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Original Research Article

Spastic co-contraction is directly associated with altered cortical beta oscillations after stroke

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Abstract (196 words)

Objective: Spastic co-contraction is a motor-disabling form of muscle overactivity occurring after a stroke, contributing to a limitation in active movement and a certain level of motor impairment. The cortical mechanisms underlying spastic co-contraction remain to be more fully elucidated, the present study aimed to investigate the role of the cortical beta oscillations in spastic co-contraction after a stroke.

Method: We recruited fifteen post-stroke subjects and nine healthy controls. The subjects were asked to perform active elbow extensions. In the study, multimodal analysis was performed to combine the evaluation of three-dimensional elbow kinematics, the elbow muscles electromyographic activations, and the cortical oscillatory activity.

Results: The movement-related beta desynchronization was significantly decreased in stroke subjects compared to healthy participants. We found a significant correlation between the movement-related beta desynchronization and the elbow flexor activation during the active elbow extension in stroke subjects. When compared to healthy subjects, stroke subjects exhibited significant alterations in the elbow kinematics and greater muscle activation levels.

Conclusions: Cortical beta oscillation alterations may reflect an important neural mechanism underlying spastic co-contraction after a stroke.

Significance: Measuring the cortical oscillatory activity could be useful to further characterize neuromuscular plasticity induced by recovery or therapeutic interventions.

Keywords: Neuronal Plasticity; Muscle Hypertonia; Brain Injuries; Movement; Upper Extremity

Highlights:

- Spastic co-contraction is directly associated with alterations in movement kinematics and upper limb motor function.
- Altered cortical oscillatory activity may reflect an important mechanism underlying spastic co-contraction.
- Cortical beta oscillations may be a potential marker for motor recovery in stroke subjects.

1. Introduction

Spastic paresis is a disabling motor syndrome occurring after corticospinal pathway damage caused by, for example, a stroke. Considered as a neural disorder, spastic paresis associates motor paresis and antagonist overactivity (Baude et al., 2018; Gracies, 2005). One of the main features of antagonist overactivity is the spastic co-contraction which refers to an excessive degree of antagonist coactivation triggered by the volitional command of agonist muscles (Gracies, 2005). Spastic co-contraction is a disabling form of antagonist overactivity, contributing to the limitation of active movement with a deleterious impact on upper limb motor function (Chae et al., 2002; Chalard et al., 2019). Indeed, an extensive part of the literature has demonstrated the deleterious impact of spastic co-contraction on movement characteristics, including an increased duration of the movement, a lack of muscle coordination and a limitation of the active range of motion (Arene and Hidler, 2009; Chalard et al., 2019; Gross et al., 2015; Sarcher et al., 2015). Spastic co-contraction has a mainly supraspinal origin, but could be also related to spinal mechanisms: while an abnormal pattern of the supraspinal descending drive leading to a loss of motor selectivity would be the predominant mechanism underlying spastic co-contraction (Gracies et al., 1997; Schieber et al., 2009). Other mechanisms such as increased Renshaw inhibition and a reduction of reciprocal and presynaptic inhibition could contribute to the spastic co-contraction (Bhagchandani and Schindler-Ivens, 2012; Katz and Pierrot-Deseilligny, 1982; Morita et al., 2001).

Electroencephalography (EEG) is a valuable non-invasive tool to assess and quantify the cortical activity during a motor task by recording the associated oscillatory activity of the brain (Ramos-Murguialday and Birbaumer, 2015) allowing, notably, to characterize the pathological patterns of cortical activation in post-stroke subjects (Kaiser et al., 2012; Park et al., 2016). Cortical oscillations in the beta-band frequency range (13-30 Hz) over the

sensorimotor cortex are heavily involved in motor control: they are present at rest and are decreased during movement, leading to a decrease in beta power which characterizes movement-related beta desynchronization (Pfurtscheller, 2001; Pfurtscheller and Lopes da Silva, 1999). The increase in the amplitude of the movement-related beta desynchronization corresponds to increased corticospinal excitability (Pfurtscheller and Lopes da Silva, 1999; Takemi et al., 2013), and can be interpreted as an electrophysiological correlate of cortical activations involved in the generation of movement. In post-stroke subjects, movement-related beta desynchronization has been used to investigate both the relationship between beta oscillations and motor impairments, and neuroplasticity induced by rehabilitation (Rossiter et al., 2015, 2014). The authors cited above have highlighted that abnormalities in cortical oscillatory activity could be an important mechanism in motor impairment, involved in neuroplasticity following a therapeutic intervention. These studies have provided fundamental understanding of the adaptative and maladaptive neural mechanisms underlying motor behavior in post-stroke subjects enabling the provision of patient-tailored rehabilitation intervention (Koch and Hummel, 2017).

Although alterations in various physiological neural mechanisms have been suggested to be involved in the emergence of spastic co-contraction (Baude et al., 2018), to date, much remains to be understood regarding the cortical mechanisms underlying the modulation of the spastic co-contraction after corticospinal pathway damage. To fill in the gaps on this issue, the aim of the present study has been to investigate the role of the cortical beta oscillations in spastic co-contraction after a stroke. We therefore assessed beta-band cortical oscillations using movement-related beta desynchronization during active elbow extension in post-stroke subjects and healthy controls. Since a decrease in movement-related beta desynchronization has been associated with altered motor function after a stroke (Rossiter et al., 2014), we hypothesized a decrease in movement-related beta desynchronization associated with an

increase in the antagonist co-contraction in post-stroke subjects. Such a change in cortical beta oscillatory activity during a motor task could reflect the alteration of the neural mechanisms underlying the spastic co-contraction after a stroke and thus provide key new elements related to the neural mechanisms behind motor impairment in chronic stroke subjects.

2. Methods

2.1. Participants

Twenty-four adults (≥ 18 years old) voluntarily participated in this study. They were allocated into two groups: the first was composed of fifteen post-stroke participants (STROKE) (4 females; mean (± SD) age: 55 ± 11 years, mean (± SD) Fugl-Meyer Upper Extremity score: 38 ± 9, mean (± SD) Erasmus modified Nottingham Sensory Assessment: 48 ± 14) and the second comprised nine healthy controls (CO) (3 females; mean (± SD) age: 43 ± 21 years) (see Table 1 for participant demographics).

Post-stroke participants were included if they were ≥ 6 months since stroke onset and were free of any antispastic treatment for ≥ 4 months. Exclusion criteria were comprehension disorders, neurodegenerative conditions, painful paretic upper limb during movement, active elbow extension ability ≤ 20° or elbow joint contracture. The presence of spasticity or the level of motor function was neither an inclusion nor an exclusion criterion applied for post-stroke participants. All participants gave and written informed consent prior to participation. This study was approved by local Research Ethics Board (No ID-RCB: 2017-A01616-47), and was conducted in accordance with the amended Declaration of Helsinki.

2.2. Materials

Recordings were made on both sides but subsequent analyzes concerned only the non-dominant side in CO and the paretic side in STROKE.

2.2.1. Kinematics

Three-dimensional kinematic analysis was performed at 125 Hz using eight OptiTrack infrared cameras (model S250e, NaturalPoint, Corvallis, Oregon, USA). Six reflective markers were placed on both sides on the acromion, lateral epicondyle, and ulnar styloid.

2.2.2. Electromyography

Following appropriate skin preparation (Hermens et al., 2000), to reduce the impedance of skin-electrode interface below 5kΩ, surface electromyography (EMG) was acquired at 1000 Hz using Ag-AgCl bipolar electrodes in bipolar configuration with an inter-electrode distance of 20 mm using an MP150 system equipped with EMG100C amplifiers (Biopac Systems Inc., Goleta, CA, USA) with the ground electrode placed on the right mastoid. Following appropriate skin preparation (Hermens et al., 2000), the long head of the triceps brachii (TB) was taken to represent the elbow extensors, while the biceps brachii (BB), the brachioradialis (BR) and the brachialis (BA) were taken to represent elbow flexors (Staudenmann and Taube, 2015).

2.2.3. Electroencephalography

EEG was recorded at 1024 Hz using a 64 channel ActiveTwo system (BioSemi instrumentation, Amsterdam, The Netherlands), with scalp electrodes arranged according to the International 10-20 system.

Kinematics, EMG and EEG were synchronized using a common trigger controlled by the Biopac system.

2.3. Task

The task consisted of two sets of ten active elbow extension-flexion movements at a self-spontaneous speed. In a quiet room, participants were seated comfortably on a straight-backed chair with their shoulders fixed to the chair back by clavicular rings. The height of the table was adjusted to obtain an initial resting position corresponding to a shoulder flexed at 80° with internal rotation of 90°, the elbow flexed at 90° and the forearm pronated (forearm in front of the thorax). For each movement, an auditory signal indicated to the participants to start performing a full active elbow extension with the elbow lifted off the table. At the end of each elbow extension, participants had a random rest period of 8 to 15 s with their forearms resting on the table before returning to the initial position. To avoid fatigue, participants rested as needed between the two sets of ten movements.

2.4. Data analysis

Extension being a key phase for bringing the hand to a target, only elbow extension movements, the deficit of which is a frequent post-stroke complaint, were considered in this study.

2.4.1. Preprocessing

Kinematic data were low-pass filtered at 6 Hz (Cahouët et al., 2002). Raw EMG signals were 10-400 Hz band-pass filtered, full wave rectified, and smoothed at 9 Hz to obtain the linear envelopes (Chalard et al., 2019). Raw EEG signals were 3-100 Hz band-pass filtered (Tisseyre et al., 2019), and then the EEG signals were re-referenced using surface Laplacian

derivations to reduce the effect of volume conduction using a spherical spline method (Perrin et al., 1989). The filters were applied on continuous signals; all were fourth-order, zero-lag Butterworth type. The onset and offset of each elbow extension were detected with a threshold of 0.01 °/s applied on the elbow angular velocity.

2.4.2. Data processing

For each movement, the active elbow range of motion (Fig. 1.A) was computed in the horizontal plane from the Cartesian coordinates of the anatomical landmarks between the brachial and antebrachial segments. The movement duration was considered as the time in seconds required to extend the elbow at a self-spontaneous speed. The smoothness of the movement was quantified by using the normalized jerk metric defined as the number of peaks of the acceleration profile normalized by the mean angular velocity (Fig. 1.B): the greater the jerk, the less smooth was the movement (de los Reyes-Guzmán et al., 2014).

For both elbow flexors and extensors, muscle activation was computed as the ratio (expressed as a percentage) of the root mean-square value of the EMG envelope obtained during the elbow extension movement and that obtained during a maximal isometric voluntary contraction of the same muscle (Fig. 1.C & 1.D). For elbow flexor activation, the average ratio of the three corresponding muscles was used.

EEG data were segmented into epochs from -3s prior to the start and +3s after the end of the movement. A visual inspection was performed to reject epochs contaminated with artifacts. The EEG power spectrum was obtained using Morlet wavelet with the MATLAB package developed by Grinsted *et al*. and adapted by Bigot *et al* (Bigot et al., 2011; Grinsted et al., 2004). The scale resolution of the wavelet (parameter 'nvoice'), the number of scales used in the wavelet analysis (parameter 'J1') and the Morlet mother wavelet parameter (parameter 'wavenumber') were set respectively to 7, 25 and 10 to provide a satisfactory compromise

between time and resolution for the identification of oscillatory activity on the [$0.31 \cdot 10^{-2}$: 0.22 : 39.98] Hz frequency range. To cope with the inter-trial duration variability that can lead to power spectrum cancelation, a normalization procedure was used to obtain a time-frequency EEG power spectrum with time expressed as a percentage of elbow extension movement time (Fauvet et al., 2019).

The event-related EEG power were obtained by the method described by Pfurtscheller (Pfurtscheller, 2001), where a decrease / increase in power corresponds to an event-related desynchronization / synchronization respectively.

$$\text{Event-related EEG power} = 10 \times \log10\,([A/B]) \quad (1)$$

where A is the absolute power during the elbow movement (period between the movement onset and the movement offset), and B is the median of the absolute power during a baseline period ranging from -2.9 s to -0.9 s before the start of the movement.

In the current work, our analysis was focused on the movement-related beta desynchronization corresponding to the event-related EEG power on the EEG-channels over each sensorimotor cortex (Cz, FCz, CPz, C1, C2, C3, C4, C5, C6, FC1, FC2, FC3, FC4, FC5, FC6, CP1, CP2, CP3, CP4, CP5, CP6) during elbow extension in the beta-band frequency (i.e., [13-30 Hz]). To compare the subjects between them independently of the side of the brain injury (STROKE) or manual laterality (CO), we flipped EEG-data of subjects with right cerebral lesion (STROKE) or left manual laterality (CO).

2.5. Statistical analysis

To assess the potential significant differences between groups in the movement-related beta desynchronization, we identified sensorimotor EEG electrodes showing significant inter-group differences in movement-related beta desynchronization using a data-driven non-parametric cluster-based randomization test (10,000 permutations) at the topographical level

(Maris and Oostenveld, 2007; Oostenveld et al., 2011). This procedure obviates questions of multiple comparisons at multiple electrodes (type I risk of error inflation) while having a greater sensitivity than a conservative Bonferroni procedure to correct alpha-level (Maris and Oostenveld, 2007). For comparison, movement-related beta desynchronization was then considered as the mean of all the significant cluster values.

All variables showed normal distribution (Shapiro-Wilk test; $P > 0.05$) and homogeneity of variance (Levene's test; $P > 0.05$). Independent t-tests were then used to compare kinematic data (active elbow range of motion, movement duration, and smoothness), and EEG data (movement-related beta desynchronization) between STROKE and CO. A mixed analysis of variance (ANOVA) was performed with Muscle (elbow flexors vs elbow extensor) as within-subject factor and Group (STROKE vs CO) as a between-subject factor. For all analyses, the significance was set at $P < 0.05$. We expressed effect sizes of the independent t-tests in standardized terms with the Hedge's g (Lakens, 2013). The magnitude of the effect size was interpreted as small ($g > 0.2$), moderate ($g > 0.5$) or large ($g > 0.8$) (Cohen, 1977). The underlying relationship between kinematic, EMG and EEG data in STROKE was explored with multiple partial correlations allowing control of cofounding factors. We applied a threshold of 0.4 to isolate the remarkable correlations highlighting the organization of the links between variables, and generated a correlation iconography diagram (Lesty et al., 2004).

3. Results

3.1. Elbow extension kinematics are altered in STROKE

Statistical analysis revealed a significant difference with a large effect size between STROKE and CO on the active elbow range of motion ($t_{22} = 7.62$, $p < 0.001$, $g = 3.07$) (Fig 2.A), the

movement duration (t_{22} = 3.38, p = 0.003, g = 1.36) (Fig. 2.B), and the jerk (t_{22} = 2.31, p = 0.03, g = 0.92) (Fig. 2 C). In STROKE, the active elbow range of motion was decreased by 35.2 ± 2.3°, the movement duration of the elbow extension was increased by 2.8 ± 1.6 s, and the normalized jerk was increased by 2.1 ± 2.63.

3.2. EMG patterns reveal overactivity of elbow muscles in STROKE

The mixed ANOVA revealed a significant Group effect with a large effect size between two groups on elbow extensor activation and elbow flexors activation ($F_{1,22}$ = 21.8, p < 0.001, ή = 0.95) (Fig. 3), but did not reveal nor a Muscle effect ($F_{1,22}$ = 0.98, p = 0.33, ή = 0.05) nor a significant interaction between Group and Muscle ($F_{1,22}$ = 0.98, p = 0.33). In STROKE, the elbow extensor activation was increased by 21.4 ± 8%, and the elbow flexor activation was increased from 17.8 ± 7.1%.

3.3. Cortical oscillatory beta activity is altered during elbow extension in STROKE

A preliminary analysis did not reveal any differences between STROKE and CO in the absolute power during the baseline period, allowing to interpret the movement-related beta desynchronization related to the elbow extension. The time-frequency topographic plot (Fig. 4) represent the response pattern of movement-related beta desynchronization during an elbow extension for both groups. The non-parametric cluster-based permutation analysis revealed a significant difference (p < 0.01) between STROKE and CO in the movement-related beta desynchronization (Fig. 4). The topographical distribution of the cluster corresponded to the ipsilesional sensorimotor electrodes for STROKE (e.g., Cz, C1, C3, CPz, CP1, CP3, CP5, FCz, FC1, FC3). The movement-related beta desynchronization was decreased by 0.9 ± 0.3 db in STROKE compared to CO during the elbow extension movement (t_{22} = 4.66, p < 0.001, g = 1.88). It is noteworthy that a complementary analysis of

covariance on movement-related beta desynchronization with Group (STROKE vs CO) as between factor and Age as a covariable revealed a Group effect on the movement-related beta desynchronization while controlling for age ($F_{1,22} = 5.02$, $p < 0.05$). The non-parametric cluster-based permutation was also performed on the movement-related alpha desynchronization and did not reveal any differences between STROKE and CO (see supplemental material).

3.4. Remarkable correlations between EEG, EMG and kinematic data in STROKE

The correlation iconography diagram representing significant partial correlations is presented in Fig. 5. Movement-related beta desynchronization correlated positively with elbow flexor activation, meaning that greater elbow flexor activation was associated with less movement-related beta desynchronization. Elbow flexor activation correlated positively with elbow extensor activation, meaning that an increase in the elbow flexor activation was associated with an increase in the elbow extensor activation. Elbow flexor activation correlated negatively with the active elbow range of motion, meaning that an increase in elbow flexor activity during the movement was associated with a reduced active elbow range of motion. Active elbow range of motion was correlated negatively with the normalized jerk, meaning that a reduced active elbow range of motion was associated with greater acceleration peaks. Finally, the normalized jerk was positively correlated with the movement duration - the more the movement was jerky the longer it took to extend the elbow.

4. Discussion

We aimed to investigate the role of the cortical beta oscillations in the excessive increase of antagonist co-contraction in post-stroke subjects during active elbow extension. To the best of

our knowledge, this study is the first to measure cortical activity directly during an active elbow extension and to examine the cortical mechanisms underlying the spastic co-contraction in post-stroke subjects. We reported a new key finding - a strong association between movement-related beta desynchronization and the elbow flexor activation during elbow extension, providing new insights into neural mechanisms underlying contributing to the motor impairment after stroke.

In line with our hypotheses, we showed significant differences in kinematic variables between post-stroke and healthy participants. In post-stroke subjects, the active range of motion was limited, slower and less smooth. These results are consistent with the literature that has reported the impairment of the motor behavior through kinematic analyses after a stroke (Murphy et al., 2011; Murphy and Häger, 2015). Our results showed an association between the jerk and the active elbow range of motion where post-stroke subjects with a jerky movement will tend to have a limited active elbow range of motion, reflecting the impairment of various characteristics of the movement. Our results showed an association between movement duration and movement smoothness, which supports the idea of using movement duration as an easy-to-use clinical marker relating the movement characteristics (Murphy et al., 2011).

Our findings also highlighted a pathological pattern of muscle overactivity on both elbow flexors and extensors, suggesting that a motor task as simple as extending the elbow represents a considerable effort for post-stroke subjects. As previously shown (Chalard et al., 2019), we found a negative association between the elbow flexor activation and the active elbow range of motion confirming that excessive antagonist co-contraction is one of the mechanisms contributing to motor impairment (Baude et al., 2018; Chalard et al., 2019; Sarcher et al., 2015). Excessive antagonist co-contraction produces an active resistance by generating an opposite force at the elbow, which limits the active extension movement

(Gracies, 2015). Furthermore, we showed a link between elbow flexor and extensor activations in post-stroke subjects, highlighting that an excessive antagonist co-contraction could contribute to the concomitant increase of elbow extensor activation, in order to counteract the active resistance generated by the elbow flexors. Taken together, these results strongly support and confirm that spastic co-contraction is a disabling form of muscle overactivity generating abnormal patterns of motor behavior leading to alterations in movement characteristics and upper limb motor function (Baude et al., 2018; Chalard et al., 2019; Gracies, 2015).

After eliminating the cofounding factors in the partial correlations, our data do not directly link the modulation of the jerk or the movement duration with elbow muscles or cortical activations. It is likely that the soft tissue plastic rearrangements, muscle contracture or other modalities of muscle overactivity (i.e. spasticity and spastic dystonia) occurring in the spastic paresis syndrome, could have a detrimental impact on the different components of the elbow kinematics (Gracies, 2005). Moreover, beta oscillations in the ipsilesional sensorimotor cortex of post-stroke subjects emerged as the strongest feature of our data, reflecting significant differences between STROKE and CO in cortical beta oscillatory activity spread across the sensorimotor cortex. In line with other studies (Gerloff, 2006; Rossiter et al., 2014), our results indicated a decrease in movement-related beta desynchronization in post-stroke subjects during the movement. As suggested by Rossiter *et al.* (2014), post-stroke subjects may lack the ability to modulate their motor cortex during the movement, reflecting an impaired ability to generate volitional descending motor signals. Cortical oscillatory activity provides a direct measure of neuronal activity that can help us to understand the link between neuroplasticity and post-stroke motor impairment. Although the role of the movement-related beta desynchronization in post-stroke and healthy subjects is documented, the cortical neural signature underlying the control of the antagonist co-contraction in post-stroke subjects

remains unexplored. We found an association between the decrease of the movement-related beta desynchronization and the increase of elbow flexor activation (i.e. the antagonist co-contraction). In healthy subjects, some studies have suggested a specific encoding for the antagonist muscles through the cortical oscillations in the beta frequency band which could play a critical role in the modulation of antagonist co-activation (Dal Maso et al., 2017, 2012; Takemi et al., 2018). Even if correlations do not provide proof of causal relationship, our results highlight that an alteration of beta cortical oscillations represents a key mechanism associated to spastic co-contraction in post-stroke situations. Alterations in various physiological mechanisms, such as a reduction in inhibitory mechanisms (reciprocal inhibition, reduction of presynaptic inhibition), have been suggested as being involved in the emergence of spastic co-contraction (Baude et al., 2018). We can hypothesize that a decrease in the movement-related beta desynchronization could reflect the reduced cortical influences on spinal mechanisms regulating the antagonist co-activation. Considering that the modulation of cortical oscillations can reflect the balance between inhibitory and excitatory processes (Hall et al., 2011; Muthukumaraswamy et al., 2013), we can suppose that the observed decrease in cortical oscillatory activity can be due to a disturbance between inhibitory and excitatory processes. The movement-related desynchronization may also reflect the conjunction of multiple factors associated with sensory aspects of motor control, notably the potential deficit in sensory feedback during movement (Müller et al., 2003). In chronic stroke situations, it has been shown that abnormal inter-hemispheric interactions occur during movement generation and influence functional recovery (Murase et al., 2004). Although we have not investigated the potential interaction between excessive inter-hemispheric inhibition and spastic co-contraction, an inter-hemispheric inhibition may be partly involved in the decrease in the cortical oscillatory activity in the ipsilesional hemisphere (Takechi et al., 2014) and, thus, contribute to the emergence of spastic co-contraction.

5. Limitations

In this study we focused on the cortical mechanisms underlying spastic co-contraction. One limitation of this study is the variability in the subject groups with regard to the time since the stroke and localizing the lesion and that little is known about the influence of lesion location on spastic co-contraction and on the cortical oscillatory activity. Taken together, and due to the small sample of the study and the absence of a matched control group, any generalization of these results should be treated with caution.

6. Conclusion

These results suggest that cortical beta oscillation alterations may reflect an important neural mechanism underlying with spastic co-contraction of elbow muscles during movement following a stroke. The present findings extend the current concept of cortical oscillations toward an underlying neural mechanism associated with the motor impairment after a stroke. Movement-related beta desynchronization is increasingly being used to investigate the functional role of cortical oscillations in motor function, making cortical beta oscillations a potential marker for motor recovery in stroke subjects. Our study pleads for an EEG-based assessment of cortical oscillatory activity as a target to understand and characterize neuromuscular plasticity induced by therapeutic interventions.

Conflict of Interest Statement

Alexandre Chalard is employee of Ipsen Innovation within the framework of a CIFRE PhD fellowship. The other authors in this study declare that there is no conflict of interest.

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Figure captions

Figure 1. Time series representation of (**A.**) the active elbow extension, (**B.**) the acceleration profile, (**C.**) the elbow flexors activation, and (**D.**) extensor activation for a typical subject in post-stroke (STROKE) (red) and in healthy controls CO (blue). The shaded area corresponds to the standard error.

Figure 2. Bar charts representing (**A.**) the active elbow range of motion, (**B.**) the movement duration, and (**C.**) the normalized jerk for post-stroke (STROKE) (light gray) and healthy controls CO (dark gray). *, **, *** indicates a significant difference between groups ($p < 0.05$; $p < 0.01$; $p < 0.001$, respectively); vertical bars represent standard error of the mean.

Figure 3. Bar charts representing the elbow extensor activation (light gray) and elbow flexor activation (dark gray) for post-stroke (STROKE) and healthy controls (CO). *** indicates significant difference between groups ($p < 0.001$); vertical bars represent standard error of the mean.

Figure 4. Bar chart and scalp topography of movement-related beta desynchronization during the elbow extension for post-stroke (STROKE) and healthy controls (CO). White dots on the scalp topography represent the significant cluster of difference between groups obtained by the cluster-based permutation analysis. *** indicates a significant difference between groups ($p < 0.001$); vertical bars represent standard error of the mean.

Figure 5. Correlation iconography diagram representing only the significant partial correlations between movement-related beta desynchronization, elbow flexor and extensor

activations and elbow extension parameters for post-stroke (STROKE). A link between two nodes represents a significant correlation ($p < 0.05$); brackets represent the 95th confidence interval for the correlation coefficient.

Figure

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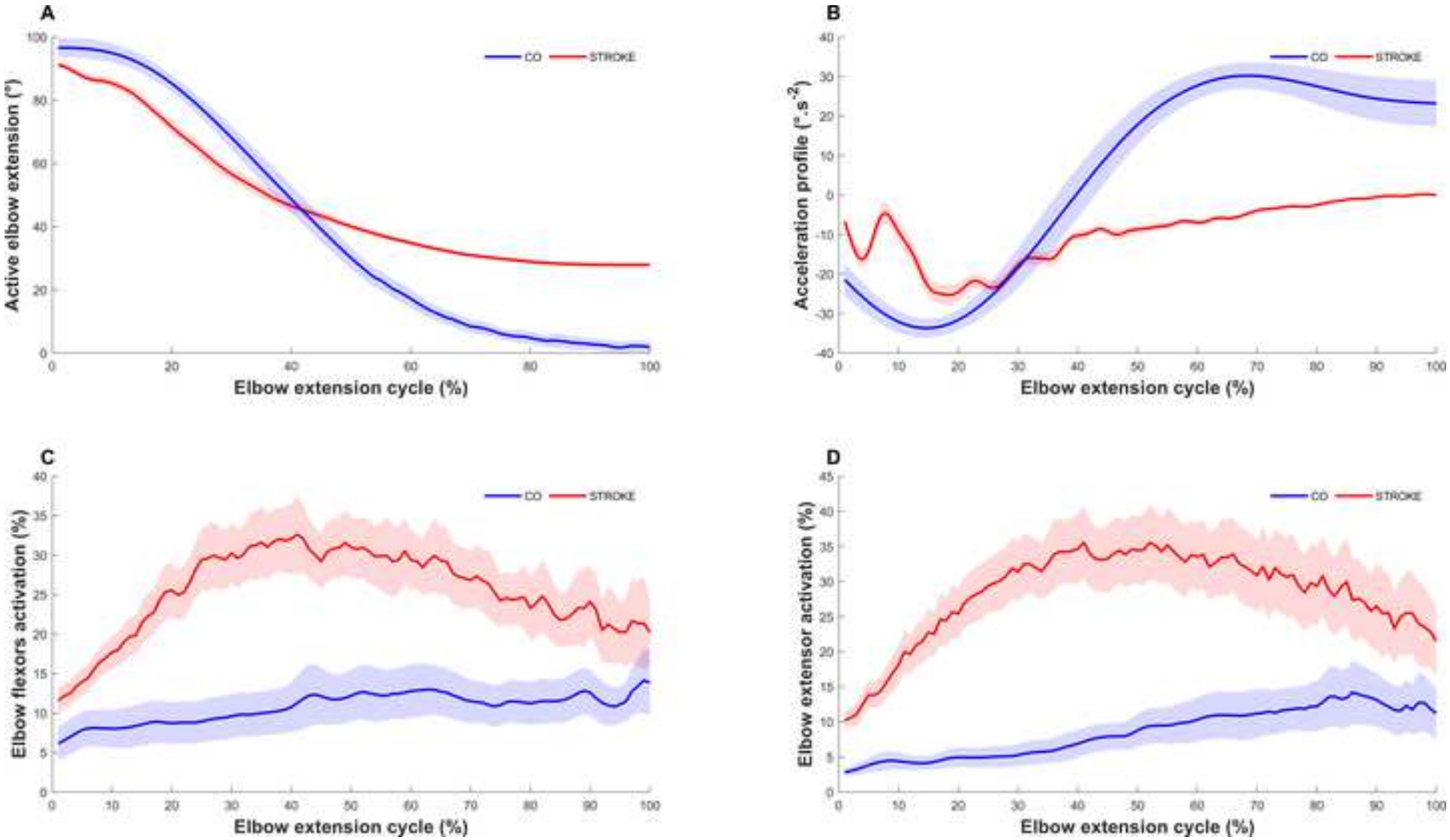

Figure

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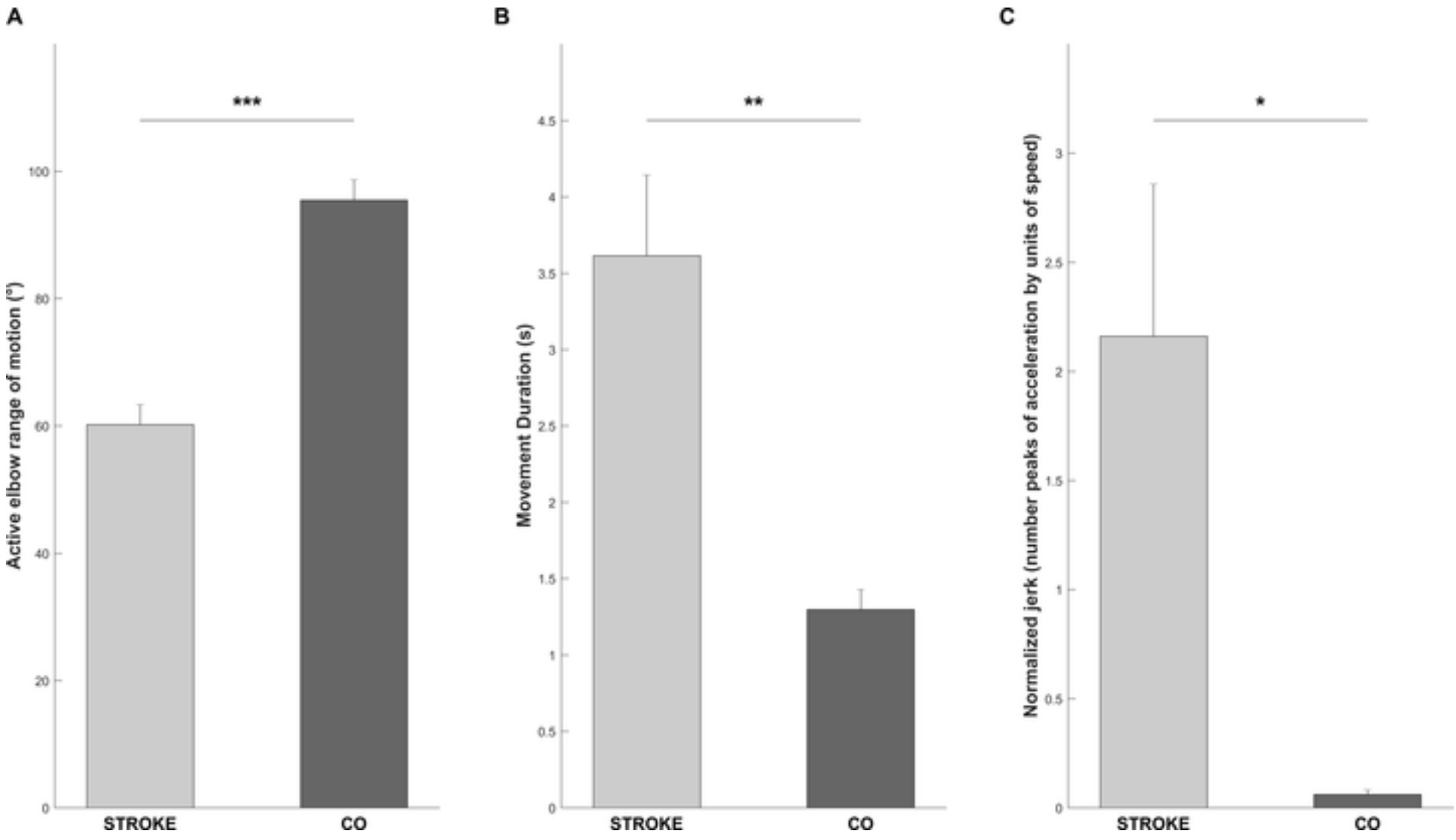

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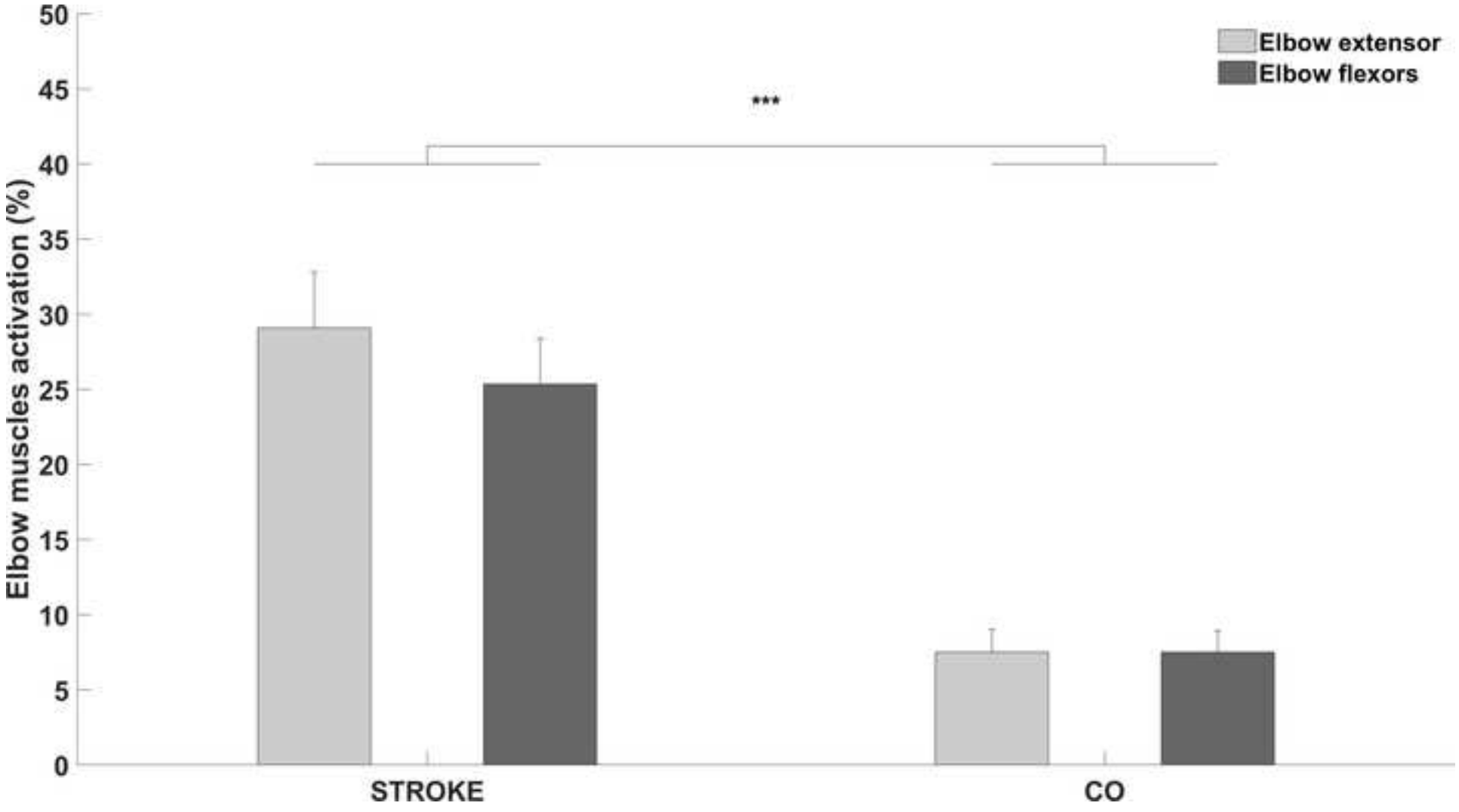

Figure

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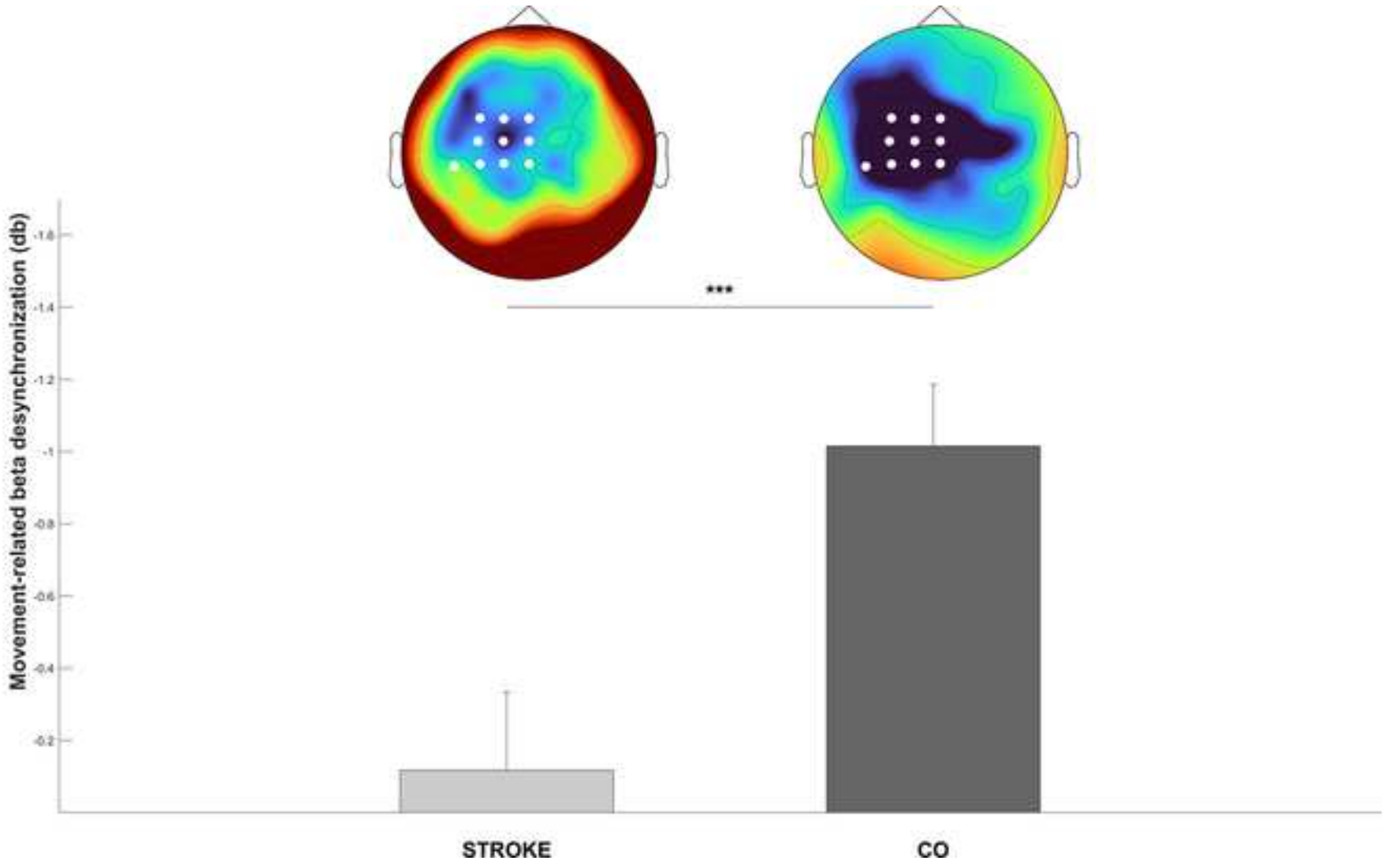
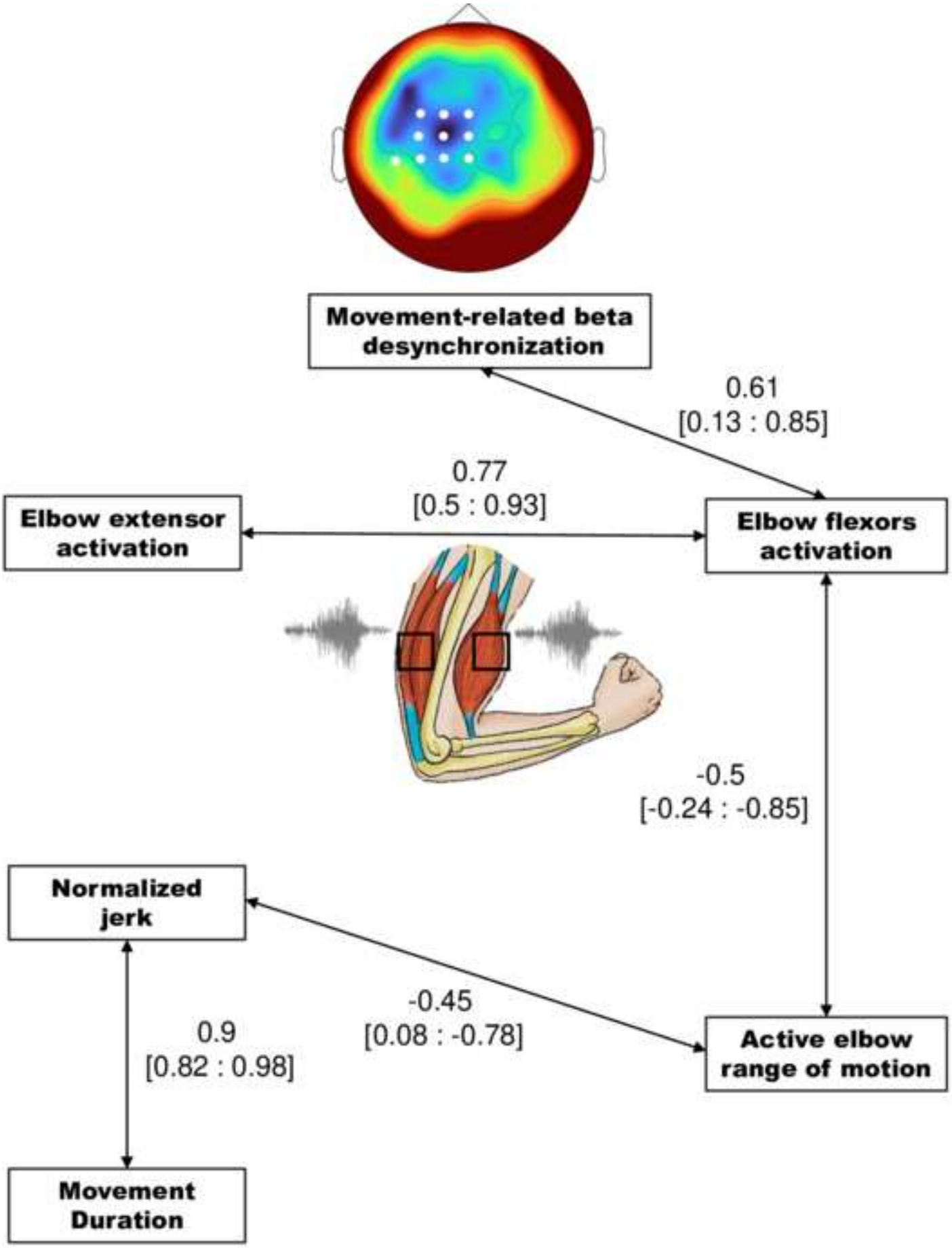



Movement-related beta desynchronization
0.61
[0.13 : 0.85]
0.77
[0.5 : 0.93]
Elbow extensor activation
Elbow flexors activation
-0.5
[-0.24 : -0.85]
Normalized jerk
-0.45
[0.08 : -0.78]
0.9
[0.82 : 0.98]
Active elbow range of motion
Movement Duration

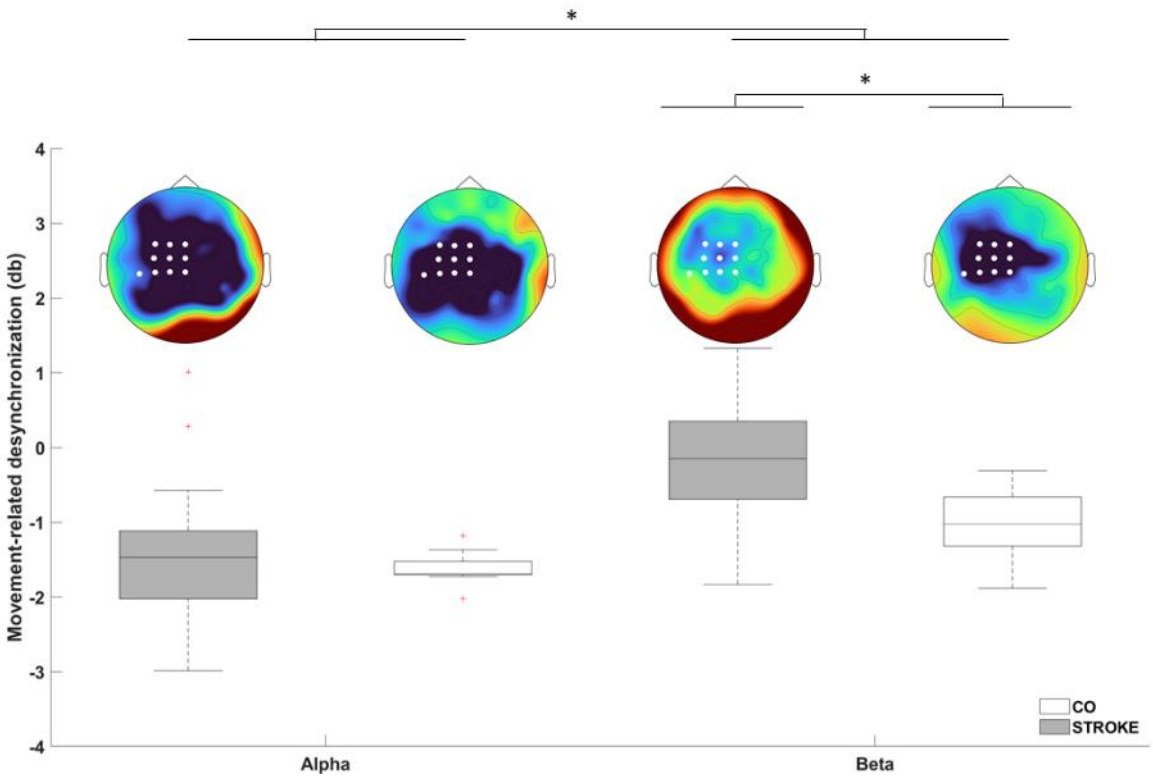
Table

Participants	Age	Sex	Time Since Stroke (months)	Stroke type, sub-type	Stroke side, location	Fugl-Meyer Upper Extremity (/66)	Erasmus modified Nottingham Sensory Assessment (/64)	Active elbow extension range of motion (°)
CONTROL (n = 9)	43 ± 21	6 M / 3 F	-	-	-	-	-	96 ± 8
STROKE 1	61	M	50	Hemorrhagic, microangiopathy	Right, basal ganglia and corona radiata	38	34	56
STROKE 2	59	M	17	Ischemic, MCA (atheroma)	Right, cortical and subcortical territories of MCA	46	27	52
STROKE 3	69	F	19	Ischemic, vertebral artery (atheroma)	Right, Pons	44	60	74
STROKE 4	65	M	74	Ischemic, carotid artery dissection	Right, cortical and subcortical territories of MCA	32	50	58
STROKE 5	50	M	29	Hemorrhagic, microangiopathy	Left basal ganglia and internal capsule	42	60	75
STROKE 6	51	F	12	Ischemic (with hemorrhagic transformation), MCA (cardio-embolic)	Left, subcortical territory of MCA	24	61	74
STROKE 7	57	M	14	Ischemic, microangiopathy	Left, posterior limb of the internal capsule	45	56	40
STROKE 8	75	M	26	Ischemic (with hemorrhagic transformation), MCA (cardio-embolic)	Left, cortical and subcortical territories of MCA	26	33	61
STROKE 9	46	M	8	Ischemic (with hemorrhagic transformation), carotid artery dissection	Left, cortical and subcortical territories of MCA	53	54	56
STROKE 10	65	M	116	Ischemic, MCA (atheroma)	Right, cortical and subcortical territories of MCA	30	62	52
STROKE 11	49	M	13	Ischemic, microangiopathy	Predominant right, Pons (paramedian)	53	63	60
STROKE 12	33	F	12	Ischemic, vertebral artery dissection	Predominant left, Pons and middle cerebellar peduncles	45	48	80
STROKE 13	33	F	12	Ischemic, carotid artery dissection	Left, subcortical territories of MCA	25	62	56
STROKE 14	57	M	18	Ischemic, MCA (atheroma)	Right, cortical and subcortical territories of MCA	41	26	66
STROKE 15	56	M	22	Ischemic, MCA (atheroma)	Right, cortical and subcortical territories of MCA	29	30	40

Table I. Participants' demographics (mean ± SD).

Abbreviations: MCA, middle cerebral artery. Cortical territory of MCA: lateral surface of the hemisphere, except for the medial part of the frontal and the parietal lobe (anterior cerebral artery), and the inferior part of the temporal lobe (posterior cerebral artery). Subcortical territory of MCA: Basal ganglia (globus pallidus, caudate, putamen), internal capsule and corona radiata.

Supplemental material: Movement-related alpha desynchronization.



Supplemental Figure 1: Boxplot and topographic representation of movement-related alpha and beta desynchronization over the ipsilesional cortex for STROKE (gray) and CO (white). White circles represent electrodes of interest (Cz, C1, C3, CPz, CP1, CP3, CP5, FCz, FC1, FC3). We performed a two-way mixed ANOVA with Frequency (Alpha vs Beta) as a within-subject factor and Group (STROKE vs CO) as a between-subject factor, on the movement-related desynchronization. The ANOVA revealed a Frequency effect ($F_{1,22}$ = 46.4, p < 0.01) and a Group x Frequency interaction ($F_{1,22}$ = 6.2, p < 0.05), post-hoc analyses showed only a significant difference for STROKE vs CO for movement-related beta desynchronization (t_{22} = 2.9, p < 0.01). The analysis did not reveal nor a Group effect ($F_{1,22}$ = 3.1, p = 0.1) nor a significant difference for STROKE vs CO for movement-related alpha desynchronization (t_{22} = 0.53, p = 0.6).