California MEDICINE

OFFICIAL JOURNAL OF THE CALIFORNIA MEDICAL ASSOCIATION

VOL. 73 SEPTEMBER 1950 No. 3

The Surgical Treatment of Portal Hypertension

W. P. LONGMIRE, JR., M.D., *Los Angeles*

SUMMARY

Portacaval shunt operations were done in 15 of 18 patients who were treated surgically for portal hypertension. In eight cases the shunt was established by splenorenal anastomosis; in seven by anastomosis of the portal vein to the side of the inferior vena cava. Of the eight patients with splenorenal shunt, two are well, four are considered improved, and two have died. Of the seven in whom the portal vein was joined to the vena cava, two are improved, one is unimproved, and four have died.

BLAKEMORE and Lord[1] and Whipple[17] stimulated renewed interest in the problem of portal hypertension with their reports in 1945 of successful venous anastomosis which permitted portal blood to flow directly into the systemic circulation, bypassing obstructions in the usual portal pathway. Their original technique of performing the venous anastomosis with vitallium tubes has been supplanted by the method of direct suture anastomosis. The principle of the venous shunt, however, remains as one of the most effective methods of relieving portal hypertension. Von Eck[5] in 1877 performed the first portacaval anastomosis in dogs, using a side-to-side technique that has become known as the Eck fistula. Whipple[18] has observed dogs with Eck fistulas for as long as eight years. These animals had occasional episodes which were attributed to disturbed protein metabolism, but they generally maintained a satisfactory state of health. Prior to the reports of Blakemore, Lord and Whipple, isolated attempts had been made to apply this principle of shunting blood directly into the systemic circulation in cases of portal hypertension. Minor branches of the portal and systemic system were usually employed and thrombosis at the site of anastomosis invariably accounted for the failure of the procedure.

Blood carried into the portal area passes through two capillary systems before returning to the heart. It passes through a set of capillaries in the viscera, from which it is collected into the veins of the portal system and is then carried to the second set of capillaries in the liver. From here, it is returned to the heart by way of the inferior vena cava. Any condition which inhibits the free flow of blood in the portal system into the inferior vena cava usually increases the pressure of the blood in all or a part of the portal system.

Patients with an elevation of the venous pressure in the portal system may be divided into two main groups as suggested by Whipple: Group I, those having an intrahepatic portal block; and group II, those having an extrahepatic block.

INTRAHEPATIC BLOCK

Intrahepatic block is usually associated with cirrhosis of the liver. The actual cause of portal hypertension in cirrhosis has not been clearly demonstrated. It has been suggested that intrahepatic fibrosis narrows the lumen of the portal vein

From the Medical Service, Wadsworth Hospital, Veterans Administration Center; and the Department of Medicine, University of California, Los Angeles.

Reviewed by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions of the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Presented before the Section on General Surgery at the 79th Annual Session of the California Medical Association, San Diego, April 30-May 3, 1950.

branches inside the liver and that the resulting increased resistance to the flow of blood is responsible for the elevated pressure.

Herrick's[7] studies of increased portal pressure in cirrhosis emphasized the communication of the portal venous system and the hepatic arterial system in their smaller branches inside the liver. The studies also emphasized the pronounced effect on portal pressure occasioned by slight increases of hepatic arterial pressure in the cirrhotic liver. In the normal liver Herrick found a rise of 1 mm. of mercury in the portal pressure for every 40 mm. elevation of arterial pressure. In the cirrhotic liver there was 1 mm. rise of portal pressure for every 6 mm. elevation of arterial pressure. Gad[6] also demonstrated that increased pressure in the hepatic artery retarded the portal flow.

McIndoe[9] failed to confirm these findings of the effect of the arterial pressure changes on the portal pressure. However, he made the important observation that only 13 per cent of fluid that perfused through the portal vein in the cirrhotic liver could be recovered from the hepatic vein; the remainder was lost through collateral vessels. McIndoe concluded that the elevated portal pressure resulted from a gradual shutting off of portal blood from the hepatic cells as interlobular fibrosis increased.

The case histories of 65 patients admitted to the Wadsworth Veterans Hospital with a diagnosis of cirrhosis of the liver have recently been reviewed. The average age of the patients in this all-male series at the time of admission was 51 years. Eighty-one per cent of the patients were between 40 and 60 years of age. All the patients with advanced cirrhosis had histories of excessive alcoholic intake and many of the acute exacerbations of their illnesses were preceded by bouts of heavy drinking.

In 33 of the cases there was evidence of esophageal or gastric varices with bleeding into the alimentary tract. Ascites was present in 50 of the patients, and 19 had both varices and ascites. Active duodenal ulcer was demonstrated in four cases.

In 58 cases with complete follow-up records, there were 30 deaths, a mortality rate of approximately 52 per cent. The immediate causes of death were as follows: Hemorrhage into the gastrointestinal tract in 15 cases, hepatic failure in 10 cases, and miscellaneous causes in five cases. The duration of the disease in relation to the cause of death is indicated in the following table:

Duration of disease	Number of patients	Living	Dead	Cause of death*		
				Hem.	Hep.	Other
0 - 3 mo.	8	4	4	1	2	1
3 mo. - 1 yr.	6	3	3	1	2	
1 yr. - 3 yr.	25	10	15	8	5	2
3 yr. - 10 yr.	14	8	6	5	1	
10 yr. plus	4	3	1			1
Unknown	1	0	1			1
Total	58	28	30	15	10	5

Mortality rate, 52 per cent.

*Hem.: Hemorrhage. Hep.: Hepatic failure.

Ratnoff and Patek[14] reported that 40 per cent of patients who have hemorrhage from esophageal varices die within one month of the first hemorrhage.

It is obvious that the prognosis for the cirrhotic patient in the past has been grave. Although portal hypertension is only a secondary manifestation of the primary disease process in the liver, a high percentage of cirrhotic patients die as a result of events directly related to the increased portal tension. It would follow that an acceptable period of palliation might be anticipated if the venous pressure in the portal system could be reduced. The most satisfactory results would be obtained in inactive cases and in those cases in which the disease process in the liver was least severe.

Blakemore[2] classified cases of cirrhosis into three groups in regard to their suitability for operation: Group I, cases in which ascites or tendency to hemorrhage is based on the inability of the damaged liver to form albumin or prothrombin in adequate amounts. Portacaval anastomosis is not indicated in this group. Group II, cases in which the liver function is adequate to furnish the required amount of protein and prothrombin but in which a severe degree of portal hypertension has supervened as a result of fibrotic contraction and periportal fibrosis. Wasting ascites and severe hemorrhage can be controlled in this group by portacaval shunt. Group III, cases in which there are varying degrees of depressed liver function plus evidence of considerable elevation of the portal pressure. These cases must be considered individually and the response of liver function under treatment observed over a period of time. Certain of the patients will be candidates for operation, but the optimum time for operation must be carefully selected. Patients with massive recurrent hemorrhage, at times, may be operated upon despite an inadequate period of preparation. If the liver function is seriously depressed at the time of operation, the prognosis is grave, even though a successful anastomosis is completed.

EXTRAHEPATIC BLOCK

There are two conditions which may cause an extrahepatic block: (1) splenic vein thrombosis, or (2) a block in the portal vein, resulting in portal hypertension.

Splenic vein thrombosis. If the block or thrombosis occurs in the distal portion of the splenic vein and interferes solely with return of blood from that organ, enlargement of the spleen occurs and there may be anemia and leukopenia, the result of so-called hypersplenism. Enlarged collateral vessels usually develop in the vasa brevia between the spleen and the stomach, but gross bleeding into the alimentary tract does not occur. For this condition splenectomy alone will provide adequate relief of the patient's symptoms. Such isolated thrombosis of the splenic vein is quite rare but it undoubtedly accounts for the occasional satisfactory result which has followed splenectomy in the treatment of patients with Banti's disease in the past.

Should the thrombus be proximal to the junction

of the splenic and coronary veins, however, and thereby obstruct the blood flow of the coronary vein from the lower esophagus and the lesser curvature of the stomach, the patient will eventually develop gastric and esophageal varices with resulting hemorrhage into the alimentary tract. Splenectomy alone in such cases will not permanently alleviate the dilatation of the gastric and esophageal veins. Such a condition occurs so regularly in cases with Banti's disease that Blakemore, Welch, Linton and others have strongly advised against splenectomy alone unless the surgeon can be certain that the pressure in the coronary vein is not elevated and that the block is confined to the splenic vein. Thrombosis of the splenic vein following splenectomy eliminates the possibility of performing a splenorenal vein anastomosis and thereby removes the best opportunity the patient has for relief of the hypertension in the coronary vein.

Portal hypertension resulting from a block in the portal vein. Although the entire visceral venous bed is usually referred to as the portal system, the portal vein *per se* is formed at the juncture of the splenic and superior mesenteric veins. This vein lies, for the most part, in the posterior part of the hepatoduodenal ligament and runs from the under or dorsal surface of the head of the pancreas to the hilus of the liver. Acute blockage of the portal vein from trauma or sudden complete thrombosis in man is usually fatal. There are three conditions which may obstruct the portal vein and produce hypertension throughout the portal system.

(a) "Cavernous transformation" of the portal vein. The origin of this condition is uncertain but it is usually thought to be a condition which arises early in the life of the individual either as a congenital malformation of the portal vein, as an extension of the obliterative process in the ductus venosus to involve the portal vein, or as a result of an inflammatory thrombosis arising from an infection in the umbilicus extending down the umbilical vein to involve the portal vein. Exploration of the hepatoduodenal ligament in later years will reveal a number of dilated, thin-walled tortuous veins instead of a single large portal vein, whence the name, "cavernous transformation."

(b) Thrombosis of the portal vein. At any age the portal vein may be the site of formation of a thrombus. Such a condition may be associated with an inflammatory process in the abdomen, or develop following trauma to the abdomen. More frequently it is idiopathic or of unknown origin. The process probably starts as a partial obstruction, but it may progress to complete occlusion of the portal vein followed by the development of collateral vessels in the hepatoduodenal ligament and other collateral areas.

(c) External compression. The portal or splenic veins may be obstructed by an adjacent inflammatory cicatrix associated with pancreatitis or cholecystitis; rarely, neighboring tumors or cysts may, by compression, interfere with portal blood flow.

SYMPTOMS ASSOCIATED WITH PORTAL HYPERTENSION

The symptoms frequently associated with portal hypertension may be grouped in the following manner, although clinically they may appear singly or in combination:

1. Effects due to enlargement of the spleen. The anemia and leukopenia in cases of Banti's disease are usually attributed to the effects of hypersplenism.

2. Effects of collateral circulation. Certain collateral vascular systems become established when portal obstruction and hypertension persist. These are described as follows:

(a) Accessory portal veins of Sappey. These are multiple small veins about the liver and portal vein, which communicate through the subdiaphragmatic and retroperitoneal areas with the systemic system. Included in this group are the vessels which course along the ligamentum teres to the umbilicus to anastomose with the epigastric veins in the abdominal wall. Clinically, the presence of such veins is often helpful in indicating portal obstruction as the superficial abdominal veins become dilated, and rarely a cluster of veins about the umbilicus, the caput medusa, may be visible.

(b) Veins of Retzius in the retroperitoneal area are important collateral vessels but are usually not demonstrable.

(c) Hemorrhoidal veins. The collateral anastomosis between the superior and inferior hemorrhoidal veins produces internal and external hemorrhoids.

(d) Cardiac and esophageal veins. Collateral pathways may be established to carry blood through these veins into the azygos system. Bleeding from these veins is, of course, one of the prime symptoms of portal hypertension.

3. Abnormal filtration through portal bed. The formation of ascitic fluid does not occur in cases in which obstruction is confined to the splenic vein. It occurs infrequently in cases in which there is obstruction of the portal vein but in which liver function is normal.

Ascites is frequently observed in the cirrhotic patient. Here it is undoubtedly produced by a combination of factors: (a) The increased hydrostatic pressure inside the branches of the portal vein; (b) disturbance of the normal constituents of the serum (a result of impaired liver function), the disturbance resulting in the passage of fluid from the veins into the peritoneal cavity. The relative importance of these two factors must be weighed in considering operation to reduce portal hypertension.

OPERATIVE PROCEDURES

The operative procedures which have been advocated in the treatment of portal hypertension or its secondary manifestations may be listed as follows:

I. Drainage procedures: (a) Insertion of glass button into abdominal wall; (b) saphenoperitoneal anastomosis; (c) pyeloperitoneal anastomosis.

II. Portal-systemic venous shunt: (a) Omento-

pexy; (b) portacaval, splenorenal and other direct venous anastomoses.

III. Direct treatment: (a) Injection of esophageal varices; (b) ligation of varicosities; (c) esophagogastric resection; (d) mediastinal packing.

Crosby and Cooney[4] in 1946 reported six cases in which patients with pronounced ascites were treated by the insertion of a glass button of original design into the lower abdominal wall; a procedure which permitted the escape of ascitic fluid into the subcutaneous tissue. All the patients were considered benefited by the procedure. The longest period of postoperative observation was 11 months. Lord[8] subsequently added two modifications to the procedure: The creation of a large subcutaneous pocket and the removal of fascia overlying the abdominal muscles. The author's experience with this procedure in three cases in which the modification of Lord was used was unsatisfactory. However, in one patient recently observed, ascites had been controlled satisfactorily for 18 months after insertion of a Crosby-Cooney glass button at another hospital. When the patient was observed by the author, intractable ascites had returned, apparently from lack of absorption of fluid in the subcutaneous tissue.

Isolated reports of successful saphenoperitoneal and pyeloperitoneal anastomoses have appeared, but the methods have never been widely used. Intraperitoneal obstruction of the ostia of such anastomoses would seem likely. The results of the various omentopexy procedures have been uniformly disappointing.

Bleeding from esophageal varices has been satisfactorily controlled by the injection of sclerosing solutions into the veins.[10, 11] Although such injections may obliterate certain of the collateral channels without reducing the portal hypertension, the procedure probably should be more widely used as a palliative measure in cases of cirrhosis with bleeding varices and extensive hepatic damage.

Som and Garlock[16] have reported two cases in which bleeding from esophageal varices was relieved by the packing of gauze about the esophagus in the upper mediastinum. It was felt that the pack promoted the formation of granulation tissue and the subsequent development of collateral vessels between the esophageal veins and the deeper venous channels of the mediastinum.

An esophagogastric resection in treatment of bleeding varicosities was reported by Phemister and Humphreys[13] in 1947. They suggested that the probable source of bleeding in cases of portal hypertension in which no gross bleeding point could be found was the engorged venocapillary network in the mucosa. Shafer[15] recently reported encouraging results in the treatment of six patients by this method.

Pattison[12] reported 14 cases of cirrhosis of the liver in which exploration was carried out. In ten of the cases a portacaval shunt was performed. There was one postoperative death. Numerous other small series or single successful cases have been reported; however, the great operative difficulties, arising chiefly from extensive collateral vessels, and the frequent occurrence of serious postoperative complications such as thrombosis at the site of anastomosis, adherence and closure of the suture line, and hepatic failure have made many surgeons question the ultimate usefulness of venous anastomosis in the treatment of this condition.

The direct shunt of blood from the portal system to the systemic circulation by means of venous anastomoses would seem to offer the most satisfactory method of reducing portal hypertension for a prolonged period. Blakemore[3] recommended anastomosis of the portal vein to the inferior vena cava whenever possible. The progress of four patients with anastomosis of this type was followed by him for more than two years. Response of hepatic function to this type of shunt did not appear to differ from that observed in patients in whom the splenorenal type of shunt had been employed. In Blakemore's series of 60 cases there were 11 postoperative deaths following portacaval anastomosis.

The following data were obtained through the study of 18 patients with portal hypertension treated surgically by the author or other members of the staff of Wadsworth Veterans Hospital:

Six of the patients are known to have died since operation. One patient who had total gastrectomy and esophagojejunostomy for hemorrhage has had no further bleeding in the three years since operation. Two were treated by splenectomy alone because a satisfactory vein for anastomosis could not be found in the splenic pedicle. One of them has shown improvement over a short postoperative period; the other has continued to have hematemesis and is unimproved.

Portacaval shunt operations were done in 15 cases. In eight of the 15, splenorenal anastomosis was used, with the following results: Two of the patients are well, four are considered improved, and two have died. In the remaining seven cases, the end of the portal vein was anastomosed to the side of the inferior vena cava. The results: Two of the patients are considered improved, one is unimproved, and four have died.

Of eight patients with intrahepatic block who were subjected to portacaval shunt, four are improved, one is unimproved, and three have died. Of ten patients with extrahepatic block, six are improved postoperatively, one is unimproved, and three have died.

In the six cases in which the patient died, the duration of life after operation, and the cause of death were:

	Survival	Cause of death
1.	7 days	Thrombosis at site of anastomosis
2.	5 days	Portal vein found thrombosed at time of exploration. Thrombus removed and portacaval anastomosis performed. Thrombus reformed with extension
3.	10 days	Hepatorenal failure
4.	3 days	Extensive pancreatic necrosis
5.	4 months	Closure of anastomosis
6.	47 days	Hepatic failure

REFERENCES

1. Blakemore, A. H., Lord, J. W., Jr.: A nonsuture method of blood vessel anastomosis, Annals of Surgery, 121:435, April 1945.

2. Blakemore, A. H.: The operation of portal caval anastomosis, New York State Journal of Medicine, 47:479, March 1947.

3. Blakemore, A. H.: Portacaval anastomosis for portal hypertension, Surgery, 26:99, July 1949.

4. Crosby, R. C., Cooney, E. A.: Surgical treatment of ascites, New England Journal of Medicine, 235:581, Oct. 17, 1946.

5. Eck, N. V.: The ligature of the portal vein, Voyeno Medical Journal, 1877.

6. Gad: Dissertation, Berlin, 1873.

7. Herrick, F. C.: An experimental study into the cause of the increased portal pressure in portal cirrhosis, Jr. Exp. Med., 9:93-104, 1907.

8. Lord, J. W.: A modification of the Crosby-Cooney operation for intractable ascites due to cirrhosis of the liver, J.A.M.A., 136:767, March 13, 1948.

9. McIndoe, A. H.: Vascular lesions of portal cirrhosis, Archives of Pathology, 5:23-42, Jan. 1928.

10. Moersch, H. J.: Treatment of esophageal varices by injection of sclerosing solution, J.A.M.A., 135:754, Nov. 22, 1947.

11. Patterson, C. O., Rouse, M. O.: The sclerosing therapy of esophageal varices, Gastroenterology, 9:391, Oct. 1947.

12. Pattison, A. C.: The use of portal caval anastomosis in portal cirrhosis, Archives of Surgery, 58:590, May 1949.

13. Phemister, D. B., Humphreys, E. M.: Gastroesophageal resection and total gastrectomy in the treatment of bleeding varicose veins in Banti's sydrome, Annals of Surgery, 126:397, Oct. 1947.

14. Ratnoff, O. D., Patek, A. J.: The natural history of Laennec's cirrhosis of the liver, Medicine, 21:207, Sept. 1942.

15. Shafer, P.: Presented at Western Surgical Society Annual Meeting, December 1949.

16. Som, M. L., Garlock, J. H.: New approach to the treatment of esophageal varices, J.A.M.A., 135:628, Nov. 8, 1947.

17. Whipple, A. O.: The problem of portal hypertension in relation to the hepatosplenopathies, Annals of Surgery, 122:449, Sept. 1945.

18. Whipple, G. H., Robscheit-Robbins, F. S., Hawkins, W. B.: Eck fistula liver subnormal in producing hemoglobin and plasma proteins on diets rich in liver and iron, Journal of Experimental Medicine, 81:171-191, Feb. 1945.