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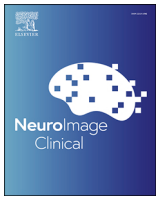

Overground gait training improves the sensorimotor cortical dynamics and mobility of persons with cerebral palsy

Morgan T. Busboom [a], Rachel K. Spooner [a,b], Liana S. Chinen [a], Sarah E. Baker [a], Brad Corr [a], Katie L. Bemis [a], Kimberley Scott [a,c], Tony W. Wilson [a,b], Max J. Kurz [a,b,*]

[a] Institute for Human Neuroscience, Boys Town National Research Hospital, Omaha, NE, USA
[b] Deparment of Pharmacology and Neuroscience, Creighton University, Omaha, NE, USA
[c] Department of Physical Therapy, Creighton University, Omaha, NE, USA

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ABSTRACT

Objective: Persons with cerebral palsy (CP) exhibit aberrant sensorimotor cortical oscillations linked to uncharacteristic motor actions and mobility, but the key physical therapy ingredients needed to offset these aberrations and drive improvements in cortical function remain unclear. This study evaluated whether overground gait training results in mobility gains that are coupled to beneficial changes in sensorimotor cortical oscillations in persons with CP. 34 persons with CP (Age = 20.58 ± 7.61yrs; Gross Motor Functional Classification Scores I-III) and 32 neurotypical (NT) controls (Age = 23.06 ± 3.79yrs) participated. Persons with CP completed 24 overground gait training sessions (3 days/week for 8 weeks). A battery of clinical assessments were used to examine changes in functional mobility. Magnetoencephalographic (MEG) imaging was used to quantify sensorimotor cortical oscillations while performing a knee extension motor task pre- and post-therapy in the CP group. Only one MEG measurement was completed by NTs. Persons with CP improved their Functional Gait Assessment scores by 8.77 % ($p < 0.001$), dynamic range by 38.94 % ($p < 0.001$), and Timed Up-and-Go (TUG) time by 15.97 % ($p < 0.001$). Post-training, sensorimotor beta oscillations during movement planning and execution became 19.95 % stronger ($p < 0.001$), approaching NT levels. Movement-related gamma oscillations became 37.88 % weaker ($p = 0.003$) and no longer differed from NTs post-therapy ($ps > 0.05$). Notably, the persons with CP who exhibited the largest increases in sensorimotor beta oscillations tended to have greater improvements in the TUG ($p = 0.025$). Overground gait training enhanced sensorimotor cortical oscillations and yielded clinically meaningful mobility gains. These neuroplastic and functional improvements may stem from gait-related tasks emphasizing movement planning, execution, and problem-solving.

1. Introduction

Cerebral palsy (CP) is an umbrella term describing a group of movement disorders resulting from an insult to the perinatal brain (Rosenbaum et al., 2007). Although CP is classified as a non-progressive neurological condition, persons with cerebral palsy often experience a decline in mobility as they age (Hurvitz et al., 2021). Maintaining mobility is an essential factor in independent self-care and active participation in society. However, therapeutic resources (*e.g.*, provider knowledge and evidence-informed interventions) aimed at supporting sustained mobility in this population are limited. As such, when individuals with CP age, they are met with a healthcare system that lacks the specialized knowledge required to address their changing mobility needs effectively (Hurvitz et al., 2021; Peterson and Hurvitz, 2021). A further concern is that many physical therapy approaches that have tried to address maintaining mobility have continued to focus primarily on the musculoskeletal system, despite the key neurological components of the condition. Optimization of long-term functional mobility outcomes will require a comprehensive approach addressing both the musculoskeletal and neurophysiological factors (Damiano, 2009). Therefore, the development of neuroscience-informed physical therapy interventions are critically needed to enhance and maintain mobility in persons with CP throughout their lifespan. Examining the neurophysiological mechanisms (*e.g.*, changes in cortical oscillatory activity in the sensorimotor

* Corresponding author at: Department of Neuroscience and Pharmacology, Creighton University School of Medicine, Institute for Human Neuroscience, Boys Town National Research Hospital, 14090 Mother Teresa Ln, Boys Town, NE 68010, USA.
E-mail address: max.kurz@boystown.org (M.J. Kurz).

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cortex) that may be impacted by these interventions should provide key insight into the response variability in treatment outcomes and help refine therapeutic paradigms aimed at prolonged functional mobility.

Ongoing magnetoencephalographic (MEG) brain imaging investigations have demonstrated clear aberrations in sensorimotor cortical oscillations in the beta (~15–30 Hz) and gamma (~70–90 Hz) frequency ranges in children and adults with CP (Busboom et al., 2022; Hoffman et al., 2019; Kurz et al., 2014; Kurz et al., 2017; Kurz et al., 2020; Kurz et al., 2024; Trevarrow et al., 2022). Decreases in beta power or event-related desynchronizations (ERD) are observed prior to movement onset and sustained throughout movement duration (Kurz et al., 2014; Cheyne et al., 2006; Engel and Fries, 2010; Gaetz et al., 2010; Wilson et al., 2010; Heinrichs-Graham et al., 2014). Such beta ERD responses have been widely connected to motor planning and movement certainty (Heinrichs-Graham and Wilson, 2015; Heinrichs-Graham and Wilson, 2016; Alegre et al., 2003; Doyle et al., 2005; Tzagarakis et al., 2010). Stronger beta ERD generally means that a population of neurons are in a more desynchronized state, while an event-related synchronization indicates the population is in a more synchronized state. Critically, the resting motor cortex is strongly synchronized at the beta rhythm, (Rempe et al.) and it is broadly accepted that stronger beta ERD responses reflect greater activation of the sensorimotor cortices (Scheeringa et al., 2011; Formaggio et al., 2008; Wilson et al., 2016). This decrease in the beta rhythm temporally overlaps with a sharp increase in movement-related gamma synchronization (MRGS), which corresponds with movement onset and is often related to motor execution (Cheyne et al., 2008; Muthukumaraswamy, 2010; Cheyne and Ferrari, 2013). Growing evidence suggests that the strength of the MRGS is influenced by cognitive factors such as distracting visual stimuli and selective attention (Gaetz et al., 2013; Grent-'t-Jong et al., 2013; Spooner et al., 2020; Wiesman and Wilson, 2020; Busboom et al., 2024; Heinrichs-Graham et al., 2018). Several investigations involving persons with CP have shown that this patient population exhibits stronger sensorimotor beta ERD responses (*i.e.,* stronger reductions in beta power relative to baseline) throughout the planning and execution stages of a knee extension motor task. In contrast, the MRGS response has been shown to be reduced in this population during motor execution. Further, these aberrations in sensorimotor cortical oscillations have been linked with slower reaction times, motor production errors, and altered mobility in persons with CP (Kurz et al., 2014; Kurz et al., 2017; Kurz et al., 2020).

Given the association between aberrant sensorimotor cortical responses and impaired motor actions and mobility, it is important to determine whether therapeutic approaches yield beneficial changes in both clinical outcomes and associated cortical oscillations. However, a limited number of studies have probed whether improvements in motor function following physical therapy interventions are associated with beneficial changes in sensorimotor cortical oscillations. Our previous work examined this relationship by investigating the connection between cortical activity and muscular power using an eight-week therapeutic power training protocol in those with CP. The results demonstrated that pre-therapy sensorimotor oscillations were weaker in persons with CP compared to neurotypical (NT) controls, while after therapy, these cortical responses no longer differed from those observed in NT controls. Further, these changes in sensorimotor cortical oscillations were associated with improvements in muscular power (Busboom et al., 2022). We have also shown that after practicing a motor task, youth with CP exhibit beneficial changes in the beta and gamma sensorimotor oscillations that are linked with improved precision of the muscular performance (Kurz et al., 2024). These findings contribute to our understanding of the neurophysiological and clinical effects of intensively practicing a motor task and current physical therapy interventions. However, the field's understanding remains limited and further research is critical to develop targeted rehabilitation strategies that address the neurophysiological deficits seen in persons with CP, with the ultimate goal of optimizing functional mobility outcomes.

One targeted therapeutic approach with the potential to reduce deficits in neurophysiology and improve functional mobility in persons with CP is overground gait training that emphasizes motor planning and execution. One example of this approach is an obstacle course with varying surfaces and heights, which promotes intrinsic motor planning and problem solving. This activity requires the participant to plan out how they are going to navigate through the course, execute the planned motor actions, and reflect and problem solve if any errors occur. Additionally, physical therapist feedback can be used to encourage the participant to self-recognize their motor plans, execution strategies, and the occurrence of any errors. For example, the physical therapist may highlight an error, such as a knocked-over obstacle, and ask the participant if they noticed when the error occurred and inquire what motor action the individual could adjust to avoid it in the next attempt. This approach contrasts with standard gait training, which tends to focus solely on mass stepping practice. Furthermore, many "green light" therapies recommended for improving motor performance for persons with CP, particularly gait speed, involve treadmill-based interventions that prioritize mass stepping practice and remove practice in a dynamic environment (Novak et al., 2020). This overemphasis on mass stepping practice targets motor execution but neglects motor planning. As stated previously, persons with CP exhibit aberrant cortical activity in motor planning, which likely has cascading effects on motor execution, underscoring the importance of incorporating both motor planning and execution strategies into gait training paradigms.

Given these considerations, the purpose of the current study was to assess changes in the sensorimotor cortical oscillations and clinical outcomes of persons with CP after undergoing eight weeks of gait training physical therapy that emphasized motor planning, execution, and problem solving. To address our hypotheses, we utilized a MEG task that required either proactive or reactive sensorimotor control. Proactive control is when adjustments in the motor command are foreseen ahead of time, while reactive control is when the motor command must be adjusted in the moment (Spooner and Wilson, 2022). This task was chosen because its motor control principles were aligned with the key ingredients of our physical therapy and our prior experiments have shown that alterations in the strength of motor-related cortical oscillations during this task are associated with the uncharacteristic motor control seen in persons with CP (Hinton et al., 2024). Based on prior MEG literature with lower extremity tasks in persons with CP,(Busboom et al., 2022; Kurz et al., 2024) we hypothesized that prior to therapy, persons with CP would exhibit stronger beta ERD and weaker MRGS responses compared to NT controls. In addition, we hypothesized that after the gait training therapy protocol, persons with CP would demonstrate improvements in mobility, exhibit weaker beta ERD and stronger MRGS responses, and that these changes in sensorimotor cortical oscillations would be coupled to changes in clinical outcomes (*i.e.*, functional mobility). Testing these hypotheses will provide key insight into how neuroscience-informed therapeutic paradigms may drive beneficial neuroplastic changes and clinical improvements in persons with CP.

2. Methods

2.1. Ethical approval

The institutional review board at Boys Town National Research Hospital reviewed and approved this investigation, and it was registered on Clinicaltrials.gov (NCT04360395). Informed consent was acquired from adult participants and parents of the adolescent participants. All adolescent participants provided verbal assent to participate in the investigation.

2.2. Participants

Thirty-four persons with CP with diplegic and hemiplegic

presentation (age = 21.56 ± 7.28 yrs.; range = 11 – 39 yrs., females = 16, Table S.1) who were classified as Gross Motor Function Classification Scale (GMFCS) levels I-III participated in this study. Persons with CP and a GMFCS I walk without difficulty but may experience challenges with speed, balance, and coordination. Those classified as GMFCS II have difficulty walking long distances and navigating uneven terrain, while those with GMFCS III experience more significant mobility impairments and commonly use a walker or forearm crutches (Palisano et al., 2008). Additionally, 32 NT controls (age = 23.06 ± 3.79 yrs.; range = 12 – 30 yrs., females = 15) participated in the MEG imaging portion of this investigation to provide data on normative oscillatory response patterns. The two groups did not significantly differ by age (p = 0.294). Exclusionary criteria included orthopedic surgery or botulinum injections within the last six months, major neurologic disease, diagnosed severe psychiatric illness, and presence of any ferrous metal implants that would interfere with MEG acquisition and/or be an MRI safety concern. None of the participants reported that they were receiving concurrent physical therapy outside of the study protocol.

2.3. Physical therapy paradigm

The gait training paradigm consisted of overground walking tasks, such as traversing obstacles with different heights and terrains, navigating stairs, and other gait-related tasks specific to individualized goals, such as a focus on endurance for community ambulation. The physical therapy was focused on three key ingredients: 1) exploratory activities that enhance the somatosensory experience through rich/novel movement, 2) activities of adequate intensity that promote gait adaptation and gait speed sustainment, 3) optimally challenging activities that emphasize planning and problem solving that requires altering the leg kinematics to meet the environmental and task constraints. The gait training was conducted by licensed physical therapists and participants completed 24 gait training sessions (1-hour session, 3 times a week for 8 weeks).

During the treatment sessions, the physical therapist provided targeted verbal feedback to encourage the participants' internal awareness of motor planning and execution to facilitate the development of new strategies for gait function. For example, if the participant was having difficulty navigating the steps in front of their home, the therapist might ask the patient to identify barriers and then discuss potential strategies. The participant would then practice these strategies during the therapy session, while refining the execution of the motor action to achieve independent stair negotiation at home. Physical assistance and passive movement of the participant's lower extremities was minimized. When support was necessary to reduce injury risk or maintain the desired activity intensity, it was provided using the ZeroG System (Aretech, Ashburn, VA) or a gait belt. However, the treatment goal prioritized minimizing the number of sessions requiring such support. Intensity for the activities was set at the participant's just right challenge point. This optimized challenge point for each participant was determined based on engagement, effort, and the participant's ability to successfully complete the activity. The participants' interest in improving in the current activity measured engagement, while effort was assessed by their persistence in successfully completing the activity. The environment in which the activities were performed were scaled to achieve the optimal challenge point.

2.4. Clinical outcomes

Participants underwent a series of mobility assessments at both pre- and post-therapy time points. These assessments were administered by licensed physical therapists and scored by a separate, single licensed physical therapist who was not involved in the gait training protocol. Testing occurred approximately one week before the start of the gait training protocol (pre) and again within one week following the completion of therapy (post). The outcome measures included: Functional Gait Assessment (FGA), dynamic range of gait speed, and Timed Up and Go (TUG). All assessments were completed with assistive devices or orthotics that the participant typically used during community ambulation. The FGA is a standardized assessment of balance and walking adaptability that is conducted over a 6-m walkway (Wrisley et al., 2004). Exemplary tasks include, walking while looking right and left, stepping over an obstacle, changing gait speeds, and tandem walking, *etc*. For scoring, two cameras (GoPro Inc., San Mateo, CA) were used to record the participants' frontal and sagittal plane performance on the respective tests. The total score across the individual FGA tests was used as the primary outcome variable. Dynamic range of gait speed demonstrates the participant's capacity to change their gait speeds and was calculated by subtracting the fast gait speed from the pre-therapy preferred gait speed. This approach normalizes the fast-as-possible gait speed to the same baseline at the respective time points. We collected six preferred and fast-as-possible gait speeds on the 4.89-m Zeno™ Walkway, and the outcome variable was the fastest speed for the respective gait assessments (ProtoKinetics, Havertown, PA) (Parati et al., 2022). Another standard clinical test, the TUG, was administered to evaluate balance, mobility and fall risk (Hassani et al., 2014). Participants initially sat on a bench, and were instructed to stand-up and walk around a cone that was positioned 3-m away and sit back down. Participants completed the test two times, and the fastest time was the outcome variable used for analysis.

2.5. MEG acquisition and experimental paradigm

MEG recordings were conducted in a two-layer magnetically shielded room using a whole head MEGIN Neo System (MEGIN, Helsinki, Finland). The recordings were collected at one time point for NT controls and pre- and post-therapy for persons with CP. The accelerometer signals and neuromagnetic responses were continuously sampled at a rate of 1 kHz using an acquisition bandwidth of 0.1–330 Hz. A fiber optic triaxial accelerometer (Optoacoustics Ltd., Israel) was secured just proximal to the participants' right lateral malleolus to quantify movement onsets (see MEG pre-processing).

During the MEG experiment, participants remained seated in a custom non-magnetic chair with their head positioned within the MEG helmet, where the sensor array was located. The experiment was a two-condition clock-based knee extension cueing task (Spooner and Wilson, 2022; Taulu and Simola, 2006). The proactive–reactive design allowed for analyses of dynamic neural mechanisms of an adaptive motor control task. Hence, participants responded with the same leg motor action for both the proactive and reactive conditions, which allowed for more straightforward interpretations of the mechanisms of adaptive cueing. To complete the task, participants maintained fixation on a centrally located crosshair while a red dot traveled in a clockwise direction around a 0.21 m circle every five seconds at a rate of 0.13 m/s. Of note, the clock-like design of the task included no numbers nor tick marks within its presentation, but instead, was meant to serve as a pacing mechanism. In both proactive and reactive conditions, a blue target interval was displayed. The participants were instructed to extend their right knee joint by ~ 15° to touch a ridged bar that was fixed to the chair and positioned at mid-shank, and to do this as quickly and accurately as possible once the red dot entered the blue target interval. During the proactive condition, the blue target interval was fixed around the 12o'clock position. While during the reactive conditions, approximately 150 ms before the red dot entered the blue target interval, the target would narrow and shift within the original target interval to one of four fixed locations (Fig. 1). Blue target interval locations for the reactive condition were presented in a pseudorandom order and every participant completed 100 proactive and 100 reactive trials. Moreover, the presentation of either a proactive or reactive condition was randomized. Prior to the start of the experiment, participants practiced exemplary proactive and reactive conditions to ensure they understood the experiment.

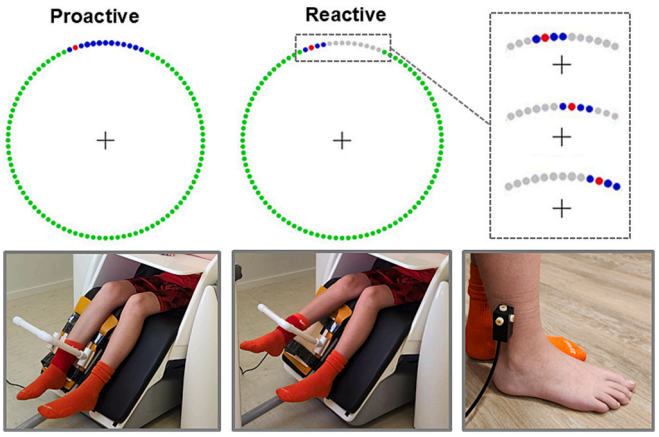

Fig. 1. Experimental Paradigm. (Top) Graphical representation of the proactive and reactive conditions. The green circle was always shown on the screen as the participant views the red dot traversing the circle in a clockwise fashion. The proactive condition is shown on the left and the reactive appears on the right. The reactive condition had four possible circumstances; essentially, 150 ms before the red dot entered the blue interval, the target window would shrink and shift within the original interval to one of four fixed locations. **(Bottom)** Participants extended their right knee as soon as the red dot entered the blue target interval of the circle. The accelerometer was placed just proximal to the participants' right malleolus and was used to quantify movement onsets. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

2.6. MEG coregistration and structural magnetic resonance imaging and processing

Prior to the MEG experiment, five coils were affixed to the head of the participant and used for continuous head localization during the MEG scan. The location of these coils, three fiducial points, and the scalp surface were digitized to determine their three-dimensional position relative to each other (Fastrak; Polhemus Navigator Sciences, Colchester, VT). Once the participant was seated in the MEG chair, electric currents with unique frequency labels (*e.g.,* 322–327 Hz) were fed to each of the five coils. The electrical currents provided a measurable magnetic field, which allowed each coil to be localized in reference to the MEG sensors throughout the recording session. The coil locations were also known in head coordinates, therefore all MEG measurements could be transformed into a common coordinate system. The coordinate system and the scalp surface points allowed for each participant's MEG data to be coregistered with their native space structural T1-weighted magnetic resonance images (MRI) prior to source reconstruction. Structural MRI scans were collected on a 3.0 T Siemens Prisma magnet (Siemens Medical Solutions USA, Inc., Malvern, PA) equipped with a 64-channel head coil and acquired with the following parameters: 1.0x1.0x1.0 mm resolution, TR = 2400 ms, TE = 3.5 ms, flip angle = 8 degrees, matrix = 256 × 256 and FOV = 256 × 256 mm. The structural MRI data were aligned parallel to the anterior and posterior commissures and transformed into standardized Talairach space. Coregistration and the transformation was performed with the Brain Electrical Source Analysis (BESA) MRI software (Version 3.0; BESA gmbH, Gräfelfing, Germany).

2.7. MEG pre-processing

Each participant's MEG data set was individually corrected for head motion that may have occurred during the experiment and was subjected to noise reduction using the signal space separation method with a temporal extension (Uusitalo and Ilmoniemi, 1997). Additionally, 0.5–200 Hz bandpass filter and a 60 Hz notch filter was applied to the data. Subsequently, cardiac and blink artifacts were removed from the data using signal-space projection and was accounted for during source reconstruction (Gross et al., 2013). The continuous magnetic time series were divided into epochs of 4000 ms duration, with 0.0 s defined as movement onset, and baseline defined as −2000 to −1500 ms before the movement onset. Movement onset was defined using the triaxial accelerometer secured just above the participant's right lateral malleolus (see Fig. 1). Briefly, the onset of movement was determined by a sharp increase in the amplitude of the resultant accelerometer signal. The sharp increase was marked as movement onset when the amplitude was 10 % above the baseline interval (*i.e.*, 300 ms prior to movement onset).

A fixed threshold method, supplemented with visual inspection was used for artifact rejection. We subsequently determined whether the number of trials remaining varied by group (three levels: CPpre, CPpost, NT controls), task condition (two levels: proactive, reactive) or their interaction using a linear mixed-effect (LME) model. We did detect a main effect of group ($F(2,95) = 11.82$, $p < 0.001$), such that NT controls ($M_{NT} = 91.45$ trials, $SE_{NT} = 2.00$ trials, 95 % CI [87.46, 95.44]) tended to have more trials than CPpre ($MC_{Ppre} = 79.15$ trials, $SE_{CPpre} = 1.94$ trials, 95 % CI [75.28, 83.02]) and CPpost ($M_{CPpost} = 82.66$ trials, $SE_{CPpost} = 1.94$ trials, 95 % CI [78.79, 86.53]), and CPpost tended to have more trials than CPpre. There was no significant main effect of task condition ($F(1,139) = 1.75$, $p = 0.189$) or interaction of group and task condition ($F(2,139) = 0.72$, $p = 0.485$). To account for the potential confounding effects of signal-to-noise ratio differences due to the differing number of accepted trials, we included the square root of the number of trials per participant as a covariate in all subsequent analyses (Junghöfer et al., 2000; Vincent, 1992; Kovach and Gander, 2016).

2.8. MEG sensor level analysis

For each sensor, the artifact-free epochs were transformed into the time–frequency domain per trial using complex demodulation (resolution: 2 Hz, 25 ms) (Maris and Oostenveld, 2007). Sensor-level data were then averaged across trials per condition and normalized per time–frequency bin using the mean power for the specific frequency during the baseline period (−2000 to −1500 ms). The specific time–frequency windows used for imaging were determined by statistical analysis of the sensor-level spectrograms across both conditions and the entire array of gradiometers. Initially, each data point in the spectrogram was 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 I error. The first stage consisted of paired-sample *t*-tests that were conducted to test for differences from baseline at each data point and the output spectrogram of *t*-values was thresholded at $p < 0.05$ to define time bins containing potentially significant oscillatory events across all participants (CP and NT), task conditions (proactive and reaction), and pre- and post-therapy sessions for persons with CP. In the second stage, time–frequency bins that survived the threshold were clustered with temporally, spectrally, and/or spatially neighboring bins that were also above the threshold ($p < 0.05$), and a cluster value was derived by summing all of the *t*-values of all the 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 the first stage) were tested directly using this distribution (Gross et al., 2001). For each comparison, 1,000 permutations were computed to build a distribution of cluster values. All imaging procedures were completed using the BESA software (BESA v7.1).

2.9. MEG source imaging

A minimum variance vector beamforming algorithm was used to calculate the source power across the whole brain volume for each neural oscillatory response based on the specific time–frequency window identified statistically at the MEG sensor level (Hillebrand et al., 2005; Hillebrand and Barnes, 2005). Briefly, the single images were

derived from the cross spectral densities of all combinations of MEG gradiometers, using only the MEG data from these time–frequency windows of interest and a baseline period of equal duration and bandwidth. This separately averaged baseline noise period was used to normalize the source power in these images per participant, which is consistent with conventional imaging pipelines (Hillebrand and Barnes, 2005; Van Veen et al., 1997; Menyhart et al., 2021). These images are typically referred to as pseudo-*t* maps with units (pseudo-*t*) that reflect noise-normalized power differences (*i.e.*, active period vs. baseline period) per 4.0 mm^3 voxel. Each participant's functional images, which were co-registered to their structural T1-weighted MRI prior to beamforming, were transformed into standardized Talairach space. This was completed by using the transform previously applied to the structural MRI volume and subsequently spatially resampled to 4.0 mm^3 resolution. The resulting images of cortical activity were averaged across all participants, conditions, and time points to provide grand averaged beamformer images per oscillatory response. This averaging enabled a data quality check and illuminated the anatomical bases of the significant oscillatory responses identified through the sensor-level analysis. We also used these grand averages to identify the peak voxel for each motor-related oscillatory response, which were the focus of our primary MEG outcomes. We chose to use this grand-averaged peak voxel approach over selecting individualized peaks for several reasons. First, in clinical populations (*e.g.*, persons with CP), anatomical and functional variability is high, especially in sensorimotor regions, and many individuals with CP exhibit more than one peak in these regions. This makes it difficult to discern which peak should be selected for the final analysis and inserts an element of subjectivity. To circumvent this difficulty and maximize rigor and reproducibility, we focused on the peak voxel derived from a grand average of each oscillatory response. This ensures that the cube of tissue is common across groups and pre/post therapy time points and that the same voxel would be used by anyone analyzing the data, thus leading to improved rigor and transparency of experimental outcomes.

Next, we extracted virtual sensor neural time series (*i.e.*, voxel time series) per participant using the peak voxel in the grand averaged beamformer image per oscillatory response. We chose to evaluate the virtual sensor neural time courses, as opposed to performing whole brain statistics, because the whole-brain maps reflect static functional activity (*i.e.*, collapsed across time) and we were interested in assessing changes in the dynamics of sensorimotor cortical oscillations. In addition, our hypotheses were focused on sensorimotor areas and thus taking a whole-brain approach would have involved thousands of statistical tests (*i.e.*, each voxel is tested independently) that were unnecessary to address our hypotheses. The neural time course was computed by applying the sensor weighting matrix derived through the forward computation to the preprocessed signal vector, which yielded a time course for the specific coordinate in source space per participant. Using the neural time course, the envelope of spectral power in the frequency bin used in the beamforming analysis was computed. The sensorimotor activity was averaged across the time window of interest for beta (motor planning and execution periods) and gamma frequencies to assess the magnitude of oscillatory responses.

2.10. Motor behavioral data

To evaluate motor performance during the MEG task, reaction time (*i.e.*, response distance from target interval onset) and the coefficient of variation of reaction time were analyzed. Specifically, both behavioral metrics were normalized by the target interval to account for differences in the interval size between the proactive and reactive conditions (*i.e.,* ratio score: response time distance from target onset / length of total target interval). Therefore, all behaviors were expressed as percent distance and/or variability from the onset of the blue target window.

2.11. Statistical analysis

LME models were used to examine the effects of group (three levels: CPpre, CPpost, NT controls), task condition (two levels: proactive, reactive) and their interaction on brain and clinical outcomes. Signal-to-noise ratio (SNR), computed as the square-root of the total number of trials per person, was included as a covariate to account for differences in accepted trials between group. Subject was included as a random effect with random intercept/slope. The model was fit using restricted maximum likelihood estimation for comparison of fixed effects. Type III sums of square were used for significance testing, with degrees of freedom calculated using the Satterthwaite method. Post-hoc pairwise comparisons were conducted using estimated marginal means (EMMs) with Tukey's adjustment for multiple comparisons. LME analyses were performed using the *lme4* and *emmeans* packages in R (version 4.4.2). Additionally, separate non-parametric Welch *t*-tests were used to probe for differences in the clinical assessments between the pre- and post-therapy time points in the CP group. Lastly, Spearman rho's correlations were used to assess relationships among the change scores in clinical outcomes and those in the strength of cortical oscillatory responses in persons with CP. The change score was calculated as the difference between post-therapy and pre-therapy values. Correlation and *t*-test analyses were performed in JASP (version 0.19.2) with a 0.05 alpha level. A Bonferroni-Hochberg adjustment was applied to adjust the alpha level to 0.025 to account for the multiple correlations (Oeffinger et al., 2008).

3. Results

3.1. Clinical outcome results

All participants completed the pre- and post-therapy clinical outcomes and the eight weeks of therapy. Participants completed an average of 22 out of 24 (94 %) physical therapy sessions. Participants with CP exhibited 8.77 % improvement in the FGA (M_{CPpre} = 19.44, SE_{CPpre} = 1.02, M_{CPpost} = 21.15, SE_{CPpost} = 1.06; $p < 0.001$), demonstrated a 38.94 % improvement in the dynamic range (M_{CPpre} = 0.43 m/s, SE_{CPpre} = 0.04 m/s, M_{CPpost} = 0.59 m/s, SE_{CPpost} = 0.05 m/s; $p < 0.001$), and completed the TUG 15.97 % faster (M_{CPpre} = 10.56 s, SE_{CPpre} = 1.27 s, M_{CPpost} = 8.88 s, SE_{CPpost} = 0.99 s; $p < 0.001$; Fig. 2). Overall, these results imply that the group of participants had notable improvements in their mobility after completing the physical therapy protocol.

The minimum clinically important difference (MCID) for gait speed is 0.1 m/s[56] and the TUG ranges from 0.7 to 1.2 unit change (Hassani et al., 2014). The MCID denotes a significant change in a clinical measure that is relevant to the participant and clearly observable by the eye. Our results exceeded these values, supporting the notion that our sample as a whole exhibited clinically meaningful improvements in these outcomes following therapy.

3.2. Motor behavioral results

A LME was conducted to predict reaction time, with group (three levels: CPpre, CPpost, NT controls), task condition (two levels: proactive, reactive), and their interaction as fixed effects. Subject was included as a random effect to account for within-subject variability. The analysis revealed a significant main effect of group ($F(2,91) = 4.42$, $p = 0.015$) and task condition ($F(1,135) = 907.76$, $p < 0.001$). The interaction between group and task condition was not significant ($F(2,135) = 2.17$, $p = 0.118$). Post-hoc comparisons using Tukey's adjustment revealed significant differences between the NT controls and CPpre group ($t(81) = -2.12$, $p = 0.013$) and between NT controls and CPpost group ($t(81) = -2.64$, $p = 0.027$). However, there was no significant difference between CPpre and CPpost groups ($t(138) = 0.53$, $p = 0.858$). The estimated marginal means (EMM) for the groups

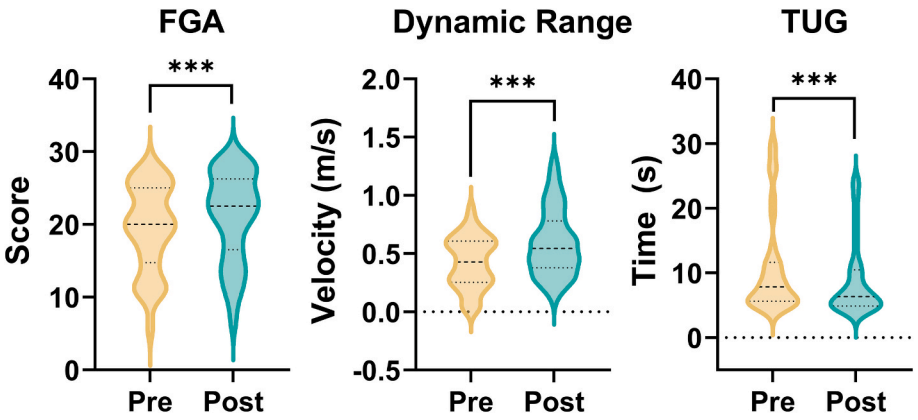

Fig. 2. Clinical Outcomes. Pre- and post-therapy outcomes in **(A)** the Functional Gait Assessment (FGA), **(B)** dynamic range of walking speed, and **(C)** Timed Up and Go (TUG).***$p < 0.001$.

indicated that NT controls responded significantly faster (EMM_{NT} = 104.65 %, SE_{NT} = 8.99 %, 95 % CI [86.76, 122.53]) than CPpre (EMM_{CPpre} = 141.11 %, SE_{CPpre} = 8.69 %, 95 % CI [123.82, 158.40]) and CPpost (EMM_{CPpost} = 137.62 %, SE_{CPpost} = 8.69 %, 95 % CI [120.33, 154.91]). Additionally, the estimated marginal means for task (t(138) = -29.67, p < 0.001) indicated that participants tended to complete the proactive condition faster ($EMM_{proactive}$ = 46.31 %, $SE_{proactive}$ = 6.73 %, 95 % CI [32.94, 59.69]) than the reactive ($EMM_{reactive}$ = 209.27 %, $SE_{reactive}$ = 6.71 %, 95 % CI [195.93, 222.60]; Fig. 3).

The same LME model was conducted for the coefficient of variation. This analysis revealed a significant main effect of task condition (F(1,143) = 163.93, $p < 0.001$), but not a main effect of group (F(2,107) = 1.63, p = 0.201) nor a group by task condition interaction (F(2,143) = 1.90, p = 0.153). Post-hoc comparisons for task condition (t(139) = 12.61, $p < 0.001$) revealed that participants tended to have a greater coefficient of variation in the proactive ($EMM_{proactive}$ = 1868.35 %, $SE_{proactive}$ = 92.86 %, 95 % CI [1684.70, 2052.01]) compared to the reactive condition ($EMM_{reactive}$ = 439.44 %, $SE_{reactive}$ = 93.31 %, 95 % CI [254.91, 623.98]; Fig. 3).

3.3. Sensor-level and source-level results

Spectrogram permutation tests revealed that there was significant beta ERD (18–28 Hz; *i.e.,* power decrease from baseline) and MRGS (64–78 Hz; *i.e.*, power increase relative to baseline) responses across sensors near the sensorimotor cortex ($p < 0.0001$, corrected). These sensor-level statistical analyses indicated that the beta ERD began 250 ms prior to movement onset (0 ms) and was sustained until 250 ms after the leg movement (*i.e.*, −250 to 250 ms), while the MRGS was observed shortly after the movement onset (*i.e.*, 50 to 350 ms; Fig. 4).

3.3.1. Beta cortical oscillations

The beta ERD response was imaged from −250 to 250 ms in each participant using a baseline of equal bandwidth and duration (18–28 Hz; −2000 to −1500 ms) to identify the underlying cortical regions. As expected, the resulting images indicated that the beta ERD occurred in the leg region of the contralateral sensorimotor cortices (Fig. 4). The peak voxel in the grand-averaged image was then used as a seed for extracting virtual sensor time series in each participant per group and visit (CPpre, CPpost, NT controls; Fig. 5). Subsequently, from the time series, we calculated the average activity seen during the motor planning (−250 to 0 ms) and execution (0 to 250 ms) time windows separately per participant. This approach allowed for us to evaluate the cortical activity in the same spatial location across participants and conditions.

Regarding the motor planning time window, a LME was conducted to evaluate beta relative power as a function of group (three levels: CPpre, CPpost, NT), task condition (two levels: proactive, reactive), and their interaction as fixed effects, while controlling for SNR. Subject was included as a random effect to account for within-subject variability. The analysis revealed significant main effects of group (F(2,90) = 11.80, $p < 0.001$) and task condition (F(1,132) = 16.89, $p < 0.001$). The

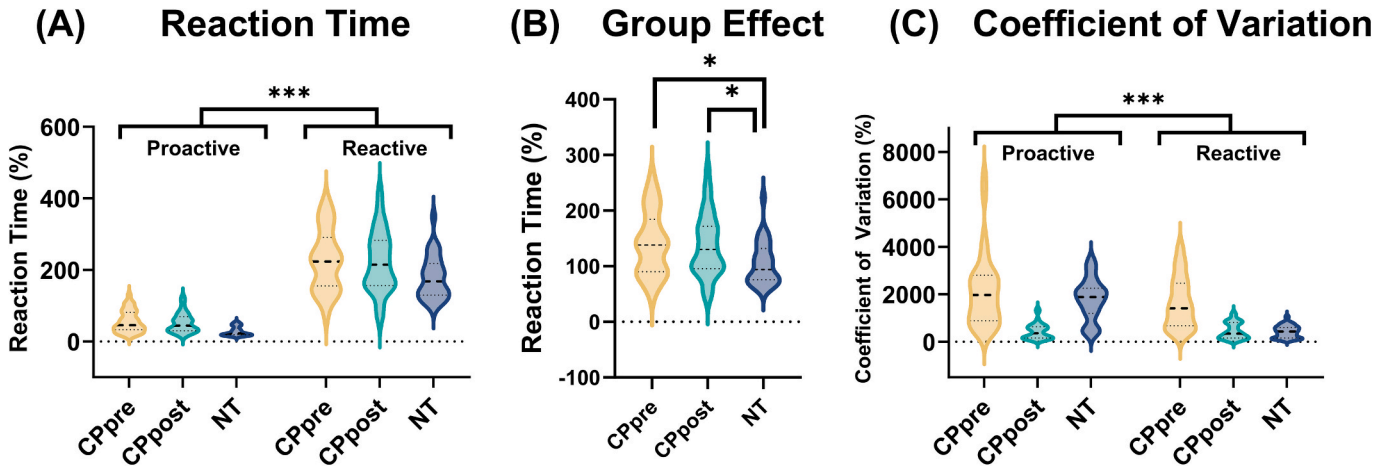

Fig. 3. MEG Behavioral Results. (A) The main effect of task indicated that all participants responded faster during the proactive compared to the reactive task condition. **(B)** The main effect of group showed that NT controls responded faster compared to CPpre and CPpost. **(C)** Movement onset was more variable during the proactive compared to the reactive condition. *$p < 0.05$, ***$p < 0.001$.

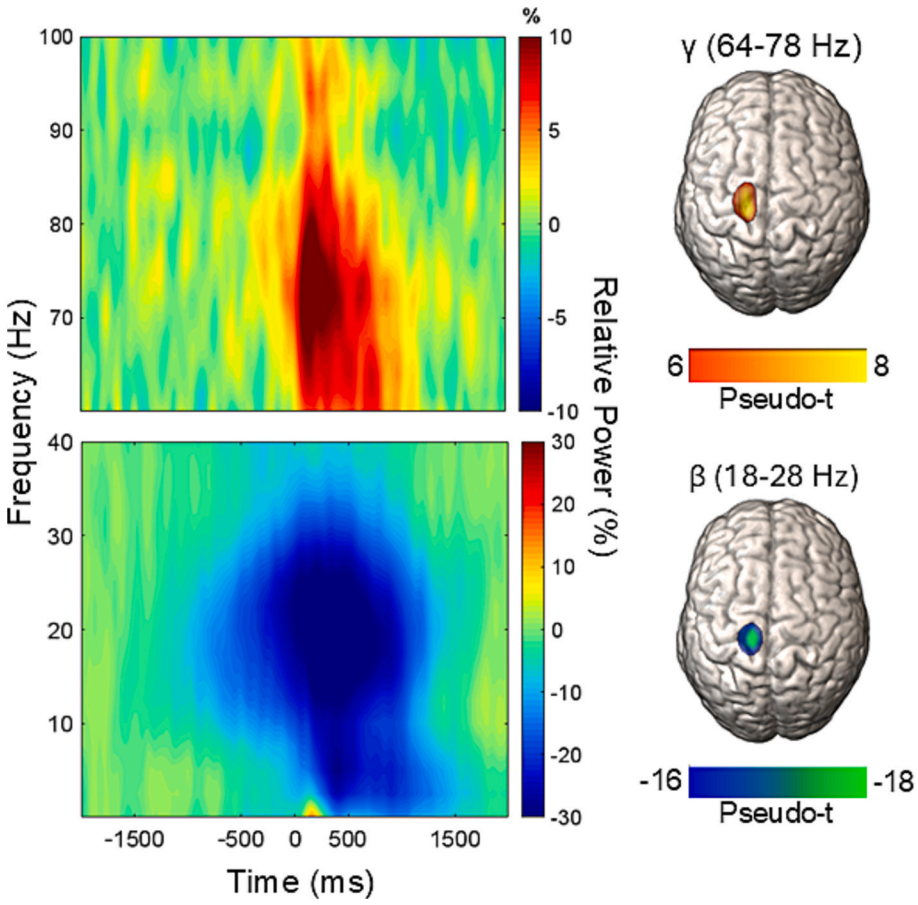

Fig. 4. Sensor and source-level neural responses. (Left) Time frequency spectrograms for a sensor near the contralateral sensorimotor cortex. The x-axis denotes time (ms) and y-axis denotes frequency (Hz). Movement onset occurred at 0 ms. The relative power of the spectrograms is expressed as a percent change from baseline (− 2000 to − 1500 ms time window). **(Right)** The significant oscillatory responses ($ps < 0.001$, corrected) in the respective spectrograms were imaged and grand averaged across both task conditions and groups. As shown in the beamformer images, the movement related gamma synchronization (MRGS) and *peri*-movement beta event-related desynchronization (ERD) were located in the motor leg region of the contralateral hemisphere.

interaction between group and task condition was not statistically significant ($F(2,132) = 0.54$, $p = 0.585$). Post-hoc comparisons using Tukey's adjustment revealed significant differences between the NT and CPpre groups ($t(77) = -3.91$, $p < 0.001$), and between NT and CPpost groups ($t(74) = -2.86$, $p = 0.015$). The CPpre and CPpost groups also differed significantly ($t(140) = 3.45$, $p = 0.002$). The estimated marginal means for the groups indicated that NT controls exhibited stronger beta ERD responses during the planning period ($EMM_{NT} = -41.45$ %, $SE_{NT} = 3.21$ %, 95 % CI [-47.84, −35.06]), while CPpost ($EMM_{CPpost} = -28.80$ %, $SE_{CPpost} = 3.01$ %, 95 % CI [-34.80, –22.80]) demonstrated beta power closer to NT control levels than the CPpre visit, but still different from the control group. Importantly, beta ERD responses were weaker in the CPpre data ($EMM_{CPpre} = -23.96$ %, $SE_{CPpre} = 3.04$ %, 95 % CI [-30.02, −17.91] compared to the CPpost results. A significant main effect of task condition was also observed ($t(138) = 4.03$, $p < 0.001$), with beta ERD responses being stronger during the reactive condition ($EMM_{reactive} = -33.70$ %, $SE_{reactive} = 2.29$ %, 95 % CI [-38.27, −29.14] compared to the proactive condition ($EMM_{proactive} = -29.10$ %, $SE_{proactive} = 2.29$ %, 95 % CI [–33.67, −24.54]; Fig. 5).

Regarding beta during the execution time window, the same LME was conducted and revealed a significant main effect of group ($F(2,90) = 14.80$, $p < 0.001$). However, there was no significant main effect of task condition ($F(1,132) = 0.47$, $p = 0.495$) or interaction ($F(2,132) = 1.70$, $p = 0.186$). Post-hoc comparisons revealed significant differences between the NT and CPpre data ($t(75) = -3.77$, $p < 0.001$) and between the NT and CPpost data ($t(72) = -2.60$, $p = 0.030$). The CPpre and CPpost data also differed significantly ($t(140) = 4.38$, $p < 0.001$). The estimated marginal means for the groups indicated that NT controls had stronger (*i.e.*, larger power reduction) beta ERD responses during the execution period ($EMM_{NT} = -43.65$ %, $SE_{NT} = 3.27$ %, 95 % CI [-50.17, −37.14]), while the CPpost data ($EMM_{CPpost} = -31.93$ %, $SE_{CPpost} = 3.08$ %, 95 % CI [-38.08, −25.79]) was weaker than the NT controls but significantly stronger than the CPpre data ($EMM_{CPpre} = -26.51$ %, $SE_{CPpre} = 3.10$ %, 95 % CI [–32.70, −20.33]; Fig. 5).

3.4. Gamma cortical oscillations

We also imaged the MRGS seen across the 50 to 350 ms time window using a baseline of equal bandwidth and duration (64–78 Hz; −1900 to −1600 ms). As expected, the resulting images indicated that the MRGS was also centered on the leg region of the contralateral sensorimotor cortices (Fig. 4). The peak voxel of the MRGS in the grand-averaged image was subsequently used as a seed for extracting virtual sensors in each participant, task condition and visit (CPpre, CPpost, NT controls; proactive, reactive Fig. 6). Specifically, we calculated the average activity seen during the time window of interest (50 to 350 ms) separately per participant, task condition and visit (for those with CP).

We used a LME similar to those described above to examine the MRGS, which revealed a significant main effect of group ($F(2,95) = 5.79$, $p = 0.004$). However, there was no significant main effect of task condition ($F(1,132) = 2.61$, $p = 0.109$) or interaction ($F(2,131) = 0.15$, $p = 0.864$). Post-hoc comparisons revealed significant differences between the CPpre and CPpost groups ($t(141) = 3.34$, $p = 0.003$). However, there was no significant difference between the NT and CPpre

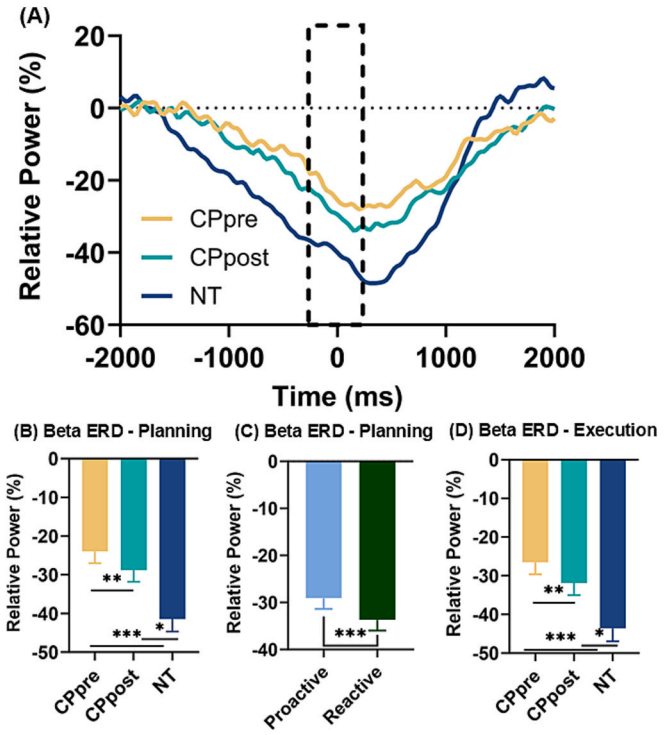

Fig. 5. Beta Oscillatory Activity. (A) Neural time courses of the beta oscillatory response extracted from the peak voxel in the sensorimotor cortices and group averaged (per visit for the CP group). The percentage change in power relative to baseline is shown on the y-axis and time is shown on the x-axis. The onset of movement is 0 ms and the black dashed box represents the time window that was imaged. As shown, the beta ERD was weaker pre-therapy and became stronger post-therapy, aligning to the pattern of beta activity observed in NT controls. **(B)** Statistical analysis revealed a significant main effect of group, with NT controls showing the strongest beta ERD during the planning period (−250 to 0 ms), CPpost showing activity approximating NT controls, and CPpre demonstrating the weakest and most abnormal beta ERD. **(C)** A main effect of task revealed that the reactive condition had significantly stronger beta ERD during the planning period compared to the proactive condition. **(D)** During the execution time window (0 to 250 ms) NT showed the strongest beta ERD, with CPpost approximating those levels, and CPpre demonstrating the weakest beta ERD. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

groups ($t(88) = -0.96$, $p = 0.602$) or between NT and CPpost groups ($t(86) = 0.86$, $p = 0.669$). The estimated marginal means for the groups indicated weaker MRGS responses in the CPpost data (EMM_{CPpost} = 12.79 %, SE_{CPpost} = 2.83 %, 95 % CI [7.16, 18.43]) compared to CPpre data (EMM_{CPpre} = 20.59 %, SE_{CPpre} = 2.86 %, 95 % CI [14.90, 26.28]), while the NT controls (EMM_{NT} = 16.42 %, SE_{NT} = 3.10 %, 95 % CI [10.25, 22.58]) did not differ from either CPpre or CPpost (Fig. 6).

3.5. Cortical oscillations and clinical correlations

For persons with CP, there was a significant positive correlation between the pre-/post-therapy change score of the TUG and the change score of the beta ERD response during the planning period (beta power was averaged between the proactive and reactive conditions for this analysis; rho = 0.390, $p = 0.025$; Fig. 7). This relationship implies that persons with CP who had a stronger motor planning beta ERD after undergoing gait training also tended to have greater improvements in the TUG. Correlations between other oscillatory activity and clinical outcomes were not significant ($ps > 0.05$). The respective correlations are shown in Table 1.

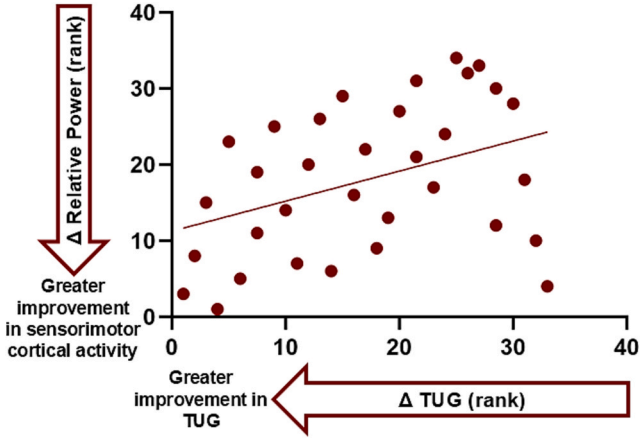

Fig. 7. Clinical and Neural Correlation. Spearman rank order correlation between the change score of the beta ERD during the planning period and the change score of the TUG. This correlation implies that participants with CP who demonstrated a stronger beta ERD from pre- to post-therapy also tended to show greater improvements in the TUG.

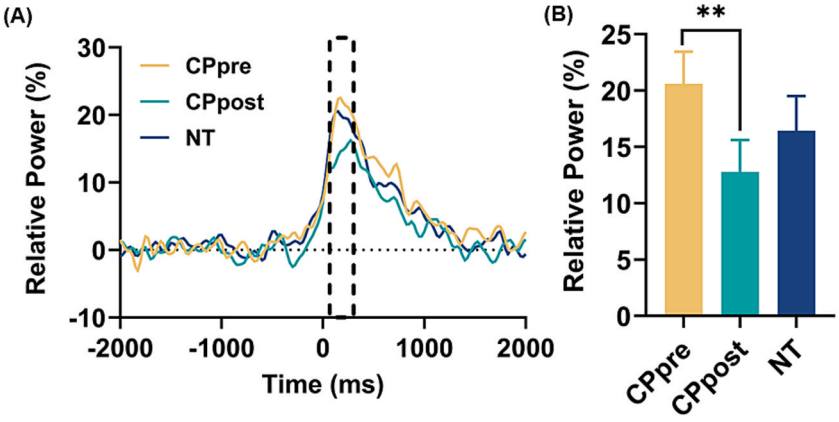

Fig. 6. MRGS Response. (A) Neural time courses of the MRGS were extracted from the peak voxel of the sensorimotor cortices and group averaged (per visit for the CP group). The percentage change in power relative to the baseline is shown on the y-axis and time is shown on the x-axis. The onset of movement is 0 ms and the black dashed box represents the time window that was imaged. As shown, the MRGS was stronger pre-therapy and became weaker after therapy in the CP group. **(B)** Statistical analyses revealed a significant main effect of group, with CPpre showing stronger MRGS compared to CPpost and NT controls not differing between CPpre or CPpost. **$p < 0.01$.

Table 1
Spearman rho correlations between the change in the beta and gamma oscillations and the change in the respective clinical outcomes.

	FGA	TUG	Dynamic Range
Beta ERD			
Motor Planning	0.083	0.39*	−0.069
Motor Execution	−0.09	−0.001	−0.068
MRGS	−0.244	0.12	0.074

ERD = Event-Related Desynchronization, MRSG = Motor Related Gamma Synchronization, FGA = Functional Gait Assessment, TUG = Timed Up-and-Go. *$p = 0.025$.

4. Discussion

As persons with CP age, their mobility tends to further decline. While neurophysiological markers of these mobility impairments have been described, physical therapy interventions aimed at these aberrant neurophysiological factors are exceedingly rare. Thus, in the current study, we used a novel gait training paradigm and examined its impact on sensorimotor cortical oscillations during a lower extremity motor task and clinical outcomes. We also investigated the relationship between these measures. Our results revealed that persons with CP demonstrated improved mobility following the eight weeks of gait training. Sensorimotor cortical oscillations revealed that NT controls had the strongest beta ERD during movement planning and execution, and NT controls had no significant differences between persons with CP for the MRGS. Furthermore, the therapeutic effect showed that the beta ERD became stronger and the MRGS became weaker following therapy in persons with CP. Importantly, we also observed a significant correlation between the TUG and beta ERD during the planning period, which makes the key link between neural and behavioral changes following therapy. Further discussion of these findings and their clinical implications are provided below.

The behavioral data from the proactive–reactive MEG task revealed significant differences between persons with CP and NT controls, with NT controls responding faster across all task conditions. Collapsed across groups, all participants had slower reaction times during the reactive compared to the proactive condition. These task condition findings are consistent with a normative study and partially align with our previous work in the upper extremity, where persons with CP demonstrated slower responses during reactive conditions but no significant difference in the proactive conditions (Spooner and Wilson, 2022; Taulu and Simola, 2006). We also observed a significant effect in the variability (*i.e.,* coefficient of variation) of participant responses, with all participants exhibiting greater variability during the proactive compared to the reaction condition. This contrasts previous work in NT controls, where increased variability was observed in the reactive conditions (Spooner and Wilson, 2022). It is plausible that participants in this sample utilized the extended response window during the proactive condition, leading to more variable response patterns. Alternatively, the heightened response variability seen here might be related to differences in the precision of the motor commands generated for the hand versus the leg. Of note, it was somewhat surprising to find that there weren't changes in the MEG behavioral variables after undergoing physical therapy, despite the noted changes in the cortical oscillations. We contend that the changes observed in the sensorimotor cortical oscillations might reflect beneficial improvements in the cortico-muscular coherence after undergoing the gait training, while the lack of significant changes in the MEG behavioral variables might suggest that there were fewer changes in the neural networks involved in performing cognitive-motor decisions. Of course, the effect size for behavioral improvements was likely smaller than that for neural changes and this may have also contributed.

Task-condition differences in the beta ERD were observed during the motor planning period, with the reactive condition eliciting a stronger beta ERD. This contrasts with what was previously found using this task in the upper extremity in persons with CP and NT controls, where there were no conditional differences in the beta ERD (Spooner and Wilson, 2022; Taulu and Simola, 2006). In persons with CP, it was postulated in this past work that the certainty of the motor action (*i.e.*, button press) irrelevant of task condition remained the same, and therefore the beta ERD cortical activity was unaffected. However, this is the first investigation to use this task in the lower extremity of persons with CP. It is possible that the lower extremities were more affected in the current sample and there was not as much certainty of the knee extension motor action compared to the button press. Hence, making the lower extremity motor action more susceptible to conditional differences for the beta ERD cortical activity. However, it should also be noted that the current sample is larger than these previous studies and thus the emergence of beta ERD conditional findings here could reflect increased sensitivity (*i.e.*, statistical power).

The sensorimotor beta ERD in persons with CP during the motor planning and execution time periods was significantly weaker (*i.e.*, less of a power reduction) compared to the NT controls. These findings differ from our previous work in which persons with CP demonstrated a stronger sensorimotor beta ERD compared to NT controls during a lower extremity motor task (Busboom et al., 2022; Kurz et al., 2014; Kurz et al., 2017; Kurz et al., 2020; Kurz et al., 2024). A substantial factor in this discrepancy may lie in the use of the adaptive motor control paradigm used in this study, which involved more cognitive engagement compared to the simpler target-matching task employed previously. To this point, the directionality between NT controls and CP in the beta ERD aligns with findings from our upper extremity research using the same paradigm and other studies involving more complex MEG tasks (Hoffman et al., 2019; Taulu and Simola, 2006). Overall, the sensorimotor beta ERD findings, while distinct from those in earlier lower extremity studies, further strengthens the notion that persons with CP have difficulty planning their motor actions, which likely has cascading effects on their ability to properly execute a motor action.

Intriguingly, the post-therapy beta ERD response in participants with CP was stronger and approximated what was seen for the NT controls. This is in agreement with recent work from our laboratory that demonstrated a similar relationship in a single-day pre-post practice investigation, where a stronger beta ERD was observed after practicing a lower extremity target-matching task (Kurz et al., 2024). A stronger beta ERD has been linked to greater movement certainty,(Tzagarakis et al., 2010) which may suggest that participants with CP, following therapy, have more confidence in selecting and executing the knee extension motor action required for both proactive and reactive task conditions. Additionally, we saw therapeutic effects for the MRGS, with the cortical activity being weaker after completing the physical therapy protocol. This finding was unexpected, as it is commonly suggested that a stronger MRGS is the result of a greater number of neurons being recruited to successfully complete the task. One possible explanation for the weaker MRGS observed post-therapy is that it may reflect increased neural efficiency in the motor cortex, where fewer neurons are required to execute the same motor command. Additionally, it could be that more neurons were not able to be recruited because of the corticospinal damage associated with CP (Condliffe et al., 2019; Papadelis et al., 2019; Wilson et al., 2011). Hence, optimization of the available cortical neurons was the only viable beneficial neuroplastic change. Alternatively, after participating in the gait training paradigm, which involved complex lower extremity motor tasks, participants may have found the MEG task less cognitively demanding, resulting in the recruitment of fewer neurons, although we would expect such a finding to emerge in higher order areas of the motor control network. Similar optimization (*i.e.*, reduction in the number of neurons recruited) has been observed in the context of stroke in those who have participated in intensive therapeutic approaches aimed at restoring function (Dong et al., 2007; Tatti et al., 2023). Based on the work of Tatti et al. (Podsiadlo and Richardson,

1991). it is also plausible that the MRGS reduction may reflect practice-related adaptation or a shift in the motor control strategy. Potentially, the participants with CP required less attention and/or executive control post-therapy. These changes may have also been connected with familiarity with the task demands at the post-therapy data collection time point. However, caution is warranted with this interpretation as there was no difference in the MEG behavioral variables. While all of these interpretations are plausible, they remain speculative and highlight the need for further investigations to understand and elucidate the mechanisms underlying the observed weaker MRGS post-therapy.

As expected, persons with CP exhibited significant improvements in clinical outcomes following eight weeks of gait training. An 8.77 % improvement in the FGA was observed, highlighting participants enhanced dynamic balance and walking adaptability. Participants also expanded their dynamic range of gait speed by 38.94 %, showing greater flexibility in transitioning from preferred gait speed to fast gait speed and exceeding the MCID for gait speed. Additionally, participants completed the TUG 15.97 % faster post-therapy and met the MCID threshold. This suggests that our sample as a whole had optimized balance and functional mobility following therapy, particularly in tasks with multiple components, such as the TUG with sit-to-stand transitions and turning around a cone. Collectively, the clinical outcome findings support that an eight-week gait training protocol using key ingredients to improve motor planning and execution can yield significant and clinically meaningful changes in dynamic balance, walking adaptability, and overall functional mobility.

When examining the relationship between change scores in oscillatory cortical activity and clinical outcomes, we found that persons with CP who exhibited a stronger post-therapy beta ERD during the planning period tended to also complete the TUG faster after therapy. This finding is notable, as the TUG is a multi-component assessment requiring sequencing of coordinated movements,(Herman et al., 2011) and has demonstrated links to cognitive processes (Körding et al., 2004). Specifically, in older adults, the TUG has been linked with behavioral measures of executive function, whereas other balance assessments that may be perceived as more cognitively demanding, such as the Dynamic Gait Index and Berg Balance Scale, were not significantly correlated with cognitive ability (Körding et al., 2004). These findings suggest that the TUG, though seemingly straightforward, may involve greater cognitive demands than previously assumed. Consistent with this, our results indicate that cortical oscillatory activity during the planning period was correlated with TUG performance, while the FGA, despite potentially being a more complex balance measure, did not show a significant relationship with cortical activity. Overall, this finding suggests that participants that were more certain in the planning of their leg motor actions had better performance on the TUG.

Lastly, it is important to highlight that the key ingredients of this intervention target both neurophysiological mechanisms and clinical presentations seen in persons with CP. The first key ingredient (*i.e.*, exploratory activities that enhance the somatosensory experience through rich/novel movement) encompasses components of the internal model theory, recognizing the sensory system as the "teacher" of the motor system (Shadmehr, 2004; Wolpert, 2007; Boyer et al., 2024). This is crucial as persons with CP often experience sensory processing deficits that can affect motor planning and execution (Cooper et al., 1995; Damiano et al., 2013; Gordon and Duff, 1999; Sanger and Kukke, 2007; Wingert et al., 2009; Kurz and Busboom, 2024; Leech et al., 2021). From a physical therapy standpoint, it is integral that interventions incorporate and draw patient's attention to sensory experiences, such as proprioceptive training, varied terrains, or visuomotor integration tasks. This incorporation may help refine motor control by strengthening the connection between the incoming sensory information and the motor output. The second key ingredient (*i.e.*, activities of adequate intensity that promote gait adaptation and gait speed sustainment) can be applied through goal-driven and optimally challenging tasks that push individuals beyond their typical activity levels and movement patterns. For example, increasing gait training intensity through bursts of fast walking, resistance on a manual treadmill, or integrating cognitive dual tasking can promote functional gains by enhancing motor efficiency and performance. Lastly, our third key ingredient (*i.e.*, optimally challenging activities that emphasize planning and problem solving that requires altering the leg kinematics to meet the environmental and task constraints) was designed to address the motor planning and problem-solving difficulties often experienced by persons with CP, which can hinder their ability to navigate dynamic environments. Clinically, strategies that utilize motor learning paradigms, [75] task variation, and real-world navigation training can enhance adaptive motor planning. These tasks encourage persons with CP to adjust their gait kinematics in response to environmental changes, such as uneven terrain or unexpected obstacles, and can improve their ability to generate and execute alternative motor strategies. Inclusion of these key ingredients into gait training paradigms may ultimately drive beneficial neuroplasticity and support more independent functional mobility for adults with CP as they navigate their communities.

5. Limitations

While this work contributes to the growing body of literature aiming to understand the neurophysiological changes related to physical therapy interventions in persons with CP, some limitations should be acknowledged. The inherent heterogeneity of the CP population is reflected in our sample, which includes variations based on the location of perinatal brain lesions, GMFCS levels, and clinical presentations (*e.g.*, hemiplegia, diplegic, *etc.*). However, due to our moderate sample size and limited statistical power, we were unable to analyze these individual differences. Another limitation is that the neurophysiological findings of this study are based on a seated-knee extension motor action task. While this allows for a controlled recording, it diverges from the full-body coordination required during walking. The generalizability of neural changes observed during the knee task to overground ambulation remains inferential. There also was no placebo-control or alternative treatment group of persons with CP. This limits our ability to derive whether the clinical and cortical changes would be observed if persons with CP participated in physical therapy interventions without the key ingredients presented in the current study. Future studies should consider including a randomized control arm to strengthen causal inferences. In addition, we did not evaluate the post-movement beta rebound (PMBR) response in our study, as our task design was more optimal for assessing the earlier beta ERD and MRGS responses. Previous work has shown that the PMBR is uncharacteristic in persons with CP (Hoffman et al., 2019). Since persons with CP often have problems integrating sensory input and rely on executive control to compensate for sensorimotor deficits, evaluating therapeutic effects on the PMBR could be a fruitful avenue for future investigations. Lastly, our experimental design was not able to disassociate the cognitive-motor changes versus the cortico-muscular changes that are due to gait training. Potentially, contrasting the sensorimotor changes seen in a MEG task that requires cognitive-motor decisions versus a task that is more execution based could advance our understanding of the mechanisms underlying therapy-induced changes in the sensorimotor cortical oscillations.

6. Conclusions

This study demonstrated that an eight-week gait training protocol with key ingredients targeting tasks that stress motor planning and execution led to clinically relevant mobility improvements and changes in the beta ERD and MRGS within the sensorimotor cortices. Specifically, participants with CP exhibited significant and clinically important differences in the FGA, dynamic range of gait speed, and TUG. The beta ERD was stronger post-therapy, while the MRGS was weaker post-therapy. Furthermore, stronger beta ERD during the planning period

was linked with faster completion of the TUG, suggesting a link between cortical oscillatory activity and functional mobility. These findings underscore the importance of integrating principles of cognitive neuroscience and adaptive motor control into gait training paradigms for beneficial neural and clinical outcomes. This work strongly contributes to the growing body of literature aimed at optimizing physical therapy interventions for persons CP.

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CRediT authorship contribution statement

Morgan T. Busboom: Writing – original draft, Formal analysis. **Rachel K. Spooner:** Writing – review & editing, Supervision, Formal analysis, Conceptualization. **Liana S. Chinen:** Writing – review & editing, Formal analysis. **Sarah E. Baker:** Writing – review & editing, Formal analysis. **Brad Corr:** Writing – review & editing, Methodology. **Katie L. Bemis:** Writing – review & editing, Methodology, Conceptualization. **Kimberley Scott:** Writing – review & editing, Methodology. **Tony W. Wilson:** Writing – review & editing, Supervision, Funding acquisition. **Max J. Kurz:** Writing – original draft, Supervision, Methodology, Funding acquisition, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.nicl.2025.103901.

Data availability

Data will be made available on request.

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