

TITLE

Acute effects of contract-relax (CR) stretch versus a modified CR technique

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ABSTRACT

Purpose: Contract-relax (CR) stretching increases range of motion (ROM) substantively, however its use in athletic environments is limited as the contractions performed in a highly stretched position require partner assistance, are often painful, and may induce muscle damage. Therefore, the acute effects of performing the contractions ‘off stretch’ in the anatomical position (stretch-return-contract [SRC]) were compared with traditional CR stretching in 14 healthy human volunteers. **Methods:** Passive ankle joint moment and dorsiflexion ROM were recorded on an isokinetic dynamometer with electromyographic monitoring of the triceps surae, whilst simultaneous real-time motion analysis and ultrasound imaging recorded gastrocnemius medialis muscle and Achilles tendon elongation. The subjects then performed CR or SRC stretches (4 × 10-s stretches and 5-s contractions) randomly on separate days before reassessment. **Results:** Significant increases in dorsiflexion ROM (4.1-4.0°; $P<0.01$) and peak passive moment (10.9-15.1%; $P<0.05$) and decreases in the slope of the passive moment curve (19.1-13.3%; $P<0.05$), muscle stiffness (21.7-21.3%; $P<0.01$) and tendon stiffness (20.4-15.7%; $P<0.01$) were observed in CR and SRC, respectively. No between-condition differences were found in any measure ($P>0.05$). **Conclusions:** Similar mechanical and neurological changes were observed between conditions, indicating that identical mechanisms underpin the ROM improvements. These data have important practical implications for the use of this stretching mode in athletic environments as performing the contractions ‘off stretch’ eliminates the pain response, reduces the risk of inducing muscle damage, and removes the need for partner assistance. Thus, it represents an equally effective, simpler, and yet potentially safer, stretching paradigm.

Keywords: Proprioceptive neuromuscular facilitation, range of motion, tendon stiffness, ultrasound.

INTRODUCTION

Both the maximal joint range of motion (ROM) and resistance to stretch during rotation (indicative of tissue stiffness) are important functional parameters that may affect muscle strain injury risk (Witvrouw et al. 2003), influence the capacity to perform activities of daily living (Mulholland et al. 2001), and are compromised with aging (Bassey et al. 1989) and disease (Duffin et al. 1999). Static muscle stretching is a commonly used technique to acutely improve ROM with these improvements thought to be attributable to several mechanisms, including reductions in tissue stiffness (Kay et al. 2015; Morse et al. 2008), altered peripheral (afferent) output (Avela et al. 1999, 2004), and dampened pain, pressure or stretch perception increasing stretch tolerance (i.e. the capacity to tolerate increased loading prior to terminating the stretch; Magnusson et al. 1996; Mitchell et al. 2007; Weppeler and Magnusson 2010). Despite the popularity of static stretching, proprioceptive neuromuscular facilitation stretching (PNF) is regularly reported as being the most effective stretching technique for acute and chronic improvements in ROM (Funk et al. 2003; Hindle et al. 2012). A common method of PNF stretching is the contract-relax (CR) technique (Sharman et al. 2006), which includes a static stretching phase for a prescribed duration, followed immediately by an intense, often maximal, isometric contraction performed in a fully stretched position. Upon completion of the contraction the joint is rotated further to again stretch the target muscle, with stretch intensity normally to the point of discomfort. While CR stretching is highly effective and often used in clinical environments to achieve rapid increases in ROM, it is not commonly used in athletic warm-up routines possibly because it normally requires an assisting partner, may be painful, and is thought to pose a greater muscle strain injury risk compared with static stretching (Beaulieu 1981).

Few studies have examined the underlying mechanisms associated with increases in ROM following CR stretching (Hindle et al. 2012; Kay et al. 2015), consequently these mechanisms remain essentially theoretical and poorly understood. Two neuromuscular mechanisms (autogenic inhibition, gate control theory) have been theorized (for review see Hindle et al. 2012). Regarding autogenic inhibition, a neuromuscular inhibition was thought to occur as the loading of the tendon during the contraction phase of CR activated/stimulated type Ib muscle afferent output from the golgi tendon organs, stimulating inhibitory spinal synapses and hyperpolarizing the dendritic ends of spinal α -motoneurons of the stretched muscle. The Ib activity would likely diminish the influence of homonymous Ia muscle afferents on the α -motoneuron pool of the stretched muscle, with the diminished reflex activity thought to allow further increases in ROM (Prentice 1983). However, several original studies have previously reported no change in electromyographic (EMG) magnitude at full ROM (Kay et al. 2015; Mitchell et al. 2009; Osternig et al. 1990), with reviews concluding autogenic inhibition was unlikely to be an

important mechanism underpinning the increase in ROM following CR stretching (Hindle et al. 2012; Sharman et al. 2006). Gate control theory posits that pressure receptors (type III afferents) activated during the contraction phase could inhibit pain perception (Mazzullo 1978), as pressure receptors are associated with larger myelinated neurons that connect to the same spinal interneurons as un-myelinated nociceptive fibers (type IV afferents) within the spinal horn (Melzack 1993). The increased activity of pressure receptors would theoretically diminish the influence of homonymous IV afferent output and pain perception, thus enabling further increases in ROM. While these neuromuscular pathways are theoretical, increased stretch tolerance (dampened pain perception) is commonly reported following CR stretching (Kay et al. 2015; Mitchell et al. 2009). Thus although autogenic inhibition has largely been discounted, a neurological contribution to the increased ROM following CR stretching is at least partly supported.

The distinct muscle-tendon (and joint) loading characteristics of various stretching methods likely result in different mechanical responses, with a key distinction between CR and other stretching techniques being the inclusion of an intense, often maximal, isometric contraction performed following the stretching phase and performed with the muscle remaining in a highly-stretched position. During passive ankle dorsiflexion stretches, more flexible subjects demonstrate greater tendon elongation with no detectable differences in the onset or magnitude of muscle activity toward the end of rotation or near full ROM (Blazevich et al. 2014), therefore tendon properties may, at least partly, influence maximum ROM. While muscular and tendinous tissues experience deformation during stretching (Blazevich et al. 2014; Morse et al. 2008), studies employing ultrasonography techniques have found muscle stiffness to be reduced after an acute bout of static stretching, whereas tendon stiffness remained unaltered (Kay & Blazevich 2009a; Morse et al. 2008). However, a recent study revealed that CR stretching acutely reduced both muscle and tendon stiffness and elicited significantly greater increases in ROM compared with a similar volume of static stretching after which only a reduction in muscle stiffness was induced (Kay et al. 2015). This broader acute adaptive response, where both muscle and tendon stiffness are influenced concurrently, offers a potentially important mechanism underpinning the superior efficacy of CR stretching for acutely increasing ROM when compared to other stretching techniques.

CR stretching is implemented to the aim of increasing ROM, often in an attempt to reduce muscle strain injury risk. However, paradoxically, performing intense muscular contractions in a highly-stretched position, where the muscle is vulnerable to injury, increases the risk of inducing tissue damage (Beaulieu 1981; Butterfield and Herzog

2006; Whitehead et al. 2003). Thus, the question should be asked whether the performance of isometric contractions in a non-stretched position *between* each passive static stretching cycle is as effective as performing the contractions *during* each passive static stretching cycle (i.e. contractions performed with the muscle in a highly-stretched position). Interestingly, several studies have reported acute reductions in tendon stiffness following maximal isometric contractions performed in the anatomical position (i.e. with the muscle off stretch; Kay & Blazevich 2009b; Kay et al. 2015; Kubo et al. 2002). Furthermore, a recent study reported concomitant increases in ROM and reductions in tendon stiffness following isometric contractions performed in the anatomical position (Kay et al. 2015), with the acute increase in ROM being similar to that observed following static stretching. Collectively, these findings suggest that substantial tendon loading, regardless of muscle length, should influence tendon stiffness and ROM. Consequently, modification of the CR stretching technique to perform the muscle contraction phase with the muscle ‘off stretch’ may provide a similar stimulus whilst reducing injury risk. Therefore, the aims of the present study were to examine the influence of an acute bout of CR stretching versus a modified CR technique (stretch-return-contract [SRC]; where the contractions are performed ‘off stretch’) on dorsiflexion ROM, maximal passive joint moment at full volitional ROM (stretch tolerance), the slope of the passive moment curve (indicative of whole muscle-tendon complex [MTC] stiffness), 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 a maximal isometric contraction were then measured. We tested the hypothesis that CR and SRC stretching techniques would produce similar increases in ROM and stretch tolerance whilst reducing muscle and tendon stiffness.

METHODS

Subjects

Fourteen recreationally active participants (8 women, 6 men; age = $26.1 \pm (\text{SD}) 9.6$ yr, height = 1.7 ± 0.1 m, and mass = 75.6 ± 13.3 kg) with no recent history of lower limb musculoskeletal injury or neurological deficit volunteered for the study after completing a pre-test medical questionnaire and giving written and informed consent. The subjects were asked to avoid any flexibility training, intense exercise and stimulant use for 48 h prior to testing. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee, and the study was completed in accordance with the Declaration of Helsinki.

Protocol

Overview

The subjects were familiarized with the testing protocol one week prior to data collection and then visited the laboratory on two further occasions under experimental conditions, with trials counterbalanced and separated by one week. During the experimental trials, the subjects performed a 5-min warm-up on a Monark cycle at 60 rpm with a 1-kg resistance load. The subjects were then seated in the chair of an isokinetic dynamometer (Biodex System 3 Pro, IPRS, Suffolk, UK) with the hip flexed to 55° and knee fully extended (0°) to ensure all plantar flexor muscles could be strongly activated and were at an appropriate length to contribute strongly to the total passive and active joint moments (Cresswell et al. 1995; Kawakami et al. 1998). The ankle was then placed in the dynamometer footplate in the anatomical position (0°) with the lateral malleolus aligned to the centre of rotation of the dynamometer. Non-elastic Velcro strapping was used to minimize heel displacement from the dynamometer footplate to provide reliable and valid ROM and passive moment data during the passive trials (Morse et al. 2008). To ensure that the degree of ankle fixation did not substantially influence the passive moment or ROM data during the pre- and post-intervention measurements, one highly experienced analyst conducted all trials to remove inter-tester variability.

Skin-mounted bipolar double differential active electrodes (model MP-2A, Linton, Norfolk, UK) were placed over the soleus (Sol), gastrocnemius medialis (GM), gastrocnemius lateralis (GL) and tibialis anterior (TA) muscles. Site preparation, electrode placement, EMG sampling, processing and normalization methods were completed as previously described (Kay & Blazevich 2009a). EMG amplitude was constantly monitored during the passive and active trials to quantify muscle activity (described later). As ankle rotation can occur during 'isometric' plantar flexion contractions performed in a dynamometer (Karamanidis et al. 2005), measurement of calcaneal movement (i.e. distal aspect of the Achilles tendon) using motion analysis was used to correct for any error induced by this joint rotation (Manal et al. 2013), which would otherwise have resulted in an overestimation of tendon length change. Four infrared digital cameras (ProReflex, Qualisys, Gothenburg, Sweden) enabled real-time motion analysis to record the movement of infrared reflective markers placed over the insertion of the Achilles at the calcaneus (see Fig. 2; *marker A*) and on the distal edge of the ultrasound probe positioned over the GM-Achilles muscle-tendon junction (MTJ) (*marker B*). A third marker was placed over the origin of the medial head of the gastrocnemius at the medial femoral epicondyle (*marker C*). Real-time ultrasound imaging (LOGIQ

Book XP, General Electric, Bedford, UK) was performed with a sampling frequency at 28 Hz using 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) to image the position (and excursion) of the GM-Achilles MTJ (see Fig. 3). The probe was positioned with the proximal end towards the origin of the medial head and the distal end towards the insertion of the Achilles tendon. The probe was then manipulated until the superficial and deep GM aponeuroses could be visualized to enable triangulation of the GM-Achilles tendon MTJ. The probe was then fixed to the skin with zinc-oxide adhesive tape to ensure consistent and accurate imaging of the MTJ during the experimental trials.

Active and passive trials

During the active trial the subjects were instructed to perform a 5-s ramped maximal isometric plantar flexor contraction to determine maximal isometric strength, EMG activity, and tendon stiffness (described later). To confirm that loading rate did not influence tendon stiffness, during the familiarization session, visual feedback of the plantar flexor joint moment was provided during the ramped contractions until the subjects reliably achieved a linear increase in moment reaching maximal voluntary contraction (MVC) after ~3 s. Furthermore, during the experimental trials the time taken by the subjects to increase plantar flexor moment from 50% to 90% MVC (the range that tendon stiffness was calculated) was recorded in the pre- and post-intervention sessions; no significant difference (pre = 2.1 ± 0.2 s, post = 2.0 ± 0.2 s; $P > 0.05$) was found. Two minutes later the subjects performed three passive dorsiflexion rotations initiated from 20° plantar flexion through to full dorsiflexion at $0.087 \text{ rad}\cdot\text{s}^{-1}$ ($5^\circ\cdot\text{s}^{-1}$) until the subject volitionally terminated the rotation by pressing a hand held release button at the point of discomfort.

Stretching Interventions

Two minutes after completing the passive ROM trials the subjects performed either the CR or SRC stretching intervention. During the CR condition the ankle was passively rotated at $0.087 \text{ rad}\cdot\text{s}^{-1}$ until reaching the point of discomfort, a position regularly used in stretch studies (Blazevich et al. 2012; Kay & Blazevich 2008, 2009a). The movement velocity was too slow to elicit a significant myotatic stretch reflex response, which ensured that full ROM was achieved and substantial stress was applied to the MTC (McNair et al. 2001). Furthermore, this ensured that moment data were reflective of the passive properties of the MTC. The subjects' ankles were held in the stretched position for 10 s and followed immediately with a 5-s ramped maximal isometric contraction (peak joint moment was obtained after ~3 s from contraction initiation and held for ~2 s) performed with the

muscle at full stretch (i.e. point of discomfort). Upon contraction cessation, the ankle was then immediately rotated again at $0.087 \text{ rad}\cdot\text{s}^{-1}$ until reaching the point of discomfort with the protocol repeated three further times giving a total duration of 60 s (i.e. $4 \times 10\text{-s}$ stretches and $4 \times 5\text{-s}$ contractions). The constant angle stretching method was chosen during the static stretching phase of the CR stretching technique as increasing ROM during the stretches (constant torque) may introduce differing levels of strain between conditions (Herda et al. 2014), which would have compromised our ability to determine whether muscle length during the contraction phase influenced ROM. Furthermore, the static stretch phase duration (10 s) was considered too short to enable further meaningful or reliable increases in ROM prior to the contraction phase. During the SRC protocol the static stretch phase was performed identically, however after the 10 s of stretching the ankle was returned to the anatomical position where the 5-s ramped maximal isometric contraction was performed. The ankle was rotated again $0.087 \text{ rad}\cdot\text{s}^{-1}$ until reaching the point of discomfort with the protocol repeated three times giving a total duration of 60 s. Two minutes later the subjects repeated the passive and active trials (see Fig. 1).

Measures

Plantar flexor moment and ROM

Maximal isometric plantar flexor moment was recorded pre- and post-intervention during the active trial to determine the influence of CR and SRC stretching on isometric strength. Peak isometric plantar flexor moment was also recorded during the four contractions performed during the CR and SRC interventions to determine the average peak loading during each intervention. Passive moment data were recorded from the third passive ROM trial to ensure thixotropic properties of the skeletal muscles did not influence the joint moment data (Proske and Morgan 1999). The passive rotation enabled ROM, peak passive moment (stretch tolerance), and the slope of the passive moment curve (indicative of MTC stiffness) to be recorded. The slope of the passive moment curve represents joint stiffness (i.e. all joint structures contribute to moment), however at the ankle the triceps surae contribute $>70\%$ to the total ankle joint moment (Murray et al. 1976), thus the slope may be considered primarily indicative of MTC stiffness. Peak passive moment was measured within a 250-ms epoch at full volitional ROM, with the slope of the passive moment curve 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 pre-stretching trials; identical joint angles were used in the post-stretching trial. Joint moment and dorsiflexion angle data were directed from the dynamometer to a high level transducer (model HLT100C, Biopac, Goleta, CA) before analogue-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 (v4.1, Biopac) and filtered with a zero lag, 6-Hz Butterworth low-pass filter.

Electromyographic (EMG) activity

Raw EMG signals from the Sol, GM, GL (i.e. the triceps surae muscle group) and TA (antagonistic muscle) were amplified (gain = 300, input impedance = 10 G Ω , common mode rejection ratio \geq 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) and stored on a personal computer running AcqKnowledge software (v4.1, Biopac). EMG signals collected during maximal volitional contractions as well as during muscle stretches were then processed using a 20- to 500-Hz band-pass filter and converted to root-mean-squared EMG with a moving symmetrical 250-ms averaging window. Additionally, a 10-ms averaging window was used on EMG data collected during muscle stretches in order to check for reflexive short-burst EMG activity that may have resulted from velocity-dependent stretch receptors (i.e. type Ia). The EMG data were then normalized as a percentage of the peak amplitude recorded during the first MVC in the pre-intervention active trial. The normalized EMG amplitude (%MVC) was used as a measure of neuromuscular activity quantified within a 250-ms epoch at peak joint moment during the active trials and at full volitional ROM during the passive trials.

Muscle and tendon length and stiffness

Ultrasound, motion analysis and dynamometry data were electronically synchronized using a 5-V ascending transistor-transistor logic (TTL) pulse. The TTL pulse simultaneously placed a marker on the AcqKnowledge (v4.1, Biopac) software while ending the capture of motion analysis and ultrasound data. Motion analysis data were then directed to and stored on a personal computer operating Track Manager 3D software (v.2.0, Qualisys). Raw coordinate data were sampled at 100 Hz and smoothed using a 100-ms averaging window prior to the calculation of Achilles tendon and GM muscle lengths. Tendon length was calculated as the distance between reflective *markers A* and *B* (using motion analysis), plus the distance from the actual MTJ position to the distal edge of the image (using ultrasound) in a method identical to that previously reported (Kay et al. 2015). Tendon stiffness was calculated as the change in plantar flexor moment from 50-90%MVC divided by the change in tendon length (Nm \cdot mm⁻¹). Muscle length was calculated as the distance between reflective *markers B* and *C* (using motion analysis), minus the distance from 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 ($\text{Nm}\cdot\text{mm}^{-1}$).

Statistical analysis

All data were analyzed using SPSS statistical software (v.20; LEAD Technologies Inc., USA) and are reported as means and 95% confidence intervals (CI). Normal distribution was assessed for pre- and post-group data 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. The effects of time were examined using separate multiple analyses of variance (MANOVA) with repeated measures to test for differences between pre- and post-intervention data in 1) peak isometric moment and EMG, and 2) ROM and peak passive moment (stretch tolerance), and 3) the slope of the passive joint moment curve (MTC stiffness), GM muscle stiffness and Achilles tendon stiffness. 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 peak passive moment in CR and SRC conditions and for ROM in the CR condition; no significant difference ($P > 0.05$) was detected in any other variable. Where significant differences were detected, paired t-tests were used to test for differences in absolute change score data between conditions. Spearman's rank correlation coefficients (r_s) were computed to quantify the linear relationship between the change in ROM and changes in peak passive moment (stretch tolerance) and the slope of the passive joint moment curve (MTC stiffness), muscle stiffness and tendon stiffness in each condition. Statistical significance for all tests was accepted at $P < 0.05$.

Reliability

Test-retest reliability was determined for peak isometric moment, peak passive moment, ROM, the slope of the passive moment curve (MTC stiffness) and muscle and tendon stiffness in the pre-test data in both conditions. No significant difference was detected between mean values ($P > 0.05$) for any measure; ICC's were 0.84, 0.93, 0.96, 0.97, 0.99, and 0.78. Coefficients of variation and SE (expressed as a percentage of the mean) were 13.4% (SE = 3.6%), 14.5% (SE = 3.9%), 5.1% (SE = 1.4%), 14.6% (SE = 3.9%), 11.1% (SE = 3.0%), and 11.6% (SE = 3.1%), respectively, for the above 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 (Kay & Blazevich 2009b; Kay et al. 2015; Kubo et al. 2002; Magnusson et al. 1996). To ensure an adequate sample size was recruited for the study, power analyses were conducted using the following parameters (power = 0.80, alpha = 0.05, effect size = 1.0, attrition = 20%). The analysis revealed that the initial sample size required to reach statistical power was 14, thus 18 subjects were recruited to account for possible attrition or data loss. Two subjects withdrew from the study with non-related injuries and two failed to complete both interventions; statistical analyses were conducted on data sets for 14 subjects who completed the testing.

RESULTS

Range of motion and stretch tolerance

A significant increase in dorsiflexion ROM (see Fig. 4) was found after CR (4.1° [CI = 2.6, 5.6]; $P < 0.01$) and SRC (4.0° [CI = 2.0, 6.0]; $P < 0.01$) stretching conditions. No difference in the increase in ROM was found between conditions ($P > 0.05$) indicating that both techniques were equally effective at increasing dorsiflexion ROM. When peak passive moment (stretch tolerance) was examined at full ROM pre- and post-intervention, a significant increase was found after CR (10.9% [CI = 4.4, 17.4]; $P < 0.05$) and SRC (15.1% [CI = 1.3, 28.9]; $P < 0.05$) stretching; no difference in the increase in stretch tolerance was found between conditions ($P > 0.05$). Significant correlations were observed between the changes in ROM and peak passive moment (stretch tolerance) in CR ($r_s = 0.63$; $P < 0.05$) and SRC conditions ($r_s = 0.71$; $P < 0.05$) indicating that changes in ROM were associated with changes in stretch tolerance after both interventions.

MTC, muscle and tendon stiffness

When the slope of the passive moment curve (indicative of MTC stiffness) was examined pre- and post-intervention, significant reductions (see Fig. 5b) were found after both CR (19.1% [CI = 9.7, 28.5]; $P < 0.05$) and SRC (13.3% [CI = 3.8, 22.8]; $P < 0.05$) stretching. No difference in the reduction in MTC stiffness was found between conditions ($P > 0.05$), indicating a similar response after each condition. To determine the influence on specific tissues, changes in muscle and tendon stiffness were also estimated separately. Significant reductions were found in tendon stiffness (see Fig. 6a) after CR (20.4% [CI = 15.2, 25.6]; $P < 0.01$) and SRC (15.7% [CI = 9.6, 21.8]; $P < 0.01$) stretching. Significant reductions were also found in GM muscle stiffness (see Fig. 6b) after CR (21.7% [CI = 17.4, 26.0]; $P < 0.01$) and SRC (21.3% [CI = 10.6, 32.0]; $P < 0.05$) stretching. No differences

($P > 0.05$) in the reduction in muscle or tendon stiffness were found between conditions, indicating a similar adaptive mechanical response. No significant correlations ($P > 0.05$) were found between the changes in ROM and changes in MTC stiffness, muscle stiffness or tendon stiffness in CR and SRC conditions, respectively.

Isometric plantar flexor moment and EMG

No significant difference was found in maximal isometric plantar flexor moment (-0.8% [CI = $-9.9, 8.3$], $P > 0.05$; 1.7% [CI = $-6.6, 10.1$], $P > 0.05$) or triceps surae EMG activity (average of Sol, GM and GL activity reported) during MVC (-11.5% [CI = $-29.0, 6.0$], $P > 0.05$; 3.0% [CI = $-8.4, 14.5$], $P > 0.05$) or EMG activity at full ROM during the passive trial (8.4% [CI = $-7.1, 23.8$], $P > 0.05$; -4.9% [CI = $-17.0, 7.1$], $P > 0.05$) following the CR and SRC conditions, respectively. These data are indicative that neuromuscular force generating capacity and reflexive muscle activity were neither inhibited nor potentiated after either condition. However, the mean voluntary isometric plantar flexor moment during the four MVCs generated at full stretch during the CR condition (147.2 ± 12.7 Nm) was significantly greater (10.6% [CI = $7.1, 14.1$]; $P < 0.05$) than the moment produced in the anatomical position during the SRC condition (131.4 ± 7.6 Nm), suggesting a greater tensile loading of the tendon during CR stretching compared with SRC stretching.

DISCUSSION

Contract-relax (CR) stretching has been commonly cited as the optimal stretching mode for achieving acute increases in ROM (Funk et al. 2003; Hindle et al. 2012), although the underlying mechanisms responsible for the efficacy of CR stretching to increase ROM remain to be established. Despite the efficacy of CR to acutely increase ROM, there is some concern that performing CR stretching can be painful and also increase muscle strain injury risk (Beaulieu 1981), which may partly explain why CR stretching is not as commonly used as static stretching for improving ROM. During muscular contractions sarcomere lengths are heterogeneous within single fibres and in different regions of the muscle, and this heterogeneity is exacerbated at long muscle lengths where the muscle operates on the descending limb of the force-length curve (Macpherson et al. 1997). Importantly, greater tissue damage is reported following contractions performed at longer muscle lengths (Butterfield and Herzog 2006; Whitehead et al. 2003) with focal damage limited primarily to the overextended sarcomeres with no disruption at other locations in the muscle fibre or in adjacent fibres (Balnave et al. 1997). Notably, the contraction phase in CR stretching is performed with the muscle held in a highly stretched position, increasing the potential for tissue damage and reducing the tensile strength of connective tissue (Butterfield and Herzog 2006; Whitehead et al.

2003). A novel finding of the present study was that similar acute increases in ROM ($\sim 4^\circ$) were observed after both the CR and SRC techniques, despite the contraction phase being performed in the anatomical rather than highly-stretched position in SRC. Importantly, as the contractions were performed with the muscle 'off stretch' during the SRC technique in the present study the risk of microscopic subcellular damage leading to muscle strain injury is substantially reduced when compared to the traditional CR technique. Given that the muscle length adopted during the muscle contraction phase of CR stretching does not appear to influence the subsequent acute gain in ROM, nor the changes in mechanical or neuromuscular responses, the SRC technique may be useful for safely improving ROM when compared to the standard CR technique. Furthermore, from a practical perspective, the SRC technique is easier to implement because it does not require partner assistance.

In addition to the relative effects of CR and SRC stretching on ROM, the mechanical responses of the MTC were examined, and similar significant acute increases in ROM ($\sim 4^\circ$) and reductions in the slope of the passive moment curve (~ 13 & 19% ; indicative of reduced MTC stiffness) were found after both SRC and CR conditions, respectively. Changes in passive moment of this magnitude concomitant with increases in ROM have been previously reported after static stretching (Kay et al. 2015; Morse et al. 2008), although few studies have employed the necessary methodology to quantify tissue-specific changes. However, where ultrasonography has been employed to assess MTC stiffness *in vivo*, reductions in muscle but not tendon stiffness have been reported (Kay & Blazeovich 2009a; Morse et al. 2008). The tissue-specific changes in stiffness following static stretching are probably expected as relaxed muscle is inherently more compliant than the tendon when measured under the current experimental conditions (Blazeovich et al. 2014). Therefore, the relatively low forces transmitted through the MTC are predominately expected to influence muscle stiffness. Consistent with previous findings (Kay & Blazeovich 2009a; Morse et al. 2008), the ultrasonography data in the present study revealed a reduced GM muscle stiffness ($\sim 21\%$) after both CR and SRC stretching, which can likely be explained by the static stretching phase being identical in both CR and SRC conditions. While reductions in muscle stiffness probably contributed to the ROM improvement, no significant correlation was found between increases in ROM and the reduction in muscle stiffness, which is indicative of other mechanisms more prominently underpinning the acute changes in ROM after CR and SRC stretching.

A distinct characteristic of CR stretching compared with other stretching techniques is the inclusion of an intense, often maximal, isometric contraction during the muscle stretch, which places substantial stress on muscular and

(unique to CR stretching) tendinous tissues. In the present study, significantly lower forces were transmitted through the tendon during the contraction phase in the SRC condition when compared with the CR condition, which is likely a consequence of the plantar flexors operating largely on the ascending limb of the force-length curve according to their force-length properties (Maganaris 2001, 2003). However, similar reductions in Achilles tendon stiffness (~16 & 21%) were found between conditions despite the reduced mechanical loading in the SRC condition. These data are consistent with a previous study (Kay et al. 2015) where a similar reduction in Achilles tendon stiffness (~22%) was observed after an acute bout of CR stretching. Tendons have been shown to withstand substantially greater loading (Kay & Blazevich 2009b) and deformation (Waugh et al. 2014) during maximal contractions than those imposed by static stretching (Blazevich et al. 2014). Therefore, maximal ROM and passive resistance to stretch are most probably dictated by the muscle's tolerance to loading and deformation rather than the tendon's, as maximum tendon tolerance is not tested during most passive stretching protocols. The relative stiffness, and consequently the deformation, of muscle and tendon are distinct during low-velocity passive joint rotations towards maximal ROM (Blazevich et al. 2014; Morse et al. 2008), however the energy transfer through the tendon and muscle are identical as these tissues are arranged in series. Therefore, reductions in tendon stiffness will lower joint moment within the MTC and thus reduce tension within the muscle at a specific joint angle. Reductions in tendon stiffness have been reported following maximal isometric contractions performed without stretch (Kay & Blazevich 2009b; Kubo et al. 2001), with concomitant increases in ROM being reported that are equivalent to the gains observed after static stretching (Kay et al. 2015). Importantly, both CR and SRC stretching techniques cause an acute reduction in muscle and tendon stiffness, and this broader adaptive response may be an important adaptation that underpins the superior efficacy of CR stretching to acutely increase ROM compared with static stretching, which only influences muscle properties (Kay & Blazevich 2009a; Morse et al. 2008).

A possible limitation of the present study was that neuromuscular reflex characteristics (e.g. M-wave/H-reflex characteristics) were not fully examined, although EMG amplitude as a measure of α -motoneuron pool reflex activity was measured at full ROM. However, the rotation velocity employed in the present study was intentionally designed to be too slow to initiate a significant myotatic reflex response (McNair et al. 2001), thus any changes in ROM should not be attributable to inhibition of the α -motoneuron pool. This was confirmed by the lack of any substantial EMG activity (< 5% MVC) at full ROM in both the pre- and post-intervention data, or significant change in EMG activity post-intervention. Thus, as no substantial activation of the musculature

occurred, changes in maximum ROM could not be notably influenced by Ib input, thus autogenic inhibition is not likely to be an important mechanism underpinning the increase in ROM following CR stretching in the present study. These data are similar to those reported in previous acute CR studies where EMG magnitude was unchanged at full ROM (Kay et al. 2015; Mitchell et al. 2009; Osternig et al. 1990). However, further study using a wider array of neuromuscular analyses at faster stretch velocities is needed to fully determine the possible role of autogenic inhibition as a mechanism underpinning the efficacy of CR stretching to increase ROM. Notwithstanding, a neurological contribution is at least partly supported by the increase in peak passive joint moment (~13%) and the strong correlations found between changes in peak passive moment (indicative of improved stretch tolerance) and ROM ($r_s = 0.63-0.71$; $P < 0.01$) after both CR and SRC stretching. These data are consistent with previous studies (Kay et al. 2015; Mitchell et al. 2009) where increased stretch tolerance was observed following an acute bout of CR stretching. Collectively, these data indicate that whilst inhibition of the α -motoneuron pool did not occur, altered pain perception is likely an important mechanism that influence ROM changes after CR stretching, however the specific neuromuscular pathways remains to be established.

CONCLUSIONS

In summary, a significant increase in ROM with reductions in both muscle and tendon stiffness and a concomitant increase in stretch tolerance were demonstrated after both CR and SRC stretching. Furthermore, the changes in ROM were significantly correlated with changes in stretch tolerance but not changes in muscle, tendon, or whole MTC stiffness. Thus, while mechanical changes in the muscle and tendon may have influenced ROM, changes in stretch tolerance may more strongly underpin the acute increases in ROM. The present study is the first to examine the effect of performing the contraction phase of CR stretching with the muscle 'off stretch'. As no differences in the changes in any measure were evident between conditions, it is likely that similar mechanisms were responsible for the (comparable) increases in ROM in CR and SRC conditions, regardless of the muscle length at which the contractions were performed. This novel finding is practically important as performing the contractions in the anatomical position (i.e. off stretch) is equally effective as CR but can be performed without partner assistance, is painless and reduces the risk of muscle damage as the contractions are performed at a shorter muscle length, therefore SRC offers a safer yet equally effective stretching model. These practical improvements may improve the capacity of individuals, coaches and clinicians to facilitate the use of this stretching mode as part of a complete injury prevention strategy in healthy and in at-risk populations in both athletic and clinical settings.

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CONFLICTS OF INTEREST

The authors declare that they have no conflict of interest.

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ABBREVIATIONS

CI	Confidence intervals
CR	Contract-relax
EMG	Electromyography
GL	Gastrocnemius lateralis
GM	Gastrocnemius medialis
ICC	Intraclass correlation coefficient
MTC	Muscle-tendon complex
MTJ	Muscle-tendon junction
MVC	Maximal voluntary contraction
PNF	Proprioceptive neuromuscular facilitation
ROM	Range of motion
Sol	Soleus
SE	Standard error
SRC	Stretch-return-contract
TA	Tibialis anterior
TTL	transistor-transistor logic

FIGURE CAPTIONS

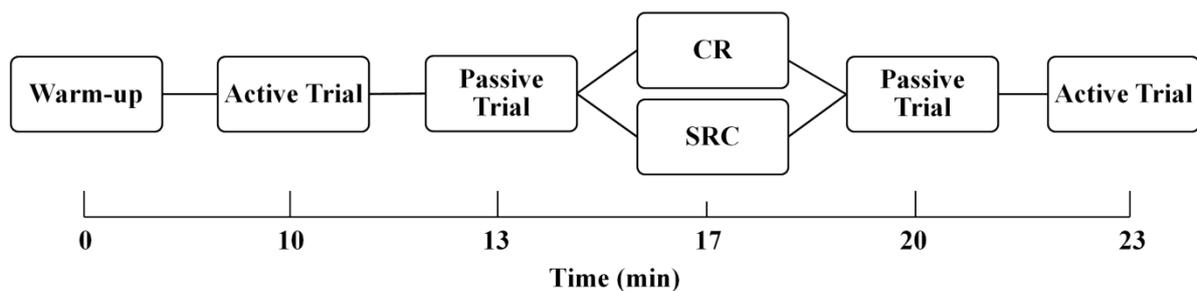


Fig. 1 Timeline of the contract-relax (CR) and stretch-return-contract (SRC) stretching protocols. At 5 min after completion of the warm-up, active and passive trials were conducted, and after 2 min further rest either the CR or SRC stretching intervention was carried out. Two minutes later, passive and active trials were repeated to determine the effects of each intervention

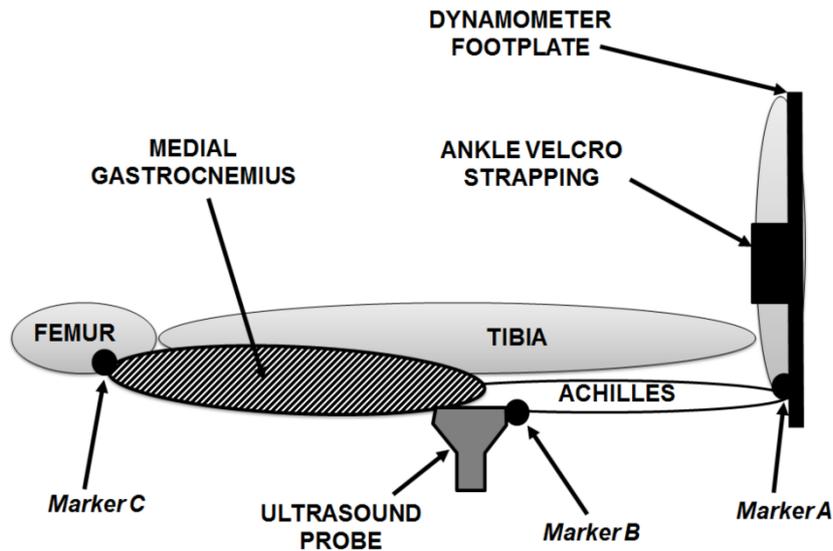


Fig. 2 Infrared reflective motion analysis marker and ultrasound probe positioning. Achilles tendon length was estimated as the distance between the reflective markers placed on the distal edge of the ultrasound probe (*marker B*) located over the gastrocnemius medialis (GM)-Achilles muscle-tendon junction (MTJ) and the insertion of the Achilles on the calcaneus (*marker A*). GM muscle length was estimated from the distance between the reflective markers placed on the distal edge of the ultrasound probe (*marker B*) and the origin of the GM muscle on the medial femoral epicondyle (*marker C*)

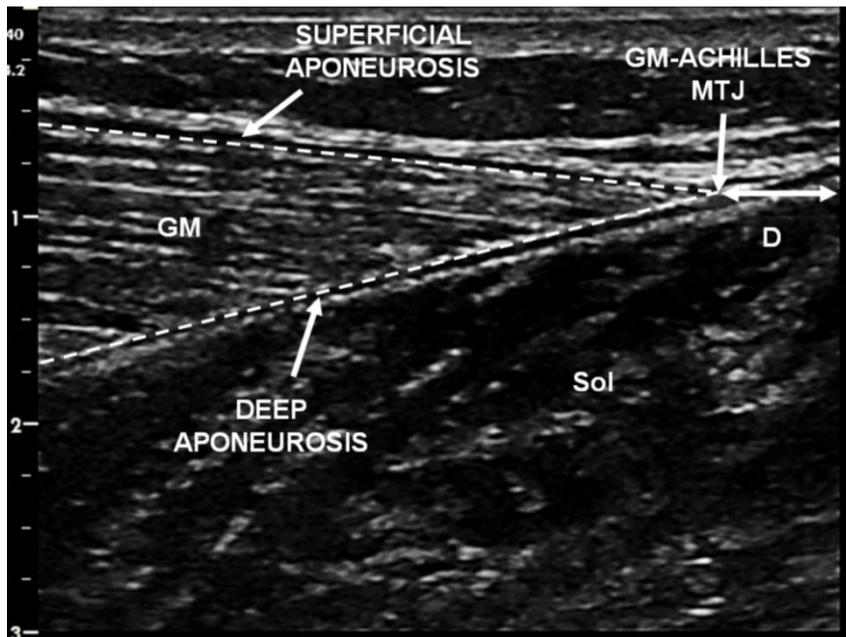


Fig. 3 Ultrasound image of the gastrocnemius medialis (GM)-Achilles muscle-tendon junction (MTJ). Real-time ultrasound imaging was used to record the position (and displacement) of the GM-Achilles MTJ. The MTJ was identified as the point where the deep GM and superficial soleus (Sol) aponeuroses and superficial GM aponeurosis merged with the Achilles tendon. Displacement of the MTJ from the distal edge of the image (D) was synchronized with motion analysis data to calculate GM muscle and Achilles tendon lengths

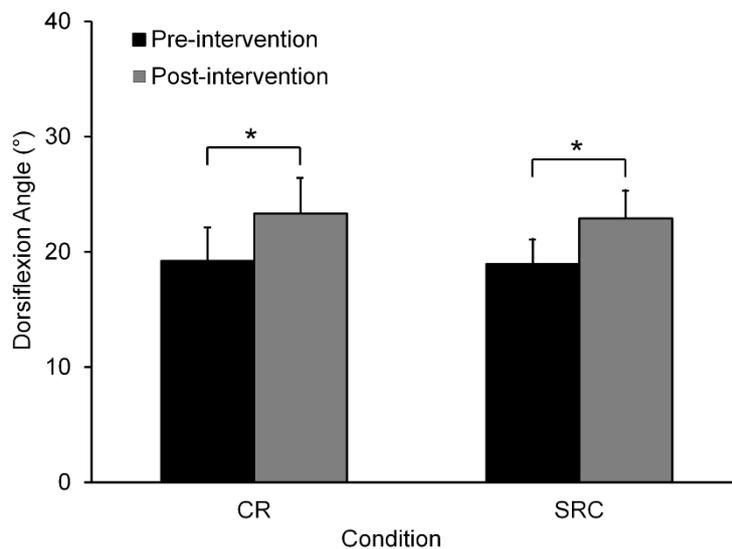


Fig. 4 Mean (\pm SE) dorsiflexion range of motion (ROM) before and after stretching. Significant increases in dorsiflexion ROM were found after contract-relax (CR; 4.1°) and stretch-return-contract (SRC; 4.0°) stretching. No difference was found in the changes in ROM between conditions. *Significant to $P < 0.01$

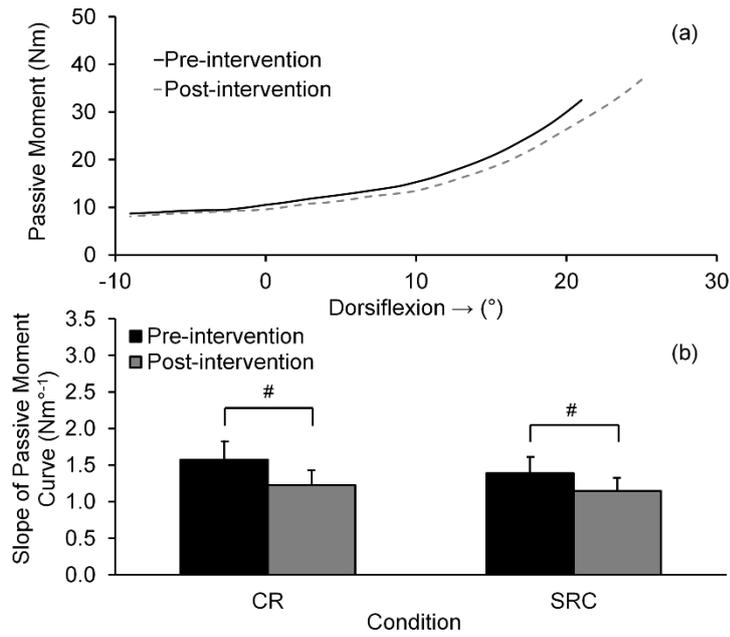


Fig. 5 Mean (\pm SE) passive plantar flexor moment before and after stretching. Passive moment (a) was reduced after stretching at all dorsiflexion angles along the joint moment-angle curve (one subject's data depicted during a contract-relax trial). Significant reductions in the slope of the passive moment curve (b) were found after contract-relax (CR; 19.1%) and stretch-return-contract (SRC; 13.3%) stretching. No difference was found in the changes in passive moment between conditions ($P > 0.05$). #Significant to $P < 0.05$

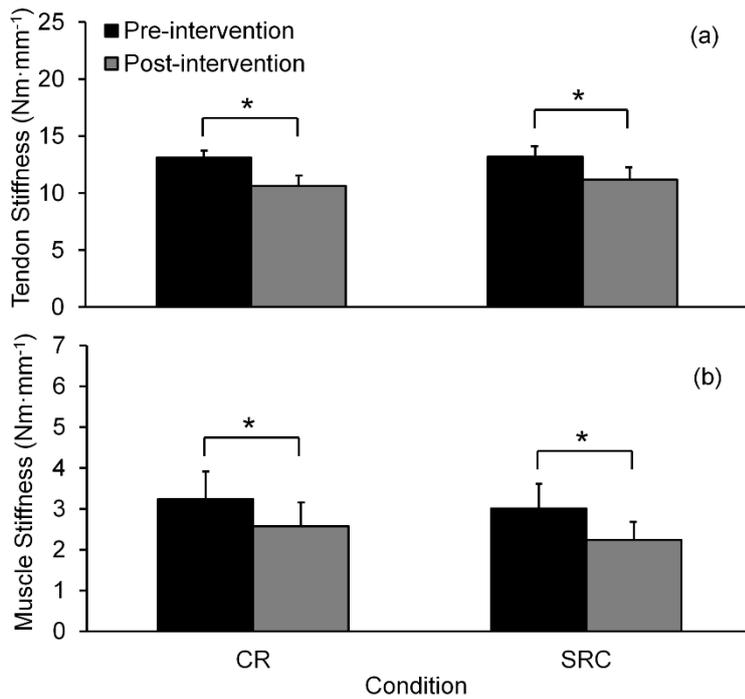


Fig. 6 Mean (\pm SE) Achilles tendon stiffness and gastrocnemius medialis (GM) muscle stiffness before and after stretching. Significant reductions in Achilles tendon stiffness (a) were observed after contract-relax (CR; 20.4%) and stretch-return-contract (SRC; 15.7%) stretching. Significant reductions in GM muscle stiffness (b) were found after CR (21.7%) and SRC (21.3%) stretching. No difference in the reductions in muscle and tendon stiffness was found between conditions ($P > 0.05$). *Significant to $P < 0.01$