

Effects of diffusion on MR signal under various microvasculature

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Introduction:

Paramagnetic deoxyhemoglobin within capillaries and veins can induce magnetic field variations around these vessels. MR signal decay is governed by both static and diffusion induced dephasing effects. It has been suggested that diffusion effects highly depends on MR pulse sequence, echo time, and vessel size. Previous studies have proposed models with simplistic assumptions such as uniform vessel radii within a voxel [1], or explore the morphology of cerebral cortex microvasculature that might help modeling [2]; however, a realistic mathematical model that can embrace the vast variety of microvasculature in various regions of the human brain has not been established to date. This also complicates the quantification of diffusion effects on the measurement of hemodynamic parameters. In this study, we aimed to shed light on how diffusion affects the MR signal under different vascular configurations pertaining to distinct regions.

Methods:

We have simulated a triple-echo asymmetric spin echo (ASE) sequence with echo times TE1/TE2/TE3 = 44/62/80 ms to acquire signal from a single voxel. Vessel orientations and capillary radii were randomly generated as previously described [1, 2]. More explicitly, vessel orientations were chosen by randomly picking a point on the uniform sphere and the inverse square root of capillary diameters were drawn from a normal distribution so as to yield radii that are mostly in the 2-7 μm range. Table 1 lists the relative cerebral blood volume (rCBV) and radii of vessels other than capillaries. The CBV was shared among these vessels in rough accordance with the results reported in [3]. Three diffusion scenarios were considered: static dephasing [diffusion coefficient $D = 0$], severely compromised diffusion [$D = 0.4\text{e-9 m}^2/\text{s}$] and normal diffusion [$D = 0.8\text{e-9 m}^2/\text{s}$]. The signal due to the analytical model proposed in [4] was also computed for reference. In all cases, only the extravascular spins contributed to the overall signal.

	White matter	Gray matter 1	Gray matter 2
rCBV [%]	2%	4%	6%
Vessel radii [μm]	2-7, 25	2-7, 25, 45	2-7, 25, 45, 80, 200

Table 1 – Simulated Configurations

Results:

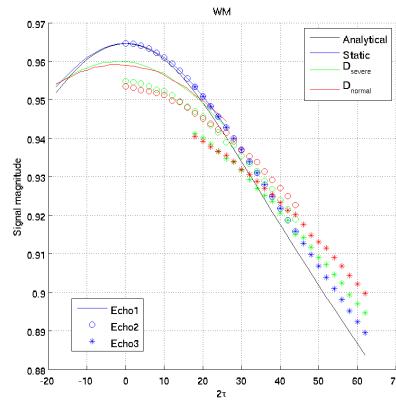


Figure 1

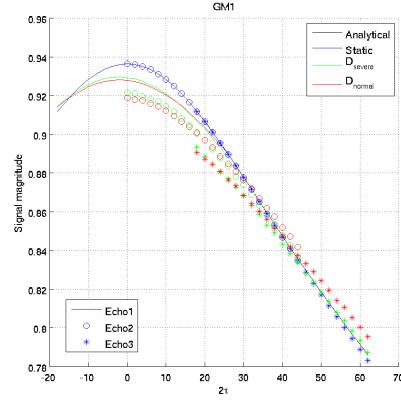


Figure 2

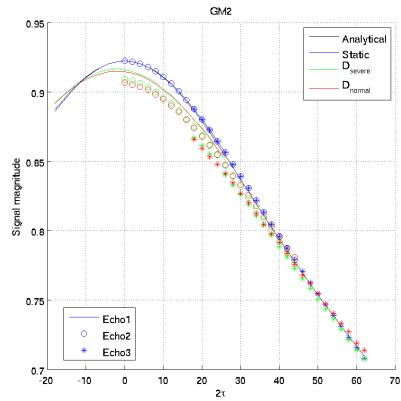


Figure 3

Figures 1, 2 and 3 show the analytically calculated curves as well as the signals under three diffusion scenarios for, respectively, the WM, GM1 and GM2 configurations. Figure 4 is simply a compilation of the normal diffusion curves, i.e. curves corresponding to the highest diffusion coefficient, out of the first three figures.

Discussion and Conclusions:

Figure 1, Figure 2 and Figure 3 consistently exhibit that the signal intensity at spin echo decreases as the diffusion coefficient is increased; however, the intensities at the gradient echoes, i.e. the second- and third-echo signal intensities, experience less reduction while the static dephasing agrees with the analytical model. This phenomenon can be explained by the fact that, when strong enough, diffusion to some extent nullifies the susceptibility effect imposed by the vessels. It suggests that MR signal decay cannot be simply described as a linear addition of two decay rates with R_2' representing the reversible static dephasing effect and R_2 representing irreversible diffusion induced dephasing effect. Another observation is how this nullification is related to the vessel size. Consistent with what was reported in [1], as the “effective” vessel size of the vasculature increases beyond about 10 μm , the effect of diffusion becomes less pronounced. This is why we see the curves almost merge for the GM2 configuration. Finally, as readily observed in the first three figures, when no diffusion exists, the three echoes line up to yield a single curve. However, as Figure 4 clearly depicts, the diffusion effect is time-dependent and causes the signals from different echoes to show different behavior over the same range of 2τ , the shift in spin echo time.

References:

[1] Boxerman et al., MRM 34: 555-566, 1995, [2] Cassot et al., Microcirculation, Vol. 16, 4:331-344, 2009, [3] Moskalenko et al., Biophysical aspects of cerebral circulation, Pergamon, Oxford, [4] Yablonski et al., MRM 32: 749-763, 1994

