnecrotics	Presinusoidal Disease
Average mean right atrial (RA) pressures (mmHg)	6.0	6.4	4.7
Average gradient between intrahepatic inferior vena cava and right atrium (mmHg)	4.9	2.7	1.7

This table indicates that there is no difference in right atrial (RA) pressures in the three groups. The table also indicates that the alcoholics had the largest RA-IVC gradient which in no case was below 4 mmHg. The patients with presinusoidal disease had normal values.

indirect portography with recording of hemodynamic parameters, it is felt that for purely diagnostic purposes, the transhepatic technique is not appropriate. The major risks of intraperitoneal hemorrhage, in a group of patients whose coagulation profiles are usually not completely normal, and iatrogenic thrombosis of the portal vein, particularly in patients whose portal flow pattern is stagnant, need not be a consideration when consistent panangiographic data can be obtained with greater safety. It should be noticed that 19 patients in our study had portal flow patterns described hemodynamically and angiographically as stagnant or reversible. Therefore, a significant portion of patients with cirrhosis are at risk from direct catheterization of the portal vein.

In order to understand the relationship between pressure phenomenona which were recorded and their implications about portal flow, it is helpful to study the microvascular pathologic factors of cirrhosis. Lunderquist and Koolpe[7] injected colored plastic solutions selectively into hepatic venules, portal venules and hepatic arteries of autopsy specimen livers. In addition to the well known obstruction of hepatic venules that occurs by progressive fibrosis in cirrhosis, this study clearly showed that the microangiopathy involved progressively larger "connections . . . between tortuous hepatic and portal vein branches."[7] When sinusoidal destruction was most severe, these anastomoses were largest. It was noticed that these anastomoses "are widened sinusoidal fragments included in the fibrotic septa and are responsible for the transmission of the portal venous pressure to the hepatic veins."[7,10] It is the progressive enlargement of hepatic arterioportal anastomoses which contributes significantly to elevation of portal pressure; ultimately, the hepatic arterial contribution to the progressively destroyed sinusoid overwhelms the capacity of hepatic venules and the portal vein becomes an outflow pathway.[9]

The study noted above[7] involved several patients from this series who died during our research project, and it validates the concept that a catheter in the wedge hepatic vein position accurately reflects portal pressure, and that portal pressure is closely related to the size of arterioportal anastomoses. We noticed that the wedge hepatic venous pressure tracings were often phasic, *i.e.* they had regular peaks which followed the "QRS" complex of the electrocardiogram and dips which corresponded to the "P" waves. This was, in effect, a highly damped arterial pressure tracing seen most clearly in patients whose portal flow had become retrograde. It is intriguing to notice that in the three patients having retrograde portal flow, in whom oxygen saturation was measured in a wedge hepatic vein blood sample, that the oxygen saturation was very nearly that of arterial blood. While the number of these determinations is not significant, these observations are congruent with the suggestions of Popper,[9] that the arterioportal shunts are transmitting both oxygenated blood and systolic pressure waves to the portal venules through the fibrous septae. This pathoanatomic evidence corroborates our clinical impression that the relationship (*i.e.* ratio) between two pressures (HWP, AoD), not their absolute values, more accurately reflects the severity of sinusoidal destruction.

The objectivity of the hemodynamic ratio $\left(\frac{\text{AoD}}{\text{HWP}}\right)$ offers a consistent standard of reference for evaluating the direction of portal flow, comparable from case to case as well as between examinations before and after operation. As anatomic studies suggest, portal blood flow direction is a continuous spectrum involving individual segments of the liver to varying degrees. Many authors[11,12,15] have evaluated the direction of portal flow radiographically, by means of splenoportography, transhepatic portography, or indirect (arterial) portography. Hepatopetal portal flow is assumed if portosystemic collateral vessels are less opacified than the intrahepatic portal radicals; when the collateral vessels seem more densely opacified, flow to the liver is presumed to be either stagnant or reversed. The spectrum of portal dynamics cannot be appreciated by these crude observations.

In Table 3 a small difference in the $\frac{\text{AoD}}{\text{HWP}}$ ratio separates patients with stagnant and hepatofugal flow, for example, in the postnecrotic group of patients. Nineteen patients in our study had stagnant or easily reversible portal flow; vasodilator augmentation of the superior mesenteric angiography, or positioning the patient supine for an initial run and oblique for a subsequent mesenteric angiogram proves the lability of portal flow (Figs. 3a and b). This is a postoperative examination in which the supine mesenteric angiogram failed to demonstrate intrahepatic portal perfusion, with pref-

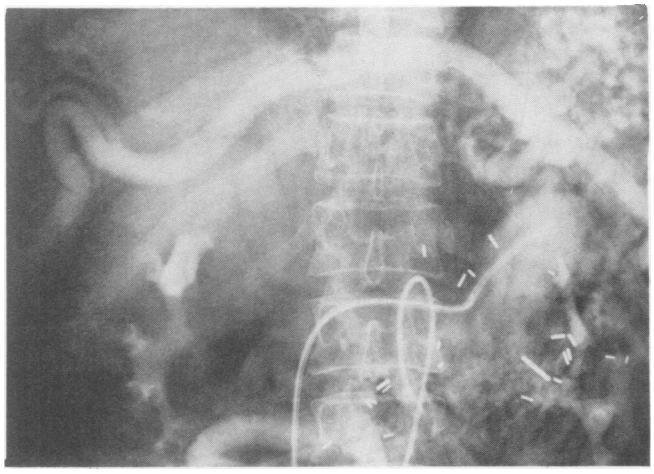

FIG. 3a. This patient developed portosystemic encephalopathy one week after a selective distal splenorenal shunt. Preoperative angiographic-hemodynamic studies revealed hepatopetal flow with an $\frac{AoD}{HWP}$ ratio of 1.6. Indirect portography following contrast injection in the superior mesenteric artery now shows hepatofugal flow with the patient supine. This accounts for the development of encephalopathy. His $\frac{AoD}{HWP}$ ratio is now 1.4, suggesting reversibility of portal flow.

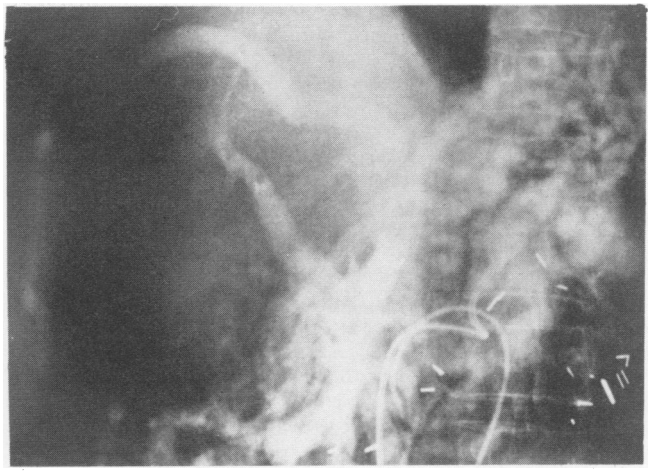

FIG. 3b. A second indirect portographic examination, performed with the patient in the right posterior oblique projection, reveals hepatopetal flow to some segments of the liver.[4] This confirms the reversibility of flow, as predicted by the $\frac{AoD}{HWP}$ ratio. Subsequently, the large collateral running along the edge of the mesentery was surgically ligated, and the encephalopathy improved.

erential filling of a large omental collateral which had not been ligated at the time the distal splenorenal shunt was performed. This patient had proven postnecrotic cirrhosis and his preoperative ratio was 1.6 (prograde flow); the $\frac{AoD}{HWP}$ ratio at the time of the second examination was 1.4, strongly suggesting reversibility of portal flow, despite the initial pictorial evidence to the contrary (Fig. 3a). This was confirmed by repeating the indirect portographic examination with the patient in the right posterior oblique projection (Fig. 3b). It is important to notice that some hepatic segments are now filled with contrast while others remained unopacified, again revealing the unevenness of hepatic destruction in different segments of the liver. The indication for postoperative angiographic examination in this case was the development of portosystemic encephalopathy. The distal shunt was angiographically patent, with no pressure gradient between the splenic vein and left renal vein; therefore, the surgical team required evidence that hepatopetal flow could be restored if the large collateral vein seen at the periphery of the radiograph were to be ligated. It is not the purpose of this paper to discuss the extensive role of spontaneous portal-systemic collaterals, which are a significant factor in the clinical aspects of portal hypertension. However, it is well known that selective obliteration of a single, large collateral vessel may increase mesenteric portal pressure sufficiently to convert stagnent flow to hepatopetal flow. The large collateral was subsequently ligated at surgery and the patient's encephalopathy improved rapidly thereafter, in accordance with the hemodynamically predicted result. The angiographic reversibility of portal flow was due to the small increase in portal vein pressure caused by the hydrostatic column of blood in the mesenteric bed, which had been raised above the level of the portal vein by positioning the patient with the left side elevated. It is speculative that had the postoperatve $\frac{AoD}{HWP}$ ratio been lower, in the hepatofugal range for postnecrotic patients, there could not have been restoration of hepatopetal flow with any angiographic manipulation. The implication, therefore, is that ligation of a large collateral would not have been beneficial given a lower $\frac{AoD}{HWP}$ ratio.

Two additional examples illustrate the value of recording hemodynamic data as an essential part of preoperative evaluation of portal hypertensive patients. One of the patients whose chart review suggested postnecrotic cirrhosis had an $\frac{AoD}{HWP}$ ratio of 2.5, a "lumpy" pull-back tracing, and a 5 mmHg gradient between the right atrium and intrahepatic inferior vena cava. The patient freely admitted an extensive history of alcohol abuse when he was told that certain laboratory results were not entirely characteristic of nonalcoholic cirrhosis. The unreliability of the history in patients with alcoholic disease is well known, and it is helpful to know that hemodynamic criteria may be a valuable adjunct

TABLE 5. *Cause of Portal Hypertension: Comparison of Clinical, Hemodynamic and Pathologic Classification*

	Chart Review and Patient History	Panangiography and Hemodynamic Results	Biopsy Results
Alcoholic	63	66	64
Nonalcoholic	28	24	26
Presinusoidal	7	8	8

Two cases having postnecrotic cirrhosis were incorrectly classified by hemodynamic parameters as having alcoholic liver disease. The 24 patients we classified as nonalcoholics, all were confirmed; the 64 surgically proven alcoholic patients were correctly predicted, and included within the 66 patients labeled hemodynamically as alcoholic. The eight proven presinusoidal patients were correctly identified.

Numbers indicate per cent.

to a carefully taken history in estimating long-term prognosis and complications.

Another patient whose clinical data suggested postnecrotic cirrhosis, had our hemodynamic criteria for presinusoidal portal hypertension, with an HWP of only 15 mmHg, a smooth "pull-back" tracing and no gradient between the right atrium and intrahepatic inferior vena cava. At surgery, a liver biopsy specimen was obtained, and histologic confirmation of our impression of an essentially normal liver was also obtained. The patient's portal hypertension was felt to be related to his massive splenomegaly, and he was treated by splenectomy rather than portosystemic shunting, with an excellent postoperative course.

One of the important phenomenona which has heretofore not been satisfactorily evaluated prior to decompressive surgery in patients having portal hypertension is an objective, reproducible parameter which can serve as a guide to the degree of portal venous perfusion of the diffusely diseased liver. Hepatopetal portal flow, with other clinically determinable criteria such as absence of ascites, lack of hepatocellular necrosis and inflammation, and absence of cardiovascular instability from acute gastrointestinal hemorrhage[1,17] permit consideration of the selective distal splenorenal shunt. The theoretic and clinical advantages of this technically more difficult shunt have been reported[1,14,16] in the properly selected patient. The flaws in previously used methods have subjected patients with partially or completely retrograde portal flow to the surgical risks of the operation but without its benefits. On the other hand, patients whose angiograms did not show antegrade portal perfusion received a nonselective (central) shunt. In those patients at our institution who were considered nonalcoholic, survival after the selective shunt was far better than reported survivals following a portacaval shunt.[16] It is hoped that a still better prognosis can be anticipated with the more accurate evaluation of portal flow direction suggested in this paper.

TABLE 6. *Incidence of Errors of Hemodynamic Classification of Cause*

Pathologic Cause	True Positives	False Positives	False Negatives
Alcoholic	64/64	2/64 (3.1%)	0/64
Nonalcoholic	24/26	0/26	2/26 (7.7%)

See legend to Table 5.

As alluded to earlier, a significantly better six year survival rate has been seen from this institution in nonalcoholic patients with cirrhosis, (89% ± 6% SE),[16] particularly for those having hepatopetal flow. Alcoholic patients with cirrhosis have only a 39% ± 14% SE six-year expected survival rate,[16] with the selective distal splenorenal shunt. Since patients may not reveal their alcohol abuse, the availability of hemodynamic parameters which we have found highly reliable for distinguishing alcoholic and nonalcoholic cirrhosis should be of interest to all those having sufficient clinical experience with portal hypertensive patients to have been misled by hemodynamic or angiographic parameters previously used. The incorrect categorization of alcoholic patients in the nonalcoholic category will alter the cohort statistics, suggesting a poorer prognosis for the correctly classified patients than should be expected. LaMont, Koff, and Isselbacher[6] note, in a current textbook of medicine, that "unexpected and unexplained cirrhosis accounts for about 10% of chronic liver disease at autopsy examination. Equally important, many patients with cirrhosis are "classified" improperly during life, and small and unrepresentative biopsy specimens from patients with a poor history often account for the errors in diagnosis."

The empiric observations of a pressure gradient of greater than 4 mmHg between the right atrium and intrahepatic inferior vena cava, and a "lumpy" pull-back tracing between the wedge and free hepatic vein positions have no anatomic explanation to date. Venacavagrams have failed to predict those patients in whom the pressure gradient should be found. Therefore, simplistic explanations such as caudate lobe encroachment are not satisfactory. Likewise, free hepatic vein contrast injections show no consistent alterations in the configuration of segmental hepatic veins adequate to explain the numerous intermediate pressure values recorded between the wedge and free positions in alcoholic patients. The longer time required for the pressure tracings in the alcoholic liver to reach the free value is consistent between examiners and patients, reproducible, and as of this time, lacking in an explanation. In those few patients who were restudied postoperatively, the presence or absence of an RA to IVC pressure gra-

dient and the characteristic shape of the pull-back tracing were unchanged; post operative changes in portal flow dynamics were accurately reflected in changes in the $\frac{AoD}{HWP}$ ratio.

An abbreviated statistical evaluation was performed because of the small number of patients in each subgroup. The clinical predictive value of the hemodynamic measurements compared well with surgical and pathlogic results, with 96 cases interpreted correctly out of 98 studied. Two patients with proven nonalcoholic cirrhosis were incorrectly labeled as alcoholic patients (Tables 5 and 6).

Determination of the gradient between the right atrium and inferior vena cava, notation of the shape of the pull-back tracings from the wedged to the free position and calculation of the $\frac{AoD}{HWP}$ ratio have been helpful in our laboratory in determining portal flow dynamics, and in confirming the origin of portal hypertension; follow-up examinations of these patients will provide definitive data on their prognosis and on the suitability of the type of surgical shunt performed. It is hoped that this will become a more valuable guide to clinicians dealing with patients having portal hypertension as more hard data becomes available.

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