

6-1955

An Evaluation Of The Methods Of Treatment Of Portal Hypertension

W.A. Altemeier

Follow this and additional works at: <https://scholarlycommons.henryford.com/hfhmedjournal>

 Part of the [Life Sciences Commons](#), [Medical Specialties Commons](#), [Public Health Commons](#), and the [Surgical Procedures, Operative Commons](#)

Recommended Citation

Altemeier, W. A. (1955) "An Evaluation Of The Methods Of Treatment Of Portal Hypertension," *Henry Ford Hospital Medical Bulletin* : Vol. 3 : No. 2 , 62-67.

Available at: <https://scholarlycommons.henryford.com/hfhmedjournal/vol3/iss2/3>

This Article is brought to you for free and open access by Henry Ford Health System Scholarly Commons. It has been accepted for inclusion in Henry Ford Hospital Medical Journal by an authorized editor of Henry Ford Health System Scholarly Commons. For more information, please contact acabr4@hfhs.org.

AN EVALUATION OF THE METHODS OF TREATMENT OF PORTAL HYPERTENSION

W. A. ALTEMEIER, M. D.*

Numerous surgical procedures have been developed for the treatment of patients with portal hypertension complicated by esophageal varices and gastro-intestinal hemorrhage, since 70 per cent of the patients with this latter complication die within two years under conservative treatment. Included among these procedures are omentopexy, ligation of coronary veins (splenectomy, thoracotomy for ligation of bleeding esophageal varices, ligation of splenic artery, portacaval shunt, splenorenal shunt, ligation of hepatic artery with or without ligation of splenic and left gastric arteries, and the Phemister procedure or its modifications.

As a resident at the Henry Ford Hospital, I recall helping Roy McClure do omentopexies, ligation of coronary veins, or splenectomies in patients with bleeding esophageal varices in an attempt to control or prevent hemorrhage. These procedures have been largely abandoned as a result of the recent advances in our knowledge and technical skill. It is also interesting to note that ligation of the splenic artery was first done by another Detroit surgeon, Alexander Blain, in a patient with Banti's disease and gastro-intestinal hemorrhage. This procedure resulted in necrosis of the spleen in that patient.

More recently, as the result of the studies of Whipple,⁽¹⁾ Blakemore,⁽²⁾ Rousselot,⁽³⁾ Linton,⁽⁴⁾ Berman,⁽⁵⁾ Reinhoff⁽⁶⁾ and others,⁽⁷⁾ two other methods of treatment have been developed, namely, a shunt between the portal and vena caval circulations and a reduction of the hepatic arterial blood flow and portal pressure by ligation of the hepatic artery with or without ligation of the splenic and left gastric arteries. The Phemister procedure⁽⁸⁾ and its modifications have been reserved generally for those patients in whom shunting procedures were impossible and other forms of treatment had failed.

Since confusion still persists in many instances as to which surgical procedure is indicated for the treatment of a given case, we have attempted to analyze our experiences in 8 cases of portal hypertension and to compare the results obtained with the different operations. We have been particularly interested in a comparison of the results following the shunt procedures and arterial ligations.

MATERIAL AND METHODS

A total of 38 cases of portal hypertension have been treated surgically during the past seven years by members of the Surgical Department of the University of Cincinnati (Table I). Four cases were treated by splenectomy alone, and thirteen were subjected to venous shunt procedures. Seven of these were splenorenal, five portacaval, and one superior mesenteroportal. In three patients with severe bleeding and shock, trans-thoracic ligation of esophageal varices was done. The remaining eighteen were subjected to ligation of the hepatic and splenic arteries with or without ligation of the left gastric artery during the past three and a half years.

The results of therapy in these patients were studied in an effort to derive any information which would aid in assessing the value and limitations of the different procedures.

*Professor of Surgery and Chairman of the Department, University of Cincinnati and Cincinnati General Hospital. Presented as the Presidential Address, Henry Ford Hospital Medical Association, Detroit, Michigan, November 13, 1954.

The causes of the portal hypertension in this series were portal cirrhosis in 23 cases, post-necrotic cirrhosis in eight, hepar lobatum in one, extrahepatic venous thrombosis in five, and cirrhosis and extrahepatic venous thrombosis in one. (Table II) The case of portal hypertension produced by congenital syphilitic hepar lobatum is apparently unique in that we have been unable to find a similar one reported.

TABLE I
PORTAL HYPERTENSION
ORIGINAL SURGICAL PROCEDURES USED

Type of Operation	Number of Cases
Thoracotomy with ligation of esophageal varices	3
Splenectomy	4
Splenorenal anastomosis	7
Portacaval anastomosis	5
Mesenterocaval anastomosis	1
Hepatic and splenic artery ligation	9
Hepatic, splenic and left gastric artery ligation	9
TOTAL	38

TABLE II
LOCATION AND CAUSE OF PORTAL BLOCK

Location of Portal Block	Number of Cases	Cause of Portal Block
Thoracotomy with ligation of esophageal varices	3	Portal cirrhosis
Splenectomy Cases:		
Extrahepatic	3	Thrombosis of portal or splenic vein
Intra and Extrahepatic	1	Cirrhosis and thrombosis of portal vein
Venous Shunt Cases:		
Intrahepatic	5	Portal cirrhosis
	5	Post-necrotic cirrhosis
	1	Hepar lobatum
Extrahepatic	2	Thrombosis portal vein
Arterial Ligation Cases:		
Intrahepatic	15	Portal cirrhosis
	3	Post-necrotic cirrhosis

The average age of the patients was 58.3 years for the thoracotomy cases, 36.7 years for the splenectomy cases, 40.5 years for those treated by shunt, and 46.7 for those treated by arterial ligation. The oldest patient was 69 and the youngest was 11 years.

All of the twenty patients treated by either thoracotomy, splenectomy, or one of the shunt procedures, had had two or more episodes of bleeding pre-operatively. In the series treated by hepatic artery ligation, thirteen patients had varying degrees of both ascites and severe gastro-intestinal hemorrhage, while four had hemorrhage only, and one had ascites only. (Table III) In general, those patients subjected to arterial ligation were obviously more seriously ill and much greater surgical risks.

Pre-operatively, each patient received a complete physical examination and routine laboratory work, as well as the following tests: blood urea nitrogen, prothrombin time, thymol turbidity, serum bilirubin, serum protein, bromsulfalene, cephalin flocculation,

and x-ray examination after barium swallow. All but five of the patients also received liver biopsies.

In the last three patients undergoing venous shunts, visualization of the portal vein immediately pre-operatively was done by intrasplenic infusion of 20 cc. of Neo-Iopax

TABLE III
INCIDENCE OF PRE-OPERATIVE HEMORRHAGE
FROM ESOPHAGEAL VARICES AND OF ASCITES

Operation	Number of Cases	Pre-operative Incidence (cases)		
		Hemorrhage Only	Ascites Only	Hemorrhage and Ascites
Thoracotomy	3	2	0	1
Splenectomy	4	3	0	1
Venous Shunt	13	10	0	3
Arterial Ligation	18	4	1	13

(70% solution) through a 19 gauge stylet needle according to the method of Leger⁽⁹⁾ and Rousselot.⁽¹⁰⁾ This proved to be quite helpful in determining the point of portal block and the selection of the surgical procedure which was indicated. Either a portacaval or splenorenal shunt could be decided upon when the block was intrahepatic and the portal and splenic veins were intact. A splenorenal shunt was performed in the presence of an obliterated portal vein but an intact splenic vein.

RESULTS

In general, the results of treatment of patients with portal hypertension are difficult to assess. The number of variable factors in a relatively small group of cases precludes any conclusions. On the other hand, several interesting observations were made in the cases treated by the various methods.

The immediate postoperative course was uncomplicated in 2 of the 38 cases.

Death occurred within three days as the result of hepatic coma and shock in all three of the patients subjected to emergency thoracotomy and suture of the bleeding esophageal varices as a desperate attempt to arrest the otherwise uncontrollable hemorrhage.

In those patients undergoing splenectomy or venous shunt procedures the immediate postoperative mortality was zero within three weeks after operation. The immediate postoperative mortality in the cases treated with hepatic and splenic arterial ligations was 11.2 per cent, however. An additional five deaths occurred among the arterial ligation cases within four and a half months, but one of these deaths was not related to the portal hypertension. Another fatality occurred in a patient who had developed recurrent bleeding 25 months after arterial ligation and who had then undergone a portacaval shunt procedure 19 months after ligation. The latter was also unsuccessful, and the patient died. A ninth death occurred in a patient in whom a portacaval shunt was done 38 months after hepatic, splenic, and left gastric arterial ligation. Hepatic failure, coma, and death occurred three and a half months after the portacaval shunt. These two patients are unique in that they are the only ones to our knowledge in whom interruption of the arterial circulation of the liver was followed by a later and secondary shunt operation. The number of deaths in the cases undergoing ligation, therefore, gives an overall mortality of 5.0 per cent for a three and a half year period after operation.

All of the seven patients subjected to a splenorenal shunt are still alive and well. Three of the five patients undergoing portacaval shunt have died, one as the result of a strangulated diaphragmatic hernia, one of recurrent gastro-intestinal hemorrhage, and one of progressive liver failure. This number of deaths in the thirteen cases treated

by venous shunt gives an overall mortality rate of 23.0 per cent.

In six of the 18 cases, 33.3 per cent, treated by arterial ligation, recurrent hemorrhage from esophageal varices has occurred. It was fatal in two and varied from minor to moderate in severity in four. In addition, generalized bleeding developed from all of the mucous membranes during hepatic coma due to liver failure in another patient, but not from the varices.

In three of the four patients who originally underwent splenectomy, recurrent and massive bleeding developed. A secondary portacaval shunt has been done successfully in one, and a Plemister procedure was performed in two when post-thrombotic obliteration of the portal vein prevented the completion of a portacaval shunt.

Of particular interest are the thirteen patients with both ascites and esophageal bleeding who were treated by arterial ligation. Eleven survived a postoperative period of four and a half months and none of these showed a recurrence of the ascites, and six have had no further episodes of bleeding.

Of the three patients treated by a venous shunt for ascites and hemorrhage, recurrent ascites occurred in one.

The results were poorer in the patients with post-necrotic cirrhosis who underwent hepatic arterial ligation. The mortality was high, all three patients with this lesion dying within three months of severe liver failure, while three of the four patients with post-necrotic cirrhosis treated by venous shunt have survived and have done well.

In addition to the fatal complications, others encountered were coma, protracted hypoproteinemia, atelectasis, laryngeal edema, delirium tremens, subcutaneous emphysema, and postoperative wound bleeding after heparinization.

COMMENTS

An analysis of these results has emphasized that patients with portal hypertension complicated by esophageal varices and gastro-intestinal hemorrhage are poor risk patients whose clinical problems are not confined to the hypertension within part or all of the portal bed. Severe disease of the liver may persist and may progress postoperatively to the point of invalidism or death, even though the hypertension be corrected. Hepatic coma may occur postoperatively, varices may persist, hemorrhagic tendencies from hypoprothrombonemia or thrombocytopenia may develop, or delayed or secondary thrombosis of the portacaval shunt may occur.

The poor results obtained with splenectomy alone in the four patients with extra-hepatic portal blocks emphasize the recommendation that splenectomy alone should be rarely done in the treatment of portal hypertension. Instead, it is our opinion that a splenorenal venous shunt should be done at the time of splenectomy. Rousselot, Blake-more and Linton have previously stressed their belief that a splenectomy should not be done in patients with portal hypertension unless one is prepared to proceed with a splenorenal shunt. Failure to do so may result in obliteration of the splenic vein post-operatively, thereby preventing a subsequent or later splenorenal procedure. In the post splenectomy cases with post-phlebotic obliteration of the portal vein, a portacaval shunt is likewise impossible, and the surgeon is faced with the necessity of doing a more difficult and tedious superior mesenterocaval anastomosis or a Plemister procedure. It is significant to note that a Plemister operation became necessary in two of the four cases treated originally by splenectomy. Both are alive and in a fair state of health three years and one year postoperatively, although the latter has had one minor episode of recurrent hemorrhage.

In general, the results which we have obtained with the splenorenal and portacaval shunts have been superior to those obtained with hepatic and splenic arterial ligation which were done for portal hypertension with recurrent hemorrhage. The mortality rate has been considerably less, both in the immediate postoperative period and during the period of follow-up. The best results were obtained with the splenorenal shunts, all patients with this procedure being alive and well.

The anastomosis used in our cases was of the end-to-side type. It is interesting to note that the studies of Preshaw, Large, and Johnson⁽¹¹⁾ of Detroit indicate that the liver with complete shunt does not tolerate damage as well as the normal liver or the liver with partial diversion of the portal flow. They infer that the already damaged human cirrhotic liver might be further embarrassed by complete diversion of the portal blood to the vena cava, and this might be an explanation for the better results obtained in our cases treated by splenorenal anastomosis.

Although it must be remembered that the cases selected for hepatic and splenic arterial ligation were generally much greater risks than those selected for venous shunt, an immediate postoperative mortality rate of 11.2 per cent and an overall mortality rate of 50.0 per cent for the arterial ligation group is significantly higher than an immediate mortality of zero and an overall mortality of 23.0 per cent in the venous shunt group.

A recurrence of hemorrhage from esophageal or rectal varices occurred in six of the sixteen patients (37.5 per cent) surviving the immediate postoperative period of three weeks after arterial ligation, while the same complication has developed in four of thirteen patients (30.7 per cent) after venous shunting. In two of the latter four cases, however, the bleeding has been minimal and limited to small hematemesis. This suggests that the results in patients with portal hypertension and hemorrhage have been better in the patients treated by splenorenal or portacaval shunts, although it must be remembered that six patients of the original eighteen treated by arterial ligations have had no bleeding since operation 33 to 42 months ago. Considering the higher mortality rate obtained with the arterial ligation procedure, however, the operation of choice appears to be a splenorenal or portacaval shunt, particularly in the greater risk patients.

Madden⁽¹¹⁾ of New York has seriously questioned the value of ligation of the hepatic and splenic arteries in the treatment of cirrhosis of the liver complicated by hemorrhage. Eight patients were treated by arterial ligation. Four died in the immediate postoperative period, one of cardiac arrest during operation, one of massive intraperitoneal hemorrhage three hours postoperatively, one of massive hematemesis forty hours postoperatively, and one of anuria on the ninth postoperateday. Only one of the four patients who survived operation is living, but has recurrent ascites and is in poor condition. One patient died seven months after operation and autopsy showed ruptured esophageal varices but no necrosis of the liver or spleen. One patient died three weeks after operation of hepatic insufficiency and hematemesis. Autopsy revealed no necrosis of the liver or spleen. Another patient died four months postoperatively of peritonitis. The bowel was perforated during paracentesis for recurrent ascites. A critical analysis of the results of Madden does not indicate that this procedure is not efficacious. Instead, it emphasizes the technical difficulties and dangers attending it.

McFadzean and Cook⁽¹²⁾ in 1953 ligated the hepatic artery in five Chinese men with portal hypertension and esophageal varices. Of four patients followed, all had recurrent bleeding and two died. The operation failed to correct the hypersplenism

which all the patients had. They concluded the operation, therefore, was useless in the treatment of portal hypertension.

The value of hepatic arterial ligation in cases of ascites, however, appears possibly to be greater than that of a venous shunt. It is interesting to note that none of these patients developed recurrent ascites if they survived the postoperative period of four and a half months. This possibility needs further study for clarification. Similar promising results have been reported by Reinhoff and Woods⁽¹³⁾ in the treatment of ascites.

The high mortality rate obtained in our cases of post-necrotic cirrhosis treated by hepatic artery ligation suggests that this procedure is too hazardous for this condition.⁽¹⁴⁾ The development of severe and fatal liver insufficiency three to five weeks postoperatively with coma, shock, massive ascites, uremia, generalized bleeding from all mucous surfaces and terminal pneumonia is a complication which will preclude the use of hepatic artery ligation in our cases of post-necrotic cirrhosis in the future.

The value of excellent medical assistance in the diagnosis and treatment of these patients cannot be over-emphasized.

SUMMARY

On the basis of this study of surgically treated cases of portal hypertension, the operative formation of a venous shunt between the portal vein and the vena cava or the splenic and renal veins, is the most satisfactory and safest procedure for the treatment of recurrent hemorrhage from esophageal varices. Patients with severe and advanced cirrhosis were greater surgical risks than those with extrahepatic blocks.

The splenorenal shunt has given the best results in our hands. Hepatic and splenic arterial ligation has given irregular results in patients with portal hypertension and hemorrhage, the results being good in only one-third of the cases. The higher overall mortality rate obtained with the arterial ligation procedure has made it more dangerous than the venous shunt. Since a very high mortality rate occurred in patients with post-necrotic cirrhosis who were subjected to hepatic arterial ligation, this condition should probably be considered a contra-indication for this operation.

BIBLIOGRAPHY

1. Whipple, A. O.: Problem of portal hypertension in relation to hepatosplenopathies, *Ann. Surg.* 122:449, 1955.
2. Blakemore, A. H.: Uortacaval shunting for portal hypertension, *Surg., Gynec. & Obst.* 94:443, 1952.
3. Rousselot, L. M.: Surgical therapy for gastrointestinal hemorrhage in portal hypertension, *Rev. Gastroenterol.* 18:575, 1951.
4. Linton, R. R.: Surgical treatment of bleeding esophageal varices secondary to cirrhosis of liver and Banti's syndrome, *Cincinnati J. Med.* 34:197, 1953.
5. Berman, J. K., Koenig, H., and Muller, L. P.: Ligation of hepatic and splenic arteries in treatment of portal hypertension; ligation in atrophic cirrhosis of liver, *A.M.A. Arch. Surg.* 63:379, 1951.
6. Rienhoff, W. F., Jr.: Ligation of hepatic and splenic arteries in treatment of portal hypertension with report of 6 cases; preliminary report, *Bull. Johns Hopkins Hosp.* 88:368, 1951.
7. Altemeier, W. A., Hoxworth, P. I., McElhinney, W. T., Giuseffi, J., MacMillan, B., and Todd, G.: The treatment of portal hypertension, *Am. Surgeon* 20:1235, 1954.
8. Plemister, D. B., and Humphreys, E. M.: Gastro-esophageal resection and total gastrectomy in treatment of bleeding varicose veins in Banti's syndrome, *Ann. Surg.* 126:397, 1947.
9. Leger, L.: Phlebographie portale par injection splénique intra parenchymateuse, *Mem. Acad. chir., Par.* 77:712, 1951.
10. Rousselot, L. M.: Personal communication to author.
11. Preshaw, D. E., Large, A., and Johnson, A. F.: Effect of portacaval venous shunt on sulfobromophthalein (Bromsulphalein) retention, *A.M.A. Arch. Surg.* 62:801, 1951.
12. Madden, J. L.: Clinical evaluation of ligation of hepatic and splenic arteries in the treatment of cirrhosis of the liver, *Rev. Gastroenterol.* 20:300, 1953.
13. McFadzean, A. J. S., and Cook, J.: Ligation of splenic and hepatic arteries in portal hypertension, *Lancet* 1:615, 1953.
14. Rienhoff, W. F., Jr., and Woods, A. C., Jr.: Ligation of hepatic and splenic arteries in treatment of cirrhosis with ascites, *J.A.M.A.* 152:687, 1953.