Side-to-Side Portacaval Anastomosis for Portal Hypertension *

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It is a generally recognized clinical observation that an increase in the blood pressure of the portal venous system commonly accompanies the advanced stage of various disease processes which produce an abnormal histological pattern in the liver; a pattern characterized by hepatic cellular destruction and increased periportal fibrosis. Many basic mechanisms of this process are poorly understood. Cirrhosis associated with chronic alcoholism and dietary deficiencies, post-hepatic cirrhosis, cholangiolytic hepatitis resulting from chronic biliary tract obstruction and cholangitis, and cirrhosis produced by chronic biliary tract obstruction in congenital biliary atresia are conditions which, while varying widely in their primary etiology, still produce similar end results in the histological architecture of the liver and, in their advanced stages, are frequently associated with portal hypertension.

In the human, occlusion of the major branches of the portal venous system is also commonly associated with an elevated venous pressure in the blocked portion of the venous bed.[1] In 1945, Whipple [32] classified the conditions which produce portal hypertension into intrahepatic block and extrahepatic block. Whether a blockage in venous flow is, in itself, enough to account for the increased venous pressure associated with the intrahepatic condition has rightfully been questioned.[30] In the extrahepatic class, it is difficult to postulate any mechanism as being responsible for the venous hypertension other than the mechanical obstruction provided by the obliterated lumen.

Esophageal varices and other enlarged, engorged veins in the mesentery, alimentary tract and abdominal wall are frequently associated with the conditions described above in which portal hypertension is present. Major bleeding into the gastrointestinal tract, when it occurs, most commonly originates from an opening in an esophageal varix. Here again, our information is not too conclusive, but the explanation advanced by Wangensteen,[31] that bleeding results from acid-peptic digestion of the esophageal mucosa and an underlying varix, would adequately account for most instances of such hemorrhage.

Since the reintroduction by Whipple [32] and Blakemore and Lord [1] in 1945 of portacaval anastomoses as a means of lowering portal venous pressure, a large volume of clinical results has accumulated [2, 8, 12-14] to support the idea that bleeding from esophageal varices may be satisfactorily controlled by lowering the portal venous pressure by portacaval anastomosis. The effectiveness of this procedure, however, has been questioned by Cohn [5] and Ripstein.[25] The original concept that such a shunt operation might also be beneficial in controlling ascites has not been substantiated.

For those who accept the rationale of portacaval anastomosis in the treatment of

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TABLE 1. *Operations for Intra-Hepatic Obstruction*

Procedure	Indications for Operation	Etiology		Results
	Bleeding Varices	Alcoholic Cirrhosis	Hepatitis	Excellent
S–S (18)	17*	13	5	18** (2 mos., 5.5 yrs.)
E–S (1)	1	1	0	1 (7 mos.)
S–R (1)	1	0	1	1 (5.5 yrs.)

* One additional S–S shunt for ascites.
** Three late deaths from hepatic failure.

bleeding esophageal varices associated with portal hypertension, there are three methods by which a sufficient volume of blood can be shunted from the portal system into the systemic circulation and, hence, effectively reduce the elevated portal venous pressure: 1) Ligation of the portal vein at the liver and anastomosis of the distal end of the vein to the side of the inferior vena cava; 2) side-to-side anastomosis of the portal vein to the inferior vena cava; and 3) splenectomy and anastomosis of the end of the splenic vein to the side of the left renal vein. The last two methods are similar because the continuity of the portal vein is left undisturbed and a normal channel remains through which some of the portal venous blood may pass through the liver before returning to the systemic circulation. In the first method, however, the portal vein is completely interrupted and all portal blood is diverted directly into the systemic circulation. Following this operation, the liver receives blood from the hepatic artery alone.

Recent reports [3, 9, 15] have emphasized the use of either the end-to-side portacaval anastomosis or the splenorenal shunt. Side-to-side portacaval anastomosis has been used sporadically since it was first successfully performed in man by Rosenstein.[16] Its disadvantages, as enumerated by various authors, include: 1) It is technically more difficult; 2) thrombosis of the anastomosis occurs more frequently; 3) there is no evidence that portal venous blood will flow into the cirrhotic liver past an adequate shunt opening into the inferior vena cava; and 4) it may actually be harmful to create a situation which will enable the hepatic arterial blood to pass through the proximal portal venous channels in a retrograde manner and into the inferior vena cava, thereby depriving the liver of a portion of its arterial blood flow and creating a vascular arrangement which might be comparable to multiple arteriovenous fistulas.

The senior author's first surgical experience with portacaval anastomosis occurred in 1946. At that time, a side-to-side shunt was performed by which adjacent incisions in the portal vein and inferior vena cava were anastomosed. The shunt subsequently closed completely when the edges of the incision healed together. The technic of anastomosis was, therefore, changed. In 1950, a report [16] was published in which 15 additional cases were described. In eight cases a splenorenal shunt was established; two patients did well, four were improved, and two died. An end-to-side anastomosis of the portal vein to the inferior vena cava was performed in seven cases; two patients were improved, one was unimproved, and four died.

Since that time, the side-to-side portacaval anastomosis has been used wherever feasible. The present series of 28 patients

with portal hypertension treated surgically includes 19 in whom portacaval anastomosis was performed for bleeding esophageal varices without operative mortality.

These satisfactory results with an operative procedure which has generally been rejected on the basis of the somewhat theoretical objections enumerated above, have prompted us to review and present our management of cases with portal hypertension.

Case Material

The cases encountered since July 1951 have been separated into three groups. Table 1 summarizes those cases in which a portacaval shunt operation was utilized for portal hypertension. A shunt operation was performed for ascites in one patient early in the series; all other patients were operated upon for bleeding esophageal varices. There were 20 males and eight females with ages ranging from four to 68 years. All patients in this group suffered from an intrahepatic obstruction, although in one a thrombus which produced a partial occlusion was removed from the portal vein during the performance of the anastomosis. In 14 cases, the clinical findings and pathologic changes in the liver were compatible with a Laennec's alcoholic cirrhosis and in six cases with a post-hepatitic cirrhosis. A side-to-side technic was used in 17 cases, an end-to-side technic in one, and a splenorenal shunt in one.

Table 2 summarizes three cases in which portacaval shunts were utilized for various conditions. Two patients were infants who were found to have an inoperable biliary atresia; a shunt operation was performed at the time of the original exploration in an effort to provide some subsequent palliation for the period when the children would inevitably develop cirrhosis. One child died two days following operation; the other survived for six months, but the presence of the shunt did not seem to influence the child's clinical course. The third patient in this group was a 78-year-old female who had a biliary stricture with recurrent bouts of cholangitis for a period of approximately eight years. She had refused operation for treatment of the stricture until she developed an extensive cirrhosis with ascites and had bled massively from esophageal varices. The stricture was repaired and a portacaval anastomosis was performed. She died seven days after operation from hepatic failure.

TABLE 2. *Shunt Procedures for Miscellaneous Conditions*

Procedure	Indication	Result
E–S	Biliary atresia	Died (2 days)
S–S	Biliary atresia	Died (6 mos.)
S–S	Biliary stricture ascites Bleeding varices	Died (7 days)

The third group, summarized in Table 3, is composed of patients who were subjected to transesophageal ligation of the esophageal varices as an emergency procedure for acute hemorrhage that had been inadequately controlled by the Sengstaken-Blakemore tube. One of these was a 46-year-old male who was operated upon during a period of acute exacerbation of a chronic alcoholic cirrhosis. Although varices were found and sutured, no single bleeding point could be identified. All of the gastric and esophageal mucosa exposed appeared edamatous and hemorrhagic, bleeding profusely from the slightest trauma. The bleeding was not controlled and the patient died a few hours following operation. The other patients were also adults with an extrahepatic type of obstruction. They uniformly had complicated histories which involved previous, unsuccessful operative procedures to control bleeding from esophageal varices. In this group, one patient died five days after operation. The other patients are alive four months, one and three-quarter years, and three years following operation. The patient who survived

TABLE 3. *Transesophageal Ligation of Varices*

Procedure	Site of Obstruction	Result
Ligation of varices	E.H.	Excellent (21 mos.)
	H.	Died (P.O.)
	E.H.	Died (5 days)
	E.H.	Good (3 yrs.)
	E.H.	Good (4 mos.)

the longest has had two episodes of bleeding since operation, one of which was serious enough to require hospitalization and transfusion. He has not bled during the past 14 months. It is interesting that among his many operations this patient had a total gastrectomy 11 years previously which apparently arrested further serious hemorrhage for a period of seven years.

Selection of Patients

A single question decided the selection of patients: Was the risk of repeated hemorrhage due to esophageal varices greater than the risk of performing a portacaval shunt? Any patient who has bled massively (or even considerably) because of esophageal varices is very likely to do so again. Such hemorrhages are not only hazardous, in the sense that any major hemorrhage is risky, but, in addition, each hemorrhage is an added insult to the diseased liver. Hepatic coma is frequently precipitated by gastro-intestinal bleeding, and repeated hemorrhages promote progressive hepatic deterioration. For these reasons, any patient in whom portal hypertension has led to bleeding was considered a candidate for portacaval shunt, although some candidates were rejected, at least temporarily, because signs of liver failure suggested an inability to survive the catabolic shock and stress of a major operation.

The most important criteria were these: 1) How good was the patient's appetite, and was he able to eat a reasonably high protein diet without clouding of consciousness? A "No" answer to this question was regarded as an ominous sign. 2) More than a trace of jaundice, fetor hepaticus, intractable ascites or anasarca, or any demonstrable systemic bleeding diathesis (whether produced by a known deficiency, such as hypoprothrombinemia, or by an unknown clotting defect) were strong relative contraindications to surgery. 3) Of the many liver function tests often taken into consideration, the most vital was the serum albumin. It was regarded as preferable to have the serum albumin normal or only slightly depressed. Nevertheless, neither a low serum albumin nor any of the other relative contraindications did, of itself, automatically cause the patient's rejection. Each case was weighed on the basis of the total picture. One patient, e.g., was temporarily rejected for operation after a major hemorrhage because manifestations of Laennec's cirrhosis—jaundice, marked hypoalbuminemia, and edema—were improving with rest, abstinence from alcohol, and a high protein diet. The patient was brought to the emergency room six weeks later with brisk esophageal bleeding. Although the serum albumin was still low and the edema was not entirely gone, it was felt that further procrastination would be inviting additional hemorrhage and cause the modest improvement to be permanently lost. After adequate transfusion and tamponade of the varices, an "emergency" portacaval shunt was successfully undertaken.

Preparation of Patients

Whenever circumstances permitted delay, an attempt was made to tide the patient over the period of active bleeding and allow recovery of strength and well-being. A diet, adequate in calories and high in both protein and vitamins, was maintained for as long a period as was practical. The degree of abnormal water retention controlled the amount of sodium chloride allowed in the diet; the maximum reduction

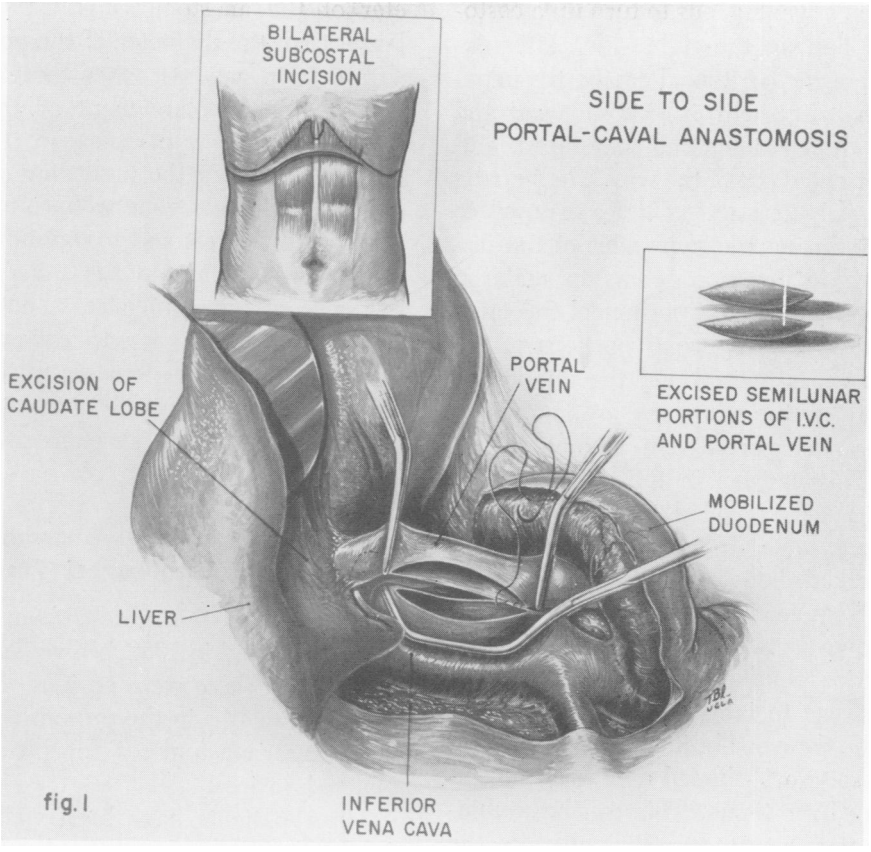

FIG. 1. Technic of side-to-side portacaval anastomosis. Semilunar segments of the portal vein and inferior vena cava are excised. A wedge-shaped segment of the caudate lobe is excised, if necessary, for adequate approximation of portal vein and inferior vena cava.

was to less than 200 mg. daily. Rapport with the patient and with the spouse or dietician preparing the meals was often essential to maintain a stringent low salt diet without reducing the protein consumption.

If the patient required surgery before an adequate hematocrit, blood volume, and serum albumin concentration could be achieved with diet and iron therapy, blood transfusions and, sometimes, infusions of salt-free human albumin were given. Vitamin K was routinely administered for reduced prothrombin concentration. Fresh whole blood was made available for the operative period, in case unexpected difficulties in hemostasis were encountered.

Operative Technic

In recent years, our results with the side-to-side anastomosis have been far superior to those originally encountered. Although a number of improvements have been made in the care of these patients, we feel that the one, possibly most, important factor has been the change from end-to-side to side-to-side anastomosis.

This operation is performed through a generous bilateral, subcostal incision with division of both rectus muscles and extension of the incision to the right side as far laterally as possible (Fig. 1). With such an incision, we have not found it necessary to enter the thorax, although in some patients

it has been advantageous to turn up a costochondral flap on the right side. After exploration of the peritoneal cavity, the hepatoduodenal ligament is exposed and the common duct is identified, mobilized, and gently retracted out of the way. The hepatic artery is palpated and at times exposed to protect it during the remainder of the dissection, if its location seems to make it vulnerable. The lateral peritoneal reflection along the first and second portions of the duodmenum is incised and the duodenum and head of the pancreas are mobilized to expose the portal vein throughout its length. This incision in the peritoneum is then extended cephalad to expose the inferior vena cava. In the cirrhotic patient with marked hepatomegaly, the caudate lobe is frequently so enlarged that it extends down between the portal vein and the inferior vena cava. If the lobe is of sufficient size to interfere with approximation of the two vessels, it has been our practice to excise a V-shaped area from the intervening liver tissue. This can be accomplished without undue difficulty in the scarred, fibrotic liver of the patient with cirrhosis. The pressure in the portal vein is measured at the beginning of operation and again after completion of the anastomosis. Following adequate mobilization of the vessels, the portal vein is occluded with two noncrushing clamps, one at the head of the pancreas, the other at the hilus of the liver. The inferior vena cava is partially occluded with a Satinsky clamp, leaving an adequate lumen in the vessel for passage of blood from the lower part of the body. To prevent the subsequent closure of the anastomosis it is essential that an elliptical segment be excised from the wall of both the inferior vena cava and the portal vein at the site of the proposed anastomosis. The two veins are then approximated with a continuous suture of 4-0 silk, interrupted in three places with stay sutures to prevent constriction of the anastomosis. The diameter of the anastomosis is made slightly larger than the diameter of the portal vein.

In certain patients, when there has been a tremendous enlargement of the spleen with evidence of hypersplenism, it has been our preference, if the preceding portion of the procedure has gone well, to extend the incision to the left and to mobilize and remove the spleen. On the other hand, if evidence of hypersplenism is not marked, it is probably preferable to leave the spleen in place. A marked decrease in the size of the spleen, following the completion of an adequate portacaval anastomosis, may be anticipated.

Results of Portacaval Anastomosis for Bleeding Esophageal Varices

Contrary to the results from our first series, reported in 1950, of five early deaths in 15 cases, there were no immediate postoperative deaths in the present series of 19 portacaval anastomoses for bleeding esophageal varices.

Three patients have been well for over five years, and seven have been well for over two years. There have been two late deaths from liver failure in this group, one occurring at two months and the other at one and one-half years after operation. The latter death occurred in a male with alcoholic cirrhosis. His progress during the first postoperative year was satisfactory. He subsequently resumed his heavy intake of alcoholic beverages and his general condition deteriorated. He died of hepatic failure without further bleeding from esophageal varices. The second patient is one of two in our series who demonstrated persistent neurologic symptoms following operation. His case is presented in detail:

Case Report

The patient was a 68-year-old bartender who had evidence of severe liver depression prior to operation. After prolonged preparation, during which time he had repeated episodes of minor gastro-intestinal tract hemorrhage, a side-to-side

TABLE 4. *Preoperative and Postoperative Studies of Hepatic Function in Unselected Cases*

Patient	Preoperative				Postoperative			
	Total Protein	Albumin	Globulin	Prothrombin Time %	Total Protein	Albumin	Globulin	Prothrombin Time %
W. M.	8.0	4.1	3.9	25	6.4	3.9	2.5	51
C. T.	5.1	2.0	3.1	67	7.8	4.1	3.7	59
W. B.	6.6	4.6	2.0	39	6.4	4.5	1.9	35
T. C.	5.6	3.2	2.4	100	6.6	2.7	3.9	56
E. J.	5.8	2.7	3.1	84	6.4	2.9	3.5	62
M. K.	5.6	2.5	3.1	69	6.4	2.8	3.6	30
R. H.	6.5	3.8	2.7	50	7.0	4.3	2.6	62
J. T.	6.9	3.3	3.6	36	7.1	3.8	3.3	85
J. B.	7.0	3.8	3.2	62	6.4	3.5	2.9	74

anastomosis was performed. He tolerated the procedure well. On the 5th day after operation he developed signs of mental confusion. With a limited protein diet and neomycin to reduce the bacterial flora of the intestinal tract, his mental status cleared, and he was discharged from the hospital 2½ weeks after operation. A month and one-half later, he returned with evidence of severe depression of hepatic function and with neurologic symptoms similar to those described by McDermott,[19] Sherlock,[29] and Hallenbeck.[9] He was confused, vague, and, at times, incoherent. There was a continuous jerky motion of his head and occasionally similar motions of the hands and thumbs. His condition steadily deteriorated. Before his death, 2 months following operation, a generalized bleeding tendency developed and he passed black and red blood per rectum. Postmortem examination showed severe Laennec's cirrhosis, a patent portacaval anastomosis, pulmonary edema, diffuse moderate gastro-intestinal hemorrhage, and cerebral edema. There was no evidence of bleeding from esophageal varices.

The other patient who suffered from neurologic symptoms has been maintained on a low protein diet and oral neomycin for 14 months since operation. On this regimen, he has progressed satisfactorily, but he had five attacks of mental confusion and stupor when attempts were made to discontinue the neomycin.*

All patients in this series have been followed periodically since operation.

Nine patients who had portacaval shunts for bleeding varices have been re-examined during the past month. A comparison of their preoperative and postoperative liver function studies is recorded in Table 4 and Figure 2. It is interesting that in this small, unselected group of patients, all but two of whom have been followed for over six months, there is a slight but definite increase in total protein and serum albumin values. The average of the prothrombin times is unchanged. However, the preoperative value represents the best level that could be obtained under intensive treatment, while the postoperative value has been determined during a period when the patients were under no specific therapy.

In no instance has there been any further postoperative bleeding from varices, despite the fact that all patients had previously had several severe hemorrhages. Acute hemorrhage from varices is routinely treated with transfusion and tamponade by means of the Sengstaken-Blakemore tube. If bleeding is not controlled, the patient is evaluated for a possible emergency portacaval anastomosis. Since a transesophageal suture of the varices is considered an operation of lesser magnitude than the

* Since submitting this report, the patient has been hospitalized to evaluate more accurately his response to changes in diet, increased rest, and withdrawal of neomycin. Under these conditions, observation over a 10-day period has failed to elicit the previously described neurologic manifestations.

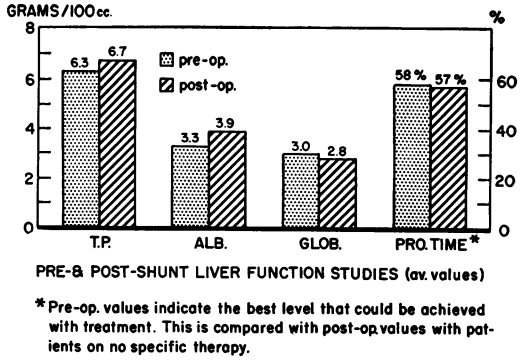

FIGURE 2

shunt procedure, the more critically ill patients are recommended for direct suture of the varix. Patients in good general condition are considered candidates for shunt operations.

Hepatic Blood Flow Studies

A clear understanding of the vascular changes in cirrhosis is essential if one or another surgical technic is to be endorsed. The altered hepatic hemodynamics, as well as the pathologic anatomy associated with cirrhosis, remains poorly understood. The accepted etiology of the accompanying portal hypertension has also been questioned. On the basis of injection studies in the experimental animal, as well as the human cirrhotic liver, Popper, Elias, and Petty [23] and Mann *et al.*[17, 18] suggest that the obstructive scarring process involves the central hepatic venous radicals as well as the portal venules. This distortion, in addition to the possible transmission of arterial pressure to the venous network through demonstrable arteriovenous and venovenous shunts, may well account for elevation of the portal pressure. In an effort to throw some light on this problem, injections of a tracer material * have been made into the hepatic artery and portal vein before and after completion of a side-to-side portacaval shunt (Fig. 3).

A peripheral portal venous radical (usually a vein in a jejunal arcade) is cannulated with polyethylene tubing, which is threaded centrally until it occupies a position well cephalad to the proposed site of portacaval anastomosis. A similar tubing is inserted into another portal venous radical and left in a peripheral position. The third tubing is threaded into the hepatic artery through one of its branches and advanced toward the liver.

One microcurie of RISA is injected into the hepatic artery and five blood samples are immediately drawn from the portal vein. After a proper time lapse, a background sample is withdrawn and two microcuries are injected into the distal portal vein; five samples are again obtained from the proximal portal vein. The portacaval shunt is then performed, and both injections are repeated, always doubling the previous dose of RISA to overcome the background and obtaining background samples just before a new injection. Five minutes following the last injection, a final background sample is obtained; this is labeled the "final count." One cc. of each sample is counted in a deep well, and the count per minute minus background is recorded. The

* RISA, or radioactive Iodinated Serum Albumin, provides a convenient source of gamma radiation for studying, from outside the body, blood flow from one area to another. RISA is readily available, is relatively inexpensive, and has a desirable half-life of eight days. Commercial RISA contains 0.3 to 0.7 millicuries per cc. and approximately 10 mg. of human serum albumin per cc. Unbound I^{131} activity is reduced at time of preparation to less than 1 per cent. For the study of blood flow through and about the liver during operation, 30 cc. of RISA, with activity of 1 microcurie per cc., was employed. The blood flow through the liver was measured directly with a scintillation counter; a crystal, measuring 1.5×0.75 inches, was placed directly over the major hepatic mass. At the same time, blood samples were obtained from the portal vein as rapidly as possible after injection of the radioactive material. After each injection, adequate time was allowed for complete mixing of the radioactive material. Subsequent injections were doubled so that the previous background could be adequately overcome.

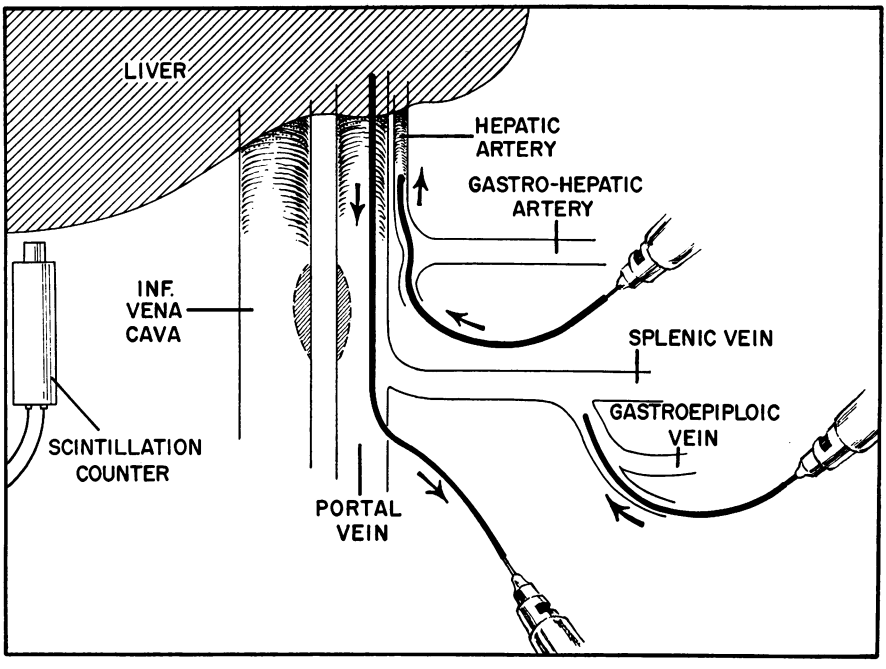

FIG. 3. Diagrammatic representation of position of catheters for injection of RISA and removal of samples of blood from portal vein. Additional evaluation of intrahepatic circulation of RISA obtained by means of scintillation counter positioned over liver.

count for each sample is related to the "final counting rate" as a percentage of the final counting rate and plotted on semi-logarithmic paper.

As a further check on the accuracy of the sample counting, a continuous monitoring of the liver is done during injection with a scintillation counter placed directly over the liver. This tube is connected to an adequate power supply and is then fed into a Berkeley "events per unit time" apparatus which gives a printed recording of the counts every other second. These values are also plotted on semi-logarithmic paper in the manner described above.

The information obtained to date in a small group of patients is summarized in a composite graph (Fig. 4). When the peripheral portal vein is injected with RISA prior to the shunt, a marked prompt concentration is detected in the proximal portal vein. As would be expected, this is not of the same magnitude as that recovered following a similar technic in normal controls. This relatively decreased value in the proximal portal vein might be explained by either stagnation of portal venous flow or loss of the isotope through collateral pathways.

Of considerable interest is the observation that, when the isotope is injected into the hepatic artery of the cirrhotic patient prior to the portacaval anastomosis, it may immediately be detected in the proximal portal vein. This finding, along with the well-known observation that the portal pressure is decreased when the hepatic artery in cirrhotic patients is transiently occluded, would suggest that not only does the hepatic arterial flow contribute to portal hypertension, but there is also actually some reflux arterial flow into the portal venous bed. This had been suspected and, in part, confirmed by Herrick [11] in 1907, and McIndoe [20] in 1928, by injection technics, and was the basis on which hepatic artery

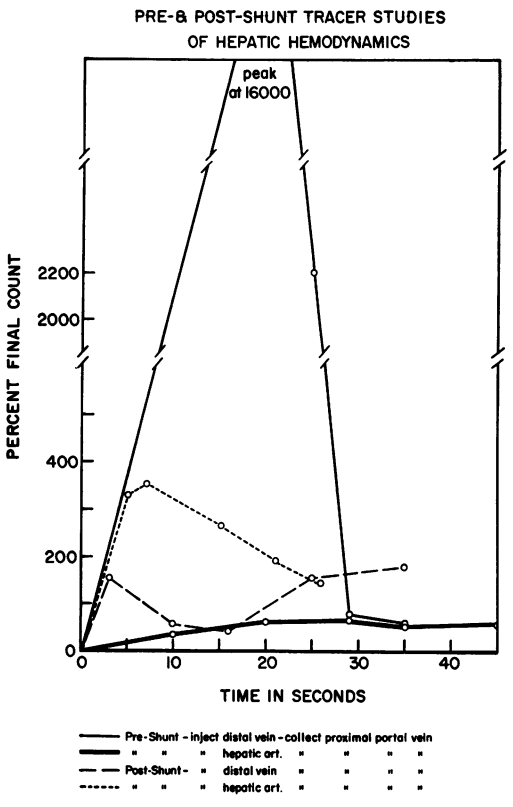

FIGURE 4

ligation was first performed by Reinhoff [24] in 1951.

Following the performance of the portacaval shunt, injection of the isotope in the distal portal vein results in minimal recovery of this material in the portal vein cephalad to the anastomosis. Under the conditions of the experiment, the major bolus of the isotope passes through the shunt into the lower pressure system of the vena cava. Additional evidence that some blood may pass beyond the anastomosis along the normal portal route is found in portal angiograms, which demonstrate radiopaque material in the intrahepatic venous tree after peripheral portal injection (Fig. 5, 6). Intrahepatic visualization occurred only after the injection of 30 cc. of 50 per cent Hypaque under moderate pressure. The material was visualized only in the inferior vena cava when a smaller amount (15 cc.) was injected more slowly.

Also of interest is the result of the post-shunt injection of the hepatic artery. This shows a significant reflux of arterial blood into the proximal portal vein, increased several times over the pre-shunt volume. As yet, one can only speculate on whether this is beneficial or harmful. In advanced cirrhosis, the liver relies heavily on its arterial supply, since a considerable portion of the portal flow by-passes the liver by way of collateral vessels. That it is not entirely devoid of portal contribution seems clear, since hepatic arterial ligation has not been uniformly fatal. Increasing the hepatic blood flow, if this is accessible at the cellular level, would obviously be desirable. Various attempts at arterializing the liver in the experimental animal has been attempted by Narath,[21] Schilling,[28] and Cohn and Herrod.[6] As yet, there is no substantial evidence that this procedure, performed in conjunction with the portacaval shunt, has resulted in any significant improvement of hepatic function. If, with the performance of a side-to-side portacaval anastomosis, the portal venous bed is decompressed and, in addition, the effective hepatic blood flow is increased, the utility of the technic may further be indicated. With the end-to-side shunt, of course, the proximal portal vein is not available for egress of the hepatic blood. At what site the arterial and venous systems communicate remains the critical question. A more accurate appraisal of the altered physiology resulting from a portacaval shunt awaits accumulation of objective functional data, since clinical evaluation of the hepatic status is, in many instances, unreliable.

Discussion

There seems to be little question that, if a patient has portal hypertension and bleeding esophageal varices associated with cirrhosis of the liver or occlusion of the portal vein, further bleeding from the esophageal veins can be satisfactorily controlled by the establishment of an adequate portacaval

anastomosis. There is no conclusive evidence at the present time that such a shunt has any direct effect on the condition of the liver, either beneficial or harmful; but, by protecting the patient from further bleeding into the alimentary tract, further deterioration of liver function may be retarded. Studies of nine patients in the present series, four months to five years after the shunt operation, revealed a slight average improvement of hepatic function as compared to the preoperative studies.

The development of neurologic complications of a similar type has been reported under three different sets of circumstances pertinent to this study: 1) In normal dogs following end-to-side portacaval shunt [4] and in a single human patient with a normal liver following radical pancreaticoduodenectomy and end-to-side portacaval anastomosis as reported by McDermott; [19] 2) in a small percentage of patients [9] following all three types of portacaval anastomoses described above, including two patients in the present series; and 3) in cirrhotic patients without portacaval anastomosis.[7]

The factors responsible for the development of these symptoms are poorly understood. The concentration of nitrogenous products in the systemic circulation seems to be of considerable importance.[10, 22]

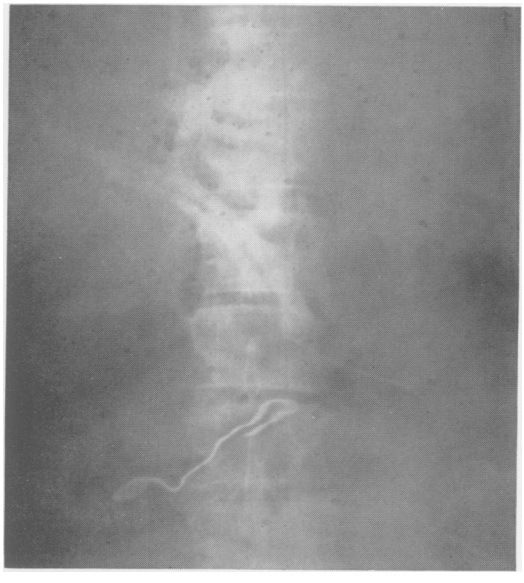

FIG. 5. Portal venogram outlining intrahepatic branches of portal vein prior to shunt.

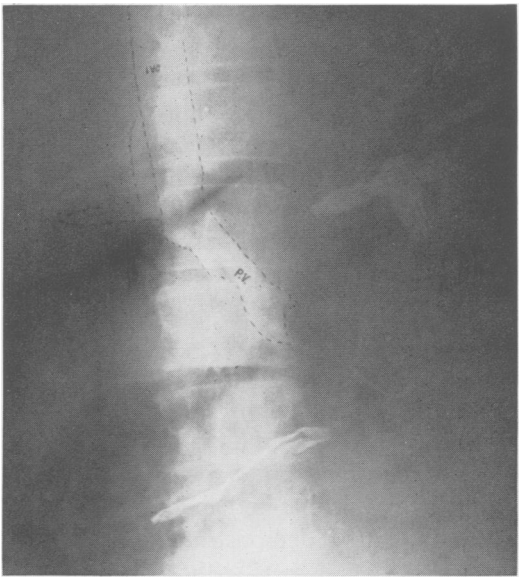

FIG. 6. Post-shunt portal venogram with 30 cc. of radiopaque solution injected into portal vein under moderate pressure. Some of the solution passes beyond the anastomosis into intrahepatic branches of the portal vein.

If the animal or the patient with a normal liver is unable to cope with the sudden increase in nitrogenous products in the systemic circulation produced by the creation of a portacaval shunt, as is suggested by the case reported by McDermott,[19] it is surprising that the patient with a damaged liver will, in most instances, tolerate the sudden diversion of the portal blood so well. It might be postulated that the cirrhotic patient has slowly created some compensating mechanisms as collateral blood flow develops between the portal and systemic circulations, so that he is better prepared for the massive diversion of portal blood following a shunt operation than is the patient with a normal liver. The final limiting factor under all conditions is the ability of the liver to extract and metabolize these nitrogenous products from either the portal or the systemic blood. When this function

drops below a critical level, the neurological symptoms and other severe symptoms of hepatic failure develop regardless of the pathway of the portal blood.

The hypothesis has been entertained by Hallenbeck and Shockett [9] that neurologic symptoms are more likely to occur after end-to-side portacaval anastomosis than after splenorenal anastomosis. The side-to-side anastomosis, like the splenorenal, maintains a potential pathway for portal blood through its usual vascular channels.

This, then, brings up the question of the hepatic and portal circulation following the side-to-side shunt. It is believed that the clinical results obtained in the present series adequately demonstrate that there is no specific damage to either the liver circulation or hepatic function created by the side-to-side shunt. The studies reported following the injection of radioactive serum albumin support the view that there is a retrograde flow of hepatic arterial blood back into the major intrahepatic branches of the portal vein in the cirrhotic patient before a shunt is established, and that this flow is markedly increased after the shunt is opened.

There is also evidence that a small amount of portal blood flows into the major intrahepatic branches of the portal vein beyond the site of the anastomotic opening after a shunt operation. The blood flow in this direction is greatly diminished as compared to preoperative levels. It might well be asked how blood in these vessels could flow in both directions. If there is evidence of a marked increase in the retrograde blood flow from the intrahepatic arteriovenous communications out through the proximal portal vein when the pressure in the vessel is reduced by an open shunt, how could portal blood be expected to pass into the liver beyond the shunt? It must be remembered, however, that these measurements are all taken under abnormal conditions in an anesthetized patient with the abdominal wall widely open.

The theory is advanced that there is a possible bi-directional flow in the portal vein cephalad to the side-to-side anastomosis. Under conditions of rest and complete relaxation, the flow of blood is, in part, from the intrahepatic, arterial circulation retrograde through the portal vein into the vena cava via the anastomosis. Under many conditions of moderate daily activity, however, blood flow and pressures in the portal system are of sufficient volume to reverse the proximal portal flow, so that the portal blood will pass beyond the anastomosis into the intrahepatic circulation. This proposal is supported by the observations of Taylor [30] who reported wide variations in portal pressure accompanying the usual daily activities. Pressures as high as 55 cm. of water were frequently recorded during coughing and choking. Forced inspiration with the glottis closed produced transient pressures as high as 160 cm. of water.

Portal venography performed at operation with the portacaval shunt opened did not demonstrate any radiopaque material in the proximal portal vein when 15 cc. of 50 per cent Hypaque were used. All of the material was seen to pass through the anastomosis. However, when 30 cc. of this material were injected under slightly increased pressure, the material could be identified in the portal vein beyond the anastomosis. Therefore, although definite proof is still lacking, it seems highly probable to us that there exists a bi-directional flow in the proximal portal vein following side-to-side portacaval anastomosis, with the direction of flow determined by changes in the pressure of the portal system during the various normal activities of the human body.

The marked improvement in our results cannot be attributed solely to a change in operative technic, since changes were also made in the selection and preparation of patients for operation. However, the current progress of our patients is sufficiently satisfactory, as compared to our earlier results and to the reports of others, to lead

us to believe that the technic may have specific advantages.

Summary

A series of 28 patients operated upon for portal hypertension is presented. Portacaval anastomosis was performed in 19 patients for bleeding esophageal varices and in one patient for ascites. There were no immediate postoperative deaths in this group. Three patients died later. A side-to-side type of anastomosis was used 18 times and end-to-side and splenorenal anastomoses were each used once. The side-to-side anastomosis preserves the normal pathway of the portal blood to the liver. There is some evidence to suggest that, with the transient changes in portal pressure associated with normal activities, a portion of the portal blood may intermittently pass beyond the shunt into the liver, and that this may, in part, be responsible for the satisfactory results obtained.

A portacaval shunt was constructed in three patients for miscellaneous conditions without beneficial results.

Transesophageal suture of varices was performed five times as an emergency procedure to control massive bleeding. There were two operative deaths in this group. Bleeding in one of these patients has been satisfactorily controlled for three years.

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Discussion

Dr. A. O. Singleton, Jr.: This has been a most interesting paper and I have enjoyed it very much. I am quite confused about this subject. I might say I was confused before I heard Dr. Longmire's paper—I was confused when I arrived here.

We have had some good results with the shunt procedure; we also have a number of other cases where we did not do a shunt, either because of the patient's refusal of operation or because we were unable to perform a shunt at operation. To our discomfiture many of these patients have done very well. I think that in this condition there are many patients who have spontaneous remissions for long periods, some with complete remission, and one must consider this in evaluation of whatever technic is used. Another thing I would like to mention is that there are cases where one finds esophageal varices without portal hypertension, and one should also remember this, especially if pressure is put on one to do some emergency type of portacaval shunt because of severe esophageal bleeding in a poor risk patient.

Dr. D. G. Mulder (closing): I should like to thank Dr. Singleton for his comments, and to show three slides to elaborate on what Dr. Longmire previously mentioned.

(Slide) This is the technique that has been utilized for the tracer isotope study. Three cannulations are performed, one into the hepatic artery, one into the peripheral portal bed, and one into the portal vein proximal to the site of the shunt. This last catheter goes well cephalad to the anastomosis to avoid any turbulence effect in the sampling. Two groups of samples are taken before the shunt, and two after the shunt. All samples are withdrawn rapidly from the proximal portal venous cannula first following injection of isotope in the peripheral portal venous bed, and then following injection into the hepatic artery. This sequence is repeated following the shunt. A continuous recording of the hepatic flow is also monitored by this scintillation counter placed over the liver.

(Slide) This is the result of the study. The first high peak on the diagram is that obtained when the peripheral portal bed was injected and samples taken proximally in the portal vein. The second tracing indicates the presence of radioactivity in the proximal portal vein following injection into the hepatic artery. These are before the shunt. Following completion of the shunt repeat samplings show again a definite but much less marked rise following peripheral portal injection, and a marked increase in radioactivity when injection is made in the hepatic artery as contrasted to the pre-shunt curve.

(Slide) We interpret these results to show a definite reflux of hepatic arterial blood through the portal bed into the proximal portal vein. Whether this is beneficial or not has at present not been established. There seems to be evidence also that some portal blood passes in its normal route into the liver following a side-to-side shunt although the major portion of it is diverted into the inferior vena cava.

Again, I want to thank Dr. Singleton for his comments and the Chair for the privilege of the floor.