Susceptibility Weighted Imaging (SWI) of Cerebral Blood Oxygenation during Voluntary Hyperventilation and Apnea

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Introduction: Cerebrovascular reactivity and oxygenation changes during respiratory challenges have clinically important implication of brain function including cerebral autoregulation and the rate of brain metabolism. Recently, such information can be non-invasively assessed by blood oxygenation level-dependent (BOLD) functional MRI (fMRI). One major limitation of fMRI is its relatively poor resolution and complicated post-processing procedure. Susceptibility-weighted imaging (SWI) is a newly developed sequence that can potentially produce high resolution venography by exploiting the magnetic susceptibility of deoxygenated blood (1). In this sense, SWI can be considered a high resolution resting state BOLD technique. Previous studies have shown that SWI is very sensitive in demonstrating oxygen saturation changes by breathing carbogen and pure oxygen (2) or ingesting caffeine (3). Both small and large veins can be directly visualized on SWI in the normal respiratory condition. The aim of this study was to assess cerebral venous blood oxygenation changes during simple voluntary breath holding (apnea) and hyperventilation using SWI at 3.0T.

Materials and Methods: Five healthy volunteers (3 men and 2 women) with a mean age of 30.8 ± 6.5 yrs participated in this study. SWI is based on a 3D gradient echo sequence with full flow compensation in 3 directions. All data were acquired on a 3T Siemens TIM system (VB13) with imaging parameters as follows: TR/TE=29/20 ms, flip angle = 15°, matrix = 512x512, FOV=220x220 mm^2, 16 slices to cover the lateral ventricular level, and slice thickness = 1.2mm. Using parallel imaging with an iPAT factor 2, the acquisition time is 1min 28sec. SWI minimum intensity projection (mIP) images were created using both magnitude and phase images over every 4 slices to produce a 5 mm-thick SWI venographic image. After a conventional T2-weighted imaging, the baseline SWI scan with normal breathing was acquired followed by the same scan but with 30~45 seconds breath-holding starting just before the scan. This is followed by a recovery period with normal breathing about 5min. Then the same SWI scan was performed after 2 min hyperventilation, which continued during the scan. Two volunteers were asked to repeat the scan on different days.

Results: SWI showed excellent image contrast of the venous vasculature that is usually not visible on conventional imaging in the baseline scan. The change in oxygenation level, indicated by the signal change due to alteration of the concentration of deoxyhemoglobin in the veins, can be clearly seen on SWI in all volunteers. As shown in *Figure 1*, compared to the baseline scan with normal breathing, the venous architecture slightly attenuated during breath-holding, but more prominent changes were seen during hyperventilation on the SWI mIP venogram. The repeat scans in the two volunteers demonstrated the same findings.

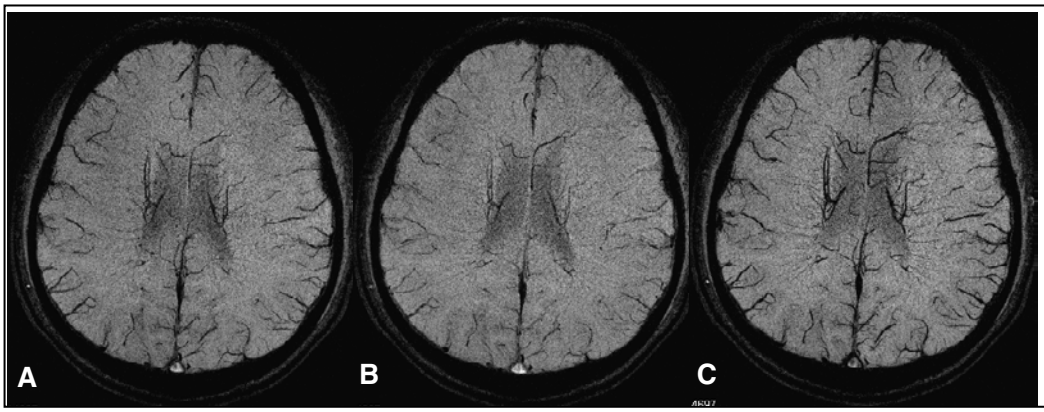

***Fig 1**. Axial SWI mIP venogram at baseline (A), breath-holding (B), and hyperventilation (C) in a healthy subject. The signal intensity changes in the venous architecture are due to oxygenation level alteration during respiratory challenge. There is a slight increase of signal in venous structures during breath-holding (B) and marked signal decrease during hyperventilation (C) as a result of different responses of vascular tone to CO_2, which is a vasoactive modulator.*

Discussion and Conclusion: This study demonstrates that cerebral blood oxygenation changes during respiratory challenge can be detected and visualized by SWI at 3.0T. Unlike BOLD fMRI, the SWI venogram specifically reveals the oxygenation change in the various sizes of veins that is not related to neuronal activity. Our data reveal the oxygenation level is much lower during voluntary hyperventilating than during breath-holding, similar to what is seen after the ingestion of caffeine (3). This is because voluntary hyperventilation can rapidly and strongly reduces cerebral blood flow (CBF) due to a large decrease of blood CO_2. This produces oxygen delivery reduction but does not decrease cerebral oxygen consumption in healthy persons (4), leading to increased concentration of deoxyhemoglobin in the veins. During breath holding (except for extreme cases), a blood flow increases significantly with a range of 59~71% in different feeding arteries as well as venous sinus (5) that transport more blood to compensate decreased oxygen saturation without increase of oxygen consumption. This study emphasizes that venous blood oxygenation is strongly influenced by CBF changes due to the modulation of CO_2 during respiratory challenge, which can be directly visualized on SWI.

References: 1) Haacke EM et al MRM (2004). 2) Rauscher A et al MRM 54(2005). 3)Haacke EM et al, ISMRM abstract (2003) 4) Van Rijen RC MRM 10 (1989). 5). De Boorder MJ et al. AJNR 35(2004).