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Interrelationship between Splenic and Superior Mesenteric Venous Circulation Manifested by Transient Splenic Arterial Occlusion Using a Balloon Catheter

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We examined the hemodynamic changes induced by transient splenic arterial occlusion using a balloon catheter to investigate the hemodynamic effect of transcatheter splenic arterial embolization—a procedure that has been used since its introduction in 1973 as therapy for hypersplenism and more recently for portal hypertension. The blood flow volume was measured in 20 patients with liver disease using an ultrasonic duplex system (Toshiba SAL50A/SDL-01A). The portal venous pressure was also measured via a 3F catheter using a transducer. The catheter was placed in position by substituting it for a 25-gauge needle that had been inserted into the portal vein under ultrasonic guidance percutaneously and transhepatically.

Splenic arterial occlusion caused a drop in splenic venous blood flow from 708 ± 487 to 241 ± 155 ml per min, in portal venous blood flow from 993 ± 439 to 807 ± 419 ml per min and in portal venous pressure from 17.4 ± 7.2 to 14.4 ± 6.1 mm Hg. The latter two reductions were less than expected from the decrease in the splenic venous blood flow volume. This phenomenon was caused by an increase in the mesenteric venous blood flow from 475 ± 126 to 630 ± 270 mm per min.

This increase may be due to a compensatory mechanism under the control of a regulatory loop in the liver or portal vein, and there seems to be a relationship between splenic and intestinal circulation in portal hypertension that maintains hepatic circulation.

Embolization of the spleen was first performed clinically by Maddison (1) in 1973 for therapy of hypersplenism. Maddison injected an autologous clot into the splenic artery and concluded that the splenic embolization-induced splenectomy had been successful. He also stated that no further episodes of gastrointestinal bleeding occurred.

Since Maddison, more than a dozen reports about splenic embolization have appeared (2–6). In respect to the hematologic effects, many authors have claimed that, although it involves complications in the form of fever, pain, pleural effusion, etc., occlusion of the splenic artery constitutes an effective, noninvasive approach to the control of splenic hyperfunction, if used with extreme caution.

In respect to the hemodynamic effects, however, there appears to be conflicting information. Some authors claim that occlusion of the splenic artery reduces the amount of blood entering the portal circulation, reduces the portal venous pressure and lowers the risk of bleeding (1, 5, 7). Owman et al. (3), however, reported that there was no change in the portal venous pressure while Zannini et al. (7) described some cases in which there was only a small reduction in the wedged hepatic venous pressure following temporary occlusion of the splenic artery, although there were a number of cases in which the reduction of the wedged hepatic venous pressure was great.

The purpose of our examination was to investigate the hemodynamic effects of occlusion of the splenic artery on portal circulation.

PATIENTS AND METHODS

Twenty patients (12 men and 8 women with an average age of 53.9 yr) were studied. Liver biopsy disclosed liver cirrhosis in 7 patients, chronic active hepatitis in 5 patients, chronic inactive hepatitis in 3 patients and idiopathic portal hypertension in another 3 patients. Angiography indicated liver cirrhosis with hepatocellular carcinoma in one patient and hepatic cyst in one patient. All patients underwent angiography, but the danger of obtaining pressure measurements in two cases together with the difficulty in penetrating gas using the Doppler beam in several cases sometimes precluded other hemodynamic examinations. Thus, there are varying numbers of patients in some of the results and figures.

The splenic-arterial occlusion studies were performed by taking advantage of routine hemodynamic examinations, which consisted of angiographical examinations, and pressure and blood flow measurements. These hemodynamic examinations are considered essential for patients with chronic liver disease in order to estimate the extent of the disease and determine treatment, and are performed at our hospital with the patient's full consent. The patients were fasting, supine and sedated by intravenous injection of 0.1 mg fentanyl and 2.5 mg droperidole.

The occlusion studies were performed after the routine hemodynamic examinations. A 7F catheter was introduced into

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the splenic artery using the Seldinger technique, and withdrawn and replaced by a balloon catheter to occlude the splenic artery. (However, the object was not necessarily to totally occlude the artery but to cause a sufficient reduction in flow volume in order to see any changes in the flow volume of other vessels. The degree of occlusion depended on the arterial anatomy of the individual.) This transient occlusion technique is in no way dangerous, and no patients complained of pain or nausea during the procedure. There were no complications which arose in connection with the technique.

A 25-gauge cholangiography needle was inserted into the intrahepatic portal branch percutaneously and transhepatically under ultrasonic guidance, and then replaced by a 3F catheter. The 3F catheter was used for portal pressure measurements in which the zero reference point was set at the midpoint between the anterior sternal and the dorsal surfaces of the patient.

The portal venous pressure was measured using a transducer, and the blood flow volume of the portal, splenic and superior mesenteric veins was measured using an ultrasonic duplex system composed of a linear-array type electronic scanner and a pulsed Doppler flowmeter (Toshiba SAL 50A/SDL 01A) (8, 9). The splenic artery was then occluded by inflating the balloon at the hilus of the spleen and the same measurements repeated—a process that took about 15 min, after which the balloon was deflated.

The procedure for measuring the blood flow was as follows. The longitudinal section of the vessel was displayed using the B-mode and the sample position for the Doppler mode set at the central portion of the vessel. Maximum blood velocity (V_{Dmax}) at the center of the vessel was measured from the Doppler spectrum obtained. The angle (θ) created between the ultrasonic Doppler beam and the blood vessel was obtained from the B-mode image.

Immediately after recording the Doppler signals, the cross-section at the sample position was scanned and the cross-sectional area (S) measured on the B-mode display. The blood flow volume of the vessel (BFV) was then calculated using the following equation (Figure 1).

$$BFV = S \times \frac{0.57\ V_{Dmax}}{\cos\theta} \times 60\ (ml/min).$$

Table 1 is a list of blood flow volumes of the portal venous system in a variety of liver diseases, as we recorded in a previous study. This study also indicated the reliability of the method by contrasting with simultaneous measurements made with an electromagnetic flowmeter (8).

Paired Student's t test was used for statistical analysis.

RESULTS

The splenic venous blood flow volume was reduced in all cases following temporary occlusion. The reduction in flow volume from an original 708 ± 487 to 241 ± 155 ml per min was statistically significant ($p < 0.01$) (Figure 2). The portal venous pressure was significantly reduced in all cases, from an average of 17.4 ± 7.2 to 14.4 ± 6.1 mm Hg ($p < 0.01$) (Figure 3). The portal venous blood flow was also significantly reduced, from 993 ± 439 to 807 ± 419 ml per min ($p < 0.01$) (Figure 4). The unexpected finding was the significant increase in the superior

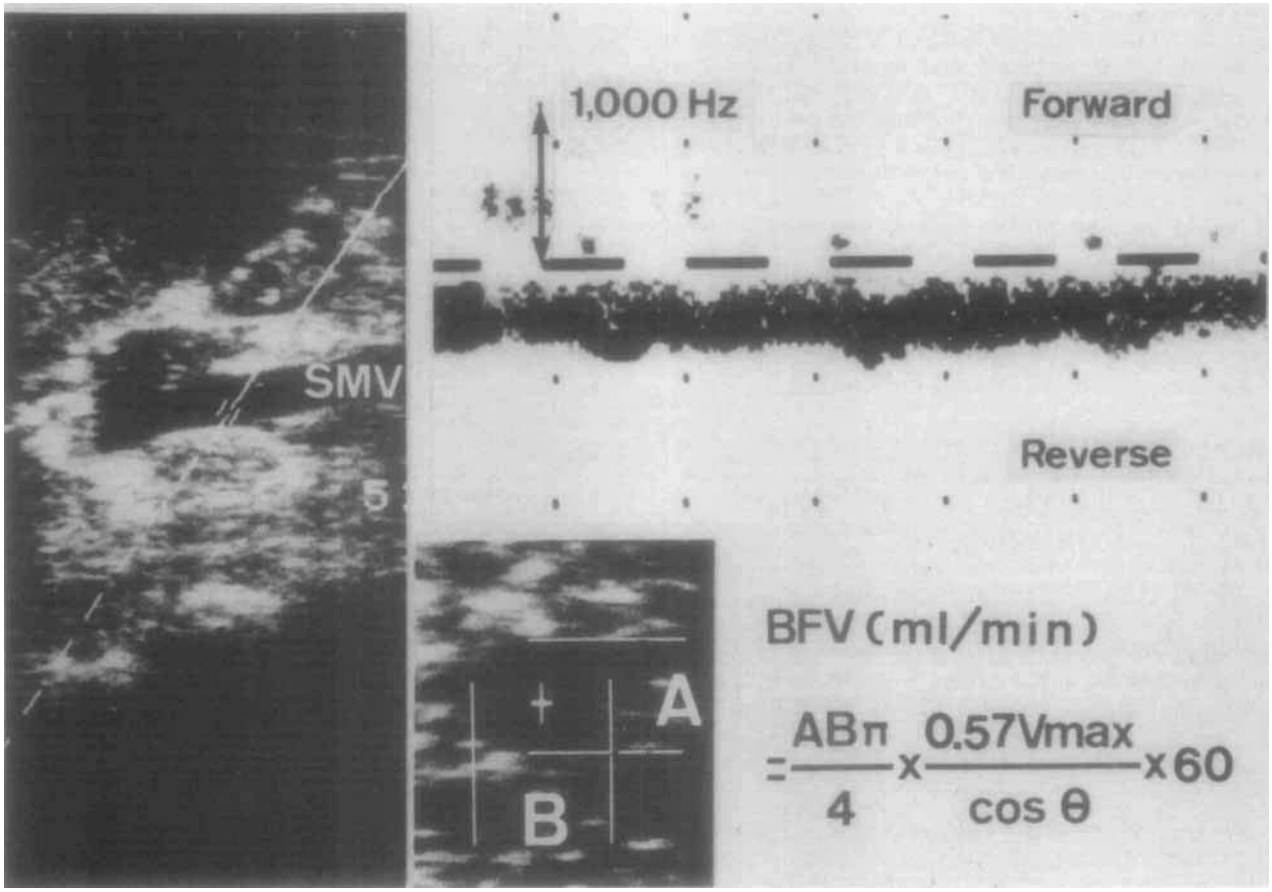

FIG. 1. Measurement of the blood flow volume (BFV) using the ultrasonic duplex system. SMV = superior mesenteric vein.

TABLE 1. Portal blood flow volume and splenic venous blood flow volume

	No. of cases	Portal blood flow volume (ml/min)	Splenic venous blood flow volume (ml/min)
Normal subjects	88	889 ± 284	450 ± 192
Chronic active hepatitis	45	853 ± 223	504 ± 147
Cirrhosis	81	881 ± 331	690 ± 465[a]
Idiopathic portal hypertension	16	979 ± 411	977 ± 538[a]
Hepatoma	39	922 ± 409	587 ± 301[b]

Values are mean ± S.D.
[a] $p < 0.001$ compared with normal subjects.
[b] $p < 0.05$ compared with normal subjects.

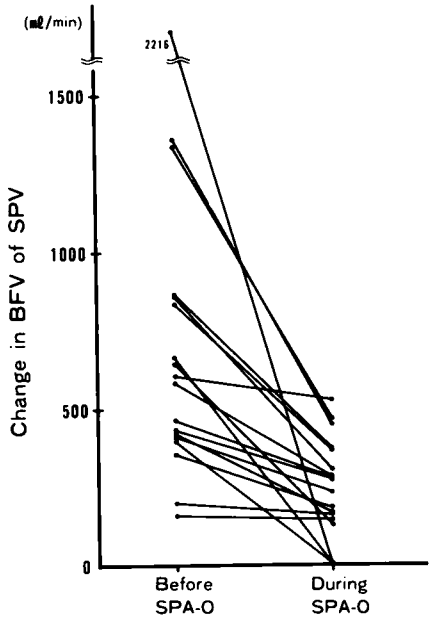

FIG. 2. Blood flow volume (BFV) of splenic vein (SPV) reduced during splenic artery occlusion (SPA-O).

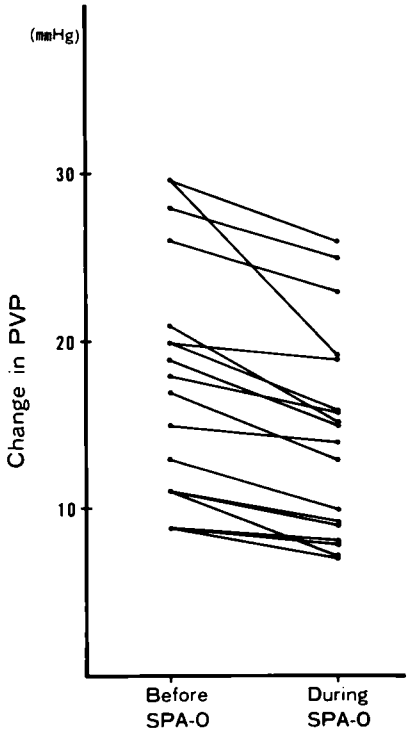

FIG. 3. Portal venous pressure (PVP) reduced during splenic artery occlusion (SPA-O).

mesenteric venous blood flow volume from 475 ± 126 to 630 ± 270 ml per min ($p < 0.05$) (Figure 5).

The splenic venous blood flow volume (in three cases) and the portal venous blood flow volume (one case) was registered as zero during temporary splenic arterial occlusion; this may be due to the limitations of the Doppler system used. The detectable blood flow velocity threshold of this system is about 6 cm per sec in practice. We therefore assumed that this value of zero indicated a value so close to zero that it could not be detected.

DISCUSSION

The portal venous pressure depends on the resistance to outflow from the liver and on the splanchnic blood flow volume (10, 11). It was therefore previously thought that portal blood flow was stagnant in the case of portal hypertension because of the increase in intrahepatic portal vascular resistance (12). In contrast to this conventional view, however, there are many reports that claim that there is also an increased blood flow into the portal system, which plays an important role in maintaining portal hypertension (13–16).

Transcatheter splenic embolization, introduced by Maddison in 1973, has been performed in patients with splenomegaly and portal hypertension, with the expectation that this nonsurgical splenectomy would result in hematologic and hemodynamic effects (2–6). Miller et al. (17), however, summarized 17 cases in which the patients survived for five or more years after surgical splenectomy and out of which seven experienced further episodes of hematemesis. Moreover, there are some reports that claim there is no change in the portal venous pressure (3), and one that describes cases in which there was only little change in the pressure following nonsurgical splenectomy—splenic arterial occlusion (7).

In this study, we noted a statistically significant change in the portal venous pressure using splenic artery occlusion, which was first reported by Viamonte et al. (18) as a method of assessing hyperdynamic portal hypertension. Other authors also described a reduction in the portal venous pressure following occlusion of the splenic or superior mesenteric arteries (2, 5, 7, 15). However, we found the reduction to be less than expected from the reduction in splenic venous blood flow.

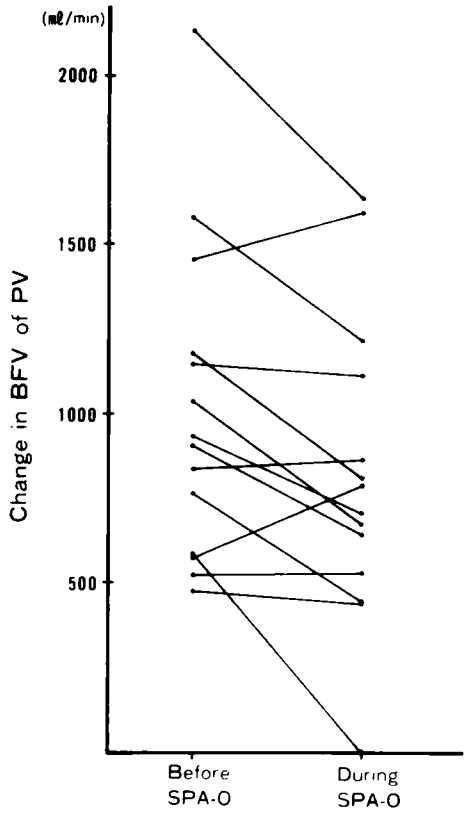

FIG. 4. Blood flow volume (BFV) of portal vein (PV) reduced during splenic artery occlusion (SPA-O).

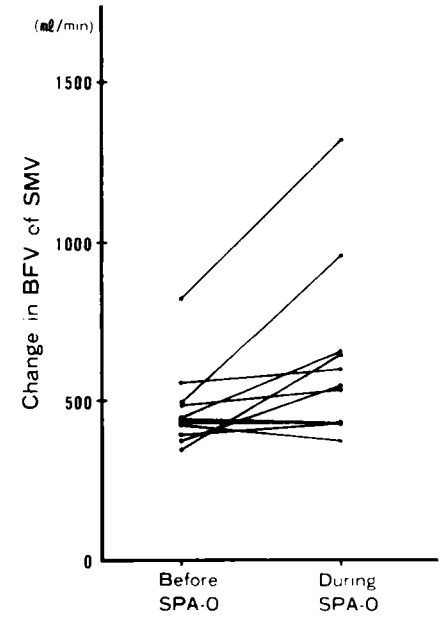

FIG. 5. Blood flow volume (BFV) of superior mesenteric vein (SMV) increased during splenic artery occlusion (SPA-O).

We also measured the change in blood flow volume of the portal, splenic and superior mesenteric veins using the ultrasonic Doppler duplex system. This has recently been used in measuring the blood flow of the portal venous system, and although there are some theoretical problems, we have been able to confirm its accuracy in a comparative study with electromagnetic flowmetry (8), and have used it in a hemodynamic study of the portal system (9, 19). Ohnishi et al. (20) have also reported a linear correlation between the velocities determined using the Doppler method and cineangiography.

We found a reduction in the blood flow volume of the splenic and portal veins, and an unexpected increase in that of the superior mesenteric vein. We assume that the reduction in portal venous pressure causes an increase in the pressure gradient between the artery and the superior mesenteric vein, and an increase in blood flow volume is therefore expected. However, the actual increase was greater than that expected from the pressure gradient. This appears to be an active compensation for the decrease in the splenic venous blood flow, lessening the change in portal venous blood flow and pressure.

There have been many reports on the relationship between the hepatic arterial and portal venous blood flow (21–23), but only few studied the splenic and intestinal blood flows (18, 24, 25). All intimate that there is a relationship between splenic and intestinal circulation. Our finding reinforces this relationship and also indicates that it may be a compensatory phenomenon.

In animals, Vorobioff et al. (26) observed that the portal venous inflow—the sum of the blood flow to the individual organs that drain into the portal vein—was increased in the cirrhotic rat, and suggested the presence of a regulatory feedback loop originating in the liver or portal vein (or both) that modulates the vasodilation and increased blood flow within the splanchnic arterial bed. They also suggested that this feedback mechanism may act to increase the portal venous blood inflow, compensating for the loss of portal blood flow through portal-systemic shunting. They indicated that the potential signal for this feedback might result from: (i) reduced oxygen delivery to the liver from the portal venous blood; (ii) reduced delivery of substrates or hepatotrophic factors such as glucagon; or (iii) neurogenic response mediated by the sympathetic nervous system or related to proposed portal venous baroreceptors (26).

We also suspect that there is a feedback mechanism in the liver or portal vein that regulates the intestinal circulation and splenic circulation; it may also regulate the hepatic arterial blood flow volume according to the work load or the blood flow demand of the liver.

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