Percutaneous Transhepatic Portography. I. Technique and Application

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Percutaneous transhepatic portography with selective catheterization of the portal vein and its tributaries was performed on 120 patients, of whom 71% had cirrhosis of the liver. The technique was improved by ultrasonically guided puncture, and the procedure was successful in 96% of the examinations. Collateral veins were visualized in 81% and esophageal or gastric varices in 69% of the patients with portal hypertension. The procedure was performed with little risk and discomfort, and portograms of high quality were obtained. Other applications of percutaneous transhepatic catheterization of the portal vein system are discussed.

Radiologic visualization of the portal venous system through injection of contrast medium has been performed by direct puncture during celiotomy [1], splenoportography [2, 3], exposures of the venous phase of celiac and mesenteric arteriography [4], percutaneous transhepatic puncture of the portal system [5, 6], injection via the reopened umbilical vein [7], and by transjugular transhepatic puncture of the portal vein [8]. Percutaneous transhepatic portography has attracted increasing attention during recent years [9–15].

In cirrhosis of the liver, percutaneous transhepatic portography evaluates hemodynamic changes, visualizes varices and other collateral systems, demonstrates the patency of the portal and splenic vein, and checks the patency of surgically established portocaval shunts [9, 13–15]. The examination is also indicated in hepatomegaly, splenomegaly, and suspected tumors along the splenoportal pathway [16].

Catheterization of the portal venous system may also be used for nonoperative obliteration of esophageal varices [17, 18]. Recently the procedure has been used to diagnose and localize endocrine pancreatic tumors [19–21]. We have used the percutaneous transhepatic portography since 1972.

Subjects and Methods

Percutaneous transhepatic portography was performed with a 27-cm-long, smooth polyethylene catheter fitted with a metal mandrin (Wiechel-Stille, Sweden), 1.00 mm ID/1.45 mm OD. It was introduced under local anesthesia in the right midaxillary line under fluoroscopic control with the patient supine. To improve the success rate of portal vein puncture, the porta hepatis was localized by ultrasonic scanning prior to portography [12]. From the ultrasonic scan, surface coordinates were marked on the patient (fig. 1). The aim was to puncture a hilar portal vein, usually the right branch. If unsuccessful, the procedure was repeated by slightly altering the direction of the catheter without completely withdrawing it from the liver. Using a flexible guide wire (PE 160) with slightly curved tip, the catheter was conducted from the intrahepatic portal vein into the branches of the portal system.

The first exposures were ordinarily made with the tip of the catheter in the splenic hilum. Sodium methylglucamine diatrizoate 76% was injected at a rate of 6 ml/sec (30 ml total). Films were exposed at one per second for 10 sec and then one every other second for 10 sec.

Portal pressure was recorded on a water manometer with the catheter tip in the main portal vein. The reference level was the midaxillary line; the recorded pressure was the mean of three readings.

Selected patients had sclerotherapy with injection of hydroxypolyaetoxy-dodecane (Aethoxysklerol, Kreussler, West Germany) in selectively catheterized varices (fig. 2). During injection, blood flow in the varices was stopped by inflation of a Sengstaken-Blakemore tube.

In patients with endocrine pancreatic tumors, the pancreatic veins were catheterized selectively and blood sampled for hormone assay. Blood samples for reference values were obtained from a percutaneous transhepatic catheterized hepatic vein.

At the end of examination, the catheter was clamped and retracted until the tip of the catheter was left in the liver parenchyma. It was withdrawn gradually during the next 6–12 hr.

From 1972 through 1978, 120 patients, 36 female and 84 male (median age, 52 years; range, 1 month to 87 years) were studied with percutaneous transhepatic portography. Eight patients were studied twice (128 total investigations). Diagnoses are shown in table 1. Two cases of portal vein thrombosis were secondary to cirrhosis.

In the evaluation of results the following definitions were used:

Successful transhepatic portography was defined as catheterization and visualization of the portal veins.

Direction of the flow in the portal vein was defined as *hepatopetal* if all contrast medium flowed through the liver, *complete hepatofugal* if contrast medium flowed to collateral veins with no flow through the liver, and *partial hepatofugal* if a great part of contrast medium flowed through collateral veins.

Liver size was defined as *normal* if the hepatogram reached the costal margin, the L1 vertebra, and the cardia; as *less than normal* if the liver was clearly within these limits; and as *enlarged* if the liver exceeded one or more of these boundaries. *Left lobe hypertrophy* denoted a liver extending far into the left hypochondrium.

Portal hypertension was defined as portal venous pressure above 20 cm of water.

Results

Percutaneous transhepatic portography was successful in 123 of the 128 investigations (96%). The main portal vein was catheterized in 106 (83%), and the splenic

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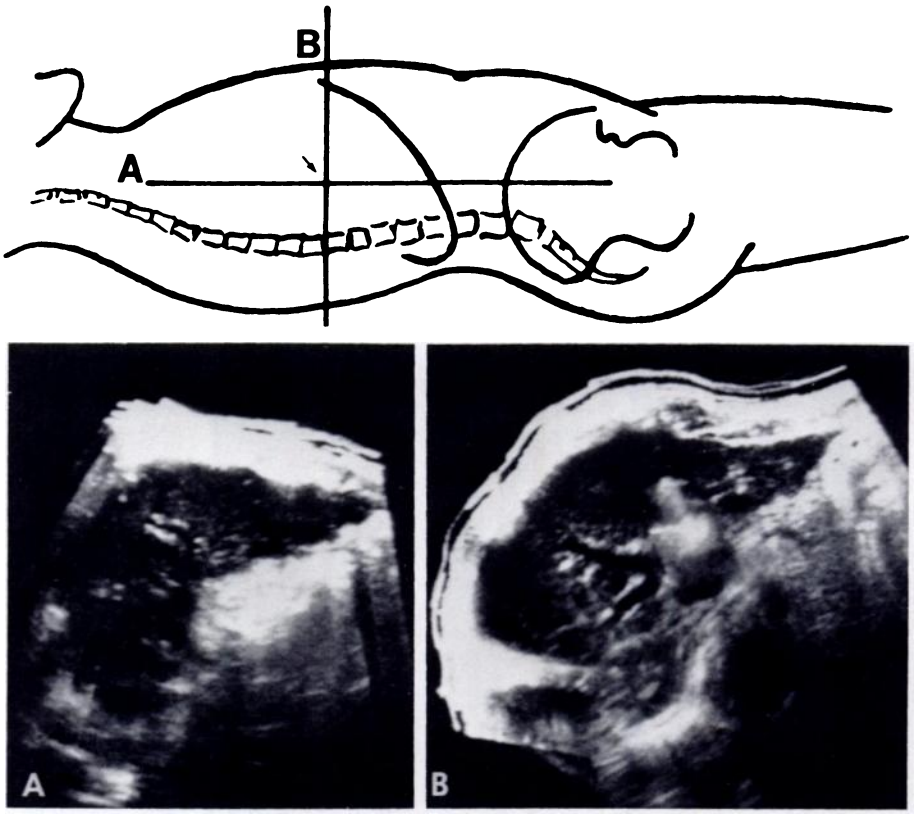

Fig. 1.—Puncture site (*arrow*) determined by scans used for localization of porta hepatis. Branching of right portal vein is shown.

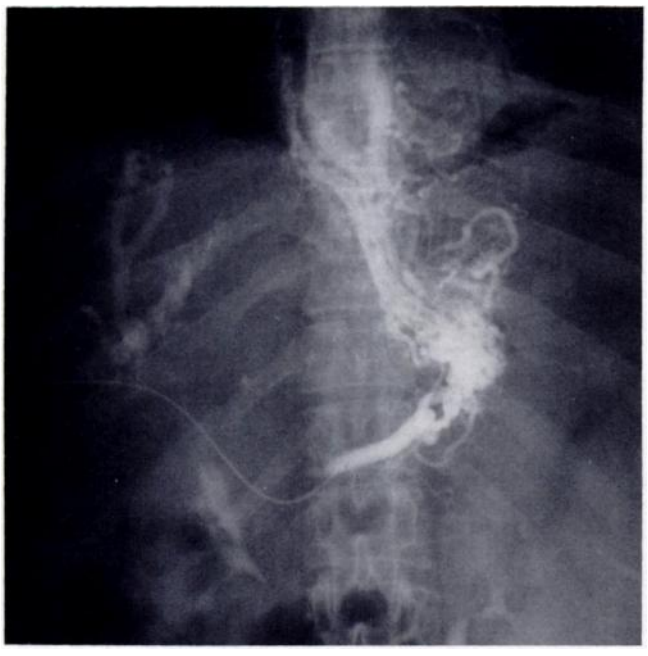

Fig. 2.—Selective catheterization of coronary vein shows esophageal and fundic varices.

TABLE 1
Diagnosis

Diagnosis	No. Patients
Intrahepatic obstruction (cirrhosis):	
Alcoholic	61
Cryptogenic	11
Chronic hepatitis	9
Primary biliary	2
Secondary biliary	2
Extrahepatic obstruction (thrombosis):	
Portal vein	6
Hepatic veins	1
Inferior vena cava	1
Biliary obstruction:	
Chronic pancreatitis	4
Pancreatic carcinoma	3
Common duct stenosis	2
Common duct stone	1
Lymphoma in porta hepatis	1
Toxic hepatitis (intrahepatic bile duct obstruction)	1
Biliary atresia	1
Other:	
Gastrinoma	12
Suspected gastrinoma	1
Insulinoma	1
Total	120

vein in 98 (77%). Portal vein catheterization was unsuccessful in six cases due to portal vein thrombosis. In an additional 11 cases the guide wire could not be passed from tortuous intrahepatic portal veins into the main portal vein.

The direction of blood flow could be estimated in 106 patients; it was hepatopetal in 35, partial hepatofugal in 59 (fig. 3), and complete hepatofugal in 12 (fig. 4). All patients with hepatofugal flow had intra- or extrahepatic obstruction.

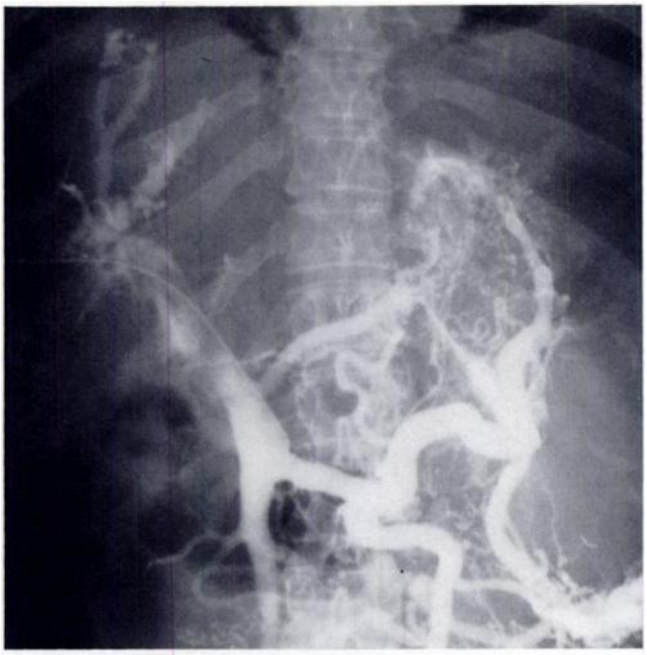

Fig. 3.—Widespread collateral veins from coronary vein, splenic hilum veins, and inferior mesenteric vein. Catheter lies in main portal stem. Flow in portal vein system is partial hepatofugal. Only sparse filling of intrahepatic portal veins. (Same patient as in fig. 2).

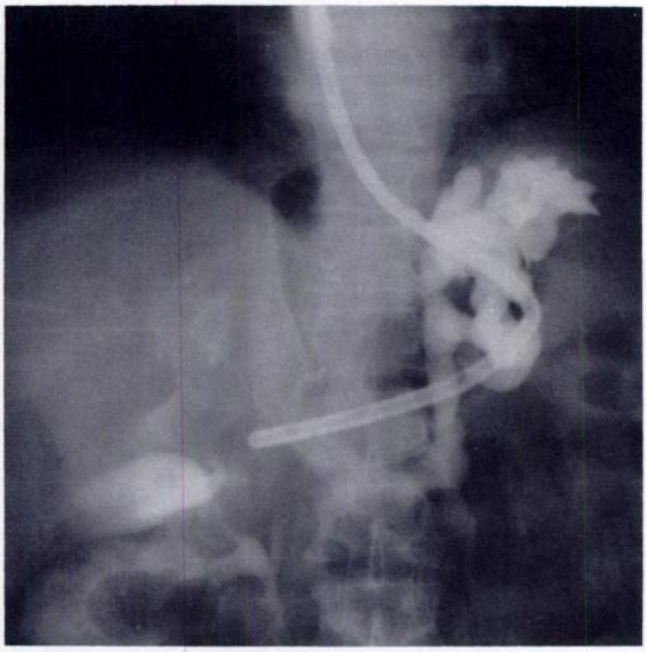

Fig. 4.—Retroperitoneal, paravertebral collaterals to left renal vein and inferior vena cava. Flow is complete hepatofugal.

The type and number of collateral veins are shown in table 2. In 18 of the patients with cirrhosis or extrahepatic obstruction no collaterals were visualized. The most commonly observed collateral pathways to the superior vena cava were the coronary veins, short gastric veins, and esophageal venous plexus (figs. 2 and 3). Next most frequently observed were collaterals to the inferior vena

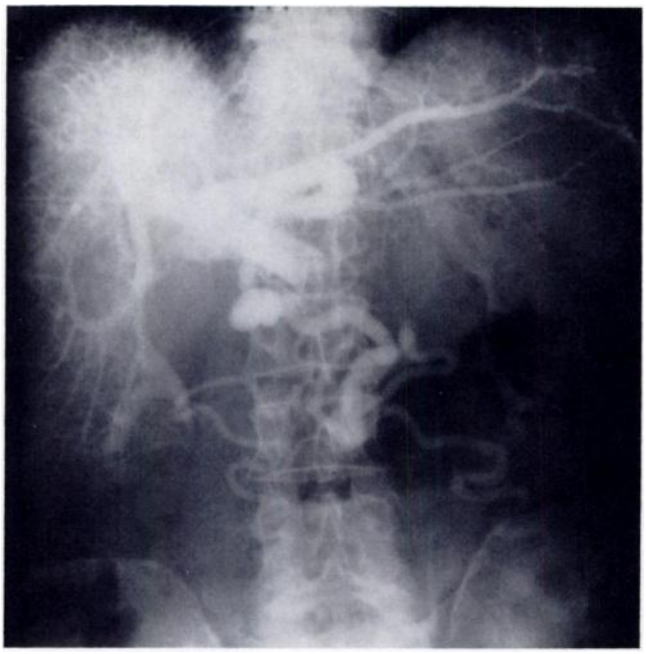

Fig. 5.—Recanalized umbilical vein connecting left portal branch and veins in abdominal wall (Caput Medusae). Left hepatic lobe is hypertrophied. Irregular and deviating intrahepatic portal vessels.

TABLE 2

Visualization of Collateral Vein System in Patients with Portal Hypertension

Type of Collateral Vein System	No. Patients (N = 94)
Coronary	60
From inferior mesenteric vein	38
Short gastric	22
Retroperitoneal-paravertebral	22
Recanalized umbilical	19
From superior mesenteric vein	13
From splenic hilum	12
From liver hilum	11
To abdominal wall	7
To left renal vein	4
Other	19

cava via the inferior mesenteric vein (fig. 3), followed by retroperitoneal veins (fig. 4), and recanalized umbilical veins (fig. 5). Less frequently collaterals via the superior mesenteric vein (fig. 6) and other types of collaterals were seen. Most patients with collateral veins had two or more collateral systems visualized. In nine patients the vena cava could be seen (fig. 4).

Of the patients with intra- or extrahepatic obstruction, 63 (69%) had visible varices. Esophageal varices alone were seen in 16 patients, and gastric varices alone in nine patients. Both esophageal and gastric varices were seen in 38 patients.

All the patients with extrahepatic portal obstruction had them demonstrated by the transhepatic portography. Stenosis of the main portal vein was demonstrated in six patients (fig. 7). The diagnoses are shown in table

3. Four of these patients had minor collateral veins, but pressures below 20 cm.

In 79 of the patients with intra- or extrahepatic obstruction, the portal venous pressure was measured. The mean portal pressure was 34.5 cm water (SD 9.6). The highest pressures were found in patients with marked collaterals to the superior vena cava. In 16 of the patients with biliary obstruction or endocrine pancreatic tumors, the mean portal pressure was 15.9 cm water (SD 5.1).

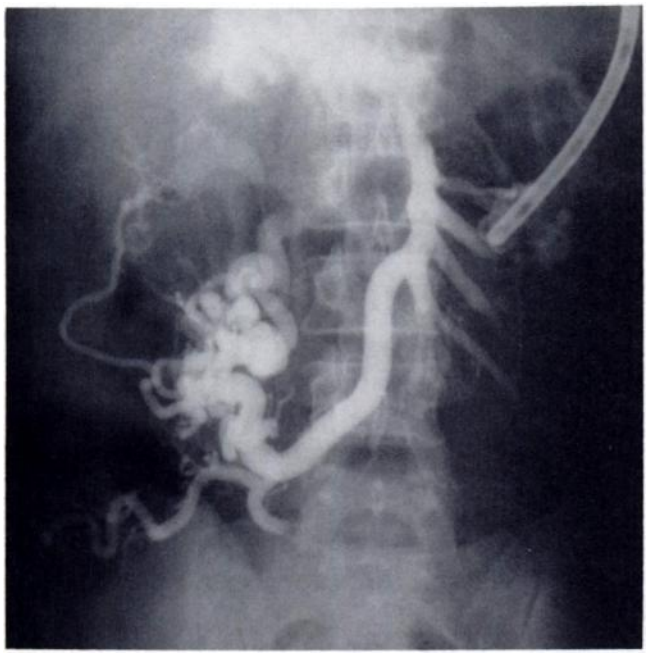

Fig. 6.—Selective catheterization of superior mesenteric vein. Collaterals around cecum and ascending colon.

The liver size could be estimated in 108 patients (90%). In 75 of the patients with cirrhosis the liver size could be estimated. The liver was normal in 40 (53%), smaller than normal in 24 (32%), and enlarged in 11 (15%); 22 (29%) had left lobe hypertrophy (fig. 5). In 33 patients without cirrhosis the liver size was normal.

In four patients 4–10 ml of 3% Aethoxysklerol was injected selectively into esophageal varices. A few minutes after injection, contrast medium flow stopped in the varices. Two of the patients had the catheter left in the coronary vein for repeated injection of Aethoxysklerol for 2 days. All patients suffered rebleeding from esophageal varices 2–4 weeks later, and portography showed recanalization of varices and formation of new collaterals to the varices.

The results of localization of the endocrine pancreatic tumors will be published elsewhere [21].

One patient had inadvertent puncture of the right flexure of colon causing no symptoms. Otherwise no complications were recorded.

Discussion

Percutaneous transhepatic puncture is the most direct route to catheterization of the portal venous system. Portography by this approach is probably the superior method for radiologic delineation of the portal venous system [22].

In normal subjects the porta hepatis is placed about 3 cm right of the vertebral spine and about the width of a vertebral body anterior to the T12 vertebra. Our portograms showed that this prediction may be as much as 7 cm off in cirrhosis. By the ultrasonic scanning method for localization of the porta hepatis, the number of puncture attempts can be reduced significantly [12].

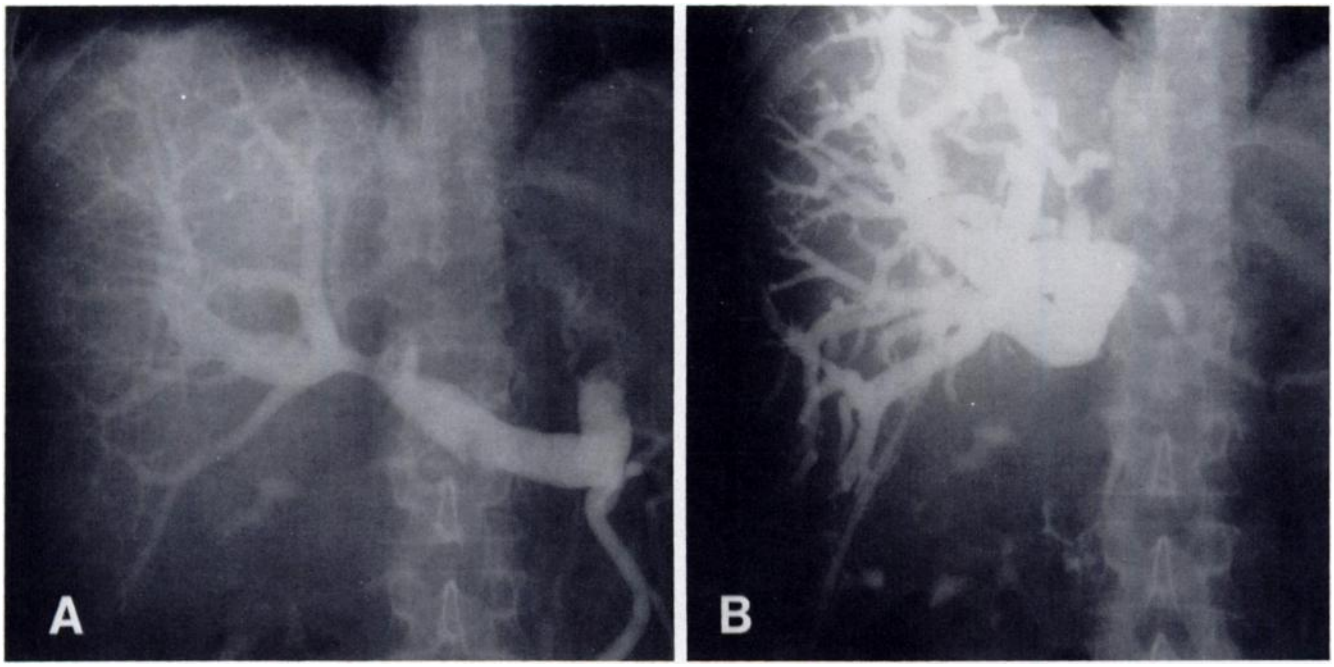

Fig. 7.— **A**, Stenosis of portal vein and no filling of left portal branch due to lymphoma in porta hepatis. **B**, Percutaneous transhepatic cholangiography. Total obstruction in porta hepatis.

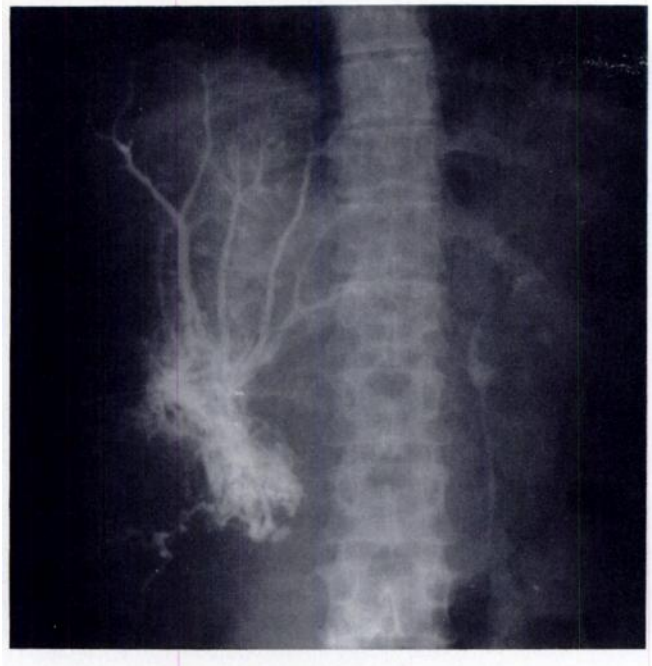

Fig. 8.—Occlusion of extrahepatic portal vein. Paraportal tortuous collaterals and some normal intrahepatic portal veins.

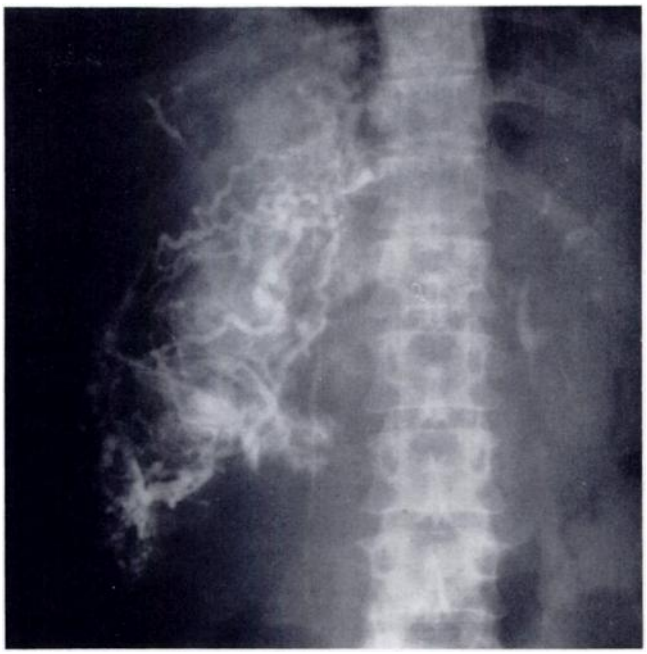

Fig. 9.—Occlusion of hepatic veins (Budd-Chiari syndrome). Intrahepatic tortuous collaterals.

TABLE 3

Diagnoses in Patients with Visualization of Stenosis of Main Portal Vein

Diagnosis	No. Patients
Carcinoma in head of pancreas	1
Gastrinoma in head of pancreas	1
Lymphoma in porta hepatis	1
Chronic pancreatitis	1
Postoperative iatrogenic stenosis	2
Total	6

Other investigators have used cholecystography or the venous phase of celiac and mesenteric arteriography to establish the anatomic position of the right portal vein [14, 15], but these methods are more cumbersome or invasive.

Puncture in the right midaxillary line secures a convenient angle of entry into the right portal branch, provides more parenchymal thickness for protection from bleeding, prevents injury of other intraabdominal organs, and eases manipulation of the catheter and guide wire.

In cirrhosis the intrahepatic portal veins are often irregular with a course markedly deviating from the normal (fig. 5) making catheterization of the extrahepatic portal system impossible in some cases. The left portal branch is often dilated, because of collateral circulation via the umbilical vein [23] or hypertrophy of the left lobe (fig. 5).

Location, number, size, and flow pattern of the collateral veins in relation to the portal venous pressure may have therapeutic implications [24]. Communications to the superior vena cava through gastroesophageal varices and a high portal pressure seem to be correlated with severe bleeding from the varices. Large communications to the inferior vena cava through retroperitoneal collaterals with opacification of the inferior vena cava and a less elevated portal pressure are correlated with minor or no gastroesophageal varices. These communications represent the most desirable route of natural decompression. Unfortunately they are a less frequent collateral system.

Extrahepatic obstruction of the portal vein is usually caused by thrombosis. With complete obstruction, the radiologic diagnosis is made by demonstration of paraportal tortuous collaterals (fig. 8). Obstruction may also be caused by tumors along the vein; it is usually incomplete and its exact site and extent can be demonstrated (fig. 7). Obstruction of the hepatic veins should be suspected when tortuous intrahepatic collaterals (fig. 9) accompany decreased or hepatofugal flow in the portal veins.

The preliminary results of obliteration of esophageal varices [17, 18] were promising, but recanalization occurred after a few weeks in about 80% of the patients [25]. The method can be used to stop actual bleeding and has been successful in about half of the cases [17, 18, 25].

The preliminary results of selective catheterization of pancreatic veins with blood sampling and hormone assay for localization of endocrine pancreatic tumors have been encouraging. The number of patients examined is still small. The largest group consists of patients with gastrinomas, in whom the localization was successful in about 80% [21].

Catheterization of the portal venous system may also be valuable in pharmacologic, metabolic, and hemodynamic studies. It is possible to leave the catheter in the portal venous system for several days for such studies.

The most often reported complications with transhe-

patic catheterizations are intraperitoneal bleeding and bile leakage from the puncture site. To avoid these, the puncture canal can be sealed with an acrylate compound [26], or by injection of autologous clots, thromboplastin, or gelatin foam [14, 15]. We have used gradual withdrawal of the clamped catheter over 6–12 hr, which allows the puncture canal time to seal. We use the same procedure in percutaneous transhepatic cholangiography. In 200 investigations, we have had two cases with bleeding episodes and two with bile leakage. However, in all four patients the catheter retracted completely in immediate connection with the procedure.

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