Studies on Portal Hypertension *

IV. The Clinical and Physiopathologic Significance of Self-Established (Nonsurgical) Portal Systemic Venous Shunts

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SPONTANEOUS DIVERSION of portal venous blood into the systemic circulation, bypassing a cirrhotic liver or an extrahepatic obstruction, is frequently observed in patients with portal hypertension. This has been usually accepted as a natural response intended to provide new outlets for the blood confined within an abnormally congested venous system. Alleviation of portal hypertension has been expected by some to follow the resulting shunting of splanchnic blood. Although the circulatory derangement responsible for this diversion of flow has been discussed extensively in the literature, there is still some difference of opinion as to its cause or ultimate effects.[13]

Whatever the nature of the primitive impulse inducing the portal blood to find new avenues of escape may be, the blood reaches the systemic circulation through a variety of pathways. Serial splenoportography has recently provided an invaluable means of delineating in living human beings this, as it is commonly called, collateral circulation of the portal bed. Hepatic blood flow determinations have rendered quantitative although indirect *in vivo* evidence concerning diversion of blood from the liver. A combination of these two methods of investigation together with portal vein and splenic pulp manometrics, in a substantially large group of patients, should result in more complete information of portal hemodynamics than that previously available from clinical observations and postmortem anatomic studies. Among the latter, however, there have been outstanding contributions,[6] the knowledge of which is essential to the understanding of the physiopathology of portal-systemic communications.

Because valves are absent in the veins of the portal system, reversal in the direction of blood flow is a frequent occurrence in the presence of portal hypertension. Thus, backflow into the mesenteric veins, pancreatic branches of the splenic vein, short gastric vessels, and the coronary vein is often observed in these cases.

While blood flowing in reversed direction into the coronary and short gastric veins can be usually demonstrated in the gastric and esophageal plexus as it gains access to the systemic circulation via the azygos system, the ultimate fate of portal blood backflowing into mesenteric, pancreatic, and other veins of the portal system seems less certain. It has been assumed that some of this blood would eventually reach the systemic circulation through normally patent communications at the level of the hemorrhoidal plexus and retroperitoneal

* Presented before the American Surgical Association, San Francisco, Calif., April 15–17, 1959.

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areas in contact with intrabdominal organs.[4, 6, 8]

If the flow of blood escaping the portal circulation through the gastric and esophageal plexus assumes sufficient importance, an unfortunate side effect may take place. Under these conditions the veins in these plexus dilate, become tortuous, sacculated and thin-walled to a point where, under the proper conditions and stimuli, they may rupture and initiate one of the most severe forms of gastro-intestinal hemorrhage.

In addition to this reversal of flow into already functioning venous components of the portal system, the splanchnic blood may also find new outlets by reopening closed or partially closed embryonic channels. Thus, channels connecting the splenic and left renal veins, patent umbilical and paraumbilical veins occasionally communicating with the deep inferior epigastric vessels, retroperitoneal, paravertebral, parietal and other not easily identifiable vessels have been demonstrated. Sometimes, these channels may acquire unusual size and become very obvious shunting pathways between the portal and systemic venous circulations. In these instances they may resemble in magnitude, and perhaps in volume of flow, surgically constructed portal-systemic venous anastomoses.

It is the purpose of this paper to report on the occurrence, behavior, and effects of the several types of natural portal-systemic shunting mechanisms as observed in a series of two hundred and three consecutive patients. Similar observations were repeated in 48 patients of the series after a portacaval anastomosis had been surgically constructed and are also herein presented. This report constitutes another phase in the comprehensive study of the normal and pathologic physiology of the portal circulation being conducted in this clinic.[10-12]

Material and Methods

Two hundred and three patients observed during the period extending from June 1, 1956 to March 31, 1959, constituted the material for this study. One hundred and thirty-five patients had cirrhosis of the liver and 15 had different types of extrahepatic obstruction of the portal system. Among the latter, the underlying pathology included carcinomas of the pancreas, stomach, and gallbladder, compression by benign pseudocyst of the pancreas, congenital obstructions of the portal tree, primary thrombosis in otherwise normal portal systems and thrombosis of the hepatic veins. Secondary bland thrombosis in veins of the portal system occurred in 13 cases of cirrhosis of the liver, and in one patient with cirrhosis and associated liver cell carcinoma. Fifty-three patients in whom no evidence of pathology in the portal system was demonstrated were used as controls. Twenty-one normal subjects not included among the 203 patients forming the basis of this report, were used as additional controls in the determination of hepatic blood flows.

Pressure in the portal system was determined in all the patients included in this report. Pressures were recorded in the portal vein during operation or in the splenic pulp in intact subjects. Whenever possible, both types of measurements were secured. Correlation between pressure in the portal vein and pressure in the splenic pulp was established and has been reported in a previous paper.[14] When the pressures at both sites were not identical, biases for the lowest and highest values were statistically analyzed. Since no significant differences between the two sets of figures was demonstrated, a bias for the highest recordings was arbitrarily adopted, and this was applied both to the control group and to the group of patients with varying degrees of pathologic involvement of the portal system.

The anterior surface of the bodies of the first lumbar vertebrae was used as a base line for correction of pressure readings determined during operation.[10, 16] When the

pressure was measured in the splenic pulp the reading was corrected to the level of a line situated 12 centimeters above the plane of the table on which the patient was lying supine. This correction line was the mean distance between the previously mentioned anterior surface of the upper lumbar vertebrae and the plane of the table as determined in 52 cadavers studied for this purpose.[14, 15]

Wedge hepatic vein pressures were determined less frequently in this series.

Multiple exposure serial splenoportograms were taken in every case and occasionally repeated at six month and one year intervals. As a rule 13 films were obtained in a one minute period during and following the rapid intrasplenic injection of 40 milliliters of 70 per cent Urokon (brand of Sodium Acetrizoate).[15] When portal portograms were taken at the time of operation, the dye was injected in the superior mesenteric vein through a catheter threaded into one of its jejunal branches.

Hepatic blood flow determinations were obtained in 89 instances in 71 patients. Colloidal aggregates of heat-denatured human serum albumin tagged with I^{131} was the material used for these determinations. The liver's specificity of uptake for this albumin complex has been reported in the literature [1-3] and tested in the surgical laboratories of this clinic.[7] The rate of disappearance from the circulation of an intravenously injected dose of the material was plotted on semilogarithmic paper and the slope of the curve was established by the proper equation. To minimize the sources of error the results were not expressed in milliliters per minute but in terms of the kinetic rate of clearance (K_c). Thus, an exact measurement of the fraction of the total circulating blood volume cleared by the liver per minute was obtained and used as a comparative value. Complete details of this technic have been described in a previous paper in this series.[12]

Liver biopsy specimens were obtained either at the time of surgery or by means of the needle biopsy technic prior to operation.

Routine clinical, laboratory and radiological studies, and less frequently esophagoscopic studies, were performed in the customary manner.

The extent of backflow into different veins of the portal system was graded in somewhat arbitrary categories and some of this material has been previously reported.[11] The presence of other entirely different pathways, believed to be for the most part reopened embryonic venous channels, was studied in the course of the present investigation and separately recorded.

The same type of studies was subsequently repeated in 48 patients in the series after surgical portacaval shunts had been performed and the results compared with the other groups of patients in this series.

Results

In this series diversion of portal blood from the liver occurred both by backflow into normally patent veins of the portal system and by circulation through reopened embryonic channels. The latter, in general, apparently provided a larger and occasionally more direct connection with the systemic circulation.

Diversion by reversed circulation in veins of the portal system (Fig. 1) was present in 97 per cent of the patients in whom elevated portal tensions were recorded. Bypassing through large connecting channels, believed to be for the most part reopened, closed, or partially closed, embryonic veins (Fig. 2), was observed in 18.6 per cent of patients of the same type.

In addition to these naturally established shunting mechanisms, practically total diversion of portal blood was accepted as existing in the group of patients with surgically established portacaval anastomoses.

PORTAL DIVERSION BY BACKFLOW INTO NORMALLY PATENT BRANCHES OF THE PORTAL SYSTEM

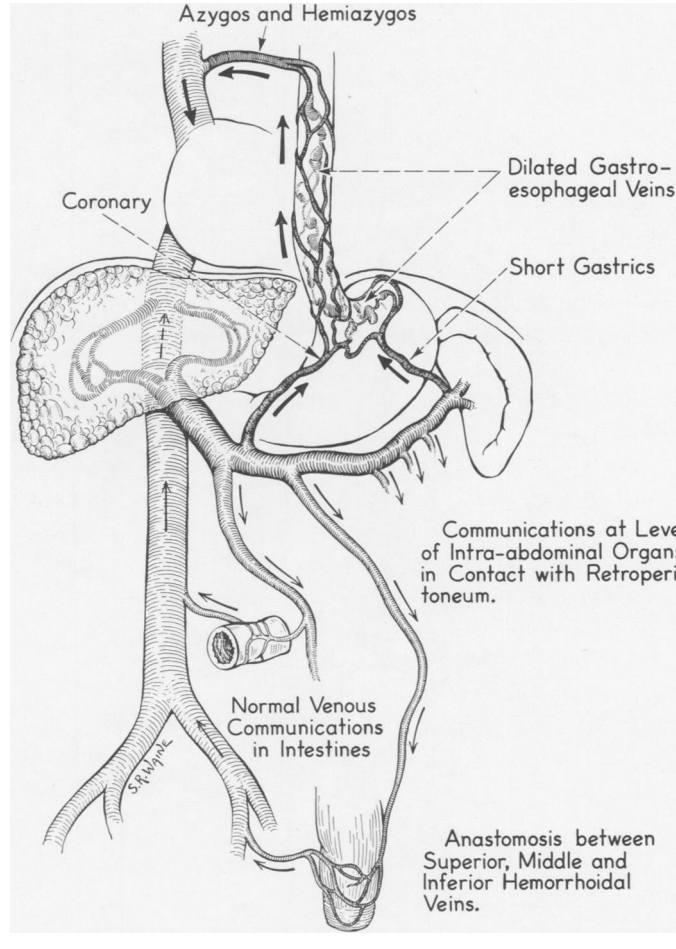

FIG. 1. Diagrammatic presentation of the main routes of portal blood diversion through normally patent branches of the portal system.

Diversion of portal blood was not observed in any of the 53 patients in the control group, nor was it present in cirrhotic patients in whom portal pressure remained within normal limits.

A. Diversion of Portal Blood by Reversed (Hepatofugal) Circulation into Normally Patent Veins of the Portal System

Reversed circulation in portal branches diverted blood from the liver with different degrees of efficiency and into different segments of the systemic circulation. Hepatofugal circulation in mesenteric, pancreatic, and smaller branches of the portal tree carried the blood into the inferior vena cava probably through normally existing communications at the level of the hemorrhoidal plexus and of retroperitoneal areas in contact with intra-abdominal organs (Fig. 1). On the other hand, hepatofugal circulation in gastric and esophageal plexus emptied portal blood into the superior vena cava via the azygos system. The gastric and

PORTAL DIVERSION THROUGH LARGE CONNECTING CHANNELS BETWEEN THE PORTAL AND SYSTEMIC CIRCULATIONS

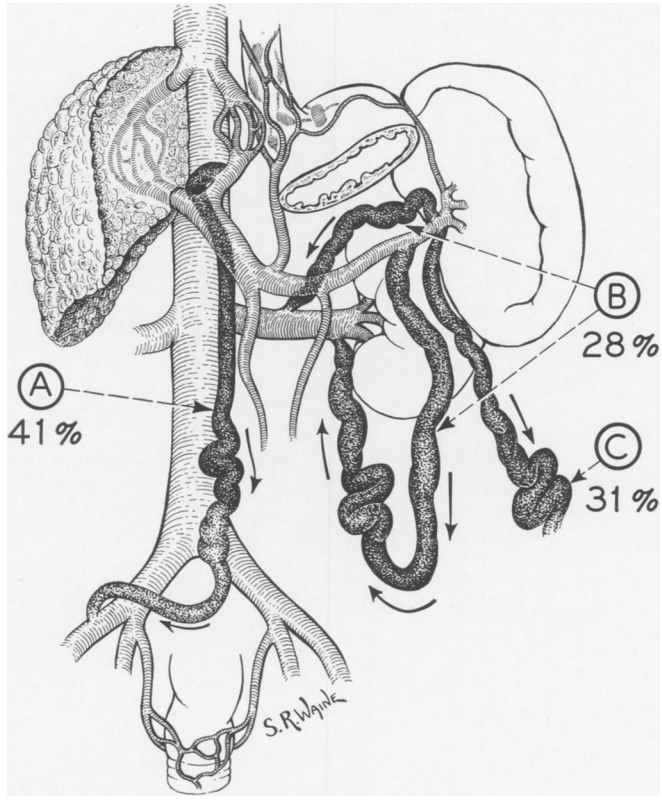

FIG. 2. Diagrammatic presentation of the main types of large channels connecting the portal and systemic circulations. A. Connections between the left intrahepatic branch of the portal vein and the iliac veins. These connections were usually mediated through the paraumbilical and deep epigastric veins. B. Channels emptying into the left renal vein. C. Channels in which the final site of communication with the systemic circulation could not be determined.

esophageal plexus were filled, for the most part, by backflow of blood into the coronary and short gastric veins.

Backflow of blood into one or more veins of the portal system was demonstrated in varying proportions in 97 per cent of the patients in this series who had an elevated portal pressure. The extent of this collateral or "supplementary" circulation was in direct linear correlation with the degree of hypertension in the portal system ($r = .99$) and this correlation was statistically significant ($P < .001$) (Table 1 and Fig. 3, 4).

Backflow of blood into veins of the portal system was not observed in any of the 53 patients in the control group.

1. Hepatofugal Circulation into Normally Patent Veins of the Portal System Emptying into the Inferior Vena Cava: Reversed circulation into the mesenteric, pancreatic, and smaller and less easily identifiable branches of the portal system, shunted splanchnic blood into the inferior vena cava presumably through normally existing venous communications. The effect of this diversion was not reflected, however, in lowered values in portal pressure. On the contrary, increasing collateralization through

TABLE 1. *Pressure* in the Portal System in 203 Patients*

Group 1		Group 2		Group 3		Group 4		Group 5		Group 6		Post-Shunt	
Case No.	Press.	Case No.	Press.	Case No.	Press.	Case No.	Press.	Case No.	Press.	Case No.	Press.	Case No.	Press.
10	225	1	220	5	300	16	296	46	303	7	345	28	265
12	70	9	360	6	246	35	440	64	410	8	465	35	90
18	152	19	203	17	90	36	283	84	415	11	527	42	200
32	183	21	215	20	283	39	277	99	510	28	485	44	247
33	85	25	170	26	350	41	245	107	403	42	410	47	220
34	75	29	247	38	179	66	340	122	468	44	400	49	230
37	148	40	228	62	343	145	380	127	380	47	433	56	200
43	120	50	385	75	280	165	427	137	281	49	540	58	250
45	90	51	131	87	226	231	305	141	375	55	370	61	210
48	228	67	434	92	230			150	265	56	440	64	110
54	168	72	193	93	325			166	322	57	540	70	205
60	120	88	258	95	364			169	392	58	530	89	220
63	150	101	171	129	276			171	310	61	355	92	230
65	143	102	385	173	265			183	303	68	340	96	290
78	275	106	230	180	162			189	210	69	375	98	190
80	150	108	150	217	288			205	227	70	462	99	380
81	210	124	153					208	338	71	530	104	270
82	185	133	205					219	470	79	460	105	280
83	245	152	238					222	414	89	285	110	170
85	273	179	241					228	295	92	440	116	295
86	275	184	215					236	288	96	326	122	330
91	144	190	265					242	310	97	350	125	200
109	276	191	153							98	420	132	190
113	123	194	210							100	445	144	130
114	203	195	217							103	415	154	250
115	225	211	195							104	480	159	210
119	105	213	160							105	441	162	250
120	210	214	239							110	293	170	370
121	113	218	230							112	470	172	85
123	186									116	650	174	190
128	90									117	360	175	130
130	240									125	465	176	210
131	215									126	570	181	270
134	196									132	390	185	220
136	93									144	480	186	170
139	155									146	390	189	170
140	150									147	345	193	210
143	276									148	396	198	335
151	175									149	383	201	305
156	326									154	480	203	180
161	73									155	351	206	230
163	140									158	282	207	200
167	70									159	510	209	185
177	160									160	356	210	207
192	121									162	455	212	200
196	77									164	500	226	182
220	290									170	465	227	136
230	97									172	318	237	328
233	135									174	440		
235	80									175	460		
238	105									176	600		
241	157									181	505		
243	195									185	580		
										186	480		
										188	470		
										193	363		
										197	257		
										198	445		
										201	415		
										202	245		
										203	423		
										204	380		
										206	473		
										207	340		
										210	388		
										212	460		
										224	430		
										225	400		
										226	440		
										227	356		
										232	490		
										237	410		
										239	377		
										240	340		
Mean:	165.49		231.07		262.94		332.56		349.50		425.47		221.35
Standard Error:	9.14		13.54		17.765		21.68		16.60		9.26		9.43
Standard Deviation:	66.56		72.92		71.06		65.03		78.06		79.63		65.33

Group 1: Normal controls
Group 2: Cirrhosis without collateralization
Group 3: Mild collateralization
Group 4: Severe collateralization without varices
Group 5: Collateralization with non-bleeding varices
Group 6: Collateralization with bleeding varices
Postshunt: Portal pressure after portacaval anastomosis

* In millimeters of water (saline).

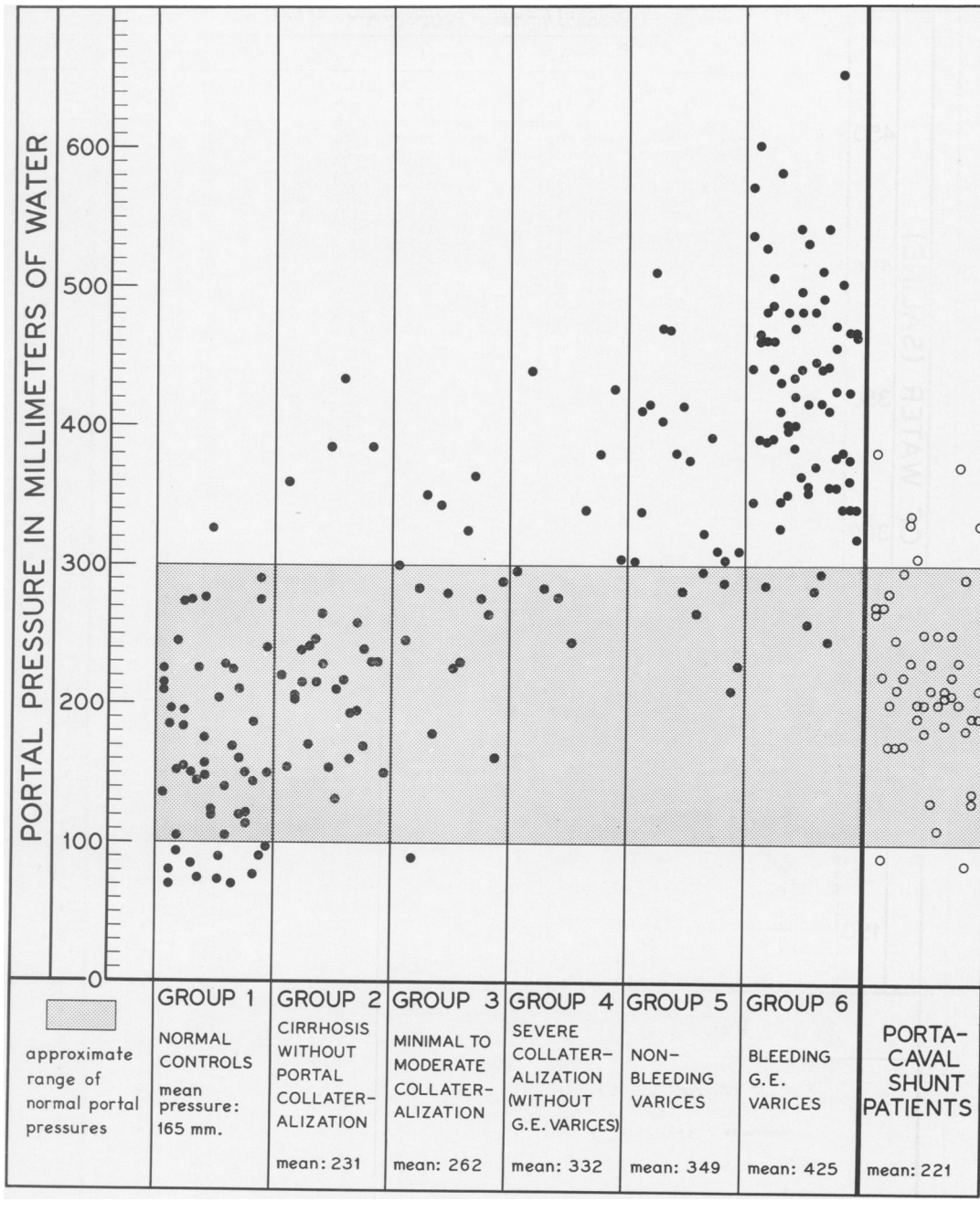

FIG. 3. Graph showing individual pressure recordings for normal controls, patients with increasing collateralization of the portal system, and patients submitted to a portal systemic surgical anastomosis (203 patients).

these veins was associated with progressively higher values in portal pressure (Groups 3 and 4 in Table 1, and Fig. 3, 4).

The rate of hepatic blood flow in patients in these groups remained essentially normal (Groups 3 and 4 in Table 2 and Fig. 5). Or

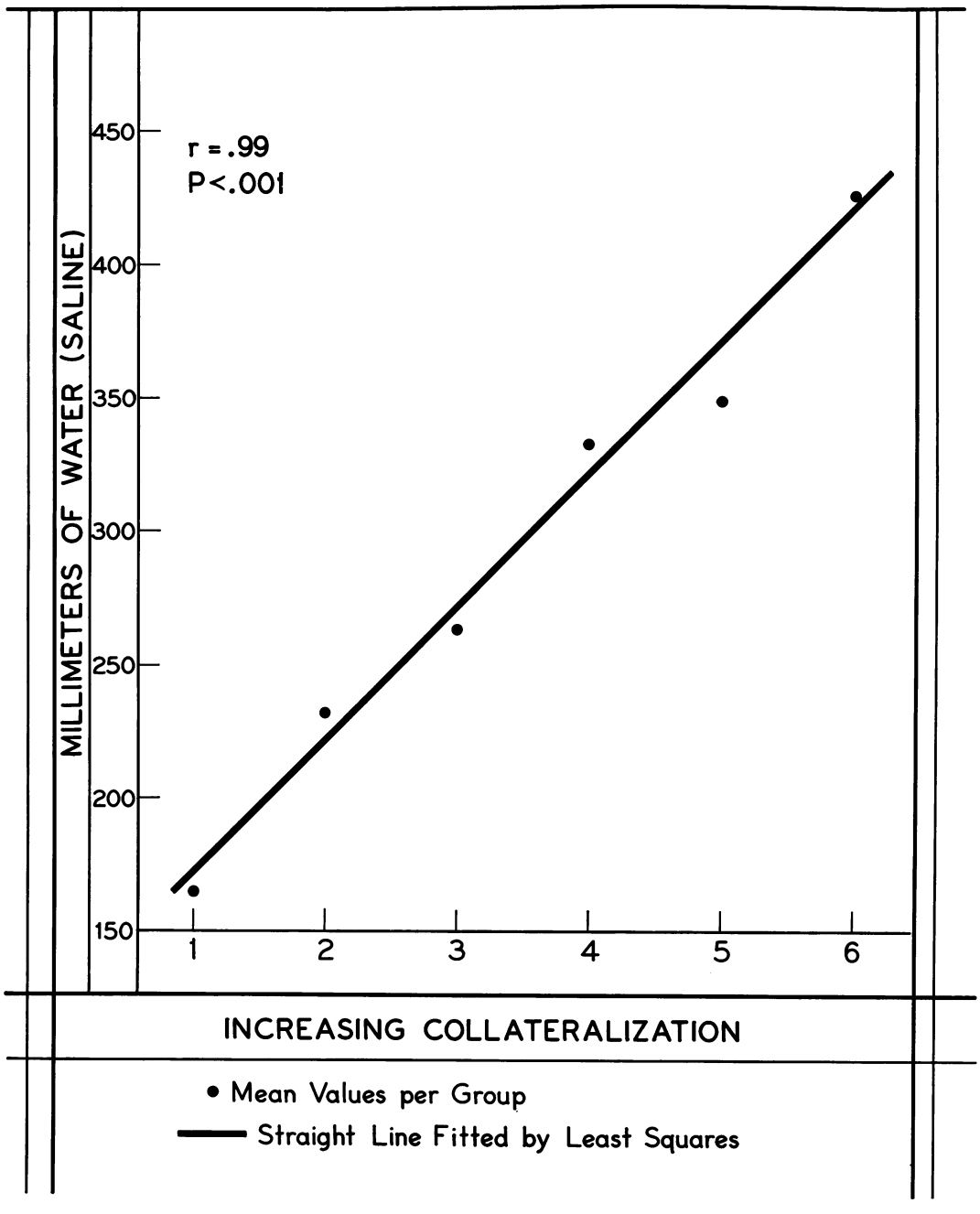

FIG. 4. Graph showing correlation between increasing collateralization of the portal system and the mean values of portal pressure. Groups are the same as in Figure 3.

at least, hepatic blood flow values were not significantly different from those obtained in the control group (Group 1 in Table 2 and Fig. 5) or in the group of cases with mild cirrhosis in which portal backflow was absent and portal pressure remained within normal limits (Group 2 in Table 2 and Fig. 5).

TABLE 2. *Rate of Hepatic Blood Flow in 71 Patients**

Group 1		Group 2		Group 3		Group 4		Group 5		Group 6		Post-Shunt	
Case No.	K_e	Case No.	K_e	Case No.	K_e	Case No.	K_e	Case No.	K_e	Case No.	K_e	Case No.	K_e
91	.29	19	.24	20	.37	16	.2)	53	.265	11	.26	44	.13
131	.26	21	.29	38	.26	36	.35	137	.23	98	.23	49	.27
136	.32	133	.36	129	.33	145	.43	141	.24	100	.19	55	.15
143	.34					165	.44	150	.45	110	.16	61	.23
C	.31							169	.17	116	.33	89	.14
C	.47							171	.26	125	.24	98	.18
C	.26									126	.25	125	.21
C	.23									132	.30	142	.16
C	.28									144	.20	154	.34
C	.26									148	.19	159	.16
C	.33									149	.12	168	.17
C	.46									154	.24	170	.26
C	.27									155	.22		
C	.21									158	.064		
C	.42									159	.26		
C	.34									170	.26		
C	.31									172	.23		
C	.40									174	.25		
C	.41												
C	.25												
C	.32												
C	.35												
C	.44												
C	.24												
C	.24												
Mean:	.3204		.2900		.3200		.3775		.2692		.2218		.2000
Standard Error:	.014788		.03299		.02625		.0307		.03534		.01432		.01760
Standard Deviation:	.07394		.05715		.04546		.0614		.08657		.06079		.06096

* In 18 patients values are mean values of two or more determinations.
Note: Groups are the same as in Table 1.

A few individual cases departed from the general pattern.

2. Hepatofugal Circulation in Veins of the Portal System Emptying into the Superior Vena Cava: Portal blood reaching the superior vena cava was carried for the most part by way of the gastroesophageal plexus which in turn emptied into the azygos system. Dilated veins in these plexus (varices) were usually filled by reversed flow of portal blood in the coronary and short gastric vessels.

In contrast with the preceding group of patients, diversion of portal blood through these channels was associated with a sharp decrease in hepatic blood flow values (Groups 5 and 6 in Table 2 and Fig. 5). This diversion did not result, however, in any alleviation of portal hypertension. On the contrary, the highest values of portal pressure in the entire series were observed in the group of patients with esophagogastric varices (Groups 5 and 6 in Table 1 and Fig. 3, 4).

The marked decrease in hepatic blood flow noted in these patients was statistically significant ($P < .001$) (Table 2 and Fig. 5). Actually, hepatic blood flow in patients with esophagogastric varices was reduced to an extent almost comparable with that

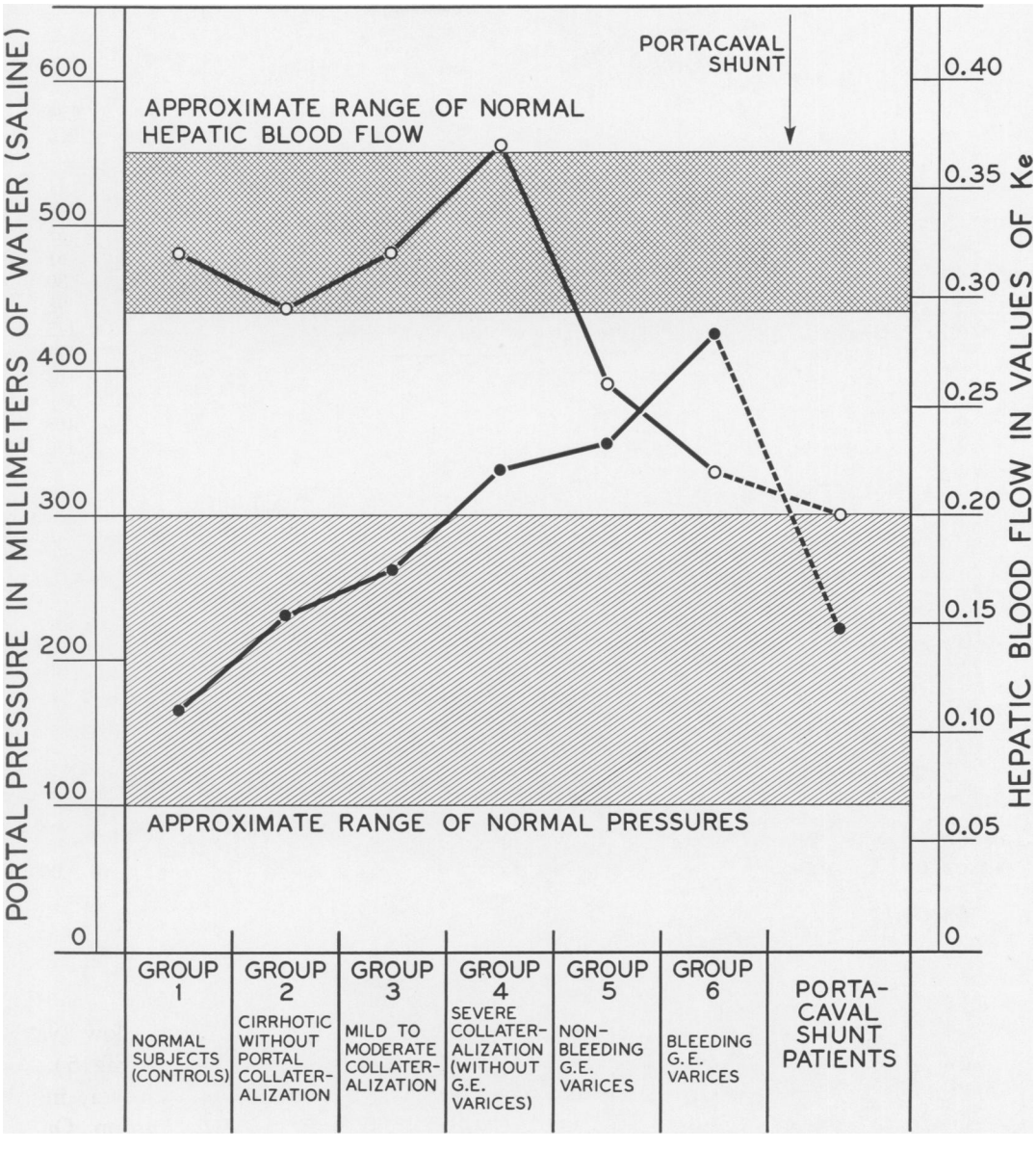

FIG. 5. Graph showing a comparison between mean values of portal pressure and hepatic blood flow in relation to the degree of collateralization of the portal system and to the effect of portacaval anastomosis (203 patients).

produced by well-functioning, surgically-constructed portal systemic venous anastomosis (Table 2 and Fig. 5).

Again, a few individual cases departed from this pattern without affecting the general trend in the series. Two of these cases may merit separate description, since they presented particularly unusual hemodynamic situations. In them (E. V., Chart No. 80243; W. V., Chart No. 85547) (Fig. 6, 7), huge dilatation of the coronary and gastroesophageal veins was observed to coexist with normal values in portal pressure. The size of the coronary vein in

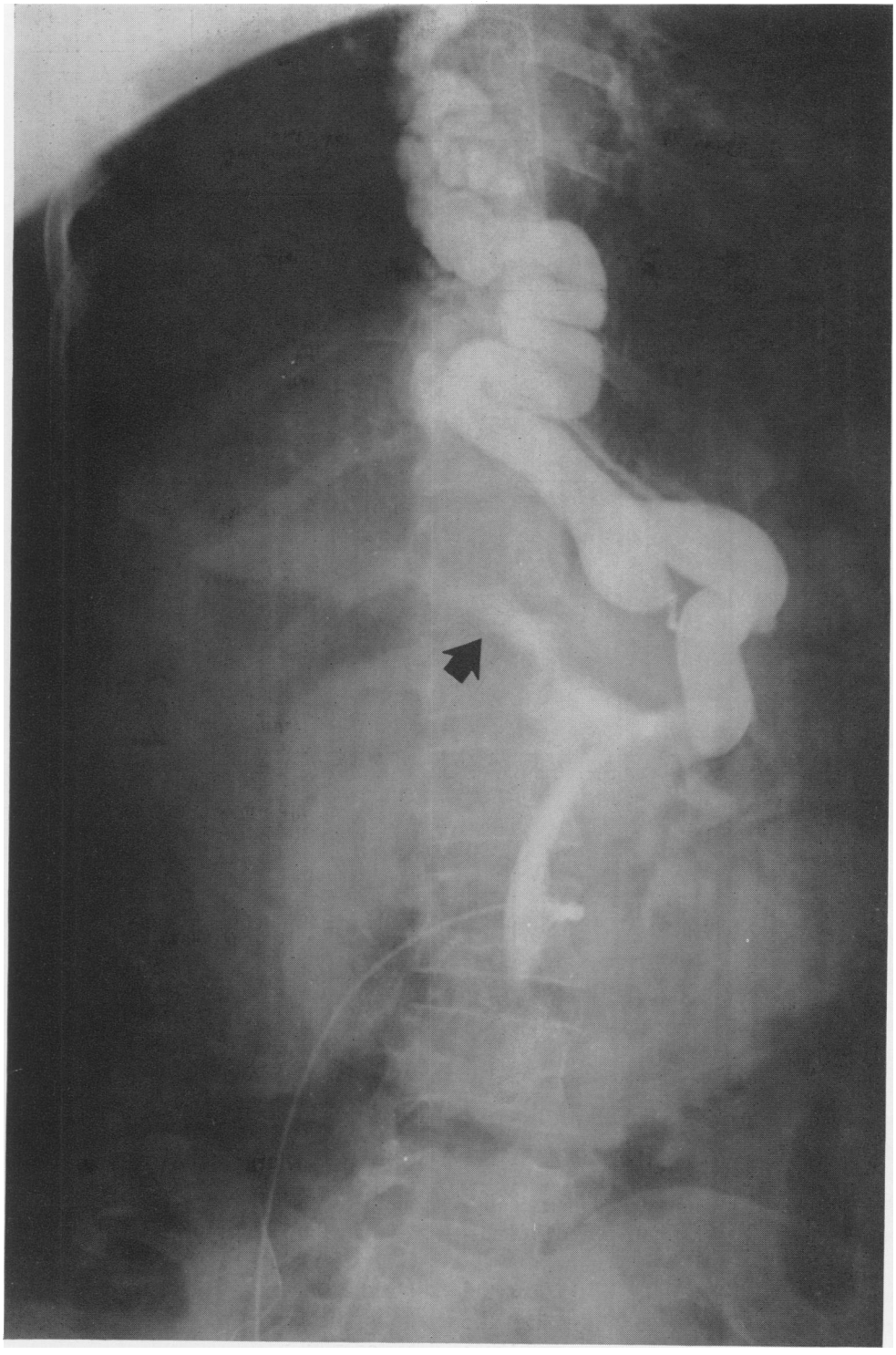

FIG. 6. Operative portal portogram of patient E. V. (Chart No. 80243), showing huge dilatation of the coronary and gastroesophageal veins. A small amount of portal blood reached the liver through the portal vein (arrow) although this was found to be anatomically patent and of normal caliber.

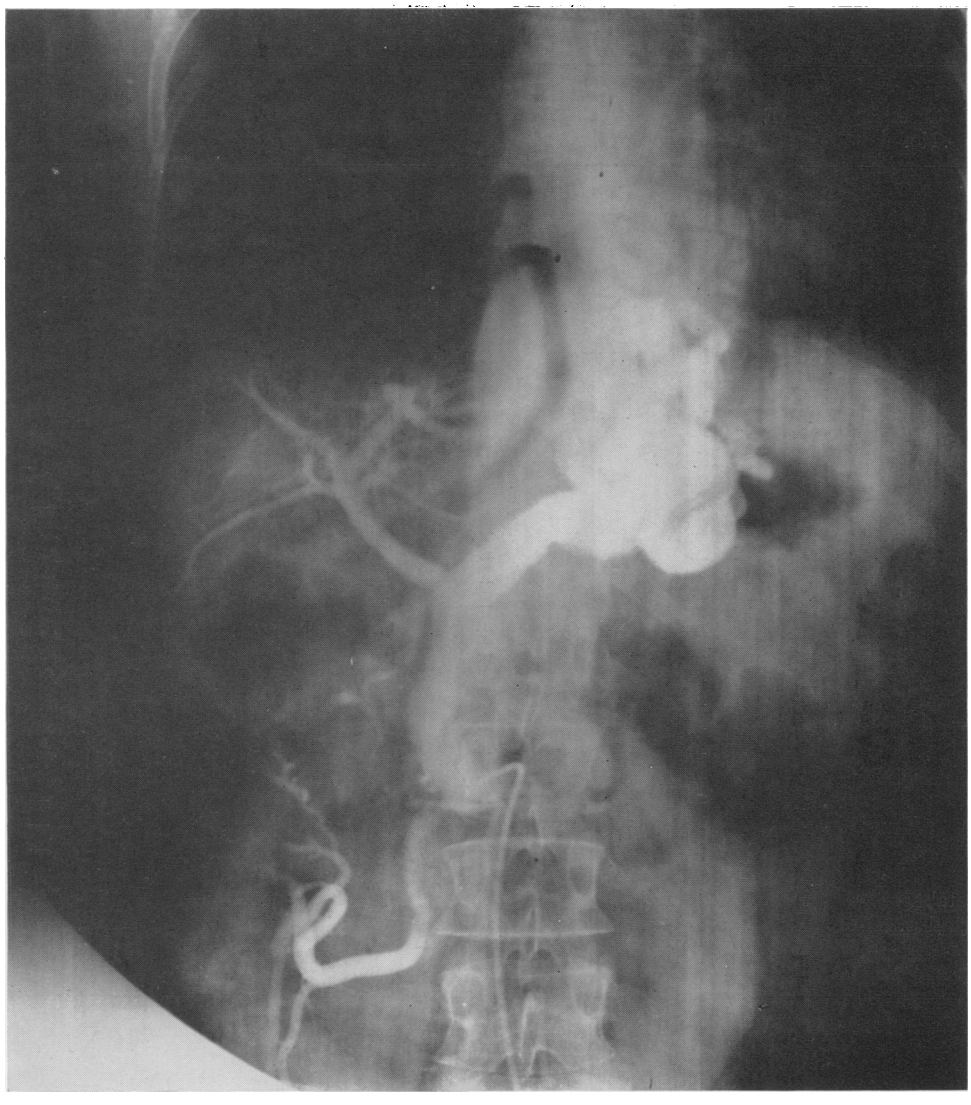

FIG. 7. Operative portal portogram of patient W. V. (Chart No. 85547), showing extreme diversion of portal blood through the coronary and gastroesophageal veins. The portal vein, of reduced diameter, apparently carries a considerably smaller volume of splanchnic blood than the gastroesophageal vessels.

both cases exceeded that of the portal vein, and a very small proportion of the splanchnic blood circulated through the portal vein and reached the liver. For all practical purposes the coronary vein acted as the end opening of the portal system, while the portal vein itself assumed the role of a side opening.[5] Contrary to what was observed in the majority of patients in this same group, esophageal varices in these two cases apparently created hemodynamic conditions capable of providing effective decompression of the portal system. The efficacy of this decompression was judged both in terms of the amount of blood diverted from the liver and also of the degree to which hypertension in the portal bed was reduced.

TABLE 3. *Patients with Large, Re-opened, Embryonic Channels Connecting the Portal and Systemic Venous Circulations*

Case No.	Demonstration of Esophagogastric Varices	Visualization of Inferior Vena Cava	Pressure* in the Portal System	Hepatic Blood Flow in Values of K_e
16	−	−	296	.298
35	−	+	440	—
39	−	−	277	—
42	+	+	410	—
58	+	−	530	—
59	+	−	NT	—
61	+	+	355	—
84	+	−	415	—
89	+	−	285	—
92	+	−	440	—
96	+	+	326	—
100	+	+	445	.190
116	+	+	650	.328
122	+	−	468	—
126	+	−	570	.258
141	+	−	375	.240
144	+	−	480	.200
145	−	−	380	.430
149	+	+	383	.120
154	+	−	480	.240
155	+	+	351	.220
158	+	−	282	.064
166	+	−	322	—
169	+	−	392	.170
172	+	+	318	.230
205	+	−	227	—
224	+	−	430	—
236	+	−	288	—
Total number of patients with communicating channels—28	Incidence of varices—85.714%	Incidence of vena cava visualization—32.14%	Mean pressure—393.14	Mean value of K_e—.2298

* In millimeters of water (saline).

B. Diversion of Portal Blood by Means of Large Embryonic Channels Connecting the Portal and Systemic Venous Circulations

In addition to reversal of flow into branches of the portal system, large communicating channels between the portal and systemic venous circulations were demonstrated in 28 patients (Table 3). Some of these channels (28%) delivered portal blood into the left renal vein (Fig. 8). Others (41%), believed to be umbilical or paraumbilical channels, originated in the left intrahepatic branch of the portal vein and joined occasionally the deep epigastric and iliac veins to finally reach the inferior vena cava (Fig. 9). In 31 per cent of the cases the final site of communication with the systemic circulation could not be determined. In nine instances the flow of blood through these channels was of sufficient magnitude to produce radiologic visualization of the inferior vena cava following the intrasplenic injection of the opaque medium.

Hepatic blood flow was markedly reduced in patients in this group, the mean

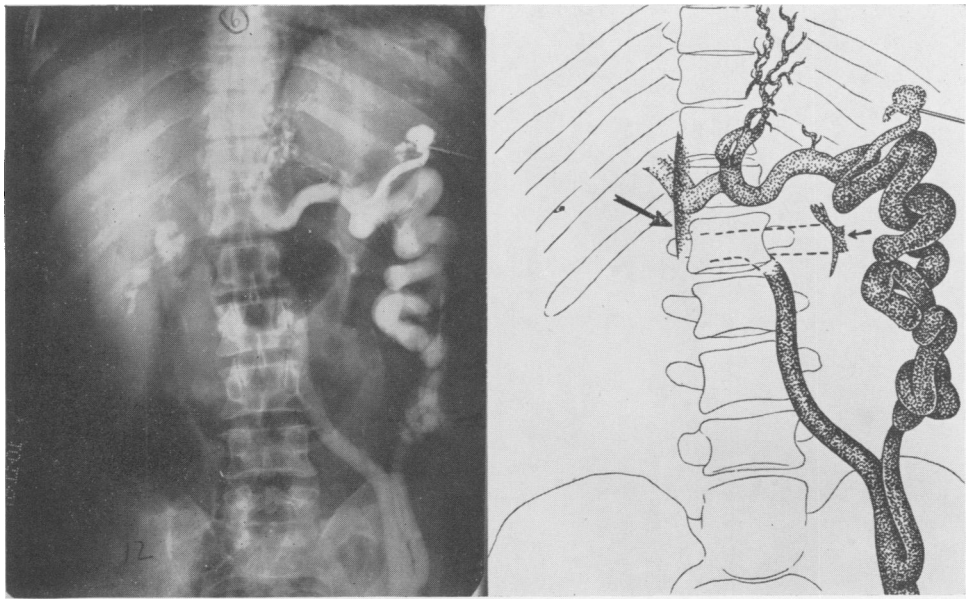

FIG. 8A.

value of K_e being .22 (Table 3). The difference in flow between this group and the control group was statistically significant ($P < .001$).

This active diversion of blood from the portal system was not generally reflected by a reduction in portal hypertension or, at least, in the majority of patients portal pressure remained within the range of clearly abnormal values. Mean value of portal pressure for this group was 393 millimeters of water (Table 3).

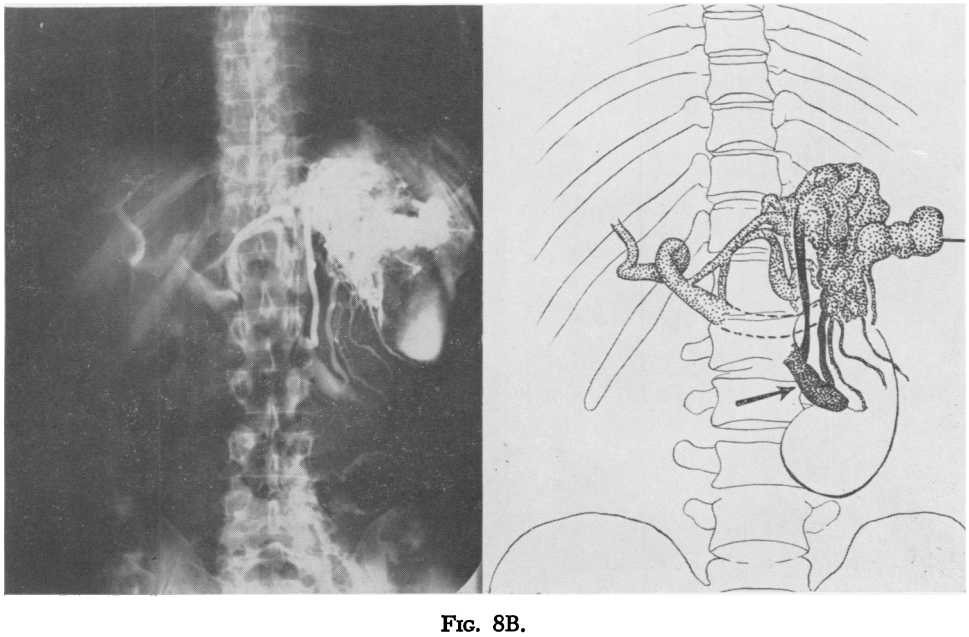

FIG. 8B.

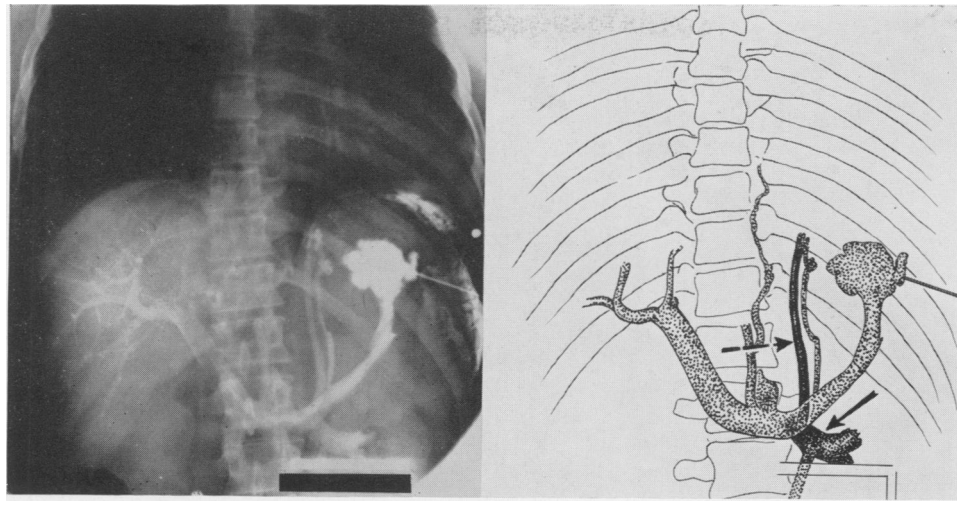

FIG. 8C.

These naturally established shunts did not prevent circulation of portal blood through esophagogastric varices which were demonstrated in 85.7 per cent of the patients in this group (Table 3), nor did they circumvent severe hemorrhage from ruptured varices. Incidence of bleeding in patients with esophagogastric varices and large natural channels was 70.8 per cent while corresponding incidence in patients with esophagogastric varices not associated with these diverting channels was 79.7 per cent (Table 4). The difference in incidence of bleeding from ruptured varices was not significant. Severity of bleeding did not appear to be affected by the presence of large embryonic shunts.

Comparison of the values of portal pressure in patients in this group having esophagogastric varices (mean pressure = 400.9 mm. of water) with the values in portal pressure of the patients with esophagogas-

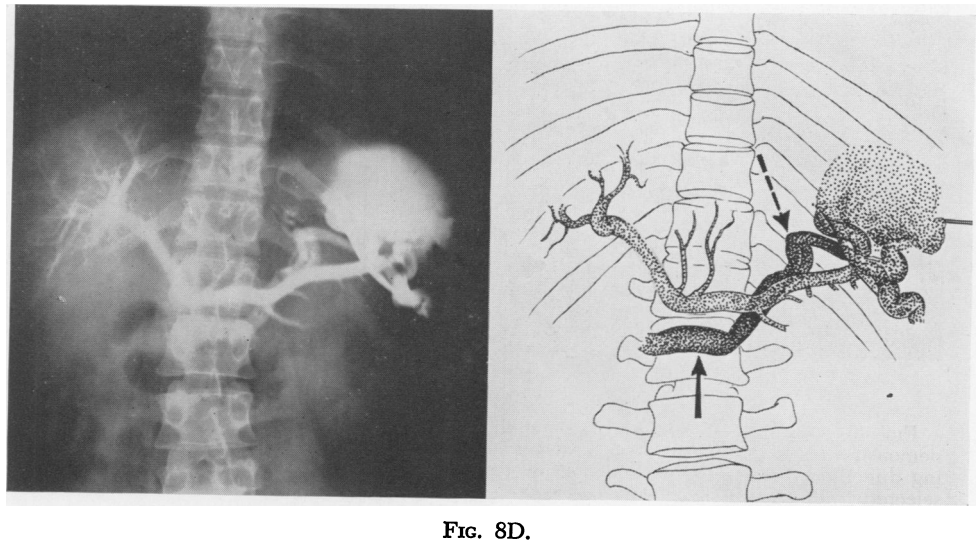

FIG. 8D.

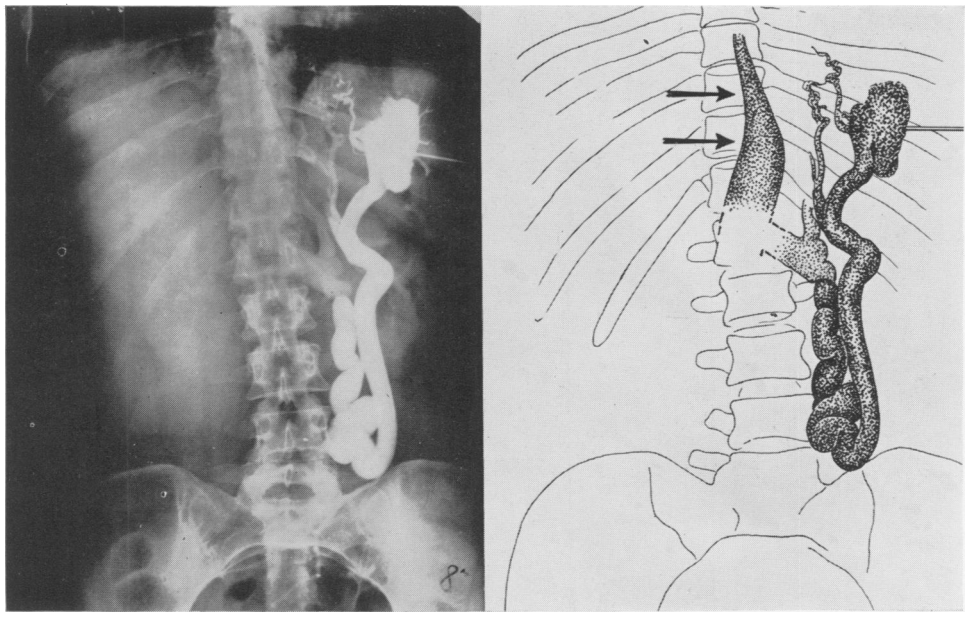

FIG. 8E.

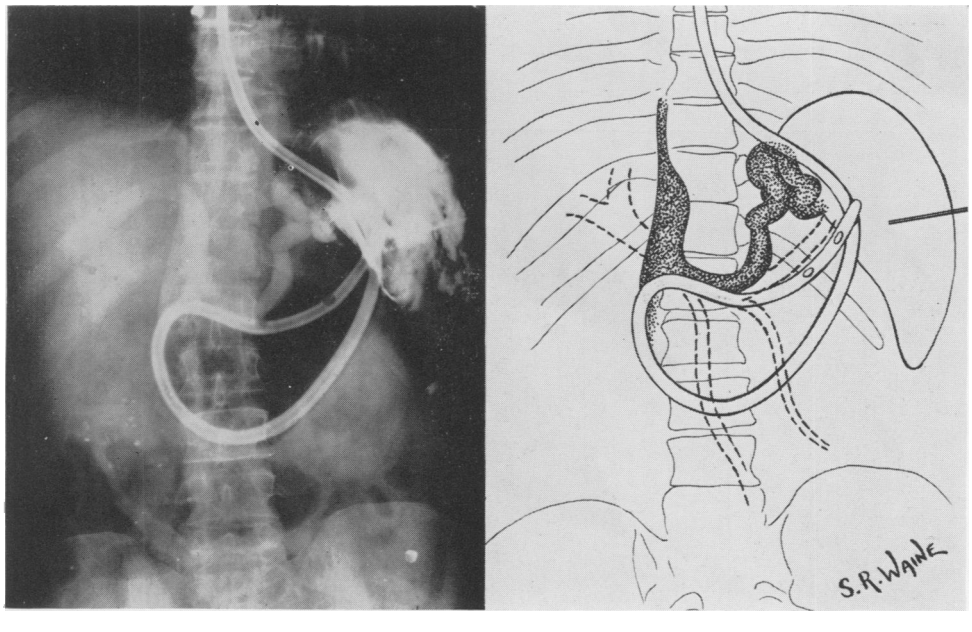

FIG. 8F.

FIG. 8. A group of splenic portograms with accompanying diagrammatic interpretations demonstrating large embryonic channels arising from the portal venous circulation and emptying directly into the systemic circulation via the left renal vein. These are representative films selected from percutaneous serial multiple-exposure splenoportograms of six different patients.

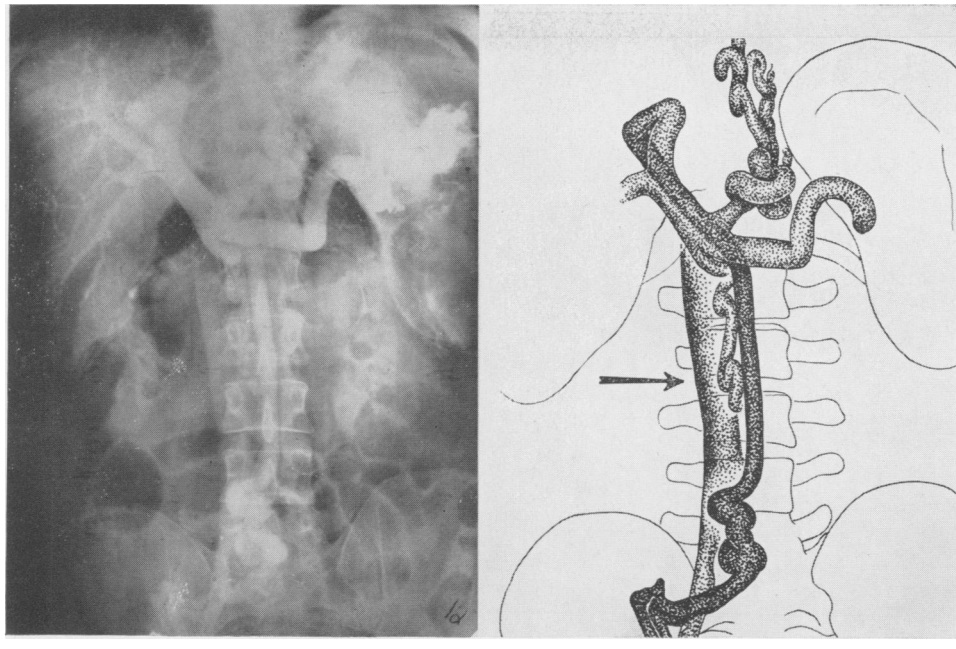

FIG. 9A.

tric varices who did not have these natural channels (mean pressure = 408.8 mm. of water) showed that the difference was not significant (Table 5).

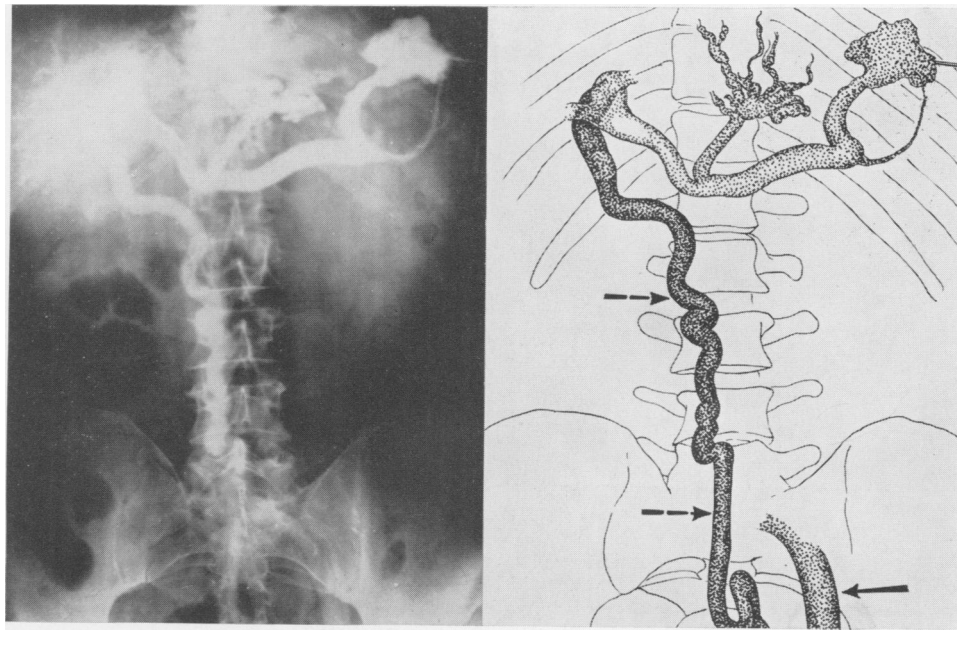

FIG. 9B.

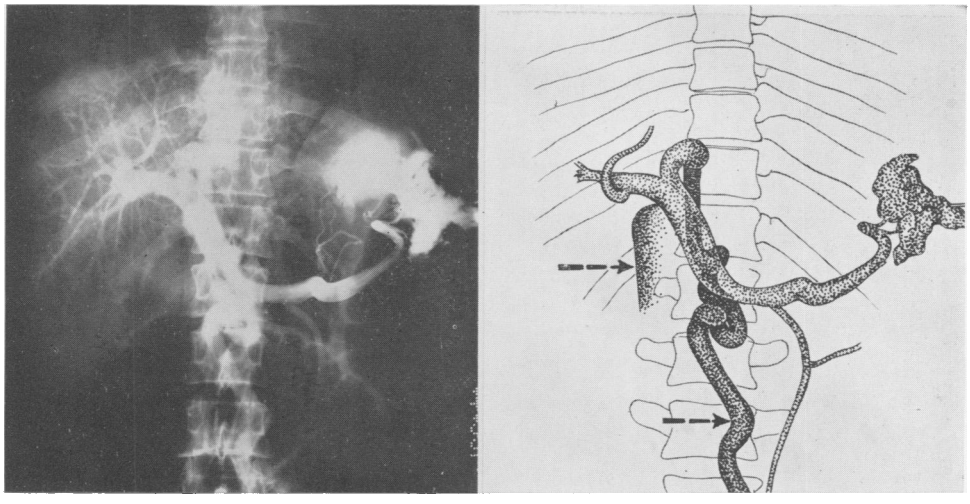

FIG. 9C.

C. Diversion of Portal Blood by Surgically Constructed Portal to Systemic Venous Anastomosis

Surgical diversion of portal blood into the systemic circulation was established in forty-eight patients in this series. Direct end-to-side portacaval anastomosis was performed in 45 patients. In three cases, a splenorenal shunt was constructed as an alternative choice. The portal vein was thrombosed in two of these cases, and in the third the use of the portal vein was precluded by an unsuccessful portacaval shunt performed at another hospital.

Hepatic blood flow, already markedly diminished in this group of patients with esophageal varices, showed some further decrease (2.1%) following portacaval anastomosis (Table 2 and Fig. 5). Mean value of K_e for the postshunt group was $0.20 \pm .06$.

Values in portal pressure after the establishment of a direct end-to-side portacaval shunt decreased sharply (Table 6 and Fig.

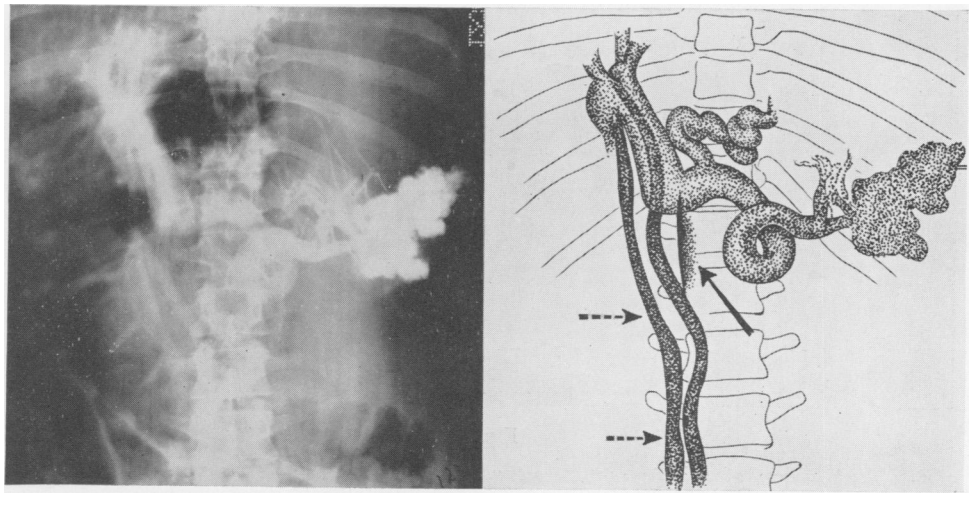

FIG. 9D.

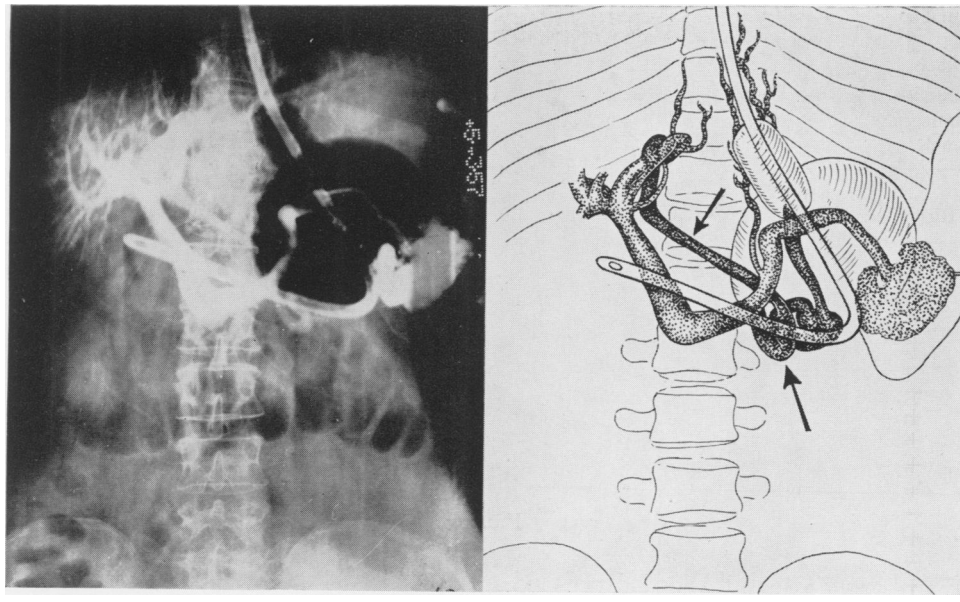

FIG. 9E.

3, 5). Reduction in portal pressure had a mean value of 217 millimeters of water, which rendered a mean value of portal pressure of 219 millimeters of water for the post-portacaval shunt group.

Splenorenal anastomosis, in the small

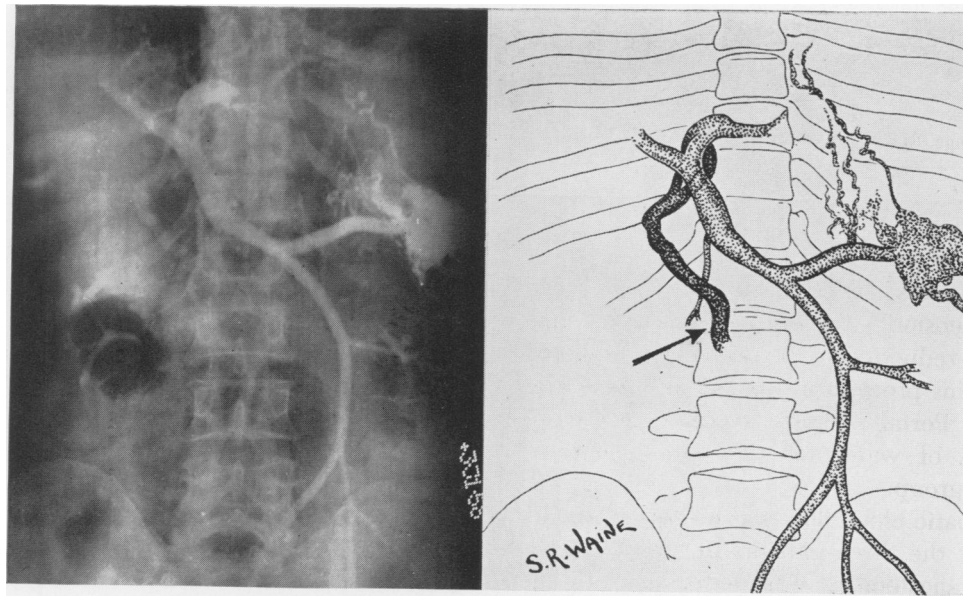

FIG. 9F.

FIG. 9. A group of splenic portograms with accompanying diagrammatic interpretations demonstrating large embryonic channels arising from the left intrahepatic branch of the portal vein. In A and B, these channels enter iliac veins and the vena cava via paraumbilical and deep epigastric veins (channel of Burrow). These are representative films selected, as are those in Figure 8, from serial multiple-exposure splenoportograms of six different patients.

TABLE 4. *Incidence of Bleeding from Ruptured Esophagogastric Varices in Relation to Its Association with Large Natural Shunts Connecting the Portal and Systemic Circulations*

Patients *with* Large Connecting Channels		Patients *without* Large Connecting Channels					
Case No.	Bleeding	Case No.	Bleeding	Case No.	Bleeding	Case No.	Bleeding
42	+	7	+	112	+	189	−
58	+	8	+	117	+	193	+
59	+	11	+	118	+	197	+
61	+	28	+	125	+	198	+
84	−	44	+	127	−	201	+
89	+	46	−	132	+	202	+
92	+	47	+	137	−	203	+
96	+	49	+	146	+	204	+
100	+	55	+	147	+	206	+
116	+	56	+	148	+	207	+
122	−	57	+	150	−	208	−
126	+	64	−	159	+	210	+
141	−	68	+	160	+	212	+
144	+	69	+	162	+	219	−
149	+	70	+	164	+	222	−
154	+	71	+	170	+	225	+
155	+	79	+	171	−	226	+
158	+	97	+	174	+	227	+
166	−	98	+	175	+	228	−
169	−	99	−	176	+	232	+
172	+	103	+	181	+	237	+
205	−	104	+	183	−	239	+
224	+	105	+	185	+	240	+
236	−	107	−	186	+	242	−
		110	+	188	+		
Percentage frequency of bleeding: 70.83%		Percentage frequency of bleeding: 79.73%					

group of patients in which it was used, proved less effective in reducing portal hypertension (Table 7). The mean value of the reduction in portal tension obtained with this procedure was 146 millimeters of water. Portal pressure averaged 242 millimeters of water for the post-splenorenal shunt group.

Hepatic blood flow was measured in only one of the three patients in whom splenorenal anastomosis was performed and the value of K_e was low (0.13).

Circulation of portal blood through gastroesophageal varices or other collaterals was no longer observed in splenoportograms after the establishment of a direct end-to-side portacaval shunt. Obviously, this investigative procedure could not be performed in patients with splenorenal shunts in whom the spleen was removed.

No incidence of bleeding from ruptured varices was observed in this series after direct end-to-side portacaval shunt.

Discussion

It would appear that a successful portal systemic shunt, either natural or surgically constructed, should be one capable of effectively counteracting the adverse effects of portal hypertension. Since, by their very

TABLE 5. *Pressure in the Portal Systems* in Patients with Esophagogastric Varices in Relation to Its Association with Large Natural Channels Connecting the Portal and Systemic Circulation*

Patients *with* Large Connecting Channels		Patients *without* Large Connecting Channels					
Case No.	Pressure	Case No.	Pressure	Case No.	Pressure	Case No.	Pressure
42	410	7	345	112	470	189	210
58	530	8	465	117	350	193	363
61	355	11	527	125	465	197	257
84	415	28	485	127	380	198	445
89	285	44	400	132	390	201	415
92	440	46	303	137	281	202	245
96	326	47	433	146	390	203	423
100	445	49	440	147	345	204	380
116	650	55	370	148	396	206	473
122	468	56	440	150	265	207	340
126	570	57	540	159	510	208	338
141	375	64	410	160	356	210	380
144	480	68	340	162	455	212	460
149	383	69	375	164	500	219	470
154	480	70	462	170	465	222	414
155	351	71	530	171	310	225	400
158	282	79	460	174	440	226	440
166	322	97	350	175	460	227	356
169	392	98	420	176	600	228	295
172	318	99	510	181	505	232	490
205	227	103	415	183	303	237	410
224	430	104	480	185	580	239	377
236	288	105	441	186	480	240	340
		107	403	188	470	242	310
		110	293				
Mean Pressure: 400.95 Standard Error: 20.57 Standard Deviation: 98.67				Mean Pressure: 408.82 Standard Error: 9.29 Standard Deviation: 79.39			

* In millimeters of water (saline).

nature, these diverting mechanisms will prevent some or all of the portal blood from perfusing the liver, any benefit to be derived from their effects upon portal hypertension should ideally overcome the negative results of hepatic bypassing.

Although clinical and anatomic studies have outlined most of the communicating pathways between the portal and systemic circulations as they occur under normal and pathologic conditions, information on the corresponding hemodynamic situations seems less precise. From the evidence collected during the present investigation in the human subject, it appears that some understanding of the intricacies of this unique type of circulatory derangement may have been gained. This, however, as with many more problems in this area, remains in need of further clarification.

In the first place, spontaneous diversion of portal blood from the liver, either accomplished by reversed flow into branches of the portal system or by circulation through reopened embryonic channels, did not appear to produce any demonstrable alleviation of portal hypertension. On the contrary, the highest values in portal pressure were recorded in the groups of patients in which collateralization of the por-

TABLE 6. *Portal Pressure Values* Before and After Direct End-to-Side Portacaval Anastomosis in 45 Patients*

Case No.	Preshunt Pressure	Postshunt Pressure	Difference
28	485	265	220
35	440	90	350
42	410	200	210
47	433	220	213
49	540	230	310
56	440	200	240
58	530	250	280
61	355	210	145
64	400	110	290
70	462	205	257
89	285	220	65
92	440	230	210
98	420	190	230
99	510	380	130
104	480	270	210
105	441	280	161
110	293	170	123
116	650	295	355
122	470	330	140
125	465	200	265
132	390	190	200
144	480	130	350
154	480	250	230
159	510	210	300
162	455	250	205
170	465	370	95
172	318	85	233
175	460	130	330
176	600	210	390
181	505	270	235
185	560	220	340
186	480	170	310
189	210	170	40
193	363	210	153
198	445	335	110
210	415	305	110
203	423	180	243
206	473	230	243
207	340	200	140
209	325	185	140
210	388	207	181
212	460	200	260
226	440	182	258
227	356	136	220
237	410	328	82
Mean:	437.77	219.95	217.82
Standard Error:	12.09	9.90	12.47
Standard Deviation:	81.13	66.41	83.66

* In millimeters of water (saline).

TABLE 7. *Portal Pressure Values* Before and After Splenorenal Anastomosis in 3 Patients*

Case No.	Preshunt Pressure	Postshunt Pressure	Difference
44	400	247	153
96	326	290	36
174	440	190	250
Mean:	388	242	146

* In millimeters of water (saline).

tal tree was most marked. Furthermore, incidence of hemorrhage from esophagogastric varices, the most dreaded complication of portal hypertension, was not decreased in the presence of any of these natural shunting mechanisms.

Secondly, it was noted that different types of communications were not equally efficient in diverting portal blood into the systemic circulation, as far as this diversion could be judged in terms of reduced hepatic blood flow and splenoportographic evidence.

As presented in the section of results, backflow into mesenteric, pancreatic, and smaller branches of the portal system was not associated with reduced values of hepatic blood flow or portal pressure. Although diversion through gastroesophageal plexus was found to coexist with markedly decreased rates of hepatic blood flow, the highest values in portal pressure were observed in the majority of these cases. Finally, the presence of large communicating channels between the portal and systemic circulation was also associated with low rates of hepatic blood flow and high values of portal pressure.

The inability of reversed circulation into mesenteric, pancreatic, and smaller branches of the portal system to lower portal pressure or decrease the rate of hepatic blood flow appears to indicate that it was of small value as a portal systemic diverting mechanism. Since the final communication between the two systems was accomplished in this instance through multiple fine vessels, the meager decompressive effect ob-

tained can, perhaps, be explained on the basis of Poiseuille's Law, which states that the volume of flow is proportionate to the fourth power of the diameter of the communicating vessel.[6] However, the implications in this law are of less clear application in explaining the markedly reduced rates of hepatic blood flow associated with the presence of esophagogastric varices. Unless definite proof can be presented that final communications between the esophagogastric plexus and the azygos systems are of a larger size than those in the mesenteric and retroperitoneal territories, the different diverting effect of these two main pathways will remain difficult of interpretation. A fact to be mentioned, however, is that the first route emptied blood into the inferior vena cava while the second reached the superior vena cava. From the evidence collected in this investigation, no further elaboration on this fact of observation seems justified at this time. In any case, if the marked reduction in hepatic blood flow observed in patients with esophagogastric varices is accepted as indicative of active diversion through these gastroesophageal pathways, one of the discriminative tests recently proposed in the differential diagnosis of upper gastro-intestinal bleeding[9] will find additional support.

The failure of the gastroesophageal pathways to decrease portal tension in spite of their apparent ability to divert portal blood from the liver, may have a more acceptable explanation. Pressure in a vascular bed, as has been demonstrated in various circulatory circuits, is dependent not only on volume of flow but also on other variables, one of the most important of which is resistance to flow. Perhaps resistance to flow in the gastroesophageal plexus remained high in the majority of patients comprising this group and was responsible for the inability of these diverting pathways to alleviate hypertension in the portal system. That such resistance may be overcome is apparently demonstrated in the two patients whose portograms are depicted in Figures 6 and 7. In these cases, the huge dilatation of the gastroesophageal plexus appears to have caused almost all of the portal blood to follow these plexus and permitted only a small proportion of it to reach the liver through anatomically patent portal veins. In these two instances, dilatation of the gastroesophageal plexus was so grossly exaggerated that practically all resistance to flow may have been overcome and portal pressure returned to normal values. These exceptional circumstances apparently created hemodynamic conditions capable of rendering effective decompression of the portal system.

In these two patients portal hypertension could be said to have been naturally cured; however, it was a "cure with defect," since a dangerous condition of hugely dilated, thin-walled gastroesophageal varices was created. One of the two patients had repeated severe hemorrhages from such varices, which may have been ruptured under the effect, usually tolerated by normal veins, of sudden elevations in pressure during coughing, choking, vomiting, etc. These elevations have been well demonstrated in normal subjects by other investigators.[17]

The presence of large embryonic channels was not accompanied by alleviation in portal pressure, but, as in the other instances, by highly abnormal pressure recordings. Hepatic blood flow rates were low in this group. However, it was impossible to determine accurately what the effect of these large natural channels alone might have been upon the rate of hepatic blood flow, since in 85.7 per cent of the patients these channels were also associated with diversion from esophagogastric varices. Separate, simultaneous studies of flows into these two pathways will be necessary to answer this question. At any rate, the very coexistence of these natural shunts with esophagogastric varices and with bleeding

from ruptured varices would tend to prove their lack of protective value against the most serious complication of portal hypertension.

Positive findings that followed the surgical establishment of a portal systemic shunt, that is, prevention of circulation of portal blood through esophagogastric varices and effective protection against hemorrhage from ruptured varices, should perhaps be evaluated against the further decrease in hepatic blood flow associated with this operation. It would appear that if the procedure is used in patients with esophagogastric varices, this further reduction (2.1%) in an already diminished hepatic blood flow will be of no great significance. In fact, the very smallness of this decrease may be responsible for the relative infrequency and mildness of ammonia intoxication generally observed in the postoperative course of these patients. Perhaps the same shunt, if performed in patients without esophagogastric varices and with values of hepatic blood flow similar to those of the control group, would be followed by symptoms of greater severity.

Finally, in explanation of the highly efficient decompressive effect of surgically constructed portal systemic shunts, it may be said that direct connection of the natural end opening of the portal system into the vena cava is perhaps the most important single factor to be considered. When a direct end-to-side portacaval anastomosis is completed, the operation utilized almost exclusively in this series, all the splanchnic blood collected in the portal vein is delivered, without interposition of vascular resistances, into a lower pressure system. Maximal decompression is usually obtained under these conditions.

The ideal operation for the patient with portal hypertension would seem to be one which would decrease the value of portal pressure while restoring the hepatic blood flow to normal limits. Such an operation is not available at the present time; until it is, the portacaval shunt at least has accomplished the first of these effects.

Summary and Conclusions

Spontaneous diversion of portal blood, either by reversed flow into branches of the portal system or through reopened embryonic channels, was not associated with alleviation of portal hypertension in this series.

The rate of hepatic blood flow was not changed in the presence of increasing collateralization until esophagogastric varices were demonstrated and only then was it sharply decreased.

Esophagogastric varices were apparently effective in decreasing hepatic blood flow, but diversion of blood through this route did not generally reduce the values of portal tension.

Large embryonic channels connecting the portal and systemic venous circulations were not associated with reductions in portal hypertension. The rate of hepatic blood flow in these cases was significantly decreased. Incidence of bleeding from ruptured varices or severity of the bleeding episodes were not affected by the presence of these large connecting channels.

Surgically constructed shunts produced a further, although not too marked, decrease in the already diminished hepatic blood flow of patients with esophageal varices. Portal pressure was sharply reduced from generally highly abnormal values to the approximate normal range. After a portacaval shunt was surgically established no incidence of backflow into esophagogastric varices or other collaterals was observed. Hemorrhage from ruptured varices did not recur.

If the interpretation of the phenomena observed is correct, the following tentative conclusions may be permissible:

1. If diversion of portal blood from the liver through collaterals and naturally es-

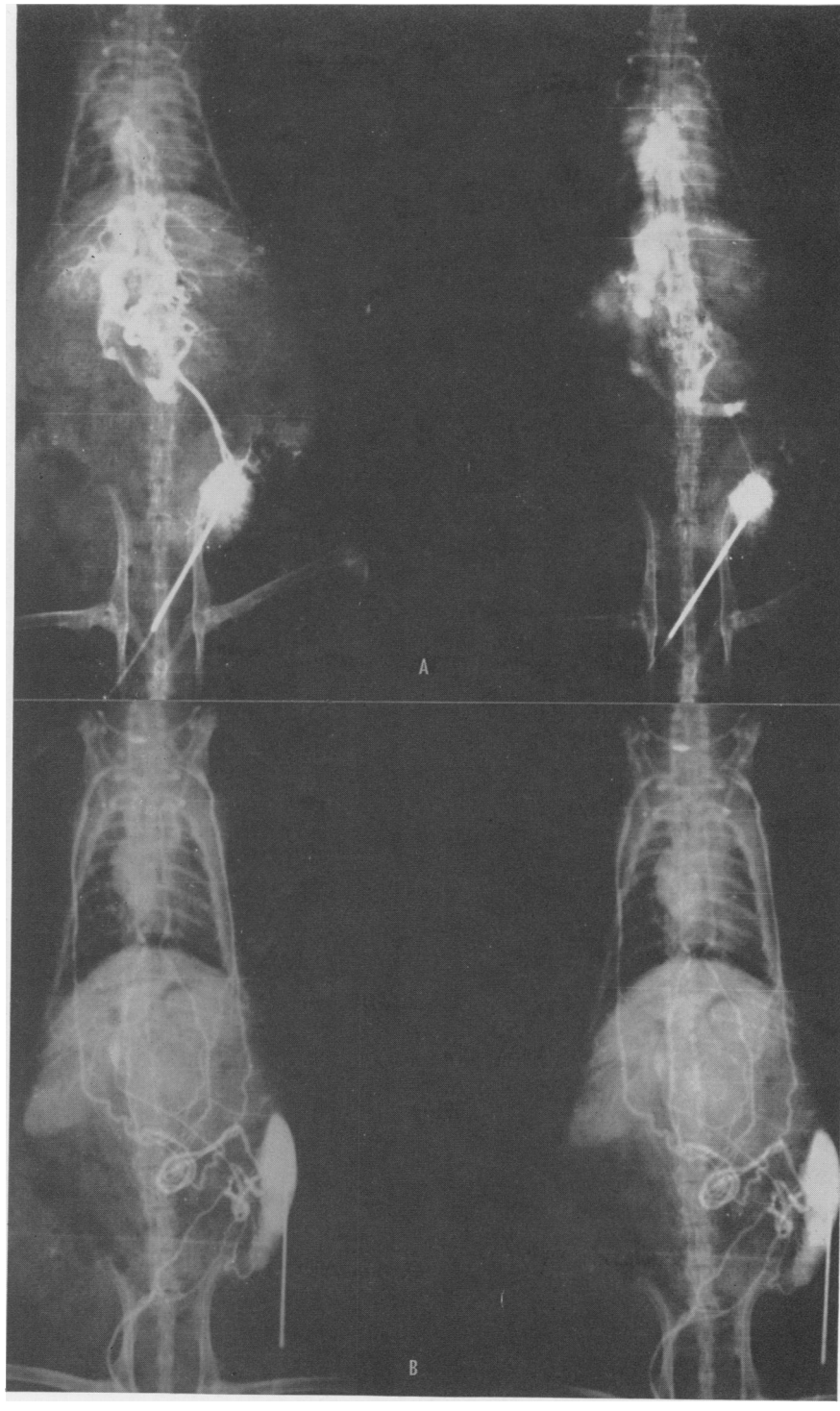

FIG. 10. Splenic portograms of two different rats showing hemodynamic situations similar to those observed in humans. A. Two films from a rapid-sequence series showing backflow into the coronary vein and dilated gastroesophageal varices. In the second film a natural shunt connecting with the left renal vein is clearly seen, as well as filling of infradiaphragmatic and supradiaphragmatic portions of the inferior vena cava. B. Caput medusa type of superficial abdomino-thoracic collateral circulation. The para-umbilical veins, the lateral thoracic, and internal mammary veins are well visualized.

tablished shunts is intended to decompress the portal system, it has failed to do so in the cases in this series.

2. Since diversion of portal blood from the liver is an undesirable situation *per se,* its failure to decompress the portal system is doubly regrettable.

3. When diversion of portal blood occurs through gastroesophageal plexus, the veins in these plexus dilate and become tortuous and sacculated. If these varices rupture, a third and most dangerously undesirable side effect follows, that is, severe gastrointestinal bleeding.

4. A surgically constructed shunt produced a small further decrease in the already diminished hepatic blood flow of patients with esophagogastric varices, but probably, it would have produced a sharp decline in patients without these varices who were demonstrated to have normal rates of hepatic blood flow. This situation will probably have to be considered if a shunt is attempted for the treatment of ascites in those patients not having esophagogastric varices.

This small further decrease in the presence of an already diminished hepatic blood flow may be responsible for the relative infrequency and mildness of ammonia intoxication observed in these cases. Perhaps the same shunt if performed in patients without esophagogastric varices and with values of hepatic blood flow similar to those in the control group would be followed by symptoms of greater severity.

Addendum

During the course of this investigation a separate experimental study was conducted in cirrhotic rats. Despite the generally accepted belief that the altered hemodynamics of portal hypertension as seen in humans is not reproducible in the experimental animal, a surprising similarity of circulatory changes was observed throughout the experiment. Practically every known portal circulatory derangement observed in patients with cirrhosis of the liver was reproduced in these animals, including the development of extrahepatic collateral circulation with the Caput medusa type of superficial abdomino-thoracic circulation and filling of paraumbilical, epigastric, internal mammary and lateral thoracic veins; hepatofugal flow into the coronary vein and gastroesophageal plexus with formation of dilated, convoluted esophagogastric varices; and the establishment of spontaneous porta-systemic venous shunts, particularly into the left renal vein, with active bypassing of the liver and filling of the inferior vena cava (Fig. 10).

A more detailed description of this experiment will be the subject of a future report.

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Discussion

Dr. Edgar J. Poth: Dr. Ravdin, Members and Guests: In view of the information we have received in these papers I think it would be interesting to recount something I have done during the past five years to relieve esophageal bleeding in patients with portal hypertension, where the other avenues for decompression, the splenic vein and the portal vein, are not available.

The esophagus is divided at the junction of the middle and distal third, closing the distal end to preserve the collaterals in the esophagus with the expectation that they would be protected from trauma, since food would not be passing over them, then re-anastomosing the stomach to the middle third of the esophagus or, better still, the interposition of a segment of jejunum, and doing a Heineke-Milkulicz pyloroplasty. This procedure has been done in several instances. The patients have had no further severe esophageal bleeding, and a significant amount of collateral venous drainage of the portal system has been preserved.

Dr. Roy Cohn: Dr. Ravdin, Gentlemen: Dr. Rousselot has pointed out the presence of veno-veno anastomoses in cirrhosis. But I should like to point out also that there has been a considerable amount of physiologic evidence for a long time that there are veno-arterial anastomoses in the presence of cirrhosis of the liver.

This physiologic information was first pointed out by Dr. Snell in 1932, who showed that the so-called walking cirrhotic had arterial oxygen saturations in the 80's; then Kowalski and Abelman, among others, have shown that in cirrhotics there is an increased cardiac output in about 30 percent of the patients.

(Slide) We have shown what others have also shown, that in cirrhosis of the liver about 30 per cent of the patients, again, who do not have evidence of renal valvular heart disease or pulmonary disease will have an increase in heart weight. In these slides, cirrhotic males, this upright line represents 400 grams, which is the upper limit of normal of the average male adult. You see the scatter here, whereas in the cirrhotic about 30 per cent of them will be above the upper limit of normal.

(Slide) The next slide shows the same thing is true in the female. We happen to have less of those than males. Therefore there must be somewhere in the body a point at which the venous blood can get into the arterial system as an obvious and a satisfactory explanation of this.

(Slide) That this does actually exist is shown in this splenal portogram, taken as postmorten angiography from Shonmaker's atlas. The injection into the portal bed shows the collateral veins going up and actually filling the pulmonary venous tree, so that because of the unique anatomy in this situation venous blood is able to pour directly into the arterial system, and therefore increase the cardiac output and desaturate the arterial blood. Thank you.

Dr. Arthur H. Blakemore: I enjoyed these papers very much. Dr. Rousselot's presentation was, I might mention, an excellent one. He brought up a subject we have puzzled over many times.

I remember one posterior communication that we traced down and it ended up in a sort of a bag