Paraumbilical Vein Patency in Cirrhosis: Effects on Hepatic Hemodynamics Evaluated by Doppler Sonography

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Doppler sonographic portal vein parameters are used for the noninvasive evaluation of portal hypertension in cirrhosis. The patency of a paraumbilical vein is a rather frequent finding in cirrhosis, which may affect hepatic hemodynamics and function. We evaluated portal and hepatic arterial parameters in 184 cirrhotic patients with and without a patent paraumbilical vein and the relationships with paraumbilical blood flow. A patent paraumbilical vein was found in 33.7% of patients. The prevalence was higher (56.8%) in Child C patients. Portal blood flow velocity (PBV) (10.8 ± 2.2 vs. 9.8 ± 2.4 cm/sec; $P < .01$) and volume (PBF) (995.0 ± 383.8 vs. 811.6 ± 318.7 mL/min; $P < .001$) were significantly higher, and effective portal liver perfusion (PLP) (portal blood flow − paraumbilical blood flow) (621.3 ± 420.8 vs. 811.6 ± 318.7 mL/min; $P < .001$) was significantly lower in patients with a patent paraumbilical vein than in those without. These differences were more evident in Child C patients (10.7 ± 2.0 vs. 8.3 ± 2.3 cm/sec; 935.7 ± 378.3 vs. 680.6 ± 239.4 mL/min; 369.0 ± 282.0 vs. 680.6 ± 239.4 mL/min). Portal vein diameter, the congestion index (CI) of the portal vein, hepatic arterial resistance indexes, and the severity of esophageal varices did not differ between the two groups. In patients with a patent paraumbilical vein, the Child-Pugh score and the prevalence of ascites were significantly higher than in those without. In conclusion, the evaluation of PBV and PBF in cirrhotic patients can provide misleading results if a paraumbilical vein is patent, underestimating the degree of portal hypertension. The CI and hepatic arterial resistance indexes are not influenced by the paraumbilical vein patency. (HEPATOLOGY 1995;22:1689-1694.)

In patients with cirrhosis, portohepatic hemodynamics can be evaluated, noninvasively, by duplex Doppler ultrasonography (DDU). Parameters evaluated by DDU are mainly related to the presence, direction, and velocity of blood flow in the portal vein. Information obtained with this technique is useful both diagnostically[1-4] and prognostically.[5,6] In cirrhosis, portal blood flow velocity is reduced because of the increased hepatic resistance, which is secondary to the anatomical distortion of the intrahepatic portal tree. As a consequence of increased resistance to portal blood flow, collateral vessels open, which allow spontaneous portal-systemic shunting. Paraumbilical veins are the most distal of the possible collateral vessels, and their opening is a frequent finding in cirrhosis,[7-15] which can be evaluated by Doppler sonography.[13-14] Because a patent paraumbilical vein represents an alternative pathway for portal blood, it may conceivably alter portal hemodynamics and, possibly, arterial hemodynamics and liver function. Doppler evaluation of portal hypertension in cirrhosis is based on the measurement of blood flow velocity in the portal vein and on the calculation of parameters related to it, such as portal blood flow volume and congestion index; therefore, the presence of a patent paraumbilical vein may interfere with Doppler evaluation of portal hypertension. And because portosystemic shunt through a patent paraumbilical vein can significantly reduce the amount of portal blood reaching the liver, it may affect liver function by decreasing the amount of portal blood flow effectively perfusing the liver. The aim of this study was to evaluate the effect of the patency of a paraumbilical vein on hepatic hemodynamics, evaluated by Doppler sonography, and liver function in cirrhosis.

PATIENTS AND METHODS

Patients. One hundred eighty-four patients (aged 20 to 78 years; mean ± SD, 55 ± 12) with cirrhosis evaluated consecutively by DDU, in which the portal vein was patent and portal hemodynamics could be studied, were enrolled in the study. Patients treated with beta-blockers or nitrates were excluded because these drugs are known to decrease portal blood velocity.[16,17] No patients were treated with other vasoactive drugs such as angiotensin-converting enzyme inhibitors, calcium antagonists, etc. Eighty-four patients were treated with diuretics (spironolactone ± furosemide). The cause of cirrhosis was alcohol induced in 71 cases, posthepatitic (virus-related) in 89 cases, and mixed posthepatitic and alcohol induced in 24. One hundred patients had no ascites; 84 had ascites. One hundred forty-nine patients had esophageal varices (F1 in 41 cases, F2 in 58 cases, F3 in 50 cases, according to the Japanese classification[18]); 35 patients had no varices. Twenty-six patients had undergone sclerotherapy of esophageal varices and were considered together with patients with F3 varices.

Abbreviations: DDU, duplex Doppler ultrasonography; PUV, paraumbilical vein blood flow mean velocity; PUF, paraumbilical vein blood flow volume; PBV, portal blood flow mean velocity; PBF, portal blood flow volume; PLP, portal liver effusion; CI, congestion index; PI, pulsatility index; RI, resistive index.

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According to the Child-Pugh classification, 59 patients were class A, 81 patients class B, and 44 patients class C. No patient had had recent gastrointestinal hemorrhage.

Informed consent was obtained from each patient, and the study protocol conformed to the 1975 Declaration of Helsinki.

Equipment. A Toshiba Sonolayer SSA-270 with Color Doppler and a 3.75 MHz sector electronic probe was used.

Duplex Doppler Sonography. All subjects were studied in the morning after an overnight fast. The transducer was positioned below the right costal margin or in the right costal spaces. The presence of a patent paraumbilical vein arising from the left branch of the portal vein was verified by color Doppler. The sample volume was positioned inside it halfway along its course through the left lobe of the liver, with a $<60°$ angle between the vessel and the ultrasound beam. The maximum blood flow velocity and the diameter of the paraumbilical vein were then measured during suspended respiration and averaged over a few seconds. Paraumbilical vein blood flow mean velocity (PUV) was calculated as maximum velocity $\times$ 0.57, according to the method of Moriyasu et al applied to the portal vein,[19] and was expressed as cm/sec. Paraumbilical vein blood flow volume (PUF) was calculated as PUV $\times$ cross-sectional area and expressed as mL/min. Each result was the mean of three to five measurements.

The portal vein was visualized longitudinally in B-mode, and the sample volume was positioned inside it immediately after the cross-point between the hepatic artery with the portal trunk, with a $<60°$ angle between the vessel and the ultrasound beam.[20] The maximum portal blood flow velocity and the portal diameter were then measured during suspended respiration and averaged over a few seconds. Portal blood flow mean velocity (PBV) was calculated according to Moriyasu et al[19] as maximum velocity $\times$ 0.57 and expressed as cm/sec. Normal values in our laboratory are 18.7 $\pm$ 3.3 cm/sec.[21]

Portal blood flow volume (PBF) was calculated as PBV $\times$ cross-sectional area, calculated from the diameter and thus assumed to be circular, and expressed as mL/min. Each result was the mean of 3 to 5 measurements. Normal values in our laboratory are 948 $\pm$ 303 mL/min.[21]

PBF really perfusing the liver (effective portal liver perfusion, PLP) was considered as PBF-PUF.

The congestion index of the portal vein (CI) was calculated according to Moriyasu et al[22] as portal vein cross-sectional area (cm^2)/PBV (cm $\times$ s). Normal values in our laboratory are 0.05 $\pm$ 0.02 cm $\times$ s.[21]

Color Doppler allowed the identification of the right and left branches of the hepatic artery. The sample volume of the Doppler system was placed inside these vessels, and the blood flow velocity wave form was recorded. Peak systolic, end diastolic, and temporal mean flow velocity were then determined, and from them the pulsatility index (PI) and the resistive index (RI) were calculated according to the following formulas[23]:

$$PI = (\text{peak systolic} - \text{end diastolic velocity})/\text{mean velocity}$$

$$RI = (\text{peak systolic} - \text{end diastolic velocity})/\text{peak systolic velocity}$$

Indexes obtained from the right and left branches of the hepatic artery were averaged to obtain the final result as recently reported.[21]

Each result was the mean of three to five measurements in each artery. Normal values in our laboratory are 0.89 $\pm$ 0.09 for PI and 0.59 $\pm$ 0.04 for RI.[21]

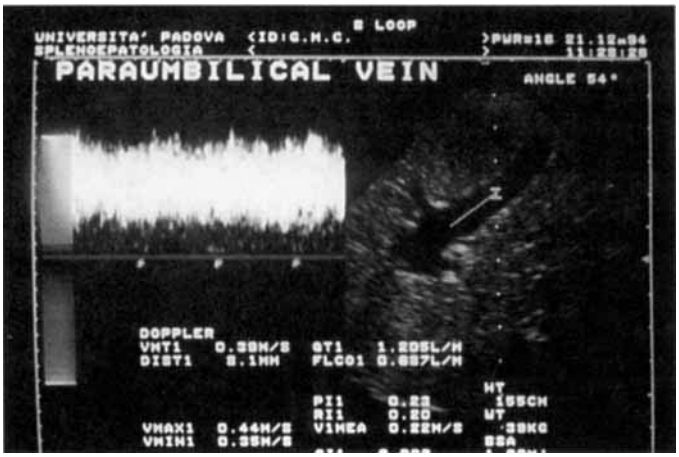

FIG. 1. Duplex Doppler sonographic image of a patent paraumbilical vein. The dots represent the Doppler beam axis. The sample volume (=) is positioned within the vein, and the time-velocity wave form is registered. From mean blood flow velocity (vlmea) and diameter (dist1), blood flow volume (flco1) is calculated.

Statistics. Results were expressed as mean $\pm$ SD, except where otherwise indicated. Differences among groups were evaluated by ANOVA and the Student's *t*-test. Wilcoxon's rank sum test was used to compare data on esophageal varices degree, ascites, and the Child-Pugh score. Linear correlation was performed for the data on PBV, PBF, PUV, PUF, PLP, PI, and RI. Spearman's rank correlation coefficient was used to correlate esophageal varices degree and the Child-Pugh score with the other parameters.

RESULTS

Figure 1 shows a patent paraumbilical vein in which blood flow velocity and volume were measured. The patency of a paraumbilical vein was evidenced in 33.7% of patients. It was present in 26.5% of Child A patients, in 29.6% of Child B patients, and in 56.8% of Child C patients. In subjects with portal hypertension (presence of esophageal varices or ascites)[13] (89% of patients), a patent paraumbilical vein was present in 34.6%. The mean value of the diameter of the paraumbilical vein was 7.2 $\pm$ 2.9 mm, of the PUV 12.2 $\pm$ 5.5 cm/sec, of the PUF 373.7 $\pm$ 358.6 mL/min. PUF was significantly higher in Child C patients (566.6 $\pm$ 426.7 mL/min) compared with Child A (197.4 $\pm$ 197.7 mL/min; $P < .01$) and Child B subjects (268.3 $\pm$ 246.9 mL/min $P < .005$). The main characteristics of the two groups of patients, those with and without a patent paraumbilical vein, are shown in Table 1.

PBV (Fig. 2) and PBF (Fig. 3) were significantly higher in patients with a patent paraumbilical vein compared with those without this feature, whereas PLP (Fig. 4) was significantly lower. The difference in PBV, PBF, and PLP between patients with and without a patent paraumbilical vein was more evident in patients with a more advanced disease, in particular Child C patients (Figs. 2 to 4).

CI and hepatic arterial resistance indexes, which were significantly higher than in our normal subjects,[21]

TABLE 1. Characteristics of Patients Divided According to the Absence (A) or Presence (B) of a Patent Paraumbilical Vein

	A (n = 122)	B (n = 62)
Sex (M/F)	81/41	45/17
Age (yrs)	55.6 ± 12.3	53.6 ± 9.9
MAP	96.0 ± 11.3	95.1 ± 8.9
Child-Pugh score	7.37 ± 1.70	8.63 ± 2.15*
Ascites (yes/no)	46/76 (37.7%)	38/24 (61.3%)†
Esophageal varices (F0/F1/F2/F3)	20/26/40/36 (16.4/21.3/32.8/29.5%)	15/15/18/14 (24.2/24.2/29/22.6%)
Diuretics	54 (44.2%)	36 (58%)
Sclerotherapy	21 (17.2%)	13 (21.0%)
PV diameter (mm)	14.2 ± 1.8	14.7 ± 2.1
PBV (cm/sec)	9.8 ± 2.4	10.8 ± 2.2‡
PBF (mL/min)	811.6 ± 318.7	995.0 ± 383.8*
PLP (mL/min)	811.6 ± 318.7	621.3 ± 420.8*
CI (cm × sec)	0.153 ± 0.060	0.149 ± 0.065
PI	1.31 ± 0.30	1.36 ± 0.29
RI	0.71 ± 0.07	0.72 ± 0.06

Abbreviations: MAP, mean arterial pressure; F0, patients without esophageal varices; F1, patients with F1 varices; F2, patients with F2 varices; F3, patients with F3 varices, according to the Japanese classification[16]; PV, portal vein; PBV, portal blood flow mean velocity; PBF, portal blood flow volume; CI, congestion index of the portal vein; PLP, effective portal liver perfusion; PI, hepatic arterial pulsatility index; RI, hepatic arterial resistive index.

*$P < .001$.

†$P < .05$

‡$P < .01$.

did not differ in patients with and without a patent paraumbilical vein (Table 1). In patients with a patent paraumbilical vein, resistance indexes measured in the left lobe were not significantly different from those measured in the right lobe (RI: right lobe: 0.72 ± 0.07; left lobe: 0.74 ± 0.07; PI: right lobe: 1.33 ± 0.33; left lobe: 1.48 ± 0.35).

The severity of esophageal varices was the same in patients with or without a patent paraumbilical vein (Table 1).

The prevalence of ascites and the Child-Pugh score were significantly higher in patients with a patent paraumbilical vein than in those without one (Table 1); the Child-Pugh score directly correlated with PUF ($r = .36$; $P < .001$) and inversely correlated with PLP ($r = -.37$; $P < .001$).

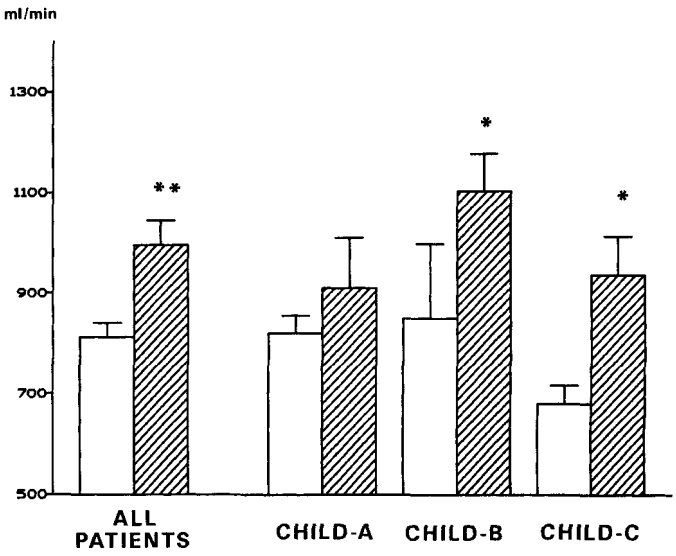

FIG. 3. Portal blood flow in cirrhotic patients with (▨) and without (□) a patent paraumbilical vein. Results are expressed as mean ± SE. **$P < .001$.

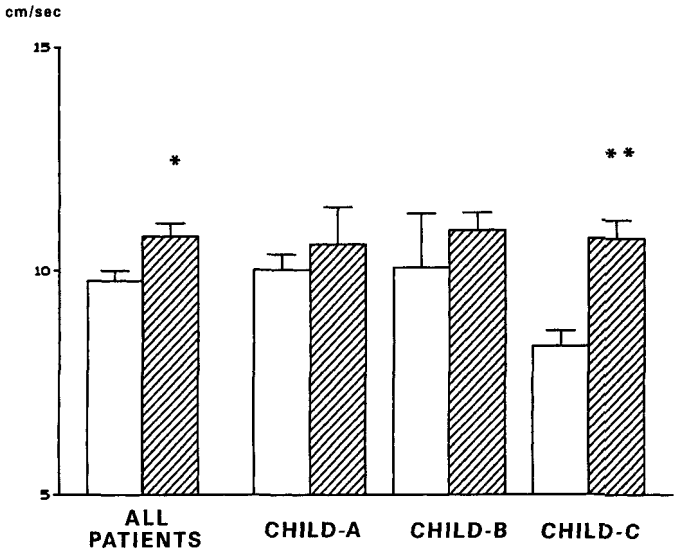

FIG. 2. Portal blood flow mean velocity in cirrhotic patients with (▨) and without (□) a patent paraumbilical vein. Results are expressed as mean ± SE. *$P < .01$; **$P < .001$.

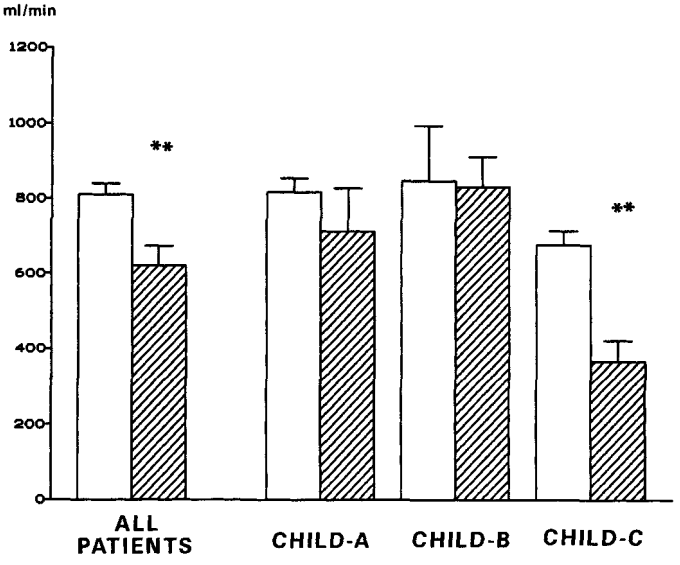

FIG. 4. Effective portal liver perfusion in cirrhotic patients with (▨) and without (□) a patent paraumbilical vein. Results are expressed as mean ± SE. *$P < .01$; **$P < .001$.

DISCUSSION

The results of the current study show that a patent paraumbilical vein, present in 33.7% of our patients with cirrhosis, influences portal but not hepatic arterial hemodynamics, as evaluated by DDU.

Concerning the prevalence of patency of the paraumbilical vein, previous studies including different numbers of patients with cirrhosis gave very different results. With portography, Lafortune et al[12] found a prevalence of 12% in patients with portal hypertension, whereas in Aagaard's study[9] performed in similar patients, the prevalence was 26%. Results obtained by sonography are even more different: the prevalence ranges from 18% (patients with cirrhosis)[14] to 82% (patients with portal hypertension).[13] Our study was performed on cirrhotic patients admitted to the hospital because of a symptomatic disease, undergoing a routine evaluation of portal hypertension by color Doppler. Most of them (89%) had portal hypertension, defined by Gibson et al[13] as "the presence of chronic liver disease and the presence of either non-malignant ascites or endoscopically proved gastroesophageal varices." The results of our study show a 33.7% prevalence of a patent paraumbilical vein among all patients studied, or 34.6% among patients with portal hypertension as previously defined. Thus, our results are in between those of other authors. These differences can be explained mainly by the different techniques used to identify a patent paraumbilical vein: sonography,[7,8,10,11] color Doppler (our study), Doppler sonography[13,14] using different probes with imaging frequencies of 3 to 7.5 MHz.[13] In particular, with regard to this last work, it must also be considered that probably the difference is mainly related to an underestimation of nonrelevant paraumbilical veins in our routine examinations. In fact, Gibson's study was performed just to determine whether the addition of pulsed Doppler imaging to conventional sonography allows discrimination between true paraumbilical veins and the apparent vein sometimes seen in the ligamentum teres in normal subjects. In that study only 51% of patients with portal hypertension had a paraumbilical vein diameter of more than 3 mm, which is considered the smallest diameter diagnostic for portal hypertension,[10] and, among our patients, only one had a paraumbilical vein diameter smaller than 3 mm.

Blood flow through a paraumbilical vein represents a portal systemic shunt, which diverts portal blood from the liver. The relevance of the shunt obviously depends on the amount of PUF. In our patients, PUF averaged 374 mL/min, which is very similar to the value obtained by Mostbeck et al.[14] The range was between 30 and 1,610 mL/min, suggesting that in some cases PUF is irrelevant, whereas in other cases it represents most or all of PBF. The relevance of PUF (in 76% of cases >100 mL/min; in 48% >300 mL/min) explains the significantly higher PBF in such patients. On the contrary, PLP was significantly lower in patients with a patent paraumbilical vein. When considering PBV, this was slightly (+10%) but significantly higher in patients with a patent paraumbilical vein. This difference was more evident (+29%) when considering only patients with a more advanced disease, in particular Child C patients. The decrease of PBV is a well-established Doppler characteristic of cirrhotic portal hypertension.[1-6] This decrease is mainly the consequence of the increased intrahepatic resistance, although PBV is affected also by alterations in splanchnic inflow and the opening of portosystemic shunts. A recent study has demonstrated that PBV has a good sensitivity and specificity in the diagnosis of portal hypertension.[4] Our results point out how PBV is strictly reliable only when a patent paraumbilical vein is excluded, particularly in the advanced phase of the disease when the patency of a paraumbilical vein causes an underestimation of the degree of portal hypertension as evaluated by PBV. The other parameter believed to express the degree of portal hypertension, the CI of the portal vein,[6,22] was exactly the same in patients with and without a patent paraumbilical vein. This finding is very interesting and is explained by the contemporary slight and not significant increase of portal vein diameter and slight and significant increase of PBV in patients with a patent paraumbilical vein. CI has been first introduced by Moriyasu et al[22] as a better parameter of portal hypertension because it takes into account contemporarily two characteristics of this condition: the increase of portal vein diameter and the decrease of PBV. A weak positive correlation has been shown between the CI and portal pressure,[22] whereas a good correlation has been shown between the CI and portal resistance.[21] Furthermore, it has been recently demonstrated that the CI is not only correlated with the degree of esophageal varices, but is also an independent predictor of early esophageal bleeding.[6] Thus, the demonstration that the CI is not influenced by the patency of a paraumbilical vein suggests that it may still be employed as an index of portal hypertension. Two factors could have influenced PBV: treatment with diuretics and sclerotherapy. We did not have the opportunity to evaluate the patients before and after these treatments, and it is not known whether they influence PBV. Nonetheless, when considering the relative number of patients treated with diuretics, this was slightly higher in the group with a patent paraumbilical vein, which had a higher PBV. Thus, because diuretics have been shown to reduce portal pressure through a decrease in portal blood flow,[23,24] they would have reduced, and not increased, PBV in patients with a patent paraumbilical vein. As far as sclerotherapy is concerned, the relative number of patients who had received this treatment was very similar in the two groups, so that portal hemodynamics would have been similarly influenced. The experimental evidence of an increase in hepatic arterial flow when portal venous flow decreases is called the "hepatic arterial buffer response" theory.[25,26] In our patients with a patent paraumbilical vein, the reduction of portal blood volume perfusing the liver efficaciously may be considered, according to the "hepatic arterial buffer response"

theory, a stimulus to increase arterial perfusion by decreasing resistance. Nonetheless, in these patients, hepatic arterial resistance indexes, which can be considered indirect indexes of hepatic arterial perfusion,[27-29] were increased, but not influenced by the patency of a paraumbilical vein. Furthermore, indexes measured in the left lobe, where the effect of the patency of a paraumbilical vein could be expected to be more evident, were even higher, although not significantly, than those measured in the right lobe. We have recently demonstrated that the increase in hepatic arterial resistance indexes in cirrhosis is mainly influenced by the anatomical distortion caused by cirrhosis, parallel to portal resistance, and is correlated with the degree of esophageal varices but not with the Child-Pugh score.[21] Thus, because the patency of the paraumbilical vein is present as an expression of portal hypertension, it is reasonable that it is associated with high hepatic arterial resistance indexes, which do not respond to the reduction of portal perfusion. Because the reduction of effective portal perfusion is not associated with an increase in hepatic arterial perfusion, it might be expected that patients with an extremely reduced liver perfusion have a more impaired liver function. This hypothesis seems to be confirmed by our results that show not only a direct correlation between PUF and the Child-Pugh score, but also an inverse correlation between PLP and the Child-Pugh score. This correlation is also present when considering together both patients with and without a patent paraumbilical vein. Zoli et al[5] have demonstrated that patients with a lower than 650 mL/min PBF have a lower cumulative probability of survival, confirming a relationship between flow and function. Nonetheless, further studies are needed to demonstrate the validity of this assumption, because the severity of cirrhosis, evaluated by the Child-Pugh score, may be responsible for the decrease of liver perfusion and for the opening of more collateral vessels.

It may be hypothesized that the opening of a distal portal-systemic shunt determines an attenuation of portal hypertension, as it reduces resistance to portal blood flow. Nonetheless, the overall effect of the shunt on portal hypertension is only partially evident, as the degree of portal hypertension (diameter of the portal vein and degree of esophageal varices) was not significantly different in patients with and without a patent paraumbilical vein. Thus, it can be hypothesized that the opening of a paraumbilical vein happens as a consequence of severe portal hypertension, which is attenuated, particularly when PUF is relevant, by this feature. These results are concordant with previous data showing that portal pressure is the same in patients with and without a patent paraumbilical vein[9] and that the patency of a paraumbilical vein does not give any protection for the development of esophageal varices.[9]

In conclusion, the evaluation of portal blood flow velocity and of portal blood flow volume by DDU in cirrhotic patients can provide misleading results if a paraumbilical vein is patent, underestimating the degree of portal hypertension. On the contrary, evaluation of the congestion index of the portal vein is not affected by the presence of a patent paraumbilical vein. Hepatic arterial resistance indexes are not affected by the patency of a paraumbilical vein, excluding that an extreme decrease of hepatic portal perfusion influences hepatic arterial resistance. Liver function seems to be related to the effective portal liver perfusion.

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