StretcHing-Induced DeFicit of MaXimal Isometric Toreque Is Restored Within 10 Minutes

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Abstract
Mizuno, T, Matsumoto, M, and Umemura, Y. Stretching-induced deficit of maximal isometric torque is restored within 10 minutes. J Strength Cond Res 28(1): 147-153, 2014—The purpose of this study was to clarify the time course of the stretching-induced decrease in maximal isometric plantar flexion torque. Nineteen women participated in 2 randomly ordered experimental trials: static 5-minute stretching or control with no stretching. The participants performed isometric maximal voluntary contractions (MVCs) of the right plantar flexor muscles, whereas electromyographic (EMG) amplitude (root mean square) was calculated for the medial and lateral gastrocnemius muscles. Measurements were conducted preintervention; immediately after intervention; and 5, 10, 15, and 30 minutes postintervention. The static 5-minute stretching trial consisted of dorsiflexion to the end range of motion and holding that position for 1 minute, 5 times, whereas the control trial consisted of 5 minutes of resting. As a result, the MVC torque was significantly decreased immediately after, and 5 minutes after the static 5-minute stretching intervention compared with the preintervention value (p < 0.05), and this change recovered within 10 minutes. However, the EMG amplitude did not change from preintervention to postintervention under any conditions. These results suggest that the deficits of static stretching are disabled in a short time after static stretching.

Key Words static stretching, plantar flexors, time course, strength

Introduction
A warm-up before competitive or regular exercise is generally done to improve performance and to reduce the risk of injuries. Static stretching is traditionally involved in a warm-up. Static stretching improves the range of motion (ROM) temporarily (21,23). However, based on recent study results, static stretching before exercise has disadvantages on a muscle’s ability to exert maximal force. This phenomenon has been named the stretching-induced force deficit (2,10). The first hypothesis is that a neural factor is involved, causing a decrease in muscle activation and reflex sensitivity. The second hypothesis is that a mechanical factor is involved, causing a decrease in stiffness of the muscle-tendon unit (MTU) that may affect the muscle’s length-tension relationship. Presently, the overwhelming consensus does not approve of static stretching before exercise, especially exercise involving higher velocity and power (5). However, several studies have suggested that static stretching has no significant effect on exercise performance or can improve performance (11,31).

As mentioned above, the time course of the stretching-induced force deficit has not been well studied. A well-known study regarding the effects of static stretching on subsequent performance was performed by Fowles et al. (5,13). These authors reported that approximately 30 minutes of static stretching reduced maximal isometric force by 28% immediately after stretching with a continued 9% impairment after 60 minutes. However, 30 minutes of static stretching was not practical for actual exercise. We previously examined the time course of stiffness of the MTU and end ROM after practical static stretching and showed 2 primary findings. First, we showed that the end ROM was significantly increased >30 minutes after static 5-minute stretching compared with prestretching values (21). Second, our study showed that the stiffness of the MTU was significantly decreased immediately after stretching; however, it recovered within 15 minutes (21). Therefore, our previous study suggests that the retention time of the stretching-induced decrease in stiffness of the MTU is shorter than the retention time of the stretching-induced increase of
end ROM. Moreover, another study investigated a shorter time course in detail and showed that stiffness of the MTU was significantly decreased immediately and 5 minutes after static 5-minute stretching compared with prestretching values. However, this stiffness recovered within 10 minutes (22).

Our previous studies did not assess muscle performance with stiffness of the MTU, end ROM, and maximal force at the same time, because of methodological limitations. Given the time course data of muscle performance after static 5-minute stretching, whether a stretching-induced force deficit is restored, in accordance with the time course of stiffness of the MTU and the end ROM, should be able to be determined. Therefore, the purpose of this study was to clarify the time course of a stretching-induced decrease in maximal isometric plantar flexion torque. This work was conducted using 2 hypotheses. The first hypothesis was that maximal isometric torque is significantly decreased immediately and 5 minutes after stretching. The second hypothesis was that maximal isometric torque is not significantly decreased 10 minutes after stretching. Therefore, a stretching-induced decrease in maximal isometric torque is restored, in accordance with the time course of stiffness of the MTU, within 10 minutes after static 5-minute stretching. The results of this study could provide information on when athletes should perform static stretching before exercise.

Methods
Experimental Approach to the Problem
This study was designed to test the hypothesis that a stretching-induced decrease in maximal isometric torque is restored, in accordance with the time course of stiffness of the MTU, within 10 minutes after static 5-minute stretching. A randomized, repeated-measures, crossover design (time [preintervention, immediately after intervention, and 5-, 10-, and 30-minute postintervention] × condition [static 5-minute stretching and no stretching control]; 6 × 2) was used to determine the acute changes of static stretching on isometric peak torque. The participants visited the laboratory on 3 occasions separated by at least 24 hours. The first visit was a familiarization trial and the subsequent 2 visits included the following experimental conditions in random order: (a) static 5-minute stretching and (b) no stretching control condition. During the familiarization trial, each participant practiced isometric peak torque assessment of plantar flexion and static stretching, to minimize any potential learning effects and to adjust to the procedures. During each experimental condition, the participants underwent preintervention isometric maximal voluntary contraction (MVC) measurement of plantar flexion, an alternative treatment intervention (static 5-minute stretching or a 5-minute rest in the supine position), and postintervention MVC measurement of plantar flexion. Before the preintervention MVC measurement, the participants were instructed to rest in a sitting position for 15 minutes in our laboratory. We performed 5 postintervention MVC measurements (immediately after the intervention, and at 5, 10, 15, and 30 minutes after the intervention) to examine the time course. This experiment was completed in summer. The participants completed all the experimental trials within 2 weeks.

Subjects
Nineteen women (mean ± SD, age: 22 ± 1 years; height: 160.3 ± 4.3 cm; weight: 54.0 ± 4.4 kg) who were university sport science students volunteered for this investigation. All the subjects were active in approximately 2–4 hours of recreational or competitive sports training or competition 1–5 times per week. No participants reported any history of recent musculoskeletal injuries or neuromuscular diseases specific to the lower limb. We did not give any instructions for eating, sleeping, and drinking habits. This study was approved by the Human Subjects Committee of Chukyo University Graduate School of Health and Sport Sciences, and complied with their requirements for the Declaration of Helsinki. Appropriate consent was obtained from each subject according to Japanese law. All the subjects were fully informed of the experimental purposes, procedures, and possible risks of the study. Each subject signed corresponding informed consent before participation.

Procedures
Maximal Voluntary Contraction Measurements. To determine peak torque, the participants performed 2 MVC measurements of plantar flexion before the treatment intervention and 1 MVC measurement at each postintervention assessment. Maximal voluntary contraction measurement of plantar flexion was performed in the right lower leg of each participant. The highest torque reached during a contraction was recorded, and the greatest value of the 2 contractions during preintervention assessment was given as the MVC. After 2 submaximal isometric contractions as a warm-up, the participants performed 2 MVC measurements at an ankle angle of 0° (= 90° between the foot and the leg) and maintained MVC for 5 seconds with 2 minutes of rest to prevent development of fatigue in the preintervention assessment. They were secured to a calibrated Biodex System3 dynamometer (Biodex Medical Systems, Inc., Shirley, NY, USA) with the knee in full extension, and the footplate fixed to the right foot. The lateral malleolus of the fibula was aligned with the axis of the dynamometer in accordance with the Biodex User's Guide (Biodex Pro Manual, Applications/Operations, Biodex Medical Systems, Inc.). To restrain movements of the ankle joint and knee joint, the foot was secured in a heel cup attached to a footplate with toe and ankle straps over the metatarsals and malleoli, and the knee was secured by straps of the dynamometer and a brace of the knee (straight position knee-joint immobilizer) (Kneebrace; Alcare, Tokyo, Japan). During the assessments, the participants were requested to give maximal effort for each trial. In this study, 2 MVC measurements were performed at the preintervention assessment because of test-retest reliability, although 1 MVC measurement was performed at each postintervention assessment. To maximize performance, verbal encouragement was given to all the participants by investigators. Planter flexion
Torque was converted from analog to digital at a sampling rate of 1.5 kHz (LX-10; TEAC, Tokyo, Japan).

In this study, the rate of torque development (RTD) at 50% MVC was calculated using a second-order polynomial regression model that was fit to the plantar flexion torque-time curve at the 7 points of 10, 20, 30, 40, 50, 60, and 70% MVC. The RTD at 50% MVC was calculated as the slope of the polynomial fit of the plantar flexion torque-time curves. The RTD was determined at 50% MVC (Figure 1). The RTD was determined in 15 participants, because 4 participants did not have completely recorded data.

**Surface Electromyography.** During MVC assessments, preamplified bipolar, active surface electrodes (DL-141; S&ME, Tokyo, Japan) were placed on the most prominent bulge of the medial and lateral gastrocnemius muscles with a fixed center-to-center interelectrode distance of 12 mm. Electromyography activity was recorded with a bandwidth of 5–2,000 Hz. Electromyography signals were transmitted to a digital data recorder at a sampling rate of 1.5 kHz (LX-10; TEAC). The EMG amplitude values were calculated using a root mean square.

**Static 5-minute Stretching Procedure.** Repeated static stretching was performed using the isokinetic dynamometer (Biodex System3; Biodex Medical Systems, Inc.) in the same fashion as previously described (21,22). Static stretching was performed in the right lower leg of each participant. The leg was secured on an isokinetic machine with the knee in full extension. The footplate attached to the isokinetic machine was fixed securely to the right foot of each participant. The right foot was passively dorsiflexed at a constant velocity of 1°·s⁻¹ from 10° of plantar flexion to a position of maximal dorsiflexion angle that provoked a sensation in the triceps surae muscle similar to a static stretching maneuver without pain. The participant stopped the dynamometer by activating the safety trigger. This position was then held at a constant angle for 1 minute. Thereafter, the footplate was returned to a position of 10° of plantar flexion. This stretching procedure was repeated 5 times. The maximal dorsiflexion angle was reassessed at each dorsiflexion. Throughout stretching, the participants were requested to relax completely and not offer any voluntary resistance.

**Reliability**
Test-retest reliability was calculated for the preintervention assessments between 2 intervention conditions for MVC, and EMG amplitude of the medial and lateral gastrocnemius muscles. For 19 participants measured >24 hours apart, the interclass correlation coefficients (ICCs) and 95% confidence intervals were 0.87 (0.70–0.95), 0.80 (0.55–0.92), and 0.77 (0.50–0.91), respectively. In addition, the within-day ICCs calculated for the static 5-minute stretching condition between 2 MVC measurements during preintervention assessment for MVC, and EMG amplitude of the medial and lateral gastrocnemius muscles were 0.95 (0.88–0.98), 0.96 (0.91–0.99), and 0.90 (0.75–0.96), respectively.

**Statistical Analyses**
The independent variables were time (preintervention, immediately after intervention, and 5,
Retention Time of the Stretching-Induced Force Deficit

10, 15, and 30 minutes postintervention), condition (static 5-minute stretching and no stretching control) and muscle (medial and lateral). The dependent variables were MVC (newton meters), RTD (newton meter per millisecond), and EMG (microvolts). To determine the interactions between time and condition, the MVC torque and the RTD were tested using 1 separate 2-way repeated measures analysis of variance (ANOVA) (time [preintervention; immediately after intervention; and 5, 10, 15, and 30 minutes postintervention] × condition [static 5-minute stretching and no stretching control]). To determine the interactions between time and condition and muscle, the EMG amplitude data were tested using 1 separate 3-way repeated measures ANOVA (time [preintervention, immediately after intervention, and 5, 10, 15, and 30 minutes postintervention] × condition [static 5-minute stretching and no stretching control] × muscle [medial and lateral]). When appropriate, follow-up analyses were performed using t-tests with Bonferroni corrections. Statistical significance was set at \( p \leq 0.05 \). All data are reported as mean ± SD.

**RESULTS**

**Maximal Voluntary Contraction Torque**

A significant 2-way interaction (time × condition) was identified (Figure 2). Post hoc testing showed that static stretching decreased isometric peak torque immediately and 5 minutes after stretching compared with that at preintervention (both \( p < 0.05 \)). However, decreased isometric peak torque was restored for 10, 15, and 30 minutes after stretching compared with immediately after stretching (all \( p < 0.05 \)). In the control condition, no significant change was identified at each postintervention assessment compared with preintervention, although repeated MVC increased isometric peak torque for 30 minutes after rest intervention compared with immediately after rest intervention (\( p < 0.05 \)). In addition, static stretching decreased isometric peak torque immediately after intervention, and 15 and 30 minutes after stretching compared with that in the control condition (\( p < 0.05 \)).

**Rate of Torque Development**

No significant 2-way interaction (time × condition) and no significant main effect were observed for condition, but a significant main effect for time was apparent. However, no significant differences were identified among any levels (Figure 3).

**Electromyography**

For EMG, no significant 3-way interaction (time × condition × muscle), no significant 2-way interaction (time × condition, time × muscle, or condition × muscle), and no significant main effect were observed for time, condition, or muscle. Electromyography amplitude

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### Table 1. Electromyography in the medial and lateral gastrocnemius muscles during isometric maximal voluntary contractions.*†

| Rest interval (min) |  |  |  |  |  |
|--------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                    | Stretching      | Control         | Stretching      | Control         | Stretching      | Control         | Stretching      | Control         |
|                    | Medial          | Lateral         | Medial          | Lateral         | Medial          | Lateral         | Medial          | Lateral         |
| Pre                | 0.24 ± 0.13     | 0.18 ± 0.10     | 0.26 ± 0.13     | 0.16 ± 0.11     | 0.24 ± 0.10     | 0.15 ± 0.06     | 0.24 ± 0.11     | 0.09 ± 0.07     |
| Immediately        | 0.27 ± 0.15     | 0.21 ± 0.19     | 0.24 ± 0.10     | 0.15 ± 0.06     | 0.24 ± 0.11     | 0.15 ± 0.06     | 0.24 ± 0.11     | 0.15 ± 0.06     |
| Post 5             | 0.24 ± 0.12     | 0.18 ± 0.10     | 0.24 ± 0.11     | 0.15 ± 0.06     | 0.24 ± 0.11     | 0.15 ± 0.06     | 0.24 ± 0.11     | 0.15 ± 0.06     |
| Post 10            | 0.24 ± 0.11     | 0.16 ± 0.07     | 0.23 ± 0.09     | 0.15 ± 0.05     | 0.24 ± 0.11     | 0.15 ± 0.06     | 0.24 ± 0.11     | 0.15 ± 0.06     |
| Post 15            | 0.24 ± 0.11     | 0.15 ± 0.08     | 0.23 ± 0.09     | 0.15 ± 0.06     | 0.24 ± 0.11     | 0.15 ± 0.06     | 0.24 ± 0.11     | 0.15 ± 0.06     |
| Post 30            | 0.26 ± 0.12     | 0.17 ± 0.09     | 0.26 ± 0.11     | 0.17 ± 0.07     | 0.26 ± 0.11     | 0.17 ± 0.07     | 0.26 ± 0.11     | 0.17 ± 0.07     |

*Pre = preintervention; immediately = just after intervention; post = postintervention.
†Values represent mean ± SD.
produced muscle activation after 0.05). p

Static 5-minute stretching-induced changes in the range of motion (ROM), stiffness of the muscle-tendon unit (MTU), and isometric maximal voluntary contraction (MVC) torque. Values include the results of this study and those of Mizuno et al. (21,22). *Reported significant difference compared with preintervention (p < 0.05).

Figure 4. Static 5-minute stretching-induced changes in the range of motion (ROM), stiffness of the muscle-tendon unit (MTU), and isometric maximal voluntary contraction (MVC) torque. Values include the results of this study and those of Mizuno et al. (21,22). *Reported significant difference compared with preintervention (p < 0.05).

The purpose of this study was to clarify the time course of a stretching-induced decrease in maximal isometric plantar flexion torque. The main finding of the present investigation was that decreased isometric peak torque immediately after static 5-minute stretching lasted for 5 minutes. However, this change recovered within 10 minutes after stretching. These results suggest that the mechanical property of the MTU may be a major contributor to the stretching-induced force deficit.

The major contributor of the stretching-induced force deficit is unclear (i.e., either a neural factor or mechanical factor). McHugh and Cosgrave (17) considered that it may be easier to initiate a neural affect than a viscoelastic effect. In contrast, several previous studies have indicated that neural effects are more transient (14) or play a smaller (19) or insignificant role in the stretching-induced force deficit (8,16,18,19,30). Therefore, the potential mechanisms underlying the stretching-induced force deficit are not completely understood, and further study has been encouraged to clarify these mechanisms (28). To extend our understanding of the stretching-induced force deficit, we compared the integrated results of our previous studies, which evaluated the time course of the end ROM and stiffness of the MTU with this study, which evaluated the time course of the isometric peak torque after static 5-minute stretching (Figure 4) (21,22). All the results of previous studies used similar research methods, although the participants were recreationally active men. The increase in the end ROM lasts 30 minutes after stretching, although the decrease in stiffness of the MTU and isometric peak torque is restored within 10 minutes. The most important finding shown in Figure 4 is that the time course of the stiffness of the MTU is in accordance with the time course of isometric peak torque. This agreement indicates that a mechanical factor may be a major contributor of the stretching-induced force deficit.

Two primary hypotheses have been proposed to explain the stretching-induced force deficit (12,10). The first hypothesis is that a neural factor is involved in the stretching-induced force deficit, and this causes decreased muscle activation and reflex sensitivity. Decreased amplitude of surface EMG during MVCs after static stretching provides evidence that the stretching-induced force deficit is attributable to a neural effect (1,2). Some peripheral mechanisms have been proposed to explain the reduced muscle activation after stretching as follows (2,4,13): (a) autogenic inhibition of the Golgi tendon reflex, (b) mechanoreceptors and nociceptors, (c) fatigue-induced inhibition, (d) joint pressure feedback inhibition because of excessive ranges of motion during stretching, and (e) stretch reflex inhibition originating from the muscle spindles. In addition, Cramer et al. (10) reported decreases in strength and muscle activation for stretched and unstretched limbs. As a result, the authors suggested that the stretching-induced decrease in maximal force production was because of a decrease in motor unit activation and firing frequency caused by an unidentified central nervous system inhibitory mechanism (10). In contrast, this study showed no significant changes in EMG amplitude, which is consistent with the findings of a previous study (29). Therefore, we conclude from the present results regarding EMG amplitude that a neural factor is not involved in the stretching-induced force deficit.

It has also been hypothesized that the stretching-induced force deficit is because of potential mechanical alterations in MTU stiffness, which affects the length-tension relationship (13). In fact, alteration of the muscle’s length-tension relationship after static stretching provides evidence that the stretching-induced force deficit can be attributed to mechanical or viscoelastic changes (26). Decreased stiffness of the MTU after static stretching may increase the resting length of the sarcomeres, which in turn may alter the length-tension relationship (9,13,15). Furthermore, alterations of the length-tension relationship and the plastic deformation of connective tissues after static stretching could limit the maximal force-producing capabilities of the MTU (13,15). Rassier et al. (26) suggested that a lengthened muscle because of an acute bout of static stretching could have a less than optimal crossbridge overlap.
that, according to the length-tension relationship, could diminish maximum muscle force output. Previous studies have also suggested that a stretching-induced decrease in evoked twitch properties might reflect the muscle’s inability to generate force because of a more compliant MTU (4,13,27). As well as these previous studies, this study also indicated that static stretching affected mechanical properties, because stretching tended to decrease the RTD at 50% MVC. In our study, this tendency was supported by the finding of a significant decrease in the RTD immediately after stretching compared with that at pre-stretching by 1-way ANOVA, whereas a 2-way ANOVA yielded no significant results. In addition, we also showed that a reduction in stiffness of the MTU and muscle was observed immediately after static-5-minute stretching (~13 and 22%, respectively) and 5 minutes after stretching (~17 and 30%, respectively) (22). Therefore, these results suggest that a mechanical factor is involved in the stretching-induced force deficit. The present results also found that the potential mechanical alterations in MTU stiffness disappear within 10 minutes.

In conclusion, the deficit of maximum isometric plantar flexion strength after static 5-minute stretching by holding the ankle joint at maximal dorsiflexion is restored within 10 minutes. This finding suggests that the defects of static stretching will be disabled in a short time after static stretching, although static stretching decreases maximum isometric strength immediately after stretching. In addition, taken together with our previous study findings, the mechanical property of the MTU may be a major contributor to the stretching-induced force deficit.

Practical Applications

As part of the warm-up routine, static stretching is commonly performed before exercise and athletic performance. This study and several previous studies (13,29) show that static 5-minute stretching decreases maximal isometric plantar strength immediately after stretching. However, this impairment is restored within 10 minutes after stretching. In general, warm-up includes not only static stretching but also jogging or running. A previous study reported that 5 minutes of running before and after static stretching did not impair performance (24). Therefore, a warm-up consisting of static stretching and activity and movement renders the stretching-induced force deficit practically ineffectual. In addition, taking into consideration the fact that preexercise stretching is usually performed at least 10 minutes before the start of competition, and that in practice, the actual time spent stretching for a single site is shorter, the disadvantages of static stretching on exercise performance are probably extremely small. Therefore, athletes should not perform static stretching within 10 minutes before a competition if they do not want to decrease maximal isometric plantar strength after static stretching. In addition, athletes should perform static stretching 10–30 minutes before a competition if they want to increase end ROM without decreasing maximal isometric plantar strength after static stretching.

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