Acute Effects of Static versus Dynamic Stretching on Isometric Peak Torque, Electromyography, and Mechanomyography of the Biceps Femoris Muscle

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Abstract

The purpose of this study was to examine the acute effects of static versus dynamic stretching on peak torque (PT) and electromyographic (EMG), and mechanomyographic (MMG) amplitude of the biceps femoris muscle (BF) during isometric maximal voluntary contractions of the leg flexors at four different knee joint angles. Fourteen men ((mean ± SD) age, 25 ± 4 years) performed two isometric leg flexion maximal voluntary contractions at knee joint angles of 41°, 61°, 81°, and 101° below full leg extension. EMG (μV) and MMG ($m \cdot s^{-2}$) signals were recorded from the BF muscle while PT values (Nm) were sampled from an isokinetic dynamometer. The right hamstrings were stretched with either static (stretching time, 9.2 ± 0.4 minutes) or dynamic (9.1 ± 0.3 minutes) stretching exercises. Four repetitions of three static stretching exercises were held for 30 seconds each, whereas four sets of three dynamic stretching exercises were performed (12–15 repetitions) with each set lasting 30 seconds. PT decreased after the static stretching at 81° ($p = 0.019$) and 101° ($p = 0.001$) but not at other angles. PT did not change ($p > 0.05$) after the dynamic stretching. EMG amplitude remained unchanged after the static stretching ($p > 0.05$) but increased after the dynamic stretching at 101° ($p < 0.001$) and 81° ($p < 0.001$). MMG amplitude increased in response to the static stretching at 101° ($p = 0.003$), whereas the dynamic stretching increased MMG amplitude at all joint angles ($p \leq 0.05$). These results suggested that the decreases in strength after the static stretching may have been the result of mechanical rather than neural mechanisms for the BF muscle. Overall, an acute bout of dynamic stretching may be less detrimental to muscle strength than static stretching for the hamstrings.

Key Words stretching-induced force deficit, EMG, MMG, hamstrings

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Introduction

Stretching is commonly performed before exercise (1) and athletic events (6,23). Traditionally, it is believed that increasing flexibility (increasing joint range of motion) will promote better performance (55) and reduce the risk of injury during strenuous exercise (56). Several studies have used muscle stretching techniques to examine various aspects of muscle function including passive force production (33–35,56), stress-relaxation (41,59,60), neuromuscular reflex patterns (22,25,62), factors contributing to muscle damage (2,31), and the mechanisms of increase in musculotendinous compliance (33,60). However, recent studies have reported that stretching before exercise or performance events actually decreases isometric (3,9,19,45) and dynamic muscle strength (12,14,29,37,45,47,48). As a result, this phenomenon has been termed the stretching-induced force deficit (19). Two primary hypotheses have been proposed to explain the stretching-induced force deficit (3,9,12,16,19,27,29,44,66): (a) mechanical factors such as decreases in muscle stiffness may affect the length-tension relationship and (b) neural factors such as altered motor control strategies and/or reflex sensitivity.

Mechanomyography (MMG) and electromyography (EMG) can provide unique information about the mechanical properties and neural activation strategies of skeletal muscles. For example, the MMG signal records and quantifies the low-frequency lateral oscillations of active skeletal muscle fibers and provides a noninvasive method to examine muscle function (5,50,57). The lateral oscillations produced by contracting muscles may reflect the mechanical counterpart of the muscle activation as measured by surface EMG (20).

It has been suggested that MMG amplitude is inversely proportional to the active stiffness of a muscle (13,15,50); therefore, stretching-induced decreases in muscle stiffness might be detected by increases MMG amplitude (16). Conversely, surface EMG reflects the algebraic sum of electrical muscle action potentials that pass within the recording areas of the EMG electrodes. Therefore, EMG amplitude quantifies muscle activation, which can be altered by the number of motor units recruited and the firing rates of the activated motor units (7,50–52). It has also been hypothesized (3,9,12,19) that static stretching reduces muscle activation, perhaps through central nervous system inhibitory mechanisms. Thus, EMG amplitude may be able to detect stretching-induced alterations in muscle activation (11,12,37). Together, MMG and EMG amplitude may be useful to test the hypotheses regarding the mechanical and neural factors, respectively, underlying the stretching-induced force deficit.

The angle-torque relationship during maximal isometric contractions (MVCs) is an additional approach to examining the mechanical factors that may be responsible for the stretching-induced force deficit. During isometric contractions, muscle fibers shorten and the tendons and aponeuroses lengthen (21). Theoretically, a stretching-induced decrease in passive stiffness of the tendon and aponeurosis will allow greater muscle fiber shortening at a given muscle length. This would affect the length-tension relationship such that, after stretching, torque would be decreased at short muscle lengths and increased at long muscle lengths. Therefore, the stretching-induced force deficit may only be apparent at muscle lengths shorter than the length for optimal force production.

Decreases in the force-producing capabilities of isolated muscle actions have been demonstrated as a result of static (9,12,14,16,19,27,37,47,53,64), proprioceptive neuromuscular facilitation (PNF) (37), and ballistic (48) stretching. Isolated muscle actions, such as isometric or isokinetic leg extensions (12,14,37,47), forearm flexions (16), or plantarflexions (19), provide well-controlled environments to examine the mechanisms underlying the stretching-induced force deficit. As a result of these studies, recommendations have been made to avoid static, PNF, and ballistic stretching before athletic performance activities that require high levels of force production. In contrast, recent investigations have reported improvements in performance after an acute bout of dynamic stretching when power output is measured during whole-body dynamic tasks (17,18,32,42) and explosive, multi-joint lower-body exercises (65). However, no previous studies have compared the acute effects of static and dynamic stretching in isolated muscle performance, which was the original model used to demonstrate the stretching-induced force deficit. The hamstring muscle was chosen in the present study because it is commonly stretched for fear of hamstring strains and because most previous studies have examined the quadriceps femoris (9,12,14,37,47), gastrocnemius (19,64), or biceps brachii (16). Therefore, the purpose of this study was to examine the acute effects of static versus dynamic stretching on peak torque (PT) and EMG and MMG amplitude of the biceps femoris muscle (BF) during MVCs of the leg flexors at four different knee joint angles.

Methods

Experimental Approach to the Problem

This study was designed to examine whether dynamic stretching elicits the same acute inhibitory influences on muscle force production as static stretching (9,19,53,64) during isometric leg flexion muscle actions at four different joint angles. EMG and MMG amplitude were recorded from the biceps femoris muscle to test the hypothesis that stretching reduces muscle activation (EMG) and muscle stiffness (MMG) (11,14,16,37). Multiple joint angles were examined to test the hypothesis that the stretching-induced force deficit is caused by alterations in the length-tension relationship (11,39,47,48).

Subjects

Fourteen healthy men ((mean ± SD) age, 25 ± 4 years; height, 177 ± 6 cm; weight, 78 ± 9 kg) volunteered for this investigation. None of the participants reported any current or ongoing neuromuscular diseases or musculoskeletal injuries specific to the ankle, knee, or hip joints. Each participant completed a pre-exercise health questionnaire and signed a written informed consent document. Of the 14 participants, 10 reported engaging in 1–8 $h \cdot wk^{-1}$ of aerobic exercise, 10 reported 1–7 $h \cdot wk^{-1}$ of resistance exercise, and 12 reported 1–4 $h \cdot wk^{-1}$ of recreational sports. None of the participants were competitive athletes; however, as a result of their reported levels of aerobic exercise, resistance training, and recreational sports, these individuals would be classified as normal, moderately active, recreationally trained participants. This study was approved by the University's institutional review board for human subjects research.

Isometric Strength Assessments

Before (pre) and after (post) the stretching exercises, isometric PT for the right hamstring muscles was measured using a Biodex System 3 isokinetic dynamometer (Biodex Medical Systems, Inc., Shirley, NY). PT was measured at four randomly ordered knee joint angles of 41°, 61°, 81°, and 101° below full extension. The participants were seated with restraining straps over the pelvis, trunk, and contralateral thigh, and the lateral condyle of the femur 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.). Two 4-second isometric MVCs of the leg flexors were performed at each joint angle with 30 seconds of rest between each MVC and 30 seconds of rest between joint angles. PT was determined as the higher of the two MVC trials at each joint angle. All isometric PT assessments were performed with a 60° angle between the thigh and torso (Figure 1). Isometric PT assessments began 4.2 ± 0.6 minutes after the completion of stretching treatments and ended 9.1 ± 0.1 minutes after the beginning of the stretching treatment.

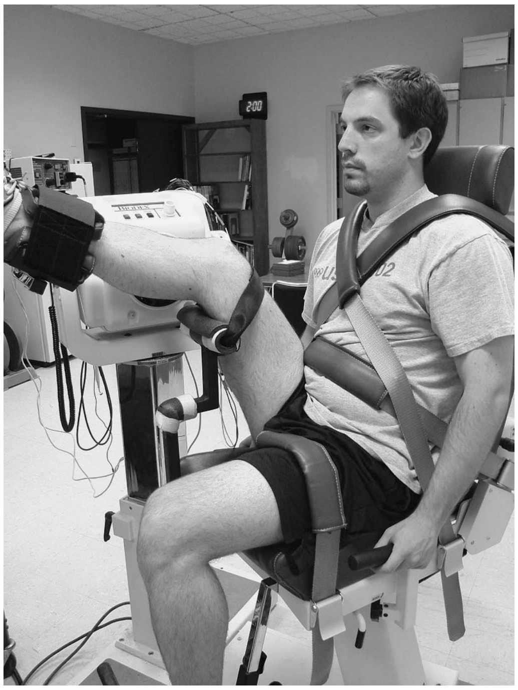

Figure 1. An example of subject positioning for the isometric strength assessments.

Static and Dynamic Stretching Exercises

Immediately after the pre-stretching isometric strength assessments, each subject performed three static or dynamic stretching exercises designed to stretch the right hamstrings. For the static stretching, four repetitions of each stretching exercise were held for 30 seconds at the point of discomfort, but not pain, as acknowledged by the subject. For the dynamic stretching, four sets of each stretching exercise were performed by repeating the stretch continuously in a slow and controlled manner for 30 seconds, which resulted in 12–15 repetitions per set. Between each static stretching repetition or dynamic stretching set, there was a 15-second rest period. The repetitions, sets, and rest periods where chosen based on previous studies (17,30) to equalize the volume of static and dynamic stretching. Consequently, the average total stretching time was 9.2 ± 0.4 minutes for the static stretching and 9.1 ± 0.3 minutes for the dynamic stretching.

For the static stretching, each subject performed one unassisted stretching exercise followed by two assisted stretching exercises. For the unassisted stretching exercise (Figure 2a), the subject positioned the heel of his right foot 5–10 cm lateral to the toes of his left foot, flexed the right leg to maintain a 5–10° angle at the knee joint, then flexed the torso downward while reaching for the right toes with both hands until the right hamstrings were stretched. After the unassisted stretching exercise, the remaining static stretching exercises were performed with the assistance of the primary investigator.

The first assisted stretching exercise (Figure 2b) was performed with the subject seated on a padded mat. The right leg was fully extended, left leg flexed, and left thigh abducted and externally rotated so that the bottom of the left foot faced the medial aspect of the right thigh. In this position, the subject flexed the torso forward while reaching for the right toes with both hands, while the investigator gently pushed the torso forward to complete the stretch. The final assisted static stretching exercise (Figure 2c) began with the subject lying supine on a padded mat with the left thigh and leg extended. The investigator knelt over the left thigh while passively flexing the subject's right thigh at the hip by applying pressure to the posterior aspects of the right leg and ankle. The right leg was flexed slightly to maintain a 5–10° angle at the knee joint, and the right foot was dorsiflexed to maintain a 90° angle at the ankle.

On a separate day, each subject performed three dynamic stretching exercises based on the methods of a previous study (36). The first dynamic stretching exercise (Figure 2d) was performed with both arms abducted and forearms extended to be horizontal to the floor. The subject flexed the right thigh while maintaining an extended leg so that the right toes approached both hands. The right thigh was then extended back to the starting position. Once completed, the subject took a regular step forward with the left leg and then repeated the dynamic stretching movement of the right thigh in a forward linear direction. The second dynamic stretching exercise (Figure 2e) involved an exaggerated step forward with the right leg while flexing the trunk at the hip and waist until both hands approached the right foot. Because of some limited forward momentum, this motion was balanced by extension of the left thigh. Once completed, the subject returned to the start position and repeated the movement in a forward linear direction. The third and final dynamic stretching exercise (Figure 2f) was performed with the hands on the hips. The subject flexed the right thigh, extended the right leg, and then took an exaggerated step forward. Once completed, the subject brought the left leg to the starting position and then repeated the dynamic stretching exercise of the right leg in a forward linear direction.

Surface Electromyography

Bipolar surface EMG electrode arrangements (Ag-Ag Cl, Quinton Quick Prep; Quinton Instruments Co., Bothell, WA) were placed along the longitudinal axis of the right BF muscle. Electrodes were placed at 50% of the distance from the ischial tuberosity to the medial epicondyle of the tibia. The center-to-center interelectrode distance was approximately 4 cm, which was selected to accommodate placement of the MMG sensors between the active EMG electrodes (14). For all EMG measurements, the reference electrode was placed over the spinous process of the 7th cervical vertebrae. Interelectrode impedance for each muscle was kept below 2,000 Ω by carefully abrading the skin and swabbing with isopropyl alcohol. The EMG signals (recorded in microvolts)

Figure 2. Examples of the (a) unassisted static stretching exercise, (b) first assisted static stretching exercise, (c) second assisted static stretching exercise, (d) first dynamic stretching exercise, (e) second dynamic stretching exercise, and (f) third dynamic stretching exercise.

were differentially amplified with a bandwidth of 1–500 Hz, input impedance of 2 MΩ (differential), common mode rejection ratio of 110 dB, maximal input voltage of ±10 V, and gain of 1,000 (EMB100C; Biopac Systems Inc., Santa Barbara, CA).

Mechanomyography

The MMG signals were detected with active miniature accelerometers (EGAS-FS-10-/VO5; Measurement Specialties, Inc., Hampton, VA) that were pre-amplified with a gain of 200, frequency response of 0–200 Hz, sensitivity of 70 $mV/m{\cdot}s^{-2}$, and range of $\pm 98\ m{\cdot}s^{-2}$. The sensor was placed over the right BF muscle between the active EMG electrodes. The accelerometers were fixed to the skin with 3M double-sided foam tape.

Signal Processing

The EMG, MMG, and torque signals were recorded simultaneously with a Biopac data acquisition system (MP150WSW, Biopac Systems, Inc.) during each isometric MVC. The torque (Nm) signal from the Biodex dynamometer and the EMG (μV) and MMG ($m{\cdot}s^{-2}$) signals were stored on a personal computer (Dell Inspiron 8200; Dell, Inc., Round Rock, TX) and expressed as root mean square (rms) amplitude values by software (LabVIEW v 7.1; National Instruments, Austin, TX). The sampling frequency was 1,000 Hz for all signals. The EMG and MMG signals were bandpass filtered (zero phase fourth-order Butterworth filter) at 10–500 Hz and 5–100 Hz, respectively, while the torque signal was low-pass filtered with a 10-Hz cutoff (zero-phase fourth-order Butterworth filter). All rms amplitude calculations were performed on the filtered signals.

MVC torque (Nm) was determined as the highest 1-second average torque value that occurred during the middle of the 4-second MVC. The same 1-second epoch used to calculate MVC torque was also used to calculate EMG and MMG amplitude during the MVC trials. The EMG and MMG amplitude values were normalized (%max) to the highest amplitude values recorded during the pre-stretching isometric strength assessments separately for each subject.

Statistical Analysis

A three-way repeated measures ANOVA (mode (static vs. dynamic) × time (pre- vs. post-stretching) × angle (101° vs. 81° vs. 61° vs. 41°)) was used to analyze the PT data. Two separate three-way repeated measures ANOVAs (mode (static vs. dynamic) × time (pre- vs. post-stretching) × angle [101° vs. 81° vs. 61° vs. 41°]) were used to analyze the normalized EMG and MMG amplitude values. When appropriate, follow-up analyses included additional lower-order ANOVAs and paired-samples *t*-tests. SPSS software (version 12.0, Chicago, IL) was used for all statistical comparisons. The α level was set at $p \leq 0.05$ to determine statistical significance.

Reliability

Previous test-retest reliability from our laboratory for PT, EMG amplitude, and MMG amplitude during maximal, voluntary, isometric leg extensions indicated that, for 12 male and seven female subjects measured 48 hours apart, the intraclass correlation coefficients (R) ranged from 0.92 to 0.93, 0.70 to 0.94, and 0.69 to 0.85, respectively, with no significant ($p > 0.05$) differences between mean values for test vs. retest.

Results

Peak Torque

For PT, there was a significant three-way interaction (mode × time × angle; $p = 0.035$). PT decreased from pre- to post-stretching at 81° ($p = 0.019$) and 101° ($p = 0.001$) for the static stretching. In addition, the PT values at 41° and 101° were lower ($p \leq 0.05$) than PT at 81° and 61° for both the static and dynamic stretching trials. No other differences were observed from pre- to post-stretching for either the static or dynamic stretching conditions (Figure 3).

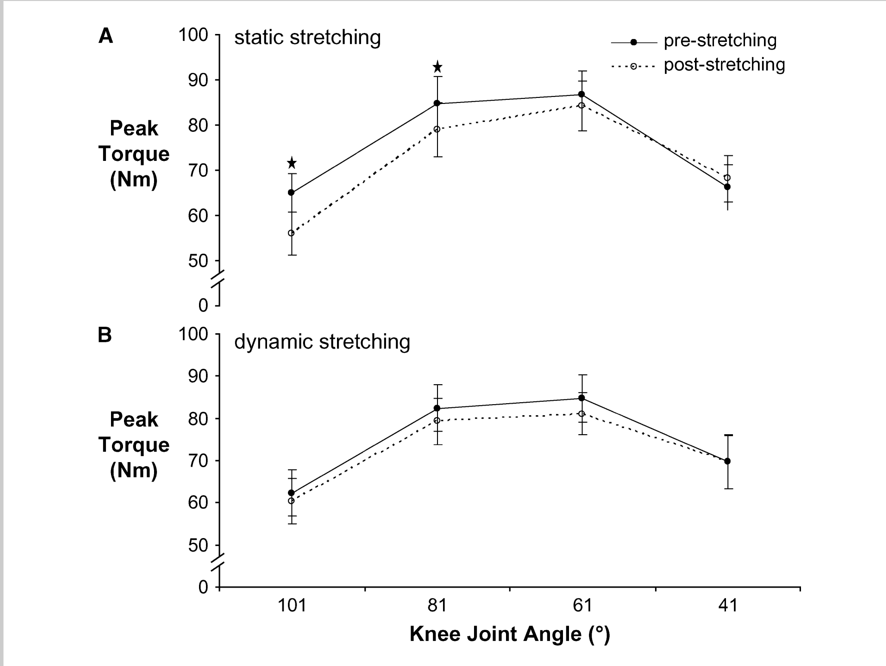

Figure 3. Isometric peak torque (Nm) plotted as a function of knee joint angle (°) during the pre- (*solid line*) and post-stretching (*dashed line*) assessments for the (a) static and (b) dynamic stretching conditions. ★Decreases from pre- to post-stretching ($p \leq 0.05$). Values are means ± SEM.

EMG Amplitude

The analyses indicated a three-way interaction (mode × time × angle; $p = 0.018$). Normalized EMG amplitude increased from pre- to post-stretching at 101° ($p < 0.001$) and 81° ($p < 0.001$) for the dynamic stretching condition; however, no other differences were observed from pre- to post-stretching for either the static or dynamic stretching (Figure 4).

MMG Amplitude

The analyses indicated no significant three-way interaction (mode × time × angle; $p = 0.994$) and no two-way interaction for mode × angle ($p = 0.316$), but there were two-way interactions for time × angle ($p = 0.006$) and mode × time ($p = 0.008$). Normalized MMG amplitude increased from pre- to post-stretching at 101° ($p = 0.003$) for the static stretching condition. MMG amplitude also increased from pre- to post-stretching at 101° ($p = 0.007$), 81° ($p = 0.004$), 61° ($p = 0.003$), and 41° ($p < 0.001$) for the dynamic stretching condition. In addition, MMG amplitude was greater at 101° than at 61° ($p = 0.002$) and 41° ($p = 0.001$) during the post-static stretching assessments, 81° and 61° were greater than 41°

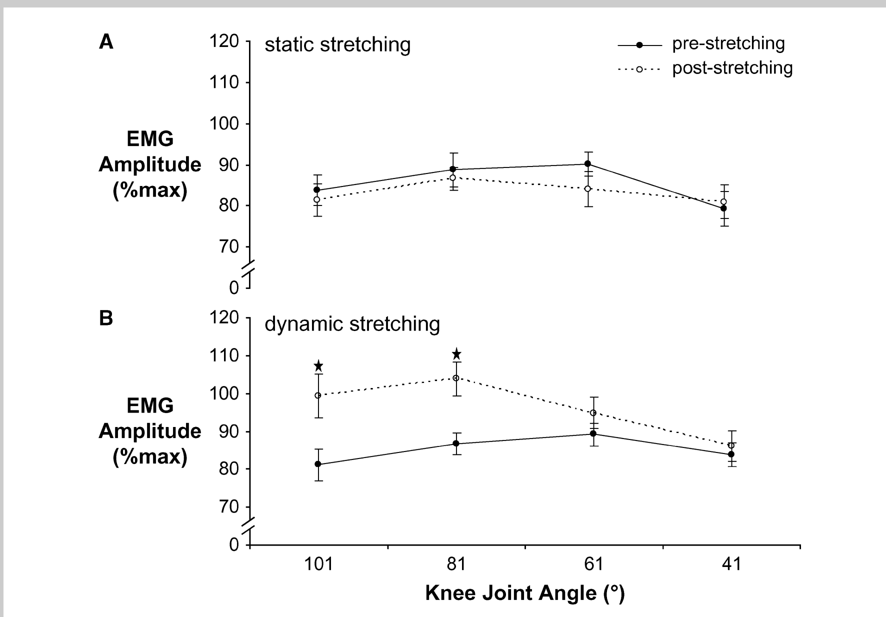

Figure 4. Normalized mean electromyographic amplitude (%max) plotted as a function of knee joint angle (°) during the pre- (*solid line*) and post-stretching (*dashed line*) assessments for the (a) static and (b) dynamic stretching conditions. ★Decreases from pre- to post-stretching ($p \leq 0.05$). Values are means ± SEM.

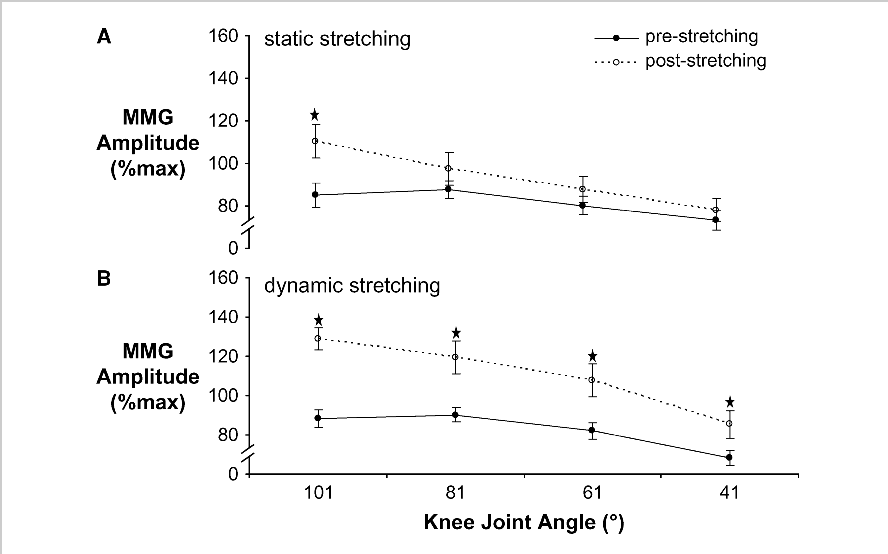

Figure 5. Normalized mean mechanomyographic amplitude (%max) plotted as a function of knee joint angle (°) during the pre- (*solid line*) and post-stretching (*dashed line*) assessments for the (a) static and (b) dynamic stretching conditions. ★Decreases from pre- to post-stretching ($p \leq 0.05$). Values are means ± SEM.

($p = 0.027$ and 0.001, respectively) during the pre-dynamic stretching assessments, and 101° and 81° were greater than 61° ($p = 0.001$ and 0.026, respectively) during the post-dynamic stretching assessments (Figure 5).

Discussion

The results of the present study indicate that the static stretching decreased isometric PT of the hamstrings muscles at knee joint angles of 101° (15.94%) and 81° (7.2%), but there were no changes in strength as a result of the dynamic stretching (Figure 3). These results are consistent with previous studies (9,19,45) that have reported acute decreases in isometric muscle strength after a bout of static stretching, which has since been termed the stretching-induced force deficit. To our knowledge, however, this is the first study to examine the acute effects of dynamic stretching on isolated muscle strength. The stretching-induced force deficit has been observed after ballistic (48), proprioceptive neuromuscular facilitation (PNF) (10,37), and static stretching (3,9,12, 14,16,19,29,45,47); however, based on the findings of the present study, dynamic stretching may not have an adverse affect on the isometric strength of the leg flexors.

Previous studies have reported acute increases in 20-m sprint performance (18), vertical jump height (17,32), and shuttle run performance (17,43) as a result of dynamic stretching. Static stretching, however, has been shown to either decrease (12,14,16,18,32,37,46,47,49,63,66) or have no affect on (33,42,53,61,65) the performance of similar tasks, such as sprinting (18,32,46), jumping (53,63,66), and agility drills (17,32,42). Yamaguchi et al. (65) reported no changes in leg extension power after static stretching; however, there was an increase in power output after the dynamic stretching. Similarly, Fletcher et al. (18) demonstrated static stretching-induced increases in 20-m sprint times, whereas the dynamic stretching improved sprint performance. Although we did not observe increases in hamstring strength as a result of the dynamic stretching, the results of the present study support the hypothesis that dynamic stretching may be less detrimental to muscle force production than static stretching. The fact that previous studies (17,18,32,42,65) have examined the acute affects of dynamic stretching on power output measured during dynamic tasks, whereas the present study measured isometric muscle strength at different joint angles, may explain the differences among findings.

Two hypotheses have been proposed to explain the stretching-induced force deficit (3,9,12,16,28,47,66): (a) mechanical factors, such as decreases in muscle stiffness and increases in the resting length of sarcomeres that alter the length-tension relationship of a muscle and (b) neuromuscular factors, such as altered motor control strategies and/or reflex sensitivity. Fowles et al. (19) reported that after 15 minutes of recovery from intense stretching, most of the decreases in muscle strength were attributable to intrinsic mechanical properties of the musculotendinous unit, rather than neural factors. It was hypothesized that the stretching may have altered the length-tension relationship and/or the plastic deformation of connective tissues such that the maximal force-producing capabilities of the muscle could be limited (19). Nelson et al. (47) also suggested that the primary mechanism underlying the stretching-induced decreases in force production was related to a decrease in musculotendinous stiffness that may alter the length-tension relationship of the muscle fibers. McHugh et al. (40) examined the angle-torque relationship during isometric leg flexion muscle actions at six different knee joint angles (80°, 65°, 50°, 35°, 20°, and 5°) as an indirect assessment of the length-tension relationship and reported that the stretching-induced force deficit was most prominent at the shorter muscle lengths. The results of the present study are consistent with those of McHugh et al. (40) and indicate decreases in isometric PT after the static stretching at the two shortest muscle lengths (101° and 81°), whereas the dynamic stretching elicited no changes in isometric PT at any of the knee joint angles. Therefore, this evidence provides tentative support to the hypothesis that static stretching causes acute alterations in the length-tension relationship that may reduce the capacity for maximal force production at short muscle lengths. It is possible that the increased EMG activity for isometric contractions at the two shortest muscle lengths after dynamic stretching is sufficient to counteract a loss of force production because of the shift in the length-tension relationship. Although force loss at short muscle lengths is consistent with a rightward shift in the length-tension relationship for isometric contractions, it follows that there should also be a force increase at muscle lengths beyond optimal length. However, an increase in knee flexion torque at 41° (longest muscle length tested) was not apparent. An additional measure at 21° would have provided a better assessment of force production in which cross-bridge formation is compromised by muscle length.

Several studies have examined the neuromuscular factors underlying the stretching-induced force deficit with both surface EMG and MMG (3,9,14,19). For example, Cramer et al. (11,14) and Marek et al. (37) reported decreases in muscle activation (EMG amplitude), but no changes in MMG amplitude, in the vastus lateralis and rectus femoris muscles after a bout of static stretching. In contrast, Evetovich et al. (16) reported no changes in EMG amplitude, but increases in MMG amplitude, for the biceps brachii after static stretching. The results of the present study support those of Evetovich et al. (16) and suggest that neither static nor dynamic stretching decrease muscle activation (EMG amplitude) but that both modes of stretching increase muscle compliance (MMG amplitude). These conflicting results between the stretching-induced changes in EMG and MMG amplitude for the quadriceps femoris (11,16,37) versus the biceps brachii (16) and BF (present study) maybe the result of muscle-specific differences in responses to stretching. For example, stretching-induced increases in MMG amplitude, but no changes in EMG amplitude, have been observed in the biceps brachii (16) and the BF. However, the studies that have reported stretching-induced decreases in EMG amplitude, but no changes in MMG amplitude, have examined the vastus lateralis and rectus femoris muscles (11,16,37). It is possible that stretching affects the activation (EMG amplitude) and stiffness (MMG amplitude) of the biceps brachii and BF differently than the quadriceps femoris muscles. Future studies should examine whether structural, architectural, and/or morphological differences among muscles influence their acute responses to stretching.

It is possible that the increases in EMG amplitude at 101° and 81° and the increases in MMG amplitude at all knee joint angles were the result of some level of post-activation potentiation (PAP). PAP is commonly defined as the transient increase in muscle contractile performance after a previous "conditioning" contractile activity (54). PAP may increase the rate constant of cross-bridge attachments (24), which in turn may allow a greater number of cross-bridges to form, resulting in an increase in force production (8). Faigenbaum et al. (17) and Yamaguchi et al. (65) hypothesized that the increases in force output after dynamic stretching were caused by an enhancement of neuromuscular function, and they implied that the dynamic stretching had a PAP effect on performance. In the present study, dynamic stretching did not improve muscular strength, although EMG amplitude increased (101° and 81°) and MMG amplitude increased, which may have reflected a potentiating effect of the dynamic stretching on muscle activation.

In theory, increases in physiological temperature will increase the compliance of both the contractile and non-contractile tissues in the muscle (58). The non-contractile proteins and connective tissues at the levels of the sarcomere and muscle fiber, respectively, provide the structural rigidity necessary for the actin and myosin filaments to generate force in series (38); therefore, temperature-related increases in the compliance of the non-contractile tissues may allow for greater lateral oscillations (i.e., MMG amplitude) during contraction. In support of this hypothesis, previous studies have demonstrated decreases in MMG amplitude caused by experimentally induced hypothermia in vivo (26) and in vitro (4). As a result, Kimura et al. (26) concluded that the surface MMG may be a useful and reliable method for monitoring the contractile properties of active skeletal muscle under a wide range of physiological temperatures. In the present study, MMG amplitude increased as a result of the dynamic stretching at all four knee joint angles; however, EMG amplitude only increased at 101° and 81° after the dynamic stretching. These results suggest that the increases in MMG amplitude cannot be fully explained by the joint angle-dependent increases in muscle activation (i.e., EMG amplitude) and may reflect increases in muscle temperature as a result of the dynamic stretching. Indeed, Fletcher et al. (18) hypothesized that improvements in power output after dynamic stretching maybe related to increases in muscle temperature. However, future studies should test this hypothesis by examining the acute effects of dynamic stretching on muscle temperature.

Practical Applications

This study was designed to test isolated muscular strength after dynamic stretching in the same manner as previous studies that first reported the stretching-induced force deficit after static stretching (3,9,12,16,19,27,29,44,66). The results of this study have implications for strength and conditioning coaches and men who perform stretching before performance events. The decreases in strength as a result of the static stretching may adversely affect the performance of athletes in sports that require high levels of force production, and these findings are consistent with previous studies (29,43,45,48,66,67). The dynamic stretching, however, did not have a detrimental effect on hamstring strength in the present study. Previous studies (17,18,32,45,65) have reported increases in power output after dynamic stretching. Therefore, our findings, in conjunction with previous studies (17,18,32,45,65), suggest that strength and conditioning professionals should consider incorporating dynamic stretching, rather than static stretching, before performance-related activities to maintain or increase muscle strength and/or power output. In addition, our findings suggest that the decreases in strength that occurred as a result of static stretching were the result of a combination of mechanical (length-tension relationship) and neural (decrease muscle activation) mechanisms. Future studies are needed to identify the mechanisms underlying the differential effects of static versus dynamic stretching on muscle strength, power output, and neuromuscular function, such as increases in muscle temperature and/or potentiation that may occur as a result of dynamic stretching. In addition, investigations are needed to determine whether dynamic stretching has the same influence on joint range of motion as static stretching, or whether certain types of athletic performance even benefit from increases in range of motion.

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