Eur J Appl Physiol (2005) 93: 530–539
DOI 10.1007/s00421-004-1199-x

ORIGINAL ARTICLE

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The acute effects of static stretching on peak torque, mean power output, electromyography, and mechanomyography

Accepted: 18 June 2004 / Published online: 15 December 2004

Abstract The purpose of this study was to examine the acute effects of static stretching on peak torque (PT), the joint angle at PT, mean power output (MP), electromyographic (EMG) amplitude, and mechanomyographic (MMG) amplitude of the vastus lateralis (VL) and rectus femoris (RF) muscles during maximal, voluntary concentric isokinetic leg extensions at 60 and 240°·s^{-1} of the stretched and unstretched limbs. Twenty-one volunteers [mean age (SD) 21.5 (1.3) years] performed maximal, voluntary concentric isokinetic leg extensions for the dominant and non-dominant limbs at 60 and 240°·s^{-1}. Surface EMG (μVrms) and MMG (mVrms) signals were recorded from the VL and RF muscles during the isokinetic tests. PT (Nm), the joint angle at PT, and MP (W) were calculated by a dynamometer. Following the initial isokinetic tests, the dominant leg extensors were stretched using four static stretching exercises. After the stretching, the isokinetic tests were repeated. PT decreased ($P \leq 0.05$) from pre- to post-stretching for the stretched limb at 60 and 240°·s^{-1} and for the unstretched limb at 60°·s^{-1}. EMG amplitude of the VL and RF also decreased ($P \leq 0.05$) from pre- to post-stretching for the stretched and unstretched limbs. There were no stretching-induced changes ($P > 0.05$) for the joint angle at PT, MP, or MMG amplitude. These findings indicated stretching-induced decreases in force production and muscle activation. The decreases in PT and EMG amplitude for the unstretched limb suggested that the stretching-induced decreases may be due to a central nervous system inhibitory mechanism.

Keywords Contralateral · Electromyography · Mechanomyography · Quadriceps femoris · Stretch

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Introduction

Static stretching is commonly performed prior to exercise (ACSM 2000) and athletic events (Beaulieu 1981; Holcomb 2000). It is believed that increasing flexibility (increasing joint range of motion) will promote better performances and reduce the risk of injury during strenuous exercise (Shellock and Prentice 1985; Smith 1994). Previous studies have used muscle stretching techniques to examine passive force production (Magnusson 1998; Magnusson et al. 1995, 1996), stress-relaxation characteristics of muscle (McHugh et al. 1992; Taylor et al. 1990; Toft et al. 1989), neuromuscular reflex patterns (Guissard et al. 1988; Hutton 1992; Vujnovich and Dawson 1994), factors contributing to muscle damage (Armstrong et al. 1993; Lieber et al. 1991), and mechanisms of increase in musculotendonous flexibility (Magnusson 1998; Toft et al. 1989). Until recently, however, few studies have examined the acute effects of stretching on performance measures (deVries 1963). Recent evidence has suggested that pre-exercise stretching may compromise a muscle's ability to produce maximal force (Avela et al. 1999; Behm et al. 2001; Cramer et al. 2004; Evetovich et al. 2003; Fowles et al. 2000; Kokkonen et al. 1998; McNeal and Sands 2003; Nelson et al. 2001b; Young and Elliott 2001). Two primary hypotheses have been developed to explain the stretch-induced strength deficit (Avela et al. 1999; Behm et al. 2001; Cramer et al. 2004; Evetovich et al. 2003;

Fowles et al. 2000; Knudson et al. 2001; Kokkonen et al. 1998; McNeal and Sands 2003; Nelson et al. 2001b; Young and Elliott 2001): (1) mechanical factors such as changes in muscle stiffness and (2) neuromuscular factors such as altered motor control strategies and/or reflex sensitivity.

Simultaneous measurements of mechanomyography (MMG) and electromyography (EMG) can provide unique information about the mechanical properties and motor control strategies during various types of muscle actions. 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 (Barry and Cole 1988; Orizio 1993; Stokes 1993). Barry and Cole (1988) and Orizio (1993) have suggested that the lateral oscillations are generated by: (1) a gross lateral movement of the muscle at the initiation of a contraction that is generated by non-simultaneous activation of muscle fibers, (2) smaller subsequent lateral oscillations occurring at the resonant frequency of the muscle, and (3) dimensional changes of the active muscle fibers. Furthermore, Gordon and Holbourn (1948) have proposed that the lateral oscillations produced by contracting muscles are reflective of the ''mechanical counterpart'' of the motor unit activity as measured by EMG. Comprehensive reviews of the technical aspects of MMG, including its validity and reproducibility, have been provided elsewhere by Orizio and colleagues (Orizio 1993; Orizio et al. 2003), Stokes and Blythe (2001), and Akataki et al. (1999).

While the amplitude of the MMG signal can be influenced by factors such as muscle tension, length, mass, intramuscular pressure, and the viscosity of the surrounding medium (Orizio 1993), it has been suggested that MMG amplitude is inversely proportional to the stiffness of an active muscle (Cramer et al. 2000a, b, 2002c; Ebersole et al. 2000; Evetovich et al. 1997, 1998, 1999; Smith et al. 1997, 1998). Recent evidence has also indicated that MMG amplitude is closely related to muscle power output (Cramer et al. 2000a, 2002a, b, c). In addition, EMG amplitude values can provide further insight regarding motor unit recruitment (Arendt-Nielsen and Mills 1985; Komi and Tesch 1979; Moritani et al. 1985; Solomonow et al. 1990; Westbury and Shaughnessy 1987). Therefore, MMG and EMG measurements may provide useful information about the mechanical and electrical changes that have been proposed to explain the stretching-induced force deficit.

Previous studies have examined the effects of stretching on maximal strength (Behm et al. 2001; Fowles et al. 2000; Kokkonen et al. 1998; Nelson et al. 2001a, b; Nelson and Kokkonen 2001), explosive force production (Young and Elliott 2001), vertical jump performance (Knudson et al. 2001; McNeal and Sands 2003; Young and Elliott 2001), concentric isokinetic peak torque (Cramer et al. 2004; Evetovich et al. 2003; Nelson et al. 2001b), and isometric force production at different joint angles (Behm et al. 2001; Fowles et al. 2000; Nelson et al. 2001a). Only one previous study has estimated the relative contributions of the mechanical and electrical mechanisms underlying the strength deficit after stretching (Fowles et al. 2000). Fowles et al. (2000) stated, however, that: ''The duration of stretch performed in this experiment is more similar to prolonged stretch procedures employed in animal experimental models and, therefore, may have limited application to sport stretching performed in conjunction with athletic performance'' (p. 1179).

Thus, limited data are available regarding the mechanical and electrical components of the strength deficit as a result of traditional static stretching. In addition, no previous studies have investigated how stretching may affect maximal, voluntary concentric isokinetic muscle actions of the stretched and unstretched leg extensor muscles. If the stretching-induced force deficit is mediated by a central nervous system mechanism, it is possible that the unstretched limb may also be affected. The purposes of this study, therefore, were to examine the acute effects of static stretching on peak torque (PT), the joint angle at PT, mean power output (MP), EMG amplitude, and MMG amplitude of the vastus lateralis (VL) and rectus femoris (RF) muscles during maximal, voluntary concentric isokinetic leg extensions at 60 and 240°·s^{-1} of the stretched and unstretched limbs.

This study was designed to: (1) test the hypothesis of Nelson et al. (2001b) that the acute effects of static stretching on PT are velocity-specific during maximal, voluntary concentric isokinetic leg extensions, (2) extend the findings of Avela et al. (1999) by examining the responses of the stretched and unstretched limbs, and (3) incorporate surface EMG and MMG signals to examine the neural (Avela et al. 1999; Behm et al. 2001; Fowles et al. 2000) and mechanical (Fowles et al. 2000; Kokkonen et al. 1998; Nelson et al. 2001a, b) factors underlying the stretching-induced decreases in force production. Based upon previous studies (Avela et al. 1999; Behm et al. 2001; Cramer et al. 2004; Evetovich et al. 2003; Fowles et al. 2000; Nelson et al. 2001b), we hypothesized that PT, MP, and EMG amplitude will decrease, while the joint angle at PT and MMG amplitude will increase in response to the static stretching.

Methods

Subjects

Twenty-one adult subjects ([7 males and 14 females, mean age (SD) 21.5 (1.3) years]) volunteered to participate in this investigation. The study was approved by the University Institutional Review Board for Human Subjects and all subjects completed a health history questionnaire and signed a written informed consent prior to testing. The experiments conducted during this

study complied with the current laws regarding human subject research in the United States.

Isokinetic testing procedure

Each subject completed a 5-min warm-up at 50 W on a stationary cycle ergometer prior to the initial isokinetic testing. Before and after the static stretching exercises, concentric isokinetic PT and MP for extension of the dominant (based on kicking preference) and non-dominant limbs were measured separately using a calibrated Cybex 6000 dynamometer (Cybex, Division of Lumex, Inc., Ronkonkoma, N.Y.) at randomly ordered velocities of 60 and 240°·s^{-1}. The subjects were in a seated position with a restraining strap over the pelvis and trunk in accordance with the Cybex 6000 User's Guide (*Cybex 6000 Testing and Rehabilitation User's Guide*, 1991). The input axis of the dynamometer was aligned with the axis of the knee, while the contralateral leg was braced against the limb stabilization bar. In addition, prior to each isokinetic assessment, gravity corrections were made for the weight of the limb. Three submaximal warm-up trials preceded three maximal muscle actions at each velocity. A 2-min rest was allowed between testing at each velocity, and a minimum of 5 min was allowed between testing for each limb. The highest PT was selected as the representative score. The joint angle at which PT occurred (degrees below full extension, where full extension = 0º) was provided by the Cybex 6000 software. MP was calculated by the Cybex 6000 software by dividing the highest work performed (area under the torque vs range of motion curve) by the muscle action time during the best working repetition.

Static stretching exercises

Immediately following the pre-stretching isokinetic tests, each subject underwent four static stretching exercises designed to stretch the leg extensor muscles of the dominant limb only, according to the procedures of Nelson et al. (2001b). Four repetitions of each stretching exercise were held for 30 s at a point of mild discomfort, but not pain, as acknowledged by the subject. Between each stretching repetition, the leg was returned to a neutral position for a 20-s rest period. The total stretching time [mean (SD)] was 16.1 (1.9) min.

Each subject performed an unassisted stretching exercise followed by three assisted stretching exercises. For the unassisted stretching exercise, the subject stood upright with one hand against a wall for balance. The subject then flexed the dominant leg to a knee joint angle of 90°. The ankle of the flexed leg was grasped by the ipsilateral hand, and the foot was raised so that the heel of the dominant foot approached the buttocks. Following the unassisted stretching exercise, the remaining stretching exercises were completed with the assistance of the primary investigator.

The first assisted stretching exercise was performed with the subject lying prone on a padded table with the legs fully extended. The dominant leg was flexed at the knee joint and slowly pressed down so that the subject's heel approached the buttocks. If the heel was able to contact the buttocks, the knee was gently lifted off the supporting surface, causing a slight hyperextension at the hip joint, to complete the stretch. To perform the second assisted stretching exercise, the subject stood with his or her back to a table and rested the dorsal surface of their dominant foot on the table by flexing the leg at the knee joint. From this position, the dominant leg extensors were stretched by gently pushing back on both the knee of the flexed leg and the corresponding shoulder. The final assisted stretching exercise began with the subject lying supine along the edge of the padded table with the dominant leg hanging from the table. The dominant leg was flexed at the knee and the thigh was slightly hyperextended at the hip by gently pressing down on the knee. Immediately after the stretching exercises, each subject sat quietly for 4.4 (1.3) min before performing the post-stretching isokinetic tests for the stretched limb and 14.9 (2.8) min before testing the unstretched limb. Because the primary purpose of this study was to examine the effects of the stretching on the stretched limb, the post-stretching isokinetic testing was always performed on the stretched limb first.

EMG measurements

Bipolar (7.62 cm center-to-center) surface electrode (Quinton Quick Prep silver-silver chloride) arrangements were placed along the longitudinal axes of the VL and RF muscles of the dominant and non-dominant limbs (Cramer et al. 2000a, b, 2002b, c; Ebersole et al. 1999). The interelectrode distances were selected to accommodate placing the MMG sensors between the active EMG electrodes (Cramer et al. 2000a, b, 2002b, c; Ebersole et al. 1999; Smith et al. 1998). The electrodes for the VL were placed over the lateral portion of the muscle at approximately the midpoint between the greater trochanter and lateral condyle of the femur. For the RF, the electrodes were placed half-way between the inguinal crease and the superior border of the patella. For all EMG measurements, the reference electrodes were placed over the iliac crests. Interelectrode impedance for each muscle was kept below 2,000 Ω by shaving the area and careful skin abrasion. The EMG signals were preamplified (gain ×1,000) using a differential amplifier (EMG 100, Biopac Systems Inc., Santa Barbara, Calif.; bandwidth = 1–5,000 Hz).

MMG measurements

The MMG signals were detected by piezoelectric crystal contact sensors (Hewlett-Packard, 21050A, bandwidth

0.02–2,000 Hz). For each muscle (VL and RF), a sensor was placed between the active EMG electrodes. A stabilizing ring, double-sided foam tape, and microporous tape were used to ensure consistent contact pressure of the MMG sensors (Bolton et al. 1989; Cramer et al. 2000a, b, 2002a, b, c; Ebersole et al. 1999).

Signal processing

The raw EMG and MMG signals were stored on a personal computer (Macintosh 7100/80 AV Power PC) and expressed as root mean square (rms) amplitude values by software (Labview, version 3.1.1, National Instruments, Austin, Tex.). The sampling frequency was 1,000 Hz for all signals. The EMG and MMG signals were bandpass filtered (fourth-order Butterworth filter) at 10–500 Hz and 5–100 Hz, respectively. The EMG and MMG amplitude values were calculated for a time period that corresponded to an approximate 30º range of motion. For example, at $60°\cdot s^{-1}$ the amplitude for 0.5 s of the EMG and MMG signals was calculated, while at $240°\cdot s^{-1}$ the amplitude for 0.125 s was calculated (Evetovich et al. 1997, 1999; Smith et al. 1998). This allowed for comparisons that were based on a standardized 30° range of motion from approximately 120° to 150° of flexion at the knee. This portion of the range of motion was selected to avoid the acceleration and deceleration phases of the movement which are typical of isokinetic dynamometers (Brown et al. 1995).

Statistical analyses

Two separate four-way repeated measures ANOVAs [time (pre- vs post-stretching) × limb (stretched vs unstretched) × muscle (VL vs RF) × velocity ($60°\cdot s^{-1}$ vs $240°\cdot s^{-1}$)] were used to analyze the EMG and MMG amplitude data. Two separate three-way repeated measures ANOVAs [time (pre- vs post-stretching) × limb (stretched vs unstretched) × velocity ($60°\cdot s^{-1}$ vs $240°\cdot s^{-1}$)] were used to analyze the PT and MP data. When appropriate, follow-up analyses included two-way repeated measures ANOVAs and paired-samples *t*-tests. All of the independent variables in the present study (time, limb, muscle, and velocity) were within-subjects, repeated measures factors. An alpha of $P \leq 0.05$ was considered statistically significant for all comparisons.

Reliability

Previous test-retest reliability from our laboratory for PT, EMG amplitude, and MMG amplitude during maximal, voluntary, concentric isokinetic leg extensions indicated that, for eight male subjects measured 48 h apart, the intraclass correlation coefficients (*R*) ranged from 0.93 to 0.94, 0.85 to 0.96, and 0.97 to 0.98, respectively, with no significant ($P > 0.05$) differences between mean values for test versus retest at either velocity (60 and $240°\cdot s^{-1}$).

Results

PT and joint angle at PT

Table 1 shows the mean (SEM) values for PT (Nm) and the joint angle at PT. For PT, the analyses indicated a significant three-way interaction (time × limb × velocity). The statistical model was decomposed by using two separate two-way repeated measures ANOVAs (time × velocity) for each limb. For the stretched limb, there was no two-way interaction (time × velocity), but there were significant main effects for time and velocity. Therefore, the marginal means for PT (collapsed across velocity) decreased from pre- to post-stretching (Fig. 1a), while the marginal means for PT (collapsed across time) were greater at $60°\cdot s^{-1}$ than $240°\cdot s^{-1}$. For the unstretched limb, there was a significant interaction (time × velocity). The model was further decomposed by using paired-samples *t*-tests (pre- vs post-stretching and $60°\cdot s^{-1}$ vs $240°\cdot s^{-1}$), which indicated that PT decreased from pre- to post-stretching at $60°\cdot s^{-1}$, but not $240°\cdot s^{-1}$, for the unstretched limb (Fig. 1b). In addition, PT at $60°\cdot s^{-1}$ was greater than $240°\cdot s^{-1}$ during the pre- and post-stretching assessments for the unstretched limb. Overall, the analyses indicated a decrease in PT from pre- to post-stretching at $60°\cdot s^{-1}$ for both the stretched and unstretched limbs as well as a decrease at $240°\cdot s^{-1}$ for the

Table 1 Peak torque, joint angle at peak torque, and mean power output values

		Pre-stretching				Post-stretching			
		Stretched limb		Unstretched limb		Stretched limb		Unstretched limb	
		$60°\cdot s^{-1}$	$240°\cdot s^{-1}$	$60°\cdot s^{-1}$	$240°\cdot s^{-1}$	$60°\cdot s^{-1}$	$240°\cdot s^{-1}$	$60°\cdot s^{-1}$	$240°\cdot s^{-1}$
Peak torque (Nm)	Mean	202.1	136.5	209.0	129.3	196.6	130.8	196.3	125.9
	SEM	11.1	9.4	11.4	8.2	10.5	8.2	9.5	7.6
Joint angle at peak torque (°)	Mean	64.0	51.9	61.3	57.2	63.0	55.0	62.4	58.3
	SEM	0.9	2.7	1.2	1.6	1.3	1.9	1.2	1.3
Mean power output (W)	Mean	131.7	234.7	119.9	225.0	121.2	240.6	120.7	235.0
	SEM	7.0	18.7	6.2	16.3	7.4	14.4	5.9	17.2

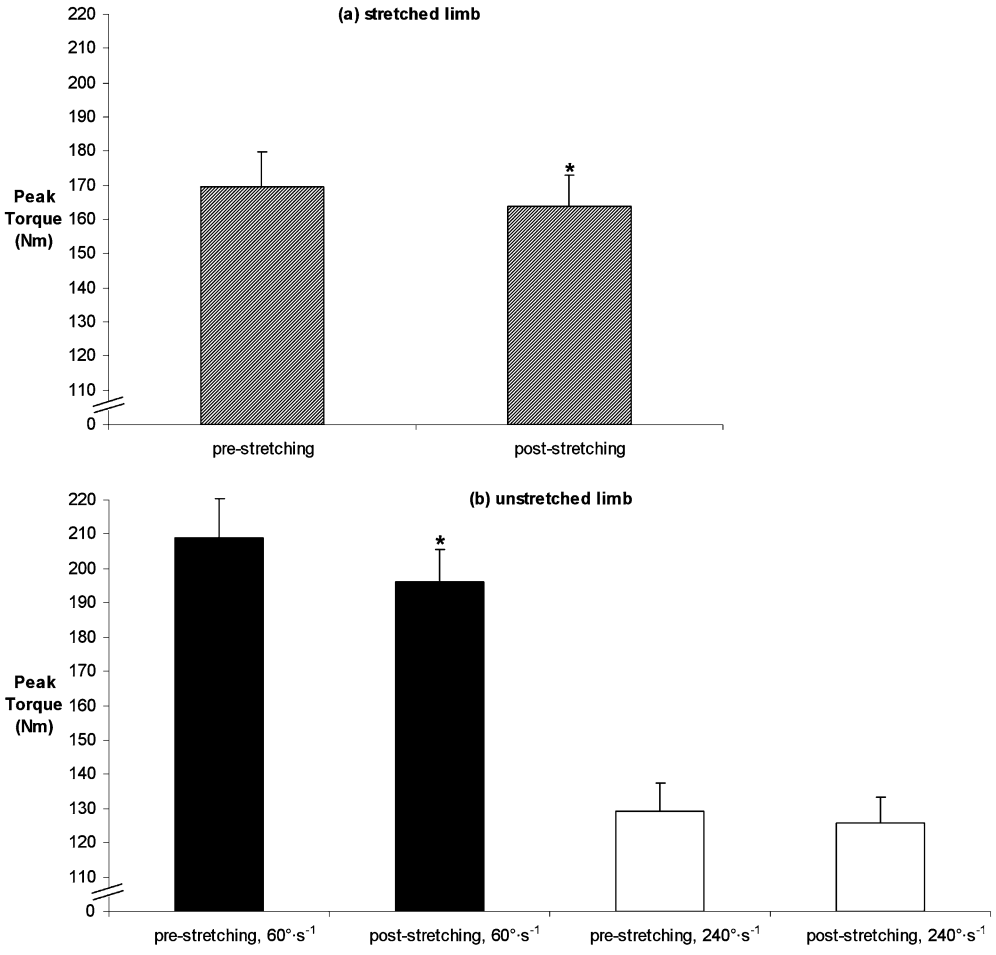

Fig. 1 a The marginal means for peak torque (collapsed across velocity, Nm) for the stretched limb decreased (*$P \leq 0.05$) from pre- to post-stretching. **b** Peak torque (Nm) for the unstretched limb decreased (*$P \leq 0.05$) from pre- to post-stretching at $60°\cdot s^{-1}$, but not at $240°\cdot s^{-1}$. Values are mean (SEM)

stretched limb only (Fig. 1a, b). Furthermore, PT at $60°\cdot s^{-1}$ was greater than at $240°\cdot s^{-1}$ for all isokinetic assessments, which was consistent with the traditional force-velocity relationship.

For the joint angle at PT, the analyses indicated no significant three- or two-way interactions (time × limb × velocity, time × limb, time × velocity, or limb × velocity), no main effects for time or limb, but a significant main effect for velocity. The model was decomposed by conducting a paired-samples *t*-test on the marginal means for velocity ($60°\cdot s^{-1}$ vs $240°\cdot s^{-1}$, collapsed across time and limb), which indicated that the joint angle at PT was greater at $60°\cdot s^{-1}$ than $240°\cdot s^{-1}$ during both the pre- and post-stretching assessments for both limbs. Overall, however, there was no change in the joint angle at PT from pre- to post-stretching in either the stretched or unstretched limbs at $60°\cdot s^{-1}$ or $240°\cdot s^{-1}$.

Mean power output

Table 1 shows the mean (SEM) values for MP (W). The analyses indicated no significant three-way interaction (time × limb × velocity), no two-way interactions (time × limb, time × velocity, or limb × velocity), no main effects for time or limb, but a significant main effect for velocity. The statistical model was decomposed by collapsing across time and limb and using a paired-samples *t*-test ($60°\cdot s^{-1}$ vs. $240°\cdot s^{-1}$), which indicated that the marginal means for MP (collapsed across time and limb) increased from $60°\cdot s^{-1}$ to $240°\cdot s^{-1}$. In summary, there was no change in MP from pre- to post-stretching in either the stretched or unstretched limbs at $60°\cdot s^{-1}$ or $240°\cdot s^{-1}$. There was, however, a velocity-related increase in MP from $60°\cdot s^{-1}$ to $240°\cdot s^{-1}$ for all isokinetic assessments, which was consistent with the traditional power-velocity relationship.

EMG amplitude

Table 2 shows the mean (SEM) values for EMG amplitude (μVrms). The analyses indicated no four-way interaction (time × limb × muscle × velocity), no three-way interactions involving limb, but a significant three-way interaction (time × muscle × velocity). The statistical model was decomposed by collapsing across limb and using two separate two-way repeated measures ANOVAs (time × velocity) for each muscle. For the VL, there was no two-way interaction (time × velocity), but there

Table 2 EMG amplitude and MMG amplitude values. *VL* Vastus lateralis muscle, *RF* rectus femoris muscle

		Pre-stretching								Post-stretching							
		Stretched limb				Unstretched limb				Stretched limb				Unstretched limb			
		60°·s^{-1}		240°·s^{-1}		60°·s^{-1}		240°·s^{-1}		60°·s^{-1}		240°·s^{-1}		60°·s^{-1}		240°·s^{-1}	
		VL	RF	VL	RF	VL	RF	VL	RF	VL	RF	VL	RF	VL	RF	VL	RF
EMG Amplitude (μVrms)	mean	454.4	848.1	623.8	925.6	555.0	920.7	739.7	928.1	471.8	847.9	608.4	902.8	490.1	799.3	659.4	902.1
	SEM	29.0	60.8	66.1	86.8	43.7	70.1	70.4	80.2	33.5	68.8	61.6	82.2	38.5	61.3	53.4	68.6
MMG Amplitude (mVrms)	mean	40.9	43.6	202.6	351.3	27.9	42.7	158.5	266.8	35.1	43.8	238.0	413.6	28.4	45.1	192.3	285.6
	SEM	10.5	9.1	42.8	43.9	6.0	6.2	34.9	37.1	7.3	7.4	85.0	58.4	5.7	6.3	47.7	37.0

were significant main effects for time and velocity. Therefore, the marginal means for EMG amplitude (collapsed across limb and velocity) for the VL muscle decreased from pre- to post-stretching (Fig. 2a), while the marginal means for EMG amplitude (collapsed across time and limb) were greater at 240°·s^{-1} than 60°·s^{-1}. For the RF, there was no two-way interaction (time × velocity), no main effect for velocity, but there was a significant main effect for time. Therefore, the marginal means for EMG amplitude (collapsed across limb and velocity) for the RF muscle decreased from pre- to post-stretching (Fig. 2b). Overall, these analyses indicated that EMG amplitude of the VL and RF muscles decreased from pre- to post-stretching for both the stretched and unstretched limbs at 60°·s^{-1} and 240°·s^{-1} (Fig. 2a, b).

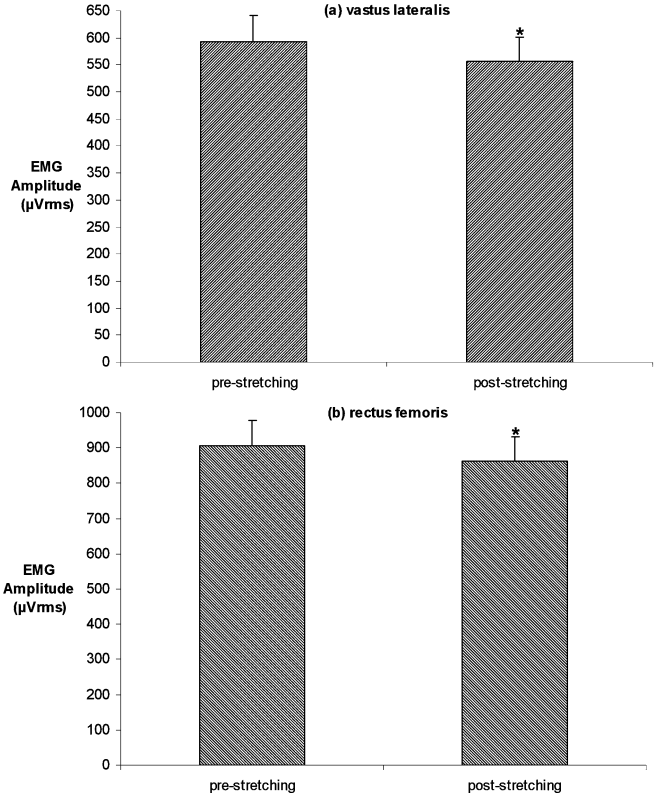

Fig. 2 The marginal means for EMG amplitude (collapsed across limb and velocity, μVrms) decreased (*$P \leq 0.05$) from pre- to post-stretching for (**a**) the vastus lateralis muscle and (**b**) the rectus femoris muscle. Values are mean (SEM)

MMG amplitude

Table 2 shows the mean (SEM) values for MMG amplitude (mVrms). The analyses indicated no four-way interaction (time × limb × muscle × velocity), no three-way interactions (time × limb × muscle; time × limb × velocity; time × muscle × velocity; limb × muscle × velocity), but there were two significant two-way interactions (muscle × velocity and limb × velocity). The statistical model was decomposed by collapsing across time and using two separate two-way repeated measures ANOVAs (muscle × velocity) for each limb. For the stretched limb, there was a significant interaction (time × velocity). The model was further decomposed by using paired-samples *t*-tests (60°·s^{-1} vs. 240°·s^{-1}), which indicated that MMG amplitude increased from 60°·s^{-1} to 240°·s^{-1} for the stretched limb. For the unstretched limb, there was no two-way interaction (time × velocity), but there was a significant main effect for velocity. Therefore, the marginal means for MMG amplitude (collapsed across time and muscle) increased from 60°·s^{-1} to 240°·s^{-1} for the unstretched limb. In summary, these analyses indicated no changes in MMG amplitude from pre- to post-stretching for either the stretched or unstretched limb at 60°·s^{-1} or 240°·s^{-1}. There were, however, velocity-related increases in MMG amplitude from 60°·s^{-1} to 240°·s^{-1} for all isokinetic assessments, which was consistent with our previous studies (Cramer et al. 2000a, 2000b, 2002c).

Discussion

The results of the present study indicated a 3.3% decrease in PT for the stretched limb at 60°·s^{-1} and 240°·s^{-1} (Fig. 1a) as a result of the static stretching. These findings were consistent with previous studies (Avela et al. 1999; Behm et al. 2001; Cramer et al. 2004; Evetovich et al. 2003; Fowles et al. 2000; Kokkonen et al. 1998; McNeal and Sands 2001, 2003; Nelson et al. 2001a, b; Young and Elliott 2001) that have reported

decreases in the force producing capabilities of a muscle following a bout of static stretching. Nelson et al. (2001b), however, reported stretching-induced decreases in isokinetic PT of the leg extensors at $60°\cdot s^{-1}$ and $90°\cdot s^{-1}$ (7.2% and 4.5% decreases, respectively), but no changes at $150°\cdot s^{-1}$, $210°\cdot s^{-1}$, or $270°\cdot s^{-1}$. It was concluded that the decreases in PT after stretching were velocity-specific and occurred primarily under the high torque production conditions associated with the slower velocities ($60°\cdot s^{-1}$ and $90°\cdot s^{-1}$), but not the lower torque production conditions at the faster velocities ($150°\cdot s^{-1}$, $210°\cdot s^{-1}$, and $270°\cdot s^{-1}$) (Nelson et al. 2001b). The present findings, as well as those of previous studies (Cramer et al. 2004; Evetovich et al. 2003), however, indicated decreases in PT at both slow ($30°\cdot s^{-1}$ and $60°\cdot s^{-1}$) and fast ($240°\cdot s^{-1}$ and $270°\cdot s^{-1}$) velocities (for the stretched limb) and suggested that the stretching-induced decreases in PT may not be as velocity-specific (for the stretched limb) as suggested by Nelson et al. (2001b).

According to Fowles et al. (2000), however, the magnitude of the inhibitory effect of stretching may be influenced by the magnitude of the stretch. Thus, even though the current study replicated the stretching protocol of Nelson et al. (2001b), the conflicting results between Nelson et al. (2001b) and those of Evetovich et al. (2003), Cramer et al. (2004), and the present study may be related to some unknown or unintended differences in stretching intensities between protocols. Future studies should examine the effects of varied doses and intensities of static stretching on muscle strength and power output.

Two primary hypotheses have been proposed to explain the stretching-induced decreases in the maximal force producing capability of a muscle: (1) neural factors such as decreased motor unit activation, firing frequency, and/or altered reflex sensitivity (Avela et al. 1999; Behm et al. 2001; Fowles et al. 2000), and (2) mechanical factors such as alterations in the viscoelastic properties of the muscle that may affect the length/tension relationship (Fowles et al. 2000; Kokkonen et al. 1998; Nelson et al. 2001a, b). Several studies have reported stretching-induced decreases in muscle activation through the use of surface (Behm et al. 2001; Fowles et al. 2000) and fine-wire (Avela et al. 1999) EMG as well as twitch interpolation (Behm et al. 2001; Fowles et al. 2000). For example, Avela et al. (1999) reported decreases in motor unit recruitment (EMG amplitude) and firing frequency (zero crossing rate) after repeated passive stretches of the plantar flexors. Using a formula by Duchateau (1995), Fowles et al. (2000) reported that 60% of the stretching-induced decreases in force production of the triceps surae (up to 15 min post-stretching) were due to neural factors. In addition, Behm et al. (2001) suggested that at least part of the stretching-induced decreases in maximal force production of the leg extensors was due to decreases in muscle activation. Evetovich et al. (2003), however, reported stretching-induced decreases in maximal, voluntary concentric isokinetic PT, but no changes in surface EMG amplitude for the biceps brachii muscle. The results of the present study support those of Avela et al. (1999), Behm et al. (2001), and Fowles et al. (2000) and indicate stretching-induced decreases in EMG amplitude at $60°\cdot s^{-1}$ and $240°\cdot s^{-1}$ for the VL and RF muscles of both the stretched and unstretched limbs. The differences between these results and those of Evetovich et al. (2003) may be related to the architectural and/or anatomical differences between the muscle groups involved (quadriceps femoris vs biceps brachii).

It has also been hypothesized that stretching-induced decreases in force production may be due to decreases in musculotendonous stiffness that affect the length/tension relationship (Fowles et al. 2000; Kokkonen et al. 1998; Nelson et al. 2001a, b). Fowles et al. (2000) reported that after 15 min of recovery, most of the decreases in force production after stretching were attributable to intrinsic mechanical properties of the muscle, rather than neural factors. In addition, previous studies (Kokkonen et al. 1998; Nelson et al. 2001a, b; Nelson and Kokkonen 2001) have suggested that the primary mechanism underlying the stretching-induced decreases in force (after 10 min of recovery) is related to decreases in muscle stiffness that may alter the length/tension relationship of the muscle fibers, causing increases in sarcomere shortening distance and velocity and, therefore, decreases in force production due to the force/velocity relationship. In our previous study (Cramer et al. 2004), we hypothesized that stretching-induced changes in the length/tension relationship may be manifested through changes in the torque versus range of motion relationship, which, in turn, may affect the joint angle at PT. In support of this hypothesis, previous studies have demonstrated stretching-induced increases in the joint angle at which maximal force production occurred during isometric (Fowles et al. 2000; Nelson et al. 2001a) and isokinetic (Cramer et al. 2004) muscle actions. The results of the present study, however, indicate no changes in the joint angle at PT from pre- to post-stretching. These findings were consistent with Nelson et al. (2001b) that reported no stretching-induced changes in the joint angle at PT for maximal, voluntary concentric isokinetic muscle actions of the leg extensors at velocities ranging from $60°\cdot s^{-1}$ to $270°\cdot s^{-1}$.

It is known, however, that the joint angle at PT is velocity-dependent and tends to occur closer to full extension as velocity increases (Kannus and Beynnon 1993). It is possible, therefore, that if static stretching affects the shortening velocity of the muscle fibers as hypothesized by Nelson et al. (2001b), then the joint angle at PT may be simultaneously affected by stretching-induced changes in the shape of the length-tension relationship and increases in the shortening velocity of the muscle fibers. Therefore, future studies should examine the effects of static stretching on the shape of the angle-torque curve (generated during isokinetic muscle actions), in addition to the joint angle at PT, as indicators of stretching-induced changes in the length-tension relationship.

Previous studies have reported decreases in vertical jumping performance (Cornwell et al. 2001; McNeal and Sands 2003; Young and Elliott 2001) as well as mean power and velocity for the bench press exercise (Fry et al. 2003) as a result of static stretching. For the present study, we hypothesized that PT and MP would decrease in response to static stretching; however, there were no changes in MP from pre- to post-stretching. MP was calculated in the present study by dividing the area under the torque versus range of motion curve (impulse) by time. It is possible, therefore, that stretching-induced changes in the length/tension relationship, though not evident by changes in the joint angle at PT, may have compensated for the decreases in PT by maintaining the area under the torque versus range of motion curve, allowing for greater force production at other joint angles during the range of motion. That is, the static stretching may have reduced the PT, but was not sufficient to alter the impulse of the torque versus range of motion relationship. Future studies should examine the effects of static stretching on the torque versus range of motion relationship for maximal, isokinetic muscle actions to determine how stretching affects torque production throughout the range of motion.

It has been suggested that MMG amplitude is indirectly related to muscle stiffness (Barry and Cole 1988; Cramer et al. 2000a, b, 2002c; Evetovich et al. 1997, 1998; Orizio et al. 1989), but directly related to muscle power output (Bodor 1999; Cramer et al. 2000a, b, 2002a, b, c). Barry and Cole (1988) and Orizio et al. (1989) have suggested that a stiff muscle may attenuate the lateral oscillations of individual muscle fibers and, therefore, decrease the amplitude of the MMG signal. In addition, muscle stiffness has been defined by Ettema and Huijing (1994) as being directly related to the number of attached myosin cross-bridges. Based on these studies (Barry and Cole 1988; Cramer et al. 2000a, 2002c; Ettema and Huijing 1994; Evetovich et al. 1997, 1998, 2003; Orizio et al. 1989), we hypothesized that static stretching would decrease muscle stiffness and increase MMG amplitude. In support of this hypothesis, Evetovich et al. (2003) reported greater MMG amplitude values after static stretching. The results of the present study, however, indicated no changes in MMG amplitude from pre- to post-stretching. The specific mechanisms underlying these conflicting results are unclear, but may be related to the different muscle groups tested (quadriceps femoris vs biceps brachii). Further studies are needed to examine the effects of static stretching on muscle stiffness and MMG amplitude to determine the mechanical factors involved in the stretching-induced decreases in muscle strength.

Bodor (1999) hypothesized that MMG amplitude is more closely related to MP than PT during maximal, voluntary concentric isokinetic muscle actions. Our previous studies have supported Bodor's (1999) hypothesis and indicated that the velocity-related patterns for MMG amplitude tracked MP, but were dissociated from PT, during maximal, concentric (Cramer et al. 2000a, 2002c) and eccentric (Cramer et al. 2002a, b) isokinetic muscle actions. Therefore, the results of the present study extended our previous findings to include the responses to static stretching and further supported Bodor's (1999) hypothesis in that MP as well as MMG remained unchanged while PT decreased as a result of static stretching. In addition, the velocity-related increases in MMG amplitude and MP from $60°·s^{-1}$ to $240°·s^{-1}$ were consistent with Bodor's (1999) hypothesis as well as our previous studies (Cramer et al. 2000a, 2002c).

Two unique findings of this study were the velocity-specific decreases in PT at $60°·s^{-1}$, but not at $240°·s^{-1}$ (Fig. 1b), as well as the decreases in EMG amplitude (Fig. 2a and 2b) for the unstretched limb from pre- to post-stretching. In a study involving repeated passive stretches of the triceps surae muscles, Avela et al. (1999) reported decreases in maximal force production and reflex sensitivity for the stretched (experimental) leg as well as similar, but nonsignificant, changes in the unstretched (control) leg. It was hypothesized that the decreases in force production, in conjunction with the observed decreases in motor unit activation and firing frequency, "could imply the occurrence of central fatigue (12) [(Gandevia 1992)], which can be caused either by supraspinal fatigue (7) [(Brasil-Neto et al. 1994)] or by changes in the inhibitory as well as disfacilitatory signals originating from the contracting muscles (3, 18) [(Bigland-Ritchie et al. 1986; Hagbarth et al. 1986)] (3, p. 1287–1288)".

The authors (Avela et al. 1999) went on to state that "if such an effect [central fatigue] could have occurred, it would also have appeared in the contralateral side (control leg), which was not the case, as demonstrated by the nonsignificant changes in the MVC of that side (p. 1288)".

Thus, the decreases in PT and EMG amplitude in the stretched and unstretched limbs in the present study, as well as previous findings (Cramer et al. 2004), provided tentative support to the hypothesis of Avela et al. (1999) that stretching-induced decreases in maximal force production may be due to a decrease in motor unit activation and/or firing frequency caused by an unidentified central nervous system (CNS) inhibitory mechanism.

In summary, the primary findings of this study were the stretching-induced decreases in PT and EMG amplitude for the VL and RF muscles of the stretched and unstretched limbs. These results support the hypotheses of previous studies (Avela et al. 1999; Behm et al. 2001; Fowles et al. 2000) that the compromised force-producing capabilities of a muscle as a result of static stretching may be due, in part, to decreases in muscle activation. Moreover, the decreases in PT and EMG amplitude for the unstretched limb provide tentative support to the hypothesis of Avela et al. (1999) that the stretching-induced decreases in muscle activation may be mediated by an unidentified CNS inhibitory

mechanism. In the present study, MP, MMG amplitude, and the joint angle at PT exhibited no changes in response to the static stretching. These results further support the hypothesis of Bodor (1999) in that MMG amplitude tracked the pattern of MP, but not PT, from pre- to post-stretching and from $60°\cdot s^{-1}$ to $240°\cdot s^{-1}$. Future studies are recommended to examine the effects of static stretching on the angle-torque relationship to determine the impact of stretching on force production throughout the range of motion.

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