

Determining the Corticospinal and Neuromuscular Responses Following a Warm-Up Protocol

Rhys Painter MHP(Sport)
Alan Pearce Ph.D.
Mohamad Rostami MS

Department of Physiotherapy, Monash University, Australia
School of Allied Health, La Trobe University, Australia.
Center on Aging, Department of Physiotherapy, University
of Social Welfare and Rehabilitation Sciences, Iran
Department of Physiotherapy, Monash University, Australia
Department of Physiotherapy, Monash University, Australia

Ashlyn Frazer Ph.D.
Dawson Kidgell Ph.D.

Background: The effect of warming-up prior to exercise on increased neuromuscular transmission speed remains largely untested. **Objective:** This study used transcranial magnetic stimulation (TMS) and peripheral nerve stimulation (PNS) to quantify neuromuscular transmission along the corticospinal tract (CST) before and after a warm-up protocol of the elbow flexors. **Method:** Using a single-group, pre-test-post-test design, 30 participants (20 male; 10 female; mean age 26.3 ± 7.4 years) completed four sets of bicep curls that aimed to increase heart rate (HR) and biceps brachii (BB) muscle temperature by a minimum of 40 beats per minute (bpm) and 1°C , respectively. Single-pulse TMS was applied to the primary motor cortex, and over the cervical and thoracic (C7-T1) areas of the spine to quantify motor evoked potentials (MEPs) and spinal evoked potentials (SEPs), respectively. Central motor conduction time (CMCT) was determined by calculating the difference in latency time of the onset of MEPs and SEPs. Peripheral motor conduction time (PMCT) was calculated following stimuli from Erb's point to the onset of the maximal compound muscle action potential twitch (M_{MAX} latency). M_{MAX} time to peak twitch was also measured. M_{MAX} amplitude was used to normalize the MEP to quantify corticospinal excitability. **Results:** Following the warm-up, significant increases in mean heart rate (44.8 ± 11.7 bpm; $P < 0.001$) and muscle temperature ($1.4 \pm 0.6^{\circ}\text{C}$; $P < 0.001$) were observed. No changes were seen in corticospinal excitability ($P = 0.39$), CMCT ($P = 0.09$), or M_{MAX} latency ($P = 0.24$). However, M_{MAX} time to peak twitch was significantly reduced ($P = 0.003$). **Conclusion:** This study has shown that exercise-based warm-ups improve neuromuscular conduction velocity via thermoregulatory processes that result in the onset of muscle contraction being more rapid, but not as a result of changes in the efficacy of neural transmission along the CST.

Citation

Painter R, Pearce A, Mohamad R, Frazer A, Kidgell D. (2020). Determining the Corticospinal and Neuromuscular Responses Following a Warm-Up Protocol. Journal of Science and Medicine; 2(2): 1-12.

Introduction

Completing a warm-up in preparation for exercise and physical activity has been suggested to improve exercise performance, with systematic evidence showing that pre-exercise activity, between 3 and 10 min, provided a positive effect in improving subsequent exercise and sporting performance [1]. Amongst a number of physiological mechanisms, it has been suggested that neural contributions of nerve conduction such as excitability and velocity can be attributed to improved performance. Narrative reviews on the benefits of warming-up [2-5] have cited the work of Karvonen [6] as evidence that neural conduction improves with warming-up. However, discussion regarding the potential changes in the central nervous system (CNS) following a warm-up is often presented in a generalized manner, without consideration of whether the changes are occurring at supraspinal or spinal levels.

Determining the changes in the CNS is well established, with peripheral nerve stimulation (PNS) [7] often being used to measure changes in nerve conduction obtained from peripheral nerves following different warm-up protocols [8-11]. With the introduction of transcranial magnetic stimulation (TMS) in the mid-1980s [12], it has become possible to measure supraspinal changes to interventions such as corticospinal excitability and central motor conduction velocity safely [13].

TMS has emerged as a technique to provide insight into the synaptic activity of the cortico-cortical circuitry of the primary motor cortex (M1) following different types of exercise interventions. TMS involves placing a magnetic coil on the scalp positioned over the M1. The current generated creates a magnetic field, which induces an electric field in the M1, which stimulates the underlying neurons. TMS activates the axons of corticospinal neurons and intracortical neurons that synapse with the cell bodies of corticospinal cells [14]. Because TMS activates several neuronal elements within the M1, it produces multiple descending volleys (D-waves and I-waves) that occur as a result of direct and indirect activation of corticospinal axons [14]. These descending volleys activate alpha-motoneurons, causing a muscle response termed the motor-evoked potential (MEP). In general, the size of the MEP is a measure of corticospinal excitability.

Single-pulse TMS can also provide a rudimentary measure of corticospinal conduction time by measuring the latency period following the stimulus to the onset of the MEP waveform. However, a more accurate measure of corticospinal conduction velocity is via the technique of central motor conduction time (CMCT) [12]. Amongst a number of different methods to measure CMCT [15], the simplest is measuring the difference in latency between the MEP waveform following magnetic stimulation of M1 and evoked potentials following spinal motor root magnetic stimulation [16], both of which are also recorded via surface electromyography (sEMG). Both MEP and CMCT data obtained in healthy participants has demonstrated strong reliability [15,17]. In healthy populations under resting conditions, the physiological conduction velocity along the corticospinal tract has been calculated to be between 65-70 m/s [18-21].

Despite general discussion regarding the potential effects of a warm-up on modulating neural transmissions with the CNS [3-5], there are limited studies to date that have determined the effects of a warm-up on the efficacy of neural transmission along the CST. In the first study to address the question regarding central conduction time following a warm-up, Pearce et al. [22] investigated central and peripheral neural conduction, using TMS and compound muscle action potential (M-wave) techniques. They measured the latency period and time to peak twitch in MEPs and M-waves, before and after an aerobic warm-up consisting of five minutes jogging exercise and using heart rate (HR) as the determinant of warming-up. There were no changes in latency of the MEP or M-wave observed. Thus, it was concluded that warming-up does not change central or peripheral neural conduction time. The authors did, however, observe a reduction in M_{MAX} time to peak twitch within the muscle itself, suggesting improved neuromuscular conduction velocity. However, there are several limitations of this study. Firstly, it is important to note that MEP latency measures presented in this previous study do not truly reflect CNS conduction time. MEP latency includes

both a central element (time from M1 stimulation to spinal motor neurons) and a peripheral element (time from spinal motor neurons to the muscle twitch). Thus, it is worthwhile to employ a technique that can segregate conduction time between the central and peripheral nervous systems following a warm-up. Using CMCT will provide a more rigorous measure than MEP latency alone. A second limitation of the study was that, despite HR changes being present, there was no temperature measurement taken to actually confirm that participants were suitably warmed-up, particularly in the muscles targeted by TMS measurements. Finally, no MEP amplitude data were presented; thus, it is not known if warming up has an effect on the efficacy of neural conduction along the CST. Therefore, the corticospinal responses to warming-up remain untested.

The aim of the present study was to investigate both central (corticospinal excitability and CMCT) and peripheral (M-wave) measures of neural conduction following a strength-based warm-up of the upper-limb muscles. Further, along with HR changes, local muscle temperature (T_{muscle}) was measured to ensure that indeed the muscle targeted by TMS and M-wave was suitably warmed-up. The hypotheses were: 1) a strength-based exercise warm-up would increase HR and T_{muscle} ; 2) corticospinal excitability would increase, as observed by an increase in MEP amplitude; and 3) that neuromuscular conduction times would reduce reflecting increased neural transmission speed to the exercised muscle.

Materials and Methods

To quantify central and peripheral neural excitability and conduction velocities, the study employed a single group pre-test-post-test design with healthy participants. In order to test the hypotheses that corticospinal excitability would increase and neural conduction times would decrease following a warm-up, single-pulse TMS and supra-maximal M-wave measures (M_{MAX}) were taken prior to and following one-bout of strength exercise with individually prescribed loads. To ensure that the warm-up protocol was sufficient, participants wore wireless telemetered heart rate monitors, and the temperature of the biceps brachii muscle (T_{muscle}) was quantified using skin thermistors.

Participants

Thirty healthy participants (20 male; 10 female; mean age 26.3 ± 7.4 years; mean height 178.9 ± 11.2 cm; mean weight 83.5 ± 10.8 kg) who were recreationally strength trained and free of musculoskeletal injury and/or neurological conditions were recruited for this study. The sample size was calculated by using peripheral and TMS conduction time effect-size data from previous investigations [9,22] which corresponded to a mean standardized effect size of 0.5. Based upon this effect size, an alpha set at 0.05 and a desired power of 0.80, a minimum of 27 participants was calculated to be required for the study. All participants, but three, were right hand dominant [23].

Participants provided informed written consent prior to the study, and were familiarized with all neuromuscular measures, the exercise protocol (being a traditional bicep curl exercise); the participant's one-repetition maximum was determined during a separate visit to the laboratory prior to the main study. The Institutional Review Board, conforming to the Declaration of Helsinki, approved the study methods.

Surface electromyography, heart-rate and temperature measures, and central and peripheral stimulation

Surface electromyography (sEMG) was used to quantify the evoked potentials (MEPs, SEPs, and M_{MAX}) pre and post warm-up in biceps brachii (Figure 1A). Following preparation of the skin with 0.5% gluconate chlorhexidine alcohol solution, two Ag-AgCl electrodes were placed 2 cm apart over the biceps brachii muscle, with the third reference electrode (ground electrode) placed over the bony prominence at the elbow (lateral epicondyle) adhering to the Non-Invasive Assessment of Muscles (SENIAM) guidelines for sEMG [24]. Recorded sEMG signals (PowerLab 4/35,

ADInstruments, Australia) were amplified (1000x) with bandpass filtering between 10 Hz and 2 kHz and digitized at 1.5 kHz for 500 ms.

Resting HR was measured using wireless telemetry (Polar, Finland) and T_{musc} quantified via a surface thermal sensor (ADInstruments, Australia) placed over the belly of the bicep brachii muscle and insulated from the air via neoprene (Figure 1B). This non-invasive technique to measure muscle temperature has previously demonstrated reliable changes in T_{musc} during the warm-up exercise [25-27]. Resting HR and T_{musc} were determined when there was less than 1 bpm or a 0.1°C change in temperature for 30 s, respectively.

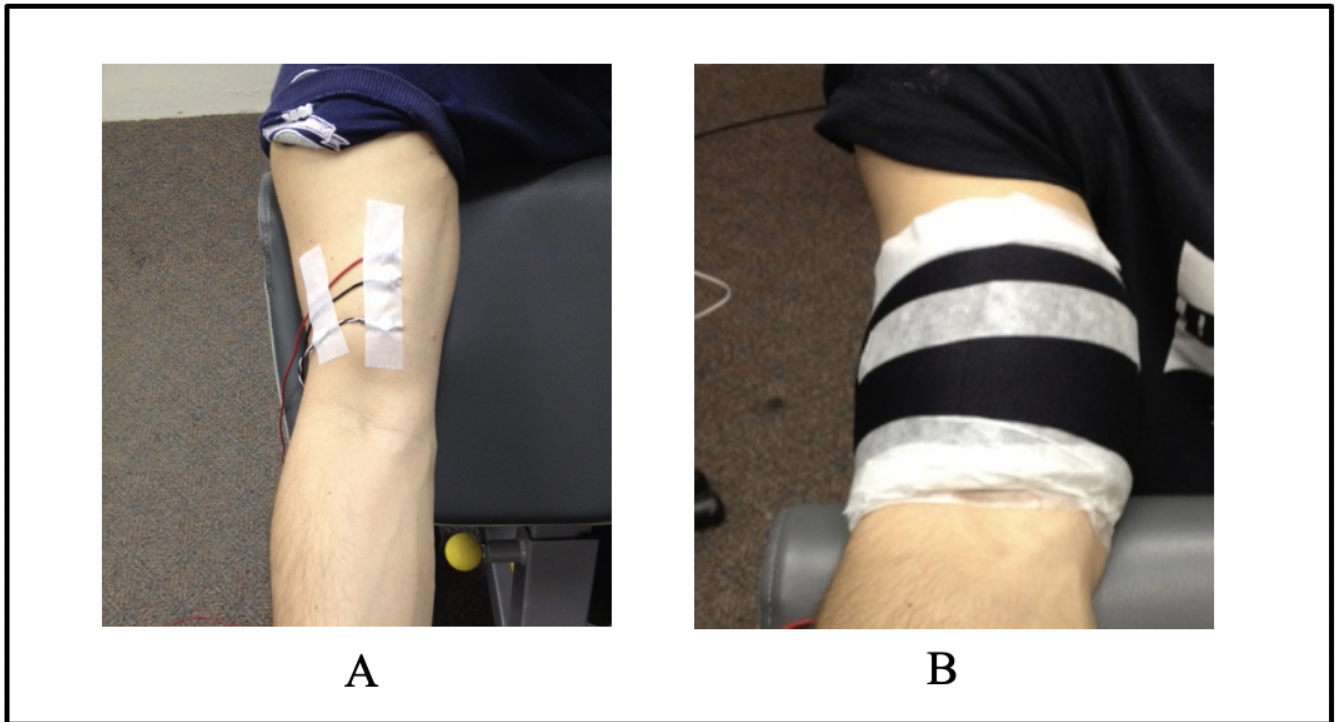


Figure 1. (A) Position of surface electrodes for EMG recording , and (B) example of neoprene insulator placed and taped over EMG electrodes and temperature sensor probes.

A Magstim 200² (Magstim, UK) was used to stimulate the M1 using a 90 mm circular coil (Magstim, UK). For stimulation at the M1, the coil was placed tangential to the skull over the motor area projecting to the biceps brachii in an antero-posterior direction (Figure 2A). To ensure reliability of the coil placement, pre and post warm-up, participants wore a snugly fitting cap marked with sites at 1 cm spacing in a latitude-longitude matrix, positioned with reference to the nasion-inion and interaural lines [28,29]. Sites near the estimated center of the area projecting to the biceps brachii muscle were explored to determine the site at which the largest MEP amplitude was observed. This site was defined as the “optimal” site [30]. Resting motor threshold (RMT) for each individual was determined by presenting five stimuli at stimulus intensity below participant’s RMT, increasing at 5% (of stimulator output) steps until no further increase in MEP amplitude was observed [31]. RMT threshold was determined as the minimum stimulus intensity to produce repeatable MEPs of 50 μV in at least 50% of five trials [15].

Peripheral motor conduction time via magnetic stimulation (PMCT-M) of the spinal nerve root evoked potentials was conducted by placing the circular coil over the spinous processes of the cervical and thoracic (C7-T1) areas of the spine [32] (Figure 2B).

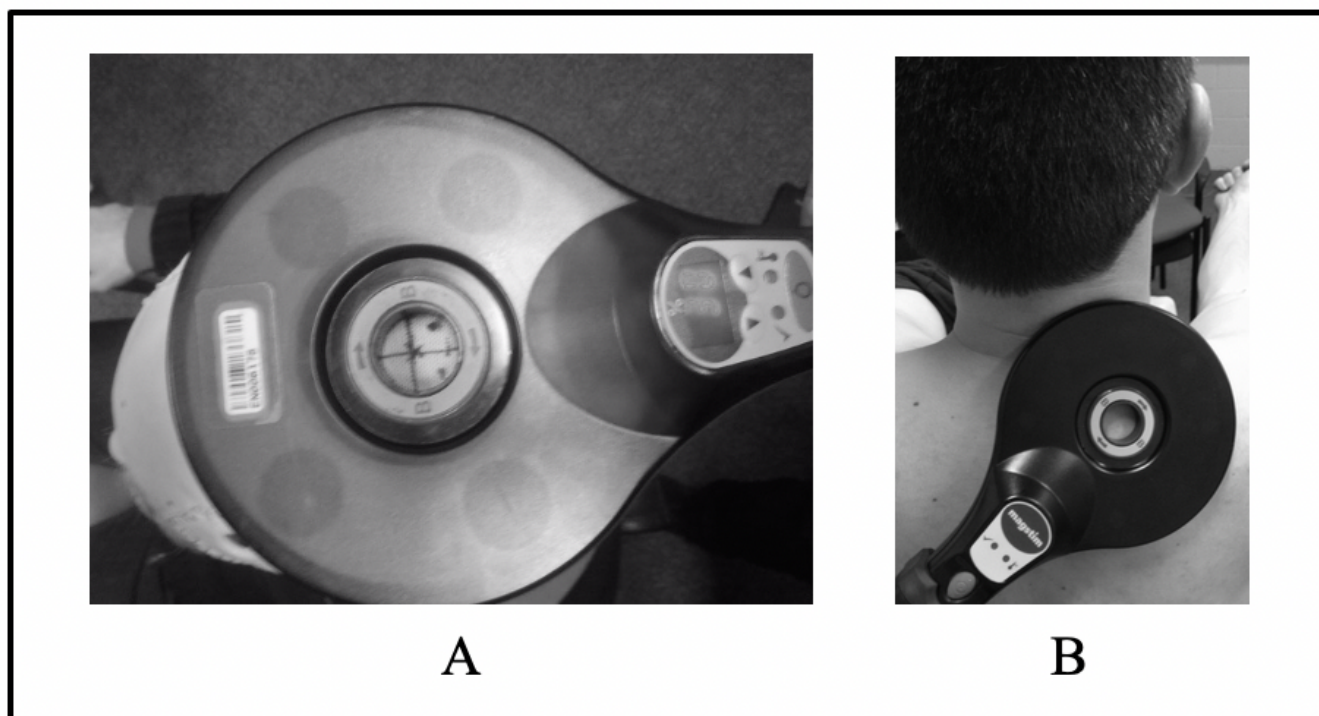


Figure 2. (A) Placement of the circular 90 mm coil over the participant's head. In order to maintain reliability of the coil placement, the participant is wearing a snugly fitted cap with identified markings 1 cm x 1 cm in all directions. (B) Placement of the circular 90 mm coil over the cervical and thoracic (C7-T1) areas of the spine.

Direct muscle responses were obtained from biceps brachii muscle by supramaximal electrical stimulation (pulse width, 200 μ s) of the brachial plexus at Erb's point (DS7A; Digitimer, Hertfordshire, United Kingdom). The stimuli were delivered while the participant sat in an upright position, with the elbow in a neutral position. An increase in current strength was applied to Erb's point until there was no further increase observed in the amplitude of the sEMG response (M_{MAX}). To ensure maximal responses, the current was increased an additional 20% and the average M_{MAX} was obtained from five stimuli, with a period of 6–9 s separating each stimulus. M_{MAX} was recorded at baseline and following the warm-up intervention.



Figure 3. *Placement of the stimulator over Erb's point for M-wave stimulation.*

Experimental Protocol

Prior to the warm-up, ten MEPs and SEPs were delivered at 150% (of stimulator output) above RMT respectively [15]. Ten peripheral stimuli were obtained prior to the intervention at 20% above M_{MAX} . To avoid stimulus anticipation, stimuli were presented at random intervals between 6-8 s apart.

The warm-up consisted of participants completing a traditional barbell bicep curl on a preacher bench. Each participant performed four sets of 15 repetitions at approximately 60% of their one-repetition max [33], with the weight being lifted calculated previously during familiarization. Participants were instructed to exercise at a tempo of 1 s from full extension to flexion and then 1 s from full flexion to extension with no pause between flexion and extension. The exercise intervention was monitored by the investigators to ensure correct technique was performed. The intervention was completed in less than ten minutes in accordance with the meta-analysis conducted by Fradkin et al. [1] and to reduce the potential for muscular fatigue. Central and peripheral nerve measures were reassessed immediately following the completion of the warm-up exercise [22].

Data and Statistical Analyses

Latency time for MEPs, SEPs and M_{MAX} were measured from the TMS stimulus artifact to the onset of the waveform. CMCT was calculated via the difference in latency time in MEP (MEP latency) and spinal root magnetic evoked potentials (PMCT-M) using the formula [15]:

$$CMCT = MEP \text{ latency} - PMCT-M$$

MEP amplitude was quantified by measuring the peak-to-peak amplitude of the signal, averaged and then normalized as a percentage of the M_{MAX} . For comparison to previous work [22], latency and time to peak twitch of the M_{MAX} were also measured. HR and T_{musc} were measured at rest and at the time of TMS and M_{MAX} measurements following the warm-up protocol.

The dependent variables of CMCT, M_{MAX} latency, normalized MEP amplitude, HR, and T_{musc} were first screened to ensure they were normally distributed. No variable's z-score of skew or kurtosis was observed to be excessive. Further, Shapiro-Wilk tests showed that the data were normally distributed (SW static range: 0.90-0.98; $P \geq 0.05$). Pre/post data were compared using paired t tests. Effect size (ES) conventions compared the magnitude of difference between pre and post means using the criteria by Hopkins [34] (trivial ≤ 0.20 ; small 0.21 – 0.59; moderate 0.60 – 1.19; large ≥ 1.20). Statistical significance was accepted at an alpha level $P < 0.05$, and all results are presented as means ($\pm$ SD).

Results

Table 1 illustrates the data of all dependent variables pre and post warm-up. There was a significant group mean increase in HR of 44.8 bpm (± 11.7 bpm) post warm-up ($t(29) = 18.36$; $P < 0.001$; $ES = 4.3$). Similarly, there was a significant group mean increase of 1.42° C (± 0.68 ° C) in T_{musc} ($t(29) = 8.89$; $P < 0.001$; $ES = 2.1$).

No changes were observed in normalized MEP amplitude following the warm-up with a group mean change of 1.5% ($\pm 7.3\%$; $t(29) = 0.87$; $P = 0.39$; $ES = 0.2$). CMCT did not change with a group mean difference in CMCT of 0.17 ms (± 0.39 ms) following the warm up ($t(29) = 1.815$; $P = 0.09$; $ES = 0.4$).

No difference was found in M_{MAX} latency, with a mean difference of 0.04 ms (± 0.15 ms; $t(29) = 1.213$; $P = 0.24$; $ES = 0.2$). However, time to peak twitch of the M_{MAX} was observed to have significantly reduced, with a mean difference in time to peak twitch of 1.07 ms (± 0.82 ms; $t(29) = 3.46$; $P = 0.003$; $ES = 0.8$). Figure 4 illustrates an example in one participant of reduced time to peak twitch.

	Pre	Post
Heart rate (bpm)	73.1 $\pm$ 14.3	118.9 $\pm$ 17.8*
T_{muscle} ($^{\circ}$ C)	32.0 $\pm$ 0.8	33.4 $\pm$ 0.8*
Normalised MEP amplitude (% M_{max})	15.4 $\pm$ 9.9	13.9 $\pm$ 11.1
MEP latency (ms)	12.1 $\pm$ 1.2	12.3 $\pm$ 1.1
PMCT-M latency (ms)	5.7 $\pm$ 1.0	5.6 $\pm$ 0.9
CMCT (ms)	6.4 $\pm$ 1.2	6.5 $\pm$ 1.1
M_{max} latency (ms)	5.2 $\pm$ 0.5	5.3 $\pm$ 0.5
M_{max} peak twitch (ms)	15.4 $\pm$ 1.5	14.4 $\pm$ 1.2*

Table 1. Group mean ($\pm$ SD) data for all dependent variables pre and post the strength-based warm up. * $P < 0.01$ (two-tailed t-test)

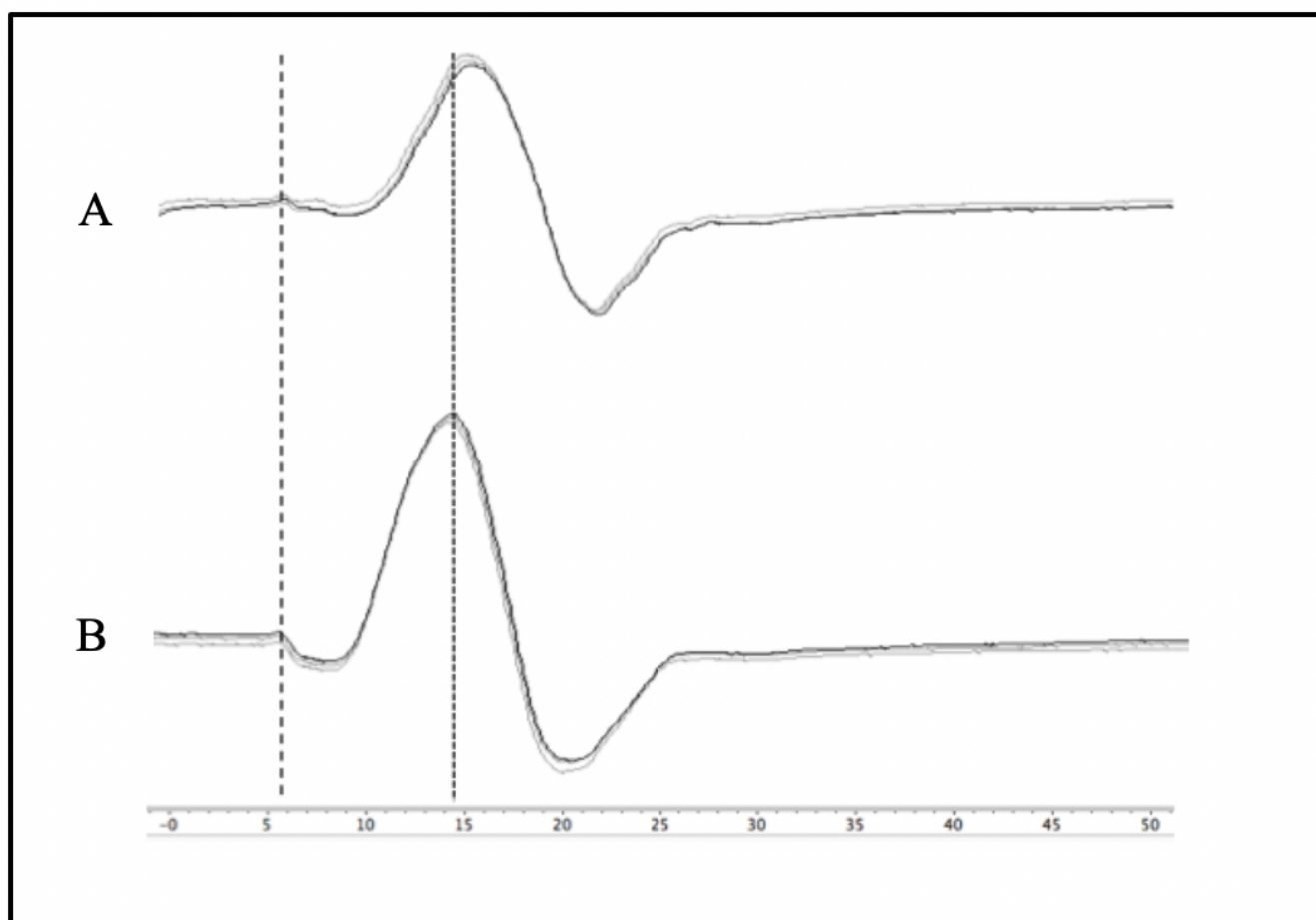


Figure 4. M_{MAX} waveform pre warm up (A) and M_{MAX} post warm up (B). Dashed line represents latency onset of waveform, and dotted line represents peak twitch.

Discussion

The results from the present study show that a strength-based warm-up, that increased the participants' mean HR by 44.8 bpm and T_{musc} of 1.38°C, had no effect on modulating the efficacy of neural transmission along the CST. However, we did observe a change in neuromuscular activation whereby the time to M_{MAX} peak twitch reduced by 7% following the warm-up.

Although no change was observed in corticospinal excitability or CMCT following the warm-up, the findings from this study are important as many warm-up routines are based upon current supposition regarding facilitation of nervous system activity in preparation for training or competition [3,4]. However, particularly with CNS responses, this is the only study that has attempted to address this hypothesis.

Contrary to the hypothesis that corticospinal excitability would increase following the warm-up, no change in MEP amplitude was observed. It is interesting that, apart from one study [22] to date, no research has examined changes in corticospinal excitability with pre-activity warm-up exercise. In an attempt to provide a directional hypothesis for this study, it was proposed that corticospinal excitability would increase following the warm-up; it seems that a warm-up does not improve the efficacy of neural condition along the CST. In fact, this finding is consistent with the acute-strength-training literature whereby a single bout of strength training has no effect on cortical excitability [35].

Although previous investigations have presented MEP latency data following a warm-up (e.g., Pearce et al [22].), MEP latency is a combination of central and peripheral measures and therefore not an accurate measure of CMCT. Thus, it was necessary that the present study examined the effects of a warm-up on CMCT by using a more robust technique. Given that we used a valid technique to measure CMCT and that we observed no change in CMCT or M_{MAX} latency, the present study concurs with a previous study [28] that neural conduction speeds do not change following a warm-up, despite significant changes in thermoregulatory processes (increases in HR and T_{musc}). A plausible explanation as to why CMCT did not alter after warming-up may be due to corticospinal neuron transmission speed being stable in healthy people, only altering (i.e., slowing) under certain pathologies such as motor neuron disease and multiple sclerosis [15,16].

Despite no change being observed in CMCT or corticospinal excitability following the warm-up, there was a change in time to peak twitch in the M_{MAX} waveform that was significantly reduced by 7%. At a minimum, this suggests that warming-up affects the peripheral nervous system by improving the onset of muscle contraction. This line of inquiry is in agreement with previous investigations that have presented reduced times to peak twitch following warming-up. For example, Van der Hoeven and Lange [9] showed a change of approximately 5% following intermittent isometric exercise in the biceps brachii muscle. More recently, Girard et al. [8] presented reductions in peak twitch time of both vastus medialis and lateralis muscles of approximately 12% following both running and strength exercise warm-ups; whilst Pearce et al [22] observed a reduction in M_{MAX} peak twitch time of ~5% and ~8.5% in the abductor pollicis brevis and gastrocnemius muscles, respectively after an aerobic-based warm-up. Collectively, these studies demonstrate that warming up improves neuromuscular conduction. Although more likely relating to strength training adaptations, neuromuscular mechanisms contributing to neuromuscular contractile changes may include motor unit behavior, such as recruitment and rate coding patterns. [36] However, for acute responses underpinning neuromuscular conduction, physiological mechanisms may be more likely to be the driving factor such as membrane hyperpolarization via increased Na^+/K^+ pumping activity [37,38] and muscle fibre swelling [39]. Greater increase in adenosine triphosphate turnover [11] may also occur, leading to individual sarcomeres becoming more rapidly activated. As a result, a combination of factors may contribute to the increase in contractile speed of the whole fibre.

In summary, this study has shown that warming up improves peripheral neuromuscular excitability with reduced time to peak twitch, but does not affect central measures such as corticospinal excitability or CMCT. Future investigations should focus on the peripheral neuromuscular responses to dynamic warm-ups, including power-based exercise, and with practices such as pre-cooling prior to warming up. Although corticospinal transmission may not alter, future investigations could explore the mechanisms supporting cognitive alertness changes following exercise using such techniques including electroencephalography or near-infrared spectroscopy that can provide data on non-motor areas of the brain.

Conclusion

The increase in HR and T_{musc} in this study provides evidence that the participants sufficiently warmed up, which improved neuromuscular excitability, but had no effect on corticospinal excitability and CMCT. Nevertheless, the present study does provide some practical implications. Firstly, pre-activity warm-up should be viewed as a method to sufficiently improve neuromuscular, rather than CNS, activation. Warm-up exercises prescribed can include both aerobic and strength-based activities, with the focus for athletes (at all levels of participation) placed on increasing HR and muscular temperature. Secondly, extending on previous findings [1], warm-up practices need only be short in duration (for example 10 min) to elicit improved neuromuscular excitability; this is important when prescribing warm-up exercises for events and activities that are limited in time or space to complete warm-ups.

Acknowledgments

The authors would like to thank Ms. Helen de Bree and Mr. Philip Seymour for previous contribution of partial data collection to this study.

Author Disclosure Statement

No funding was provided for the completion of this work. No competing financial interests exist.

References

1. Fradkin Andrea J, Zazryn Tsharni R, Smoliga James M. Effects of Warming-up on Physical Performance: A Systematic Review With Meta-analysis. *Journal of Strength and Conditioning Research*. 2010; 24(1)[DOI](#)
2. Volianitis S, Koutedakis Y, Carson RJ. Warm-up: A brief review. *Journal of Dance Medicine & Science*. 2001; 5(3):75-81.
3. Bishop David. Warm Up I. *Sports Medicine*. 2003; 33(6)[DOI](#)
4. Bishop David. Warm Up II. *Sports Medicine*. 2003; 33(7)[DOI](#)
5. Knudson DV. Warm-up and flexibility. In: Chandler TJ, Brown LE. *Conditioning for Strength and Human Performance*. Lippincott, Williams and Wilkins: Lippincott, Williams and Wilkins; 2007:166-181. [DOI](#)
6. Karvonen J. Importance of Warm-Up and Cool Down on Exercise Performance. In: Karvonen J, Lemon PWR, Iliev I. *Medicine in Sports Training and Coaching*. Karger Publishers; 1992:189-214. [DOI](#)
7. Schieppati Marco. The Hoffmann reflex: A means of assessing spinal reflex excitability and its descending control in man. *Progress in Neurobiology*. 1987; 28(4)[DOI](#)
8. Girard Olivier, Carbonnel Yvan, Candau Robin, Millet Grégoire. Running versus strength-based warm-up: acute effects on isometric knee extension function. *European Journal of Applied Physiology*. 2009; 106(4)[DOI](#)
9. Van der Hoeven J, Lange F. Supernormal Muscle Fiber Conduction Velocity During Intermittent Isometric Exercise in Human Muscle. *Journal of Applied Physiology*. 1944;

- 77(2):802-806. [DOI](#)
10. Farina Dario, Arendt-Nielsen Lars, Graven-Nielsen Thomas. Effect of temperature on spike-triggered average torque and electrophysiological properties of low-threshold motor units. *Journal of Applied Physiology*. 2005; 99(1)[DOI](#)
 11. Gray SR, De Vito G, Nimmo MA, Farina D, Ferguson RA. Skeletal muscle ATP turnover and muscle fiber conduction velocity are elevated at higher muscle temperatures during maximal power output development in humans. *Am J Physiol Regul Integr Comp Physiol*. 2006; 290(2):376-382. [DOI](#)
 12. Barker A.T., Freeston I.L., Jalinous R., Jarratt J.A.. Clinical Evaluation of Conduction Time Measurements in Central Motor Pathways Using Magnetic Stimulation of Human Brain. *The Lancet*. 1986; 327(8493)[DOI](#)
 13. Rossi Simone, Hallett Mark, Rossini Paolo M., Pascual-Leone Alvaro. Safety, ethical considerations, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research. *Clinical Neurophysiology*. 2009; 120(12)[DOI](#)
 14. Kidgell Dawson J, Pearce Alan J. What Has Transcranial Magnetic Stimulation Taught Us About Neural Adaptations To Strength Training? A Brief Review. *Journal of Strength and Conditioning Research*. 2011; 25(11)[DOI](#)
 15. Sandbrink F. The MEP in clinical neurodiagnosis. In: Wasserman EM, Epstein CM, Ziemann U, Walsh V, Paus T, Lisanby SH. *The Oxford Handbook of Transcranial Stimulation*. Oxford University Press: Oxford University Press; 2008:237-283. [DOI](#)
 16. Kobayashi Masahito, Pascual-Leone Alvaro. Transcranial magnetic stimulation in neurology. *The Lancet Neurology*. 2003; 2(3)[DOI](#)
 17. van Hedel Hubertus J. A., Murer Christian, Dietz Volker, Curt Armin. The amplitude of lower leg motor evoked potentials is a reliable measure when controlled for torque and motor task. *Journal of Neurology*. 2007; 254(8)[DOI](#)
 18. Rothwell J. C., Thompson P. D., Day B. L., Dick J. P. R., Kachi T., Cowan J. M. A., Marsden C. D.. Motor cortex stimulation in intact man: 1 General characteristics of EMG responses in different muscles.. *Brain*. 1987; 110(5)[DOI](#)
 19. Eisen Andrew A., Shtybel Wayne. AAEM minimonograph #35: Clinical experience with transcranial magnetic stimulation. *Muscle & Nerve*. 1990; 13(11)[DOI](#)
 20. Ugawa Yoshikazu, Genba-Shimizu Kieko, Kanazawa Ichiro. Electrical Stimulation of the Human Descending Motor Tracts at Several Levels. *Canadian Journal of Neurological Sciences / Journal Canadien des Sciences Neurologiques*. 1995; 22(1)[DOI](#)
 21. Earl J.E, Schmitz R.J, Arnold B.L. Activation of the VMO and VL during dynamic mini-squat exercises with and without isometric hip adduction. *Journal of Electromyography and Kinesiology*. 2001; 11(6)[DOI](#)
 22. Pearce Alan J., Rowe Grant S., Whyte Douglas G.. Neural conduction and excitability following a simple warm up. *Journal of Science and Medicine in Sport*. 2012; 15(2)[DOI](#)
 23. Bryden M.P.. Measuring handedness with questionnaires. *Neuropsychologia*. 1977; 15(4-5)[DOI](#)
 24. Hermens HJ, Freriks B, Merletti R, Stegeman D, Blok J, Rau G, Disselhorst-Klug C, Hägg G. European Recommendations for Surface ElectroMyoGraph. Roessingh Research and Development: The Netherlands; 1999.
 25. Brajkovic Dragan, Ducharme Michel B., Webb Paul, Reardon Frank D., Kenny Glen P.. Insulation disks on the skin to estimate muscle temperature. *European Journal of Applied Physiology*. 2006; 97(6)[DOI](#)
 26. Tomaras Elias K., MacIntosh Brian R.. Less is more: standard warm-up causes fatigue and less warm-up permits greater cycling power output. *Journal of Applied Physiology*. 2011; 111(1)[DOI](#)
 27. Cheung SS. Neuromuscular Response to Exercise Heat Stress. *Med Sci Sport Exer.*. 2008; 53:39-60. [DOI](#)
 28. Pearce A. J., Thickbroom G. W., Byrnes M. L., Mastaglia F. L.. Functional reorganisation of the corticomotor projection to the hand in skilled racquet players. *Experimental Brain Research*. 2000; 130(2)[DOI](#)
 29. Kidgell Dawson J., Pearce Alan J.. Corticospinal properties following short-term strength

- training of an intrinsic hand muscle. *Human Movement Science*. 2010; 29(5)[DOI](#)
30. Kidgell Dawson J, Stokes Mark A, Castricum Troy J, Pearce Alan J. Neurophysiological Responses After Short-Term Strength Training of the Biceps Brachii Muscle. *Journal of Strength and Conditioning Research*. 2010; 24(11)[DOI](#)
 31. Pearce Alan J., Clark Ross A., Kidgell Dawson J.. A Comparison of Two Methods in Acquiring Stimulus-Response Curves with Transcranial Magnetic Stimulation. *Brain Stimulation*. 2013; 6(3)[DOI](#)
 32. Samii Ali, Luciano Carlos A., Dambrosia James M., Hallett Mark. Central motor conduction time: Reproducibility and discomfort of different methods. *Muscle & Nerve*. 1998; 21(11)[DOI](#)
 33. Baechle TR, Earle RW, Wathen D. Calculating estimated one repetition maximum. *Essentials of strength training and conditioning*. Human Kinetics Publishers: Human Kinetics Publishers; 2008:395-425.
 34. Hopkins W. A Scale of Magnitudes for Effect Statistics 2002. *A new view of statistics*.
 35. Mason Joel, Frazer Ashlyn K., Pearce Alan J., Goodwill Alicia M., Howatson Glyn, Jaberzadeh Shapour, Kidgell Dawson J.. Determining the early corticospinal-motoneuronal responses to strength training: a systematic review and meta-analysis. *Reviews in the Neurosciences*. 2019; 30(5)[DOI](#)
 36. Duchateau Jacques, Semmler John G., Enoka Roger M.. Training adaptations in the behavior of human motor units. *Journal of Applied Physiology*. 2006; 101(6)[DOI](#)
 37. Hicks A., Fenton J., Garner S., McComas A. J.. M wave potentiation during and after muscle activity. *Journal of Applied Physiology*. 1989; 66(6)[DOI](#)
 38. Hess Christian W., Mills Kerry R., Murray Nicholas M. F., Schriefer Thomas N.. Magnetic brain stimulation: Central motor conduction studies in multiple sclerosis. *Annals of Neurology*. 1987; 22(6)[DOI](#)
 39. van der Hoeven J. H., Van Weerden T. W., Zwarts M. J.. Long-lasting supernormal conduction velocity after sustained maximal isometric contraction in human muscle. *Muscle & Nerve*. 1993; 16(3)[DOI](#)