Phase Contrast Measurements of Microscopic Cardiac-Induced Brain Tissue Pulsations

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Introduction

Magnetic resonance images can be made sensitive to micron-scale motion by the addition on motion-encoding gradient pulses. Coherent motion produces phase changes, while incoherent motion leads to phase dispersion and signal loss. However, such sequences are equally sensitive to unwanted patient motion. We model and eliminate the effects of both field inhomogeneity and rigid-body head motion and present measurements of micrometer-scale brain tissue motion caused by changes in blood pressure through the cardiac cycle. Measuring and analyzing such motion may provide important information relating to mechanical stress, strain, brain compliance and intracranial pressure in diseased and injured brains.

Methods

A standard 2D diffusion weighted, single shot spin echo EPI sequence was modified to allow cardiac gating and storage of the phase information. Data were acquired on a 1.5T clinical GE MRI scanner, with TE/TR = 100ms/2 RR, acquisition matrix 128x128, and motion encoding by two 2.2G/cm trapezoidal gradients of duration 25 ms, separated by 40 ms. The data acquisition was repeated twice (to test reproducibility) for motion-encoding gradients along the ±x, ±y, and ±z directions as well as with no motion-encoding. 4 healthy volunteers (male, ages 34-55) were scanned, and sets of images acquired at 25 different time points separated by 40 ms through the cardiac cycle.

The measured phase at each point in an image is modeled as

$$\theta_{Measured} = \theta_{Pulsation} + \theta_{Susceptibi\,lity} + \underline{m} \cdot \underline{r} + c + 2\pi n$$

where $\theta_{Measured}$ is the measured phase, $\theta_{Pulsation}$ is the phase due to cardiac-induced brain motion, $\theta_{Susceptibility}$ is the phase due to magnetic field inhomogeneities, $\underline{m}$ and c are linear (with position $\underline{r}$) and constant phase terms related to rigid-body rotation and translation respectively [1], with the final term due to phase wrapping of the data to the range [0, 2π]. The phase data was first unwrapped using a 2D phase unwrapping algorithm [Goldstein's branch cut algorithm, 2]; susceptibility-induced phase differences were removed by subtraction of the phase data from images acquired with no motion encoding gradients; finally a 2D linear least squares fit was used to eliminate the linear and constant phase terms. Hence the cardiac-induced phase term, $\theta_{Pulsation}$ was determined which varies linearly with the tissue velocity (as in conventional phase contrast MRI).

Results

With the parameters chosen, a tissue displacement of approximately 7 µm over the 40 ms sampling time is sufficient to give a phase difference of 1 radian. In all subjects the motion is observed to be greatest during systole (Fig 1, 94ms following cardiac trigger), and weak during diastole (334ms following cardiac trigger). The displacement in the x-direction during systole clearly shows a lateral motion relative to the center of the brain. Repeated measurements with the same and opposite motion-encoding gradient amplitudes show the technique to be reproducible for a given subject and scan session. While the amplitudes of the motion observed in the 4 subjects varied substantially, the spatial and temporal (through the cardiac cycle) distributions of the motion were very similar.

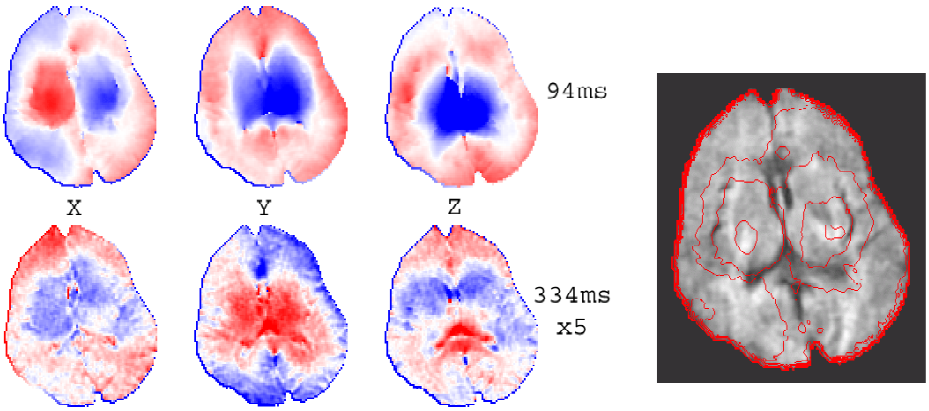

Fig. 1: Calculated brain tissue displacement maps measured in the x , y and z directions (top) 94ms and (bottom, displacement multiplied by 5 for clarity) 334ms following ECG trigger. Tissue displacement in +/- directions are marked with blue/red. (right) Corresponding T2-weighted magnitude image, with each overlaid contour line corresponding to 10µm displacements in the x-direction at 94ms after trigger.

Discussion

We have demonstrated that micron-scale cardiac-induced tissue displacements in three dimensions can be determined rapidly (using EPI), quantitatively and reproducibly, despite the confounding effects of patient motion and magnetic field inhomogeneity. These preliminary experiments did not include monitoring of, for example, blood pressure, but instead simply demonstrate the feasibility of this approach. Further studies are required to include measurement of physiological status; comparison of disease to control subjects; and modeling of the mechanical properties of the brain.

Conclusions

The technique described has the potential to accurately and non-invasively determine the 3D velocity distribution and hence the mechanical properties (stress, strain) of the brain. It offers the exciting prospect of a non-invasive test that may have important clinical applications to, for example, monitoring of intracranial pressure, brain injuries and hydrocephalous.

Acknowledgement

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References

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