

EVIDENCE OF VASCULAR STEAL IN THE CEREBRAL CIRCULATION

J. A. Stainsby¹, J. Poubanc², A. Crawley², D. M. Mandell², J. S. Han³, J. A. Fisher³, and D. J. Mikulis²

¹GE Healthcare, Toronto, Ontario, Canada, ²The Toronto Western Hospital, Toronto, Ontario, Canada, ³Anesthesiology, Toronto General Hospital, Toronto, Ontario, Canada

Introduction: Cerebral vascular reactivity (CVR) is a promising technique for investigating the brain's autoregulatory capacity [1]. Such studies can be performed using a BOLD MR imaging technique combined with inhaled carbon dioxide (CO₂) as the vasodilatory stimulus. In normal brain tissue the CO₂ stimulus prompts global cerebral vasodilation, resulting in a global increase in BOLD signal. In diseased tissue, regions of negative BOLD signal can occur. The underlying physiologic process that results in a negative BOLD signal has not been conclusively determined. Here we present support for flow shunting away from diseased tissue, the so-called "steal" hypothesis.

Theory: One hypothesis that has been proposed that could contribute to a negative BOLD signal response is a "steal hypothesis" [2]. This hypothesizes that regions of damaged tissue may have limited or no capacity to vasodilate during the CO₂ stimulus and thus limited or no capacity to drop the flow resistance for blood flow. The flow resistance will drop in tissue that does vasodilate and this should result in a preferential shunting of flow to healthy, vasodilating tissue. If this hypothesis is true, then we expect that the timing of the flow response in the damaged tissue should be dictated by the timing of the resistance changes in the global (healthy) tissue and not dominated by the arrival and presence of the CO₂ stimulus at the site of the damaged tissue. In this situation we would expect to see regions of tissue with negative reactivity, with delayed blood transit times relative to regions of healthy tissue but with BOLD signal response times that are not delayed relative to regions of healthy tissue.

Methods: We retrospectively analyzed CVR data acquired as part of standard clinical protocols at our institution acquired using a BOLD EPI acquisition on a GE 3T Signa scanner on patients that also received a dynamic susceptibility contrast (DSC) study during the same examination. From the BOLD EPI data, maps of the CVR response (% signal change per mmHg) and maps of a time delay (TD) parameter characterizing the BOLD response times [3] were generated. From the DSC data, maps of the time to minimum signal (TM) were made. Regions of interest in an area of negative reactivity and in a region of positive reactivity with the shortest TM time were drawn.

Results: Sample CVR, TD and TM maps are shown in Figures 1-3. The corresponding regions of interest for signal analysis are shown in Figure 4. The mean TM time in the region of negative reactivity (5.0 s) is much longer than the TM time in the region of positive reactivity (2.6 s) whereas the TD value is similar, and in fact shorter, in the region of negative reactivity (9.8 s) compared to the region of positive reactivity (13.9 s).

Discussion: The tissue in the region demonstrating negative reactivity shows significantly delayed blood transit times on the DSC maps. This suggests that the delivery of the CO₂ stimulus (which is carried in the blood from the lungs) will be correspondingly delayed. However, a measure that is characteristic of the time response of the BOLD signal changes is similar, and actually slightly shorter in the region of negative reactivity. This characteristic time is influenced both by blood transit times and by vascular response times (time over which the vasodilatory response to CO₂ develops). The lengthened response time in normal tissue is consistent with regions of strong reactivity. In addition, the differences in this characteristic time compared to the transit delay differences (measured by TM) has previously been shown to be dominated by a lengthened return to baseline of the BOLD signal following the end of the CO₂ stimulus [4]. The time course of the BOLD signals does not exhibit any significant relative delays between the two regions. This behavior strongly supports the hypothesis of a flow "steal" phenomenon since the BOLD signal response in the area of negative reactivity appears to happen prior to the arrival of the CO₂ stimulus.

References: [1] A Vesely, MRM, 45, 2001, [2] A Shiino, et al, JCBFM, 23, 2003, [3] J. Poubanc, et al., Proc ISMRM, #1235, 2005, [4] A. Hetzel, et al., Cerebr Dis, 16, 2003.

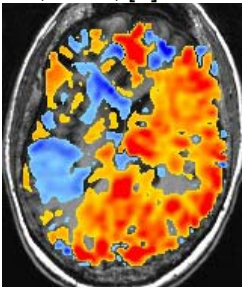


Figure 1: CVR map, dark blue to dark red represent - 0.56 to +0.56 %signal / mmHg

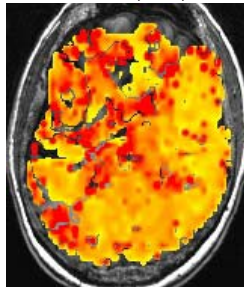


Figure 2: Characteristic BOLD time delay map. Colors range from 0 (yellow) to 45 (dark red) seconds.

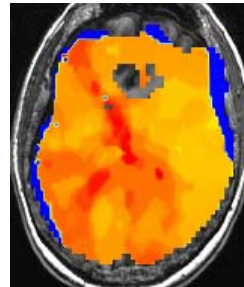


Figure 3: Time to minimum map from DSC study. Colors range from 22 sec (yellow) to 32 sec (dark red)

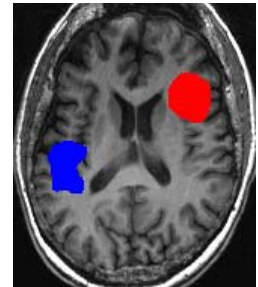


Figure 4: ROIs used for analysis. Blue = region in area of negative reactivity. Red = remote region in normal tissue.