Oxygenation and hematocrit dependence of blood T2 and T2* at 3T

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INTRODUCTION

3T is becoming a new standard for clinical MR scanners. Quantification of the relaxation times of different tissue types at 3T is needed to identify tissue pathologies or to quantify functional activity in the brain. Values of T_1 and T_2(*) of white matter (WM) and gray matter (GM), and T_1 of blood have been published [1-3], Here, we report in vitro blood T_2(*) measured as a function of oxygenation and hematocrit under physiological conditions at 3T. These values provide a means to calculate the BOLD response inside vasculature under conditions of known oxygenation change and, using a parenchymal model, allow us to simulate the intravascular contribution to the parenchymal BOLD response, similar to Silvennoinen et al. [4]. When combined with extravascular responses [5], the total BOLD effect can be simulated [4].

MATERIALS & METHODS

BOLD Modeling. We consider a slow-exchange, 5-compartment voxel model consisting of GM parenchyma (arterioles + capillaries + venules + tissue), WM, CSF, arteries, and veins. The total signal in a voxel is [4]

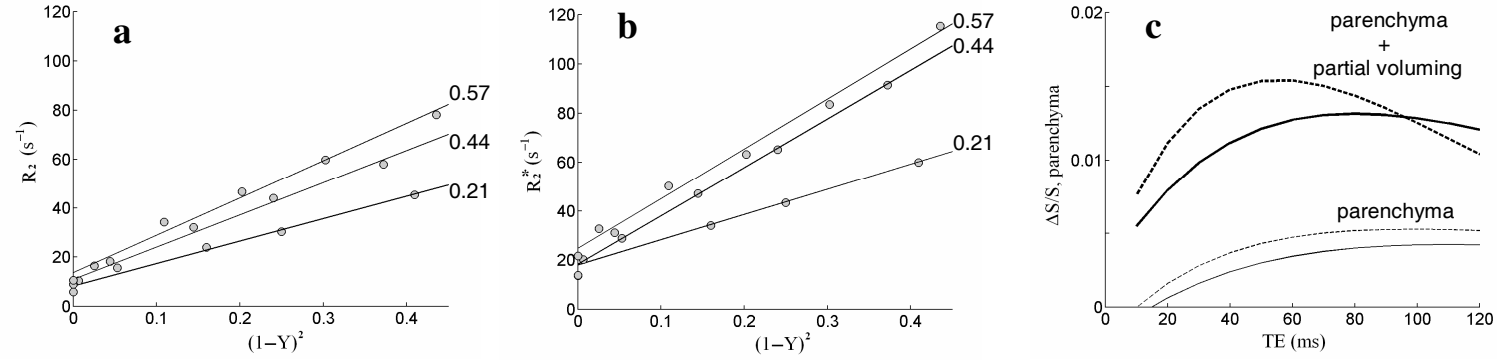
$$S_{voxel} = x_{GM} S_{GM} + x_{WM} S_{WM} + x_{CSF} S_{CSF} + x_{art} S_{art} + x_{vein} S_{vein} \qquad (1)$$

The sum of molar fractions (x_i) inside the voxel is normalized to 1; the signal intensities (S_i) are calculated from T_1 and T_2(*) of the different tissue types and the experimentally used TE, TR, and flip angle. The GM parenchyma is considered a subcompartment within which the microvascular blood volume changes during brain activation, but the total water volume remains constant; detailed calculations can be found in [4]. Together with the measured T_2 and T_2* of blood, Eq. (1) yields the total signal changes in spin-echo (SE) and gradient-echo (GRE) BOLD experiments.

MRI. A home-build blood perfusion system was used to maintain oxygenation level and blood circulation during MR experiments. All experiments used bovine blood and were performed on a 3T Philips Achieva scanner at 37°C. We used single spin echo sequence with TE = 20-200ms and TR = 3s to measure T_2, and single gradient echo sequence with TE = 10-70ms and TR= 3s to measure T_2*. Relaxation times were determined from single exponential fits to signal intensity as a function of TE. Data were analyzed with a quadratic model [6]: $R_2^{(*)} = A^{(*)} + B^{(*)}(1-Y) + C^{(*)}(1-Y)^2$ with $B^{(*)} = 0$, for the dependence on the oxygen saturation fraction, Y.

RESULTS & DISCUSSION

Figs. a and b show R_2 (= $1/T_2$) and R_2*(=$1/T_2$*) as functions of $(1-Y)^2$ at three hematocrit (Hct) levels. The R-squares of the linear regressions are >0.98, suggesting that the assumption of B* = 0 is valid. This quadratic dependence is in line with findings at 1.5T and 4.7T [4]. The data show that 65-90% of the gradient echo BOLD effect in blood is due to contributions of changes in T_2, depending on Hct. **Fig. c** shows SE (solid line) and GRE (dashed line) BOLD simulations for typical visual activation changes in blood flow, oxygenation, and volume [4]. This is done for pure GM parenchyma (thin lines) and GM parenchyma with partial voluming of 10% CSF [7], 5% WM, 2% veins, 0.8% arteries. The parenchymal signals change by 0.30% for SE and 0.43% for GRE at TE = 50ms. With the partial volume of 2% veins whose T2(*) sensitively depend on the blood oxygenation level, the effect rises to 1.2% for SE and 1.5% for GRE



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