



RESEARCH ARTICLE

Power training alters somatosensory cortical activity of youth with cerebral palsy

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Abstract

Objective: Our prior magnetoencephalographic (MEG) investigations demonstrate that persons with cerebral palsy (CP) have weaker somatosensory cortical activity than neurotypical (NT) controls, which is associated with reduced muscular strength and mobility. Power training can improve lower extremity isokinetic strength, muscular power, and walking performance of youth with CP. Potentially, these clinically relevant improvements are partially driven by changes in somatosensory processing. The objective of this investigation was to determine if power training has complementary changes in muscular function and somatosensory cortical activity in youth with CP. **Methods:** A cohort of youth with CP ($N = 11$; age = 15.90 ± 1.1 years) and NT controls ($N = 10$; Age = 15.93 ± 2.48 years) participated in this investigation. Youth with CP underwent 24 power training sessions. Pre-post bilateral leg press 1-repetition maximum (1RM), peak power production, 10-m walking speed, and distance walked 1-min were used as outcome measures. MEG neuroimaging assessed the changes in somatosensory cortical activity while at rest. NT controls only underwent a baseline MEG assessment. **Results:** Youth with CP had a 56% increase in 1RM ($p < 0.001$), a 33% increase in peak power production ($p = 0.019$), and a 4% improvement in 1-min walk ($p = 0.029$). Notably, there was a 46% increase in somatosensory cortical activity ($p = 0.02$). **Interpretation:** These results are the first to show that power training is associated with improvements in muscular function, walking performance, and the resting somatosensory cortical activity in individuals with CP. This treatment approach might be advantageous due to the potential to promote cortical and muscular plasticity, which appear to have carryover effects for improved walking performance.

Introduction

Cerebral palsy (CP) is one of the most prevalent pediatric movement disorders worldwide, with 3 in 1000 children receiving a diagnosis in the United States alone.¹ CP is primarily characterized by musculoskeletal impairments that are the result of a prenatal or perinatal injury to the developing brain.² Although the initial brain insult remains static, as the child grows, secondary musculoskeletal alterations and motor impairments develop.³ The muscles tend to be significantly weaker and have decreased fascicle

length and a reduction in the cross-sectional area compared to neurotypical (NT) controls.⁴ Moreover, previous work has demonstrated that youth with CP appear to lack the ability to maximally activate their motor units compared to NT controls.^{5,6} As a result, the ability of youth with CP to perform activities of daily living might be associated with their diminished walking capacity.⁷

Strength training has been one of the primary treatment approaches for youth with CP since it targets the muscular weakness that is often reported to be partly related to the decreased walking capacity in this patient

population.^{8,9} However, the outcomes from this treatment approach have been mixed with many participants demonstrating minimal walking improvements.^{8,9} These perplexing outcomes have been perceived to be related to the idea that maximal strength is not necessary to perform many activities of daily living. Rather, deficiencies in the ability to rapidly recruit the available motor units and rate coding of the respective motor units may play a larger role in the walking deficits seen in youth with CP. Recognizing these neurophysiological deficits, the current therapeutic interests have shifted from strength training toward high-velocity power training.

Power training involves the production of rapid muscular contractions performed at submaximal force production levels,¹⁰ whereas traditional strength training involves heavier loads moved at slower velocities. Power training has been found to be equally effective as strength training in increasing muscular strength.¹¹ Furthermore, prior investigations have identified power training to be more efficacious in increasing movement velocity, muscle power production, and the walking capacity in youth with CP.^{12–16} Although it is recognized that power training can promote muscular plasticity,^{13,15} far less consideration has been given to the potential changes in the nervous system or the origin driving these changes. Our recent magnetoencephalographic (MEG) brain imaging investigation has begun to probe the possible neurophysiological mechanisms responsible for the improved muscular performance.¹⁷ These results suggest that power training has the potential to result in changes in the sensorimotor cortical activation that are linked with the ability to produce greater leg muscular power. Hence, suggesting that the driver of the muscular performance gains might be associated with improved motor cortex function.

The definition of CP has evolved to also include alterations in somatosensation and perceptions.² This inclusion is due to the accumulating evidence of altered proprioception, stereognosis, and tactile discrimination reported in the clinic.^{18–24} Hand sensory deficits have been shown to be closely associated with upper extremity motor impairments,^{25,26} prediction of grip force,²⁷ and an individual with CP's capacity to learn a novel upper extremity motor skill.²² Although very few investigations have examined lower extremity somatosensory function in CP,^{28–30} these studies have reported that the somatosensory deficits also persist in the lower extremities, specifically deficits in position awareness of the knee²⁹ and hip.³⁰ Furthermore, these lower extremity sensory deficits were found to influence postural control, gait, and the motor performance of youth with CP.^{28,31,32}

MEG and electroencephalography (EEG) brain imaging studies have identified that the neurophysiological underpinnings of the altered somatosensory processing noted in youth with CP are partly due to weaker somatosensory

cortical oscillations in theta-alpha (4–14 Hz) and beta (18–34 Hz) activity.^{33–36} These attenuated frequency-specific neuronal oscillations appear to be related to deficits in the ankle plantar flexor strength and walking performance of youth with CP.^{34,35} In addition to measuring oscillatory activity, several studies have also evaluated the time-domain somatosensory cortical responses. These investigations have consistently found similar outcomes for youth with CP, in that the somatosensory-evoked cortical activity for both upper and lower extremities is attenuated and, in some cases, has longer response latencies.^{33,37–42} Although our recent MEG neuroimaging investigation has identified that power training can alter the sensorimotor cortical activity,¹⁷ its influence on the sensory processing deficits seen in this patient population remains unknown. It is possible that the heightened feedback from the sensory afferent during the high-velocity leg performance might provide critical information for learning how to recruit the motor units. However, this conjecture has yet to be assessed.

The primary aim of this investigation was to use MEG brain imaging to evaluate potential changes in the somatosensory cortical responses of youth with CP after 8 weeks of a lower extremity power training protocol. We hypothesized that: (1) at baseline, the youth with CP would have weaker somatosensory cortical activity when compared with NT controls, (2) that youth with CP would exhibit increased strength, muscular power production, and walking performance following power training, and (3) that somatosensory cortical activity would trend toward what is seen in the NT controls following power training.

Methods

Participants

Using the effect size (1.3) for the pre-post difference in the lower extremity peak power seen in youth with CP after undergoing a high-velocity power training protocol,¹³ six participants will provide >85% power to detect a similar difference at a 0.05 alpha level. Hence, we enrolled 11 participants with CP that had either a spastic diplegic or hemiplegic presentation (age = 15.9 ± 1.1 years; 8 males; Gross Motor Function Classification Score [GMFCS] levels I–III) for this investigation. Individuals with GMFCS levels of I–II typically ambulate independently, although with slowed gait speed and uncharacteristic gait patterns.⁴³ Individuals with GMFCS level of III require assistive devices to ambulate, such as crutches, ankle-foot orthoses, or wheeled walkers. An additional 10 NT controls (age = 15.93 ± 2.48 years; 5 males) completed the MEG imaging portion of this experiment. Their data were used to determine whether the potential neurophysiological changes seen in the youth with CP were

trending toward what is seen in NT individuals. The Institutional Review Board reviewed and approved this investigation. Informed consent was acquired, and the youth assented to participate in the experiment. This investigation was registered and performed under the following ClinicalTrials.gov Identifier: NCT03555708.

Power training protocol

The power training protocol was conducted by licensed pediatric physical therapists three times a week for 8 weeks (B. C. & H. R.) (Citation: Moreau, N.G., Power training intervention protocol. 2018: Louisiana State University Health Sciences Center - New Orleans.). Hence, there were 24 total sessions and a required 1-day of rest between each training session. Each treatment session began with a 5-min warm-up that consisted of overground walking and practicing leg presses below resistance and velocity thresholds. The training consisted of unilateral and bilateral leg presses on a Total Gym GTS® system (San Diego, CA, USA), which primarily target the quadriceps followed by the hip extensors and plantar flexors. The Total Gym GTS® system allowed for squat-like movement patterns to be performed while safely reclined in a supine position for postural support that was tailored to each participant depending on his/her initial level of capability. The sled was positioned at a 30-degree incline for all participants, and weights were added to a weight bar to increase resistance. The treatment loads utilized were individualized for each participant. At 30-degrees, the participant would be lifting 50% of their body weight when the sled angle was at 30°. Since the 1-repetition maximums for all of the subjects were greater than their body weight, there was no need to place the sled at an angle that was <30°. The individualized training loads were between 40% and 80% of the participant's 1-repetition maximum (1RM) with progression toward 80%. The training consisted of the participant performing 6 sets of 5 repetitions for each leg separately and bilaterally. The instructions for the concentric phase were to push as fast as possible, and for the eccentric phase to lower the weight in a slow and controlled fashion over a 1–2 sec time period. To minimize fatigue, 1–2 min of rest were given between sets. The training procedures utilized in this investigation were similar to what has been used in the strength and conditioning literature for the optimal development of muscular power.⁴⁴

Clinical outcomes

The Total Gym GTS® system was retrofitted with a linear cable sensor (SGD-120-3, TE Connectivity, Chatsworth, CA, USA) and a custom LabView program was used to quantify the sled velocity at a 1 kHz sampling rate. Power

was calculated as the product of the velocity and the amount of weight lifted. A true 1RM was attempted with all participants on the Total Gym. In cases where 1RM was not achievable due to the weight capacity of the Total Gym GTS, approximate 1RM was calculated with the Brzycki equation (Brzycki, 1993). The amount of weight added to the sled during the power test was 50% of the participant's baseline 1RM. Five trials were performed for the power test, and the largest peak power produced across the respective trials was used as the outcome variable. The pre- and post changes in the 1RM were also used to assess the changes in the muscular performance.

Additional clinical outcomes measures included a 1-min walk test and the 10-m preferred walk test. During the 1-min walk test, the participants walked back and forth in a 40-m hallway and were instructed to walk as far and fast as possible during the time interval. The 10-m walk test was performed at the preferred walking speed.

MEG acquisition and experimental paradigm

The participants with CP underwent pre/post MEG imaging, while the controls completed a single MEG scan. All recordings were conducted in a one-layer magnetically shielded room with active shielding engaged for advanced environmental noise compensation. With an acquisition bandwidth of 0.1–330 Hz, neuromagnetic responses were sampled continuously at 1 kHz using a MEGIN MEG system (Helsinki, Finland) with 306 sensors, including 204 planar gradiometers and 102 magnetometers. 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.⁴⁵

Throughout the MEG experiment, the participants were seated in a custom-made nonmagnetic chair with their head positioned within the MEG helmet-shaped sensor array while focusing their gaze on a fixation cross. Electrical stimulation was applied to the right tibial nerve using external cutaneous stimulators connected to a Digitimer DS7A constant current stimulator system (Digitimer Limited, Letchworth Garden City, UK). A single-pulse, unilateral electrical stimulation was applied using electrodes that were affixed to the skin overlying the right tibial nerve. The intensity of stimulation was set to the individual's motor threshold where there was an overt muscle twitch in the digits of the foot. During the experiment, a 200 µsec constant-current square wave single-pulse stimulation was applied every 2 sec for 4 min yielding a total of 120 trials. The stimulus intensity used for the baseline MEG assessment was the same for the post-power training MEG assessment.

MEG coregistration

Four coils were affixed to the head of the participant 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 participant was positioned for the 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 the 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 were coregistered with an MRI prior to source reconstruction.

MEG preprocessing

Cardiac artifacts were removed from the data using signal-space projection, which was accounted for during source reconstruction.⁴⁶ The continuous magnetic time series was divided into epochs of 1100 msec duration, from -500 to 600 msec with the baseline being defined as -400 to -100 msec and 0.0 msec being stimulation onset. Epochs containing artifacts (e.g., eye blinks, muscle artifacts, etc.) were rejected based on a fixed-threshold method using individual amplitude and gradient thresholds and supplemented with a visual inspection. The number of trials accepted between groups was not significantly different (CP = 110.55 ± 5.6 , TD = 112.5 ± 4.19 , $p = 0.31$), nor did the trials significantly differ between pre- and post sessions in the group with CP (pre = 110.76 ± 2.35 , post = 110.44 ± 1.62 , $p = 0.71$).

Sensor-level analysis

The artifact-free epochs were averaged across trials to generate a mean time series per sensor and participant, and the specific time windows used for subsequent source imaging were determined by statistical analysis of the sensor-level time series across all participants using the entire array of gradiometers. Each data point in the time series was initially evaluated using a mass univariate approach based on the general linear model. To reduce the risk of false-positive results while maintaining reasonable sensitivity, a two-stage procedure was followed to control for Type 1 error. In the first stage, paired-sample *t*-tests were conducted to test for differences from baseline at each data point and the output time series of *t*-values was

threshold at $p < 0.05$ to define time bins containing potentially significant phase-locked activity across all participants. In stage two, the time points that survived the threshold were clustered with temporally and/or spatially neighboring time points that were also above the threshold ($p < 0.05$), and a cluster value was derived by summing all the *t*-values of all data points in the cluster. Nonparametric permutation testing was then used to derive a distribution of cluster values and the significance level of the observed clusters (from stage one) was tested directly using this distribution.⁴⁷ For each comparison, 1000 permutations were computed to build a distribution of cluster values. Based on these analyses, the time windows that contained significant phase-locked events across all participants were used to guide subsequent time-domain source level analysis.

Source imaging (sLORETA)

Time-domain source images were computed using standardized low-resolution brain electromagnetic tomography (sLORETA).⁴⁸ The resulting whole-brain maps were 4-dimensional estimates of current density per voxel, per time sample across the experimental epoch. These data were normalized to the sum of the noise covariance and theoretical signal covariance, and thus, the units are arbitrary. These maps were then averaged temporally over the time windows identified in the sensor-level analysis. The resulting maps were then grand-averaged across all participants to determine the location of the peak voxel. From this peak voxel, the sLORETA units were extracted to derive estimates of the time-domain response amplitude for each participant. All imaging procedures were done with the Brain Electrical Source Analysis (BESA) software (BESA v7.0; Grafelfing, Germany). For a more detailed description of our imaging methodology, see Wiesman and Wilson.⁴⁹

Statistical analysis

Separate paired sample *t*-tests were used to evaluate the potential pre/post change in the peak somatosensory cortical activity and respective clinical outcome measures. In addition, independent samples *t*-tests were used to examine how pre- and post-training somatosensory cortical responses compared to the NT control group. All statistical analyses were conducted at a 0.05 alpha level.

Results

Participants with CP had a 95% compliance with the power training protocol (22.8 out of 24 sessions) and completed all clinical outcome assessments. Two participants with CP were excluded from the final MEG analysis due to technical issues with the stimulator during either

the pre- or post acquisition time points. Hence, the final analysis of the pre/post MEG data was comprised of 9 youth with CP and 10 NT controls.

Clinical outcomes

Overall, the youth with CP increased their bilateral 1RM (pre = 158.3 ± 24.7 kg, post = 247.5 ± 41.5 kg, $p < 0.001$, Fig. 1A) and peak muscular power production (pre = 509.9 ± 64 W, post = 677.1 ± 114.3 W, $p = 0.019$, Fig. 1B) following the power training. The youth with CP also had a significant improvement in the distance they could walk for 1-min (pre = 74.5 ± 9.2 m, post = 80.8 ± 8.4 m, $p = 0.029$, Fig. 1C). The 6.4 unit change in the 1-min walk represented a medium minimum clinically important difference (MCID = 5.6)⁵⁰ suggesting that the improvements were clinically discernable. However, there were no changes in the preferred 10-m walking speed (pre = 1.09 ± 0.11 m/sec, post = 1.09 ± 0.09 m/sec, $p = 0.492$). Lastly, there were no correlations with the magnitude of the change in the respective clinical outcomes and the GMFCS levels of the participants (1RM $p = 0.873$, peak power $p = 0.988$, 1-min walk $p = 0.841$).

MEG findings

Sensor-level permutation testing identified that the somatosensory cortical activity was significantly different from baseline during the 115–200 msec time window. Source activity estimates were averaged across this window and across all participants. The resulting grand-averaged sLORETA image demonstrates that the source of the neural response was the leg region of the somatosensory cortices (Fig. 2A). As shown in Figure 2A, there was a robust increase in neural power within the leg region of

the contralateral somatosensory cortices. The plot represents the average time course of the somatosensory cortical activity extracted from the peak voxel in the grand average sLORETA image. As shown, the magnitude of the somatosensory cortical response during the 115–200 msec time window was significantly weaker in the individuals with CP compared with controls prior to starting the power training (NT = 323.53 ± 71.15 AU, CP pre = 136.78 ± 46.45 AU, $p = 0.02$). However, the somatosensory cortical response (i.e., pre vs. post) notably increased after the participants with CP completed the power training (CP post = 199.98 ± 46.45 AU, $p = 0.02$, Fig. 2) and was not different from the controls after completing the power training ($p = 0.32$, Fig. 2). Lastly, there was no correlation with the magnitude of the amount of change in the somatosensory cortical activity and the GMFCS levels of the participants ($p = 0.799$).

Discussion

The primary aim of this investigation was to examine the neurophysiological changes in the somatosensory cortices of youth with CP after completing a power training intervention. To this end, we utilized MEG brain imaging to quantify the somatosensory cortical activity that serves the foot while at rest and a series of clinical assessments of lower extremity muscular function and walking performance. Our results are the first to show that power training has the potential to promote improvements in somatosensory cortical activity. Furthermore, our results indicate that these neurophysiological changes are associated with the ability of youth with CP to improve their lower extremity muscular strength and power production. Overall, these results imply that power training has the potential to result in muscular changes and neuroplastic

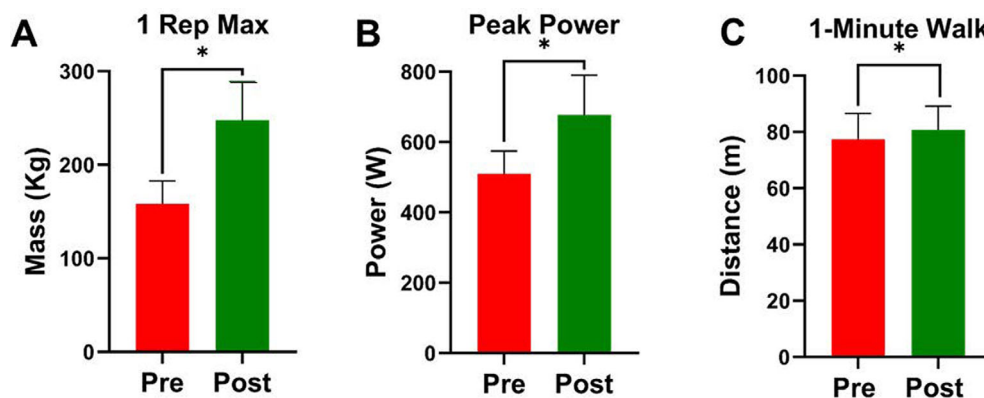


Figure 1. Clinical outcomes. Pre- and post-power training differences in muscular strength (A), muscular power (B), and distance walked in 1-min (C). Overall, these results show that the individuals with cerebral palsy had clinically relevant improvements in their muscular strength, muscular power, and ability to walk further in 1-min. All values are mean \pm standard error. * $p < 0.05$.

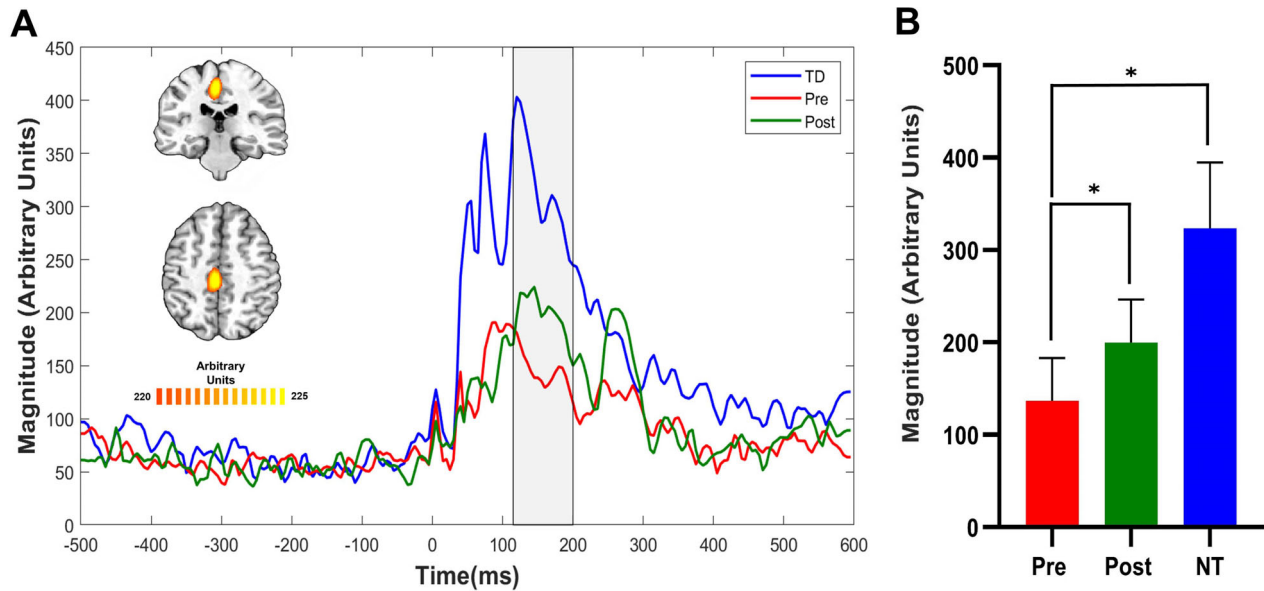


Figure 2. Somatosensory cortical activity. (A) Grand-averaged sLORETA images of the source of the cortical somatosensory response (inset). The blue trace is the average neural time course for the controls, whereas the red time course is the average for the participants with cerebral palsy pre-power training, and green corresponds to post-power training. Time (msec) is denoted on the *x*-axis, with 0 msec defined as the stimulus onset, and the magnitude of the evoked response (arbitrary units) denoted on the *y*-axis. The gray box designates the evaluated time window. (B) Average somatosensory cortical activity across the 115–200 msec time window. All values are mean \pm standard error. * $P < 0.05$.

changes. Further discussion of our experimental findings appears in the following sections.

Compared with NT controls, our results show that the somatosensory cortical responses before undergoing power training were reduced in amplitude for youth with CP. This is in line with several neuroimaging studies that have previously identified weaker somatosensory processing in youth with CP, in response to both tactile and electrical stimulation paradigms.^{18,20,34,35,41,42,51–53} Hence, there is substantial evidence that the altered activity seen in the somatosensory cortices likely contributes to the perceptual deficits that are reported clinically for this patient population. The weaker somatosensory cortical activity seen in the youth with CP could be dependent on several possible neurophysiological mechanisms. For one, the weaker activity might be related to fewer neurons and/or synaptic connections in the cortical area that represents the foot response to the peripheral stimulations. Alternatively, it is possible that the weaker somatosensory activity might be related to heightened γ -aminobutyric acid (GABA) interneuronal activity. The latter scenario would align with prior PET studies that have shown youth with CP tend to have increased GABA_A receptor binding potential within the sensorimotor cortices.^{54,55}

Our results also demonstrated improvements in lower extremity strength (56.4% change), peak muscular power

production (32.8% change), and distance walked in 1-min (4.4%). The minimal clinically important change score (MCID) for the distance walked for 1-min was medium indicating that the improvements were clinically discernable.⁵⁰ However, a previous investigation utilized ecologically relevant motor tasks (i.e., walking, stair climbing, and pushing a chair) as its method for power training demonstrated a greater improvement in the 1-min walk test (13% change) than what was seen here.¹⁵ We suspect that these discrepancies might be explained by the training specificity of our approach. In other words, our protocol utilized a leg press motor task and not tasks that specifically trained walking performance *per se*. Nonetheless, our results do demonstrate that the power training exercises prescribed for the Total Gym GTS still might have a good potential for improving the distance youth with CP can walk for 1-min.

The key finding of this study was that the somatosensory cortical activity that serves the foot while at rest became notably stronger after the participants with CP completed the power training. Furthermore, the strength of the somatosensory cortical activity after power training was similar to what was seen in the controls. Together these results imply that the somatosensory cortical processing was vastly improved in the participants with CP. The clinical gains seen after power training have primarily been attributed to alterations in the muscle and/or

activation of the motor cortices.^{17,56} Our results imply that improvements in muscular strength and power seen after the training might also be partially attributed to alterations in the somatosensory cortical processing. Power training requires the participant to pay attention to the lower extremity's performance to gauge the speed of the concentric contraction as they learn how to effectively recruit the available motor units and coordinate the lower extremity joint movements to meet the task demands. We suspect that the directed attention is attributed to the recalibration of the somatosensory cortical processing seen in this investigation. This notion is aligned with a prior behavioral investigation that identified learning a motor task heightens the sensory perceptions of NT controls.⁵⁷

Primate animal models and high-field fMRI studies have shown that the cytoarchitecture of the primary somatosensory cortex is organized into Brodmann Areas 3a, 3b, 1, 2, and 5.^{58–63} The consensus is that Area 3a processes sensory information from the Ia afferents, while Areas 3b, 1, and 2 are involved in the processing of afferent information from the mechanoreceptors.^{64,65} Area 5 has been shown to be involved in the processing of joint proprioceptive information.⁶⁶ Prior EEG and MEG studies stimulating the median nerve have suggested that the early components of the evoked somatosensory fields (i.e., N20) reside in Area 3b of the primary somatosensory cortices, while the later (i.e., P100) components correspond to Area 1 and the SII.^{67,68} Similar MEG findings have been reported for the tibial nerve, although the later components have been more closely associated with Area 5.⁶⁹ Our statistical permutation testing approach suggested that the somatosensory cortical activity evoked after stimulating the tibial nerve was primarily different during the latter 115–200 msec time window. This would suggest that the altered somatosensory cortices activity seen after power training might represent the improved activity of the neural generators in Area 5 that are involved in the processing of joint position sense.

Finally, our results imply that the site of the improved somatosensory function primarily resides at the cortical level. However, one should also consider whether this altered cortical activity might be reflective of changes occurring at the spinal cord and/or in the peripheral receptors. Prior animal models have shown that inhibition at the cortical level can result in the corticospinal tracts terminating in the dorsal horn of the spinal cord that is typically occupied by the interneurons that process afferent sensory information.⁷⁰ Furthermore, it has been demonstrated that increases in the cortical activity through forced use of the impaired limb can alter the corticospinal terminations toward a normal topology and

that this reorganization is associated with improved motor function.⁷¹ Based on this evidence, it is possible that the forced use of the impaired legs during power training heightens the activity of the sensorimotor cortices, which, in turn, alters the organization of the spinal cord neural generators for improved processing of the peripheral afferent feedback. Potentially, this downstream neuroplasticity might improve the signal-to-noise levels of the sensory feedback that is sent to the cortex. This, in turn, could improve the strength of the somatosensory cortical activity. Although this premise seems plausible, further research is necessary to identify the neurophysiological changes that are occurring at the spinal cord and cortical levels to better delineate the mechanisms that are driving the somatosensory cortical changes seen after power training.

Study limitations

Although our power analysis indicated that our sample size was adequate, the outcomes from this investigation may not be generalizable to the wider population of individuals with CP. Specifically, there might be relevant differences in the cortical and muscular performance changes seen in patient populations with different GMFCS levels and presentation types (e.g., hemiplegic vs. diplegic). Upon completion of this investigation, it was also apparent that the tenants of producing a high-velocity movement were unfamiliar to many of our participants. Furthermore, there were some challenges for the therapist to gauge if the leg press was performed with sufficient velocity. Potentially, real-time feedback that is provided to the patient as well as the physical therapists during the power training might promote even greater cortical and clinically relevant changes. Clinical tests of sensory function were also not included in this investigation. However, we are somewhat cautious in listing this as a limitation as these clinical tests require the participant to self-report their sensations, which may bias the reliability of these metrics. We contend that the somatosensory neurophysiological metrics used in this investigation may provide a more objective measure of somatosensory processing since they do not rely on patient reports. That being said, the MEG methods employed in this investigation were acquired while at rest. Potentially, the somatosensory processing may be different for functional/weight-bearing tasks. Lastly, the power training was only performed with the youth with CP and not the controls. We suggest that the premise that power training improves somatosensory cortical activity would be further strengthened if similar changes were also demonstrated in controls.

Conclusions

The overall outcomes of this investigation imply that changes in the strength of the somatosensory cortical activity likely occur after individuals with CP complete a power training protocol. These cortical changes appear to complement the changes in the peak muscular power production and strength. Together these results suggest that power training may promote neuroplastic changes in the somatosensory cortices that might partially drive the improvements in lower extremity motor function. The positive outcomes portrayed in this study support the need for further studies that explore the neurophysiological tenants of power training for youth with CP.

Author Contributions

Conceptualization of hypotheses and experimental design (MJK, TW, NGM), design of physical therapy intervention (NGM), performed physical therapy intervention (BC, HR), data collections (HB, BC, SB, HR, MJK), data processing and statistical analyses (HB, MT, MJK, TW), draft and conceptualization manuscript (HB, TW, MJK), editing of final manuscript (all authors)

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Conflict of Interest

The authors have no conflict of interest to declare.

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