Concentric muscle contractions before static stretching minimize, but do not remove, stretch-induced force deficits

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Kay AD, Blazevich AJ. Concentric muscle contractions before static stretching minimize, but do not remove, stretch-induced force deficits. \textit{J Appl Physiol} 108: 637–645, 2010. First published January 14, 2010; doi:10.1152/japplphysiol.01135.2009.—The effects of concentric contractions and passive stretching on musculotendinous stiffness and muscle activity were studied in 18 healthy human volunteers. Passive and concentric plantar flexor joint moment data were recorded on an isokinetic dynamometer with simultaneous electromyogram (EMG) monitoring of the triceps surae, real-time motion analysis of the lower leg, and ultrasound imaging of the Achilles-medial gastrocnemius muscle-tendon junction. The subjects then performed six 8-s ramped maximal voluntary concentric contractions before repeating both the passive and concentric trials. Concentric moment was significantly reduced (6.6%; \( P < 0.01 \)), which was accompanied by, and correlated with \(( r = 0.60–0.94; P < 0.05)\), significant reductions in peak triceps surae EMG amplitude (10.2%; \( P < 0.01 \)). Achilles tendon stiffness was significantly reduced (11.7%; \( P < 0.01 \)), but no change in gastrocnemius medialis muscle operating length was detected. The subjects then performed three 60-s static plantar flexor stretches before being retested 2 and 30 min poststretch. A further reduction in concentric joint moment (5.8%; \( P < 0.01 \)) was detected poststretch at 90\% of range of motion, with no decrease in muscle activity or Achilles tendon stiffness, but a significant increase in muscle operating length and decrease in tendon length was apparent at this range of motion \(( P < 0.05)\). Thirty minutes after stretching, muscle activity significantly recovered to pre-maximal voluntary concentric contractions levels, whereas concentric moment and Achilles tendon stiffness remained depressed. These data show that the performance of maximal concentric contractions can substantially reduce neuromuscular activity and muscle force, but this does not prevent a further stretch-induced loss in active plantar flexor joint moment. Importantly, the different temporal changes in EMG and concentric joint moment indicate that a muscle-based mechanism was likely responsible for the force losses poststretch.

triceps surae; joint moment; tendon stiffness; electromyography

WARM-UP ROUTINES THAT ARE performed before intense physical activity commonly include cardiovascular work, muscle stretching, and progressively intense muscular contractions (1, 2, 8, 55). The performance of maximal isometric contractions has been shown to reduce tendon stiffness (31, 33) and to potentiate muscle force (6, 44), although this effect is inconsistent (12, 25, 27). The stretching routines are primarily employed to reduce musculotendinous stiffness and to increase functional range of motion (ROM) (2) in an attempt to reduce injury (2, 8, 55). McHugh et al. (39) revealed that lower levels of flexibility were found in those individuals who exhibited greater pain, strength loss, and creatine kinase activity after the eccentric exercise, indicating that those with a stiffer muscle-tendon complex (MTC) may be at greater risk of muscle-strain injury. Paradoxically, a highly compliant MTC may increase joint instability and increase the risk of injury to structures within the joint complex (54). This has prompted speculation that there may be an optimal stiffness for performance enhancement, while minimizing the risk of injury (11). Accordingly, there is still some debate as to the efficacy of stretching (24, 50, 53).

Nonetheless, significant reductions in force and power production have also been reported following passive stretching (29, 30, 48, 51). Such a stretch-induced decrease in muscle force might substantially compromise neuromuscular, and therefore movement, performance. So the development of methods to mitigate or remove this effect is an important goal for practitioners who are invested in optimizing performance while reducing injury risk. A possible mechanism underpinning the decrease in force after stretching is reduced neuromuscular activity [electromyography (EMG)], which has been reported to occur concurrently with (4, 16, 22, 30), and be correlated with, reductions in force (30). However, given that the changes in force could not be fully explained by the changes in neuromuscular activity (30) and that losses in force have also been reported in the absence of any change in EMG (15, 29, 52), other mechanisms must be at least partially responsible for the force deficits.

Poststretch reductions in passive moment (29, 30, 42), indicative of changes in the mechanical properties of the MTC, could be a second mechanism. Examining the influence of interventions on the mechanical properties of the tendon is important, as decreased tendon stiffness may reduce the rate of force development (9, 19, 33) and active muscle length (33, 37) and increase neuromechanical delay (18, 26). However, Kay and Blazevich (30) and Morse et al. (42) reported that moderate-duration stretches (3 and 5 min, respectively) did not affect Achilles tendon stiffness or reduce muscle operating length, which could attenuate maximal force in the human plantar flexors, according to its force-length relationship (35, 36). In contrast, Kubo et al. (32) reported a significant decrease in Achilles tendon stiffness and muscle operating length after 10 min of static plantar flexor stretch, although this was not associated with a reduced isometric force output. Interestingly, similar reductions in tendon stiffness have been reported following repeated isometric contractions (33, 37). Collectively, these results suggest that the duration and intensity of tissue strain imposed by either stretching or strong muscular contractions may determine whether changes in tendon stiffness occur.
In a recent study (31), it was shown that a series of maximal voluntary isometric contractions (MVICs) completely removed the effects of subsequent passive stretch on muscle-tendon mechanical properties, neuromuscular activity (EMG), and force production in the plantar flexors. This is despite the stretch protocol being identical to one that had previously resulted in significant changes in these measures (30). However, this apparent prophylactic effect occurred largely because the isometric contractions themselves had a detrimental effect on concentric force and neuromuscular activity. Consequently, the overall performance decrement from both the muscular contractions and stretch was similar to that found with passive stretching alone (31). These changes are very similar to those that have been reported, or hypothesized, to occur after acute passive stretching (5, 14, 16, 22). This is suggestive of stretching and isometric contractions having similar mechanical and neurophysiological effects on the MTC, which may explain apparent prophylactic effect of the isometric contractions on subsequent stretch. The significant changes seen after the performance of isometric contractions also suggest that the intervention may not be a useful preperformance strategy, even though it mitigates subsequent force deficits in response to stretch.

Although concentric force was decreased following the MVIC intervention, an encouraging finding was that the effects of the MVIC intervention were contraction mode dependent, as no change in isometric force was evident (31). Given that any mechanical changes in the MTC would have been expected to influence both isometric and concentric force production, and that EMG was only reduced in the concentric trial, the effect of MVICs on neuromuscular activity appears also to be contraction mode dependent. Furthermore, if peripheral mechanisms were responsible for the reductions in EMG (4), a decrease in isometric and concentric force would again be expected. These arguments leave central mechanisms (16, 23, 31) as being most likely to underpin the reduction in neuromuscular activity during the concentric contraction mode. Thus the possibility exists that contractions of a different mode (e.g., concentric rather than isometric) might mitigate the effects of a subsequent period of passive stretch without first compromising neuromuscular activity and muscle force production.

The negative effects of stretch on force production are widely reported (29, 30, 38, 43); however, the examination of stretch in isolation may limit the external validity of many of these studies to preperformance routines. Our previous study (31) revealed that the negative effects of stretch on force production could be removed, if stretch was performed after a series of isometric contractions. However, these contractions induced similar force deficits to those observed following stretch in isolation (30); therefore, the prophylactic benefit is not practically useful. Concentric contractions are commonly performed by athletic and clinical populations before intense physical activity, yet, surprisingly, there are no data available to determine whether they have an influence on the commonly described stretch-induced force reduction. Measuring neuromuscular changes in response to stretch subsequent to the application of an intervention that had measurable effects on the MTC would be an effective paradigm for elucidating the mechanisms that underpin the stretch-induced force deficit phenomenon. The results of such an examination would also inform best practice as to the design of research studies and optimize the structure of preperformance routines, since concentric actions are commonly performed during warm-up activities before strength testing. Thus the aim of the present study was to examine the effects of a series of maximal concentric plantar flexor contractions and a subsequent period of static muscle stretching on Achilles tendon stiffness, gastrocnemius medialis muscle operating length, active (concentric) and passive ankle joint moment, and neuromuscular (EMG) activity in the triceps surae (TS) MTC.

**MATERIALS AND METHODS**

**Subjects**

Eighteen active participants (9 women and 9 men; age = 21.0 ± 3.3 yr, mass = 73.1 ± 16.4 kg, height = 1.7 ± 0.1 m, body mass index < 27 kg/m²), with no recent history of lower limb injury or illness, volunteered for the study after completing a pretest medical questionnaire and giving written and informed consent. A priori power analyses based on previous research (14, 29, 32, 42) suggested that 15 participants were needed to achieve statistical power (0.8). The subjects were asked to refrain from intense exercise, flexibility training, and stimulant use for 48 h before testing. Ethical approval was granted by the Ethics Committee’s of The School of Sport and Education at Brunel University and The School of Health at The University of Northampton, in accordance with the declaration of Helsinki.

**Protocol**

**Overview.** The subjects were initially familiarized with the testing protocol 1 wk before data collection. During the experimental sessions, the subjects performed a warm-up on a Monark cycle for 5 min at 60 revolutions/min with a 1-kg resistance load, producing a constant 60-W power output. The subjects were then seated in the chair of an isokinetic dynamometer (Biodex System 3 Pro, IPRS, Suffok, UK) with the knee fully extended (0°), ensuring that the gastrocnemii contributed significantly to plantar flexor joint moment during contraction (18), and were placed under significant stress during the stretch protocol (28). The ankle was placed in the neutral position (0°) with the sole of the foot perpendicular to the shank, and positioned in the dynamometer footplate with the lateral malleolus aligned to the center of rotation of the dynamometer. The subjects’ ankles were passively dorsiflexed through their full ROMs at 0.087 rad/s (5°/s), before they performed a maximal concentric plantar flexor contraction at 0.087 rad/s through their full ROM. The concentric intervention was then performed, which required the subjects to perform a series of six ramped maximal voluntary concentric plantar flexor contractions (MVCCs). Two minutes after completing the intervention, the passive and active trials were repeated to determine whether there were any effects of the concentric contractions. Two minutes after the subject completing the active trial, the subjects’ ankles were rotated (dorsiflexion) at 0.087 rad/s to stretch the plantar flexors; three 60-s static plantar flexor stretches (as described below) were imposed with 60 s of rest after each stretch. The passive and active trials were then repeated 2 min and 30 min poststretch to determine the impact of stretch. The order and time of the experimental protocol, including the passive and concentric trials and concentric and stretch interventions, is depicted in Fig. 1.

**Passive ankle moment.** The subjects were seated in the chair of an isokinetic dynamometer (Biodex System 3 Pro, IPRS) with the hip flexed to 55°, the knee fully extended (0°), and the unshod foot strapped into the dynamometer footplate, as previously described (30, 31). The subjects’ ankles were passively rotated at 0.087 rad/s through their full ROM; the subjects voluntarily terminated the rotation at the point of discomfort by pressing a hand-held release button. EMG was constantly monitored throughout the passive trials to ensure the TS...
were inactive (see EMG recording below). Passive moment was recorded throughout the trial and then normalized to the maximum passive joint moment obtained in the first trial (%Mpas). To account for interindividual differences in joint flexibility/ROM, moment data were analyzed at 50, 70, and 90% of maximum ROM. Data were not analyzed at joint angles <50% of ROM, as passive joint moment reliability was lower at these joint angles (coefficients of variance increased from 1.9% at 50% of ROM to 5.3% at 10% of ROM), limiting the change detecting significant change (30). Full ROM was calculated from the force trace inflection point, where a clear change in the slope of the passive moment curve occurred (see Fig. 3, in Ref. 30), to peak dorsiflexion ROM. As described previously (30), joint angles below this point did not reflect the normal curvilinear force-length MTC relationship, which was indicative of the MTC being at slack length and would reduce the validity of passive moment being indicative of the passive stiffness of the MTC.

Concentric plantar flexor ankle moment. The subjects were inclined to 85° at the hip to ensure that the center of rotation of the ankle remained in line with the center of rotation of the dynamometer, as described previously (30, 31). The subjects’ ankles were rotated through their dorsiflexion ROM at 0.087 rad/s until reaching the point of discomfort. The subjects then maximally contracted the plantar flexors isometrically until maximal isometric moment was reached (i.e., there was a visible plateau in the moment trace) before the footplate of the dynamometer was released at 0.087 rad/s. The subjects continued to maximally contract the plantar flexors through their full ROM. Concentric plantar flexor moment was normalized to the maximum plantar flexor moment attained during the MVIC (%MVIC). 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%), to remove interindividual variations in flexibility (30, 31). Analysis was not conducted at joint angles <50% of ROM, as the slow concentric velocity (5°/s) resulted in a total contraction period of ~12 s, which may have induced substantial fatigue. 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, Goleta, CA) before analog-to-digital conversion at a 2,000-Hz sampling rate (model MP150, Data Acquisition, Biopac). The signals were directed to a personal computer running AcqKnowledge (version 3.8.2, Biopac) software and filtered using a 20- to 500-Hz band-pass filter. The filtered signal was vectorized root-mean-squared EMG with a 250-ms sample window, and then normalized as a percentage of the peak amplitude recorded during a MVIC. The normalized EMG amplitude (%MVIC) was used as a measure of neuromuscular activity; the EMG signals for Sol, GM, and GL were averaged to obtain an amplitude representative of the TS muscle group activity (30, 31). The antagonist TA EMG data were processed and normalized using the same method. The normalized joint moment and normalized TS EMG amplitude were used to calculate the EMG-to-moment ratio. A fast-Fourier transformation of the 1-s sample of the filtered (non-root-mean-squared) EMG was used to calculate the mean and median EMG frequency at 90% of ROM.

Muscle and tendon length and stiffness. Motion analysis. Movement of the ankle in the dynamometer footplate was recorded during the passive, concentric, and stretch trials using real-time motion analysis with three infrared digital cameras (ProReflex, Qualisys, Gothenburg, Sweden) operating Track Manager 3D software (version 1.8.226, Qualisys). These recorded the position and movement of infrared reflective markers placed over the medial femoral epicondyle, representative of the origin of the GM (see Fig. 2; marker A), and over the calcaneum, representative of the insertion of the Achilles tendon (marker B). Ultrasound imaging was used to locate the GM-Achilles musculotendinous junction (MTJ), and a third marker (marker C) was placed over the MTJ position aligned with adhesive zinc-oxide hypoechoic tape. Data were sampled at 100 Hz, and raw coordinate data were smoothed using a 100-ms averaging window before the calculation of Achilles tendon and GM muscle lengths.
640 STRETCH-MEDIATED FORCE LOSSES

Fig. 2. Infrared reflective marker (motion analysis) and ultrasound probe positioning. Gastrocnemius medialis (GM) muscle length was estimated from the distance between the reflective markers B and C, positioned over the origin of the GM muscle on the medial femoral epicondyle and hypoechoic tape, positioned over the GM-Achilles musculotendinous junction (MTJ), respectively. Achilles tendon length was estimated from the distance between the reflective markers A and C, positioned over the insertion of the Achilles on the calcaneus and hypoechoic tape, positioned over the GM-Achilles MTJ, respectively.

ULTRASOUND. The position and excursion of the GM-Achilles MTJ were recorded using real-time ultrasound video imaging (LOGIQ Book XP, General Electric, Bedford, UK) from a wideband linear probe (8L-RS, General Electric) with a 39-mm wide field of view and coupling gel (Ultrasound gel, Dahlhausen, Cologne, Germany) between the probe and skin. The probe was orientated with the proximal end toward the origin of the medial head, and the distal end positioned toward the insertion of the tendon. The position of the probe was manipulated until the deep aponeurosis between GM and Sol could be visualized, and then the probe was affixed with zinc-oxide adhesive tape perpendicular to the skin. This protocol ensured consistent and accurate imaging of the MTJ and the hypoechoic tape throughout the trial (see Fig. 3). Ultrasound images were sampled at 28 Hz, the MTJ and middle of the hypoechoic area (indicative of reflective marker C) positions were manually digitized (Peak Motus, Englewood, CO), and raw coordinate data were smoothed using a 100-ms moving average. Given that the ultrasound and motion analysis data were sampled at different frequencies (28 and 100 Hz, respectively), the ultrasound data were exported to a spreadsheet, where a linear regression was employed to calculate the displacement of the MTJ from the middle of the hypoechoic area at the specific time used on the motion analysis system to ensure both data sets were synchronized before calculation of the distance between the MTJ and the middle of the hypoechoic area.

CALCULATIONS. Data from the dynamometer, motion analysis, and ultrasound systems were synchronized using a 5-V ascending transistor-to-transistor logic pulse, which was exported to the AcqKnowledge software (version 3.8.2, Biopac) and triggered the capture of the ultrasound data. A time-line regression to a specific ROM could then be determined using the transistor-to-transistor logic pulse marker placed on the AcqKnowledge data and the last image on the ultrasound recording. Achilles tendon length was calculated using motion analysis data as the distance between reflective marker A (Achilles insertion) and marker C (marker aligned to the hypoechoic tape), minus the distance from actual MTJ position to the middle of the hypoechoic tape (see Fig. 3; D) determined in the synchronized ultrasound image. GM muscle length was calculated as the distance between reflective marker B (GM origin) and marker C, plus the distance from actual MTJ position to the middle of the hypoechoic area (see Fig. 3; D). Achilles tendon stiffness was calculated by dividing the change in ankle moment by tendon length change (Nm/mm) during concentric trials. Due to the test-retest protocol employed in the present study, where data were analyzed at the same ROM, moment arms were not determined, since they were nonchanging and would not have influenced the results; thus, tendon “force” (joint moment per moment arm) was not derived.

Data Analysis

All data were analyzed using SPSS statistical software (version 11.5; LEAD Technologies); group data are reported as means ± SD and median and mean frequencies. Separate analyses of variance with repeated measures were used to test for differences in EMG amplitude and mean and median frequencies. Post hoc analyses of variance with repeated measures were used to test for differences in 1) concentric and passive plantar flexor moment; and 2) GM muscle and Achilles tendon lengths and tendon stiffness. Post hoc analyses with Bonferroni correction were used to further examine changes in measures where statistical significance was reached. Pearson’s product moment correlation was used to determine relationship between post-MVCC reductions in moment and changes in EMG amplitude. Statistical significance for all tests was accepted at P < 0.05. Test-retest reliability for these techniques has been documented previously (30).

RESULTS

The present study protocol included two interventions: 1) six ramped MVCCs; and 2) 3 min of passive static stretching. For clarity, the results are separated into two sections in line with these interventions.

Concentric Intervention

A significant reduction (mean = 6.6 ± 1.0%) was detected in concentric moment at 50% (7.3%; P < 0.05), 70% (7.1%; P < 0.01), and 90% (5.5%; P < 0.01) of ROM following the MVCC intervention (see Fig. 4). A significant decrease in peak TS EMG amplitude (mean = 10.2 ± 3.1%) was observed at 50% (7.3%; P < 0.01), 70% (13.5%; P < 0.01), and 90% (9.9%; P < 0.01) of ROM following the MVCC intervention.
were significant at 50% (r = 0.60; P < 0.05), 70% (r = 0.78; P < 0.01), and 90% (r = 0.94; P < 0.01) of ROM (see Fig. 6), indicating that the subjects who had the greater reductions in EMG tended also to exhibit the greatest loss of active joint moment. Similar correlations were also detected in individual muscle EMG amplitudes and joint moment (P < 0.05; data not shown).

No significant differences in muscle or tendon lengths were detected during the concentric trial (P > 0.05) following the MVCC intervention, indicating that muscle operating length had not changed; reliability of these methods to detect significant change in tendon length were modeled and have been described previously (31). However, a significant reduction in tendon stiffness from 10.3 ± 1.6 Nm/mm pre-MVCC to 8.9 ± 0.9 Nm/mm post-MVCC (11.7 ± 3.7%; P < 0.01) was observed (see Fig. 7) as the magnitude of tendon deformation (pre-MVCC = 15.0 ± 1.7 mm; post-MVCC = 16.0 ± 1.3 mm) was similar, despite the lower muscle force.

During the passive trials, a trend toward a reduced joint moment occurred at 50 and 70% of ROM, which became significant (3.9%; P < 0.01) at 90% of ROM (see Fig. 8). A significant increase in Achilles tendon length (0.8 ± 0.1%; P < 0.01) was detected at 50% (0.8%; P < 0.01), 70% (0.8%; P < 0.01), and 90% (0.7%; P < 0.01) of ROM, accompanied by a significant decrease in GM muscle length (0.4 ± 0.0%; P < 0.01) at 50% (0.4%; P < 0.01), 70% (0.4%; P < 0.01), and 90% (0.3%; P < 0.01) of ROM.

Stretch Intervention

A trend toward a further reduction in concentric joint moment was found at 50 and 70% of ROM poststretch, compared with post-MVCC data, which became significant at 90% of ROM (5.8%; P < 0.01). Joint moment remained significantly depressed after 30 min of rest at all joint angles compared with baseline data (9.2 ± 1.2%), indicating that force-generating capacity had not recovered (see Fig. 4). No significant reductions were detected in EMG amplitude or frequency immediately poststretch in any muscle. TS EMG amplitude (see Fig. 5) increased significantly at 30 min poststretch at all joint angles (P < 0.01) compared with poststretch data, such that it was no longer significantly reduced relative to baseline (P < 0.05). Similar recovery patterns existed for individual muscle EMG amplitudes (data not shown), which suggested that neuromuscular activity had significantly recovered.

![Graph showing correlation between post-MVCC reductions in moment and TS EMG at 90% of ROM. Significant correlations were detected between reductions in TS EMG amplitude and moment deficit at all joint angles measured (r = 0.60–0.94; P < 0.01).](http://jap.physiology.org/)

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**Fig. 4.** Joint moment during maximal concentric plantar flexion at 50, 70, and 90% of range of motion (ROM). Subjects performed a maximal concentric plantar flexion contraction through full ROM before and after six MVCC contractions, and 2 and 30 min after three 60-s stretches. Significant reductions were seen post-MVCC at all joint angles, with further reductions detected after stretch, which remained depressed 30 min later. MVIC, maximal voluntary isometric contraction. *Significant to P < 0.05. *Significant to P < 0.01 compared with baseline.

**Fig. 5.** Triceps surae (TS) electromyography (EMG) amplitude during maximal concentric plantar flexion at 50, 70, and 90% of ROM. Neuromuscular activity (EMG) was examined before and after six MVCC contractions, and 2 and 30 min after three 60-s stretches. Significant reductions were seen post-MVCC at all joint angles; no further effect was seen immediately poststretch. Significant increases were seen at 30 min poststretch, such that EMG was no longer depressed. *Significant to P < 0.01 compared with baseline.

**Fig. 6.** Correlation between post-MVCC reductions in moment and TS EMG at 90% of ROM. Significant correlations were detected between reductions in TS EMG amplitude and moment deficit at all joint angles measured (r = 0.60–0.94; P < 0.01).
No significant change in Achilles tendon stiffness (see Fig. 7) was detected poststretch at any ROM during the concentric contraction and remained significantly reduced relative to baseline after 30 min, indicating that tendon stiffness had not recovered. A significant decrease in Achilles tendon length (0.9 ± 0.1%; P < 0.01) was detected poststretch at 50% (0.8%; P < 0.01), 70% (0.9%; P < 0.01), and 90% (1.0%; P < 0.01) of ROM, indicative of the reduced force transmitted through the tendon.

Within the passive trials, further reductions were detected in passive moment (mean = 5.8 ± 0.6%) following the stretch protocol at 50% (5.0%; P < 0.01), 70% (6.1%; P < 0.01), and 90% (6.2%; P < 0.01) of ROM, indicating the reduced joint moment was attributable to reduced muscle stiffness as Achilles tendon stiffness remained unchanged poststretch. Passive joint moment increased significantly at 30 min poststretch (see Fig. 8) at all joint angles (P < 0.01), compared with poststretch data, and was no longer significantly reduced compared with baseline (1.7 ± 1.4%; P < 0.05).

DISCUSSION

The present study is the third of a series of distinct studies (30, 31) examining the effects of stretch on force production in isolation and when combined within a multi-intervention protocol preceded by intense muscular contractions. The same participants, stretch protocol, and measures were examined in all three studies, enabling a comparison of the data and the influence of stretch in isolation and when preceded by isometric or concentric contractions on force production. The aim of the present research was to examine the effects of a series of maximal concentric plantar flexor contractions and a subsequent period of static muscle stretching on Achilles tendon stiffness, neuromuscular activity (EMG), and concentric joint moment. The main finding from the present study was that the six-repetition MVCC protocol itself significantly impaired active concentric plantar flexor moment production, but then the subsequent 3-min static stretch intervention resulted in further losses of muscular force. The performance of isometric contractions has previously been reported to result in a similar reduction in concentric force (31), but, unlike the concentric contractions in the present study (which were of a similar duration and volitional intensity), those isometric contractions completely removed the subsequent effects of stretch. Perhaps of even greater significance was that, although there was less total decline in concentric moment following the concentric contraction-stretch combination (10.4%; present study) than previously shown for the isometric contraction-stretch combination [13.4%; Kay and Blazevich (31)], the significant reduction in concentric moment remained after a 30-min rest period study when concentric contractions preceded the stretching (9.2%; present study). The implications of these findings are substantive for the design of research studies, as the warm-up imposed on subjects before the stretch seems to strongly influence the stretch-induced loss of force. Discrepant findings between previous studies could have conceivably occurred through the choice of different warm-up routines. Although there are a number of methodological differences between studies, it is interesting to note that several studies that included repeated contractions before stretch (7, 17, 20) reported no effect of stretch, whereas studies omitting these contractions reported significant reductions in muscular performance (14, 22, 52). These results also have important practical implications for the formulation of pre-performance routines in normal, athletic, and clinical populations, where maximal force production within the plantar flexors is an important performance component; the performance of fatiguing maximal concentric contractions does not completely abolish the stretch-induced decline, but seems to be associated with a lesser recovery of the force after stretch.

The performance of maximal contractions before the testing of peak muscle strength and power has previously been reported to enhance force production (6, 12, 44), a phenomenon termed postactivation potentiation (PAP). However, their efficacy in this regard is debated due to equivocal reports in the literature (12, 25, 27). Furthermore, significant reductions in force and EMG activity have also been reported following similar intermittent contractions (49). Following the series of maximal contractions performed in the present study, a significant reduction in joint moment and EMG amplitude was detected, clearly indicating that PAP did not occur and that some level of fatigue was induced. Interestingly, Chiu et al.
(12) reported that, while PAP occurred in a highly trained athlete group, no potentiating effect was detected within a recreationally active group. Considering the subject demographic used in the present study (recreationally active), the present data are consistent with those of Chiu et al.

Typically, the reduced concentric moment might be explained by metabolic fatigue induced by the MVCC intervention; however, this is unlikely, as the intermittent contractions would not have induced local muscular ischemia (49). Furthermore, the clear reduction in EMG amplitude was highly correlated ($r = 0.94$) with the decrease in joint moment, but there was no change in the EMG-to-moment ratio. This is suggestive of an inability of the subjects to activate the $\alpha$-motoneuron pool rather than a decrease in muscle force at a given level of activation. Furthermore, there was no change in the mean or median EMG frequencies, which would be expected to decrease if muscular fatigue was present (13). Increased peripheral inhibition (4) of the $\alpha$-motoneuron pool from type 1b muscle afferents (golgi tendon organs), possibly as a result of the decreased tendon stiffness, may explain the reduced EMG activity. However, this too is unlikely, as a similar isometric intervention used in a previous study, which significantly reduced tendon stiffness, resulted in no change in isometric force, despite both concentric force and EMG being reduced (31). Furthermore, both isometric and concentric force would be attenuated, if peripheral inhibition was responsible for the reduced EMG activity. Therefore, these data are likely to be indicative of a decrease in central neural drive (16, 23, 49).

Although this has been reported following intermittent maximal contractions of similar repetition and duration (49), the present methods did not enable the location (i.e., spinal or supraspinal) of the reduced descending neural drive to be determined. Further research is required to reveal the location of the mechanisms underpinning the losses in EMG activity.

Importantly, despite there being a reduction in tendon stiffness after the MVCCs (measured during the concentric contractions), there was no change in the operating length of GM. Assuming that GM muscle length is indicative of the whole TS length, the decreased active concentric moment recorded after the MVCCs could not be explained by a reduced operating length of the TS affecting their force-length properties (35, 36). This finding is consistent with our previous reports of a lack of change in muscle length after stretch (30) or muscle contraction (31) interventions. Therefore, this commonly hypothesized mechanism underpinning reductions in joint moment (15, 22, 28, 52) can be removed as a candidate. Given that tendon stiffness is important for performance and injury risk, possible differences between subjects is an important consideration.

Efforts were made to ensure obese subjects were not included within the study (body mass index < 27 kg/m²); however, recently preobese (25–29.99 kg/m²) individuals have also been reported to have higher stiffness values than normal (21). This may be a limitation of the present study, and further research should be conducted on normal, preobese, and clinically obese subjects to determine the effects of these contractions on tendon stiffness.

Probably the most intriguing finding of the study was that 3 min of stretch resulted in a further decrease in concentric moment, which was accompanied by an increase in muscle operating length (i.e., a decrease in tendon length), but not a further decrease in EMG amplitude. Thus, after concentric muscle actions, it seems possible that muscle force-generating capacity can be compromised by stretching, even when neuromuscular activity is not. Similar decreases in muscle force production without a change in EMG have been previously reported after an acute bout of static stretching (29, 52), but we recently showed that the present stretch protocol reduced concentric moment concomitantly with a decrease in EMG when stretching is performed in isolation (30), or to have no effect on either active moment or EMG when it is performed after a series of isometric contractions (31). Indeed, in addition to the stretch protocol being identical between these studies, the same subject sample was recruited for the present study, which improves our confidence in the comparisons. Also, the concentric contractions conducted in the present study involved the performance of an 8-s contraction with joint moment increasing to maximum after 4 s and then decreasing to zero by 8 s. This facilitates the comparison of results to those of the previous study (31), where isometric contractions were performed with a 4-s increase to maximum and then a 4-s decrease to zero in joint moment. Importantly, joint moment remained significantly depressed 30 min later (9.2% compared with baseline), despite EMG recovering fully. Thus, while a decrease in neuromuscular activity might be reflective of, or associated with, other changes that might impact on force-generation capacity, it does not seem that a reduced activity was either the main cause of the additional force decline detected poststretch in the present study, or important for the maintenance of these losses 30 min later.

Although reduced neuromuscular activity was associated with the initial reduction in joint moment following the concentric intervention, it was clearly not a mechanism underpinning the additional losses incurred poststretch or the maintenance of these losses 30 min later. Plantar flexor joint moment and EMG have previously been shown to recover after 30 min when stretching was performed in isolation (4, 22, 30) and when stretching was preceded by a series of isometric contractions (31). However, the additional losses in joint moment found poststretch in the present study occurred without any change in EMG. Similar poststretch reductions in joint moment, without any change in EMG, have been reported previously (29, 52). Unfortunately these studies did not determine the temporal effects of stretch, which limits our ability to determine whether these losses were transient. In the absence of any change in neuromuscular activity, muscle-based hypotheses have been suggested to partially explain these poststretch force losses. The significant reduction in passive moment detected poststretch in the present study was attributed to reduced muscle stiffness, in agreement with other studies (30, 42). This could be explained by the thixotropic properties of muscle tissue, where muscle stiffness can be affected by prior contraction or stretch history (41, 45, 46), although Morse et al. (42) suggested that this was an unlikely mechanism able to explain the reduction in muscle stiffness. Alternatively, the reduced stiffness may be indicative of damage to the muscular cytoskeleton, which could affect contractile behavior (56), calcium infiltration (3, 34), and the excitation-contraction coupling process (10, 34), which may explain the reduced force generation. Regardless, the specific impairment to this process remains unknown, and further research is required, although the mechanism underpinning the maintenance of these force losses appears not to be of neurological origin.
In summary, the effects of concentric contractions (MVCCs) and passive stretch on joint moment, neuromuscular activity, and MTC mechanics were examined. In agreement with a previous isometric intervention study (31), significant reductions were detected in concentric joint moment and EMG amplitude post-MVCC. Furthermore, the significant correlation \((r = 0.94)\) between reductions in joint moment and reduced muscle EMG amplitude confirmed our previous findings \([r = 0.90;\) Kay and Blazevich (31)] that reduced neuromuscular activity was the most likely mechanism underpinning these losses. The absence of any change in the frequency content of the EMG signal is suggestive of central mechanisms affecting the activation of the α-motoneuron pool. Importantly, a further reduction in concentric joint moment was found poststretch when no change in EMG occurred; 30 min later, EMG fully recovered, while concentric moment remained impaired, implicating a muscle-based mechanism underpinning these additional force losses. Therefore, stretching in isolation (30) or when preceded by isometric contractions (31) can temporarily (<30 min) reduce plantar flexor force; however, additional and long-lasting (>30 min) reductions in force occur when concentric contractions precede stretch, which may influence performance and injury risk. These findings have important implications for research study design, as the warm-up imposed on subjects before stretch seems to strongly influence the impact of stretch. Furthermore, the results also have important practical implications in the formulation of neuromechanical properties of the triceps surae muscle complex. *J Appl Physiol* 86: 428–434, 2002.


DISCLOSURES

No conflicts of interest are declared by the authors.

REFERENCES


