

Measuring Venous Blood Oxygenation using Quantitative Susceptibility Mapping: A Study using Acetazolamide Challenge in Patients with Chronic Stenosis of Major Arteries

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Target Audience: Radiologists and Clinical Scientists Interested in Venous Oxygenation level and Cerebrovascular Reserve

Introduction:

Oxygenation Extraction Fraction (OEF) is an important measure of tissue metabolism and can provide valuable information about tissue viability during pathologic conditions such as ischemia¹. Furthermore, OEF measured at resting state as well as under pharmaceutical stress² could also provide valuable prognostic information for patients with chronic cerebrovascular diseases. Assuming that arterial blood oxygen saturation is constant, oxygenation of venous blood is inversely related to OEF. Because of deoxygenated hemoglobin is paramagnetic, it demonstrates oxygenation-level-dependent positive magnetic susceptibility value that can be measured using quantitative susceptibility mapping (QSM)³. In this study, we studied the feasibility of using QSM to measure the venous blood oxygenation level at baseline as well as under Acetazolamide (Diamox) challenge in a group of with chronic stenosis or occlusion of major arteries including the carotid.

Method:

Eleven patients (mean \pm SD age = 50.1 \pm 14.6 years; 7 Females) with chronic stenosis of their internal carotid and/or middle cerebral arteries were included in this study. MRI scans were performed at 3T (Tim Trio, Siemens Medical System, Erlangen) with a 12-channel head coil. Before Diamox injection, a 3D flow-compensated multi-echo gradient echo sequence was acquired for QSM with the following parameters (TR = 45ms, 6 echoes, first TE = 6.1ms, echo spacing = 5.7ms, flip angle = 12, FOV = 256 x 256mm, Matrix = 256 x 256, slice thickness = 2mm, 80 slices, GRAPPA factor = 2). One gram of Diamox was then slowly infused over a period of about 3 minutes while patients were lying still in the scanner. The 3D GRE sequence was then repeated approximately 16 minutes after the completion of Diamox infusion, when the cerebrovascular response approaches plateau. QSM processing is performed using a software package developed in-house. The phase image from each echo was first unwrapped using a Laplacian method followed by spherical mean filtering to remove the background field to generate the filtered field map. The filtered field maps from each echo were then averaged to increase the signal-to-noise ratio. An L1-norm optimization based method⁴ was used to reconstruct susceptibility map from the mean field map. Post-diamox QSM images were registered to the pre-diamox images to correct for the effect of head motions. A ROI was then placed in the straight sinus in the mean susceptibility images, and mean susceptibility values were then measured (Fig 1.).

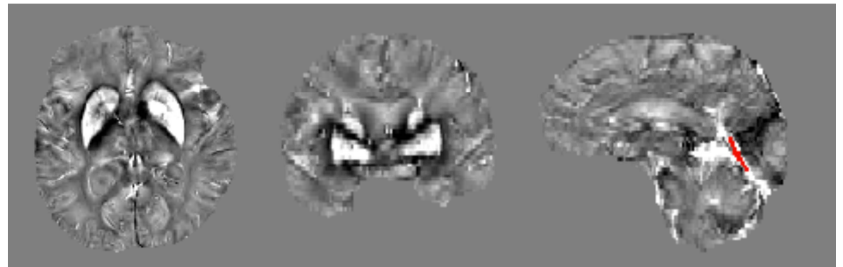


Fig 1. shows QSM images in three different planes and the placement of ROI in the straight sinus (red region).

Results & Discussions:

Since deoxygenated blood has high susceptibility value, veins show as high signal intensity on QSM images (Fig 2). Mean (SD) susceptibility value of the straight sinus among all the subjects was 0.16 (0.05) ppm and 0.13 (0.03) ppm before and after Diamox injection respectively. Among the 11 patients, 8 of them have reduced susceptibility value in straight sinus after Diamox injection, suggesting reduced oxygenation extraction fraction. This is consistent with increased cerebral blood flow due to Diamox challenge under constant cerebral metabolic rate of oxygen.

Conclusions:

Veins show as high signal intensity in QSM images due to paramagnetism of deoxygenated hemoglobin in venous blood. Diamox challenge, which increases the cerebral blood flow, decreases the oxygen extraction fraction. QSM with Diamox challenge is feasible and may provide valuable information in patients with chronic stenosis of major arteries.

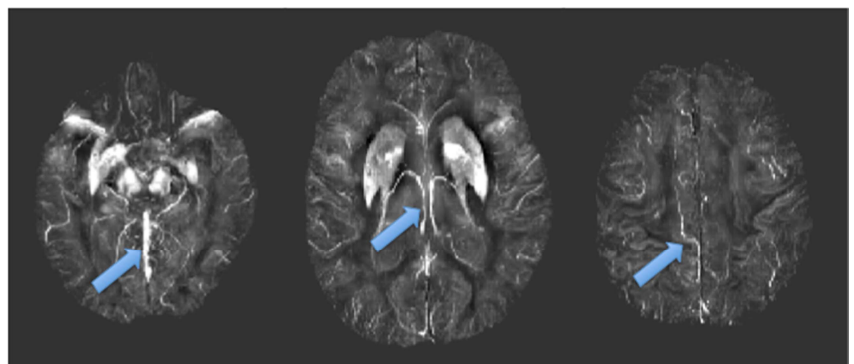


Fig 2. Veins with deoxygenated blood have high susceptibility values on MIP (maximum intensity projection) image of QSM.

References:

1. Christen T, et al. AJNR. Doi:10.3174/ajnr.A3070. 2. Gupta A, et al. Stroke. 2012; 43:2884-91. 3. Haacke EM, et al. JMRI. 2010. 32:663-676. 4. Qiu D, et al. AJNR. 35:1085-1090.