Aberrant Synchrony in the Somatosensory Cortices Predicts Motor Performance Errors in Children with Cerebral Palsy

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ABSTRACT

Cerebral palsy (CP) results from a perinatal brain injury that often results in sensory impairments and greater errors in motor performance. Although these impairments have been well catalogued, the relationship between sensory processing networks and errors in motor performance has not been well explored. Children with CP and typically developing age-matched controls participated in this investigation. We used high-density magnetoencephalography (MEG) to measure event-related oscillatory changes in the somatosensory cortices following tactile stimulation to the bottom of the foot. In addition, we quantified the amount of variability or errors in the isometric ankle joint torques as these children attempted to match a target. Our results showed that neural populations in the somatosensory cortices of children with CP were desynchronized by the tactile stimulus, while those of typically developing children were clearly synchronized. Such desynchronization suggests that children with CP were unable to fully integrate the external stimulus into ongoing sensorimotor computations. Our results also indicated that children with CP had a greater amount of errors in their motor output when they attempted to match the target force, and this amount of error was negatively correlated with the degree of synchronization present in the somatosensory cortices. These results are the first to show that the motor performance errors of children with CP are linked with neural synchronization within the somatosensory cortices.

Key Words: MEG, Motor Control, Variability, Error Correction, Sensory, Tactile
INTRODUCTION

It well recognized that children with cerebral palsy (CP) often have sensory deficits that limit their proprioception, stereognosis and tactile discrimination (Clayton et al., 2003; Cooper et al., 1995; Sanger and Kukke, 2007; Wingert et al., 2009; Auld et al., 2012). Diffusion tensor imaging (DTI) studies have suggested that these deficits are related to the extent of the structural damage along the thalamocortical tracts (Trivedi et al., 2008; Hoon et al., 2009; Trivedi et al., 2010). A few previous studies that used DTI have provided some insight on this symptomatic relationship, by showing that a greater amount of damage present in the thalamocortical tracts is related to reduced muscular strength and a more severe Gross Motor Function Classification Score in children with CP (Trivedi et al., 2010; Hoon et al., 2009). Since CP can involve damage to both the sensory and motor tracts, it is unclear whether these sensory deficits generally contribute to the motor impairments seen in these children, or if they are more independent symptoms that share a common cause (i.e., perinatal brain damage). Further understanding of how the deficient somatosensory processing in children with CP interacts with the motor system is paramount for understanding the movement impairments seen in these children.

Prior experimental work has established that the brain maintains and updates an internal model that is used to predict the ideal muscular synergies to achieve a motor goal (Shadmere, 2004; Wolpert, 2007; Kording et al., 2004). Outcomes from these experiments have shown that an internal model of the motor system is used to formulate a motor plan based on the available sensory feedback, and this plan is transformed into a motor command that contains the predicted muscle synergies for achieving the goal state (Figure 1). During the execution of the motor command, the internal sensory feedback that occurs is compared with the expected sensory
feedback of the internal model, and any mismatch between the expected and returned sensory feedback is used to make online error corrections in the movement trajectory (Shadmere, 2004; Wolpert, 2007; Kording et al., 2004). The notion that the motor impairments seen in children with CP may be partly attributable to deficient processing by the sensorimotor cortices, and related error checking networks, is a novel concept that has only recently been explored (Kulak et al., 2005; Kulack et al., 2006; Kurz & Wilson, 2011; Kurz et al., 2012; Teflioudi et al., 2011; Riquelme & Montoya, 2010). Intuitively, improper transmission of sensory feedback would limit the child’s ability to make initial adjustments in the motor plan, and to execute online error corrections to the motor command.

The impression that the movement abnormalities seen in children with CP may be a product of deviant somatosensory processing has gained favorable attention over the last few years. Several studies have used magnetoencephalography (MEG) and electroencephalography (EEG) to examine neural oscillatory activity in the somatosensory cortices following sensory stimulation. The overwhelming consensus from these investigations has been that somatosensory evoked-potentials/fields for the hand, foot, and lips are diminished, and in some cases they are latent in children with CP (Kulak et al., 2005; Kulack et al., 2006; Kurz & Wilson, 2011; Kurz et al., 2012; Teflioudi et al., 2011; Riquelme & Montoya, 2010). Results from a fMRI study has provided further support for these outcomes by showing that the somatosensory BOLD response is diminished when sensory feedback is provided to the hand (Wingert et al., 2010). Moreover, a recent EEG study has extended these findings by revealing that aberrant event-related potentials...
are strongly correlated with the two-point tactile discrimination deficits often seen in children with CP (Maitre et al., 2012). Altogether these experimental results indicate that the somatosensory cortices of children with CP may not adequately process sensory feedback. Intuitively, abnormally suppressed somatosensory processes should affect the motor performance in children with CP; however, this link has not been established.

Our prior investigation showed that children with CP have a greater amount of errors when they attempt to generate an isometric lower extremity muscular force that matches a target (Arpin et al., 2013). Based on our scientific model presented in Figure 1, these errors may arise during the sensorimotor transformation, the motor output, and/or the sensory feedback stages. In this investigation, we hypothesized that these errors are strongly associated with the contribution of the somatosensory cortices at the sensorimotor transformation and sensory feedback stages. To this end, the purpose of our investigation was to evaluate the relationship between oscillatory activity of the somatosensory cortices, and the amount of error seen in ankle-joint motor performance of children with CP. Our primary hypothesis was that greater errors in motor performance would be correlated with diminished oscillatory activity in neuronal groups of the somatosensory cortices.

MATERIALS AND METHODS

Participants

Eleven children with a diagnosis of either spastic diplegic or hemiplegia CP (Age = 14.5 ± 0.7 yrs.; Height = 1.61 m; Weight = 52.6 kg) and a Gross Motor Function Classification System (GMFCS) score between I-III participated in this investigation. Children classified as level I can walk with minimal limitations, children classified as level II have limitations walking
long distances and wore orthotics, and the child classified as level III required a walker to assist in ambulation (Palisano et al. 1997). Further details on the children with CP who participated in the experiment are shown in Table 1. Children with known large occupying lesions and/or volume loss were not included in our investigation because it may affect the results if they are mapped to common space. An additional 11 age and gender matched TD children (Age = 14.1 ± 0.7 yrs.; Height = 1.64 m; Weight = 56.8 kg) served as a control group. The Institutional Review Board at the University of Nebraska Medical Center reviewed and approved this investigation. Informed consent was acquired from the parents and the children assented to participate in the experiment.

Motor Experimental Methods

On a separate day from the MEG experiment, an isokinetic dynamometer (Biodex Inc., Shirley, NY) was used to measure the steady-state isometric torques generated by the ankle plantar flexors for the participating children. The largest torque generated from two maximum isometric plantar-flexion contractions was used to establish the child’s maximum voluntary torque (MVT). The child then performed two submaximal steady-state isometric contractions at 20% of their maximum voluntary torque. We selected this percentage because our prior experimental work has shown that compared to TD children, children with CP have greater errors in maintaining this torque level with their ankle (Arpin et al., 2013). The target and the torque exerted were displayed as boxes on a large monitor that was positioned ~1 meter away from the subject at eye level. The box that represented the exerted torque moved vertically as the child applied greater force to the apparatus. The child was instructed to apply a torque such
that the box was within the target force box, and to hold the force as accurately as possible for 30 seconds (Figure 1). The maximum on the visual feedback (vertical) scale was twice the target value. The child was given ample time to practice achieving the target torque before the two actual trials were recorded. These two trials were then averaged together for all data measures.

The voltage output from the torque motor was read by custom LabVIEW (National Instrument Inc., USA) software and sampled at 1 kHz by a 14-bit National Instruments analogue-to-digital converter. The coefficient of variation (CV = [Standard Deviation of Torque/Mean Torque] x 100) was used to assess the amount of variability or error present in the middle 15 seconds of the steady-state torque.

MEG Experimental Paradigm

Throughout the somatosensory experiment, the children were seated in a custom-made nonmagnetic chair with their head positioned within the MEG helmet-shaped sensor array. The children were instructed to close their eyes and a unilateral tactile stimulation was applied to the bottom of the foot at the first metatarsal using a small airbladder. The same foot used for the motor experiment was used for the MEG experiment. For each child, more than 135 trials were collected using an inter-stimulus interval that varied randomly between 2900 and 3300 ms.

MEG Data Acquisition

All recordings were conducted in a one-layer magnetically-shielded room with active shielding engaged for advanced environmental noise compensation. During data acquisition, participants were monitored via real-time audio-video feeds from inside the shielded room. With an acquisition bandwidth of 0.1 – 330 Hz, neuromagnetic responses were sampled continuously
at 1 kHz using an Elekta Neuromag system (Helsinki, Finland) with 306 MEG sensors, including 204 planar gradiometers and 102 magnetometers. Using the MaxFilter software (Elekta), each MEG data set was individually corrected for head motion during task performance and subjected to noise reduction using the signal space separation method with a temporal extension (Taulu et al. 2005).

**MEG Coregistration & Structural MRI Processing**

Four coils were affixed to the head of the subject and were used for continuous head localization during the experiment. Prior to the experiment, the location of these coils, three fiducial points and the scalp surface were digitized to determine their three-dimensional position (Fastrak 3SF0002, Polhemus Navigator Sciences, Colchester, VT, USA). Once the child was positioned for MEG recording, an electric current with a unique frequency label (e.g., 322 Hz) was fed to each of the four coils. This induced a measurable magnetic field and allowed each coil to be localized in reference to the sensors throughout the recording session. Since coil locations were also known in head coordinates, all MEG measurements could be transformed into a common coordinate system. With this coordinate system (including the scalp surface points), each participant’s MEG data was coregistered with structural T1-weighted MRI data prior to source space analyses. Structural MRI data were aligned parallel to the anterior and posterior commissures and transformed into the Talairach coordinate system (Talairach and Tournoux 1988) using BrainVoyager QX version 2.2 (Brain Innovations, The Netherlands).

**MEG Pre-Processing**

Artifact rejection was based on a fixed threshold method, supplemented with visual
inspection. The data analysis epochs were a total duration of 1.2 s (-0.5 s to +0.7 s), with the onset of the mechanical stimulation defined as time 0.0 s, and the baseline defined as -0.5 s to 0.0 s. Artifact-free epochs for each sensor were transformed into the time-frequency domain using complex demodulation (resolution: 2.0 Hz, 25 ms) and averaged over the respective trials. The power of each time-frequency bin was normalized by dividing it by the amount of power present in the respective frequency’s baseline period. This normalization procedure allowed for the visual inspection of power changes that were present in sensor space, and these spectrograms were inspected for maximal responses in the fronto-parietal region (since our experiment focused on somatosensory stimulation). The MEG sensor with the maximum response (increase or decrease) in the fronto-parietal region was then chosen and this sensor was averaged across the group to derive the precise time-frequency band of interest. Based on this procedure, we identified 4-14 Hz frequency range from 25 to 275 milliseconds (see Figure 2).

MEG Source Imaging

A minimum variance vector beamforming algorithm was employed to calculate the source power across the entire brain volume (Gross et al., 2001). The single images were derived from the cross spectral densities of all combinations of MEG sensors within the time-frequency ranges of interest, and the solution of the forward problem for each location on a grid specified by input voxel space. Following convention, the source power in these images was normalized per subject using a separately averaged pre-stimulus noise period of equal duration and bandwidth (van Veen et al., 1997; Hillebrand et al., 2005). Thus, the normalized power per voxel was computed for 4-14 Hz frequency band over the entire brain volume per participant at 4.0 x
Group effects were examined using a random effects analysis for the time-frequency component of interest (TFC), and one-sample t-tests were conducted to determine the activation patterns present in each group. The statistical parametric maps were initially thresholded and a cluster-based correction method (i.e., 40 contiguous voxels) was applied to the supra-threshold voxels to reduce the risk of false positive results. Thus, we imaged these responses using beamforming and statistically evaluated the resulting 3D maps of functional brain activity using a mass univariate approach based on the general linear model. A t-test was used to determine if there were differences in the coefficient of variation of the isometric ankle joint torque generated by the children with CP and the TD children. In addition, we performed Pearson product moment and Spearman rho correlations using each participant’s amplitude value in the peak voxel of the group-level statistical parametric map and the coefficient of variation of the isometric ankle torque.
RESULTS

Children with CP exhibited 4-14 Hz desynchronization in the medial wall of the contralateral postcentral gyrus ($P < 0.05$, cluster-corrected; Figure 3A; peak Talairach coordinates (x,y,z): -7,-23,67), whereas TD children had strong synchronization of neuronal discharges in this same brain area ($P < 0.05$, cluster-corrected; Figure 3A; peak Talairach coordinates: -16,-41,58). This pattern of responses gave rise to a significant group effect ($P < 0.05$, cluster-corrected) in this same region of the medial postcentral gyrus (Figure 3B; peak Talairach coordinates: -10,-29,64). These results indicate that the responsiveness of primary somatosensory cortices to the external afferent feedback was weaker and aberrant in the children with CP.

The children with CP had weaker MVT compared with the TD children (CP = 0.36 ± 0.07 Nm/Kg; TD = 0.98 ± 0.14 Nm/Kg; $P=0.0001$). In addition we found that the children with CP also had a larger CV while attempting to sustain the 20% MVT steady-state torque values (CP = 17.5 ± 7%; TD = 2.7 ± 0.5%; $P = 0.01$), implying that they had greater errors in their ability to adjust the force output to remain at the target value. The Pearson product moment calculated using the data from all of the children showed that there was a negative correlation between the CV and the amount of activity in the somatosensory cortices ($r = -0.50; P=0.01$), indicating that greater errors in matching the target force were related to less synchronization in the somatosensory cortices. Further interrogation of the individual groups revealed that there was a negative correlation between the CV and the amount of activity in the somatosensory cortices.
(r= -0.53; P=0.04) in the children with CP (Figure 4), but not for the TD children (r= 0.03; P=0.46).

The Spearman rho calculated using the data from all of the children indicated that the rank order correlation between the CV and the amount of activity in the somatosensory cortices was marginally significant (rho = -0.32; P= 0.08). Similar results were found when we evaluated the separate rank order correlations for the children with CP (rho = -0.42; P=0.1), but not the TD children (rho = -0.15; P= 0.34). This pattern of results is consistent with those obtained using the Pearson product-moment correlations, and we expect the marginal findings simply reflect the small sample size used in this investigation.

To further explore the results, we divided the data from the children with CP into spastic diplegic (n=8) and hemiplegic (n=3) presentation groups. Qualitatively, it was apparent that the amplitude value in the peak voxel of the group-level statistical parametric maps was generally weaker in the children who had a spastic diplegic presentation. In addition, the children with a spastic diplegic presentation also had a larger CV while attempting to sustain the 20% MVT steady-state plantarflexion torques (Spastic diplegia mean CV = 22.7%; Hemiplegia mean CV = 3.8%). These exploratory results give the qualitative impression that children with a spastic diplegic presentation may be more likely to have uncharacteristic activity of the somatosensory cortices and greater errors in their motor performance.
DISCUSSION

Our results indicated that neural populations representing the foot in the somatosensory cortices abnormally desynchronized in response to tactile stimulation in children with CP. In TD children, these same neural populations exhibited increased synchronization in response to the tactile stimulation. These results provide new support to the notion that the somatosensory cortices in children with CP have an atypical response to external stimuli (Kulak et al., 2005; Kulack et al., 2006; Kurz & Wilson, 2011; Kurz et al., 2012; Teflioudi et al., 2011; Maitre et al., 2012). Based on our scientific model shown in Figure 1, it is likely that the inability of the somatosensory cortices to properly respond to sensory feedback would have a downstream effect on the sensorimotor transformation and error checking stages. The impact on the sensorimotor transformation stage is especially salient since a large portion of the corticospinal tract originates in the postcentral gyrus (Lemon and Griffiths, 2005).

The children with CP had greater errors in their ability to match and sustain the target force with their ankle. This result concurs with prior experiments that have shown that children with CP have larger errors when attempting to control the force production by the muscles of the hands or the lower extremity joints (Valvanno & Newell, 1998; Arpin et al., 2013; Bandholm et al., 2009). Interestingly, there was a negative correlation between the amount of error in motor performance and the amount of synchronization present in the somatosensory cortices in the 4-14 Hz frequency band. This indicates that greater errors occurred in the motor output when there was less synchronization in neural populations of the somatosensory cortices, which provide feedback to the motor system. This finding provides further support for the notion that the motor performance errors seen in children with CP are partly related to the responsiveness of the sensorimotor cortices to afferent feedback.
Variability is a prominent feature of the motor output of both TD children and children with CP. In part, some of these variations arise from subtle adjustments in the motor command to reduce errors in motor performance, while others arise from noise that is present at all levels of the nervous system (Churchland et al., 2006; Faisal et al., 2008). Noise at the sensory level can be problematic if it is equal to or greater than the incoming sensory information (Faisal, et al., 1998). When this occurs, it may be difficult for the somatosensory cortices to discern if there has been a change in afferent feedback. Prior studies have shown that the variability seen in the motor output can be largely attributed to sensory estimation errors (Osborne et al., 2005). Since it is well established that children with CP often have sensory deficits, we suspect that they may have a heightened amount of noise present during the transmission of sensory information (Clayton et al., 2003; Cooper et al., 1995; Sanger and Kukke, 2007; Wingert et al., 2009; Auld et al., 2012). Potentially, the noise induced errors may hamper the ability of the somatosensory cortices to discern changes in the afferent feedback. We suspect that this may partly explain the uncharacteristic desynchronized response to external stimulation in this investigation.

Numerous transcranial magnetic stimulation (TMS) and fMRI investigations have shown that the sensorimotor cortices of children with CP often dynamically rewire themselves throughout development. For example, in children with more severe hemiplegic presentations, the ipsilateral homologue cortices often assume the role of the damaged contralateral cortices that would normally be involved in the control of movement (Carr et al., 1993; Holmstron et al., 2010; Vandermeeren et al., 2003b; Thickbroom et al., 2001; Staudt et al., 2002). A recent fMRI study has also reported that bilateral activation of the sensorimotor cortices can occur when children with spastic diplegic and quadraplegic presentations perform a finger-to-thumb opposition task (Lee et al., 2013). Altogether, these data clearly support the notion that the
sensorimotor cortices of children with CP may attempt to compensate by recruiting a larger network of brain regions for controlling movement and responding to sensory feedback. The children included in our investigation did not display such compensations in the somatosensory cortices when the foot was stimulated. We suspect that the degree and location of perinatal damage and/or behavioral experience may instigate the neuroplastic changes that encompass a larger network of brain regions. However, whether this potential compensation represents an optimization of the available cortical networks, or deviant plasticity that further degrades the child’s motor performance remains unknown.

A considerable amount of scientific evidence from animal models has shown that the topographical organization and activation of the somatosensory cortex is use dependent (Jenkins et al., 1990; Merzenich et al., 1983). In addition, a DTI study has shown that the functional connectivity of the white matter fiber tracts can be improved in children with CP after an intensive therapeutic protocol (Trivedi et al., 2008). These results suggest that the somatosensory deficits noted in children with CP may be somewhat reversible. We suspect that employing an intensive sensory-training program (touch, proprioception, vibration and stereognosis) in with children with CP may help to resynchronize the somatosensory cortical response to the afferent feedback, and promote beneficial structural improvements in the corticospinal and thalamocortical tracts (cf. Riquelme et al., 2013). Potentially, improvements in the synchronization of the somatosensory cortices to the afferent feedback would reduce the errors that are present in the motor performance of children with CP. However, further studies are warranted to test these hypotheses.
Acknowledgements

Funding for this project was provided by the Hattie B. Munroe Foundation, a research support fund grant from the Nebraska Health System and the University of Nebraska Medical Center, and the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) of the National Institutes of Health (1R21HD077532-01).
Table 1. Demographics of the participating children with cerebral palsy. GMFCS = Gross Motor Function Classification Score.

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age</th>
<th>Gender</th>
<th>GMFCS</th>
<th>Type of CP</th>
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<tr>
<td>1</td>
<td>13</td>
<td>Female</td>
<td>III</td>
<td>Spastic Diplegia</td>
</tr>
<tr>
<td>2</td>
<td>17</td>
<td>Male</td>
<td>I</td>
<td>Hemiplegia</td>
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<td>17</td>
<td>Male</td>
<td>I</td>
<td>Spastic Diplegia</td>
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<td>13</td>
<td>Male</td>
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<td>Spastic Diplegia</td>
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<td>Female</td>
<td>I</td>
<td>Spastic Diplegia</td>
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Figure Captions

Figure 1. Conceptual scientific model of the sensorimotor transformation, execution and sensory feedback stages that are involved in completing a goal oriented isometric motor task with the ankle musculature. These stages are based on an internal model that is used to predict the muscular synergies that will best match the desired motor outcome. Sensory information is used during the formulation of the motor plan, and online corrections in the evolving motor pattern.

Figure 2. Average Time-Frequency Spectra in Controls and Children with CP During Somatosensory Stimulation. Time (in ms) is denoted on the x-axis, with 0 ms defined as stimulation onset. Frequency (in Hz) is shown on the y-axis. The average patterns of event-related spectral changes during the foot stimulation task, expressed as percent difference from baseline (-500 to 0 ms), are shown in the children with CP (A) and the control group (B). In each case, the fronto-parietal MEG sensor with the greatest response amplitude was chosen for each participant and these were averaged across the person’s respective group. As shown, the children with CP (A) exhibited a decrease in 4-14 Hz band during the 25-275 ms time window, whereas the healthy children showed a strong increase in this same time-frequency band window (4-14 Hz, 25-275 ms). Note that the color scale for each plot is shown to the right, and that the scale for controls is twice that of the children with CP.

Figure 3. (A) Statistical parametric maps (SPMs) of the 4-14 Hz response from 25 to 275 ms post-stimulation for the typically-developing (TD) children (top) and children with CP (bottom). Both maps have been thresholded at \( P < 0.05 \), cluster-corrected. As can be discerned, both the
children with CP and the TD children had activation in the medial wall of the contralateral postcentral gyrus. In addition, it is apparent the children with CP had a desynchronized response (blue) to the external stimulus, while the TD children had a synchronized response (orange). (B) 2D SPM’s of the group effect for the 4-14 Hz response (25-275 ms). Images have been thresholded at $P < 0.05$, cluster-corrected. As shown, the group difference was in the same region of the medial postcentral gyrus, and was weaker and aberrant in the children with CP. The images are displayed following the radiological convention (R=L).

Figure 4. Scatter graph of the relationship between the coefficient of variation of the isometric torque and the peak activation in the somatosensory cortices of the children with cerebral palsy. The graph shows that a larger amount of variability or errors in the steady state isometric torques was related to a decreased activity in the somatosensory cortices to the tactile stimulation.
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