Moderate-duration static stretch reduces active and passive plantar flexor moment but not Achilles tendon stiffness or active muscle length

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Kay AD, Blazevich AJ. Moderate-duration static stretch reduces active and passive plantar flexor moment but not Achilles tendon stiffness or active muscle length. \textit{J Appl Physiol} 106: 1249–1256, 2009. First published January 29, 2009; doi:10.1152/japplphysiol.91476.2008.—The effects of static stretch on muscle and tendon mechanical properties and muscle activation were studied in fifteen healthy human volunteers. Peak active and passive moment data were recorded during plantar flexion trials on an isokinetic dynamometer. Electromyography (EMG) monitoring of the triceps surae muscles, real-time motion analysis of the lower leg, and ultrasound imaging of the Achilles-medial gastrocnemius muscle-tendon junction were simultaneously conducted. Subjects performed three 60-s static stretches before being retested 2 min and 30 min poststretch. There were three main findings in the present study. First, peak concentric moment was significantly reduced after stretch; 60\% of the deficit recovered 30 min poststretch. This was accompanied by, and correlated with ($r = 0.81$; $P < 0.01$) reductions in peak triceps surae EMG amplitude, which was fully recovered at 30 min poststretch. Second, Achilles tendon length was significantly shorter during the concentric contraction after stretch and at 30 min poststretch; however, no change in tendon stiffness was detected. Third, passive joint moment was significantly reduced after stretch, and this was accompanied by significant reductions in medial gastrocnemius passive muscle stiffness; both measures fully recovered by 30 min poststretch. These data indicate that the stretching protocol used in this study induced losses in concentric moment that were accompanied by, and related to, reductions in neuromuscular activity, but they were not associated with alterations in tendon stiffness or shorter muscle operating length. Reductions in passive moment were associated with reductions in muscle stiffness, whereas tendon mechanics were unaffected by the stretch. Importantly, the impact on mechanical properties and neuromuscular activity was minimal at 30 min poststretch.

triceps surae; force deficits; tissue mechanics; electromyography

PREPERFORMANCE STRETCHING routines, which are commonly used by athletes in the belief that they aid performance and reduce injury risk, have come under increased scrutiny following equivocal support from research as to their influence on injury risk (12, 30, 33, 36, 37) and reports that they induce significant decrements in force and power production (2–10, 13–15, 23, 24, 28–30, 32, 34, 35, 39). Although these force and power reductions have obvious performance implications, the mechanisms underpinning these potential losses are not fully understood. Furthermore, the time course of force decrements and the possible mechanical and physiological changes that cause them have yet to be fully elucidated. Further study is of particular importance for the triceps surae muscle group because of its relative importance in lower limb force and power production during locomotion.

Two primary mechanisms implicated in the stretch-induced force losses include: 1) a reduced neuromuscular activation (2, 3, 10) and 2) changes in the mechanical properties of muscle-tendon complex (MTC) (8–10, 13, 28, 29, 35), which might result in the muscle operating at a different point on its length-tension relation. Reductions in neuromuscular activity [measured by electromyography (EMG)] have been reported simultaneously with stretch-induced force losses after 30 min (10) and 1 h (2) of intermittent static stretching, although the relationship between the changes in EMG and changes in force development has not been explicitly examined. Nonetheless, significant decreases in plantar flexor force have also been reported to occur without a change in neuromuscular activity after 4 × 15 s (13) static stretches and after 5 × 120 s (35) static stretches. Furthermore, Fowles et al. (10) reported the complete recovery of neuromuscular activity 15 min poststretch, whereas plantar flexor moment remained impaired 1 h poststretch. These results are suggestive that a mechanism other than a reduction in neuromuscular activity being at least partly responsible for the force decrements.

An alternate theory is that acute stretching reduces tendon stiffness, which would decrease the active muscle length at a given force level; in the human plantar flexors, the decrease in muscle length would reduce the maximal force according to its force-length relationship (18, 19). A significant reduction in Achilles tendon stiffness has previously been reported after 10 min of static plantar flexor stretch (16). In contrast, 5 min of plantar flexor stretch (27) resulted in Achilles stiffness remaining unaffected while gastrocnemius medialis (GM) muscle stiffness decreased. The intensity of stretch may also impact on the strain experienced by the MTC tissues and any subsequent mechanical changes. Common practice and recommendation is to stretch to the point of discomfort (4, 9, 13, 31); however, this is a highly subjective position, which may impact the findings. Nonetheless, significant differences in study design do not allow a clear determination of the effects of static stretch on tendon stiffness, although both studies reported significant reductions in passive moment, indicating that there were substantive changes in the mechanical properties of the MTC.

Some studies have revealed no change in passive moment following three 45-s stretches (21) or a single 90-s stretch (22), indicating that the increased range of motion (ROM) that

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succeeded the stretch was attributable to an increased “stretch tolerance” rather than any change in the mechanical properties of the tissues. Together, these studies are also suggestive that longer duration stretches (> 5 min) might impact mechanical properties of the MTC, whereas shorter duration stretches might not. Furthermore, some recent studies (13, 14, 30–32, 39) have indicated a clear dose-response effect where reductions in maximal force become more significant with longer stretch durations. Although a number of studies have reported a negative effect of acute stretching on force production (2, 3, 5, 10, 13–15, 23, 24, 30, 32, 34, 35, 39), the extensive stretch durations (10–60 min) used in many studies do not reflect the durations of stretch commonly employed by athletes before performance. Limited data exist describing the effects of stretch on changes in muscle force production, neuromuscular activity, and mechanical properties of the MTC when more commonly used stretch durations (< 3 min) are utilized. Furthermore, few studies have examined the effects of stretch on both active and passive force within the same study protocol, and, to our knowledge, none have included simultaneous neuromuscular activity (EMG) and muscle-tendon imaging protocols to determine the possible contributions of changes in neuromuscular activity and muscle-tendon mechanical properties to the changes in active and passive forces. Given the above, the present study aimed to determine the effects of three 60-s static stretches on active (concentric) and passive plantar flexor force and to determine whether any decrement persisted to 30 min poststretch. Second, we examined neuromuscular activity (EMG) in the triceps surae and correlated the changes in joint moment with changes in EMG amplitude to determine the strength of its relationship. Third, in addition to neuromuscular activity, the present study used a combined approach, where GM muscle operating length and Achilles tendon stiffness were also measured in an attempt to elucidate the relative importance of the mechanisms underpinning possible stretch-induced force losses.

MATERIALS AND METHODS

Subjects

Fifteen active participants (7 women and 8 men; age 20.2 ± 2.4 yr, mass 68.2 ± 13.4 kg, height 1.7 ± 0.1 m) with no recent history of lower limb injury or illness volunteered for the study after providing written and informed consent. No significant difference in stretch-induced force deficits between the sexes (34) has been reported, so the use of men and women should not influence the results. The subjects refrained from intense exercise, flexibility training, and stimulant use of men and women should not influence the results. The subjects induced force deficits between the sexes (34) has been reported, so the research was conducted in accordance with the Declaration of Helsinki.

Overview

The subjects were initially familiarized with the testing protocol 1 wk before data collection. They visited the laboratory on two occasions separated by 1 wk, once under control conditions (no stretch) to determine the reliability of the measures and once for the experimental condition (stretch intervention). The order of testing was randomized to reduce any possible learning effect with 50% undertaking the stretch intervention first and the remaining subjects completing the control condition first. During the experimental sessions, the subjects performed a warm-up on a Monark cycle for 5 min by cycling at 60 revolutions/min with a 1-kg resistance load producing a power output of 60 W. The subjects then sat upright in the chair of an isokinetic dynamometer (Biodex System 3 Pro, IPRS, Suffolk, UK) with knee fully extended (180°), the ankle in neutral position (0°) with the sole of the foot perpendicular to the shank, and the lateral malleolus aligned to the center of rotation of the dynamometer. The subjects’ ankles were passively rotated through their full ROM at 0.087 rad/s (5°/s) before they performed a maximal concentric plantar flexor contraction at 0.087 rad/s through the full ROM. By rotating the ankle (dorsiflexion), three 60-s static plantar flexor stretches were imposed with 60 s of rest after each stretch. They were subsequently retested after stretch (2 min) and at 30 min poststretch.

Stretch Protocol

The subjects’ ankles were passively rotated through their full ROM at 0.087 rad/s until reaching the point of discomfort, a subjective position regularly used in stretch studies (4, 9, 13, 31). The velocity of the movement was too slow to elicit a significant myotatic stretch reflex response (23, 25, 26), ensuring that full ROM was achieved and a substantial stress was applied to the MTC. The subjects were held in the stretched position for 60 s and then released at 0.087 rad/s, returning the foot to a fully plantar-flexed position. The stretch protocol was repeated twice (after 60 s of rest), giving a total stretch duration of 180 s. EMG amplitude, joint moment, ROM, movement velocity, ultrasound imaging of the muscle-tendon junction (MTJ), and video imaging of the lower leg were continuously and synchronously recorded throughout the stretch period, as described below.

EMG Recording

Before electrode placement, the skin was shaved and the skin was lightly abraded to reduce skin resistance. The sites were swabbed with ethanol to remove residual skin cells and oils and to further minimize the small chance of infection. Skin-mounted bipolar double-differentiated active electrodes (model MP-2A, Linton, Norfolk, UK) were positioned over the central portion of the muscle bellies of the medial gastrocnemius (GM), lateral gastrocnemius (GL), soleus (Sol), and tibialis anterior (TA). EMG was constantly monitored during passive trials to ensure that the muscles were inactive and during the maximum concentric plantar flexion trials to allow quantification of muscle activity. EMG signals were amplified (gain 300, input impedance 100 MΩ, common mode rejection ratio 100 dB at 65 Hz) and then directed to a high-level transducer (model HLT100C, Biopac, Goleta, CA) before being converted from analog to digital at a 2,000-Hz sampling rate (model MP150 Data Acquisition, Biopac). Signals were then directed to a personal computer running AcqKnowledge (v3.8.2) software (Biopac) where they were filtered using a 20- to 500-Hz band-pass filter. The filtered signal was finally converted to root mean squared EMG with a 250-ms sample window and normalized as a percentage of the peak amplitude recorded during a maximal isometric contraction. The normalized RMS EMG amplitude was used as a measure of neuromuscular activity; the normalized RMS EMG signals for GM, GL, and Sol were also averaged to obtain a representative activity of the triceps surae (TS) muscle group. The antagonist TA EMG data were processed and normalized using the same method.

Muscle and Tendon Length and Stiffness

Motion analysis. Real-time motion analysis using three ProReflex cameras (Qualisys, UK) operating Track Manager 3D (v.1. 8.226) software recorded the position of infrared reflective markers placed on the Achilles insertion at the calcaneum (see Fig. 1; marker A) and on the origin of the medial head of the gastrocnemius at the medial femoral epicondyle (marker B). Another marker was placed over the GM MTJ (marker C) with adhesive hypoechoic tape placed on the skin aligned with this marker. Data were sampled at 100 Hz and smoothed using a 100-ms moving average. J Appl Physiol • VOL 106 • APRIL 2009 • www.jap.org
Achilles tendon length was estimated as the distance between infrared reflective markers placed on the calcaneus (marker A) and hypoechoic tape (marker B) parallel to the ultrasound probe. MG, gastrocnemius medialis.

Ultrasound. Real-time ultrasound video imaging (LOGIQ Book XP, General Electric, Bedford, UK) using a wide-band linear probe (8L-RS, General Electric) with a 39-mm-wide imaging field of view and coupling gel (Ultrasound gel, Dahlhausen, Cologne, Germany) between the probe and skin was used record the GM-Achilles MTJ position. The probe was orientated along the longitudinal axis of the GM-Achilles MTC. When the location of the MTJ was ascertained, the probe was positioned so that both the superficial and, importantly, the deep aponeurosis between GM and Sol were apparent, ensuring accurate and reliable identification of the MTJ. The probe was affixed with zinc-oxide adhesive tape perpendicular to the skin to maintain a constant position, which ensured consistent imaging of the MTJ and the hypoechoic tape throughout the trial (see Fig. 2). The probe was orientated with the distal end positioned toward the insertion of the tendon and proximal end toward the origin of the medial head. Ultrasound video imaging was sampled at 28 Hz, manually digitized (Peak Motus, Englewood, CO) and smoothed using a 100-ms moving average.

Calculations. Ultrasound, motion analysis, and dynamometer data were synchronized using a 5-V ascending transistor-transistor logic pulse, which placed a marker on the ROM data that was exported to the AcqKnowledge (v3.8.2) software and triggered the capture of ultrasound data. A time-line regression to a specific ROM could then be determined using the marker on the ROM trace and the last image on the ultrasound recording. Tendon length was calculated as the distance between reflective markers A (Achilles insertion) and C (marker aligned with hypoechoic tape over estimated MTJ position), minus the distance from actual MTJ position (determined with ultrasound) to the hypoechoic tape (appearing as a black line on the ultrasound image), according to Fig. 2. GM muscle length was calculated as the distance between reflective markers B (GM origin on the medial femoral epicondyle) and C (marker aligned with hypoechoic tape over estimated MTJ position), plus the distance from actual MTJ position (determined with ultrasound) to the hypoechoic tape (appearing as a black line on the ultrasound image). Tendon stiffness was calculated by dividing tendon length change by the change in ankle moment during concentric trials, and GM muscle stiffness was calculated by dividing GM muscle length change by ankle moment change during passive trials. Moment arms were not determined in the present study because it was nonchanging and would not have influenced the results; thus tendon “force” (joint moment per moment arm) was not derived.

Passive ankle moment. The subjects were reclined to 55° at the hip to enable a full ROM about the ankle to be attained; initial testing showed that subjects felt substantial pain at the back of the knee and a lesser ankle ROM when the subjects were seated with a greater hip flexion angle (85 and 70°). The subjects’ ankles were passively rotated through their full ROM at 0.087 rad/s until reaching the point of discomfort; this is a stretch intensity regularly utilized in stretch studies (4, 9, 13, 31). The subjects volitionally terminated the stretch by pressing a handheld release button. EMG was constantly monitored throughout the passive trials to ensure the TS were inactive. Passive moment was recorded throughout the trial and then normalized as a percentage of the maximum prestretch passive joint moment. The normalized moment data at 10, 30, 50, 70, and 90% of maximum ROM were used for analysis so as to account for interindividual differences in joint flexibility/ROM. Full ROM was calculated from the inflection point (mean angle = 0.8 ± 4.8° dorsiflexion), where a clear change in the slope of the passive moment curve occurred (see Fig. 3), to the volitional end of the ROM.

Concentric plantar flexor ankle moment. The subjects’ ankles were passively rotated through their ROM at 0.087 rad/s until reaching the point of discomfort before they maximally contracted the plantar flexors isometrically. The footplate of the dynamometer was released once maximal isometric moment was attained (i.e., there was a visible plateau in the moment trace), which enabled the subjects to continue to maximally contract the plantar flexors through a concentric plantar flexion movement at 0.087 rad/s through their full ROM. During familiarization, the subjects’ moment data were observed during contraction, and trials were repeated until they could consistently achieve a plateau during isometric phase and a linear reduction during the concentric phase (see Fig. 4). This was considered to be indicative
Coefficients of variation (expressed as a percentage of the mean) ranged from 0.3 to 0.4%; no significant difference was detected between mean values (P > 0.05). Test-retest reliability for concentric moment, passive moment, muscle length, and tendon length was conducted on 15 subjects within the control condition. The ICCs ranged from 0.79 to 0.90 and from 0.83 to 0.99, 0.99, and 0.99, respectively. Coefficients of variation ranged from 2.3 to 3.2%, from 1.8 to 5.2%, and from 0.2 and 0.4 to 0.5%, respectively.

RESULTS

There was no change in active or passive joint moment, EMG amplitude, tendon and muscle length, or muscle and tendon stiffness after 5 min of rest (control condition; P > 0.05). Within the stretching condition, however, there were significant decreases in peak concentric moment (mean = 5.0 ± 0.3%) measured at 50% (5.2%; P < 0.05), 70% (4.6%; P < 0.05), and 90% (5.0%; P < 0.01) of ROM (see Fig. 5). Post hoc t-tests revealed that all these decreases occurred after the stretch, but there was no significant difference detected at 30 min poststretch.

A decrease in peak EMG amplitude was observed after stretch in GM measured at 70% (10.8%; P < 0.05) and 90% (16.4%; P < 0.01) of ROM, and a significant reduction was seen in GM at 50% ROM (11.1%; P < 0.05). TS activity (mean EMG in GL, GM, and Sol) was also significantly reduced at 90% (9.2%; P < 0.05) of ROM, and statistical significance was almost obtained at 50% (P = 0.053) and 70% (P = 0.066) of ROM (see Fig. 6) with effect sizes of 0.62 and 0.46, respectively. Post hoc t-tests revealed that all these decreases occurred after the stretch and had all significantly increased at 30 min poststretch to, or above, prestretch levels, indicating that EMG had fully recovered.

The Pearson’s product moment correlations computed between changes in triceps surae EMG and decreases in joint moment were significant at 50% (r = 0.81; P < 0.01) and 70% (r = 0.65; P < 0.05) of ROM, indicating that the subjects who had the greater reductions in EMG tended also to exhibit the greatest loss of active joint moment (see Fig. 7). These significant correlations were also seen in all three plantar flexor muscles individually at 50 and 70% of ROM (data not shown). However, the strongest correlations were seen in TS EMG, suggesting that averaging of the individual EMG signals to create a single EMG representative of the muscle group activation produced stronger relationships with moment deficits. Repeated-measures ANOVA revealed no significant differences in the changes in EMG between each of the plantar flexor muscles.

Intratester reliability for the manual digitizing of ultrasound MTJ position was conducted on five subjects’ passive trials. The intraclass correlation coefficients (ICCs) ranged from 0.98 to 0.99 and coefficients of variation (expressed as a percentage of the mean) ranged from 0.3 to 0.4%; no significant difference was detected between mean values (P > 0.05). Test-retest reliability for concentric moment, passive moment, muscle length, and tendon length was conducted on 15 subjects within the control condition. The ICCs ranged from 0.79 to 0.90 and from 0.83 to 0.99, 0.99, and 0.99, respectively. Coefficients of variation ranged from 2.3 to 3.2%, from 1.8 to 5.2%, and from 0.2 and 0.4 to 0.5%, respectively.

Data Analysis

All data were analyzed using SPSS statistical software (v.11.5, LEAD Technologies, Chicago, IL); all data reported are means ± SE. Separate ANOVA with repeated measures were used to test for differences in 1) peak concentric and passive plantar flexor moment, 2) peak TS amplitude (EMG), 3) change in peak EMG amplitude between muscles poststretch, 4) muscle and tendon length, and 5) muscle and tendon stiffness. Post hoc t-tests were used to further examine changes in measures where statistical significance was reached. Pearson’s product-moment correlation was used to determine relationship between poststretch reductions in moment and changes in EMG. Statistical significance for all tests was accepted at P < 0.05.

Reliability

Fig. 4. Joint moment recorded during a concentric plantar flexion trial. The subjects performed an isometric contraction (marker A) until a plateau was reached (marker B), and then the footplate of the dynamometer was released and a concentric contraction (marker C) was performed through the full ROM. Moment data were then normalized as a percentage of the prestretch maximum plantar flexor moment during the maximum voluntary isometric contraction (%MVIC). Full ROM was converted to a percentage with 100% being full dorsiflexion and 0% being full plantar flexion, and moment data were analyzed at 50, 70, and 90% of ROM.

of a consistent maximal contraction, with the linear decrease representative of the force-length characteristics of the TS (18, 19). Concentric plantar flexor moment was normalized as a percentage of the prestretch maximum plantar flexor moment during the maximum voluntary isometric contraction. Again, since subject flexibility was variable, maximal concentric moment was recorded throughout the full ROM but data were analyzed only at 50, 70, and 90% of the full ROM, calculated between full plantar flexion (0%) and full dorsiflexion (100%). Analysis was not conducted at 10 or 30% of ROM because the slow concentric velocity (5%‘s) used resulted in a total contraction period of ~12 s, and this may have significantly influenced the results at these joint angles. To ensure that the centre of rotation of the ankle remained in line with the center of rotation of the dynamometer, the subjects were inclined at the hip to 85°; initial testing revealed that lower inclinations resulted in a visibly greater degree of ankle movement in the footplate, hip elevation, and sliding of the upper body in the dynamometer seat. During testing, joint moment, joint angle, and angular velocity data for both passive and active trials were directed from the dynamometer to a high-level transducer (model HLT100C, Biopac) before analog-to-digital conversion at a 2,000-Hz sampling rate (model MP150 Data Acquisition, Biopac). The data were then directed to a personal computer running AcqKnowledge software (v3.8.2, Biopac) and filtered with a double-pass 6-Hz Butterworth low-pass filter.

Fig. 5. Normalized maximal joint moment recorded during concentric plantar flexion trials. *Significant decreases (P < 0.05) in plantar flexor joint moment were observed at 50, 70, and 90% of ROM after stretch (*).
There were three major findings of the present study. First, there were significant reductions in peak concentric joint moment measured at 50, 70, and 90% of ROM after stretch (mean = 5.0 ± 0.3%), which were accompanied by, and correlated with (r = 0.81; P < 0.01), significant reductions (90% of ROM) in EMG amplitude (mean = 8.3 ± 1.0%). Concentric moment partially recovered (59.0 ± 12.6%), whereas EMG fully recovered after 30 min of rest. Second, Achilles tendon length during the concentric contractions was significantly reduced (mean = 1.4 ± 0.1%) and muscle operating length was significantly increased poststretch, but there were no significant differences at 10 or 30% of ROM.

DISCUSSION

There was a significant decrease detected immediately poststretch in tendon length (mean = 1.4 ± 0.1%) measured during the concentric contraction during the active trials at 50% (1.3%; P < 0.05), 70% (1.5%; P < 0.05), and 90% (1.5%; P < 0.05) of ROM (see Fig. 8). This was accompanied by a significant increase in muscle operating length (1.0 ± 0.1%) at 50% (1.0%; P < 0.05), 70% (0.9%; P < 0.05) and 90% (1.0%; P < 0.05) of ROM. Post hoc t-tests revealed that the poststretch reduction in tendon length and increase in muscle operating length remained apparent 30 min poststretch compared with prestretch data. However, no significant differences were observed at any joint angle in tendon stiffness (P > 0.05).

There were significant decreases in passive joint moment (mean = 5.1 ± 1.6%) measured during the passive trials at 50% (4.0%; P < 0.05), 70% (6.9%; P < 0.01) and 90% (4.3%; P < 0.01) of ROM (see Fig. 9) but no significant differences at 10 or 30% of ROM. Post hoc t-tests revealed significant reductions after stretch and subsequent, significant increases 30 min later compared with poststretch data, such that muscle stiffness returned to prestretch levels (see Fig. 10).

There were significant reductions in muscular cocontraction at 50, 70, and 90% of ROM after stretch (mean = 5.5 ± 1.8%) at 50% (4.3%; P < 0.01), 70% (7.6%; P < 0.01), and 90% (4.7%; P < 0.01) of ROM, but no significant differences were seen at 10 or 30% of ROM. Post hoc t-tests revealed significant reductions after stretch compared with prestretch data, and they revealed subsequent, significant increases 30 min later compared with poststretch data, such that muscle stiffness returned to prestretch levels (see Fig. 10).

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Fig. 6. Normalized triceps surae electromyograph (EMG) recorded during concentric plantarflexion trials. Significant decreases in EMG amplitude (P < 0.05) were observed at 90% of ROM after stretch (*), and statistical significance was almost obtained at 50% (P = 0.053) and 70% (P = 0.066) of ROM.

Fig. 7. Correlation between changes (decreases) in active joint moment and changes in triceps surae EMG amplitude. Significant correlations (P < 0.01) were observed at 50 and 70% ROM between poststretch joint moment reductions and mean plantar flexor EMG [i.e., average of lateral gastrocnemius, gastrocnemius medialis (GM), and Sol].

Fig. 8. Achilles tendon length recorded during concentric plantar flexion trials. Significant decreases in tendon length (P < 0.05) were observed at 50, 70, and 90% ROM after stretch (*) and at 30 min poststretch (*).

Fig. 9. Normalized passive joint moment recorded during passive dorsiflexion trials. Significant decreases (*) in passive joint moment were observed at 50, 70, and 90% ROM after stretch (P < 0.05); no significant reductions were observed at 10 or 30% of ROM.

mm, poststretch = 174.6 ± 7.1 mm) or GM muscle length (prestretch = 273.8 ± 6.7 mm, poststretch = 274.6 ± 7.0 mm) at any ROM during the passive trials, although consistent trends at all joint angles (indicating longer muscle and shorter tendon length) were apparent. GM muscle stiffness was significantly reduced (mean = 5.5 ± 1.8%) at 50% (4.3%; P < 0.01), 70% (7.6%; P < 0.01), and 90% (4.7%; P < 0.01) of ROM, but no significant differences were seen at 10 or 30% of ROM. Post hoc t-tests revealed significant reductions after stretch compared with prestretch data, and they revealed subsequent, significant increases 30 min later compared with poststretch data, such that muscle stiffness returned to prestretch levels (see Fig. 10).

POSTSTRETCH FORCE LOSSES

There were significant correlations (r = 0.81; P < 0.01), significant reductions (90% of ROM) in EMG amplitude (mean = 8.3 ± 1.0%), concentric moment partially recovered (59.0 ± 12.6%), whereas EMG fully recovered after 30 min of rest. Second, Achilles tendon length during the concentric contractions was significantly reduced (mean = 1.4 ± 0.1%) and muscle operating length was significantly increased poststretch, but there were no significant differences at 10 or 30% of ROM.

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POSTSTRETCH FORCE LOSSES

There were three major findings of the present study. First, there were significant reductions in peak concentric joint moment measured at 50, 70, and 90% of ROM after stretch (mean = 5.0 ± 0.3%), which were accompanied by, and correlated with (r = 0.81; P < 0.01), significant reductions (90% of ROM) in EMG amplitude (mean = 8.3 ± 1.0%). Concentric moment partially recovered (59.0 ± 12.6%), whereas EMG fully recovered after 30 min of rest. Second, Achilles tendon length during the concentric contractions was significantly reduced (mean = 1.4 ± 0.1%) and muscle operating length was significantly increased poststretch, but there were no significant differences at 10 or 30% of ROM.
was no significant change in tendon stiffness. This indicates that the reduced tendon length resulted from a decrease in muscle force despite the muscle operating closer to the plateau of its force-length curve. Third, there were significant decreases in passive joint moment poststretch at 50, 70, and 90% of ROM (mean = 5.0 ± 1.2%), which were accompanied by significant reductions in GM muscle stiffness (measured at those joint angles; mean = 5.5 ± 1.8%), but there was no detectable change in tendon stiffness. Both passive joint moment and muscle stiffness returned to baseline levels after 30 min of rest. Despite these changes being seen at the most dorsiflexed joint positions, there were no significant reductions in passive joint moment, Achilles tendon stiffness, GM muscle length, or Achilles tendon length during the passive trials at 10 or 30% of ROM.

The consistent and significant reductions (P < 0.05) in active concentric plantar flexor moment and peak EMG amplitude through a range of joint angles (50, 70, and 90%) are consistent with previous reports of decreases in muscle force and EMG amplitude after acute periods of static stretch (2–10, 14, 28, 29, 35). In fact, the peak plantar flexor moment and triceps surae EMG decreases were well correlated (r = 0.81; P < 0.01), which is indicative of a reduced muscle activity being partly responsible for the losses in force production; over 65% of the variability in moment changes can be explained by the changes in EMG amplitude. This reduction in EMG might indicate a decrease in central neural drive (11) or peripheral inhibition of the a-motoneuron pool by group I muscle afferents (2). However, the underlying mechanism(s) implicated with stretch-induced reductions in EMG remains unclear and needs to be examined in more detail in future studies. The 5% force decrease seen in the present study is less than has been reported previously (7–23%; Refs. 2, 3, 10, 24, 34, 35). One difference between this study and others is that a shorter stretch duration was imposed presently, which may have limited the force losses according to the well-known dose-response relationship (13, 14, 30–32, 39). Interestingly, the significant reduction in force was not present after 30 min of passive rest (59.0 ± 12.6% recovery) with EMG fully recovered to, or slightly above, prestretch levels. Fowles et al. (10) reported similar recovery patterns after 30 min of passive rest (43% recovery in joint moment; full recovery in EMG). These different temporal responses of muscle force and EMG are indicative of other mechanisms being at least partly responsible for force losses. For example, the decrease in muscle stiffness may indicate microtrauma to the cytoskeleton, which might possibly affect the excitation-contraction coupling process (1, 6, 17, 38), however, this remains speculative and further research is required to determine whether a muscle based component is associated with stretch-induced force deficits.

The second main finding of the present study was that there was no evidence for a change in tendon stiffness. Decreased tendon stiffness has been suggested to be a possible mechanism for reduced moment poststretch (13) as this would decrease muscle length at a given joint angle for the plantar flexors, as the leftward shift in its force-length curve would cause it to operate further down its ascending limb (18, 19). In fact, we found that tendon elongation was significantly reduced at each joint angle (50, 70, and 90% of ROM) when measured during concentric contraction and that it occurred simultaneously with a reduced joint moment. Thus the muscle operated at a longer, and presumably stronger, length after stretch, yet force production was reduced. Our findings of a lack of change in tendon stiffness are consistent with those of Morse et al. (27), who found no change in tendon stiffness following 5 min stretch, but they are not in agreement with Kubo et al. (16), who reported decreases in tendon stiffness after 10 min of stretch (16). These different results might indicate a dose-response effect of stretch on changes to tendon stiffness or may be reflective of differing methods of measuring tendon stiffness [e.g., Morse et al. (27) measured tendon elongation at the MTJ, whereas Kubo et al. (16) measured at the fascicle insertion onto the deep aponeurosis]. Although alterations in muscle and tendon stiffness have also been hypothesized to contribute to the stretch-induced force losses (8–10, 13, 28, 29, 35), no studies had previously measured muscle or tendon length changes post-stretch to determine whether a more compliant tendon would result in the muscle operating at a shorter length. The present study is the first to remove this as a mechanism of stretch-induced force deficits. Although muscle operating length did change, it actually increased, which should have increased joint moment according to its force-length properties (18, 19). Given that a decrease in muscle force was not caused by a reduction in muscle operating length, that it cannot be fully explained by decreases in EMG, and that there was a clear change in muscle mechanical properties, we speculate that a muscle (perhaps sarcomere)-based mechanism is present. Further research is required to more clearly determine the mechanisms responsible for reductions in muscle force after stretch.

The third main finding of the present study was that the significant reduction in passive moment was accompanied by significant reductions in GM muscle stiffness, measured at 50, 70, and 90% of ROM. The reductions in passive joint moment are consistent with those reported by others where stretch duration exceeded 5 min (16, 20) and those of Morse et al. (27), who reported similar decrements in passive joint moment accompanied by reduced muscle stiffness but unaltered tendon stiffness. However, studies imposing shorter durations of stretch (90–135 s; Refs. 21, 22) have reported no change in passive joint moment. Collectively, these findings are indicative of a dose response where only longer durations of stretch (>3 min) consistently reduce passive moment.

Interestingly, no significant reductions in passive moment were found at 10 or 30% of ROM. This might be due to passive moment reliability (measured in the control group) being lower at
these angles; coefficients of variation increased from 1.9% at 50% of ROM to 5.3% at 10% of ROM, under these conditions statistical significance may be more difficult to detect. Alternatively, the data might indicate that the effects of stretch are muscle length dependent, with decreases occurring only at longer lengths. The measurement of joint moment at several joint angles and the determination of the inflection point may be important methodological considerations, as analysis of passive joint moment at planar-flexed ROMs (i.e., below the inflection point) could produce unreliable data and mask the effects of stretch.

In summary, the present study is the first to specifically compare the effects of stretch on both active and passive joint moment through a range of joint angles, including analysis of neuromuscular and mechanical mechanisms that are implicated in the stretch-induced force deficits. Reductions in peak concentric moment were correlated with reduced muscle EMG indicating that a significant proportion of the reduction in force (~65%) can be explained by muscle activity. Possible reductions in muscle operating length resulting from a decrease in tendon stiffness were plainly not a factor. Clearly, an as yet unidentified mechanism must also impact on force production after stretch. The reductions in passive moment were accompanied by reductions in muscle stiffness, while tendon stiffness remained unaffected by the stretch, which is in agreement with previous data (27) showing that increased joint flexion can be attributed to decreased muscle stiffness. Importantly, no significant deficits in active and passive joint moment, EMG amplitude and muscle stiffness were found 30 min poststretch, suggesting the effects of these shorter duration stretches (3 min in total) were transient. Therefore, the performance of physical tasks requiring high levels of plantar flexor muscle force is unlikely to be compromised following moderate duration passive stretch, and the previously reported negative impact of stretch on force production might be of lesser practical importance when tasks are performed at a reasonable time period (~30 min) after the stretch.

REFERENCES


