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Impact of the EMG normalization method on muscle activation and the antagonist-agonist co-contraction index during active elbow extension: Practical implications for post-stroke subjects

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ABSTRACT

Electromyographic (EMG) raw signals are sensitive to intrinsic and extrinsic factors. Consequently, EMG normalization is required to draw proper interpretations of standardized data. Specific recommendations are needed regarding a relevant EMG normalization method for participants who show atypical EMG patterns, such as post-stroke subjects. This study compared three EMG normalization methods ("isometric MVC", "isokinetic MVC", "isokinetic MVC kinematic-related") on muscle activations and the antagonist-agonist co-contraction index. Fifteen post-stroke subjects and fifteen healthy controls performed active elbow extensions, followed by isometric and isokinetic maximum voluntary contractions (MVC). Muscle activations were obtained by normalizing EMG envelopes during active movement using a reference value determined for each EMG normalization method. The results showed no significant difference between the three EMG normalization methods in post-stroke subjects on muscle activation and the antagonist-agonist co-contraction index. We highlighted that the antagonist-agonist co-contraction index could underestimate the antagonist co-contraction in the presence of atypical EMG patterns. Based on its practicality and feasibility, we recommend the use of isometric MVC as a relevant procedure for EMG normalization in post-stroke subjects. We suggest combined analysis of the antagonist-agonist co-contraction index and agonist and antagonist activations to properly investigate antagonist co-contraction in the presence of atypical EMG patterns during movement.

1. Introduction

Raw electromyographic (EMG) signals are sensitive to both intrinsic (such as anatomical and physiological characteristics) and extrinsic (such as electrode configuration or placement, skin preparation) factors (Burden, 2010). EMG normalization, which refers to the conversion of the EMG signal to a relative scale by a reference value, is thus a key step in enabling (i) proper interpretation of standardized data, and (ii) comparison between muscles or individuals (Halaki and Ginn, 2012). The method used for EMG normalization influences the shape of EMG patterns, which makes its choice critical to accurately present the muscle activation for a given muscle and to permit correct interpretation of the amplitude and temporal variations of EMG signal intensity.

The most common method is to normalize the EMG envelope during a task under investigation to the maximum peak value obtained during isometric maximum voluntary contraction (MVC) (Yang and Winter, 1984). Depending on the task of interest, it has been reported that EMG signals normalized using such "isometric MVC" normalization may reach values above 100% (Jobe et al., 1984). This suggests this method may be not accurate enough to reveal the maximum activity level, and may be inappropriate for dynamic movement (Mirka, 1991). To address this issue, the maximum EMG value obtained during isokinetic MVC can be used as the reference EMG value in order to normalize the EMG envelope under dynamic conditions (Fernández-Peña et al., 2009). Using such "isokinetic MVC" normalization, the reference EMG value is calculated for a comparable joint range-of-motion at a similar velocity to the task under investigation. It is, however, not always possible to realize an isokinetic protocol due to experimental limitations (El

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Mhandi and Bethoux, 2013). An alternative method for EMG normalization is to use the maximum EMG value reached during the task under investigation as the reference value (Yang and Winter, 1984). However, this method tends to reduce the variability between individuals since it makes the reference value relative to the task and not to the maximum capacity of the muscle (Halaki and Ginn, 2012). Although this method may be suitable for comparing EMG patterns over time, it cannot enable consistent and reliable comparison of activity between muscles, tasks and individuals.

In healthy participants, recent literature reviews have highlighted that "isometric MVC" normalization produces similar results to "isokinetic MVC" normalization (Burden, 2010; Halaki and Ginn, 2012). A recommendation has been made stating that "isometric MVC" normalization is sufficient to provide normalized EMG values with enough confidence to assess muscle activity during active movement for healthy subjects (Burden, 2010).

It is well established that clinical populations such as post-stroke subjects present neuromuscular alterations during movement reflected by abnormal EMG muscle activation patterns (Ma et al., 2017). Among them, the spastic co-contraction corresponds to an excessive activity of antagonist muscles during the active movement (Banks et al., 2017; Gracies, 2005; Ma et al., 2017) which seem to be overstated with muscle lengthening due to alteration of force-length and force-velocity relationships after brain injury (Gracies, 2005; Sarcher et al., 2017). The choice of a suitable method of EMG normalization appears especially relevant for post-stroke subjects who present such atypical patterns of EMG activity.

It has been shown that "isometric MVC" normalization can yield unpredictable results in subjects with altered neuromuscular control (Ettinger et al., 2016). While EMG analysis is increasingly used in both the upper limb assessment and rehabilitation of post-stroke subjects during active movements (Klein et al., 2018; Zarantonello et al., 2017), there is still a substantial lack of data supporting any recommendation for an EMG normalization method in participants who exhibit an atypical EMG pattern. Apart from EMG-based assessment of muscle activation, the issue of EMG normalization is also of major relevance in the clinical context to quantify an EMG-based antagonist-agonist co-contraction index, which is likely to reflect the level of spastic co-contraction. Previous work highlighted a relationship between the level of spastic co-contraction and the range-of-motion restriction (Chalard et al., 2019; Sarcher et al., 2015), highlighting the importance of quantifying the antagonist-agonist co-contraction index in order to improve the motor function of such patients.

To address the relevance given to different EMG normalization methods under dynamic conditions in participants with atypical muscle activations patterns, the present study assessed the impact of three methods for EMG normalization on muscle activation and antagonist-agonist co-contraction in post-stroke subjects during active elbow extension. In this study we focused on three EMG normalization methods corresponding to "isometric MVC" normalization, "isokinetic MVC" normalization and "isokinetic MVC kinematic-related" normalization. We hypothesized that "isokinetic MVC" normalization and "isokinetic MVC kinematic-related" normalization would provide more accurate EMG-normalized measurements than "isometric MVC" normalization by considering muscle dynamics during active elbow extension in post-stroke subjects.

2. Methods

2.1. Participants

Thirty adults (≥18 years) allocated into two groups participated in this study: the first group comprised fifteen post-stroke participants (HEMI); the second comprised fifteen healthy controls (CO). The participants demographics are presented in Table 1. For HEMI, spasticity was assessed using Tardieu scale and motor impairment with the Fugl-

Table 1
Participant demographics (median ± interquartile range).

Participants	Sex	Age (y)	Mass (kg)	Brain injury side	Disease course (mo)	FMA-UE (/66)	Spasticity[1]		Isometric Torque (N·m/kg)		Isokinetic Torque (N·m/kg)		Isokinetic Speed (deg·s^{-1})	
							Elbow flexors	Elbow extensors	Extension*	Flexion*	Extension	Flexion	Extension	Flexion
Control (n = 15)	9 Male 6 Female	42 ± 20	67 ± 19	–	–	–	–	–	0.52 ± 0.34	0.74 ± 0.38	–	–	–	–
HEMI (n = 15)	13 Male 2 Female	55 ± 11	75 ± 14	8 Right 7 Left	20 ± 20	40 ± 12	2 ± 0.5	1 ± 2	0.31 ± 0.07	0.34 ± 0.03	0.24 ± 0.06	0.39 ± 0.16	30 ± 8.5	35 ± 15

Abbreviations: FMA-UE, Fugl-Meyer Assessment score for Upper Extremity.
* Indicates a significant difference between HEMI and CO ($p < 0.05$).
[1] Spasticity of elbow flexors and extensors was assessed using the Tardieu scale.

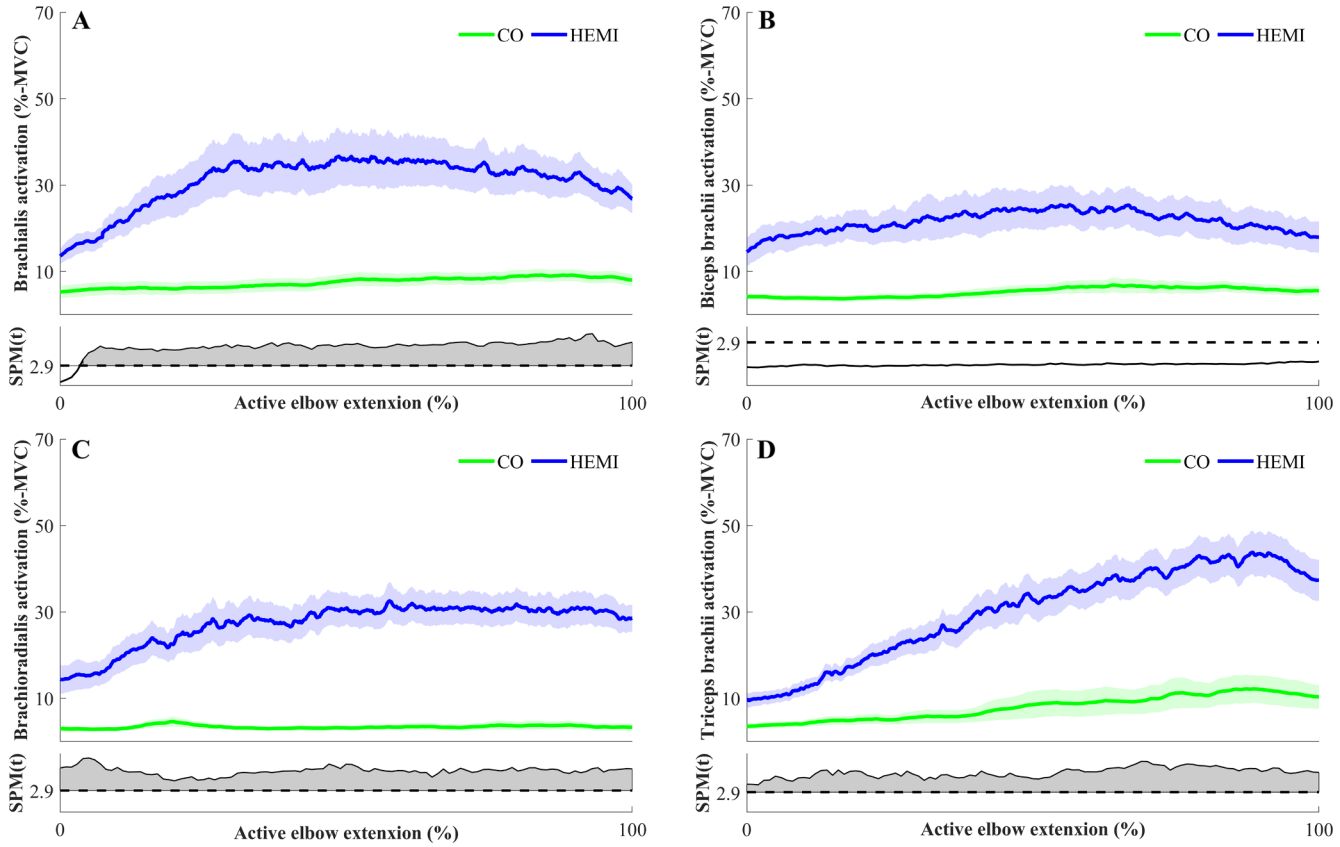

Fig. 1. Muscle activations normalized by M_{Isom} during active elbow extension for: A. brachialis (BA), B. biceps brachii (BB), C. brachioradialis (BR), and D. triceps brachii (TB). The upper panel represents the muscle activation and standard error for CO (green) and HEMI (blue). The lower panel represents the SPM(t) test statistic continuum, the dashed line corresponding to the significance level threshold. Whenever the test statistic continuum SPM(t) exceeds the threshold ($p < 0.05$), significance is reached and the p-values are reported by shaded gray areas. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Meyer Upper Extremity Assessment. Post-stroke participants were included if they were ≥6 months since stroke onset and were free of any anti-spastic treatment for ≥4 months. Potential participants with comprehension disorders, neurodegenerative conditions, painful paretic upper limbs during movement or an active elbow extension ability ≤20° were excluded. All participants gave informed consent prior to participation. This study was approved by the local Research Ethics Board (No ID-RCB: 2017-A01616-47).

2.2. Experimental design

The experimental protocol consisted of two consecutive steps. In the first step, three-dimensional kinematics and EMG data were simultaneously collected during repeated active elbow extension movements at spontaneous speed. The second step was to perform isometric and isokinetic maximum voluntary contractions (MVC) during which EMG measurements were taken, together with joint angle, angular velocity, and torque provided by a calibrated dynamometer.

2.3. Materials

2.3.1. Kinematics

The three-dimensional kinematics of upper limbs were collected at 125 Hz using eight Optitrack infrared cameras (model S250e, software Motive:Tracker 1.8.0; NaturalPoint, Corvallis, Oregon, USA). Twelve reflective markers were placed on the following positions: the spinous process of C7, the sternal notch, both sides of the acromion, the lateral epicondyle, both the ulnar and radial styloid, and the head of the second metacarpus.

2.3.2. Electromyography

Surface EMG was acquired at 1 kHz with a MP150 system (Biopac Systems Inc., Goleta, CA, USA) with the ground electrode placed on mastoid process. After suitable skin preparation, rectangular self-adhesive bipolar pairs of disposable Ag/AgCl surface electrodes with a 10 mm recording diameter were placed with a 10 mm inter-electrode distance (Afsharipour et al., 2019). The long head of the triceps brachii (TB) was taken to represent the elbow extensors; the biceps brachii (BB), the brachioradialis (BR) and the brachialis (BA) were taken to represent the elbow flexors (Staudenmann and Taube, 2015). A verification procedure was performed to limit crosstalk among biceps brachii, brachialis and triceps brachii.

As was done in Banks et al. (2017) during gait experiments, the agonist or antagonist role assigned to these muscles was fixed to their biomechanical function during elbow extension.

2.3.3. Dynamometry

Elbow joint angle, angular velocity, and net torque were recorded at 1 kHz using an isokinetic dynamometer (Con-Trex MJ; CMV AG, Dubendorf, Switzerland).

For each experimental step, data synchronization was achieved using a common timing signal controlled by the Biopac system.

2.4. Procedure

2.4.1. Active elbow extension movements

Participants were seated on an upright chair with shoulders fixed to the chair back by clavicular rings. The height of the table was adjusted to obtain an initial resting position corresponding to shoulder flexion of

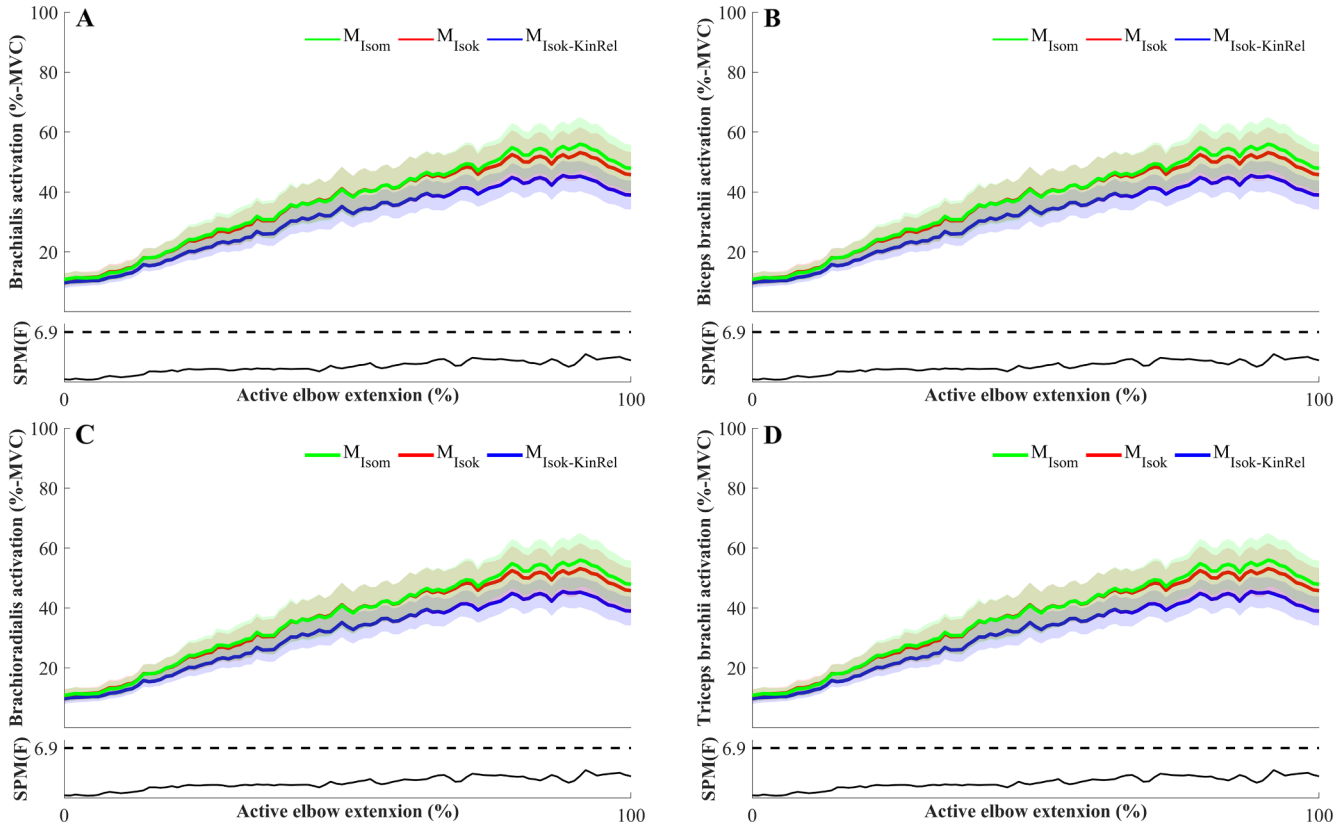

Fig. 2. Muscle activations during active elbow extension for HEMI for: A. brachialis (BA), B. biceps brachii (BB), C. brachioradialis (BR), and D. triceps brachii (TB). The upper panel represents the muscle activation and standard error normalized by M_{Isom} (green), M_{Isok} (red) and $M_{Isok\text{-}KinRel}$ (blue). The lower panel represents the SPM(F) test statistic continuum, the dashed line corresponding to the significance level threshold. Whenever the test statistic continuum SPM(F) exceeds the threshold ($p < 0.05$), significance is reached and the p-values are reported by shaded gray areas. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

80° with internal rotation of 90°, the elbow flexed at 90° and the forearm in a neutral position. Participants were asked to perform two sets of ten active elbow extension movements at spontaneous speed. For each movement, an auditory signal requested the participants to perform a full active elbow extension with the elbow off the table. At the end of elbow extension, participants had a 10-second rest with their forearm on the table. To avoid fatigue, participants were allowed to rest for as long as needed between the two sets.

2.4.2. Isometric and isokinetic MVC

Participants were seated on the dynamometer chair with their upper body strapped, the glenohumeral joint positioned at 90° flexed and internally rotated, and the forearm positioned in a neutral position. During isometric MVC, participants performed three 5-second maximum contractions in both flexion and extension directions with the elbow flexed at the middle of the angular extension movement range recorded during elbow extension-flexion movements. During isokinetic MVC, participants performed three maximum contractions in both concentric and eccentric modes. During each isokinetic contraction, the elbow angular range-of-motion and velocity matched the average corresponding values observed in each mode during elbow extension movements (see Table 1). Participants had a 1-min rest between contractions and a 3-min rest between directions or modes. No participant reported any pain or discomfort that would interfere with the production of force during MVC.

2.5. Data processing

2.5.1. 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 (Amarantini and Bru, 2015). Net torque was low-pass filtered at 15 Hz (Bassan et al., 2015). All filters were fourth-order, zero-lag Butterworth type.

2.5.2. Active elbow extension

Kinematic data were obtained from the filtered Cartesian coordinates of the anatomical markers. The onset and offset of each active elbow extension were detected with a threshold of 0.01°/S applied on the elbow angular velocity.

2.5.3. Muscle activations

At each time point of active elbow extension, muscle activation was computed by normalizing the EMG signal of each muscle to its EMG reference using the following three normalization methods:

- isometric MVC [M_{Isom}] EMG normalization: The preprocessed EMG signal was normalized to its EMG reference value obtained during isometric MVC. The M_{Isom} EMG reference value was calculated as the root mean square (RMS) value of the EMG linear envelope on the 2-second window where the elbow net torque was highest.
- isokinetic MVC [M_{Isok}] EMG normalization: The preprocessed EMG signal was normalized to its EMG reference value obtained during isokinetic MVC. The M_{Isok} EMG reference value was calculated as the RMS value of the EMG linear envelope on a centered 100 ms window when the participant reached the middle of the active elbow extension range of motion. The M_{Isok} EMG reference value was computed using data collected in concentric mode for elbow extensors (TB) and in eccentric mode for elbow flexors (BB, BR, BA).
- isokinetic MVC kinematic-related [$M_{Isok\text{-}KinRel}$] EMG normalization:

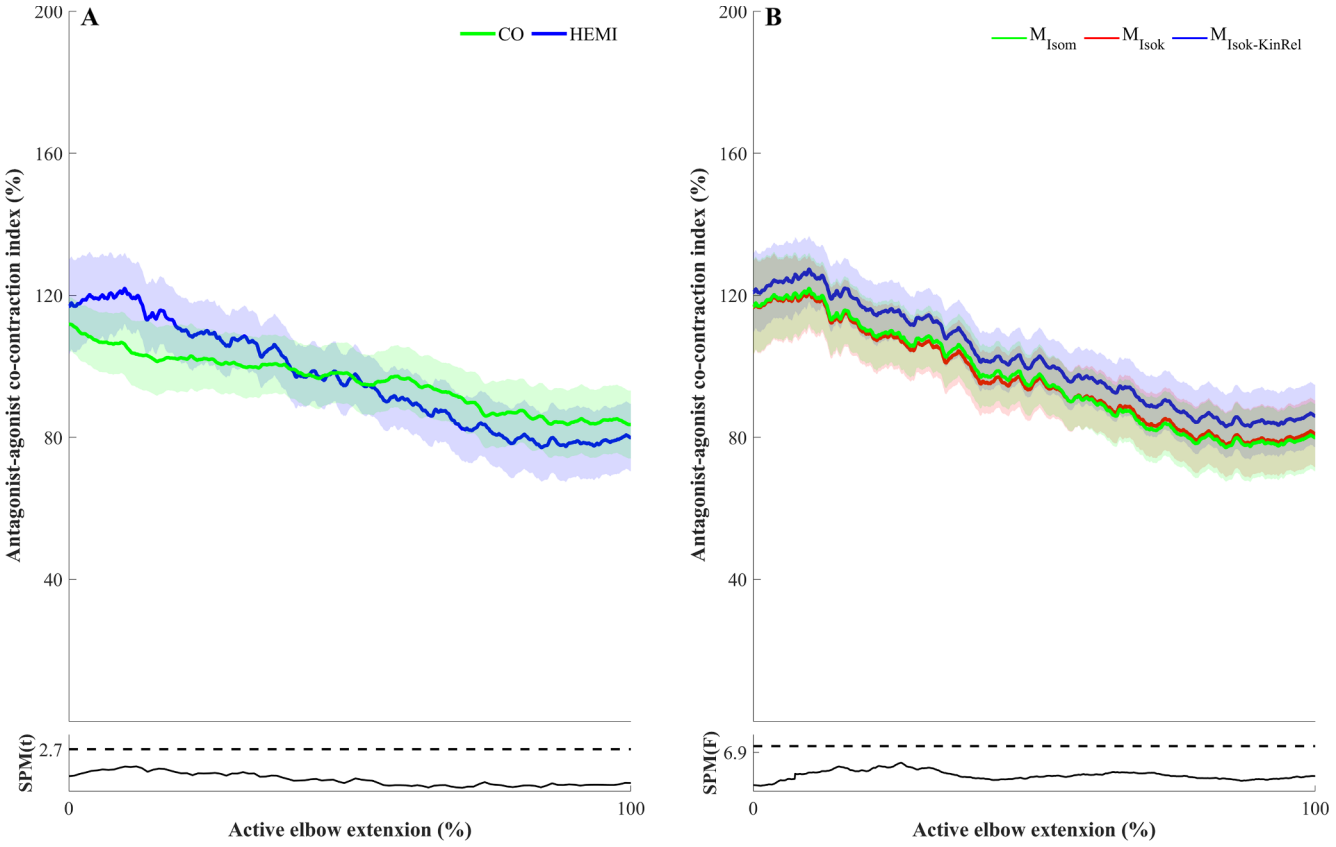

Fig. 3. A. Antagonist-agonist co-contraction index during active elbow extension. The upper panel represents the antagonist-agonist co-contraction index and standard error normalized by M_{Isom} for CO (green) and HEMI (blue). B. Antagonist-agonist co-contraction index during active elbow extension for HEMI. The upper panel represents the antagonist-agonist co-contraction index and standard error normalized by M_{Isom} (green), M_{Isok} (red) and $M_{Isok\text{-}KinRel}$ (blue). The lower panel represents the SPM test statistic continuum, the dashed line corresponding to the significance level threshold ($p < 0.05$). Whenever the test statistic continuum SPM exceeds the threshold, significance is reached and the p-values are reported by shaded gray areas. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

At each percent value of the active elbow range of motion during each extension movement, the preprocessed EMG signal was normalized to its EMG reference value obtained, defined as the RMS value of the EMG linear envelope at a sliding window centered on the same percent value of the active elbow extension range of motion during isokinetic MVC. The $M_{Isok\text{-}KinRel}$ EMG reference values were computed using data collected in concentric mode for elbow extensors and in eccentric mode for elbow flexors.

2.5.4. Antagonist-agonist co-contraction index

For each EMG normalization method, the antagonist-agonist co-contraction index (CCI) was computed from muscle activation (i.e., normalized EMG signals) during each of the active elbow extensions (Falconer and Winter, 1985):

$$CCI = 2 \times \left(\frac{EMG_{Flexors}}{EMG_{Extensors} + EMG_{Flexors}}\right) \times 100 \quad (1)$$

where $EMG_{Flexors}$ is the mean of the three elbow flexor (i.e., BB, BR and BA) activations recorded, and $EMG_{Extensors}$ is the activation of TB.

2.6. Statistical analysis

The statistical analysis consisted of two steps: (i) the first being a preliminary analysis to investigate the presence of atypical EMG patterns in post-stroke subjects compared to healthy subjects, and (ii) the second investigating the effect of the normalization method on muscle activations and the antagonist-agonist co-contraction index in the presence of atypical EMG patterns. For each analysis we used Statistical Parametric Mapping (SPM) which provides a framework to enable statistical comparisons between entire time series data rather than data reduction or selected features (Friston, 2007). In brief, SPM computes a statistic test at each point in the time series, thereby forming a test statistic continuum. To control for multiple comparisons, a critical threshold was computed using random field theory which describes probabilistic behavior of random curves and accounts for the smoothness and temporal increment of the data (Pataky et al., 2013; Pataky et al., 2015). In order to control a Type I error rate, a critical threshold $\alpha = 0.05$ was set (above which only 5% of random curves of the same smoothness would exceed). If the test statistic continuum exceeded the critical threshold, a significant difference is deemed to exist. In order to test the differences in EMG patterns between groups, SPM independent t-tests were performed between HEMI and CO on muscle activations and the antagonist-agonist co-contraction index normalized by M_{Isom}. In order to test the effect of the normalization method (i.e., M_{Isom} vs. M_{Isok} vs. $M_{Isok\text{-}KinRel}$) in HEMI, SPM one-way repeated-measures ANOVA were performed on muscle activations and the antagonist-agonist co-contraction index. All the analyses were conducted using the open-source package "SPM1D" written in Python (Pataky, 2012); in the present study the significance threshold was set at $p < 0.05$. All variables showed normal distribution (Shapiro-Wilk test; $P > 0.05$) and homogeneity of variance (Levene's test; $P > 0.05$).

3. Results

3.1. Inter-group comparisons for muscle activations and the antagonist-agonist co-contraction index

The analysis revealed significant differences during the whole movement for BA, BR and TB (Fig. 1A, C and D), with a significant cluster exceeding the critical threshold ($SPM_t > 2.98$; $p < 0.05$). No significant inter-group difference was found either for BB activation (Fig. 1B) or for the antagonist-agonist co-contraction index during the active elbow extension (Fig. 3A).

3.2. Inter-group normalization method comparison for muscle activation and the antagonist-agonist co-contraction index

The intra-group comparisons revealed no difference between the three methods of normalization either for muscle activations, or for the antagonist-agonist co-contraction index (all, $SPM_F < 6.98$; $p > 0.05$) (Figs. 2 and 3B).

4. Discussion

The aim of this study was to investigate the impact of three EMG normalization methods on muscle activation and on the antagonist-agonist co-contraction index – i.e., an EMG-normalized derived variable used to estimate the antagonist co-contraction level – during active elbow extension in post-stroke subjects. As previously shown (Chalard et al., 2019), our results revealed atypical EMG patterns characterized by increased activity of the elbow flexors and extensors during the active elbow extension in such subjects.

4.1. Isometric MVC normalization is relevant for EMG normalization in post-stroke subjects

In order to consider atypical EMG activity patterns in the stretch position occurring in post-stroke subjects, we made the initial hypothesis that “isokinetic MVC” normalization may be different than “isometric MVC” normalization due to the consideration of force-length and force-velocity relationships. However, and contrary to our initial hypothesis, our results failed to show any significant difference between the three methods of normalization (M_{Isom}, M_{Isok} and $M_{Isok\text{-}KinRel}$) investigated among post-stroke subjects. The similarity of the results obtained using either “isometric MVC” normalization or “isokinetic MVC” normalization may admittedly be explained by a uniform relationship between EMG muscle activation on the one hand, and by muscle-length and elongation velocity on the other hand. This uniform relationship is likely to reflect the absence of the influence of elbow position or angular velocity on EMG amplitude during maximum voluntary contraction (Burden, Trew, and Baltzopoulos, 2003; Burden and Bartlett, 1999). Nevertheless, the absence of any difference between the three methods of EMG normalization provides new practical insights regarding the EMG methodology to be used in post-stroke subjects. Our findings support the evidence that “isometric MVC” normalization is sufficient for accurately assessing muscle activation and the antagonist-agonist co-contraction index during an active movement in post-stroke subjects. The novel practical implications arising from these results are the use and the relevance of the “isometric MVC” normalization method to normalize EMG in a post-stroke population.

4.2. Assessment of antagonist-agonist co-contraction in the presence of atypical EMG patterns

In addition to the aim of this study, our results challenge the relevance of an antagonist-agonist co-contraction index to properly characterize the antagonist co-contraction in the presence of atypical EMG patterns. Based on the sole interpretation of the antagonist-agonist co-contraction index, it is not possible to conclude that post-stroke subjects exhibit significant excessive antagonist co-contractions (Banks et al., 2017). Indeed, our analysis revealed a concomitant increase in both agonist and antagonist muscle activation in post-stroke subjects compared to healthy controls. This general increase in muscle activation reflects pathological EMG patterns related to the loss of motor selectivity between agonist and antagonist muscles during active elbow extension (Schieber et al., 2009). Such atypical agonist activation patterns can lead to the underestimation of the antagonist-agonist co-contraction index, highlighting the inadequacy of using only a ratio between agonist and antagonist muscles to assess antagonist co-contraction in post-stroke subjects. We thus underline the importance of taking a critical look at the quantification of the antagonist co-contraction using the antagonist-agonist co-contraction index in the presence of atypical EMG patterns. To avoid misleading conclusions on antagonist co-contraction, and to properly detect atypical EMG patterns in post-stroke subjects, we recommend concurrent investigation of individual muscle activation of both agonist and antagonist muscles.

5. Limitations

Any generalization of these results should be viewed with caution since we only investigated the impact of three EMG normalization procedures during an active elbow extension in post-stroke subjects. Future studies should investigate the reproducibility of the observed differences in order to improve the applicability of the results.

6. Conclusion

Our findings extend existing advice on EMG normalization in post-stroke subjects exhibiting atypical EMG patterns during voluntary contractions. Based on its practicality and feasibility, we recommend the use of EMG reference values determined during isometric MVC to normalize EMG in post-stroke subjects in a relevant way, either during upper limb isometric contractions or active movements. In addition, our results underline that the assessment of an antagonist-agonist co-contraction index should be systematically combined with the analysis of agonist and antagonist muscle activation to properly highlight the atypical EMG patterns during movement in post-stroke situations.

Declaration of Competing Interest

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

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