Intrahepatic Veno-Occlusive Disease in Cirrhosis with Chronic Ascites: *

Diagnosis by Hepatic Phlebography and Results of Surgical Treatment

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IN MOST PATIENTS with cirrhosis of the liver, ascites appears late in the course of the disease as one manifestation of hepatic decompensation. With prolonged medical treatment and improvement in hepatic function, control or disappearance of ascites frequently occurs.[5, 29] However, there are cirrhotic patients who develop chronic ascites which becomes resistant to prolonged and intensive medical treatment in the absence of clinical and laboratory evidence of rapidly progressive hepatic decompensation. Such patients have been compared to patients with Budd-Chiari syndrome with hepatic vein occlusive disease and chronic massive ascites disproportionate to measureable impairment of hepatic function.

Several observers have suggested that in cirrhotic ascites there is markedly increased resistance to flow through the venous outflow tract of the liver and that augmentation of venous drainage of the liver is required for control of ascites.[21, 25, 38, 39] Anatomical evidence of gross obstructive lesions of the *major* hepatic veins, in support of the *outflow block* concept, is usually lacking in cirrhosis with ascites. Corrosion cast preparations [11, 18, 28] and phlebographic studies of autopsy livers [27] have demonstrated that deformities of the *smaller intrahepatic* veins do occur in cirrhosis. Micropathological reconstruction studies have indicated that such deformities may be produced by extrinsic pressure and displacement of the smaller hepatic vessels by nodules of regenerating parenchyma.[14] Involvement of adventitia-free intrahepatic veins may be more severe than of intrahepatic portal radicles which have adventitia. In living patients with cirrhosis, the degree and extent of obliterative changes in the hepatic veins are not made apparent by conventional diagnostic studies such as liver biopsy, hepatic function tests, the levels of portal or hepatic vein wedge pressures, or splenoportography.

A study was undertaken to determine the value of hepatic vein catheterization and cine-phlebography as a method for studying abnormalities of the hepatic veins in living cirrhotic patients and, if possible, to correlate changes in the hepatic veins with clinical response to treatment of ascites. The reported results of surgical treatment of patients with documented hepatic veno-occlusive disease and ascites were reviewed, since it was thought that indications of the importance of hepatic outflow tract obstruction in the genesis of ascites might be reflected in surgically altered hepatic hemodynamics. Hepatic vein catheterization and cinephlebography was performed on four patients without liver disease, 11 patients with cirrhosis and ascites, one patient with recurrent bleeding from

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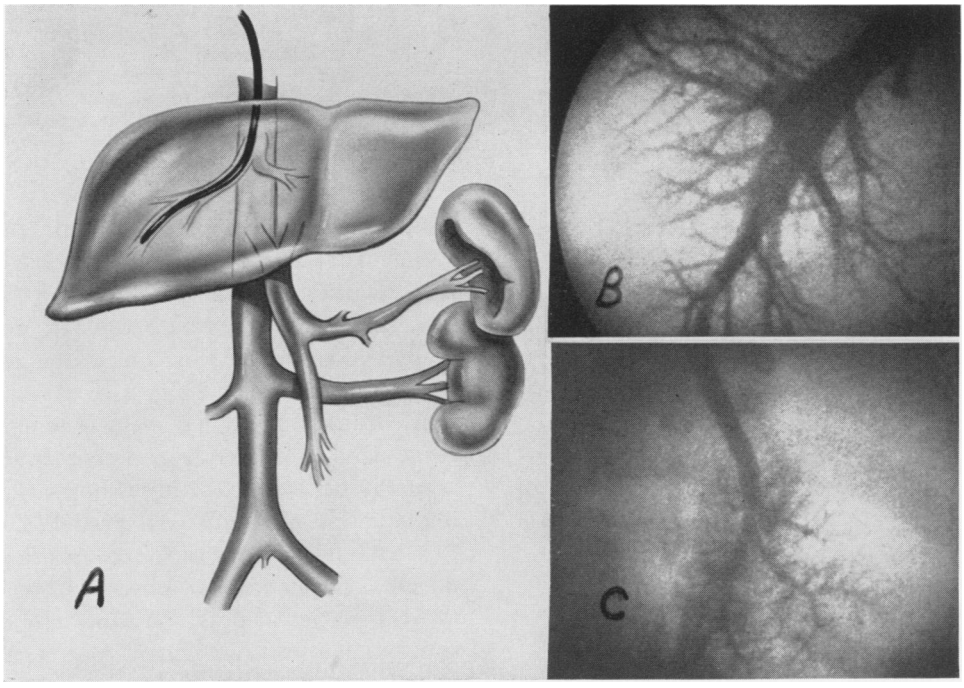


FIG. 1. A. Usual positioning of the hepatic vein catheter after introduction by fluoroscopic control through a right antecubital vein. B. Phlebogram of a normal right main hepatic vein showing smooth tapering walls, uniform filling pattern, and four distinct orders of branch caliber. C. Phlebogram of a normal left main hepatic vein showing lobular collecting veins and areas of cloud-like sinusoidal filling with excessive injection pressure. (Courtesy Arch. Surg.)

varices but without ascites, and one patient with congenital cystic disease of the liver and chronic ascites.

Methods

Hepatic Vein Catheterization and Cinephlebography. Retrograde catheterization of the hepatic veins was performed as described by Taylor and Myers [36] (Fig. 1). A 100 cm. woven Nylon 6F or 8F cardiac catheter with a single end hole was passed initially for measurement of inferior vena caval and free and wedged hepatic vein wedge pressures in as many branches in all lobes as could be entered with the catheter. This catheter was then replaced by an angiographic catheter having terminal side holes. With the catheter tip lying free in a major hepatic vein, injection of warmed 90 per cent diatrizoate (Hypaque®) was made at 3.0 to 7.0 kg. pressure to deliver 10 to 35 ml. in one to six seconds while cine-radiographs were exposed using a five-inch image intensifier on 35 mm. film at 54 frames per second. In most patients, orienting visualization was obtained by hand injection of small amounts of contrast medium. In patients who were found to have severe obliterative changes in the hepatic veins, hand injection alone was adequate for filling branches of all calibers. In normal subjects pressure injection was necessary to obtain complete segmental visualization.

Operative Studies. Studies at operation were performed through an abdominal incision during endotracheal cyclopropane anesthesia. Free portal vein and inferior vena cava pressures were obtained by direct needle puncture using a saline manometer zeroed at the level of the respective vessels. The portal vein was then tempo-

rarily occluded and pressures recorded on the hepatic side of the clamp (*occluded hepatic portal pressure*) and on the splanchnic side of the clamp (*occluded splanchnic portal pressure*). In one case (Case 14), portal vein flow was measured using a noncannulating gated sine wave electromagnetic flowmeter.

Management. All patients with ascites were hospitalized and given a diet containing approximately 10 mEq. of sodium daily. Paracenteses were performed for discomfort and prior to needle biopsy of the liver, splenoportography, or hepatic vein catheterization. For purposes of this study, ascitic patients were arbitrarily classified according to their response to a graded program of treatment (Table 1):

A. Diuresis and control of ascites within two to six weeks of dietary sodium restriction and mercurial diuretic.
B. No response to A after two to six weeks but response within one month when acetazolamide or thiazide diuretic was added.
C. No response to A or B within one month but response within one month after spironolactone diuretic was added.
D. No response to A, B, or C after at least six months of continuous treatment on an outpatient basis and in the hospital but had no recurrence of ascites following portacaval shunt. Two patients in this group were given prednisone without response in conjunction with all other agents.

Results

Characteristics of the Normal Hepatic Veins. In the four subjects without liver disease, the hepatic veins were found to have smooth walls which tapered peripherally and multiple eccentrically placed radial side branches (Fig. 1 b, c). At least four orders of branch caliber could be recognized consistently, ranging from the major lobar trunks to tiny arborizations of immediately postsinusoidal branches. With the catheter tip in the free position, sinusoidal filling was not observed but, with the tip in the wedged position, cloud-like sinusoidal filling did occur. At the end of injection all orders of branches emptied rapidly. During inspiration contrast medium streamed directly into the right atrium and, during expiration, refluxed caudad

TABLE 1. *Data of 17 Patients Studied by Hepatic Vein Catheterization and Cine-phlebography*

Patient	Age, Sex	Liver Disease	Bleeding Varices	Duration Ascites	Clinical* Response	Phlebographic Classification	Comment
1.	35, F	0	0	—	—	0	Normal
2.	18, F	0	0	—	—	0	Normal
3.	42, M	0	0	—	—	0	Normal
4.	41, M	0	0	—	—	0	Normal
5.	32, M	PN	+	—	—	I	
6.	16, F	PN	0	3 mo.	B	II	
7.	68, M	PN	0	12 mo.	B	II	E-S shunt 4 mos. earlier
8.	35, F	Cystic	0	48 mo.	B	II	
9.	52, M	L	0	5 mo.	C	III	
10.	58, F	PN	0	3 mo.	C	III	
11.	65, M	PN	0	6 mo.	C	III	Associated portal vein thrombosis
12.	71, M	L	0	36 mo.	C	III	
13.	45, F	PN	0	36 mo.	C	III	
14.	71, F	PN	0	36 mo.	D	IV	
15.	72, M	PN	0	8 mo.	D	IV	
16.	42, F	PN	+	7 mo.	D	IV	
17.	35, M	PN	0	30 mo.	D	IV	

PN, Postnecrotic cirrhosis; L, Laennec's cirrhosis.
* See text.

into the hepatic vena cava. With continuous visualization of all phases of filling and emptying by cine technic, it was apparent that variation in the volume and pressure of injection affected the number of branches filled but did not otherwise result in artefacts giving the appearance of anatomical abnormality. These observations are in accord with those of Tori [37] who described the normal phlebographic anatomy of the hepatic veins in ten subjects.

Hepatic Vein Abnormalities in Cirrhosis. In the 12 cirrhotic patients who were studied, five general types of changes in the hepatic veins were observed. These were 1) rigidity and loss of tapering contour of first, second, and third order vessels; 2) reduction in the number of second, third, and fourth order branches visualized; 3) reduction in caliber of all orders of branches; 4) irregularity, tortuosity, and segmental stenoses of second, third, and fourth order branches; and 5) sinusoidal filling, with or without retrograde portal branch filling, during injection of the catheter in the free position. In all instances in which contrast medium was forced retrograde into portal radicles by the force of injection, completion of injection was followed by drainage of medium in prograde direction towards the sinusoids and not into the portal trunk.

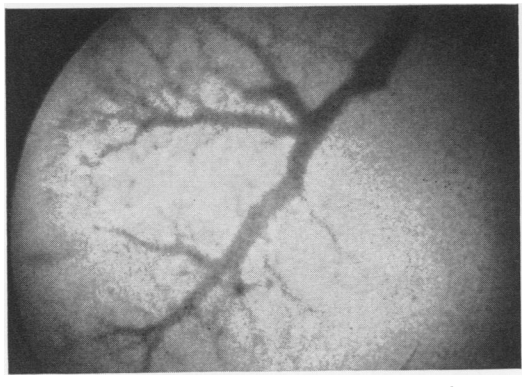


FIG. 2. Phlebogram of a right main hepatic vein showing Class II veno-occlusive disease with patchy non-visualization of third and fourth order branches and tortuosity of second and third order branches; Case 7. (Courtesy Arch. Surg.)

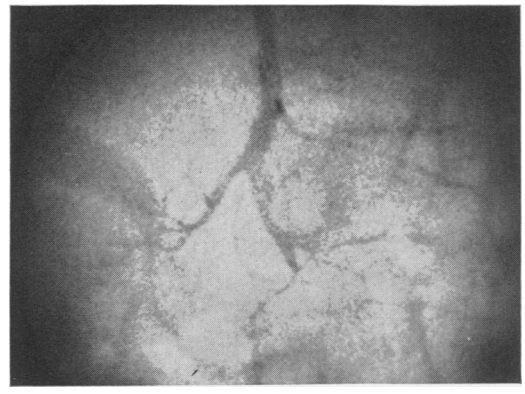


FIG. 3. Phlebogram of a right posterior lobar trunk showing Class III veno-occlusive disease with reduction in caliber and number of branches and widespread nonvisualization of third and fourth order branches; Case 10.

When it was possible to catheterize multiple branches in both right and left lobes of the liver, wide variation in the type of deformities within the same lobe was apparent. In general, the degree and extent of these changes was more or less uniform throughout the liver and an arbitrary phlebographic classification of individual patients was made, based upon the preponderance of changes in all areas visualized. In the absence of functional data indicating relationships between hemodynamic factors and the types of anatomical

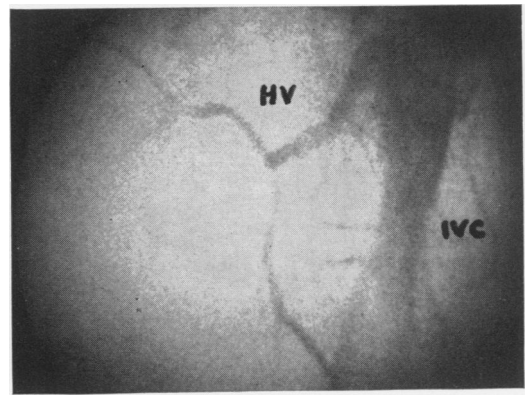


FIG. 4. Only the tip of the catheter could be passed into the ostium of the right main hepatic vein in this patient with Class IV veno-occlusive disease. There is almost complete obliteration of all orders of branches and simultaneous injection of the inferior vena cava; Case 14. (Courtesy Arch. Surg.)

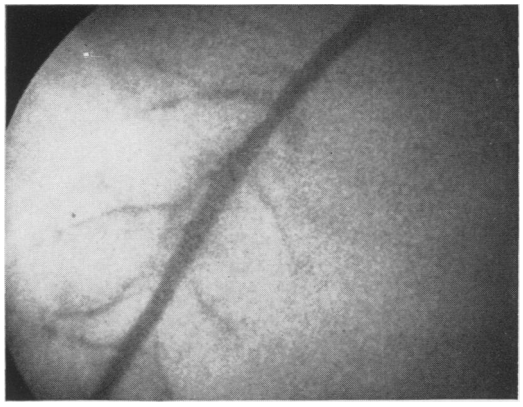


FIG. 5. Phlebogram of the right main hepatic vein showing Class IV changes with narrowing of the primary trunk, paucity of branches, and no visualization of third and fourth order branches; Case 15. (Courtesy Arch. Surg.)

deformity, the primary factors considered in the descriptive classification were segmental stenoses and reduction in numbers and caliber of vessels in the areas visualized. Class I included patchy loss of third and fourth order branches. Class II included widespread changes of mild degree involving the smaller branches (Fig. 2). Class III included predominant nonfilling of third and fourth order veins, gross tortuosity of all branches, and multiple areas of stenosis of second and third order branches (Fig. 3). Class IV changes included gross alteration of venous architecture with nonvisualization of the majority of second, third, and fourth order branches, loss of tapering contour of remaining primary and secondary trunks, and, in one patient (Case 16), the appearance of thrombotic occlusion of the right main hepatic vein (Fig. 4–7).

Comparison of Phlebographic Changes with Clinical Response to Treatment of Ascites. Within the limitations of the small series of observations, there was a remarkably close correlation between the severity of obliterative changes in the hepatic veins and the response of ascites to the outlined program of medical treatment (Table 1). All four of the patients with severe veno-occlusive disease had ascites which did not respond to prolonged and intensive treatment but they did not reform ascites from nine to 20 months after portacaval shunt. Five patients with Class III hepatic veno-occlusive disease required aldosterone inhibitor in addition to sodium restriction and other diuretics for control of ascites. Two cirrhotic patients with ascites and one patient with hepatic cystic disease and ascites were thought to have Class II

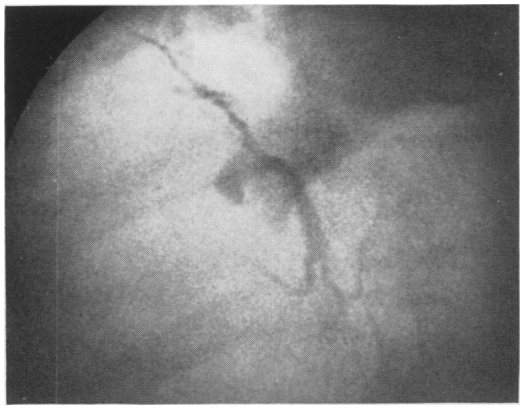


FIG. 6. Attempted phlebogram of the right main hepatic vein in Case 16; the catheter tip could not be inserted further than the sharp cut-off which has the appearance of the cephalic end of a thrombus occluding the main trunk. This interpretation is consistent with the history of rapid onset of intractable ascites after an unsuccessful attempt at portacaval shunt. (Courtesy Arch. Surg.)

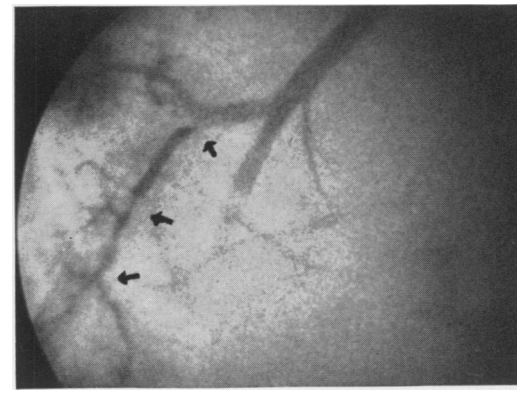


FIG. 7. Phlebogram of the right main hepatic vein showing Class IV changes with reduction in number and caliber of visualized smaller branches, tortuosity of second and third order branches, and multiple stenoses of second and third order branches in Case 17.

changes and they clinically responded to sodium restriction and mercurial and thiazide diuretics. One cirrhotic patient who had bled repeatedly from varices but had never had ascites showed phlebographic patchy loss of third and fourth order hepatic veins and mild tortuosity of second and third order trunks.

Nine and 12 months after the initial study, two Group C patients (Case 12, 13) became refractory to all treatment including 800 mg. of spironolactone daily. This observation, in addition to the fact that four of the five Group C patients had previously responded to lesser therapeutic measures in the months prior to the study, suggests that their hepatic veno-occlusive disease was progressive. Unfortunately, follow up hepatic phlebography of Cases 12 and 13 was impossible. The patient with extensive congenital cystic of the liver with minimal involvement of the kidneys presented an unusual combination of hepatic veno-occlusive disease without measureable impairment of hepatic function. The encroachment by fluid filled cysts, ranging from a few millimeters to 15 cm. in diameter, upon all orders of hepatic veins (Fig. 8) resulted in elevation of the portal and hepatic vein wedge pressures (Table 3). Esophageal varices were not demonstrable as they were in a similar patient reported by Sabeh and Edwards.[31]

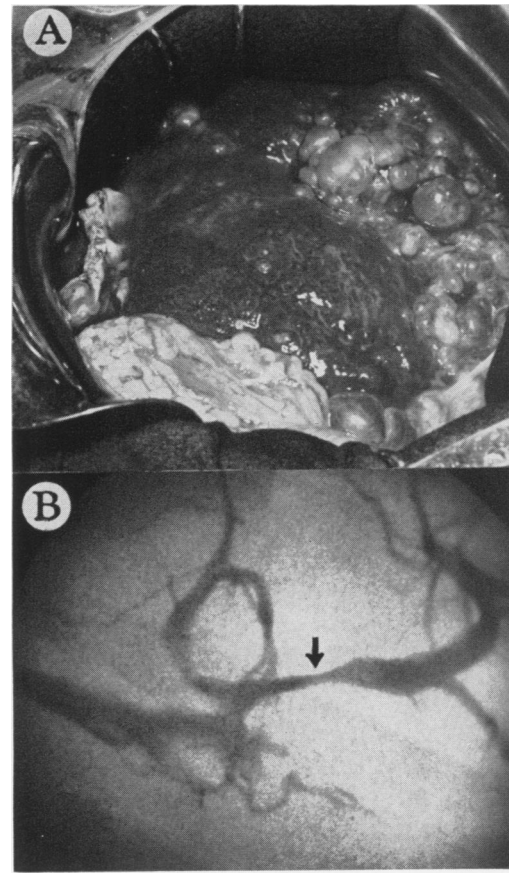


FIG. 8. A. Operative photograph of Case 8 showing extensive congenital cystic disease associated with no measurement impairment of hepatic function but with chronic ascites. B. Left hepatic phlebogram showing marked distortion of smooth walled hepatic veins by intrahepatic cysts. Multiple stenoses (arrow) throughout all lobes of the liver were present. (Courtesy Arch. Surg.)

Functional and Hemodynamic Changes in Cirrhosis with Hepatic Veno-Occlusive Disease. The conventional hepatic function tests of the four Group D patients with Class IV hepatic veno-occlusive disease yielded results expected in any patient with chronic cirrhosis and ascites (Table 2). The low serum albumin levels in Cases 14 and 15 were apparently related to frequent paracenteses prior to admission since serum albumin levels rose postoperatively in both patients. The levels of operative portal pressure and preoperative hepatic vein wedge pressure were not significantly different from those observed in patients with milder hepatic vein changes (Table 3) or in reported studies of patients with cirrhosis with or without ascites.[9] Of interest was the finding of different segmental wedge pressures in the same lobe of the liver (Table 3) even when contrast material was gently injected to confirm the wedged position. The levels of wedged pressure did not necessarily correspond to the severity of anatomical changes in the segmental veins catheterized. A possible reason for this may be illustrated in Figure 7 which shows multiple stenoses of second order branches; with the catheter tip wedged in such a

TABLE 2. *Admission Data of Four Patients with Cirrhosis and Medically Resistant Ascites Associated with Hepatic Vein Occlusive Disease*

	Case 14	Case 15	Case 16	Case 17
Age, years	71	72	41	35
Sex	F	M	F	M
Type of cirrhosis	PN	PN	PN	PN
Duration of ascites, months	36	8	7	30
Number of paracenteses	22	13	5	12
Volume of paracenteses	12–20 L,	3–9 L,	8 L,	10–14 L,
Hepatomegaly	1 cm.	0	5 cm.	8 cm.
Splenomegaly	2 cm.	5 cm.	8 cm.	10 cm.
Esophageal varices	+	+	+	+
Hemorrhage from varices	0	0	2	0
Pleural effusion	0	+	0	0
Peripheral edema	+	+	0	+
24 hour urine sodium, mEq/L	5.4	2.0	4.4	3.8
Albumin/Globulin, Gm.%	2.4/3.6	1.9/5.1	3.1/3.5	3.0/3.3
BSP, 45 minute retention	25%	—	16%	33%
Total bilirubin, mg.%	0.8	1.8	0.5	1.6
SGOT, units	20	37	20	14
Alkaline phosphatase, units	21	6	—	4.7
Cephalin flocculation, units	+++	+++	0	0
Thymol turbidity, units	2.0	3.2	2.0	7.5
Prothrombin %	68	50	58	50
Prothrombin % after vitamin K	100	58	58	50

stenosis, a lower pressure may be encountered because of local collateral drainage than when the tip is wedged in a terminal third order branch.

Results of Surgical Treatment of Ascites with Hepatic Veno-Occlusive Disease. Surgical attempts to control ascites in 30 patients with hepatic veno-occlusive disease, proven by phlebography, autopsy, or suggestive history and liver biopsy evidence of severe centrilobular congestion, were reviewed. The reporting authors and the patients' underlying or associated disease, operation, and the results are summarized in Table 4. Six patients operated upon in the terminal stages of their disease died in shock within one or two days after operation. Five patients survived

TABLE 3. *Preoperative Hepatic Catheterization and Operative Portal Pressures*

Case	Clinical Group	Phlebo. Class	Operative Portal Pressures (mm. Hg) FPP	OHPP	OSPP	IVCP	Hepatic Vein Pressures (mm. Hg) FHVP	WHVP	IVCP	WHVP*
7.	B	II	—	—	—	—	3.5	18.0	2.0	16.0
							5.5	23.0	2.0	21.0
								29.0	2.0	27.0
8.	B	II	20.0	—	—	4.0	13.0	28.0	6.0	22.0
9.	C	III	—	—	—	—	10.0	31.0	6.0	25.0
10.	C	III	—	—	—	—	9.5	26.5	7.5	19.0
11.	C	III	—	—	—	—	10.0	35.0	9.0	26.0
								18.0	9.0	9.0
12.	C	III	—	—	—	—	5.0	34.0	5.0	29.0
								41.0	5.0	36.0
13.	C	III	—	—	—	—	6.0	18.0	2.0	16.0
14.	D	IV	29.7	30.7	18.4	12.3	—	—	—	—
15.	D	IV	16.1	16.1	16.1	6.1	10.0	27.0	6.0	21.0
17.	D	IV	25.4	25.4	25.4	10.0	10.0	30.0	4.0	26.0
18.	C	?	30.7	28.2	33.8	9.2	19.0	26.5	6.5	20.0
							11.5	20.5	6.4	
19.	C	?	15.3	11.5	16.0	8.4	8.3	28.0	8.0	20.0

Abbreviations: FPP, Free portal pressure; OHPP, Occluded hepatic portal pressure; OSPP, Occluded splanchnic portal pressure; IVCP, Abdominal inferior vena cava pressure; FHVP, Free hepatic vein pressure; WHVP, Wedged hepatic vein pressure; IVCP, Hepatic inferior vena cava pressure; WHVP*, Corrected wedge pressure.

TABLE 4. *Reported Surgical Results in Hepatic Vein Occlusive Disease with Chronic Ascites Resistant to Medical Treatment*

Authors	Age, Sex	Disease	Operation	Follow up and autopsy findings
Sohval[35]	30, F	Polycythemia vera	Omentopexy	Died in shock in 12 hours. Thrombosis of HV, PV, SV, IVC, and SMV with recanalization of SMV.
Blakemore[2]	28, F	Leukemia	Splenorenal shunt	No ascites for 8 years; death from leukemia. Shunt occluded, HV recanalized.
Jonas and Lawrence[13]	19, F	Cirrhosis	Laparotomy	Died in 6 hours. Postnecrotic cirrhosis with HV stenoses and caval stenosis.
Brink and Botha[3]	18, F	Trauma	End to side portacaval	Died a few hours postop. Stenoses of HV at junction with vena cava.
Norris[26]	30, M	Idiopathic	Ligation HA and SA	No effect on ascites. Bled from varices 5 months and fatally 2 years later. Endophlebitis HV.
Fitzgerald, *et al.*[8]	22, F	Polycythemia vera	Splenorenal shunt	No ascites at one year.
McAllister and Barker[20]	35, F	Polycythemia vera	Side to side portacaval	No ascites at 5 years.
Eisenmenger and Nickel[6]	18, F	Postnecrotic cirrhosis	End to side portacaval	Shunt for variceal bleeding. Postop. intractable ascites and died in a few weeks. Endophlebitis HV.
	14, F	Endophlebitis HV	Splenorenal shunt	No ascites for 3 years. Died with cerebral hemorrhage.
McDermott[22]	?, M	Bush tea poisoning	Side to side, vein graft	Ascites-free for a period then ascites recurred followed by downhill course and death. Thrombosis of bridging graft.
Imanaga[12]	?	Cirrhosis	Described 1 ileoentectropy and 6 sternal-omentopexies with remark that all were improved.	
Madden[19]	?	Cirrhosis	Suprahepatic poudrage	Two patients, died with hepatic failure 2 and 3 weeks postop. No adherence of liver to diaphragm because of rapid reaccumulation of ascites.
Mackby[17]	23, M	Idiopathic	Splenorenal shunt	Died in 24 hours. Thrombosis of HV, intrahepatic PV.
Erlik, *et al.*[7]	33, F	Idiopathic	Side to side portacaval	No ascites 4 months postop.
		Idiopathic	Splenorenal shunt	Two patients, died in 24 hours. Both had HV thrombosis.
Kimura, *et al.*[15]	25, M 53, F 29, M	Idiopathic Idiopathic Idiopathic	Membranotomy Membranotomy Membranotomy	All three patients had ascites and edema with caval membrane at the diaphragm and HV stenosis. One death and two free of ascites at 3 and 6 months.

TABLE 4.—(*Continued*)

Authors	Age, Sex	Disease	Operation	Follow up and autopsy findings
Britton, Brown, Shirey	71, F	Postnecrotic cirrhosis	Side to side portacaval	Ascites-free for 20 months. Died with perforated duodenal ulcer.
	72, M	Postnecrotic cirrhosis	Side to side portacaval	Ascites-free 9 months.
	41, F	Postnecrotic cirrhosis	Splenorenal	Ascites-free 22 months.
	35, M	Postnecrotic cirrhosis	Side to side portacaval	Ascites-free 13 months.

Abbreviations: HV, Hepatic veins; PV Portal vein; SV, Splenic vein; IVC; Inferior vena cava; HA, Hepatic artery; SA, Splenic artery; SMV, Superior mesenteric vein.

briefly then died with hepatic failure or postoperative complications. Two of the latter are of special interest. The first patient, reported by McDermott [22] had chronic massive ascites treated by side to side portacaval shunt employing a venous "H" graft. The patient was free of ascites for a period of time but ascites recurred and death followed progressive deterioration. Autopsy revealed thrombosis of the "H" graft and hepatic vein endophlebitis of the type seen with bush tea poisoning. The second patient, reported by Eisenmenger and Nickel [6] had an end-to-side portacaval shunt performed for recurrent hemorrhage from varices without peroperative ascites. Massive intractable ascites developed postoperatively followed by death within a few weeks. Autopsy showed a patent shunt and severe hepatic vein endophlebitis of the idiopathic type.

Of the 19 patients who survived and left the hospital, 11 were free of ascites for follow up periods extending from three months to eight years. Nine of these had splenorenal or side to side portacaval shunts and two had disruption of suprahepatic vena caval membranes.[15] Six patients treated with sternalomentopexy and one by ileoentectropy were said to be improved but no further information given.[12] One patient treated by hepatic and splenic artery ligation, to reduce hepatic inflow, continued with ascites unchanged until death two years later with bleeding varices.[26] From this review, it would appear that in the presence of severe obstruction of the venous outflow tract of the liver control or cure of ascites may best be obtained surgically by measures which augment venous drainage of the liver.

Discussion

Hepatic vein catheterization and phlebography as a diagnostic method was reported in 1953 by Tori [37] who described normal hepatic vein anatomy in ten subjects and abnormalities associated with hepatic neoplasm and myeloid leukemia. Leevy and Gliedman [16] pointed out the value of hepatic phlebography in detecting hepatic metastases. Brink and Botha [3] were the first to confirm by preoperative phlebography the diagnosis of Budd-Chiari syndrome. This technic appears to be satisfactory for the study of anatomical changes in the hepatic veins of living cirrhotic patients. Despite the coarse grain of fast motion picture film, there was sufficient clarity of detail to permit recognition of individual hepatic veins from the size of the small interlobular collecting veins up to the major lobar trunks. Cinephlebography has certain advantages over single exposure technic in that all phases of filling and emptying, the effects of respiration, and the direction of flow of contrast medium may be visualized.[4] Single exposure

phlebography may have serious limitations, as indicated by two reports in which erroneous interpretation of flow direction was made.[9, 38] In these reports, single exposure roentgenograms showing retrograde portal branch filling during pressure injection of wedged hepatic vein catheters were presented as visual proof of the portal vein functioning as a venous outflow tract of the liver. In the present study employing cinephlebography, it was apparent that contrast medium which was forced retrograde through the sinusoids into portal radicles then drained back toward the sinusoids in normal direction of flow when injection was discontinued (Fig. 10).

In this small series, there appeared to be a close correlation between the severity of obliterative changes in the hepatic veins and intractability of ascites. Further studies of cirrhotic patients with chronic portal hypertension but without ascites are necessary to determine whether comparable changes in the hepatic veins may occur without ascites. It is possible that venous abnormalities due to nodular regeneration

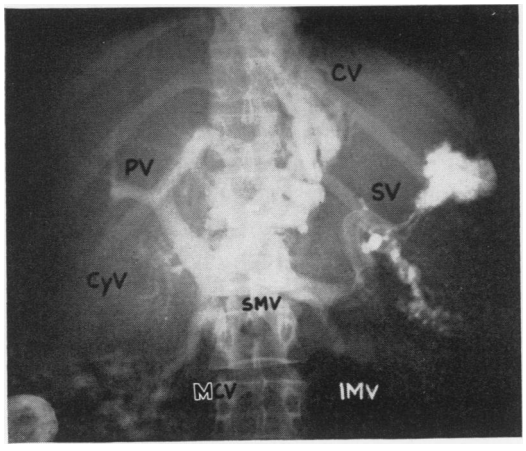


FIG. 9. Percutaneous splenoportogram of Case 16 demonstrating prograde flow into the portal vein and the liver despite virtually complete obstruction of the right hepatic veins. Serial films demonstrated simultaneous filling of the splenic vein (SV), the inferior mesenteric vein (IMV), superior mesenteric vein (SMV) and its middle colic branch (MCV) anastomosing with body wall veins, the cystic vein (CyV), and the portal vein (PV).

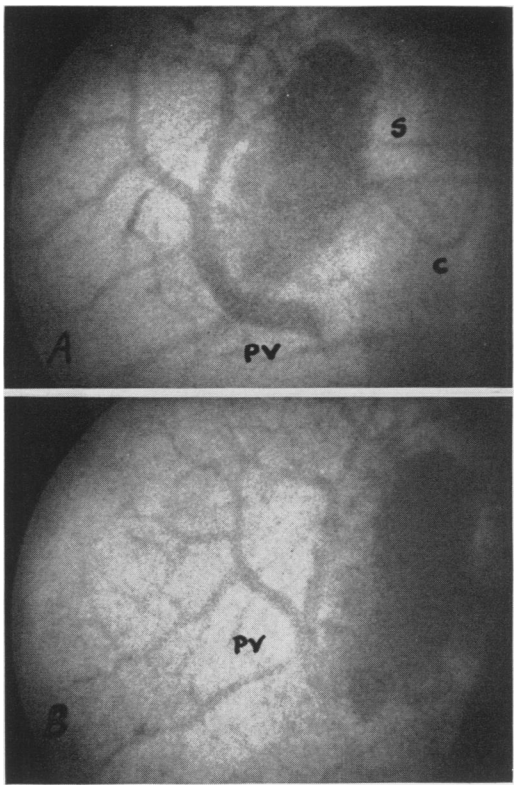


FIG. 10. A. Wedged phlebogram of the caudate lobe in Case 14 showing the catheter (C), a globular area of sinusoidal filling (S), and from the sinusoidal area retrograde filling of the right branch of the portal vein. B. A frame taken a few seconds after completion of injection showing drainage of contrast medium peripherally in the portal radicles toward the sinusoids.

of liver tissue involve both portal and hepatic vein branches in varying degree, and that in certain patients disproportionate involvement of the hepatic veins may occur. In this circumstance, ascites may appear early in the course of the disease as a result of impairment of hepatic venous drainage rather than as a manifestation of progressive hepatic deterioration.

To a limited extent, these studies in living patients provide anatomical support for the *outflow block* concept of intractable ascites. Although experimental and clinical ascites may result from obstruction of hepatic drainage, the mechanisms of ascites formation in all cirrhotic patients need not necessarily be due to hepatic veno-occlu-

TABLE 5. *Comparative Operative Portal Pressures**

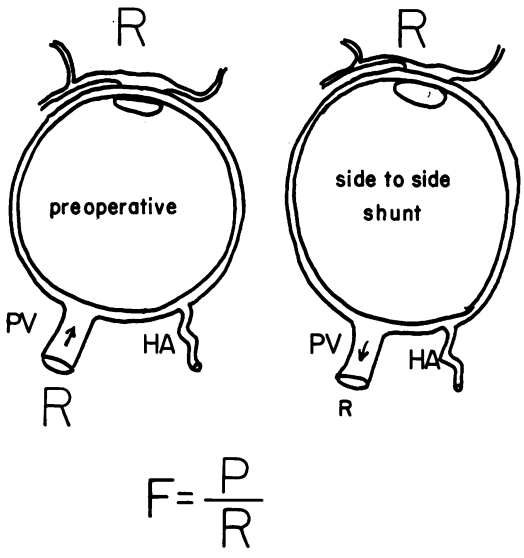
	FPP	OHPP	Change from FPP	OSPP	Change from FPP
Bleeders without ascites or history of ascites. (25 cases)	30.2	18.8	−38%	37.2	+20%
Bleeders with controlled ascites or history of ascites. (7 cases)	23.8	20.7	−13%	25.0	+ 5%
Chronic resistant ascites (4 cases)	23.5	23.8	+ 1.3%	19.8	−16%

* All pressures in mm Hg. as mean pressures.
FPP, Free portal pressure; OHPP, Occluded hepatic portal pressure; OSPP, Occluded splanchnic portal pressure.

sive disease.[24] On the other hand, in cirrhotic patients who do have severe veno-occlusive disease, impairment of hepatic drainage undoubtedly contributes to intractability of ascites. In such patients the nature of the obstructive process is such that spontaneous improvement, such as may occur with recanalization of thrombotic occlusion superimposed upon idiopathic stenosis or endophlebitis secondary to senecio alkaloid poisoning,[10, 23, 34] is unlikely to occur.

Advocates of the *outflow block* concept have suggested that a high outflow tract resistance may result in reversal of portal flow within the liver and that the portal vein thus becomes a venous outflow tract of the liver.[21, 25, 38, 39] Evidence cited to support this contention includes the observation that in certain cirrhotic patients with intractable ascites the occluded portal pressures may be reversed.[21, 25] Presumably, with a gradient pressure higher in the liver than in the portal bed, there is reversal of portal flow. Observations during the present study do not support this interpretation of reversal of operative occluded portal pressures. All four patients with Class IV veno-occlusive disease demonstrated prograde portal flow during splenoportography (Fig. 9). In Case 14, who had reversal of operative occluded portal pressures, reversal of intrahepatic portal flow was not seen when retrograde portal filling was observed during hepatic vein injection of contrast medium and at operation a prograde portal trunk flow of 300 ml./min. was recorded by flowmeter. Other investigators employing electromagnetic flow meters under similar circumstances have observed increased hepatic arterial flow with portal trunk occlusion at operation but have not observed reversal of portal flow in the absence of a portacaval shunt.[24, 32] Furthermore, comparison of operative occluded portal pressures in three groups of cirrhotic patients with bleeding varices without ascites, bleeding varices and controlled ascites, and intractable ascites, respectively, suggests that reversal of occluded portal pressures is an indication of increased hepatic arterial flow and the severity of outflow tract resistance and not of reversal of portal flow (Table 5).

Supporters of the side-to-side portacaval shunt for the treatment of refractory cirrhotic ascites contend that high sinusoidal pressure, secondary to outflow tract obstruction, may be relieved by hepatic decompression through the shunt (Fig. 11). Direct evidence that retrograde portal flow from the liver through the shunt may occur in patients with high outflow tract resistance has been obtained by phlebography[30] and by operative flow meter studies.[24] Indirect evidence is implied in the observation that relief of ascites in the



RETROGRADE PORTAL FLOW FOLLOWING SIDE TO SIDE PORTACAVAL SHUNT.

FIG. 11. Schematic representation of the hepatic lobule preoperatively (left) indicating high resistance in the deformed hepatic veins and in the portal bed. In order to maintain flow (F) into the liver through the portal vein (PV), sinusoidal pressure (P) must be high. After side-to-side portal shunt (right), with a low resistance to retrograde flow into intrahepatic portal radicles and through the shunt, hepatic arterial flow (HA) may be partly diverted into the shunt and sinusoidal hypertension relieved.

patients with Budd-Chiari syndrome was obtained only with surgical measures which augmented hepatic venous drainage. The success of the end-to-side portacaval shunt in controlling intractable ascites in certain cirrhotic patients raises the obvious question of the importance of outflow tract obstruction and of hepatic decompression in the genesis and surgical treatment of ascites.[1] It is possible that in most cirrhotic patients with ascites, near normal portal inflow, and moderate outflow tract deformity and resistance, ascites forms as a transudative process exceeding the drainage capacity of the hepatic lymphatics, as suggested by Rousselot.[24] Under these circumstances, portal diversion alone might reduce sinusoidal hypertension and ascites formation. The failure of the end-to-side shunt and hepatic artery ligation, both measures designed to reduce hepatic inflow, to control ascites in patients with Budd-Chiari syndrome suggests that in patients with severe outflow tract obstruction hepatic decompression is required for control of ascites.

Conclusions

Retrograde hepatic vein catheterization and cine-phlebography is a satisfactory method for the study of pathological changes in the hepatic veins not apparent by conventional diagnostic studies in living cirrhotic patients.

Chronic cirrhotic ascites resistant to medical treatment may be associated with severe hepatic vein occlusive disease without rapid hepatic decompensation.

Chronic cirrhotic ascites associated with severe hepatic veno-occlusive disease may be relieved by portal-systemic shunts which preserve the portal vein intact.

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