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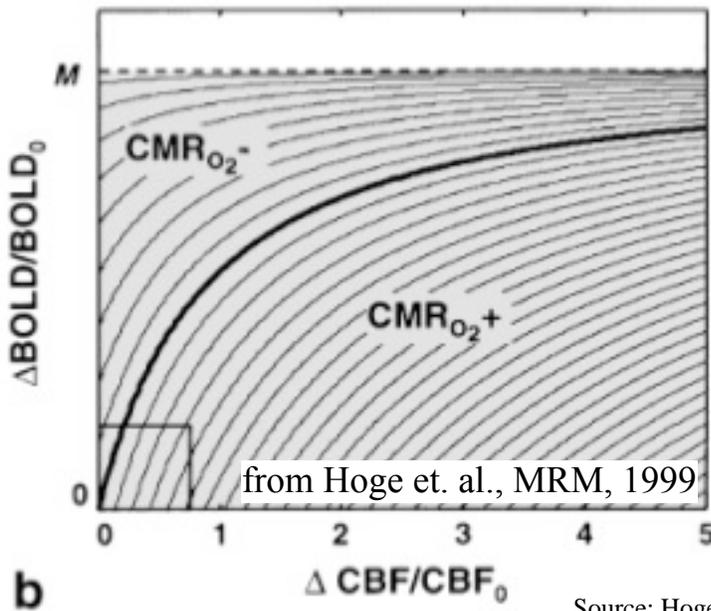
HST.583 Functional Magnetic Resonance Imaging: Data Acquisition and Analysis  
Fall 2008

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## Problem Set 3: Imaging Physiology Block

Due: October 31, 2008

1. The figure below shows the BOLD effect's "ceiling" (i.e.  $M$ ), a theoretical region where a change in blood flow will not yield a change in the BOLD signal. From a physiological standpoint, why do you think this could happen?



Source: Hoge, R., et al. *MRM* 42 (1999): 849-63.

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2. SE-BOLD theoretically can eliminate extravascular BOLD signal around larger veins, thereby more specifically targeting late capillaries and early venules. Why would this be advantageous? Given conventional scanners available at most imaging centers, why do you think this is not routinely done?
3. List two benefits and two drawbacks of going to higher fields for BOLD imaging? Explain.
4. Right now there are pulse sequences that can acquire data at incredibly fast rates; with TRs on the order of a few milliseconds. Explain why this may *not* be incredibly useful for doing BOLD fMRI?

Three photos removed due to copyright restrictions.

Figure 1a, b, and c in Schwartz, W. J., et al. "Metabolic Mapping of Functional Activity in the Hypothalamo-Neurohypophysial System of the Rat." *Science* New Series 205, no. 4407 (August 17, 1979): 723-725.

5. The figure on the left from Schwartz and colleagues summarizes one of the first experiments to use autoradiographic techniques to map glucose metabolism (1977). They subjected rats to an osmotic load which stimulated cell bodies in the PVN and SON (a). Interestingly, they saw a large increase in metabolism in the posterior pituitary - the terminal end of PVN/SON axons (c), but did *not* see an increase in metabolism near the SON/PVC nuclei (c). Comment on this finding in light of Logothetis' observation that fMRI signals are more highly correlated to LFPs than APs.

6. In lecture 3, we asserted that the BOLD response is roughly *linear*. In reality, there are definitely some non-linear components to the signal. Speculate on where non-linearities could arise, considering the problem from a neuronal, metabolic, and vascular point of view (i.e. one example for each; i.e. three total).
7. CBF increases in response to neural activation. Most evidence suggests that this occurs by increasing capillary blood velocity, *not* by capillary recruitment. Imagine for a second, that this is actually wrong; i.e. there *is* significant capillary recruitment during neuronal activation. Does this alter our view of the oxygen limitation model as an explanation of why CBF increases significantly more than  $CMRO_2$  during activation?

8. (Extra Credit) As we saw in Lecture 4, water diffusing around small vessels experience a range of fields, such that phase offsets acquired in the first half of the experiment are different from those acquired in the second half. This leads to an *inability* to completely refocus the signal during a spin echo experiment, resulting in a small but measurable phase change between activated and baseline states (*circled*). This  $\Delta$ phase alters the  $T_2/R_2$  and produces a modest change in the SE-BOLD signal (*thick red double arrow*).

We also discussed why this effect is reduced around large vessels (*thin red double arrow*): the field size is large compared to the diffusion size; water molecules feel a *constant* field, leading to a *linear* phase accrual, which can be reversed with a spin echo.

We did not discuss, however, the fact that the effect peaks around vessels with radius of  $\sim 7 \mu\text{m}$ , and *decreases* with smaller vessels. At a vessel radius of  $1 \mu\text{m}$  (for example), the spin echo sequence does *not* cause much of  $T_2/R_2$  change (*thick blue arrows*). Explain why this is the case.

