

Do Practical Durations of Stretching Alter Muscle Strength? A Dose–Response Study

ERIC D. RYAN¹, TRAVIS W. BECK¹, TRENT J. HERDA¹, HOLLY R. HULL², MICHAEL J. HARTMAN³, JEFFERY R. STOUT⁴, and JOEL T. CRAMER¹

¹Biophysics Laboratory, Department of Health and Exercise Science, University of Oklahoma, Norman, OK; ²NY Obesity Research Center, College of Physicians & Surgeons Body Composition, Columbia University, New York, NY; ³Human Performance Laboratory, Department of Exercise Science and Health Promotion, Florida Atlantic University, Davie, FL; and ⁴Metabolism and Body Composition Laboratory, Department of Health and Exercise Science, University of Oklahoma, Norman, OK

ABSTRACT

RYAN, E. D., T. W. BECK, T. J. HERDA, H. R. HULL, M. J. HARTMAN, J. R. STOUT, and J. T. CRAMER. Do Practical Durations of Stretching Alter Muscle Strength? A Dose–Response Study. *Med. Sci. Sports Exerc.*, Vol. 40, No. 8, pp. 1529–1537, 2008. **Purpose:** To examine the time course (immediate, 10, 20, and 30 min) for the acute effects of 2, 4, and 8 min of passive stretching (PS) on isometric peak torque (PT), percent voluntary activation (%VA), EMG amplitude, peak twitch torque (PTT), rate of twitch torque development (RTD), and range of motion (ROM) of the plantarflexors. **Methods:** Thirteen volunteers (mean \pm SD age, 22 \pm 3 yr) participated in four randomly ordered experimental trials: control (CON) with no stretching, 2 min (PS₂), 4 min (PS₄), and 8 min (PS₈) of PS. Testing was conducted before (pre), immediately after (post), and at 10, 20, and 30 min poststretching. The PS trials involved varied repetitions of 30-s passive stretches, whereas the CON trial included 15 min of resting. PT, %VA, EMG amplitude, PTT, and RTD were assessed during the twitch interpolation technique, whereas ROM was quantified as the maximum tolerable angle of passive dorsiflexion. **Results:** PT decreased ($P \leq 0.05$) immediately after all conditions [CON (4%), PS₂ (2%), PS₄ (4%), and PS₈ (6%)] but returned to baseline at 10, 20, and 30 min poststretching. %VA and EMG amplitude were unaltered ($P > 0.05$) after all conditions. PTT and RTD decreased ($P \leq 0.05$) immediately after the PS₄ (7%) and the PS₈ (6%) conditions only; however, these changes were not sufficient to alter voluntary force production. There were also increases ($P \leq 0.05$) in ROM after the PS₂ (8%), the PS₄ (14%), and the PS₈ (13%) conditions that returned to baseline after 10 min. **Conclusion:** Practical durations of stretching (2, 4, or 8 min) of the plantarflexors did not decrease isometric PT compared with the CON but caused temporary improvements in the ROM, thereby questioning the overall detrimental influence of PS on performance. **Key Words:** STRETCHING-INDUCED FORCE DEFICIT, EMG, MUSCLE ACTIVATION, RANGE OF MOTION

Stretching is commonly performed in a variety of settings (i.e., clinical, fitness, and athletic) with the intent to increase flexibility or the pain-free range of motion (ROM) about a joint. Clinically, reduced ankle ROM is related to several leg disorders, including Achilles tendinitis (17) and plantar fasciitis (29). In the fitness and athletic setting, stretching is often performed with the belief that increasing flexibility will improve performance (32) and/or reduce the risk of injury (9). However, these traditionally accepted concepts have been heavily scrutinized by many recent literature reviews (31,34) and original research studies (3,11,27,28,36). For example, recent

studies have suggested that preexercise stretching may temporarily compromise a muscle's ability to produce maximal force—a phenomenon that has since been termed the “stretching-induced force deficit.” In fact, stretching has been reported to reduce isometric force production (3,11), concentric isokinetic peak torque (PT) (4,5,10), and muscle strength endurance (28) in untrained subjects. In addition, stretching has been shown to decrease sprint speed (27,38) and vertical jump height (16) in trained athletes; however, these findings have been challenged by other studies that have reported no changes in vertical jump height (35) and concentric isokinetic PT (8) in collegiate women's basketball players. Overall, though, the majority of studies have reported some stretching-induced decrements in performance (31,34) in both trained and untrained participants, and we are aware of no studies that have demonstrated any acute improvements in performance as a result of stretching.

There have been two main hypotheses proposed to explain the stretching-induced force deficit: a) neural factors, such as decreases in muscle activation, and b) mechanical factors, such as decreases in musculotendinous stiffness that may affect the muscle's length–tension relationship and/or sarcomere shortening velocity. Previous

Address for correspondence: Joel T. Cramer, Ph.D., Biophysics Laboratory, Department of Health and Exercise Science, University of Oklahoma, 1401 Asp Avenue, HHC Room 12, Norman, OK 73019-6081; E-mail: jrcramer@ou.edu.

Submitted for publication November 2007.

Accepted for publication February 2008.

0195-9131/08/4008-1529/0

MEDICINE & SCIENCE IN SPORTS & EXERCISE®

Copyright © 2008 by the American College of Sports Medicine

DOI: 10.1249/MSS.0b013e31817242eb

studies have shown stretching-induced decreases in muscle activation using surface electromyography (EMG) (3–5,11) and the twitch interpolation technique (3,11). Cramer et al. (5) also reported stretching-induced decreases in PT and surface EMG amplitude in both the stretched and the unstretched (contralateral) leg extensor muscles. The authors suggested that the stretching-induced force deficit may be related to an unidentified central nervous system inhibitory mechanism. On the other hand, it has been suggested that the stretching-induced decreases in force production are due to alterations in the mechanical or contractile properties of the musculotendinous unit (4,13). For example, several studies have suggested that stretching may increase the resting length of the sarcomeres, which may alter the muscle's length–tension relationship, and/or influence the rate of sarcomere shortening velocity (4,13). Herda et al. (13) demonstrated joint-angle-specific decreases in isometric torque production of the hamstrings after static stretching that were most evident at the two shortest muscle lengths. Changes in evoked muscle twitch properties as a result of stretching have also been reported, which may be related to decreases in musculotendinous stiffness (3,11). Nevertheless, the potential neural and mechanical mechanisms underlying the stretching-induced force deficit are not completely understood, and further research has been encouraged (31) to elucidate these mechanisms. Furthermore, it is unclear whether these potential underlying mechanisms are different for untrained subjects as opposed to elite athletes (8,27,35,38), and more research is needed to exploit this difference in training status.

The study by Fowles et al. (11) is perhaps one of the most commonly cited articles regarding the stretching-induced force deficit. These authors were the first to examine the underlying mechanisms and time course for the stretching-induced force deficit. Fowles et al. (11) reported that 30 min of passive stretching reduced isometric PT by 28%, and a 9% force deficit was still present at 1 h poststretching. These results as well as those of previously mentioned studies (2,20,27) have raised significant concerns among practitioners and health professionals that preexercise stretching may have detrimental effects on muscle strength and athletic performance. However, Fowles et al. (11) admittedly used an exaggerated 30-min stretching protocol that was "...similar to prolonged stretch procedures employed in animal experimental models..." (p. 1179). The authors concluded that "Further testing with a stretching protocol more similar to that regularly performed in the athletic context should be evaluated under the controlled conditions of this study" (p. 1187). To date, however, no studies have examined the potential dose–response relationship that may govern the stretching-induced force deficit. Therefore, the primary purpose of the present study was to extend the findings of Fowles et al. (11) and to examine the acute effects of shorter stretching durations (2, 4, and 8 min) on plantarflexion strength (isometric PT; PT), activation [percent voluntary activation

(%VA) and surface EMG amplitude], twitch properties [peak twitch torque (PTT) and rate of torque development (RTD)], and ankle ROM in moderately active, recreationally trained subjects. The secondary purpose of this study was to examine the time course for any acute effects of passive stretching by assessing neuromuscular function immediately after the stretching and at 10, 20, and 30 min poststretching.

Based on previous studies (5,11,14,36), we hypothesized that passive stretching would elicit a transient, dose-dependent decrease in muscle strength and activation, such that the longer stretching durations would elicit the greatest force deficits that would remain depressed for longer periods of time (11). This study was also designed to identify the mechanisms underlying the stretching-induced force deficit by examining the neural (%VA and EMG amplitude) and mechanical (PTT, RTD, and ROM) aspects of voluntary and evoked isometric plantarflexion (36). It was further hypothesized that any improvements in ankle ROM will also be dose dependent, with the longer durations of stretching yielding greater and more prolonged increases in ROM than shorter durations. In addition, because the primary purpose of the present study was to extend the findings of Fowles et al. (11), we chose to study moderately active recreationally trained subjects to be consistent with their sample demographics. Therefore, overall we hypothesized that examining the stretching-induced changes in PT, %VA, EMG amplitude, PTT, RTD, and ROM would provide information underlying the stretching-induced force deficit and determine whether the duration of stretching influences the time course for these changes.

METHODS

Subjects. Seven men (mean \pm SD age, 24 ± 4 yr; stature, 178 ± 7 cm; mass, 83 ± 12 kg) and six women (21 ± 1 yr; 157 ± 5 cm; 55 ± 6 kg) volunteered for this investigation. No one reported any current or ongoing neuromuscular diseases or musculoskeletal injuries specific to ankle, knee, or hip joints. None of the participants were competitive athletes; however, due to their reported levels of aerobic exercise (4 ± 1 h·wk⁻¹), resistance training (4 ± 3 h·wk⁻¹), and recreational sports (3 ± 2 h·wk⁻¹), these participants might be best classified as normal, moderately active, and recreationally trained. This study was approved by the University Institutional Review Board for Human Subjects Research, and all participants completed an informed consent form and a preexercise testing health status questionnaire. Using the procedures described by Gravetter and Wallnau (12) for estimating sample sizes for repeated-measures designs, a minimum sample size of $n = 6$ was required to reach a statistical power ($1 - \beta$) of 0.80 based on the findings of Fowles et al. (11).

Experimental design. A randomized repeated-measures design [time (pre- vs posttreatment vs 10 vs 20 vs 30 min posttreatment) \times condition (CON vs PS₂ vs PS₄

vs PS₈); 5 × 4] was used to examine the acute effects of repeated passive stretching (PS) on isometric PT, percent voluntary muscle activation (%VA), passive range of motion (PROM), PTT, RTD, and EMG amplitude of the soleus (SOL) and medial gastrocnemius (MG) muscles during isometric maximal voluntary contractions (MVCs) of the plantarflexors. The participants visited the laboratory on five occasions each separated by 3–7 d. The first visit was a familiarization trial, and the subsequent four visits included the following experimental conditions in random order: a) control (CON) condition, b) PS for 2 min (PS₂), c) PS for 4 min (PS₄), and d) PS for 8 min (PS₈) of the plantarflexors. During each experimental condition, the participants underwent the pretreatment assessments, the treatment intervention, and the posttreatment assessments. For the CON condition, the pre- and posttreatment assessments were separated by a 15-min resting period, which was equivalent to the total duration of the 8-min PS condition (including rest between stretches). The posttreatment assessments occurred immediately after the PS or the CON conditions as well as 10, 20, and 30 min after the PS or the CON conditions to examine the time course for any effects. All experimental trials were performed at the same time of day (±2 h) for each subject.

Familiarization trial. Three to five days before the experimental trials, each participant signed the informed consent form, completed the health status questionnaire, and practiced the PT, the PROM, and the %VA assessments to assure that they were comfortable with the procedures and to minimize any potential learning effects.

Isometric strength assessment. To determine PT (N·m) and %VA, each participant performed two 5-s isometric MVCs before the experimental conditions and one 5-s isometric MVC at each posttreatment assessment (posttreatment and 10, 20, and 30 min posttreatment). All MVCs were performed at a neutral ankle joint angle (0° = 90° between the foot and the leg) with a leg flexion angle of 0° below the horizontal plane (full extension) on a calibrated Biodex System 3 dynamometer (Biodex Medical Systems, Inc., Shirley, NY, USA). The participants were seated with restraining straps over the pelvis, the trunk, and the thigh, with the lateral malleolus of the fibula 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.). In addition, the foot was secured in a heel cup attached to a footplate with toe and ankle straps over the metatarsals and malleoli. Two minutes of rest was allowed between the pretreatment MVCs. The MVC trials were used to assess voluntary PT (before the superimposed twitch) as well as %VA (see below). The participants were instructed to give a maximum effort for each trial, and strong verbal encouragement was provided by the investigators.

Percent voluntary activation. The twitch interpolation technique was used to determine %VA, PTT, and RTD. Transcutaneous electrical stimuli were delivered to the tibial

nerve using a high-voltage (maximal voltage, 400 V) constant-current stimulator (Digitimer DS7A, Hertfordshire, UK). The cathode was a metal probe (8 mm diameter) with the tip covered in a saline-soaked sponge, which was pressed over the tibial nerve in the popliteal fossa. The anode was a 9 × 5-cm rectangular self-adhesive electrode (Durastick Supreme, Chattanooga Group, Hicton, TN, USA) that was positioned between the patella and the tibial tuberosity. Single square wave stimuli (1 ms in duration) were used to determine the optimal probe location (20 mA) as well as the maximal compound muscle action potential (M-wave) with incremental amperage increases (2–100 mA). Once a plateau in the peak-to-peak M-wave was determined, despite amperage increases, 20% was added to the amperage that yielded the highest peak-to-peak M-wave to assure a supramaximal stimulus. Doublets (two single stimuli delivered successively at 100 Hz) were administered with the supramaximal stimulus intensity during the MVC trials to increase the signal-to-noise ratio and to minimize the series elastic effects on evoked torque production (7). In accordance with the twitch interpolation procedure, a supramaximal doublet was administered 350–500 ms into the MVC plateau (superimposed twitch) and then again 3–5 s after the MVC trial at rest (potentiated twitch). The comparison of the interpolated twitch to a resting, potentiated twitch using a doublet stimuli has been recommended by Shield and Zhou (33). %VA was calculated with the following equation (33):

$$\%VA = \left[1 - \left(\frac{\text{superimposed twitch}}{\text{potentiated twitch}} \right) \right] \times 100$$

Passive range of motion. The PROM of the plantarflexors was determined for each participant during the pre- and posttreatment assessments using the Biodex isokinetic dynamometer programmed in passive mode. The maximum tolerable torque threshold was determined for each individual during the familiarization trial as the point of discomfort, but not pain, as verbally acknowledged by the subject during a series of passive stretches of the plantarflexors. The dynamometer lever arm passively dorsiflexed the foot at an angular velocity of 5°·s⁻¹ until the torque threshold was met and held for 5 s, similar to the procedures of Muir et al. (26). PROM (°) was calculated as the ROM attained from 0° (neutral) to the maximum tolerable point of passive dorsiflexion.

Surface electromyography. Preamplified bipolar, active surface electrodes (EL254S, Biopac Systems Inc., Santa Barbara, CA, USA; gain, 350) with a fixed center-to-center interelectrode distance of 20 mm were placed over MG and SOL muscles. For the SOL, the electrodes were placed along the longitudinal axis of the tibia at 66% of the distance between the medial condyle of the femur and the medial malleolus. The electrodes for the MG were placed on the most prominent bulge of the muscle in accordance with the recommendations of Hermens et al. (15). A single

pregelled, disposable electrode (Ag-Ag Cl, Quinton Quick Prep, Quinton Instruments Co., Bothell, WA, USA) was placed on the spinous process of the seventh cervical vertebrae to serve as a reference electrode. To reduce interelectrode impedance and to increase the signal-to-noise ratio, local areas of the skin were shaved and cleaned with isopropyl alcohol before placement of the electrodes.

Signal processing. The EMG and torque signals were recorded simultaneously with a Biopac data acquisition system (MP150WSW, Biopac Systems, Inc.) during each isometric MVC. The torque (N·m) signal from the dynamometer and the EMG (μ V) signals recorded from the SOL and MG were sampled at 2 kHz. All signals were stored on a personal computer (Dell Inspiron 8200, Dell, Inc., Round Rock, TX, USA) and processed off-line using custom written software (LabVIEW v 7.1, National Instruments, Austin, TX, USA). The EMG signals were digitally filtered (zero-phase fourth-order Butterworth filter) with a pass band of 10–500 Hz. The torque signal was low-pass filtered with a 10-Hz cutoff (zero-phase fourth-order Butterworth filter) and was gravity-corrected so that the baseline torque value was 0 N·m. All subsequent analyses were performed on the filtered signals.

Isometric MVC torque (N·m) was calculated as the average torque value during the 0.25-s epoch taken immediately before the superimposed twitch. Consequently, the same (concurrent) 0.25-s epochs were selected from the EMG signals to calculate the time domain (amplitude) values during the MVC trials. The time domain was represented as the root mean square amplitude value.

PTT and RTD were calculated based on the potentiated twitch evoked at rest during the twitch interpolation procedure. PTT was defined as the highest average torque value achieved for any 20 consecutive data points (i.e., 0.01-s epochs) during the potentiated twitch. RTD was determined as the highest slope value (torque / time) calculated for any 20 consecutive data points (i.e., 0.01-s epochs) from the onset of torque production to the location of the PTT.

Passive stretching. The repeated PS of the right plantarflexor muscles was performed on the isokinetic dynamometer in the same fashion as the PROM assessments. The dynamometer passively dorsiflexed the foot until the predetermined torque threshold was met. The dynamometer maintained this constant passive torque (39), which stretched the plantarflexors for 30 s and was then released for 20 s (8) in accordance with the procedures of Yeh et al. (39). Each stretch was repeated until the specific time under stretch for each condition was completed (i.e., the PS₂ condition involved four 30 s stretches for a total of 2 min of time under stretch).

Reliability. Based on the procedures described by Weir (37), test-retest reliability was calculated for all the prestretching assessments during each experimental trial (CON, PS₂, PS₄, and PS₈) for PT, EMG amplitude of the SOL and MG, and ROM. For 13 subjects measured 3–7 d

apart, the intraclass correlation coefficients (ICC) were 0.94, 0.83, 0.76, and 0.87, respectively, with no significant ($P > 0.05$) differences among mean values. In addition, the within-day ICCs calculated for the CON condition across time (pre-, post-, 10, 20, and 30 min poststretching) for PT, EMG amplitude of the SOL and MG, and ROM were 0.98, 0.93, 0.92, and 0.97, respectively, with no significant ($P > 0.05$) differences among mean values.

Statistical analyses. Five separate two-way repeated-measures ANOVAs [time (pre vs post vs 10 min vs 20 min vs 30 min posttreatment) \times condition (CON vs PS₂ vs PS₄ vs PS₈)] were used to analyze the PT, %VA, PTT, RTD, and PROM data. Surface EMG amplitude values were normalized to the pretreatment MVC values for all posttreatment conditions (18); therefore, one separate three-way repeated-measures ANOVA [time (pre- vs posttreatment vs 10 min vs 20 min vs 30 min) \times condition (CON vs PS₂ vs PS₄ vs PS₈) \times muscle (SOL vs MG)] were used to analyze the normalized EMG amplitude data. When a significant interaction was found, follow-up analyses were performed using one-way repeated-measures ANOVAs with Bonferroni corrections. An alpha of $P \leq 0.05$ was used to determine statistical significance.

RESULTS

Table 1 contains the mean \pm SE values for each of the dependent variables (PT, %VA, EMG amplitude, PTT, RTD, and PROM).

Peak torque. There was no significant two-way interaction (condition \times time, $P = 0.165$) and no main effect for condition ($P = 0.770$), but there was a significant main effect for time ($P = 0.050$). PT decreased from pre- to posttreatment for all conditions (CON, PS₂, PS₄, PS₈); however, PT returned to pretreatment values for the remaining periods (10, 20, and 30 min posttreatment) (Fig. 1).

Peak twitch torque. There was a significant two-way interaction (condition \times time, $P = 0.047$). PTT decreased from pre- to poststretching for PS₄ ($P = 0.031$) and PS₈ ($P = 0.041$) conditions but then returned to the pretreatment values for the remaining periods (10, 20, and 30 min posttreatment) (Fig. 2).

Rate of torque development. There was a significant two-way interaction (condition \times time, $P = 0.011$). Like PTT, RTD decreased from pre- to poststretching for PS₄ ($P = 0.033$) and PS₈ ($P = 0.017$) conditions but then returned to pretreatment values for the remaining periods (10, 20, and 30 min posttreatment) (Fig. 3).

Passive range of motion. There was a significant two-way interaction (condition \times time, $P < 0.001$). PROM increased pre- to poststretching for the PS₂ ($P = 0.002$), PS₄ ($P < 0.001$), and PS₈ ($P < 0.001$) conditions but then returned to pretreatment values for the remaining periods (10, 20, and 30 min posttreatment) (Fig. 4).

Percent voluntary activation. There was no significant two-way interaction (condition \times time, $P = 0.303$)

TABLE 1. Pre- and posttreatment and 10-, 20-, and 30-min mean (SE) values of the dependent variables for the CON, PS₂, PS₄, and PS₈ treatments.

Condition	Isometric MVC PT (N·m)	%VA	Variables					
			EMG Amplitude for the Soleus (%MVC)	EMG Amplitude for the Medial Gastrocnemius (%MVC)	PTT (N·m)	RTD (N·m·s ⁻¹)	ROM (°)	
CON	Pretreatment	129.5 (12.8)	96.1 (0.9)	100.0	100.0	37.3 (2.7)	504.5 (37.3)	26.9 (1.5)
	Posttreatment	125.6 (13.3)*	96.0 (1.8)	95.0 (4.1)	99.3 (3.4)	37.4 (2.6)	495.2 (34.9)	26.0 (1.6)
	10 min	124.8 (12.9)	95.1 (1.3)	94.0 (5.2)	94.2 (4.3)	37.0 (2.5)	491.6 (37.2)	26.5 (1.6)
	20 min	126.1 (13.0)	96.0 (1.9)	100.1 (4.1)	94.7 (5.6)	36.9 (2.4)	486.6 (34.0)	27.0 (1.6)
	30 min	127.3 (12.9)	96.8 (1.5)	101.6 (2.7)	96.0 (3.7)	36.6 (2.5)	477.6 (35.1)	26.7 (1.4)
PS ₂	Pretreatment	129.9 (10.6)	96.9 (1.0)	100.0	100.0	37.8 (2.3)	509.5 (32.0)	27.4 (1.6)
	Posttreatment	127.3 (10.4)*	97.8 (0.6)	101.9 (5.4)	90.8 (4.1)	37.4 (2.4)	501.6 (32.6)	29.5 (1.6)*
	10 min	128.8 (11.5)	96.7 (1.1)	103.1 (6.0)	94.9 (5.5)	37.9 (2.3)	514.8 (32.7)	26.5 (1.5)
	20 min	126.2 (12.0)	96.3 (1.5)	98.6 (5.4)	89.6 (6.0)	37.7 (2.2)	502.9 (31.6)	26.5 (1.5)
	30 min	124.5 (10.7)	97.4 (1.2)	98.2 (6.4)	85.5 (4.2)	37.0 (2.1)	498.2 (29.6)	26.9 (1.4)
PS ₄	Pretreatment	130.7 (12.1)	97.6 (0.6)	100.0	100.0	37.0 (2.4)	496.9 (34.2)	26.6 (1.7)
	Posttreatment	124.2 (11.5)*	97.7 (0.7)	106.0 (6.1)	100.5 (4.7)	34.7 (2.4)*	468.2 (32.1)*	30.3 (2.0)*
	10 min	127.2 (13.1)	96.6 (1.0)	96.6 (3.4)	100.8 (5.1)	36.7 (2.5)	498.6 (33.1)	27.1 (1.7)
	20 min	132.2 (12.3)	99.1 (0.5)	108.0 (5.6)	95.7 (3.4)	37.8 (2.4)	495.8 (34.2)	26.8 (1.6)
	30 min	130.5 (14.1)	95.9 (1.4)	99.0 (4.7)	96.0 (5.8)	37.5 (2.5)	499.1 (35.2)	26.7 (1.6)
PS ₈	Pretreatment	134.7 (13.1)	97.0 (0.9)	100.0	100.0	38.3 (2.8)	513.2 (40.0)	26.6 (1.8)
	Posttreatment	125.3 (11.4)*	98.5 (0.5)	95.8 (6.1)	97.5 (4.1)	35.9 (2.6)*	477.2 (35.7)*	30.2 (2.1)*
	10 min	131.4 (13.2)	97.8 (0.5)	110.6 (6.8)	97.3 (4.5)	37.6 (2.6)	504.8 (38.1)	27.7 (1.9)
	20 min	133.4 (12.5)	96.8 (1.2)	117.3 (6.6)	103.7 (4.4)	37.7 (2.6)	507.2 (38.0)	27.0 (1.6)
	30 min	130.5 (12.7)	98.8 (0.4)	104.0 (6.7)	105.5 (6.3)	37.8 (2.6)	508.9 (38.7)	27.2 (1.6)

* indicates a significant ($P \leq 0.05$) difference from pre- to post-treatment.

and no main effects for condition ($P = 0.264$) or time ($P = 0.709$). %VA did not change from pre- to posttreatment for any condition.

EMG amplitude. There was no significant three-way interaction (condition \times time \times muscle, $P = 0.266$) and no significant two-way interactions for condition \times time ($P = 0.216$), condition \times muscle ($P = 0.497$), or time \times muscle ($P = 0.105$). In addition, there were no significant main effects for condition ($P = 0.484$), time ($P = 0.907$), or muscle ($P = 0.051$). Therefore, EMG amplitude did not change from pre- to posttreatment for any condition.

DISCUSSION

There has been a growing concern that stretching before exercise or athletic events may cause transient but detrimental decreases in muscle strength (31,34) in both

untrained and trained individuals. According to a citation index (ISI Web of Science®; <http://portal.isiknowledge.com>), Fowles et al. (11) is the most commonly cited study (compared with the others cited in this article) examining the acute effects of stretching on muscle strength. These authors (11) demonstrated that 30 min of PS immediately decreased plantarflexor strength by 28%, whereas 9% strength decrements were still present 1 h after the stretching. However, the authors (11) admittedly used an exaggerated stretching duration (30 min of time-under-stretch) and suggested that "...further testing with a stretching protocol more similar to that regularly performed in the athletic context should be evaluated under the controlled conditions of this study" (p. 1187). To address this question, more recent studies have examined shorter stretching durations for the plantarflexors and have reported 10% decreases after 20 min of stretching (14), 7% decreases

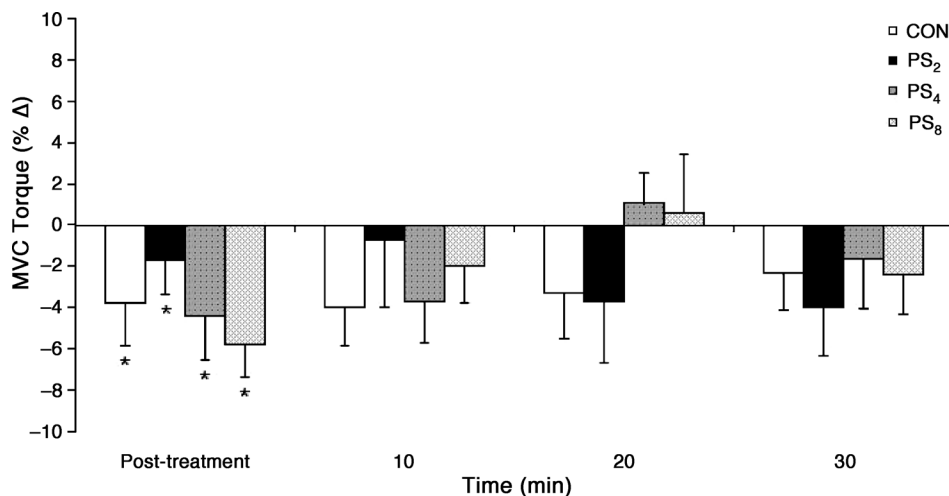


FIGURE 1—Percent change in isometric PT as a result of the CON, the 2-, the 4-, and the 8-min PS treatments. *Significant decrease from pre- to posttreatment ($P < 0.05$). Values represent the percent changes (means \pm SEM).

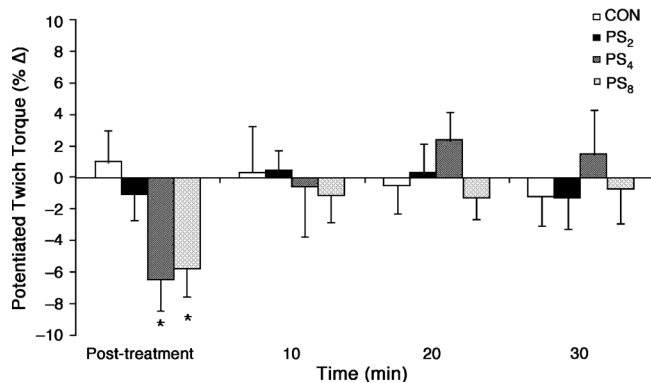


FIGURE 2—Percent change in potentiated twitch torque as a result of the CON, the 2-, the 4-, and the 8-min PS treatments. *Significant decrease from pre- to posttreatment ($P < 0.05$). Values represent the percent changes (means \pm SEM).

after 10 min of stretching (36), and 0.3–3.6% nonsignificant decreases after a 5-min warm-up and 1–4 min of stretching (1,40). The results of the present study extended these previous findings using the controlled conditions of Fowles et al. (11) and indicated that more practical stretching durations of 2, 4, and 8 min (PS₂, PS₄, and PS₈, respectively) did not alter plantarflexor strength when compared with the CON condition (Fig. 1). Thus, our findings suggested that merely sitting dormant for 15 min resulted in the same decrease in plantarflexor strength as any of the practical stretching durations for moderately active, recreationally trained individuals.

To illustrate how the results of the present study have extended our understanding of the stretching-induced force deficit, Figure 5 shows the percent changes (%Δ) in plantarflexor strength in response to various durations of stretching. The results from several studies are presented in Figure 5—all of which used similar research methods and relatively untrained subjects. Together, these results (Fig. 5) suggested that the stretching-induced force deficit may be governed by a dose–response relationship. Specifically, the Fowles et al. (11), the Herda et al. (14), and the Weir et al. (36) studies demonstrated significant decreases in strength after 30, 20, and 10 min of stretching, respectively. Most importantly, however, these decreases followed a curvilinear pattern (Fig. 5) in which the 30-min stretching protocol of Fowles et al. (11) caused the greatest decrease in strength (28% decrease), followed by the 20-min protocol of Herda et al. (14) (10% decrease) and the 10-min protocol of Weir et al. (36) (7% decrease). In the present study, however, there was a nonsignificant 6% decrease in plantarflexor strength after 8 min of stretching (Fig. 5). Thus, these findings, in conjunction with those from previous investigations (11,14,36), suggested that there may be a threshold stretching duration between 8 and 10 min that may distinguish between significant and nonsignificant decreases in plantarflexor strength in untrained individuals. However, future studies are necessary to test this hypothesis and to examine whether this dose–

response relationship is muscle specific and/or specific to the mode of stretching (e.g., static, ballistic, dynamic, or proprioceptive neuromuscular facilitation stretching). In addition, it is unclear whether the same dose–response relationship would be present in well-trained athletes.

Two general hypotheses have been suggested to explain the stretching-induced force deficit: a) neural factors that involve decreases in muscle activation (3,5,11) and b) mechanical factors that involve alterations of the length–tension relationship, force–velocity relationship, and/or the viscoelastic properties of the muscle (4,13). For example, Fowles et al. (11) reported that most of the force loss within the initial 15-min poststretching period was due to an impaired ability to activate all available motor units. Other studies have reported similar acute decreases in muscle activation using surface EMG and/or the twitch interpolation technique in the plantarflexors (11,13) and leg extensors (3,5). Fowles et al. (11) suggested that the temporary inability to fully activate the stretched muscle may be related to a persistent Golgi tendon organ reflex, mechanoreceptor and nociceptor pain feedback, and/or fatigue-related mechanisms. However, Herda et al. (14) recently reported distinct similarities between the stretching- and the vibration-induced decreases in muscle activation, which tentatively suggested that stretching, like vibration, may cause a temporary inhibition of gamma loop function. It is thought that prolonged vibration decreases the ascending feedback from the muscle spindles, which result in a disfacilitation in Type II motor unit recruitment (22). This hypothesis may also help to explain the apparent central nervous system inhibition demonstrated by stretching-induced decreases in muscle torque production and surface EMG amplitude in the stretched and unstretched, contralateral limbs (5).

It should be noted, however, that not all studies have reported stretching-induced decreases in muscle activation. For example, Weir et al. (36) found no changes in %VA in the plantarflexors when examining the acute effects of only 10 min of stretching. Longer durations of stretching have

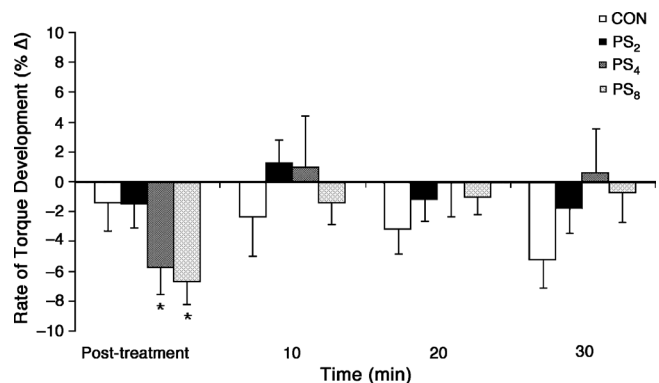


FIGURE 3—Percent change in RTD as a result of the CON, the 2-, the 4-, and the 8-min PS treatments. *Significant decrease from pre- to posttreatment ($P < 0.05$). Values represent the percent changes (means \pm SEM).

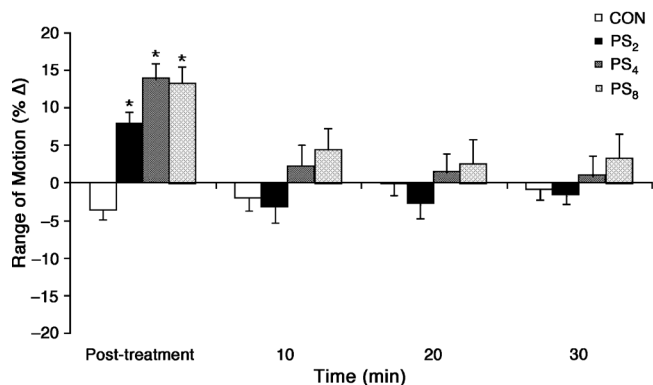


FIGURE 4—Percent change in PROM as a result of the CON, the 2-, the 4-, and the 8-min PS treatments. *Significant decrease from pre- to posttreatment ($P < 0.05$). Values represent the percent changes (means \pm SEM).

elicited decreases in %VA in the plantarflexors (11,14) and the leg extensors (3). The results of the present study were consistent with the findings of Weir et al. (36) and suggested that shorter durations of stretching may not diminish muscle activation in the plantarflexors. In contrast, larger muscle groups such as the leg extensors (4,5,25) have demonstrated reductions in muscle activation after 8 min of stretching. Therefore, it is possible that the stretching-induced decreases in muscle activation may be muscle specific, such that the suboptimal activation of the larger, proximal muscles may be amplified after stretching, whereas the near fully activated, distal muscles (19) require longer durations of stretch to diminish muscle activation. Future studies are needed, however, to test this hypothesis. It should also be noted that most of the studies that have examined the underlying mechanisms responsible for the stretching-induced force deficit have used nonathletes. It is possible that the neuromuscular adaptations that generally accompany the training programs of athletes may affect how athletes respond to stretching. Therefore, more studies are also needed to examine athletes under similar controlled conditions.

The other primary hypothesis that is often used to explain the stretching-induced force deficit involves the potential mechanical alterations in musculotendinous stiffness, which may decrease the force producing capabilities of muscle. In theory, decreases in musculotendinous stiffness after stretching may increase the resting length of the sarcomeres, which in turn may alter the length-tension relationship (4,11,13). Previous studies have suggested that stretching-induced decreases in evoked twitch properties (3,11,30) may reflect the muscle's inability to generate force due to a more compliant musculotendinous unit. For example, Rosenbaum and Henning (30) found a reduction in Achilles tendon tap force and rate of force development after 3 min of static stretching. Similarly, Fowles et al. (11) reported decreases in PTT at three joint angles (0° , 10° , and 20° of dorsiflexion), which lasted up to 1 h poststretching. The results of the present study also demonstrated dose-dependent decreases in

twitch properties (PTT and RTD) for the PS₄ and PS₈ stretching conditions. These findings were consistent with previously reported decreases in musculotendinous stiffness after 7.5 min of stretching (24) but no changes in stiffness after shorter durations of stretching (~ 2 min) (23,26). Therefore, our findings, in conjunction with previous studies on musculotendinous stiffness (3,11), supported the hypothesis that there is an inverse relationship between muscle twitch properties and musculotendinous compliance. However, it should be noted that the altered twitch properties after the PS₄ and the PS₈ stretching conditions were not sufficient to cause significant decreases in voluntary PT or %VA when compared with the CON condition. It is possible that the decreases in PT after practical stretch durations (such as PS₂, PS₄, and PS₈) may only be detectable at shorter muscle lengths (13). Future studies should examine the muscle twitch properties and voluntary PT at various joint angles after practical stretching conditions such as those of the present study.

Similar to the stretching-related mechanical alterations in the musculotendinous unit are the improvements in ROM that are often observed after stretching. Indeed, a previous study reported temporary (up to 3 min) increases in joint ROM after shorter durations (~ 2 min) of static stretching (6). The results of the present study were consistent with these previous findings (6) and indicated that passive dorsiflexion ROM increased after the PS₂ (8% increase), the PS₄ (14% increase), and the PS₈ (13% increase) conditions (Fig. 4). Magnusson et al. (23) have suggested that increases in ROM after shorter stretching durations (1.3–2.25 min) may be due to increases in stretch tolerance, whereas ROM increases after longer durations of stretching (7.5 min) may also be due to decreases in musculotendinous stiffness (24). Interestingly, our findings indicated that 2, 4, and 8 min of PS resulted in similar increases in ROM immediately after the stretching; however, ROM returned to baseline by 10 min poststretching (Fig. 4). Therefore,

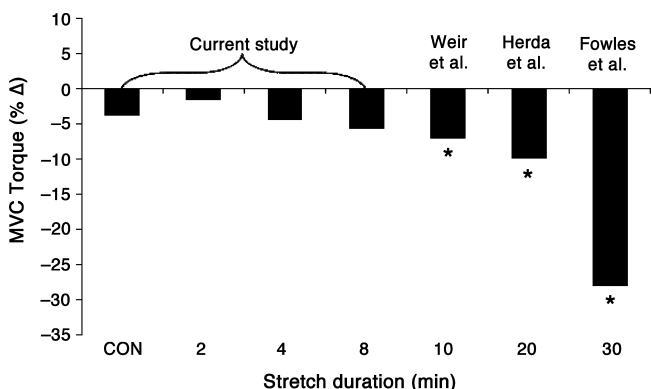


FIGURE 5—A hypothetical graph demonstrating the dose-dependent relationship between the duration of passive stretch and the decreases in isometric plantarflexion strength. Values include the results of the current study and of Weir et al. (36), Herda et al. (13), and Fowles et al. (11). *Reported significant decrease from pre- to posttreatment ($P < 0.05$).

whatever the mechanisms are underlying the stretching-induced improvements in ROM (i.e., stretch tolerance and/or musculotendinous stiffness), they appear to be short-lived. It is possible that the transient improvements in ROM were due to the muscles' inherent viscoelastic nature, suggesting that "...muscle has a strong tendency to return to its resting or genetically and biomechanically determined length" (p. 1187) (11).

In summary, our results indicated that there were no decreases in voluntary PT after the PS₂, the PS₄, and the PS₈ stretching conditions when compared with the CON condition. In addition, no stretching-induced changes in %VA or surface EMG amplitude were observed in the present study, which suggested that 2, 4, or 8 min of PS of the plantarflexors may not alter muscle activation. However, there were temporary dose-dependent decreases in potentiated twitch properties (PTT and RTD) after the PS₄ and the PS₈ conditions, which suggested that the altered mechanical properties of muscle contraction may have been due to stretching-induced increases in musculotendinous compliance, yet these changes were not sufficient to alter voluntary PT. In addition, the present findings were unique in that all stretching durations (PS₂, PS₄, and PS₈) resulted in similar increases in dorsiflexion ROM. However, the improvements in ROM were short-lived and had diminished by 10 min poststretching. Therefore, from a practical standpoint, our findings indicated that PS durations of 2,

4, and 8 min for the plantarflexors did not alter plantarflexion strength but did improve ROM (albeit temporarily). Because many preexercise or precompetition stretching routines occur well before 10 min before the start of exercise or competition, the adverse effects of practical stretching durations on plantarflexor strength may be minimal. Nevertheless, positive chronic adaptations to regular stretching regimens have been reported (21,31,34); therefore, it may be reasonable to include stretching exercises after cardiovascular or resistance training exercise sessions rather than before. Furthermore, the current findings, in conjunction with similar previous studies (11,14,36), implied a dose-response relationship (Fig. 5) between the duration of stretch and the magnitude of the stretch-induced force deficit in the plantarflexor muscles. Specifically, there may be a threshold between 8 and 10 min of stretching, such that durations of stretching above the threshold may significantly decrease plantarflexor strength (Fig. 5). In addition, it should be noted that the subjects in this study were only moderately active and recreationally trained; they were not athletes. Therefore, future studies are needed using the same controlled conditions to determine whether the dose-response relationship for the stretching-induced force deficit is similar for elite athletes and whether it is muscle or joint angle specific.

The results of the present study do not constitute endorsement by ACSM.

REFERENCES

- Alpkaya U, Kocaja D. The effects of acute static stretching on reaction time and force. *J Sports Med Phys Fitness*. 2007;47(2):147-50.
- Behm DG, Bambury A, Cahill F, Power K. Effect of acute static stretching on force, balance, reaction time, and movement time. *Med Sci Sports Exerc*. 2004;36(8):1397-402.
- Behm DG, Button DC, Butt JC. Factors affecting force loss with prolonged stretching. *Can J Appl Physiol*. 2001;26(3):261-72.
- Cramer JT, Beck TW, Housh TJ, et al. Acute effects of static stretching on characteristics of the isokinetic angle-torque relationship, surface electromyography, and mechanomyography. *J Sports Sci*. 2007;25(6):687-98.
- Cramer JT, Housh TJ, Weir JP, Johnson GO, Coburn JW, Beck TW. The acute effects of static stretching on peak torque, mean power output, electromyography, and mechanomyography. *Eur J Appl Physiol*. 2005;93(5-6):530-9.
- Depino GM, Webright WG, Arnold BL. Duration of maintained hamstring flexibility after cessation of an acute static stretching protocol. *J Athl Train*. 2000;35(1):56-9.
- Desbrosses K, Babault N, Scaglioni G, Meyer JP, Pousson M. Neural activation after maximal isometric contractions at different muscle lengths. *Med Sci Sports Exerc*. 2006;38(5):937-44.
- Egan AD, Cramer JT, Massey LL, Marek SM. Acute effects of static stretching on peak torque and mean power output in National Collegiate Athletic Association Division I women's basketball players. *J Strength Cond Res*. 2006;20(4):778-82.
- Ekstrand J, Gillquist J, Moller M, Oberg B, Liljedahl SO. Incidence of soccer injuries and their relation to training and team success. *Am J Sports Med*. 1983;11(2):63-7.
- Evetovich TK, Nauman NJ, Conley DS, Todd JB. Effect of static stretching of the biceps brachii on torque, electromyography, and mechanomyography during concentric isokinetic muscle actions. *J Strength Cond Res*. 2003;17(3):484-8.
- Fowles JR, Sale DG, MacDougall JD. Reduced strength after passive stretch of the human plantarflexors. *J Appl Physiol*. 2000;89(3):1179-88.
- Gravetter FJ, Wallnau LB. *Statistics for the Behavioral Sciences*. 6th ed. Belmont: Thomson Wadsworth; 2004:263-9.
- Herda TJ, Cramer JT, Ryan ED, McHugh MP, Stout JR. Acute effects of static versus dynamic stretching on isometric peak torque, electromyography, and mechanomyography of the biceps femoris muscle. *J Strength Cond Res*. 2008;22(3):809-17.
- Herda TJ, Ryan ED, Smith AE, et al. Acute effects of passive stretching versus vibration on the neuromuscular function of the plantar flexors. *Scand J Med Sci Sports*. (in press).
- Hermens HJ, Freriks B, Merletti R, et al. European Recommendations for Surface Electromyography: Results of the SENIAM Project: Roessingh Research and Development; 1999. p. 52.
- Holt BW, Lambourne K. The impact of different warm-up protocols on vertical jump performance in male collegiate athletes. *J Strength Cond Res*. 2008;22(1):226-9.
- Kaufman KR, Brodine SK, Shaffer RA, Johnson CW, Cullison TR. The effect of foot structure and range of motion on musculoskeletal overuse injuries. *Am J Sports Med*. 1999;27(5):585-93.
- Keenan KG, Farina D, Maluf KS, Merletti R, Enoka RM. Influence of amplitude cancellation on the simulated surface electromyogram. *J Appl Physiol*. 2005;98(1):120-31.
- Kent-Braun JA, Le Blanc R. Quantitation of central activation failure during maximal voluntary contractions in humans. *Muscle Nerve*. 1996;19(7):861-9.
- Kokkonen J, Nelson AG, Cornwell A. Acute muscle stretching

- inhibits maximal strength performance. *Res Q Exerc Sport*. 1998;69(4):411–5.
21. Kokkonen J, Nelson AG, Eldredge C, Winchester JB. Chronic static stretching improves exercise performance. *Med Sci Sports Exerc*. 2007;39(10):1825–31.
 22. Kouzaki M, Shinohara M, Fukunaga T. Decrease in maximal voluntary contraction by tonic vibration applied to a single synergist muscle in humans. *J Appl Physiol*. 2000;89(4):1420–4.
 23. Magnusson SP, Aagaard P, Nielson JJ. Passive energy return after repeated stretches of the hamstring muscle-tendon unit. *Med Sci Sports Exerc*. 2000;32(6):1160–4.
 24. Magnusson SP, Simonsen EB, Aagaard P, Kjaer M. Biomechanical responses to repeated stretches in human hamstring muscle in vivo. *Am J Sports Med*. 1996;24(5):622–8.
 25. Marek SM, Cramer JT, Fincher AL, et al. Acute effects of static and proprioceptive neuromuscular facilitation stretching on muscle strength and power output. *J Athl Train*. 2005;40(2):94–103.
 26. Muir IW, Chesworth BM, Vandervoort AA. Effect of a static calf-stretching exercise on the resistive torque during passive ankle dorsiflexion in healthy subjects. *J Orthop Sports Phys Ther*. 1999;29(2):106–13; discussion 114–105.
 27. Nelson AG, Driscoll NM, Landin DK, Young MA, Schexnayder IC. Acute effects of passive muscle stretching on sprint performance. *J Sports Sci*. 2005;23(5):449–54.
 28. Nelson AG, Kokkonen J, Arnall DA. Acute muscle stretching inhibits muscle strength endurance performance. *J Strength Cond Res*. 2005;19(2):338–43.
 29. Riddle DL, Pulisic M, Pidcoe P, Johnson RE. Risk factors for plantar fasciitis: a matched case-control study. *J Bone Joint Surg Am*. 2003;85-A(5):872–7.
 30. Rosenbaum D, Hennig EM. The influence of stretching and warm-up exercises on Achilles tendon reflex activity. *J Sports Sci*. 1995;13(6):481–90.
 31. Rubini EC, Costa AL, Gomes PS. The effects of stretching on strength performance. *Sports Med*. 2007;37(3):213–24.
 32. Shellock FG, Prentice WE. Warming-up and stretching for improved physical performance and prevention of sports-related injuries. *Sports Med*. 1985;2(4):267–78.
 33. Shield A, Zhou S. Assessing voluntary muscle activation with the twitch interpolation technique. *Sports Med*. 2004;34(4):253–67.
 34. Shrier I. Does stretching improve performance? A systematic and critical review of the literature. *Clin J Sport Med*. 2004;14(5):267–73.
 35. Unick J, Kieffer HS, Cheesman W, Feeney A. The acute effects of static and ballistic stretching on vertical jump performance in trained women. *J Strength Cond Res*. 2005;19(1):206–12.
 36. Weir DE, Tingley J, Elder GC. Acute passive stretching alters the mechanical properties of human plantar flexors and the optimal angle for maximal voluntary contraction. *Eur J Appl Physiol*. 2005;93(5–6):614–23.
 37. Weir JP. Quantifying test-retest reliability using the intraclass correlation coefficient and the SEM. *J Strength Cond Res*. 2005;19(1):231–40.
 38. Winchester JB, Nelson AG, Landin DK, Young MA, Schexnayder IC. Static stretching impairs sprint performance in collegiate track and field athletes. *J Strength Cond Res*. 2008;22(1):13–18.
 39. Yeh CY, Chen JJ, Tsai KH. Quantifying the effectiveness of the sustained muscle stretching treatments in stroke patients with ankle hypertonia. *J Electromyogr Kinesiol*. 2007;17(4):453–61.
 40. Young W, Elias G, Power J. Effects of static stretching volume and intensity on plantar flexor explosive force production and range of motion. *J Sports Med Phys Fitness*. 2006;46(3):403–11.