Hemodynamic Changes with Cirrhosis of the Liver:

Control of Arteriovenous Shunts during Operation for Esophageal Varices

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ADMIXTURE of arterial and venous blood is a normal finding in the portal vein and can be explained by the presence of arteriovenous shunts in the viscera drained by the portal vein. The presence of these shunts has been well documented by previous reports from our laboratory and the work of others.[22, 23, 30, 31, 32]

In patients with cirrhosis there is evidence of increased arterial flow through these shunts. A high oxygen saturation has been found in the portal vein by Keys and Snell [16] and by Peters and Womack.[23] An increased number of gross and microscopic arteriovenous shunts in liver, esophagus, and spleen removed from cirrhotic patients has been demonstrated. [23] Lesions that look like angiomas have been noted in the gastric mucosa of cirrhotic patients with gastrointestinal bleeding. On microscopic examination these lesions prove to be arteriovenous communications, and in several patients they have appeared to be the site of hemorrhage. Hales *et al.*[11] demonstrated enlarged intrahepatic arteries in the liver of patients with cirrhosis, suggesting increased hepatic artery flow. His injection studies also showed admixture of the arterial with the venous perfusate.

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Increased cardiac output in patients with cirrhosis has been documented, but is unexplained.[5, 17, 18] Adrenalin has been shown to open arteriovenous shunts in the portal system, cause an increase in portal vein flow,[2] and an increase in cardiac output; whereas the decrease in portal vein flow produced by pituitrin is associated with a decrease in cardiac output.[1] Peters and Carter [24] have shown in dogs a relationship between cardiac output and portal vein flow. Nokano and DeSchryver [21] demonstrated a direct relationship between cardiac output and flow through an arteriovenous fistula. A hypothesis to explain the increase in cardiac output in patients with cirrhosis is that the portal vein and hepatic blood flow are increased due to increased flow through the multiple arteriovenous fistulae.

Alterations in hemodynamics found in the presence of a systemic arteriovenous shunt have been reported.[8, 14, 21, 29] These are decrease in peripheral resistance, accompanied by increase in cardiac output, stroke volume and pulse rate. Diastolic and even systolic pressure may fall while pulse pressure increases. If the hypothesis above is valid, alterations in circulatory dynamics similar to these should be present in cirrhotics.

To test this hypothesis cardiac output, arterial and right atrial pressures, and arte-

rial and venous oxygen saturation have been studied in a group of patients with cirrhosis of the liver and in a control group of hospitalized patients. Also evaluated is an operation designed to remove or to decrease blood supply to the abnormal arteriovenous shunts in the portal system and decrease gastric acidity.

Method

Study of Hemodynamics

Thirty-seven patients with cirrhosis and 34 control patients were used for the hemodynamic study in this series. The diagnosis of cirrhosis was usually made by liver biopsy, but in a few patients it was made on the basis of clinical findings of esophageal varices, ascites, abnormal liver chemical studies, and a history of excessive alcoholic intake. In no instances were the subjects in either group acutely ill. Patients used for control were hospitalized patients with no evidence of hepatic disease. They included patients prior to elective thoracotomy, patients awaiting minor operations, or patients admitted for diagnostic evaluation.

The studies were performed with the patients supine, without medication, and having carried out their usual hospital activities. A Cournand needle was placed into a brachial artery and a No. 110 PE catheter threaded through a thin-walled needle in the femoral vein up into the right atrium. Its position was verified by the recording of a pressure tracing typical of the right atrial pressure.

Oxygen saturation was measured on blood samples from the arterial and right atrial catheters using an American Optical Reflectance Oximeter, Model 10800. These samples were obtained while the patient breathed room air and each determination was made within a minute after collection.

Dye-dilution curves were inscribed after injection of indocyanine green dye using a Waters densitometer. The resulting curves were analyzed using the principles of Stewart [27] and Hamilton [12, 13] for the determination of cardiac output and central blood volume. The latter indicates the amount of blood from the site of injection to the site of sampling.

Surface area was derived from a DuBois chart using the height and weight of the patient. Cardiac index, stroke volume, stroke index and peripheral resistance were calculated from the information already described. Peripheral resistance is presented in units as suggested by Green,[10] rather than multiplying by a constant. For example, if we assume a cardiac output of 90 cc. per second and a mean arterial pressure of 90 mm. Hg, mean pressure divided by cardiac output would give a resistance of one unit.

Total blood volume was determined by the method described by Buckwalter,[3] using radioactive iodinated serum albumin. This was related to the surface area of the patient. Controls for blood volume determinations were made on patients without liver disease. These were not the same patients used for the controls of the other studies.

To evaluate further whether a hyperdynamic state [9] existed in these patients the mean systolic ejection rate (stroke volume in cubic centimeters divided by the length of systole in seconds) and the left ventricular stroke work (stroke volume times the mean arterial pressure) were calculated.

The mean of each type of determination from the patients with cirrhosis was compared to that of the controls by the Students T test. The relationship between cardiac output and RISA blood volume and between central blood volume and RISA blood volume was evaluated by the coefficient of correlations. Data from these two relationships were then subjected to linear regression analysis.

Operative Series

Fifty-one individuals were operated upon for bleeding esophageal varices at the

North Carolina Memorial Hospital during the 12-year period from July, 1953 to July, 1965. The basic operation consists of an incision through the chest in the seventh or eighth interspace; after opening the diaphragm the spleen is removed, a long portion of the greater curvature of the stomach is resected and particular attention is paid to dividing arterial branches to the cardia. The type of vascular distortion found dictates the next portion of the operation. If large esophageal varices are the main problem, they are oversewn; should an ulcer appear at the esophagastric junction, a vagotomy and pyloroplasty are performed. Large gastric varices or smaller angiomas in the upper stomach require multiple ligation with transfixion sutures. A liver biopsy is taken and a gastrostomy tube inserted in most cases.

The degree of hepatic disease and reserve was estimated using criteria established by Wantz and Payne.[28] The number of blood transfusions given preoperatively, during the operation, and after operation, has been used to indicate the amount of blood lost.

Follow up was obtained in all patients and results have been studied using the life table analysis method described by Cutler.[4] Results are compared to current reports of 5-year survival following portal systemic shunts.

Results

Hemodynamic Studies

A summary of the hemodynamic studies is listed in Table 1. The control and cirrhotic groups had no significant difference between mean values of arterial mean blood pressure, pulse pressure and diastolic pressure, right atrial pressure, pulse rate, venous and arterial oxygen saturation and arteriovenous oxygen differences.

Mean cardiac index in cirrhotic patients, 4.1 liters per minute, was 41 per cent higher than that in controls, 2.9 L/min. ($p <$

TABLE 1. *Results of Circulatory Studies*

Study	Mean of Controls	No. in Study	Standard Deviation	Standard Error	Mean of Cirrhotics	No. in Study	Standard Deviation	Standard Error	Significance*
Cardiac index L./min./M.^{2}	2.9	32	0.6	0.1	4.1	35	1.2	0.2	<0.001
Stroke index ml./M.^{2}	36	30	8.8	1.6	51	34	15.6	2.7	<0.001
Peripheral resistance units	1.2	12	0.28	0.08	0.8	35	0.30	0.05	<0.001
Total blood volume ml./M.^{2}	2,950	24	247	50	3,296	23	576	128	<0.005
Central blood volume ml./M.^{2}	761	25	180	40	953	23	360	80	<0.02
Mean systolic ejection rate ml./systolic sec/M.^{2}	112	15	26	7	153	28	55	10	<0.01
Stroke work gram meters	73	15	25	6	98	28	30	6	<0.001
Arterial mean blood pressure mm. Hg	89	11	7.8	2.5	85	34	13.3	2.3	>0.5
Pulse rate beats/minute	81	29	15.5	2.9	83	34	14.5	2.5	>0.3
Arterial oxygen saturation per cent	95	29	2.0	0.4	94	33	2.7	0.5	>0.1
Right atrial oxygen saturation per cent	68	12	9.3	2.7	72	24	8.8	1.8	>0.2
Right atrial pressure mm. Hg	5	6	2.0	0.8	5	23	1.2	0.3	>0.4

* Significance (p) of the difference between the mean of control patients and those with cirrhosis.

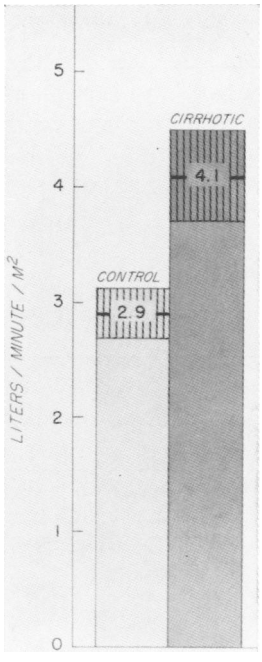

FIG. 1. Bar graph comparing cardiac index in cirrhotic and control groups. Area between the horizontal lines represents twice the standing error on either side of the mean. The same applies for Fig. 2–6.

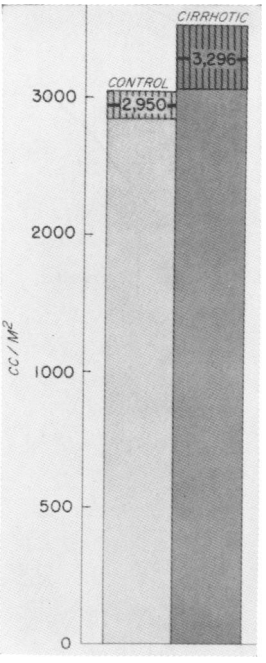

FIG. 3. Bar graph comparing total blood volume.

0.001) (Fig. 1). This was due to an increase in the mean stroke index in patients with cirrhosis rather than a difference in pulse rate. Mean peripheral resistance was 33 per cent lower in cirrhotics ($p < 0.001$) (Fig. 2).

Mean blood volume per square meter as determined by radioactive iodinated serum albumin was increased 12 per cent in cirrhotic patients ($p < 0.005$) (Fig. 3). Mean central blood volume per square meter was increased by 25 per cent in patients with cirrhosis ($p < 0.02$) (Fig. 4).

The coefficient of correlation between cardiac index and blood volume per square meter was +0.25, not significantly different from zero correlation. However, correlation between central blood volume per square meter and blood volume per square meter

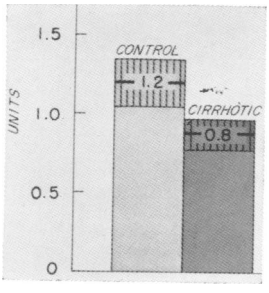

FIG. 2. Bar graph comparing peripheral resistance.

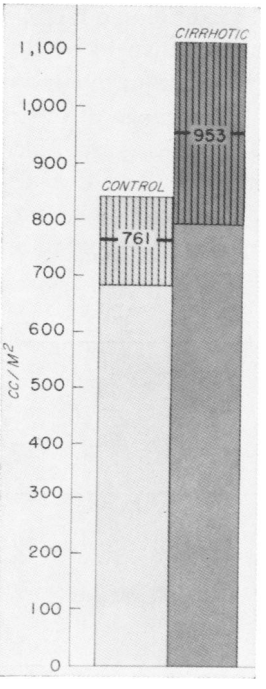

FIG. 4. Bar graph comparing central blood volume.

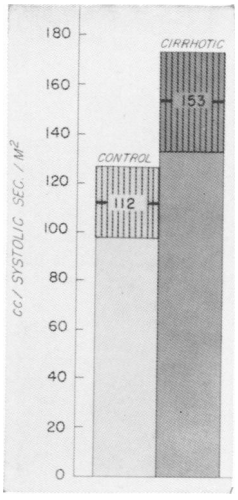

FIG. 5. Bar graph comparing mean systolic ejecting rate.

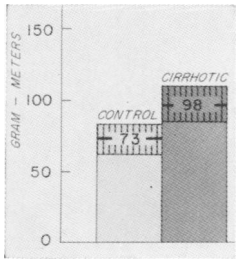

FIG. 6. Bar graph comparing stroke work.

as determined with RISA was +0.56. Thus as the central blood volume increased, the blood volume increased; a relationship that was shown to be linear when subjected to a least square analysis ($p < 0.0001$).

The mean of mean systolic ejection rates for patients with cirrhosis was 37 per cent greater than for controls and the mean stroke work was increased 34 per cent in cirrhotic patients (Fig. 5, 6).

TABLE 2. *Mortality According to Age Distribution in Operative Series*

Age	Patients	Still Alive
0–9	3	1
10–19	5	2
20–29	3	1
30–39	8	3
40–49	12	5
50–59	13	5
60–69	7	1
Total	51	18

TABLE 3. *Mortality According to Diagnoses in Operative Series*

	Total	Still Alive
Laennec's cirrhosis	25	10
Non-alcoholic (adult) cirrhosis	17	5
Juvenile cirrhosis	3	0
Childhood varices (no cirrhosis)	5	2
Splenic artery aneurysm	1*	1

* Does not contribute to follow up; operated within last 6 months.

Operative Series

Of the 51 patients operated upon, 34 were men and 17 women, with an age distribution as listed in Table 2. Diagnoses in this group of patients are listed in Table 3.

Three general types of operations have been performed. Type I is splenectomy only, Type II is splenectomy plus resection of the superior one half of the greater curvature of the stomach, and ligation of the left gastric or ascending branch of the

TABLE 4. *Operation Type, Grade, and Operative Mortality*

	I Splenectomy		II I and Ligation Gastric Blood Supply		III II and Plication of Varices		All Operation Types	
	Total	Died	Total	Died	Total	Died	Total	Died
Grade								
A	0	0	6	0	4	1	10	1
B	3	0	8	5	6	1	17	6
C	2	1	12	7	10	7	24	15
All grades	5	1	26	12	20	9	51	22

left gastric artery or both, and Type III is Type II plus intraluminal ligation of esophageal varices. We no longer consider splenectomy alone an adequate operation for bleeding esophageal varices. Twenty-seven (53%) operations were performed as emergencies to control bleeding.

Death in the postoperative period prior to discharge or within 30 days of operation occurred in 22 patients. Distribution of postoperative mortality by operation type and clinical grade of disease is listed in Table 4. Of five patients who had no splenectomy, three died postoperatively. The 22 patients who died in the postoperative period received an average of 19 units of blood in the preoperative period, whereas the patients who lived received only 10 units. A breakdown of the time of transfusion is given in Figure 7.

Seventeen of 22 patients who died in the postoperative period were operated upon as an emergency. Only ten of 29 patients who survived operation were operated upon as emergencies. Of the 22 patients who died all but one were Grade C or were operated upon to control acute hemorrhage.

Death in 13 was due to liver failure and to continued bleeding in six. Respiratory failure, a ruptured esophagus due to a Sengstaken-Blakemore tube, and uncontrollable convulsions each accounted for one death.

Survival as calculated by the life table is shown in Table 5. Five-year survival excluding postoperative mortality was 75 per cent. Including postoperative mortality 43 per cent survived.

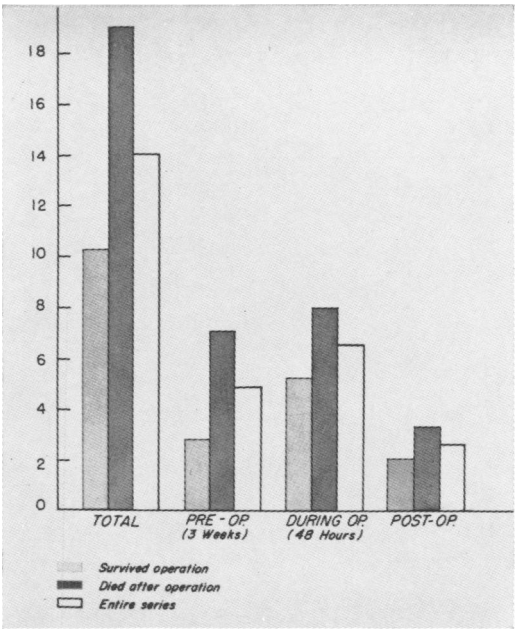

FIG. 7. Bar graph depicting: 1) the mean of the total number of transfusions during hospitalization of the patients who survived operation, those who died, and the total group; 2) the same for the preoperative period; 3) the same for the time of operation; and 6) the same for the postoperative period.

Seven patients survived more than five years (Table 6). All these patients were in the clinical Grades A or B, and in four the cirrhosis was not associated with ingestion of alcohol.

Upper gastrointestinal bleeding occurred in 13 patients following discharge from the hospital. Table 7 lists some of the details. Two had an inadequate primary operation

TABLE 5. *Life Table Analysis of Survival Following Operation* (100% Follow Up)

Years after Operation	Alive at Beginning of Interval	Died during Interval		Withdrawn Alive during Interval	Effective Number Exposed to Risk of Dying in Interval	Cumulative % Survival through Interval Including Postop. Deaths	Cumulative % Survival through Interval Excluding Postop. Deaths
		From Disease	From Other Causes				
0–1	51	25*		2	50.0 (28.0)	50	89
1–2	24	1	1	2	22.5	48	85
2–3	20	2	1	1	19.0	46	81
3–4	16	1		4	14.0	43	75
4–5	11		1	3	8.5	43	75

* 22 classified as postoperative deaths.

TABLE 6. *Patients Surviving Longer Than 5 Years*

Patient	Age Sex	Grade	Etiology Varcies	Type Operation	Time since Operation (Years)	Comment
MP	41 F	A	Non-alcoholic cirrhosis	II	6	Splenectomy performed 3 years after first operation, well since first operation.
MG	39 F	B	Non-alcoholic cirrhosis	III (Resect distal esophagus)	8	Several mild bleeding episodes in last 3 years.
HE	38 M	A	Alcoholic cirrhosis	II	7	Intraluminal ligation varices 1 year after first operation; continued to drink—massive bleeding episode 5 years—died suicide.
WB	47 M	A	Non-alcoholic cirrhosis	II	8	Well since first operation.
AL	50 M	B	Alcoholic cirrhosis	III (Resect distal esophagus)	7	Mild bleeding episode 6 years; treated with tube tamponade; well since.
HB	25 M	B	Non-alcoholic cirrhosis	I	10	One year after first operation rebled and had intraluminal ligation of varices, a partial gastric resection, and vagotomy —he continued to have episodes of bleeding and 5 years after first operation the left gastric artery was ligated and vessels along lesser and greater curvature ligated; he has done well since except for difficulties from regional enteritis.
CB	39 M	A	Alcoholic cirrhosis	III	11	Several episodes of bleeding after first operation—2 years later had a resection of greater curvature of stomach, a vagotomy and pyloroplasty; well since.

consisting of only a splenectomy and required a second operation after which A. N. died from liver failure. Seven more patients were reoperated upon and six survived. Four were not reoperated upon, two of whom died from acute hemorrhage. Eight of 13 patients are still living. The majority of the 29 patients who survived operation were able to return to gainful occupations. Those that continued to drink alcohol have not done as well. No cerebral symptoms were manifest in the survivors.

Discussion

Results of these studies of circulatory dynamics in the cirrhotic patients demonstrate a hyperkinetic state of the cardiovascular system and are similar to those found in patients with peripheral arteriovenous shunts. The increase in cardiac output, stroke volume, blood volume and decrease in peripheral resistance results from an increase in the amount of flow through arteriovenous anastomoses in the portal system.

The hyperkinetic state is further evidenced by increase in mean systolic ejection rate and stroke work in patients with cirrhosis. Both of these values reflect the increased stroke volume with normal pulse rate and blood pressure, and indicate the increased burden placed on the heart in this condition.

A similar alteration in circulatory dynamics in patients with cirrhosis has been described by Kowalski *et al.*[17, 18] They reported an increase in cardiac output in cirrhotic patients which was even greater if ascites were present. Murray *et al.*[20] report similar findings in a series of cirrhotic

TABLE 7. *Patients Who Rebled following Original Operation*

Patient	Age Sex	Original Operation	Time Interval to Second Operation (Months)	Second Operation	Total Time since First Operation (Years)	Comments
KP	49 M	II	17	II (Religated vessels to stomach, vagotomy)	4	Three mild bleeding episodes since last operation; still drinking.
JW	14 M	II	6	III	3	Childhood varices, two mild bleeding episodes since last operation.
GD	14 M	III No ligation gastric	9	III (Ligation left gastric)	1	Childhood varices, well for the year since last operation.
HE	38 M	II	13	III	7	Death due suicide: One massive bleeding episode 6 years after first operation while continuing to drink.
RL	3 M	III	6	III (Religate varices)	3	Childhood varices, five mild bleeding episodes since last operation.
HB	25 M	I	10	III (No ligation left gastric or greater curvature)		
			61	III (Ligation left gastric and resect greater curvature)	10	Well for 4 years since last operation—enterostomy for regional ileitis
AN	13 M	I	13	III	1	Juvenile cirrhosis: died following second operation of liver failure.
CB	39 M	III	22	III (Resect greater curvature; vagotomy, pyloroplasty)	11	Well for 8 years since last operation.
FW	61 M	III (No vessel ligation; no resection of greater curvature)	1	III (Ligation left gastric)		
			3	III	0	Died following third operation of liver failure.
MG	39 F	II			6	Alive, no bleeding episode since 1963.
AL	50 M	II			7	Treated tube tamponade in 1963; well since.
JF	25 F	III			0	Died due to bleeding 3 months after operation.
RD	42 M	II			3	Died due to bleeding 3 years after operation.

patients. They suggest that most patients with cirrhosis expire of liver failure before congestive heart failure is manifested. Del Guercio's [5] recent extensive review of the hemodynamics in a series of cirrhotics suggested the final downfall in many instances was related to cardiac failure rather than liver failure. In his patients with massive bleeding the venous pressure was elevated despite a fall in cardiac output, indicating

an overloaded heart. It is possible that many patients thought to have died of liver failure expired from unrecognized cardiac failure complicating the cirrhotic syndrome.

Total blood volume was increased in patients with cirrhosis and the correlation between the increase in total and central volumes places a significant amount of the total increase in the central compartment. The validity of the measurement of the central blood volume has been questioned.[19] However, since this is a comparative study any error was introduced in both groups and the difference between the two should be valid. Whether the large central blood volume is an early indication of a failing heart is not clear.

Animals with an arteriovenous fistula and an otherwise hyperdynamic cardiovascular system may have a normal pressure, pulse rate and right atrial pressure. Elkin and Warren[6] and Frank *et al.*[8] have found the increase in cardiac output to result primarily from an increase in stroke volume. Tachycardia, hypotension and elevation of right atrial pressure apparently develop when an increase in the stroke volume alone cannot maintain adequate cardiac output to both perfuse tissues and provide for increased flow through the low resistance shunt. Lack of alteration in blood pressure, pulse rate, right atrial pressure and oxygen saturation in this study of cirrhotic patients primarily reflects the selection of patients with compensated cirrhosis. As previously stated, none of the patients were in seriously ill condition at the time of the circulatory studies.

A lowered arterial oxygen saturation and an increased arteriovenous difference in patients with cirrhosis has been demonstrated in previous studies.[7, 16] This has been attributed to right-to-left shunts in the lungs known to be functioning in patients with cirrhosis. The finding of a normal mean arterial oxygen saturation in patients with cirrhosis in this series may be a consequence of selection of patients with well compensated disease and with minimal pulmonary right to left shunting of blood. Fritts[7] suggested a decreased affinity of hemoglobin for oxygen as a cause for oxygen unsaturation in patients with cirrhosis. Since we examined blood samples immediately, it is conceivable that sufficient time had not elapsed for desaturation to occur. It is also possible that, had we determined the partial pressure of oxygen in the blood samples, some pulmonary arteriovenous shunting would have been demonstrated.

Thus a concept that accounts for the basic abnormalities of the circulation present in cirrhosis of the liver centers on the presence of numerous microscopic arteriovenous shunts which in turn provoke a hyperdynamic local and systemic state. The anastomotic channels known to be present cause or aggravate disease when the amount of blood flowing through them is too great. The derangement of this humoral control may be the result of hepatic cell damage. Intra- or extrahepatic obstruction of the portal vein is not essential for the development of esophageal varices but an increase in flow in the portal bed is a prerequisite. Although these shunts are scattered throughout the body as evidenced by spider angioma and pulmonary arteriovenous shunts, their malfunction in the portal system causes the majority of the symptoms. An increase in flow through arteriovenous shunts in the gastric and esophageal submucosa will cause a rise in the mucosal vein pressure and produce varices or angiomata. Arteriovenous shunts in the spleen and stomach lead to an increase in flow and oxygen saturation in the portal vein. An increase in portal vein flow can increase portal vein pressure or, if resistance in the liver falls, the pressure may remain the same. Thus a varix can exist with or without portal vein hypertension, depending on the resistance to flow through the liver.

Intrahepatic hepatic artery to portal vein shunts may cause functional obstruction to

portal vein flow even though no anatomic obstruction can be demonstrated. This possibility has been demonstrated by studies of local hemodynamics around an arteriovenous fistula.[15] Turbulence and the high pressure associated with arterial blood flow into the "peripheral" vein causes a dynamic obstruction to flow through the "peripheral" or, in this case, portal vein. This causes an apparent increased resistance to flow in the portal vein, and coupled with increased flow, portal hypertension results. Even reverse flow in the portal vein which has been described can be explained by the presence of greatly increased flow through intrahepatic arteriovenous shunts.

The clinical results speak for themselves. Postoperative mortality is too high, but compares favorably with other series if the large number of poor risk patients and emergency operations are considered. Rodriguez[25] recently reported a series of patients in whom portal systemic shunts were created. Fourteen were done as emergencies and ten died. Poor risk patients made up 31 per cent of Rodriguez' series with a 48 per cent operative mortality. Satterfield[26] did not do emergency portal systemic shunts. Although he did not grade his patients, 68 of 80 had serum albumin levels greater than 3 Gm./100 cc. which would probably classify them as Grade A.

In the two reported series, however, few patients were operated upon for acute hemorrhage and a smaller percentage than in our series was classed as Grade C. If we correct our series by eliminating patients in Grade C to make the population more comparable to the other series, results are obviously superior as shown by the top curve in Figure 8.

It is obvious that better methods of selecting patients for operative treatment are needed and perhaps hemodynamic data of the type presented will be of value in making such a selection.

The hyperdynamic state of the circulation in patients with cirrhosis described by

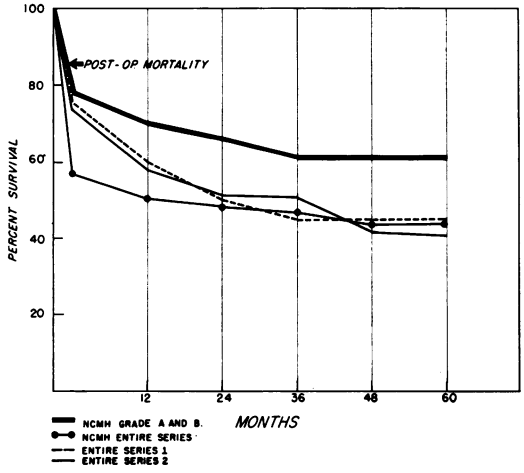

FIG. 8. Plot of mortality statistics of this series compared with those of Rodriquez and Satterfield. The top curve is corrected curve eliminating the grade C patients to more nearly match the degree of illness in this series with that of the other two.

others[5, 17, 18, 20] is confirmed in these comparative studies and—together with enlarged arteriovenous communications in the upper gastrointestinal tract, liver and spleen—can be explained rationally by our hypothesis, which identifies the fundamental circulatory derangement in cirrhosis as increase in flow of blood through shunts in the upper gastrointestinal tract, liver and spleen. An hypothesis which identifies the fundamental derangement as obstruction to flow in the portal vein cannot account for the hyperdynamic state of the circulation or enlargement of the arteriovenous communications.

No operation can restore these vascular abnormalities to normal. However, it seems more rational to use an operation which attempts to reduce flow through arteriovenous communications rather than one which diverts portal vein blood away from an already taxed liver into the vena cava. The operation reported here is at least as effective as are other operations in the control of hemorrhage and in preservation and prolongation of life. It is superior in the avoidance of the crippling cerebral symptoms which often follow portal systemic shunts.

Summary and Conclusion

An increase in cardiac output, blood volume, stroke work, and systolic ejection rate, and a decrease in peripheral resistance are found in patients with cirrhosis of the liver.

These hemodynamic alterations are similar to those found in patients with systemic arteriovenous fistula and suggest the presence of multiple arteriovenous fistulas in patients with cirrhosis of the liver.

Fifty-one patients during a 12-year period have been operated upon to remove as many of these arteriovenous shunts as possible and to decrease the arterial flow into the portal system. An unusual number of poor-risk patients may have attributed to a high operative mortality. Five-year survival, excluding postoperative mortality, was 75 per cent. We recommend this operation for the treatment of esophageal varices.

Acknowledgment

This work is obviously based on the physiologic concepts set forward and given to us by Dr. Nathan A. Womack. Many of the patients were operated on by him. His modesty keeps us from being able to include his name as co-author. The authors are indebted to him for his advice and counsel and the stimulation and support he provided us in the physiologic investigations and in the compilation of the clinical results.

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Discussion

Dr. George Johnson, Jr. (Closing): It is apparent from this data presented by Dr. Peters that better means of selecting patients with cirrhosis and esophageal varices for surgery are needed. The reports of Conn and Garleau, comparing the results of prophylactic portocaval shunts and conservative therapy for esophageal varices, also suggest that we must examine the indications for surgery. Hemodynamic studies such as those presented by Dr. Peters may be of great value in answering these questions. Although the difference in the mean of the various studies performed was highly significant, there was considerable individual variation in the group of patients with cirrhosis. Perhaps those patients with cirrhosis and the hypodynamic cardiovascular syndrome should have an operation of the type presented; others may be selected for conservative therapy. Studies to evaluate this concept are being carried out in our laboratory at the present time.

We have also been impressed by the number of long-term survivors in the clinical series. Eighteen of these patients are still living; seven patients are living after more than 5 years, and two after more than 10 years. The results of this evaluation of a surgical attack, proposed by Dr. Womack more than 10 years ago and aimed at correcting the basic physiological defects in this disease, have encouraged us to recommend the operation to you as a sound surgical approach in the treatment of esophageal varices.