Spiral diffusion tensor imaging at 7T - Fiber tracking on a rat model of brain trauma

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Introduction

In our previous work [1], we have demonstrated the feasibility and the accuracy of a fast Diffusion Tensor Imaging (DTI) sequence implemented on a Magnex 7T small-bore system with 12 cm diameter equipped with shield gradients (G_{max} = 200 mT/m). The method consists of a Twice Refocused Spin Echo (TRSE) sequence [2] combined with an interleaved Variable-Density Spiral (VDS) k-space acquisition [3]. The aim of this work was to apply this TRSE-VDS-DTI sequence to *in-vivo* traumatic brain injury (TBI) in rats to measure diffusivities, fractional anisotropy (FA) and especially to track corpus callosum fibers in order to detect the presence of axonal rupture.

Material and methods

Diffuse TBI was induced according to the impact acceleration model [4]. Two groups of rats were studied using DTI: a "Trauma group" (n = 5) in which rats were submitted to TBI and a "Sham group" (n = 5) in which rats were submitted to the same surgical preparations as the "Trauma group" but without TBI. Rats were anesthetized, tracheotomized and artificially ventilated. Two catheters were inserted into the femoral artery and vein to monitor arterial blood pressure, blood gases and for intravenous administration of anesthetic agents (α-chloralose, pancuronium). During experiments, rat body temperature, mean arterial blood pressure and arterial blood gases (PaO_2, $PaCO_2$, arterial pH) were maintained constant. Two diffusion gradient sampling schemes: Dual Gradient 6 directions (DG6) [5] and Icosahedral 21 directions (IC21) [6] were implemented and used. In order to minimize the effect of eddy currents, calibrations of the TRSE sequence using the signal of polybutadiene: $(CH_2\text{-}CH\text{=}CH\text{-}CH_2)_n$, a polymer with a very slow diffusion coefficient ($D = 10^{-15}$ mm^2/s^{-1}) were performed for each diffusion gradient orientation with a gradient factor-b in the range of [100-2000 s/mm^2] which correspond to none signal attenuation. The objective of these calibrations was to find the optimum durations of the gradient diffusion for a given echo time (TE = 50ms), which were found equal to ($\delta_1 = \delta_4$ = 3.5 ms and $\delta_2 = \delta_3$ = 7.5 ms - see Fig 1). Signal reproducibility during two hours was also tested on the polymer. Optimal imaging parameters avoiding off-resonance artifacts in VDS acquisition (acquisition time per interleaf then number of interleaves) were determined on water phantom then celery using a FOV of 50 mm and a matrix size of 128 × 128. Finally, using a gradient b-value of 1000 s/mm^2 (corresponding to less than 2% polymer signal attenuation) and a number of 16 interleaves, DG6 (16 averages) and IC21 (8 averages) gradient sampling schemes were validated on healthy rat brain using actively decoupled volume/surface coils for emission/detection, respectively. The repetition time (TR) was set to 2000 ms resulting in a total acquisition time of 1h00 and 1h30 for DG6 and IC21 respectively. During the experiment, DTI was performed at 1h00 and at 4h30 post TBI with DG6 gradient sampling scheme and at 2h00 post TBI with IC21. The Mann-Withney test was used to compare groups of data (Sham vs. Trauma and DG6 vs. IC21), and Anova two-way analysis of variance to compare repeated measurements (*: p<0.05). Fiber tracking imaging was performed using the "*MedInria DTI Track*" software. *MedInria DTI Track* uses Log-Euclidean metrics, which are shown to be well adapted to DTI [7].

Results and discussion

DTI derived values obtained with TRSE-VDS-DTI sequence from a water phantom (Mean Diffusivity: MD = 23 × 10^{-4} mm^2/s at 25°C and FA = 0.07), from celery ($D_\perp = 6 \times 10^{-4}$ and $D_{/\!/} = 15 \times 10^{-4}$ mm^2/s) and from healthy rat brain (MD = 8 × 10^{-4} mm^2/s, FA = 0.25 in the cortex and FA = 0.55 in the corpus callosum) were in accordance with published results [8,9]. With both gradient sampling schemes, a significant decrease of MD in the "Trauma group" vs. the "Sham group" was observed in the cortex (26 ± 2 % decreases, $p < 0.05$) as well as in the corpus callosum (41 ± 5 % decreases, $p < 0.05$). These results suggest that cellular edema is predominant following diffuse TBI. There was no significant difference between FA values in the cortex. In the corpus callosum FA indices were significantly lower ($p < 0.05$) in the "Trauma group" (FA = 0.46 ± 0.04) than in the "Sham group" (FA = 0.55 ± 0.04). No significant temporal evolution was observed following trauma. Fiber tracking imaging shows systematic axonal rupture in the corpus callosum in the "Trauma group" while, in the "Sham group", fibers in this region are tracked with a regular continuity without any rupture in accordance with published results [10]. (Fig. 2).

Conclusion

In this work, using the validated TRSE-VDS-DTI sequence on our 7T system, we demonstrate the feasibility of fiber tracking imaging to visualize the rupture of axonal fibers in the corpus callosum in rats following TBI. In addition to the MD and FA decrease following TBI which suggests the predominant cellular edema and the presence of diffuse axonal injury respectively, this new finding is of great interest for the early management of patients with severe TBI. Cerebral edema characterization and white matter integrity following TBI are accessible with DTI and fiber tracking imaging, respectively, within the same MRI acquisition.

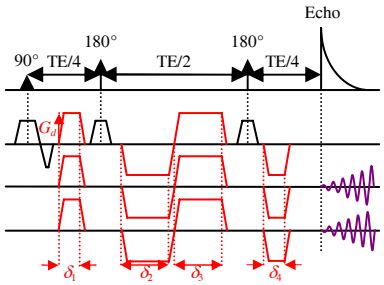

Fig. 1: Spiral twice refocused spin echo diffusion tensor imaging sequence.

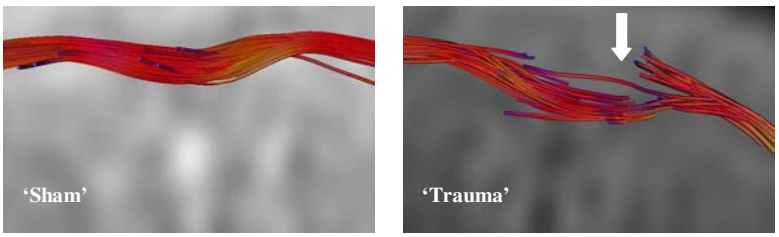

Fig. 2: Fiber tracking images of the center of the corpus callosum of a 'Sham' and a 'Trauma' rat.

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