

Assessing Portal Vein Contribution on Hepatic Perfusion based on Arterial Spin Labeling MRI

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Target Audience: Radiologists and MRI scientists engaged in functional liver imaging.

Purpose: The human liver has a dual blood supply that comes from the portal vein and hepatic artery. The contributions from each are altered predictably in many diseases, including primary and metastatic liver tumors, liver cirrhosis and hepatic-renal syndrome(1, 2). Because most liver pathologies affect blood flow regionally, globally, or both, imaging-based approaches are often preferred (3). In addition to PET or CT-based perfusion techniques, dynamic contrast enhanced (DCE) MRI differentiates the hepatic arterial and portal venous components according to their different tracer arrival time (4). Although arterial spin labeling (ASL) MRI has been applied for hepatic perfusion (5, 6), currently there is no ASL-based approach to quantify the portal venous fraction.

Methods: The hepatic portal vein formed by the confluence of the mesenteric and splenic veins, also receives blood from the gastric and cystic veins. However, the portal vein and the proper hepatic artery are often intertwined with each other (Fig. 1), so it is difficult to individually tag each vessel. Meanwhile, hemodynamics within hepatic supplying vessels vary significantly, i.e., mean velocity of 12 cm/s and 35 cm/s in portal vein and splenic vein (7), respectively, and a peak velocity of 58 cm/s in the hepatic artery. Therefore, an ECG-triggered, FAIR-based pulse ASL technique with QUIPPS II (8) and background suppression (9) was used in this study to quantify both portal venous and the combined hepatic perfusion; thus avoiding the potential variation on tagging efficiency.

As illustrated in Fig 1, the imaging slice was placed above the splenic vein branch and just below the heart to minimize RF saturation of the spleen. The elimination of hepatic artery contribution during portal vein perfusion was achieved by the application of additional regional saturation before FAIR tagging, which saturates the entire heart and a major portion of the descending aorta above the celiac trunk branch. Any residual arterial contamination from aorta and hepatic artery was eliminated by the application of in-plane saturation RF pulses one second after FAIR labeling. This step also ensures the complete filling of tagged blood within portal vein and its tributaries.

Eight healthy subjects were recruited in this IRB-approved study. All experiments were performed on a 3T Siemens Trio scanner. The labeling duration (TI) was 3000 ms; the post saturation delay (TD) was 2000 ms such that the effective duration of labeling bolus was 1000 ms. TR was 8s to allow for complete replenishing of blood. MR parameters for single shot True-FISP were: voxel size of $1.7 \times 1.7 \times 6 \text{ mm}^3$; echo train of 1.43 ms. Each label/control pair was acquired within a single breath-holding of 12-16 s. Total of 16 repetitive measurements was conducted. Due to large hepatic transit time, single intravascular ASL signal model was adopted for perfusion quantification.

Results: Fig. 2 presents typical examples of hepatic perfusion quantification in this study. Artifacts caused by large supplying hepatic vessels are evident despite the fact that a tagging delay (TI) of 3 second was adopted. This is mainly caused by the slow portal venous flow. Portal vein perfusion is significantly lower than the combined hepatic perfusion, demonstrating the feasibility of separating two sources of hepatic perfusion. Across 8 healthy control subjects, the mean portal vein fraction is $73 \pm 9\%$ (60- 88%), consistent with the reported values in literature.

Conclusion and Discussion: In this study, we demonstrated the feasibility of pulsed ASL-based approach to estimate the portal venous fraction in hepatic perfusion. A long delay time (TI=3s) and a post-labeling in-plane saturation (1s) was adopted to account for the delayed arrival of splanchnic blood. The proposed approach will be able to detect global and regional alterations in hepatic flow pattern in liver disease patients.

References: 1. Pandharipande, et al, Radiology 2005;234:661; 2. Breedis, et al, AmJPhao 1954;30:969; 3. Itai, et al, Radiology 1997; 202:306; 4. Koh, et al, Radiology 2008;249:301; 5. Katada, et al, JpnJRadiol 2012;30:863; 6. Martirosian, et al, EJNMMI 2010; 37:S52; 7. Brown, et al, Gut 1989;30:503; 8. Wong, et al, MRM 1998;39:702; 9. Ye, et al, MRM 1997;37:226;

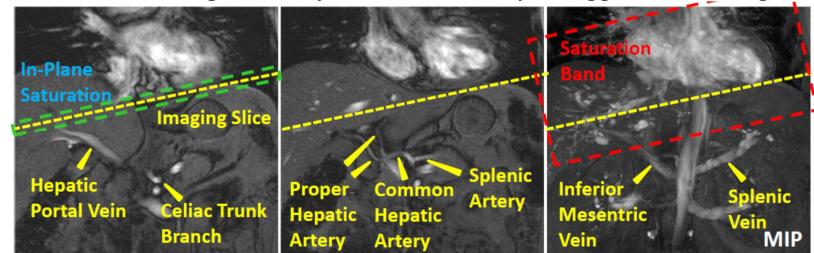


Figure 1. Blood supply for hepatic perfusion: portal vein and its tributaries. Common and proper hepatic artery arises from the celiac trunk branch of the descending aorta. Regional saturation band (before FAIR prep) and in-plane saturation (1s after FAIR prep) eliminates hepatic arterial contamination during portal vein perfusion quantification.

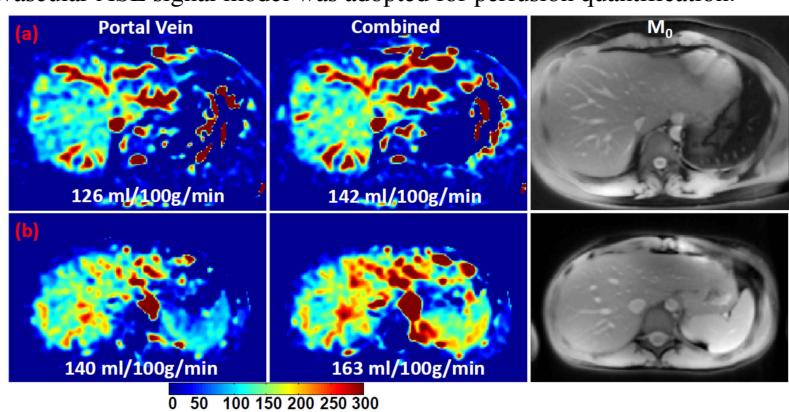


Figure 2. Map of hepatic portal vein and the combined hepatic perfusion contribution estimated on two healthy subjects. The suppression on aorta blood perfusion signal during portal vein ASL is demonstrated from the reduced ASL signal in spleen as illustrated in (b). The estimated portal vein fraction is 88% (a) and 84% (b). The mean portal vein fraction from all studies is $73 \pm 9\%$.