HEPATIC VEIN PRESSURE DETERMINATION AND PHLEBOGRAPHY IN THE EVALUATION OF PORTAL HYPERTENSION*

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In the light of the knowledge we now have of the subject, portal pressure determination and splenoportography are unquestionably valuable in the detection and diagnosis of portal hypertension.

Yet, not only do these tests entail a measure of risk for the patient, but they cannot always provide, at least as far as the angiographic aspect of the problem is concerned, all the data that would be required for a correct assessment of the degree of intrahepatic vascular involvement. Such a limitation originates from the fact that (*i*) in portal hypertension the blood stream is slowed down, and occasionally deviated through collateral channels, (*ii*) cases may even occur in which a prevalent stream is established or complete mixing of streams does not occur, and (*iii*) as recently reported, functional blocks at the hepatic hilum level are fairly often revealed by splenoportography. For these reasons, all patients reporting to our General Surgical Clinic to be examined for portal hypertension during the last 10 years have been examined by the method of hepatic vein phlebography and pressure determination.

Table I gives a summary of the portal hypertension cases who were submitted to our tests during the above-

TABLE I. PERSONAL CASES—A SUMMARY

Types of portal hypertension	*Investigated*	*Operated upon*	*Forms observed*	
Prehepatic obstruction	28	22	Portal thromboses:	
			In splenectomized subjects	8
			In splenomegalic subjects	11
			In cirrhotic and splenomegalic subjects	3
			Total	22
Intrahepatic obstruction	210	129	Cirrhoses:	
			Postnecrotic	36
			Alcoholic	73
			Prim. biliary	5
			Secondary to extrahepatic cholestasis	15
			Total	129
Post-hepatic obstruction	7	1	Right atrial malignancy	1

mentioned period, as classified according to the pre-, intra- or post-hepatic location of their obstructions. Out of a total of 245, 152 patients underwent surgery to establish a portosystemic derivation, and we were thus able, in a fairly large number of cases, to check the correctness of our pressure readings against surgical findings, as well as to compare the angiographic data we had obtained with the macroscopic and histopathological pictures provided by liver biopsies.

METHOD

Twin-lumen, balloon catheters† were used, which allowed both pressure readings to be obtained under free-flow, obstruction and block conditions, and angiograms to be taken of hepatic veins under block.

In our opinion, block pressure readings (Fig. 1) provide a way for checking occlusive pressure findings (parti-

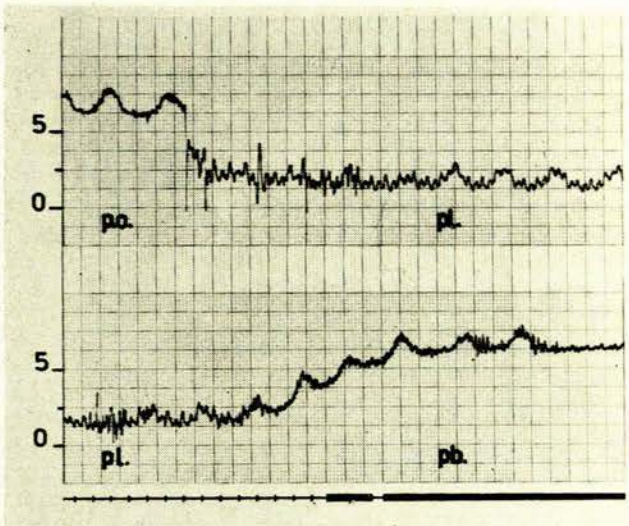

Fig. 1. Hepatic vein pressure readings in a healthy subject. Pressure values in mm.Hg. p.o. — occlusive pressure; p.l. — free-flow pressure; p.b. — blockade pressure (obtained by inflating the catheter balloon at the level of a major hepatic vein).

cularly when collateral circulation is present), while a comparison between block or occlusive pressure data and free-flow pressure readings enables the porto-suprahepatic gradient to be determined. In turn, the portacaval gradient can be determined from caval pressure readings.

Contrast-medium injections under block provide a good visualization of the suprahepatic district as cannot be obtained by other methods, mostly on account of the high pressure conditions that are often found in this area.

RESULTS

Pressure and Phlebographic Findings in Prehepatic Obstructions

Pressure and angiographic findings obtained in 28 cases of portal hypertension induced by a prehepatic obstruction are reported in Table II.

TABLE II. PORTAL HYPERTENSION FROM PREHEPATIC OBSTRUCTION (28 CASES INVESTIGATED, 22 OPERATED UPON)*

Pressure readings (mean values)			*Angiographic findings*	
Pre-operative hepatic veins (mm.Hg)		*Intra-operative vena porta root ($cm.H_2O$)*	*Hepatic vein phlebogram*	*Intra-op. portal phlebogram*
Occlus. press.	11			
Block press.	14	Portal press. 50	Bundled branches with interv. anastomoses	Portal thrombosis Portosystemic collateral circul. patterns
Free-flow press.	6			
V. cava inf.	3			
Gradients:				
Portacaval	9			
Portahepatic	5			

* The site of obstruction was correctly diagnosed in 22 cases (all those operated upon).

On account of the existence of a prehepatic obstruction, pressure readings obtained at hepatic vein level were found to be either normal or just above normal, as were

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†USCI No. 8.1/2 f. Dotter-Lukas type and Rush' No. 10 ch.

the portacaval and portahepatic gradients; while pre-operative portal pressure readings were the highest among all those obtained in portal hypertension cases submitted to surgery.

As far as phlebography was concerned, the most significant phlebograms showed a fairly regular vein pattern, occasionally associated with clustered divisional branches. In such cases, collateral circulation in the form of intervenous, intralobular anastomoses was a constant, conspicuous finding (Fig. 2).

Whenever a more complete inoculation could be performed, a certain degree of irregularity was evidenced, at least in cases where the prehepatic block was of longer standing. In 3 cases where marked vascular changes had been revealed by the angiograms, with altered patterns which definitely indicated a cirrhosis of the liver, surgery showed that the latter condition was associated with portal thrombosis

Pressure and Phlebographic Findings in Intrahepatic Obstructions

The most significant pressure and angiographic findings associated with 210 cases where portal hypertension had been induced by intrahepatic obstructions are reported in Table III. Of these, 129 underwent surgery (73 alcoholic cirrhoses, 36 postnecrotic cirrhoses and 20 biliary cirrhoses, some of them primary and some brought about by extrahepatic biliary stagnation).

TABLE III. PORTAL HYPERTENSION FROM INTRAHEPATIC OBSTRUCTION (210 CASES INVESTIGATED, 129 OPERATED UPON)*

Pressure readings (mean values)				*Angiographic findings*	
Pre-operative hepatic veins (mm.Hg)		*Intra-operative vena porta root ($cm.H_2O$)*		*Hepatic vein phlebogram*	*Intra-op. portal phlebogram*
Occlus. press.	35			Rigid vessels	
Block press.	35			Cut-off vess.	Rigid vessels
Free-flow press.	12	Portal press.	40	Distorted vess.	Distorted vessels
V. cava inf.	3			Acinous, mottled pattern	
Gradients:					
Portacaval	32				
Portahepatic	23				

* The site of obstruction was correctly diagnosed in 129 cases (all those operated upon). Correct pressure readings were done in 90 cases (70% of those operated upon) and undervalued in 39 cases (30% of those operated upon).

Occlusive pressure readings were virtually identical—at least as far as surgically-treated cases were concerned—while high portahepatic and portacaval gradients were noted. We felt that the degree of liver damage ought to be determined, as gauged by the degree of hypertension, the latter ranging from 18 to 40 mm.Hg.

Pressure readings likewise evidenced some increase in caval pressure, with a consequent reduction of the portacaval gradient in patients suffering from portal hypertension complicated by ascites.

Basically, phlebogram indications may be described as showing:

(*a*) Skimpier venous ramifications, the process being apparently proportional to the severity of the condition.

(*b*) Upset vasal structures resulting from the irregular arrangement and subdivision of individual venous branches (displacements and axial deviations), uneven distribution of vascular density and changes in the angles formed by collateral veins with their original trunk.

(*c*) Morphological changes of individual vasal elements in the form of irregular outlines, sudden strictures and/or obliterations (Fig. 3). (In some cases these alterations were just noticeable, while in others they appeared more obvious, and a relationship was apparent between the changes and the extent to which cirrhosis had evolved.)

(*d*) Alterations noted in the parenchymal injection stage, wherein the uniform opacity or cluster patterns of normal angiograms were replaced in cirrhotic cases by an irregularly mottled pattern, with an alternation of opaque and clear areas. (It should be noted here, however, that such a finding requires a considerable degree of contrast medium penetration, and may therefore be overlooked if the vessels are inadequately filled.)

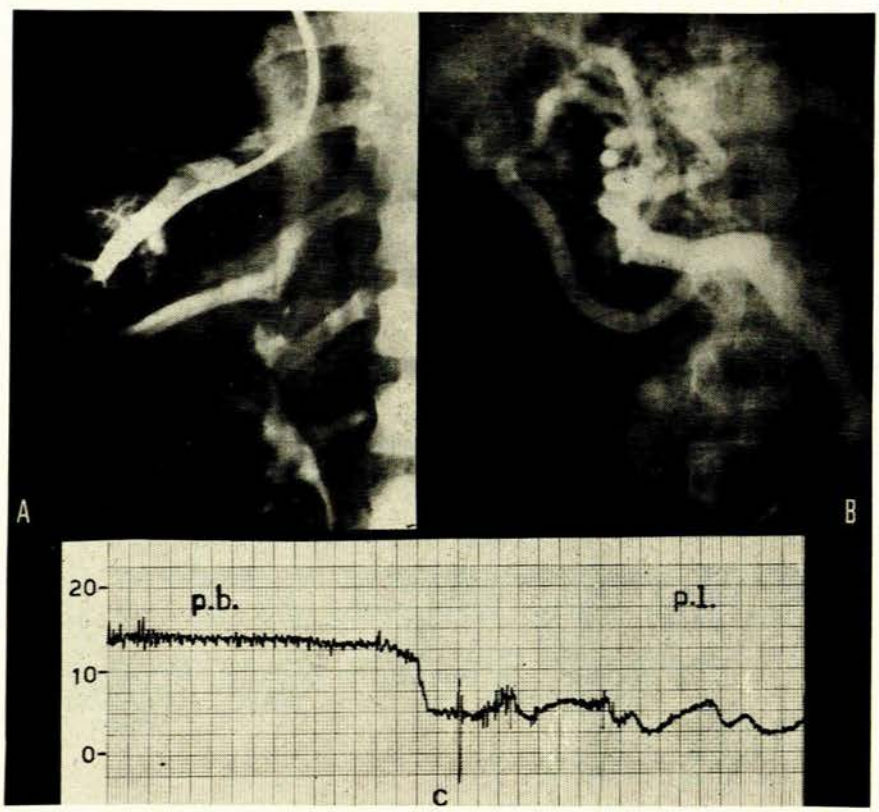

Fig. 2. Portal hypertension induced by prehepatic obstruction (portal cavernoma). A: Angiogram of the R.H. hepatic venous system. Vessel outlines have a fairly regular appearance, with conspicuous intersystemic collateral circulation patterns featuring separate outlets into the vena cava. B: Intra-operative portal phlebogram. Irregular portal vessel distribution throughout hepatic hilum demonstrates a cavernous angioma with conspicuous collateral circulation patterns. C: Hepatic vein pressure readings (mm. Hg), with blockade pressure (p.b.) slightly above normal.

(*e*) A possible visualization of vena porta branches, readily identified from their different distribution against the background of the hepatic shadow (intersystemic anastomoses), or of intervenous shunts (intrasystemic anastomoses). The former finding was noted in approximately 30% of the cases, and fairly consistently with primary biliary cirrhoses. The vena porta branches are most likely inoculated through newly-established direct connections between the hepatic veins and the portal system (Fig. 4). The latter finding (intervenous intersystemic anastomoses) was not apparently specific of cirrhosis, as it was constantly observed in biliary stagnation, echinococcal conditions and malignancies of the liver. Cirrhotic forms were nearly constant in those of a biliary origin, but in those of the Laënneckian kind they were present in no more than 20 cases. Two points which we could not conclusively determine were whether intervenous anastomoses are associated with ascitogenous or haemorrhagic cirrhosis, and whether any direct relationship exists between the presence of anastomoses and their magnitude, on the one hand, and the severity of the hypertensive condition on the other. Actually, while no such connection could be demonstrated, we would be rather inclined to assume that the condition might be somehow related to the stage of the disease, as it was often found in initial cirrhosis with enlargement of the liver, but never in long-standing forms associated with atrophy.

Pressure and Phlebographic Findings in Post-hepatic Obstruction

The results of our tests performed on 7 patients whose hepatic conditions were the result of stagnation, as well as on 2 with a caval obstruction syndrome (Table IV), appear to warrant the following remarks:

As far as pressure determinations are concerned, high occlusive, free-flow and caval pressure levels were noted in patients with a right-hand heart imbalance, where average values were 14, 14 and 13 mm.Hg respectively. It is therefore important that these readings can be extended to all explorable districts, as a diagnosis of this condition is grounded on high occlusive and free-flow pressure data, but first and foremost on caval hypertension findings. The result of all

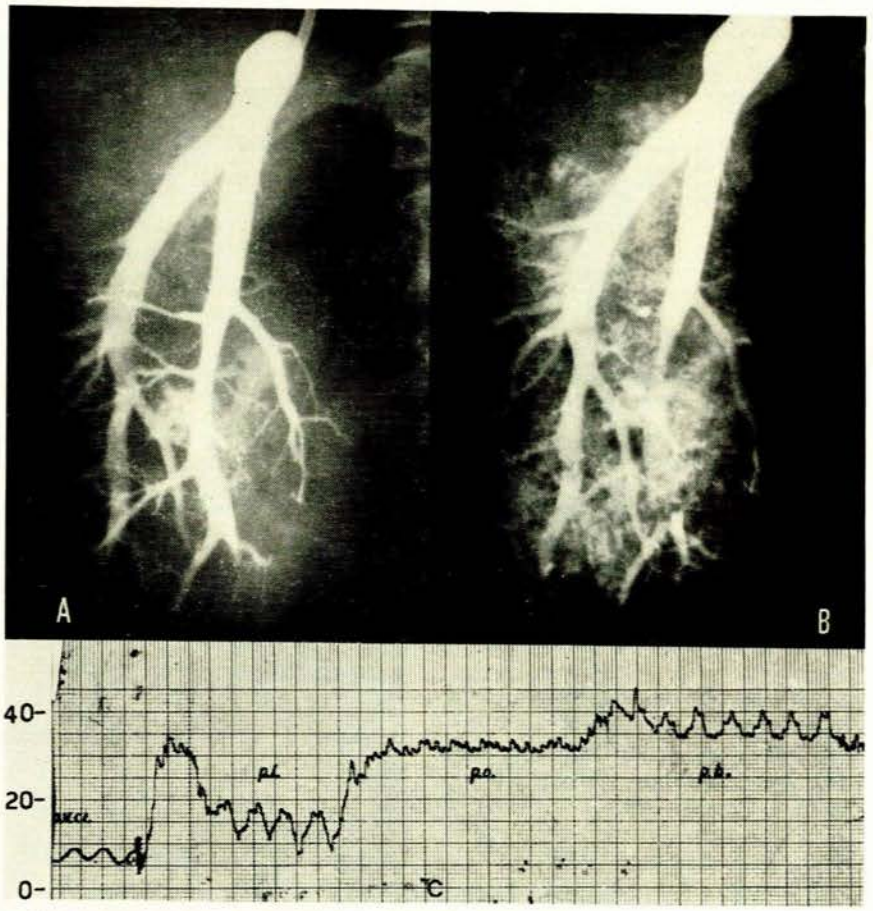

Fig. 3. Portal hypertension induced by intrahepatic obstructions (alcoholic cirrhosis). A: Angiographic stage featured by underfilling of R.H. hepatic venous system. Note 'starving' of venous branches accompanied by distortion. B: Parenchymatographic stage, featuring unevenly 'mottled' vascular pattern. C: Pressure determinations—occlusive (p.o.) and blockade (p.b.) pressure readings of 35 mm.Hg were obtained.

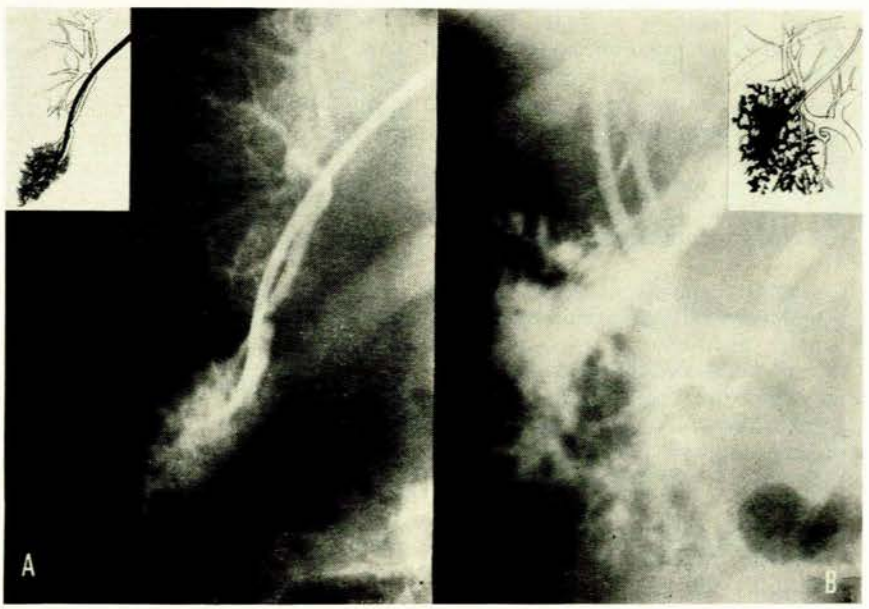

Fig. 4. Portal reflux patterns. A: Alcoholic cirrhosis—as a small peripheral segment was injected, typical vascular alterations became evident, together with a pronounced portal-branch reflux. B: Biliary cirrhosis. Note irregular intraparenchymatous vascular pattern, with phlebogram evidencing both R.H. and L.H. portal branches and the presence of intervenous reflux patterns between hepatic veins

TABLE IV. PORTAL HYPERTENSION FROM POST-HEPATIC OBSTRUCTION (7 CASES INVESTIGATED, 1 OPERATED UPON)*

Right heart imbalance			*Caval or hepatocaval obstruction*	
Pressure readings (mean values) (mm.Hg)		*Angiographic findings hepatic veins*	*Pressure readings (mean values) (mm.Hg)*	*Angiographic findings vena cava*
Occlus. press.	14	Widened vessels	Vena cava inferior 35	Vasal obstruction
Block press.	15	Open confluent angles		Refluxes
Free-flow press.	14			Collateral circulation
Vena cava inf.	14			
Gradients:				
Portacaval	1			
Portahep.	1			

* The site of obstruction was correctly diagnosed in 7 cases (all those investigated).

the data so obtained, depending on how seriously the venous reflux is impaired, may be a considerably reduced, if not nullified, portacaval pressure gradient.

With these patients, phlebograms are typical on account of the presence of dilated vessels arranged in a fan-like pattern and with wide branch-off angles. Although no alterations of the morphology of the finer veins were ever demonstrated by our parenchymatograms, such alterations may be noted in cases of chronic stagnation in the event of the hepatic damage evolving in a cirrhotic or precirrhotic direction and tending to induce a secondary portal hypertension.

Particularly interesting was the documentation we obtained of a right atrial malignancy in which the congestive liver condition induced by the restricted caval reflux was so serious as to have been complicated by ascites, while liver function appeared to be very seriously impaired. Pressure readings obtained in the superior and inferior venae cavae (since the suprahepatic veins could not be reached owing to the catheter being bent by the atrial outgrowth) were approximately 40 mm.Hg in both districts. An angiocardiogram subsequently provided additional evidence of the restricted venous reflux at atrial level on account of the tumour, while an ascending cavogram showed the block to be located at that level and the reflux to be occurring into the right-hand suprahepatic system (Fig. 5).

Another significant example of portal hypertension induced by a post-hepatic obstruction was offered by a case of Budd-Chiari's syndrome where only pressure readings and phlebograms allowed a diagnosis to be made of the condition. The symptoms had been an enlarged liver and ascites. As catheterization was performed, two facts were noted, viz.:

(*a*) The catheter could not be advanced to the level of the hepatic veins inlets or along the intrahepatic portion of the inferior vena cava (normal pressure readings were obtained for the unobstructed subdiaphragmatic portion of the inferior vena cava).

(*b*) The contrast medium proved the presence of a caval obstruction, whereas a thin vascular network could be noted in the regions of the suprahepatic inlets, whereupon ascending cavography by retrograde catheterization showed the location of the caval obstruction, extending over some 4 inches (corresponding to the intrahepatic portion of the cava), while caval circulation was being ensured by peri- and intraspinal collateral channels. Caval pressure readings for that tract were at the 35-mm.Hg level (Fig. 6).

CONCLUSIONS

The results we have been able to obtain by applying our hepatic phlebography and pressure-reading methods to 245 patients suffering, or suspected to be suffering, from portal hypertension have provided conclusive evidence of the complete reliability of the method in clinical diagnosis.

It proves to be irreplaceable with splenectomized patients; it allows the degree of portal hypertension to be accurately determined from occlusive and block pressure

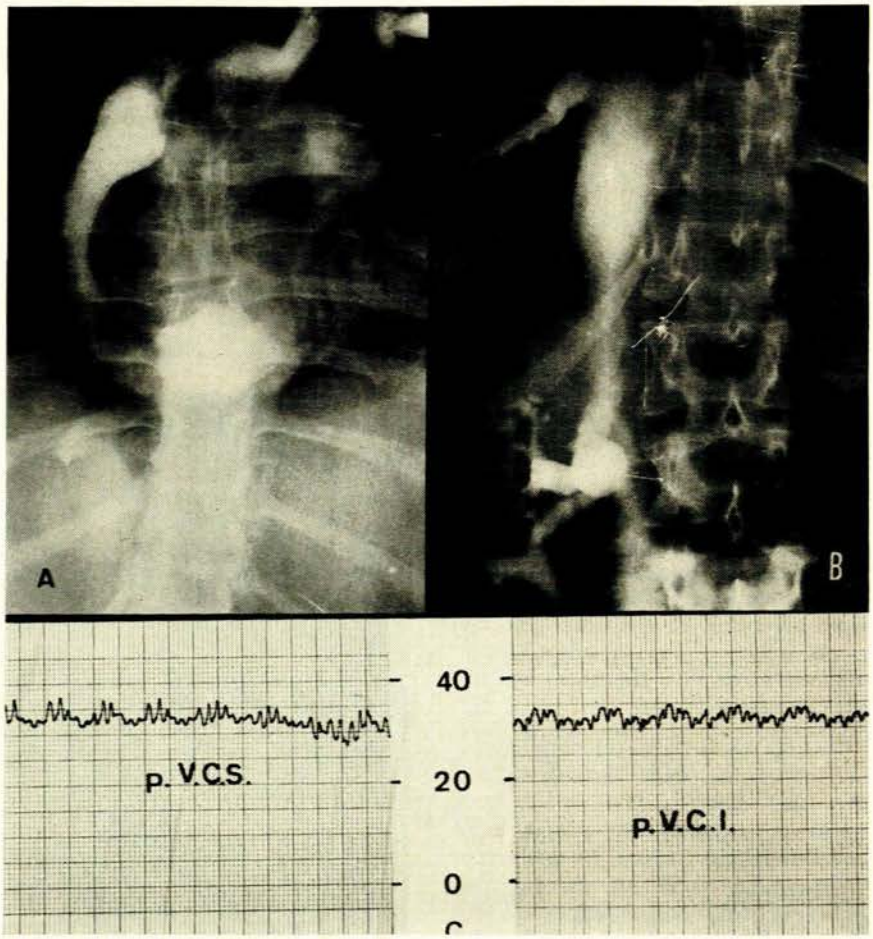

Fig. 5. Portal hypertension originating from a post-hepatic obstruction (R.H. atrium tumour). A: Angiocardiogram showing severely impaired atrial filling, resulting in lamination of contrast medium flow. B: Ascending caval phlebogram. Note complete throttling off of contrast medium at atrial inlet level, with reflux into R.H. hepatic and renal veins. C: Superior and inferior caval pressure readings (30 mm.Hg).

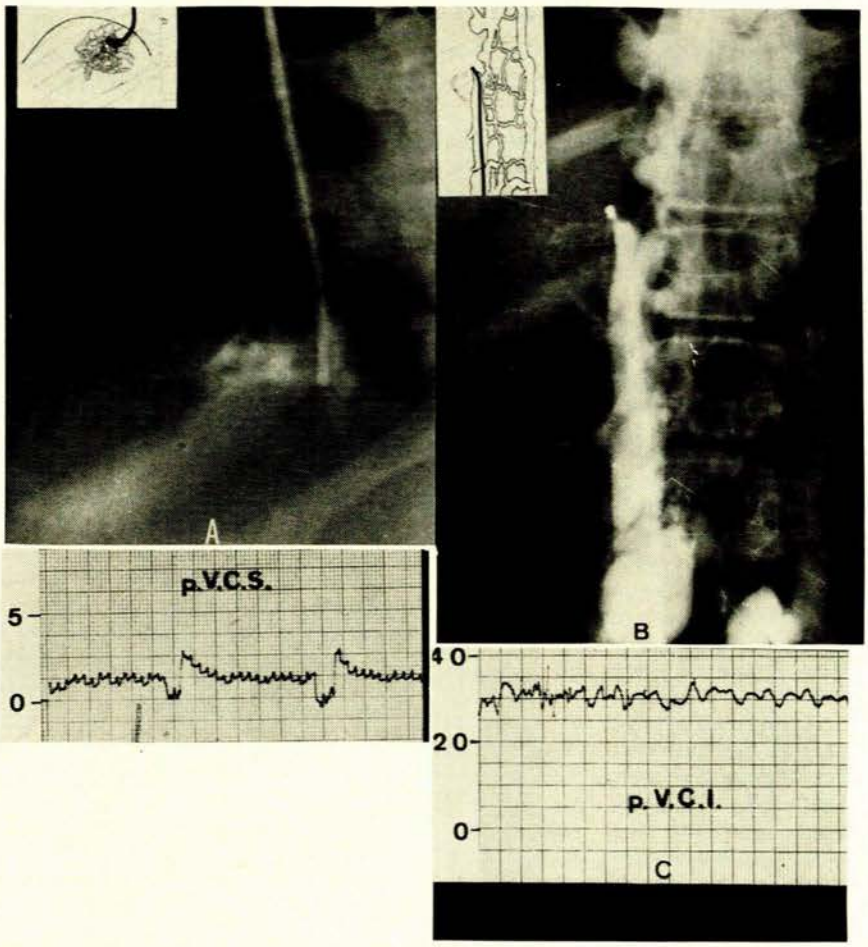

Fig. 6. Hypertension induced by post-hepatic obstruction (Budd-Chiari's syndrome). A: Catheter is detained at hepatocaval inlet, resulting in incomplete contrast medium injection. B: Caval phlebogram—catheter detained in intrahepatic section of vena cava—several compensatory collateral circulation patterns demonstrated by contrast medium injection. C: Pressure readings with normal values were obtained for the supradiaphragmatic vena cava *vs.* 35 mm.Hg for the inferior vena cava. p.V.C.I. = inferior vena cava pressure; p.V.C.S. = suprahepatic inferior vena cava pressure.

readings; and it provides a means for detecting the existence or threat of a cirrhotic condition of the liver (often the cause of portal hypertension), as well as for assessing its type and stage with sufficient accuracy.

Clinical experience, more than theoretical considerations, substantiates our contention that the method discussed should be preferred to splenoportography—at least in all cases where hypertension is not the result of an obstruction in the splenoportal trunk—although even in this instance the possibility of the obstruction being diagnosed through a mere hepatic catheterization cannot be ruled out. The latter test has been providing us with ample grounds for concluding in favour of surgery for subjects with portal thrombosis without having to resort to splenoportography, a technique which we performed only in cases where the need for it was recognized.

SUMMARY

Suprahepatic vein catheterization with related pressure and phlebographic findings, as applied to over 200 subjects suffering—or suspected to be suffering—from portal hypertension, made possible:

1. The determination of individual free, occlusive and block pressure readings (and, with the block pressure readings, enabled one to check occlusive pressure under various portal hypertension conditions).

2. Early detection of cirrhotic-type liver damage from altered suprahepatic vein branches in portal hypertension induced by intrahepatic obstruction.

3. Investigation of phlebographic features which are typical of suprahepatic veins in cases of prehepatic obstruction.

4. Identification of portal hypertension due to suprahepatic or suprahepatic-caval obstruction.

5. Investigation of phlebographic features typical of portal hypertension induced by Hanot's disease.

6. The determination of the process whereby collateral circulation patterns are established under varying portal hypertension conditions.

On the grounds of experience and of the results so obtained, suprahepatic vein pressure determination and phlebography should be regarded as a basically important method for an accurate diagnosis of portal hypertension.