Doppler Study of Mesenteric, Hepatic, and Portal Circulation in Alcoholic Cirrhosis: Relationship Between Quantitative Doppler Measurements and the Severity of Portal Hypertension and Hepatic Failure

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To determine the relationship between quantitative Doppler parameters of portal, hepatic, and splanchnic circulation and hepatic venous pressure gradient (HVPG), variceal size, and Child–Pugh class in patients with alcoholic cirrhosis, we studied forty patients with proved alcoholic cirrhosis who underwent Doppler ultrasonography, hepatic vein catheterization, and esophagoscopy. The following Doppler parameters were recorded: time-averaged mean blood velocity, volume flow of the main portal vein flow, and resistance index (RI) of the hepatic and of the superior mesenteric artery. Doppler findings were compared with HVPG, presence and size of esophageal varices, and Child–Pugh class. There was a significant inverse correlation between portal velocity and HVPG ($r = -.69$), as well as between portal vein flow and HVPG ($r = -.58$). No correlation was found between RI in the hepatic artery or superior mesenteric artery and HVPG. No correlation was found between portal vein measurements and presence and size of varices. Severe liver failure was associated with lower portal velocity and flow. In patients with alcoholic cirrhosis, only portal vein blood velocity and flow, but neither hepatic nor mesenteric artery RI, are correlated to the severity of portal hypertension and to the severity of liver failure. (HEPATOLOGY 1998;28:932-936.)

The introduction of duplex Doppler ultrasonography has been an important breakthrough in the evaluation of splanchnic hemodynamics. Qualitative evaluation of the portal circulation is being widely used in patients with cirrhosis. Some hemodynamic parameters have been proposed for the assessment of liver cirrhosis. The first parameters studied were focused on the portal vein blood flow.[1-3] More recently, other parameters have been suggested for the study of the hepatic and superior mesenteric arteries, in which blood flow may be altered by liver diseases affecting mesenteric and hepatic arterial resistances.[4-6] It has been shown that portal as well as hepatic and splanchnic arterial Doppler parameters are significantly different in patients with liver disease and healthy subjects. However, few data are available regarding the relationship between these parameters and the severity of portal hypertension or the impairment of liver function in patients with cirrhosis. The present study was therefore designed to investigate the relationship between quantitative Doppler parameters of portal, hepatic, and splanchnic circulation and the clinical, biological, endoscopic, and hemodynamic parameters commonly used to assess the prognosis of patients with alcoholic cirrhosis (i.e., Child–Pugh class, variceal size, and portal pressure),[7-11] and to determine the value of Doppler parameters in predicting the severity of portal hypertension.

PATIENTS AND METHODS

Patients. In a recent 16-month period (from January 1996 to May 1997), 52 patients with known alcoholic liver disease and suspected cirrhosis, who had been referred to our department for transjugular liver biopsy, were prospectively evaluated by duplex Doppler ultrasonography. Liver disease was considered to be alcohol related when alcohol consumption was estimated to be more than 80 g/day for more than 10 years and when no other cause was evident. Liver biopsy established the diagnosis of cirrhosis in 41 patients, of whom 1 patient was excluded because of hepatocarcinoma associated with cirrhosis. The remaining 40 patients constituted the study population. There were 35 men and 5 women, with a mean age of 52 years ± 8 (age range, 34 to 76 years). All patients gave their informed consent to inclusion in the study.

Acording to Pugh's modification of Child's criteria,[7] 13 patients were class A, 9 patients class B, and 18 patients class C. Endoscopy was performed in all patients, and varices were found in 27 (67%). Nine patients had had variceal bleeding, and 4 of these patients had undergone sclerotherapy of varices. At endoscopy, the size of the varices was graded into three groups according to a previously published classification.[8] Thirteen patients had grade 1, 12 patients grade 2, and 2 patients grade 3 varices. Among patients with previous sclerotherapy, varices were grade 2 in 2 patients and grade 3 in 2 patients. Twenty-two patients had ascites, and 13 patients were receiving diuretics.

Portal pressure was determined by the hepatic venous pressure gradient (HVPG), obtained by subtracting the free hepatic venous pressure from the wedged hepatic venous pressure. After an overnight fast, hepatic vein catheterization was performed percutaneously through the jugular vein and pressure was recorded in both the wedged (occluded) and the free positions. Wedging of the

Abbreviations: HVPG, hepatic venous pressure gradient; TAMV, time-averaged mean blood velocity; HA, hepatic artery; RI, resistance index; SMA, superior mesenteric artery.

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catheter was confirmed by contrast media injection after recording pressure. Wedged hepatic vein pressure was recorded on a Statham strain gauge in at least three locations in each patient, usually in the right lobe. The mean of these three different measurements was considered. Hepatic vein catheterization and measurement of hepatic vein pressures were performed by the same operator in all patients.

Duplex Doppler Examinations. All subjects were studied in the morning after an overnight fast. Both Doppler examination and HVPG measurement were performed within the shortest time interval possible (less than 2 hours in most of the cases and never more than 2 days). All Doppler studies were performed with an ATL Ultramark 9 HDI color and pulsed Doppler unit with a 3.5-MHz probe and a low-value high-pass filter. The following hemodynamic parameters were measured on the portal, hepatic, and mesenteric circulation:

1. Time-averaged mean blood velocity (TAMV) (cm/s) of the main portal vein was measured in its mid-portion, where the hepatic artery (HA) crosses the portal vein, on oblique subcostal scans. The axial size of the sample volume was adjusted to encompass the portal vein lumen in its entirety. The angle between the Doppler beam and the long axis of the vessel was made to be less than 6O°. TAMV was automatically calculated on samples of the Doppler signal lasting more than 4 seconds.

2. The diameter of the portal vein (in mm) was measured in longitudinal section, at the exact site of the Doppler sample volume.

3. Portal flow volume (mL/min) was calculated as TAMV × cross-sectional area, assumed to be circular and calculated from the diameter. For each patient, a patent paraumbilical vein arising from the left branch of the portal vein was searched for by color Doppler imaging. In patients with a patent paraumbilical vein, paraumbilical flow volume was measured. Effective blood flow was considered as portal blood flow volume minus paraumbilical flow volume, as reported by Sacerdoti et al.[12] and was used in subsequent statistical analysis. TAMV data of patients with a patent paraumbilical vein were excluded.

4. The HA resistance index (RI) was measured on the proper HA, at its crossing of the portal vein, by using a Doppler sample length of 3 to 9 mm. The peak systolic (S) and peak end-diastolic (D) Doppler frequency shifts were measured manually on the time-frequency Doppler spectrum by calipers, and the RI was automatically calculated as RI = (S − D)/S.

5. The superior mesenteric artery (SMA) RI was calculated on the SMA with a 2- to 7-mm Doppler sample length put about 2 cm downstream of the vessel's origin from the aorta.

Doppler ultrasound parameters were measured in triplicate, and results were averaged. The Doppler investigations were performed by the same well-trained operator, who was blinded to results of laboratory tests, esophagoscopy, and portal pressure.

Statistical Analysis. Results were expressed as means ± SD. Correlations between TAMV, portal flow volume, diameter of the portal vein, HA RI, SMA RI, and HVPG were evaluated by linear regression analysis and correlation coefficient calculation. Analysis of variance was used to compare Doppler ultrasound quantitative parameters and HVPG among the different groups of patients classified according to the esophageal varices grade and the Child–Pugh class. For statistical analysis, patients with grade 3 and 2 varices were mixed because of the small number of patients with grade 3. In addition, patients were divided into hemodynamically severe (HVPG >12 mm Hg) and mild (HVPG ≤12 mm Hg) portal hypertension, and also were separated on the basis of the direction of the portal blood flow. Student's *t* test was used for comparison of quantitative parameters between two groups, and the χ^2 test with Yates' correction for comparison of qualitative variables. Results were considered significant when *P* was <.05.

RESULTS

Table 1 shows the main features of the 40 patients of the study population. Qualitative analysis of the portal flow by Doppler ultrasound showed an hepatofugal flow in 9 patients and a patency of the paraumbilical vein in 4 patients. Thus, measurement of TAMV could be taken into account in only 36 patients.

TABLE 1. Characteristics of the Study Population (n = 40)

Sex (M/F)	35/5
Age (yrs)	52 ± 8
Child–Pugh Class	A: 13
	B: 9
	C: 18
Varices	grade 0: 13
	grade 1: 13
	grade 2: 12
	grade 3: 2
HVPG (mm Hg)	17.6 ± 5.5 (8; 29)
PV diameter (mm)	11.6 ± 5.4 (3.2; 18.1)
TAMV (cm/s)	5.4 ± 7.8 (−12.3; 25.7)
PVF (mL/mn)	325 ± 464 (−501; 1,230)
HA RI	0.66 ± 0.10 (0.43; 0.83)
SMA RI	0.81 ± 0.04 (0.73; 0.88)

NOTE. Results are expressed as mean ± 1 standard deviation (range).
Abbreviations: PV, portal vein; PVF, portal flow volume.

There was a significant correlation between HVPG and TAMV, as well as between HVPG and portal vein flow ($P < .001$; $r = -.69$ and $-.58$, respectively) and in patients with hepatopetal flow ($P < .05$; $r = -.49$ and $-.37$, respectively). Figures 1 and 2 are scatterplots that show the relationship between HVPG and TAMV, and between HVPG and portal vein flow, respectively. There was no significant correlation between HVPG and portal vein diameter ($r = .003$), HA RI ($r = .01$), or SMA RI ($r = .10$). Thirty-one patients had hemodynamically severe portal hypertension and 9 patients had mild portal hypertension. Patients with severe portal hypertension had significantly ($P < .05$) lower TAMV (4.2 vs. 12.5 cm/s) and portal vein flow (244 vs. 606 mL/min) than

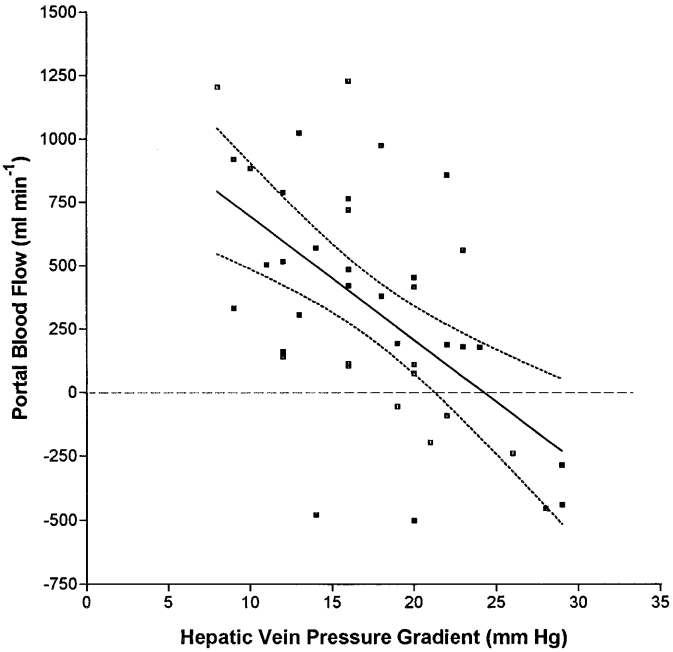

FIG. 1. Relationship between portal blood flow and HVPG (n = 40). There is a significant ($P < .001$) correlation between portal blood flow and HPVG ($r = -.58$).

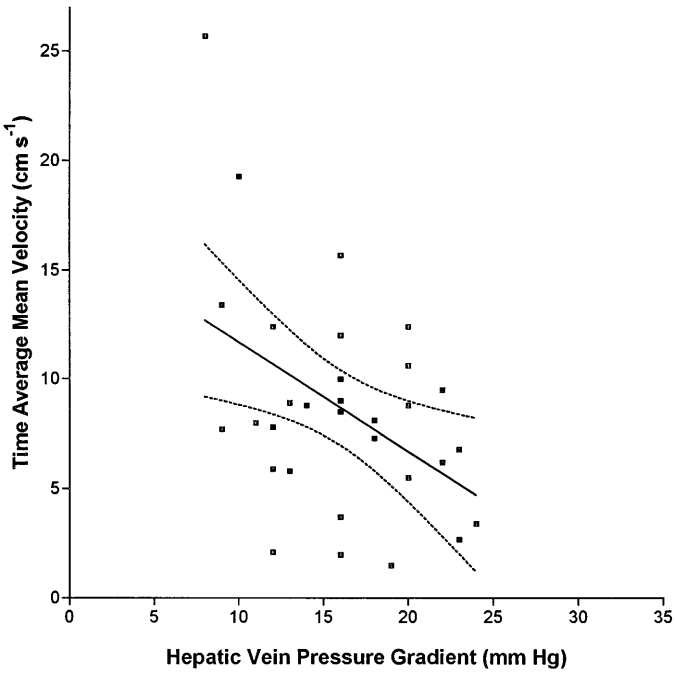

FIG. 2. Relationship between time-averaged mean velocity and HPVG (n = 36). There is a significant ($P < .001$) correlation between portal blood flow and HPVG ($r = -.69$).

patients with mild portal hypertension. There was no statistically significant difference in HA and SMA RIs between patients with severe and patients with mild portal hypertension (Table 2). There was no statistically significant difference in the varices grade between the groups with moderate and with severe portal hypertension, whereas more severe liver failure (Child B or C) was associated with severe portal hypertension.

At endoscopy, esophageal varices were absent in 12 patients, grade 1 in 14 patients, and grade 2 or 3 in 14 patients. There was no significant difference in portal velocity and flow and in SMA RIs between these three groups (Table 3). HA RI was significantly higher in the group with grade 2 or 3 varices than in the groups with grade 0 and grade 1 varices. There was no significant difference in portal pressure according to the varices grade.

TABLE 3. Doppler and Pressure Data According to the Presence and Size of Esophageal Varices

	Patients Without Esophageal Varices (n = 12)	Patients With Esophageal Varices Grade 1 (n = 14)	Patients With Esophageal Varices Grade 2 or 3 (n = 14)	Statistical Significance
TAMV (cm/s)	4.7 ± 9.8 (n = 11)	5.2 ± 7.1	8.2 ± 6.9	NS
PVF (mL/min)	253 ± 564 (n = 12)	334 ± 425 (n = 14)	379 ± 435 (n = 14)	NS
HA RI	0.61 ± 0.10 (n = 12)	0.65 ± 0.09 (n = 14)	0.72 ± 0.07 (n = 14)	$P < .05$
SMA RI	0.81 ± 0.03 (n = 12)	0.81 ± 0.04 (n = 14)	0.82 ± 0.05 (n = 14)	NS
HVPG	16.1 ± 6.1 (n = 12)	17.5 ± 5.1 (n = 14)	18.9 ± 5.5 (n = 14)	NS

NOTE. Results are expressed as mean ± 1 standard deviation. HA RI in patients with grade 2 or 3 varices is significantly superior to HA RI in patients with grade 0 varices.

Table 4 shows Doppler and pressure parameters according to Child–Pugh classification. Less severe liver disease (Child A) was associated with significantly lower TAMV, lower portal vein flow values, and lower HVPG when compared with Child B or C liver disease. Among the class B or C patients, 46% (11/24) had a TAMV of less than 2 cm/s and 48% (13/27) had portal vein flow of less than 150 mL/min, whereas none of the 13 class A patients had such values. Other Doppler parameters were not correlated with the severity of liver disease.

Table 5 shows data concerning arterial Doppler parameters, portal pressure, varices grade, and Child class of patients divided on the basis of portal flow direction. HA and SMA RIs did not significantly differ in relation to the direction of portal flow. The portal pressure and the prevalence of severe liver failure (Child B or C) were significantly higher in patients with hepatofugal flow. There was no correlation between the presence and grade of the varices and the direction of the portal flow.

DISCUSSION

Several Doppler studies have shown the value of quantitative measurements in differentiating cirrhotic patients from

TABLE 2. Doppler Parameters, Varices and Child Data According to the Severity of Portal Hypertension

	Patients With Severe Portal Hypertension (HVPG >12 mm Hg)	Patients With Mild Portal Hypertension (HVPG ≤12 mm Hg)	Statistical Significance
TAMV (cm/s)	4.2 ± 7.4 (n = 28)	12.5 ± 6.8 (n = 8)	$P < .05$
PVF (mL/mn)	244 ± 462 (n = 31)	606 ± 367 (n = 9)	$P < .05$
HA RI	.66 ± .10 (n = 31)	.67 ± .09 (n = 9)	NS
SMA RI	.81 ± .04 (n = 31)	.82 ± .03 (n = 9)	NS
Varices (0/1/2-3)	(8/11/12)	(4/3/2)	NS
Child (A/B/C)	(5/9/17)	(8/0/1)	$P < .05$

NOTE. Results are expressed as mean ± 1 standard deviation. TAMV and PVF were significantly higher in patients with mild portal hypertension. Less severe liver failure was associated with mild portal hypertension.

TABLE 4. Doppler and Pressure Data According to the Severity of Liver Failure Assessed by Child–Pugh's Classification

	Patients From Child–Pugh's Class A	Patients From Child–Pugh's Class B	Patients From Child–Pugh's Class C	Statistical Significance
TAMV (cm/s)	10.8 ± 6 (n = 12)	3.6 ± 5.8 (n = 7)	2.4 ± 8.2 (n = 17)	$P < .05$
PVF (mL/min)	685 ± 296 (n = 13)	177 ± 369 (n = 9)	140 ± 472 (n = 18)	$P < .05$
HA RI	.68 ± .09 (n = 13)	.69 ± .08 (n = 9)	.64 ± .11 (n = 18)	NS
SMA RI	.82 ± .04 (n = 13)	.82 ± .04 (n = 9)	.81 ± .03 (n = 18)	NS
HVPG (mm Hg)	13 ± 4 (n = 13)	21.6 ± 3.7 (n = 9)	18.9 ± 5.1 (n = 18)	$P < .05$

NOTE. Results are expressed as mean ± 1 standard deviation. Patients with Child A disease has significantly higher portal velocity and flow, and lower portal pressure than patients with Child B or C disease.

TABLE 5. Doppler Parameters, Varices, and Child Data According to the Direction of Portal Flow

	Patients With Hepatopetal Flow (n = 31)	Patients With Hepatofugal Flow (n = 9)	Statistical Significance
HA RI	.67 ± .10	.63 ± .08	NS
SMA RI	.81 ± .04	.81 ± .04	NS
HVPG	16 ± 4.6	23.1 ± 5.2	$P < .05$
Child (A/B/C)	(13/6/12)	(0/3/6)	$P < .05$
Varices (0/1/2-3)	(8/11/12)	(4/3/2)	NS

NOTE. Results are expressed as mean ± 1 standard deviation. The portal pressure and the rate of severe liver disease (Child B or C) was significantly higher in patients with hepatofugal flow.

healthy subjects. A significant decrease in portal vein velocity and flow[13-16] and a significant increase in HA RI[5,6] have been consistently reported in cirrhotic patients compared with healthy subjects. However, because there is some overlap of data between these two groups, such measurements are not currently used for the diagnosis of portal hypertension in clinical practice. To our knowledge, only a few studies[6,17] have focused on the value of quantitative Doppler analysis to predict the severity of portal hypertension. The present study was mainly designed to compare Doppler ultrasound parameters with hemodynamic and endoscopic parameters widely used to assess the severity of portal hypertension in patients with cirrhosis.

It has been well established that the gradient between wedged and free hepatic venous pressures closely reflects the portal pressure in patients with alcoholic cirrhosis. In contrast, this gradient underestimates portal pressure in patients with nonalcoholic cirrhosis,[18] which is why we did not include such patients in our study population. Furthermore, because the cause of cirrhosis may influence the hemodynamic pattern,[16] the inclusion of a homogenous group of patients with solely alcoholic cirrhosis may facilitate the analysis of hemodynamic Doppler parameters. We used the HVPG value of 12 mm Hg to differentiate severe from mild portal hypertension because this is considered to be the threshold level for risk of variceal hemorrhage.[19] Moreover, the aim of the pharmacological treatment of portal hypertension is to reduce the portal pressure to 12 mm Hg or less.[20] We correlated Doppler parameters with the Child–Pugh class and with the size of esophageal varices, which are the main prognostic factors in patients with portal hypertension caused by alcoholic cirrhosis.

We found a close inverse correlation between HVPG and portal vein velocity ($r = -.63$) and flow ($r = -.58$). Our results seem more encouraging than those reported by Moriyasu et al.[17] about the use of portal velocity and flow measurements in predicting the severity of portal hypertension. In patients with cirrhosis, Moriyasu et al. did not show any correlation between HVPG and portal vein flow, and showed only a weak correlation ($r = .20$) between HVPG and congestion index. However in Moriyasu et al.'s study,[17] mean velocity of the portal vein was not directly obtained from the Doppler spectrum but was derived from maximal velocity using a standard coefficient, and the paraumbilical vein flow was not sought or subtracted from the portal vein flow. The discrepancy between their study and ours may also be related to differences in patient populations. Moriyasu et al.'s study included patients with various causes of cirrhosis and only patients with hepatopetal flow; in contrast, our study included only patients with alcoholic cirrhosis, and 22.5% of them had reversed flow in the portal vein. Therefore, our cirrhotic patients probably had more severe liver disease than Moriyasu's patients. The design of our study, which included patients referred for transjugular biopsy, explains the severity of the liver disease in our patients and the high prevalence of reversed portal flow in our study. Transjugular access is the method of choice to obtain liver samples in patients with severe liver failure in order to decrease the risk of bleeding. The prevalence of reversed portal flow is variable, from 3.1% to 22% in the literature.[21,22] Discrepancy is likely caused by the different selection of patient populations. Studies that included a high percentage of patients with severe liver failure,[22] as ours, also show high rate of hepatofugal flow.

It is not surprising that we did not find any correlation between portal vein flow or HVPG and esophageal varices size. Previous hemodynamic studies have shown the absence of correlation between the degree of portal hypertension and the development of esophageal varices in patients with HVPG greater than 12 mm Hg.[9,20] In the same way, it has been shown that there is no relationship between the presence of a reversed flow in the portal vein and the presence and size of esophageal varices.[21] The lack of relationship between portal hemodynamic parameters and development of esophageal varices could mean that, unlike some other spontaneous or surgical portosystemic anastomoses, gastroesophageal collaterals are unable to drain an amount of blood sufficient to significantly lower the portal flow.

When considering liver function failure, we observed that patients with severe liver disease (class B or C) had lower portal velocity and flow. This supports previous reports,[16,23] which show an inverse correlation between portal vein velocity and flow and the Child–Pugh score.

Some recent studies concern the Doppler assessment of hepatic arterial hemodynamics in patients with cirrhosis.[5,6,24] Most studies have shown that HA RI is increased in cirrhotic patients.[5,6] We did not find any correlation between HA RI and portal hemodynamic parameters. HA RI value in cirrhosis results from two mechanisms with opposite effects. On one hand, distortion of hepatic lobules and reduction of vascular spaces caused by cirrhosis increase HA resistance. On the other hand, the HA buffer response mechanism[25] may decrease HA resistance in response to a decrease in portal liver perfusion to maintain total blood flow.[26] This may explain why HA RI yielded no predictive value of the severity of portal hypertension in patients with alcoholic cirrhosis. In the same way, despite the buffer arterial response mechanism, there was no significant decrease of the HA in patients with hepatofugal flow, likely because more severe portal hypertension present in these patients results in increased hepatic arterial resistances.

An increase in mesenteric arterial flow resulting in an increased portal venous inflow is classically described in advanced stages of cirrhosis.[27] Thus, a decrease of SMA RI may be expected in those stages. However, we did not find any correlation between the SMA RI and the severity of the portal hypertension. We believe that the SMA RI does not reflect only the circulatory resistance in the mesenteric arterial and capillary bed, but more likely the sum of downstream resistances, including the mesenteric arterial and capillary bed as well as mesenteric and portal venous and hepatic vascular resistances. The same mechanism has been

hypothesized to explain that splenic artery RI did not decrease in cirrhosis despite the increase in splenic blood flow caused by splenic enlargement.[28]

In conclusion, our study demonstrates that in patients with alcoholic cirrhosis, only portal vein blood velocity and flow, but neither HA nor SMA RI, exhibit a significant correlation with HVPG and with the severity of liver failure. However, because of the scattering of the data, the quantitative measurement of portal velocity or flow has a limited utility in predicting either hepatic venous gradient or severity of liver failure in an individual patient. The prognostic value of our data regarding the clinical outcome in alcoholic cirrhosis has yet to be established. Further studies, including correlation with long-term clinical follow-up, are needed to compare the prognostic values of Doppler measurements, portal pressure, variceal size, and Child–Pugh score.

REFERENCES

1. Burns P, Taylor K, Blei AT. Doppler flowmetry and portal hypertension. Gastroenterology 1987;92:824-826.
2. Ohnishi K, Saito M, Koen H, Nakayama T, Nomura F, Okuda K. Pulsed doppler flow a criterion of portal venous velocity: comparison with cineangiographic measurements. Radiology 1985;154:495-498.
3. Lomas DJ, Britton PD, Summerton CB, Seymour CA. Duplex doppler measurements of the portal vein in portal hypertension. Clin Radiol 1993;48:311-315.
4. Sabba C, Ferraioli G, Genecin P, Colombato L, Buonamico P, Lerner E, Taylor KJW, et al. Evaluation of postprandial hyperemia in superior mesenteric artery and portal vein in healthy and cirrhotic humans: an operator-blind echodoppler study. HEPATOLOGY 1991;13:714-718.
5. Joynt LK, Platt JF, Rubin JM, Ellis JH, Bude RO. Hepatic artery resistance before and after standard meal in subjects with diseased and healthy livers. Radiology 1995;196:489-492.
6. Sacerdoti D, Merkel C, Bolognesi M, Amodio P, Angeli P, Gatta A. Hepatic arterial resistance in cirrhosis with and without portal vein thrombosis: relationships with portal hemodynamics. Gastroenterology 1995;108: 1152-1158.
7. Pugh RN, Murray-Lyon IM, Dawson JL, Pietroni MC, Williams R. Transection of the oesophagus for bleeding oesophageal varices. Br J Surg 1973;60:646-649.
8. Vinel JP, Schuller P, Herrieu C, Cales P, Cassigneul J, Pascal JP. Devenir des malades après éradication des varices oesophagiennes par sclérose endoscopique: résultats d'une étude prospective. Gastroenterol Clin Biol 1986;10:584-588.
9. Lebrec D, De Fleury P, Rueff B, Nahum H, Benhamou JP. Portal hypertension, size of esophageal varices and risk of gastrointestinal bleeding in alcoholic cirrhosis. Gastroenterology 1980;79:1139-1144.
10. Merkel C, Bolognesi M, Bellon S, Zuin R, Noventa F, Finucci G, Sacerdoti, et al. Prognostic usefulness of hepatic vein catheterization in patients with cirrhosis and esophageal varices. Gastroenterology 1992; 102:973-979.
11. Vorobioff J, Groszmann RJ, Picabea E, Gamen M, Villavicencio R, Bordato J, Morel I, et al. Prognostic value of hepatic venous pressure gradient measurements in alcoholic cirrhosis: a 10-year prospective study. Gastroenterology 1996;111:701-709.
12. Sacerdoti D, Bolognesi M, Bombonato G, Gatta A. Paraumbilical vein patency in cirrhosis: effects on hepatic hemodynamics evaluated by doppler sonography. HEPATOLOGY 1995;22:1689-1694.
13. Zoli M, Marchesini G, Cordiani MR, Pisi P, Brunori P, Trono A, Pisi E. Echo-doppler measurement of splanchnic blood flow in control and cirrhotic subjects. J Clin Ultrasound 1986;14:429-435.
14. Ohnishi K, Sato S, Pugliese D, Tsunoda T, Saito M, Okuda K. Changes of splanchnic circulation with progression of chronic liver disease studied by echo-doppler flowmetry. Am J Gastroenterol 1987;82:507-511.
15. Gaiani S, Bolondi L, LiBassi S, Santi V, Zironi G, Barbara L. Effect of meal on portal hemodynamics in healthy humans and in patients with chronic liver disease. HEPATOLOGY 1989;9:815-819.
16. Zironi G, Gaiani S, Fenyves D, Rigamonti A, Bolondi L, Barbara L. Value of measurement of mean portal flow velocity by doppler flowmetry in the diagnosis of portal hypertension. HEPATOLOGY 1992;16:298-303.
17. Moriyasu F, Nishida O, Ban N, Nakamura T, Sakai M, Miyake T, Uchino H. Congestion index of the portal vein. AJR Am J Roentgenol 1986;146: 735-739.
18. Pomier-Layrargues G, Kusielewicz D, Willems B, Villenueve JP, Marleau D, Côte J, Huet JM. Presinusoidal portal hypertension in non-alcoholic cirrhosis. HEPATOLOGY 1985;5:415-418.
19. Garcia-Tsao G, Groszmann RJ, Fisher RL, Conn HO, Atterbury C, Glickman M. Portal pressure, presence of gastroesophageal varices and variceal bleeding. HEPATOLOGY 1985;5:419-424.
20. Groszmann RJ, Bosch J, Grace N et al. Hemodynamic events in a prospective randomized trial of propranolol versus placebo in the prevention of a first variceal hemorrhage. Gastroenterology 1990;99:1401-1407.
21. Gaiani S, Bolondi L, Li Bassi S, Zironi G, Siringo S, Barbara L. Prevalence of spontaneous hepatofugal portal flow in liver cirrhosis. Gastroenterology 1991;100:160-167.
22. Letard JC, Boustieres C, Romy P, et al. Inversion du débit sanguin portal au cours de la cirrhose. Gastroenterol Clin Biol 1993;17:578-583.
23. Ljubicic N, Duvnjak M, Rotkvic I, Kopjar B. Influence of the degree of liver failure on portal blood flow in patients with liver cirrhosis. Scand J Gastroenterol 1990;25:395-400.
24. Vassiliades VG, Ostrow TD, Chezmar JL, Hertzler GL, Nelson RC. Hepatic arterial resistive indices: correlation with the severity of cirrhosis. Abdom Imaging 1993;18:61-65.
25. Lautt WW, Greenway CV. Conceptual review of the hepatic vascular bed. HEPATOLOGY 1987;7:952-963.
26. Platt JF, Rubin JM, Ellis JH. Hepatic artery changes in portal vein thrombosis. Radiology 1995;196:95-98.
27. Vorobioff J, Bredfeldt JE, Groszmann RJ. Hyperdynamic circulation in portal hypertensive rat model: a primary factor for maintenance of chronic portal hypertension. Am J Physiol 1983;244:52-57.
28. Bolognesi M, Sacerdoti D, Merkel C et al. Splenic doppler impedance indices: influence of different portal hemodynamic conditions. HEPATOLOGY 1996,23:1035-1040.