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Original Article

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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.

6

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18

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26 **Abstract** (200 words)

27 Electromyographic (EMG) raw signals are sensitive to intrinsic and extrinsic factors.

28 Consequently, EMG normalization is required to draw proper interpretations of standardized

29 data. Specific recommendations are needed regarding a relevant EMG normalization method

30 for participants who show atypical EMG patterns, such as post-stroke subjects. This study

31 compared three EMG normalization methods (“isometric MVC”, “isokinetic MVC”,

32 “isokinetic MVC kinematic-related”) on muscle activations and the antagonist-agonist co-

33 contraction index. Fifteen post-stroke subjects and fifteen healthy controls performed active

34 elbow extensions, followed by isometric and isokinetic maximum voluntary contractions

35 (MVC). Muscle activations were obtained by normalizing EMG envelopes during active

36 movement using a reference value determined for each EMG normalization method. The results

37 showed no significant difference between the three EMG normalization methods in post-stroke

38 subjects on muscle activation and the antagonist-agonist co-contraction index. We highlighted

39 that the antagonist-agonist co-contraction index could **underestimate** the antagonist co-

40 contraction in the presence of atypical EMG patterns. Based on its **practicality and feasibility**,

41 we recommend the use of isometric MVC as a relevant procedure for EMG normalization in

42 post-stroke subjects. We suggest combined analysis of the antagonist-agonist co-contraction

43 index and agonist and antagonist activations to properly investigate antagonist co-contraction

44 in the presence of atypical EMG patterns during movement.

45

46 **Keywords:** upper extremity, hemiplegia; brain injury; antagonist co-contraction; muscle

47 hypertonia

48

49

50

51 **Introduction**

52

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

62

63 The most common method is to normalize the EMG envelope during a task under investigation
64 to the maximum peak value obtained during isometric maximum voluntary contraction (MVC)
65 (Yang & Winter, 1984). Depending on the task of interest, it has been reported that EMG signals
66 normalized using such “isometric MVC normalization” may reach values above 100% (Jobe,
67 Moynes, Tibone, & Perry, 1984). This suggests this method may be not accurate enough to
68 reveal the maximum activity level, and may be inappropriate for dynamic movement (Mirka,
69 1991). To address this issue, the maximum EMG value obtained during isokinetic MVC can be
70 used as the reference EMG value in order to normalize the EMG envelope under dynamic
71 conditions (Fernández-Peña, Lucertini, & Ditroilo, 2009). Using such “isokinetic MVC
72 normalization”, the reference EMG value is calculated for a comparable joint range-of-motion
73 at a similar velocity to the task under investigation. It is, however, not always possible to realize
74 an isokinetic protocol due to experimental limitations (El Mhandi & Bethoux, 2013). An
75 alternative method for EMG normalization is to use the maximum EMG value reached during

76 the task under investigation as the reference value (Yang & Winter, 1984). However, this
77 method tends to reduce the variability between individuals since it makes the reference value
78 relative to the task and not to the maximum capacity of the muscle (Halaki & Ginn, 2012).
79 Although this method may be suitable for comparing EMG patterns over time, it cannot enable
80 consistent and reliable comparison of activity between muscles, tasks and individuals.

81

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

87 It is well established that clinical populations such as post-stroke subjects present
88 **neuromuscular alterations during movement reflected by abnormal EMG muscle activation**
89 **patterns (Ma et al., 2017)**. Among them, the spastic co-contraction corresponds to an excessive
90 activity of antagonist muscles during the active movement (Banks, Huang, Little, & Patten,
91 2017; Gracies, 2005; Ma et al., 2017) which seem to be overstated with muscle lengthening
92 due to alteration of force-length and force-velocity relationships after brain injury (Gracies,
93 2005; Sarcher et al., 2017). The choice of a suitable method of EMG normalization appears
94 especially relevant for post-stroke subjects who present such atypical patterns of EMG activity.
95 It has been shown that “isometric MVC normalization” can yield unpredictable results in
96 subjects with altered neuromuscular control (Ettinger, Weiss, Shapiro, & Karduna, 2016).
97 While EMG analysis is increasingly used in both the upper limb assessment and rehabilitation
98 of post-stroke subjects during active movements (Klein, Li, Hu, & Li, 2018; Zarantonello,
99 Stefani, & Comel, 2017), there is still a substantial lack of data supporting any recommendation
100 for an EMG normalization method in participants who exhibit an atypical EMG pattern. Apart

101 from EMG-based assessment of muscle activation, the issue of EMG normalization is also of
102 major relevance in the clinical context to quantify an EMG-based antagonist-agonist co-
103 contraction index, which is likely to reflect the level of spastic co-contraction. Previous work
104 highlighted a relationship between the level of spastic co-contraction and the range-of-motion
105 restriction (Chalard *et al.*, 2019; Sarcher *et al.*, 2015), highlighting the importance of
106 quantifying the antagonist-agonist co-contraction index in order to improve the motor function
107 of such patients.

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

118

119 **Methods**

120

121 **Participants**

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

126 **Assessment.** Post-stroke participants were included if they were \geq 6 months since stroke onset
127 and were free of any anti-spastic treatment for \geq 4 months. Potential participants with
128 comprehension disorders, neurodegenerative conditions, painful paretic upper limbs during
129 movement or an active elbow extension ability $\leq 20^\circ$ were excluded. All participants gave
130 informed consent prior to participation. This study was approved by the local Research Ethics
131 Board (No ID-RCB: 2017-A01616-47).

132

133 ***Experimental design***

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

140

141 ***Materials***

142 ***Kinematics***

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

148 ***Electromyography***

149 Surface EMG was acquired at 1 kHz with a MP150 system (Biopac Systems Inc., Goleta, CA,
150 USA) with the ground electrode placed on mastoid process. After suitable skin preparation,

151 rectangular self-adhesive bipolar pairs of disposable Ag/AgCl surface electrodes with a 10 mm
152 recording diameter were placed with a 10 mm inter-electrode distance (Afsharipour, Soedirdjo,
153 & Merletti, 2019). The long head of the triceps brachii (TB) was taken to represent the elbow
154 extensors; the biceps brachii (BB), the brachioradialis (BR) and the brachialis (BA) were taken
155 to represent the elbow flexors (Staudenmann & Taube, 2015). A verification procedure was
156 performed to limit crosstalk among biceps brachii, brachialis and triceps brachii.

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

159 *Dynamometry*

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

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

164

165 ***Procedure***

166 *Active elbow extension movements*

167 Participants were seated on an upright chair with shoulders fixed to the chair back by clavicular
168 rings. The height of the table was adjusted to obtain an initial resting position corresponding to
169 shoulder flexion of 80° with internal rotation of 90°, the elbow flexed at 90° and the forearm in
170 a neutral position. Participants were asked to perform two sets of ten active elbow extension
171 movements at spontaneous speed. For each movement, an auditory signal requested the
172 participants to perform a full active elbow extension with the elbow off the table. At the end of
173 elbow extension, participants had a 10-second rest with their forearm on the table. To avoid
174 fatigue, participants were allowed to rest for as long as needed between the two sets.

175

176 *Isometric and isokinetic MVC*

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

188

189 ***Data processing***

190 *Preprocessing*

191 Kinematic data were low-pass filtered at 6 Hz (Cahouët, Martin, & Amarantini, 2002). Raw
192 EMG signals were 10-400 Hz band-pass filtered, full wave rectified, and smoothed at 9 Hz to
193 obtain the linear envelopes (Amarantini, & Bru, 2015). Net torque was low-pass filtered at
194 15 Hz (Bassan *et al.*, 2015). All filters were fourth-order, zero-lag Butterworth type.

195 *Active elbow extension*

196 Kinematic data were obtained from the filtered Cartesian coordinates of the anatomical
197 markers. The onset and offset of each active elbow extension were detected with a threshold of
198 0.01 °/S applied on the elbow angular velocity.

199 *Muscle activations*

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

- 203 • isometric MVC [M_{Isom}] EMG normalization: The preprocessed EMG signal was normalized
204 to its EMG reference value obtained during isometric MVC. The M_{Isom} EMG reference value
205 was calculated as the root mean square (RMS) value of the EMG linear envelope on the 2-
206 second window where the elbow net torque was highest.
- 207 • isokinetic normalization [M_{Isok}] EMG normalization: The preprocessed EMG signal was
208 normalized to its EMG reference value obtained during isokinetic MVC. The M_{Isok} EMG
209 reference value was calculated as the RMS value of the EMG linear envelope on a centered
210 100 ms window when the participant reached the middle of the active elbow extension range
211 of motion. The M_{Isok} EMG reference value was computed using data collected in concentric
212 mode for elbow extensors (TB) and in eccentric mode for elbow flexors (BB, BR, BA).
- 213 • isokinetic kinematic-related [$M_{Isok-KinRel}$] EMG normalization: At each percent value of the
214 active elbow range of motion during each extension movement, the preprocessed EMG
215 signal was normalized to its EMG reference value obtained, defined as the RMS value of the
216 EMG linear envelope at a sliding window centered on the same percent value of the active
217 elbow extension range of motion during isokinetic MVC. The $M_{Isok-KinRel}$ EMG reference
218 values were computed using data collected in concentric mode for elbow extensors and in
219 eccentric mode for elbow flexors.

220

221 *Antagonist-agonist co-contraction index*

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

225
$$CCI = 2 \times (\text{EMG}_{\text{Flexors}} / (\text{EMG}_{\text{Extensors}} + \text{EMG}_{\text{Flexors}})) \times 100 \quad (1)$$

226 where $\text{EMG}_{\text{Flexors}}$ is the mean of the three elbow flexor (i.e., BB, BR and BA) activations
227 recorded, and $\text{EMG}_{\text{Extensors}}$ is the activation of TB.

228

229 ***Statistical analysis***

230 The statistical analysis consisted of two steps: i) the first being a preliminary analysis to
231 investigate the presence of atypical EMG patterns in post-stroke subjects compared to healthy
232 subjects, and ii) the second investigating the effect of the normalization method on muscle
233 activations and the antagonist-agonist co-contraction index in the presence of atypical EMG
234 patterns. For each analysis we used Statistical Parametric Mapping (SPM) which provides a
235 framework to enable statistical comparisons between entire time series data rather than data
236 reduction or selected features (Friston, 2007). In brief, SPM computes a statistic test at each
237 point in the time series, thereby forming a test statistic continuum. To control for multiple
238 comparisons, a critical threshold was computed using random field theory which describes
239 probabilistic behavior of random curves and accounts for the smoothness and temporal
240 increment of the data (Pataky, Robinson, & Vanrenterghem, 2013; Pataky, Vanrenterghem, &
241 Robinson, 2015). In order to control a Type I error rate, a critical threshold $\alpha = 0.05$ was set
242 (above which only 5% of random curves of the same smoothness would exceed). If the test
243 statistic continuum exceeded the critical threshold, a significant difference is deemed to exist.
244 In order to test the differences in EMG patterns between groups, SPM independent t-tests were
245 performed between HEMI and CO on muscle activations and the antagonist-agonist co-
246 contraction index normalized by M_{Isom} . In order to test the effect of the normalization method
247 (i.e., M_{Isom} vs. M_{Isok} vs. $M_{\text{Isok-KinRel}}$) in HEMI, SPM one-way repeated-measures ANOVA were
248 performed on muscle activations and the antagonist-agonist co-contraction index. All the
249 analyses were conducted using the open-source package “SPM1D” written in Python (Pataky,

250 2012); in the present study the significance threshold was set at $p < 0.05$. All variables showed
251 normal distribution (Shapiro-Wilk test; $P > 0.05$) and homogeneity of variance (Levene's test;
252 $P > 0.05$).

253

254

255 **Results**

256

257 ***Inter-group comparisons for muscle activations and the antagonist-agonist co-contraction***
258 ***index***

259 The analysis revealed significant differences during the whole movement for BA, BR and TB
260 (Fig. 1.A, 1.C and 1.D), with a significant cluster exceeding the critical threshold ($SPM_t > 2.98$;
261 $p < 0.05$). No significant inter-group difference was found either for BB activation (Fig. 1.B)
262 or for the antagonist-agonist co-contraction index during the active elbow extension (Fig. 3.A).

263

264 ***Inter-group normalization method comparison for muscle activation and the antagonist-***
265 ***agonist co-contraction index***

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

269

270

271 **Discussion**

272 The aim of this study was to investigate the impact of three EMG normalization methods on
273 muscle activation and on the antagonist-agonist co-contraction index (CCI) – i.e., an EMG-
274 normalized derived variable used to estimate the antagonist co-contraction level – during active

275 elbow extension in post-stroke subjects. As previously shown (Chalard *et al.*, 2019), our results
276 revealed atypical EMG patterns characterized by increased activity of the elbow flexors and
277 extensors during the active elbow extension in such subjects.

278

279 *Isometric MVC normalization is relevant for EMG normalization in post-stroke subjects*

280 In order to consider atypical EMG activity patterns in the stretch position occurring in post-
281 stroke subjects, we made the initial hypothesis that “isokinetic MVC normalization” may be
282 **different** than “isometric MVC normalization” due to the consideration of force-length and
283 force-velocity relationships. However, and contrary to our initial hypothesis, our results failed
284 to show any significant difference between the three methods of normalization (M_{Isom} , M_{Isok}
285 and $M_{Isok-KinRel}$) investigated among post-stroke subjects. The similarity of the results obtained
286 using either “isometric MVC normalization” or “isokinetic MVC normalization” may
287 admittedly be explained by a uniform relationship between EMG muscle activation on the one
288 hand, and by muscle-length and elongation velocity on the other hand. This uniform
289 relationship is likely to reflect the absence of the influence of elbow position or angular velocity
290 on EMG amplitude during maximum voluntary contraction (Burden, Trew, & Baltzopoulos,
291 2003; Burden & Bartlett, 1999). Nevertheless, the absence of any difference between the three
292 methods of EMG normalization provides new practical insights regarding the EMG
293 methodology to be used in post-stroke subjects. Our findings support the evidence that
294 “isometric MVC normalization” is sufficient for accurately assessing muscle activation and the
295 antagonist-agonist co-contraction index during an active movement in post-stroke subjects. The
296 novel practical implications arising from these results are the use and the relevance of the
297 “isometric MVC normalization” method to normalize EMG in a post-stroke population.

298

299 *Assessment of antagonist-agonist co-contraction in the presence of atypical EMG patterns*

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

317

318 ***Limitations***

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

323

324 ***Conclusion***

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

333

334 **Conflict of Interest Statement**

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

337

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345

346

347

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424

Table 1. Participant demographics (median \pm interquartile range).

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

* Indicates a significant difference between HEMI and CO ($p < 0.05$). ¹Spasticity of elbow flexors and extensors was assessed using the Tardieu scale.

Abbreviations: FMA-UE, Fugl-Meyer Assessment score for Upper Extremity.

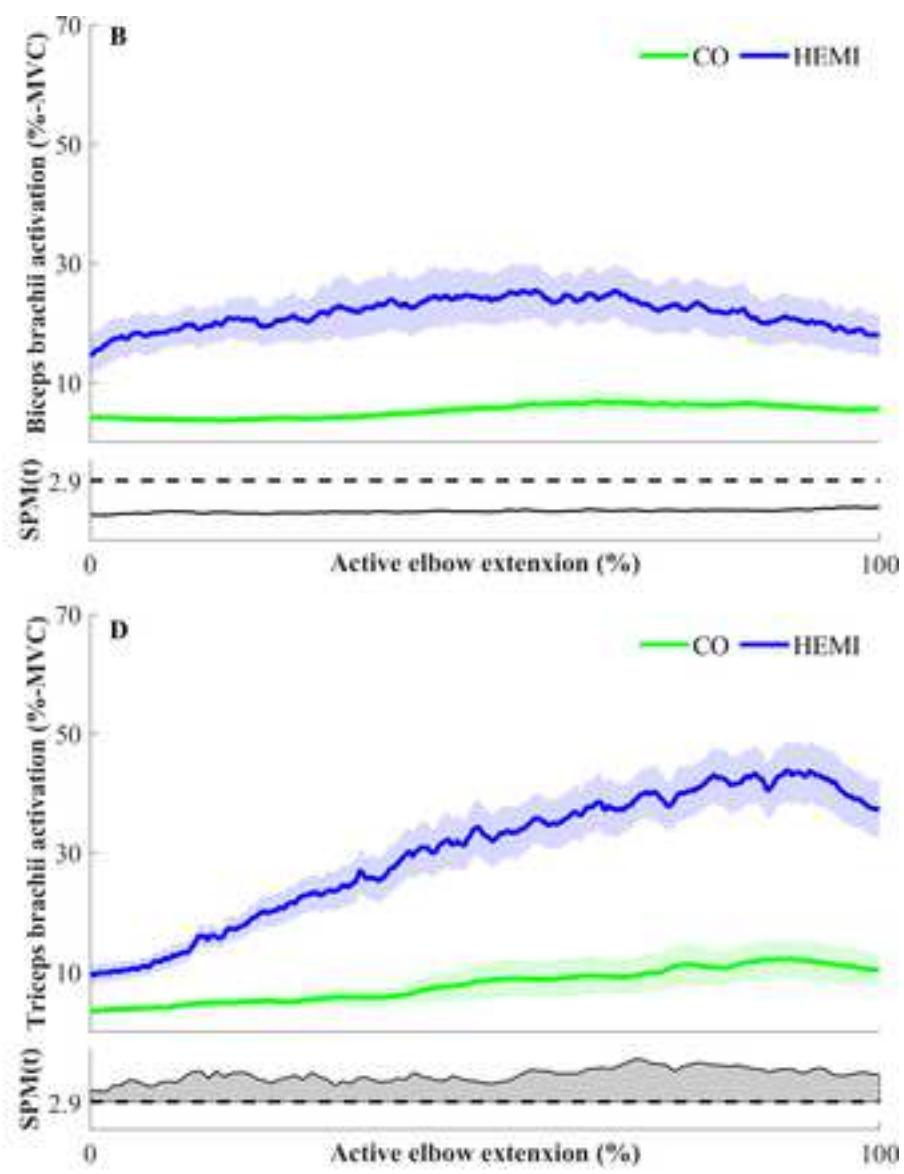
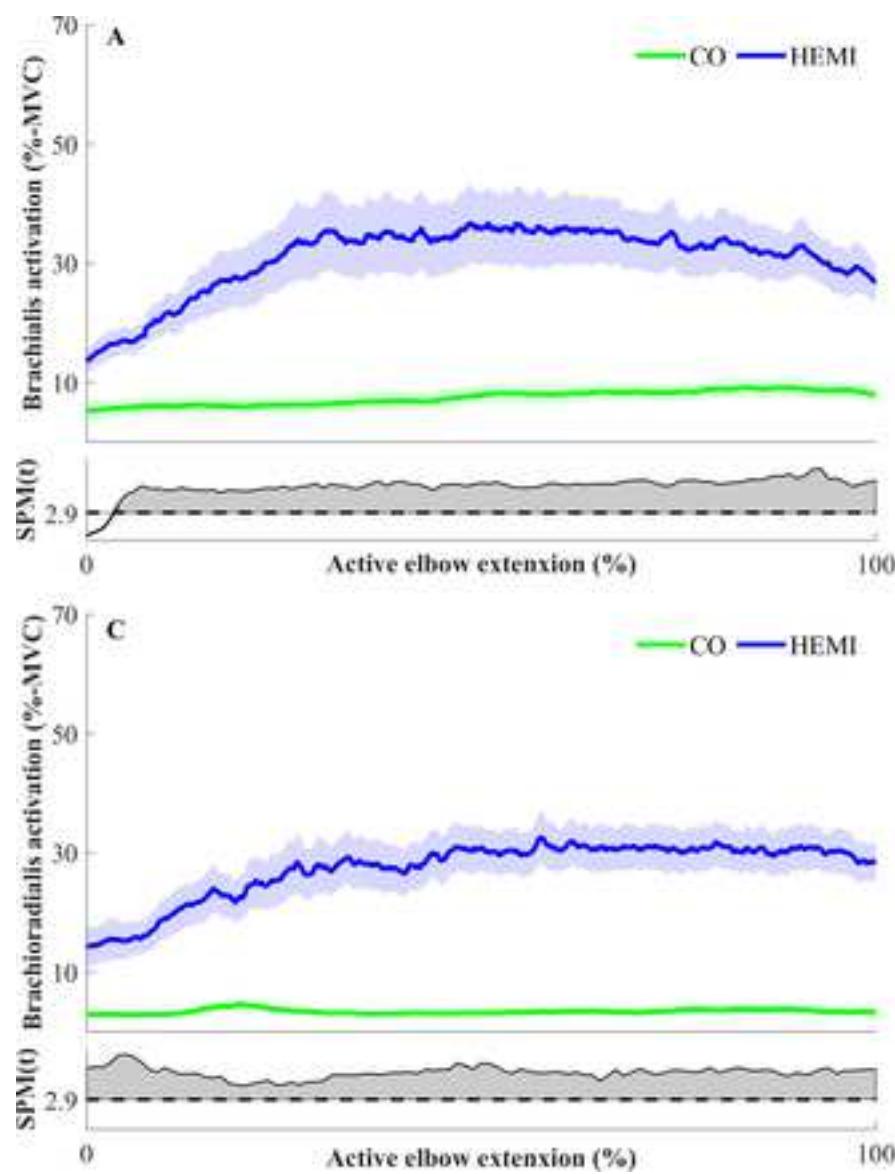
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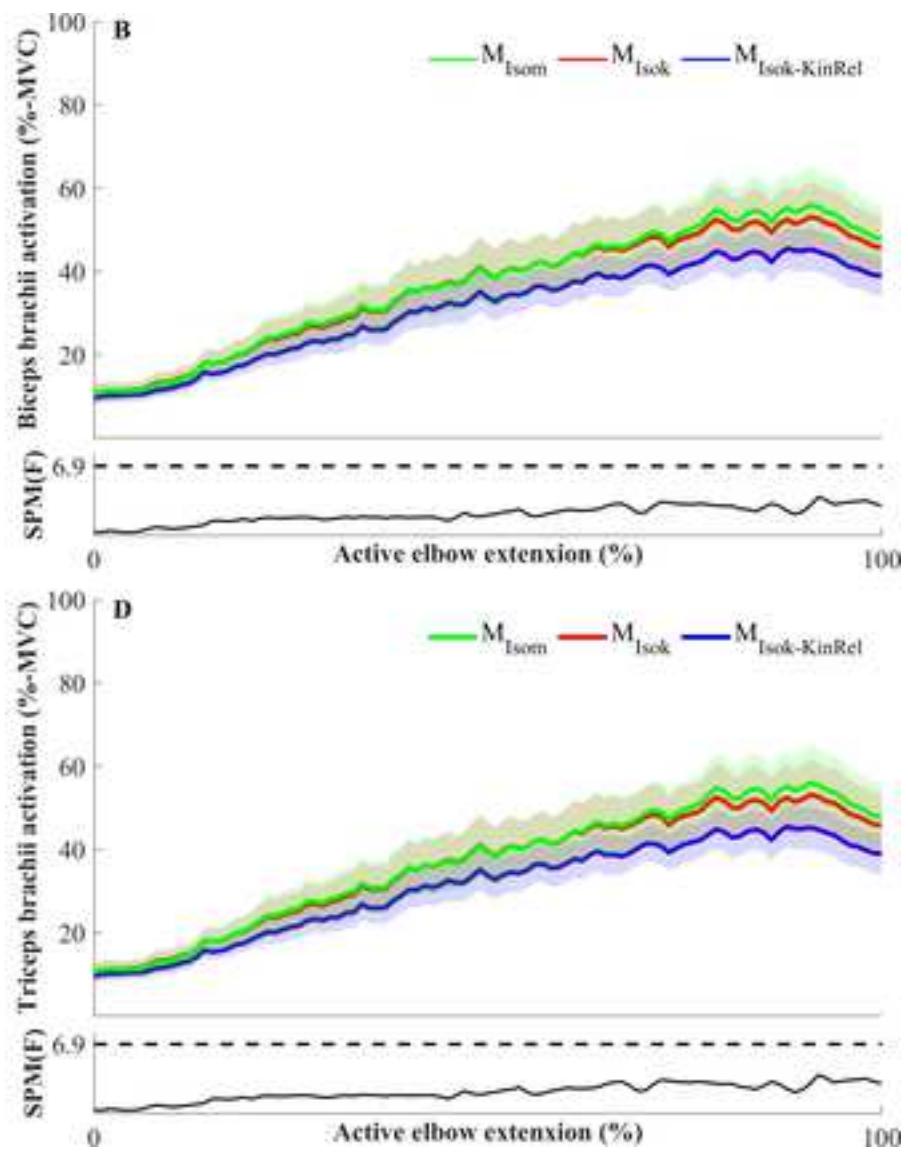
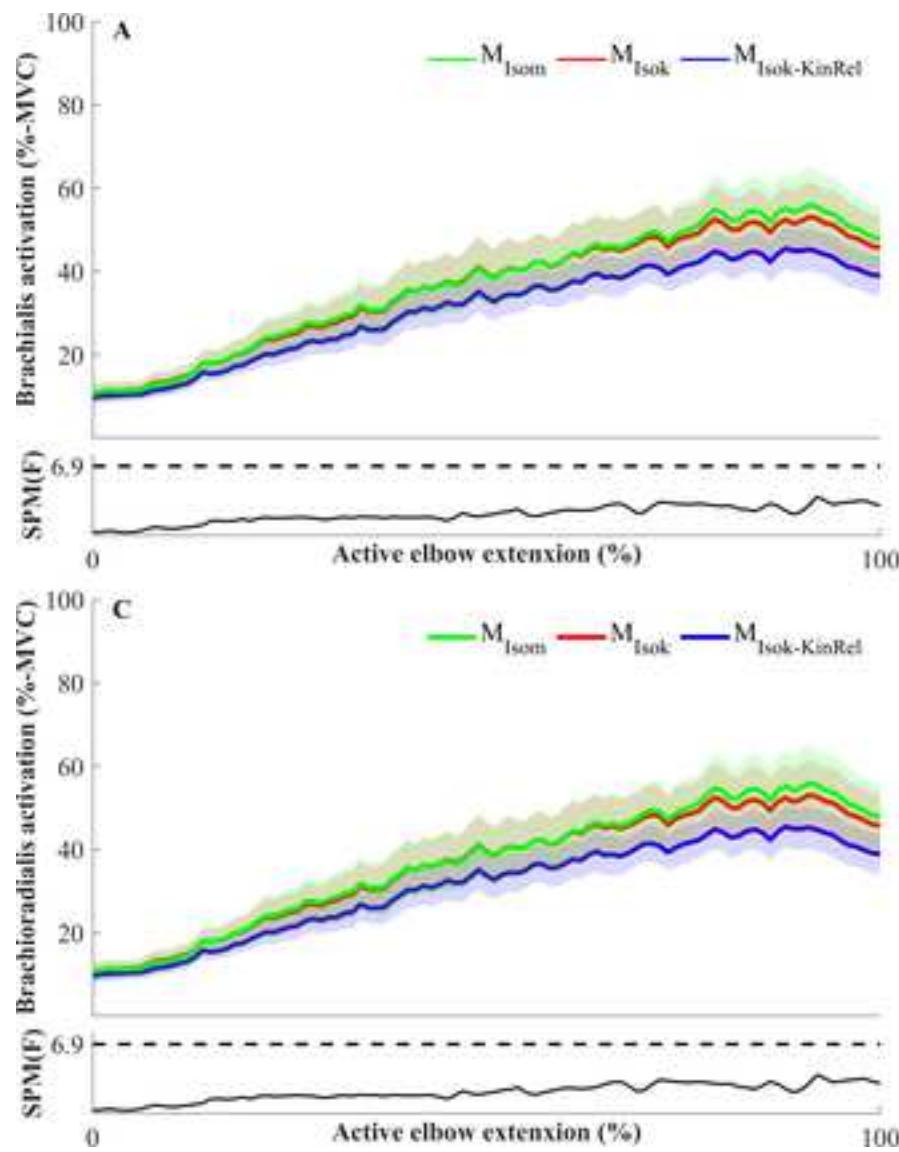
Figure 1. Muscle activations normalized by M_{Isom} during active elbow extension for: A. brachialis (BA), B. biceps brachii (BB), C. brachioradialis (BR), and D. triceps brachii (TB). The upper panel represents the muscle activation and standard error for CO (green) and HEMI (blue). The lower panel 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.

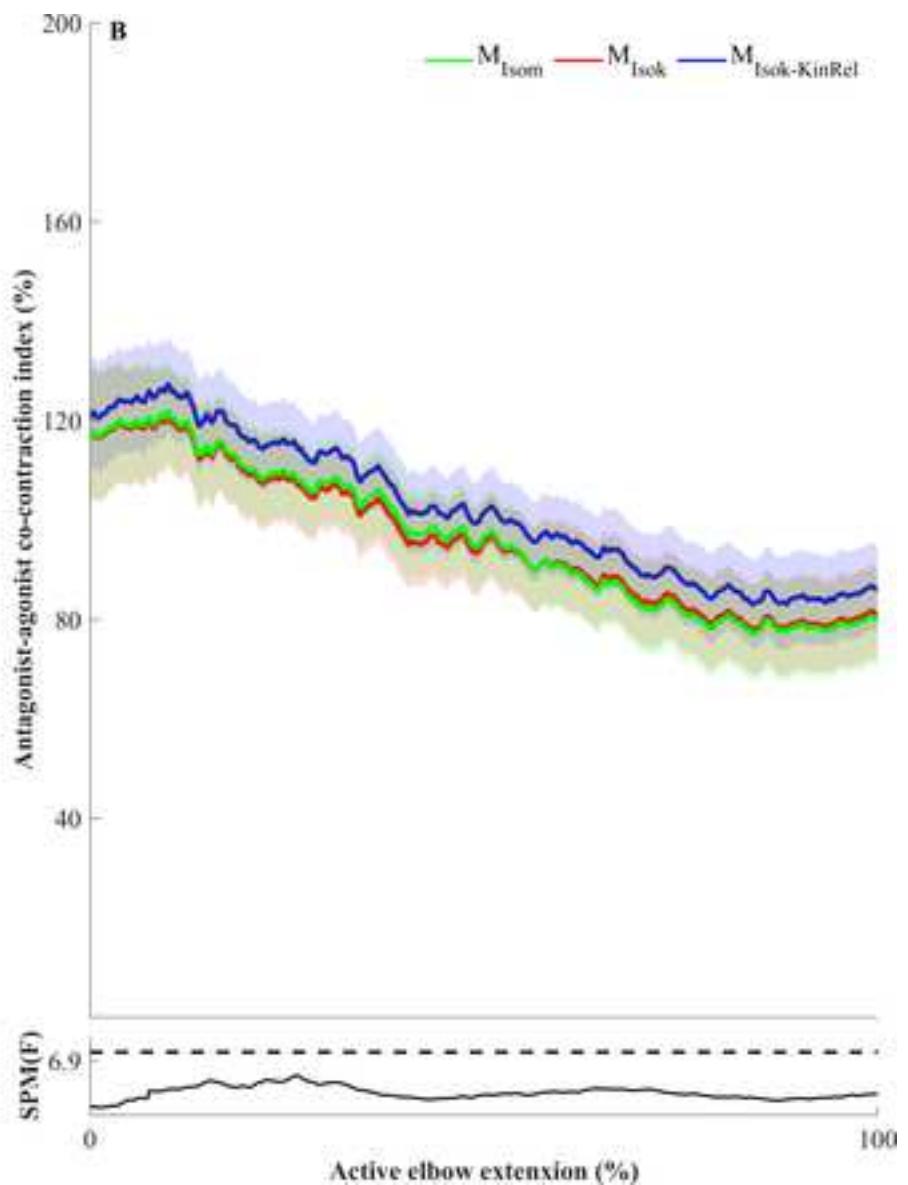
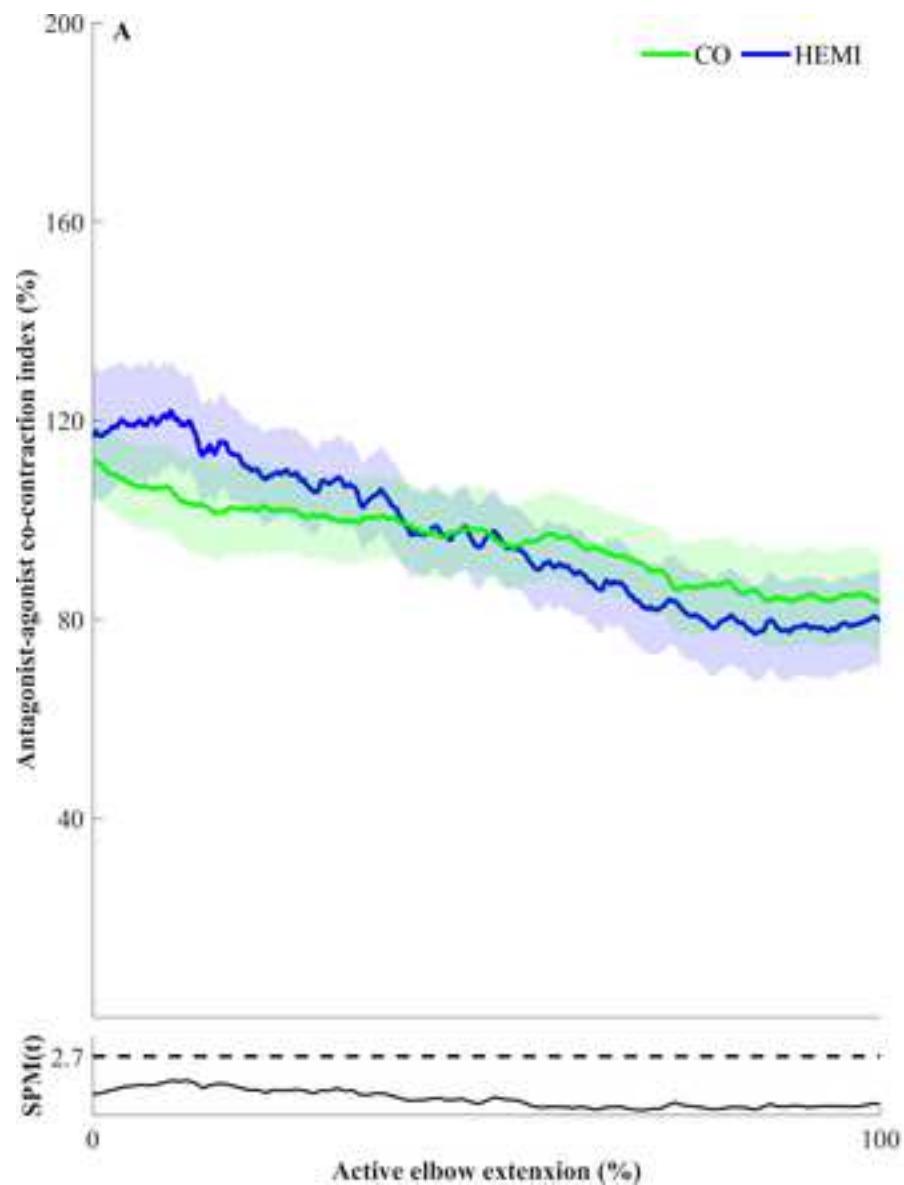
Figure 2. Muscle activations during active elbow extension for HEMI for: A. brachialis (BA), B. biceps brachii (BB), C. brachioradialis (BR), and D. triceps brachii (TB). The upper panel represents the muscle activation and standard error normalized by M_{Isom} (green), M_{Isok} (red) and $M_{Isok-KinRel}$ (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.

Figure 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 $M_{Isok-KinRel}$ (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.









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subjects.

Conflict of Interest Statement

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



All authors have made substantial work according the ICJME guidelines and have read and approved the submitted manuscript. The manuscript has not been submitted elsewhere nor published elsewhere.