Preoperative Assessment of Portal Hypertension

W. Dean Warren, M.D., John J. Fomon, M.D.,
Manuel Viamonte, M.D., Robert Zeppa, M.D.

From the Departments of Surgery and Radiology, University of Miami School of Medicine and Jackson Memorial Hospital, Miami, Florida

Bleeding from esophageal varices can almost always be controlled by portacaval shunt but many patients are saved from hemorrhage to die subsequently of liver failure. Of those who survive a significant number endure the morbidity of chronic encephalopathy.[3, 4, 9] No single criterion can be used in selection of patients for nonoperative or operative therapy or in determining the type of operation to be performed. Three categories of assessment are useful in selection of patients. These are 1) clinical evaluation, 2) laboratory evaluation of hepatic function, and 3) appraisal of hemodynamic alterations. Based on previous work it was postulated that abrupt changes in hepatic portal blood flow profoundly influence the prognosis for patients with portal hypertension.[10, 11] This premise augments and extends rather than replaces evaluations predicated on clinical and laboratory data. It seeks reasons for the well-being of one group of patients and the morbidity of another following portacaval shunt so that the result of treatment can be predicted more accurately.

Methods

Technics used to study hepatic and splanchnic vascular physiology were: estimation of total hepatic blood flow, radioisotope liver scan, hepatic vein catheterization, splenoportography or indirect portography, visceral angiography and in some instances umbilical vein catheterization:

1. Estimated Hepatic Blood Flow (EHBF). Thirty microcuries of Au^{198} are injected through a needle into an unobstructed antecubital vein and the disappearance rate is monitored over the temple using a $2 \times 2''$ sodium iodide, thalium activated crystal as the scintillation probe. The probe is attached to a ratemeter * which in turn is connected to a strip recorder.** The rate of removal of Au^{198} from the blood is assumed to be constant; that is, the amount removed is proportional to the amount in the blood at a given time. The disappearance curve represents the amount of radioactive colloidal gold in the blood at given times following injection. The height of the curve in millimeters is measured at specified times, one, two, two and a half, three, three and a half and four minute intervals (Fig. 1). These values are plotted on semilogarithmic paper and a straight line is drawn to fit the points. The one-minute point is regarded as the "mixing" period and ignored. The line is extrapolated to time zero giving N_0, the N value at time zero, expressed in millimeters. The time at which the N value is one half of N_0 is $t\frac{1}{2}$. $T\frac{1}{2}$ is the value used to estimate k, the relative rate of decrease of N, that is,

Presented at the Annual Meeting of the Southern Surgical Association, December 6–8, 1966, Boca Raton, Florida.

This work was partially supported by the U. S. Public Health Service, Grant No. HE 08411-04 and No. 1 MO 1 FR00251 with the Clinical Research Facility of the University of Miami School of Medicine, Miami, Florida.

* Nuclear-Chicago Ratemeter Model 1620B.

** Texas Instrument Recti-Riter Strip Recorder Model No. RRMA-T-2.

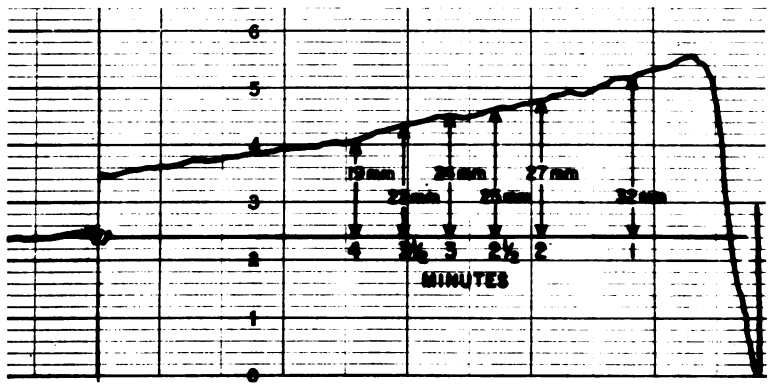

FIG. 1. Disappearance curve of Radioactive Colloidal Gold. With the recorder running at a speed of three quarters of an inch each minute it usually takes 20 minutes for the curve to reach a baseline. The baseline is extended to the point of injection. The height of the curve at the various time intervals is measured and it is these values that are plotted on semilogarithmic paper.

the rate of decrease per unit time per unit N. Mathematically the relationship is expressed:

$$\frac{N}{N_0} = e - kt$$

(e is the base of the natural or Napierian Logarithm, a number approximately equal to 2.718).

When $N = \frac{1}{2}N_0$, $t = (t\frac{1}{2})$, commonly known as the half-life or period of time during which half the Au^{198} is expected to be removed from the blood. Then, the above equation becomes:

$$\frac{1}{2} = e - K(t\frac{1}{2})$$

Taking the natural logarithm of both sides,

$$\ln(\frac{1}{2}) = -K(t\frac{1}{2})$$
$$-0.693 = -K(t\frac{1}{2})$$

therefore

$$K = 0.693/t\frac{1}{2}$$

The K value is obtained by dividing the constant 0.693 by $t\frac{1}{2}$ and represents the fraction of extrasplanchnic blood volume perfusing the liver per minute.[7]

2. **Liver-Spleen Scan.** An average dose of 150 μc of radioactive colloidal gold is injected into a peripheral vein. Thirty minutes later the abdomen is scanned with a 5″ Picker Magna Scanner.

3. **Hepatic Vein Catheterization.** A No. 6 or No. 7 open-tip catheter without side holes is introduced into the basilic vein in the antecubital fossa. Using continuous electrocardiographic and fluoroscopic television monitoring the catheter is advanced through the right side of the heart and into the inferior vena cava where it is positioned in an hepatic vein. The catheter is passed to the wedged position and a pressure tracing is recorded. An abrupt change in pressure or in the character of the wave form or both assures the operator that the catheter is wedged. The catheter is then withdrawn into the free hepatic vein while continuously recording pressure. This procedure is repeated several times, usually in at least two hepatic veins.[6] The pressure in the free hepatic vein (FHV) is subtracted from the wedged hepatic vein pressure (WHV) to give the corrected sinusoidal pressure. Once more the catheter is wedged and a *wedged hepatic venogram* obtained; 12 ml. of 75% Hypaque are injected at a rate of 2 ml. per second. Two films are exposed each second for the first three seconds and one each second for the following six seconds.

4. **Splenoportography.** Under local anesthesia a small stab wound is made in the left posterior axillary line in the ninth interspace. While the patient holds his breath a polyethylene catheter is threaded over a spinal needle and introduced into the splenic pulp and the needle is removed. The catheter is left in place and respirations are resumed. Following aspiration of

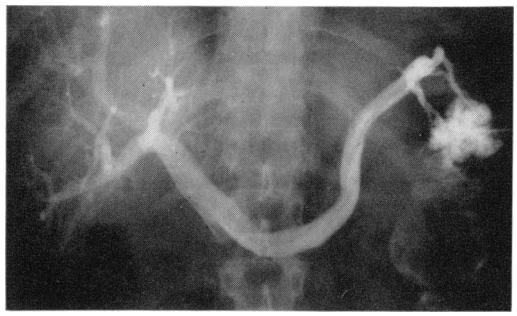

FIG. 2a. Hepatopetal portal flow was excellent with filling of vessels to the periphery of the liver in both patients. Late films showed dense hepatograms. Collateral vessels are absent.

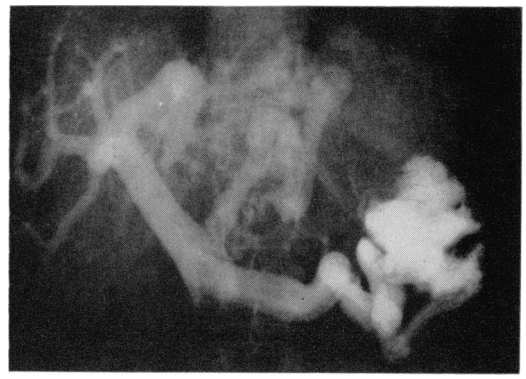

FIG. 2b. A large coronary vein forms large varices in the gastroesophageal area, though portal flow to the liver was good.

blood from the catheter a test dose of contrast material is injected and observed on the fluoroscopic television screen. Once the catheter is well positioned 50 ml. of 75% Hypaque are injected at a rate of 10 ml. per second. X-ray exposures are made at a rate of two each second for three seconds and then one each second for 14 seconds.

5. Visceral Arteriography and Indirect Portography. Using the percutaneous transaxillary or preferably the transfemoral route a double curved open tip catheter with side holes is introduced into the *splenic* artery. Test injections are made and the fastest delivery rate that will not cause recoil of the catheter tip is selected for final injection. The total amount of contrast material varies with the size of the spleen, most often 30–60 ml. of 75% Hypaque are injected at a rate of 8–12 ml. per second. Films are exposed at a rate of two each second for three seconds and then at one each second for 14 seconds. The tip of the catheter is next introduced into the common hepatic artery and 30 ml. of 75% Hypaque are injected at a rate of 10–12 ml. per second. Films are exposed at a rate of four each second for three seconds and one each second for eight seconds.

In some patients celiac and superior mesenteric artery catheterizations are done simultaneously through separate catheters. To satisfactorily opacify the portal system a large amount of Hypaque, 60–80 ml. is used, with as rapid an injection rate as possible.

6. Umbilical Vein Catheterization. The obliterated umbilical vein is dissected extraperitoneally, forcibly dilated and a catheter is introduced and advanced into the left branch of the portal vein.[1] A total of 40 ml. of 75% Hypaque are injected at a rate of 10 ml. each second. Film exposure is made at a rate of two each second for six seconds and one each second for 14 seconds.

Results

Seventy-four patients were studied preoperatively, at the time of operation, or postoperatively; many were studied repeatedly. In forty-five patients technically satisfactory splenoportograms were obtained prior to treatment. The anatomic location, calibre, patency and extraluminal abnormalities of the portal vein and its collateral circulation were recorded. In addition, the amount of contrast material going to the liver in relation to that diverted through collateral channels was estimated. The appearance and degree of the hepatic blush or hepatogram on late films was helpful in assessing the amount of portal flow going to the liver. With high hepatopetal flow the blush was prominent and when the flow was predominantly hepatofugal it was poor or absent. Intermediary flow could also be gauged. Intrahepatic portal vein branches

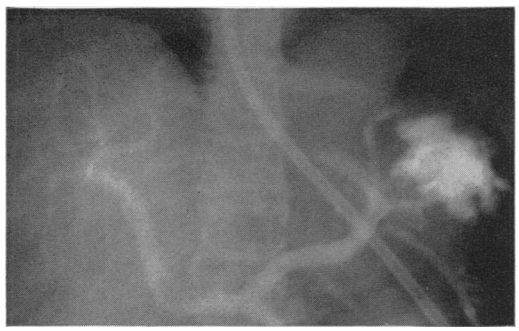

FIG. 3. The concentration of contrast medium in the portal vein and its branches was reduced. The vascular tree within the liver was distorted and on late films the liver blush was poor. The inferior mesenteric vein filled as did a vessel arising from the splenic vein.

TABLE 1. *Comparison of Estimation of Hepatic Portal Flow by Splenoportography and Hepatic Venography*

		Hepatic Venogram				
	Grade	1	2	3	4	Total
Splenoportogram	1	5	2	0	0	7
	2	0	0	0	0	0
	3	0	4	2	0	6
	4	0	0	0	3	3
	Total	5	6	2	3	16

Statistical analysis reveals a highly significant ($p < 0.01$) correlation between the two technics. In Hepatic Venography Categories one, three and four, the status of hepatic portal flow has been correctly assessed in all cases.

in cirrhosis were typically distorted and frequently failed to opacify to the periphery of the liver. An important feature of splenoportography is inability to prove organic occlusion of the portal vein. Although patency of the vein usually can be ascertained by complete filling, failure to visualize the vein may be due to spontaneous reversal of flow, with the portal vein an outflow tract from the liver. This diagnostic problem is clarified by wedged hepatic venography. On the basis of radiographic appearance the degree of interference with portal flow to the liver was estimated in stages: Stage I, normal or only slightly restricted portal flow (Fig. 2), Stage II modate reduction (Fig. 3), Stage III severe restriction of flow (Fig. 4). Total lack of opacification of the portal vein was termed Stage IV (Fig. 5).

Hepatic vein catheterization was performed on 26 patients. With successful injection of dye in the wedged position sinusoids were always seen on the roentgenogram. An irregular outline of the sinusoid with filling defects, was characteristic of patients with cirrhosis. A well visualized, sharply defined sinusoidal pattern, usually 4–6 cm. in its longest diameter was indicative of satisfactory sinusoidal filling. On the other hand, "flooding" the sinusoidal bed can lead to errors in interpretation because in this circumstance contrast medium may be forced into a portal vein that is the site of strong hepatopetal flow. Flooding is characterized by a very dense collection of dye with obliteration of the *variegated* pattern of the sinusoids (Fig. 6). Care should be taken to control the volume and rate of injection and to avoid catheterization of small lobular veins such as those in the caudate lobe. In the latter circumstance, the catheter may be truly obstructive due to the fact that collateral flow is limited and lead to incorrect interpretations of pressures and venograms.

One important use of hepatic venography has been assessment of hepatic portal flow (Table 1). Four categories of patients were identified. In Category 1 there was excellent filling of the sinusoidal bed but no filling of the portal system. This was characteristic of patients who had normal or mildly restricted portal venous flow to the liver (Fig. 7). Category 2 was characterized by retrograde flow into the portal system with the hepatopetal flow opacifying portal veins in adjacent lobules (Fig. 8). In Category 3 portal flow was almost static with retrograde filling of intrahepatic branches of the portal vein, but no true hepatofugal flow. This pattern was characteristic of portal vein thrombosis as well

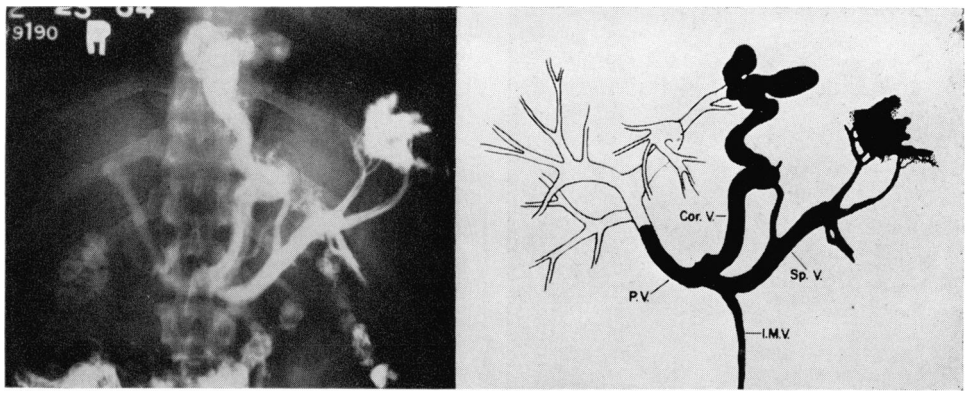

FIG. 4. Portal flow to the liver was severely restricted in this patient. The portal vein was never well opacified and an hepatogram was lacking on late films. The splenic vein (Sp.V.), inferior mesenteric vein (I.M.V.) were opacified. The coronary vein (Cor.V.) formed huge varices at the gastroesophageal junction.

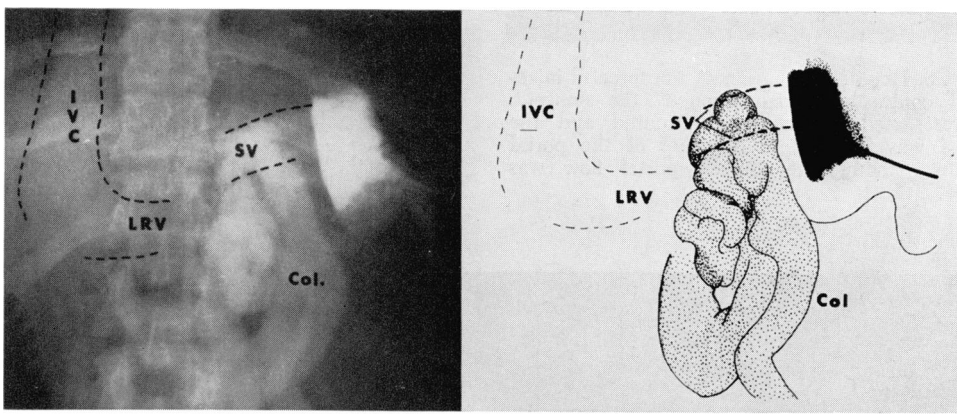

FIG. 5a. A large, tortuous collateral vein (Col.) filled from the splenic vein (S.V.) and formed a spontaneous shunt with the left renal vein (L.R.V.). From there the contrast material entered the inferior vena cava (I.V.C.). Gastroesophageal varices were not present.

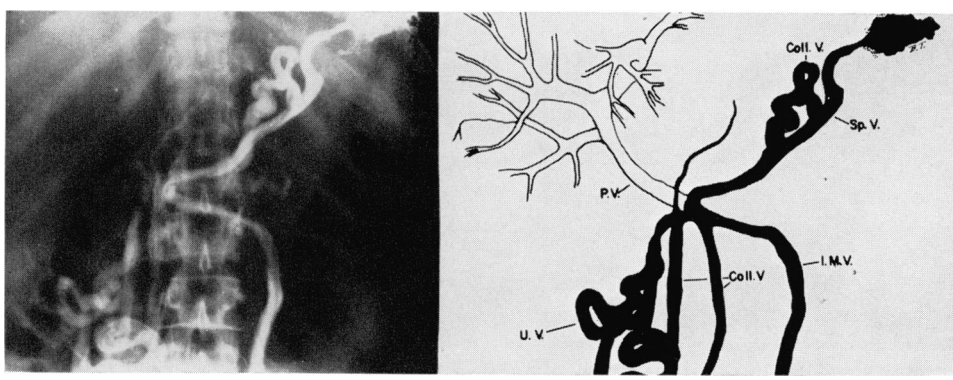

FIG. 5b. The splenic vein (Sp.V.), a collateral (Coll.V.) arising from it, the inferior mesenteric vein (I.M.V.), two other collateral veins (Coll.V.) and umbilical veins (U.V.) are all filled with contrast material. Lack of opacification of the portal vein was functional rather than occlusive (see Fig. 10).

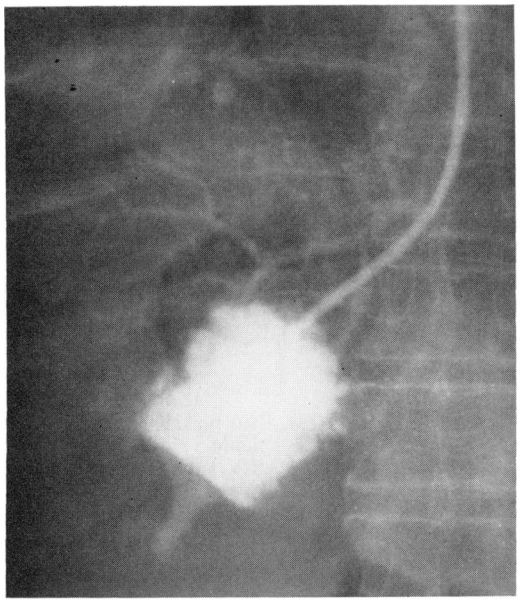

FIG. 6. Hepatic Venogram. A too forceful injection of contrast material "flooded" the sinusoid. The variegated pattern was obscured and the material was forced into branches of the portal vein even though hepatopetal portal flow was good.

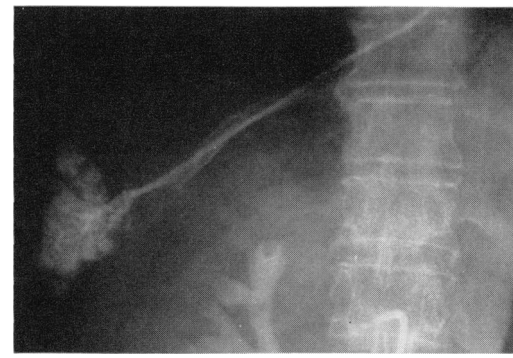

FIG. 7. Wedged Hepatic Venogram, Category I. Injection of contrast material resulted in a sinusoidogram. The filling defects and lack of homogenicity within it are characteristics of cirrhosis. The contrast medium escaped only by way of the hepatic vein without any filling of the portal system. All six patients with this pattern have had good to excellent portal flow to the liver.

as of advanced cirrhosis (Fig. 9). In Category 4 injection of dye resulted in retrograde hepatofugal flow through the portal vein and into the splanchnic circulation (Fig. 10). In such instances the portal vein

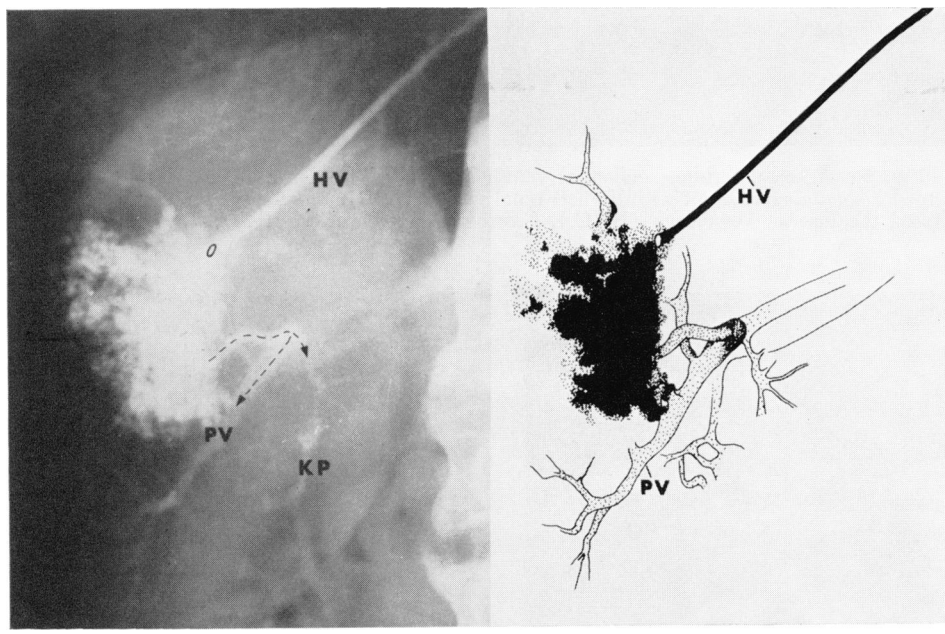

FIG. 8 Wedged Hepatic Venogram, Category II. The sinusoidogram was consistent with a diagnosis of cirrhosis. With the catheter wedged in an hepatic vein (H.V.) contrast material filled small hepatic veins and branches of the portal vein (P.V.). On subsequent films the material cleared slowly indicating sluggish portal flow. The kidney pelvis (K.P.) was opacified by a previous injection.

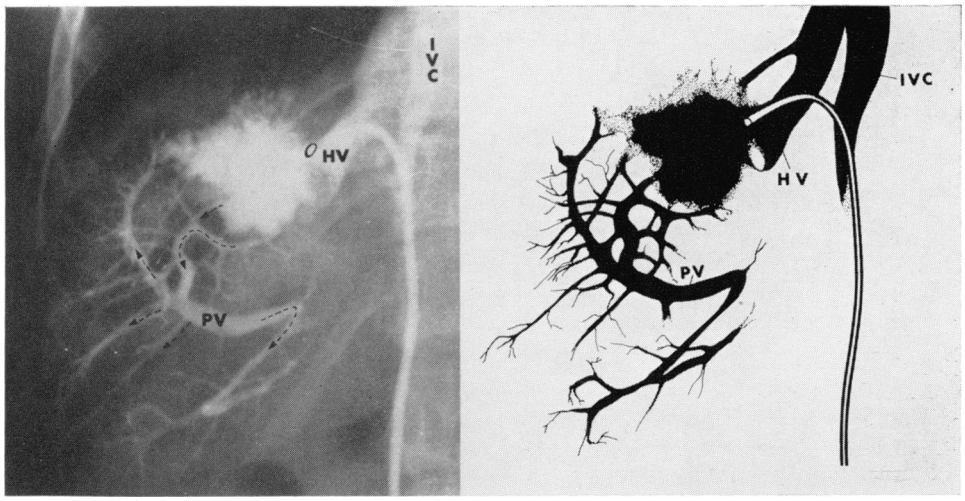

FIG. 9. Wedged Hepatic Venogram, Category III. There is virtual complete filling of the right portal system. The architecture of the veins was normal in this non-cirrhotic patient with thrombosis of the portal vein as proved by indirect portography.

was not opacified on the splenoportogram, and wedged hepatic venograms differentiated organic occlusion from an hemodynamic process. On splenoportography three patients had no evidence of hepatic portal flow and were deemed Stage IV. In all three patients on wedge hepatic venograms the portal vein was patent and filled in a retrograde direction (Category 4). In this small group there was agreement in the estimation of hepatic portal flow by both splenoportography and hepatic venography. Of 13 patients who were judged by splenoportography to be Stage III, severe restriction of hepatic portal flow, six had hepatic venograms. In two there was agreement and in four the restriction shown by hepatic venography was moderate (Category 2). No patient with moderately restricted flow estimated by splenoportography (Stage II) had hepatic venography performed. On splenoportography eighteen patients had good hepatopetal portal flow (Stage I). Seven of these had hepatic venograms performed. In five there was agreement in estimation of hepatic portal flow. In two the flow was thought to be only slightly restricted by splenoportography (Stage I) but was judged moderately restricted by hepatic venography (Category 2). Conversely in six patients in whom portal venules failed to fill on the hepatic venogram good flow to the liver was always demonstrated by portography (Table 1).

Hepatic blood flow was estimated (EHBF) prior to treatment in all patients in whom it was feasible to perform the test. In 44 patients the values obtained were

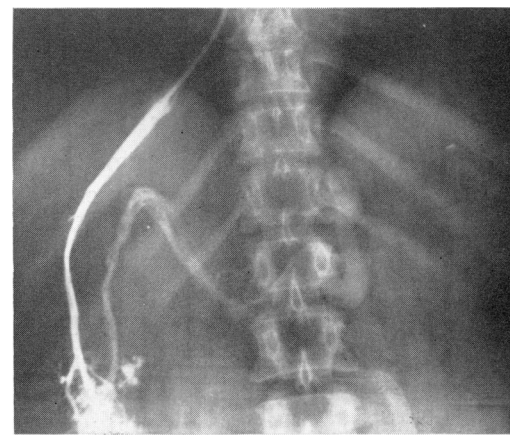

FIG. 10. Wedged Hepatic Venogram, Category IV. An hepatic vein was catheterized and the portal, coronary and inferior mesenteric veins filled indicating that the portal vein was functioning as an outflow tract. This venogram was performed on the same patient whose splenoportogram is illustrated in Figure 5.

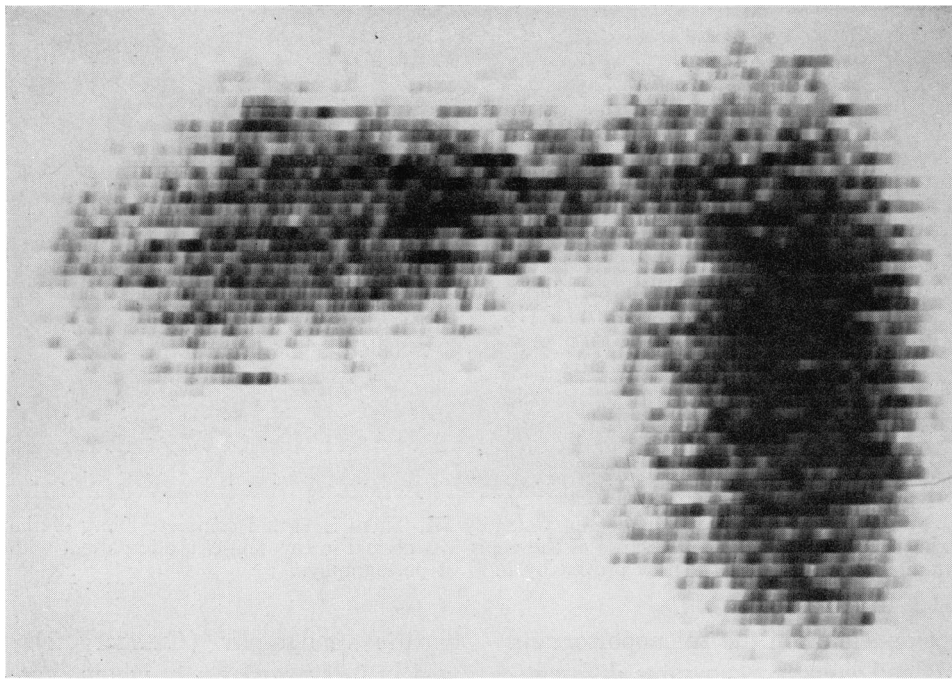

FIG. 11a. Liver Scan. A large spleen with high Au^{198} uptake by the reticuloendothelial tissue caused a spuriously high value when total hepatic blood flow was estimated. Uptake in the contracted liver lacked homogenicity. This was characteristic of diffuse parenchymal disease of the liver.

compared with the estimation of blood flow to the liver estimated by another method for determining hepatic blood flow. The normal K value in this institution is 0.29 ± 0.05. In almost all instances a liver scan was performed immediately following estimation of total hepatic blood flow. Relatively accurate estimation of liver and spleen size was possible and estimation of extrahepatic Au^{198} uptake was useful in interpreting results. Evaluation of the status of liver parenchyma was not always accurate. On occasion it was possible to predict that the parenchyma was either normal or had single or multiple filling defects or was diffusely diseased. A prominent caudate lobe, if present, could usually be recognized (Fig. 11).

In nine patients hepatic venography but not splenoportography was performed. Two had severe reduction of portal flow to the liver. In four the reduction was moderate and in three it was slight. Twenty-three patients had good portal perfusion of the liver judged by splenoportogram, hepatic venogram or both. In 17 of these hepatic blood flow was estimated. The average K value for the group was 0.23 ± 0.07. Eighteen had severely reduced hepatic portal flow, and the average K value obtained from 15 of these patients was 0.16 ± 0.05. Fifteen patients had moderate restriction of portal flow and EHBF measured in 12, showed an average K value of 19 ± 0.05. The differences in EHBF between patients with severely restricted flow and those with relatively good flow was significant to $p < 0.005$ (Table 2).

In 27 patients a corrected sinusoidal pressure (CSP) was calculated by substracting the pressure in the free hepatic vein (FHV) from that obtained with the catheter in the wedged position (WHV). Pressures between 6 and 14 mm. Hg signified mild por-

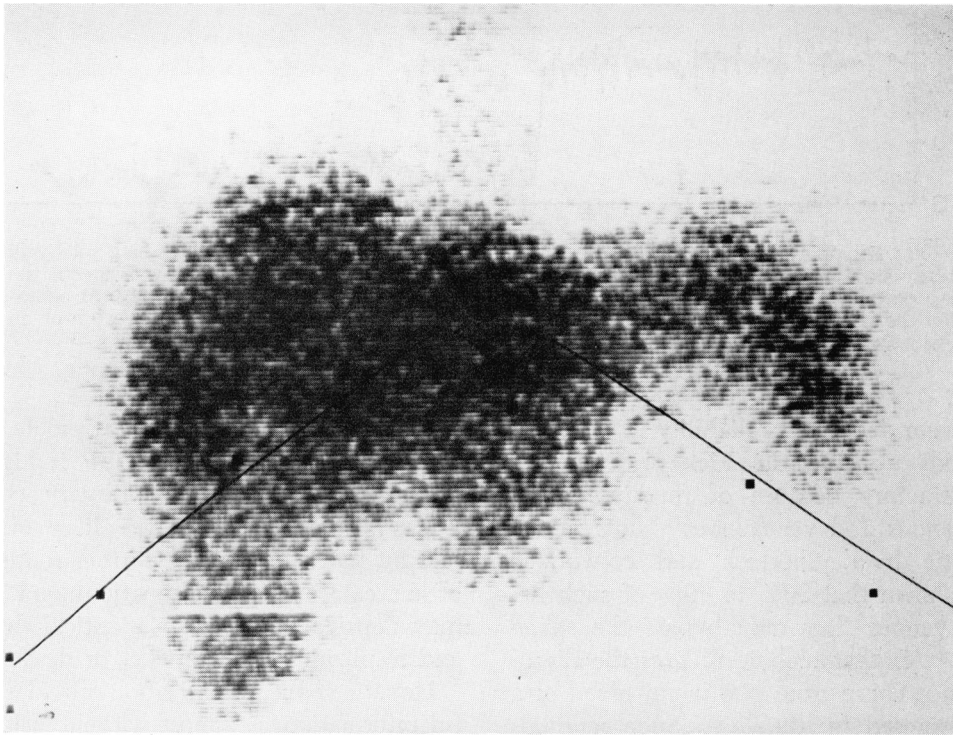

FIG. 11b. The spleen was of normal size. Because it and the bone marrow took up only a small proportion of the gold the estimation of hepatic blood flow was more accurate. The large liver was the site of diffuse parenchymal disease and a caudate lobe was easily seen.

tal hypertension, between 15 and 20 mm. Hg moderate hypertension, and above 20 mm. Hg severe hypertension (Fig. 12). Normal sinusoidal pressures are characteristic of extrahepatic portal vein thrombosis of the non-cirrhotic type. High pressures do not necessarily occur in far advanced portal hypertension and may be seen in patients with an early stage of the disease.

Splenoportograms and esophagograms as means to demonstrate varices in the gastroesophageal area were compared. Varices could be diagnosed more accurately from splenoportograms than from esophagograms. Thirty seven patients who had splenoportograms also had esophagograms. Estimations of the degree of varices by the two methods did not agree, but only in three instances were varices seen on the splenoportogram and not on the esophagogram. The converse was true once. The severity of varices as determined by splenoportography did not necessarily correlate to the degree of portal hypertension.

Discussion

Accurate measurements of total hepatic blood flow would be most helpful in the study of portal hypertension. Unfortunately, available technics to estimate hepatic blood

TABLE 2. *EHBF in Patients with Restricted Hepatic Portal Blood Flow*

	Portal Flow Estimated by Splenoportography and/or Hepatic Venography		
	Slightly Reduced (I)	Moderately Reduced (II)	Severely Reduced (III & IV)
No. pts.	17	12	15
EHBF	K 0.23 ±0.07	K 0.19 ±0.05	K 0.16 ±0.05*

* Falsely high values due to increased extrahepatic uptake of Au^{198} and highest percentage of splenic venous flow bypassing the liver.

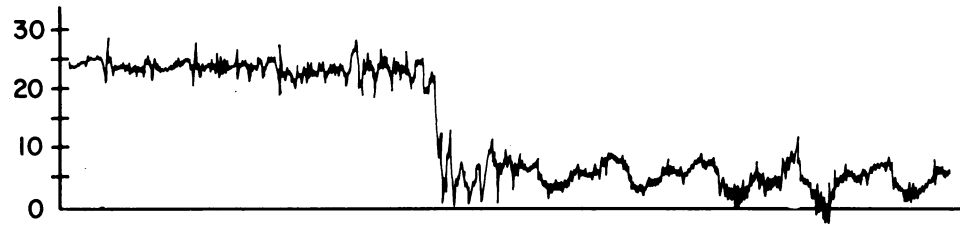

FIG. 12. Wedge and Free Hepatic Vein Pressure Curves. With the patient in a relatively steady state the typical wedged hepatic pressure curve was 24 mm. Hg. The pressure in the free portal vein was 5 mm. Hg giving a corrected sinusoidal pressure of 19 mm. Hg. At operation the corrected free portal pressure was only 6 mm. Hg, a falsely low value because of an unsteady state.

flow have decreased reliability in cirrhosis. Methods utilizing the Fick principle are unsatisfactory because of functioning arteriovenous and venovenous shunts in the cirrhotic liver. Sherlock and co-workers have shown that 30% to 40% of such hepatic venous flow may bypass the sinusoids.[2, 8] Another technic utilizes the disappearance curve from peripheral blood of a dye excreted by the liver. Such methods are inaccurate because of variability of hepatic extraction in cirrhosis.[5] A third method, the one used in this study, is simple, easy to repeat and depends on extraction of radioactive colloidal gold by the Kupfer cells. The accuracy of the method is predicated on the false assumption that all gold injected into a peripheral vein is removed by the liver on its first passage through that organ. This, of course, is not true, as some of the gold is removed by other reticuloendothelial tissues. Abnormally high uptake of gold by a normal or enlarged spleen will give a falsely high value for hepatic blood flow when a significant fraction of splenic venous flow bypasses the liver. Care must be exercised in performance of the test and in interpretation of the curve. If the curve does not rise or rises and drops a small degree the calculation of $T\frac{1}{2}$ is inaccurate. However, curves in the latter category are almost always seen in patients with other evidences of reduced hepatic blood flow. The clinical value of EHBF is in identifying patients with either very high or extremely low flows. Usually when the EHBF is high, a portacaval shunt, under ordinary conditions, abruptly deprives the liver of significant flow. Conversely, further reduction of an extremely low hepatic blood flow may deprive the liver to a critical degree and result in encephalopathy or death.

Hepatic vein catheterization provides useful information, is almost without risk and has become a standard procedure. Except in presinusoidal obstruction or following a portacaval shunt, wedged hepatic pressures closely approximate free portal pressures. Pressures measured both at the time of hepatic vein catheterization and at operation are converted to corrected pressures for increased accuracy. For example, a WHV pressure of 17 mm. Hg might be interpreted as moderate portal hypertension and would be if the pressure in the free hepatic vein and inferior vena cava were 2 mm. Hg. However, if this latter pressure were 13 mm. Hg portal hypertension would not be present. Establishment of the level of portal hypertension is important in interpretation of pressures obtained at the time of operation. Extreme variations in hemodynamics during operation may result from anesthesia, changes in intraabdominal pressure due to the incision, or release of ascitic fluid or loss of blood. This probably accounts for the impression that consistent elevation in portal venous pressure does not occur in cirrhosis of the liver. How-

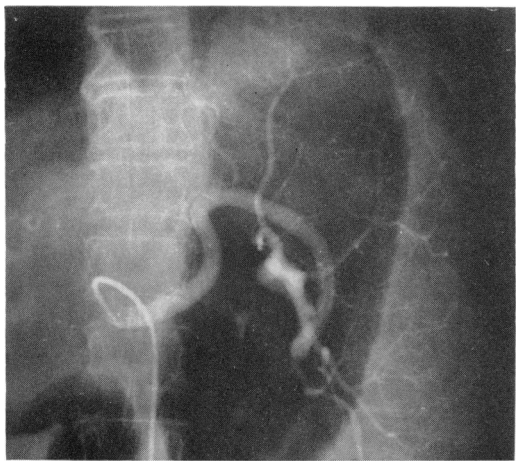

FIG 13a. Splenic Arteriogram. Arterial phase. The splenic artery was opacified and branched within a large spleen.

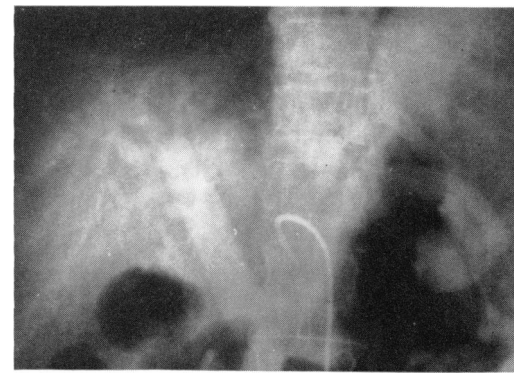

FIG. 13b. During the venous phase the portal vein and its intrahepatic branches were seen. A large coronary vein opacified huge esophageal varices.

ever, with wedged hepatic pressures, the level of sinusoidal pressure can be measured under basal conditions with the patient conscious. The procedure need not be hurried and if there are changes in the patient's condition these can be corrected so that a stable state may be achieved. With such data, pressures measured at operation which would seemingly be contradictory can be interpreted.

Radiographic estimation of portal venous flow to the liver usually is most easily determined by splenoportography. However, there are instances in which this procedure is either unsuccessful, too hazardous or cannot be performed. When properly performed and interpreted, wedged hepatic venography correlates well with alterations of portal flow to the liver and has proven to be a useful diagnostic tool. In patients with spontaneous reversal of portal flow hepatic venograms are the *sine qua non* of diagnosis.

The splenoportogram is usually the single best method for estimating portal hemodynamics. The pliable catheter used decreases but does not eliminate the possibility of splenic injury. When operation to control bleeding from the spleen would be hazardous splenoportography is not advisable. It is also avoided in severe thrombocytopenia, severely decreased prothrombin activity or marked jaundice or ascites.

Indirect portography is safer than splenoportography. The only contraindications are those of any intraarterial percutaneous study and because of added information obtained and safety this examination is being used increasingly both in preoperative and postoperative evaluations of portal hemodynamics. At times it is impossible to catheterize the splenic artery. With huge splenomegaly and loading of the portal system the contrast medium may be so diluted in the venous phase as to provide unsatisfactory opacification. When successful the information obtained from the venous phase of the examination is similar to that obtained from splenoportography (Fig. 13). Immediately following catheterization of the splenic artery the catheter is positioned in the hepatic artery. In presinusoidal hypertension and when the EHBF is relatively high and portal venous hepatic perfusion is decreased the hepatic artery may appear unduly large, perhaps because of compensatory hypertrophy. In such instances there is a prominent liver blush on late films. The intrahepatic branches of the hepatic artery have a characteristic appearance in hepatic cirrhosis. If the liver is of normal size or enlarged the arteries may

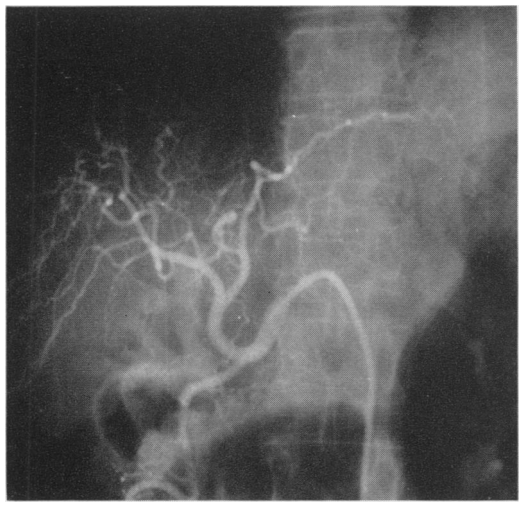

FIG. 14. Hepatic Arteriogram. Some of the distorted intrahepatic arterial branches are coiled, characteristic changes in a cirrhotic liver. Hepatic artery is normal in size.

appear relatively undistorted. When there is fibrosis and a small liver, the branches are reduced in diameter, distorted and often coiled (Fig. 14). In the event that splenic or hepatic artery catheterization is unsuccessful the celiac axis may be injected or both the celiac and superior mesenteric arteries through separate catheters may be injected simultaneously (Fig. 12).

Portal hypertension is a complex syndrome in which multiple changes have occurred in the vascular physiology of the splanchnic system. Surgically constructed portacaval shunts produce acute, pronounced alterations in the new "basal conditions" to which the organism has adjusted. Some of the changes are accepted as being beneficial to the patient, such as the rerouting of splanchnic venous flow away from bleeding esophageal varices. Other changes are acknowledged to be deleterious, for example, the sudden cessation of high volume portal venous flow to the liver. Rational therapy requires a detailed assessment of hemodynamics. Only with such information can effective choice between nonoperative and operative therapy be made. If operation is to be carried out preoperative study is also helpful in selecting the type of procedure.

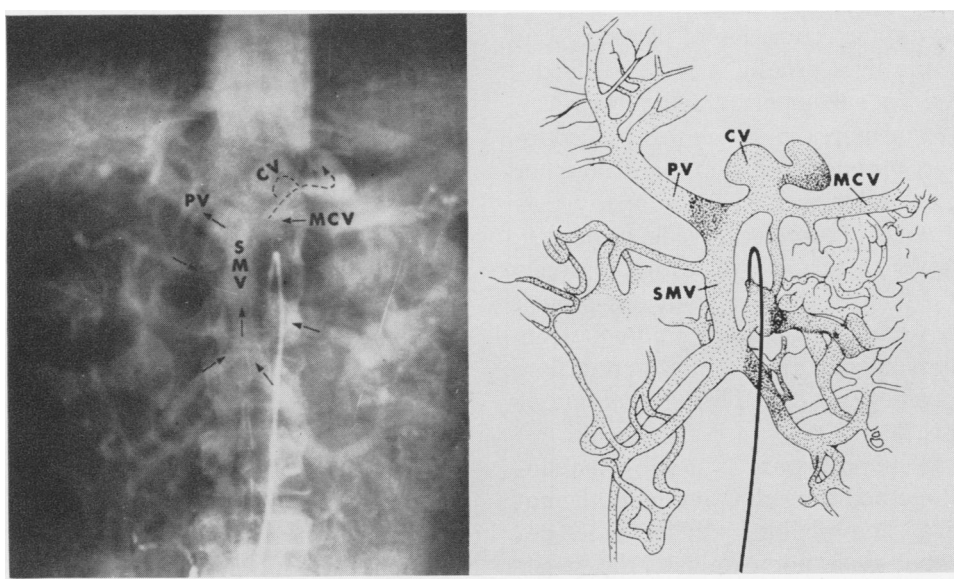

FIG. 15. Superior mesenteric arteriogram, venous phase. Because of previous splenectomy this method was used to opacify the portal system. The portal vein (P.V.) was well seen and flow to the liver was good. The coronary vein (C.V.) formed a large varix. The superior mesenteric vein (S.M.V.) and middle colic vein (M.C.V.) were also seen.

Since vascular physiologic changes are numerous the problem of predicting the effects of a portacaval shunt is complicated. When an attempt is made to relate such general changes to an individual patient the difficulties are compounded. One patient may have only small esophageal varices with no portal flow to the liver while another may have large varices with good hepatic portal flow. Studies of large numbers of patients with portal hypertension emphasize the tremendous variability in vascular patterns and hemodynamics in patients with cirrhosis of the liver. Changes produced by portal systemic shunting procedures are related not only to the effects of the operation but also to the preoperative hemodynamics in a given patient. Our approach is to define as clearly as possible the physiologic and anatomic features in individual patients. Using knowledge of changes induced by operation, some idea of the total effect of the operation may be obtained.

Summary

1. Hepatic and splanchnic vascular physiologic features were investigated preoperatively in 74 patients with either cirrhosis of the liver or presinusoidal portal vein obstruction. The methods involved estimation of total hepatic blood flow with Au^{198}, liver-spleen scan, hepatic vein catheterization, splenoportography or indirect portography, visceral angiography and in a few instances umbilical vein catheterization.

2. Splenoportography was the most useful single examination in the preoperative assessment of portal hypertension.

The following information can be obtained by this technic: a) establishment of the diagnosis of portal hypertension, b) location of collateral veins, including esophageal varices, c) estimation of the volume of intrahepatic and extrahepatic portal venous flow, d) establishment of patency—but not occlusion—of the portal vein.

Splenoportography is the best way to grade esophageal varices and assess portal venous flow to the liver.

3. Hepatic vein catheterization was shown to be a valuable preoperative study especially for the study of seriously ill patients. With both manometric and venographic studies the following may be accomplished: a) diagnosis of portal hypertension and differential diagnosis of intrahepatic vs. extrahepatic obstruction of the portal vein, b) assessment of portal venous flow to the liver, c) a definitive differential diagnosis between occlusive obstruction and spontaneous reversal of flow in the portal vein.

Characteristic patterns with hepatic venography were identified which clearly delineate either very good or very restricted portal venous flow to the liver. The determination of pressures in the basal state is most helpful in interpreting pressures measured during operation.

4. The Au^{198} technic for estimation of hepatic blood flow shows significant differences between mild (or early) and severe portal hypertension. It is most useful in screening patients with near normal or very low hepatic blood flow. The liver-spleen scan is helpful in assessing the significance of extrahepatic uptake of Au^{198} in individual patients.

5. Visceral angiography with indirect portography is most useful in postsplenectomy patients and when splenoportography is contraindicated. Umbilical vein catheterization has been utilized only in selected cases.

6. The ability to define precise vascular physiologic changes in individual patients with portal hypertension is becoming more important.

References

1. Bayly, J. H.: The Portal Vein and Liver Portography Utilizing the Umbilical Vein. J. Nat. M. A., 57:267, 1965.
2. Bradley, S. E., Ingelfinger, F. J., Bradley, G. P. and Curry, J. J.: Estimation of He-

patic Blood Flow in Man. J. Clin. Invest., 24:890, 1945.
3. Callow, A. D., Lloyd, J. B., Ishihara, A., Ponsdomenech, E., O'Hara, E. T., Chalmers, T. C. and Garceau, A. J.: Interim Experience with a Controlled Study of Prophylactic Portacaval Shunt. Surgery, 57:123, 1965.
4. Hallenbeck, G. A., Wollaeger, E. E., Adson, M. A. and Gage, R. P.: Results After Portal Systemic Shunts in 120 Patients with Cirrhosis of the Liver. Surg. Gynec. Obstet., 116:435, 1963.
5. Leevy, C. M., Mendenhall, C. L., Lesko, W. and Howard, M.: Estimation of Hepatic Blood Flow with Indocyanine Green. J. Clin. Invest., 41:1169, 1962.
6. Myers, J. D. and Taylor, W. J.: An Estimation of Portal Venous Pressure by Occlusive Catheterization of an Hepatic Venule. J. Clin. Invest., 30:662, 1951.
7. Restrepo, J. E., Warren, W. D., Nolan, S. P. and Muller, W. H., Jr.: Radioactive Gold Technique for the Estimation of Liver Blood Flow: Normal Values and Technical Considerations. Surgery, 48:748, 1960.
8. Shaldon, S., Chiandussi, L., Guevara, L., Caesar, J. and Sherlock, S.: The Estimation of Hepatic Blood Flow and Intrahepatic Shunted Blood Flow by Colloidal Heat-denatured Human Serum Albumin Labeled with I^{131}. J. Clin. Invest., 40:1346, 1961.
9. Sherlock, S.: Personal Communication.
10. 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.
11. Warren, W. D., Restrepo, J. E., Respess, J. C. and Muller, W. H.: The Importance of Hemodynamic Studies in Management of Portal Hypertension. Ann. Surg., 158:387, 1963.

Discussion

Dr. George Johnson (Chapel Hill): Dr. Mahorner. An approach to the surgical treatment of portal hypertension associated with cirrhosis of the liver that is based on systemic and portal hemodynamics such as just described has merit. The authors are to be commended for the establishment of more rational criteria for the operation performed on these patients.

A significant number of patients with cirrhosis and portal hypertension have been shown to have a hyperdynamic cardiovascular system with an increase in cardiac output.

If a portacaval shunt is to be performed on these patients, it is important to know the effects of this procedure on the systemic hemodynamics. The first slide, please.

(Slide.) In a series of dogs, cardiac outputs were performed using a dye dilution technic at weekly intervals of 3 to 6 weeks. A sham operation was then performed on six dogs in which the portal vein and inferior vena cava were dissected but no shunt performed.

As you can see, the cardiac outputs in the postoperative period in red, were not significantly different from those in the preoperative period, in blue. The mean difference for the pre- and postoperative periods were not significant in the six animals. The next slide, please.

(Slide.) However, when a portacaval shunt was performed, there was a rise in cardiac output in each animal with a mean increase of 54 cc./Kg./min., a rather large and significant change.

That this increase in cardiac output following a portacaval shunt occurs in humans has recently been reported by Even of France after a study of 12 patients.

In the cirrhotic patient with a hyperkinetic cardiovascular system, creation of a portacaval shunt would thus appear to add an even greater burden on the heart and vascular system.

I sincerely enjoyed Dr. Warren's presentation and, Dr. Mahorner, I sincerely appreciate the courtesy of the floor. Thank you.

Dr. W. Dean Warren (Closing): I would like to thank Dr. Johnson for his comments.

As many of you know, the changes which he describes, in cardiac output, cardiac index, *etc.*, are actually seen in patients with cirrhosis before the creation of a portacaval shunt, and the best data available would indicate that a portacaval shunt increases this abnormality slightly. However, I don't believe that this is one of the most bothersome problems in the large number of patients we have followed after the creation of a portacaval shunt.

The biggest problem which follows shunts is delayed hepatic death in which the patient survives the operation but progressively deteriorates to die of hepatic failure, usually within 2 years.

Second is the severe morbidity manifest by encephalopathy or continued weakness, fluid retention, *etc.* To my knowledge, we have not had any patient who was bothered significantly by chronic cardiac failure based on the hyperdynamic situation which Dr. Johnson has mentioned.

However, I certainly think that their study of non-shunting procedures in the cirrhotic is one of fundamental importance and one which you should all consider as you approach the problem of management of these very difficult patients.

Thank you.