Snapshot MR-OEF for Simultaneous Imaging of Tissue Oxygenation and CVR

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Introduction: Cerebral Oxygen Extraction Fraction (OEF) has been shown to be an independent predictor of stroke risk [1]. Increased hemispheric OEF is linked to poor long term outcomes. Furthermore, the NIH/NINDS Progress Review Group has recently named tissue oxygenation imaging as a primary research goal in its August 2013 review. We have developed a means of quantifying OEF with MRI using PARSE [2,3]. Observation of OEF alteration under mild physiologic stress induced by breath-holding or throughout the cardiac cycle reflects cerebrovascular reserve which may provide complimentary information on collateralization, and correlate with favorable outcome to revascularization therapy. We present here an ICA analysis of raw free-induction decay signals, which is completely independent of the normal bias associated with anatomic land-marking.

Methods: We acquire a 2D k-space volume using a novel rosette trajectory in 65 ms for simultaneous imaging of OEF and vascular reserve. Seventeen subjects were tested under 2 transient stress conditions: breath-hold and cardiac-gated. *Breath-hold:* 20 susceptibility weighted measurements were taken ($\Delta t = 3$ secs) with an induced 15 second breath-hold. *Cardiac-gated:* Because of the speed of PARSE (65 ms) we are able to see the transient hemodynamic fluctuations induced from the cardiac cycle. Images were acquired in 25 ms intervals from the R-wave trigger. By acquiring 20 PARSE datasets and inducing a mild stress condition we are able to observe frequency shifts ($\delta\omega$) resulting from increased de-oxyhemoglobin in the draining veins of the head, similar to BOLD contrast. These 4-10 Hz shifts are de-noised using ICA with spatial coordinates defined as the length along the PARSE readout and temporal domain being the 20 time points separated by Δt= (3 sec for breath-hold and 25 ms for cardiac-gated). ICA uses blind-source separation to extract time-courses, which correspond to bulk signal enhancement associated with the stressor. Only the time courses that showed an enhancement of greater than 20% during the physiologic stress state were used to reconstruct the de-noised free-induction decay signal; allowing us to create a signal containing only the dynamic components.

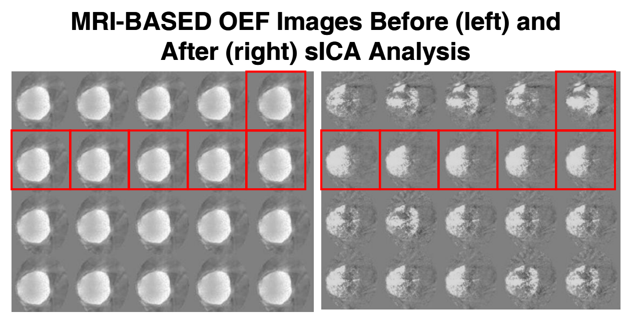

Figure 1: Twenty consecutive MR-OEF images acquired with a breath-hold (red boxes) show the sensitivity of ICA-processed MR-OEF images. Note the Occipital region OEF enhancement at baseline, and frontal shift in OEF under stress.

Results: Measured mean OEF in normal brain parenchyma of 36.87 ± 6.6% and regions in symptomatic patients reaching 84.05 ± 4.54% correlate well with literature. Figure 1 shows a comparison between 20 consecutive MR-OEF images created before and after ICA analysis. Though little can be deduced from the time-course created before de-noising, the ICA images clearly show a frontal shift in OEF during the period of induced stress followed by a return to the baseline after the stress is removed. OEF images taken during the first 125 ms of the cardiac cycle in a symptomatic patient with a right ICA stenosis are shown in Figure 2. Asymmetric hemispheric OEF (right hemispheric 13.06% elevation) is clearly visible. We also see a non-uniform flush in of de-oxygenated hemoglobin with a subsequent uneven outflow, unseen in healthy volunteers, suggesting regions of compromised cerebral vascular reserve.

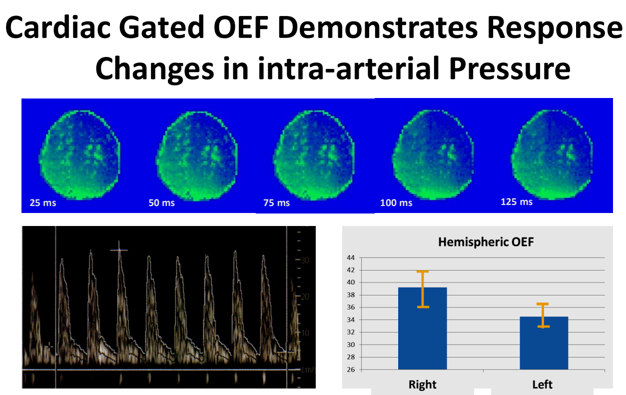

Figure 2: OEF images taken in the first 125 ms of the cardiac cycle in a symptomatic patient with a right ICA stenosis. Notice the early flush and subsequent flow out, showing sensitivity to intra-arterial pressure waveforms (seen in lower left). Also notice the increased OEF in the patient's right hemisphere

Discussion/Conclusion: We have found that MR-PARSE has high sensitivity to frequency shifts induced by transient alterations in de-oxyhemoglobin. The use of ICA to extract and quantify cerebrovascular reactivity represents a new and simple, non contrast approach to stratifying patients toward therapies to prevent stroke.

References: [1] Derdeyn, Brain 2002, [2] Menon, JCBFM 2014, [3] Twieg, MRM 2002

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