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Neurophysiological abnormalities in the sensorimotor cortices during the motor planning and movement execution stages of children with cerebral palsy

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Abstract

AIM—This investigation used magnetoencephalography (MEG) to examine the neural oscillatory responses of the sensorimotor cortices during the motor planning and movement execution stages of children with typical development and children with cerebral palsy (CP).

METHOD—The study involved 13 children with CP (nine males, four females; mean [SD] age 14y 3mo [9mo], range 10–18y; height 1.61m [0.08m]; weight 52.65kg [13kg]), and 13 age- and sex-matched children with typical development (height 1.64m [0.06m]; weight 56.88kg [10kg]). The experiment required the children to extend their knee joint as whole-head MEG recordings were acquired. Beamformer imaging methods were employed to quantify the source activity of the beta-frequency (14–28Hz) event-related desynchronization (ERD) that occurs during the motor planning period, and the gamma-frequency (~50Hz) event-related synchronization (ERS) that occurs at the motor execution stage.

RESULTS—The children with CP had a stronger mean beta ERD during the motor planning phase and reduced mean gamma ERS at the onset of movement.

INTERPRETATION—The uncharacteristic beta ERD in the children with CP suggests that they may have greater difficulty planning knee joint movements. We suggest that these aberrant beta

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ERD oscillations may have a cascading effect on the gamma ERS, which ultimately affects the execution of the motor command.

A considerable amount of research effort has been placed on cataloging and quantifying the impairments seen in the final motor output of children with cerebral palsy (CP). These efforts have revealed that children with CP may present a wide variety of musculoskeletal impairments, and this has promoted the idea that the movement deficits seen in these children primarily reside in the musculoskeletal machinery. Unfortunately, treatment strategies that have focused on musculoskeletal impairments (i.e. surgical and strength training) have had mixed results, and their outcomes have not been clearly successful.[1,2] More recent therapeutic trends have shifted toward a task-orientated approach that focuses on the neurological impairments first, with the musculoskeletal impairments resulting from the neurological insult as a secondary issue. This approach places greater emphasis on the neurological basis for the impaired timing of the muscle activation patterns that result in co-contractions, spasticity, and weakness.[3] Despite this new paradigm shift, there is still only limited insight into the neurological foundation for how children plan and execute their movements.

Neural oscillatory activity in the sensorimotor cortices has received significant attention in electrophysiological studies of the motor system because it has been linked to the processes that occur during the planning and execution stages of movements. A plethora of electroencephalography (EEG) and magnetoencephalography (MEG) experiments have shown that cortical oscillatory activity across the sensorimotor cortices decreases in the beta frequency range (15–30Hz) before the onset of movement.[4–8] This decrease in the power of the beta band frequency, commonly termed beta desynchronization, is thought to reflect task-related changes in the firing rate of local populations of neurons as they begin to prepare for the specific demands of the pending movement. The consensus is that this beta event-related desynchronization (ERD) is related to the formulation of a motor plan, because it occurs well before the onset of movement and is influenced by the certainty of the movement pattern to be performed.[4–8]

Invasive electrocorticographic (ECoG) studies have also shown that the beta ERD is followed by an increase (or synchronization) in the high gamma frequency range (~50Hz) as the motor plan is executed.[9] This high frequency activity is restricted to a smaller population of neurons within the primary motor cortex and appears to follow the homuncular organization common in rolandic regions. Only within the past 5 years have MEG studies reported gamma-band neural oscillatory activity during movement, and the few studies that exist have shown that gamma band oscillations are concentrated in the pre-central gyrus and closely yoked to the onset of muscular activation.[5,6,10] Based on these initial findings, it has been proposed that the rapid and temporally succinct gamma response reflects the motor command execution signal. While the central role of beta and gamma neural oscillatory activity before and at the movement onset is well appreciated, there has been limited effort to use this knowledge to more precisely characterize the neurological basis of motor deficits seen in children with CP. Applying this knowledge has the potential to elucidate whether children with CP have motor planning deficits, or whether their poor motor control partly

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resides in the feed-forward motor execution command, or whether both of these alternatives play a significant role.

In this investigation, we used high-density MEG and beamforming methodology to examine the stage-like neural oscillatory activity in the sensorimotor cortices of children with CP, and children with typical development as they initiated the extension of their knee joint to a physical target. The knee joint was selected for this investigation because it is well recognized as a critical factor that limits the mobility of children with CP.[11] Our primary hypothesis was that children with CP would have altered beta ERD during the motor planning stage. Our secondary hypothesis was that children with CP would have an altered gamma ERS at the onset of the movement.

METHOD

Thirteen children with a diagnosis of either spastic diplegia (*n*=9) or hemiplegia CP (*n*=4) and in Gross Motor Function Classification System (GMFCS) levels I to III participated in this investigation (mean [SD] age 14y 3mo [9mo]; height 1.61m [0.08m]; weight 52.65kg [13kg]). One child with spastic diplegia was in GFMCS level I, five were in GMFCS level II, and three were in GMFCS level III. All of the children with a hemiplegia presentation had a GMFCS score of I. None of the recruited children had known large gray or white matter lesions that would have affected the cortical structure. Thirteen age- and sex-matched children with typical development (mean [SD] age 14y 1mo [9mo]; height 1.64m [0.06m]; weight 56.88kg [10kg]) also participated in this investigation and served as a comparison group. The Institutional Review Board at the University of Nebraska Medical Center reviewed and approved the protocol for this investigation. Informed consent was acquired from the parents and the children assented to participate in the study according to the Declaration of Helsinki.

Experimental paradigm

Throughout the knee-movement task, participants were seated in a custom-made nonmagnetic chair with their head positioned within the MEG helmet-shaped sensor array. Participants were instructed to fix their gaze on the physical target and to perform a single knee-movement every 4.1 to 4.7 seconds (variable interstimulus interval). An auditory tone was used to pace the child to extend their knee joint by 15° to touch a ridged bar that was fixed to the chair and positioned just proximal of the malleolus. Each participant performed approximately 115 trials and the total recording time was ~8.5 minutes. Before the MEG recording, all participants practiced the movement while positioned in the MEG recording chamber. A video camera was used to monitor the child during the experiment to ensure that he/she was successfully performing the motor task.

Magnetoencephalography 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 0.1–330Hz, neuromagnetic responses were sampled

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continuously at 1kHz using an Elekta Neuromag system (Elekta, Helsinki, Finland) with 306 MEG sensors, including 204 planar gradiometers and 102 magnetometers. Using the MaxFilter software (Elekta, Helsinki, Finland), each MEG dataset was individually corrected for head motion during task performance, and subjected to noise reduction using the signal space separation method with a temporal extension.[12]

Magnetoencephalography co-registration and structural magnetic resonance imaging processing

Four coils were fixed to the head of the participant and were used for continuous head localization during the experiment. Before 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 participant was positioned for MEG recording, an electric current with a unique frequency label (e.g. 322Hz) 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 co-registered with structural T1-weighted magnetic resonance imaging (MRI) data using three external landmarks (i.e. fiducials) and the digitized scalp surface points before source space analyses. Structural MRI data were aligned parallel to the anterior and posterior commissures and transformed into the Talairach coordinate system[13] using the volumetric subspace warping method implemented in BrainVoyager QX version 2.2 (Brain Innovations, Maastricht, the Netherlands).

Magnetoencephalography pre-processing

Artifact rejection was based on a fixed threshold method, supplemented with visual inspection. The data analysis epochs were a total duration of 4 seconds (–1.5s to 2.5s), with the auditory cue defined as time 0.0 seconds, and the baseline defined as –0.7 seconds to –0.2 seconds. Artifact-free epochs for each sensor were transformed into the time-frequency domain using complex demodulation (resolution: 2.0Hz, 25ms) 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. Based on this procedure, we identified that the beta ERD (14–28Hz) occurred from 0.4 to 0.9 seconds, and the high-frequency gamma ERS (38–56Hz) was from 0.75 seconds to 1.15 seconds. All passbands and time bins were chosen to focus on maximum responses across the sample, thus sacrificing some precision on the single-participant level to achieve a consistent analytical approach across participants.

Magnetoencephalography source imaging

The minimum variance vector beamforming algorithm was employed to calculate the source power across the entire brain volume.[14] 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

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input voxel space. Following convention, the source power in these images was normalized per participant using a separately averaged pre-stimulus noise period of equal duration and bandwidth.[15] Thus, the normalized power per voxel was computed for the selected frequency bands (beta ERD: 14– 28Hz, 0.4–0.9s; gamma ERS: 38–56Hz, 0.75–1.15s) over the entire brain volume per participant at 4.0 × 4.0 × 4.0mm resolution. Each participant's functional images, which were co-registered to anatomical images before beamforming, were transformed into a standardized space using the transform previously applied to the structural MRI volume.[13] MEG pre-processing and imaging used the Brain Electrical Source Analysis (BESA) software, version 5.3.2; (BESA GmbH, Gräefelfing, Germany), and MEG-MRI co-registration used BrainVoyager QX software, version 2.2.

Group effects were examined using a random effects analysis for the respective beta and gamma time-frequency components (TFC). One-sample *t*-tests were conducted to determine the activation patterns present in each group per TFC. In both cases (task and group effects), 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. We then 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 masking procedure was used to focus the analysis to the following areas: basal ganglia, bilateral cerebellum, pre- and post-central gyri, parietal cortices, and supplementary motor area. The mask was implemented in the Wake Forest University (WFU) Pickatlas using the automated anatomical labeling template.[16,17]

RESULTS

Beta event-related desynchronization

The children with typical development had a wide area of activation that included pre-central and post-central gyri, with a separate strong cluster in the basal ganglia ($p<0.001$, cluster-corrected). In the cortex, there were separate maximas in a very medial area (leg) and near the motor hand-knob feature of the pre-central gyrus (i.e. hand/arm area; Fig. 1). For the children with CP, the volume of activation was also large, but was centered on the post-central gyrus and was more restricted to the midline (Fig. 1). The group effect showed significantly stronger beta ERD present in the children with CP ($p<0.01$, cluster-corrected), with the significant areas being medial post-central gyrus stretching posterior to the superior parietal lobule (Fig. 2). No areas showed greater beta ERD activity in the children with typical development. The amplitude of the peak voxel in the group level image was extracted from each participant's functional map (e.g. single participant data) and is shown in Figure. S1 (online supporting information).

Gamma event-related synchronization

The children with typical development had a robust gamma ERS near the midline in the pre-central and post-central gyri, with other small islands of activity stretched across motor areas of the left hemisphere ($p<0.001$, cluster-corrected; Fig. 3). Activation was also significant in the cerebellum and the right prefrontal cortex. The children with CP showed no regions with

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significant gamma ERS responses, although small patches of gamma ERD were detected ($p<0.01$, cluster-corrected; Fig. 3). Finally, the group effect indicated significantly stronger gamma ERS near the midline in the pre-central and post-central gyri, and superior parietal areas of children with typical development ($p<0.01$, cluster-corrected; Fig. 4). The amplitude of the peak voxel in the group level image was extracted from each participant's functional map (e.g. single participant data) and is shown in Figure S2 (online supporting information).

DISCUSSION

Our results showed that children with CP had a stronger beta ERD and a reduced gamma ERS compared with the children with typical development. Since the beta ERD occurs during the motor planning stage, it is plausible that the altered cortical activity may reflect greater difficulty in selecting the proper muscular coordination to initially extend the knee joint. This assumption is in line with previous MEG research that has shown that changes in the beta ERD are related to the certainty of the motor performance.[7,8] In regard to the gamma ERS, children with CP failed to generate this response and this may be a direct consequence of difficulties in formulating an adequate motor plan. Taken together, these results suggest that the movement deficits noted in children with CP are partly a result of the aberrant neural oscillatory activity that occurs before the onset of movement.

The spatial location of the beta ERD was different between the children with CP and the children with typical development. The beta ERD for the children with CP was located in the post-central gyrus and superior parietal lobule. This suggests that the oscillatory activity that occurs during the motor planning stage may have a greater dependency on the neuronal groups that are involved in processing sensory information. This result was somewhat unexpected given that previous MEG and functional MRI results have shown that the responsiveness of the somatosensory cortices to external stimuli is diminished in children with CP.[18–21] Potentially, the critical distinction is that in our investigation the activity of the somatosensory cortices may have been used to select and/or compute an active movement pattern, while the previous studies were based on a passive external stimulation paradigm. Since children with CP have been noted to have sensory deficits, we speculate that the presence of the beta ERD in their sensory processing networks may represent a greater burden on these networks while formulating the motor plan.

There is considerable evidence to show that the high frequency gamma ERS emanates from a smaller population of neurons that are somatotopically-organized within the primary motor cortex and are tightly coupled with the onset of movement.[5,6,10] The children with typical development displayed typical gamma ERS responses located medially over the leg region of the pre- and post-central gyrus during the experimental motor task. Contrary to this finding, the high frequency gamma oscillations were uncharacteristically desynchronized for the children with CP, with maximas along the left post-central gyrus in the medial leg region. We suspect that the abnormal gamma activity may actually be a downstream effect of the neural oscillatory beta activity that occurs during the motor planning stage. Intuitively, if the sensorimotor cortices cannot properly formulate a motor plan, then the oscillatory activity associated with the motor execution command will be faulty.

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It has been well-established in previous experimental work that the brain maintains and updates an internal model that is used to predict the ideal muscular synergies to achieve a motor goal.[22] The internal model is used to formulate a motor plan, which is transformed into a feed-forward motor command that is assumed to achieve the goal state. The majority of research on the motor impairments seen in children with CP has focused on the final motor output, and potential deficits in the motor plan have been largely ignored. Outcomes from the few behavioral investigations that have evaluated motor planning deficits in children with CP have shown that these children have difficulty in anticipating their grip forces, take longer to meet a prescribed target force, and take longer to plan sequential movements.[23–25] All together these behavioral results suggest that children with CP may not have an appropriate internal model of their musculoskeletal system, which limits their ability to develop an effective motor plan that will meet the motor goal. It is likely that the children with CP who participated in this investigation may have had a faulty internal model for predicting the outcomes of the knee extension trajectory. In principle, the formulation and updating of the internal model is a result of the sensory consequences of the selected motor command.[22] Therefore, we suspect that a poorly developed model may be related to the somatosensory deficits that are often noted in children with CP.[18–21]

Conclusion

Our results show that children with CP have uncharacteristic somatosensory cortical oscillations during the motor planning and execution stages. These results provide new insight into the potential neural basis for the motor planning and control deficits seen in these children. Further studies are required to link changes in these stage-like oscillations to the biomechanical deficiencies seen in the movement patterns of children with CP. Moreover, we suggest that the beta ERD and gamma ERS measures used in this investigation may provide a new metric for gauging the success of the current task-orientated treatment approaches that have a neurological basis.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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ABBREVIATIONS

ERD	Event-related desynchronization
ERS	Event-related synchronization
MEG	Magnetoencepholagraphy
TFC	Time-frequency components

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What this paper adds

- Children with CP have uncharacteristic beta event-related desynchronization during the motor planning period.
- The sensorimotor cortices have difficulty computing the proper motor output.
- Children with CP have weaker gamma ERS during motor execution.
- The sensorimotor cortices have difficulty executing the motor plan.
- Improving the motor planning stage may improve the final motor performance.

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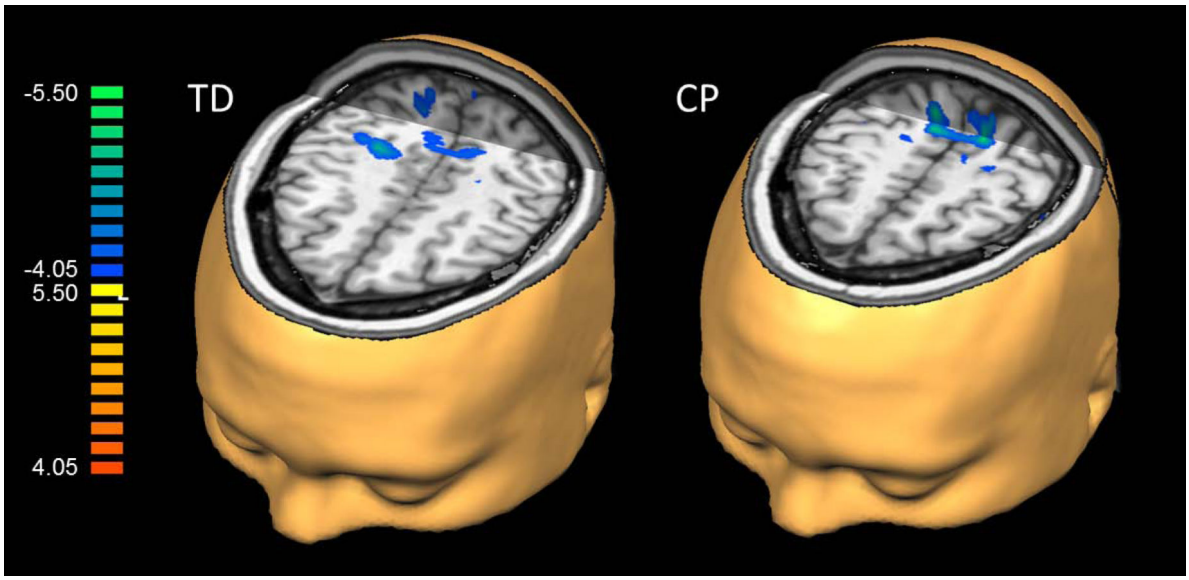

Figure 1.

Statistical parametric maps of the beta event-related desynchronization task effect in children with typical development (TD) and children with cerebral palsy (CP). Both maps have been thresholded at (p<0.001, cluster-corrected) and the scale of t-values is shown on the left. As can be discerned, both groups activated medial sensorimotor areas within the pre-central and post-central gyri, probably corresponding to the sensorimotor leg representation.

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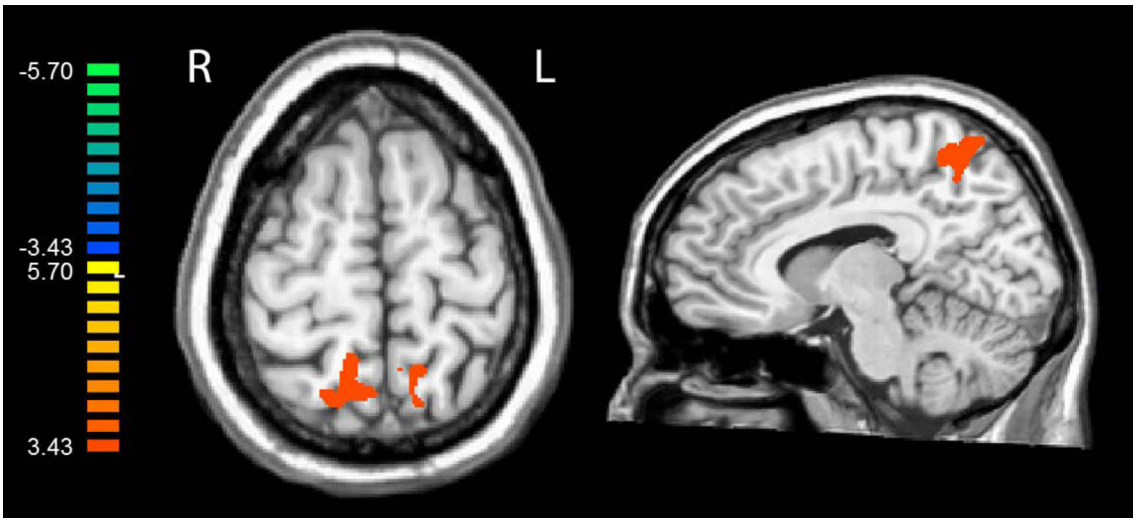

Figure 2.

Two-dimensional statistical parametric maps of the group effect for the beta event-related desynchronization (ERD) response. Images have been thresholded at (p<0.01, cluster-corrected) and are displayed following the radiological convention (R=L). Scale of t-values appears to the left. As shown, the children with cerebral palsy exhibited significantly stronger beta ERD responses relative to children with typical development in the post-central gyri and superior parietal lobule.

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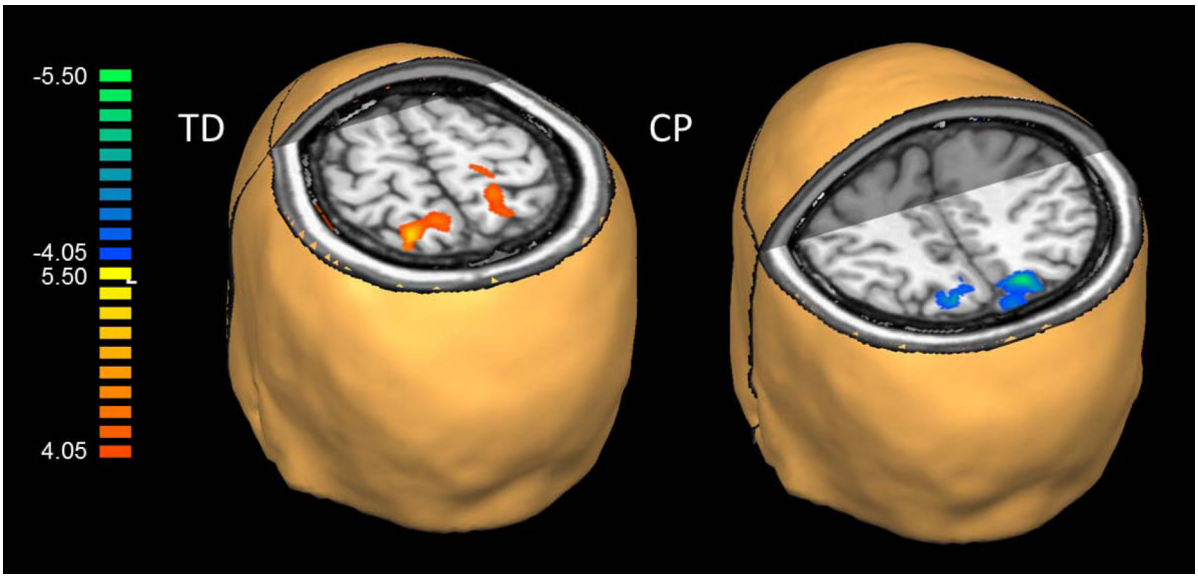

Figure 3.

Statistical parametric maps of the gamma event-related synchronization (ERS) task effect in children with typical development (TD) and children with cerebral palsy (CP). Both maps have been thresholded at (p<0.001, cluster-corrected) and the scale of t-values is shown on the left. As can be discerned, children with typical development activated medial regions along the pre-central and post-central gyri, as well as anterior areas of the superior parietal lobule. In contrast, children with CP showed no regions with significant gamma ERS, but instead exhibited gamma event-related desynchronization in the superior parietal and surrounding area.

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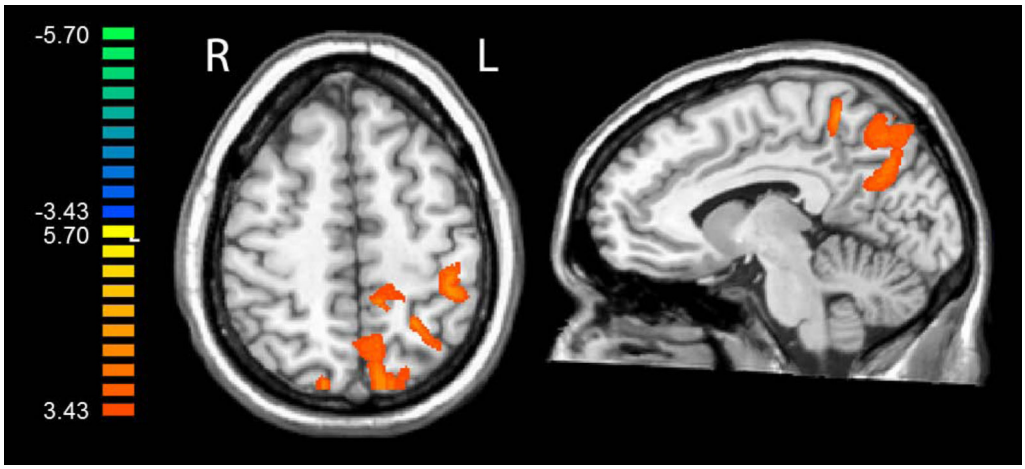

Figure 4.

Two-dimensional statistical parametric maps of the group effect for the gamma event-related synchronization. Images have been thresholded at (p<0.01, cluster-corrected) and are displayed following the radiological convention (R=L). Scale of t-values appears on the left. As shown, the children with typical development exhibited significantly stronger gamma responses relative to children with cerebral palsy in the medial post-central gyri and superior parietal lobule.