Portal Vein contribution to hepatic perfusion estimated using a Triple Inversion Recovery ASL Technique

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INTRODUCTION: The increase in the liver blood flow resistance is one of the common consequences of chronic liver injury like fatty, alcoholic, and autoimmune liver disease among others, and leads to the main complication of this disease: ascites, encephalopathy and esophageal varices bleeding1. The increase in the intra-hepatic vascular resistance induces many hemodynamics changes. One of the earliest hemodynamic changes is the decrease in the portal vein contribution to the liver perfusion. This is mainly due because the portal vein is a “low pressure” system compare with the hepatic artery (the other source of liver perfusion) which is a “high pressure” system. Many techniques have been proposed to detect these changes, mainly with invasive procedures. MRI techniques have been also proposed, however most of the techniques require the use of intravascular contrast agent. We have previously demonstrated the feasibility to selectively visualized the intrahepatic portal vein using a new ASL technique that does not require a subtraction step (TIR-ASL)2,3. In this work we sought to study the utility of this technique to indirectly quantify the portal vein contribution to the liver perfusion in healthy volunteers and cirrhotic patients.

METHODS: TIR-ASL uses a Triple Inversion Recovery pre-pulses and exploits the ability of the two non-selective Inversion-Recovery prepulses to null background signal while maintaining the signal of labeled blood using a regional inversion pulse (Fig.1). With the optimal selection of the inversion times TI1 and TI2, it is possible to null the static tissue and just keep the signal of the targeted blood. This sequence can be used either in one or two heartbeats interval, obtaining the same effect but increasing the labeling time (TI1+TI2) (Fig.2). We used this technique to obtain the portal vein angiogram and estimate the intrahepatic portal vein blood volume (IPVBV) that enters in two heartbeats into the hepatic circulation in healthy volunteers and cirrhotic patients, by labelling the abdominal main venous system below the liver (Fig.3). We estimated IPVBV by the segmentation of the intrahepatic portal vein pixels based in their intensity using homemade Matlab software. We corrected these measurements by the liver volume (LV) and we estimated the ratio IPVBV/LV. We estimated LV using a 3D Inversion Recovery sequence with an inversion time of 500ms. We tested this protocol in 6 healthy volunteers and 2 patients with chronic liver disease Child-Pugh B. All the images were obtained on a 1.5T Achieva Gyroscan MR scanner (Philips Healthcare, Best, NL).

RESULTS: We successfully obtained the intrahepatic portal vein angiograms. Fig. 4 shows the difference between the portal vein angiograms obtained in 2 heartbeats in a healthy volunteer and a patient with advance cirrhotic diseases. The median LV (cm³) and the IPVBV (cm³) show a significant difference in both groups. The IPVBV/LV ratio was significantly lower in the cirrhotic patients group suggesting a lower portal vein contribution to the liver perfusion (Fig. 5).

CONCLUSIONS: This preliminary result showed that it is possible to detect portal vein liver perfusion defect in advanced cirrhotic patients compared to healthy volunteer. This technique has the advantages of not requiring contrast agent injection or a subtraction step like most classic ASL techniques. Studies in patients with different level of cirrhosis are now warranted to investigate its clinical usefulness.