Grey/white matter contrast in phase images: is it susceptibility or is it not?

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Introduction Recently great interest has been devoted to the tissue contrast observed in phase images, not only in veins and iron rich regions (1) but also contrast between WM and cortical GM (2). The tissue phase variations are thought to originate from either tissue susceptibility variations (3) or variations of macromolecules content affecting the water chemical shift (4). The two mechanisms are expected to display different spatial patterns: the susceptibility contribution should be a convolution of the tissue distribution with the dipole field kernel (5) while the macromolecular contribution is expected to be characterized simply by the tissue distribution. In this study we aimed at estimating the susceptibility and/or macromolecular contribution to the gray matter (GM) / white matter (WM) phase contrast by taking advantage of this spatial information.

Methods Three subjects were scanned in a 7T head scanner (Siemens, Germany), equipped with head gradient coil and an 8-channel RF transceiver (Rapid, Germany). All subject followed the same protocol: (1) T_1 weighted MP2RAGE (6) with TR/TI1/TI2=8/1/3.2secs, excitation pulses for each GRE module $\alpha1/\alpha2=4/5$, (2) 3D GRE with TR=29ms, $\alpha=18$ and 5 equally spaced echoes with TE=3.25/8.45/13.65/18.85/24.05, for both images the bandwith was 200Hz/px; isotropic resolution of 0.65 mm and matrix size 320x320x256 to facilitate co-registration.

Using the odd echoes of the 3D GRE (which are inherently flow compensated relative to each other) of each loop of the rf-coil, two field maps (based on the 3rd and 1st echoes and 5th and 1st echoes) were computed and combined in a weighted fashion (7), taking into account the pixel based T_2^* estimate. Finally, the field maps from separate coils were combined, weighted with the respective magnitude images. This allowed the computation of "singularity free" field maps. R2* maps were also obtained from the 3 odd echoes.

The MP2RAGE image was coregistered to the 3DGRE and then used for CSF/GM/WM/arterialBLOOD segmentation. by thresholding based on MP2RAGE intensity histograms. A fifth segment representing venous blood obtained from the R_2^* map ($R_2^*>60s^{-1}$). The frequency shift generated by each of the tissue segments was then calculated (5). The fieldmap, the individual tissue images obtained from segmentation and the predicted frequency shifts due to a susceptibility origin were high-pass filtered. This was done to reduce the contribution of large scale frequency shifts on the field map, while keeping the same spatial frequency information on the field map and the other images. Finally, the high-pass filtered field map was fitted by means of matrix inversion considering the following model:

$$\begin{aligned}\text{FieldMap} = {} & b(0)+b(1)*WM+b(2)*GM+b(3)*CSF+b(4)*\text{VenousBlood}+b(5)*\text{ArtBlood}+ \\ & b(6)*WM_\chi+b(7)*GM_\chi+b(8)*CSF_\chi+b(9)*\text{VenousBlood}_\chi+b(10)*\text{ArtBlood}_\chi+b(11)*\text{AirTissue}_\chi\end{aligned} \quad \text{Eq. 1}$$

where WM, GM, CSF, VenousBlood and ArtBlood stand for the contributions to the measured fieldmap of non-susceptibility origin and the terms with subscript χ represent field shift contributions of susceptibility origin. As the high-pass filter will not eliminate all undesirable field variations, and because WM and GM properties are expected to vary throughout the brain, the fitting routine was performed in overlapping regions of 40x40x40 pixels. Three fits were performed: (i) Eq. 1 without the terms WM_χ, GM_χ, CSF_χ; (ii) Eq. 1 without the terms WM, GM, CSF (iii) Eq. 1 completely

Results The yellow arrows in Fig. 1 point out representative regions of GM where the fitting procedure succeeded in explaining the contrast via a macromolecular related frequency shift argument (seen by the low value of the fit residual images in the 3rd column), and failed to explain it via a susceptibility origin (visible by the large contrast present on the residuals of the 4th column). This observation suggests that local frequency shifts between WM and GM explains a bigger fraction of the WM/GM contrast than fields generated by the difference of susceptibility between the two tissues which was estimated to be of 0.015±0.005ppm, which is significantly smaller than necessary to explain the GM/WM phase contrast (3). The segmented model has some limitations as it does not distinguish WM tracts, such as the optic radion or the corpus callosum (yellow stars on subject 1 and 3) that have different phase and T_2^* contrast from the neighboring WM. Vessels and arteries seem to be left in the residuals, which is not surprising due their small size, which implies that the segmentation will be a very coarse and overestimation of their shape and sizes.

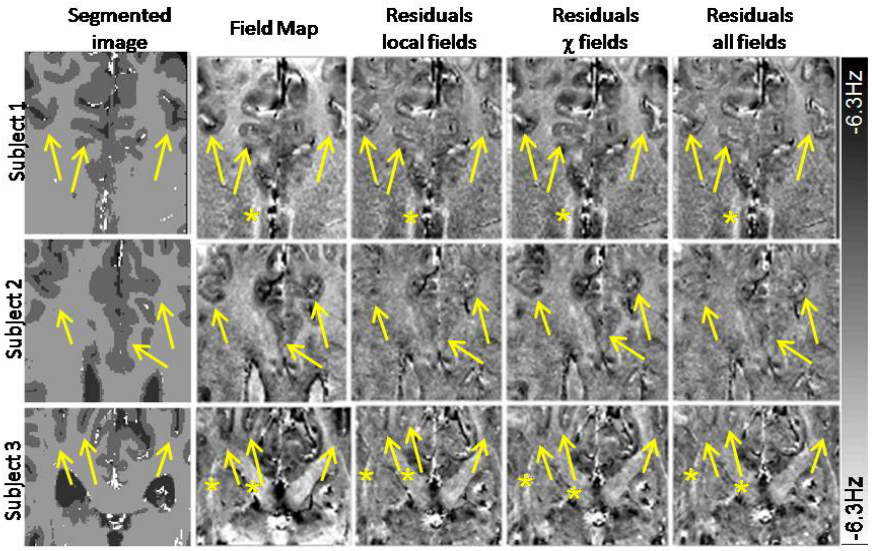

Figure 1 – Data corresponding to transverse slices of three subjects are shown in the three rows. First column shows the segmentation obtained from the T1 and R2* images. Second column shows the field maps measured. Third, fourth and fifth column show the residuals of the fit (i), (ii) and (iii).

Discussion Care should be taken interpreting the results obtained, as fitting susceptibility originated field shifts could be more sensitive to a poor segmentation model, due to both the large frequency shift values that can be expected at boundaries of tissues with different magnetic susceptibilities and to the shape dependence of this fields. Nevertheless, this preliminary results point to a bulk frequency shift in the origin of WM/GM contrast.

References (1) Haacke et al. Magn. Res. Med., 2004, 52, 612-618 (2) Abduljalil et al, J. Magn. Res. Imag., 2003, 18, 284-290 (3) J. Duyn et al, PNAS, 2007, 104, 11796-11801; (4) Zhong et al, Neuroimage, 2008, 40, 1561-1566.; (5) Marques et al., Conc. Magn. Res. B, 2005,25, 65-78; (6) Marques et al. Proc. ISMRM 2008, 1393; (7) Gruetter et al., Mag. Res. Med., 2003, 29, 804-811