Hepatic Artery Flow Improvement After Portacaval Shunt:

A Single Hemodynamic Clinical Correlate

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We have documented a highly significant increment in hepatic arterial flow following a portacaval shunt in patients with cirrhosis of the liver and portal hypertension. In contrast with other hemodynamic variables, the increment in arterial flow was directly related to morbidity, hospital mortality, and long term survival. Patients with increments smaller than 100 ml/min had the worst clinical results. They accounted for all of the hospital mortality, the largest incidence of encephalopathy, and the worst long term cumulative survival rates. The extent of the increment was not related directly to the type of shunt but, rather, to some intrinsic capability of the cirrhotic liver to increase its arterial flow in response to the relief of sinusoidal hypertension produced by the shunt. This capability appears related to the degree of entrapment of the hepatic arterioles by the fibrous tissues of cirrhosis. This encasement of arterioles should change the elastic properties of the hepatic arterial bed and we propose to measure these properties by determining the characteristic input impedance of the arterial bed.

WITH REMARKABLE EXCEPTIONS, it can be said that portacaval shunts are compromise solutions for otherwise unsolvable situations, i.e., death from exsanguinating gastroesophageal hemorrhage or, more rarely, wasting ascites refractory to all forms of conservative treatment. Although shunts are effective means of correcting these situations, the 10-year life expectancy for patients with cirrhosis of the liver subjected to these operations is less than 20%.[31] Some patients are unable to cope with the initial insult of the operation and die during their hospital stay. Furthermore, the quality of life of the survivors is compromised by a 30% incidence of moderate to severe portalsystemic encephalopathy. And yet, there are sufficient instances of patients leading long and productive lives as to discourage any thoughts of abandoning this operation.

Because of the previous considerations, it was only logical that many investigators, including ourselves, would endeavor to find means of identifying those patients who would have poor results from those that might have reasonable outcomes. Leaving aside patients whose condition is obviously deteriorated, clinical and laboratory findings have not been very helpful means for this identification and this might be one of the reasons why surgeons began to look into the possible significance of several hemodynamic variables. For a while, it seemed reasonable that patients with large pre-shunt portal flow should do worse than patients with portal flow markedly reduced by diversion through collateral pathways. Here, the reasoning was that abrupt diversion of a large portal flow by the shunt should produce more harmful effects than diversion of a small flow which had been gradually reduced during the natural course of the disease. As a consequence of this reasoning, there was a proliferation of methods to estimate pre-shunt portal flow using a variety of free and occluded pressure measurements as well as radiologic and radioactive studies.[20,39,40,41,43] In two previous communications we reported lack of correlation between various estimates of

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portal flow and the magnitude of the actually measured flow and between the latter and clinical course of our patients.[7,28] We also discussed the hemodynamics of the occluded portal pressures and showed their lack of relation to the portal flow in the absence of occlusion. Other works in the literature have sought clinical correlations with a presumed spontaneous reversal of portal flow in cirrhosis.[9] Recently, we analyzed the case for and against this spontaneous reversal and concluded that the evidence was mostly circumstantial and that, if existing at all, its incidence must be so rare as to have only marginal clinical significance.[29,30]

Among several editorial comments on our results[2,35,37] one, by T. B. Reynolds, expressed the hope that hepatic

TABLE 1. *Data Ordered by Magnitude of Post-shunt Increment in Hepatic Arterial Flow*

	Hepatic Artery Flow (ml/min)				Hepatic limb Flow†						
Pt. No.	Increment	Pre	Post-shunt	Shunt type*	Prograde	Retrograde	Encephalopathy	Hospital Mortality	Dead at (yr, mos)	Lost to Followup (yr, mos)	Alive at (yr, mos)
92	0	680	680	E.S.	‡—	—	+	–	3 4	– –	– –
123	0	130	130	E.S.	‡—	—	+	+	– –	– –	– –
147	25	420	445	S.S.	§—	—	–	+	– –	– –	– –
149	25	295	320	S.S.	105	—	–	–	1 6	– –	– –
102	30	290	320	E.S.	‡—	—	+	–	4 0	– –	– –
106	30	290	320	E.S.	‡—	—	–	–	4 8	– –	– –
97	40	860	900	E.S.	‡—	—	+	–	8 11	– –	– –
126	40	180	220	S.S.	250	—	–	–	0 4	– –	– –
152	45	185	230	S.S.	§—	—	–	+	– –	– –	– –
90	70	350	420	E.S.	‡—	—	+	–	9 10	– –	– –
101	70	380	450	E.S.	‡—	—	+	+	– –	– –	– –
130	70	210	280	S.S.	200	—	+	+	– –	– –	– –
142	70	410	480	S.S.	§—	—	+	+	– –	– –	– –
150	70	410	480	S.S.	§—	—	+	–	0 5	– –	– –
146	75	310	385	S.S.	470	—	–	–	0 2	– –	– –
138	90	230	320	E.S.	‡—	—	+	+	– –	– –	– –
133	100	220	320	S.S.	270	—	–	–	0 3	– –	– –
122	105	625	730	E.S.	‡—	—	–	–	– –	– –	8 0
132	120	420	540	S.S.	—	980	–	–	0 5	– –	– –
98	130	250	380	E.S.	‡—	—	+	–	0 4	– –	– –
125	130	220	350	E.S.	‡—	—	+	–	6 9	– –	– –
131	140	280	420	S.S.	—	340	–	–	– –	4 2	– –
121	160	550	710	E.S.	‡—	—	–	–	2 1	– –	– –
103	170	610	780	S.S.	680	—	+	–	0 1	– –	– –
153	170	310	480	S.S.	§—	—	–	–	– –	3 1	– –
141	220	510	730	E.S.	‡—	—	–	–	– –	3 3	– –
91	250	230	480	S.S.	—	288	–	–	– –	8 8	– –
94	250	490	740	E.S.	‡—	—	–	–	2 5	– –	– –
115	255	425	680	E.S.	‡—	—	–	–	5 1	– –	– –
114	270	410	680	E.S.	‡—	—	–	–	3 9	– –	– –
99	295	135	430	S.S.	¶ 0	0	+	–	– –	6 10	– –
116	315	295	610	E.S.	‡—	—	–	–	0 2	– –	– –
154	330	280	610	S.S.	—	440	–	–	– –	3 1	– –
136	340	280	620	S.S.	—	660	–	–	5 1	– –	– –
143	370	430	800	S.S.	—	625	–	–	– –	3 7	– –
135	400	280	680	S.S.	—	720	–	–	2 5	– –	– –
117	410	490	900	E.S.	‡—	—	+	–	– –	– –	8 4
145	410	410	820	S.S.	—	520	–	–	– –	– –	4 11
120	420	420	840	E.S.	‡—	—	–	–	– –	6 1	– –
151	425	395	820	S.S.	—	380	–	–	– –	– –	4 2
139	430	210	640	S.S.	§—	—	–	–	– –	0 9	– –
107	445	295	740	E.S.	‡—	—	–	–	2 6	– –	– –
134	460	420	880	S.S.	§—	—	–	–	– –	– –	5 10
144	470	410	880	S.S.	—	1295	–	–	– –	– –	4 11
148	500	380	880	S.S.	—	410	–	–	4 8	– –	– –
156	510	480	990	S.S.	—	230	–	–	– –	– –	3 6
137	700	420	1120	S.S.	—	920	–	–	2 10	– –	– –

* E.S.: End-to-side anastomosis. S.S.: Side-to-side anastomosis.
† Refers to direction of flow in hepatic limb of a side-to-side portacaval anastomosis.
‡ Not applicable.
§ Not measured.
¶ Stagnant flow.

arterial flow measurements might still be useful. Actually, on the basis of a preliminary series of measurements, we proposed to the American Surgical Association in 1968 that there should be a correlation between the post-shunt increment in hepatic arterial flow and the clinical course of our patients.[6] Now, with an increasing number of measurements of pre and post-shunt hepatic arterial flow and with a prolonged course of follow-up of our patients, we begin to find statistically significant patterns which point to the post-shunt increment in hepatic arterial flow as the only clinical correlate so far uncovered. These patterns appear to be the first meaningful clue towards an understanding of the relationship between hemodynamics and clinical results. Despite the complexity of the problem, the fundamental features of our interpretation appear sound and we will present them to the best of our current understanding.

We recognize that our results are retrospective but, because of the information and understanding they provide, they should pave the way for future applications and predictions. In the section of Discussion of this report we introduce the concept of a new variable, the characteristic input impedance of the hepatic arterial bed, which should be related to the elastic properties of the bed. We further propose to relate these elastic properties to the capability of the hepatic arterial bed to increase its flow in response to the lowering of sinusoidal pressure produced by the portacaval shunt. Within our present technology, we can measure this variable immediately before the construction of the shunt by using the ratio of instantaneous pressure to instantaneous flow in the hepatic artery. It is conceivable that some future estimates of this variable could be made preoperatively at the time of selective hepatic artery catherization for angiographic studies.

Materials and Methods

Forty-seven patients admitted to the medical and surgical services of St. Vincent's Hospital and Medical Center of New York from January 1966 through April 1972, are the subjects of this study. These 47 patients were selected from our larger overall experience with portacaval shunt only on the basis of having obtained technically successful measurements of hepatic artery flow before and after the construction of a portacaval shunt. No patients were excluded and they were in no other way unique. An end-to-side portacaval anastomosis was constructed in 20 patients and a side-to-side shunt in the remaining 27.

The followup data were available on all operative survivors for a period of 3 years 6 months to 9 years 10 months with a mean followup period of 6 years and 10 months. Nine patients were lost to followup (mean 4 years and 11 months) and they were treated by the actuarial method for cumulative survival rates.[4] All the patients were followed by the same team of surgeons who performed or supervised their portacaval shunts.

TABLE 2. *Increment in Hepatic Artery Flow After Portacaval Shunt (in ml/min)*

Number of patients	47
Pre-shunt flow:	364 ± 21 S.E.
Post-shunt flow:	582 ± 34 S.E.
$p \ll 0.001$	

Flows were measured with successive generations of the square wave electromagnetic flowmeter of Denison and Spencer (Carolina Medical Electronics, Winston Salem, North Carolina). Statistical treatment of the data and graphic plots were obtained using a small digital computer and attached x-y plotter (Hewlett-Packard, Models 9100-A and 9125-A).

Results

The raw data from which all our computations were derived are given in Table 1. They could also be used for further computations if desirable in the future. There was a statistically highly significant increment in hepatic arterial flow after the establishment of a portacaval anastomosis: $P \ll 0.001$ (Table 2). There was no correlation between the size of the pre-shunt hepatic artery flow and its post-shunt increment ($r = 0.11$, $P < 0.7$) or between the latter and the magnitude of the pre-shunt portal venous flow ($r = 0.13$, $P < 0.9$). The difference in increment in hepatic arterial flow between the 20 patients with end-to-side anastomosis and the 27 patients with side-to-side anastomosis had a borderline statistical significance: $P < 0.05$ (Table 3). However, these results changed markedly when the patients with side-to-side shunts were divided in groups according to the direction of venous flow in the hepatic limb of their anastomosis. We had these measurements in 20 of the 27 patients with side-to-side shunts. In 13 of these patients, hepatic blood exited the liver via the hepatic limb of the anastomosis. The average volume flow leaving the liver via this newly created accessory outflow tract was 596 ± 86 S.E. (ml/min). One patient had stagnant flow in

TABLE 3. *Increment in Hepatic Arterial Flow After Various Types of Portacaval Anastomosis in 47 Patients (ml/min)*

End-to-side:	172 ± 32 S.E.	$P < 0.05$
All side-to-side:	260 ± 37 S.E.	
Side-to-side with prograde flow:	80 ± 21 S.E.	$P < 0.001$
Side-to-side with retrograde flow:	381 ± 43 S.E.	
End-to-side:	172 ± 32 S.E.	$P < 0.001$
Side-to-side with retrograde flow:	381 ± 43 S.E.	

TABLE 4. *Increment in Hepatic Artery Flow in Relation to Encephalopathy (ml/min)*

Number of patients	47
Encephalopathy:	110 ± 28 S.E.
No encephalopathy:	275 ± 31 S.E.
$P < 0.001$	

TABLE 5. *Post-shunt Increment in Hepatic Artery Flow (ml/min) and Hospital Course of Patients*

Number of patients	47
Hospital mortality:	53 ± 12 S.E.
Survivors:	252 ± 28 S.E.
$P < 0.01$	

the hepatic limb of the shunt with only to and fro motion of the blood synchronous with respiration. In six other patients with side-to-side shunts, splanchnic blood continued entering the liver via the hepatic limb of the anastomosis and this prograde flow averaged 329 ± 86 S.E. (ml/min). Patients with prograde flow in the hepatic limb of their side-to-side shunt had a smaller increment in hepatic artery flow than those who had retrograde flow and the difference was statistically highly significant: $P < 0.001$ (Table 3). The difference between the larger increment in hepatic artery flow in patients with retrograde flow in their side-to-side shunt and that of patients with end-to-side anastomosis was also highly significant: $P < 0.001$ (Table 3). Patients with persistent flow into the liver via the hepatic limb of their side-to-side shunts had the smallest increment of this entire group (Table 3).

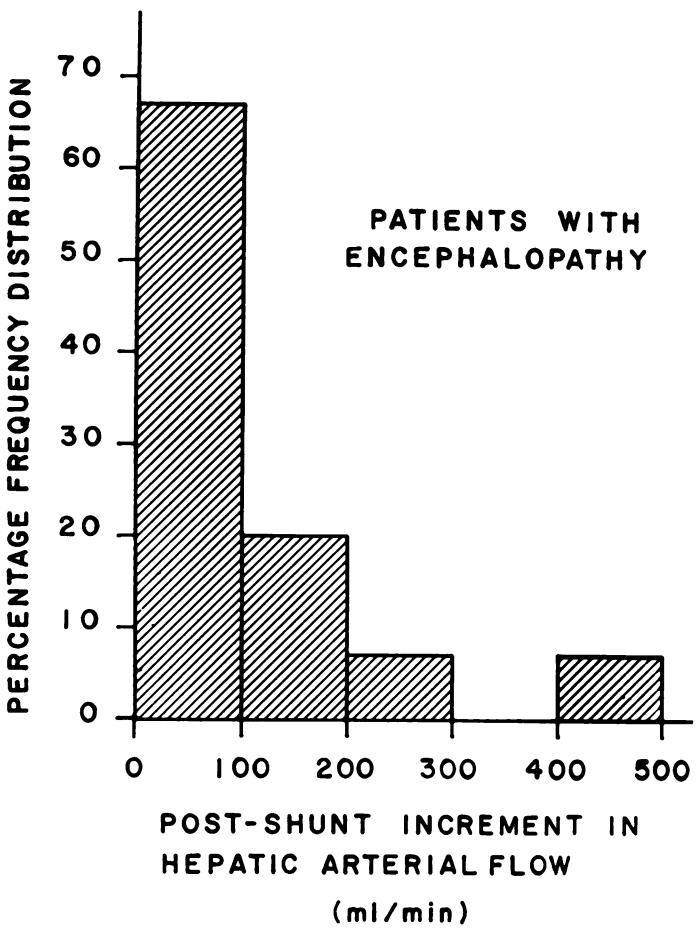

FIG. 1. Frequency distribution of post-shunt increments in hepatic arterial flow in patients suffering from portalsystemic encephalopathy. Note that almost 70% of these patients had increments smaller than 100 ml/min.

In our series, there were 15 patients (32%) who had moderate to severe portalsystemic encephalopathy following the shunt. The anastomosis was of the end-to-side type in 10 of these patients and of the side-to-side variety in the remaining 5 patients. A test of significance for the different incidence of encephalopathy according to the type of shunt gave a $X^2 = 5$ with a probability value of $P < 0.025$. However, the difference between the small post-shunt increment in hepatic arterial flow of the patients who developed encephalopathy and the larger increment of those who were free of this complication was statistically highly significant: $P < 0.001$ (Table 4). The frequency distribution in Fig. 1 shows that almost 70% of the patients having post-shunt encephalopathy had increments in hepatic arterial flow smaller than 100 ml/min. Actually, 10 of the 17 patients (59%) with increments smaller than 100 ml/min had encephalopathy, while only 5 of the 30 patients (17%) with increments larger than 100 ml/min suffered from this complication ($X^2 = 8.9$; $P < 0.003$). Note also that only 2 of the 22 patients (7%) with increments larger than 200 ml/min had encephalopathy.

Seven of our 47 patients (15%) died in the hospital while 40 survived their hospital stay. Three of the 7 patients who died had an end-to-side anastomosis and four had a side-to-side anastomosis. The difference was not statistically significant ($x^2 = 0.0003$). However, there was a significant statistical difference between the very small increment in hepatic artery flow of the patients who died in the hospital and the larger increment of the patients who survived: $P < 0.01$ (Table 5). All of the 7 patients who died during their hospital stay had increments smaller than 100 ml/min and, consequently, none of the 30 patients with increments larger than 100 ml/min died during their hospitalization. Eleven of the 17 patients (65%) with increments smaller than 100 ml/min were dead within one year of their portacaval shunt while only 4 of the 30 patients (13 per cent) with increments larger than 100 ml/min died within one year ($X^2 = 13.2$; $P < 0.0005$). In the overall series, 32% of patients died during the first year following the portacaval shunt, and their increment in hepatic arterial flow (135 ± 33 S.E. ml/min) was significantly smaller ($P < 0.01$) than that of

those surviving this period of observation (267 ± 32 S.E. ml/min). The increment in hepatic arterial flow in this group of patients was not related to the type of shunt they received. The increment in those having end-to-side shunts (169 ± 64 S.E. ml/min) was not significantly different ($P < 0.4$) from that in patients having side-to-side shunts (114 ± 37 S.E. ml/min).

The difference in survival in relation to the magnitude of the post-shunt increment in hepatic arterial flow can be more clearly seen in Fig. 2 where, using the actuarial method,[4] we plotted cumulative survival rates vs time. The projected 10-year survival rate for patients who had post-shunt increments in hepatic arterial flow larger than 100 ml/min was 40% against a projected zero 10-year survival rate for those having increments of less than 100 ml/min. The difference between the two survival rates was statistically highly significant ($X^2 = 6.8$; $P < 0.008$) and the probability of surviving, calculated from the ratios of the sums of expected and observed mortality, was three times greater for patients with increments larger than 100 ml/min. When the two groups were combined (curve not plotted), the projected 10-year survival rate approached the usual 20% figure.

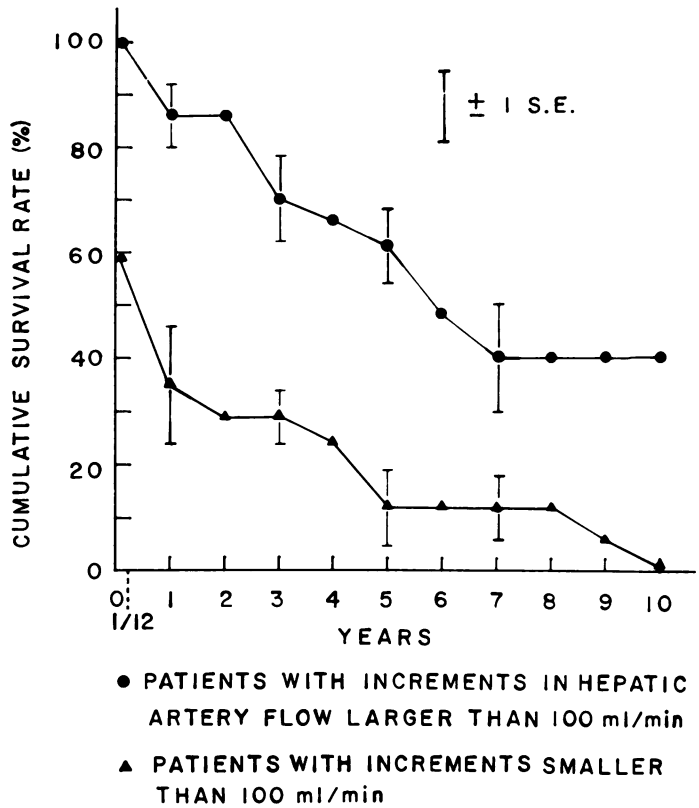

FIG. 2. Cumulative survival curves constructed using the actuarial method (see text) in patients who had post-shunt increments in hepatic arterial flow larger than 100 ml/min and smaller than this quantity. Note that the 1/12 marking on the abscissa corresponds to hospital mortality. The difference between the two curves was statistically highly significant ($X^2 = 6.8$; $P < 0.008$).

Discussion

The discussion of our results is not simple and, in some instances, predisposes to circular thinking. There are areas in which it is not always obvious how to establish proper cause-effect relationships between the variables accessible to our measurements and the observed results. There could also be "hidden" variables which were operating without our recognition. At the cost of some artificiality, and inevitable overlapping of subjects, we will discuss the most important issues under separate subheadings before attempting to integrate them into a unified overall view. We will also indicate some new avenues of investigation which appear logical consequences of our present results.

Pathophysiology of post-shunt increment in hepatic arterial flow

A mutual influence between the two inflow tracts of the liver, the portal vein and the hepatic artery, was suspected to exist in cirrhosis after early perfusion studies of organs removed from cadavers.[21] With the advent of practical electromagnetic flowmeters, some relationships were demonstrated in live human subjects submitted to surgical interventions. That clamping of the portal vein produces changes, usually increases, in hepatic arterial flow was clearly shown in the early studies of Schenk and associates,[36] Ferguson,[13] and Price and associates.[33] The latter also observed post-shunt increments in hepatic arterial flow in a small group of patients subjected to portacaval anastomosis and they thought that some resistance in an "unknown location" must have been lowered by the operation. We believe that their thinking was correct and will indicate the most probable location of such resistance at the same time that we offer a plausible mechanism to account for its lowering after construction of a portacaval shunt. We will use current knowledge of the behavior of denervated terminal vascular beds where the relationship between arteriolar, capillary, and venous pressures and resistances have been studied in detail under controlled conditions.* We must emphasize that the equation given in the footnote was derived for denervated vascular beds and cannot account for any neurochemical-vascular feedback loop. However, it is a very useful expression to visualize the interaction of the

* The value of the capillary pressure (p_c), a variable of great interest to us, is given by an equation derived by Pappenheimer and Soto-Rivera[32]:

$$p_c = [(r_v/r_a)(p_A + p_V)][1 + (r_v/r_a)]^{-1}$$

where r_v and r_a are respectively venous and arteriolar resistance, and p_A p_V are the arterial and venous pressure.

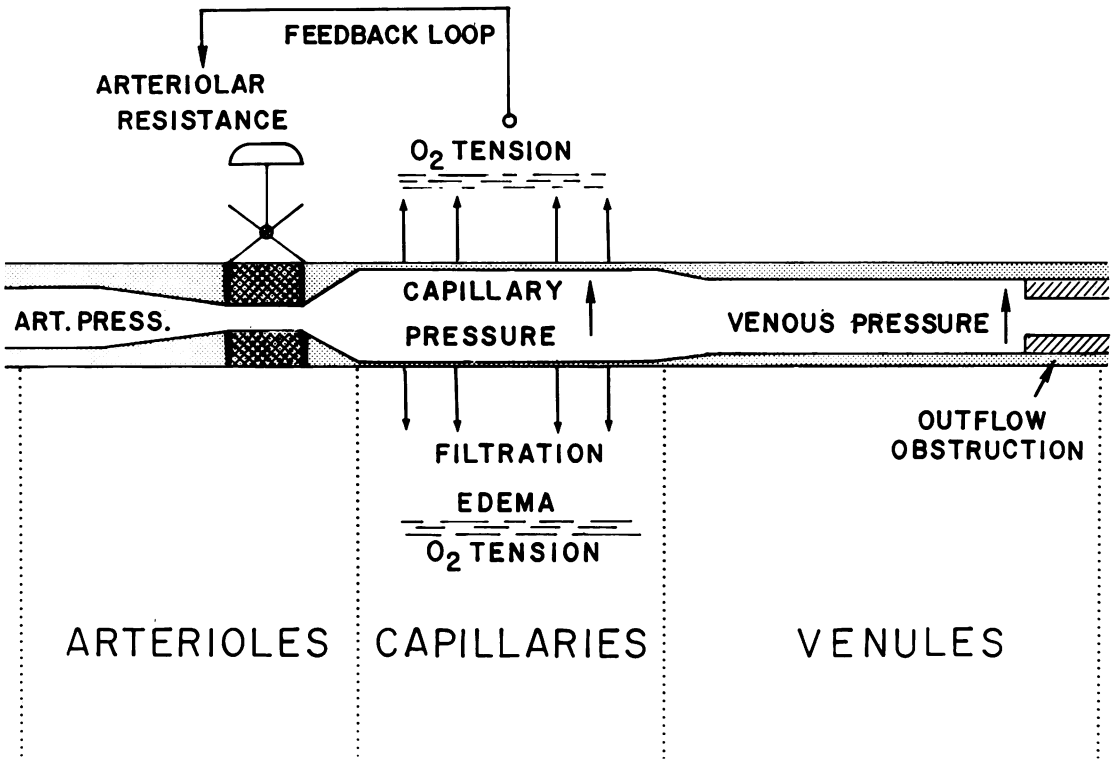

FIG. 3. Schematic representation of the sequence of events that develops after outflow obstruction of any terminal vascular bed. The controlled variable which sets up the feedback loop leading to arteriolar constriction and diminished arterial flow is the low tissue oxygen tension resulting from increasing capillary pressure and excessive filtration into the extravascular spaces.

variables involved. In cirrhosis with portal hypertension there is impairment of venous outflow, the so-called postsinusoidal obstruction, which leads to a sequence of increased venous resistance, increased venous pressure, and increased sinusoidal pressure. The arterial reservoir "does not see" these important but comparatively small changes in pressure (in relation to its own high pressure) because of the presence of a normally high arteriolar resistance which acts as an isolating resistor. The terms large and small, as used here, have only relative significance. Thus, a large increment in venous outflow resistance with respect to its normal value, is still small when compared with the normal arteriolar resistance which dissipates almost 90% of the pressure energy imparted to the blood by the ejecting ventricle. Nevertheless, the resulting elevation in capillary pressure has important consequences. This elevation of sinusoidal pressure in cirrhosis has been repeatedly documented by hepatic vein "wedged" pressure measurements. A high venous outflow resistance in cirrhosis has been also documented by the relatively large gradient between the abnormally elevated sinusoidal pressure obtained from the "wedged" position and the relatively small free hepatic vein or caval pressures. In contrast, a very small presinusoidal resistance has been demonstrated by the elegant studies of Reynolds and associates,[34] who showed negligible differences between the sinusoidal pressure measured from the hepatic vein "wedged" position and the simultaneous value of the portal pressure measured from a catheter inserted into the portal vein via the recanalized umbilical vein. For the purpose of our discussion, the data of Reynolds and associates are important because they will permit us to make very good approximations of sinusoidal pressure from the value of the free, not the occluded, portal pressure.

Contrary to the simplified case of denervated terminal vascular beds, the case of the hepatic circulation is significantly more complex. First, the capillary or sinusoidal bed has the extra inflow provided by the portal venules which contributes to the value of the sinusoidal pressure. The now more complicated network with complex relationships between pressures, resistances, and flows can be best visualized using the steady state analysis of the Wheatstone bridge analogy which we have given in detail in previous communications.[28,29] Second, the neuro-chemical-vascular feedback loops of the system should be considered in the analysis.

An important consequence of the elevated sinusoidal pressure secondary to outflow obstruction is the increment in transmural pressure across the sinusoidal wall, i.e., the difference between the pressure of the plasma within the sinusoids (P_{pl}) and that of the extravascular interstitial fluid (P_{if}).* According to the equation for fluid movement across a capillary wall given in the footnote,

* Fluid movement (FM) across a capillary wall is given by[17]:

$$FM = k[P_{pl} - \Pi_{pl} - P_{if} + \Pi_{if}]$$

which, disregarding the grouping of signs and variables, says that filtration into the extravascular spaces is favored by an increase in transmural pressure or hydrostatic gradient ($P_{pl} - P_{if}$), by an increased osmotic gradient ($\Pi_{if} - \Pi_{pl}$) and by a larger permeability of the capillary wall (k).

we see that all the conditions for an increased filtration into the extravascular tissues are met in the cirrhotic liver: the hydrostatic gradient is increased by elevation of the sinusoidal or plasma pressure; the osmotic gradient is increased (toward the extravascular spaces) by lower levels of plasma proteins; the filtration constant (k) is known to be the highest of any capillary bed in the body even under normal conditions,[15,18] and there is a presumption that it may be further increased in cirrhosis.[26] Findings of such large structures as red blood cells in the thoracic duct lymph of some patients with cirrhosis[10] may support this presumption. Excessive transudation of fluid into the extravascular spaces in cirrhosis has been shown by us using intraparenchymal deposition of contrast medium[26] and it can also be presumed from the increased rates of thoracic duct lymph found by Dumont.[11] The extreme case of excessive transudation leads to weeping of hepatic lymph from the hilar structures of the liver which, if exceeding the capability of the peritoneal surfaces to absorb it, results in ascites. The fact that not all patients with cirrhosis and portal hypertension develop ascites indicates that the causes discussed above may be necessary but not sufficient to produce the accumulation of ascitic fluid. How other causes may be required to coexist cannot be included within the limits of this report.

As in other types of edema, transudation of plasma into the hepatic extravascular spaces tends to be a self-limiting phenomenon. This is apparently the result of efforts to prevent the accumulation of a large fluid barrier that would severely impair interchanges of oxygen and other products between the capillaries and the extravascular spaces. One way of limiting transudation is to reduce the inflow of blood into the capillaries. This prevents further elevations in sinusoidal pressure as the disease progresses and further impairs venous outflow. Probably, the first to suffer is the portal inflow because the splanchnic venous bed cannot elevate its pressure beyond some 500 to 600 mm H_2O (37 to 44 mm Hg) and the gradient for portal flow, i.e., the pressure differential between the splanchnic venous reservoir and the sinusoidal bed becomes progressively reduced. With this reduced gradient, splanchnic blood finds it easier to take the route of collateral pathways having lower downstream pressures and portal inflow diminishes by an average of more than one-half the normal value. In many patients, portal inflow is very small or even ceases.[27–29] Reducing hepatic arterial flow is a more complicated problem and cannot be achieved passively as is the case with the portal inflow. This is due not only to the higher head of pressure in the arterial reservoir but also to the active throttle effect of the arteriolar resistance. It is known that physiological preparations with obstructed venous outflow, elevated capillary pressure, and increased filtration rates, have decreased tissue oxygen tension.[14] In these preparations,

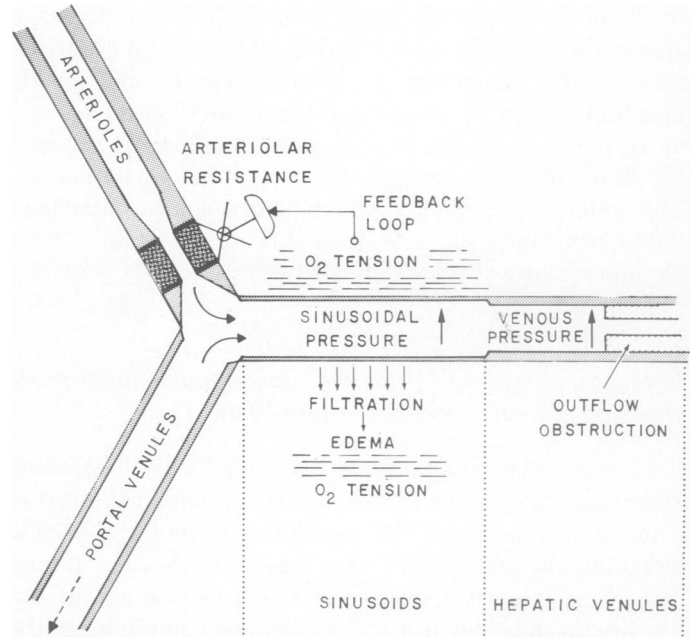

FIG. 4. A similar situation as in Fig. 3 for the more complicated hepatic circulation. Here, not only the arterioles but also the portal venules contribute to the value of the sinusoidal pressure. Portacaval shunts diminish sinusoidal pressure by removing the portal contribution, the case of end-to-side shunts, or by creating a portal accessory outflow tract, the case of side-to-side shunts. The broken arrow in the portal venules indicates removal of sinusoidal blood by the accessory outflow tract with consequent relief of sinusoidal hypertension.

the diminished oxygen tension appears to set up a feedback loop which increases the arteriolar resistance and diminishes the arterial inflow. It seems that the price of a lower rate of perfusion is less costly than that of a decreased oxygen tension. The role of the tissue oxygen tension as the controlled variable has been shown in several tissues,[14] and there is no reason to believe that this would not be true for the hepatic tissues of cirrhotic livers with outflow obstruction. A clinical example of this arteriolar constriction, and reduced arterial flow, is found in the venous outflow obstruction of patients with thrombophlebitis.[5] Fig. 3 is a schematic representation of the sequence of events in physiological preparations with venous outflow obstruction, and Fig. 4 shows the more complicated arrangement in the hepatic circulation.

Within the above scheme of nature, it becomes possible that a portacaval shunt by reducing the sinusoidal pressure would deactivate the feedback loop and reverse the process, i.e., increase the reduced arterial inflow. However, it is important to note, at this point, that there was no proportionality in our patients between the magnitude of the post-shunt increment in hepatic arterial flow and the size of the pre-shunt arterial inflow, a finding that may indicate that other factors in addition to those discussed may be involved in determining the size of the post-shunt increment. We will return to this subject in the next section of this discussion. In the meantime, we will only state that there are two plausible

mechanisms by which portacaval shunts may decrease sinusoidal pressure: by removing all the portal contribution to the sinusoids, as is the case in end-to-side anastomosis or by creating an accessory outflow tract, as is the case in the side-to-side shunts with reversal of flow in the hepatic limb of the anastomosis. The side-to-side shunts which show some splanchnic blood still entering the liver via their hepatic limb should occupy a place in between the two previous situations.

Influence of type of portacaval anastomosis upon post-shunt increment in hepatic arterial flow

In our initial computations we found only borderline statistical differences in post-shunt increment in hepatic arterial flow between the patients who had end-to-side anastomosis and those who had side-to-side shunts. However, when we divided the side-to-side shunts according to the direction of flow in their hepatic limb, the difference in favor of those who had reversed flow became statistically highly significant as was shown in Table 3. We will attempt to discuss the meaning of these results although we should warn the reader that this is a subject that may readily lead into the pitfalls of circular reasoning.

There is no question that in our series, all side-to-side shunts which "succeeded" in reversing the flow in their hepatic limb were associated with the largest post-shunt increments in hepatic arterial flow. It seems reasonable that this newly created outflow tract would relieve the outflow obstruction and, therefore, decrease the sinusoidal pressure to the largest extent. In purely hydraulic terms, this would result in the largest post-shunt increment in hepatic arterial flow. On the other hand, the side-to-side shunts which "failed" to reverse the flow and continued delivering splanchnic blood into the sinusoids should reduce the sinusoidal pressure even less than the end-to-side shunts which remove all of the portal inflow into the sinusoids. This reasoning would agree with the fact that patients having side-to-side shunts which "failed" to reverse flow in their hepatic limb had the smallest increment in hepatic arterial flow of the entire group. It may also point to the undesirability, with respect to increasing hepatic arterial flow, of operations which deliberately preserve portal inflow.[42]

The arguments in the previous paragraph appear reasonable, perhaps too reasonable for those of us who have become accustomed to distrust oversimplified schemes. In the first place, we have no right to assume that some side-to-side shunts "failed" while others "succeeded" in reversing the flow in the hepatic limb of the anastomosis. This seems to imply that the shunts which "failed" were mechanically inferior or defective shunts, an implication which might be true, but for which we have absolutely no evidence. Perhaps the strongest objection to this oversimplified reasoning is the fact that the post-shunt increment in hepatic arterial flow was not proportional to the increment in the gradient of pressure between the arterioles and the sinusoids. A few computations will clarify this point. For the best case, a portacaval shunt would lower the portal pressure from say, 30 mm Hg to about 15 mm Hg.[27] If we assume that the sinusoidal pressure is lowered by an approximately equal amount, a not entirely unreasonable assumption, we can estimate the gradient or pressure differential for hepatic arterial flow before and after construction of the shunt. Any errors introduced by this assumption would be in the direction of overestimating the extent of lowering of sinusoidal pressure and would add strength rather than weakness to our next argument. For a mean arterial pressure of approximately 100 mm Hg, the increase in gradient produced by the shunt would be about 20% and this quantity would not vary very much from patient to patient since pre and post-shunt values of portal pressure vary within a very narrow range.[27] Despite this rather constant increment in gradient for post-shunt hepatic arterial flow, the actual increment in flow varied from as little as zero to as much as 200%.

Portacaval shunts do appear to increase hepatic arterial flow by lowering the sinusoidal pressure and, to a certain extent, it is possible that the greater the lowering the larger the increase in arterial flow. In this sense some types of shunts may appear superior to others. However, as just calculated, the variation in post-shunt arterial pressure gradient is not within the range of the observed variation in the post-shunt increment in hepatic arterial flow. Therefore, this variation must be related to some intrinsic ability, or inability, of the cirrhotic liver to increase its arterial flow once that it has been given fair, good, or excellent improvements in the pressure differential.

A "hidden" variable for post-shunt increment in hepatic arterial flow and a plausible mechanism limiting the magnitude of such increment

Granting that portacaval shunts, by lowering sinusoidal pressure, initiate the sequence of events leading to post-shunt increments in hepatic arterial flow, we are forced to conclude that other important mechanisms must also determine the final size of the increment. From the previous section, it is clear that with a constant arterial pressure, the rather limited range of improvement in the pressure differential for post-shunt arterial flow cannot account for the large range of variation of the actual increment in this flow. Clearly, within the restricted range of change of the pressure gradient, the large variations

in flow can only be ascribed to adjustments of the only remaining determinant of flow, the hepatic arteriolar resistance. It is entirely conceivable that a hepatic arteriolar resistance, with its value increased above normal by the feedback mechanisms proposed in the first section of this discussion, would tend to return to more normal values once the variable controlled by the feedback loop is relieved. All the available evidence points to diminished tissue oxygen tension produced by a large fluid barrier secondary to sinusoidal hypertension as this controlled variable. Consequently, hepatic arteriolar resistance should decrease after a portacaval shunt and this would result in a corresponding improvement in hepatic arterial flow. However, two facts of observation complicate the interpretation of this issue. First, the post-shunt increment in hepatic arterial flow was not proportional to the pre-shunt magnitude of the arterial flow and, second, patients with identical post-shunt decreases in sinusoidal pressure had very different increments in hepatic arterial flow. Both these facts seem to point to some limiting factor determining the extent of lowering of the hepatic arteriolar resistance for any given magnitude of the improvement in the pressure gradient. We propose that this factor, or "hidden" variable, be the degree of encasement of the arterioles by the dense fibrous tissues of cirrhosis. Thus, firmly entrapped arterioles may not be able to dilate sufficiently to yield large post-shunt increments in hepatic arterial flow.

The working hypothesis offered above would explain why a small pre-shunt arterial flow, presumably the result of a severely "clamped" arteriolar resistance, does not necessarily result in a large post-shunt increase once the cause for the severe constriction is relieved. Thus, the post-shunt increase in hepatic arterial flow may not only be a function of the extent of reduction of the pre-shunt flow but also a function of the capability of the arterioles to dilate within their fibrous environment in response to lowering of sinusoidal hypertension. Actually, large pre-shunt flows may be the result of either modest increases in arteriolar resistance or, quite the opposite, the result of arterioles unable to constrict their lumen because they are severely encased in fibrous tissues. An entire family of intermediate and converse situations can be readily postulated and they would be consistent with the lack of proportionality between the post-shunt increment in hepatic arterial flow and either the magnitude of the pre-shunt arterial flow or the extent of improvement of the pressure gradient.

As will be discussed in the next section, we have identified retrospectively small post-shunt increments in hepatic arterial flow with unfavorable clinical results. Therefore, it would be of utmost importance to find means of predicting the capability of the hepatic arterioles to dilate and increase their flow once that sinusoidal hypertension has been relieved by a portacaval shunt. In the final section of this discussion we will examine some promising avenues of investigation that might lead to future useful pre-shunt predictions.

Clinical correlations

Our results showed clear correlations between the size of the post-shunt increment in hepatic arterial flow, the incidence of encephalopathy, the occurrence of hospital and early mortality, and the projected long term survival. They open the way to a better understanding of the relationship between hemodynamic variables and clinical results and, perhaps, to the development of future criteria for a more reasonable selection of patients. In the meantime, our findings should be taken with caution and should not be used as a basis to offer or deny shunt operations to any particular group of patients. Within this restrictive framework, we will discuss the possible clinical implications of our results. Of necessity, some aspects of the discussion will remain speculative.

On first examination our data indicates that if we had not operated on the patients who had post-shunt increments smaller than 100 ml/min, we would have cut by one-half the incidence of encephalopathy, eliminated the hospital mortality, and doubled the projected 10-year survival rate. As a general statement this is a correct statement, however, and irrespective of the fact that we did not know the extent of the increment until we completed the shunt, there are other considerations which limit its range of application to individual patients. Although 65% of the patients in this group were dead before the end of one year, there were two patients who lived free of encephalopathy for one year and one-half and almost 5 years respectively. These two patients would have been denied a shunt if a criterion for selection requiring increments in hepatic arterial flow larger than 100 ml/min would have been accessible and acceptable to us and, probably, this denial would have been unfair to the patients. More debatable would have been the case of four patients in this group who survived from over three years to almost 10 years but who were afflicted by serious encephalopathy. In summary, we may say that although the general response of this group was significantly poorer, there were a few individual instances in which survival could be considered as reasonably long. The philosophical questions as to the choice between a poor quality of life and no life at all cannot be resolved by us. On the other hand, the 11 patients who either died in the hospital or a few months after discharge could have definitely been spared the anguish and suffering of a truly major surgical ordeal which did not benefit them in any way. We should

emphasize that categorizing patients by a single variable is a very difficult undertaking and it is remarkable that we have succeeded to the degree discussed here. Actually, mortality and morbidity of these patients is affected by many variables which, among the more obvious, are progressive liver disease, smoldering hepatitis, continued alcoholism, age group, and many others. Furthermore, measurements with electromagnetic flowmeters are subjected to errors and artifacts which may distort the data for individual patients while still preserving the general tendencies of the groups. There are two patients in our series, patients 132 and 144, in whom one of the measurements must have been in error since the flow in the accesory outflow tract created by a side-to-side shunt was larger than the hepatic inflow. Two final facts of observation are pertinent to the discussion of this group of patients with post-shunt increments in hepatic arterial flow smaller than 100 ml/min. One of them is that all patients in this group had died at the time of closing our study. The other is that within the patients with prograde flow in the hepatic limb of their side-to-side shunt, all but one died in less than 4 months following the operation. The latter finding poses the question as to whether or not patients who do not establish an accesory outflow tract once that they are given the opportunity to do so might belong in a particularly poor risk group.

Considering now the group of patients who had post-shunt increments in hepatic arterial flow larger than 100 ml/min, the analysis presents fewer ambiguities. Only 17% of these patients suffered from encephalopathy, an incidence almost one-half of that in our overall experience with this complication. Almost 90% of the patients in this group survived more than one year and, at the time of closing our study, 23% were known to be alive and 30% had been lost to followup. Treating these figures by the actuarial method yields a projected 10-year survival rate of 40% which is double that expected in our overall experience with patients with cirrhosis subjected to portacaval shunt. Anderson and associates, in an important article from Starr's laboratories,[1] have emphasized the suitability of the actuarial method for analysis of series of patients following a surgical event. In essence, the cumulative survival rates computed by this method do not consider patients lost to followup as being alive, which would bias the series in favor of the surgical treatment, or as being dead, which would bias the series against the surgical treatment. Additionally, the method provides a place for patients who may die of entirely unrelated causes such as automobile accidents or myocardial infarction.

End-to-side and side-to-side shunts were evenly distributed among the patients with increments in hepatic arterial flow larger than 100 ml/min. Although more patients with side-to-side anastomosis were alive or lost to followup at the end of the study there were no clear statistical differences with respect to the patients having end-to-side anastomosis. Three patients suffering from encephalopathy had end-to-side shunts while two patients afflicted by this complication had side-to-side shunts. Obviously, the difference is not significant. However, of the two patients with side-to-side shunts, one had prograde flow and the other stagnant flow in the hepatic limb of their shunt. Again, the question poses itself as to whether or not the patients who do not establish an accessory outflow via the hepatic limb belong in a group of particularly poor risk patients. Conversely, none of the patients with retrograde flow in the hepatic limb suffered from encephalopathy, a fact that might be due to the combined portal and hepatic decompressive effect of a side-to-side shunt advanced originally by McDermott[19] and Welch.[44] That this decompressive effect requires retrograde flow in the hepatic limb of the shunt appears obvious from our data, a fact that may also question the desirability of operations that deliberately preserve portal inflow.[42] The question cannot be resolved from our data but it appears pertinent and should be investigated by surgeons who use this type of operations. A similar question could be asked concerning operations designed to arterialize the portal bed. In this case the sinusoids instead of being relieved from the hypertension created by the disease will receive the full impact of a high arterial pressure which has not been dissipated by any arteriolar resistance.

Future avenues of exploration

Our results have opened the way for the exploration of new approaches to evaluate the capability of the hepatic arterial bed to increase its flow in response to the relief in sinusoidal hypertension produced by a portacaval shunt. In a previous section we have proposed that entrapment of hepatic arterioles by the fibrous tissues of cirrhosis is the limiting factor, or "hidden" variable, determining the magnitude of the post-shunt increment in arterial flow. Thus, firmly entrapped arterioles may not be able to dilate sufficiently to give substantial increments in flow. Conversely, arterioles not so firmly encased might yield those substantial increases measured in some patients. If entrapment of arterioles by fibrous tissues is indeed our "hidden" variable, its effects should be reflected in changes in the elastic properties of the hepatic arterial bed. In turn, changes in elastic properties should induce changes in the characteristic input impedance of the arterial bed. We propose to measure this characteristic impedance as a new variable that might predict the magnitude of the increment in hepatic arterial flow produced by a portacaval shunt.

The theory of characteristic input impedance has been developed by analogy with electrical networks theory and its measurement is obtained from the instantaneous ratio of pressure to flow in the main artery feeding any terminal vascular bed.[8,38] The characteristic input impedance of the pulmonary arterial bed has been measured in dogs and man by cardiovascular physiologists,[22–25] and has recently made its entrance in the surgical literature.[12] As a complex number, the input impedance has both a modulus and a phase angle. The modulus provides general information concerning the elastic properties of the arterial bed while the changes in phase angle are related to the location of rigid segments within the bed. The latter changes should be useful for an evaluation of the extent of encasement of the hepatic arterioles by the fibrous tissues of cirrhosis.

We propose to measure the characteristic input impedance of the hepatic arterial bed during operation, but before constructing the shunt. We further propose to correlate the value of the impedance with the magnitude of the increment in hepatic arterial flow after clamping the portal vein and with that after constructing the shunt. If a good correlation between the value of the impedance and the magnitude of the increment does exist, this measurement may become a reasonable predictor of the increment and, consequently, of the clinical outcome. This phase of the investigation need not be too long because we have already the long term information which correlates increments in hepatic arterial flow with clinical results and what we need to find out is if the impedance correlates with the increment. We also plan to explore the possibility of obtaining some approximation to the value of the impedance at the time of preoperative selective hepatic artery catheterization for angiographic studies. In an indirect way, the concepts presented in this section should be implicit in the philosophy behind Kessler's evaluation of patients from measurements of flow within a pump circuit attached to the subject.[16]

Conclusions

We have documented statistically significant increments in hepatic arterial flow following a portacaval shunt in patients with cirrhosis of the liver. By analogy with the arteriolar constriction observed in patients with venous obstruction secondary to thrombophlebitis, we postulate that the hepatic venous outflow obstruction of patients with cirrhosis and portal hypertension constrict the hepatic arterioles and reduces arterial flow. The presumed mechanism includes a feedback loop initiated by an abnormally elevated sinusoidal pressure, excessive transudation of fluid into the extravascular spaces, and diminished tissue oxygen tension. A portacaval shunt by relieving sinusoidal hypertension would deactivate the loop, release the arteriolar constriction, and increase the hepatic arterial flow.

The magnitude of the post-shunt increment in hepatic arterial flow was directly related to the morbidity, hospital mortality, and long term survival of our patients. Taken literally, our results indicate that if we had not operated on patients with increments in hepatic arterial flow smaller than 100 ml/min, we would have cut by one-half the incidence of encephalopathy, eliminated the hospital mortality, and double the projected 10-year survival rate. However, pending further confirmation, these results should be taken with caution. It is remarkable to realize how clinical outcome is so closely related to hepatic perfusion by arterial blood even in the absence of whatever portal factors are removed by the shunt.

Presumably, portacaval shunts decrease sinusoidal pressure by eliminating or reducing portal inflow into the sinusoids or be creating an accessory outflow tract. Although side-to-side shunts should produce the largest decrease in sinusoidal pressure this would only be accomplished if the hepatic limb of the shunt drains hepatic blood into the vena cava. If the hepatic limb does not operate as an accessory outflow tract and some portal blood continues entering the liver, they would decrease sinusoidal pressure to even a lesser extent than end-to-side shunts which remove all of the portal contribution to the sinusoids. In our series, patients with this persistent prograde flow had the worst clinical results and appeared to be a particularly poor risk group.

During our analysis it became clear that irrespective of the type of shunt, the magnitude of the increment in hepatic arterial flow was regulated by some intrinsic capability of the cirrhotic liver to increase its arterial flow. We postulate that such intrinsic capability, or "hidden" variable, is the extent of entrapment of the hepatic arterioles by the fibrous tissues of cirrhosis and propose its evaluation from measurements of the characteristic input impedance of the hepatic arterial bed. Measurements of this type are derived from the instantaneous ratio of pressure to flow in the artery feeding a vascular bed. They have been already performed in the pulmonary arterial bed of dogs and man. We hope to learn to predict the magnitude of the increment in hepatic arterial flow from the value of the impedance measured at operation immediately before construction of the shunt. From this learning we might be able to make reasonable estimations of the impedance at the time of preoperative selective hepatic artery catheterization for angiographic studies.

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Discussion

Dr. John H. C. Ranson (New York, New York): Clinical studies by Dr. Richard Kessler at New York University have also shown a close relationship between the ability of the hepatic artery to maintain total hepatic blood flow following diversion of portal blood, and prognosis after portacaval shunt. Since the adverse effects of diverting portacaval shunts appear to be related in part to a decrease in the total quantity of hepatic blood flow, Mailard, Adamsons, and others have suggested preservation of hepatic blood flow by arterialization of the portal vein stump. There have, however, been difficulties in delivering arterial blood to the portal vein at physiologic pressures.