Use of the relationship between phase and magnetic susceptibility for assessment of assumed contrast agent distributions in vivo: Application to $\Delta R2^*$ maps in dynamic susceptibility contrast MRI

E. Lindgren¹, L. Knutsson¹, D. van Westen², F. Ståhlberg^{1,3}, and R. Wirestam¹

¹Dept. of Medical Radiation Physics, Lund University, Lund, Sweden, ²Radiology, Skane University Hospital, Lund, Sweden, ³Dept. of Diagnostic Radiology, Lund University, Lund, Sweden

Introduction

Quantification of magnetic susceptibility based on MRI phase data is a promising tool for obtaining additional knowledge about brain structure and composition [1, 2]. This method may also become useful in connection with contrast agents [3], since the introduction of contrast agent into a compartment is reflected by increased susceptibility. However, susceptibility quantification by deconvolution of measured phase maps is an ill-posed inverse problem to which an optimal solution is presently lacking, although promising approaches have been proposed [1, 2]. An interesting intermediate step would be to compare measured phase maps Φ_m (reflecting true local susceptibility in the object) with artificial phase maps Φ_a calculated by convolution of an assumed susceptibility distribution. In the present study, $\Delta R2^*$ maps were assumed to represent contrast agent concentration C, as is traditionally the case in dynamic susceptibility contrast MRI (DSC-MRI). However, there are strong indications that T2* relaxivities differ between different *in vivo* compartments, for example, between blood and tissue, and a comparison between artificial phase maps (based on convolution of $\Delta R2^*$ data) and measured phase maps might thus serve as an interesting supplement to previous simulations related to this topic [4].

Method

The relationship between phase $\Phi(x, y, z)$ and local magnetic susceptibility of the object $\chi(x, y, z)$ is given by Eq. 1 [5, 6]:

$$\Phi(x,y,z) = \gamma \cdot B_0 \cdot TE \cdot FT^{-1} \left(\frac{1}{3} - \frac{k_z^2}{K^2} \right) \otimes \chi(x,y,z),$$
 [1]

where " FT^{I} " denotes the inverse Fourier transform, k_z is the z-axis coordinate in k-space and $K^2 = k_x^2 + k_y^2 + k_z^2$. Convolution (denoted " \otimes ") was carried out by multiplication in k-space. Signal and phase data were acquired, following a bolus injection of gadolinium contrast agent (0.1 mmol/kg bw, Dotarem), using a flow-compensated EPI pulse sequence (3 T Philips Achieva, TE=29 ms, voxel size=1.72×1.72×5 mm³, 23 non-angulated axial slices, 1 mm slice gap, temporal resolution 1.36 s). Measured phase maps were constructed using pair-wise subtraction and subsequent summation [7]. Maps of $\Delta R2^*$ = -(1/TE)·ln(S/S₀) at peak concentration of contrast agent were calculated and converted to artificial phase maps according to Eq. 1, under the assumption that $\Delta R2^* \propto C \propto \chi$. In DSC-MRI, $\Delta R2^*$ has often been used as an approximation to contrast agent concentration, under the implicit assumption that the T2* relaxivity is equal for all compartments (e.g., blood, normal tissue and pathological tissue). In order to assess the validity of this assumption, the artificial phase maps were compared with measured phase data.

Results

In Figure 1, maps of $\Delta R2^*$ (a), artificial phase (b) and measured phase (c) are shown for one subject, together with a corresponding phase difference map Φ_m - Φ_a (d). Visual inspection implied that differences between measured and artificial phase were obvious in the vicinity of large vessels in the Sylvian fissure region, but more subtle differences can be anticipated also in other regions.

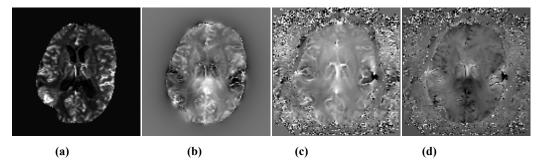


Figure 1. Images showing (a) $\Delta R2^*$, (b) artificial phase, (c) measured phase and (d) difference between measured and artificial phase.

Discussion

The phase difference map, displayed in Fig. 1d, highlights differences between measured and artificial phase in the vicinity of large vessels. This seems consistent with the, most likely simplified, assumption that the T2* relaxivity is equal in all compartments, an assumption that formed the basis of the $\Delta R2^*$ maps that represented susceptibility in the present study. Previous simulations have predicted that the T2* relaxivity in tissue is higher than in whole blood, and the present results are not inconsistent with these findings. Interestingly, the phase difference map does not exclude the possibility of minor relaxivity differences also in other regions. The present methodology is in some respects somewhat suboptimal and can be optimized in future applications. For example, the spatial resolution was limited, and a small gap was used between slices, leading to potential uncertainties in the artificial phase maps. The presented concept could, however, provide an interesting way to estimate relative differences in relaxivity between different compartments, and to further characterize the non-linear $\Delta R2^*$ -vs-concentration relationship in blood. An approximate relaxivity map could potentially be useful for correction of $\Delta R2^*$ maps in order to obtain more reliable perfusion estimates in DSC-MRI.

References: [1] Schmueli et al., MRM 62, 1510 (2009); [2] Wharton et al., MRM 63, 1292 (2010); [3] Petridou et al., NeuroImage 50, 491 (2010); [4] Kjølby et al., MRM 56, 187 (2006); [5] Marques & Bowtell, Concepts Magn Reson B 25B, 65 (2005); [6] Salomir et al., Concepts in Magn Reson B 19B, 26 (2003); [7] Akbudak & Conturo, MRM 36, 809 (1996).