Title

REDUCTIONS IN ACTIVE PLANTARFLEXOR MOMENT ARE SIGNIFICANTLY CORRELATED TO STATIC STRETCH DURATION

Running Head

MOMENT DECREMENTS AFTER STRETCH

Keywords: Moment, stretching, correlation, pre-performance, triceps surae

ABSTRACT

The present study investigated the effects of static stretch duration on peak isometric plantarflexor moment and passive ankle moment in seven healthy volunteers. After passive ankle plantarflexor moment was recorded on an isokinetic dynamometer, the peak isometric ankle moment was measured with simultaneous EMG monitoring of the medial gastrocnemius. Subjects subsequently performed a single 5-s stretch, a single 15-s stretch, four 5-s stretches, four 15-s stretches, or no stretch (control), before being re-tested. All subjects randomly completed each condition with a 24-hr rest between testing occasions. The main finding of the study was a significant correlation between reductions in peak moment and stretch duration (r=0.68; p<0.05), which became significantly different from the control group after four 15-s stretches

(16.7+5.3%; $p<0.05$). There was a similar decrease in passive moment (20.9+1.3%; $p<0.05$) after each stretch condition, but this was not accompanied by a change in hysteresis. EMG recorded during the maximal plantarflexion did not change significantly after stretching. These data are the first to show a duration-dependent effect of stretch on force (moment) production of the plantarflexors. Further research is required to elucidate the mechanisms responsible as alterations in the stiffness properties of the muscle-tendon complex or muscle recruitment cannot completely explain the changes.

INTRODUCTION

Pre-performance routines involving stretching have often been performed by athletes under the premise that they can minimise the risk of injury and improve performance (Alter, 1996). Prolonged passive stretching results in a decrease in overall musculotendinus stiffness (Avela et al., 1999) and an increase in joint range of motion (ROM), with the increased ROM often associated with a reduction in musculotendinous injury rates (Alter, 1996). More compliant individuals also exhibit reduced soreness and force loss following eccentric exercise (McHugh et al., 1999). However, several reviews of the literature have revealed equivocal results and have not supported the contention of stretching as an injury preventative measure (Gleim & McHugh, 1997; Thacker, 2004; Weldon & Hill, 2003; Witvrouw et al., 2004).

Not only has acute stretching rarely been associated with reduced rates of injury, but significant decrements in force and power production have been reported (Avela et al., 1999, 2004; Brandenburg, 2006; Fowles et al., 2000; Knudson & Noffal, 2005; Kokkonen et al., 1998; Weir et al., 2005). Further research has also revealed stress relaxation (Magnusson, 1998; McHugh et al., 1992), plastic deformation (Taylor et al., 1990), altered neuromuscular activation and reflex patterns (Avela et al., 1999; Fowles et al., 2000) and a decreased sensitivity to pain (Magnusson et al., 1996) after prolonged stretch. Significant decreases were reported by Avela et al. (1999) in maximal isometric plantarflexion torque (23.2%) and maximal EMG (19.9%) after 1-hr of intermittent passive stretching, and Fowles et al. (2000) reported decreases of 28% and 15% in plantarflexor torque and EMG after 30-min of intermittent stretching. These studies initially suggest reduced activation as the mechanism for impaired force production, however, Fowles et al. (2000) also reported EMG fully recovered 1-hr post-stretch while torque remained impaired, suggesting a separate mechanism other than neural partly responsible for torque losses. In agreement with this, Weir et al. (2005) revealed a significant decrease in plantarflexor torque (7%) with no associated change in activation following 10-min static stretch (5 x 120-s).

While current data are predominantly indicative of a negative effect of stretching on force production (Avela et al., 1999, 2004; Brandenburg, 2006; Fowles et al., 2000; Knudson & Noffal, 2005; Kokkonen et al., 1998; Weir et al., 2005), the extensive stretch durations (30-60 min) used in some studies (Avela et al., 1999; Fowles et al., 2000) are not consistent with those used by athletes as part of their

pre-performance routine. Currently, limited data exist describing the effects of stretch duration on muscle force production. Brandenburg (2006) revealed significant decreases in hamstring torque following 15-s and 30-s static stretches but no difference in torque losses between stretching protocols. Alternatively, Knudson & Noffal (2005) revealed a logarithmic decline of torque production following repeated 10-s static stretch with statistically significant differences from the control group appearing after 40-s of stretch. However, it is unclear whether their testing of maximum force intermittently with the stretch influenced their findings. Due to equivocal results in the literature, there is some dispute as to the impact of stretch, and stretch duration, on neuromuscular performance, and therefore whether static stretching should be performed as part of an athlete's pre-performance routine. The present study aimed to determine the effects of the duration of static stretch on peak isometric and passive plantarflexor moments with measurements being done over several test sessions rather than in a single session. The triceps surae muscle group was chosen because of its relative importance in lower-limb power generation during running and jumping movements. Furthermore, the significant muscle size and long tendon makes it highly susceptible to manipulation by stretch.

METHODS

Subjects

Seven active participants (4 men and 3 women; age=25.5+5.3-y, mass=70.4+14.1-kg, height=1.7+0.1-m) with no recent lower limb injury or

illness volunteered for the study. Subjects refrained from intense exercise, flexibility training and stimulant use for 24-hr prior to testing. Ethical approval was granted by the Ethics Committee of the Department of Sport Sciences at Brunel University.

Overview

Subjects completed a full familiarisation of all procedures during this session. Range of motion (ROM) about the ankle joint was also determined using an isokinetic dynamometer (Kin-Com 500-Hz, Chattanooga, Tennessee, USA) by passively rotating the foot until the subject could tolerate no further increase in dorsiflexion. This was then used as the point to which stretches would be performed during testing. Prior to all sessions a five-minute jogging warm up was performed on a treadmill at 8km·hr^{-1} before peak isometric plantarflexion moment, passive moment and hysteresis were measured about the ankle joint. All procedures were completed whilst the subject lay supine with their knee 20° from full extension, the foot was strapped securely to the footplate of the dynamometer and the upper body was firmly secured to the bench.

Over a 5-day period and at the same time of day, subjects randomly performed one of five different stretch protocols: 1) a single 5-s stretch, 2) a single 15-s stretch, 3) four 5-s stretches, 4) four 15-s stretches, or 5) no stretch (control). Two minutes separated pre- and post-testing in all conditions with the stretching being completed 15-s before passive moment was re-tested and 60-s before MVC was re-tested to ensure consistent recovery time for all conditions. Stretching was performed by rotating the footplate at 0.35 rad·s^{-1} until the ankle reached the

maximum ROM as determined previously; it was then held stationary for the allotted time. Statically holding the individual at a constant joint angle is a commonly performed protocol routinely used by individuals to stretch the muscle-tendon complex (Yeh et al., 2007).

Passive moment and hysteresis

Subjects lied supine on the bench such that the centre of rotation of the dynamometer arm during passive and maximum voluntary contraction (MVC) trials was adjacent to the lateral malleolus of the ankle (i.e. the subject was moved slightly during MVC trials so that the ankle was adjacent to the dynamometer's centre of rotation during MVC as the foot deformed and the straps securing the subject stretched slightly). The ankle was then moved through a ROM between 20° of plantarflexion and 20° of dorsiflexion at a velocity of 3.14 $rad \cdot s^{-1}$. Peak isometric ankle moment (PIAM), joint angle and angular velocity were recorded during two trials and saved to a personal computer using Spike2 (v.4) software (CED, England). Passive moment data were smoothed by application of a moving average with a 100-ms averaging window and the peak passive moment was taken as the moment developed at 20° of dorsiflexion. Hysteresis was calculated as the mean area between the loading and unloading curves during the passive trials.

(Figure 1 here)

Peak Isometric Ankle Moment

The PIAM was determined as the maximum ankle moment measured during a 5-s maximal isometric plantarflexion against the footplate with the ankle at 20° dorsiflexion; subjects were instructed to contract the triceps surae maximally. Moment data were smoothed with a moving average (100-ms window).

Muscle activation

An electromyogram (EMG) recording of GM was taken using bi-polar electrodes (30-mm H87V ARBO electrodes, Kendall, Germany) positioned over the central aspect of the muscle belly, parallel to the fascicles of the muscle; a third (ground) electrode was placed on the midline of the patella of the same limb. Prior to electrode placement, the skin was shaved and the skin lightly abraded to reduce skin resistance and the site swabbed with ethanol to remove residual skin cells and oils, and minimise the chance of infection. Electrode sites were marked after testing on day one to ensure precise repeated placement on subsequent testing days. EMG was constantly monitored during passive test trials to ensure muscles were inactive, and during maximum isometric plantarflexion trials to allow quantification of muscle activity. EMG signals collected at a sampling frequency of 1000-Hz were directed to a CED 1902 signal conditioner where they were amplified (1000×), filtered using 20-Hz high-pass and 500-Hz low-pass filters, full-wave rectified and smoothed with a 500 ms moving average by a personal computer running Spike2 (v.4) software. Averaged EMG (aEMG) amplitude, used as a measure of muscle activation, was taken as the peak of the smoothed, rectified EMG curve. The triceps surae have been reported to produce in the range of 72% (Murray et al., 1976) to 93% (Giddings et al., 2000) of joint

moment during plantarflexion, with GM EMG signals being representative of plantarflexion moment (van Zandwijk et al., 2000), thus peak isometric ankle moment was used as an estimated measure of the muscle group's maximal isometric force generating capacity.

(Figure 2 here)

Statistical Analysis

Paired t-tests were used to determine significant differences between pre- and post-intervention data for the control (no stretch) condition. A Multivariate Analysis of Variance (MANOVA) was used to test for differences between dependent variables, with main effects being further analysed by Tukey's post-hoc test. Pearson's Product Moment Correlation Coefficient was used to quantify the linear relationship between stretch duration and the change in moment. All analyses were performed using SPSS statistical software (v.10; LEAD Technologies Inc., USA). Statistical significance was accepted at an alpha level of < 0.05.

RESULTS

(Table 1 here)

There was no change in active or passive moments, hysteresis, or EMG after 2-min complete rest within the no stretch (control) condition ($p>0.05$). A

significant correlation was revealed between losses in peak ankle moment and stretch duration (r=0.68; p<0.01).

(Figure 3 here)

Force losses post-stretch when compared to the control condition were non significant following 5-s (p=0.92), 15-s (p=0.70), four 5-s (p=0.43), and only became significantly different after four 15-s stretches (p=0.001).

(Figure 4 here)

No significant post-stretch change occurred in aEMG during the MVC test (F=0.51; p>0.05). There was a trend toward a decrease in aEMG and further analysis revealed large effect sizes after 15-s (0.75) and four 15-s (1.72); however, these small changes in aEMG did not correspond to the change in peak moment produced in each condition.

(Figure 5 here)

There was a significant reduction in the passive moment measured at 20° of dorsiflexion (F=3.34; p<0.05), which was similar for all stretch conditions compared with the no stretch condition (control).

(Figure 6 here)

Changes in passive moment accounted for only 11.1% of the reduction in peak ankle moment during the four 15-s stretch protocol. The change in passive moment was not accompanied by a change in passive hysteresis (F=0.52; p>0.05), so changes in active and passive plantarflexor moment and hysteresis were not consistent with each other.

DISCUSSION

Substantial data exist showing a decrease in muscle force production after periods of acute static stretch (Avela et al., 1999, 2004; Brandenburg, 2006; Fowles et al., 2000; Knudson & Noffal, 2005; Kokkonen et al., 1998; Weir et al., 2005). Despite this, the effect of stretch duration on the magnitude of force decrement has received little attention. The present research examined the effects of stretch duration on peak plantarflexor moment, passive ankle moment and hysteresis of the triceps surae MTC and joint structures. The main findings were: 1) decreases in peak ankle moment were significantly correlated with stretch duration (r=0.68; p<0.05); 2) peak ankle moment was significantly

reduced (16.7+5.3%) after four 15-s static stretches only compared to the control condition; 3) passive moment was similarly reduced after all stretching protocols compared to the no-stretch condition (20.9+1.3%; $p<0.05$); 4) aEMG and passive hysteresis were unaffected in all stretch conditions compared to the no stretch condition ($p>0.05$).

The reductions in peak ankle moment within the current study are in agreement with Knudson & Noffal (2005) who revealed a logarithmic decline in torque production during a handgrip test following repeated 10-s static stretches. However, in their study torque was measured intermittently between the stretches (i.e. 10-s stretch followed by torque recording), so it is unclear whether the effects were due to the stretch, or a synergistic effect of the stretches with maximum torque contractions. Our data are in agreement with their data, even though our data were collected on separate testing days. The results of both of these examinations clearly suggest a duration-dependant effect of stretch on force losses. Knudson & Noffal (2005) also revealed significant losses only after 40-s of stretch compared to the control group, which is also consistent with findings in the current study. However, a possible limitation of the current study was sample size (seven subjects) which may give rise to a false negative being reported on the significance of the reductions in peak torque in the shorter duration stretches due to large standard deviations. While very large p values reported in these durations (5-s ($p=0.92$) and 15-s ($p=0.70$)) make it unlikely that a false negative has been reported, caution should be taken when proposing that shorter duration stretches had no significant impact on reductions in torque due to limited statistical power. However, we feel it is important to reiterate that the

main aim of the study was to determine whether a dose-response relationship existed, and not when these reductions became significant.

The present data show a decreased resistance to stretch, or increased compliance of joint and/or series elastic (SEC) and parallel elastic (PEC) components. This is particularly interesting since these effects were prominent even though the subjects had completed a comprehensive warm-up, which would have increased tendon compliance (Kubo et al., 2001a), and that of other tissues crossing the joint. Kubo et al. (2001b) and Taylor et al. (1990) have reported an increased tendon compliance post-stretch. In the absence of a reduction of activation, Weir et al. (2005) suggested a mechanical or architectural modification following stretch as a possibility for the reductions observed in force. Thus, if stretching increased compliance of the tendon then the muscle length during MVC would have decreased attenuating force production in accordance with the force-length curves of the soleus and gastrocnemius as reported by Maganaris (2001, 2003). This is in agreement with Nelson et al. (2001) who reported force losses post-stretch only at 162° (i.e. long muscle length) during isometric knee extension close to the ascending limb-plateau region of the force-length curve. However, it is important to note that tendon length was not measured in the present study and mechanisms responsible for these losses cannot be determined or whether these reductions were transitory or remain apparent for some time post-stretch. Therefore, the length of time for which these deficits continued post-stretch, and mechanisms responsible, needs to be investigated to determine their significance to performance.

While the main goal of the present research was to examine performance changes rather than the mechanisms underpinning them, the data offer some insight into possible mechanisms. One possible mechanism hypothesised to explain force losses post-stretch is reduced activation (Avela et al., 1999; Fowles et al., 2000). However, Fowles et al. (2000) reported that EMG recovered fully after 15-min while torque remained impaired 1-hr post-stretch after stretching of the plantarflexors for 30-min. This suggests the recovery of muscle activation may be somewhat independent of the recovery of force generating capacity and importantly indicates mechanisms other than a loss of neural drive must therefore have been partly responsible for these force losses. Interestingly, Weir et al. (2005) reported a significant decrease in plantarflexor torque (7%) with no associated change in activation with relatively shorter duration stretches (5x2-min), which is in agreement with the present study. Furthermore, decreases in passive moment in the present study were not time-dependent, therefore a combination of mechanical and neurological mechanisms may need to be considered for the decreases seen in peak ankle moment, which is consistent with the findings of Fowles et al. (2000). A limitation of the present study was that EMG was recorded from the medial gastrocnemius (GM) and not the lateral gastrocnemius (GL) or soleus (SOL). However, Avela et al. (2004) reported similar reductions in activation from the gastrocnemius and soleus following stretch, which indicates a consistent change in EMG of the plantarflexors, therefore, it could be suggested that GM was largely indicative of the whole triceps surae muscle complex. Nonetheless, we cannot confirm a lack of change of activation in other muscles after the stretch routines.

In summary, our data support the contention that short duration stretches influence the passive properties of the muscle-tendon-joint complex and we report for the first time that the torque decrement after passive stretching of the plantarflexors depends on the duration of the stretches imposed. While time-dependence of stretch has been shown previously in other muscle groups (e.g. Knudson & Noffal, 2005), we tested the effect of each stretch duration on a separate occasion in order to rule out possible synergistic effects of alternating stretches and muscle contractions impacting performance.

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Table/Figure Legends

Table 1. Mean and SD values for passive and peak plantarflexor moments pre- and post-stretch in all stretching conditions.

Stretch protocol	*Passive moment (N.m)*		*Peak moment (N.m)*	
	Pre-stretch	*Post-stretch*	*Pre-stretch*	*Post-stretch*
No Stretch	23.3+6.2	23.8+5.2	215.4+39.3	219.2+41.9
5-s	26.4+10.5	21.9+10.5	220.6+57.8	216.7+52.1
15-s	23.6+7.0	19.5+5.7	213.3+46.3	205.1+51.4
4x5-s	24.2+7.6	20.3+5.4	219.4+48.3	207.1+51.1
4x15-s	21.3+4.9	17.0+4.8	232.0+47.8	193.3+52.8

Figure 1. Loading and unloading curves for passive plantarflexion. During the passive trials on the dynamometer, the ankle joint was moved from 20° of plantarflexion to 20° of dorsiflexion at a velocity of 3.14 rad·s^{-1}. Hysteresis was considered equivalent to the area between the loading and unloading curves.

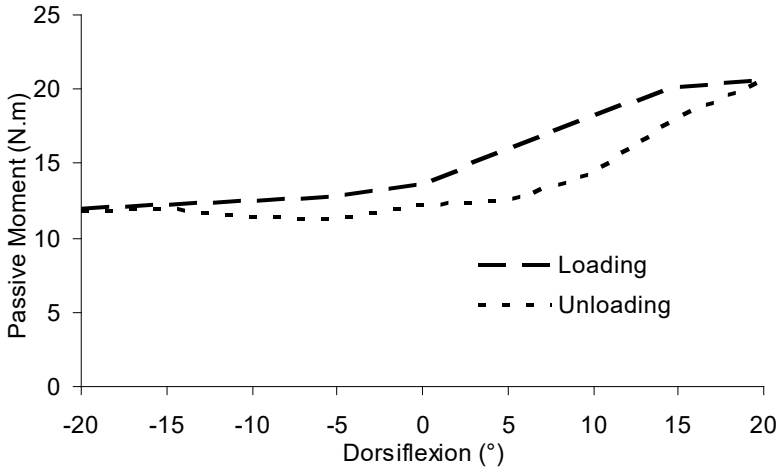

Figure 2. Moment and aEMG recorded during isometric plantarflexion of one subject. Peak ankle moment was used as a measure of maximum triceps surae force production. The peak of the rectified aEMG (500-ms moving average) was taken as a measure of muscle activation.

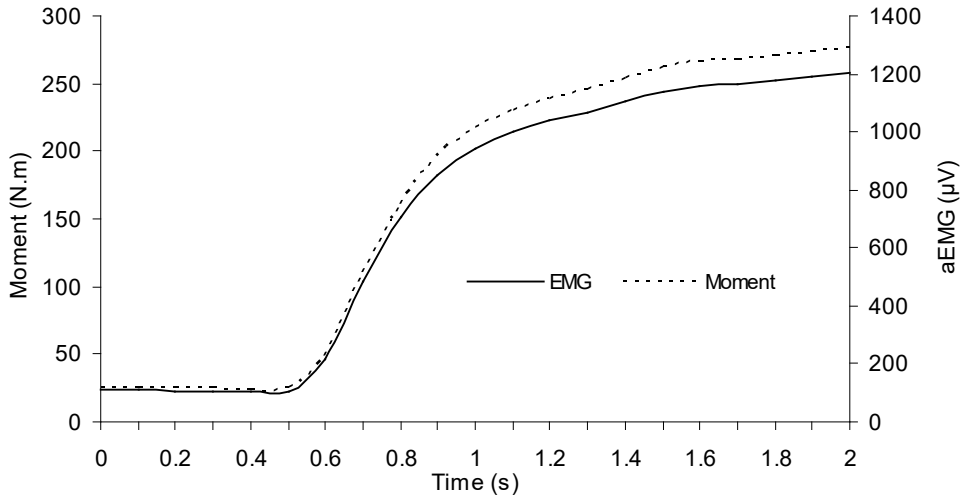

Figure 3. Relationship between stretch duration and mean percentage change in peak ankle moment. A significant correlation was observed between duration of stretch and force decrement (r=0.68; p<0.01).

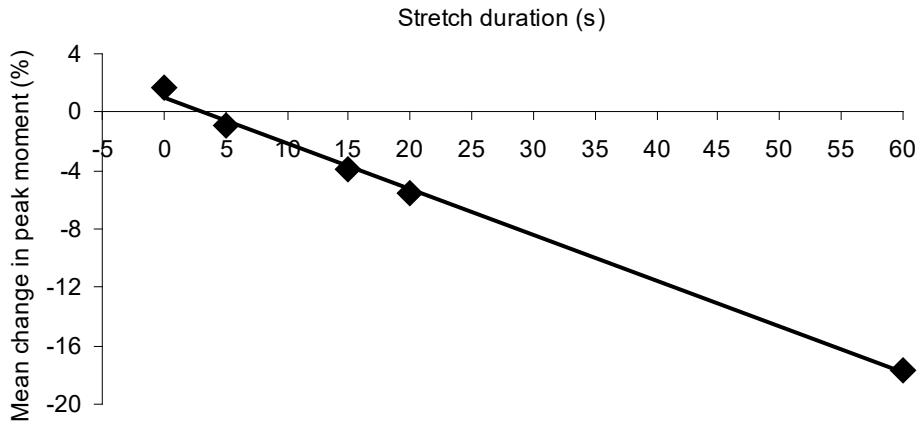

Figure 4. Change in peak moment after stretch (durations indicated). There was a step-wise reduction in moment with increased stretch duration with a significantly reduced moment after four 15-s stretches, compared to the control (no stretch) condition (p<0.05). *Significantly different from control condition.

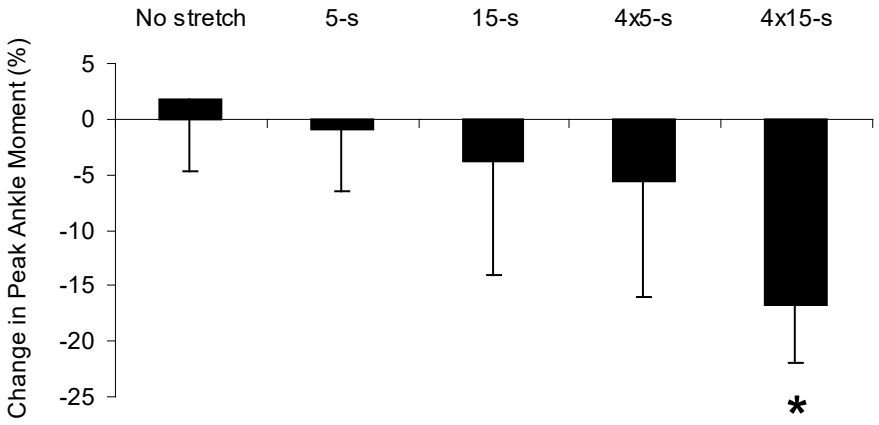

Figure 5. Pre- and post-stretch peak aEMG (durations indicated). There was no significant change in aEMG within any stretch protocol pre to post or when compared to control group (p>0.05).

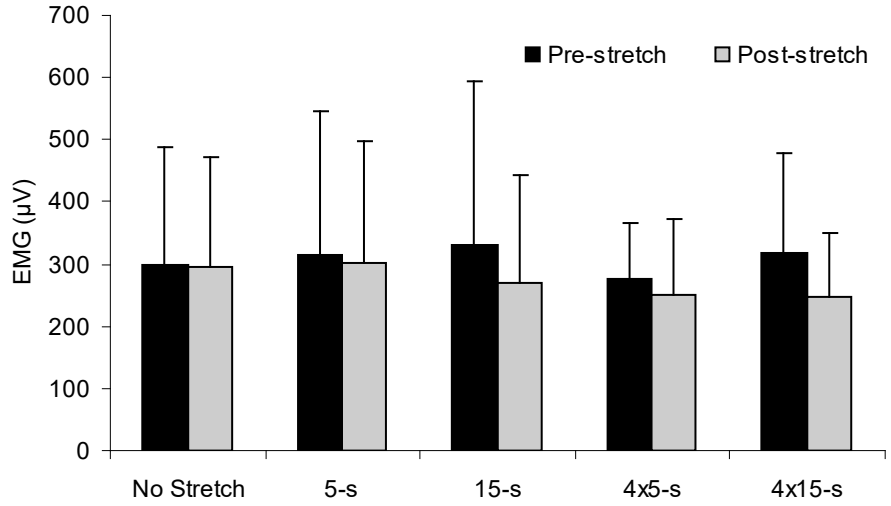

Figure 6. Change in passive moment after stretch (durations indicated). Mean changes were divided by the mean pre-stretch moment data to determine mean (+SD) percent change in moment. There was a significant decrease in passive moment after stretching compared to the control (no-stretch) condition (p<0.05). *Significantly different from control condition.