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The Time Course of Musculotendinous Stiffness Responses Following Different Durations of Passive Stretching

Stretching prior to competition or exercise has been traditionally recommended and practiced for many years.[35] It is generally thought that stretching will improve performance[40] and reduce the risk of injury.[35] However, recent reviews have questioned the efficacy of pre-event stretching.[34,38,44,45] These reviews have suggested that there is not enough evidence to support the claims that stretching improves performance[34,38] or reduces the risk of injury.[44,45]

Indeed, several recent studies have indicated that an acute bout of stretching reduces muscle strength,[7,12] power output,[3] and balance performance.[2] It has also been recently emphasized[44] that the limited experimental evidence[4,33] does not clearly support the efficacy of stretching prior to athletic events to reduce the risk of injury. Shrier[39] and Witvrouw et al[45] have even hypothesized that stretching prior to performance may increase the risk of injury, although this has not been validated. Despite this evidence, a recent study by Shehab et al[37] reported that stretching is still included prior to competition in high school athletics because of its presumed injury reduction benefits. Thus, the need for further research on the acute effects of stretching and the dissemination of this evidence is apparent.

Many studies that examine the acute effects of stretching use changes in joint range of motion as the primary outcome variable.[1,6,31] However, Magnusson et al[25] stated that "joint range of motion, a single measurement in time in a static system, provides limited information about the behavior of the muscle-tendon unit." The passive biomechanical properties of the muscle-tendon unit (musculotendinous stiffness [MTS]) may provide more comprehensive evidence when studying the efficacy of stretching to reduce the likelihood of musculotendinous strain injuries. MTS measurements are usually calculated from the force or torque curves that are recorded during passive stretches.[20]

• **STUDY DESIGN:** Repeated-measures experimental design.

• **OBJECTIVE:** To examine the acute effects of different durations of passive stretching on the time course of musculotendinous stiffness (MTS) responses in the plantar flexor muscles.

• **BACKGROUND:** Stretching is often implemented prior to exercise or athletic competition, with the intent to reduce the risk of injury via decreases in MTS.

• **METHODS AND MEASURES:** Twelve subjects (mean ± SD age, 24 ± 3 years; stature, 169 ± 12 cm; mass, 71 ± 17 kg) participated in 4 randomly ordered experimental trials: control with no stretching, and 2 minutes (2_{min}), 4 minutes (4_{min}), and 8 minutes (8_{min}) of passive stretching. The passive-stretching trials involved progressive repetitions of 30-second passive stretches, while the control trial involved 15 minutes of resting. MTS assessments were conducted before (prestretching), immediately after (poststretching), and at 10, 20, and 30 minutes poststretching on a Biodex System 3 isokinetic dynamometer.

• **RESULTS:** MTS decreased ($P<.05$) immediately after all stretching conditions (2_{min}, 4_{min}, and 8_{min}). However, MTS for the 2_{min} condition returned to baseline within 10 minutes, whereas MTS after the 4_{min} and 8_{min} passive-stretching conditions returned to baseline within 20 minutes.

• **CONCLUSIONS:** Practical durations of passive stretching resulted in significant decreases in MTS; however, these changes return to baseline levels within 10 to 20 minutes.

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• **KEY WORDS:** compliance, passive, stiffness, strain injury, stretch

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Therefore, the amount of passive tension can be quantified at any point in the range of motion. In theory, less passive tension will be recorded from the musculotendinous unit at the same absolute joint angle after a bout of stretching, which will result in a decrease in MTS.[4,10,43] Thus, although passive stretching has reported to not change the ultimate force at failure,[10] it is thought that changes in MTS after stretching are more indicative of the ability of a musculotendinous unit to resist strain injuries within the normal range of motion than simple changes in joint range of motion.[25]

Traditionally, it is believed that the inclusion of stretching activities prior to exercise and/or athletic events may reduce the risk of musculotendinous strain injuries by decreasing MTS.[4] However, shorter practical durations of stretch (1.00-2.25 minutes) have resulted in no changes in MTS,[22-24,28,30] while longer durations of stretch (2.5-30.0 minutes) have resulted in decreases in MTS.[7,25,32] The dose-response relationship between the duration of stretching and the MTS response has yet to be fully understood. In addition, because many stretching routines are performed 15 to 60 minutes prior to competition or exercise,[5,46] the time course for the stretching-induced decreases in MTS needs to be precisely determined. This was acknowledged recently by Morse et al,[29] who noted that "as discussed by Magnusson et al,[22] the time course of the in vivo physiological adaptations associated with static stretching remains unresolved." Fowles et al[7] previously reported that MTS remained depressed for 30 minutes; however, the stretching procedure included 30 minutes of passive stretching (PS), which is well beyond any practical stretching duration. Therefore, the purpose of the present study was to extend the findings of Fowles et al[7] and address the recommendations of Morse et al[29] to determine the time course (up to 30 minutes poststretching) for the acute effects of practical stretching durations (2-8 minutes) on MTS.

METHODS

Subjects

Seven men (mean ± SD age, 24 ± 4 years; height, 178 ± 7 cm; mass, 82 ± 12 kg) and 5 women (age, 21 ± 1 years; height, 157 ± 5 cm; mass, 56 ± 7 kg) volunteered for this investigation. This study was approved by the University of Oklahoma Institutional Review Board for Human Subjects Research, and all participants completed an informed consent form and a pre-exercise testing health status questionnaire. No one reported any current or ongoing neuromuscular diseases or musculoskeletal injuries specific to the ankle, knee, or hip joints. None of the participants were competitive athletes; however, due to their reported levels of aerobic exercise (median [range], 4 [4.5] h/wk), resistance training (6 [5.5] h/wk), and recreational sports (3 [4.5] h/wk), these participants might be best classified as normal recreationally active college-aged subjects.

Experimental Design

A randomized, repeated-measures, crossover design (time [prestretching versus poststretching versus 10 minutes versus 20 minutes versus 30 minutes posttreatment] × condition [control versus 2_{min} versus 4_{min} versus 8_{min}]) was used to examine the acute effects of PS on MTS. The participants visited the laboratory on 5 occasions, each separated by 3 to 7 days. The first visit was a familiarization trial and the subsequent 4 visits included the following experimental conditions in random order: (*a*) control condition, (*b*) PS for 2 minutes (2_{min}), (*c*) PS for 4 minutes (4_{min}), and (*d*) PS for 8 minutes (8_{min}). During each experimental condition, the participants underwent the pretreatment assessments, the treatment condition, and the posttreatment assessments. For the control condition, the pretreatment and posttreatment assessments were separated by a 15-minute rest, which was equivalent to the total duration of the 8 minutes PS condition (including rest between stretches). The posttreatment assessments occurred immediately after the treatment conditions and at 10, 20, and 30 minutes after the treatment conditions to examine the time course. All experimental trials were performed at the same time of day (±2 hours) for each subject.

Familiarization Trial

Three to 5 days prior to the experimental trials, each participant signed the informed consent form, completed the health status questionnaire, and practiced the MTS assessments to ensure that they were comfortable with the procedures and to minimize any potential learning effects.

Musculotendinous Stiffness

To determine MTS, each participant underwent 2 pretreatment assessments prior to the PS and 1 assessment at each posttreatment assessment period (posttreatment, and 10 minutes, 20 minutes, and 30 minutes posttreatment). All participants were seated with restraining straps over the pelvis, trunk, and thigh on a calibrated Biodex System 3 dynamometer (Biodex Medical Systems, Inc, Shirley, NY). The knee was in extension, with the thigh and lower leg parallel to each other and to the horizontal plane. The lateral malleolus of the fibula was aligned with the input axis of the dynamometer, in accordance with the Biodex User's Guide (*Biodex Pro Manual, Applications/Operations*; Biodex Medical Systems, Inc, Shirley, NY). The foot was secured in a thick rubber heel cup attached to a footplate, with straps over the toes and metatarsals (distal to the malleoli), so that the straps did not impede any passive foot movement at the ankle joint.

During the familiarization trial, the maximum tolerable torque threshold was determined for each individual as the point of discomfort, but not pain, as verbally acknowledged by the subject during a series of passive stretches of the plantar flexors. This predetermined torque threshold was then used for all subsequent experimental trials as the

maximum tolerable point of stretch. The dynamometer lever arm passively dorsiflexed the foot at an angular velocity of 5°/s until the torque threshold was met and held for 5 seconds, similar to the procedures of Muir et al.[30] Position (degrees) and torque (Nm) values were sampled from the dynamometer during the MTS assessments, which provided the passive angle-torque curves (**FIGURE 1**, bottom). To be consistent with previous studies conducted by Magnusson and colleagues,[22,24,25] MTS values ($Nm \cdot deg^{-1}$) were calculated using a fourth-order polynomial regression model that was fit to the angle-torque curves, according to the procedures recently described by Nordez et al.[32] As described previously by Herda et al,[12] joint angle specific stiffness values were calculated as the slope of the polynomial fit angle-torque curves. MTS was determined at every fourth degree during the final 13° range of motion (at 1°, 5°, 9°, and 13°) (**FIGURE 1**) that was common to every testing period (prestretching, poststretching, 10 minutes, 20 minutes, and 30 minutes posttreatment). Therefore, the same absolute positions (degrees) were used for each subject to calculate MTS at each testing period. No gravity correction was performed, as based on the methods of Salsich et al,[36] who indicated that the foot constituted approximately 1.4% of the body's mass and suggested that this mass can be considered negligible.[12]

Surface Electromyography

Preamplified bipolar, active surface electrodes (EL254S; Biopac Systems, Inc, Santa Barbara, CA), with a gain of 350 and a fixed center-to-center interelectrode distance of 20 mm, were taped over the medial gastrocnemius (MG) and soleus (SOL) muscles. For the SOL, the electrodes were placed along the longitudinal axis of the tibia at 66% of the distance from the medial condyle of the femur to the medial malleolus. The electrodes for the MG were placed on the most prominent bulge of the muscle, in accordance with the recommendations

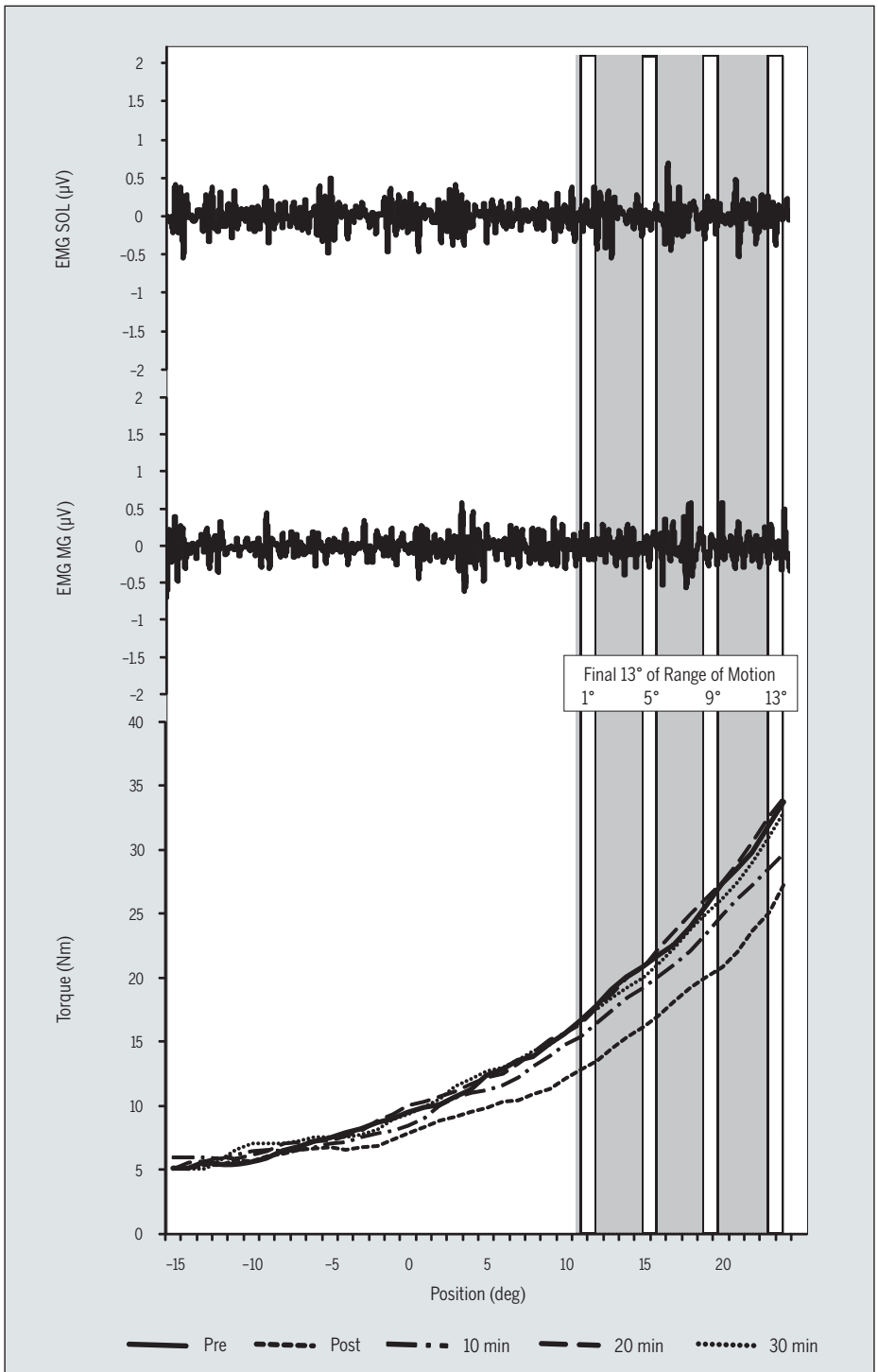

FIGURE 1. An example of the electromyographic (EMG) signals from the soleus (top) and medial gastrocnemius (middle), during a single, passive, angle-torque curve of the plantar flexors (bottom), taken from a subject during the pretreatment assessment (solid line) of an 8_{min} stretching condition. For visualization, the posttreatment, and 10, 20, and 30 minutes posttreatment raw angle-torque curves recorded for this subject were also plotted. The large rectangular shaded area represents the final 13° range of motion that was common to all testing periods (pretreatment, posttreatment, and 10, 20, and 30 minutes posttreatment). The vertical white boxes represent every fourth degree in the final 13° range of motion (1°, 5°, 9°, and 13°), where musculotendinous stiffness (MTS) and EMG amplitude values were calculated and used for analysis.

of Hermens et al.[13] A single, pregelled, disposable electrode (Ag-Ag Cl Quinton Quick Prep; Quinton Instruments Co, Bothell, WA) was placed on the spinous process of the seventh cervical vertebra to serve as a reference electrode. To reduce interelectrode impedance and increase the signal-to-noise ratio, local areas of the skin were shaved and cleaned with isopropyl alcohol prior to placement of the electrodes.

Signal Processing

The torque (Nm), position (degrees), and EMG (μV) signals were sampled simultaneously at 1 kHz, with a Biopac data acquisition system (MP150WSW; Biopac Systems, Inc, Santa Barbara, CA) during each passive angle-torque curve. All signals were stored on a personal computer (Dell Inspiron 8200; Dell, Inc, Round Rock, TX) and processed off-line using custom-written software (LabVIEW, Version 8.2; National Instruments, Austin, TX). The EMG signals were digitally filtered (zero-phase fourth-order Butterworth filter), with a pass band of 10 to 500 Hz. The torque and position signals were low-pass filtered, with a 10-Hz cutoff (zero-phase lag, fourth-order Butterworth filter). All subsequent analyses were performed on the filtered signals.

During the passive torque-angle curves, EMG amplitude for the MG and SOL muscles was calculated with a root-mean-square (rms) function for 4 separate 200-millisecond epochs that corresponded with each MTS angle (1°, 5°, 9°, and 13°). **FIGURE 1** shows where these epochs were extracted to represent muscle activation during the MTS assessments. These 200-millisecond epochs were chosen because the dynamometer passively dorsiflexed the foot at 5°/s; thus, 200 milliseconds corresponded with a 1° change in range of motion. In addition, all EMG amplitude values (μVrms) were normalized to the pretreatment isometric maximal voluntary contraction (MVC) of the plantar flexors that was conducted at each trial prior to the first pretreatment MTS assessment.[16] Therefore, EMG amplitude values during the passive torque curves were represented as a percentage of the maximal EMG values recorded during the MVC (%MVC).

TABLE Pretreatment, Posttreatment, 10-, 20-, and 30-Minute Mean (SE) Values for Musculotendinous Stiffness for the Control, 2_{min}, 4_{min}, and 8_{min} Treatments

	Musculotendinous Stiffness (Nm/°)			
	1°	5°	9°	13°
Control				
Pretreatment	0.65 (0.04)	0.84 (0.06)	1.07 (0.09)	1.38 (0.12)
Posttreatment	0.70 (0.05)	0.89 (0.07)	1.14 (0.08)	1.45 (0.12)
10 min	0.69 (0.06)	0.88 (0.08)	1.11 (0.10)	1.41 (0.12)
20 min	0.68 (0.06)	0.84 (0.08)	1.08 (0.10)	1.40 (0.12)
30 min	0.69 (0.05)	0.86 (0.08)	1.09 (0.11)	1.42 (0.12)
2_{min}				
Pretreatment	0.65 (0.05)	0.84 (0.06)	1.06 (0.08)	1.33 (0.11)
Posttreatment	0.54 (0.04)*†	0.72 (0.05)*†	0.93 (0.07)*†	1.21 (0.11)*†
10 min	0.69 (0.04)	0.85 (0.05)	1.07 (0.08)	1.36 (0.13)
20 min	0.70 (0.04)	0.87 (0.06)	1.09 (0.10)	1.36 (0.18)
30 min	0.68 (0.04)	0.84 (0.07)	1.04 (0.10)	1.31 (0.13)
4_{min}				
Pretreatment	0.64 (0.05)	0.87 (0.06)	1.13 (0.11)	1.33 (0.10)
Posttreatment	0.50 (0.04)*†	0.63 (0.05)*†	0.85 (0.08)*†	1.15 (0.12)*†
10 min	0.62 (0.05)†	0.80 (0.06)†	1.00 (0.08)†	1.24 (0.12)†
20 min	0.65 (0.05)	0.82 (0.06)	1.03 (0.08)	1.27 (0.11)
30 min	0.64 (0.06)	0.82 (0.07)	1.04 (0.08)	1.28 (0.11)
8_{min}				
Pretreatment	0.68 (0.05)	0.86 (0.07)	1.08 (0.09)	1.40 (0.14)
Posttreatment	0.50 (0.04)*†	0.65 (0.05)*†	0.86 (0.07)*†	1.15 (0.10)*†
10 min	0.62 (0.06)†	0.76 (0.07)†	0.97 (0.09)†	1.22 (0.12)†
20 min	0.67 (0.06)	0.81 (0.07)	0.99 (0.09)	1.25 (0.11)
30 min	0.67 (0.06)	0.81 (0.07)	1.00 (0.09)	1.28 (0.12)

* *Indicates a significant ($P \leq .05$) difference from pretreatment to posttreatment.*
† *Indicates a significant ($P \leq .05$) difference from the control condition.*

Passive Stretching

Repeated PS of the right plantar flexor muscles was performed on the isokinetic dynamometer in the same fashion as the MTS assessments. The dynamometer passively dorsiflexed the foot until the predetermined torque threshold established during the initial pretesting familiarization trial was met. The dynamometer maintained this constant passive torque,[12,47] which stretched the plantar flexors for 30 seconds. Twenty seconds of rest was allowed between each PS repetition. The PS repetitions were repeated until the designated time under stretch for each condition was satisfied (the 2_{min} condition involved four 30-second PS repetitions for a total of 2 minutes of time under stretch).

Statistical Analyses

A 3-way mixed-factorial ANOVA (time [prestretch versus poststretch versus 10 minutes versus 20 minutes versus 30 minutes] × condition [control versus 2_{min} versus 4_{min} versus 8_{min}] × angle [1° versus 5° versus 9° versus 13°]) was used to analyze the MTS data. A 4-way mixed-factorial ANOVA (time [prestretch ver-

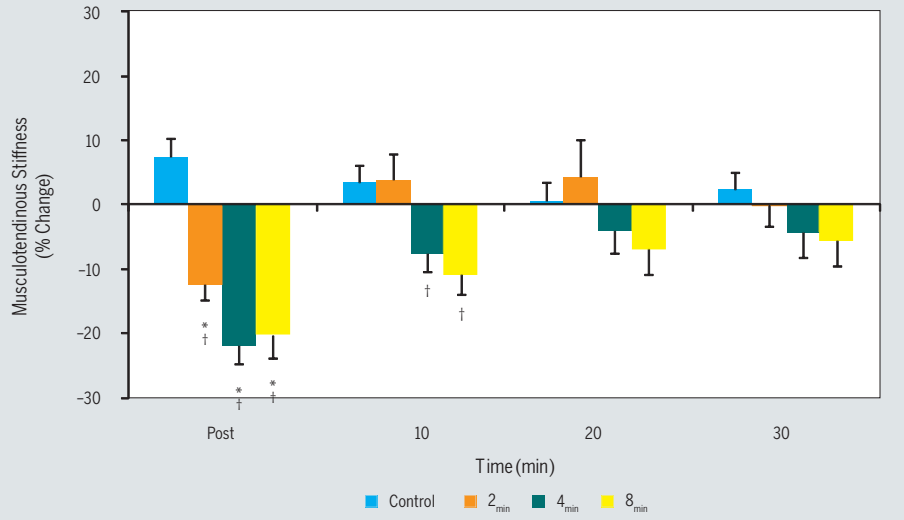

FIGURE 2. Percent change in musculotendinous stiffness (MTS) as a result of the control, 2_{min}, 4_{min}, and 8_{min} of passive stretching treatments. *A significant decrease from pretreatment to posttreatment ($P<.05$). †A significant difference from the control condition. Values represent the percent changes from the pretreatment condition (means ± SEM).

sus poststretch versus 10 minutes versus 20 minutes versus 30 minutes] × conditon [control versus 2_{min} versus 4_{min} versus 8_{min}] × angle [1° versus 5° versus 9° versus 13°] × muscle [SOL versus MG]) was used to analyze the normalized EMG amplitude data. When appropriate, follow-up analyses were performed using lower-order ANOVAs and *t* tests with Bonferroni corrections. An alpha of $P \leq .05$ was used to determine statistical significance. Statistical analyses were performed using SPSS Version 14.0 (SPSS Inc, Chicago, IL).

RESULTS

The **TABLE** contains the mean ± standard error (SE) values for MTS at each time point (prestretching, poststretching, 10-, 20-, and 30-minutes posttreatment) for each condition (control, 2_{min}, 4_{min}, and 8_{min}). Partial eta-squared (η_p^2) values are also reported below to reflect the magnitude of the change following each treatment.

Musculotendinous Stiffness

The ANOVA indicated no 3-way interaction ($P>.782$, $\eta_p^2 = 0.040$), no 2-way interactions for time by angle ($P = .331$, $\eta_p^2 = 0.097$), and condition by angle ($P = .236$, $\eta_p^2 = 0.122$), but a significant 2-way interaction for condition by time ($P<.001$, $\eta_p^2 = 0.357$). There was also a main effect for angle ($P<.001$, $\eta_p^2 = 0.850$). MTS increased during the final 13°-range motion ($1°<5°<9°<13°$). These differences were observed for each condition (control, 2_{min}, 4_{min}, and 8_{min}) and each time (prestretching, poststretching, 10-, 20-, and 30-minutes posttreatment). For the condition-by-time interaction, MTS decreased from prestretching to posttreatment for the 2_{min}, 4_{min}, and 8_{min} conditions, but was unchanged for the control condition (**FIGURE 2**). In addition, MTS for the 4_{min} and 8_{min} conditions was still lower than the control at 10 minutes poststretching (**FIGURE 2**).

Electromyographic Amplitude

The ANOVA resulted in only 1 main effect for muscle ($P = .008$, $\eta_p^2 = 0.491$). Normalized EMG amplitude values were greater for the SOL than the MG. However, there were no stretching- or stiffness-related changes in EMG amplitude for the SOL (mean, 0.18% MVC) and MG (mean, 0.28% MVC).

DISCUSSION

The primary findings of the present study were that all stretching conditions (2_{min}, 4_{min}, and 8_{min}) reduced MTS immediately following the stretching exercises (**FIGURE 2, TABLE**). MTS remained lower than the control for 10 minutes poststretching after the 4_{min} and 8_{min} PS conditions. However, MTS returned to baseline within 10 minutes after the 2_{min} PS condition and within 20 minutes after the 4_{min} and 8_{min} PS conditions. A similar study by Fowles et al[7] reported that 30 minutes of PS decreased MTS for up to 30 minutes in the plantar flexors. Although the authors admitted that "...the duration of stretch performed in this experiment is more similar to prolonged stretch procedures employed in animal experimental models and, therefore, may have limited application to sport stretching performed in conjunction with athletic performance."[7] Therefore, the results of the present study extended those of previous studies[7,25] and suggested that the time course for the decreases in MTS may be dose-dependent for more practical stretching durations of 2 to 8 minutes (**FIGURE 2**).

In theory, an acute bout of stretching should reduce the amount of passive tension exhibited by a musculotendinous unit at any similar joint angle. It has been suggested that the stretching-induced decrease in passive tension (measured by a decrease in MTS) will reduce the amount of strain on the musculotendinous unit throughout a given range of motion, thereby reducing the risk of strain injury.[4,43] However, if decreasing MTS is the primary goal of stretching to reduce the risk of strain injuries, the duration of stretching necessary to elicit lasting decreases in MTS has yet to be determined.[22,25,30,32] Some studies have suggested that there may be a threshold for stretching duration that is necessary to cause a decrease in MTS.[22-24] For example, Magnusson and colleagues[22-24] reported no changes in MTS of the hamstrings following PS durations of 1.5

to 2.25 minutes. In contrast, Nordez et al[32] and Magnusson et al[25] showed that longer stretching durations (2.5 and 7.5 minutes, respectively) reduced MTS in the hamstrings. Similar findings have been demonstrated in the plantar flexors, such that longer durations of stretch (5-30 minutes) reduced MTS,[7,29] while shorter durations (1-2 minutes) had no effect on MTS.[28,30] Therefore, the results of the present study, in conjunction with those of previous studies,[28,30] suggested that if there is a threshold necessary for causing a decrease in MTS in the plantar flexor muscles, it may be approximately 2 minutes of passive stretching. Albeit, 2 minutes of PS only caused transient decreases in MTS that returned to baseline within 10 minutes. However, as the stretching duration was increased to 4 or 8 minutes, the reduction in MTS lasted longer before returning to baseline within 20 minutes.

It should be noted that Muir et al[30] showed no changes in MTS after 2 minutes of stretching the plantar flexors, whereas the present study showed significant decreases in MTS following all PS durations (including 2_{min}). It is possible that the differences between our findings and those reported by Muir et al[30] may be due to the differences in stretching treatments. Muir et al[30] used a passive constant-angle stretching protocol (the angle at which the stretch occurs is held constant), whereas the current study employed a constant-torque stretching protocol (the torque at which the stretch occurs is held constant). Recently, Yeh et al[47,48] reported that a constant-torque stretching treatment was more effective in reducing MTS of the plantar flexors for patients with hypertonicity. It was suggested that the constant-torque stretching resulted in greater "muscle creep" or strain relaxation, which may translate to greater reductions in MTS than constant-angle stretching.[47,48] It is possible that constant-torque stretching induces greater work on the musculotendinous unit for a given time, when compared to constant-angle stretching. Future studies are needed to determine which mode of stretching is most effective for reducing MTS.

There have been several hypotheses proposed to explain the mechanisms responsible for the stretching-induced decreases in MTS, which include increases in tendon compliance,[17,19] increases in fascicle length,[7,11] and alterations in intramuscular connective tissues.[8,25,29,42] For example, McHugh et al[27] suggested that MTS may be related to "tendon-aponeurosis extensibility." Kubo and colleagues[17,19] supported this hypothesis and showed that 5 to 10 minutes of static stretching decreased tendon stiffness when measured with an ultrasound device. However, conflicting data have been reported[18] that suggest that changes in MTS may be unrelated (r = 0.19) to tendon stiffness. In contrast, Halar et al[11] suggested that stretching-induced decreases in MTS may be predominately related to the changes in the muscle rather than the tendon. Fowles et al[7] supported this hypothesis in a single subject, by showing that muscle fascicles of the soleus and the lateral and medial gastrocnemius increased in length by 8, 8, and 2 mm, respectively, following 30 minutes of PS. In addition, Gajdosik[8] indicated that the noncontractile muscle proteins (composing the sarcomeric cytoskeleton), such as titin and desmin, may be lengthened during PS, which could also contribute to the decreases in MTS.

A common rationale used to explain the decreases in MTS following stretching is an alteration in intramuscular connective tissue.[8,29,42] Taylor et al[42] found that 10 repeated isometric contractions, compared to 10 passive stretches, of the rabbit anterior tibialis muscle resulted in similar decreases in passive tension. The authors[43] concluded that the decreases in passive tension were likely mediated by alterations in the connective tissues. More recently, Morse and colleagues[29] reported that 5 minutes of PS elicited a distal shift of the musculotendinous junction in the medial gastrocnemius muscle, which were not attributed to increases in tendon compliance or muscle fascicle lengths. The authors suggested, therefore, that the lengthening or deformation of intramuscular connective tissues was responsible for the decreases in MTS. Although the precise mechanism underlying the decreases in MTS cannot be determined from the present study, it is possible that the rapid return to baseline for MTS observed after 2_{min} PS, and the delayed return after 4_{min} and 8_{min} PS, may be related to the connective tissue and muscle "viscoelastic recoil."[7] Magnusson[22] suggested that this rapid return to baseline in MTS is an important property of the musculotendinous unit that contributes to elastic recoil during locomotion.[41] Future studies are needed to identify the time course of the mechanisms responsible for stretching-related decreases in MTS, particularly after practical durations of PS, and how these reductions relate to active muscle injury reduction.

Theoretically, it is possible that the PS that occurs during a MTS assessment may elicit the stretch reflex, which would cause activation of the stretched muscles. Thus, the torque curve that is recorded during a MTS measurement could be contaminated with active force production as well as passive tension. Furthermore, it has been suggested that any decreases in MTS observed after a bout of stretching may result from a stretching-induced inhibition of reflex activity.[15] Consequently, there would be a decrease in the active portion of the torque curve that could be erroneously interpreted as a decrease in MTS. However, many previous studies[9,23-26,28] have reported minimal surface EMG responses (<1% MVC) during PS and MTS assessments, which suggests that reflex-initiated activation of the stretched muscles does not occur under controlled conditions. Specifically, when the rate of stretch is slow enough (approximately 5°/s) there is a diminished likelihood that the stretch reflex would cause active force production during the MTS assessment.[14] The results of the present study indicated that mean surface EMG amplitude never exceeded

0.65% of the MVC value during any of the MTS values calculated at final 13° of ankle dorsiflexion. This lack of muscle activation is in agreement with previous findings in both human[23-26,28] and animal models,[43] and suggests that the stretching-induced changes in MTS are due to changes in the mechanical properties of the musculotendinous unit.

CONCLUSION

The results of the present study indicated that all PS interventions (2_{min}, 4_{min}, and 8_{min}) caused decreases in MTS immediately after stretching. Although MTS returned to baseline within 10 minutes for the 2_{min} PS condition and within 20 minutes for the 4_{min} and 8_{min} PS conditions. If reductions in MTS aid in the prevention of musculotendinous strain injuries, then these data suggest that practical durations of stretching (2 to 8 minutes) for the plantar flexors should be performed 10 to 20 minutes prior to the start of competition or exercise. However, given that many precompetition routines are recommended to be performed 15 to 60 minutes prior to competition,[5,46] and that the addition of continuous exercise for 30 minutes following PS has little influence on passive energy absorption,[21] it is possible that pre-event stretching has limited clinical significance in the prevention of musculotendinous injuries.[39,44]

KEY POINTS

FINDINGS: Practical durations of passive stretching (2 to 8 minutes) reduced MTS immediately poststretching; however, MTS returned to baseline within 10 minutes for the 2_{min} condition, and within 20 minutes for the 4_{min} and 8_{min} conditions.

IMPLICATION: If decreases in MTS aid in the prevention of musculotendinous injuries, stretching should be performed within 20 minutes prior to the start of competition or exercise.

CAUTION: This study only assessed the plantar flexor muscles, although similar findings have been reported in other muscles (eg, hamstrings), direct extrapolation of these results should be limited to the plantar flexor muscles.

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