Amnestic cognitive impairment correlates with hippocampal spectroscopy in aged human brain

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Introduction: Although it would be anticipated that hippocampal spectroscopy measurements are of significant interest for studies of amnestic cognitive impairment (ACI) and aging, there are relatively few reports evaluating its use in correlation with neuropsychological performance (1,2). This may be surprising, given the success and more common use of hippocampal spectroscopy in the evaluation of temporal lobe epilepsy. We describe work correlating neuropsychological performance measures with hippocampal spectroscopy at 4T, finding significant lateralized relationships of NAA/Cr to assessments of language memory (category fluency) and dementia severity (Blessed score).

Methods: All studies were performed using a Varian 4T INOVA whole body MR system with a TEM ^{1}H volume head coil. The spectroscopic studies were acquired from a plane angulated along the planum temporale using a localized adiabatic sequence (3D-LASER). Bilateral hippocampi were maximally included in this sequence, including white matter lateral to the temporal horns of the ventricles (Fig.1). The adiabatic localization allows excellent elimination of lipid resonances from the bony (mastoid and frontal) sinuses both laterally and anteriorly such that no further lipid suppression was needed. Water suppression was provided by use of an optimized water suppression pulse. Within the 19.2x19.2 FOV (slice thickness, 10mm), spectroscopic imaging was performed at 24x24 rectangular encoding scheme to provide a 0.64cc nominal voxel volume (duration, 19.2min). The data were processed using a convolution difference of 250Hz (to minimize residual broad water components) followed by 3Hz of gaussian broadening and spectral Fourier transformation. A hanning filter was used to apodize the data in the spatial domain to minimize PSF ripple, resulting in an isotropic resolution of approximately 10mm. Quantitative T1 tissue segmentation was acquired in the same spectral plane to allocate tissue contributions to the spectral measures. Eleven elderly patients (10 completed; age 81±4.9yo) and 10 young controls (36±12yo) were studied.

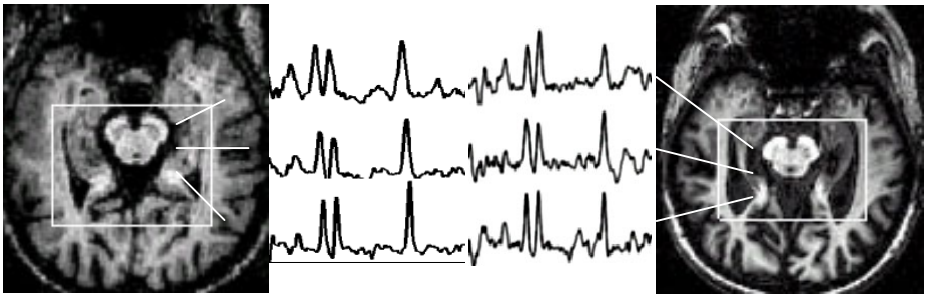
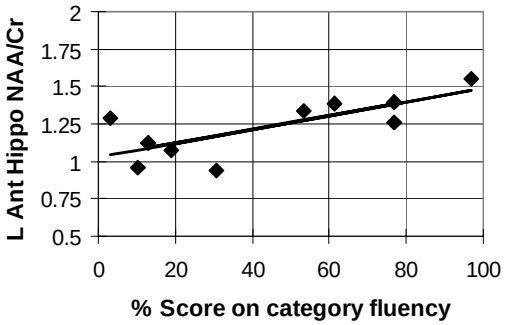


Fig 1. A (Left) Scout and spectra from a healthy aged volunteer. B (Right) spectra and scout from a ACI aged volunteer. The NAA/Cr ratio is depressed throughout the right hippocampus of the (B) volunteer. The white boxes indicate the localized volumes. Fig. 2 (Below) Correlation between anterior hippocampus NAA/Cr with category fluency performance.

Results: Fig. 1 demonstrates scout and spectra from an aged and ACI volunteer, showing the depression of the NA/Cr ratio seen particularly in the ACI patient. Evaluating all the aged subject data, a significant relationship was found in assessing the spectral data from the left anterior hippocampus with regards to verbal memory intensive performance (Fig 2; R = +0.75, $p<0.015$) a left hemispheric task. The assessment of the right anterior hippocampus found no significant relationship with category fluency. However, the volume of abnormality in the right hippocampus did correlate significantly with the Blessed score, with R=0.70, $p<0.025$.



Conclusions: Spectroscopic imaging of NAA/Cr can evaluate with high sensitivity the dysfunctionality of the hippocampus. As seen in epilepsy, NAA/Cr is significant for lateralized abnormalities. The category fluency performance is expected to emphasize left hippocampal function while the Blessed, as a broad based dementia score, would be expected to weight towards the right hippocampus. None of the present volunteers were diagnosed with frank Alzheimers' disease; thus our findings suggest that hippocampal metabolic abnormalities can occur early in concert with amnestic disability. Given the primary role of hippocampal pathology in Alzheimers' disease, these data suggest that hippocampal NAA/Cr can be used to segregate patients who have high versus low probability of developing Alzheimers' dementia.

References: 1) Kantarci K et al, Dement Geriatr Cogn Disord. 14(4):198-207 2002; 2) Dixon R et al Brain. 125:2332-41 2002