0270-9139/89/0906-0815$02.00/0
HEPATOLOGY

Vol. 9, No. 6, pp. 815-819, 1989
Printed in U.S.A.

Effect of Meal on Portal Hemodynamics in Healthy Humans and in Patients with Chronic Liver Disease

STEFANO GAIANI, LUIGI BOLONDI, SILVIA LI BASSI, VITTORIO SANTI, GIANNI ZIRONI AND LUIGI BARBARA
Istituto di Clinica Medica e Gastroenterologia, Università di Bologna, 40138 Bologna, Italy

The effect of a standard Italian meal on portal hemodynamics was evaluated in 12 normal subjects, in 11 patients with chronic active hepatitis and in 11 patients with liver cirrhosis using duplex Doppler ultrasound, which allows a noninvasive assessment of portal blood flow. In the fasting state, the portal vein caliber was significantly higher in patients with liver cirrhosis than in normal subjects and patients with chronic active hepatitis, whereas the mean flow velocity in the portal vein was significantly lower in this group. Basal flow volume of the portal vein was greater in patients with liver cirrhosis than in normal subjects and patients with chronic active hepatitis. Sixty minutes after the standard meal, we observed both in normal subjects and in patients with chronic active hepatitis a significant increase of mean caliber, mean velocity and flow volume in the portal vein, whereas in patients with liver cirrhosis, these parameters remained almost unchanged. In addition, the examination of individual patterns showed that flow velocity and flow volume in the portal vein decreased in some cirrhotic patients after the meal. This behavior is probably related to the hypertensive state in the splanchnic venous bed and diversion of splanchnic blood flow into spontaneous portosystemic collaterals.

Splanchnic vasodilation and hyperemia occur following intake of food in animals (1) and healthy humans (2), owing to the interaction of intrinsic (change in arteriolar transmural pressure and/or increase in vasodilator tissue metabolites) or extrinsic mechanisms (autonomic nervous system) (3–5) and the effect of gastrointestinal hormones, namely gastrin, cholecystokinin and glucagon, as demonstrated by experimental studies (6, 7). Our knowledge of the mechanisms involved in splanchnic postprandial hemodynamics is derived mainly from experimental investigations in animals, whereas measurement of splanchnic venous flow in humans has been limited by the fact that it required, until few years ago, complex and invasive techniques such as electromagnetic flowmetry (8, 9) or cineangiography (10), and noninvasive methods were not feasible. The development of ultrasound pulsed Doppler technology allows measurement of flow velocity in deep abdominal vessels (11, 12) and, by simultaneously measuring cross-sectional area of the vessel, it provides quantitative information about blood flow volume (13). Several reports have recently demonstrated the reliability of this method in evaluating the portal venous system in normal subjects and patients with portal hypertension (14–18), even though some limitations and possible source of errors have been stressed (13, 19, 20; Burns PN, Blei AT. Gastroenterology 1988; 95:260–261, Correspondence).

Using a duplex Doppler device, we investigated changes induced by a standard meal on portal flow in normal subjects and in patients affected by chronic liver disease, with the aim to detect differences of the hemodynamic response associated with the presence of portal hypertension.

PATIENTS AND METHODS

Twelve healthy subjects (NS) (mean age ± S.D. = 48.5 ± 7.8 years; seven males and five females), 11 patients with chronic active hepatitis (CAH) (mean age = 42.5 ± 9.8 years; five males and six females) and 11 patients with liver cirrhosis (LC) (three alcoholic cirrhosis and eight of viral origin; mean age = 43.8 ± 7.6 years; six males and five females) were submitted to the study. The diagnosis of CAH and LC was made on the basis of liver biopsy. Esophageal varices at endoscopy (21) and some of the ultrasonographic signs of portal hypertension (dilated portal vein, portosystemic collaterals, splenomegaly with dilated splenic radicles, diminished response to deep respiration in splenic vein and superior mesenteric vein, ascites) (22–27) were demonstrated in all patients with LC and were absent in patients with CAH. None of them had ascites. Informed consent was obtained from each patient.

We utilized real-time ultrasound equipment with a 3.5-MHz linear array transducer (Ansaldo-Hitachi AUC 940) provided by pulsed Doppler device operating at a frequency of 3.5 MHz. The study was performed in each case after an overnight fast and 60 min after the end of a standard Italian meal of 1,100 kilocalories (proteins 18%, lipids 39%, carbohydrates 43%). In three NS and in three LC, the duplex Doppler study was also repeated 90 and 120 min after the meal.

In each examination, the following parameters were evaluated: (i) caliber of the portal vein; this is measured, during suspended respiration, as the mean value between the anteroposterior and the transverse diameter of the vessel at its largest point (in mm); (ii) mean velocity (V_{mean}) of the portal venous flow (in cm per sec), and (iii) volume (F) of portal venous flow (in ml per min).

The flow volume was also expressed in relation to the weight (ml per min per kg), in order to avoid eventual influence of the size of patients.

Received March 28, 1988; accepted November 18, 1988.
Address reprint requests to: Luigi Bolondi, M.D., Istituto di Clinica Medica e Gastroenterologia, Università di Bologna, Policlinico S. Orsola, via Massarenti 9, 40138 Bologna, Italy.

The portal vein was first ultrasonographically identified with longitudinal scans parallel to the axis of the vessel, and then the sample volume was positioned into the vein. Our equipment is able to display the angle between the ultrasonic beam and the longitudinal axis of the vessel and we choose an angle lower than 60 degrees, because large angles affect the accuracy of flow velocity calculation. Flow velocity is directly calculated by the equipment from the Doppler spectral analysis, utilizing the formula:

$$V = Fd \cdot C/2Fo \cdot \cos \alpha$$

as described by Gill (13), where F_d is the Doppler frequency shift, C is the velocity of ultrasound in the tissues (1,500 m per sec), F_o is the emitted ultrasonic wave frequency and α is the angle of incidence between the ultrasonic beam and the longitudinal axis of the vessel.

In this study, we considered the "mean velocity of flow" (V_{mean}) in the portal vein. Utilizing a sample volume whose size nearly corresponds to the vessel diameter, it is possible to obtain the "even insonation" (19). In this way, V_{mean} can be determined directly on Doppler spectral analysis without making assumptions regarding the spatial flow profile in the vessel. In a group of five NS, we measured portal flow velocity both directly calculating V_{mean} from Doppler spectral analysis and measuring V_{mean} by the conversion of maximal flow velocity (14).

An overestimation of the actual mean velocity by this method may, however, result, owing to the partial loss of peripheral slow components of the flow by the sample volume (which can be modified by the operator only regarding its longitudinal axis), and to the low-cut filter. These errors are still constant in the observations before and after a meal and they can reduce the accuracy in evaluating the actual mean velocity, but the differences found between the fasting and fed states are not significantly affected.

V_{mean} measurement was made on Doppler traces of 4 to 6 sec in order to avoid eventual flow fluctuations, which are also reduced by examining the patients during suspended respiration. In all patients, the velocity of portal flow was calculated as the mean value of three consecutive examinations. Volume of the portal flow (F) was obtained by the formula:

$$F = V_{mean}\, \pi r^2$$

where r represents the half-diameter of the portal vein.

In order to assess the intra- and interobserver variation of portal flow measurement by pulsed Doppler, five subjects were examined three times each by two different operators (L. B. and S. G.); values obtained showed an overall intra- and interobserver variation lower than 10%.

Statistical Analysis. Portal vein caliber, V_{mean} and F are presented as means ± S.D. Increases of portal vein caliber, V_{mean} and flow volumes were also expressed, in each group, in terms of maximal percentage variation over basal values and were compared among the three groups of patients (NS, CAH, LC).

Basal values and percentage increases after a meal in the three groups were compared by one-way analysis of variance (ANOVA) and by the nonparametric Kruskal-Wallis test when necessary (28). Student's t test for nonpaired values was used to compare portal vein caliber, V_{mean} and F separately in the different groups (NS, CAH, LC). Student's t test for paired values was utilized to compare portal vein caliber, V_{mean} and F in each group in basal conditions and after a meal.

RESULTS

Caliber of the Portal Vein. Portal vein calibers (mean ± S.D.) before and after the standard meal are reported in Table 1. In the fasting state, the mean caliber was similar in NS (10.5 ± 1.5 mm) and in CAH (10.5 ± 2.1 mm), whereas in patients with LC it was significantly higher (14 ± 2.4 mm) ($p < 0.005$; ANOVA: $p < 0.0005$). Sixty minutes after the standard meal, the caliber of the portal vein significantly increased in NS (11.9 ± 1.7 mm) ($p < 0.0005$) and in CAH (11.5 ± 1.5 mm) ($p < 0.025$), corresponding to an increase of 14 and 11.4%, respectively. In patients with LC, the increase was very slight and not significant (14.3 ± 2.1 mm). ANOVA showed a statistical difference of the caliber increase in the three groups ($p < 0.025$).

Individual patterns (Fig. 1) show that the caliber increased in 10 of 12 NS (83%), whereas dilatation of the portal vein was evident in six of 11 CAH (54%) and in only three of 11 patients with LC (27%).

Velocity of the Portal Flow. Mean values of flow velocity in the portal vein before and after the standard meal are reported in Table 2. In patients with LC, the velocity of portal blood flow (12.4 ± 2.3 cm per sec) was significantly lower than in NS (16 ± 4.1 cm per sec) ($p < 0.005$) and in patients with CAH (17.3 ± 4.1 cm per sec) ($p < 0.005$) (ANOVA: $p < 0.005$). The standard meal induced a significant increase in portal flow velocity both in NS (19.7 ± 4.8 cm per sec) (+24%) ($p < 0.025$) and in CAH (20.4 ± 4.9 cm per sec) (+18.2%) ($p < 0.05$). ANOVA showed a statistical difference of velocity increase in the three groups ($p < 0.025$). The examination of individual patterns (Fig. 2) demonstrates that the velocity of portal flow increased in all patients but one, both in NS (Fig. 3A) and in CAH. Flow velocity remained almost unchanged in LC (12.7 ± 3.1 cm per sec). Among these patients, the velocity of portal flow did not actually change in one, whereas it slightly increased in six and in the remaining four the meal induced a decrease of portal flow velocity (Fig. 3B), up to 30%.

TABLE 1. Effect of standard meal on portal vein caliber

	Before meal	After meal	% increase
NS (n = 12)	10.5 ± 1.5[a,c]	11.9 ± 1.7[c]	+14
CAH (n = 11)	10.5 ± 2.1[b,d]	11.5 ± 1.5[d]	+11.4
LC (n = 11)	14.0 ± 2.4[a,b,e]	14.3 ± 2.1[e]	+3

The values are expressed in mm (means ± S.D.) and as mean percentage increase. *a* to *e* indicate levels of significance by Student's t test between values with the same letter: *a* = $p < 0.0005$; *b*, *c* = $p < 0.005$; *d* = $p < 0.025$; *e* = not significant. Analysis of variance between groups before meal: $p < 0.0005$; between percentages of increase: $p < 0.0005$.

TABLE 2. Effect of a standard meal on blood flow velocity in the portal vein

	Before meal	After meal	% increase
NS (n = 12)	16 ± 4.1[a,c]	19.7 ± 4.8[c]	+24
CAH (n = 11)	17.3 ± 4.1[b,d]	20.4 ± 4.9[d]	+18.2
LC (n = 11)	12.4 ± 2.3[a,b,e]	12.7 ± 3.1[e]	+3.2

The values are expressed in cm per sec (means ± S.D.) and as mean percentage increase. *a* to *e* indicate levels of significance by Student's t test between values with the same letter: *a*, *b* = $p < 0.005$; *c* = $p < 0.025$; *d* = $p < 0.05$; *e* = not significant. Analysis of variance between groups before meal: $p < 0.0005$; between percentages of increase: $p < 0.025$.

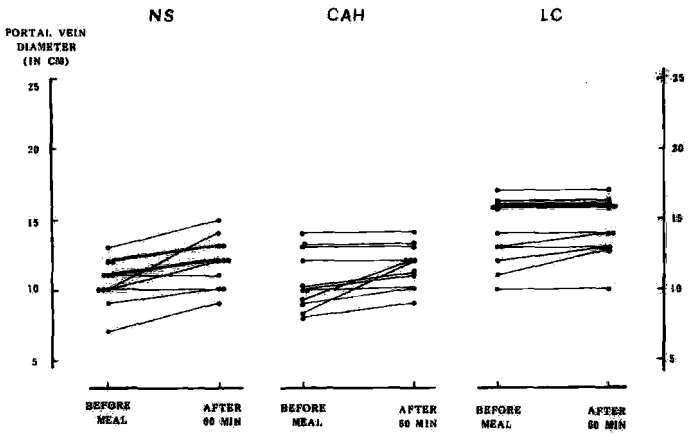

FIG. 1. Caliber of the portal vein before and 60 min after a standard meal in normal subjects (NS) and in patients with chronic active hepatitis (CAH) and with liver cirrhosis (LC). The caliber increased in 10 of 12 NS (83%) and in six of 11 CAH (54%). In patients with LC, in whom basal caliber (means ± S.D.) was significantly greater (14 ± 2.4 mm) than in NS (10.5 ± 1.5 mm) ($p < 0.005$) and in CAH (10.5 ± 2.1 mm) ($p < 0.005$), caliber increased only in three of 11 (27%).

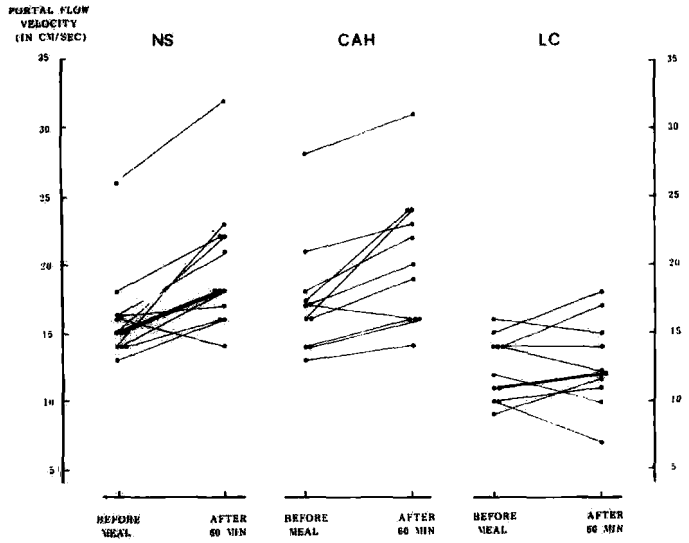

FIG. 2. Flow velocity in the portal vein before and 60 min after a standard meal in normal subjects (NS) and in patients with chronic active hepatitis (CAH) and with liver cirrhosis (LC). Flow velocity increased in all patients but one in NS and CAH groups. In the 11 patients with LC, in whom basal velocity was significantly lower (12.4 ± 2.3 cm per sec) than in NS (16 ± 4.1 cm per sec; $p < 0.005$) and in CAH (17.3 ± 4.1 cm per sec; $p < 0.005$), it slightly increased in six, remained unchanged in one and decreased after the meal in four cases.

Flow Volume of the Portal Vein. Mean values of portal vein flow volume are reported in Table 3. In basal conditions, flow volume was not different in NS (832 ± 245 ml per min; 12.5 ± 4.1 ml per min per kg) and in CAH (891 ± 293 ml per min; 13.0 ± 3.9 ml per min per kg), whereas in patients with LC it was greater (1,160 ± 426 ml per min; 17.7 ± 7.9 ml per min per kg), with a poor statistical significance (ANOVA: $p = 0.054$ and $p = 0.07$). After the standard meal portal flow volume increased in NS by 59% (1,312 ± 433 ml per min; 19.5 ± 5.8 ml per min per kg) ($p < 0.0005$ for both parameters) and by 49% in patients with CAH (1,261 ± 302 ml per min; 18.4 ± 3.9 ml per min per kg) ($p < 0.0005$ for both parameters), whereas in LC it did not significantly change (+8.5%) (1,262 ± 480 ml per min; 19.2 ± 8.7 ml

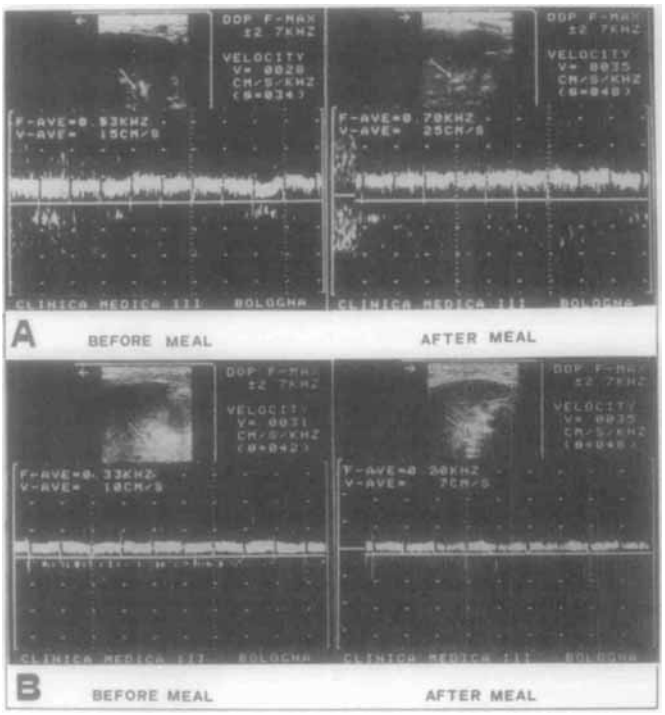

FIG. 3. Duplex Doppler sonography of the portal vein before and after a standard meal in a normal subject (A) and in a patient with liver cirrhosis (B). Right subcostal longitudinal scans. In the normal subject, flow velocity greatly increased after the meal, whereas in the patient with liver cirrhosis, portal flow velocity frankly decreased in the postprandial state.

TABLE 3. Effect of a standard meal on the portal vein flow volume

	Before meal	After meal	% increase
NS (n = 12)	832 ± 245[a,c]	1,312 ± 433[c]	+59
CAH (n = 11)	891 ± 293[b,d]	1,261 ± 302[d]	+49
LC (n = 11)	1,160 ± 426[a,b,e]	1,262 ± 480[e]	+8.5

The values are expressed in ml per min (means ± S.D.) and as mean percentage increase. *a* to *e* indicate levels of significance by Student's t test between values with the same letter: *a* = $p < 0.025$; *c*, *d* = $p < 0.0005$; *b*, *e* = not significant. Analysis of variance between groups before meal: $p < 0.0005$; between percentages of increase: $p < 0.0005$.

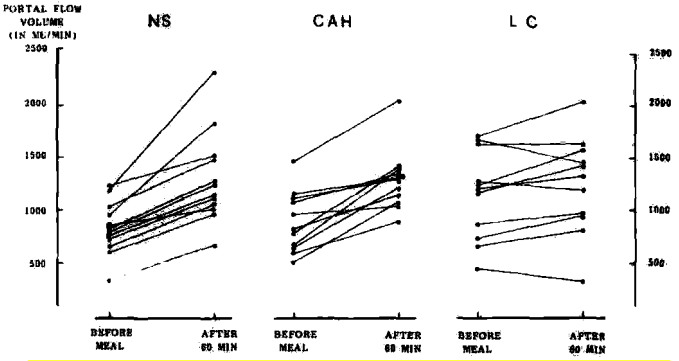

FIG. 4. Flow volume in the portal vein before and after a standard meal in normal subjects (NS) and in patients with chronic active hepatitis (CAH) and with liver cirrhosis (LC). In NS and CAH groups, flow volume constantly increased in all patients after the meal. In patients with LC, in whom basal flow volume was greater (1,160 ± 426 ml per min) than in NS (832 ± 245 ml per min; $p < 0.025$) and in CAH (891 ± 292 ml per min; NS), flow volume decreased in three, did not change in one and moderately increased in the remainder.

per min per kg). The rate of flow volume increase was statistically different in the three groups of patients (ANOVA: $p < 0.0005$; NS vs. CAH, not significant; NS vs. LC $p < 0.0005$; CAH vs. LC $p < 0.005$). In the same way as the velocity of portal flow, flow volume decreased in some patients (three of 11) with LC, whereas it constantly increased in all NS and in patients with CAH (Fig. 4).

The duplex Doppler study performed at 90 and 120 min after a meal in three NS and three patients with LC showed that maximal portal flow increase occurs after 60 min.

DISCUSSION

Using the duplex Doppler technique, we demonstrated a significant increase of portal flow in the fed state both in normal subjects and in patients with chronic active hepatitis, but not in patients with liver cirrhosis.

The accuracy of duplex Doppler in investigating portal flow has been demonstrated by recent papers in which this method was compared with cineangiography (14) and electromagnetic flowmetry (15). However, the estimation of the mean velocity of blood flow by pulsed Doppler may involve some error because of the exclusion of lower frequencies by the use of a high-pass filter. Such error may be minimized by the conversion of mean velocity to maximal velocity. With reference to this point, we compared mean velocity of blood, directly determined by Doppler spectral analysis, with the values of mean velocity calculated by the conversion of maximal velocity (14), obtaining a good correlation. It is reasonable to affirm that pulsed Doppler measurement of portal flow reflects the actual blood flow in the portal vein even though the absolute value expressed in ml per min may not correspond to the real flow volume. Doppler flowmetry is therefore suitable for monitoring acute hemodynamic changes in the portal system, such as those induced by a standard meal. Morphological changes in the normal portal venous system following a meal have been recently evaluated by ultrasonography (29); in this study, the mean caliber of the portal vein increased by 47.5% 1 hr after a meal, whereas in our group of NS the increase was markedly lower (+14%). Okazaki et al. (30) have studied portal blood flow in normal subjects and in patients with liver cirrhosis using the Octoson pulsed Doppler system. Their data are partially in agreement with ours; they reported a 125% increase in portal flow 60 min after a meal in normal subjects and a 15% increase in patients with liver cirrhosis. Our patients showed a lower flow increase (by 59% in NS and by 8.5% in LC). These various hemodynamic responses may be correlated in both cases with the composition of the meal administered, whereas all these studies confirm that maximal flow increase occurs after 60 min. In a recent study, Darnault et al. (31), using pulsed Doppler technique, reported a postprandial increase of portal flow volume by 80% in normal subjects and by 26% in patients with liver cirrhosis. In these patients, flow increase was greater in comparison with that which occurred in our experience (+8.5%) and in Okazaki's group (+15%), probably in relation to different degrees of severity of liver cirrhosis.

In NS the increase in flow volume was consequent to the increase in caliber and in flow velocity, except in one case in which flow velocity slightly decreased after a meal and flow volume rose due to the increase of the caliber (and cross-sectional area) alone.

The postprandial portal venous inflow in healthy subjects seems to be essentially determined by the increased inflow from the mesenteric arterial bed, as recently confirmed by Jaeger et al. (12) with duplex Doppler. In this mechanism, arteriolar vasodilatation probably plays a prominent role. It is not yet established whether splanchnic venous bed dilatation is a passive or active event (3), although experimental studies suggest a portal venous response to several vasoactive substances (32–34).

In patients with CAH, the increase in portal blood flow was slightly and not significantly lower than in NS (49% vs. 59%), with a rise of flow velocity in all patients but one. Four patients of this group had a portal vein caliber ranging from 12 to 14 mm, which is considered "possibly pathological" (23), and it did not change after a meal. This aspect may suggest an initial congestion and lack of compliance of splanchnic venous vessels which prevents portal dilatation in response to the meal and may represent an initial sign of portal hypertension. The remaining patients of this group responded to a meal essentially as controls, probably because they did not have a significant increase of portal pressure and portosystemic collaterals had not developed.

In our group of LC, we found a significantly higher basal portal flow in comparison with control subjects. The same pattern was found in the group of Darnault et al. (31). Regarding this point, it is important to emphasize that LC may show different patterns of portal flow volume as described by Ohnishi et al. (16), probably reflecting different evolutive stages of the disease and presence of portosystemic collaterals. In our group with LC, mean portal flow showed a minimal increase (from $1,160 \pm 426$ to $1,262 \pm 480$ ml per min, corresponding to +8.5%). Examining individual patterns, it should be remarked that flow volume increased in seven patients by 9 to 33%, remained unchanged in one patient and decreased by 6 to 30% in three patients (Fig. 4). These data are probably correlated with the increased intrahepatic resistances and the hypertensive state in the splanchnic venous system of the cirrhotic patients which prevents further increase in splanchnic venous flow and with the diversion of blood flow by portosystemic collaterals; this phenomenon probably played a predominant hemodynamic role in the three patients in whom portal flow decreased after a meal.

Vasoactive intestinal mediators, which are involved in the mechanism of postprandial hyperemia (3, 32–34), might play a vasodilating effect also on collateral circulation, and this may result in a further decrease in portal flow.

From a clinical point of view, we believe that a reduced flow velocity in the portal vein associated to a lack of response of portal flow to a meal may represent an indirect sign of the presence of increased intrahepatic

resistances and/or spontaneous portosystemic shunts, thus suggesting portal hypertension in patients in whom other morphological findings are missing on conventional real-time ultrasound.

REFERENCES

1. Reininger EJ, Sapirstein LA. Effect of digestion on distribution of blood flow in the rat. Science 1957; 126:1176–1178.
2. Norryd C, Dencker H, Lunderquist A, et al. Superior mesenteric blood flow during digestion in man. Acta Chir Scand 1975; 141:197–202.
3. Richardson PDI, Withrington PG. Liver blood flow. I. Intrinsic and nervous control of liver blood flow. Gastroenterology 1981; 81:159–173.
4. Lanciault G, Jacobson ED. The gastrointestinal circulation. Gastroenterology 1976; 71:851–859.
5. Granger DN, Richardson PDI, Kvietys PR, et al. Intestinal blood flow. Gastroenterology 1980; 78:837–845.
6. Bowen JC, Pawlik W, Fang WF, et al. Pharmacologic effects of gastrointestinal hormones on intestinal oxygen consumption and blood flow. Surgery 1975; 78:515–519.
7. Chou CC, Hsieh CP, Dabney JM. Comparison of vascular effects of gastrointestinal hormones on various organs. Am J Physiol 1977; 232:H34–H39.
8. Price JB Jr, Voorhees AB Jr, Britton RC. Operative hemodynamic study in portal hypertension. Significance and limitations. Arch Surg 1967; 95:843–851.
9. Moreno AH, Burchell AR, Rousselot LM, et al. Portal blood flow in cirrhosis of the liver. J Clin Invest 1967; 46:436–445.
10. Sovak M, Soulen RL, Reichle FA. Blood flow in the human portal vein. A cineradiographic method using particulate contrast medium. Radiology 1971; 99:531–536.
11. Taylor KJW, Burns PN, Woodcock JP, et al. Blood flow in deep abdominal and pelvic vessels: ultrasonic pulsed Doppler analysis. Radiology 1985; 154:487–493.
12. Jaeger K, Bollinger A, Valli C. Measurement of mesenteric blood flow by duplex scanning. J Vasc Surg 1986; 3:462–469.
13. Gill RW. Pulsed Doppler with B-mode imaging for quantitative blood flow measurement. Ultrasound Med Biol 1979; 5:223–235.
14. Ohnishi K, Saito M, Koen H, et al. Pulsed Doppler flow as a criterion of portal venous velocity: comparison with cineangiographic measurements. Radiology 1985; 154:495–498.
15. Moriyasu F, Ban N, Nishida O, et al. Clinical application of an ultrasonic duplex system in the quantitative measurement of portal blood flow. J Clin Ultrasound 1986; 14:579–588.
16. Ohnishi K, Saito M, Nakayama T, et al. Portal venous hemodynamic in chronic liver disease: effects of posture change and exercise. Radiology 1985; 155:757–761.
17. Ohnishi K, Saito M, Sato S, et al. Direction of splenic venous flow assessed by pulsed Doppler flowmetry in patients with large splenorenal shunt: relation to spontaneous hepatic encephalopathy. Gastroenterology 1985; 89:180–185.
18. Patriquin H, Lafortune M, Burns PN, et al. Duplex Doppler examination in portal hypertension: technique and anatomy. Am J Roentgenol 1987; 149:71–76.
19. Burns PN, Jaffe CC. Quantitative flow measurements with Doppler ultrasound: techniques, accuracy and limitations. Radiol Clin North Am 1985; 23:641–657.
20. Burns PN, Taylor KJW, Blei AT. Doppler flowmetry and portal hypertension (Editorial). Gastroenterology 1987; 92:824–826.
21. Japanese Research Society for Portal Hypertension (Chairman: K Inokuchi). The general rules for recording endoscopic findings on esophageal varices. Jpn J Surg 1980; 10:84–93.
22. Subramanyam BR, Balthazar EJ, Madamba MR, et al. Sonography of porto-systemic venous collaterals in portal hypertension. Radiology 1983; 146:161–166.
23. Bolondi L, Gandolfi L, Arienti V, et al. Ultrasonography in the diagnosis of portal hypertension: diminished response of portal vessels to respiration. Radiology 1982; 142:167–172.
24. Bolondi L, Mazziotti A, Arienti V, et al. Ultrasonographic study of portal venous system in portal hypertension and after porto-systemic surgical shunts. Surgery 1984; 95:261–269.
25. Weinreb J, Kumari S, Phillips G, et al. Portal vein measurements by real time sonography. Am J Roentgenol 1982; 139:497–499.
26. Di Candio G, Campatelli A, Mosca F, et al. Ultrasound detection of unusual spontaneous portosystemic shunts associated with uncomplicated portal hypertension. J Ultrasound Med 1985; 4:297–305.
27. Hill MC, Dach JL, Shawker TH. Ultrasonography in portal hypertension. In: Benhamou JP, Lebrec D, eds. Clinics in gastroenterology, Vol. 14. London: W. B. Saunders, 1985: 83–104.
28. Snedecor GW, Cochran WG. Statistical methods, Ed. 7. Ames, Iowa: The Iowa State University Press, 1980.
29. Bellamy EA, Bossi MC, Cosgrove DO. Ultrasound demonstration of changes in the normal portal venous system following a meal. Br J Radiol 1984; 57:147–149.
30. Okazaki K, Miyazaki M, Onishi S, et al. Effects of food intake and various extrinsic hormones on portal blood flow in patients with liver cirrhosis demonstrated by pulsed Doppler with the Octoson. Scand J Gastroenterol 1986; 21:1029–1038.
31. Darnault P, Bretagne JF, Raoul JL, et al. Effects of food intake on portal blood flow and superior mesenteric venous blood flow in patients with liver cirrhosis demonstrated by pulsed Doppler. Proceedings of the 6th Congress of the European Federation of Societies for Ultrasound in Medicine and Biology, Helsinki, Finland, June 14–18, 1987: 70.
32. Hanson KM, Post JA. Splanchnic vascular responses to the infusion of prostaglandins A1, A2 and B1. Pharmacology 1976; 14:166–186.
33. Richardson PDI, Withrington PG. The effect of intraportal injection of noradrenaline, adrenaline, vasopressin and angiotensin on the hepatic venous vascular bed of the dog: marked tachyphylaxis to angiotensin. Br J Pharmacol 1977; 59:293–301.
34. Richardson PDI, Withrington PG. Responses of the simultaneously-perfused hepatic arterial and portal venous vascular beds of the dog to hystamine and 5-hydroxytriptamine. Br J Pharmacol 1978; 64:581–588.