In Vivo Measurement of Oxygenation Changes after Stroke Using Susceptibility Weighted Imaging

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Introduction: Oxygenation level is a vital parameter to monitor in intensive care units for both stoke and traumatic brain injury (TBI) patients (1). Although positron emission tomography (PET) is available for the measurement of oxygenation level, O-15 PET is invasive, has low spatial resolution, and is not as widely used anymore because of the role that functional MRI plays today in studying the brain. SWI provides a novel and non-invasive approach to this problem with high spatial resolution. By measuring blood vessel phase changes, we can obtain blood oxygen saturation levels in vivo and measuring changes in oxygen saturation over time is viable.

Materials and Methods: 10 ischemic stroke patients aged from 20 to 74 years were recruited in this study. There were 6 males and 4 females. All patients were not treated with thrombolysis during this study. NIH stroke scales (NIHSS) were assessed by an experienced neurologist. Both MRI scans and NIHSS were acquired at the three time points: 1) < 24 hours; 2) 2-3 weeks; and 3) 2 months after stroke symptoms occurred. All MR imaging was performed on a 1.5T magnet (GE Signa HD1.5T) with an 8 channel head coil. The SWI sequence used the following parameters: TE/TR=40/50 ms, FA=20°, BW=122Hz/pixel, TH=2 mm, FOV=256mm x 256mm, and an acquisition matrix=512x512. After the acquisition, the SWI data were post-processed by high pass filtering with a central matrix size of 64x64 (2). Figure 1 shows how to measure the phase differences from the same vessel $\Delta\varphi_a$, $\Delta\varphi_b$ for the two MRI scans. Here $\Delta\varphi_a$, $\Delta\varphi_b$ are the differences between venous and surrounding brain tissue. Equation [1] is used to calculate oxygenation change $\Delta Y_{b,a}$ (3,4). The average oxygen saturation level Y is assumed to be 0.55 (4). For each patient, we chose 20 veins that could be clearly seen in all three MRI scans, 10 vessels were located in the stroke hemisphere around the affected region and the other 10 were in the non-stroke hemisphere (Figure 2). The phase differences $\Delta\varphi_1$, $\Delta\varphi_2$ and $\Delta\varphi_3$ between venous and surrounding brain tissue were for the first, second and third sets of SWI phase data, respectively. We obtained the oxygen saturation level changes $\Delta Y_{2,1}$ and $\Delta Y_{3,2}$ for each of the vessels as follows:

$$-\frac{\Delta\varphi_b - \Delta\varphi_a}{\Delta\varphi_a} = \frac{\Delta Y_{b,a}}{1-Y} \qquad [1]$$

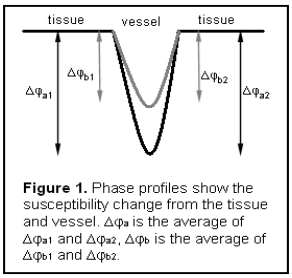

Figure 1. Phase profiles show the susceptibility change from the tissue and vessel. $\Delta\varphi_a$ is the average of $\Delta\varphi_{a1}$ and $\Delta\varphi_{a2}$, $\Delta\varphi_b$ is the average of $\Delta\varphi_{b1}$ and $\Delta\varphi_{b2}$.

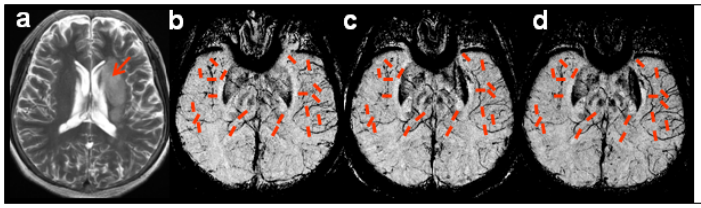

Figure 2. Vessel selection for a stroke patient. a) maximum intensity projection of T2 weighted imaging shows stroke affected area (red arrow). b), c) and d) minimum intensity projections of SWI processed phase images with 20 measured vessels which are crossed by red line for both hemisphere at (b) < 24 hours, (c) 2 – 3 weeks, (d) 2 months after the onset of stroke.

Results and Discussions: All patients underwent three NIHSS assessments. To evaluate NIHSS change, the later NIHSSs had the earlier ones subtracted from them to obtain $\Delta NIHSS_{2,1}$ and $\Delta NIHSS_{3,2}$. The oxygenation changes shown for each hemisphere were the average over the ten vessels. The correlations between the oxygenation changes and NIHSS changes for both hemispheres are illustrated in Figure 3. There was a negative correlation between NIHSS changes and oxygen saturation changes. The correlation coefficients and the p-values were r = -0.62 (p > 0.05) for Figure 3a; r = -0.70 (p < 0.05) for Figure 3b; r = -0.02 (p > 0.05) for Figure 3c; and r = -0.31 (p > 0.05) for Figure 3d. Both hemispheres demonstrated different profiles: the stroke affected side of the brain showed moderate to strong correlation between the oxygenation change and NIHSS change; however the normal side of brain had a weaker association. Our study demonstrated that after acute ischemic stroke, the clinical outcomes correlated with the oxygen saturation level around the stroke area. Previous studies proved that increasing brain oxygenation level in stroke region could increase the regional oxygen extraction fraction (OEF) (5), and improve brain tissue viability. In turn, the patient's clinical outcome improves. The regression model shown in Figure 3b was significant. The reasons why at the early stage of the stroke (3a) the correlation was not strong in the stroke hemisphere was because of the angiogenesis induced at the early stage of ischemia stroke (6), especially in the penumbra, which will influence the phase measurement $\Delta\varphi$. Although we plotted the oxygenation results averaged over 10 vessels, we found that temporal changes of oxygenation were vessel and region specific.

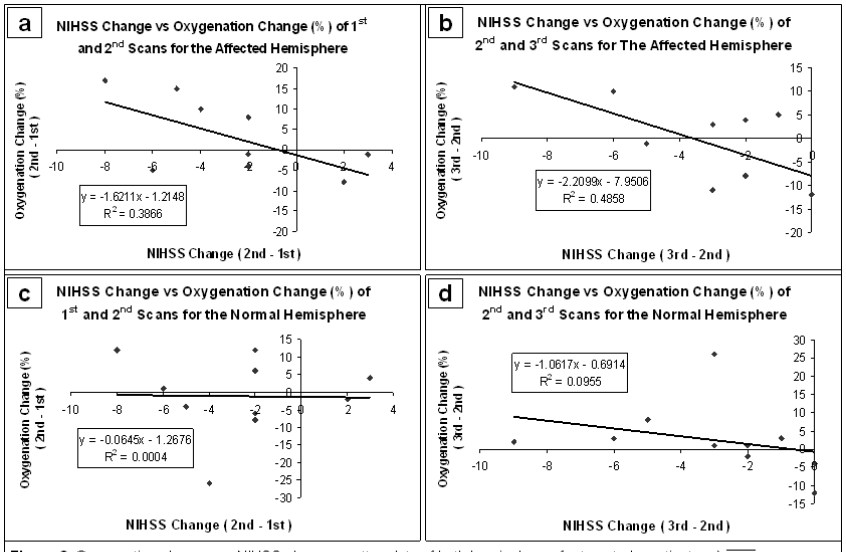

Figure 3. Oxygenation change vs. NIHSS change scatter plots of both hemispheres for ten stroke patients. a) $\overline{\Delta Y_{2,1}}$ vs. $\Delta NIHSS_{2,1}$ for the stroke hemisphere; b) $\overline{\Delta Y_{3,2}}$ vs. $\Delta NIHSS_{3,2}$ for the stroke hemisphere; c) $\overline{\Delta Y_{2,1}}$ vs. $\Delta NIHSS_{2,1}$ for the non-stroke hemisphere; d) $\overline{\Delta Y_{3,2}}$ vs. $\Delta NIHSS_{3,2}$ for the non-stroke hemisphere. Here $\overline{\Delta Y_{2,1}}$ and $\overline{\Delta Y_{3,2}}$ are the average over 10 vessels for each hemisphere.

Conclusions: SWI provided a novel method to measure the oxygenation change of the human brain in vivo in a non-invasive manner. By measuring the phase difference between the veins and surrounding brain tissue in SWI phase imaging, it is possible to estimate the relative changes of oxygenation over time with high spatial resolution. The results obtained by this study indicate that the oxygen saturation change may predict clinical outcomes of the stroke patient. The venous oxygenation changes demonstrated that SWI could reveal a dynamic panorama of venous vasculature response to the stroke in a temporal and spatial pattern.

References: 1) Furlan M, et al. Ann Neurol. 1996;40:216-226. 2) Haacke EM, et al. Magn. Reson. Med. 2004;52:612-618. 3) Haacke EM, et al. Magnetic Resonance Imaging: Physical Principles and Sequence Design. John Wiley and Sons 1999:741-779. 4) Haacke EM, et al. Human Brain Mapping. 1997;5:341-346. 5) Yamauchi H, et al. Journal of Nuclear Medicine. 1999:40:1992-1998. 6) Zhang ZG, et al. J Clin Invest. 2000:106, 829-838.