Arterialization of the Portal Vein in Conjunction with a Therapeutic Portacaval Shunt

Hemodynamic Investigations and Results in 75 Patients

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Seventy-five cirrhotic patients were submitted to peroperative hemodynamic investigations including flow and pressure studies. Sixty-two patients with a hepatopedal portal flow underwent a therapeutic end-to-side portacaval shunt (PC) in conjunction with arterialization of the portal vein and 13 with a stagnant flow a PC shunt alone. Thirty-five patients were operated on in emergency and 40 electively. In 61 patients portal flow was correlated with maximum perfusion pressure ($r = 0.66$), and in 33 patients with the reduction of corrected sinusoidal pressure induced by the occlusion of the portal vein ($r = 0.72$). Operative mortality, which was 3.5% for 57 class A and B patients and 55.5% for 18 class C patients, differed significantly ($p < 0.05$) in emergency between arterialized (14.8%) and nonarterialized patients (62.5%). At the time this study was ended on July 15, 1981, the follow-up was over two years for all the patients. The five-year actuarial survival rate of the arterialized patients was 48% for the whole group and 56% for class A and B patients; the overall incidence of chronic encephalopathy was 20%. It is concluded that arterialization is a safe surgical procedure that could be beneficial in respect with operative mortality in emergency, late survival, and tolerance to portacaval shunt. However, a prospective randomized study such as the one undertaken in December 1979 is the only method to prove clearly that arterialization is really able to minimize the risk of encephalopathy and to prolong the long-term survival after portacaval shunt.

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THE SURGICAL DECOMPRESSION of portal hypertension is not infrequently associated with a progressive deterioration of hepatic function. During the last 15 years, two different approaches have been explored to avoid these complications: selective decompression of oesophageal varices and arterialization of the portal vein.

Arterialization of the hepatic stump of the portal vein in conjunction with end-to-side portacaval shunt is based on the hypothesis that the increased incidence of hepatic failure after portosystemic shunting is at least partly related to the abrupt decrease of the total hepatic blood flow.[11] Indeed, some clinical reports have suggested that the incidence of postshunt encephalopathy was effectively decreased after arterialization.[2,11,15] However, the reported experience of this procedure is limited to a few hundred cases,[2,15] and several questions should be answered before a wider clinical application is recommended. Firstly, the indications for arterialization should be more precisely defined on a hemodynamic basis, since a wide range of residual portal flow is observed in cirrhotic patients,[13] and the compensation of the hepatic artery following end-to-side portacaval shunt is unpredictable from preoperative data.[4] Secondly, practical means should be available to the surgeon to prevent arterialization from exceeding the initial portal pressure and flow since histologic damage to the liver parenchyma has resulted, in experimental animals, from an excessive arterial inflow into the intrahepatic portal bed.[17] Thirdly, more information is needed about the influence of arterialization on the operative risk of portacaval shunt, both in emergency and electively, and also on the long-term survival; potential complications should be known.

An answer to these questions is brought at least partly by the retrospective study of the authors' experience with 75 patients operated on between September 1973 and May 1979; hemodynamic investigations, operative techniques, early and late mortality, complications, and results are described. According to the data collected in

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the course of the study, the patients with a hepatopedal portal flow were submitted to complete portal decompression in conjunction with an arterialization of the hepatic stump of the portal vein while the patients with a stagnant portal flow underwent an end-to-side portacaval shunt.

Clinical Material and Methods

Patients

The series includes 75 patients with portal hypertension who had suffered at least one episode of gastrointestinal bleeding related to rupture of varices as proved by endoscopy and/or barium studies. Portal hypertension was related to a sinusoidal and postsinusoidal block in 72 patients (two thirds of whom were alcoholics) and to a presinusoidal block in three patients (sarcoidosis, chronic vitamin A or arsenic intoxication). The operative risk was assessed according to Child's classification: 37 patients being in class A, 20 in class B, and 18 in class C (Table 1). Thirty-five patients were operated on in emergency, either because the bleeding was not controlled by balloon tamponnade and/or continuous intravenous vasopressin, or because the hemorrhage recurred soon after release of the medical means. Forty patients were operated on in elective conditions after full recovery from the last bleeding.

At the beginning of the study, it was decided to perform an arterialization only in patients with a hepatopedal portal flow; accordingly, 62 patients were submitted to complete portal decompression in conjunction with an arterialization of the portal vein while 13 patients with a stagnant portal flow underwent a standard portacaval shunt. The clinical characteristics of these patients are detailed in Table 2.

Deaths occurring in the hospital during the postoperative period were recorded as operative deaths. The surviving patients were regularly examined at the follow-up clinic, at three-month intervals during the first postoperative year, at six-month intervals during the second postoperative year, and yearly thereafter. At the time this study was ended on July 15, 1981, the follow-up was over two years for all the patients. Encephalopathy was evaluated according to the clinical criteria of Voorhees et al.[24] and was defined as acute or chronic when it lasted, respectively, for less or more than 30 consecutive days.[18]

Methods

Surgical techniques. The operation was performed through a right subcostal incision; after completion of the peroperative hemodynamic investigations, a standard end-to-side portacaval anastomosis was performed.

TABLE 1. *Child's Classification*

	PCS* with ART†			PCS*		
	Total	Elective	Emergency	Total	Elective	Emergency
A	33	24	9	4	4	—
B	18	9	9	2	1	1
C	11	2	9	7	—	7
Total	62	35	27	13	5	8

* PCS = end to side portacaval shunt.
† ART = arterialization of the hepatic stump of the portal vein.

In patients with a hepatopedal portal flow, arterialization of the intrahepatic portal bed was achieved by means of a saphenous vein graft that was interposed between the infrarenal aorta and the hepatic stump of the portal vein. Great care was taken to ensure that arterialization was not excessive by keeping deliberately the flow and the pressure in the hepatic portal bed below their initial values; to achieve that purpose, banding of the prosthesis was realized in 16 patients.

Hemodynamic investigations. The corrected sinusoidal pressure (CSP), defined as the difference between the wedged hepatic vein pressure and the free hepatic vein pressure,[25] was measured before surgery and at various stages of operation by means of a Swan-Ganz balloon catheter, introduced percutaneously through the right jugular vein and advanced into one of the main hepatic veins. The balloon catheter was connected to a pressure transducer (Statham X. M. 5327/00) and a paper recorder (Cardiopam 3 M Phillips). The free hepatic vein pressure was recorded first; the balloon was then expanded with 0.6 ml of air and the wedged hepatic vein pressure recorded.[19] The ΔCSP was calculated as the difference between the values of CSP recorded during the operation, with the portal vein being open or occluded. After operation, the CSP was monitored during 36 to 48 hours; in the later follow-up, the measurement of CSP was repeated when complications, such as ascites and/or jaundice, occurred.

TABLE 2. *Clinical Data*

	PCS* with ART†	PCS*
Number of patients	62	13
Mean age (range)	50.7 (22–68)‡	48.8 (35–68)‡
Sex ratio (M:F)	2:1	1.1:1
Intrahepatic block		
Sinusoidal and postsinusoidal	60	12
% alcoholic cirrhosis	58	66
Presinusoidal	2	1

* PCS = end-to-side portacaval shunt.
† ART = arterialization of the hepatic stump of the portal vein.
‡ Range.

TABLE 3. *Portal Flow and Pressure Studies*

	61 Patients	33 Patients
Q^{PV} (ml/min.)	484 ± 41 (0/1600)	440 ± 56.5 (0/1250)
MPP (cm H_2O)*	19.8 ± 1.37 (−3/42)	
PPP (cm H_2O)	8.6 ± 0.72 (−3/27)	
ΔCSP (mmHg)†		3.57 ± 0.62 (−3/11)

Q^{PV} = Portal flow; MPP = Maximum perfusion pressure; PPP = Portal perfusion pressure; ΔCSP = Reduction of corrected sinusoidal pressure induced by peroperative portal occlusion.

Data expressed as $\bar{X}$ ± SEM (Range).

* Correlation between Q^{PV} and MPP: $r = 0.668$: $p < 0.001$.

† Correlation between Q^{PV} and ΔCSP: $r = 0.72$; $p < 0.001$.

At the beginning of the procedure, the portal pressure was measured by direct puncture with a water manometer in the portal vein (free portal pressure, FPP) and on both sides of a clamp occluding the portal vein (splanchnic occluded portal pressure [SOPP] and hepatic occluded portal pressure [HOPP]). The maximum perfusion pressure (MPP) (difference between the splanchnic occluded portal pressure and the hepatic occluded portal pressure) and the portal perfusion pressure (PPP) (difference between the free portal pressure and the hepatic occluded portal pressure) were calculated.[16] At the end of operation, the pressure was taken in the portal vein above the portacaval shunt and, when appropriate, in the arterialized hepatic stump of the portal vein (HAPP). The pressure was measured also by direct puncture in the inferior vena cava, before and after the portacaval shunt.

The portal blood flow (Q^{PV}) was measured with an electromagnetic flowmeter (Devices blood flow meter, 3765, Devices Limited, Great Britain) in 61 patients. The statistical analysis of the peroperative hemodynamic data obtained in these patients, which forms part of this report, shows that, in the authors' experience and in contrast to the data reported by others,[3] the residual portal blood flow can be inferred with a good approximation from pressure differences induced by the clamping of the portal vein; therefore this indirect method of flow estimation was used in the 13 remaining patients; a single case had no flow evaluation. As the result of this peroperative portal flow evaluation, each patient could be categorized as having either a hepatopedal or a stagnant portal flow even when direct measurement was not available.

The flow through the venous graft (*i.e.*, the arterial inflow into the hepatic portal bed) was measured with the flowmeter in 49 out of the 62 patients who underwent arterialization; it was indirectly estimated from the restituted hepatic portal (HAPP) and sinusoidal pressures in the other patients with exception of three cases. Recording of the hepatic artery flow permitted measurement of the total hepatic blood flow in six patients.

Statistical methods. Means were compared using Student t-tests for paired and nonpaired observations; proportions were compared with chi-square test without or with Yates' correction or with Fisher exact test; linear correlation was calculated using least squares method.[9,22] Survival curves were drawn according to the actuarial method.[6]

TABLE 4. *Selection for Arterialization*

	Hepatopedal Flow n = 49	Stagnant Flow n = 12
Q^{PV} (ml/min.)	583 ± 40 (100/1000)	77 ± 9 (0/100)
MPP (cm H_2O)	23.3 ± 1.22 (6/42)	5.5 ± 1.4 (−3/17)
PPP (cm H_2O)	10.1 ± 0.72 (2/24)	2.3 ± 0.7 (−3/7)

Q^{PV} = Portal flow; MPP = Maximum perfusion pressure; PPP = Portal perfusion pressure.

Data expressed as $\bar{X}$ ± SEM (Range).

Results

Peroperative Hemodynamic Data

Portal flow studies. The 61 patients whose portal flow was measured with the flowmeter before portacaval shunting (Table 3) had a mean portal flow of 484 ± 41 ml/min., a mean maximum perfusion pressure of 19.8 ± 1.37 cm H_2O and a mean portal perfusion pressure of 8.6 ± 0.72 cm H_2O. Maximum perfusion pressure was correlated ($r = 0.668$, $p < 0.001$) with the portal flow. In a subgroup of 33 patients in whom the simultaneous measurement of CSP was achieved, ΔCSP was also found to be correlated ($r = 0.72$, $p < 0.001$) with the portal flow. Among these 61 patients who had a direct portal flow measurement, 49 were classified into the hepatopedal portal flow group (group a) and 12 into the stagnant portal flow group (Table 4).

In the 13 patients not submitted to direct flow measurement, the maximum perfusion pressure ranged from 10 to 30 cm H_2O (mean: 20.9 ± 1.75) and the portal perfusion pressure from 5 to 18 cm H_2O (mean: 11 ± 1.32) in 12 patients, while the last patient had negative (−3 cm H_2O) MPP and PPP; these 12 patients (group b) were allocated to the hepatopedal portal flow group on the basis of the correlation observed between MPP and portal flow in the 61 patients whose flow was directly measured; for the same reason, the remaining patient was allocated to the stagnant portal flow group. In summary, 61 patients* were considered to have a hepatopedal portal flow and were submitted to arterialization

* In order to avoid misunderstanding, the fact should be stressed that the 61 patients who were considered to have a hepatopedal portal flow (groups a and b) are not the same as the 61 patients whose portal flow was measured directly with the flowmeter.

TABLE 5. *Comparison of Blood Flow and Pressures in the Hepatic Portal Bed Before and After Arterialization*

	n	Before PCS	After PCS and ART
Hepatic portal blood flow (ml/min)	49	583 ± 40	342 ± 24*
Intrahepatic portal pressure (cm H_2O)	57	36.7 ± 0.73	32.2 ± 0.99*
Corrected sinusoidal pressure (mmHg)	23	15.86 ± 0.93	12.34 ± 0.93*

* $p < 0.001$. Data expressed as $\bar{X}$ ± SEM.

of the hepatic stump of the portal vein in conjunction with complete portal decompression as was the single patient who had no blood evaluation (to make a total of 62 arterialized patients); the 13 patients who were considered to have a stagnant portal flow underwent a standard portacaval shunt.

Comparison of blood flow and pressure in the hepatic portal bed before and after arterialization. The hepatic portal blood flow was 583 ± 40 ml/min before PC shunting and 342 ± 24 ml/min after arterialization in 49 patients. The intrahepatic portal pressure was 36.7 ± 0.73 cm H_2O before PC shunting and 32.2 ± 0.9 cm H_2O (HAPP) after arterialization in 57 patients. The corrected sinusoidal pressure was 15.86 ± 0.93 mm Hg before PC shunting and 12.34 mmHg ± 0.93 after arterialization in 23 patients (Table 5). The differences between the values recorded before shunting and after arterialization were highly significant ($p < 0.001$) and corresponded approximately to a reduction by one third of the initial flow and by one fifth of the initial pressure in the hepatic portal bed.

Total hepatic blood flow before and after arterialization. The data of the total hepatic blood flow measured in six patients before and after portacaval shunt and arterialization are listed in table 6. Despite the small number of data and individual variations, they indicate that arterialization in these six patients restored the total hepatic blood flow at a slightly lower level than preshunt values. About two thirds of the portal vein flow measured before shunting were delivered to the liver through the arterialization while a substantial increase of the flow through the hepatic artery was observed in three patients despite arterialization.

Operative Mortality.

The operative mortality was 16% for the whole group of 75 patients, 11.2% (7 out of 62) for the arterialized patients and 38.4% (5 out of 13) for the patients not arterialized. The difference between the two subgroups was not statistically significant except for emergency cases: four out of 27 arterialized patients (14.8%) and five out of eight patients not arterialized (62.5%) died during the postoperative period. Details of operative mortality are given in table 7.

The main cause of operative death was hepatic failure in eight patients, exsanguination from oesophageal rupture due to prolonged balloon tamponnade in one patient with incidental hepatoma, stroke in one patient, and pulmonary complications related to tuberculosis in one patient; one single death was possibly related to hyperarterialization (intractable ascites with renal failure). Postmortem examination performed in each case showed that all vascular anastomoses were patent.

Encephalopathy, Long-term Survival and Late Fatalities

Arterialized patients. Among the 55 patients who survived beyond the first month, 15 (27%) showed encephalopathy, acute in four cases (7%) and chronic in 11 (20%); four of the latter patients already had encephalopathy before surgery. Acute encephalopathy was seen exclusively in alcoholics and was precipitated by heavy drinking in all cases but one; chronic encephalopathy was more frequent in patients who underwent emergency surgery. Twelve out of the 15 patients were dead at the time of assessment after a median survival time

TABLE 6. *Hepatic Blood Flow (ml/min)*

	Before PCS and ART			After PCS and ART		
Patients	Portal Vein	Hepatic Artery	Total	Arterialization	Hepatic Artery	Total
1	600	150	750	650	150	800
2	200	150	350	150	200	350
3	600	175	775	400	400	800
4	1000	100	1100	700	100	800
5	300	200	500	150	200	350
6	1200	200	1400	600	600	1200
Mean	650	160	810	440	275	715

TABLE 7. *Operative Mortality*

		PCS + ART	PCS
Emergency	9/35 = 25.7%*	4/27 = 14.8%*	5/8 = 62.5%*
Elective	3/40 = 7.5%*	3/35 = 8.5%	0/5 = 0%
Class A-B	2/57 = 3.5%*	2/51 = 3.9%	0/6 = 0%
Class C	10/18 = 55.5%*	5/11 = 45.5%	5/7 = 71.4%

* $p < 0.05$.

of 16 months (end-stage liver failure in ten cases and bleeding duodenal ulcer or hepatoma in two cases). The median follow-up time of the three survivors was 62 months at the time of assessment.

The actuarial survival curves of the 62 patients who were arterialized are shown in Figures 1 and 2; the five-year survival rate was 48% for the whole group and 56% for class A and B patients.

Among the 55 operative survivors of the arterialized group, 24 patients had died by mid-July 1981. The rate of late mortality was higher for class C patients (five out of six) than for class A and B patients (19 out of 49) but was the same for all patients whether operated on electively or in emergency (43%). Death occurred during the first postoperative year in seven cases, between one and two years in seven, between two and three years in five, between three and four years in two, between four and five years in one case and over five years in two cases. The cause of death was liver failure (18), acute viral hepatitis (one), bleeding duodenal ulcer (one), neoplasm (two), stroke (one), and nephrotic syndrome (one).

Patients with standard PC shunt. The eight patients who survived the procedure were alive at the time of assessment (median survival time: 31 months; range: 27–48 months), three of them suffering from chronic encephalopathy.

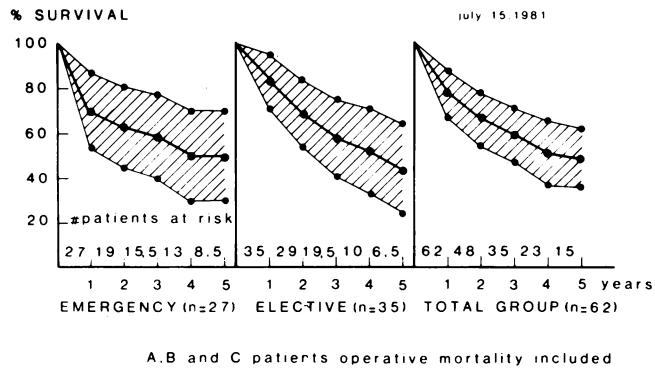

FIG. 1. Actuarial survival curves of 62 arterialized patients. Interval of 95% confidence is represented by the striped area. Numbers of patients at risk during each postoperative year are indicated at the bottom of the figure above the abscissa.

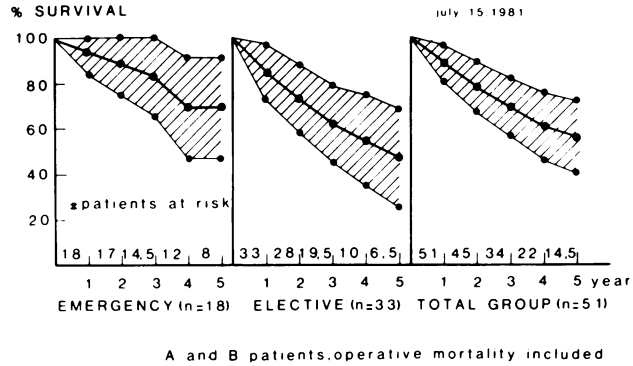

FIG. 2. Actuarial survival curves of 51 class A and B patients. Interval of 95% confidence is represented by the striped area. Numbers of patients at risk during each postoperative year are indicated at the bottom of the figure above the abscissa.

Patency of the Arterialization

Patency of the prosthesis was investigated directly in 31 patients, either by aortography (22), surgical exploration (five) or autopsy (four). The prosthesis was patent in 26 out of the 31 patients (84%) after a median postoperative interval of nine weeks for the radiograph-examined patients, two weeks for the surgically explored and nine months for the autopsied patients. In the patients with patent prosthesis, a systolic murmur could usually be heard in the right upper quadrant of the abdomen, on the surface of the liver or under its lower margin. In the other 31 patients, no direct exploration was performed, but a murmur was present in 28 (90%), the longest follow-up with a murmur being eight years. None of the patients explored by aortography showed feeding of the varices by collaterals of the arterialized intrahepatic portal system.

Postoperative Ascites and Jaundice in Arterialized Patients

Massive and intractable ascites developed in six patients either immediately (three patients) or during the second or the third postoperative week (three patients); only one of these patients had ascites before operation. The patency of PC shunt and venous graft was proved by angiography in all instances. The CSP was recorded repeatedly; in comparison with the preoperative value, it had increased considerably in five patients, reaching at least twice the preoperative level, while it had decreased in one patient. The five patients with an increased CSP were surgically re-explored between day one and day 16; the flow through the venous graft was found to have approximately doubled in each patient. The venous graft was ligated in three patients and narrowed by a banding in two patients. The first patient encoun-

tered with this complication died shortly after reoperation; the other patients were cured of ascites and survived the complication. In the sixth patient with a decreased CSP, a Leveen shunt was placed at 16 months with a good result. Transient ascites was observed in four patients between the first postoperative day and the sixth week. A nonsurgical cause was identified in every case and responded to medical treatment in three cases; one patient with hepatic failure died. None of these patients showed a significant increase of CSP; vascular anastomoses were patent in all instances.

Severe jaundice, exceeding 10 mg % of serum bilirubine, was observed in eleven patients, either in the early postoperative period (six patients) or later between three weeks and six months (five patients). The jaundice resulted from acute viral B hepatitis in two patients and from non-A non-B acute hepatitis in two patients (with two deaths). Progressive hepatic failure leading to hepatic coma and death was observed in six patients; in three of these, hepatic failure was possibly precipitated by hyperarterialization. Alcoholism was also responsible for severe jaundice in one patient; cure followed abstinence.

Discussion

The world experience of arterialization of the portal vein in conjunction with a portacaval shunt that was collected in 1977 by Adamsons[2] amounted to 246 cases (including the authors' first 14 cases)[15] among which only 24 had been operated on in emergency conditions. By May 1979, the authors' own experience had been extended to the 62 cases that form the basis of this report. In the collected series, the operative mortality of arterialization averaged 17% after elective surgery; the influence of this procedure on hospital mortality in emergency situations and on long-term survival was unknown, owing to the lack of detailed studies and the small number of cases.

In the authors' experience, arterialization did not increase the low operative mortality of elective cases, but it significantly reduced the operative mortality in emergency (14.8%) which used to be much higher in the authors' previous experience[23] and averaged 40% in several reported series of the standard portacaval shunt.[8,12,14] However, the operative mortality remained prohibitive in class C patients despite concomitant arterialization; therefore these patients are now treated in this institution by endoscopic sclerosis of oesophageal varices.

The addition of arterialization to portacaval shunting undoubtedly increases the operative trauma and the length of operation; nevertheless, no postoperative death was directly attributed to the procedure itself in the collective reviews of Maillard[11] and Adamsons,[2] with the exception of one case of Maillard. In the authors' own series, arterialization "per se" contributed to death in a single patient owing to the delayed recognition that intractable ascites was possibly related to secondary increase of the flow through the venous prosthesis. Arterialization can thus be considered as a safe surgical procedure.

Up to now, no data have been published concerning the long-term survival of arterialized patients. The five-year survival rate of the 62 patients (operative deaths included) is 48%; it reaches 56% for class A and B patients operated on in elective conditions. The only, but still disputable, comparison could be made with the three controlled series[7,18,21] of the therapeutic portacaval shunt whose data combined by Conn[5] gave a survival rate of about 44% after four years; a slight improvement in long-term survival could be observed in the series of arterialized patients despite a much higher proportion of class C patients (17%) and emergency operations (43%) than in the controlled studies. Reduction of postshunt encephalopathy has been the major purpose of the various teams involved in clinical arterialization. Maillard[11] and Adamsons[2] have presented encouraging results; the incidence of 27% that was observed among 55 surviving patients is lower than that reported by Jackson et al.,[7] Resnick et al.,[18] and Rueff et al.[21] in their randomized studies (respectively, 34, 50, and 41%). However the authors' follow-up is shorter, and it is well known that the incidence of postshunt encephalopathy increases with time. Whether arterialization really minimizes the risk of encephalopathy and prolongs the long-term survival could be clarified only by a prospective randomized study such as the one undertaken in December 1979.

The purpose of arterialization is the prevention of an abrupt decrease in hepatic blood flow, but this rationale has often been questioned inasmuch as there seems to be no correlation between the extent of preshunt portal flow and either the hospital or long-term mortality or the incidence of portal systemic encephalopathy, as shown by Burchell et al.[3] Moreover the same authors[4] have shown that the increment in arterial flow was the only hemodynamic variable directly related to morbidity, hospital mortality and long-term survival. This observation, however, supports the view that residual total hepatic blood flow after a PC shunt has some bearing on the tolerance to the operation, and it implies that arterialization of the portal vein could be beneficial for at least those patients whose arterial flow does not increase adequately in response to the relief of sinusoidal hypertension. That hepatic arterial flow can fail to increase commensurately with the diverted portal flow is

demonstrated by the correlation (r = 0.72), observed in 33 of the patients, between the portal flow and the peroperative reduction of the corrected sinusoidal pressure in response to the occlusion of the portal vein. Furthermore, the data obtained in a few of the cases in which the hepatic artery flow was measured (Table 6) suggest that arterialization of the portal vein limits but does not preclude the increase of the flow through the hepatic artery; detailed peroperative flow measurements made in more recent cases appear to confirm this observation.

The authors share Adamsons' opinion[2] that arterialization is indicated when hepatopedal portal flow has been proven by peroperative hemodynamic investigations; the lack of rationale and the fear of complications have led to the avoidance of arterialization in patients with a spontaneous reversal of portal flow. Since excessive arterialization has to be avoided as shown by experimental studies,[17] peroperative hemodynamic investigations should comprise reliable evaluation of both the residual portal flow and the arterial flow subsequently restored into the intrahepatic portal bed.

In order to avoid occasional errors, the authors think that direct flow measurement should be completed by pressure studies. Maximum perfusion pressure is an indirect but easily available method for evaluating the portal flow; its value has been denied by Burchell and coworkers[3] but the authors' own data in 61 patients have shown a correlation (r = 0.66) between MPP and the portal flow measured with the flowmeter. It is hard to explain the contradiction between these two studies that concern similar numbers of patients except that some electromagnetic measurements might have been erroneous as suggested by Burchell and coworkers.[3] In the authors' experience, the peroperative recording of the CSP also gives an evaluation of the portal flow which was found in 33 patients to be in correlation (r = 0.72) with the reduction of CSP induced by the occlusion of the portal vein.

To prevent postoperative complications and further damage to the cirrhotic liver and to allow adaptation of the hepatic artery, great attention was paid in this series to limit the arterial flow into the intrahepatic portal bed to about two thirds of the pre-existing portal flow.

To attain this purpose, banding of the prosthesis was required in 16 patients before termination of the operation. Despite the precautions that were taken during surgery, several patients developed postoperative complications that were possibly related to progressive increase of the flow through the prosthesis. Intractable ascites were observed in five patients with a considerable increase of CSP over the peroperative values; re-exploration showed that the flow through the venous prosthesis had approximately doubled by comparison with the values recorded at the end of the first operation. Reduction of the flow achieved by total or partial ligation of the prosthesis was followed by disappearance of ascites and the cure of four patients. A cholostatic type jaundice was observed by Maillard[10] in one patient whose blood pressure in the prosthesis had increased from 20 mmHg at the first operation to 40 mmHg at the second. Reduction of the pressure by partial ligation of the prosthesis was followed by a rapid decrease of jaundice. The authors experienced the same complication in one patient, and the banding of the prosthesis also resulted in a steep decrease of jaundice over the next days. Maillard[11] reported that there was no relation between increase in sinusoidal pressure and increase in total hepatic blood flow. In the authors' experience however, increase in sinusoidal pressure has been a useful guide for detecting excessive arterialization when intractable ascites or cholostatic jaundice developed after operation. If arterialization is the elected procedure for preventing the decrease in total hepatic blood flow resulting from decompression of the portal bed, the authors suggest that besides the peroperative hemodynamic investigations that are mandatory, sinusoidal pressure should be monitored after operation and repeatedly measured in case of complications possibly related to excessive arterialization.

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