Perfusion MRI of the Human Cervical Spinal Cord using Arterial Spin Labeling

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Target audience: MR physicists and physicians interested in spinal cord hemodynamic assessment.

PURPOSE: Spinal cord (SC) perfusion is involved in post-traumatic recovery processes and is responsible for secondary injuries, hence it is a critical function to assess and monitor in order to establish correct diagnosis and prognosis of SC injured patients. Previous studies have demonstrated the feasibility of SC perfusion measurements on small animals using Arterial Spin Labeling (ASL) techniques, and correlations were found with SC injuries [1]. Furthermore, we have recently showed preliminary results, demonstrating the feasibility of ASL perfusion imaging on human SC [2]. This report summarizes our recent advances in this field.

METHODS: All experiments were performed on a 1.5T MRI scanner (Siemens, Erlangen, Germany) on healthy volunteers. Blood labeling strategy: The ASL technique consists of acquiring two images of the same slice: 1/ containing labeled-blood (i.e. inverted) and 2/ containing relaxed unlabelled blood. The difference image, ΔM, obtained after a transit time TI provides measurement of local tissue perfusion. Since the SC vascular network is particularly complex, exhibiting segmented blood arrivals, bi-directional vessels and watershed area, as well as patient-dependency, a FAIR labeling strategy [3] was used to ensure that a maximal amount of blood coming from various feeding arteries was labeled by the global inversion pulse before entering the slice of interest. CSF signal suppression: A dedicated CSF suppression module (Fig. 1) was played prior to the FAIR labeling/control pulses to minimize the ΔM signal arising from inflowing CSF that contaminates the perfusion image. In addition, data filtering (Hamming apodization) was performed to further reduce the spreading of residual CSF signal (Fig. 2). Finally, to overcome artifacts arising from pulsatile motion of CSF, ECG triggered acquisitions were performed with timings set such that the readout module occurs at the quiescent phase for the cervical SC [4] (~250 ms after the R wave). Imaging: A HASTE readout module, less prone to susceptibility artifacts, was preferred to EPI. Sagittal orientation was chosen first to evidence brain and SC perfusion at the same time, allowing for direct comparison. Further acquisitions were performed in the axial plane to improve delineation of SC structure. In both cases an imaging/inversion slice thickness ratio of 2 (6mm/12mm) was chosen after determination of the adiabatic inversion (15.4ms sech) and excitation pulse-profiles by Bloch equation simulations. All above-mentioned features were developed in-house and implemented in combination with the product HASTE imaging sequence. Sixty NEX data (30 control/label pairs, acquisition time 10 min) were acquired and further registered using the FSL FLIRT algorithm [5] to reduce the impact of patient motion.

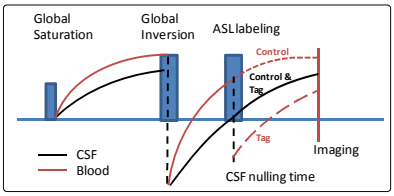

Fig. 1: CSF suppression strategy. The ASL labeling pulses are applied at the CSF signal nulling point: the CSF stays unlabeled while blood is regularly labeled. With such a scheme inflowing CSF should produce zero ΔM.

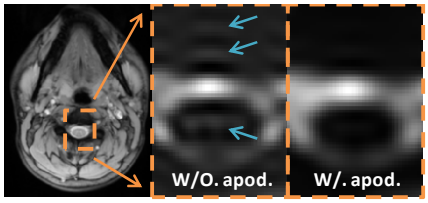

Fig. 2: Effect of apodization on the MR signal seen in the SC. Left: anatomic T2*w image. Middle & right: ΔM images w/o. and w/. apodization, acquired without CSF suppression. Secondary lobes of the CSF signal contaminate surrounding tissues (blue arrows) when no apodization is performed.

RESULTS: Figure 3 shows brain and SC perfusion images acquired in the sagittal plane for various TIs. From these images one can appreciate blood arrival in SC tissues. Moderate signal is seen in the ventral side of the SC at TI = 2s and spreads to the whole SC at TI = 3s (blue arrows), evidencing longer transit time compared to brain. An axial acquisition performed at TI = 3s is illustrated in Fig. 4. Here the ventral horn of the SC gray matter shows significant perfusion signal relative to white matter (see T2*-w image for SC structure comparison). A percentage of signal change (ΔM/M0) of about 0.9% was measured at TI = 3s in the SC for both orientations (Fig. 5). For comparison, a 2% signal change is typically obtained on brain gray matter using the same ASL sequence.

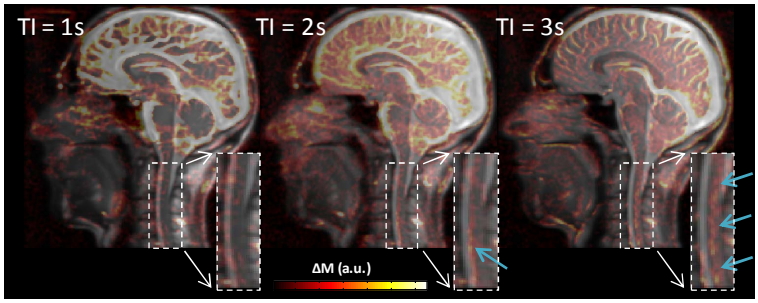

Fig. 3 : Colored ΔM images overlaid on grayscale anatomic images as a function of TI. In plane resolution of 1.9x1.9 mm^2.

DISCUSSION: Presented results are preliminary but promising as they likely depict SC vascular pattern. Current investigations are focused on optimizing the inversion time, and on improving the robustness, which currently suffers from various effects such as sub-optimal CSF suppression scheme, patient motion, partial volume effects, and stability of the ECG gating. In particular, the strong arterial signal that remains at TI =3s in the radial arteries (see Fig. 4), suggests that longer TIs would be desirable. In this perspective, experiments at higher field strength (e.g. 3T) would certainly be advantageous to improve the signal to noise ratio (longer T1s at higher B0). Image registration was also critical in our long experiments and a better robustness may be obtained with non-linear registration algorithms.

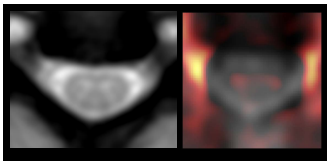

Fig. 4 : Axial perfusion image. Right: Colored ΔM images overlaid on grayscale control for TI = 3s. In plane resolution of 1.25x1.25 mm^2. Left: anatomic T2*-w image for comparison.

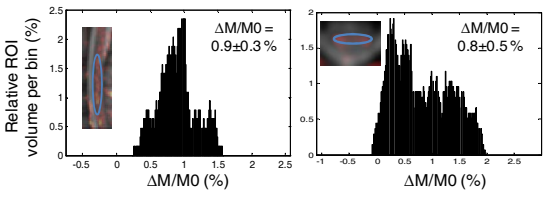

Fig. 5 : Histogram of ΔM/M0 in the SC for both sagital (left) and axial (right) orientation. Similar percentage of signal changes were measured.

CONCLUSION: This study was intended as a demonstration of human SC perfusion measurement using ASL MRI. Although the presented methods require more reliability, this work should pave the way to future developments leading to robust SC perfusion measurements, hence providing a valuable clinical tool for SC disease characterization.

REFERENCES: [1]. Duhamel et al., MRM 2009; 62(2):430 [2]. Girard et al., Proc ISMRM Perfusion Workshop 2012 [3]. Kim, MRM 1995; 34:293 [4]. Summers et al., AJNR 2006; 27:1952 [5]. Jenkinson et al., NeuroImage 2002; 17(2):825