

Combining segmentation and arterial spin-labeling to evaluate cerebral blood flow within vascular territories in children with sickle cell disease

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Purpose: Sickle cell disease (SCD) is a devastating hematological disease with manifestations including vaso-occlusive events leading to vasculopathy, brain injury and stroke. Earlier studies demonstrated generalized or territorial hyperperfusion and localized hypoperfusion of gray matter. We used MRI arterial spin-labeling (ASL) to quantify cerebral blood flow (CBF) of gray matter (GM), white matter (WM), and abnormal white matter (AWM) delineated by automated segmentation and parceled manually into vascular territories.

Method: All MR imaging was performed without contrast on a 1.5-T (Siemens Medical Systems, Iselin, NJ) whole-body imager. Patients underwent a clinical imaging protocol after IRB-approved informed consent had been obtained from the patient, parent, or guardian, as appropriate. Prospective data was collected for two age-defined cohorts of SCD patients (14 between 5-10, 14 between 11 and 18 years of age). ASL data was acquired with a pulsed ASL Q2TIPS [2] (TR/TE = 2200/13 ms) sequence developed as a work-in-progress from Siemens Medical Systems. This sequence calculated 5 slices 5mm thick with a spacing of 2.5mm centered at the basal ganglia. Conventional MR imaging sets included T1-weighted (TR/TE = 552/9 ms), PD- and T2-weighted (TR/TE1/TE2 = 4470/16/109 ms), and FLAIR (TR/TE/TI = 9140/112/2400 ms) sequences with 27 slices 5mm thick without gap covering the entire brain. The conventional MR images were coregistered to the ASL images using a normalized mutual information algorithm developed in-house. The coregistered PD and T2 imaging set provided the necessary detail for manually drawing twelve cerebral vascular territories for the 5 slices analyzed. The territories included the left and right: anterior cerebral artery (ACA), ACA Perforators, middle cerebral artery (MCA), MCA Perforators, posterior cerebral artery (PCA), and thalamic regions. The four conventional MR images were analyzed with a neural network segmentation based on a Kohonen Self-Organizing Map [1] after RF correction was performed to differentiated GM, WM, CSF, and AWM volumes for each vascular territory. CBF was quantified from the ASL data as described by Luh [2]. CBF was calculated for GM and WM in each vascular territory of each slice. Results were compared to a smaller published study of SCD patients [3].

Results and Discussions: CBF values for the vascular territories are shown graphically in Figure 1. To facilitate the comparison to the previous study, values from individual hemispheres were combined in the graph. CBF values for the segmented GM were similar to those for manually defined GM regions reported by Oguz. Values for the ACA perforators, MCA perforators, and thalamus were not reported in the previous study. Using the normalized PD and T2W images provided improved contrast for drawing the vascular territories. Automated segmentation may reduce errors associated with partial volume effects and will facilitate unbiased and automated analysis in large group studies.

Conclusion: This study replicates the findings of elevated resting CBF in SCD patients. Elevated CBF in large vessels, as measured by ultrasound, is a well established risk factor for subsequent stroke in this population. ASL probes perfusion at the parenchymal level and may provide a more sensitive indicator of brain at risk for subsequent vascular event. Automated segmentation of GM and WM for CBF quantification will facilitate prospective studies of cerebral vasculopathy and the efficacy of different treatment regimens.

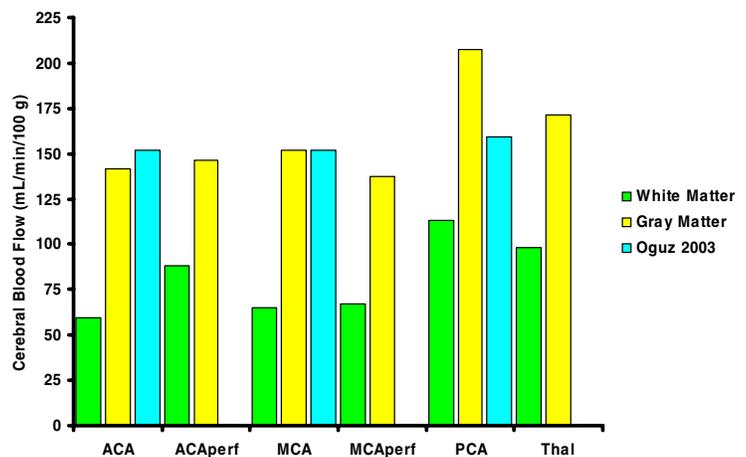


Figure 1 – Cerebral blood flow (CBF) in vascular territories. Green bars represent the normal white matter (WM), the yellow bars gray matter (GM), and the light blue values from GM reported by Oguz [3]. The plot demonstrates the similarity between the current technique and the previous study as well as the differences in CBF between GM and WM.

References: 1. Glass JO, *MRM* 52, 2004. 2. Luh W-M, *MRM* 41, 1999. 3. Oguz KK, *Radiology* 227, 2003.