

## Quantitative Cerebral Blood Flow Measurements in Symptomatic Versus Asymptomatic Intracranial Atherosclerotic Disease Using Bookend DSC-MR PWI Technique with Vascular Territory ROI Analysis

Alexander Korutz<sup>1</sup>, Parmede Vakili<sup>1</sup>, Renee Qian<sup>2</sup>, Ali Habib<sup>2</sup>, Justin Vranic<sup>2</sup>, Michael C Hurley<sup>2</sup>, Ali Shaibani<sup>2</sup>, Timothy J Carroll<sup>2</sup>, and Sameer A Ansari<sup>2</sup>

<sup>1</sup>Northwestern University, Chicago, IL, United States, <sup>2</sup>Northwestern University

### Purpose:

The third-leading cause of death in the United States is stroke with approximately 800,000 victims per year. Intracranial atherosclerotic disease (ICAD) is responsible for 7-10% of all strokes, but the risk of recurrent stroke in this population approaches nearly 12-22% at 1 year despite optimum medical management with antiplatelet/antithrombotic medications (1). Although the role of surgical or endovascular intervention in severe ICAD has been definitively relegated to patients failing medical management, we have yet to develop a risk stratification scheme to identify this susceptible population prior to refractory TIAs/strokes. We studied the differences in quantitative cerebral blood flow (qCBF) value measurements as measured by the Bookend dynamic susceptibility contrast (DSC-MR PWI) technique using vascular territory ROI analysis in symptomatic and asymptomatic patients with ICAD (2).

### Materials and Methods:

Using our institution's PACS and medical record databases we retrospectively identified 10 patients (6M:4F; 61+/-14 years) with moderate to severe intracranial stenoses (>50%) as identified with MRA within or immediately distal to the Circle of Willis (A1/A2 ACA, M1/M2 MCA, and P1/P2 PCA) that had a correlative DSC-MR PWI scan at time of diagnosis or presentation. Vascular territories were divided into symptomatic and asymptomatic subgroups that contained acute infarcts on diffusion-weighted MRI versus those that were infarct free respectively. Automated vascular territory ROI analysis was performed to include both gray and white matter within each respective arterial distribution on each subject's MR PWI maps. Individual qCBF values were generated for each vascular distribution from the DSC-MR PWI scan. Vascular territory ROI analysis for the MCA distribution was further subdivided into the inferior division (iMCA), superior division (sMCA), and M1 perforators (pMCA). qCBF values for individual territories downstream of intracranial stenoses were compared using the two-tailed Student's t-test.

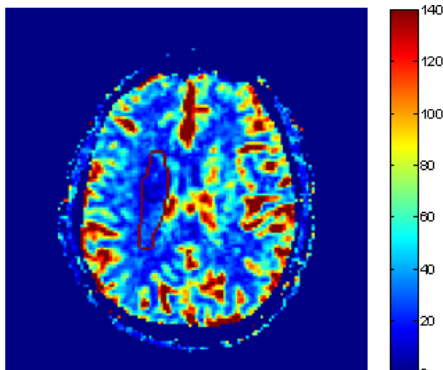


Figure 1 Quantitative CBF map of a symptomatic patient with a severe right M1 MCA stenosis and subsequent hypoperfusion. Outline of right M1 MCA

### Results:

7 vascular territories (1 ACA, 2 sMCA, 2 iMCA and 2 pMCA) were placed in the symptomatic subgroup, presenting with acute infarcts, and 9 vascular territories (2 sMCA, 2 iMCA, 2 pMCA and 3 PCA) were placed in the asymptomatic subgroup, infarct free. The mean qCBF for all vascular territories (gray and white matter) containing acute infarcts was 27.7 +/- 5.8 mL/min/100g and for all vascular territories which were infarct free was 39.9 +/- 9.4 mL/min/100g. The difference between qCBF values in vascular territories with and without infarcts was statistically significant ( $p < 0.01$ ). Figure 1 shows the qCBF map of a symptomatic patient with severe stenosis in the right M1 MCA. There is subsequent hypoperfusion in the perfused vascular territory, which is outlined in red.

### Conclusion:

Quantitative CBF measurements using the Bookend DSC-MR PWI sequence and vascular territory ROI analysis may assist in stratifying patient risk in the setting of moderate to severe ICAD as a marker for stage 2 hemodynamic compromise. Statistical differences in qCBF values between symptomatic and asymptomatic patients suggest earlier detection of unstable or refractory intracranial stenoses may be possible, allowing for more aggressive medical management or intervention.

**References** 1) Gupta R, Stroke 2008 2) Shin W et al, MRM 2007