

An Empirical Investigation of Motion Effects During eMRI of Interictal Epileptiform Spikes

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Introduction: We recently developed a functional neuroimaging technique called *encephalographic MRI* (eMRI) [1]. Our method acquires concurrent scalp electroencephalography (EEG) and rapid gradient-echo EPI; it attempts to measure an MR signal more directly linked to neuronal electromagnetic activity than existing methods such as BOLD-fMRI. We imaged fast (20-200 ms), high amplitude (> 50 μ V on EEG) cortical discharges in a cohort of focal epilepsy patients. We found easily detectable MR magnitude and phase changes concurrent with interictal EEG spikes with a lag of milliseconds. Due to the time scale of the responses, localized changes in blood flow are unlikely to cause the MR signal changes. While the precise underlying mechanisms are unclear, in this study we empirically investigate one potentially important confounding variable – motion.

Head motion in the scanner affects EEG recording due to the presence of B_0 . Even if the head is securely restrained, tiny head movements can produce high-amplitude “interictal spike-like” artifacts on EEG. By Faraday's law, motion-related change in the area of the interelectrode loops normal to B_0 induces emf in the electrode leads. Bulk head motion accompanying interictal spikes could also cause MR signal increases similar to our results due to inflow of unsaturated spins into the imaging plane. Since the spins in the slice of interest are excited periodically and rapidly during imaging, they do not return to equilibrium, but reach a pseudo-steady state. Any change in head position relative to the slice profile will perturb this steady state, and a transition to a new steady state will occur. This transient state, which can last for several acquisitions following the displacement will affect the MR signal.

Methods

A. Concurrent EEG and Head Motion Tracking: We acquired concurrent EEG (sampling rate 250 Hz) and head motion outside the MR scanner in 3 epilepsy patients. Head motion was tracked using an electromagnetic tracking device with a positional accuracy of 0.7 mm (Aurora; Northern Digital Inc., Waterloo, Ontario, Canada). The subject was seated in a relaxed position. The motion sensor was attached to the EEG cap and recorded 6 parameters (3 translation, 3 rotation) for each time point. The motion tracking system transmitted a single TTL pulse to the EEG device corresponding to each position measurement (sampling rate 20 Hz). Temporal alignment of the EEG and the motion data was based solely on these markers.

B. Healthy Subject “Twitch” Scans: MR imaging for this IRB-approved study was performed using 3-T scanners (Signa, General Electric, Milwaukee, USA with 8-channel head coil; Magnetom Trio; Siemens Healthcare, Erlangen, Germany with 12-channel head coil). We acquired concurrent EEG and MR in 3 healthy subjects. The subjects were instructed to lie still for a period and then nod their head slightly or twitch for brief periods of time (< 1 sec). We acquired single-slice gradient-echo EPI one axial slice at a time. Scan parameters were: TR 47 ms, TE 22 ms, flip angle 20°, image matrix 64 X 64, 28 cm field of view, 5 mm slice thickness, pixel bandwidth 7.8 kHz. Each acquisition block consisted of between 512 and 700 images. We recorded 32-channel EEG (1 subject) and 64-channel EEG (2 subjects) at 5000 Hz continuously using an MR-compatible EEG machine (BrainProducts, Munich, Germany). Temporal alignment of EEG and MR was based solely on the TTL pulses delivered to the EEG system by the MR scanner at each TR. We analyzed the MR magnitude and phase data separately and looked for head motion-related signal changes.

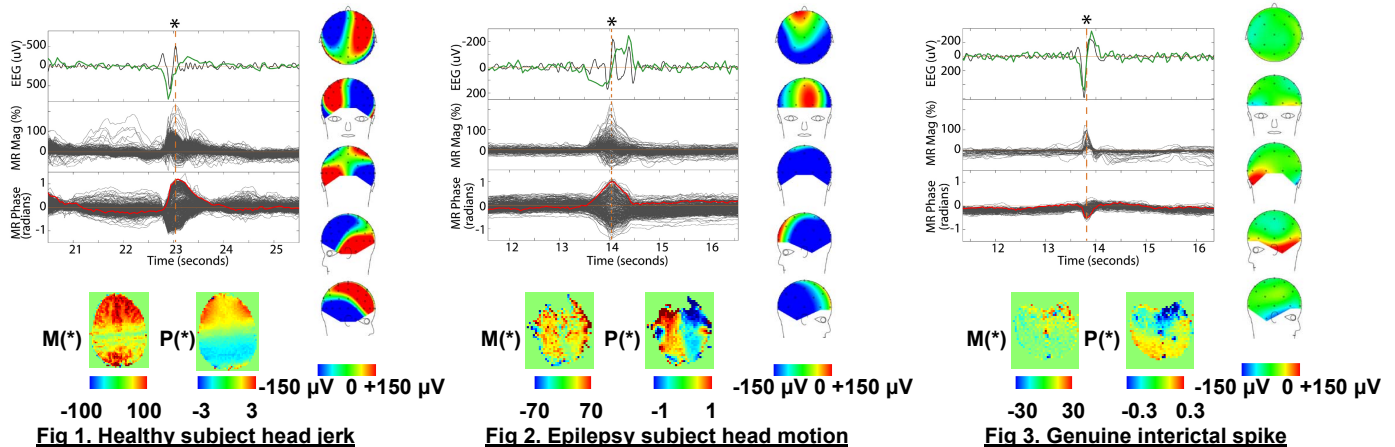
C. Epilepsy Patient Scans: We imaged 15 epilepsy patients with the concurrent EEG-MR scanning setup described in (B). We obtained informed consent from all patients. Subjects were instructed to lie still. A board-certified epileptologist reviewed the EEGs and identified interictal spikes and EEG segments with motion. We analyzed the MR magnitude and phase data and compared the interictal spike-related signals to the motion-related signal.

Results: We found no measurable head motion accompanying the interictal spikes, to an accuracy of 0.7 mm (rms). Temporal correlation of the motion tracker output with EEG yielded a correlation coefficient of 0.0043. In the imaging experiments in both healthy and epilepsy subjects, we found that motion caused confounding signal changes in both MR and EEG. Figs 1-3 show 5-sec of EEG and MR (magnitude, phase) for motion in healthy subjects (Fig 1), motion in an epilepsy subject (Fig 2), and an interictal spike in an epilepsy subject (Fig 3). We found that it was possible to differentiate motion in both healthy and epilepsy subjects from genuine interictal spikes via scalp EEG potential maps (5 views shown corresponding to time *). Motion induces widespread changes in scalp potential; in comparison, interictal spikes are localized. Temporal derivative (green, top plots) of MR phase time course (red, bottom plots) tracks EEG closely for the case of the interictal spike but this relationship was not maintained for the motion case. $M(^*)$ and $P(^*)$ are MR magnitude (%) and phase change images at time (*).

Discussion: Head motion measurement outside the scanner and the localized nature of the EEG scalp potential maps during interictal spikes make bulk head motion an unlikely generator of the large spike-related MR signal changes. While focal micromotions of brain tissue and cerebrospinal fluid during interictal spikes have never been reported, they may be a possible signal source. Lorentz forces[2] act on diffusing ions in the presence of magnetic fields. However, recent work[3] suggests that the ion velocities may be too slow for Lorentz forces to affect diffusion at typical field strengths. Another contrast mechanism may be an apparent displacement of the imaging slice due to a transient local magnetic field during the slice select gradient. This could cause excitation of the given slice along with a neighboring slice, bringing unsaturated spins into the field of view[4]. Rapid neuronal cell swelling may occur during interictal spikes, but the extent of these volume changes is unknown. Transient loss of hydration shells of cations may release bound water into the free water pool, increasing proton density and causing MR signal increases. Migration of hydrated ions during intense neuronal activity may be another potential mechanism. Further work is required to precisely identify the mechanisms underlying the observed MR signal changes.

References:

1. P. Sundaram et al. MRM, In Press, 2010. 2. T.-K. Truong et al. JMR, 2008 3. R. Wijesinghe et al. JMR, 2010 4. V. Renvall et al. PNAS, 2009



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