CORRELATION BETWEEN T2-RELAXOMETRY AND PROTON MRS ABNORMALITIES IN DIFFERENT EPILEPSY SYNDROMES

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***Introduction**:* MR investigations show typical changes in patients with temporal lobe epilepsy (TLE), including tissue signal change assessed by T2 relaxometry, and brain metabolite abnormalities by Proton-MRS. It is not known how these measurements relate to each other. Here we analyse the relationship of T2 relaxometry and MRS in TLE (lesional TLE and TLE with normal conventional MR; MR negative TLE) and in idiopathic generalised epilepsy (IGE), where no obvious brain abnormalities are expected.

Methods: We assessed 40 patients and 40 healthy controls; 23 patients had lesional TLE, nine had MR-negative TLE and eight had IGE. MR imaging was performed on a 3T GE LX Horizon scanner (Milwaukee, WI, US). T2 relaxometry was acquired in 24 slices (whole brain coverage), with eight TEs between 29 and 231ms, FOV 24cm. Maps of tissue T2 relaxation time were generated using proprietary GE software (Functool®), which fitted a single exponential to signal intensity values of corresponding pixels from each image (TE) per slice location. Region of interest analysis was performed bilaterally in the hippocampus, anterior temporal lobe (ATL), frontal lobe, amygdala, thalamus and caudate.

Bilateral temporal and frontal lobe single voxel spectra were acquired (TR, 3.0 s; TE 30 ms). Concentrations of *N*-acetylaspartate (NA), creatine (Cr), phosphocholine (Cho), myoinositol (mI), and glutamine/glutamate were obtained using LCModel. Analysis of covariance, with age included as a covariate, was performed to assess for differences between the three patient groups and controls. Regression analysis was used to assess the association between areas of significantly increased T2 relaxation time with metabolite concentrations. The only area to show a significant side-to-side difference was the hippocampus: ipsilateral T2 relaxation time in lesional TLE was increased, compared to the contralateral side (p=0.0005). Other regions, patients with MR negative TLE or IGE, and MRS results showed no side-to-side difference. Therefore, apart from the T2 data from the hippocampus, results are presented as the mean of the two sides. The level of significance was set at 5%.

***Results**:* In lesional TLE, the signal intensity of T2 maps was increased in the ipsilateral and contralateral hippocampus, ATL, and amygdala (table). MRS showed a reduction of NAA in both the temporal and frontal lobe, and of Cr in the frontal lobe (table). Hippocampal T2 signal increase was correlated with increased mI (p=0.02, r=0.4, F=5.8). The anterior temporal lobe T2-signal increase was correlated with reduced frontal lobe Cho (p=0.04, r=0.4, F=4.6). In MR-negative TLE, T2 signal intensity was increased in the hippocampus, ATL, and thalamus. MRS showed a reduction of NAA in the frontal lobe, and of Cho in both the temporal and frontal lobes. Hippocampal signal increase was associated with frontal lobe NAA deficits (p=0.008, r=0.6, F=9.7). Patients with IGE had no T2 changes, but showed a reduction of frontal lobe NAA, Cho and Cr concentration.

Conclusion: The results confirm the known abnormalities in lesional TLE. Interestingly, increased hippocampal T2 relaxation times were associated with increased mI concentration. This fits well with theoretical models that suggest both parameters relate to gliosis. In MR-negative TLE, subtle T2 changes were observed in the hippocampus, ATL, and thalamus confirming earlier reports (Scott et al. Brain 2003,126:1968-74). Hippocampal T2 change was correlated with frontal lobe NAA, suggesting that both changes may reflect an epi-phenomenon of the epilepsy. The metabolite changes in IGE are in agreement with a recently published report (Simister et al. Neurology 2003,61:897-902), and suggest mild frontal lobe abnormalities. In conclusion, both MRS and T2 relaxometry reveal abnormalities in patients with normal MR on conventional imaging, but they do not appear to relate directly to the seizure focus.

	controls	TLE		IGE
		lesional	MR negative	
Proton-MRS (Institutional Units)				
frontal lobe				
NAA	5.7 (± 0.5)	5.2 (± 0.7)*	5.3 (± 0.5)*	5.3 (± 0.5)*
mI	2.9 (± 0.5)	2.7 (± 0.7)	2.7 (± 0.4)	3.0 (± 1.2)
Cho	1.2 (± 0.2)	1.2 (± 0.2)	1.1 (± 0.1)*	1.1 (± 0.2)*
Glu	7.6 (± 1)	7.1 (± 1.1)	7.8 (± 1.2)	8.0 (± 1)
Cr	4.0 (± 0.4)	3.8 (± 0.5)*	4.0 (± 0.4)	3.8 (± 0.3)*

	controls	TLE		IGE
		lesional	MR negative	
T2-relaxometry (% normal values)				
Hc	100 ± 3	ipsi: 118±13*	104 ± 5 *	100 ± 4
		contra: 104 ±6*		
ATL	100 ± 6	107 ± 10 *	106 ± 4*	103 ± 4
frontal	100 ± 7	101 ± 7	101 ± 6	96 ± 7
amygdala	100 ± 7	106 ± 6 *	105 ± 5	105 ± 7
thalamus	100 ± 5	103 ± 5	104 ± 4 *	100 ± 8
caudate	100 ± 6	97 ± 8	98 ± 9	100 ± 9

* p<0.05