

Lack of Neuromuscular Origins of Adaptation After a Long-Term Stretching Program

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Context: Static stretching is commonly used during the treatment and rehabilitation of orthopedic injuries to increase joint range of motion (ROM) and muscle flexibility. Understanding the physiological adaptations that occur in the neuromuscular system as a result of long-term stretching may provide insight into the mechanisms responsible for changes in flexibility. **Objective:** To examine possible neurological origins and adaptations in the Ia-reflex pathway that allow for increases in flexibility in ankle ROM, by evaluating the reduction in the synaptic transmission of Ia afferents to the motoneuron pool. **Design:** Repeated-measures, case-controlled study. **Setting:** Sports medicine research laboratory. **Participants:** 40 healthy volunteers with no history of cognitive impairment, neurological impairment, or lower extremity surgery or injury within the previous 12 mo. **Intervention:** Presynaptic and postsynaptic mechanisms were evaluated with a chronic stretching protocol. Twenty subjects stretched 5 times a wk for 6 wk. All subjects were measured at baseline, 3 wk, and 6 wk. **Main Outcome Measures:** Ankle-dorsiflexion ROM, $H_{max}:M_{max}$, presynaptic inhibition, and disynaptic reciprocal inhibition. **Results:** Only ROM had a significant interaction between group and time, whereas the other dependent variables did not show significant differences. The experimental group had significantly improved ROM from baseline to 3 wk (mean 6.2 ± 0.9 , $P < .001$), 3 wk to 6 wk (mean 5.0 ± 0.8 , $P < .001$), and baseline to 6 wk (mean 11.2 ± 0.9 , $P < .001$). **Conclusions:** Ankle dorsiflexion increased by 42.25% after 6 wk of static stretching, but no significant neurological changes resulted at any point of the study, contrasting current literature. Significant neuromuscular origins of adaptation do not exist in the Ia-reflex-pathway components after a long-term stretching program as currently understood. Thus, any increases in flexibility are the result of other factors, potentially mechanical changes or stretch tolerance.

Keywords: ankle, soleus, lower extremity, Hoffmann reflex, flexibility

Clinicians commonly use static-stretching methods to increase joint range of motion and muscle flexibility during the treatment and rehabilitation of orthopedic injuries.^{1,2} Increasing flexibility is commonly considered an important element of athletic performance. Therefore, understanding the physiological adaptations that occur in the neuromuscular system as a result of long-term stretching may provide insight into the mechanisms responsible for changes in flexibility. Current theory suggests that neurological elements are integral to increasing flexibility.² Changes in the neural aspects of stretching are related to and can be assessed by measuring changes in

motoneuron-pool excitability of a given muscle.²⁻⁵ The Hoffmann reflex (H-reflex) is a measurement tool that can be used to assess motoneuron-pool excitability⁶⁻⁸ and synaptic transmission.

Several studies have identified changes in the H-reflex resulting from static stretching.^{1-4,9} It has been reported that after static stretching the amplitude of H-reflex decreases,⁹⁻¹¹ but this neurological change dissipates once stretching is stopped. Furthermore, the extent of H-reflex decrease has been reported to be related to the magnitude of the stretch.^{3,11} It has been suggested that inhibition of the H-reflex during stretching promotes muscle lengthening by reducing neural input to the motoneuron pool of the stretched muscle.^{1,9} These results were obtained using acute stretching protocols in which neural changes were assessed during the actual performance of the given protocol. Few studies, however, have investigated the neural mechanisms underlying the adaptation to long-term muscle stretching. Specifically, it is not clear how the role of the spinal reflexes or the excitability of the motoneuron pool changes during the physiological adaptation to repeated bouts of static stretching.

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One study² to date has addressed the central question of whether long-term adaptations by spinal reflexes occur after a long-term static-stretching program. In that study, subjects were asked to perform passive static stretching of the plantar-flexor muscles 5 times per week for 6 weeks.² At 4 weeks, increases in flexibility were observed at rest, but the H-reflex amplitude was not different than pretraining values.² After 6 weeks of training, a significant decrease in H-reflex was observed with additional increases in flexibility.² Since some improvements in flexibility were observed before changes in the H-reflex, the authors concluded that increases in flexibility during the first month of the stretching protocol resulted primarily from mechanical adaptations such as increases in the length of the muscle-tendon unit and reduced passive torque,² supporting the mechanical findings of others,¹²⁻¹⁵ and that the long-term increases in ankle flexibility resulted from decreased reflex activity.²

Research indicates that the decrease in motoneuron-pool excitation during stretching is caused by both presynaptic and postsynaptic mechanisms.^{2,3} In an acute stretching regimen, changes in presynaptic mechanisms were found to contribute to small changes in H-reflex amplitudes while large changes were attributed to changes in postsynaptic mechanisms.³ Guissard et al³ hypothesized that the H-reflex changes observed after 6 weeks of static stretching were due to both presynaptic and postsynaptic mechanisms; however, the relative contribution of each was not assessed.

It has been suggested that presynaptic and postsynaptic mechanisms could effectively modulate the H-reflex without changing motoneuron excitability.³ Without thorough evaluation of both the presynaptic and postsynaptic mechanisms, however, the origins of the changes in the excitability of the motoneuron pool observed after 6 weeks of long-term stretching remain unknown. Therefore, to adequately assess the neurological adaptations that occur with long-term stretching, presynaptic and postsynaptic mechanisms must be evaluated in conjunction with the response of the H-reflex.

Therefore, the primary purpose of this study was to evaluate the neurological changes that occur with increases in flexibility in the soleus during a 6-week static-stretching program. Specifically, we evaluated how long-term stretching affects ankle dorsiflexion, the $H_{\max}:M_{\max}$, and the levels of presynaptic and disynaptic reciprocal inhibition. We hypothesized that the reduction in synaptic transmission of Ia afferents to the motoneuron pool as a result of long-term stretching may be related to corresponding increases in the level of presynaptic and/or disynaptic reciprocal inhibition. In addition, evaluation of $H_{\max}:M_{\max}$ may provide additional insight into the excitability changes of the motoneuron pool with increasing flexibility.

Methods

Study Design

We employed a repeated-measure, case-controlled design with 2 independent variables: group (experimental, con-

trol) and time (baseline, 3 wk, and 6 wk). The dependent measures were $H_{\max}:M_{\max}$, presynaptic inhibition, disynaptic reciprocal inhibition, and ankle-dorsiflexion passive range of motion (PROM) and were taken at each data collection. Qualified subjects were randomly assigned to either the experimental (stretching) or control group. 10 men and 10 women were assigned in each group. Neuromuscular and flexibility testing occurred after 3 and 6 weeks on the same day of the week and at the same time of day. During testing days, we conducted a follow-up on compliance with the experimental group and then tested ankle dorsiflexion through PROM, $H_{\max}:M_{\max}$, the level of presynaptic inhibition, and disynaptic reciprocal inhibition for all subjects.

Participants

Forty healthy subjects, 20 women and 20 men (22.37 ± 3.13 y, 174.28 ± 9.29 cm, 77.72 ± 17.71 kg), with no history of cognitive impairment, neurological impairment, or lower extremity surgery or injury within the previous 12 months volunteered for this study. Each subject signed an informed-consent document that had been previously approved by the university's institutional review board.

Procedures

Subject Preparation. Surface electromyography (MP100, BIOPAC Systems Inc, Santa Barbara, CA) was used to measure the H-reflex and M-wave. After the skin was shaved, abraded, and wiped with isopropyl alcohol, pregelled, self-adhesive disposable vinyl Ag-AgCl recording electrodes (1 3/8-in.; EL 503, BIOPAC Systems Inc) were placed over the soleus and tibialis anterior muscle bellies and the ipsilateral lateral malleolus.

An electrical stimulator (S88, Grass Instruments Inc) was used to elicit nerve responses. To stimulate the soleus, an unshielded 12-mm stimulating electrode (EL 212, BIOPAC Systems Inc) was applied to the skin over the posterior tibial nerve behind the knee. To be used for presynaptic measurements, another stimulating electrode was placed over the common peroneal nerve adjacent to the fibular head to stimulate the tibialis anterior. To identify the correct placement location, the stimulating electrodes were moved over the nerves until a muscle response was seen in both EMG recordings. Once the correct location was found, the stimulating electrodes were taped to the subject's skin and outlined with permanent marker to ensure that they were placed in the same location during subsequent data collection.¹⁶ One dispersal pad, or anode (3 cm²), was placed on the distal thigh above the knee, while a second anode was placed on the musculature lateral and distal to the stimulating electrode of the tibialis anterior. Signa Gel electrode gel (Parker Laboratories, Fairfield, NJ) was liberally applied on both the stimulating electrodes and both dispersal pads. A constant current unit (CCU1, Grass Instruments Inc, W. Warwick, RI) and a stimulation-isolation unit (SIU5, Grass Instruments Inc) were employed to limit the risk of electrical shock.

Subject Positioning. All subjects were tested in a specific prone position for the H-reflex and M-wave EMG measurements (see Figure 1). The ankle-, knee-, and hip-joint angles used for each individual during the initial testing session were measured with a goniometer, recorded, and referenced for the following testing sessions.

Measurement. To capture peak-to-peak amplitude of the H-reflex and M-waves, EMG measurements were collected at a rate of 2000 samples/s. H-reflex and M-wave recruitment curves were mapped using AcqKnowledge waveform-acquisition software for Microsoft Windows (AcqKnowledge Software v 3.7.3, 1992–2002, Biopac Systems, Inc, Goleta, CA).

$H_{max}:M_{max}$. Motoneuron-pool excitability was determined by assessing the $H_{max}:M_{max}$ obtained by percutaneous electrical stimulation of the tibial nerve (via 1-ms pulses) and measuring the corresponding EMG of the soleus. After the initial low stimulus intensity of 0.5 mV, intensity was increased by 0.5 mV for each subsequent stimulus, with the stimulus being delivered twice between increases in intensity. This protocol was followed until the maximum H-reflex and M-wave amplitudes had been attained.¹⁷

Conditioned Stimulation. The level of presynaptic and disynaptic reciprocal inhibition was evaluated by measuring the soleus H-reflex. However, the H-reflex measurement was altered due to a prior stimulation of the common peroneal nerve (conditioned stimulation) and activation of the tibialis anterior. Once the recording

electrodes, stimulating electrode, and dispersal pad had been applied, the stimulation intensity to motor threshold of the tibialis anterior was identified by the EMG recording and detected physically by the same investigator.^{7,18} This intensity was maintained throughout testing.^{7,18}

Presynaptic Inhibition. Stimulation of the tibialis anterior preceded the soleus stimulation at a time point between 80 and 120 milliseconds. The average soleus H-reflex response of 5 conditioned stimulations was evaluated at delays of 80, 85, 90, 95, 100, 105, 110, 115, and 120 milliseconds. We identified optimal delay by discovering the time by which the greatest inhibition in the soleus was present over the average of 5 conditioned trials.^{19,20} The sequence was as follows: A conditioned stimulus of the tibialis anterior over the common peroneal nerve occurred first, then a subject-dependent delay (80–120 ms), followed by stimulation to the soleus.

Once the optimal inhibition delay was identified, we began presynaptic inhibition testing to be included in the data analysis. We first measured a simple soleus H-reflex (unconditioned stimulation) before the initiation of the M-wave without the influence of a preceding tibialis anterior stimulation. Then, we waited 10 seconds and elicited a conditioned stimulation to the tibialis anterior and measured the resulting soleus H-reflex. The combination sequence of the conditioned and unconditioned was repeated 10 times. Therefore, 10 trials of the unconditioned H-reflex (no tibialis anterior conditioning) were assessed, as well as 10 trials of the conditioned H-reflex.¹⁷ Presynaptic inhibition was evaluated by subtracting the



Figure 1 — Subject in prone testing position with the left ankle preserved at 90°. A standardized trunk, head, and hand position was maintained by the use of a body pillow.

conditioning amplitude from the unconditioned amplitude of the soleus. The result was then multiplied by 100 to get a percentage:

$$\frac{(\text{Unconditioned response} - \text{conditioned response})}{\text{unconditioned response}} \times 100$$

The average of the 10 trials was used for data analysis.

Disynaptic Reciprocal Inhibition. The stimulation of the tibialis anterior preceded the H-reflex measure of the soleus muscle by 2 to 4 milliseconds.²¹ In 1987, Crone et al¹⁸ demonstrated that with this short latency period (1–3.5 ms), the inhibition is disynaptic in origin, and its low threshold indicates that it is caused by activation of group I muscle afferents. Therefore, we assume that the inhibition is mediated via a pathway similar to the disynaptic reciprocal inhibitory pathway and, therefore, due to postsynaptic mechanisms.

Therefore, the conditioned measurement included a stimulus of the tibialis anterior over the common peroneal nerve first, then a 2- to 4-millisecond delay, followed by stimulation to the soleus. The average soleus H-reflex response of 5 conditioned stimulations was evaluated at delays of 2, 2.5, 3, 3.5, and 4 milliseconds. We identified optimal delay by discovering the time by which the greatest inhibition in the soleus was present over the average of 5 conditioned trials. The combination sequence of the conditioned and unconditioned was repeated 10 times; 10 trials of the unconditioned H-reflex were assessed, as well as 10 trials of the conditioned H-reflex. Disynaptic reciprocal inhibition was evaluated by subtracting the conditioning amplitude from the unconditioned amplitude of the soleus. The result was then multiplied by 100 to get a percentage. The average of the 10 trials was used for data analysis.

Range of Motion. Ankle-dorsiflexion PROM was evaluated using the following protocol. Blindfolded subjects were tested in the semireclined position with

their hip at 90° of flexion and knee angle fixed at 60° of flexion on a Biodex isokinetic dynamometer (Biodex System 3, Biodex Medical Systems Inc, Shirley, NY). The exact positionings of height, angles, and distance were recorded during the initial testing session to limit experimental error and changes in muscle length at subsequent testing sessions. A passive dorsiflexion test was used to measure soleus extensibility or ROM. Once a subject was ready, the left ankle was moved to a set starting position of 90°. During 30 seconds of relaxation, the subject was then instructed to relax the muscles of the lower leg and to concentrate on the sensation of the stretch. The Biodex then passively moved the ankle into dorsiflexion at 2°/s. Subjects used a handheld stop switch to stop ankle dorsiflexion at the maximal tolerable stretch. The final position was designated as the point of maximal passive dorsiflexion recorded by the Biodex.

A total of 4 trials were given per data collection. The first trial was used during each session to familiarize the subject with the protocol. The average of the final 3 trials was used in data analysis to represent ankle-dorsiflexion PROM.

Stretching Protocol. After baseline neurological and flexibility testing, each subject in the experimental group was instructed to perform 30 static-stretching sessions over a 6-week period. Thus, the experimental group performed the following protocol 5 times per week while the control group did not stretch. In each session, the subjects were instructed to perform each of the 3 stretches 5 times for 30 seconds at their maximum tolerance level of stretch. They were also asked to rest for 30 seconds between stretches. Therefore, the total time spent stretching was 7.5 minutes, and the total time spent relaxing was 7.5 minutes during each session, which lasted approximately 15 minutes. All subjects in the experimental group were instructed on proper stretching technique using diagrams (Figure 2). These

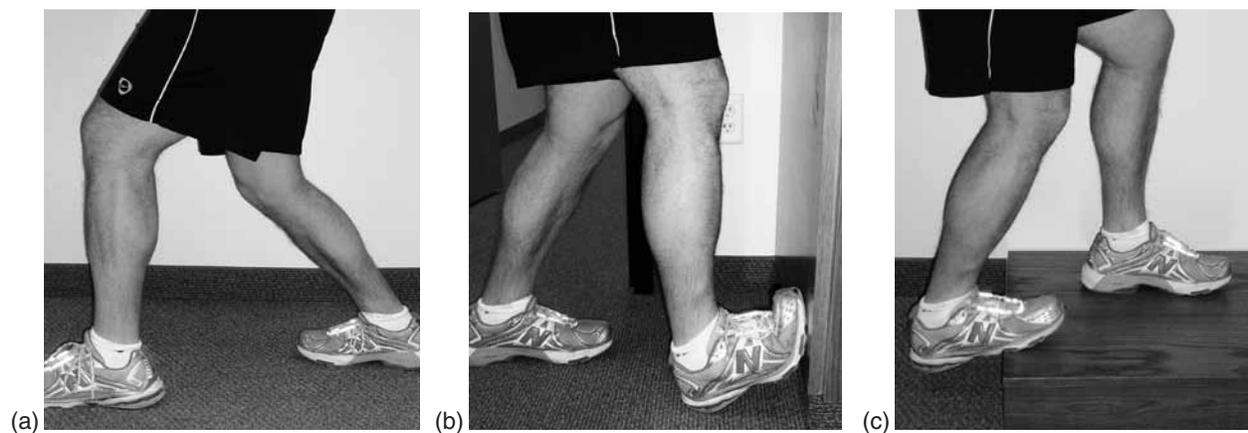


Figure 2 — (a) Standing calf stretch: Left leg was bent at 10–20% of flexion (or knee approximately 12–14 cm in front of left toe). (b) Forefoot on wall standing calf stretch: Subject placed the forefoot of the left leg on the wall while maintaining 10–20% knee flexion. (c) Calf stretch on step: Subject placed forefoot on the step while the heel hung off over the edge and the knee remained slightly bent to isolate the soleus.

stretches were chosen because they have been shown in previous work to elicit changes in the flexibility of the soleus muscle.² The static-stretching program was designed to increase flexibility at the maximum tolerance level of dorsiflexion for each subject. The subjects were directed to record in a journal the date and time of each stretching session that they completed and to bring the journal to each of the subsequent 2 testing sessions in the laboratory. All subjects reported 100% compliance to the stretching protocol.

Statistical Analysis

We used a 2×3 repeated-measures MANOVA factorial design. In the event of a significant group \times time interaction, univariate repeated-measures analyses were assessed. Paired *t*-tests were used to determine mean differences only when the repeated-measures MANOVA yielded a significant *F*-ratio for the group \times time interaction. Alpha level was set at .05 and Bonferroni adjustment was applied to limit the chance of type I error in all 5 subsequent univariate analyses.

In addition, we calculated test-retest reliability using values for ankle dorsiflexion PROM and $H_{\max}:M_{\max}$ and percentage of presynaptic inhibition and disynaptic reciprocal inhibition found in the soleus muscle. The intersession reliability over 2 trials was estimated using an intraclass correlation coefficient ($ICC_{2,1}$; *R*).²² The 2 measures were taken at baseline and at 6 weeks with the control subjects. The advantage of employing this method is that we could look at 2 sets of scores at a time and analyze the effect of 1 source of error.²² All statistical analyses were performed using SPSS software, version 11.5 (SPSS, Inc, Chicago, IL).

Results

To determine the effects of randomization to experimental groups on baseline outcome variables, we ran independent *T*-tests. Among our 4 outcome variables, baseline range of motion was on average greater in the control than in the experimental group ($P = .005$). Among the other 4 outcome variables, there were no baseline differences between the control and experimental groups ($P \geq .266$).

In an analysis of covariance with 6-week change in range of motion as the dependent variable, experimental group as the independent variable, and baseline range of motion as the covariate, there was a significantly greater change in range of motion in the experimental group than in the control group ($P < .05$). In the experimental group, the change in range of motion after adjusting for the baseline difference in range of motion was 11.37° (95% CI range 9.99 – 12.76°). By contrast, in the control group, the change in range of motion after adjusting for the baseline difference in range of motion was 0.59° (95% CI range $-.79^\circ$ to 1.98°).

The 2×3 repeated-measures MANOVA indicated a significant group \times time interaction (Wilks's $\Lambda = 16.36$, $P < .001$). Post hoc analysis revealed a significant ($F_{1,38}$

$= 150.60$, $P < .001$) difference in ankle-dorsiflexion PROM. As seen in Figure 3, the experimental group had significantly improved PROM from baseline to 3 weeks (mean $6.2^\circ \pm 0.9^\circ$, $P < .001$), 3 weeks to 6 weeks (mean $5.0^\circ \pm 0.8^\circ$, $P < .001$), and baseline to 6 weeks (mean $11.2^\circ \pm 0.9^\circ$, $P < .001$). Specifically, from baseline to 3 weeks, 3 weeks to 6 weeks, and baseline to 6 weeks, the experimental group had a 23.5%, 15.2%, and 42.3% increase in ankle-dorsiflexion PROM, respectively (see Figure 3).

No group \times time interactions were observed for $H_{\max}:M_{\max}$, presynaptic inhibition, and disynaptic reciprocal inhibition outcome measurements. However, in the experimental group, baseline to 3 weeks yielded a 7.2% decrease, 3 weeks to 6 weeks yielded a 2.1% increase, and baseline to 6 weeks yielded a 5.3% overall decrease in soleus motoneuron excitability (Figure 4). However, there were no statistically significant neurological differences ($P > .05$).

The intersession reliability ($ICC_{2,1}$) of the measures performed on the control subjects to assess the stability and consistency of the measurement protocol was found to have high consistency ($H_{\max}:M_{\max}$ [$R = .808$], ankle-dorsiflexion PROM [$R = .977$], presynaptic inhibition [$R = .810$], and disynaptic reciprocal inhibition [$R = .786$]).

Discussion

The primary purpose of this study was to evaluate the neurological changes that occur with increases in flexibility in the soleus during the implementation of a 6-week static-stretching program. Specifically, we evaluated how long-term stretching affects ankle dorsiflexion, $H_{\max}:M_{\max}$, and the levels of presynaptic and disynaptic reciprocal inhibition. The lack of significant motoneuron excitability changes at 6 weeks corresponding with a significant increase of flexibility with our protocol limited that investigation and contrasted with the current literature.²

Few studies have investigated the mechanisms of adaptation to long-term or chronic muscle stretching, thus limiting understanding of whether increases in flexibility result from mechanical or neural elements. The theory remains that both elements remain integral in increasing flexibility; however, their time courses are different.² It is now well documented that H-reflexes are decreased while performing a static muscle stretch,^{9–11} and it was recently suggested that neural mechanisms at presynaptic and postsynaptic levels are involved.³ Specifically, it was reported that presynaptic mechanisms were responsible for changes during small-amplitude stretches, whereas postsynaptic mechanisms were responsible for changes during large-amplitude stretches.³ The impact of these studies is that neural changes contributed to increased flexibility during an active stretching regimen by changing the tonic Ia-reflex-pathway activity, but the effect dissipated or was removed once the stretching ended.^{1,11} Therefore, the central question that we addressed is whether long-term adaptations of the spinal reflex occur and remain after a chronic static-stretching program. The

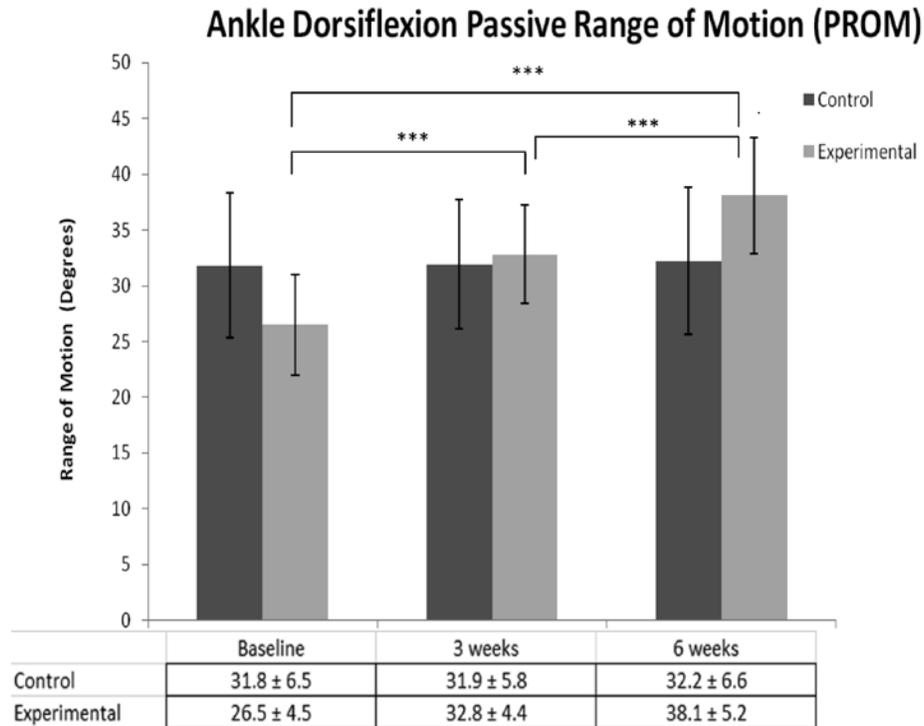


Figure 3 — Illustration of PROM in ankle dorsiflexion at baseline, after 3 weeks, and after 6 weeks (mean ± SD). *** $P < .001$.

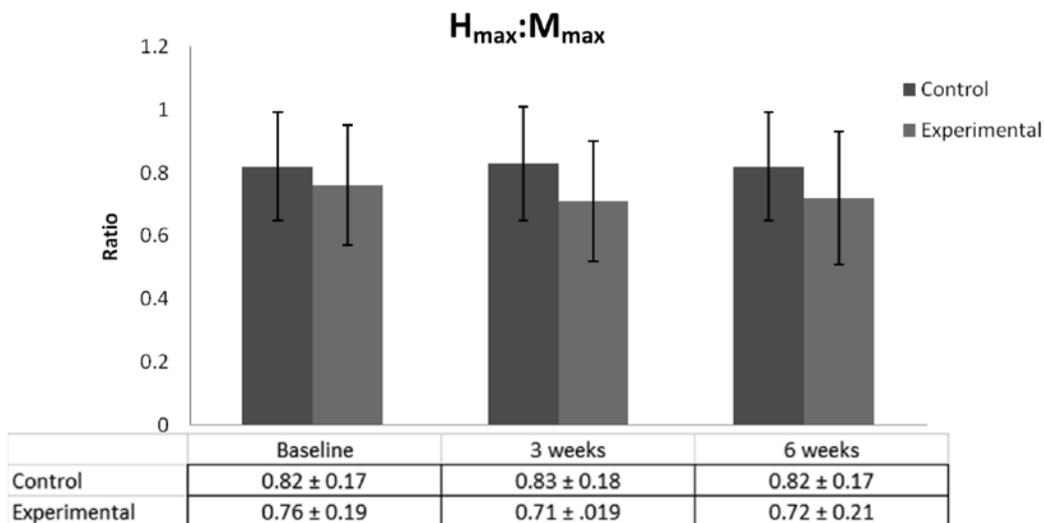


Figure 4 — Illustration of the $H_{max}:M_{max}$ at baseline, after 3 weeks, and after 6 weeks (mean ± SD).

results from our study indicate that the neural elements of the Ia-reflex pathway did not adapt to a chronic stretching program, so the 44% increase in ankle dorsiflexion after 6 weeks and 30 sessions of stretching was not due to statistically significant neurological adaptations in the soleus.

To our knowledge, only 1 prior study has addressed this central question of whether long-term adaptations

of the Ia spinal reflex occur and remain after a chronic static-stretching program. That study found that the H-reflex or neural aspects are inhibited or decreased after training with increased flexibility. Furthermore, it was reported that a significant level was only reached when 30 static-stretching sessions had been completed in 6 weeks of training 5 times per week.² Specifically,

Guissard and Duchateau² revealed a 14% inhibition in motoneuron excitability with an increase of 30.8% dorsiflexion, whereas we found that a 5.3% inhibition occurred compared with pretraining values with a 42.3% increase in PROM. It must be noted, however, that there were differences in the stretching programs between studies. Our stretching program solely isolated the soleus muscle, whereas Guissard and Duchateau² incorporated the gastrocnemius and soleus in theirs. Furthermore, the contrast in reported results between our study and that of Guissard and Duchateau² may also be due to the differences in sex distribution in subjects. They had an unbalanced sex distribution, with twice as many males as females, whereas our study had an equally balanced sex distribution. Our study was underpowered to look into the effect of sex, but this should be investigated in future studies.

Research indicates that the decrease in motoneuron-pool excitation during stretching is caused by both presynaptic and postsynaptic mechanisms.^{1,4} Guissard and Duchateau² hypothesized that the H-reflex changes observed after 6 weeks of static stretching were due to both presynaptic and postsynaptic mechanisms; however, the relative contribution of each to the H-reflex decrease was not assessed. We were unable to address this central question as the amount of influence each mechanism has on the inhibition of motoneuron-pool excitability due to nonsignificant statistical results after 6 weeks.

The neural changes or H-reflex amplitude changes were negligible, and we can surmise that it was mainly the mechanical adaptations that contributed to increased flexibility over 3 weeks and 6 weeks. Therefore, our observations support the findings of Guissard and Duchateau² that significant mechanical changes in the muscle occur before the significant neural changes.² Therefore, these increases in flexibility are thought to be related to the viscoelastic properties of muscle tissue,²³ which are commonly attributed to the passive elastic tensions in both connective tissues and myofibrils.²⁴ Another possible explanation, formulated by Magnusson et al²³ and Halbertsma et al,²⁵ is that the increased range of motion from stretching, albeit the hamstrings, was the result of increased stretch tolerance rather than a change in mechanical or viscoelastic properties of the muscle, stretch tolerance being determined as the subject's tolerance to the stretch by bringing the stretch position to the point of onset of pain. However, our findings refute the prior conclusions that neural adaptations have occurred with significant increases in flexibility at 6 weeks and support the theories of increased stretch tolerance and/or mechanical theories that are used to explain increased extensibility whereby the muscles are viscoelastic structures, and therefore elastic change occurs.

Our study has certain limitations that readers should consider when interpreting our results. Although an a priori power analysis was not performed, we did use the methodology of Guissard and Duchateau² to determine a practical and feasible justification of sample size.²⁶ They had a sample size of 12 subjects (8 male and 4 female)

and yielded a 14% change in neurological activity ($P < .01$). A concern we had with this methodology was the unbalanced distribution of males and females. We therefore selected a larger and more balanced sample of 10 men and 10 women in both the control and experimental groups ($n = 40$), and we included outcome variables that were not assessed by Guissard and Duchateau.² Thus, according to Bacchetti,²⁶ we have practical justification of significant power with our outlined methodology of 20 subjects per group.

In addition, when considering the task specificity of neural adaptations after training we realize that adaptations of the H-reflex can be task and training specific. Schubert et al²⁷ recently found that corticospinal excitability was increased or decreased after training, but this was not a general effect as it could not be detected at rest. Therefore, the stretching methods employed in this study were essentially the training task and focused on the soleus muscle due to the bent-knee nature of the stretching intervention. However, the data we reported measured the neural adaptations at rest and not during the stretching task, which is when the adaptations may have occurred.

Conclusions

In summary, it is evident that 6 weeks and 30 sessions of static stretching increased the flexibility of maximal ankle dorsiflexion; however, the improvement of flexibility was not due to statistically significant neuromuscular origins or adaptations. Therefore, mechanical and/or stretch tolerance has a greater influence on the increase in ankle-dorsiflexion PROM after a long-term stretching protocol. Future research should increase the duration of the stretching and testing protocol to evaluate when or if neurological influences are present. In addition, we would recommend that future studies investigate potential sex differences regarding the neurological changes with stretching. Furthermore, evaluating the neurological influences during the stretching activity may provide greater insight into whether neuromuscular origins of adaptation are training or task specific.

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