Can passive stretch inhibit motoneuron facilitation in the human plantar flexors?

Gabriel S. Trajano, Laurent B. Seitz, Kazunori Nosaka, and Anthony J. Blazevich

Centre for Exercise and Sports Science Research, School of Exercise and Health Sciences, Edith Cowan University, Joondalup, Western Australia, Australia

Submitted 9 September 2014; accepted in final form 16 October 2014

Can passive stretch inhibit motoneuron facilitation in the human plantar flexors? J Appl Physiol 117: 1486–1492, 2014. First published October 23, 2014; doi:10.1152/japplphysiol.00809.2014.—The purpose of the present study was to examine the possible inhibitory effect of passive plantar flexor muscle stretching on the motoneuron facilitatory system. Achilles tendon vibration (70 Hz) and triceps surae electrical stimulation (20 Hz) were imposed simultaneously in 11 subjects to elicit contraction through reflexive pathways in two experiments. In experiment 1, a vibration-stimulation protocol was implemented with the ankle joint plantar flexed (+10°), neutral (0°), and dorsiflexed (−10°). In experiment 2, the vibration-stimulation protocol was performed twice before (control), then immediately, 5, 10, and 15 min after a 5-min intermittent muscle stretch protocol. Plantar flexor torque and medial and lateral gastrocnemius and soleus (EMGmed, EMGsoleus) EMG amplitudes measured during and after (i.e., self-sustained motor unit firing) the vibration protocol were used as an indicator of this facilitatory pathway. In experiment 1, vibration torque, self-sustained torque and EMGmed, EMGsoleus were higher with the ankle at −10° compared with 0° and +10°, suggesting that this method is valid to assess motoneuronal facilitation. In experiment 2, torque during vibration was reduced by ~60% immediately after stretch and remained depressed by ~35% at 5 min after stretch (P < 0.05). Self-sustained torque was also reduced by ~65% immediately after stretch (P < 0.05) but recovered by 5 min. Similarly, medial gastrocnemius EMG during vibration was reduced by ~40% immediately after stretch (P < 0.05), and EMGsoleus during the self-sustained torque period was reduced by 44% immediately after stretch (P < 0.05). In conclusion, passive stretch negatively affected the motoneuronal amplification for at least 5 min, suggesting that motoneuron disfacilitation is a possible mechanism influencing the stretch-induced torque loss.

Ia afferent; persistent inward current; muscle force

IT IS WELL ESTABLISHED THAT A bout of passive muscle stretching can acutely reduce maximal force production (31). Several lines of evidence support that a reduction in central drive to the muscle has a considerable involvement in this phenomenon (2, 14, 30, 50, 51). However, the mechanisms underpinning this reduced central drive after stretching remain unclear. Speculatively, stretch-sensitive muscle proprioceptive structures (e.g., group Ia muscle spindle afferents) might be desensitized after prolonged passive stretching, which could ultimately affect one of the motoneuron facilitatory processes. Facilitatory modulation at the motoneuron can be exerted by (1) the development of persistent inward currents (PICs), and/or (2) increased monoaminergic drive, which can depolarize the resting membrane and hyperpolarize the spike threshold (20). PICs are a voltage-dependent characteristic of spinal motoneurons that amplify and prolong synaptic input when activated, changing the input-output relationship and producing sustained depolarization, especially in low-threshold motoneurons (21). This amplification allows the motoneurons to fire at the higher frequencies necessary to produce maximal levels of muscular force (26).

The amplification of motoneuronal responses to excitatory postsynaptic potentials has been studied primarily in animal preparations using steady synaptic input imposed by tendon vibration (19, 28, 34, 35), which selectively activates muscle spindle Ia afferents (40). Despite the fact that animal preparations provide a more controlled environment to study PICs, tendon vibration reflexes have been used in human experiments to improve our understanding of PICs and their influence on muscular force output (41, 48). When a high-frequency vibration is applied to the tendon, it generates a train of Ia afferent impulses, inducing progressive excitation of the homonymous motoneurons and eliciting PICs in these motoneurons (19). The slow increase in involuntarily isometric force during the vibration sequence and, even more, the visibly sustained force that persists after the vibration is ceased provide remarkable evidence for the presence of PICs (21). Another marked characteristic of this amplification is its joint position dependency, where PIC development has been demonstrated to be greater when a muscle receives synaptic input while its antagonist is held at a shortened length (27). Additionally, during repetitive activation, PICs elicit a progressive increase in neuronal excitability, which is commonly referred to as the wind-up effect (3, 49). Thus the presence of a sustained muscular force after vibration cessation, its angle-dependent characteristics, and a progressive augmentation in response to repeated stimulation can be taken as indirect evidence for PIC development in humans.

When performed in isolation, the tendon vibration reflex typically recruits only low-threshold motor units, resulting in small force outputs (17, 29, 32). Recently, however, the imposition of high-frequency tendon vibration during electrically induced muscular contractions has elicited forces as high as 50% of maximal voluntary contraction (MVC), providing evidence of higher-threshold units being recruited in response to the additional input from electrical stimulation (36, 37). This stimulation-vibration technique can provide insights into the presence of PICs, not only in low-threshold motor units but also in higher-threshold units, which contribute more to maximal force production according to the size principle of motor unit recruitment (22). Thus the utilization of electrical stimulation superimposed onto high-frequency tendon vibration provides a unique opportunity to investigate the functional ability of the nervous system to develop PICs in humans, regardless of the exact location of its modulation.
Given that muscle stretching results in an acute central drive depression, a reduction in stretch-dependent afferent feedback after stretch, previously demonstrated as a reduction in H-reflex amplitude (2), might speculatively impact PIC development and thus central (spinal) drive. The main aim of the present study was to examine the effect of muscle stretching on PIC development. The first specific aim, therefore, was to determine whether muscular force and electromyographic (EMG) responses to simultaneous Achilles tendon vibration and muscle electrical stimulation would exhibit joint angle dependency and elicit a stepwise increase in magnitude with repeated stimulation (i.e., a wind-up effect), consistent with PIC-like properties in the human plantar flexors. The second purpose of the present study was to determine whether an acute bout of passive plantar flexor muscle stretching impairs the force and EMG responses to simultaneous tendon vibration and muscle electrical stimulation. It was hypothesized that 1) vibration-induced contractions would be more pronounced at longer muscle lengths and exhibit a wind-up effect; and 2) passive stretch would decrease the reflexive plantar flexor contraction force and triceps surae muscle activity elicited by Achilles tendon vibration.

METHODS

Ethical Approval

The procedures performed during this research were approved by the Edith Cowan University Human Research Ethics Committee and were in agreement with the Declaration of Helsinki. All participants read and signed an informed consent document.

Subjects

Eleven healthy subjects (9 men and 2 women; mean ± SD: age, 28.9 ± 4.7 yr; height, 1.77 ± 0.9 m; body mass, 74.8 ± 8.6 kg) without neuromuscular impairment volunteered for the study. The subjects reported not being engaged in flexibility training for at least 6 mo before the study. The subjects also refrained from vigorous exercise and alcohol consumption for 24 h, and stimulant (e.g., caffeine) use for 12 h, before testing.

Study Design and Overview

All data collection was performed in a single session lasting ~1 h and 30 min, during which the subjects performed two experiments. Prior to experiment 1, the subjects were seated upright in the chair of an isokinetic dynamometer (Biodex System 3 Pro, Biodex Medical System, Shirley, NY) with the knee fully extended (0°) and ankle at neutral (0°) position. They were then instructed to practice four voluntary submaximal isometric plantar flexion contractions (two contractions at 60% and two at 80% of perceived maximal effort) to become familiar with the contractions and to precondition the tendon for subsequent strain (38). This was done to ensure its mechanical properties, and thus vibration transmission, were unaffected by the muscle stretch (43, 30). After practice, two MVCs were performed with a 1-min passive rest interval, and the contraction with the maximum torque was recorded for subsequent analysis. Verbal encouragement and visual feedback were provided during all MVCs. The muscle EMG was also recorded simultaneously from soleus (Sol), medial gastrocnemius (MG) and lateral gastrocnemius (LG).

Experiment 1. Experiment 1 was designed to determine whether the torque produced by the electrical stimulation superimposed on tendon vibration (vib-stim) would exhibit muscle length dependence, which is typically found in animal models and suggested to be a marked PIC characteristic. Thus the knee remained fully extended throughout experiment 1, and the torque and EMG amplitude elicited by vib-stim was evaluated with the ankle in three different joint positions: neutral (0°), plantar flexion (+10°), and dorsiflexion (−10°). These joint angles were chosen because a previous study has reported minimal changes in tendon length (~2%) when the ankle joint was passively rotated through these angles (4). This would minimize the effect of joint rotation on tendon mechanical properties and thus vibration transmission. In this experiment, evidence for a wind-up effect was also sought by examining joint torque production after application of repeated electrical stimulations.

Experiment 2. Experiment 2 was designed to investigate the effect of acute passive stretching on the reflexive torque and muscle activity elicited by vib-stim. Subjects were assessed with the ankle in a dorsiflexed position (−10°) at 5 min (control 1) and 1 min (control 2; prestretch) before the stretching as well as immediately, 5 min, 10 min, and 15 min after.

Tendon Vibration and Superimposed Muscle Stimulation (Experiments 1 and 2)

A constant-current electrical stimulator (DS7, Digitimer, Welwyn Garden City, UK) was used to deliver an electrical square-wave stimulus (1-ms pulse width) to the plantar flexor muscle belly through two self-adhesive electrodes (9 × 5 cm, Dura-Stick II, Chattanooga Group, Hixon, TN). The cathode was placed distal to the popliteal crease and the anode over the distal myotendinous junction of Sol. For all electrical stimulations, the intensity necessary to reach 20% of MVC with a 0.5-s 20-Hz tetanic stimulation was used.

The Achilles tendon was mechanically vibrated at 70 Hz and 1 mm of amplitude by a vibrator (VibraQ, Perth, Western Australia, Australia). The tip of the vibrator was firmly attached to the tendon with a clip to maintain steady pressure at a fixed position on the tendon, and the vibration was applied continuously for 33 s. The pressure of the vibrator alone, without mechanical vibration, did not cause any noticeable increments in resting plantar flexor torque. Ten seconds after vibration onset, five 2-s bursts of 20-Hz electrical stimulation separated by 2-s intervals were also applied (Fig. 1). Five bursts of electrical stimulation were used because PICs have a tendency to grow larger during repeated activation (i.e., wind-up effect) (3, 49). Based on pilot data, greater amounts of reflexive torque could be elicited with five bursts of electrical stimulation, and this progressive increase in torque would be an additional evidence of PICs during the vib-stim protocol.

Voluntary and Evoked Torque Measurements (Experiments 1 and 2)

The peak isometric plantar flexor torque, assessed during MVCs (described previously), was the only voluntary torque measurement used in this study and was used to normalize the EMG (see below) elicited by the vib-stim protocol. The involuntary “reflexive torque” was measured as the mean torque in a 500-ms window at two time points: 1) during vibration 500 ms after the first (last) burst of electrical stimulation [torque vibration (Tvib)]; and 2) 3 s after vibration ceased [torque sustained (Tstim)] (Fig. 1). Also, the torque during vibration after the first burst of electrical stimulation was measured (500-ms window), and the difference between it and Tvib was taken as an indicative of a wind-up effect caused by repetitive stimulation. After the torque returned to baseline levels at each time point, an extra 20 Hz-tetanic stimulation (using the parameters described above) was delivered. Subjects were instructed to relax and not to voluntarily contract the plantar flexors during the vib-stim protocol. As low-frequency electrical stimulation applied to resting muscle usually does not involve reflexive pathways, the peak torque (Tstim_ref) was used to determine whether the stretch protocol affected the muscle’s contractile potential, i.e., the ability to produce torque without central command. All of the torque values were presented as changes from the baseline (resting) value, as plantar flexor muscles impose a small passive torque, even when the muscle is relaxed.
Measurement of Muscle Activity (EMG)

Surface EMG was recorded from Sol, LG, and MG using a bipolar electrode configuration at a 4,000-Hz analog-digital conversion rate (bandwidth 20–450 Hz) using the Bagnoli-8 Main Unit EMG system (DelSys). Electrodes were positioned according to SENIAM’s recommendations (23). The interelectrode distance was 1 cm, and a reference electrode was placed on the lateral malleolus. The skin under the electrodes was shaved, abraded, and cleaned with alcohol to reduce the interelectrode resistance below 5 kΩ. EMG data were also recorded during the stretching maneuvers to ensure that muscle activation remained below 5% of the maximal value; a small muscle activity response is often seen even when the subjects are required to remain completely relaxed (4). Muscle activity was expressed as the root-mean-square EMG amplitude (500-ms averaging window) measured for each muscle [Sol (EMGsOL); LG (EMGLG); and MG (EMGMG)] over the same time period as the torque measurements (Tvib and Tsust). Ankle joint torque, joint angle, and EMG data were simultaneously recorded using LabChart version 6.1.3 software (PowerLab System, ADInstruments, NSW, Australia).

Muscle Stretching Protocol

The stretch procedures were performed on an isokinetic dynamometer with the muscles relaxed. The plantar flexors were stretched five times, separated by 10-s nonstretch intervals, by rotating the ankle into dorsiflexion at 5°/s until a maximal tolerable stretch was attained and then held at the stretched position for 1 min. This 5-min stretch protocol was chosen because previous studies showed that a similar 5-min stretch could reduce maximal voluntary torque and neural drive to the muscle (50, 51).

Statistical Analysis

Separate one-way repeated-measures ANOVAs were performed to compare changes in all variables (Tvib, Tsust, wind-up effect, EMGSOL, EMGLG, EMGMG) at different joint angles (experiment 1; neutral, plantar flexion, and dorsiflexion). Similarly, separate one-way repeated-measured ANOVAs were used to compare changes in Tvib, Tsust, EMGSOL, EMGLG, and EMGMG over time (experiment 2; before and immediately, 5, 10, and 15 min after stretch). Pairwise comparisons were performed as follow-up tests. Statistical significance was set at an α-level of 0.05. Intraclass correlations were computed to evaluate reliability of Tvib and Tsust torque measurement between control 1 and control 2 time points. All data are presented as means ± SD.

RESULTS

Experiment 1

Torque. MVCs were on average 132 ± 23 Nm. There were significant effects of joint angle on Tvib (P = 0.000), Tsust (P = 0.001), and the wind-up effect (P = 0.005). Post hoc analyses revealed that Tvib and Tsust were higher when the ankle joint was in dorsiflexion compared with both plantar flexion (71%, P = 0.000, and 69%, P = 0.004, respectively) and the neutral position (67%, P = 0.01, and 60%, P = 0.007, respectively) (Fig. 2). The wind-up effect was greater (72%, P = 0.005, and 85%, P = 0.008, respectively) when ankle joint was in dorsiflexion compared with plantar flexion and the neutral position.
Muscle activity. There was a significant effect of joint angle on EMGSol amplitude when measured during Tvib ($P = 0.016$) and Tsust ($P = 0.025$) (Fig. 3). Post hoc analyses showed that EMGSol amplitude was 32% ($P = 0.018$) greater when measured during Tvib and 28% ($P = 0.029$) greater when measured during Tsust when the ankle was held in dorsiflexion compared with plantar flexion. Similarly, there was a joint angle effect on EMGLG ($P = 0.045$). Post hoc analyses revealed that it was 27% ($P = 0.046$) greater when measured during Tvib when the muscle was held in dorsiflexion compared with plantar flexion.

Experiment 2

Reflexive torque. Intraclass correlation values describing the reliability of Tvib and Tsust between control 1 and control 2 (i.e., before muscle stretch) were 0.95 and 0.96, respectively, suggesting that the measures were reliable. There was a significant time effect for both torque measures (Tvib, $P = 0.026$; Tsust, $P = 0.04$), with post hoc analyses indicating that Tvib was reduced by 60% ($P = 0.008$) immediately after stretch and remained depressed by 32% ($P = 0.008$) at 5 min after stretch (see Fig. 4). Torque remained elevated after vibration cessation; however, Tsust magnitude was also reduced by 65% ($P = 0.012$) immediately after stretch and remained depressed by 42% at 5 min after stretch ($P = 0.001$). In addition, there was a significant time effect for wind-up ($P = 0.044$), with post hoc analyses revealing that wind-up effect was reduced by 62% ($P = 0.002$) immediately after stretch.

Muscle contractile capacity ($T_{\text{stim,rest}}$). There was no significant time effect ($P = 0.86$) for $T_{\text{stim,rest}}$, suggesting that the muscle’s contractile force capacity was not affected by the stretch protocol.

Muscle activity. A significant time effect was found for EMGMG amplitude when measured during Tvib ($P = 0.039$) and for EMGSol when measured during Tsust ($P = 0.006$). Post hoc analyses revealed that EMGMG amplitude during Tvib was reduced by 41% ($P = 0.018$) immediately after stretch, and EMGSol amplitude measured during Tsust was reduced by 44% immediately after stretch ($P = 0.042$). However, they were both recovered by 5 min after stretch and were increased by 16 and 10% ($P = 0.02$), respectively, by 15 min (Fig. 5).

DISCUSSION

Little is known about the effect of acute passive muscle stretching on motoneuron facilitatory pathways. The novel findings of this study were that 1) the vib+stim protocol showed joint angle dependence of torque production and muscle activity during vib+stim as well as after its cessation, self-sustained firing after stimulation cessation, and a wind-up effect consistent with PIC-like behavior; 2) passive muscle stretching decreased both the torque and muscle activity elic-
itted by the vib+stim protocol; and 3) the poststretch inhibition lasted at least 5 min and was fully recovered by 10 min. These findings support the hypothesis that passive stretching inhibits reflex-induced PIC development in the human plantar flexors.

One important aim of the present study was to test whether the stimulation protocol could elicit contractions consistent with PIC development. To test this hypothesis, a combined vibration-electrical stimulation (vib+stim) protocol was applied with the ankle joint in three different positions (plantar flexion, neutral, and dorsiflexion). Experiments in decerebrate cats reveal a joint angle-dependent modulation of PICs when a steady synaptic input is imposed by tendon vibration (27). This angle-dependent modulation seems to be caused by an increase in disynaptic Ia reciprocal inhibition that occurs when the antagonist muscle (i.e., tibialis anterior) is held at a long length, exciting muscle spindle primary (Ia) afferents and increasing agonist inhibition (27). In the present study, the ankle joint angle-modulated reflexive torque and muscle activity were consistent with expectation, according to the results of studies in animal models (i.e., greater amplification when the agonist muscle is held at a longer length, and thus the antagonist was held shorter). The increase in reflex-induced torque and muscle activity when the ankle was dorsiflexed, together with the apparent self-sustained firing after vibration (and stimulation) cessation and the presence of a wind-up effect caused by repetitive activation, can be taken as indirect evidence of the development of PICs using the present protocol. Previous studies have used tendon vibration to estimate the contribution of PICs in human motoneurons, especially in patients with motor impairment (41, 48), and self-sustained firing behavior has already been reported in the literature after low-intensity contractions elicited by tendon vibration (17, 29, 32). However, this appears to be the first study to demonstrate joint angle-dependent modulation of reflexive contractions evoked by tendon vibration in humans, increasing the body of evidence supporting the possibility that contractions elicited by tendon vibration are mediated by PICs. Also, the significantly greater EMG<sub>Sol</sub> amplitudes observed during T<sub>vib</sub> and T<sub>sust</sub> with the ankle in dorsiflexion suggest that sustained motor unit firing was present during and after vibration cessation. PIC amplification more typically produces self-sustained firing in low-threshold motoneurons (34), and the finding that EMG<sub>Sol</sub>, but not EMG<sub>LG</sub> or EMG<sub>MG</sub>, amplitude was greater in the dorsiflexed position during T<sub>sust</sub> is consistent with this, given that Sol is known to consist predominantly of fibers with a lower recruitment threshold (i.e., type I fibers) (16). Thus the utilization of this protocol as an indirect and relative measure of PIC development in human studies appears to be justified.

In the present study, evidence was presented for the first time for the inhibitory effect of passive muscle stretching on motoneuronal amplification. In fact, the possibility that passive stretching might decrease I<sub>a</sub> afferent efficiency has been dem-

---

**Fig. 5.** A: Sol, MG, and LG EMG amplitude measured after EMG<sub>vib</sub> immediately before, immediately after, and 5, 10, and 15 min after muscle stretching. B: a similar response was observed for EMG<sub>sust</sub>. *P < 0.05 compared with immediately before stretch.

**Fig. 6.** Example of torque data obtained during the vib+stim protocol (torque produced by the electrical stimulation superimposed on tendon vibration) at 1 min before the stretching (A), as well as immediately after (B), and 5 min (C) and 10 min after stretching (D).
Passive Stretch Inhibits Motoneuronal Amplification  ·  Trajano GS et al.

The present data expand on previous findings (2) by showing that moderate-duration (5 min) static muscle stretching impairs the ability to develop PICs in the plantar flexors. It is well known that motoneurons rely on a PIC-mediated facilitatory system that increases the gain of synaptic input to achieve maximal discharge frequency and thus produce maximal levels of muscular force (21, 26). PICs have marked characteristics, such as self-sustained firing, wind-up, and greater amplification when the agonist muscle is held at longer lengths, and these characteristics were demonstrated in the protocol used in the present study to elicit reflexive torque. Reductions in reflexive torque (T\textsubscript{ vib}) production measured during vibration and especially reductions in the ability to sustain the torque without synaptic input (self-sustained torque; T\textsubscript{sust}) can be interpreted as a reduction in PIC development (see Fig. 6). Importantly, T\textsubscript{sust} and T\textsubscript{ vib} were statistically recovered by 5 and 10 min poststretch, respectively, suggesting that the inhibitory effects of passive stretch did not last for longer than 10 min. This temporal profile is consistent with previous data showing that the reduced neural drive associated with force loss in response to muscle stretch should be recovered by at least 15 min poststretch using a similar muscle stretching protocol (50, 51). Unfortunately, voluntary force production was not measured at 5 and 10 min poststretch in previous studies, so a precise temporal comparison cannot be done. Additionally, the clear lack of changes in T\textsubscript{stim,rest} shows that the muscle’s ability to produce force through direct electrical stimulation was not affected, suggesting that any changes in reflexive torque production must have been caused by central rather than peripheral (i.e., muscle based) mechanisms. Moreover, poststretch reductions in EMG\textsubscript{MG} amplitude during T\textsubscript{ vib}, as well as EMG\textsubscript{Sol} amplitude during T\textsubscript{sust}, provide strong evidence for PIC-related reductions in motor unit activity after stretch. It is also interesting to note that an increase in EMG\textsubscript{Sol} amplitude during T\textsubscript{ vib} and T\textsubscript{sust} was found 15 min after stretch, suggesting the possibility of a facilitatory effect subsequent to the initial inhibitory effect. However, increases in muscle force production subsequent to the poststretch force loss have not been previously reported, so the functional significance of this finding is unclear.

Although clear evidence for modulation of PIC decrements with muscle stretching was observed, it was not possible to determine the precise location of this modulation. For example, presynaptic mechanisms could result in a reduced efficiency of the Ia pathway (2), including muscle-spindle desensitization (12), increases in Ia afferent thresholds (18), prolonged presynaptic inhibition (24, 25, 42), and even neurotransmitter depletion at Ia nerve endings (11). Alternatively, postsynaptic mechanisms might involve a prolonged activation of inhibitory interneurons (13, 46). Regarding interneuronal inhibition, another possible mechanism could be the activation of other propriocceptive structures during muscle stretching. For instance, it has been clearly shown in a series of experiments that stretch-sensitive free nerve endings are responsible for the homonymous-inhibitory clasp-knife reflex in response to large-amplitude stretch of the extensor muscles in decerebrate cats, with the inhibitory effects persisting after stretch cessation (5, 6, 7, 8). Thus it is reasonable to speculate that prolonged stretch might also activate free nerve endings in healthy humans, inducing a similar inhibitory mechanism within the spinal circuitry. Also, the contribution of supraspinal mechanisms cannot be ruled out. Human experiments have consistently demonstrated the possible involvement of cortical structures in response to stimulation of stretch-sensitive afferents (9, 10, 39, 47). Therefore, to better understand the precise mechanisms underpinning this prolonged inhibition, further studies should examine the adaptation of spinal circuitry in animal models after passive stretching, as well as determine the possible contribution of supraspinal mechanisms to this phenomenon.

In summary, the present data indicate that motoneuronal facilitation, mediated by PICs, is negatively affected for at least 5 min after prolonged (5 min) passive stretching. This conclusion is based on the significant reduction in the torque elicited by tendon vibration as well as self-sustained torque, with a concomitant reduction in EMG\textsubscript{Sol} and EMG\textsubscript{MG} amplitudes immediately after muscle stretch. The stretch protocol used in this study did not affect the muscle’s ability to produce contractile force, so force changes were not likely of peripheral origin. Future studies may focus on strategies to upregulate PIC activity (e.g., increasing monoaminergic drive) to mitigate the acute force-reducing effects of passive muscle stretching.

ACKNOWLEDGMENTS

The authors thank Dr. Christopher Thompson and Prof. C. J. Heckman for insightful discussions about the manuscript.

GRANTS

This research was funded by the Edith Cowan University PhD fund.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: G.S.T. and A.J.B. conception and design of research; G.S.T. and L.B.S. performed experiments; G.S.T. analyzed data; G.S.T., L.B.S., K.N., and A.J.B. interpreted results of experiments; G.S.T. prepared figures; G.S.T. and A.J.B. drafted manuscript; G.S.T., L.B.S., K.N., and A.J.B. edited and revised manuscript; G.S.T., L.B.S., K.N., and A.J.B. approved final version of manuscript.

REFERENCES


