Diffusion Measurement of Human Optic Nerve Using Fat Suppressed Diffusion Turbo FLASH Sequence

J. G. Xu[1], S-K. Song[2]

[1]Chemistry, Washington University in St. Louis, St. Louis, Missouri, United States, [2]Radiology, Washington University in St. Louis, St. Louis, Missouri, United States

Introduction

The human optic nerve is ideal for study of the correlation between diffusion tensor imaging (DTI) and neurological disability resulting from diseases such as multiple sclerosis (MS) [1]. However, conventional echo planar imaging based diffusion sequences are extremely sensitive to the off-resonance effects resulting from susceptibility differences at the air-tissue interface and bony structures near the optic nerve, leading to substantial image distortion and intensity variation. To address this issue, a diffusion sequence based on turbo fast low angle shot (turbo FLASH) was developed and evaluated. The sequence employs slice selective, phase cycling, and centric-order turbo FLASH as the readout of a diffusion weighting preparation with fat suppression. This sequence was tested on phantoms and then used to measure the apparent diffusion coefficient (ADC) and relative diffusion anisotropy in the optic nerve of two healthy volunteers.

Methods

We adopted the twice refocused spin echo scheme for the diffusion preparation period (orange, Fig. 1) to minimize eddy-currents [2]. After preparation, a second 90° pulse tipped the diffusion prepared magnetization back along the z-direction. A four step phase cycling ($90°_{x/-x/y/-y}$) of this driven equilibrium pulse was necessary to correct for T_1 contamination and the residue eddy-current induced phase distortion [3-4]. The turbo FLASH readout module (blue, Fig. 1) used a centric-order phase encoding scheme. Gradient spoiling was applied along both slice selective (SS) and read out (RO) axes with rewinding phase encoding (PE) gradients at the end of each step. A chemical shift selective fat saturation pulse was inserted between the preparation and imaging periods (black, Fig. 1). Spoilers flanking the fat saturation pulse were used to eliminate the fat signal at the center of *k*-space and crush unwanted coherences. Diffusion measurement of grey and white matter in healthy human brain using this sequence is comparable with literature values. Minimum distortion, especially at the lower part of the brain, can be appreciated (Fig. 2).

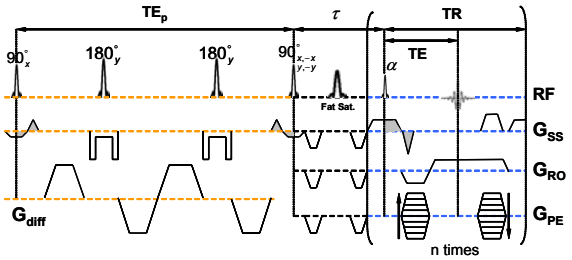

Figure 1. Diffusion turbo FLASH sequence diagram.

In vivo DTI data was acquired using a 4-element phased array "optic nerve" coil (Stark Contrast, Erlangen, Germany) on a 3-tesla Allegra scanner (Siemens AG, Erlangen, Germany). Relevant imaging parameters were: slice thickness = 2.5 mm, TE_p = 55 ms, τ = 10 ms, TR/TE = 3.5/1.58 ms, magnitude averages = 32, receiver bandwidth = 480 Hz/pixel, flip angle α = 10°, field of view (FOV) = 320 × 150 (read × phase), and matrix size = 192 × 192. It should be noted that the intrinsic phase-encoding resolution is limited by a point spread function (PSF) due to T_1 recovery during each phase encoding step. The full width half maximum (FWHM) of the PSF is given by $FWHM_{PE} = 2 \cdot FOV_{PE} \cdot (TR/T_1 - \ln \cos\alpha)/\pi$ [5]. At the given FOV_{PE}, flip angle, TR, and brain T_1 at 3-tesla (~ 800 - 1000ms), the in-plane resolution is determined and smoothed to 1.7 mm^2 before tensor calculation (i.e., the latter echoes in the turbo FLASH echo train was used to fill the repetition time (TR_{total} = 3000 ms) for signal-to-noise ratio (SNR) improvement rather than for phase encoding). Two diffusion sensitizing factors or *b* values = 0 and 800 s/mm^2 were used to calculate diffusivities. Six diffusion gradient directions follow the oblique dual gradient scheme to minimize TE_p.

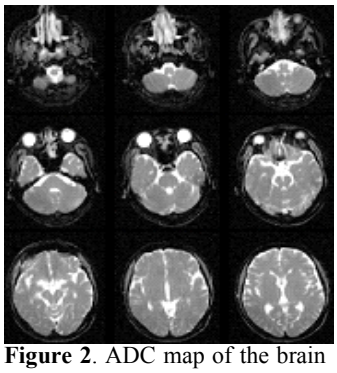

Figure 2. ADC map of the brain of a healthy volunteer.

Results and Discussions

The human intra-orbital optic nerve is surrounded by orbital fat. Hence fat saturation is essential to eliminate chemical shift artifacts in gradient echo based sequences. For practical receiver bandwidths, the chemical shift could amount to 0.5 - 1.5 pixels width. Such shift is substantial for the small structure (3 - 4 mm in diameter) of the optic nerve considering the resolution limitation from the PSF. The effect of chemical shift artifact is demonstrated in non-diffusion weighted (b_0) image (Fig. 3A) and the calculated ADC map (Fig. 3B) without fat saturation as compared with fat saturation (Figs. 3C and 3D, respectively). The intensity loss in non-fat-saturated optic nerves in the images is probably due to signal cancellation from the fat signal that is partially out-of-phase with water signal. With fat suppression, ADC (Fig. 4A) and scaled relative anisotropy (sRA) were measured to be 0.72 ± 0.15 μm^2/ms and 0.17 ± 0.09 (n = 2; mean ± range), respectively in the healthy optic nerve. The anterior-posterior orientation of the nerve fiber is reflected as green (red: left-right; blue: superior-inferior) in the color coded anisotropy map (Fig. 4B).

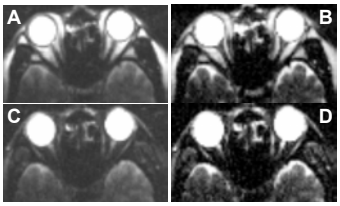

Figure 3. non-fat-saturated b_0 image (A), ADC map (B) comparing with fat-saturated b_0 image (C), and ADC map (D).

In addition to the inherently low SNR and limited spatial resolution of the diffusion turbo FLASH sequence, other limitations include: (i) fat saturation is only achieved in the center of k-space (the latter echoes are still contaminated by fat signal due to the short fat T_1) and (ii) the phase cycling implemented herein without navigator echo is susceptible to patient motion. To avoid phase cycling, the current sequence can be slightly modified to adopt a diffusion turbo STEAM scheme [6].

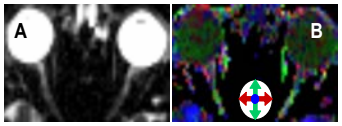

Figure 4. ADC (A) and color coded anisotropy (B) maps of the optic nerve from a volunteer.

Conclusion

Diffusion weighted turbo FLASH sequence offers an alternative to image the human optic nerve with minimum susceptibility distortion. Fat suppression is essential in this sequence for accurate diffusion measurement of structures surrounded by fat, such as the optic nerve.

Reference [1] Trip, et al. *NeuroImage* in Press, 2005. [2] Reese, et al. *Magn. Reson. Med.* **49**, 177-182, 2003. [3] Coremans, et al. *J. Magn. Reson.* **124**, 323-342, 1997. [4] Thomas, et al. *Magn. Reson. Med.* **39**, 950-960, 1998. [5] Nolte, et al. *Magn. Reson. Med.* **44**, 731-736, 2000. [6] Rieseberg, et al. *Magn. Reson. Med.* **54**, 486-490, 2005.