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RESEARCH ARTICLE

Sensory Processing

A single exposure to prolonged flexor carpi radialis muscle vibration increases sensorimotor cortical areas activity

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Abstract

Prolonged local vibration (LV) is thought to promote brain plasticity through repeated Ia afferents discharge. However, the underlying mechanisms remain unclear. This study therefore aimed at determining the acute after-effects of 30-min LV of the flexor carpi radialis muscle (FCR) on sensorimotor (S1, M1) and posterior parietal cortex (PPC) areas activity. Sixteen healthy participants were tested before and immediately after 30 min of FCR LV. Electroencephalographic signals were recorded during isometric submaximal wrist flexions. Time-frequency analyses were performed at source levels during contraction preparation, contraction initiation, force plateau, and relaxation. After LV, the results showed an increase in α and β desynchronizations in the source activity for the estimated M1, S1, and PPC during contraction preparation ($P \leq 0.05$) and contraction initiation ($P \leq 0.05$; except for PPC in the β band: $P = 0.07$), and a greater α desynchronization in M1, S1, and PPC ($P < 0.01$) during force plateau. No LV-induced changes were observed during relaxation. Prolonged LV on the upper limb could increase estimated cortical activity within M1, S1, and PPC areas during subsequent isometric contractions. This could be due to LV-induced Ia afferents inputs projecting onto cortical areas through proprioceptive pathways, and likely triggering brain use-dependent plasticity.

NEW & NOTEWORTHY Prolonged local vibration (LV) is thought to promote brain plasticity, yet the underlying mechanisms remain unclear. In the present study, we used electroencephalography in healthy subjects and found increased activity in primary motor, primary somatosensory, and posterior parietal areas after a single exposure to LV. This may be due to LV-induced Ia afferents inputs projecting onto cortical areas through proprioceptive pathways, and likely triggering brain plasticity.

EEG; local vibration; M1; PPC; S1

INTRODUCTION

Local vibration (LV) applied on a muscle or its tendon at high frequency (≈ 100 Hz) and low amplitude (< 2 mm) is known to induce both acute and chronic neuromuscular adaptations through repeated Ia afferents discharge projecting at both spinal and cortical levels (1). As such, prolonged LV has emerged in the past decades as a new training (2) and rehabilitation modality (3). In patients with stroke as an example, the repetition of LV sessions has been reported to induce an improvement of motor function (4, 5) and a

decrease in spasticity (6, 7). Although this may be the result of neuroplasticity, the neurophysiological mechanisms underlying the beneficial effects of LV interventions are still poorly understood and further studies are required to better understand LV-induced effects at the cortical level.

In healthy subjects, an increase in contralateral primary somatosensory (S1) and motor (M1) cortex activity during LV exposure has been demonstrated using functional magnetic resonance imaging (fMRI) (8–10). Electroencephalography (EEG) can also be used to record brain rhythmic oscillations induced by LV. Main outcomes are oscillations in α “mu



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rhythms" (8–12 Hz) and β (15–35 Hz) bands, both related to M1-S1 activity (11), with α rhythms being associated to somatosensory sensations and β rhythms associated to both movements and somatosensory sensations (12). Those oscillations can be investigated through event-related desynchronization (ERD) and event-related synchronization (ERS). ERD is usually interpreted as an enhanced activity (i.e., when compared with a baseline period) in M1-S1 neuronal population, as known to occur during movement preparation and movement execution (13, 14). Conversely, ERS is commonly visualized in the β band (i.e., β rebound) after the completion of a movement (15) and therefore reflects a reduced activity in M1-S1 neuronal population (13, 14). Such β rebound can also be observed after somatosensory stimulation, reflecting a return to baseline of sensorimotor networks (16). In two separate studies, bilateral M1-S1 α ERD (17) and bilateral M1-S1 β ERD (18) were reported during biceps brachii LV being applied while participants remained relaxed. In a recent study, α and β ERD were also described during biceps and triceps tendons LV (19). Similarly, a contralateral M1-S1 β ERD was observed during LV applied to the Achilles tendon (20). Altogether, these results suggest an increased sensorimotor cortical activity during LV (17, 18, 20), in a similar way as it was observed during movement execution (15). This increase in cortical activity during LV could potentially lead to an acute use-dependent cortical plasticity when LV exposure is prolonged, as it is observed after prolonged nerve electrical stimulation (21, 22).

Immediately after 10 min of LV, it was reported an increased contralateral α ERD at the level of electrodes placed over M1-S1 while performing an isometric contraction. This finding suggests a greater M1-S1 activity during a contraction when performed after the LV (23), which could be explained by an increase of cortical excitability (24). Actually, greater cortical excitability after prolonged LV has already been suggested by studies combining transcranial magnetic stimulation (TMS) and corticospinal tract electrical stimulation (25–27). Moreover, a LV-induced increase in the excitability of the sensorimotor pathways has been suggested in a TMS study (28). Indeed, repeated Ia afferents discharge elicited by LV directly projects to S1 at cortical level, so the increase in sensorimotor pathways excitability may rely on topographically and functionally specific reciprocal connections between S1 and M1 (29). Accordingly, an enhanced activity of the reciprocal connections between S1 and M1 was previously reported after LV exposure in an EEG study (17). Besides S1-M1 activity and connectivity, the role of the parietal posterior cortex (PPC) in afferent processing has not yet been investigated after prolonged LV. It could be of interest since parietal areas are known to receive specific somatosensory afferent inputs and to be functionally connected with M1 and S1, with a major role in motor control (30, 31).

Although previous research has demonstrated increased activity in M1-S1 areas during LV, the literature is inconclusive regarding cortical activity after prolonged LV. We, therefore, recorded EEG during repeated isometric contractions performed after 30 min of LV applied to the right flexor carpi radialis muscle (FCR). We calculated ERD/ERS in M1-S1 and PPC. We hypothesized a greater desynchronization in M1-S1 and PPC when performed after LV.

MATERIALS AND METHODS

Participants

Sixteen healthy participants (10 men and 6 women; age: 27 ± 6 yr; stature: 170 ± 11 cm; mass: 68 ± 8 kg) were included in the experiment. All participants were free from neurological disease and musculoskeletal injury. The study was approved by the institutional ethics committee and conformed to the Declaration of Helsinki (except for registration in a database). Written informed consent was obtained from each participant prior to the study begin.

Experimental Procedure

Participants visited the laboratory for a single session lasting ~ 2 h. During the entire experiment, participants were seated in a comfortable chair, the left arm totally relaxed and the right arm installed on a custom-built ergometer (Fig. 1).

Baseline measurements began with a standardized warm-up of 10 submaximal isometric contractions, gradually increasing the force produced to reach the maximum wrist flexion force. Then, participants performed three 5-s maximally voluntary contractions (MVCs) spaced by 60 s of rest. During MVCs, participants were instructed to contract as hard as possible and were verbally encouraged by the experimenter. The best MVC was retained to set the target for subsequent contractions (i.e., 10% MVC), as displayed on the screen of the computer. Participants were then familiarized with the repeated isometric contractions procedure. Specifically, participants had to perform sequences initiated by 2 s of contraction preparation, followed by 7 s of contraction of isometric wrist flexion and then 5 s of relaxation before starting a new sequence. Each steps of the sequence were punctuated by a beep sound as displayed in Fig. 2. During contraction preparation and relaxation, participants were instructed to remain relaxed and for their whole body to be as still as possible. During contractions, participants were instructed to contract as quickly as possible and to maintain 10% MVC as stable as possible.

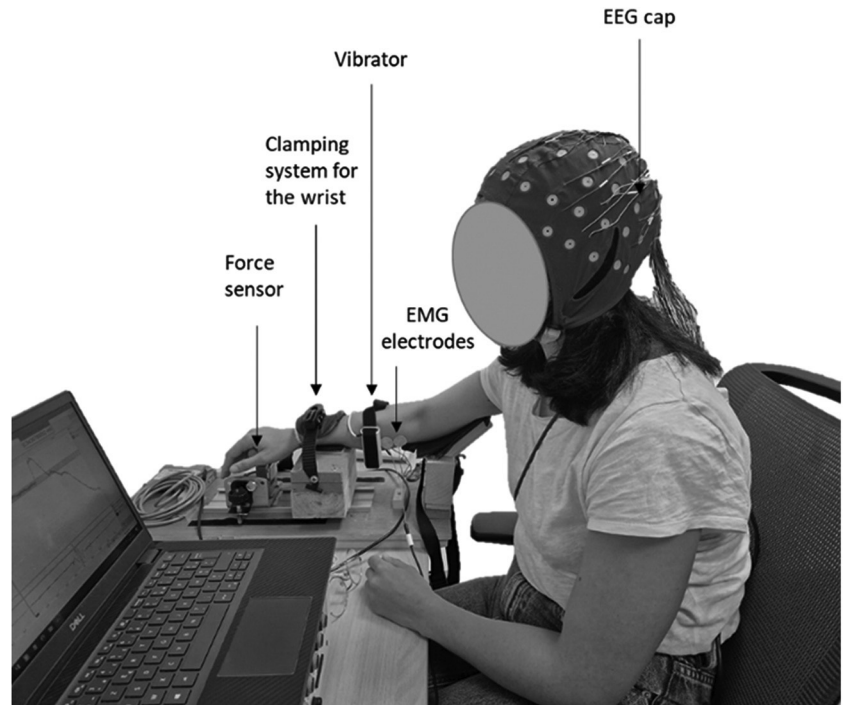
When participants were familiarized (i.e., when they were able to perform at least three consecutive sequences without any error), the EEG recording began while participants had to complete 30 consecutive sequences as previously presented. Those measurements were performed at baseline (CON-1), after 10 min of rest (CON-2; i.e., to investigate the effects of a control period), and after 30-min LV (POST-VIB). LV (100 Hz with an amplitude of 1 mm; VB 115, Techno Concept, Mane, France) was applied on the right FCR muscle belly for three times for 10 min with 1 min of rest in-between. During control and LV periods, participants were asked to remain fully relaxed and to focus their attention on a cross placed at 1 m at eye level.

Instrumentations and Preprocessing

Force recording.

Voluntary isometric wrist flexion force was recorded with a custom-built ergometer. The right arm of the participants was restrained in an orthosis with an elbow angle of 120° , a shoulder abduction of 20° , and no shoulder flexion. The forearm was locked in a pronation position with a clamping system at the wrist and a force sensor positioned in the palm of

Figure 1. Illustration of the custom-built ergometer with placement of the vibrator, electromyographic (EMG) electrodes, and EEG cap.



the hand to measure the strength in wrist flexion. This position was maintained throughout the entire session.

Electrophysiological recordings.

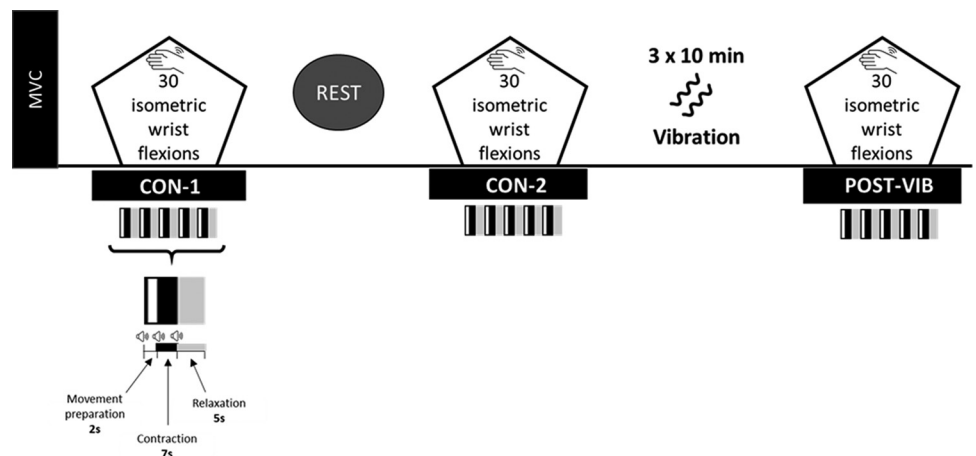
EEG activity was recorded continuously from 64 Ag/AgCl surface electrodes embedded in an elastic cap (10-20 system, EEGCap64SE; Bioelettronica, Italy). The signal was preamplified at the electrode sites and postamplified with direct coupled amplifiers, digitized at a sampling rate of 2,048 Hz (OT BioLab +, Bioelettronica). The signals of each electrode were referenced to the mean signal of all 64 electrodes. EEG signals were resampled at 1,024 Hz and filtered offline with a 1–120 Hz band-pass digital filter and a notch filter at 50 Hz. Independent component analyses were performed to delete eyes artifacts. EEG signals during the 30 sequences were segmented into 30 epochs from the beginning of the contraction preparation phase (i.e., 2 s before the start of the contraction; –2 s) to the end of the relaxation period (12 s; see Fig. 3).

After artifact rejection based on visual inspection, 21 epochs in total were included in the analyses for each participant, corresponding to 70% of valid data on average per participant. Epochs were averaged for each participant and each time point (i.e., CON-1, CON-2, and POST-VIB). All the pre-processing and processing of EEG data was implemented in Brainstorm software (32).

Electromyographic recording.

Participants were first prepared by shaving, gently abrading the skin, and then cleaning it with isopropyl alcohol. Electromyogram (EMG) of the right FCR was recorded with pairs of self-adhesive surface electrodes placed on the muscle belly (Electrode EMG—CDE Series, OT Bioelettronica, Italy) in a bipolar configuration with a belly-belly montage. Signal was band-pass filtered according to EEG signals (1–120 Hz band-pass), rectified and analyzed offline using Brainstorm software.

Figure 2. Overview of the experimental design. EEG was recorded at baseline (CON-1), after 10 min of rest (CON-2), and after 30 min of local vibration (LV) (POST-VIB).



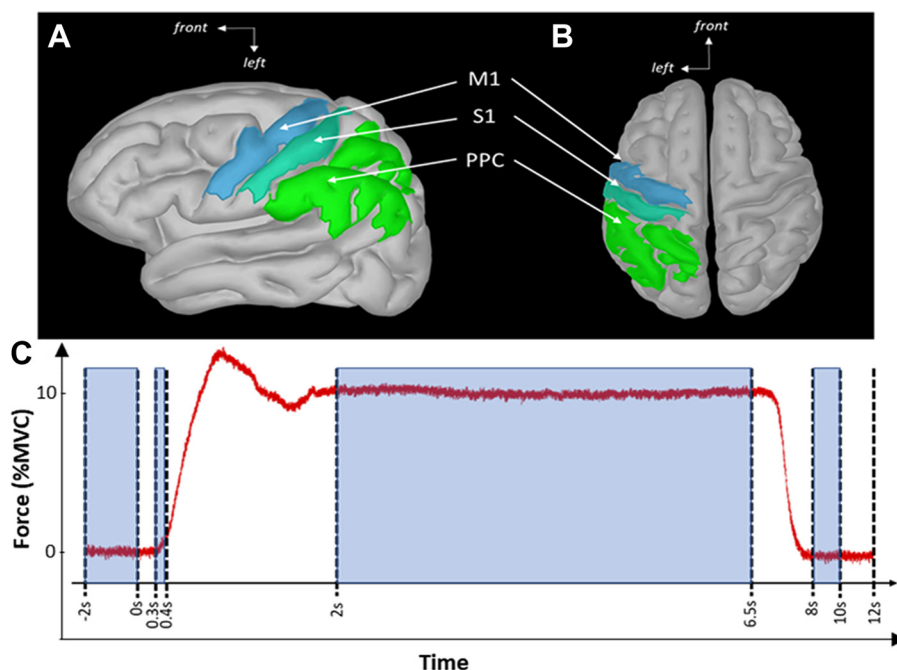


Figure 3. Overview of the selected regions of interest for the estimated source activity within the cortex, calculated based on the 64-channel EEG recordings: left primary motor cortex (M1), left primary somatosensory cortex (S1), and left posterior parietal cortex (PPC) in a side view (A) and a top view (B). C: illustration of a typical sequence where participant alternates contraction preparation, contraction at 10% maximally voluntary contraction (MVC) and then relaxation. Time windows identified for the EEG analyses are presented in blue: contraction preparation [−2 s; 0 s], contraction initiation [300 ms; 400 ms], force plateau [2; 6.5 s] and relaxation [8; 10 s].

Data Analyses

Force and electromyographic analysis.

For each contraction, the reaction time was measured from the trigger signaling the beginning of the contraction to the beginning of the visual force contraction (i.e., when force signal increased by 1 N). The reaction time was measured for each contraction and averaged for CON-1, CON-2, and POST-VIB for each participant. Reaction time was identical across time points (CON-1: 380 ± 55 ms, CON-2: 390 ± 85 ms, POST-VIB: 370 ± 74 ms, $P = 1$), allowing to define the same windows of interest for the EEG analysis.

During the force plateau [2; 6.5 s], EMG activity was calculated as the root mean square (RMS) of the signal.

Source localization and time-frequency analyses.

Time-frequency analyses were conducted in the EEG source space to enhance spatial resolution (estimated at 5–9 cm) (33). Source localization techniques were used to minimize issues related to volume conduction and reference electrodes (34) while accounting for interindividual cortical morphology variability. Neural sources were estimated with dynamical Statistical Parametric Mapping (35), implemented in the Brainstorm software (32), which interprets each electrode signal as an unconstrained dipole with equal weight contributions. Each source was derived from a linear recombination of signals from all 64 electrodes. The kernel matrix in Brainstorm was used to project sensor data to specific cortical locations through a three-shell boundary element model (i.e., scalp, outer skull, and inner skull), applied to the Montreal Neurological Institute (MNI Colin27, 15,000 vertices) brain template. In the absence of individual MRI scans, we used standardized 10-20 system coordinates for reliable spatial placement of the EEG channels. At the source level, data were transformed into the time-frequency domain using Morlet wavelet transforms and averaged across sources in each region of interest (36). The 1-Hz central frequency (full width at half

maximum, time central = 3 s) of the wavelet was selected as an optimal compromise between temporal and spectral resolution (36), yielding smooth, interpretable time-frequency plots that offered a good compromise between temporal and spectral resolutions (36). We computed α (8–12 Hz) and β (15–35 Hz) event-related desynchronization/synchronization (ERD/ERS) relative to a baseline period (from 10 s to 12 s in each sequence, corresponding to the end of the relaxation phase and 4 to 2 s before movement initiation). Four different time windows were selected for ERD/ERS calculation: contraction preparation [−2 s; 0 s], contraction initiation [300 ms; 400 ms], force plateau [2; 6.5 s], and relaxation [8; 10 s], allowing analysis over oscillatory cycles (Fig. 3C). ERD/ERS average power in the α and β bands was calculated for each window and expressed as a percentage of the baseline power (32). The regions of interest were defined based on the Destrieux cortical atlas (37), targeting the left M1, S1, and PPC regions (contralateral hemisphere, see Fig. 3, A and B), corresponding to Brodmann areas BA4, BA3, and BA7 + BA40 + BA39. According to this atlas, M1 corresponds to the precentral cortex with 164 vertices, S1 corresponds to the postcentral cortex with 120 vertices, and PPC to the inferior and superior parietal cortex with 408 vertices. For each region of interest, three sources per vertex were calculated based on all 64 EEG electrode signals, totaling 492 sources for M1, 362 for S1, and 1,224 for PPC. Although signals from all electrodes contributed to source calculations, proximity-based weighting was applied, giving higher influence to electrodes nearest each region of interest. For left M1, the primary electrodes were C1, C3, FC3, and C5; for left S1, C1, C3, CP1, and C5; and for left PPC, CP1, CP3, and P5. This approach allowed for a more refined differentiation of activity between M1 and S1 than what could be achieved at the sensor level alone.

Statistical Analyses

All data are presented as medians, with error bars indicating [min; max] values. The Shapiro–Wilk test and Levene’s

test were used to assess normality and homogeneity of variances, respectively. The normality test for ERD/ERS data was not satisfied, so paired Wilcoxon tests with a false discovery rate (FDR) correction were applied to control for multiple comparisons.

We conducted separate comparisons for each frequency band (α and β) between CON-1 and CON-2 (control condition) and between CON-2 and POST-VIB (experimental condition) across each cortical area (M1, S1, PPC) and four time windows, resulting in a total of 48 paired Wilcoxon tests. For EMG RMS data, the normality test also failed, and a one-level Friedman test was used for comparisons across conditions (CON-1, CON-2, and POST-VIB).

Within the results, both the V value and P value from the paired Wilcoxon tests are presented, where the V value indicates the sum of ranks for positive differences. The significance level was set at $P \leq 0.05$ for all analyses. Statistical analyses were performed using R software (v.1.3.1093).

RESULTS

Isometric contraction analysis was performed on the 16 tested participants. No differences were found between CON-1 and CON-2 (i.e., control condition) for all parameters (time-frequency) ($P > 0.05$) and only vibration effect will be presented below (i.e., comparison between CON-2 and POST-VIB).

Contraction Preparation [−2 s; 0 s]

During contraction preparation, an increase in cortical desynchronization in the α band was observed in the estimated M1 ($V = 109$, $P = 0.05$), S1 ($V = 121$, $P = 0.02$), and PPC ($V = 117$, $P = 0.01$) at POST-VIB compared with CON-2 (Fig. 4A). An increase in cortical desynchronization in the β band was similarly found in the estimated M1 ($V = 114$, $P = 0.05$), S1 ($V = 113$, $P = 0.02$) but not in PPC ($V = 103$, $P = 0.07$) at POST-VIB compared with CON-2 (Fig. 4B).

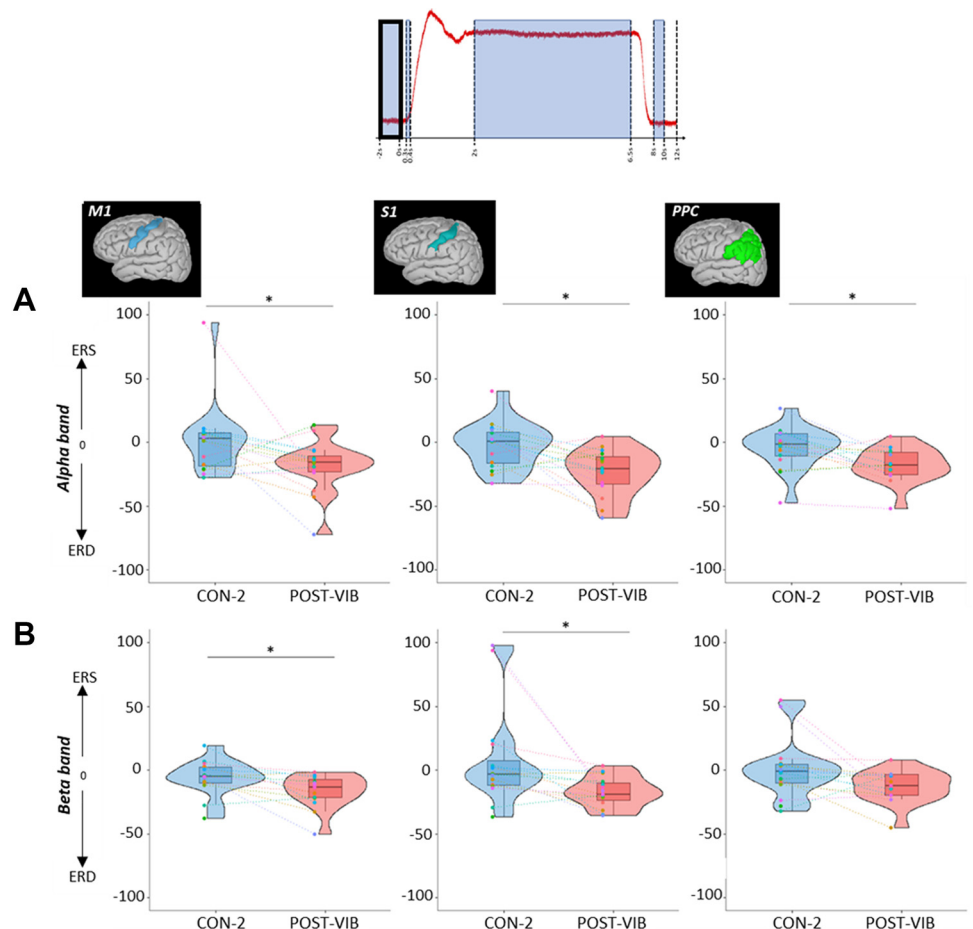
Contraction Initiation [300 ms; 400 ms]

During contraction initiation, an increase in cortical desynchronization in the α band was observed in the estimated M1 ($V = 133$, $P = 0.03$), S1 ($V = 133$, $P = 0.01$), and PPC ($V = 122$, $P = 0.03$) at POST-VIB compared with CON-2 (Fig. 5A). An increase in cortical desynchronization in the β band was similarly found in the estimated M1 ($V = 113$, $P = 0.05$), S1 ($V = 111$, $P = 0.05$) but not in PPC ($V = 118$, $P = 0.07$) at POST-VIB compared with CON-2 (Fig. 5B).

Force Plateau [2 s; 6.5 s]

During force plateau, EMG RMS was not different across time (CON-1: $44.3 \pm 15 \mu\text{V}$, CON-2: $31.7 \pm 13 \mu\text{V}$, POST-VIB: $22.5 \pm 11.5 \mu\text{V}$, $P = 0.27$). An increase in cortical desynchronization in the α band was observed in the estimated M1 ($V = 125$, $P = 0.04$), S1 ($V = 127$, $P = 0.04$), and PPC ($V = 132$, $P =$

Figure 4. Boxplot of event-related desynchronization (ERD)/event-related synchronization (ERS) activities during movement preparation. Individual values ($n = 16$, 6 women) are presented using the median, and with the error bars representing [min, max]. ERD/ERS activities are expressed as percentage of baseline values. **A:** ERD/ERS in the estimated primary motor cortex (M1), primary somatosensory cortex (S1), and posterior parietal cortex (PPC) regions before (after 10 min of rest, CON-2) and after prolonged vibration (after 30 min of local vibration, POST-VIB) in the α band (paired Wilcoxon for each cortical area). **B:** ERD/ERS in the estimated M1, S1, and PPC regions before (CON-2) and after prolonged vibration (POST-VIB) in the β band (paired Wilcoxon for each cortical area). *Significant differences between CON-2 and POST-VIB.



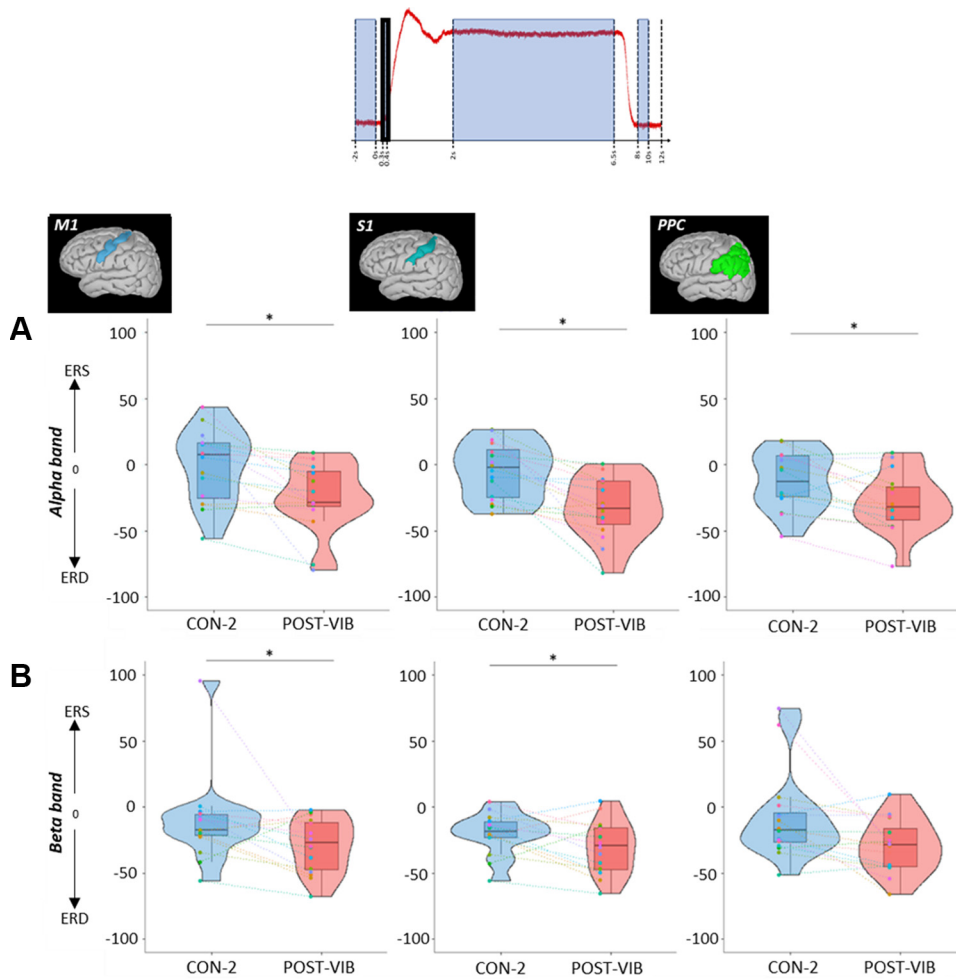


Figure 5. Boxplot of event-related desynchronization (ERD)/event-related synchronization (ERS) activities during contraction initiation with individual values ($n = 16$, 6 women) using the median, and with the error bars representing [min, max]. ERD/ERS activities are expressed as percentage of baseline values. **A:** ERD/ERS in the estimated primary motor cortex (M1), primary somatosensory cortex (S1), and posterior parietal cortex (PPC) regions for pre (after 10 min of rest, CON-2) and post vibration (after 30 min of local vibration, POST-VIB) in the α band (paired Wilcoxon for each cortical area). **B:** ERD/ERS in the estimated M1, S1, and PPC regions for pre (CON-2) and post vibration (POST-VIB) in the β band (paired Wilcoxon for each cortical area). *Significant differences between CON-2 and POST-VIB.

0.03) at POST-VIB compared with CON-2 (Fig. 6A). Cortical activity in the β band was not modulated at POST-VIB compared with CON-2 [M1 ($V = 92$, $P = 0.29$), S1 ($V = 92$, $P = 0.38$), PPC ($V = 90$, $P = 0.11$)] (Fig. 6B).

Relaxation [8 s; 10 s]

During relaxation, no changes in the α band were observed in the estimated M1 ($V = 106$, $P = 0.11$), S1 ($V = 99$, $P = 0.10$) nor PPC ($V = 79$, $P = 0.08$) at POST-VIB compared with CON-2 (Fig. 7A). Cortical activity in the β band was not modulated after vibration compared with CON-2 [M1 ($V = 58$, $P = 0.29$), S1 ($V = 57$, $P = 0.25$), PPC ($V = 54$, $P = 0.24$)] (Fig. 7B).

DISCUSSION

The aim of the present study was to investigate how prolonged LV modulates cortical activity during repeated isometric contractions of the FCR. We present three main observations. First, prolonged LV resulted in increased α ERD (i.e., desynchronization) in the estimated contralateral M1, S1 and PPC and increased β ERD within M1 and S1 during contraction preparation. Second, we observed LV-induced increased α and β ERD in the estimated M1, S1 and increased α in PPC during contraction initiation. Third, during force plateau, we observed increased α ERD in the estimated M1, S1, and PPC without any change in the β band. These main

findings provide support for the interpretation that prolonged LV can facilitate broad-band oscillations desynchronization across sensorimotor and parietal cortex areas.

From a methodological point of view, all analyses in the present study were performed at sources level instead of EEG electrodes level. Electrical signals recorded at each electrode originate from a variety of brain regions, as electrical activity propagates throughout the brain (i.e., due to volume conduction). Therefore, it is difficult to interpret the signal recorded at one electrode as solely reflecting the activity of the brain regions directly beneath. By performing analyses at the source level, we were able to reduce volume conduction issues typically associated with EEG (38) and more accurately infer the activity of our regions of interest (i.e., M1, S1, and PPC).

Oscillatory EEG activity is determined by several factors such as neurons intrinsic properties, neurotransmitters release, and strength of the interconnections between neural networks. When there is a decrease in the power of this oscillatory activity, as compared with a baseline condition, it is referred to an ERD (i.e., event-related desynchronization) (13). Indeed, α and β desynchronizations are classically found in M1-S1 around 2 s prior to voluntary movement, reflecting an activation of cortical networks in sensorimotor areas, such desynchronization being still observed during actual movement (24, 39) and related to an increase in

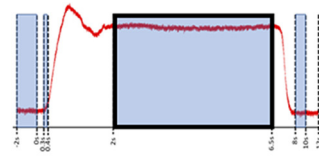
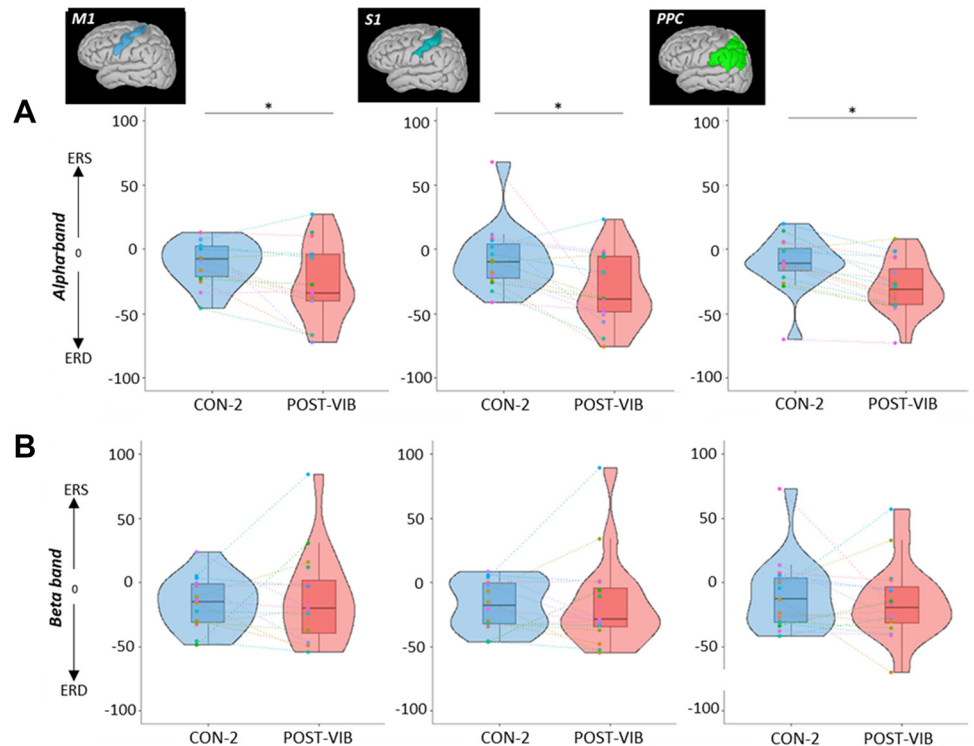


Figure 6. Boxplot of event-related desynchronization (ERD)/event-related synchronization (ERS) activities during force plateau with individual values ($n = 16$, 6 women) are presented using the median, and with the error bars representing [min, max]. ERD/ERS activities are expressed as percentage of baseline values. **A:** ERD/ERS in the estimated primary motor cortex (M1), primary somatosensory cortex (S1), and posterior parietal cortex (PPC) regions for pre (after 10 min of rest, CON-2) and post vibration (after 30 min of local vibration, POST-VIB) in the α band (paired Wilcoxon for each cortical area). **B:** ERD/ERS in the estimated M1, S1, and PPC regions for pre (CON-2) and post vibration (POST-VIB) in the β band (paired Wilcoxon for each cortical area). *Significant differences between CON-2 and POST-VIB.



cortical excitability (24). In our study, we observed an increase in α and β bands desynchronization in M1-S1 during contraction preparation and contraction initiation after prolonged LV. We also showed LV-induced increased α desynchronization in M1-S1 during force plateau. These findings suggest increased M1-S1 cortical activity after LV. The α and β frequency bands have been associated with various brain functions, reflecting distinct neurophysiological mechanisms. In particular, α rhythms might reflect the integration of somatosensory information (40, 41) whereas β rhythms might reflect motor command processing (42).

Prolonged LV has been reported to acutely decrease spinal loop excitability (1), likely as a result of motoneuron excitability depression (26, 27, 43). Those acute after-effects of prolonged LV can take up to around 20 min to recover (44). Such decreased motoneuron output after prolonged LV can lead to a decreased motor unit firing rates (45), explaining the commonly observed vibration-induced decrease in maximal force production capacities (1). In the context of our study where participants had to perform submaximal contractions, this could also have induced an increase in the cortical drive to maintain the same level of voluntary force, i.e., trying to compensate for decreased motoneuron excitability. If this holds true, this would provide a potential explanation for the increased β desynchronization in the estimated M1-S1 we showed during contraction preparation and contraction

initiation. Then when contraction reaches a plateau, the absence of greater β desynchronization at POST-VIB could suggest a similar cortical drive as compared with the baseline condition (i.e., CON-2) despite still increased somatosensory processing (i.e., increased α desynchronization in M1-S1, see below). A similar lack of β desynchronization has already been described during the maintenance of a stable force (46) such as episodes of postural maintenance (42). This result is in line with the study of Lopez et al. (23) demonstrating greater α desynchronization at the level of electrodes placed over M1-S1, but no change in the β band, during an isometric contraction performed after LV when compared with before LV exposure. This would suggest that once motor units are recruited, LV-induced decreased motoneuron excitability has less influence on the neural drive needed to perform the low-intensity isometric contractions we requested from the participants.

The increase in α desynchronization in the estimated M1-S1 during contraction preparation, contraction initiation, and force plateau after prolonged LV, could also be related directly to the increase in Ia afferent cortical projections to S1 induced by LV (47), increasing in turn cortical excitability (26, 27, 43). This may rely on topographically and functionally specific reciprocal connections between S1 and M1 (48) as suggested by an acute increased sensorimotor integration for some responders when investigated immediately after prolonged LV (28). Similarly, the increase in PPC activity observed after

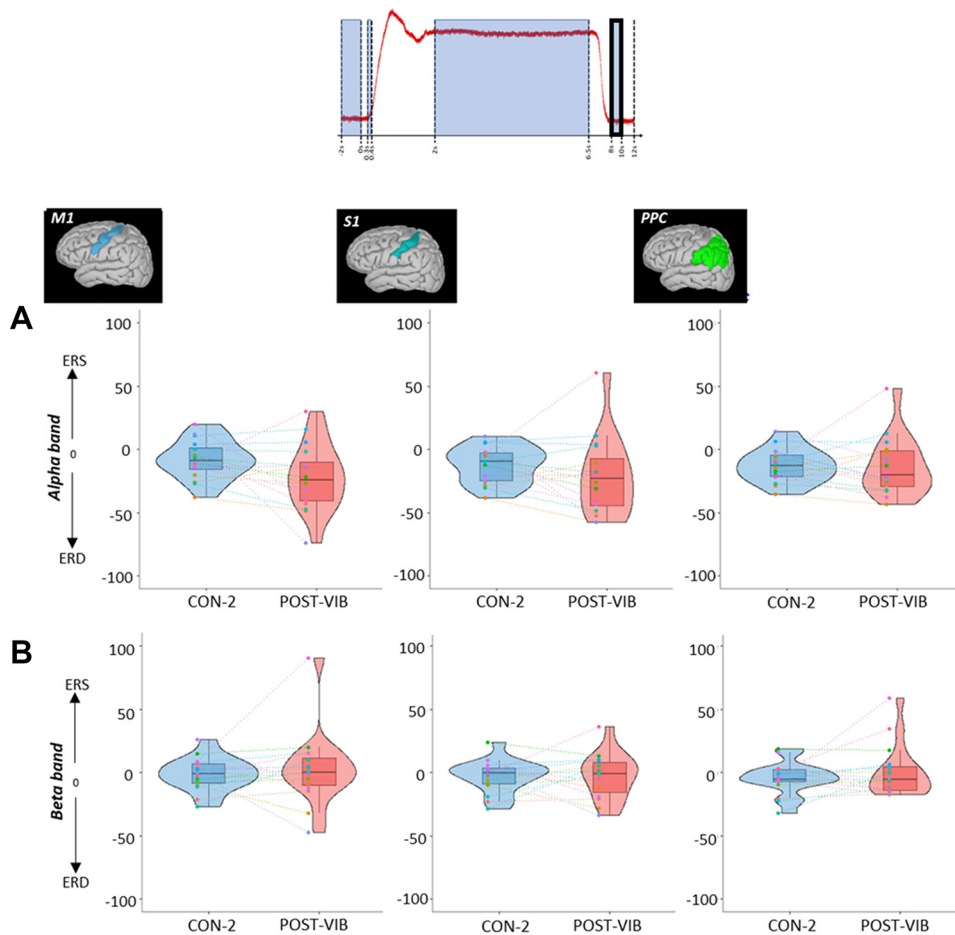


Figure 7. Boxplot of event-related desynchronization (ERD)/event-related synchronization (ERS) activities during relaxation with individual values ($n = 16$, 6 women) are presented using the median, and with the error bars representing [min, max]. ERD/ERS activities are expressed as percentage of baseline values. **A:** ERD/ERS in the estimated primary motor cortex (M1), primary somatosensory cortex (S1), and posterior parietal cortex (PPC) regions for pre (after 10 min of rest, CON-2) and post vibration (after 30 min of local vibration, POST-VIB) in the α band (paired Wilcoxon for each cortical area). **B:** ERD/ERS in the estimated M1, S1, and PPC regions for pre (CON-2) and post vibration (POST-VIB) in the β band (paired Wilcoxon for each cortical area). *Significant differences between CON-2 and POST-VIB.

prolonged LV could be consistent with the increase in Ia afferent discharge induced by LV as specific reciprocal connections exist also between S1 and PPC and between the thalamus and PPC (49). The increase in the estimated PPC activity could be then related to an increase in proprioceptive inputs integration, PPC playing an important role in sensory information processing, in particular during hand movement (50). This is further suggested by findings of improvement of proprioception acuity after proprioceptive pathways electrical stimulation (51, 52). In this way, patients suffering from lesions in the PPC have difficulty performing certain motor tasks (53) and present a marked neglect of their contralateral body side (51), mainly due to a loss of sensory inputs, including proprioceptive ones.

During relaxation, we did not observe any β rebound at CON-2 nor at POST-VIB as it could have been expected from the literature. Interestingly, previous investigation has defined β rebound as a variable parameter, varying across conditions (54). Moreover, β rebound is thought to reflect cortical inhibition of motor activity and may be directly related to the level of force produced, a higher force output resulting in greater β rebound (55). The first hypothesis explaining the lack of β rebound observed in our study could therefore be the low level of force used for our repeated isometric contractions (55). Given that β rebound reflects the integration of both visual inputs and proprioceptive feedbacks, a second hypothesis could be

the disruption of their congruence by LV (16). Moreover, we cannot rule out the possibility that the baseline period we used as a reference (i.e., from 10 s to 12 s within each sequence, i.e., corresponding to the end of the relaxation phase) could have been still during the β rebound (55), then preventing its observation in the present study.

One limitation of this study is the absence of a sham condition for all participants to highlight the LV effect, however to our knowledge there are no convincing ways to apply a sham condition when considering the effect of prolonged LV exposure. Another limitation of this study is the shorter duration of the control period compared with the LV period. Indeed, to ensure the consistency of attention and cortical activity of the participants across the experiment we preferred to reduce this period.

Conclusions

In conclusion, we investigated cortical activity modulation induced by prolonged LV during repeated isometric wrist flexions. Compared with control, an increase in contralateral source activity for M1-S1 has been demonstrated during contraction preparation, contraction initiation, and force plateau after prolonged LV. For the first time, EEG analyses were performed at sources level, confirming LV-induced greater activity in M1-S1. Moreover, we demonstrated an increase in PPC activity after prolonged LV. Altogether, these results could be related to the increase in Ia afferents discharge induced by

LV, likely inducing increased cortical drive to compensate for decreased motoneuron excitability on one hand and increasing sensorimotor integration on the other as a result of reciprocal connections between S1 and M1 and between S1 and PPC. The present results provide an interesting neurophysiological model for the explanation of the chronic LV effects described in rehabilitation. Future research should test this model after repeated LV sessions.

DATA AVAILABILITY

All data are available at <https://www.doi.org/10.17605/OSF.IO/AXYN9>.

DISCLOSURES

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

AUTHOR CONTRIBUTIONS

C.P., M.F., and T.L. conceived and designed research; C.P., M.F., and N.Z. performed experiments; C.P., M.F., A.A., and C.-E.B. analyzed data; C.P., M.F., A.A., C.-E.B., and T.L. interpreted results of experiments; C.P. and M.F. prepared figures; C.P., M.F., and T.L. drafted manuscript; C.P., M.F., N.Z., A.A., C.-E.B., and T.L. edited and revised manuscript; C.P., M.F., N.Z., A.A., C.-E.B., and T.L. approved final version of manuscript.

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