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# The effect of rolling massage on the excitability of the corticospinal pathway

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**Abstract**

The aim of the present study was to investigate the alterations of corticospinal excitability (motor evoked potential, MEP) and inhibition (silent period, SP) following rolling massage of the quadriceps muscles. Transcranial magnetic and femoral nerve electrical stimuli were used to elicit MEPs and compound muscle action potential (Mmax) in the vastus lateralis and vastus medialis muscles prior to and following either: i) 4 sets of 90-s rolling massage (ROLLING) or ii) rest (CONTROL). One series of neuromuscular evaluations, performed after each set of ROLLING or CONTROL, included three MEPs and one Mmax elicited every 4 s during 15 s submaximal contractions at 10% (experiment 1, n = 16) and 50% (experiment 2, n = 10) of maximal voluntary knee extensions (MVC). The MEP·$Mmax^{-1}$ ratio and electromyographic activity recorded from VL at 10% MVC demonstrated significantly lower values during ROLLING than CONTROL ($P < 0.05$). The ROLLING did not elicit any significant changes in muscle excitability (Mmax area) and duration of TMS-induced SP recorded from any muscle or level of contraction ($P > 0.05$). The findings suggest that rolling massage can modulate the central excitability of the circuitries innervating the knee extensors however, the observed effects are dependent on the background contraction intensity during which the neuromuscular measurements are recorded.

Key words: massage, transcranial magnetic stimulation, afferent feedback receptors, corticomotor pathway, motoneurone.

Résumé

Le but de cette étude était d’investiguer les modifications d’excitabilité (potentiel évoqué moteur, PEM) et d’inhibition (période de silence, PS) corticospinale à la suite d’un massage par rouleau des quadriceps. La stimulation magnétique transcrânienne et la stimulation électrique du nerf fémoral ont été utilisées pour évoquer des PEMs et des potentiels d’action musculaires composés (Mmax) sur les muscles vastus lateralis et vastus medialis avant et après : i) 4 séries de 90-s de massage par rouleau (ROLLING) ou ii) une période équivalente de repos (CONTROL). Les évaluations neuromusculaires, réalisées après chaque série de ROLLING ou CONTROL, comprenaient trois PEMs et un Mmax évoqués toutes les 4 s pendant une contraction sous-maximale à 10% (étude 1, n = 16) et 50% (étude 2, n = 10) de la force maximale volontaire (FMV). Le rapport MEP·$Mmax^{-1}$ et l’activité électromyographique enregistrée sur VL à 10% de FMV étaient significativement plus faibles pour ROLLING que pour CONTROL ($P < 0{,}05$). En revanche, ROLLING n’induisait aucune modification significative de l’excitabilité du muscle (aire de Mmax) ou de la durée des PSs, quel que soit le niveau de contraction ($P > 0{,}05$). Ces résultats suggèrent que le massage par rouleau peut moduler l’excitabilité centrale des voies innervant les muscles extenseurs du genou. Cependant, les effets dépendent l’intensité de contraction pendant laquelle l’évaluation neuromusculaire est réalisée.

Mots-clés : massage, stimulation magnétique transcrânienne, récepteurs sensoriels, voie cortico-spinale, motoneurone.

## Introduction

Self myofascial release (SMFR) technique using foam roller and roller massager is used extensively in rehabilitation and athletic settings to promote soft-tissue extensibility and enhance recovery from training (for review, see Beardsley and Škarabot, 2015). Previous studies suggest that this technique may enhance range of motion (MacDonald et al. 2013; Sullivan et al. 2013; Halperin et al. 2014; Bradbury-Squire et al. 2015; Behara and Jacobson 2017), pressure pain threshold (Pearcey et al. 2015; Aboodarda et al. 2015; Cavanaugh et al. 2017) and arterial dilation and vascular plasticity (Okamoto et al. 2014). A "neurophysiological model" has been proposed to explain the influence of SMFR on the musculoskeletal functions. This model focuses on the mechanical pressure that a roller massage apparatus exerts on the mechanoreceptors, proprioceptors and pain receptors encapsulated in the fascia (for review, see Beardsley and Škarabot 2015). It has been suggested that activation of these sensory receptors alters the self-regulatory dynamics of the autonomic nervous system and consequently modifies the muscle tissue extensibility (for review, see Schleip 2003 a,b; Beardsley and Škarabot 2015).

One aspect of the SMFR technique that has not been explored is the role that it may play in the modulation of the corticospinal pathway (central) excitability throughout the activation of the afferent feedback receptors. It is well established that repeated somatosensory input (via activation of sensory receptors) can modulate the responsiveness of the motor and sensory cortical circuitries (Fourment et al. 1996; Carson et al. 1999; Ridding and Taylor, 2001; Kaelin-Lang et al. 2002). Several studies have used transcranial magnetic stimulation (TMS) and reported an increase in the excitability of the corticomotor pathway following activation of the afferents sensory receptors with muscle and tendon vibration (Siggelkow et al. 1999; Steyvers et al. 2003; Souron et al. 2017). This contrasts with no change in corticospinal excitability with manual massage (Dishman and Bulbulian, 2001). Conversely, studies that used Hoffmann's reflex (H-reflex) amplitude found a reduction in excitability of the spinal motoneurone during manual massage (Morelli et a. 1991; Goldberg et al. 1992; Sullivan et al. 1991, 1993; Behm et al. 2013). However, there is no documented study that has explored the influence of the rolling massage on the responsiveness of the corticospinal pathway innervating the massaged muscle group.

Understanding the effects of rolling massage on acute corticomotor responses may reveal the mechanistic basis of the adaptations that may occur in the central nervous system following the chronic use of SMFR. Therefore, the aim of the present study was to investigate the influence of rolling massage on the corticospinal and peripheral responses of the knee extensor muscles. Based on previous massage studies, it was hypothesized that rolling massage will inhibit corticospinal excitabilities.



## MATERIALS and METHODS

### *Experiment 1*

***Participants.*** Sixteen recreationally active male participants (height 175.5 $\pm$ 7.8 cm, body mass 79.4 $\pm$ 9.1 kg, age 27.2 $\pm$ 8.8 yrs) volunteered for this study. Fifteen participants were determined as right-leg dominant based on the preferred leg used to kick a ball (Kovaleski et al. 1999). Individuals with neurological conditions, cardiovascular complications, or surgery or injury to the knee structures were excluded from the study. After explaining the experimental procedures, participants completed the TMS safety checklist (Rossi et al. 2011) and the Physical Activity Readiness Questionnaire-Plus form (Canadian Society for Exercise Physiology, 2011). Participants also signed a letter of informed consent prior to participating in the study.

Participants were instructed to abstain from alcohol, caffeine, nicotine, and strenuous physical activity for at least 24-hours prior to the experimental sessions. Ethical approval for this study was granted by the Health Research Ethics Authority of the Memorial University of Newfoundland (HREB #14.118).

***Research design.*** Participants visited the laboratory on three separate occasions separated by at least 24 hours. The first session involved familiarizing the participants with the experimental protocol and obtaining informed consent. During the next two sessions, the order of which was randomized, the participants performed one of the two intervention protocols: i) four sets of 90s rolling massage (ROLLING) applied on the quadriceps muscles or ii) time matched rest (CONTROL). A series of neuromuscular evaluations were performed before (baseline) and following each set of intervention (rolling massage or rest). All measurements and the rolling massage were performed on the right leg.



***Experimental set up.*** Electromyography and stimulating electrodes were placed on the participants' muscles and peripheral nerve, respectively (see below). During experimental protocol, participants were seated in a custom-built knee extension chair with the hip and knee positioned at 90° (Button and Behm, 2008). In order to avoid contribution from the upper body during knee extensions, two straps were placed around the trunk and waist and participants were instructed to cross their arms across their chest. The right ankle was inserted into padded ankle cuffs attached to a strain gauge (Omega engineering Inc., LCCA 250, Don Mills, Ontario) via a non-extensible strap. The data from the strain gauge was sampled at a rate of 2,000-Hz, amplified (×1000), digitally converted (AcqKnowledge III, Biopac Systems Inc., Holliston, MA) and monitored on a computer screen.

Before initiation of the neuromuscular evaluations, participants performed a warm-up for the knee extensor muscles. Warm-up consisted of 2 sets of 12 submaximal isometric contractions at 50% of estimated MVC. The contractions were intermittent: 2-s contraction followed by 2-s rest. Following warm-up, two 4-s isometric knee extension MVCs were performed at baseline. Two minutes of rest was given between the MVCs. Another MVC was performed immediately after completion of the interventions (ROLLING or rest) in each experimental session. Participants were encouraged to generate maximal force output as fast as possible.

The maximal force derived from the baseline MVCs was used to calculate 10% of MVC. This value was shown on the computer screen, which participants used as a guideline. The participants were instructed to sustain the knee extension force just above the guideline for 15 s during which three TMS and one peripheral nerve electrical stimulus (PNS) (Figure 1) were elicited. The time interval between the stimuli was 4 s and the first stimulus was delivered 2 s after initiation of knee extension contractions. Thus, the stimuli were delivered at 2, 6, 10 and 14 s. The sequence of TMS and PNS stimuli was randomly assigned for each participant.



Rolling massage was applied on the quadriceps muscles using a Theraband® roller massager (Hygienic Corporation, Akron, OH). The roller massager was 24 cm in length and 14 cm in circumference and composed of a hard rubber material with low amplitude, longitudinal grooves surrounding a plastic cylinder (Halperin et al. 2014). Rolling massage was applied over the belly of the quadriceps muscle, along the length of VL, VM and rectus femoris muscles, at a slow pace (2 s proximally and 2 s distally). Participants provided feedback regarding the level of perceived pain during the rolling massage and the intensity of applied force (with a depth of ~ 1-3 cm over quadriceps muscle) was adjusted accordingly to ensure a value of 7/10 on the visual analogue scale (VAS) was maintained (Halperin et al. 2014; Aboodarda et al. 2015).

***Electromyography (EMG).*** Surface EMG activity was measured using pairs of self-adhesive Ag-Ag Cl electrodes (Kendall MediTrace foam electrodes, Chicopee, MA) positioned 2 cm apart (centre to centre) on the vastus lateralis (VL) and vastus medialis (VM) muscles of the right leg in the direction of the underlying muscle fibers (Hermens et al. 1999). A ground electrode was placed on the patella bone of the same leg. In order to decrease skin resistance and ensure an inter-electrode impedance of <5 kΩ, the skin was shaved, abraded, and cleaned with an isopropyl alcohol swab. All EMG signals were amplified (Biopac System Inc., DA 100: analog to digital converter MP150WSW; Holliston, MA) and recorded with a sampling rate of 2,000 Hz using a commercially designed software program (AcqKnowledge III, Biopac System Inc.). EMG activity was filtered with a Blackman −61 dB band-pass filter between 10–500 Hz, amplified (bi-polar differential amplifier, input impedance = 2 MΩ, common mode rejection ratio > 110 dB min, gain × 1000), analog-to-digitally converted (12 bit) and stored for further analysis.

***Peripheral nerve stimulation.*** To determine the size of compound muscle action potential (Mmax), the peripheral nerves innervating the quadriceps muscle were stimulated by a single stimulus at the femoral nerve using a constant-current stimulator (DS7AH; Digitimer, Hertfordshire, UK). The surface stimulating electrodes were secured at the femoral triangle (cathode; Kendall MediTrace foam electrodes, Chicopee, MA) and between the greater trochanter and superiliac projections (anode; 9 × 5 cm, Dura-Stick II, Chattanooga Group, Hixson, TN). The intensity of the stimuli (70 - 340 mA; square-wave pulse duration: 200 μs; 400 V maximum voltage) was increased incrementally until Mmax was observed. The current intensity was then increased by an additional 30% to ensure supramaximal stimulation. This stimuli intensity was used for the remainder of the experimental session. Mmax was also used to normalize MEP area to account for changes in peripheral neuromuscular propagation.

***Transcranial Magnetic Stimulation.*** TMS induced motor evoked potential (MEP) responses of the quadriceps muscles were evoked using a single TMS pulse. During voluntary isometric knee extensions (10% of MVC), TMS pulses were manually delivered to the motor cortex using a magnetic stimulator (Magstim 2002 , The Magstim Company Ltd., Whitland, UK) and a 110-mm double-cone coil (maximum output of 1.4 T) to induce a posteroanterior current. Participants wore a latex swim cap on which the coil location was drawn. The coil was positioned at the vertex marked on the scalp as the intersection of the lines drawn from nasion to inion and from tragus to tragus. TMS intensity was increased stepwise to produce a MEP amplitude of approximately 20% of VL and VM muscle Mmax during brief contractions at 10% MVC. The group means stimulation intensities for contractions at 10 and 50% of MVC were 61 ± 14% and 47 ± 9% of maximum stimulator output, respectively.

***Experiment 2***

Ten recreationally active male participants (height 176.2 ± 6.83 cm, body mass 78.9 ± 8.4 kg, age 27.6 ± 6.6 yrs) completed the same protocol as experiment 1, with the exception of the intensity of MVC knee extensions, which was changed to 50%. Participants included seven participants from experiment 1 and three new participants.

***Outcome measures.*** MEP and Mmax areas were measured from the initial deflection of signal from baseline to the second crossing of the horizontal axis. The duration of the silent period (SP) was assessed as the interval from the MEP stimulus artifact to the return of the continuous EMG by visual inspection (Schnitzler and Benecke 1994). The MEP responses were divided by the corresponding Mmax recorded at each contraction to calculate MEP·$Mmax^{-1}$ ratio. In order to eliminate the effect of day-to-day variations on MEP and Mmax responses, all post-intervention values (i.e. measurements following each set of rolling massage or rest) were normalized to the average of the two baseline measurements at the same contraction intensity. The background EMG (root mean square; rmsEMG) of the VL and VM were quantified over 500 ms duration prior to the point of each stimulus (TMS and PNS) at each target force. In order to evaluate the central drive during contractions, the rmsEMG values were normalized to the amplitude of Mmax recorded at each contraction. The magnitude of the baseline and post-intervention peak MVC force outputs were measured in each experimental session.



***Statistical Analysis***. Statistical analyses were computed using SPSS software (Version 16.0, SPSS, Inc, Chicago, IL). Assumption of normality (Shapiro-Wilk test) and sphericity (Mauchley test) were tested for all of the dependent variables. If the assumption of sphericity was violated, the corrected value for non-sphericity with Greenhouse-Geisser epsilon was reported. In order to determine the effect of rolling massage on corticospinal responses of the quadriceps muscles, a two-way analysis of variance (ANOVA) with repeated measures (2 conditions × 4 sets of interventions) was used for all variables. A two-way ANOVA with repeated measure (2 conditions × 2 time points) was performed to measure the influence of the rolling massage on

MVC force output. If results showed a significant main effects or interactions, Bonferroni post-hoc test was used to identify differences trials. The effect size (ES) was calculated converting partial eta-squared to Cohen`s d (Cohen, 1988) to provide a better understanding about the magnitude of the statistical significance between different measures. According to Cohen (1988), the magnitude of effect size can be classified as small ($0.2 \leq d < 0.5$), medium ($0.5 \leq d < 0.8$), and large ($d \geq 0.8$). This process was repeated for all variables recorded at either 10 or 50% of MVC experiments. Significance was defined as $p < 0.05$.

## Results

The ROLLING did not cause any significant change in the post-intervention MVC force output as well as the muscle excitability (Mmax area) at either 10 or 50% MVC (all $P > 0.05$). Additionally, no significant change was observed for the SP recorded from VL or VM during contractions at either 10 or 50% MVC ($P > 0.05$). The absolute values for the neurophysiological parameters are presented in Tables 1 and 2.



### *Experiment 1*

***MEP Area***. The MEP·Mmax$^{-1}$ ratio recorded from VL at 10% MVCs demonstrated a significantly lower value (condition effect: $F_{1,15} = 4.75$, $P = 0.046$, $d = 1.12$) during the ROLLING compared to the CONTROL session (Figure 1 and 2). No significant difference was observed for the MEP·Mmax$^{-1}$ recorded from VM at this contraction intensity.

***rmsEMG.*** The rmsEMG recorded from VL (normalized to Mwave) exhibited a significantly lower value (condition effect: $F_{1,15} = 7.91$, $P = 0.016$, $d = 1.62$) following ROLLING than CONTROL across the 4 sets of intervention (Figure 3). The difference between the two conditions showed similar pattern for the VM rmsEMG however the data demonstrated a trend to significance ($F_{1,15} = 3.93$, $P = 0.07$, $d = 1.14$).

***Experiment 2***

***MEP Area.*** No significant change was observed for the VL and VM MEP·$Mmax^{-1}$ ratio at this intensity ($P > 0.05$).

***rmsEMG.*** The rmsEMG recorded from VL and VM at 50% of MVC did not demonstrate any difference between two conditions ($P > 0.05$).

**Discussion**

The principal findings of the present study are: (i) ROLLING modulated (reduced) the corticospinal responses recorded from VL at 10% of MVC, (ii) no significant difference was observed in the peripheral excitability (Mmax) of the VL after the two conditions; thus these findings suggest that the observed modulations in MEP and rmsEMG responses at 10% of MVC were due to the adaptations in the central motor pathway controlling the activity of the VL. The MEP and rmsEMG recorded from VL and VM at 50% of MVC exhibited no difference between the two conditions. Overall, the results indicate that rolling massage disfacilitates the central excitability of the circuitries innervating the massaged muscles (specifically VL). However, this effect is only evident at low level of contractions (e.g. 10% of MVC) where minimum central drive is required to recruit the low threshold spinal motoneurones and motor units.

To best of our knowledge, this is the first study to quantify the effect of rolling massage on central and peripheral excitability of a muscle group. Indeed, several studies have examined the effect of other mechanical stimuli such as tendon vibration (Siggelkow et al. 1999; Kossev et al. 1999; Steyvers et al. 2003) and manual massage (Dishman and Bulbulian, 2001) on alteration of the corticomotor pathway responses. However, due to differences in the characteristics of the mechanical pressure applied on the tissue, the findings of the present study can not be directly compared with these studies. For instance, during the muscle and tendon vibration, a low muscle vertical displacement (0.5 mm) and moderate to high frequency stimuli (75-120 Hz) were applied (Siggelkow et al. 1999; Steyvers et al. 2003); whereas during ROLLING a high muscle vertical pressure (with a depth of ~ 1-3 cm) and low pace of rolling massage (i.e. 2 s from proximal to distal and 2 s from distal to proximal) were exerted. Nonetheless, a general comparison between the effects of the two mechanical stimuli indicates that the local vibration (high frequency/low mechanical pressure) facilitated the corticospinal excitability (Siggelkow et al. 1999; Kossev et al. 1999; Steyvers et al. 2003) whereas ROLLING (low frequency/high mechanical pressure) resulted in the reduction of central motor responses. A possible factor leading to this divergent result could be the activation of different afferent sensory receptors by local vibration and ROLLING. It is well established that the low amplitude innocuous vibration activates primary spindle afferents and consequently enhances the excitability of corticospinal projections to the target muscle (Kossev et al. 1999; Smith and Brouwer, 2005). Conversely, a deep tissue massage can evoke multidimensional sensory pathways including mechanoreceptors, proprioceptors and muscle nociceptors mediated by group III and IV afferents (Goldberg et al. 1992). Several investigators have postulated that activation of Golgi tendon organs, secondary muscle spindle afferents and group III and IV pain receptors can inhibit central excitability in the massaged muscles (Goldberg et al. 1992; Sullivan et al. 1991, 1993; Behm et al. 2013). Interestingly, the magnitude of this inhibitory response was greater following deep tissue massage compared to a light massage (Goldberg et al. 1992). In the present study, the magnitude of mechanical pressure applied during ROLLING was adjusted based on the pain perception. Given that a high amplitude mechanical pressure was administered during ROLLING and participants experienced 7/10 pain sensation, it seems quite plausible to speculate that ROLLING activated a wide range of somatosensory inputs including inhibitory afferent pathways mediated by Golgi tendon organs and muscle nociceptors.

Another intriguing result of the present study was that the MEP and rmsEMG exhibited distinctive responses when neuromuscular evaluations were performed at 10 and 50% of MVC. Specifically, despite that the neuromuscular evaluations at 10% of MVC revealed a depression of VL MEP and rmsEMG responses, the two measures exhibited no difference between ROLLING and CONTROL at 50% of MVC. The reason for this finding remains unclear; however, it can be suggested that the mechanical stimuli exerted by ROLLING had a selective inhibitory effect on the low threshold motoneurones which are contributing to low intensity contractions (10% of MVC). In line with this explanation, Bradbury-Squire and colleagues (2015) showed a reduction in VL EMG activity during a lunge action following 5 sets of 60-s rolling massage intervention. These investigators suggested that the lower EMG could be due to a reduction in the spinal motoneurone excitability. Caution should be taken in accepting this interpretation in the context of the present study because we did not measure spinal motoneurone responses. In fact, the changes in the MEP amplitude and rmsEMG (normalized to Mwave) give access to the excitability of the entire corticospinal pathway (above the neuromuscular junction) including the motor cortical and spinal motoneurones (Gandevia et al. 1999; Taylor et al. 2002). Thus, our data does not specifically determine whether the depression in the central excitability was due to a reduction in the responsiveness of the motor cortical neurons, the spinal motoneurone and/or the corticospinal transmission. Given that we did not find any alteration in the duration of the SP, it could be inferred that the reduction in the central excitability following ROLLING could not be due to a GABAnergic intracortical inhibition. Further studies are required to quantify the effect of rolling massage on the acute and chronic adaptations of the cortical and spinal segments of the central nervous system.

Investigating the influence of rolling massage on maximal force output was not the main purpose of the present study, as our previous experiments had demonstrated that the technique did not alter the maximal force generating capacity (Sullivan et al. 2013; Halperin et al. 2014; Cavanaugh et al. 2017). In line with our previous findings, the MVC force output did not show any significant change following ROLLING. The data suggest that, although rolling massage can modulate the corticospinal excitability responses, it does not cause any change in the maximal force out.



Although the investigators attempted to exert a fairly equal mechanical amplitude and frequency of ROLLING over both VL and VM muscles, it is not clear why the MEP and rmsEMG recorded from the VM did not show similar results to VL. A plausible explanation for different responses of VM and VL might be that the VL is the primary knee extensor during low intensity isometric knee extensions (Zhang et al. 2003). Therefore, our data suggest that different segments of quadriceps muscle may demonstrate various responses to ROLLING depending on the background contraction intensity.

A methodological consideration for the current study is that a 24 to 48 hours interval was assigned between the two intervention sessions. Although there is no documented research that has explored the potential long-term adaptation of corticomotor responses following rolling massage, our cross-over study design warrants further considerations. In addition, the current study does not directly evaluate the influence of ROLLING on activation of muscle spindles and group III and IV afferent receptors located in the quadriceps muscle. Thus, further studies with more sophisticated neurophysiological measurements of afferent and efferent reflexive pathways are required to elucidate the influence of rolling massage on neuromuscular performance.

In conclusion, the results in the present study suggest that the rolling massage technique could modulate the responsiveness of corticospinal circuitries innervating the knee extensor muscles. However, the observed effects were highly dependent on the background knee extension voluntary contractions during which the neuromuscular measurements were recorded.

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**Conflict of interest.** The authors report no conflicts of interest associated with this manuscript.

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**TABLES**

Table 1. The absolute values for the neurophysiological parameters recorded from knee extenosrs (VL and VM) at 10% of MVC at the baseline and following the four sets of the two interventions (CONTROL and ROLLING).

| **VL** | | **Baseline** | **Set 1** | **Set 2** | **Set 3** | **Set 4** |
|---|---|---|---|---|---|---|
| MEP/Mmax * | CONTROL | .27 (.09) | .28 (.13) | .27 (.08) | .28 (.14) | .28 (.08) |
| | ROLLING | .33 (.12) | .34 (.18) | .30 (.15) | .30 (.12) | .29 (.12) |
| rmsEMG/Mmax * | CONTROL | .0062 (.0028) | .0061 (.0028) | .0063 (.0029) | .0060 (.0029) | .0062 (.0030) |
| | ROLLING | .0067 (.0023) | .0059 (.0018) | .0056 (.0015) | .0058 (.0017) | .0057 (.0021) |
| SP (ms) | CONTROL | 167.3 (82.2) | 172.8 (84.7) | 174.9 (85.2) | 169.2 (83.3) | 170.1 (84.4) |
| | ROLLING | 169.4 (81.3) | 177.4 (75.2) | 169.1 (77.1) | 173.8 (78.9) | 180.6 (86.2) |
| **VM** | | **Baseline** | **Set 1** | **Set 2** | **Set 3** | **Set 4** |
| MEP/Mmax | CONTROL | .35 (.25) | .38 (.28) | .33 (.18) | .32 (.24) | .36 (.19) |
| | ROLLING | .44 (.20) | .50 (.33) | .41 (.20) | .47 (.19) | .45 (.25) |
| rmsEMG/Mmax | CONTROL | .0045 (.0016) | .0046 (.0016) | .0047 (.0017) | .0045 (.0014) | .0049 (.0012) |
| | ROLLING | .0060 (.0028) | .0059 (.0028) | .0054 (.0025) | .0058 (.0032) | .0057 (.0029) |
| SP (ms) | CONTROL | 177.4 (84.6) | 185.3 (83.9) | 183.1 (82.4) | 179.6 (84.9) | 179.3 (89.7) |
| | ROLLING | 173.5 (77.3) | 177.8 (81.7) | 177.5 (82.8) | 177.8 (82.4) | 183.2 (81.5) |
| | | **Baseline** | **-** | **-** | **-** | **Post-intervention** |
| MVC force (N) | CONTROL | 659.0 (134.6) | - | - | - | 667.8 (148.2) |
| | ROLLING | 602.5 (68.6) | - | - | - | 603.6 (122.2) |

Note. MEP: motor evoked potential; Mmax: maximal compound muscle action potential; rmsEMG: root mean square of electromyographic activity; SP: silent period; VL: vastus lateralis and VM: vastus medialis; MVC: maximal voluntary knee extensions. * denotes a significant condition effect ($p < .05$).

Table 2. The absolute values for the neurophysiological parameters recorded from knee extenosrs (VL and VM) at 50% 0f MVC at the baseline and following the four sets of the two interventions (CONTROL and ROLLING).

| **VL** | | **Baseline** | **Set 1** | **Set 2** | **Set 3** | **Set 4** |
|---|---|---|---|---|---|---|
| MEP/Mmax | CONTROL | .80 (.19) | .80 (.20) | .77 (.21) | .79 (.26) | .77 (.28) |
| | ROLLING | .72 (.16) | .70 (.19) | .65 (.17) | .73 (.20) | .66 (.22) |
| rmsEMG/Mmax | CONTROL | .029 (.015) | .026 (.014) | .028 (.015) | .028 (.014) | .029 (.016) |
| | ROLLING | .030 (.009) | .032 (.010) | .030 (.006) | .032 (.011) | .031 (.009) |
| SP (ms) | CONTROL | 121.2 (29.1) | 120.4 (29.3) | 123.8 (35.2) | 119.9 (30.1) | 120.5 (33.9) |
| | ROLLING | 111.3 (22.1) | 110.3 (18.9) | 112.4 (81.9) | 112.2 (22.4) | 106.4 (24.1) |
| **VM** | | **Baseline** | **Set 1** | **Set 2** | **Set 3** | **Set 4** |
| MEP/Mmax | CONTROL | .73 (.18) | .72 (.24) | .72 (.20) | .72 (.20) | .68 (.18) |
| | ROLLING | .64 (.16) | .59 (.16) | .59 (.11) | .62 (.17) | .57 (.14) |
| rmsEMG/Mmax | CONTROL | .032 (.015) | .029 (.014) | .031 (.017) | .031 (.018) | .031 (.017) |
| | ROLLING | .027 (.009) | .029 (.010) | .025 (.008) | .028 (.009) | .029 (.011) |
| SP (ms) | CONTROL | 118.4 (29.6) | 115.8 (29.9) | 120.4 (36.9) | 117.8 (31.6) | 116.2 (32.0) |
| | ROLLING | 111.9 (26.3) | 109.1 (19.8) | 109.3 (22.9) | 109.4 (22.0) | 105.6 (25.2) |
| | | **Baseline** | **-** | **-** | **-** | **Post-intervention** |
| MVC force (N) | CONTROL | 695.6 (111.6) | - | - | - | 737.9 (97.0) |
| | ROLLING | 750.4 (129.8) | - | - | - | 725.3 (108.4) |

Note. MEP: motor evoked potential; Mmax: maximal compound muscle action potential; rmsEMG: root mean square of electromyographic activity; SP: silent period; VL: vastus lateralis and VM: vastus medialis; MVC: maximal voluntary knee extensions.

**Figure 1**. Representative traces from a single subject for the MEPs and Mmax recorded from VL at 10% of MVC at the baseline and following each set of intervention (CONTROL and ROLLING). MEP: motor evoked potentials; Mmax: compound muscle action potential.

**Figure 2.** The mean and SD of MEPs (normalized to Mwave) recorded from VL at 10% (panel A) and 50% MVCs (panel B). * denotes a significantly lower value (*P = 0.046*) during the ROLLING compared to the CONTROL session.

**Figure 3.** The mean and SD of rmsEMG (normalized to Mwave) recorded from VL at 10% (panel A) and 50% MVCs (panel B). * denotes a significantly lower value (*P = 0.041*) following the ROLLING compared to the CONTROL session.

**FIGURE 1**

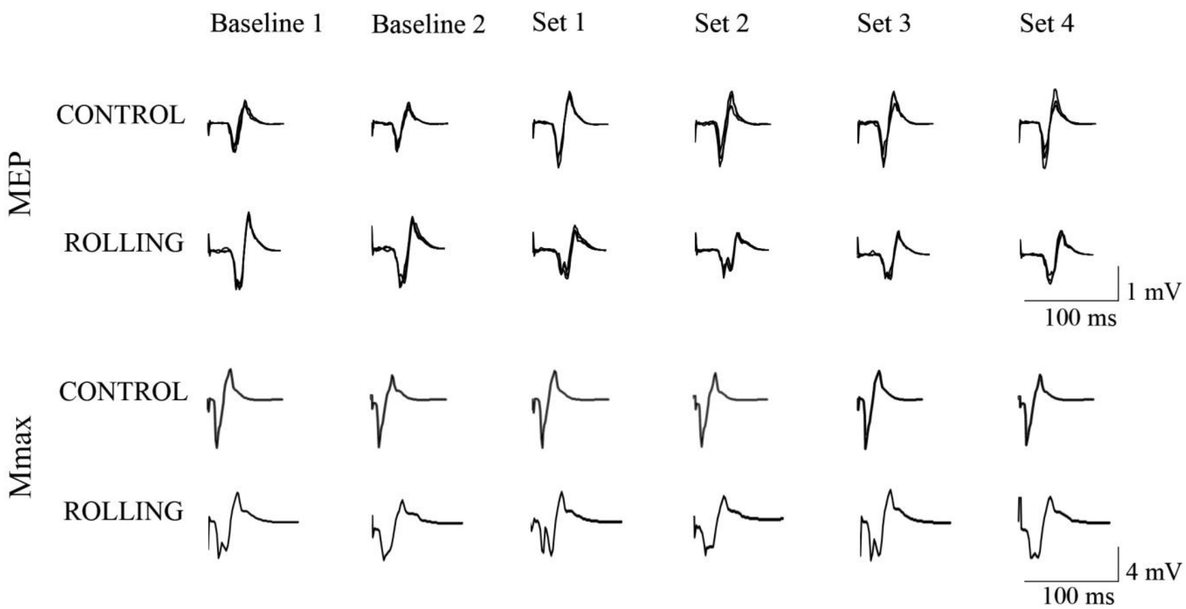

Baseline 1
Baseline 2
Set 1
Set 2
Set 3
Set 4
MEP
CONTROL
ROLLING
1 mV
100 ms
Mmax
CONTROL
ROLLING
4 mV
100 ms

**FIGURE 2**

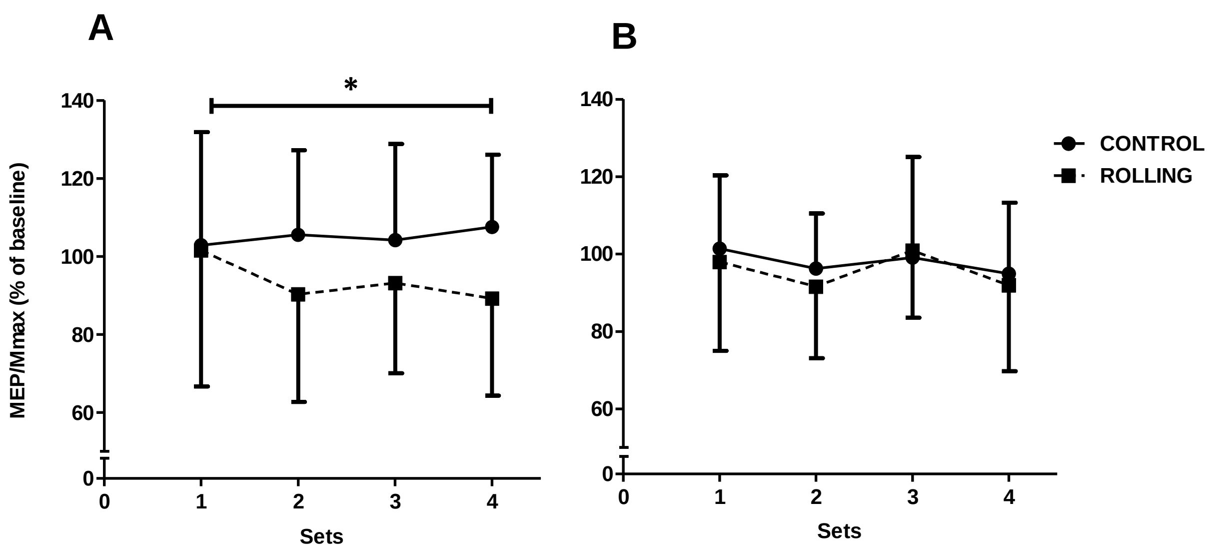
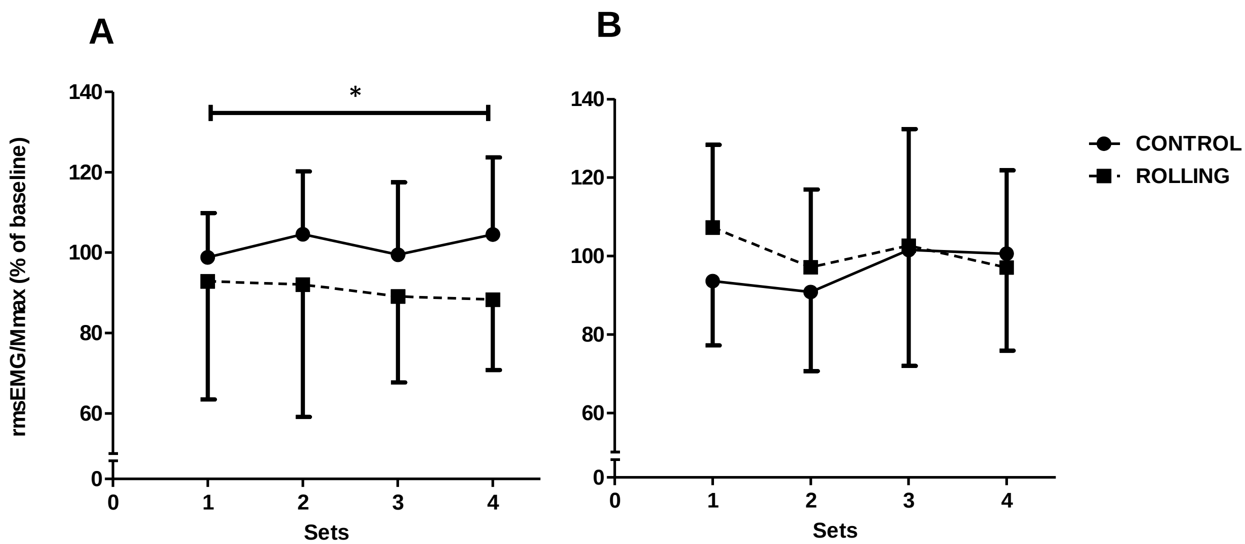

A
B
*
140
120
100
80
60
0
MEP/Mmax (% of baseline)
0
1
2
3
4
Sets
CONTROL
ROLLING

**FIGURE 3**


A
B
*
140
120
100
80
60
0
rmsEMG/Mmax (% of baseline)
0
1
2
3
4
Sets
CONTROL
ROLLING