Host Tissue Dependence on the Tracer Relaxation Effect in Spin Echo

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Introduction

The in vivo concentration of MRI contrast agents (CA) cannot be measured directly and is commonly determined indirectly using their relaxation effect. This requires knowledge of the CA relaxivity in the target tissue. Quantitative perfusion studies involve measurement of CA concentration during a bolus passage. Relaxation is commonly converted into concentrations assuming identical relaxivities for tissue and blood (1,2). It is known that the relaxivity of blood pool CA depends significantly on the host tissue for gradient echo (GE), (10). The spin echo (SE) pulse sequence is promising in perfusion measurements because SE is more sensitive to the microvasculature, (12). Understanding the relaxation effect of brain tissue and blood in the presence of CA is important for the quantification of perfusion MRI, functional MRI and to vessel size imaging.

The aim of the current study was to compare the tissue relaxation obtained using SE at 1.5 T and newly measured blood relaxation data at 1.5 T.

Methods

The experiment was performed as follows. A phantom consisting of test tubes containing whole blood (hct = 0.3 and hct = 0.5) with five different concentrations of GdDTPA-BMA (in the range 1 to 15 mM) was used (Figure 1). Imaging was performed at 1.5 T (Siemens Sonata) using a multiple single echo pulse SE sequence with TE = 3.9, 10, 20, 30, 40, 50, and 60 msec. Relaxation rates were calculated from a mono-exponential fit of the signal intensity versus echo time data and the dose response as a function of blood hct was plotted.

The calculation of the tissue relaxation was performed using the signal simulation program described in Ref. (10) for both gray matter (GM) (ζ = 0.06) and white matter (WM) (ζ = 0.025), where ζ is the blood volume.

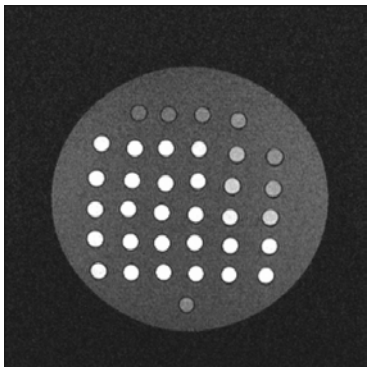

Figure 1 The blood phantom

Results

The change in relaxation rate, ΔR_2, of CA for SE is shown in Figure 2 as a function of the tissue concentration of CA, c. The latter is related to the blood concentration, c_b, via $c_t = c_b/\zeta$. This dependence is nonlinear which reflects the proportionality of ΔR_2, to the power 2/3 of the magnetic susceptibility of blood in the static dephasing regime, (4). The relaxation of tissue versus CA concentration is much smaller than for GE due to rephasing of the dephasing effect caused by the extravascular field inhomogeneities, as also found by Boxerman et al. (12).

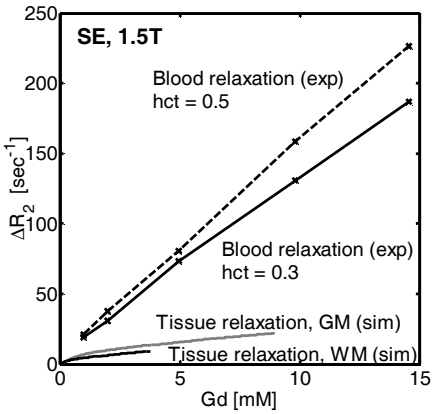

Figure 2 The change in relaxation of tissue (simulations) and blood (experiments).

Discussion

The relaxation of Gd in blood derived from SE sequences is almost as large as for GE, Ref. (10). This can be understood qualitatively in terms of the microscopic contribution (on the molecular level) and the mesoscopic contribution (due to the compartmentalization of the contrast agent in plasma) to the relaxation effect. The latter depends on the cell shape, hct, and the rate of exchange between plasma and erythrocytes. A quantitative theory is therefore currently missing.

The tissue relaxation is much smaller for SE than for GE, so in order to get a significant tissue signal drop in perfusion a higher concentration of CA is required. Note that using a high concentration of CA gives rise to signal void of the AIF signal that comes from pure blood, as demonstrated for GE in (13).

It was demonstrated previously (6) that the perfusion measurements using dynamic susceptibility contrast inherently overestimate the cerebral blood flow and volume due to the significant difference in the relaxation effect of the CA in brain tissues compared to blood, (10). In this study (figure 2) we demonstrate that for SE the tissue and the blood relaxation effect depends on many factors, and that the grey-white matter contrast is affected. The complicated shape of the relaxation of tissue in the presence of CA for SE (nonlinearity and CBV-dependence) is important for all methods that involve the relaxation effect of different tissues.

References

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