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*Review*

# Hepatic Outflow Obstruction Created by Balloon Occlusion of the Hepatic Vein: Induced Hepatic Hemodynamic Changes and the Therapeutic Applications of Hepatic Venous Occlusion with a Balloon Catheter in Interventional Radiology

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**Hepatic outflow obstruction created by balloon occlusion of the hepatic vein induces characteristic angiographic findings in the occluded area: prolonged enhancement on hepatogram followed by reversed portal opacification on the hepatic arteriogram and perfusion defect on the arterial portogram. The following induced hepatic hemodynamic changes are suggested: hepatic arterial flow increases, and the portal vein acts as a draining vein with slow reversed flow. These unique hemodynamic changes enhance the effect of hepatic interventional therapies. In transcatheter arterial infusion, increasing hepatic arterial flow and absence of portal inflow can bring about a high concentration of drugs, the presence of which is greatly protracted due to outflow blockage. In transcatheter arterial chemoembolization, reversed portal flow can allow portal embolization in addition to arterial embolization. In microwave coagulation therapy and radiofrequency ablation therapy, decreasing portal flow can cause larger areas of coagulation. Further, the technique of hepatic venous occlusion has potential therapeutic applications.**

**Key words:** liver, hepatic vein, obstruction, blood supply, therapy

Interventional radiology has developed rapidly in the last few decades with remarkable evolution of the technologies and imaging modalities, and now is accepted as an effective minimally invasive treatment in a variety of territories. Hepatic vascular interventions including transcatheter arterial infusion (TAI) and transcatheter arterial chemoembolization (TAE) were developed to treat malignant hepatic tumors, and have been applied mainly to inoperable tumors. However, their therapeutic effect is not satisfactory; a relatively high recurrence rate and limited prognosis have been shown. In an attempt to enhance their therapeutic effect, a number of drugs, embolic agents, and their combinations have been introduced and tested. As a different strategy, Rousselot and Grossi *et al*. performed hepatic outflow blockage by double balloon occlusions of the inferior vena cava (IVC) at the orifice of the hepatic veins (HVs) during TAI of anticancer drugs using dogs in the 1960s [1-4]. They successfully demonstrated highly concentrated drugs in the hepatic parenchyma, HV, lymph nodes, and the thoracic ducts. However, their methods were quite invasive due to IVC occlusion with a sudden decrease of pulse pressure and cardiac output about 10 min after the blockage. Subsequently, Kanazawa *et al*. improved their

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methods by using the technique of hepatic venous occlusion (HVO) with a balloon catheter selectively catheterized into the HV, and confirmed the safety of the improved methods [5]. They also showed the unique angiographic findings induced by HVO, which gave rise to studies regarding temporary HVO with a balloon catheter. Herein, we review the articles presenting relevant radiological findings, hepatic hemodynamic changes under temporary HVO, and the therapeutic applications of HVO with a balloon catheter.

## Radiological findings under hepatic venous occlusion

The liver has a unique hemodynamics due to its dual blood supply from the hepatic arteries (HAs) and portal veins (PVs). The normal liver receives approximately 75–80% of its blood supply from the PVs and 20–25% from the HAs, both of which mix, flow into the sinusoids, and finally drain into the HVs [6]. PV hemodynamics is variable and can be affected by various factors, including chronic liver disease, posture, diet, and exercise [7, 8]. HA hemodynamics changes to compensate for changes in PV hemodynamics [8, 9]. Thus, if the hepatic vein is occluded, what can be expected to happen to the hepatic hemodynamics?

In membraneous obstruction of the suprahepatic IVC or HV, called primary Budd-Chiari syndrome, the characteristic angiographic findings are well known [10]. Liver, renal, or adrenal tumors may obstruct or compress IVC or major HV, and sometimes are accompanied by tumor thrombus. Hypercoagulable states, use of oral contraceptives, trauma, pregnancy, or collagen-vascular disorders may also compromise the hepatic outflow. Angiographic findings similar to those of primary Budd-Chiari syndrome are observed in the above-mentioned situations, which are known as secondary Budd-Chiari syndrome.

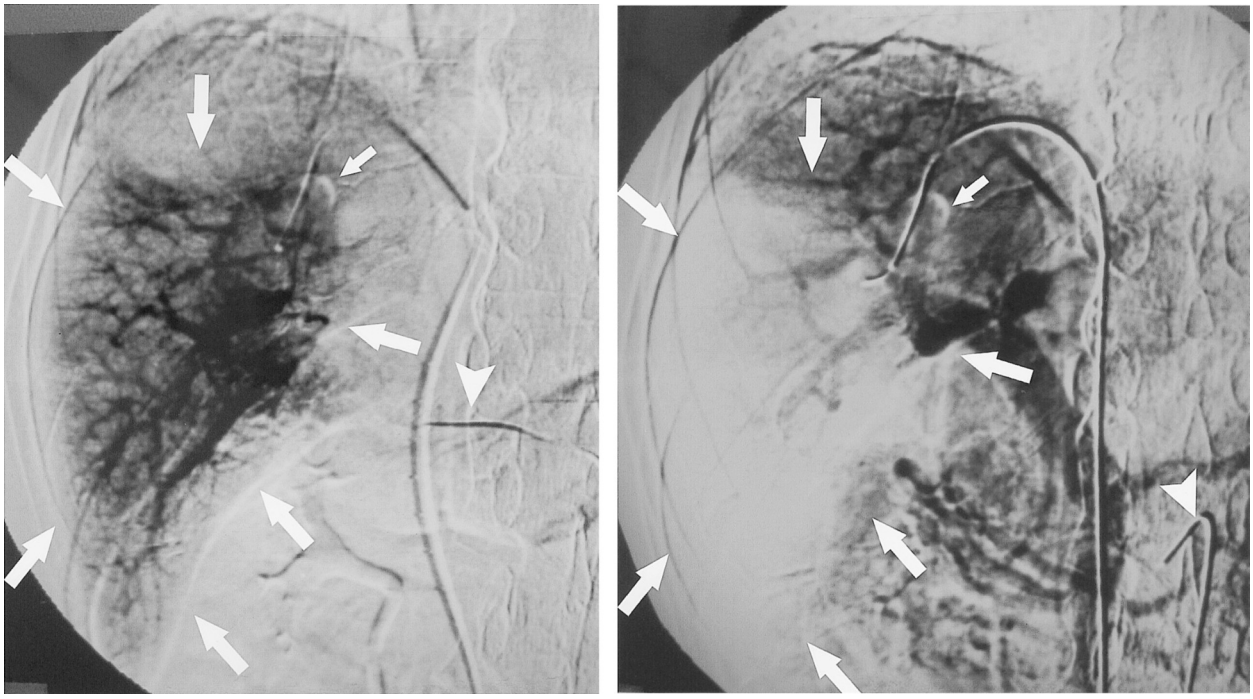


**Fig. 1** Hepatic arteriogram (**A**) and arterial portogram (**B**) under HVO. The occluded area (large arrows) indicates prolonged enhancement on hepatogram with hepatofugal portal opacification on the hepatic arteriogram and perfusion defect with less portal opacification on the arterial portogram. Small arrows demonstrate an inflated balloon in the RHV. Arrowhead indicates a catheter introduced into the common hepatic artery (**A**) or into the superior mesenteric artery (**B**). (Panels (**A**) and (**B**) are reprinted with permission from Hiraki T., Altered Hemodynamics in the Liver Caused by Temporary Occlusion of the Hepatic Vein: Evaluation with Doppler Ultrasonography in 14 Patients. Radiology (2001) 220: 357–364.)

Budd-Chiari syndrome, whether primary or secondary, usually represents the chronic or subacute phase of hepatic outflow obstruction. Kanazawa *et al.* investigated angiographic findings in the acute phase of HVO with a balloon catheter in pigs [5]. Angiographic findings in the occluded area immediately after balloon occlusion of the HV were still similar to those of Budd-Chiari syndrome: prolonged enhancement on hepatogram with more opacification of peripheral small arterial branches followed by reversed portal opacification on the hepatic arteriogram (Fig. 1A) and perfusion defect with less opacification of portal branches on the arterial portogram (Fig. 1B). They also performed scintigrams with hepatic arterial infusion of Technetium-99 m macroaggregated albumin over 60 min, which showed significant increase in radioactivity in the occluded area. By the same methods, they confirmed similar angiographic findings in humans [11].

Computed tomography (CT) during injection of contrast medium through the catheter introduced into the HA (CT hepatic arteriography; CTHA) or into the superior mesenteric artery (CT arterial portography; CTAP) has been shown to improve the ability to demonstrate and differentiate hepatic tumors. Murata *et al.* performed CTHA and CTAP during occlusion of each HV (left (L), middle (M), right (R), and inferior right (IR) HV) in humans [12, 13]. Those findings exactly reflected the angiographic results: the occluded areas were presented as prolonged hyperattenuated regions at CTHA and hypoattenuated regions at CTAP. By using CT, the region of each hepatic venous drainage was clearly demonstrated: the left lobe was drained by the LHV; the ventral part of the anterior segment and medial segment except the ventromedial part by the MHV; the dorsal part of the anterior segment and ventral part of the posterior segment by the RHV; and the dorsocaudal part of the right lobe by the IRHV. There was no overlap of the affected areas among the HVOs except in the case of the LHV and MHV.

## Hepatic hemodynamic changes induced by hepatic venous occlusion

Based on the angiographic findings under HVO, the following induced hepatic hemodynamic changes are suggested: enhanced hepatogram suggests increase in HA flow; reversed portal opacification suggests drainage of HA flow into the PVs; prolonged hepatogram can be explained by slower drainage of HA flow into the PVs resulting from the fact that the pressure gradient between the HAs and PVs is smaller than that between the HAs and HVs in the non-occluded area. However, those angiographic findings may also be influenced by other factors; *e.g.*, enhanced hepatogram can be induced only by the absence of PV inflow without contrast material; reversed portal opacification may be enhanced by the artificial pressure caused by injection of contrast material.

Hiraki *et al.* evaluated the hemodynamic changes under temporary HVO in humans with Doppler US, which allowed quantitative evaluation and eliminated the above influential factors [14]. They examined the velocity of the RHA with a Doppler-tipped guide wire up to 120 sec after balloon occlusion of the RHV. The velocity of the RPV and the Doppler signals of the PV branch in the occluded area were examined before and after RHVO. The velocity of the RHA decreased slightly or did not change for 15–30 sec after RHVO, and then increased rapidly to reach a plateau at around 75–90 sec with 1.5–2.0 times the velocity before RHVO (Fig. 2). Conversely, the velocity of the RPV decreased significantly after

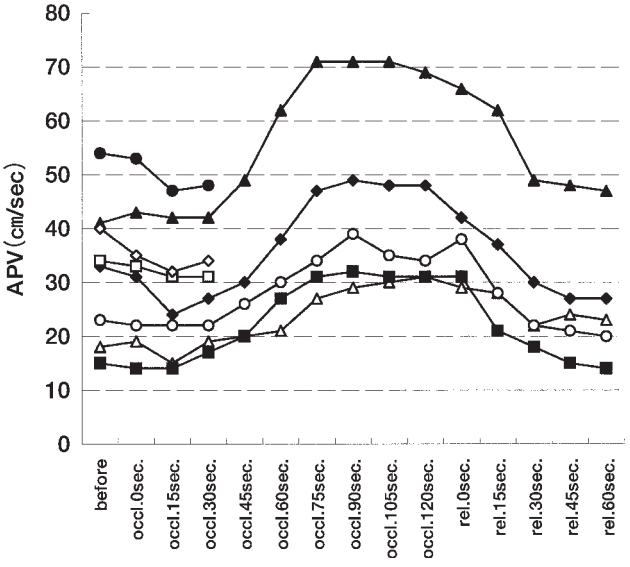


**Fig. 2** Changes in average peak velocity (APV) of the RHA with time after RHVO. APV of the RHA decreases slightly or does not change for 15–30 sec after RHVO, and then increases rapidly and reaches a plateau at around 75–90 sec with 1.5–2.0 times the velocity of that before RHVO. After release of RHVO, APV returns close to the baseline. occl. = occlusion; rel. = release (Graph is reprinted with permission from Hiraki T., Altered Hemodynamics in the Liver Caused by Temporary Occlusion of the Hepatic Vein: Evaluation with Doppler Ultrasonography in 14 Patients. Radiology (2001) 220: 357–364.)

RHVO. Doppler signals of the PV branch in the occluded area presented no signal in most patients (10/13 patients), decreased hepatopetal flow in 2 patients, and hepatofugal flow in 1 patient (Fig. 3). Their quantitative data suggested the following hemodynamic changes with time after HVO [15] (Fig. 4). (i) Immediately after HVO, blood from the HA and PV continues to flow into the sinusoids, which causes sinusoidal stagnation and expansion elevating sinusoidal pressure. At first, elevating sinusoidal pressure mainly decreases PV flow and affects HA flow to a lesser degree because PV pressure is much lower and closer to sinusoidal pressure than HA pressure is (Fig. 4B). (ii) Thereafter, decrease in PV flow induces compensatory increase in HA flow, leading

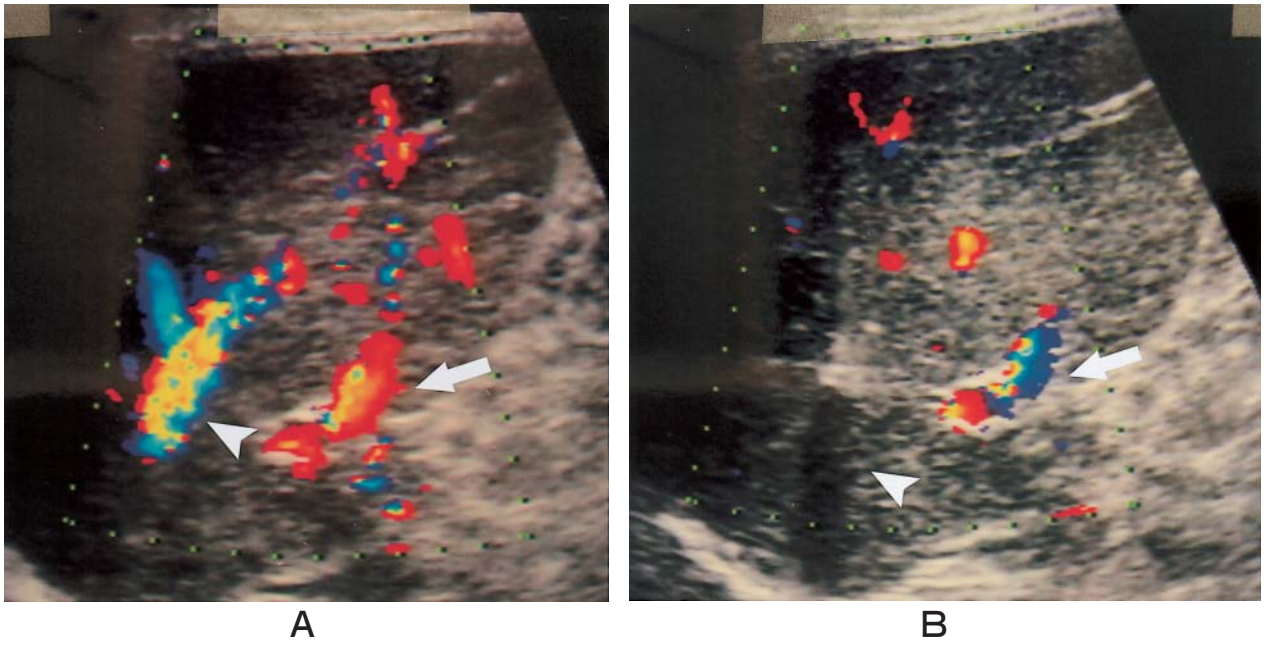


**Fig. 3** Color Doppler images in the occluded area before (**A**) and after (**B**) RHVO. Doppler signal of the PV branch in the occluded area (arrows) changes from hepatopetal before RHVO to hepatofugal after RHVO. Doppler signal of the RHV (arrowheads) changes from hepatofugal before RHVO to no signal after RHVO. (Panels (**A**) and (**B**) are reprinted with permission from Hiraki T., Altered Hemodynamics in the Liver Caused by Temporary Occlusion of the Hepatic Vein: Evaluation with Doppler Ultrasonography in 14 Patients. Radiology (2001) 220: 357–364.)

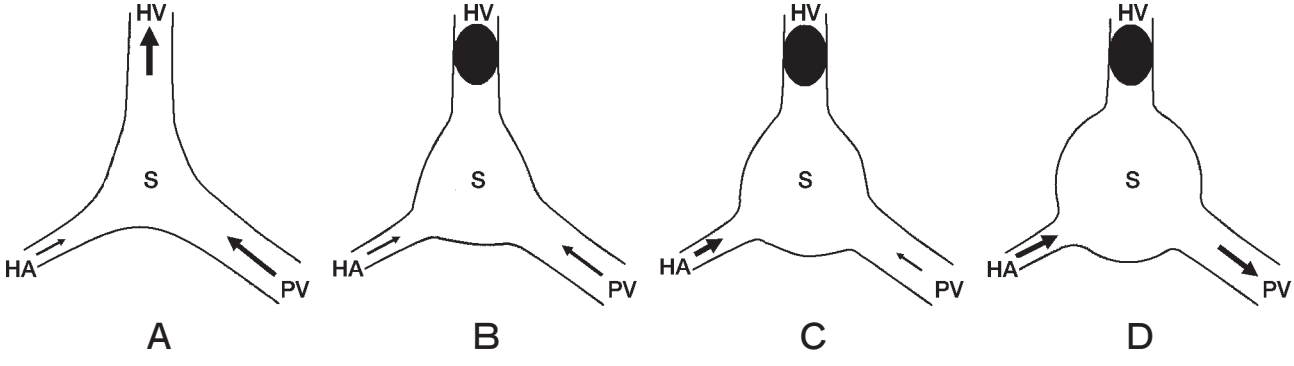


**Fig. 4** The schema of hemodynamic changes with time after HVO. (**A**) Before HVO, the sinusoid receives approximately 75–80% of its blood from the PV and 20–25% from the HA, which mix together, flow into the sinusoid (S), and drain into the HV. (**B**) Immediately after HVO, sinusoidal stagnation and expansion starts, leading to decrease in PV flow and slight decrease or no change in HA flow. (**C**) Then, decrease in PV flow induces compensatory increase in HA flow, leading to more sinusoidal stagnation and expansion. PV flow is slightly hepatopetal or stops before sinusoidal pressure exceeds the portal pressure. (**D**) After the sinusoid is fully expanded and its pressure exceeds the portal pressure, HA flow drains into the PV.

to more sinusoidal stagnation, and expansion. PV flow continues to decrease, but it is still slightly hepatopetal or stops until sinusoidal pressure exceeds the portal pressure (Fig. 4 C). Thus, the question is raised: where does persistent HA flow drain? They expected that (iii) after HA flow fully expands the sinusoids and their pressure overcomes the portal pressure, persistent HA flow could probably drain into the PV (Fig. 4D); however, their study focused on the very acute phase of HVO and thus did not show PV drainage except in one patient.

Pathologic findings in Budd-Chiari syndrome appear to support their expectation of PV drainage. Congestion and prominent distention of the sinusoids are identified mainly in the centrilobular regions, while the sinusoids around the portal triads are relatively spared [16]. Hepatocytes around the patent PV survive, whereas parenchyma in the centrilobular region and around the thrombosed PV become extinct with loss of hepatocytes and fibrosis [17]. These results very likely suggest that in Budd-Chiari syndrome, *i.e.*, in the subacute or chronic phase of hepatic outflow obstruction, hepatic arterial blood flows into the sinusoids around the portal triads, supplying the surrounding hepatocytes and then draining into the patent PV.

Interestingly, HA flow increases in spite of the elevated peripheral vascular resistance due to the outflow blockage. Although mechanisms to regulate the hepatic blood flow are not yet fully understood, neurogenic factors such as sympathetic activity and humoral factors such as glucagon or platelet-activating factor have been suggested to play a role [18–20]. Further, hemodynamic autoregulation mediated by adenosine, a potent vasodilator, is likely one of the main mechanisms, the one called "hepatic arterial buffer response" by Lautt *et al*. [21–23]. Their experimental results using cats suggested that adenosine is continuously released in the fluid surrounding the portal triad at the pre-sinusoidal level (the so-called Mall space); the concentration of adenosine is regulated by washout by PV and HA flow in the portal triad, and when PV flow decreases, less adenosine is washed out and a higher concentration of adenosine leads to increase in HA flow because of its vasodilating effect. If their theory can be applied to humans, the mechanism for overcoming the elevated resistance and increasing the HA flow can be explained.

## Therapeutic applications of hepatic venous occlusion in interventional radiology

The hepatic hemodynamic changes induced by HVO can provide advantages to hepatic interventional therapies. In TAI and TAE for malignant hepatic tumors, absence of PV inflow and increased HA flow can bring about a higher concentration of drugs in the occluded area, which lasts for a longer duration. Moreover, in TAE, reversed PV flow may allow embolization of the PVs in addition to the HAs; *i.e.*, dual embolization. The effect of dual embolization for hepatocellular carcinomas (HCCs) was first explored in 10 patients by Nakao *et al*. [24]. In their study, PV embolization was achieved by percutaneous transhepatic PV puncture. They successfully created infarction of the affected area containing tumors, resulting in complete necrosis of tumors in 4 of 5 patients in whom histologic results were available. In 1 patient, a portion of the tumor cells remained viable due to a technical failure. The remaining 5 patients without histologic results had no evidence of tumor recurrence for 2–17 months. Although classical HCCs are predominantly supplied by HA flow alone, capsular invasion of tumor, small intrahepatic metastases, and early HCCs can be related to PV supply [25–27]. Surrounding parenchymal infarction caused by dual embolization can offer sufficient safety margin in the treatment. Thus, dual embolization likely improved the effect of TAE alone, although their results were preliminary.

Higashihara and Okazaki *et al*. performed TAE during HVO in 6 patients with HCC, aiming at dual embolization by using the technique of HVO [28, 29]. Retrograde opacification of PV branches was demonstrated in the occluded area on fluoroscopy during embolization, which suggested embolization of PV branches by iodized oils. No local recurrence was identified during follow-up of 1–17 months (mean, 6 months) in any of the 6 patients. All of the 4 patients with more than 3-month follow-up showed atrophy of the treated area containing the tumors. Their results are quite encouraging, and their technique is an easier and less invasive method to achieve dual embolization than the technique with percutaneous transhepatic PV puncture. However, regrettably, no further results of TAE during HVO have not been reported to date.

Percutaneous coagulation or ablation therapy using microwave (microwave coagulation therapy; MCT) and radiofrequency (radiofrequency ablation; RFA) induces

coagulated necrosis of tissue by heat and has been shown to be a feasible, safe, and effective therapy for malignant hepatic tumors. Although a variety of electrode needles are available, some limitations remain to be resolved. One limitation is the coagulated area obtained per session is restricted, partly because the cooling effect by the abundant hepatic blood flow limits the thermal spread. Therefore, multiple needle insertions are required during the same treatment session, especially in large lesions. HVO is 1 method of decreasing the hepatic blood flow resulting from significantly decreased PV flow.

Takamura *et al*. examined and compared the diameters of coagulated areas by MCT using porcine normal livers in the following 6 groups: no vessel occlusion, occlusion of the HA, occlusion of the PV, combined occlusion of the HA and PV, occlusion of the HV, and combined occlusion of the HA and HV [30]. Coagulated areas were significantly larger in the last 4 groups than in the first 2 groups. Although the groups with combined occlusion of the HA and PV and of the HA and HV had the largest coagulated area, there were no significant differences among the last 4 groups. Hence, they concluded that the coagulated area was enlarged mainly by decreased PV flow, and that HVO was an easy and safe method to obtain a sufficiently enlarged coagulated area. The same group examined the extent of coagulated areas by MCT in human cirrhotic livers with HCCs in 2 groups, those with HA embolization alone and those with combination of HA embolization and HVO, revealing significantly larger coagulated areas in the latter group [31]. In a later study, the same group showed promising results in patients with metastatic hepatic tumors treated with MCT with combined balloon occlusion of the HA and HV [32]. The same scenario can be applied to RFA; de Baere *et al*. showed, in humans, larger coagulated areas induced by RFA with HV or PV balloon occlusion than those without occlusion [33]. The larger coagulated areas provided by HVO can probably reduce not only the number of needle-insertions and thereby the procedural time and risks including peritoneal hemorrhage and tumor-cell dissemination, but also the local recurrence rate, especially in cases with large lesions.

When the technique of HVO is applied to the treatment of hepatic tumors, it should be taken into consideration that the hemodynamic changes under HVO in tumors can differ from those in non-tumorous parenchyma. Kanazawa *et al*. compared tumor-vascularity of HCCs with HVO to that without HVO angiographically in 24 patients [34]. Increase in tumor-vascularity was not observed with HVO except in 1 patient with a prominent arteriovenous shunt. Murata *et al*. examined the attenuation of HCCs with and without HVO with the use of CTHA [35]. HVO decreased the attenuation in 27 of 28 HCCs in contrast to the surrounding non-tumorous parenchyma. Those results suggested that hepatic hemodynamic autoregulation by mediators such as adenosine does not exist within HCCs. The following reasons are suggested: (a) because most of the HCCs are originally supplied by HA alone, the theory that increase in HA flow is accompanied by decrease in PV flow after HVO cannot be applied to HCCs. (b) Advanced HCCs can destroy the normal structures that secrete the mediators. (c) Even if the mediators were secreted, arterial tumor vessels could not respond to them because they lack a muscular layer. Decreased tumor vascularity of HCCs does not reduce the effect of TAE using the technique of HVO when the following method is adopted: TAE is first performed without HVO for the treatment of a large extent of HCCs supplied by HA, followed by TAE with HVO for the portion of HCCs supplied by PV and the surrounding parenchyma. In MCT or RFA under HVO for hepatic tumors, decreased tumor vascularity can contribute to achieve larger coagulated areas.

In addition to the above applications, the technique of HVO with a balloon catheter has other potential applications in hepatic interventions. Percutaneous transhepatic portal embolization (PTPE) has been performed in the resected lobe before extended hepatectomy to prevent postoperative hepatic failure, offering compensatory hypertrophy of the remaining (non-embolized) lobe accompanied by atrophy of the resected (embolized) lobe. Although this method has been proven to be effective to enlarge the non-embolized lobe, it is an invasive method sometimes accompanied by limited enlargement of the non-embolized lobe. One of the reasons for its limited results may be compensatory increase in HA flow after PTPE, which reduces atrophy of the embolized lobe. Because TAE during HVO can provide dual embolization, it might be an alternative to PTPE; it might increase atrophy of the embolized lobe and hypertrophy of the non-embolized lobe. Further, it could be an easier and less invasive procedure.

Bleeding events from the intrahepatic portal vein forming intrahepatic or subcapsular hematoma, hemoperitoneum, or hemobilia can be associated with hepatic trauma including iatrogenia due to liver biopsy, per-

cutaneous transhepatic puncture of the bile duct or PV, and creation of a transjugular intrahepatic portosystemic shunt. If the bleeding is massive and the patient's condition is therefore hemodynamically unstable, the use of HVO with a balloon catheter could offer safe, easy, and fast bleeding control by significantly decreasing or stopping portal inflow, allowing successful subsequent interventional or surgical management.

## Problems and unresolved issues

Some problems and unresolved issues exist in therapeutic applications of HVO. When the target area extends over areas affected by 2 or more HVs or when marked hepatic veno-venous anastomoses exist reducing or eliminating the occlusive effect, one or more additional venous accesses and catheterizations are required. To examine the exact extent and determine the draining vein of the target area, CT scan is essential. To explore the presence of veno-venous anastomoses, hepatic venogram during balloon occlusion (occlusion hepatic venogram) must be performed before the treatment. Hepatic veno-venous anastomoses are usually accompanied by preexisting hepatic veno-occlusive conditions, and sometimes associated with diaphragmatic hernia, Osler-Weber-Rendu disease, and/or congestive liver [36]. Kawazawa *et al.* [11] and Hiraki *et al.* [14] showed marked hepatic veno-venous anastomoses in 1 of 10 patients and in 4 of 18 patients, respectively. Novak *et al.* performed occlusion hepatic venograms in 20 patients with portal hypertension and found intrahepatic and extrahepatic collaterals in 8 patients [37]. In our opinion, hepatic veno-venous anastomoses often accompany cirrhotic livers because the hepatic venules are commonly stenosed by the surrounding fibrosis in cirrhotic livers. Moreover, HVO continuing for the duration of treatment might develop new veno-venous anastomoses.

Although Hiraki *et al.* confirmed the increased HA flow in the occluded area after HVO in patients with a noncirrhotic liver, they did not examine HA flow in cirrhotic livers. Several reports have shown a blunted hepatic arterial buffer response to the altered PV flow in cirrhotic livers compared to noncirrhotic livers [8, 9]. However, it remains unclear whether the same can be applied under the situation of HVO. Further investigation should be made to clarify this point because the technique of HVO is often applied in cirrhotic livers for the treatment of HCCs.

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