Ultrashort Echo Time (UTE) Imaging in Multiple Sclerosis

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Target Audience: Neuroradiologists, Neurologists, Multiple Sclerosis Investigators

Purpose: Multiple Sclerosis (MS) is an autoimmune disorder characterized by focal white (WM) and gray matter (GM) lesions, iron deposition in the central GM, and brain atrophy. MRI is sensitive to focal white matter lesions which appear as hyperintensities on T2-weighted and T2-FLAIR imaging.[1] Normal appearing white matter (NAWM) seen with these techniques can reveal abnormalities when imaged with more advanced techniques. Using multicomponent analysis of T2 relaxation, protons with T2s of about 15-20 ms have been attributed to myelin water and correlated with myelin loss.[2] Magnetization transfer using the cross relaxation between myelin protons and tissue water, can also indirectly detect abnormalities in NAWM.[3] Ultrashort TE (UTE) imaging allows direct access to tissue protons with T2*s less than 1 ms. These are invisible with conventional sequences.[4] Studies in the rat spinal cord, rat optic nerve, and frog sciatic nerve suggest such ultrashort T2* signals may originate from the lipid protons of myelin.[5, 6] In this study we report on the use of an inversion recovery UTE (IR-UTE) sequence to target ultrashort T2* components in cadaveric MS brain.

Methods: All imaging was performed on a Sigma HDx 3T Scanner (GE Healthcare, Milwaukee, WI) using a volume head coil. An adiabatic inversion recovery (IR) pulse was used to invert the longitudinal magnetization of long T2 tissue components. The UTE data acquisition began after the delay time (TI) at which the inverted longitudinal magnetization of the long T2 components in white matter reached the null point. Short T2 components were not inverted due to significant transverse relaxation during the relatively long adiabatic inversion process, and were subsequently detected by the 2D UTE data acquisition. A second echo was acquired at which time residual long T2 components were detected while the short T2 components had decayed to zero or near zero. The second echo image was subtracted from the first to allow selective visualization of ultrashort T2 components. IR-UTE imaging used the parameters: TR 1500ms, FOV = 24 cm, Matrix =256x256, Bandwidth = 125 kHz, TE = 0.01 ms 2.5 ms, 5ms, 7.5 ms; TI varied between 100 – 500 ms and was chosen to optimize long T2 component nulling. Human cadaver brain specimens were imaged prior to fixation. After fixation, brains were immersed in phosphate buffered saline and reimaged. Image analysis was performed in ImageJ and Matlab using custom scripts for T1, T2, T2* as described previously.[7] Regions of interest were drawn manually.

Results: *White Matter (WM):* WM had high signal on IR-UTE images. There were areas of signal loss which corresponded to T2 hyper intense areas on T2WI, but also extended into NAWM (**Figure 1**). *Gray Matter:* Cortical GM was low signal on IR-UTE imaging. The central GM and thalamus had higher signal with intensity approaching or exceeding that of the short T2 components seen in white matter, as well as areas of signal loss (**Figure 2**). T2* relaxometry showed frontal WM compoments had a typical T2* of about 200 us (**Figure 3)**.

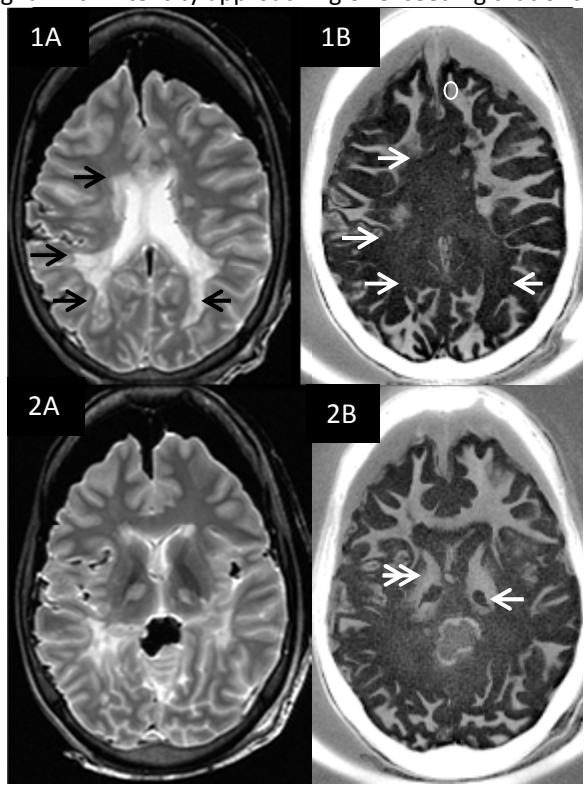

Figure 1(A) Spin Echo T2 Weighted: TR 4000, TE 67.78ms, MS lesions appear hyperintense (**black arrows**) **(B)** IR-UTE: TR=1500 TE=0.01 TI=420 ms subtracted by TE = 2.5 ms echo: WM appears bright. Myelin loss extends beyond the lesions shown in **1A (white arrows), circle** represents ROI of WM for **Figure 3**

Figure 2 (A) Spin Echo T2 Weighted: TR 4000, TE 67.78ms **(B)** IR-UTE: TR=1500 TE=0.01 TI=420 ms after subtraction by TE = 2.5 ms image. The anterior internal capsule **(double arrow)** and deep gray matter appear bright with a low signal lesion **(arrow)**

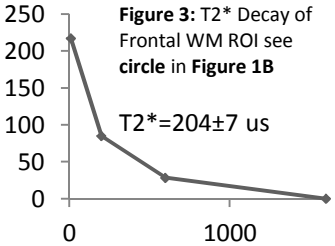

Figure 3: T2* Decay of Frontal WM ROI see **circle** in **Figure 1B**

Discussion: IR-UTE imaging of WM shows areas of signal loss from short T2 components in locations that appear normal on T2WI. The high signal intensity of the central GM may be secondary to iron deposition resulting in T1 and T2 shortening. By imaging previously invisible tissue components, the IR-UTE sequence may improve MRI based disease assessment.

Conclusion: IR-UTE imaging of multiple sclerosis allows direct imaging of the short T2 components of white matter. It shows high contrast between normal and abnormal white matter. The IR-UTE sequence also shows gray matter lesions. High signal intensity is seen in the deep gray matter probably secondary to iron deposition in MS.

References

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