Imaging and Characterizing Brain Iron via MRI at 3T in Living Humans: Implications for neurodegenerative disease research & MRI signal validation

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Introduction

We are investigating non-invasive, MRI-based detection and quantification of iron compounds associated with neurodegenerative disorders such as Alzheimer’s, Parkinson’s and Huntington’s diseases, vascular dementias, and hereditary ataxias. The goal is to evaluate the relationship between presence of iron and the severity of disease and disease progression in living humans using MR relaxometry imaging at 3T [1-3]. Our aim is to identify, at moderately high resolution, the specific iron compounds responsible for T2 and T2* shortening in human brain MR images. This project on the Phillips 3T instrument is developing in parallel with high field MRI (600 MHz instrument) and with synchrotron x-ray fluorescence of cadaver tissues, and of iron standards. Iron standards are also imaged in the 3T instrument for optimizing pulse sequence parameters for human use.

Experimental

Iron standards were examined using the 3T Phillips instrument in order to obtain T1, T2 and T2* relaxation times for specific concentrations and particle forms. Data were also collected from a living human brain using spin echo sequences for the T1 and T2 estimates, and gradient echo for the T2* estimates. Subsequent analysis will cross-correlate relaxometry maps obtained from living brain at 3T, with relaxometry maps obtained from cadaverous tissue in the 600 Mhz instrument, the latter maps validated by synchrotron techniques to identify specific iron compounds.

Results and Discussion

Protocols are currently under development on the Phillips 3T instrument to determine relaxation parameters (T1, T2 and T2*) in living human brain. In order for the relaxometry pulse sequences to be practical to use with patients having neurological disorders, the imaging must be able to be carried out within a relatively short total time, as well as producing the requisite quantitative precision. We are presently able to obtain whole brain images with 2 mm isotropic voxels, and six to eight decay times to fit each of the three exponential decays, in under 30 minutes. Future studies of specific disorders may concentrate on specific regions of interest (e.g. for Parkinson’s disease, globus pallidus and substantia nigra) possibly permitting smaller voxels to be obtained within the short imaging times needed to accommodate patients.

Conclusions

We have been partially successful in developing protocols to obtain T1, T2, and T2* in living human brains as well as in iron standards. This is unfunded pilot work currently in progress.

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References