Isometric contractions reduce plantar flexor moment, Achilles tendon stiffness, and neuromuscular activity but remove the subsequent effects of stretch

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Kay AD, Blazevich AJ. Isometric contractions reduce plantar flexor moment, Achilles tendon stiffness, and neuromuscular activity but remove the subsequent effects of stretch. *J Appl Physiol* 107: 1181–1189, 2009. First published July 30, 2009; doi:10.1152/japplphysiol.00281.2009.—The effects of isometric contractions and passive stretching on muscle-tendon mechanics and muscle activity were studied in 16 healthy human volunteers. First, peak concentric and passive ankle joint moment data were recorded on an isokinetic dynamometer with electromyographic monitoring of the triceps surae; real-time motion analysis of the lower leg and ultrasound imaging of the Achilles-medial gastrocnemius muscle-tendon junction were simultaneously conducted. Second, the subjects performed six 8-s maximal voluntary isometric contractions (MVICs) before repeating the passive and active trials. Although there was no decrease in isometric joint moment after MVICs, peak concentric moment was significantly reduced (11.5%, *P* < 0.01). This was accompanied by, and correlated with (*r* = 0.90, *P* < 0.01), significant reductions in peak triceps surae electromyographic amplitude (21.0%, *P* < 0.01). Achilles tendon stiffness (10.9%, *P* < 0.01) and passive joint moment (4.9%, *P* < 0.01) were also significantly reduced. Third, the subjects performed three 60-s static plantar flexor stretches before being retested 2 and 30 min after stretch. The stretch protocol caused no significant change in any measure. At 30 min after stretching, significant recovery in concentric moment and muscle activity was detected at dorsiflexed joint angles, while Achilles tendon stiffness and passive joint moment remained significantly reduced. These data show that the performance of MVICs interrupts the normal stretch-induced losses in active and passive plantar flexor joint moment and neuromuscular activity, largely because concentric strength and tendon properties were already affected. Importantly, the decrease in Achilles tendon stiffness remained 30 min later, which may be an important etiological factor for muscle-tendon strain injury risk.

triceps surae; force deficits; tissue mechanics; electromyography

**Preperformance Warm-up Routines** are commonly promoted and are specifically designed to prepare an individual for high-intensity physical activity and reduce the risk of injury (9, 56). The routines typically include cardiovascular warm-up, stretching, and strong muscular contractions (with progressing intensity), which promote increased peripheral blood flow to the working muscle, elevated intramuscular temperature, enhanced neural conduction velocity, increased range of motion (ROM), and decreased viscosity and stiffness of the muscle-tendon complex (MTC) (1, 2, 9, 26, 27, 31, 32, 56). The stretching routines conducted within the warm-up protocol are employed primarily to increase functional ROM and reduce MTC stiffness in an attempt to reduce injury risk, although their effect in this regard is still debated (21, 49, 53, 55). The strong muscular isometric contractions have been shown to alter mechanical properties of the MTC, which may optimize neuromuscular recruitment and force production. Accordingly, some athletes use maximal isometric contractions within their warm-up protocol to potentiate neuromuscular recruitment for optimal performance.

Recently, significant reductions in force and power production were reported immediately after passive muscle stretching (7, 12–16, 19, 26–29, 37, 38, 42–44, 48, 50, 52, 57). Although there are numerous possible mechanisms underpinning the decrease in force after stretching, two primary mechanisms include 1) reduced neuromuscular activation (4, 5, 15, 19, 27) and 2) altered mechanical properties of the MTC (14, 16, 19, 26, 42, 43, 52). Stretch-induced reductions in electromyographic (EMG) activity have been reported concurrently with force losses after 30 min (19) and 1 h (4) of intermittent static stretching, and stretch-induced force deficits within the plantar flexors have been strongly correlated with the reductions in triceps surae (TS) EMG amplitude (~65% explained variance, *P* < 0.01) (27). However, the changes in active joint moment cannot be fully explained by changes in neuromuscular activity, so a separate mechanism must be partially responsible for the force deficits.

Poststretch reductions in passive moment (26, 27, 31, 35, 36, 41), indicative of changes in the mechanical properties of the MTC, could impact the force-generating capacity of important muscle groups, such as the TS, if there was a decrease in the series stiffness (and, in particular, a decrease in tendon stiffness), which would cause the muscle to operate at a shorter, weaker length (33, 34). Indeed, Kubo et al. (31) reported a significant decrease in Achilles tendon stiffness and muscle operating length after 10 min of static plantar flexor stretch, although this finding was not associated with a reduced isometric force output. In contrast, Morse et al. (41) and Kay and Blazevich (27) reported that shorter-duration (<5-min) stretches did not affect Achilles tendon stiffness or reduce muscle operating length (27). Interestingly, Kubo et al. (32) reported similar reductions in tendon stiffness after fifty 3-s isometric contractions. Collectively, these studies suggest that the duration and intensity of tissue strain imposed by stretching or strong muscular contractions may determine whether changes in tendon stiffness occur. Identification of interventions that alter the mechanical properties of the tendon is important, inasmuch as reduced tendon stiffness may increase...
neuromechanical delay (17, 22, 30), reduce the rate of force development (11, 18, 32), and decrease the active muscle length (32), which could attenuate maximal force in the human plantar flexors according to its force-length relationship (33, 34). Altered mechanical properties have been hypothesized to contribute to poststretch reductions in force production (14, 16, 19, 26, 42, 43, 52), and given that similar changes in tendon stiffness may be induced by intense contractions (32), the greater intensity of tissue strain imposed during these contractions may mitigate or remove the effects of subsequent stretch. An understanding of the effects of maximal contractions combined with stretching is important for two reasons. 1) Some researchers (7, 13–15, 37, 47), but not others (35, 36, 41), have included maximal isometric contractions in the warm-up or as part of the experimental model before testing the effects of static stretch on MTC mechanics, neuromuscular activation, and force production. The impact of these contractions on subsequent stretch-induced force losses and the mechanical or neuromuscular mechanisms associated with these losses has not been directly measured. The inclusion of these contractions may modify MTC mechanics before the stretch intervention, which may reverse, mitigate, or compound the effects of subsequent stretch. 2) Warm-up protocols commonly include maximal contractions and stretching, so examination of each activity in isolation does not allow estimation of their combined effects on tendon properties and force production.

To gain a more comprehensive understanding of the impact of isometric contractions and static stretch on force production, it is necessary to quantify muscle activity and muscle (or tendon) length changes simultaneously, within a multi-intervention protocol. The aims of the present study were as follows: 1) to determine the effects of six 8-s ramped maximal voluntary isometric contractions (MVICs) on Achilles tendon stiffness, gastrocnemius medialis (GM) muscle operating length, active (concentric) and passive plantar flexor joint moment, and neuromuscular (EMG) activity of the TS and 2) to determine the influence of these contractions on the well-documented effects of stretch by quantifying the additional effects of these measures 2 and 30 min after stretch.

MATERIALS AND METHODS

Subjects

Sixteen active individuals (8 women and 8 men, 20.2 ± 2.6 yr old, 65.5 ± 10.5 kg, 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. The subjects were asked to avoid intense exercise, stretching, and stimulant use for 48 h before testing. Ethical approval was granted by the Ethics Committees of the School of Sport and Education at Brunel University and the School of Health at the University of Northampton, and the study was conducted in accordance with the Declaration of Helsinki.

Protocol

Overview. The protocol is similar to that used in our previous study (27). The subjects visited the laboratory on three occasions, each separated by 1 wk. They were initially familiarized with the testing protocol 1 wk before data collection and then visited the laboratory on two further occasions, once under control conditions (no stretch) and once under the experimental condition, in a randomized order. During the experimental sessions, the subjects performed a 5-min warm-up on a Monark cycle at 60 rpm with a 1-kg resistance load producing a power output of 60 W. Each subject was then seated upright in the chair of an isokinetic dynamometer (Biodex System 3 Pro, IPRS, Suffolk, UK) with the knee fully extended (0°) to ensure that the gastrocnemius was also placed under significant stretch and contributed significantly to plantar flexor joint moment (17, 25). The ankle was placed 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 dynamometer footplate initially rotated the ankle to full plantar flexion (~30°), and then the ankle was passively rotated to its maximum dorsiflexion ROM at 0.087 rad/s (5°/s); in this position, the subject performed a maximal concentric plantar flexor contraction at an angular velocity of 0.087 rad/s. Subsequently, the subject produced six 8-s ramped MVICs with the ankle in neutral position (0°) before repeating the passive and active trials to determine any effects of isometric contractions. Three 60-s static plantar flexor stretches were then imposed by rotation of the ankle to full dorsiflexion ROM, with 60 s of rest between each stretch. The subject subsequently repeated the passive and active trials 2 and 30 min after stretch to determine the effects of stretch. The order and time of the experimental protocol, including the passive and concentric trials and isometric and stretch interventions, are shown in Fig. 1.

Passive ankle moment. While seated with a hip angle of 55° and the knee extended, the ankle was passively rotated through the full ROM at 0.087 rad/s. The subject was instructed to volitionally terminate the rotation by pressing a hand-held release button at the point of discomfort. Passive moment was recorded throughout the trial and then normalized (as a percentage) to the maximum prestretch passive joint moment. To account for interindividual differences in joint flexibility/ROM, moment data were analyzed at 50, 70, and 90% of maximum ROM. Full ROM was calculated from the passive joint moment inflection point (mean angle = 0.8 ± 4.8° dorsiflexion), where a clear change in the slope of the passive moment curve occurred (27), to the volitional end of the ROM.

Concentric plantar flexor ankle moment. While seated with an 85° hip angle and the knee extended, the dynamometer rotated the ankle through its ROM at 0.087 rad/s until reaching the point of discomfort. The subject then maximally contracted the plantar flexors until maximal isometric moment was attained (i.e., there was a visible plateau in the moment trace) before the footplate of the dynamometer was released at 0.087 rad/s. This enabled the subject to continue to maximally contract the plantar flexors through the full ROM (Fig. 2). Concentric plantar flexor moment was normalized as a percentage of

![Fig. 1. Timeline of maximal voluntary isometric contractions (MVICs) and stretch interventions. At 5 min after completion of the warm-up, passive and active concentric trials were conducted, and after 2 min the MVIC intervention was carried out. After 2 min, passive and active trials were repeated to determine the effect of the MVIC intervention. Stretch intervention was initiated 2 min later, with passive and active trials repeated 2 and 30 min after stretch to determine effects of the stretch intervention.](http://jap.physiology.org/)

### STUDY PROTOCOL

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the maximum plantar flexor moment measured during the MVIC (%MVIC). Maximal concentric moment was recorded throughout the full ROM; data were analyzed only at 50% (mean angle = 7.7 ± 3.8°) plantar flexion, 70% (mean angle = 0.7 ± 5.3°) plantar flexion, and 90% (mean angle = 6.2 ± 6.8°) dorsiflexion) of the full ROM, calculated between full plantar flexion (0%) and full dorsiflexion (100%), to remove interindividual variations in flexibility. Analysis was not conducted at joint angles <50% of ROM, inasmuch as the slow concentric velocity (5/s) resulted in a total contraction period of ~12 s and incurred substantial fatigue. During testing, joint moment, joint angle, and angular velocity data for 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 data were then directed to a personal computer running AcqKnowledge software (version 3.8.2, Biopac) and filtered with a double-pass 6-Hz Butterworth low-pass filter.

**Maximal isometric contractions.** At 2 min after completion of the concentric trial, the ankle was passively rotated from full plantar flexion at 0.087 rad/s until it reached the anatomic position (0°). The subject then produced a ramped maximal isometric plantar flexor contraction, with maximal moment reached ~3 s after contraction initiation (visible plateau in the moment curve); the subject slowly reduced force to zero (~8 s total contraction time). The ramped contractions allowed determination of tendon deformation, which enabled calculation of tendon stiffness. The ankle was then returned to a plantar-flexed position (25°), and this process was repeated after a 30-s rest, with the subject completing a total of six contractions during the isometric trial. At 2 min after completion of the six isometric contractions, the subject repeated the passive and active trials for determination of any effects of the isometric contractions (Fig. 3).

**Stretch protocol.** At 2 min after completion of the second concentric trial, the ankle was passively rotated at 0.087 rad/s through its full ROM until reaching the point of discomfort, a position regularly used in stretch studies (7, 16, 26, 27, 47). The movement velocity was too slow to elicit a significant myotatic stretch reflex response (37, 39, 40), which ensured that full ROM was achieved and a substantial stress was applied to the MTC. The ankle was 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. After 60 s of rest, the stretch protocol was repeated (twice), giving a total stretch duration of 180 s.

**EMG recording.** Site preparation, electrode placement, EMG sampling, processing, and normalization methods were completed as previously described (27). Skin-mounted bipolar double-differentiated active electrodes (model MP-2A, Linton, Norfolk, UK) constantly monitored the EMG activity of the soleus (Sol), gastrocnemius medialis (GM), gastrocnemius lateralis (GL), and tibialis anterior (TA). EMG signals were amplified (gain = 300, input impedance = 10 GΩ, common mode rejection ratio ≈100 dB at 65 Hz) and directed 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 signals were then directed to a personal computer running AcqKnowledge software (version 3.8.2), where they were filtered using a 20- to 500-Hz band-pass filter. The filtered signal was converted to root-mean-squared EMG with a 250-ms sample window and normalized as a percentage of the peak amplitude recorded during an MVIC. The normalized EMG amplitude (%MVIC) was used as a measure of neuromuscular activity; the normalized EMG signals for GM, GL, and Sol were then averaged to reflect the representative activity of the TS muscle group (27). The antagonist TA EMG data were processed and normalized using the same method.

**MUSCLE AND TENDON LENGTH AND STIFFNESS.** Motion analysis. Movement of the ankle in the dynamometer footplate was recorded using a real-time motion analysis system with three infrared digital cameras (ProReflex, Qualisys, Gothenburg, Sweden) operating Track Manager 3D software (version 1.8.226, Qualisys). Infrared reflective markers were placed over the insertion of the Achilles at the calcaneus (Fig. 4, marker A), over the origin of the medial head of the gastrocnemius at the medial femoral epicondyle (marker B), and over the GM-Achilles muscle-tendon junction (MTJ, marker C), with adhesive zinc oxide-hypoechoic tape placed on the skin aligned with this marker. Raw coordinate data were sampled at 100 Hz and smoothed using a 100-ms averaging window before the calculation of Achilles tendon and GM muscle lengths.

**ULTRASOUND.** The GM-Achilles MTJ was identified (Fig. 5) using real-time ultrasound video imaging (LOGIQ Book XP, General Electric, Bedford, UK) and a wide-band 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 then affixed perpendicular to the skin to maintain a constant position with zinc oxide adhesive tape, which ensured consistent imaging of the MTJ and the hypoechoic tape throughout the trial. Ultrasound images were sampled at 28 Hz, and the position of the MTJ was manually digitized (Peak Motus, Englewood, CO) and smoothed using a 100-ms moving average.

**CALCULATIONS.** Motion analysis, ultrasound, and dynamometer data were synchronized using a 5-V ascending transistor-transistor logic pulse, which triggered the capture of ultrasound data (preceding 27 s of data) and simultaneously placed a marker on the ROM trace on the AcqKnowledge software (version 3.8.2). GM muscle length was calculated as the distance between reflective markers B and C plus the distance from actual MTJ position (determined with ultrasound; Fig. 5). Tendon length was calculated as the distance between

**Fig. 2. Pre- and post-MVIC moment data from 1 subject’s trial. Joint moment was recorded before release of the dynamometer and remained reduced throughout ankle range of motion (ROM) at all joint angles after the MVIC intervention.**
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 within the control condition ($P > 0.05$). The present study protocol included two interventions: 1) six ramped MVICs followed by 2) 3 min of passive static stretching. For clarity, the results have been separated into two sections in line with these interventions.

Isometric Intervention

There was no significant difference in peak isometric joint moment between the first and sixth isometric contractions ($P > 0.05$), indicating that fatigue was not induced. A significant increase in Sol EMG amplitude was detected (15.5%, $P < 0.05$); however, no change in EMG was detected in the other TS muscles (GL and GM) or when EMG was averaged across the muscles (3.5%, $P > 0.05$). No change in TA EMG was observed, indicating that co-activity of the antagonist muscle was unchanged.

Significant reductions (mean $11.5 \pm 1.3\%$) were detected in concentric moment at 50% (12.9%, $P < 0.05$), 70% (11.4%, $P < 0.01$), and 90% (10.4%, $P < 0.01$) of ROM following the MVIC intervention (Fig. 6). A significant decrease in peak TS EMG amplitude (21.0 ± 0.3%) was observed after MVIC intervention at 50% (20.8%, $P < 0.01$), 70% (20.7%, $P < 0.01$), and 90% (21.3%, $P < 0.01$) of ROM during the concentric contractions (Fig. 7). Similar reductions ($P < 0.01$) in all EMG amplitudes (GL mean = 23.4 ± 2.6%, GM mean = 22.6 ± 3.0%, and Sol mean = 16.3 ± 1.8%) were detected. A significant reduction in TA EMG amplitude (13.7 ± 1.8%, $P < 0.05$) was also detected, suggesting that the MVIC intervention also decreased muscular co-activity.

Pearson’s product-moment correlations computed between reductions in TS EMG and decreases in joint moment were significant at 50% ($r = 0.90$, $P < 0.01$), 70% ($r = 0.73$, $P < 0.01$), and 90% ($r = 0.74$, $P < 0.01$) of ROM (Fig. 8).

Reliability

Previous test-retest reliability was determined from our laboratory (27) by calculating the intraclass correlation coefficients (ICC) for intratester reliability of the manual digitizing of ultrasound MTJ excursion from the hypoechoic tape ($n = 5$). ICCs ranged from 0.98 to 0.99; no significant difference was detected between mean values ($P > 0.05$). Coefficients of variation (expressed as a percentage of the mean) were also calculated and ranged from 0.3 to 0.4%. Test-retest reliability was calculated for concentric moment, passive moment, and muscle and tendon length during the control condition ($n = 15$). The ICC ranged from 0.79 to 0.90, 0.83 to 0.99, 0.99, and 0.99, and coefficients of variation ranged from 2.3 to 3.2%, 1.8 to 5.2%, 0.2%, and 0.4 to 0.5%, respectively.

Fig. 4. Reflective marker (motion analysis) and ultrasound probe positioning. Achilles tendon length was estimated from the distance between reflective markers placed over the insertion of the Achilles tendon on the calcaneus (marker A) and hypoechoic tape (marker C) placed over the gastrocnemius medialis (GM)-Achilles muscle-tendon junction (MTJ). GM muscle length was estimated from the distance between reflective markers placed over the origin of the GM muscle on the medial femoral epicondyle (marker B) and hypoechoic tape (marker C) placed over the GM-Achilles MTJ.

Fig. 5. Ultrasound image of GM-Achilles MTJ. Position and displacement of reflective markers A and C minus the distance from the actual MTJ position (27). Tendon stiffness was calculated by dividing tendon length change by change in ankle moment.

Data Analysis

All data were analyzed using SPSS statistical software (version 11.5, LEAD Technologies, Chicago, IL); group data are means ± SE, and change data are means ± SD. The study protocol included two interventions, isometric contractions (MVICs) and static stretches. Paired t-tests were used to test for differences in 1) peak isometric moment and EMG and 2) muscle and tendon length and stiffness between the first and sixth isometric contractions. Separate ANOVAs with repeated measures were used to test for differences in 1) concentric and passive plantar flexor moment, 2) peak TS amplitude (EMG), and 3) muscle and tendon length and stiffness after MVICs. Pearson’s product-moment correlation was used to determine the relationship between post-MVIC reductions in moment and changes in EMG amplitude.

After the static stretch intervention, separate ANOVAs with repeated measures were used to test for differences in 1) peak concentric and passive plantar flexor moment, 2) peak TS amplitude (EMG), and 3) muscle and tendon length and tendon stiffness. Post hoc t-tests with Bonferroni’s correction were used to further examine changes in measures where statistical significance was reached. Statistical significance for all tests was accepted at $P < 0.05$. 

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indicating that the subjects who had the greater reductions in EMG amplitude tended also to exhibit a greater loss of active joint moment. Significant correlations were also detected in individual muscle EMG amplitudes and joint moment at all joint angles ($P < 0.01$; data not shown).

Achilles tendon and GM muscle lengths were measured during the first and sixth contractions at 30, 50, 70, and 90% of MVIC (determined in the 1st contraction). Mean data revealed a trend toward a longer tendon ($187 \pm 7$ and $190 \pm 7$ mm for MVIC at 1st and 6th contractions, respectively) and shorter GM muscle ($243 \pm 5$ and $241 \pm 5$ mm for MVIC at 1st and 6th contractions, respectively) length at all MVIC percentages, which became significant ($1.2\%$, $P < 0.01$) at 90% MVIC and resulted in the muscle operating at a shorter length during the sixth isometric contraction.

After the MVIC intervention, there were no significant differences in muscle or tendon length at any ROM during the concentric trial ($P > 0.05$), indicating that the stretch protocol did not affect force production or neuromuscular activity. Peak concentric moment increased significantly 30 min after stretch at 70 and 90% of ROM compared with immediately after stretch (Fig. 6) and was no longer significantly depressed relative to baseline ($4.7 \pm 2.4\%$). Peak EMG amplitudes in GM, GL, Sol, and TS (Fig. 7) increased significantly.

Stretch Intervention

There was no change ($P > 0.05$) in concentric joint moment or EMG from pre- to poststretch, indicating that the stretch protocol did not affect force production or neuromuscular activity. Peak concentric moment increased significantly 30 min after stretch at 70 and 90% of ROM compared with immediately after stretch (Fig. 6) and was no longer significantly depressed relative to baseline ($4.7 \pm 2.4\%$). Peak EMG amplitudes in GM, GL, Sol, and TS (Fig. 7) increased significantly.
icantly 30 min after stretch at 70 and 90% of ROM compared
with immediately after stretch and were no longer significantly
depressed relative to baseline (GM = 7.3 ± 3.0%, GL = 4.8 ±
4.4%, Sol = 2.3 ± 4.8, TS = 3.2 ± 1.1%). No significant
recovery in moment or EMG was detected at 50% of ROM
(P > 0.05).

Pearson’s product-moment correlations computed between
the recovery in TS EMG and the recovery in concentric joint
moment were significant at 70% (r = 0.63, P < 0.05) and 90% (r = 0.73, P < 0.01) of ROM, with the subjects who achieved
greater recovery in EMG tending also to exhibit a greater
recovery of active joint moment. Significant correlations were
also detected in individual muscle EMG amplitudes and joint
moment (P < 0.05; data not shown).

During the concentric trials, no significant change in GM
muscle length, Achilles tendon length, or Achilles tendon
stiffness (Fig. 9) was detected after stretch at any joint angle,
indicating that muscle operating length at these joint angles did
not change after stretch. Also there was no significant change
in passive moment (Fig. 10), Achilles tendon stiffness and
length, or GM muscle length (P < 0.05) at any joint angle
during the passive trials. Thus there was no significant recov-
ery in these variables after 30 min.

DISCUSSION

The aims of the present research were 1) to determine the
effects of six MVICs on Achilles tendon stiffness, passive and
concentric force production, and neuromuscular activity
(EMG) and 2) to determine whether there were any additional
changes in these measures after 3 min of static stretch. After
the MVIC intervention, active (concentric) joint moment was
significantly lower at all joint angles (mean = 11.5 ± 1.3%,
P < 0.01), and this was accompanied by, and correlated with
(r = 0.90, P < 0.01), reduced TS EMG amplitude (mean =
21.0 ± 0.3%, P < 0.01). These changes could not be attributed
to the initial concentric contraction, inasmuch as no change in
any measure (P > 0.05) was detected under control conditions,
i.e., concentric protocol followed by 5 min of rest and a repeat
of the concentric trial. Also, metabolic muscle fatigue could
not account for the force depression, inasmuch as isometric
joint moment did not change between the first and sixth (last
isometric contractions. The MVIC intervention also resulted in
a significant decrease in passive joint moment (mean = 4.9 ±
0.7, P < 0.01), indicating a decreased stiffness of the MTC or
joint capsule. The findings of a trend toward a longer tendon
length during the passive joint rotation and a significantly
reduced tendon stiffness during the maximal concentric con-
traction (mean = 10.7 ± 1.3%, P < 0.01) suggest that the
reduced passive joint moment was attributable to a reduction in
tendon stiffness. Importantly, the stretch intervention imposed
after the MVIC did not cause a further change in muscle-
tendon properties, neuromuscular activity, or force-generating
capacity of the TS muscles (P > 0.05). Finally, significant
increases were detected in active joint moment and EMG
amplitude in the most dorsiflexed positions after 30 min of
passive recovery following the stretch intervention, but passive
moment and Achilles tendon stiffness remained depressed.

The present data indicate that active concentric plantar flexor
moment decreases after MVICs, which are accompanied by,
and highly correlated with, decreases in EMG amplitude. In the
present study, we found a linear relationship between the
decreases in force and EMG amplitude, such that >81% of
the variability in moment changes was explained by the
changes in EMG amplitude. Although the strongest correlation
was found when the EMG amplitudes of the three TS muscles
were averaged (r = 0.90, P < 0.01), significant correlations
were also found for GM (r = 0.63, P < 0.05), GL (r = 0.68,
P < 0.01), and Sol (r = 0.85, P < 0.01) muscles individually.
The reduction in EMG measured in the concentric contractions
is intriguing, because no decrease in force or EMG amplitude
was seen during the isometric contractions. Typically, reduc-
tions in EMG amplitude result from decreased central neural
drive (14, 15, 20) or peripheral inhibition or disfacilitation
of the α-motoneuron pool by associated muscle afferents (4). The
present data are suggestive of central, rather than peripheral,
mechanisms influencing EMG amplitude, since peripheral al-
terations would be expected to influence EMG during the
isometric and concentric contractions. Furthermore, peripheral
inhibition of the α-motoneuron pool from increased Golgi
tendon organ activity should increase co-activity of the TA
muscle. However, significant reductions were detected in TA
EMG, lending further support to the idea that central, rather
than peripheral, mechanisms are responsible for the reductions
in neuromuscular activity. We are unaware of other studies
that have reported contraction mode-dependent changes in muscle
force and EMG; therefore, further research is required, inas-
much as these results may have practical implications for
muscle performances that rely on concentric force production.

Despite the strong relationship, the reduced active joint
moment could not be completely explained by the decrease in
EMG. Another possible mechanism of force reduction is a
change in tendon stiffness, inasmuch as this could result in a
change in muscle operating length (32); the leftward shift in its
force-length curve would cause the plantar flexors to operate
farther down their ascending limb (33, 34). This hypothesis
appears to be supported by the significant reduction in tendon
stiffness during the concentric trial after MVICs in the present
study (10.7%). However, ultrasound imaging of the MTJ
revealed no change in tendon length (<0.1 mm) or muscle
operating length after MVICs during the concentric trials,
despite an 11.5% reduction in concentric moment. Thus the
decrease in force production combined with a greater tendon

\[ P < 0.05, \#P < 0.01. \]
Compliance allowed the muscle to work at the same length at a given joint angle, so a leftward shift in the force-length curve was not present and cannot explain the reduced muscle force. To ensure that the present methods could detect a change in tendon length, a linear regression was employed to model tendon deformation during a ramped MVIC. This model calculated a projected 2-mm reduction in tendon deformation from the 11.5% reduction in joint moment seen in the present data; we are confident that the present methods would be able to detect this small, but significant, change.

Although altered force-length characteristics were clearly not a mechanism for the force depression, the decreased tendon stiffness might have functional consequences. Tendon structures account for 42.5–60% of total work done during the concentric phase (8, 10, 51) of the stretch-shortening cycle exercise, where the concentric action of the MTC is preceded by eccentric loading. The stiffness of tendinous structures has also been significantly correlated with rate of force development, maximal isometric force, and vertical jump height (11, 18, 32, 54) and inversely correlated with neuromechanical delay (17, 22, 30). Therefore, a reduction in the stiffness of the tendon may impede fast force transmission through the tendon onto the bone, thus attenuating joint moment. However, a more compliant tendon would store more energy and transfer less force to the musculature during eccentric loading of the MTC, cited as a factor related to injury (24). Therefore, the decrease in tendon stiffness may provide a prophylactic effect and reduce the risk of muscle strain injury. Also, if a decrease in stiffness is reflective of a decreased tensile strength of the tissue, then there might be an increase in the risk of tendon strain injury. However, these two properties are not always functionally related, and strong muscular contractions are commonly performed in athletic populations, and with a paucity of tendon rupture reported in the literature (in the healthy tendon), we believe this is unlikely. Nonetheless, the reduced maximal joint moment associated with this intervention may increase the injury risk, inasmuch as strength has also been cited within the etiology of muscle strain injury (46). Thus the impact of these contractions on MTC injury risk is unclear.

The isometric contraction protocol was chosen to ensure that a consistent amount of work was completed between subjects and similar strain was applied to the tendinous tissues to enable valid postintervention statistical analyses. However, a range of maximal contraction modes are commonly performed during athletic preperformance routines, and rapid eccentric or concentric loading of the MTC may induce effects different from those observed following the maximal isometric contractions performed in the present study. Therefore, these data cannot be generalized to athletic populations where different preperformance regimens may be used. Further research is needed to determine the effects of contraction mode on force production and the mechanical properties of the MTC.

Notwithstanding the possible differences in the modality of the preperformance contractions employed in the present study, the examination of the effects of stretch after a series of intense muscle contractions is important given that many individuals perform a progressively intense warm-up in addition to stretching before maximal exercise bouts. Some research studies (7, 13–15, 37, 47), but not others (35, 36, 41), have included maximal contractions in the warm-up or as part of the experimental model before testing the effects of static stretch on joint moment. However, to our knowledge, no studies have examined whether these contractions might mitigate or compound the effects of subsequent stretching. Clearly the MVIC intervention resulted in significant decreases in active (concentric) and passive joint moment. A novel finding of the present study was that no further reductions in concentric joint moment, EMG, passive moment, or MTC stiffness were found after stretch, despite use of a stretch protocol that has previously resulted in substantial changes in these variables (27). Thus the negative effects of stretch appear to be dependent on whether MVICs are performed before stretch. The impact of prior maximal isometric contractions not only has important methodological implications for stretch-based studies but also has significant practical implications for athletes where maximal concentric contractions are essential to performance. Significant improvements in muscle performance (6, 23, 45) have been reported following the implementation of prior maximal isometric contractions. Again, further research is required to determine whether similar reductions are realized when other contraction modes (e.g., concentric) are used before stretch and whether they also mitigate the subsequent effects of stretch.

Interestingly, after 30 min of rest, there was a significant recovery of concentric joint moment (64% recovery of the deficit) in the most dorsiflexed positions. This was accompanied by, and correlated with (r = 0.73, P < 0.01), significant recovery in EMG amplitude (82%), such that force and EMG were no longer significantly reduced compared with baseline levels. However, tendon stiffness and passive joint moment remained significantly depressed. Previously, stretch-based interventions (19, 27) resulted in similar recovery patterns in active joint moment and EMG amplitude; however, passive joint moment tended to fully recover to baseline (19, 27). These disparate results in the recovery of passive moment may be explained by the different mechanical effects of these interventions on MTC tissues. The present data clearly indicate that the reduction in passive joint moment after MVICs was attributable to a decreased tendon stiffness, which remained apparent after 30 min. However, moderate-duration (<5-min) stretch interventions (27, 41) have resulted in no change in tendon stiffness, with decreases in passive moment suggested to originate from a decreased muscle stiffness. Furthermore, the reduction in muscle stiffness appears to be transient, dissipating after 30 min, indiacting only a temporary effect of stretch on muscle stiffness. The continued depression of tendon stiffness after 30 min of MVICs, while concentric force recovered, may have important prophylactic injury implications, inasmuch as tendon stiffness (24) and muscle force (46) have been implicated in muscle strain injury risk. Further research is required to determine the long-term effects of MVICs on force production, MTC mechanics, and muscle strain injury risk.

In summary, the present study is the first to specifically examine the effects of prestretch MVICs as part of a comprehensive warm-up to determine the subsequent effect of stretch on joint moment, neuromuscular activity, and MTC mechanics. Significant reductions in tendon stiffness, active (concentric) and passive plantar flexor joint moment, and EMG amplitude occurred after the MVIC intervention. This was unlikely to result from local muscle fatigue, inasmuch as there was no reduction in isometric force. Significant correlations were found between the reductions in joint moment and decreases in
EMG amplitude, indicating that a substantial proportion of the reduction in force (~81%) could be attributed to a reduced neuromuscular activity. Although tendon stiffness was decreased, reductions in muscle operating length were clearly not a mechanism implicated in reductions of active force. An important finding of the present study was that no significant decrease in active or passive joint moment, EMG, or MTC mechanics was evident when the MTC was then stretched passively for 3 min. This suggests that the use of MVICs in a warm-up routine might mitigate the widely reported negative effects of moderate-duration (<3-min) stretch within the plantar flexors, but only because concentric force was already depressed. This finding has important implications for research and warm-up intervention designs. The significant increase in active joint moment and EMG amplitude 30 min after stretch suggests that physical tasks requiring high levels of plantar flexor muscle force are unlikely to be compromised at this time. However, decreased tendon stiffness and reduced passive joint moment remained and may have important implications for injury risk for the TS-Achilles MTC.

REFERENCES


