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ABSTRACT

KAY, A. D., J. HUSBANDS-BEASLEY, and A. J. BLAZEVICH. Effects of Contract–Relax, Static Stretching, and Isometric Contractions on Muscle–Tendon Mechanics. Med. Sci. Sports Exerc., Vol. 47, No. 10, pp. 2181–2190, 2015. Introduction: Loading characteristics of stretching techniques likely influence the specific mechanisms responsible for acute increases in range of motion (ROM). Therefore, the effects of a version of contract–relax (CR) proprioceptive neuromuscular facilitation stretching, static stretching (SS), and maximal isometric contraction (Iso) interventions were studied in 17 healthy human volunteers. Methods: Passive ankle moment was recorded on an isokinetic dynamometer, with EMG recording from the triceps surae, simultaneous real-time motion analysis, and ultrasound-imaging-recorded gastrocnemius medialis muscle and Achilles tendon elongation. Subjects then performed each intervention randomly on separate days before reassessment. Results: Significant increases in dorsiflexion ROM (2.5°–5.3°; P < 0.01) and reductions in whole muscle–tendon stiffness (10.1%–21.0%; P < 0.01) occurred under all conditions, with significantly greater changes detected following CR stretching (P < 0.05). Significant reductions in tendon stiffness were observed after CR stretching and Iso (17.7%–22.1%; P < 0.01) but not after SS (P > 0.05), whereas significant reductions in muscle stiffness occurred after CR stretching and SS (16.0%–20.5%; P < 0.01) but not after Iso (P > 0.05). Increases in peak passive moment (stretch tolerance) occurred after Iso (6.8%; P < 0.05), CR stretching (10.6%; P = 0.08), and SS (5.2%; P = 0.08); no difference in changes between conditions was found (P > 0.05). Significant correlations (r ranged from 0.69–0.82; P < 0.01) were observed between changes in peak passive moment and maximal ROM under all conditions. Conclusions: Although similar ROM increases occur after Iso and SS, changes in muscle and tendon stiffness are distinct. Concomitant reductions in muscle and tendon stiffness after CR stretching suggest a broader adaptive response that likely explains its superior efficacy in acutely increasing ROM. Although mechanical changes appear tissue-specific between interventions, similar increases in stretch tolerance after all interventions are strongly correlated with changes in ROM. Key Words: PROPRIOCEPTIVE NEUROMUSCULAR FACILITATION, RANGE OF MOTION, TENDON STIFFNESS, STRETCH TOLERANCE, ULTRASONOGRAPHY

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aximal joint range of motion (ROM) and resistance to joint rotation within that range (i.e., resistance to stretch) are important physical characteristics influencing one’s capacity to perform activities of daily living and athletic tasks (34) and are affected considerably by aging (4) and disease (10). Nonetheless, although muscle stretching is commonly practiced, relatively little is known about the underlying mechanisms that influence ROM, in particular, or its change in response to acute and chronic muscle stretching training. Despite static stretching (SS) being the most commonly used stretching mode, proprioceptive neuromuscular facilitation (PNF) stretches are regularly reported as being more effective for increasing ROM (25,27). The distinctive characteristic of PNF is that a brief (sometimes maximal) isometric contraction (Iso) is performed while the muscle is held on stretch (1). Two common methods of PNF stretching are the contract–relax (CR) and CR agonist contract techniques (37). The CR method includes an SS phase followed by an intense Iso of the stretched muscle, immediately followed by a further stretching phase. The CR agonist contract method requires an additional contraction of the agonist (i.e., opposing the muscle group being stretched) muscle during the stretch, before the subsequent additional stretch of the target muscle. However, despite these techniques being commonly employed in clinical environments to achieve rapid increases in ROM, they are not commonly used in athletic warm-up routines possibly because they normally require an assisting partner, may be painful, and may pose greater muscle strain injury risk compared with SS (5). Despite their efficacy, limited data have described the specific underlying mechanisms associated with changes in ROM following these modes of stretch, which is problematic because determining mechanisms may...
allow researchers to determine a priori whether these interventions may be useful in different clinical populations, to understand why such stretch interventions elicit different responses in different individuals, and to offer information that allows us to modify the techniques to optimize/improve acute and chronic responses to stretching.

Two neuromuscular mechanisms have been traditionally theorized to underpin the significant improvements in ROM achieved through CR stretching: autogenic inhibition and gate control theory (13). Autogenic inhibition may occur during the contraction phase of CR stretching, as increased activity from Type I muscle afferent fibers within Golgi tendon organs acts to hyperpolarize the dendritic ends of spinal α-motoneurons of the stretched muscle. This output could reduce the effectiveness of homonymous Type Ia muscle afferent output during stretch, inhibiting the activation of the α-motoneuron pool, thus possibly enabling further increases in ROM (1,36).

Intuitively, a reduction in α-motoneuron pool activity may enable further increases in ROM, but there is no direct evidence of a causal relationship. However, Golgi tendon organ activity substantially decreases or ceases once the contraction has terminated, with several studies reporting increased resting EMG activity immediately following the contraction phase of CR stretching (25,32). Thus, autogenic inhibition is unlikely to be the primary underlying mechanism explaining either the increases in ROM or the superiority of CR stretching in increasing ROM above other stretching modalities (37). Although recent reviews have generated ambiguity over the involvement of autogenic inhibition (13,37), other inhibitory neurological mechanisms may explain the efficacy of CR stretching (38). Gate control theory suggests that increased output from Type III muscle afferents during the contraction phase of CR stretching could inhibit pain perception (28).

Pressure receptors have larger myelinated neurons and connect to the same spinal interneurons within the spinal horn as unmyelinated nociceptive fibers (Type IV afferents) (31); therefore, increased activity could theoretically dampen pain perception and enable further increases in ROM (37). However, increased peak passive torque at full volitional ROM, indicative of dampened pain perception or increased stretch tolerance (i.e., the capacity to tolerate increased loading before terminating the stretch), has also been commonly reported following SS (27,39). Thus, an increase in the ROM at which stretch sensation, discomfort, or pain perception is perceived or tolerated (i.e., stretch tolerance) is a common characteristic across stretch modalities and may not explain the superior ROM outcomes associated with CR stretching.

Acute increases in ROM following a single static (passive) muscle stretching session are frequently reported with concomitant reductions in muscle–tendon complex (MTC) stiffness (16,17,22,24,26,33), reduced neuromuscular reflex response (2,3), and increased stretch tolerance (27,39). Therefore, despite the relatively lower levels of tissue loading imposed during SS compared with CR stretching, mechanical changes in musculotendinous tissues are notable and may underpin the increases in ROM or at least influence receptor activity and/or these afferent pathways. Although dynamometry-based passive moment data are often used in the quantification of MTC stiffness, ultrasonography provides the opportunity to examine the influence of stretch on specific tissues, although relatively few studies have employed this methodology in vivo during muscle stretching. During stretching, both muscular and tendinous tissues experience deformation (i.e., strain); however moderate-duration SS (3–5 min) has been reported to reduce muscle stiffness without influencing Achilles tendon stiffness (17,33), which is indicative of a muscle-based response underpinning increases in ROM. However, acute reductions in tendon stiffness have been reported following repeated maximal isometric (18,20) and concentric (19) contractions, where relatively greater tissue loading occurs within the tendon. Collectively, data from these studies suggest that the intensity and location of tissue strain may influence the change in tendon stiffness and, thus, the specific site of mechanical changes in the MTC. It may be hypothesized, therefore, that CR stretching might impose significant strain on both muscle (because of MTC stretch) and tendon (during the muscle contraction phase), offering a unique stimulus for decreases in both muscle stiffness and tendon stiffness. These may be directly (by reducing stiffness) or indirectly (through alterations in afferent feedback) associated with the reductions in resistance to stretch and increased ROM after CR stretching. Despite this possibility, testing of mechanical theories associated with the increased ROM following CR stretching has been limited to examinations of viscoelastic stress relaxation and creep responses (13), with no studies examining the acute effects of CR stretching on muscle and tendon stiffness.

The aims of the present study were to examine the influence of a version of CR stretching (i.e., MTC stretch plus muscle contraction), SS (i.e., MTC stretch only), and maximal Iso (i.e., muscle contraction causing tendon stretch) on dorsiflexion ROM, the slope of the passive joint moment curve (MTC stiffness), maximal passive joint moment at full volitional ROM (stretch tolerance), gastrocnemius medialis (GM) muscle stiffness, and triceps surae EMG activity measured during a passive joint stretch. The acute effects of these interventions on Achilles tendon stiffness, maximal isometric plantar flexor joint moment, and peak triceps surae EMG activity during maximal Iso were also measured. We tested the hypothesis that CR stretching would produce significantly greater increases in ROM and stretch tolerance while reducing muscle and tendon stiffness, whereas SS would influence only muscle stiffness and Iso would influence only tendon stiffness.

**MATERIALS AND METHODS**

**Subjects**

Seventeen recreationally active participants (nine women and eight men; mean ± SD age, 25.6 ± 8.8 yr; mean ± SD mass, 74.8 ± 11.8 kg; mean ± SD height, 1.7 ± 0.1 m) with no recent history of lower limb injury or illness volunteered for the study after providing a written informed consent...
form. The subjects were asked to avoid intense exercise, muscle stretching, and stimulant use for 48 h before testing. Ethical approval was granted by The University of Northampton’s Ethics Committee, and the study was completed in accordance with the Declaration of Helsinki.

Protocol

Overview. Subjects were fully familiarized with the testing protocols 1 wk before data collection, and they then visited the laboratory on three further occasions under experimental conditions, with trials conducted in randomized order separated by 1 wk. During the experimental trials, the subjects performed a warm-up for 5 min on a Monark cycle at 60 rpm, with a 1-kg resistance load. The subjects were then seated on the chair of an isokinetic dynamometer (Biodex System 3 Pro; IPRS, Suffolk, UK) with the knee fully extended (0°) to ensure that all plantar flexor components were influenced by the interventions and contributed significantly to passive and active joint moments (15). The foot was then strapped to the dynamometer footplate in the anatomical position (0°), with the sole of the foot perpendicular to the shank and with the lateral malleolus aligned with the center of rotation of the dynamometer. Nonelastic Velcro strapping was used to minimize heel displacement from the dynamometer footplate during passive and active trials to provide reliable and valid ROM and passive moment data during the passive trials (33). To confirm that the degree of ankle fixation did not substantially influence the passive moment data during measurements, one highly experienced analyst conducted all trials in order to remove intertester variability. To further confirm the reliability of these methods, we measured the day-to-day reliability of passive moment before each intervention (pretest data); analysis of the data indicated very high reliability (intraclass correlation coefficient (ICC), 0.95; SE, 3.0%). The subjects then performed maximal isometric plantar flexor contraction to determine maximal isometric joint moment and peak EMG activity (root-mean-squared (RMS) amplitude; described later). This was followed 2 min later by three passive dorsiflexion rotations initiated from 20° plantarflexion through to full dorsiflexion at 0.087 rad s⁻¹ (5° s⁻¹) to determine dorsiflexion ROM and peak passive moment at full ROM (stretch tolerance). Two minutes after completing the passive trials, the subjects performed one of three interventions (CR stretching, SS, or Iso; specific details provided later). Two minutes after completing the intervention, the subjects repeated the passive and active trials.

Dynamometry data. Subjects were seated on the dynamometer chair with the hip flexed to 55°, knee fully extended (0°), and ankle in the anatomical position (0°). The subjects then produced a ramped maximal isometric plantar flexor contraction, with maximal joint moment reached ~3 s after contraction initiation and held for 2 s (i.e., there was a visible plateau in moment trace), followed by an identical dorsiflexor contraction. The ramped plantar flexor contraction allowed maximal strength to be determined but also enabled tendon deformation to be captured using sonography, which allowed tendon stiffness to be calculated when combined with joint moment data (17–19). To confirm that the loading rate during ramped contraction did not influence tendon stiffness, the subjects repeated ramped contractions using visual feedback during the familiarization session until they had reliably achieved a linear increase in joint moment reaching maximal voluntary contraction (MVC) after ~3 s. During ramped contractions in the experimental trials, the time interval between 50% MVC and 90% MVC (the range over which tendon stiffness was calculated) was recorded in the preintervention and postintervention sessions. No significant difference (pre, 2.1 ± 0.1 s; post, 2.0 ± 0.1 s; P > 0.05) in the 50%–90% MVC time interval (indicative of tendon loading rate) was found. Two minutes after the subjects had completed isometric tests, their ankles were passively rotated through their full ROM at 0.087 rad s⁻¹ (5° s⁻¹) until they voluntarily terminated the rotation by pressing a handheld release button at the point of discomfort (6, 7). The passive rotations were performed three times, with the slope of the passive moment curve (indicative of MTC stiffness), peak passive moment (stretch tolerance), and ROM data measured from the third trial to ensure that muscular thixotropic properties did not influence the data. The slope of the passive moment curve was calculated as the change in plantar flexor moment through the final 10° of dorsiflexion (in the linear portion of the passive moment curve) in the prestretching trials; these identical joint angles were used in poststretching analysis. Joint moment and angle data were directed from the dynamometer to a high-level transducer (model HLT100C; Biopac, Goleta, CA) before analog-to-digital conversion at a 2000-Hz sampling rate (model MP150 Data Acquisition; Biopac). The data were then directed to a personal computer running AcqKnowledge software (version 4.1; Biopac) and filtered with a zero-lag, 6-Hz Butterworth low-pass filter before maximal ROM and passive joint moment were determined. Peak passive moment was measured within a 250-ms epoch at full volitional ROM.

EMG recording. Electrode site preparation, electrode placement, and EMG sampling, processing, and normalization methods were completed as described previously (17–19). EMG activities of GM, gastrocnemius lateralis, soleus, and tibialis anterior were monitored using skin-mounted bipolar double-differential active electrodes (model MP-2A; Linton, Norfolk, UK). EMG signals were preamplified by the electrode (gain, 300; input impedance, 10 GΩ; common-mode rejection ratio, >100 dB at 65 Hz) and then directed to a high-level transducer (model HLT100C; Biopac) before analog-to-digital conversion at a 2000-Hz sampling rate (model MP150 Data Acquisition; Biopac). The EMG signals were then directed to a personal computer running AcqKnowledge software (version 4.1; Biopac), filtered using a 20- to 500-Hz bandpass filter, and then converted into RMS EMG data with a 250-ms sample window. RMS EMG data were then normalized as a percentage of the peak amplitude recorded.
during the first maximal voluntary Iso. The normalized EMG amplitude was used as a measure of neuromuscular activity during the active trials (volitional activity) and at the end of ROM during the passive trials (reflexive activity), with antagonist tibialis anterior EMG data processed and normalized using the same method. During the active and passive trials, EMG activity was measured within a 250-ms epoch at peak joint moment and full volitional ROM, respectively.

**Muscle and tendon stiffness and elongation.**

**Motion analysis.** Real-time motion analysis using four infrared digital cameras (ProReflex; Qualysys, Gothenburg, Sweden) and operating Track Manager 3D software (version 2.0; Qualysys) were used to record the movement of infrared reflective markers during the trials. Using methods previously described (17–19) to calculate Achilles tendon and GM muscle length and elongation, we placed reflective markers over the insertion of the Achilles at the calcaneus (Fig. 1, marker A) and on the distal edge of the ultrasound probe positioned over the GM–Achilles muscle–tendon junction (MTJ) (Fig. 1, marker B). A third marker was placed over the origin of the medial head of the gastrocnemius at the medial femoral epicondyle (Fig. 1, marker C). Raw coordinate data were sampled at 100 Hz and smoothed using a 100-ms averaging window before the calculation of Achilles tendon length and GM muscle length.

**Ultrasound.** Real-time ultrasound images (LOGIQ Book XP; General Electric, Bedford, UK) were recorded at 28 Hz using a wide-band linear probe (8 L-RS; General Electric) with a 39-mm-wide field of view. Coupling gel (Ultrasound gel; Dahlhausen, Cologne, Germany) was used between the probe and the skin to image the GM–Achilles MTJ (Fig. 2). The probe was positioned perpendicular to the skin with zinc oxide adhesive tape to ensure consistent imaging of the MTJ during the trials. The distance between the MTJ and the distal edge of the ultrasound image was manually digitized (LOGIQ Book XP; General Electric).

**Calculations.** A 5-V ascending transistor–transistor logic pulse triggered the capture of ultrasound data (preceding

15 s of data), ended the capture of motion analysis data, and simultaneously placed a pulse trace on AcqKnowledge software (version 4.1; Biopac) to synchronize motion analysis, ultrasound, and dynamometer data. Tendon length was calculated as the sum of the distance between reflective markers A and B (using motion analysis) and the distance from the actual MTJ position to the distal border of the image (using ultrasound), using a method similar to that previously reported (17), where MTJ distance was measured to a hypoechoic area (beneath a tape affixed to the skin) in the image. Digitization of the position of the MTJ to the edge of the ultrasound image was employed as the ultrasound probe was permanently fixed to the skin for the duration of the test; thus, reliable positioning was ensured. Furthermore, removal of the tape affixed to the skin eliminated the hypoechoic area in the ultrasound image, enabling the MTJ to remain clearly visible throughout the recording, thus improving MTJ digitization. Tendon stiffness was calculated as the change in plantar flexor moment from 50% MVC to 90% MVC divided by the change in tendon length (N·mm·m–1). Muscle length was calculated as the distance between reflective markers B and C (using motion analysis) minus the distance from the actual MTJ position to the distal border of the image. Muscle stiffness was calculated as the change in plantar flexor moment through 10° of dorsiflexion (in the linear portion of the passive moment curve) divided by the change in muscle length (N·mm·m–1).

**Interventions.** Two minutes after completing the passive ROM trials, subjects performed one of three interventions. Under SS conditions, the ankle was passively rotated at 0.087 rad·s–1 through to full ROM until the subject reached the point of discomfort—a position regularly used in stretch studies (16,17). Movement velocity was too slow to elicit a significant myotatic stretch reflex response (29,30), which
ensured that full ROM was achieved and substantial stress was applied to the MTC. This ensured that the moment recorded was considered reflective of the passive properties of the plantar flexors. The subject’s ankle was held in the stretched position for 15 s and then released, returning the foot to 20° plantarflexion.

The stretch protocol was then repeated three times with 15-s rests, giving a total stretch duration of 60 s. Such stretch durations are likely to be achievable in clinical and other contexts, and previous research has shown that significant increases in ROM (35,40) and decreases in MTC stiffness (16,40) result from stretches of equal and lesser duration. During subsequent stretches, the subject was encouraged to stretch to a greater joint angle to ensure that substantial stress was imposed on the tissues and to more accurately reflect current stretching practices. CR stretching was performed using methods similar to those used in SS, except that the stretch was held passively for 10 s followed immediately by a 5-s ramped maximal Iso. Although—in traditional CR stretching—the muscle would be immediately stretched to its new ROM before the stretch and contraction phases are repeated, in the present study, the subject’s limb was returned to the anatomical position to allow for similar rest periods between stretches across interventions. After 15 s of rest, the protocol was repeated three times. Under isometric conditions (Iso), the ankle was passively rotated at 0.087 rad s⁻¹ until it had reached the anatomical position (0°), where it was held for 10 s before a 5-s ramped maximal Iso (i.e., identical to the contraction performed during CR stretching) was performed. After 15 s rest, the protocol was repeated three times. Two minutes after each intervention, the passive and active tests were repeated to determine the influence of the interventions on dorsiflexion ROM, the slope of the passive joint moment curve (MTC stiffness), maximal passive joint moment (stretch tolerance), Achilles tendon and GM muscle stiffness, and maximal isometric plantar flexor joint moment and triceps surae EMG activity.

Data Analysis

All data were analyzed using SPSS statistical software (version 17.0; LEAD Technologies, Chicago, IL); condition data are presented as mean ± SE, and change data are presented as mean ± SD. The study protocol included three interventions: CR stretching, SS, and Iso. Normal distribution for pregrou and postgroup data in all variables was assessed using Kolmogorov–Smirnov and Shapiro–Wilk tests; no significant difference (P > 0.05) was detected in any variable, indicating that all data sets were normally distributed. Separate repeated-measures multivariate ANOVA were used to test for differences between preintervention and postintervention data in 1) peak isometric moment and EMG; 2) ROM and peak passive moment (stretch tolerance); and 3) the slope of the passive joint moment curve (MTC stiffness) and GM muscle and Achilles tendon stiffness. Where significant differences were detected, separate repeated-measures ANOVA were used to test for differences in absolute change score data between interventions. Post hoc t-tests with Bonferroni correction were used to further examine changes in measures where statistical significance was reached. Normal distribution was also examined for change score data in all variables using Kolmogorov–Smirnov and Shapiro–Wilk tests; a significant difference (P < 0.05) was detected for changes in ROM, but no significant difference (P > 0.05) was detected in any other variable. Spearman’s rank correlation coefficients (rₛ) were computed to quantify the linear relationship between changes in ROM and changes in peak passive moment (stretch tolerance) and the slope of the passive joint moment curve (MTC stiffness) under each condition. Statistical significance for all tests was set at P < 0.05.

Reliability

Test–retest reliability was determined for peak isometric moment, peak passive moment (stretch tolerance), ROM, slope of the passive moment curve (MTC stiffness), muscle stiffness, and tendon stiffness in pretest data across conditions. No significant difference was detected between mean values (P > 0.05) for any measure (ICC, 0.89, 0.97, 0.97, 0.95, 0.80, and 0.96). The coefficients of variation and SE (expressed as percentage of the mean) were 9.5% (SE, 2.3%), 7.8% (SE, 1.9%), 4.4% (SE, 1.1%), 12.4% (SE, 3.0%), 11.1% (SE, 2.7%), and 4.4% (SE, 1.1%), respectively, for the abovementioned variables.

Sample Size

Effect sizes (Cohen’s d) were calculated from mean changes in variables (ROM, muscle and tendon stiffness, and peak passive moment) from previous studies employing similar methods (17,18,33). To ensure adequate statistical power for all analyses, we conducted power analysis for tendon stiffness (the variable with the smallest effect size) using the following parameters: variable, tendon stiffness; power, 0.80; α = 0.05; effect size, 0.95; attrition, 20%. Analysis revealed that the initial sample size required for statistical power was 15; thus, 20 subjects were recruited to account for possible attrition. Three subjects withdrew from the study because of nonrelated injuries. Statistical analyses were conducted on data sets for 17 subjects who completed the testing.

RESULTS

Range of motion. A significant increase in dorsiflexion ROM (Fig. 3) was found after CR stretching (5.3° ± 4.6°; P < 0.01) and SS (2.6° ± 3.5°; P < 0.01), as well as after Iso (2.5° ± 2.2°; P < 0.01). A significant difference (F = 4.3; P < 0.05) in changes in ROM was detected across the three interventions (Fig. 3). Post hoc analysis revealed significantly greater increases in ROM after CR stretching compared with both SS and Iso (P < 0.05) but no difference between SS and Iso (P > 0.05).

Stretch tolerance. A significant increase in peak passive moment (measured at full volitional ROM) was found...
after Iso (6.8 ± 10.2%; P < 0.05), but the change after CR stretching (10.6 ± 18.8%; P = 0.08) and SS (5.2 ± 16.8%; P = 0.08) did not reach statistical significance. Nonetheless, no difference in changes in peak passive moment was found among the three conditions (P > 0.05). Significant correlations (Fig. 4) between changes in ROM and changes in peak passive moment after CR stretching (r_s = 0.80; P < 0.01), SS (r_s = 0.82; P < 0.01), and Iso (r_s = 0.69; P < 0.01) were observed, suggesting that changes in ROM were associated with changes in the peak torque tolerated after each intervention.

**MTC stiffness.** Significant reductions in the slope of the passive joint moment curve were found after CR stretching (21.0% ± 11.3%; P < 0.01), SS (10.1% ± 12.2%; P < 0.01), and Iso (10.1% ± 11.8%; P < 0.01), indicating a significant reduction in MTC stiffness (Fig. 5). A significant difference (F = 4.9; P < 0.05) in the reductions in passive moment across the three interventions was detected. Similar to ROM changes, post hoc analysis revealed significantly greater reductions in passive moment after CR stretching compared with both SS and Iso (P < 0.05), but there was no difference in changes following SS and Iso (P > 0.05). As the mean changes in MTC stiffness after SS plus Iso were almost arithmetically equal to changes following CR stretching, we compared the changes in stiffness after CR stretching with SS plus Iso, where a significant correlation was detected (r_s = 0.66; P < 0.01). No significant correlations were observed between reductions in MTC stiffness and increases in ROM after CR stretching, SS, or Iso intervention (P > 0.05).

**Achilles tendon stiffness and GM muscle stiffness.** Significant reductions in tendon stiffness (Fig. 6A) were found after CR stretching (22.1 ± 24.1%; P < 0.01) and Iso intervention (17.7 ± 20.8%; P < 0.05), but not after SS (1.7 ± 8.2%; P > 0.05). No difference in reduction in tendon stiffness was found between CR stretching and Iso intervention (P > 0.05). Significant reductions in muscle stiffness (Fig. 6B) were also found after CR stretching (20.5% ± 8.9%; P < 0.01) and SS (16.0% ± 12.3%; P < 0.01), but not after Iso intervention (3.0% ± 7.0%; P > 0.05). No difference in reduction in muscle stiffness was found between CR stretching and SS (P > 0.05).

**Maximal isometric plantar flexor moment and EMG.** No significant difference in maximal isometric plantar flexor moment or EMG activity (during maximal contraction or passive rotation at full ROM) was found following any
generating capacity and reflexive muscle activity were retained 

DISCUSSION 

intervention (P > 0.05), indicating that neuromuscular force generating capacity and reflexive muscle activity were retained after all interventions.

Increases in ROM immediately following muscle stretching have been largely attributed to either increases in stretch tolerance (27,38,39) or changes in the mechanical properties of the MTC (22,24,26,33), although few studies have employed the requisite methodology to localize tissue-specific changes within the MTC. In the present study, significantly greater increases in ROM and reductions in the passive moment measured at predetermined joint angles during a plantar flexor stretch were observed following acute CR stretching compared to static (passive) stretching or maximal Iso, in agreement with our hypothesis. In addition, we observed moderate to strong correlations between increases in ROM and increases in peak passive joint moment (r = 0.80; P < 0.01), which is considered an indication of greater “stretch tolerance,” after CR stretching. Regarding mechanical changes, both muscle stiffness and tendon stiffness were reduced following CR stretching, whereas SS influenced only muscle stiffness and Iso influenced only tendon stiffness. In fact, the total decrease in MTC stiffness after CR stretching (~21%) was almost arithmetically equal to the changes in MTC stiffness after SS (~10%) plus Iso (~10%). The concomitant reductions in muscle and tendon stiffness after CR stretching are consistent with previous studies, where reductions in muscle stiffness were reported after SS (17–19,33) yet reductions in tendon stiffness were reported after maximal contractions (18–20), perhaps indicating that the separate effects of SS and Iso were achieved by the singular imposition of CR stretching.

To determine whether the changes in MTC stiffness following CR stretching could be explained by changes experienced following SS and Iso, we compared the changes in MTC stiffness after CR stretching with the summed changes in MTC stiffness following SS and Iso, revealing a significant correlation (r = 0.66; P < 0.01). Despite the significant correlation, more than 50% of the changes in stiffness remain unexplained with this method; thus, the separate loading strategies of SS and Iso do not appear to fully explain the changes in stiffness following the CR stretching method included within the study. A more complex testing model—using a range of stretch and contraction intensities and durations to explore the relationship of the magnitude of separate and concurrent changes in muscle and tendon stiffness with changes in MTC stiffness—may provide a more comprehensive assessment of this relationship. The present methods used motion analysis, in addition to ultrasonography, to correct for possible ankle rotation during the ramped Iso, overestimating tendon length change measurements and compromising stiffness calculations. However, a possible limitation of this method is that a linear three-dimensional motion analysis model using two reflective markers was used to calculate Achilles tendon length. A curved path using multiple reflective markers may more accurately reflect Achilles tendon length (11), with substantial error introduced when measurements are taken over multiple joint angles. However, error with the linear model is negligible in the anatomical position (0.8%; ref 11); thus, any error in tendon length is likely minimal in the present study. Furthermore, acute changes in stiffness and test–retest reliability data were very high (ICC, 0.95) in the present study; thus, we are confident that the present methods accurately captured changes in stiffness. These are the first data to confirm that CR stretching acutely influences both muscle stiffness and tendon stiffness, which is indicative of a broader adaptive response that offers a possible new mechanism for the reported superiority of CR stretching for acute ROM enhancement and reduction in resistance to stretch.

Historically, autogenic inhibition has been theorized as an important mechanism explaining the superior effects of CR stretching for acute ROM enhancement (1,36). Increased activity of Type Ib muscle afferents during the contraction phase was thought to hyperpolarize the dendritic ends of spinal α-motoneurons of the stretched muscle, minimizing or removing the influence of stretch-induced Type-Ia-mediated reflexive activity (29,30). However, this mechanism is unlikely in the present study, as the low velocity of joint rotation during stretching was imposed in an attempt to minimize or remove Type-Ia-mediated reflexive activity (30) and to isolate tissue mechanical responses as a possible underlying mechanism. The lack of any substantial EMG activity (<5% MVC) at full ROM in both preintervention and postintervention data or any significant pre-to-post change in EMG at

FIGURE 6—Achilles tendon stiffness and GM muscle stiffness preintervention and postintervention. Significant reductions in tendon stiffness (A) were found after CR stretching (22.1%) and Iso (17.7%), but not after SS (1.7%). No difference in the reductions in tendon stiffness was found between CR stretching and SS. Significant reductions in muscle stiffness (B) were found after CR stretching (20.5%) and SS (16.0%), but not after Iso (3.0%). No difference in the reductions in muscle stiffness was found between CR stretching and SS. *Significant at P < 0.05. # Significant at P < 0.01.
full ROM is indicative of minimal Type Ia or Ib reflexive involvement influencing maximal ROM or poststretch changes in ROM. These data are similar to those reported in previous acute CR stretching studies where EMG magnitude was unchanged or even increased at full ROM (25, 32); thus, autogenic inhibition is an unlikely mechanism explaining either the increase in ROM following CR stretching or the significantly greater increase in ROM compared with the other conditions. However, other neuromuscular adaptations contributing to the gains in ROM cannot be discounted (8). In fact, a neurological contribution is supported by the increase in peak passive moment (stretch tolerance) detected after CR stretching (10.6%). However, increases in peak passive moment were also detected after Iso (6.8%) and SS (5.2%) and, importantly, these increases were not significantly different between conditions. Furthermore, moderate to strong correlations ($r_c = 0.69–0.82; P < 0.01$) between changes in peak passive moment and changes in ROM were observed under each condition, indicative of altered Type III or IV afferent activity influencing pain perception and the magnitude of changes in ROM (27, 39). The present changes in peak passive moment are strong evidence that stretch tolerance is an important mechanism associated with acute increases in ROM, regardless of stretching mode; however, it is unlikely to explain CR stretching’s efficacy to acutely increase ROM compared to other stretching modes as similar changes were observed between conditions. Relatively low peak forces were applied to the MTC during SS (34.1 ± 4.2 Nm) compared to either CR stretching (151.7 ± 13.2 Nm) or Iso (123.3 ± 3.1 Nm). Despite this lower loading intensity, a significant increase in ROM and a reduction in MTC stiffness were observed, although these changes were less than those elicited by CR stretching. Interestingly, a reduction in muscle stiffness of a magnitude similar to that elicited by the CR stretching protocol was observed, which is in accordance with previously reported changes after SS (17–19, 33). Although both muscle and tendon deformed during the SS manoeuvre, the lower intensity of loading experienced during SS was more likely to cause muscle stretch rather than tendon stretch, as the tendon is inherently stiffer than relaxed muscle tissue during plantar flexor stretches with the knee extended (6, 14, 33). The majority of studies have reported no change in tendon stiffness following plantar flexor SS (16, 33), with few studies (9, 14, 22) reporting acute reductions in tendon stiffness with substantially longer stretch durations (5–20 min). Notwithstanding, no studies using shorter (i.e., <5 min) and potentially more practically/clinically relevant durations of SS have reported a reduction in tendon stiffness. Thus, the duration of stretch may be a key determinant of the likelihood and location of stiffness change within the MTC. The intensity of stretching likely influences acute responses, as greater changes in muscle stiffness are reported after constant torque versus constant angle stretching (12). However, our aim was to determine whether tendon loading (i.e., Iso and CR stretching) contributed to the increase in ROM; thus, identical stretching phases were performed across interventions (i.e., constant angle method). Continual ROM increases during the stretch phase (i.e., constant torque method) are difficult to control as some subjects may feel unable to increase ROM further, introducing differing levels of strain between conditions and compromising our ability to determine whether tendon loading influenced ROM. Furthermore, constant torque stretches produce a more intense and sometimes painful stretch that may not be suitable in sensitive populations (i.e., clinical or injured populations). However, to ensure that substantial stress was achieved during each stretch and to more closely reflect current practice, we encouraged the subjects to push each successive stretch to a greater joint angle as their stretch tolerance increased. Collectively, the findings of the present study and other studies point to an acute muscle-based adaptive response following moderate-duration SS as an important mechanism either directly (by reduced muscle stiffness) or indirectly (by altered afferent activity) underpinning the increases in ROM. As the duration of stretch or tissue loading may influence the likelihood and location of changes in tissue stiffness, an Iso intervention was used to specifically impose an isolated high-intensity loading to the tendon while reducing strain in the muscle. Substantially higher forces are transmitted through the tendon during Iso (21) compared to SS (18), resulting in greater tendon deformation (6). We hypothesized that this would increase the likelihood of changes in tendon stiffness. The Iso protocol was chosen in order to provide a similar level of tendinous tissue loading as the CR stretching intervention to determine the impact of tendinous stretch alone on changes in ROM. Significantly greater loading was observed during both Iso and CR stretching protocols compared with SS; however, loading during CR stretching was also significantly greater than in the isometric protocol. This is likely a consequence of gastrocnemius and soleus force–length properties, which would have operated on the ascending limb of their force–length curve in the present experiments (23, 24). Despite the difference in loading magnitude, a substantial reduction in tendon stiffness (~18%) was found after Iso, which was similar to the change found after CR stretching (~22%). However, the reduction in tendon stiffness after Iso occurred without a change in muscle stiffness and was associated with a similar and significant increase in ROM (~3°) as SS. The increase in ROM achieved with a concomitant reduction in tendon stiffness following the Iso intervention is a novel finding and provides further support for the concept that acute mechanical changes (from muscle stretching or muscular contractions) influence the increases in ROM. Despite similar increases in ROM being observed following both SS and Iso, the location of changes in tissue stiffness was clearly distinct, with increases in ROM poststretch being attributable to reductions in muscle stiffness but with increases in ROM following Iso being attributable to reductions in tendon stiffness. However, whether these muscle-based or tendon-based mechanical changes directly (reduced stiffness) or indirectly (altered afferent feedback) influenced ROM remains to be established. Regardless, the present findings have clear methodological implications, as performing
contractions in the anatomical position results in similar mechanical changes in tendon stiffness as CR stretching. Thus, modifying CR stretching to perform the contraction phase in the anatomical position, rather than in the highly stretched position, removes the need for partner assistance, decreases the likelihood of tissue damage and muscle strain injury (5), and results in a simpler technique that may be more widely used in clinical and athletic populations.

In summary, the present study is the first to examine the acute effects of muscle-dominant versus tendon-dominant tissue loading using CR stretching, static (passive) stretching, and maximal Iso on joint ROM, MTC stiffness, maximal passive joint moment (stretch tolerance), muscle and tendon stiffness, and EMG activity. Although significant increases in ankle joint ROM and reductions in MTC stiffness were evident after all interventions, the increase in ROM after CR stretching was significantly greater. Furthermore, reductions in stiffness were tissue-specific and distinct between interventions, with SS acutely reducing muscle stiffness, Iso reducing tendon stiffness, and CR stretching reducing both muscle stiffness and tendon stiffness. Clearly, the mode of tissue loading is an important determinant of changes in tissue stiffness, with SS being sufficient to reduce muscle stiffness but with Iso and CR stretching influencing tendon stiffness. The present data provide clear evidence that tissue-specific imaging is essential to determine the influence of such interventions on tissue-specific MTC properties and to enable possible underlying mechanisms associated with changes in ROM to be identified. The present data provide novel and compelling evidence for a mechanical mechanism underpinning acute changes in ROM after muscle stretching, with the greater efficacy of CR stretching in acutely increasing ROM likely being attributable to concomitant reductions in muscle and tendon stiffness. Notwithstanding the clear mechanical differences between conditions, significant correlations between changes in peak passive moment and changes in ROM were also observed under all conditions. This result is considered good evidence that a neurological adaptation (i.e., increased stretch tolerance) is also important for acute increases in ROM.

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