Some Limitations of Splenic Portography:

I. Incidence, Hemodynamics and Surgical Implications of the Nonvisualized Portal Vein *

ALBERT R. BURCHELL,** M.D., AUGUSTO H. MORENO,*** M.D., WILLIAM F. PANKE, M.D., LOUIS M. ROUSSELOT, M.D.

From the Departments of Surgery, St. Vincent's Hospital and Medical Center of New York and New York University School of Medicine, New York City

SPLENIC PORTOGRAPHY has gained increasing acceptance in the evaluation of anatomic and hemodynamic changes associated with portal hypertension. With an experience of more than 900 examinations we have had a reasonable opportunity to assess the usefulness of this method as well as to become aware of some of its inherent limitations. Although our overall experience has been most satisfactory, an analysis of some of the limitations of portography would seem appropriate at this time.

Apart from innate hazards and technical failures which could be met by the occasional practitioner, there are instances in which a splenic portogram, although technically successful, does not provide complete information as to the integrity of the splenorenal axis. The best example would be when a portogram demonstrates esophagogastric varices and other routes of hepatic bypass, allowing for a diagnosis of portal hypertension, but the portal vein, or the entire splenorenal axis, is not outlined by contrast medium, leaving unknown whether the portal vein is patent or occluded. In our earlier experiences with splenic portography we assumed that nonvisualization was associated with occlusion and, on this basis, a few patients were denied portacaval anastomosis. This assumption was shown to be incorrect by subsequent surgical exploration or postmortem examination, demonstrating that some of these patients had a completely normal portal vein.

Whether a patent portal vein is not visualized because the contrast medium is diverted through hepatic bypass routes [13] or because blood actually is flowing away from the liver in the portal vein,[4, 12] the fact remains that splenic portography does not permit differentiation beween an occluded and patent nonvisualized portal vein.[3] We viewed the following as pertinent considerations: 1) incidence of nonvisualization in a large series of splenic portograms; 2) incidence of nonvisualized portal veins which actually are patent and thereby suitable for portacaval anastomosis; 3) possible usefulness of other methods to determine the patency of a nonvisualized portal vein; 4) the best approach to surgical management of patients with portal hypertension and a nonvisualized portal vein; 5) hemodynamic significance of a nonvisualized but patent portal vein. The last consideration may relate to the hy-

* Submitted for publication November 18, 1964.

** Postdoctoral Research Fellow, National Institutes of Health.

*** Career Scientist Award I-123, Health Research Council of the City of New York.

This work was supported in part by Research Contract U-1032 from the Health Research Council of the City of New York and PHS Grant HE 06087-06.

potheses that in certain cirrhotic patients, particularly those with associated ascites, the portal vein may already have assumed the role of accessory outflow tract.[2, 14, 15]

This report is based on 56 patients with portal hypertension in whom the portal vein was not visualized during an otherwise satisfactory splenic portogram and in some of whom additional information was obtained by intrahepatic parenchymal deposition of contrast medium [5-10] and by electromagnetic measurements of direction and rate of flow in the portal vein.

Clinical Material and Methods

Nine hundred and four splenic portograms were performed in 735 patients by a consistent technic reported elsewhere.[11, 13] In 56 patients technically successful splenic portograms failed to visualize the portal vein. Not included in this group are patients with splenoportographic evidence of portal thrombosis or obstruction, when bridging collaterals or irregular outline of the portal vein confirmed the presence of thrombosis and eliminated the diagnostic problem. In patients with nonvisualized portal veins additional studies were performed: 1) direct portograms at operation by cannulation of a mesenteric branch in six patients; 2) intrahepatic parenchymal deposition of contrast medium in 18 patients (in 5 this study was repeated, for a total of 23); 3) direct measurement of direction and rate of flow in the portal vein at operation (square wave electromagnetic flowmeter and noncannulating probes) in 14 patients. A Schonander rapid film changer was used to record 14 intrahepatic depositions and nine were recorded by cinefluorography.

Anatomic patency or occlusion of the portal vein was verified in 45 of 56 patients whose portal veins were not visualized by splenic portography. Verification was obtained by operative exploration, postmortem examination or intrahepatic parenchymal deposition of contrast medium. Of 11 patients in whom the condition of the portal vein was not determined, six died and postmortem examination was not obtained, two are alive and a positive diagnosis has not yet been established and three have been lost to follow up.

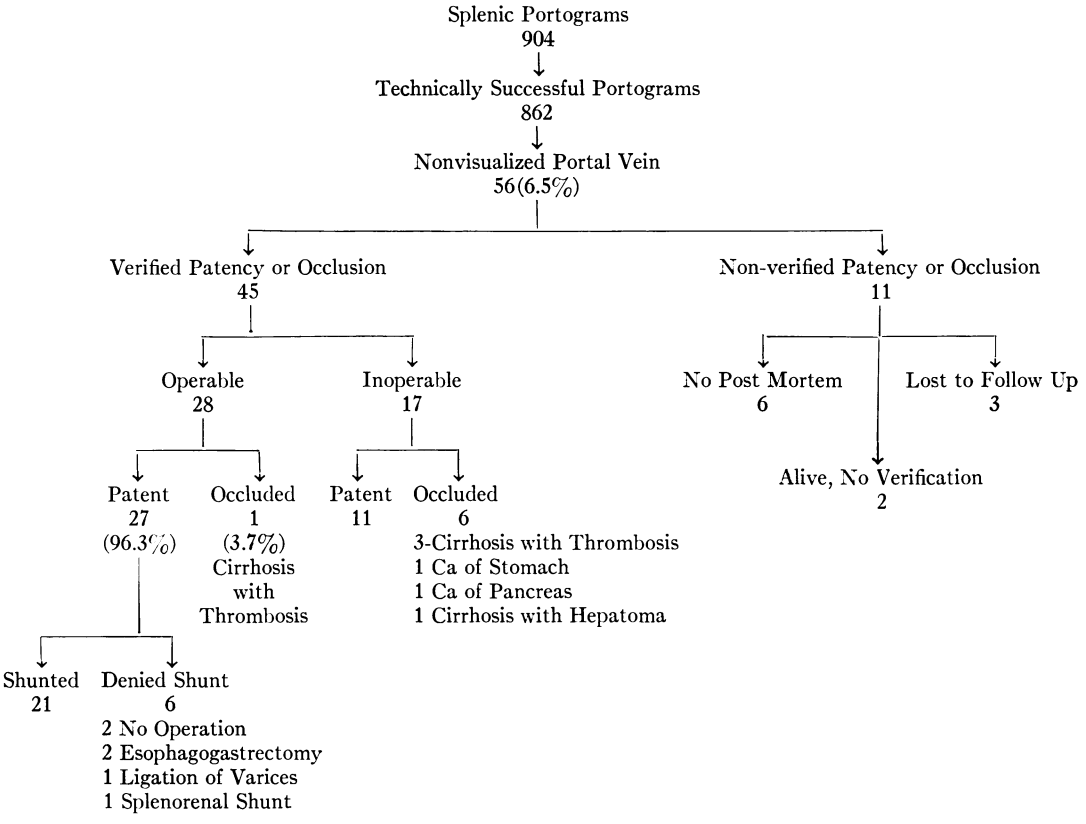
Results

Incidence of Nonvisualization of the Portal Vein

Of 904 splenic portograms in this series, 862 were considered technically successful on the basis of satisfactory deposit of contrast medium in the splenic pulp, lack of significant subcapsular suffusion and sufficient opacification of the splenic or collateral veins. Accordingly, sufficient contrast medium was available to outline the portal vein, but it was not visualized in 56 patients (6.5%).

Our *overall incidence* of failure to visualize the portal vein may not reflect the true clinical significance of this diagnostic limitation of the technic, since the patients we have studied by portography in a comprehensive research program had such a wide range of pathologic involvement. The severity of portal hypertension varied from early uncomplicated cases in our patients who did not require surgical intervention to advanced cases in patients for whom any form of operation had become contraindicated. Somewhere between these two extremes was a group in need of shunt and in satisfactory condition to undergo the procedure. This latter group is usually in the majority on the average surgical service. In our experience with 152 such patients the portal vein was not outlined in 27 technically satisfactory splenic portograms (17.8%). This *relative incidence* should be more meaningful clinically. Thus, splenic portography indicated the presence of portal hypertension and esophagogastric varices but did not show the portal vein in more than one sixth of those patients

TABLE 1. *Summary of Distribution of Patients with a Nonvisualized Portal Vein*



requiring a decision regarding surgical intervention.

Incidence of Anatomically Patent Portal Veins Not Visualized During Splenic Portography

Anatomic patency was verified in 38 of 45 patients whose portal veins were not visualized during technically successful splenic portography, an *overall incidence* of 84 per cent. Of seven patients with proved occlusion, four had cirrhosis with secondary portal thrombosis, one had cirrhosis with a hepatoma extending into the portal vein and two had extrahepatic portal block due to carcinomatous invasion from the stomach and pancreas, respectively.

Here again, these data represent a heterogeneous group of patients. When those with malignant and advanced liver disease are discounted, there are 28 patients requiring and able to undergo portacaval anastomosis. In 27 the vessel was patent, a *relative incidence* of 96.3 per cent. A diagram of the incidence of nonvisualized portal veins and anatomic patency is presented in Table 1.

Usefulness of Other Methods to Determine Patency of Nonvisualized Portal Veins

Portal Portography. Direct portography by cannulation of a venous mesenteric branch was performed during operation in six patients in whom the portal vein was not visualized by previous splenic portography. In four patients a normally patent portal vein was outlined; in two the portal vein was not visualized and the contrast

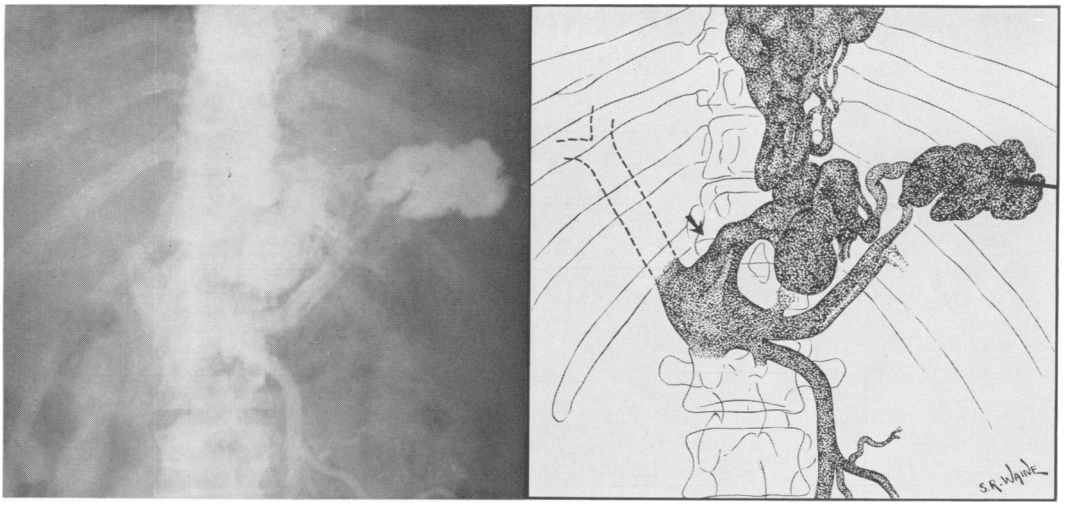


FIG. 1. Bypass of the liver via the coronary system.
In 45% of cases of a nonvisualized but patent portal vein the predominant route of hepatic bypass was noted to be over a dilated coronary system (arrow) to the azygos and superior vena cava.

material was diverted through a very large coronary vein and a left adreno-renal-caval "natural shunt," respectively. In one patient failure of visualization by both splenic and portal portography was interpreted as representing occlusion and operation was discontinued. Postmortem findings, following death from abrupt varical hemorrhage, demonstrated the portal vein to be patent. In the other patient operation was continued, a patent vessel was found and a satisfactory shunt was constructed. These results have discouraged our routine use of this method following nonvisualization, although we still believe that it has substantial merit for the post-splenectomy patient.

Intrahepatic Parenchymal Deposition. This method, originally devised to examine the natural outflow tracts of the liver—the hepatic vein system—gained some unexpected usefulness in the study of portal hemodynamics. Although the method permitted patency to be established in eight of 14 patients whose portal vein was not visualized by splenic portography, its primary value was the demonstration of the hemodynamics responsible for nonvisualization. We do not believe that this procedure, which is not without potential dangers, is justified for routine clinical use.

Surgical Management of Patients with Portal Hypertension and Nonvisualized Portal Veins

According to our data, although splenic portography fails to visualize the portal vein in about one of six patients who require and can tolerate operation, there is about a 96 per cent probability of patency in such instances. Accordingly, pre- or intraoperative use of other diagnostic methods would not seem necessary, and operative exploration of all patients in this category would seem indicated. Our practice is to perform a limited subcostal incision and immediately palpate and then explore the portal vein. In 21 of 22 patients the vein was found to be patent, the incision was completed and the anastomosis was accomplished. If the portal vein is occluded an alternative type of anastomosis (splenorenal or caval mesenteric) could be established, either at that time or as a subsequent procedure.

The consequences of assuming that a

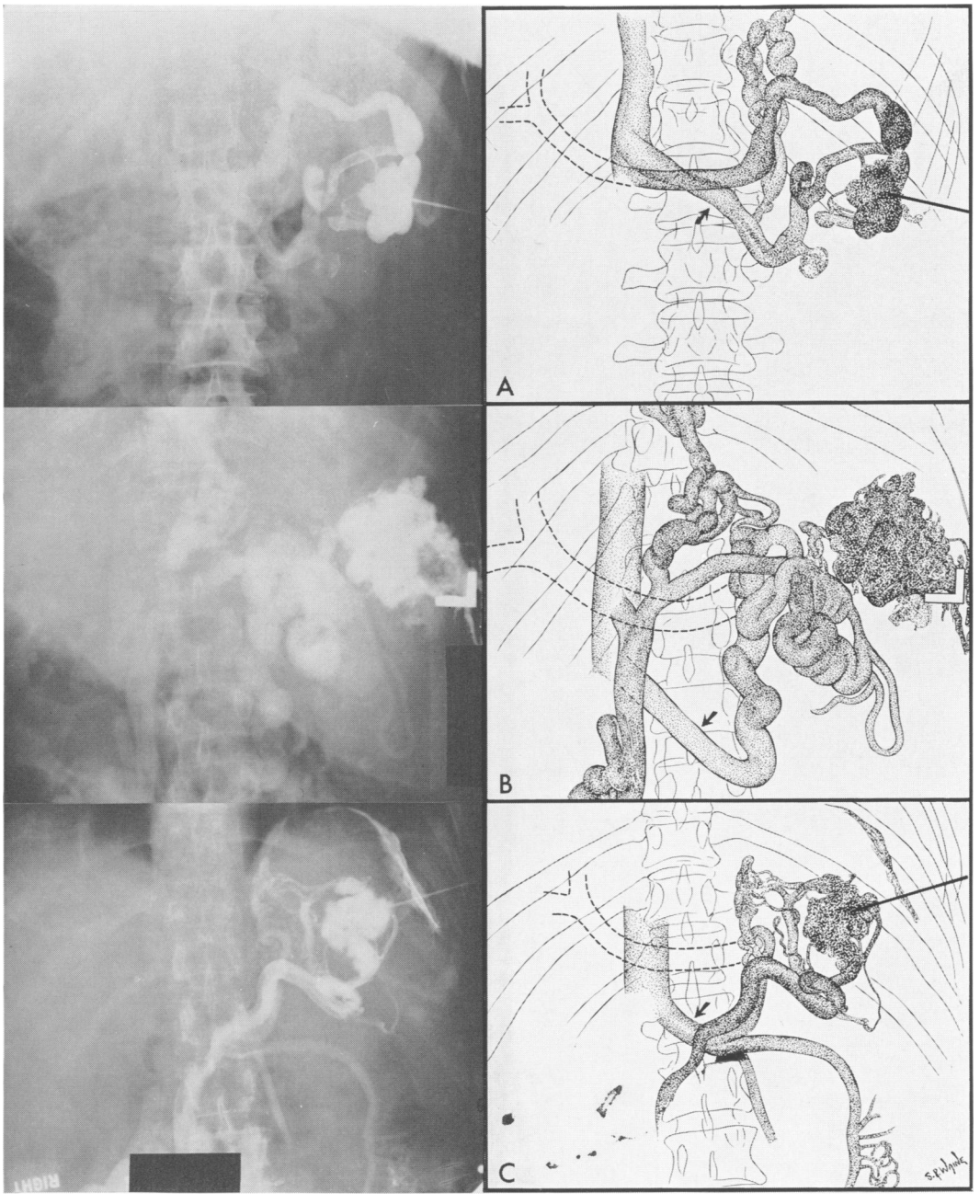
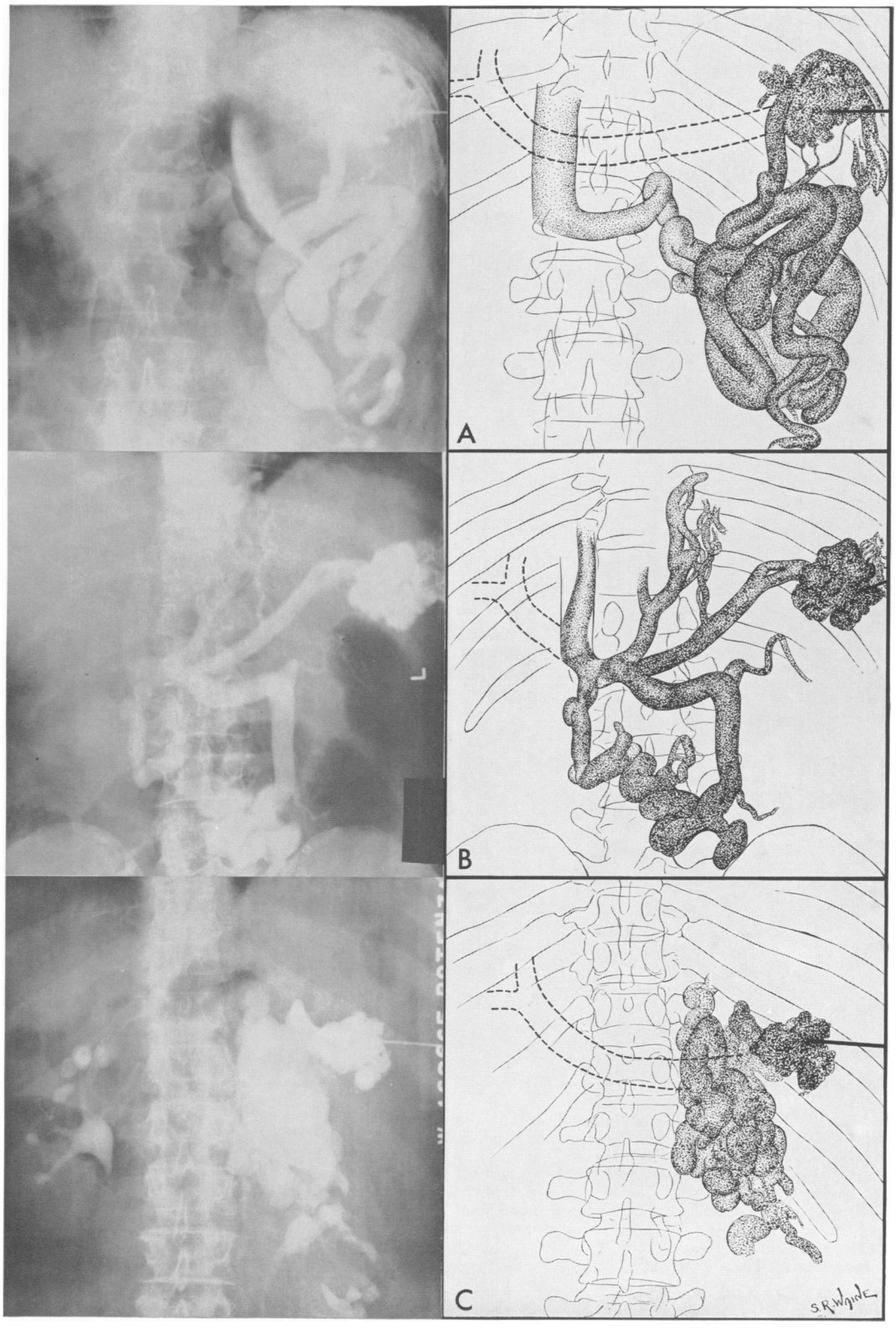


FIG. 2. Adreno-renal-caval "natural shunt" about the liver.

In another 45% of patients with a nonvisualized but patent portal vein the primary route of hepatic bypass was over an adreno-renal-caval "natural shunt" (arrows) to the inferior vena cava. In A, there is the slightest suggestion of a small amount of contrast outlining the portal vein, but insufficient for a diagnostic conclusion as to vessel patency. In B and C neither the splenic nor portal veins are outlined, a phenomenon that occurred in 53% of subjects with an adreno-renal-caval route of bypass and only 12% of patients with a coronary type of bypass. As discussed in the text, this complete lack of information concerning major vessel patency poses a particular tactical problem in the surgical management of such patients.


A
B
C
S.R. Wynne

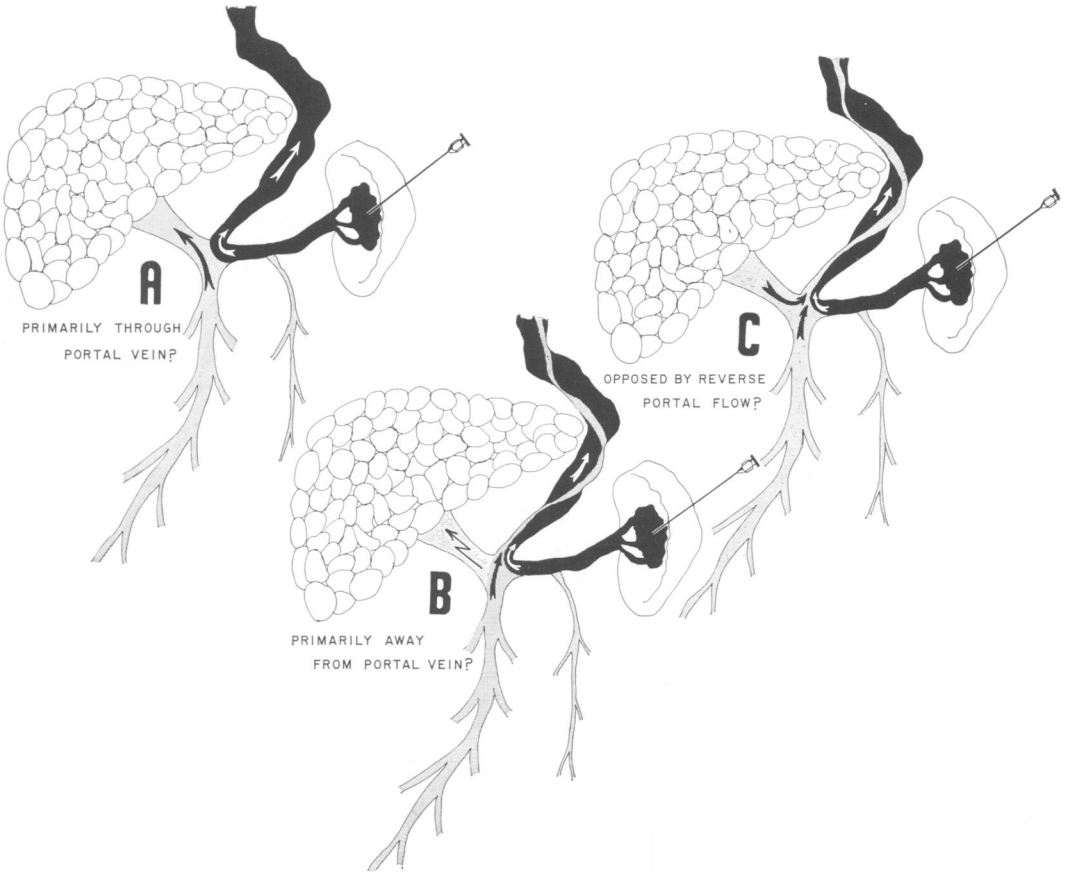


FIG. 4. Three theoretical possibilities as to the hemodynamic arrangement that results in nonvisualization of the portal vein.

A. Nonvisualization of the portal vein is the result of the simple interposition of a large bypassing collateral between the site of injection in the spleen and the portal vein itself. The portal vein still continues to carry to the liver the large volume of "unstained" blood from the rest of the splanchnic vascular bed.

B. As in A, the contrast-stained blood from the spleen has been diverted away from the liver by a collateral vessel that is interposed before the origin of the portal vein. However, most of the splanchnic venous return is also diverted away from the liver, so that the flow of "unstained" splanchnic blood through the portal vein is greatly reduced or even ceases completely.

C. It might be postulated that the contrast deposited in the spleen cannot enter and opacify the portal vein because of the opposition of true reverse flow of hepatic arterial blood in the portal vein.

The data obtained by means of direct electromagnetic flowmeter studies and other radiographic technics that supports our favoring the second possibility (B) is presented and discussed in the text.

FIG. 3. Unidentified routes of hepatic bypass.

In 10% of cases of a nonvisualized but patent portal vein, the route of hepatic bypass may be via enormously dilated and unidentifiable venous channels that can result in a variety of unusual radiographic patterns. The finding of such bizarre pathways of collateralization with a nonvisualized portal vein should not distract the surgeon from the statistical fact of a high incidence of patency in such portal veins and should not serve as a deterrent from surgical exploration.

TABLE 2. *Portal Venous Flow in 14 Cirrhotic Patients with Portal Hypertension and a Nonvisualized Portal Vein (Square-Wave Electromagnetic Flowmeter)*

Patient	ml./min.	ml./Kg./min.	Direction
A. A.	0	0	Stagnant
E. S.	0	0	Stagnant
F. E.	0	0	Stagnant
A. H.	80	0.6	Toward liver
P. O'C.	110	1.6	Toward liver
R. K.	128	1.6	Toward liver
W. McM.	158	2.2	Toward liver
E. C.	190	2.6	Toward liver
A. C.	340	4.5	Toward liver
R. C.	438	8.8	Toward liver
R. W.	578	8.7	Toward liver
P. C.	610	8.7	Toward liver
V. P.	631	6.6	Toward liver
M. C.	1144	20.6	Toward liver
Mean ± S.D.	314.8 ± 319.5	4.75 ± 5.47	

nonvisualized portal vein is occluded were demonstrated by six patients denied portacaval anastomosis during our early experience with splenic portography. In the first patient operation was discontinued after both splenic and portal portography failed to outline the portal vein. The patient was discharged and died of recurrent varical hemorrhage. The second and third patients received extensive esophagogastric resections; one died of recurrent varical hemorrhage and the other following complications of colonic esophageal replacement. The fourth patient died of varical hemorrhage after transesophageal ligation of varices. The fifth patient was not operated upon and died with massive ascites and spontaneous perforation of the umbilicus. At postmortem examination a completely patent portal vein was found in all five patients. The sixth patient had a splenorenal shunt and is alive.

Hemodynamic Significance of the Nonvisualized but Patent Portal Vein

In most patients in this category, routes of hepatic bypass were so prominent and dilated that a large amount of splanchnic blood, perhaps most of it, seemed to be diverted. In about one half of the patients bypass was established through a massively dilated coronary system (Fig. 1). In the other half the primary route appeared to be a large adreno-renal-caval "natural shunt" (Fig. 2), except for four patients in whom there was a variety of communicating pathways (Fig. 3).

In 12 patients not only the portal vein but the entire splenoportal axis was not visualized; the contrast medium injected into the spleen was diverted into systemic circulation via large "natural shunts" without outlining the splenic portal vein and without indicating the fate of blood in the remainder of the splanchnic bed (Fig. 2, 3). From the diagnostic and tactical points of view these patients presented a particularly difficult problem because information was lacking as to the availability of both the portal and splenic veins for either a portacaval or a splenorenal anastomosis.

When the portal vein is not outlined following a satisfactory deposit of contrast medium into the splenic pulp there are several alternatives. 1) Routes of hepatic bypass are interposed between the site of injection and the portal vein, the latter still carrying to the liver "unstained" blood from the remaining areas of the splanchnic bed (Fig. 4A). 2) Most splanchnic blood is diverted via the routes of hepatic bypass and flow of "unstained" blood in the portal vein becomes very small or ceases (Fig. 4B). 3) Contrast medium cannot enter the portal vein because it is opposed by hepatic blood actually flowing away from the liver (Fig. 4C). The third possibility, suggested by other investigators [4] and at one time by

TABLE 3. *Portal Venous Flow in 6 Normal Subjects (Square-Wave Electromagnetic Flowmeter)*

Patient	ml./min.	ml./Kg./min.	Direction
C. O'B.	869	13.4	Toward liver
J. R.	1,161	19.1	Toward liver
E. W.	1,345	20.7	Toward liver
A. H.	1,413	22.6	Toward liver
M. G.	1,491	23.5	Toward liver
V. S.	1,514	26.3	Toward liver
Mean ± S.D.	1,299 ± 223	20.9 ± 4.1	

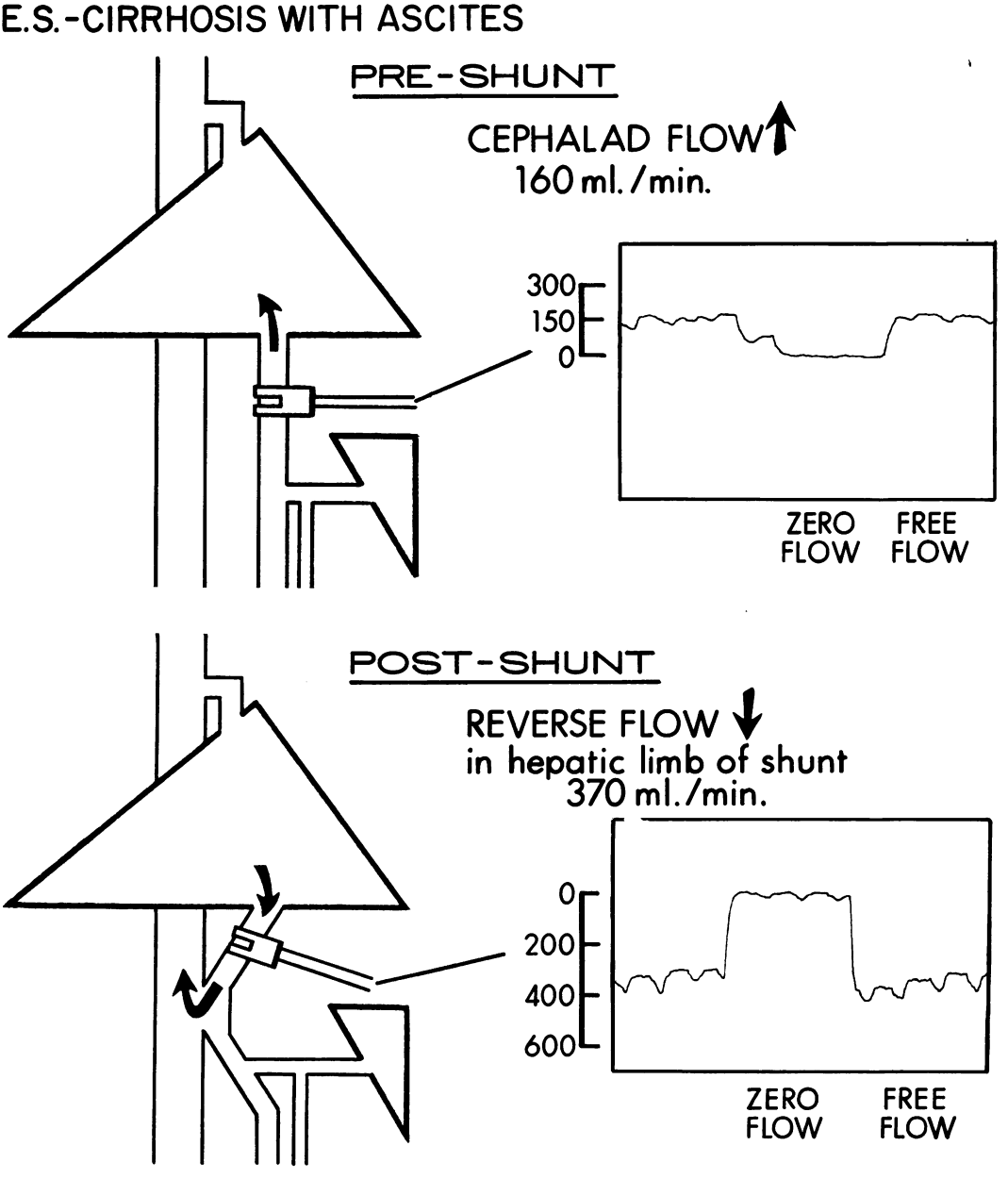


FIG. 5. Electromagnetic flowmeter demonstration of true reverse flow in the cephalic limb of the portal vein following a side-to-side portacaval shunt.

Prior to shunt, this patient had a greatly diminished portal flow although in the normal direction towards the liver as shown in the upper recording by the electromagnetic flowmeter. After completion of the side-to-side anastomosis, the lower recording demonstrates actual reversal of portal flow, with the cephalic limb of the side-to-side shunt now functioning as an accessory outflow tract. Such reverse flow was never observed in the pre-shunt intact portal vein of 72 cirrhotic patients studied by this technic.

ourselves,[12] supports the premise that following postsinusoidal obstruction in cirrhosis the portal vein decompresses the liver by spontaneously assuming the role of an accessory outflow tract.[2, 14, 15] It is noteworthy that 43 of 56 patients (77%) did

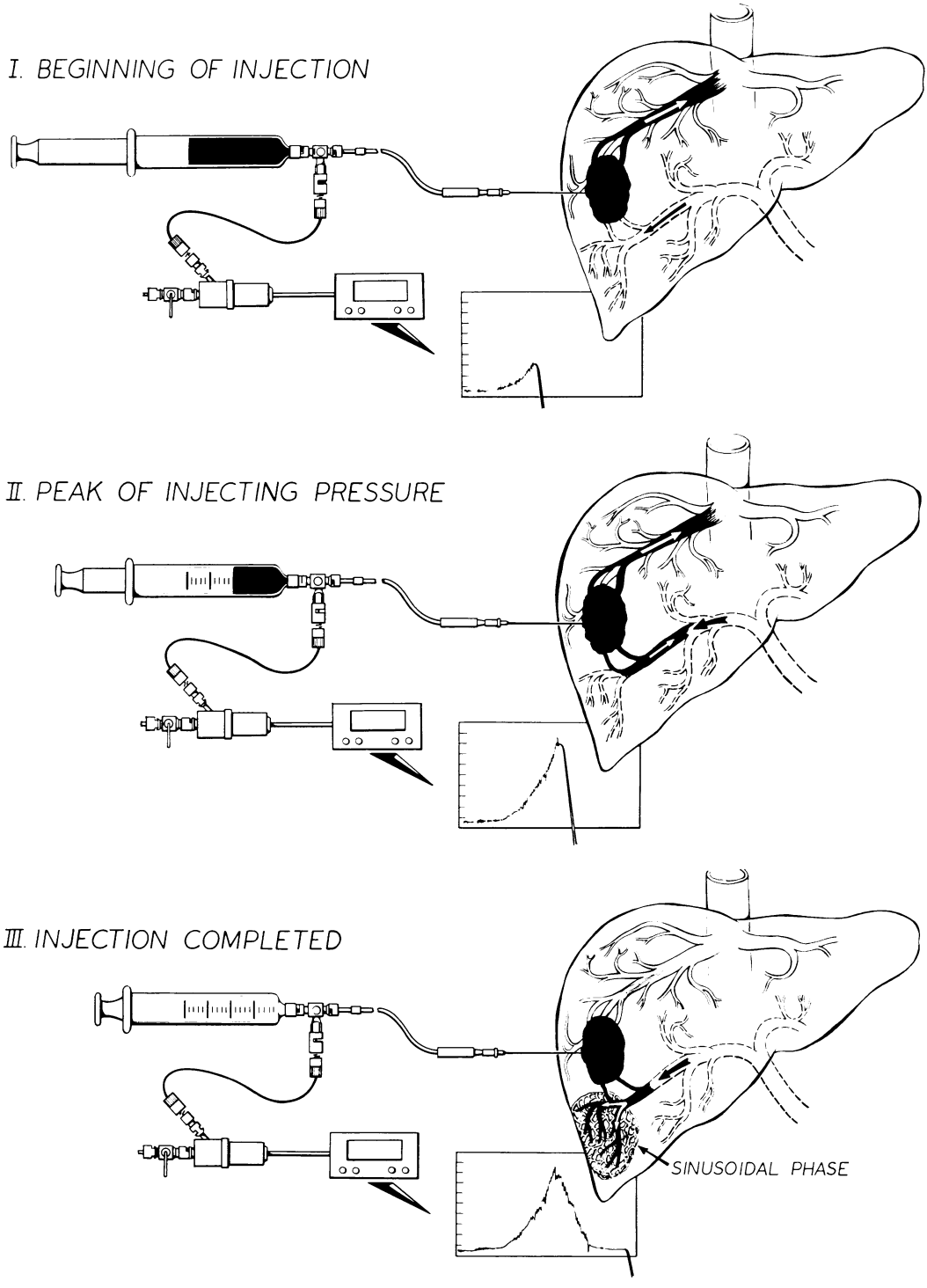


FIG. 6. Mechanism of retrograde visualization of the portal vein following intrahepatic parenchymal deposition of contrast in the pre-shunt patient.

I. From the beginning of the injection, the normal outflow of the liver, the hepatic venous system, was seen to drain the deposition of contrast. Minimal pressure of injection, as measured by an

have significant ascites, a condition in which spontaneous reverse portal flow usually has been implicated.

On the basis of two different methods of examination, present data favors the second possibility.

Direct Measurement of Rate and Direction of Flow in the Portal Vein. Measurements of rate and direction of flow in the portal vein in 14 cirrhotic patients in whom this vessel, although patent, was not visualized during splenic portography are presented in Table 2. Measurements in six patients with normal hepatic hemodynamics, taken during operations in the upper abdomen, are presented as controls in Table 3.

The mean rate of flow in the former group was only one fourth of that in the control group. One patient had a normal flow rate, and another had one half the normal mean. About one half of the patients had values less than one tenth the normal mean. (In three patients flow was stagnant, with only some to-and-fro motion within the portal vein coinciding with the respiratory cycles). There was no instance in which reverse flow in the portal vein could be observed. Except for the case with normal flow, this group of patients had only one sixth of normal flow and less than two thirds the flow observed in a larger group of 72 cirrhotics to be reported elsewhere.[4a] This drastically reduced flow rate apparently is insufficient to incorporate the contrast medium injected into the spleen. In the single exception, strategic location of a large collateral vessel between the spleen and the portal vein may have been responsible for the failure of the contrast medium to reach the liver.

Although theoretically possible, preshunt reverse flow in the portal vein was observed neither in this series nor in the larger group of 72 cirrhotic patients.[4a] These same flowmeters have recorded reversed flow in the portal vein after the construction of a side-to-side portacaval shunt in both cirrhotic patients (Fig. 5) and normal dogs.[1] (It is interesting that reversed flow after side-to-side shunt is not necessarily the result of postsinusoidal obstruction since it is observed in the normal animal.)

Intrahepatic Parenchymal Deposition of Contrast Medium. While most of the radiopaque material was removed by hepatic veins, some was forced into portal branches (against the incoming flow of splanchnic blood) during the peak of the injection pressure, after which it resumed the normal direction of portal flow and was transported into smaller branches and the sinusoids (Fig. 6).

After using this method in about 140 patients, and particularly with the use of fluorographic high-speed motion pictures, certain aspects of the hemodynamic derangement in portal hypertension became evident. Contrast material could be forced into portal branches more and more easily as the incoming flow of splanchnic blood was further reduced by the cirrhotic process. When directly measured portal flow

---

electromanometer, has resulted in no reflux of contrast into the portal venous system against the head of pressure of the incoming portal flow.

II. With the peak of injecting pressure, some contrast was artifactually forced back into the portal system, overcoming to some degree the normally forward momentum of portal flow. This phenomenon occurred in varying extent in all examinations, being barely perceptible in the normal subject with a normal portal inflow, and being quite extensive in cirrhotics with a reduced portal inflow. In patients with a stagnant portal flow, reflux of contrast occurred in the most prominent manner.

III. With cessation of the injecting pressure, the hemodynamic situation was no longer disturbed by the examination itself, and forward progress to the liver of the contrast medium that had been refluxed into the portal vein was then consistently observed. This could be appreciated only by the cine technic.

This fortuitous artefact of portal reflux has enabled us to observe the subsequently undisturbed portal-hepatic circulation and has led to our abandonment of the notion of spontaneous reverse portal flow.

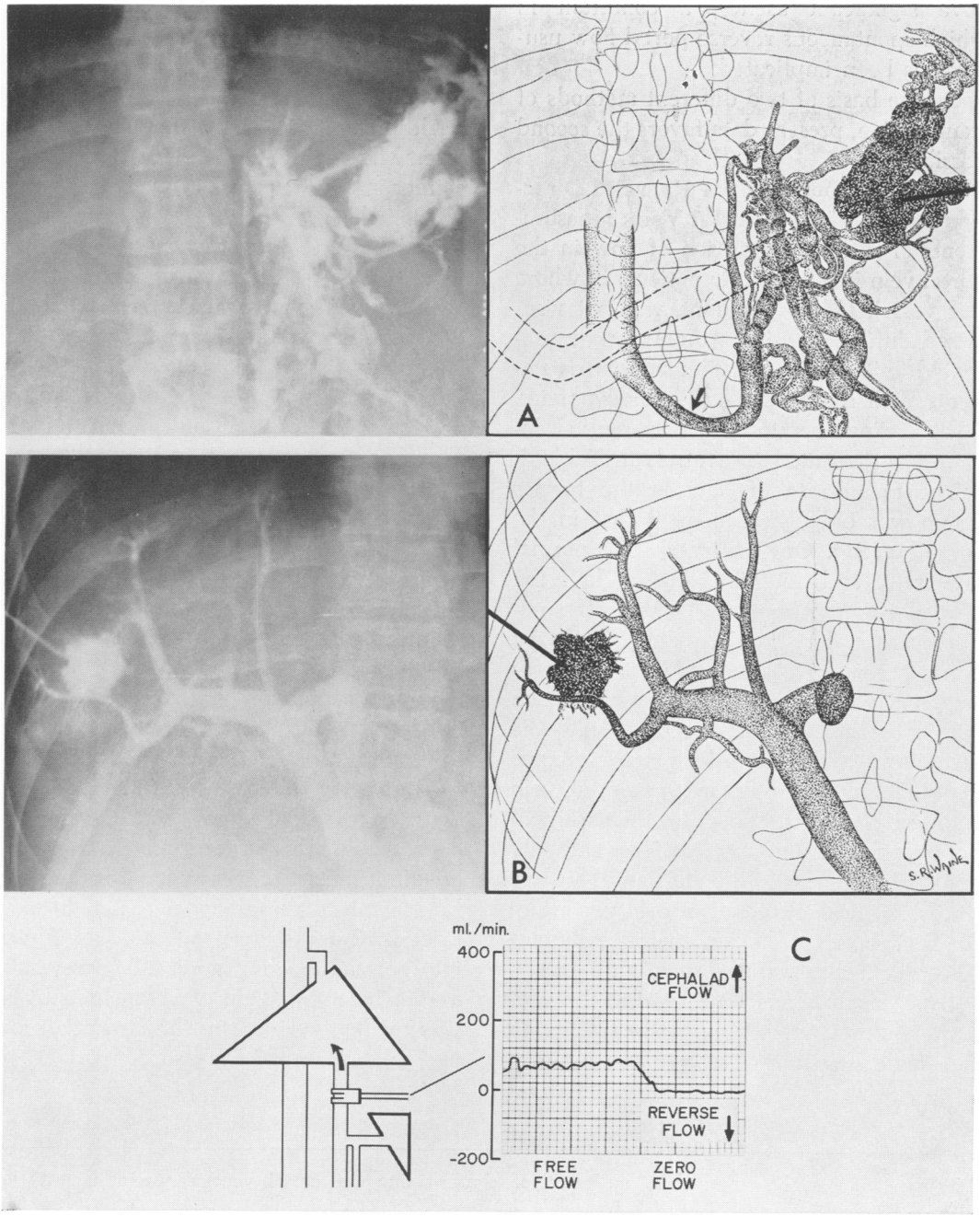


FIG. 7. A composite of studies in a patient with a nonvisualized but patent portal vein.

A. Splenic portography. Despite a technically successful procedure, the portal vein has not been visualized. Presence of collateral vessels, including varices, allows a diagnosis of portal hypertension, but no information concerning vessel patency or availability for a shunting procedure has been obtained.

B. Intrahepatic parenchymal deposition of contrast medium. The peak force of the injecting pressure has forced the contrast medium in a retrograde fashion back into the portal venous system, well outlining the portal vein. The cine examination demonstrated definite forward flow of this contrast in the portal vein following cessation of the disruptive pressure of injection.

became very small or stagnant, contrast medium could be forced into the main portal vein or into collaterals and transported by splanchnic blood into varices or other communications. With subsiding injection pressure, contrast medium within the main portal vein would resume normal direction of flow and perfuse the liver. In patients with stagnant flow, contrast medium forced into the portal vein was seen to remain within its lumen as long as examination was continued. As stated previously patency was established in eight of 14 portal veins not visualized during splenic portography (Fig. 7); in two, stagnant flow was diagnosed by intrahepatic deposition and confirmed at operation by direct electromagnetic measurement. Only high-speed motion pictures can reveal the true nature of hemodynamic events following intrahepatic injections; still films can be misleading, suggesting erroneously the presence of spontaneous reverse portal flow.

True reverse flow, as occurs following side-to-side portacaval shunt, is demonstrated readily by intrahepatic deposition.[9] Even with minimal injection pressure the deposit is rapidly drained by portal branches while the portal vein demonstrates active flow (Fig. 8). In one patient reported previously [9] this was shown more than 3 years after the original operation.

Results of intrahepatic parenchymal deposition of contrast medium in patients whose portal vein, though patent, was not visualized by splenic portography indicate that nonvisualization may follow severe reduction in, rather than reverse, portal flow and corresponding hepatic bypass of splanchnic blood. In both respects, the radiographic results agree with those of direct measurements. It should be noted that this analysis does not consider any possible effect of changes in cardiac output, venous return or mesenteric arterial flow that may occur in cirrhosis and play a role in the phenomenon of portal vein nonvisualization.

That patients with nonvisualized portal veins, and with extreme reduction in portal inflow, may represent a more advanced degree of cirrhotic distortion cannot be disregarded. Only additional studies of morbidity and survival rates following portacaval shunt will indicate whether prognosis is less favorable than that of patients in whom the portal vein is visualized.

Summary and Conclusions

In a series of 904 splenic portograms 6.5 per cent of portal veins were not visualized. Of 152 patients whose condition both allowed and required portacaval shunt this incidence was 17.8 per cent.

Anatomic patency was proved in 84 per cent of nonvisualized portal veins. In a subgroup of 28 patients operated upon, the incidence of patency was 96 per cent.

Routine diagnostic use of other methods to examine portal patency, such as portal portography or intrahepatic parenchymal deposition of contrast medium, would seem not justified for routine use, although the latter was of some investigative value.

Results of both intrahepatic parenchymal deposition of contrast medium and electromagnetic determination of direction and rate of portal flow suggest that failure to visualize a patent portal vein results from a drastic reduction in portal flow and corresponding hepatic bypass of splanchnic flow.

C. Electromagnetic determination of pre-shunt portal flow. At operation, the flowmeter recording in the same patient prior to shunt demonstrated a definite forward flow although of greatly reduced volume confirming the visual demonstration of forward flow obtained by cinefluorography, and validating the concept that extensive reflux into the portal system only occurs in the presence of a greatly reduced portal inflow. No patients in a group of 72 cirrhotics have demonstrated pre-shunt reverse flow.

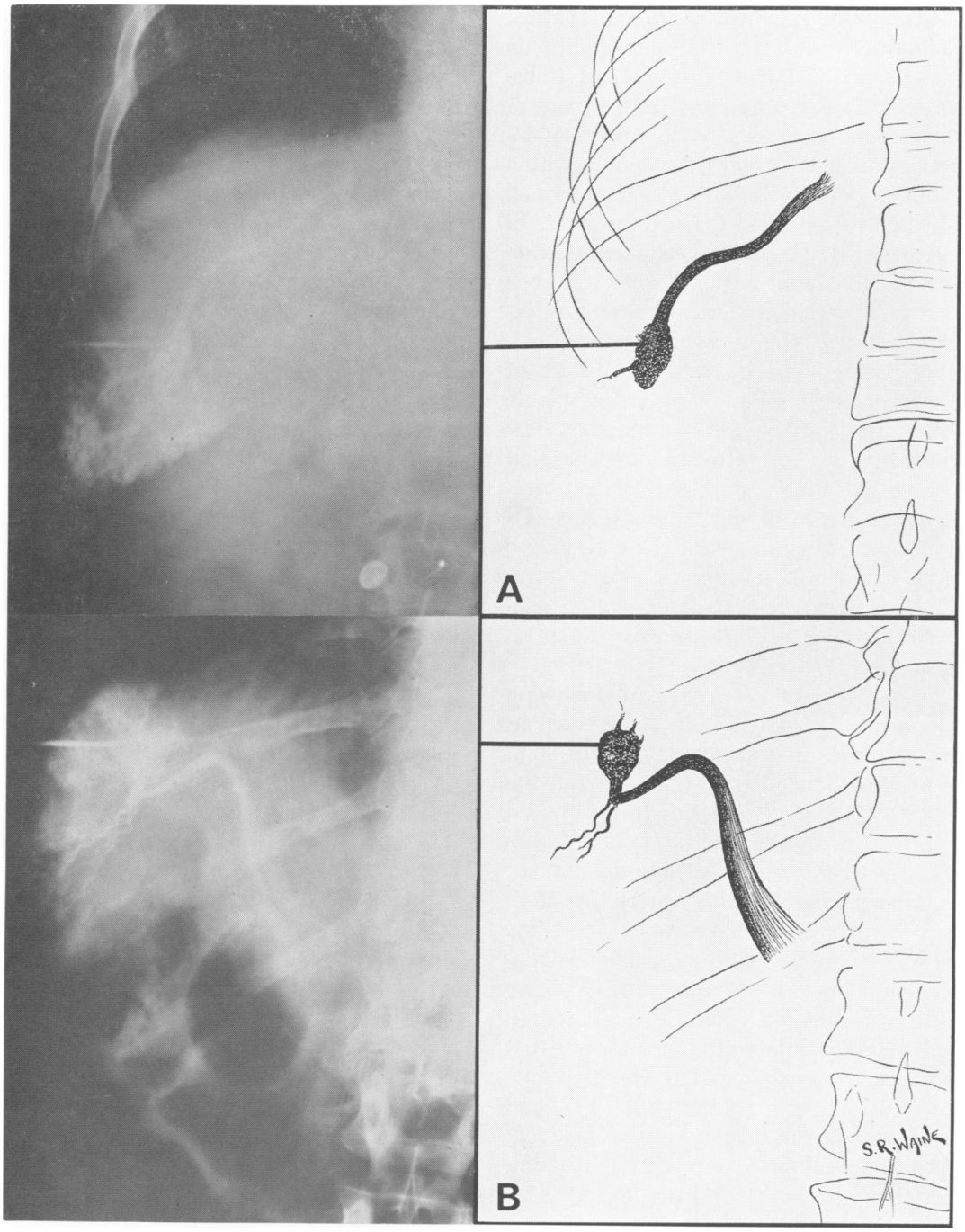


FIG. 8. Radiographic demonstration of true reverse flow in the portal vein following a side-to-side shunt.

A. Prior to shunt, the deposit of contrast medium in the hepatic parenchyma was drained in the normal fashion via the hepatic venous system.

B. Following a side-to-side shunt, the intrahepatic deposition of contrast demonstrated definite reverse flow in the cephalic limb of the portal vein, characterized by graceful streamlining and rapidity of flow directly into the cava even after dissipation of the pressure of injection. This phenomenon was demonstrated best by the cine technic, and was never observed in the pre-shunt intact portal vein.

No spontaneous reversal of portal flow was found.

Portal vein nonvisualization represents the most common and clinically most significant limitation of splenic portography, imposing upon the surgeon the diagnostic problem of vessel patency. The documentation by this study of a patency rate of 96 per cent has helped to resolve this dilemma by reassuring the surgeon in his decision to carry out a portacaval shunt. Accordingly, the recognition and analysis of this limitation of the method has in no way dampened our enthusiasm for the overall usefulness and value of splenic portography.

References

1. Bono, R. F., Moreno, A. H., Rousselot, L. M. and Burke, J. H.: On the Ability of the Portal Vein to Act as a Hepatic Outflow Tract in the Normal Dog. Surg. Forum, **13**:320, 1962.
2. Child, C. G., III and Donavan, A. J.: Current Problems in Management of Patients with Portal Hypertension. J.A.M.A., **163**:1219, 1957.
3. Coomaraswamy, R. P., Del Guercio, L. R. M., Miller, H., State, D. and Elkin, M.: Splenoportography and Portal Vein Thrombosis in Patients with Cirrhosis of the Liver. Surg., Gynec. & Obstet., **118**:560, 1964.
4. Ekman, C. A.: Portal Hypertension, Diagnosis and Surgical Treatment. Acta Chir. Scand., **Suppl. 222**, 1957.

4a. Moreno, A. H., Burchell, A. R., Panke, W. F., Slafsky, S. F. and Burke, J. H.: Rate and Determinants of Portal, Collateral and Mesenteric Blood Flow in Cirrhosis with Portal-Systemic Bypass. Submitted for publication, J. Clin. Invest. 1965.

5. Moreno, A. H., Rousselot, L. M., Burchell, A. R., Bono, R. F. and Burke, J. H.: Response of Hepatic Vein Sphincters to Intraparenchymal Injection of Radiopaque Iodinated Compounds into the Liver of Anesthetized Dogs. Physiologist, **4**:76, 1961.
6. Moreno, A. H., Rousselot, L. M., Burchell, A. R., Bono, R. F. and Burke, J. H.: Studies on the Outflow Tracts of the Liver. I. On a Method for the Functional Demonstration of the Outflow Tracts of the Liver and its Application to the Study of Hepatic Hemodynamics in Normal and Cirrhotic Rats. Ann. Surg., **155**:412, 1962.
7. Moreno, A. H., Rousselot, L. M., Burchell, A. R., Bono, R. F. and Burke, J. H.: Studies on the Outflow Tracts of the Liver. II. On the Outflow Tracts of the Canine Liver with Particular Reference to its Regulation by Hepatic Vein Sphincter Mechanisms. Ann. Surg., **155**:427, 1962.
8. Moreno, A. H., Rousselot, L. M., Burchell, A. R., Bono, R. F., Slafsky, S. F. and Burke, J. H.: Concepts on the Etiology of Cirrhotic Ascites. Clin. Res., **10**:395, 1962.
9. Moreno, A. H., Ruzicka, F. F., Rousselot, L. M., Burchell, A. R., Bono, R. F., Slafsky, S. F. and Burke, J. H.: Functional Hepatography. A Study of the Hemodynamics of the Outflow Tracts of the Human Liver by Intraparenchymal Deposition of Contrast Medium, with Attempts at Functional Evaluation of the Outflow Block Concept of Cirrhotic Ascites and the Accessory Outflow Role of the Portal Vein. Radiology, **81**:65, 1963.
10. Moreno, A. H.: Perpetuation of Cirrhotic Ascites as a Result of Altered Regulation of Venous Return. Bull. N. Y. Acad. Med., **40**:80, 1964.
11. Panke, W. F., Bradley, E. G., Moreno, A. H., Ruzicka, F. F., Jr. and Rousselot, L. M.: Technique, Hazards and Usefulness of Percutaneous Splenic Portography. J.A.M.A., **169**:1032, 1959.
12. Panke, W. F., Moreno, A. H. and Rousselot, L. M.: The Diagnostic Study of the Portal Venous System. Med. Clin. N. Amer., **44**:727, 1960.
13. Rousselot, L. M. and Burchell, A. R.: Portal Venography and Manometry. An Appraisal of the Altered Hemodynamics in Cirrhosis and Non-cirrhotic Lesions with Portal Hypertension. *In* Schiff, L., Ed. Diseases of the Liver. Second Edition. Phila., J. B. Lippincott, 1963.
14. Welch, C. S., Attarian, E. and Welch, H. F.: The Treatment of Ascites by Side-to-side Portacaval Shunt. Bull. N. Y. Acad. Med., **34**:349, 1958.
15. Welch, C. S. and Margulies, E. J.: The Effect of Side-to-side Portacaval Shunt on Experimentally Produced Ascites in Dogs. Surg. Forum, **11**:280, 1960.