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# Effect of Sensory Attenuation on Cortical Movement-Related Oscillations

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## Contents

# Effect of sensory attenuation on cortical movement-related oscillations

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## Abstract

This study examined the impact of induced sensory deficits on cortical, movement-related oscillations measured using electroencephalography (EEG). We hypothesized that EEG patterns in healthy subjects with induced sensory reduction would be comparable to EEG found after chronic loss of sensory feedback. EEG signals from 64 scalp locations were measured from 10 healthy subjects. Participants dorsiflexed their ankle after prolonged vibration of the tibialis anterior (TA). Beta band time frequency decompositions were calculated using wavelets and compared across conditions. Changes in patterns of movement-related brain activity were observed following attenuation of sensory feedback. A significant decrease in beta power of event-related synchronization was associated with simple ankle dorsiflexion after prolonged vibration of the TA. Attenuation of sensory feedback in young, healthy subjects led to a corresponding decrease in beta band synchronization. This temporary change in beta oscillations suggests that these modulations are a mechanism for sensorimotor integration. The loss of sensory feedback found in spinal cord injury patients contributes to changes in EEG signals underlying motor commands. Similar alterations in cortical signals in healthy subjects with reduced sensory feedback implies these changes reflect normal sensorimotor integration after reduced sensory input rather than brain plasticity.

**NEW & NOTEWORTHY** Transient attenuation of sensory afferents in young, healthy adults led to similar changes in brain activity found previously in volunteers with incomplete spinal cord injury. Beta band power associated with ankle movement in these controls was attenuated after prolonged vibration of the tibialis anterior. Evoked potential measurements suggest that prolonged vibration reduces phasing across trials as the mechanism behind this attenuation of cortical activity.

## INTRODUCTION

Disruption of sensory inputs may alter cortical rhythms and drive changes in well-documented but poorly understood oscillatory patterns associated with motor commands. Neural oscillations have long been theorized to facilitate coordination and communication between different cortical regions (Schnitzler and Gross 2005). Beta band activity (15–30 Hz) in particular has been strongly associated with motor control (Kilavik et al. 2013). These neural oscillations in the beta frequencies shift in magnitude and spatial orientation after spinal cord injury (SCI), possibly in response to sensory deficits (Kokotilo et al. 2009). We hypothesized that sensory attenuation in healthy adults could induce a comparable shift in cortical oscillations within the framework of a simple motor task.

Synchronization of dynamic systems occurs in a diverse array of contexts, particularly relevant to the function of the human brain, where intrinsic mechanisms of both single and populations of neurons have been observed to impart coordinated rhythmical activity (Boccaletti et al. 2002; Wang 2010). This synchronous activity in the cortex has been noted across a wide range of frequencies and structural regions (Buzsáki 2004; Feige et al. 2000; Gerloff et al. 1998). Neural oscillations found in the beta frequency range (15–30 Hz) of electroencephalography (EEG) signals demonstrate a distinctive trend over motor areas during movement commands. Specifically, neurons of the motor cortices desynchronize, producing an event-related desynchronization (ERD) during movement initiation and are subsequently followed by a significant event-related synchronization (ERS) (Pfurtscheller and Lopes da Silva 1999). The ERD is associated with a decrease in beta band power, while ERS produces an increase in power, above baseline. These modulations in beta band power have been implicated in numerous cortical roles, from a broad association with motor commands to specific functions ranging from the processing of sensory inputs to cortical anticipation of future events (Reyns et al. 2008; van Ede et al. 2011; Zhang et al. 2008). Although there is no definite consensus on a comprehensive framework for these modulations, there is a growing agreement toward the role of oscillatory signals in mediating the coordination of different local networks, spatially distributed across the brain (Caplan et al. 2003; Laughlin and Sejnowski 2003; Müller-Putz et al. 2007).

Coherence measurements between cortical sources and EMG have provided evidence that afferent pathways directly contribute to sensorimotor beta band oscillations (Mima and Hallett 1999; Witham et al. 2011). Impairment of these pathways from incomplete SCI have been observed to attenuate ERS, along with an amplification of the ERD compared with controls (Gourab and Schmit 2010). Significant increases in activation magnitude occur during motor tasks in sensorimotor areas while several contradictory studies have observed no change or even reduced activations compared with controls during movement (Alkadhi et al. 2004; Castro et al. 2007; Hotz-Boendermaker et al. 2008). Spatially, posterior shifts of motor representation and a tendency for cortical activations to be displaced toward the deafferented limb representation are prominent trends associated with SCI (Cramer et al. 2005; Green et al. 1998; Lotze et al. 2006). Similar disruptions in cortical activity have also been reported in patients with various forms of sensory deafferentation (Cassim et al. 2001; Kristeva et al. 2006; Reyns et al. 2008). The consistent observations of changes in brain activity reported after loss of afferent feedback suggests that there is a link between the processing of sensory information and modulation of cortical oscillations.

Either changes in ERS associated with SCI could occur directly as a result of the loss of sensory feedback to the cortex, or changes could be associated with cortical plasticity secondary to reorganization associated with sensorimotor losses (Behrman et al. 2006; Curt et al. 2008; Raineteau and Schwab 2001). A number of investigators have argued that changes in brain activation patterns are due to plasticity. For example, Hotz-Boendermaker et al. (2008) argue that increased brain activation in SCI subjects is the result of plasticity in response to a higher cognitive threshold needed to perform motor tasks. Similarly, Alkadhi et al. (2004) argue that increased M1 activity is caused by plastic changes in excitability after the loss of sensory input. Cramer et al. (2005) attribute the decrease in the magnitude of activity they found with a cortical loss of function resulting from SCI. To gain insight into the relative contributions of direct sensory loss on cortical signals vs. cortical plasticity, we sought to measure brain signals in young healthy subjects with different levels of sensory feedback.

The objective of this study was to investigate the effects of attenuated proprioceptive feedback on EEG signals of healthy subjects during a simple ankle motor task using prolonged tendon and muscle vibration, previously demonstrated to depress Ia afferents (Shinohara 2005). We hypothesized that the attenuation of sensory input would induce a similar change in beta band modulation as found in SCI, consisting of an attenuation of ERS, magnified ERD, and a posterior spatial shift of activity.

# METHODS

## Experimental protocol.

Ten healthy, neurologically intact volunteers participated in this study (5 men, 5 women, age range 20–35 yr). Written, informed consent was obtained from all participants and the study protocol was approved by the Marquette University Institutional Review Board (protocol number HR-2395). Participants came to a university laboratory and performed a series of simple movements of the ankle or received electrical stimulation of the common peroneal nerve during EEG recordings of brain activity. Vibration of the tibialis anterior (TA) was applied before movements for some trials.

Four different types of conditions were randomly interleaved across five blocks and consisted of the following: *1*) no vibration before a visual cue for ankle dorsiflexion movement, *2*) 10 ± 1 s prolonged vibration of the TA followed immediately by a visual cue for ankle dorsiflexion after the stop of vibration, *3*) electrical stimulation (e-stim) of the TA at rest, and *4*) 10 ± 1 s vibration of the TA followed 0.5 s later by e-stim after the stop of vibration. There was a total of 50 trials of each condition, separated by a 10 ± 1-s interval when the subject was instructed to remain at rest. The visual-cued movements consisted of a brisk dorsiflexion of the right ankle after a visual cue presented on a screen (visual fixation cross at center of screen, green border presented around the edge of the screen to cue movement), while seated in a comfortable position. Subjects were seated on a reclined seat, with the legs hanging vertically off the seat. The ankle was free to move, loaded only by the weight of the foot. EMG was used to quantify the relative size of each contraction. E-stim consisted of a 2-Hz stimulus, 2.5-s duration, (stimulator model D67A, Digitimer, Letchworth Garden City, UK) applied to the common peroneal nerve where it crosses the head of the fibula. Test stimuli were applied to different points on the skin near the head of the fibula to identify a site producing a twitch with low-amplitude stimulus. A bar electrode with two 1-cm-diameter contacts (2.5 cm between electrodes) was used to deliver a stimulus at 90% of motor threshold, identified by visual observation of twitch contraction before beginning the test protocol. The e-stim was used to produce an evoked potential, to obtain a measure of the cortical response to the ascending sensory signal. For vibration, a DC motor (Faulhaber Minimotor, Schönaich, Germany) was used to rotate two eccentric weights (6 g) that attached to the motor's shaft (1 weight on each end of the motor) to produce the vibration (~10-N peak force). The motor and weights were encased within a plastic tube, which was securely strapped onto the subject's lower leg, centered on the TA muscle belly. A custom LabVIEW program was used to send a voltage command signal, producing a 70-Hz vibration. Vibration was applied to condition the sensory afferents of the muscle (Ribot-Ciscar et al. 1998) so that they would be less responsive during the movement.

## Data acquisition.

Signals from a 64-channel, active electrode EEG cap (ActiCap, Brainproducts), using the modified 10–20 convention (Modified Combinatorial Nomenclature) and FCz reference, were sampled at 2,000 Hz and bandpass filtered between 1 and 500 Hz (Synamps 2 amplifier, Compumedics Neuroscan). Electrode

impedance was maintained below 20 kΩ using high-viscosity electrolyte gel (SuperVisc, Brainproducts). Electromyography (EMG) recordings were taken from the TA and the medial gastrocnemius (MG) muscles using wireless electrodes (Trigno, Delsys), sampled at 2,000 Hz and bandpass filtered between 10 and 350 Hz.

## Data analysis.

Offline data analysis was conducted using the Fieldtrip and EEGLab toolboxes, Brainstorm (Tadel et al. 2011), and custom scripts (MATLAB, MathWorks). EEG data were rereferenced from the FCz electrode to a whole head average reference and epoched by movement onset as defined by TA EMG (5 s before event or vibration and 5 s after) or e-stim pulse (500 ms before event and 500 ms after). Epochs with gross artifacts were removed according to a *z*-score threshold above 2 standard deviations of the mean (mean 4.5 out of 50 epochs for each condition across all subjects), while blink artifacts were removed through independent component analysis using the extended runica (Makeig et al. 1996) and ADJUST algorithms for determining artifact related components (Mognon et al. 2011).

Brain signals associated with dorsiflexion movement were characterized by event-related desynchronization (ERD) followed by event-related synchronization (ERS) of the beta frequency band (13–35 Hz). A beta band time frequency (TF) decomposition of the Cz electrode was calculated using Morlet wavelets. The TF decompositions were smoothed over time using a Savitzky–Golay smoothing filter (2nd order polynomial, frame size: 20% of epoch length), averaged across epochs and referenced to a baseline rest period either before the onset of vibration in conditions with prolonged vibration or the visual cue in conditions without vibration as a percent change in power. Local ERD and ERS minima and maxima of individual subject's TF decompositions were identified using a custom MATLAB masking algorithm identifying regions of interest consisting of >75% maximum power or <50% minimum power. Minimum (ERD) and maximum (ERS) TF power values between the no-vibration and prolonged-vibration conditions were then compared using a paired *t*-test (α = 0.05).

Phasing differences across trials may have underscored changes observed in the evoked potential following prolonged vibration. To better characterize the effect of vibration on cortical activity, phase coherence of the E-stim conditions (no vibration/prolonged vibration before E-stim) was calculated using an intertrial coherence (ITC) method (Makeig et al. 2002; Tallon-Baudry et al. 1996):

$$\mathrm{ITC}(\mathrm{time,frequency}) = \left| \frac{\sum \frac{X}{\sqrt[2]{X^{*}X}}}{n_{\mathrm{trials}}} \right| \leq 1,$$

summed across trials, where $X$ = Fourier power spectrum (real and imaginary values).

Maximum ITC values between the no-vibration and prolonged-vibration conditions were compared using a paired *t*-test (2 tailed, α = 0.05) to evaluate the difference in latencies of evoked potentials across trials.

Possible differences in the motor characteristics of ankle dorsiflexion following prolonged vibration were quantified using EMG of the TA and MG. EMG data were rectified and the root mean square (RMS) was calculated using a 200-point sliding window with 50% overlap. Muscle activation was enveloped using a Hilbert and *z*-transform to find EMG amplitudes and latencies. Differences in amplitudes and latencies for the TA/MG EMG between the no-vibration and prolonged-vibration conditions were assessed using a paired *t*-test ($\alpha = 0.05$).

The spatial topography of beta band modulations and evoked potentials on the cortex were characterized using the Brainstorm toolbox. Source localizations of ensemble averaged EEG signals onto a common cortical surface (*MNI/*Colin27 brain, 1-mm resolution) were prepared using a boundary element method forward model (OpenMEEG) and a whitened, minimum-norm inverse solution (Baillet et al. 2001). Sources for individual subjects were *z*-score normalized to a baseline at rest for group averages. Cortical sources for the e-stim conditions with and without prolonged vibration were cross-correlated for each vertex of the MNI/Colin27 brain mesh to localize evoked potential phasing differences found previously with ITC. The mean norm of the cross-correlated e-stim values were calculated from three cortical regions of interest (ROI) located in the left hemisphere: frontal (caudal middle and superior frontal gyri), sensorimotor (precentral, postcentral and paracentral gyri), and parietal (inferior and superior parietal gyri), using a gyral based Desikan-Killiany cortical atlas (Desikan et al. 2006).

## RESULTS

Prolonged TA vibration muted the ERD and ERS signals associated with dorsiflexion movements. Maximum beta band synchrony (ERS) of the group-averaged Cz electrode was significantly attenuated from 138 ± 51 to 93.8 ± 52% (%Δ in power from baseline rest, $P = 0.0199$) after prolonged vibration compared with ankle dorsiflexion without vibration (Fig. 1). Maximum desynchrony (ERD) of beta band power at movement onset was also observed to be attenuated from 34.1 ± 15 to 22.1 ± 13% (%Δ in power from baseline rest, $P = 0.0557$) after prolonged vibration.

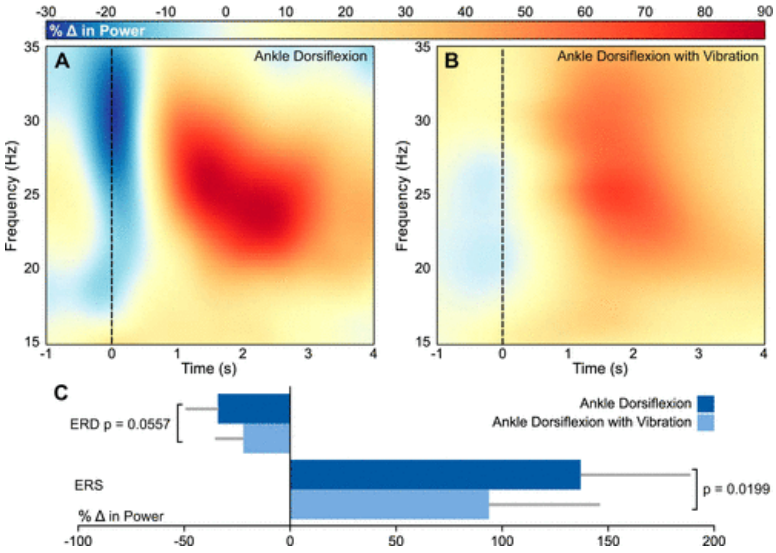


**Fig. 1.** Group-averaged time frequency decomposition of the Cz electrode, normalized to percent change from baseline rest, of ankle dorsiflexion (*A*) and ankle dorsiflexion after sensory attenuation using prolonged vibration

(*B*). Dashed line represents movement onset as defined by tibialis anterior (TA) EMG. Maximum and minimum power values of the time frequency (TF) decomposition, identified as the mean power of regions of interest (ROIs) consisting of either >75% of maximum power or <50% of minimum power, were used in a paired *t*-test to determine significance, while error bars denote standard deviation (*C*). ERD, event-related desynchronization; ERS, event-related synchronization.

Despite the changes in the brain signals, the motor output was similar for the vibration and no-vibration tests. The corresponding motor output, as measured by EMG of the TA and MG presented in Fig. 2, was not statistically different in mean or variance (*t*-test/*F*-test) after vibration in either latency (TA paired *t*-test/*F*-test: *P* = 0.88/0.26) or amplitude (TA: *P* = 0.31/0.27) across subjects. Thus, there were no apparent differences in the motor output for the vibration and no-vibration conditions.

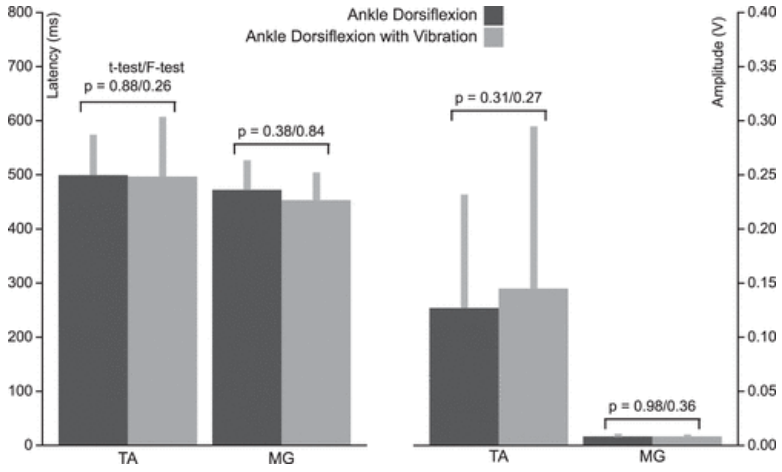


**Fig. 2.** Latency of movement onset after the visual cue as defined by TA and medial gastrocnemius (MG) EMG (*left*). EMG amplitude of TA and MG during movement (*right*).

There were slight changes in the localization of modeled cortical current sources with TA vibration. Cortical sources of ankle dorsiflexion during movement initiation were localized to frontal, medial motor, and sensorimotor areas associated with motor planning, execution, and control of the leg (Fig. 3*A*). These cortical sources shifted posteriorly into medial, superior parietal areas during beta band event-related synchronization, shown at the time point corresponding to maximum power in Fig. 3*B*. After prolonged vibration, cortical sources were observed to shift posteriorly during movement initiation, while largely confined to the same regions during event-related synchronization (Fig. 3*B*).

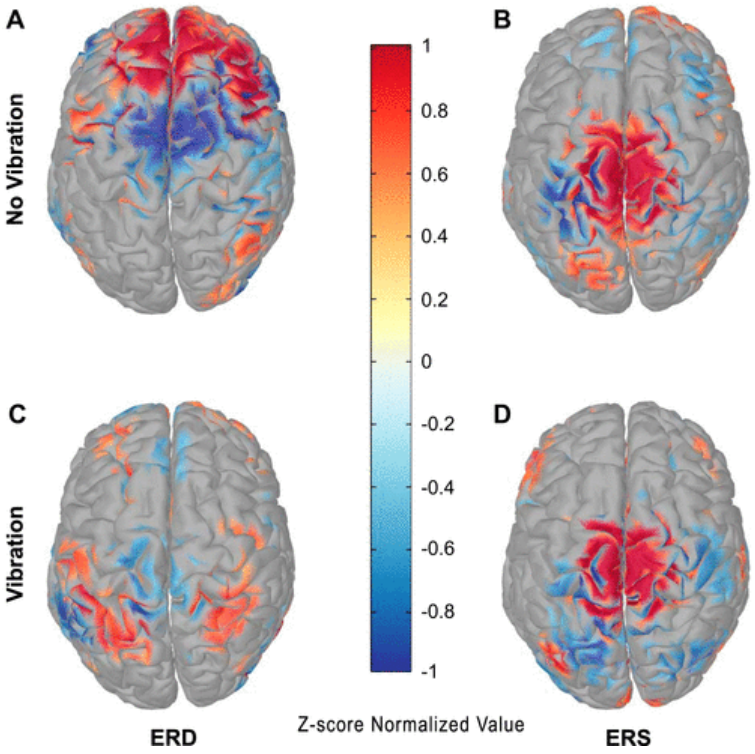


**Fig. 3.** Source localization averaged across all subjects of ankle dorsiflexion during movement onset (*A*) and maximum TF power (*B*), in addition to movement onset (*C*) and maximum TF power (*D*) after prolonged vibration.

Evoked potentials using electrical stimulation of the TA were measured to characterize the effect of prolonged vibration on cortical activity. Group ensemble averaged EEG signals from over the Cz electrode during the e-stim conditions showed several positive and negative peaks following stimulation (Fig. 4*A*). A strong phasing of the e-stim evoked response across trials was observed in the ITC (maximum value: 0.46) corresponding with the N80 peak (Fig. 4*B*) and was attenuated (paired *t*-test: $P = 0.052$) following prolonged vibration (maximum value: 0.35) (Fig. 4*C*). Spatial localization of the phasing between the e-stim evoked response with and without prolonged vibration showed that components of the evoked response after prolonged vibration phase lagged the evoked response without vibration by ~30 ms in the frontal and sensorimotor areas (Fig. 4*D*).

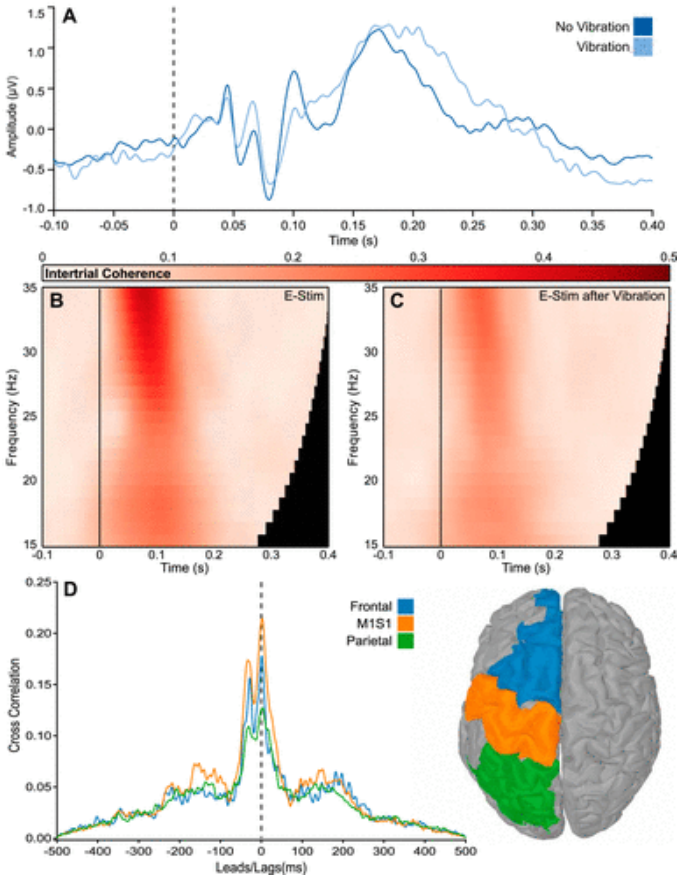


**Fig. 4.** Grand average (Cz electrode) of the electrical stimulation (e-stim) evoked response with and without prolonged vibration (*A*), intertrial coherence of the e-stim evoked response (*B*) intertrial coherence of the e-stim evoked response after prolonged vibration (*C*), and grand average cross-correlation map across regions of interest (*D*).

## DISCUSSION

Acute changes in patterns of movement-related brain activity were observed following attenuation of sensory feedback in young, healthy subjects. We measured a significantly decreased beta power in ERS with simple ankle dorsiflexion after prolonged vibration of the TA. Beta band power shifts in premotor and sensorimotor cortical oscillations were not accompanied by a change in the timing or magnitude of ankle dorsiflexion as measured by EMG. Prolonged TA vibration decreased the ITC of evoked potentials, indicating that sensory feedback was disrupted, likely due to increased variance in the timing of afferent sensory information processed by the cortex. Our findings provide evidence toward beta band oscillations holding a significant role in the integration of sensory input with cortical motor commands.

Beta band oscillations measured from our participants modulated with basic cued ankle dorsiflexion, consistent with beta band modulations in other motor tasks. The pronounced rebound in beta power following a discrete movement was characteristic of ERS (along with its preceding ERD counterpart) in EEG measurements (Pfurtscheller 1992). From upper and lower limb movements to active, passive, and imagined activations, the reproducibility with which ERS has been observed across a wide variety of motor paradigms supports the idea that this prominent feature of motor commands must be of some

cortical importance (Demandt et al. 2012; Müller-Putz et al. 2007). Proposed theories are numerous, including the inhibition of motor networks to maintain certain motor states (Gilbertson et al. 2005; Pfurtscheller et al. 1996), resetting the sensorimotor network in preparation for further sensory inputs (Gaetz and Cheyne 2006; Zhang et al. 2008), and even the modulation of attention or correctness of movement (Koelewijn et al. 2008; van Ede et al. 2011).

Although a consistent decrease in ERS within an individual subject was found when comparing the no-vibration ankle dorsiflexion with the prolonged vibration trial, we observed considerable variation between subjects in overall magnitude, phasing, and frequency in ERD and ERS. Previous studies have observed large variations in phase and frequency when looking at beta band corticomuscular and intermuscular coherence of healthy controls (Kilner et al. 1999; Riddle and Baker 2005). In addition, reorganization of the cortex after SCI has shown to be highly variable according to numerous factors such as time and age (Moxon et al. 2014). It is likely that a combination of natural variations in cortical structure (Gaser and Schlaug 2003; Maguire et al. 2000), slight differences in experimental methods such as electrode placement, and the complicated underlying dynamics of how these beta band oscillations interact with afferent pathways would lead to significant between subject differences.

The transient nature of the sensory attenuation used in our study and its acute effect on beta ERS for healthy, young subjects suggests that these oscillations are changing within the confines of a normal, fully functioning mechanism for sensorimotor integration, rather than permanent changes in brain structure and connectivity from neural plasticity associated with permanent sensory loss. Attenuated beta ERS in motor commands have been reported in several chronic conditions linked to somatosensory deficits such as incomplete SCI, neuropathy, and amyotrophic lateral sclerosis and as a result of normal aging (Bizovičar et al. 2014; Gourab and Schmit 2010; Labyt et al. 2006; Reyns et al. 2008). Long-term sensory deafferentation from these conditions may imply a structural reorganization of cortical sensorimotor networks (Feige et al. 2000). However, the prolonged vibration we used to attenuate proprioceptive feedback was interleaved with “normal” ankle movements, providing a transitory disruption of proprioceptive input, yet still leading to a decrease in the beta power ERS in healthy, young adults. Similar decreases in motor beta oscillations were found after an ischemic nerve block to temporarily deafferent healthy controls (Cassim et al. 2001). A simple increase in cortical baseline from prolonged vibration would not account for the measured decrease in ERD. Instead, the immediate modulation of beta power ERS after sensory attenuation in otherwise normal subjects and the return back to normal magnitudes of beta power during trials without the vibratory stimulus may support the idea that these oscillatory signals play an active role in mediating the coordination of sensorimotor networks.

An increase in cortical beta power may reflect the processing of proprioceptive feedback in relation to the execution of a motor command. Beta power modulations have previously been associated with proprioceptive afferents (Alegre et al. 2002; Gaetz and Cheyne 2006; Keinrath et al. 2006). In addition, these oscillations have been theorized to underlay the comparison of motor predictions with their respective motor outcomes (Arnal and Giraud 2012; Koelewijn et al. 2008; Kristeva et al. 2006). Holding an active role in mediating motor plans corresponds to previous observations of beta band cortical activity having direct, functional links to movement in both passive measurements of EMG coherence and slowing of voluntary movement after direct cortical stimulation at these frequency ranges using transcranial magnetic stimulation (Kristeva et al. 2007; Pogosyan et al. 2009). The short latency and

continuous feedback of proprioceptive afferents would provide an effective error signal that could generalize across a wide variety of conditions.

Substantial proprioceptive feedback to alpha motor neurons is channeled through excitatory Ia afferents from muscle spindles sensitive to muscle length (Houk et al. 1981). Vibratory evoked potentials have been traced to regions in the somatosensory cortex, while oscillations reflecting the frequency of vibration have been observed in the prefrontal cortex (Romo et al. 1999; Spitzer, Wacker, and Blankenburg 2010; Tobimatsu et al. 1999, 2000). Prolonged vibration has been observed to reduce feedback from Ia afferents resulting from a number of subcortical factors including heighted discharge threshold, presynaptic inhibition, and neurotransmitter exhaustion (Curtis and Eccles 1960; Hayward et al. 1986; Hultborn et al. 1987). Cortically, we were able to measure the ITC of phase-locked e-stim evoked potentials from the Cz electrode, which decreased following prolonged vibration. The decreased coherence was not a result of an attenuation in amplitude, but because of a phase shift in certain components of the evoked potential occurring earlier in the premotor and sensorimotor cortex. This may suggest that prolonged vibration attenuates Ia afferent sensory feedback cortically by increasing the variance of feedback over a period of time rather than a simple reduction of firing rate or amplitude.

### Conclusions

Cortical beta band modulations have been well characterized to modulate with motor commands. We observed that attenuation of sensory feedback in young, healthy adults leads to a corresponding decrease in beta band synchronization magnitude. This acute, yet temporary change in beta oscillations suggests that these modulations are a mechanism for sensorimotor integration, rather than a mere byproduct of cortical activity.

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## DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

## AUTHOR CONTRIBUTIONS

J.J.L. and B.D.S. conceived and designed research; J.J.L. and B.D.S. performed experiments; J.J.L. analyzed data; J.J.L. and B.D.S. interpreted results of experiments; J.J.L. prepared figures; J.J.L. drafted manuscript; J.J.L. and B.D.S. edited and revised manuscript; J.J.L. and B.D.S. approved final version of manuscript.

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