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PROGRAM #

Numerical Simulations of the Oxygenation Dependence of the T2 and T2* of Whole Blood using a Deterministic Diffusion Model

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INTRODUCTION:

Changes in blood oxygenation cause changes in susceptibility - related intravascular frequency shifts and changes in intravascular spin coherence. In the context of functional MRI (fMRI), both of these effects are weighted by the relative intravascular signal, which is dependent, among other things, on the T2* and T2 of blood. Extravascular spin coherence; modulated by blood susceptibility - induced extravascular gradients, is not weighted by blood T2 and T2*.

To accurately predict the blood oxygenation level dependent (BOLD) signal changes obtained either with gradient-echo or spin-echo sequences, and across possible vessel distributions, (voxels having a homogeneous distribution of capillaries to voxels that are completely within intravascular space), it is necessary to accurately know the oxygenation - dependence of whole blood T2 and T2*. Studies of the T2 of blood have been published (1-4), but none, to date, have directly studied blood T2*.

A simplified model of red blood cells in plasma, based on the deterministic diffusion methodology of Wong et al.(5) was created using physiologic and biophysical parameters obtained from the literature. The simulated results convergently matched experimental the results of Wright et al.(3) and Hoppel et al.(6).

The hemoglobin susceptibility related effects on blood T2 and T2* are also simulated across a range of possible biophysical and physiologic conditions. The relative significance of these effects are assessed in the context of existing BOLD contrast models.

METHODS and RESULTS:

The basic simulation methodology (5) involves stepwise 2-D Gaussian convolution and phase rotation to simulate diffusion in the presence of off-resonance effects. The inter-step time was 0.25 μ s. Field overlap from adjacent red blood cells was considered. Blood susceptibility values of Weisskoff et al. (7) were used. Red blood cell geometry was simplified to randomly oriented and distributed infinite cylinders having 2.5 μ m radii. First, using a multi-echo simulation with identical parameters to those used experimentally in (3), blood T2 vs. %HbO₂ curves were generated at different inter - 180 pulse times (τ). The best fit to the data of Wright et al. (2) was with D equal to 1.8 μ m²/ms, shown in Figure 1. Field strength = 1.5 T. Hematocrit = 44. T₂₀ = 250 ms.

The Δ R2* and Δ R2 that occur with a change in whole blood %HbO₂ from 60% to 70% were also simulated at different values of D. Figure 2 shows Δ R2* and Δ R2 on the left axis and Δ R2*/ Δ R2 on the right axis. At D = 1.8 μ m²/ms, the ratio was 1.5; corresponding to the Δ R2*/ Δ R2 ratio for whole blood reported by Hoppel et al. (6) using a similar oxygenation perturbation.

While the model makes several simplifying assumptions, simulated dependencies of the transverse relaxation of blood on hematocrit, cell integrity, and field strength showed general agreement with literature values.

CONCLUSIONS:

The implications of these studies are important in the context of fMRI: whole blood T2 and T2* effects are significant in several ways. One is regarding voxels that are completely contained within a draining vein (100% blood volume - as may be the case for many voxels in high resolution fMRI studies). With an activation - induced %HbO₂ change from 60% to 70%, simulated gradient-echo and spin-echo percent signal changes (TE=60ms) are

30% and 18% respectively. These "large vessel effects," which are caused by frequency shifts within red blood cells and which are vessel orientation - independent, are not removed by the use of spin-echo sequences.

Even in voxels that have a relatively small blood volume fraction, the intravascular component of fMRI signal changes is highly significant (8). Accurate determination of blood relaxation rates is necessary for the assessment of these intravascular dephasing effects.

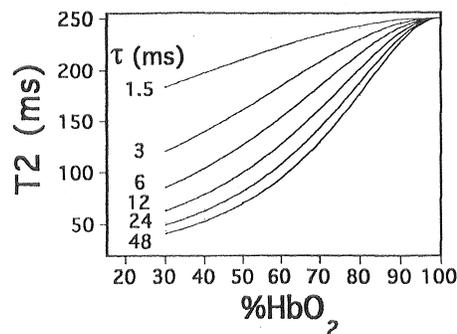


Figure 1: Hct = 44, B₀ = 1.5 T, T₂₀ = 250 ms, cell radius = 2.5 μ m. Curves obtained at this value of D (1.8 μ m²/ms), give the best fit to those of Wright et al.(3).

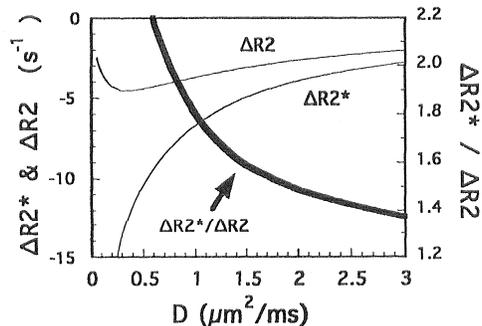


Figure 2: Dependence of Δ R2* and Δ R2 (left axis) and Δ R2*/ Δ R2 (right axis) on D. The change in %HbO₂ was from 60% to 70%. Hct = 44. T₂₀ = 250 ms, τ = 48 ms. The ratio at D = 1.8 μ m²/ms (\approx 1.5) matches reported results of Hoppel et al. (6).

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