4D MR Velocity Mapping using PC VIPR to Quantify Blood Flow In Portal Hypertension

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Introduction: The blood supply to the liver is unique, as it has a dual supply from the portal vein (PV) and the hepatic artery (HA). Under normal conditions the PV and HA contribute ~80% and ~20% of the flow, respectively. Portal hypertension (PH) is a clinical syndrome characterized by a pathological increase in portal venous pressure, in response to an increase intrahepatic vascular resistance. Clinically, the severity of PH can be assessed by direct portal vein catheterization or hepatic vein wedge pressure, to determine the PV-to-systemic venous pressure difference. It is an invasive procedure with risk of complications. Therefore, non-invasive quantification of hepatic hemodynamics would be highly desirable. This has been a challenging proposition using methods such as ultrasound or 2D phase contrast MRI, because volumetric coverage of complex and variable anatomy with high spatial resolution is needed. Recent developments in “4D flow” phase contrast methods 3-5 have shown exciting promise to achieve this goal. The purpose of this work was to quantify blood flow to the liver in patients with portal hypertension and compare with healthy controls using radically undersampled 4D MR velocity mapping (5-point PC-VIPR).

Methods: 5-point PC VIPR data from 10 Patients with portal hypertension (58.6±6.73years; 88.4± 6.7 kg; 8 male, 2 female) and 7 controls with no history of liver disease (32.2±10.1 years, 85.7±8.7 kg; 4 male, 3 female) were analyzed in this IRB-approved, HIPAA-compliant study. Written informed consent was obtained prior to participation. In the patients, the presence of chronic liver disease was confirmed by medical history and by a median MELD score of 9.

MR imaging was performed on a 3T clinical scanner (Discovery MR 750, GE Healthcare, Waukesha, WI) with a 32-channel phased array body coil (Neocoi, Pewaukee, WI). Image parameters included: FOV=32x32x22 cm, TR/TE=6.1-7.8/2.1-3.2 ms, flip angle=60-100°, isotropic spatial resolution of ~1.3 mm. Adaptive respiratory gating with an acceptance window of 50% lead to approx. 11 min long scan times. 10 time frames/RR cycle were reconstructed using retrospective ECG triggering and temporal view sharing.

Segmentation of the hepatic vasculature was performed using a commercial software (MIMICS, Materialise, Ann Arbor, MI). A flow visualization package (EnSight, CEI Inc., Apex, NC) was used to create and export cut-planes perpendicular to the main axis of the flow in the portal vein (PV), splenic vein (SV), superior mesenteric vein (SMV), supraceliac aorta (SCAo) and hepatic artery (HA). Figure 1 shows the anatomical model of the hepatic vasculature of a control subject as derived from the PC VIPR angiogram.

Flow measurements were conducted using a Matlab environment software 6 in which the cut-planes at 10 time frames over the cardiac cycle were imported and contoured in the regions of interest. In Black lines perpendicular to the flow direction in the vessels show the cut-plane locations in Fig. 1.

Conservation of mass was performed for internal consistency and validation by measuring blood flow at the porta-splenic confluence in two ways: 1) the PV blood flow was measured at three different locations (see Fig 1), and 2) SV and SMV flows were added and compared to the measured PV flow (see the inset in Fig 1).

Results: Figure 2 summarizes the flow measurements from the SC Ao, PV, HA and the ratios between them. Blood flow through the SC Ao was not found to be significantly different between PH patients (3.6 ± 1.8 L/min; range = 2.0 – 7.2 L/min) and controls (3.5 ± 0.6 L/min; range = 2.7 – 4.5 L/min) (p=0.9), however, larger variability was observed in the PH patients. Similar situation occurred in the PV flow, where, there was a small increase but not significant in the patients (1.3 ± 0.9 L/min; range = 3.2 – 0.4 L/min) compared to the controls (1.1 ± 0.4 L/min; range = 0.7 – 2.0 L/min) (p=0.6) and larger variability in the PH patients. The portal flow measurements were in good agreement with data found in the literature. The ratio of the portal vein flow to the total blood flow to the abdomen (Q PV(Q SCAo /Q PV)) was found to be similar between the two groups. Similar behavior was demonstrated by the ratio of the portal blood flow to the total blood flow to the liver (Q PV(+Q HA)), which was found to be similar in patients and controls.

The two validation methods that are based on conservation of mass showed similar results. The first method (measuring the flow in the PV at 3 locations) revealed an error of less that 7 ± 5 % relative to the total portal vein flow, while the second method (Q PV+Q SMV+Q SV) showed a relative error of 6 ± 4 %.

Summary: This study demonstrates the feasibility of using 4D PC-VIPR to quantify the blood flow to the liver not only in healthy controls but also in patients with portal hypertension. High variability of blood flow through the supraceliac aorta and portal vein were observed as the main differences between PH patients and controls. It is important to clarify that the severity of the patients involved in this study was relatively mild based on their MELD score (range: 1 – 16). The individual contribution of the portal vein and hepatic artery to total liver flow agreed with known values (~80%) in both groups. The error observed using the conservation of mass measurements was relatively small, demonstrating internal consistency of the flow measurements at the portosplenic confluence.


Acknowledgements: We acknowledge support from the NIH (R01 DK083380, R01 DK088925, R01HL072260 and RC1 EB010384), the Coulter Foundation, and GE Healthcare.