Whole-Brain N-Acetylaspartate Quantification: Performance Comparison of NAA Versus Lipid Nulling

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Background and Purpose

N-acetylaspartate (NAA), the second most abundant amino acid derivative in the mammalian brain, is almost exclusive to neurons and their processes and, therefore, is regarded, as a marker for their health and density. Due to its unique specificity, NAA is the most frequently examined neuro-metabolite and its concentration has been reported to decline in all other central nervous system (CNS) diseases. Since many neurological disorders are diffuse throughout the CNS, it is frequently desired to gauge their global load through their surrogate whole-brain NAA (WBNAA) loss. Although the NAA yields the most prominent peak in brain proton MR-spectroscopy (1H-MRS), it resonates at or near and is, consequently, obscured by, the much more intense skull bone marrow and subcutaneous lipids signals. As a result, the cortex, i.e., most of the brains gray matter, is, unfortunately inaccessible to standard localized 1H-MRS. To address the lipid contamination problem as well as the repositioning misregistration issue in serial studies, a WBNAAspectral editing sequence has been proposed. In this study we compared its performance against a new lipid-nulling approach in order to evaluate the precision for the same measurement time as well as serially.

Material and Methods

The two sequences in Fig. 1 were implemented on a 3T whole body imager using its standard transmit/receive head coil (Siemens AG, Erlangen, Germany). Both were run on seven human volunteers who consented through the proper Institutional Review Board process. The first sequence, (Fig. 1, left) nulls the NAA every second shot (1) and the add-subtract scheme destructively interferes the lipids which are thermal every shot. It is compared with the new variant (Fig. 1, right) which nulls the lipids using Inversion Recovery and its result is shown in Fig. 2. T1 for lipids was determined experimentally in vivo.

Both sequences used a 135-T2 binomial pulse for selective excitation and additional water and lipids suppression. Acquisition commenced immediately, i.e., the effective TE~0 ms and, therefore, both sequences are non-T2*, since the TR of both sequences is long, 10 s, T1-weighting is minimized. Weighted. Four cycles of the established four-step sequence in took 160 s, and 40 repetitions of the sequence in Fig. 2, took the same time. Post processing of the MR signals done with in-house software.

Results and Discussion

For visual comparison purposes, the spectra from the same male volunteer are shown in Fig. 2. Both sequences accomplish the goal of NAA editing- and lipids signal deletion. However, the intra-subject (serial) coefficient of variance (CV) associated with the NAA (10.6%) was significantly lower (p=0.0285) than the corresponding CV for Lipid nulling (CV = 19.7%). Similarly, the likelihood ratio test from the mixed model analysis of the normalized individual observations at each time point indicated that the within-subject within-time CV associated with NAA nulling (5.8%) was significantly lower (p=0.012) than the corresponding CV for lipid nulling (8.6%). Therefore, we conclude that when speed is a more pressing requirement than precision, the lipid-nulling sequence is a viable alternative. For precision, however, NAA-nulling is the approach of choice despite its twofold, but still rather modest (~5min) time penalty.

Reference

1. Gonen, et.al. Neurology 11:15