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ORIGINAL ARTICLE

Passive muscle stretching impairs rapid force production and neuromuscular function in human plantar flexors

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Abstract

Purpose We examined the effect of muscle stretching on the ability to produce rapid torque and the mechanisms underpinning the changes.

Methods Eighteen men performed three conditions: (1) continuous stretch (1 set of 5 min), (2) intermittent stretch (5 sets of 1 min with 15-s inter-stretch interval), and (3) control. Isometric plantar flexor rate of torque development was measured during explosive maximal voluntary contractions (MVC) in the intervals 0–100 ms (RTD_{V100}) and 0–200 ms (RTD_{V200}), and in electrically evoked 0.5-s tetanic contractions (20 Hz, 20 Hz preceded by a doublet and 80 Hz). The rate of EMG rise, electromechanical delay during MVC (EMD_V) and during a single twitch contraction (EMD_{twitch}) were assessed.

Results RTD_{V200} was decreased ($P < 0.05$) immediately after continuous (− 15%) and intermittent stretch (− 30%) with no differences between protocols. The rate of torque development during tetanic stimulations was reduced ($P < 0.05$) immediately after continuous (− 8%) and intermittent stretch (− 10%), when averaged across stimulation frequencies. Lateral gastrocnemius rate of EMG rise was reduced after intermittent stretch (− 27%), and changes in triceps surae rate of EMG rise were correlated with changes in RTD_{V200} after both continuous ($r = 0.64$) and intermittent stretch ($r = 0.65$). EMD_V increased immediately (31%) and 15 min (17%) after intermittent stretch and was correlated with changes in RTD_{V200} ($r = -0.56$). EMD_{twitch} increased immediately after continuous (4%), and immediately (5.4%), 15 min (6.3%), and 30 min after (6.4%) intermittent stretch ($P < 0.05$).

Conclusions Reductions in the rate of torque development immediately after stretching were associated with both neural and mechanical mechanisms.

Keywords Rate of force development · Explosive force · Flexibility · Force transmission

Abbreviations

ANOVA	Analysis of variance
CI	Confidence interval
EMD	Electromechanical delay
EMD_{twitch}	Electromechanical delay during the electrically evoked twitch
EMD_V	Electromechanical delay during voluntary contraction
EMG	Electromyogram
LG	Lateral gastrocnemius
M_{max}	Maximal compound action potential amplitude
MVC	Maximal voluntary contraction
PICs	Persistent inward currents
RER	Rate of electromyogram rise
RFD	Rate of force development
RTD	Rate of torque development
RTD_I	Involuntary rate of torque development
RTD_V	Voluntary rate of torque development
SOL	Soleus
VFT	Variable-frequency train of stimulation

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Introduction

There is extensive evidence that acute passive muscle stretching lasting ≥ 60 s can decrease subsequent maximal voluntary force production (i.e., peak force) and that this force loss is largely attributable to neural mechanisms (Behm et al. 2016; Trajano et al. 2017). However, much less attention has been given to its effects on the ability to produce rapid force, usually measured as the rate of force (or torque) development (RFD or RTD), i.e., the first derivative of force/torque with respect to time. The rate of force development (henceforth, referred as the rate of torque development) has been of clear, recent scientific interest, as it seems to be more strongly related to athletic and functional daily task performances than traditional maximal torque measurements (i.e., peak force) (Maffiuletti et al. 2016). A recent meta-analysis revealed an average reduction of 4.5% in the rate of torque development immediately after passive stretching (Simic et al. 2013), indicating that stretching may induce clinically relevant changes in it. Nonetheless, the mechanisms underpinning the reduction are not yet known, so it is not possible to develop potential interventions to reduce or mitigate the effect.

The ability to produce high levels of torque in a short period of time can be influenced by several factors including the number of motor units recruited and their firing rates (van Cutsem et al. 1998; Aagaard et al. 2002; Klass et al. 2008), the rate of sarcoplasmic Ca^{2+} release (Nielsen 2009) and/or myofibrillar Ca^{2+} sensitivity (Abbate et al. 2002), and the muscle–tendon unit's ability to transmit force rapidly (related to electromechanical delay) (Waugh et al. 2013). Thus, changes in any of these factors could negatively affect the rate of torque development, and a comprehensive evaluation is necessary to better understand the possible effect of passive muscle stretch on them.

Regarding neuromuscular factors that are thought to most prominently influence the rate of torque development (Duchateau and Baudry 2014), it is possible to gain insight into the role of neural (efferent) drive in determining the rate of torque production by quantifying the rate of rise in electromyogram (EMG) amplitude (RER; i.e., the gradient of EMG/time trace) during fast, maximal voluntary muscle contractions, and then normalizing it to the muscle's maximal compound action potential (M_{max}) amplitude. This normalization would account for possible (peripheral) changes in fiber membrane characteristics, location of innervation zones and electrode detection volumes (Un et al. 2013; Jenkins et al. 2014). Specifically, the presence of initial high-frequency motor unit discharges (i.e., doublets) has been shown to significantly increase the rate torque development in voluntary muscle contractions (van Cutsem et al. 1998). Also, this phenomenon has been replicated by comparing the rate of torque development evoked by a standard, constant train of (tetanic) neuromuscular electrical stimulation (e.g., 20 Hz) against a similar, but variable-frequency train with an initial doublet (e.g., first two pulses at 10 ms inter-spike interval) (Binder-Macleod and Kesar 2005). A comparison of the rates of torque developed at different frequencies of tetanic electrical stimulation might also provide information related to the role of calcium-related mechanisms. It is important to note that faster rates of force rise observed during a variable-frequency train with an initial doublet have been suggested to be associated with transient increases in myoplasmic Ca^{2+} availability, myofibrillar Ca^{2+} sensitivity (i.e., the sensitivity of the actin–myosin interaction to a given concentration of free myoplasmic Ca^{2+}) (Binder-Macleod and Kesar 2005; Nielsen 2009), and rapid saturation of Ca^{2+} binding to troponin C (Bakker et al. 2017). The increase in Ca^{2+} binding to troponin C would increase the number of cross-bridges that can bind to actin over a given period of time, causing specifically a greater rate of force development (Bakker et al. 2017). Thus, a comparison between the rate of torque development elicited by a tetanic electrical stimulation during a continuous frequency train (i.e., 20 Hz) against a similar train with an initial doublet, could shed light on possible changes related to Ca^{2+} binding to troponin C in in vivo study designs. Also, additional information related to changes in free myoplasmic Ca^{2+} concentration can be gathered by comparing the ratio between the torque produced by 20- and 80-Hz tetanic stimulation, as commonly done in studies of muscle fatigue and exercise-related muscle damage (Martin et al. 2004). The use of such tests, therefore, may provide in vivo evidence for changes in calcium handling within muscle fibers.

The time delay between the arrival of electrical signals at the muscle and the detection of a mechanical force response, i.e., the electromechanical delay (EMD) (Cavanagh and Komi 1979), can be measured during voluntary (Cavanagh and Komi 1979) or electrically evoked contractions (Zhou et al. 1995). It is considered to provide information related to force transmission along the series elastic components in the muscle–tendon unit (Nordez et al. 2009) during asynchronous (i.e., voluntary) or synchronous (i.e., time-locked electrically evoked contractions) activation. This is because a considerable part of the electromechanical delay results from the time necessary to transmit the force within the muscle–tendon unit (Nordez et al. 2009). Passive stretch could cause a longer electromechanical delay by temporarily reducing stiffness of the series elastic components within the muscle–tendon unit (Costa et al. 2010; Esposito et al. 2011). Indeed, increases in electromechanical delay have been correlated with the reduction in fast torque production during plantar flexions (Waugh et al. 2013). Thus, examination of

the electromechanical delay may provide information related to possible impact of changes in the in vivo mechanical properties of series elastic components and their potential impact on rate of torque development.

With respect to the muscle function changes after muscle stretching, we have observed a greater reduction in maximal voluntary torque and neural drive after repetitions of short-duration (e.g., 60 s) passive muscle stretches (i.e., intermittent stretching) than longer periods of constant stretch (i.e., continuous stretch) (Trajano et al. 2014a). However, it is not known whether these two stretching protocols differently affect the rate of torque development and, if so, whether the difference between the protocols is associated with any differences in neuromuscular processes. Given the purported importance of the rate of torque development to muscle function and task performance (Maffiuletti et al. 2016), it is of great interest to compare the effects of different stretch methods on the rate of torque development and the mechanisms that underpin them. Therefore, the purpose of the present study was to determine the effect of intermittent versus continuous muscle stretching on muscular rate of torque development and to provide information relating to the mechanisms underpinning possible changes. We hypothesized that intermittent stretch would provoke a greater reduction in the rate of torque development through a significant attenuation of efferent neural drive to the muscle (i.e., a reduction in M-wave normalized rate of EMG rise).

Materials and methods

Participants

Eighteen recreationally active young men (1.75 ± 0.1 m; 72.7 ± 12.6 kg; 26.8 ± 4.5 years) without any neuromuscular impairment volunteered to participate in the study. They reported not being engaged in any type of flexibility training in the last 6 months prior to the data collection. They were instructed to avoid vigorous exercise and alcohol consumption for 24 h, and caffeine use for at least 6 h, prior to testing. All participants read and signed the informed consent document and the Edith Cowan University Human Research Ethics Committee approved this study (Approval number: 6965). This study was conducted according to the principles expressed in the Declaration of Helsinki. Data collected during this experiment relating to maximal voluntary contraction (MVC) strength and joint range of motion have been published previously (Trajano et al. 2014a).

Study design and overview

Participants were tested on four occasions separated by at least 48 h at the same time of day. The first visit was used as a familiarization session, whilst the subsequent three visits were used for the completion of the following experimental conditions in a randomized, counterbalanced order: (1) one 5-min continuous stretch; (2) five sets of 1-min intermittent stretches (15-s inter-stretch interval); and (3) control (or no stretch). The subjects were tested immediately before, and then immediately, 15 and 30 min after each intervention. During familiarization, the intensities of all electrically evoked muscle and nerve stimulation measurements were determined, the rapid maximal voluntary contractions were practiced, and the maximum tolerable passive torque during stretch was measured. In the experimental sessions, performed at the same time of day as the familiarization session, the subjects warmed up on a Monark cycle for 5 min at 60 rpm with a 1-kg load. The subjects were then seated upright in the chair of an isokinetic dynamometer (Biodex System 3 Pro, IPRS, Suffolk, UK) with the knee in full extension (0º), the ankle in the neutral position (0°; plane of foot relative to tibia) with the sole of the foot perpendicular to the shank, and the lateral malleolus of the fibula aligned to the center of rotation of the dynamometer. Ankle joint torque, joint angle and EMG data were simultaneously recorded using LabChart v.6.1.3 Software (PowerLab System, ADInstruments, NSW, Australia).

Muscle stretching protocol

All stretch procedures were performed on an isokinetic dynamometer with the subjects seated (0° knee angle and 70° hip flexion) and instructed to keep their muscles relaxed. The plantar flexors were stretched by rotating the ankle into dorsiflexion at 5° s^{-1} until the passive resistance reached 90% of the maximal tolerable stretch torque, as measured during the familiarization session, and the joint angle was continually adjusted toward dorsiflexion during the stretch to maintain the passive torque within 5 N m of the initial stretch torque level. With this design, stretches in both conditions were of the same intensity and duration, differing only in the number of intervals.

Stimulation procedures

Muscle stimulation (tetanic contractions)

A constant current electrical stimulator (DS7, Digitimer Ltd, Welwyn Garden City, UK) was used to deliver electrical square-wave stimuli (0.5-ms pulse width) to the plantar flexor muscle belly through two self-adhesive electrodes (9 × 5 cm, Dura-Stick® II, Chattanooga Group, Hixon, USA) during the tetanic stimulations. The cathode was placed distal to the popliteal crease and the anode was placed over the distal myotendinous junction of soleus. For all tetanic

stimulations, the intensity necessary to reach 50% of MVC with a 0.5-s tetanus at 80 Hz was used. These stimulation parameters typically evoke muscle contractions largely via motor axons with no detectable H-reflex when delivered via muscle belly electrical stimulation (Bergquist et al. 2011). In the present study, all the EMG traces during electrical stimulation were visually inspected and no evidence of significant contribution of excitatory reflexes (e.g., H waves or asynchronous activity) was found. Three tetanic stimulations with the same duration (0.5 s) were delivered to test the rate of torque development during three different stimulation patterns: (1) 20 Hz train; (2) variable-frequency train (i.e., 20 Hz train with the first two pulse s at 100 Hz); and (3) 80 Hz train. The comparison of these three types of tetanic stimulation would allow us to determine whether the stretch could affect the rate of force development in low- (20 Hz) and high-frequency (80 Hz) stimulations, measured as the 20:80 ratio, and during a typical variable-frequency train of stimulation (VFT), which is usually associated with an enhanced rate of torque development (Binder-Macleod and Kesar 2005), measured as the 20:VFT ratio. We used these measures to determine whether the stretch protocols could elicit a frequency-dependent change in the rate of torque development, which were assumed to provide information on calcium-related mechanisms (concentration, sensitivity, and the rate of binding to troponin C) (see "Introduction" and "Discussion" sections for details) (Martin et al. 2004).

Nerve stimulation (twitch)

The same electrical stimulator was used to deliver an electrical square wave pulse of 1-ms width stimuli to the posterior tibial nerve via a cathode electrode (Ag–AgCl, 10 mm) fixed to the popliteal fossa and an anode electrode of large size (9 × 5 cm, Dura-Stick® II, Chattanooga Group, Hixon, USA) placed on the anterior surface of the knee. The intensity for a single twitch was set at 120% of the intensity required to elicit M_{max}, to ensure that a supramaximal current stimulus was used.

Rate of torque development (RTD)

The torque signal was sampled at 2000 Hz. The voluntary rate of torque development (RTD_V) was measured using the raw torque trace during two maximal voluntary plantar flexor contractions where the subjects were instructed to rotate the foot away from the body "as fast and as hard as possible" and the highest value was used. RTD_V was calculated as the average change in torque per time interval from the torque onset to 100 (RTD_{V100}) and 200 ms (RTD_{V200}). To identify whether changes in the rate of torque development were proportional to the overall reductions in torque generating capacity, RTD_{V200} normalized to MVC ($RTD_{V200/MVC}$) is also presented. Involuntary rate of torque development (RTD_I) was calculated during tetanic contractions (20 Hz, RTD_{20Hz}; variable-frequency train, RTD_{VFT}; and 80 Hz, RTD_{80Hz}) from the torque onset to 250 ms. Torque onset was defined as the point where torque increased 3 N m above baseline but did not fall below baseline for the duration of the 3-s contractions. Baseline values were visually inspected and the average value was used to determine the onset value and this point was visually confirmed.

Electromyographic (EMG) measurement

Surface EMG was recorded from soleus (SOL) and lateral gastrocnemius (LG) using a bipolar electrode configuration at a 4000 Hz analog–digital conversion rate (bandwidth 20–450 Hz) using the Bagnoli-8 Main Unit EMG system (DelSys, Inc., MA, USA). Electrodes were placed following SENIAM recommendations. The inter-electrode distance was 1 cm and a reference electrode was placed on the fibula's lateral malleolus. The skin under the electrodes was shaved, abraded and cleaned with alcohol to reduce the inter-electrode resistance below 5 kΩ. During the stretch protocols, EMG data were also recorded to ensure that muscle activation remained below 10% of the maximal value; a small activity response is normally detected even when the subjects are asked to remain completely relaxed (Blazevich et al. 2012).

Rate of EMG rise (RER)

To determine the rate of EMG rise, the root mean square of the band-pass filtered lateral gastrocnemius and soleus EMG signals were calculated. The rate of EMG rise was calculated as the average change in the EMG amplitude per time interval from the EMG onset to 100 ms during MVC. The 0–100 ms time window was chosen due to its good measurement reliability (ICC = 0.91). The EMG onset was defined as the first point where the EMG amplitude exceeded 0.01 mV from the baseline and did not fall below this value for at least 10 ms prior to the MVC. This was performed in a semi-automatic way where a non-blinded investigator determined the EMG baseline before the initiation of MVC and the software searched for the first value exceeding the baseline by 0.01 mV. This point was then visually inspected and confirmed by the investigator as the EMG onset. The repeatability of this method was determined by re-measuring a subset of 20 contractions, and was found to exhibit an intraclass correlation of 0.99 and a coefficient of variation of 3.9%. To account for possible effects of peripheral changes, rate of EMG rise of each muscle was normalized to the RMS value of its respective M_{max} amplitudes (RER/M). Also, the normalized values for lateral gastrocnemius and soleus were summed and considered as an indication of triceps surae

muscle excitation. Although the use of M_{max} normalization in fatigue studies has been questioned (Rodriguez-Falces and Place 2018), recent evidence suggested that this procedure appropriately accounts for changes in electrode recording volume (Lanza et al. 2017).

Electromechanical delay (EMD)

The electromechanical delay was measured during the electrically evoked twitch (EMD_{twitch}) and the maximal voluntary contraction (EMD_V). EMD_{twitch} was calculated as the time lag between the start of the compound muscle action potential (M-wave) and the torque onset (Costa et al. 2010). For the calculation of EMD_V, the band-pass filtered rectified LG EMG signal was used and the time between the EMG onset and the torque onset calculated.

Statistical analysis

Shapiro–Wilk's tests were used to verify the assumption that the data were normally distributed. SPSS (IBM, version 23) was used to perform a three-way repeated-measures ANOVA to compare changes in the rate of torque development in the three stimulation frequencies (20 Hz, variable-frequency, 80 Hz) between conditions (control, continuous and intermittent) over time (before, immediately after, and 15 and 30 min after). Separate two-way repeated-measures ANOVAs were performed to compare changes in all other variables between conditions over time. Pairwise comparisons with Bonferroni corrections were performed when a significant interaction effect was detected. Repeated-measures Bland–Altman within-subject correlation coefficients were computed using RStudio (Version 1.0.153) and rmcorr R package to determine the relationships between the rate of torque development and the rate of EMG rise and voluntary electromechanical delay across time points (Bakdash and Marusich 2017). Strength of the correlations was interpreted using the following criteria (Cohen 1988): trivial, $r < 0.1$; small, $r = 0.1$–0.3; moderate, $r = 0.3$–0.5; large, $r = 0.5$–0.7; very large, $r = 0.7$–0.9; and nearly perfect, $r > 0.9$. Statistical significance was set at an α level of 0.05. Data are presented in mean ± SD and 95% confidence interval (95% CI).

Results

RTD_V

No interaction effect (condition × time) was found for RTD_{V100} ($P = 0.275$), but a significant effect was observed for RTD_{V200} ($P = 0.002$). Post hoc analysis revealed a significant reduction immediately after both intermittent (− 30.2 ± 27.7%; 95% CI − 43.0 to − 17.4%; $P < 0.001$) and continuous (− 15.1 ± 22.6%; 95% CI − 25.6 to − 4.7%; $P < 0.045$) stretching with no difference from baseline being found at 15 min (Figs. 1, 2). No significant changes were found during the control condition. There was no interaction effect for $RTD_{V200/MVC}$ ($P = 0.093$) (Table 1).

RTD_I

No three-way interaction effect (condition × time × frequency) was observed for RTD_I, suggesting there were no differences in the changes over time between the three stimulation frequency conditions (20 Hz, VFT and 80 Hz). In addition, no two-way interaction effect (condition × time) was observed for 20:VFT ($P = 0.54$) or 20:80 ($P = 0.81$) ratios. However, there was a significant condition × time interaction effect ($P = 0.007$) for RTD_I with a significant reduction (collapsed across frequencies) immediately after both continuous (− 8.1 ± 8.1%; 95% CI − 10.3 to − 6.0%, $P = 0.001$) and intermittent (− 10.1 ± 11.3; 95% CI − 13.1 to − 7.1%, $P = 0.001$) stretching.

RER/M

There was a significant condition × time interaction for RER/M_{LG} ($P = 0.008$). Post hoc analyses revealed a significant reduction in RER/M_{LG} immediately after intermittent stretching (− 27.1 ± 29.2; 95% CI − 13.6 to − 40.7; $P = 0.006$). No significant condition × time interaction was

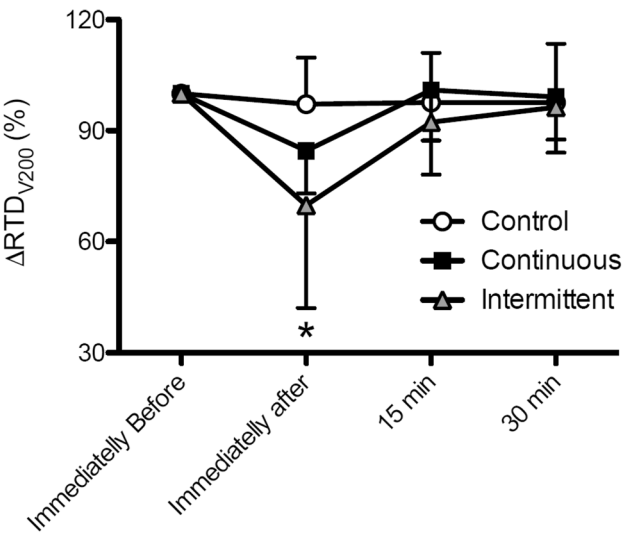

Fig. 1 Normalized changes (mean ± SD) in the rate of torque development measured during voluntary contractions in 0–200 ms (RTD_{V200}) from baseline (100%) immediately, 15 min and 30 min after continuous stretch, intermittent stretch and control conditions. RTD_{V200} was reduced immediately after both stretch protocols but returned to baseline levels 15 min after. Data are presented as mean ± SD

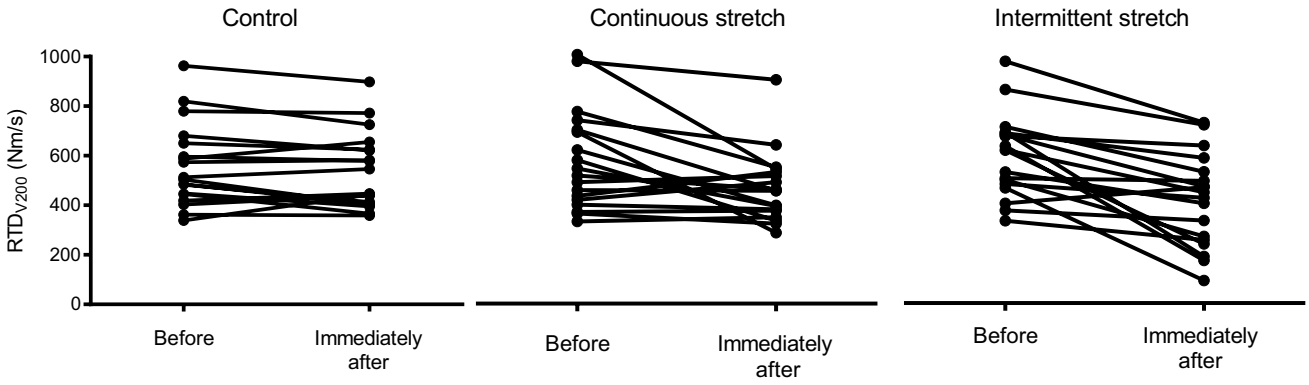

Fig. 2 Absolute values of RTD_{V200} (in N m) before and immediately after each condition for individuals

found for RER/M_{SOL} ($P = 0.7$) and RER/M_{TS} ($P = 0.34$). However, there was a significant, large correlation between RTD_{V200} and RER/M_{TS} across time points ($r = 0.64$, $P = 0.000$) in the continuous stretch condition. Also, there was a large correlation ($r = 0.65$, $P = 0.000$) between RTD_{V200} and RER/M_{TS} across time points in the intermittent stretch condition. There was no significant condition × time effect for the maximal M-wave amplitude ($P = 0.64$).

EMD_{twitch} and EMD_V

There was a significant interaction effect (condition × time) for EMD_{twitch} ($P = 0.037$). Post hoc analyses revealed a significant increase in EMD_{twitch} immediately (5.4 ± 7.6; 95% CI 1.9–8.9%, $P = 0.046$), 15 min (6.3 ± 6.2%; 95% CI 3.5–9.2%, $P = 0.003$), and 30 min (6.4 ± 4.5%; 95% CI 4.3–8.5%, $P < 0.001$) after intermittent stretch. Additionally, there was a significant ($P = 0.039$) increase in EMD_{twitch} immediately after continuous stretch (4.0 ± 5.6%; 95% CI 1.5–6.6%) with no significant difference from the baseline being found at 15 and 30 min, and no changes in the control condition.

A significant condition × time interaction was found for EMD_V ($P = 0.03$), with a significant increase in EMD_V being observed immediately (31.1 ± 33.5%; 95% CI 15.6–46.5%, $P = 0.009$) and 15 min after (16.9 ± 19.8%; 95% CI 7.8–26.1%, $P = 0.015$) the intermittent stretch. No changes were found in the continuous stretch or control conditions. A large correlation ($r = -0.56$, $P = 0.000$) was found between RTD_{V200} and EMD_V across time points in the intermittent stretch condition.

Discussion

The main findings of the present study were that: (1) both continuous (1 set of 5 min) and intermittent (5 sets of 1 min) stretching reduced the voluntary rate of plantar flexor torque development (RTD_{V200}) immediately after stretching, although this recovered by 15 min (Fig. 1); (2) the electrically evoked (involuntary) rate of torque development was also reduced immediately after both stretch interventions irrespective of the frequency pattern of stimulation; (3) the rate of lateral gastrocnemius excitation (measured as the rate of EMG rise normalized to the maximal M-wave amplitude) after intermittent stretching was significantly reduced, and the correlations between changes in triceps surae rate of EMG rise and changes in voluntary rate of torque development indicate that greater reductions in the rate of muscle excitation were associated with greater reductions in rate of torque development; (4) involuntary electromechanical delay was increased immediately after both stretch protocols and returned to baseline levels by 15 min after continuous stretch, but remained prolonged by 30 min after intermittent stretch, and (5) voluntary electromechanical delay was increased immediately and 15 min after intermittent stretching only, and the changes observed immediately after stretching were associated with changes in voluntary rate of torque development.

Based on previous data reporting the effects on maximal torque production (Trajano et al. 2014a), we hypothesized that intermittent stretch would trigger a greater reduction in voluntary rate of torque development when compared to the continuous stretch. However, although the mean decrease was ~ twofold greater immediately after intermittent (− 30%) than continuous (− 15%) stretching, the difference was highly variable between individuals (see Fig. 2) and did not reach statistical significance. This high variability (including subjects with large reductions in rate of torque development) cannot be attributed to measurement error (RTD_{V200} ICC = 0.94) and is also commonly observed in peak torque data (which is also highly reliable, ICC = 0.97) obtained after passive stretching, and it has been reported and discussed previously (Trajano et al. 2014a). It is possible that the substantial decrease in rate of torque development observed in some participants in the present study (e.g., four

Table 1 Torque and EMG data before and immediately, 15 and 30 min after control, continuous and intermittent stretching

	Control				Continuous				Intermittent			
	Before	Immediately after	15 min after	30 min after	Before	Immediately after	15 min after	30 min after	Before	Immediately after	15 min after	30 min after
RTD_{V100} (N m/s)	565 ± 179	503 ± 174	535 ± 176	527 ± 199	549 ± 226	402 ± 207	507 ± 198	504 ± 172	530 ± 201	377 ± 203	461 ± 178	514.4 ± 182
RTD_{V200} (N m/s)	558 ± 167	537 ± 155	541 ± 158	540 ± 157	542 ± 188	436 ± 134*	535 ± 154	525 ± 147	558 ± 155	387 ± 175*	509 ± 140	527 ± 128
$RTD_{V200/MVC}$	3.2 ± 0.5	3.1 ± 0.5	3.1 ± 0.5	3 ± 0.4	3.1 ± 0.7	3 ± 0.8	3.2 ± 0.7	3.1 ± 0.6	3.2 ± 0.7	2.9 ± 0.7	3.1 ± 0.6	3.2 ± 0.5
RTD_{20Hz} (N m/s)	186 ± 33	185 ± 36	178 ± 39	177 ± 39	189 ± 36	173 ± 43	175 ± 34	172 ± 35	185 ± 41.5	166 ± 38	162 ± 37	167 ± 40
RTD_{VFT} (N m/s)	196 ± 41	194 ± 41	187 ± 47	188 ± 45	199 ± 41	182 ± 46	187 ± 37	186 ± 40	195 ± 46	178 ± 45	175 ± 48	180 ± 47
RTD_{80HZ} (N m/s)	267 ± 64	275 ± 61	268 ± 72	266 ± 68	277 ± 61	263 ± 69	272 ± 57	269 ± 57	269 ± 70	260 ± 71	255 ± 72	264 ± 76
20:80 ratio (%)	71 ± 7	68 ± 6	67 ± 6	67 ± 6	69 ± 6	67 ± 8	67 ± 9	65 ± 8	70 ± 7	64 ± 7	65 ± 5	65 ± 5
20:VFT ratio (%)	96 ± 5	95 ± 5	96 ± 6	95 ± 4	96 ± 4	95 ± 5	94 ± 6	93 ± 4	97 ± 5	94 ± 6	95 ± 5	95 ± 4
RER/M_{LG}	0.5 ± 0.35	0.48 ± 0.34	0.5 ± 0.28	0.53 ± 0.34	0.47 ± 0.24	0.4 ± 0.23	0.46 ± 0.23	0.46 ± 0.25	0.51 ± 0.31	0.35 ± 0.3*	0.55 ± 0.33	0.57 ± 0.27
RER/M_{SOL}	0.34 ± 0.3	0.32 ± 0.25	0.39 ± 0.29	0.37 ± 0.28	0.36 ± 0.30	0.32 ± 0.32	0.36 ± 0.32	0.38 ± 0.35	0.52 ± 0.36	0.47 ± 0.39	0.50 ± 0.34	0.55 ± 0.42
RER/M_{TS}	0.83 ± 0.43	0.8 ± 0.43	0.89 ± 0.42	0.9 ± 0.43	0.82 ± 0.38	0.72 ± 0.41	0.82 ± 0.4	0.84 ± 0.44	1.02 ± 0.44	0.83 ± 0.52	1.05 ± 0.43	1.11 ± 0.42
EMD_V (ms)	53.4 ± 13.7	53.4 ± 10.5	53.9 ± 14.2	50.3 ± 10.4	58.4 ± 14.4	62.3 ± 25.1	58.6 ± 14.1	63.4 ± 24.3	48.7 ± 10.6	63.9 ± 22.2*	56.4 ± 13.9*	55.8 ± 14
EMD_{twitch} (ms)	27.6 ± 2.6	27.4 ± 2.3	27.7 ± 2.3	28 ± 3.4	27 ± 3.5	28.1 ± 4.2*	28.3 ± 4.6	28.1 ± 4.3	26.3 ± 4.2	27.6 ± 3.9*	27.9 ± 3.9*	28 ± 4.2*

Values are means ± SD. RTD_{V100}, rate of torque development from 0 to 100 ms; RTD_{V200}, rate of torque development from 0 to 200 ms; $RTD_{V200/MVC}$, rate of force development normalized to the maximal voluntary contraction; RTD_{20Hz}, rate of torque development during 20 Hz tetanic stimulation; RTD_{VFT}, rate of torque development during variable-frequency tetanic stimulation; RTD_{80Hz}, rate of torque development during 80 Hz tetanic stimulation; 20:80 ratio, the ratio RTD_{20Hz} and RTD_{80Hz}; 20:VFT ratio, the ratio between RTD_{20Hz} and RTD_{VFT}; RER/M_{LG}, the rate of EMG rise normalized by the M-wave lateral gastrocnemius, soleus (RER/M_{SOL}), and triceps surae (RER/M_{TS}); EMD_V, voluntary electromechanical delay; EMD_{twitch}, involuntary electromechanical delay

*$P < 0.05$

participants had reductions > 50%), as well as reduction in peak torque and voluntary activation observed previously in the same subject cohort (Trajano et al. 2014a), could result from the ischemia and pain caused by the muscle stretching. It is well documented that static stretching induces significant ischemia in the muscle (Trajano et al. 2014a), and in some lower-limb muscles extreme reductions in peak force and voluntary activation have been observed after a 2-min maximal voluntary contraction followed by 2 min of ischemia (Kennedy et al. 2015). In the study of Kennedy et al. (Kennedy et al. 2015), the average peak (MVC) force decreased to below 40% of its maximal value, while voluntary activation fell on average below 70% immediately after ischemia, suggesting that large reductions in the ability to voluntarily drive lower-limb muscles can be triggered even after ischemia and pain have subsided. Such magnitudes of effect are similar to those observed in the rate of torque development in the present study. Nonetheless, the decrement in rate of torque development was fully recovered by 15 min after both stretching conditions. The persistent reduction (30 min) in muscle function (peak torque) previously reported in the same subjects after intermittent stretch (Trajano et al. 2014a) was not observed for rate of torque development, suggesting that the negative effect of stretching on muscle function might more predominately affect measures of maximal than explosive torque. Thus, moderately long (5 min) periods of both intermittent and continuous moderate-duration passive stretch can temporarily reduce the ability to produce rapid force in human plantar flexors, although this reduction is fully recovered by 15 min after stretching.

The rate of torque development measured during electrically evoked tetanic contractions was reduced regardless of the stimulation frequency (20 Hz, variable-frequency, or 80 Hz), indicating that the muscle–tendon unit itself was compromised with respect to rate of torque development capacity. While these reductions were consistent between subjects, they were small and not correlated with reduction in RTD_{V200} (see Figs. 3 and 4). In addition, we could not detect any changes in the 20:VFT or 20:80 ratios, indicating a lack of clear change in the force–frequency relationship (de Ruiter et al. 1999). Disproportionate reductions in involuntary rate of torque development between the lower frequency (20 Hz) and lower frequency with initial doublet (20 Hz with doublet), i.e., the 20:VFT ratio, conditions could be taken as indicative of alterations in mechanisms such as the saturation of Ca^{2+} binding to troponin C and other mechanisms influencing myofibrillar Ca^{2+} sensitivity (Binder-Macleod and Kesar 2005; Bakker et al. 2017). Similarly, reductions in the ratio of involuntary rate of torque development produced by low- versus high-frequency tetanic stimulations, i.e., the 20:80 ratio, may indicate whether passive stretching affected the amount of calcium released by the sarcoplasmic reticulum and consequently the concentration of myoplasmatic-free Ca^{2+} (Balog 2010). However, in the present study, there was a similar reduction in rate of torque development measured in all stimulation conditions so there was no clear evidence to support a specific calcium-related mechanism. The stretching might therefore be considered to have promoted multiple changes within the muscle–tendon unit, including mechanical alterations within the muscle. The assumption that mechanical alterations may have occurred that would influence force transmission is supported by the observations of an increase in involuntary electromechanical delay immediately after both stretching conditions. The ramifications of this are discussed in more detail below.

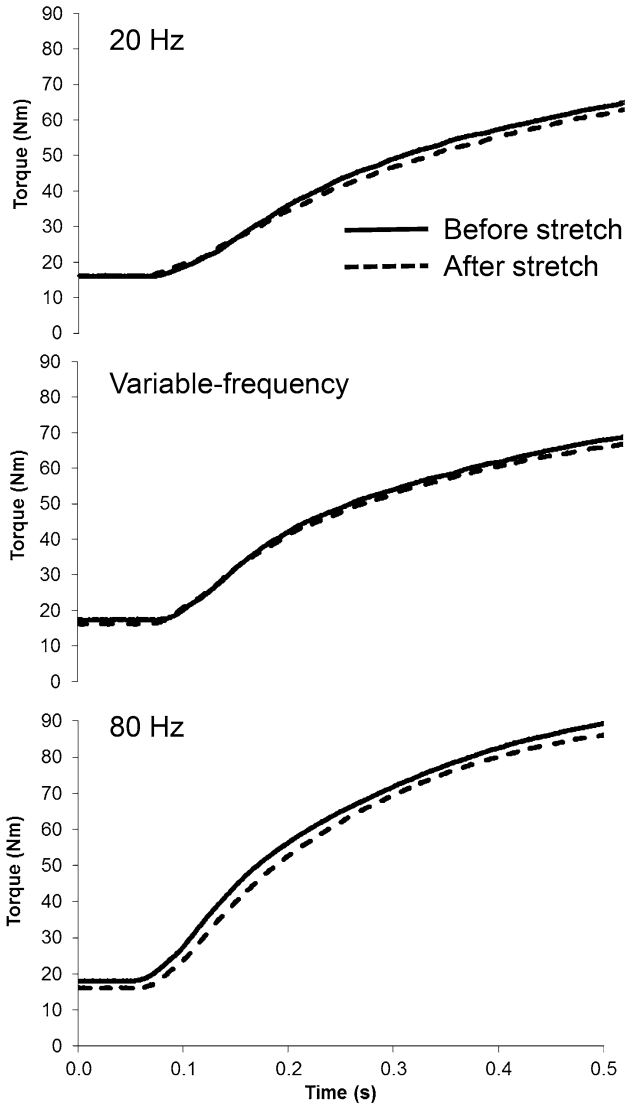

Fig. 3 Example of raw torque traces during 20 Hz (top panel), variable-frequency (middle panel) and 80 Hz (bottom panel) tetanic stimulation obtained from one subject before (solid line) and immediately after (dashed line) intermittent stretching

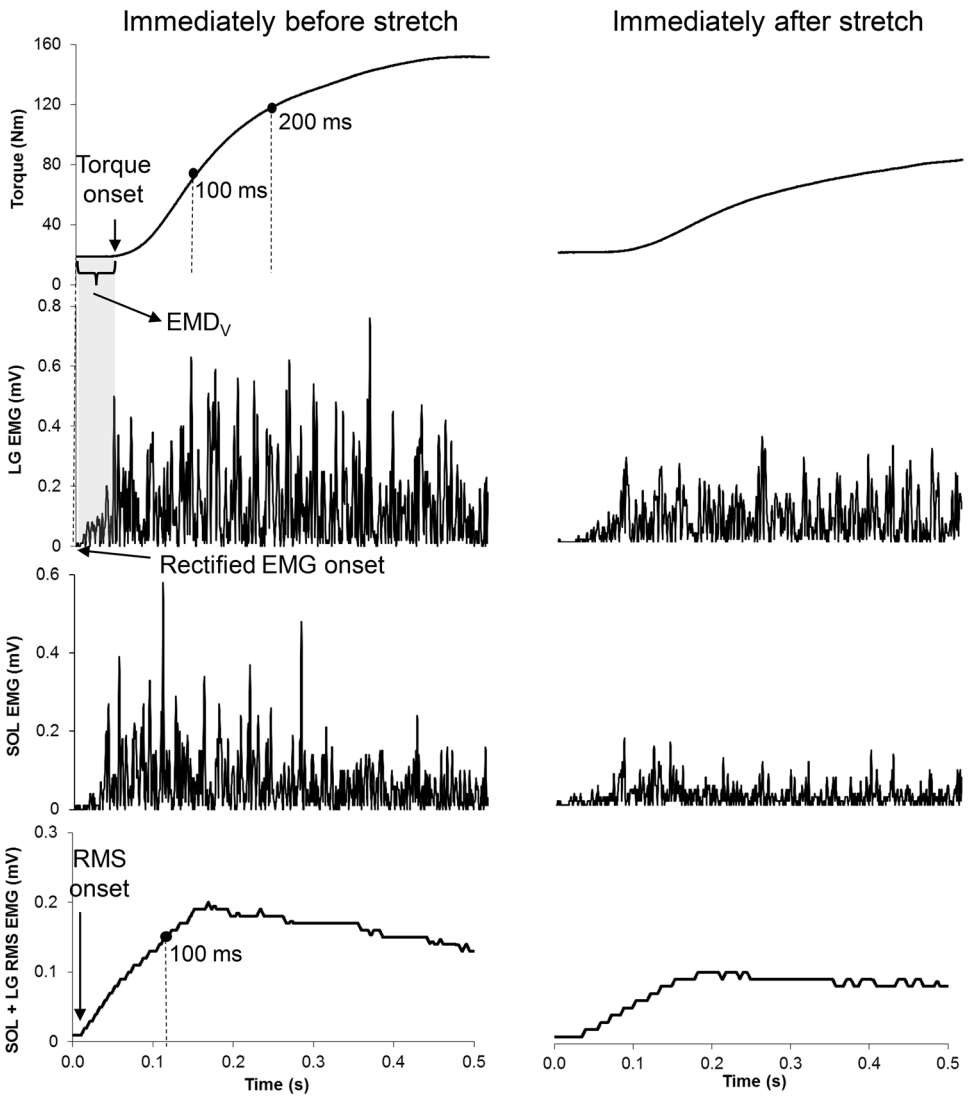

Fig. 4 Example of raw torque, rectified lateral gastrocnemius (LG) and soleus (SOL) EMG, and the summed SOL and LG root-mean-squared (RMS) data obtained from one participant during a voluntary contraction immediately before (left) and after (right) intermittent stretching. Example of measurement of torque onset, rate of torque development at 100 and 200 ms, rectified LG EMG onset, voluntary electromechanical delay (EMD_V), RMS EMG onset, and the rate of EMG rise at 100 ms. A slower rise of the voluntary torque trace with respect to time can be observed immediately after stretching (top right panel) when compared to before stretching (top left panel). The same pattern can be observed for the rise of the RMS EMG signal before (bottom left panel) and after stretching (bottom right panel)

A significant reduction was observed in the normalized rate of EMG rise measured in lateral gastrocnemius after intermittent stretching. This suggests that intermittent stretching reduced the rate at which lateral gastrocnemius motoneurons were excited. In addition, the changes observed in triceps surae (lateral gastrocnemius + soleus) rate of EMG rise immediately after both stretching conditions were strongly associated with the changes in rate of torque development measured from 0 to 200 ms (Fig. 5). Using a linear model, it was found that 42% of the variability in rate of torque development was associated with changes in rate of EMG rise after intermittent stretching and 41% of the variability was associated with changes in rate of EMG rise after continuous stretching. Thus, reduction in the ability of the nervous system to rapidly recruit the lateral gastrocnemius motor units might have contributed to the large reduction in the rate of torque development observed after intermittent stretching. When muscles contract rapidly, the recruitment threshold of motor units is decreased (i.e., motor units are recruited at a lower force level in comparison with slow, ramped contractions) and the initial firing rates of these units are remarkably high (e.g., doublet discharges) (Desmedt and Godaux 1977; Duchateau and Baudry 2014). The decrease in recruitment thresholds and the presence of doublet discharges are indicative of an increase in motoneuron excitability, possibly resulting from motoneuronal facilitation (Heckman and Enoka 2012). In particular, plateau potentials caused by the facilitation exerted by persistent inward currents (PICs) have been suggested to be a potential mechanism for doublet discharges in human motoneurons (Kudina and Andreeva 2010). We have previously provided evidence that passive stretching might cause disfacilitation at the motoneuronal level, preventing the occurrence of plateau potentials and thus self-sustained motor unit firing (Trajano et al. 2014b). This disfacilitation could have

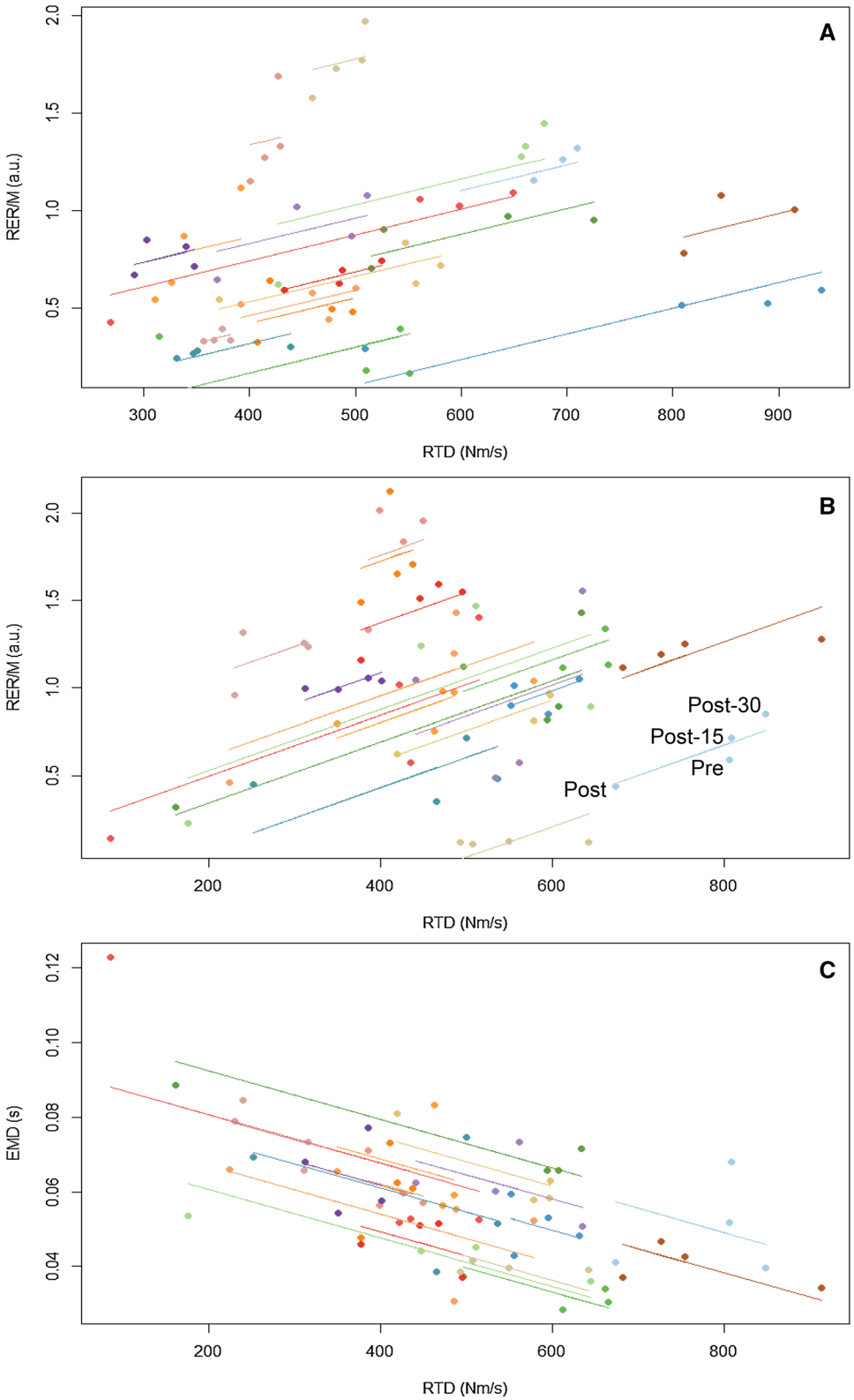

Fig. 5 Repeated-measures correlations plot illustrating the association between the rate of force development (RTD) and: **a** the rate of EMG rise (RER/M) during the continuous stretch condition, **b** the RER/M during the intermittent stretch condition, **c** the voluntary electro mechanical delay (EMD) during the intermittent stretch condition. Separate parallel lines are fitted to the data from each participant along time (pre, post, post-15 and post-30) and are represented by different colors

attenuated the typical recruitment threshold reduction observed during fast contractions [compared to slow, ramped contractions (Desmedt and Godaux 1977)] and reduced motor unit firing rates, particularly at contraction onset when initial high-frequency discharges occur that seem to be of fundamental importance to achieving higher rates of torque development. Future studies using high-density EMG arrays in conjunction with motor unit

decomposition techniques might shed light on the recruitment patterns of motor units during the force rise phase of muscular contraction after muscle stretching.

Another finding of the present study was that there was an increase in voluntary electromechanical delay only after intermittent stretching, which was observed immediately and 15 min after stretch, and large correlations were observed between changes in voluntary electromechanical delay and changes in rate of force development immediately after intermittent stretching. Also, there was an increase in involuntary (electrically stimulated) electromechanical delay immediately after both stretching protocols that were observed immediately after continuous stretch and immediately, 15 min and 30 min after intermittent stretch. It seems that both stretch protocols increased the electromechanical delay when the muscle was activated synchronously (time-locked activation of all motor units) via single pulse electrical nerve stimulation (i.e., EMD_{twitch}). This is in agreement with previous research reporting an acute increase in electromechanical delay measured during electrically evoked contractions immediately after passive stretching (Costa et al. 2010; Esposito et al. 2011). In contrast, only intermittent stretching prolonged the electromechanical delay when the muscle was activated in a physiological, asynchronous manner via voluntary motor command, but, importantly, this prolonged time to transmit the force was associated with slower rates of torque development. Collectively, these results suggest that there was a slower force transmission through the muscle–tendon unit, which could suggest an increase in its compliance, with this change being of greater significance to voluntary force production after intermittent stretching. An increase in compliance in the series elastic components has been associated with a longer electromechanical delay (Nordez et al. 2009), which may ultimately reduce the rate of force development (Maffiuletti et al. 2016). However, it has been previously shown that stretching does not decrease tendon stiffness if the muscle–tendon unit has been pre-conditioned (i.e., warm-up and sub-maximal contractions) prior to passive stretching (Morse et al. 2008; Kay and Blazevich 2009), as was the case in the present study. Thus, it is possible the stretching protocols might have affected the stiffness of titin or connective tissues within the muscle itself, such as perimysium or aponeurosis.

In summary, both continuous and intermittent muscle stretching performed for 5 min reduced voluntary rapid force production ability in the human plantar flexors. We present evidence to support the hypothesis that neural factors are associated with reduction of rate of torque development (RTD_{V200}) observed immediately after intermittent stretching. This conclusion is supported by the significant reduction observed in the lateral gastrocnemius rate of EMG rise after intermittent stretching and the large correlations observed between changes in RTD_{V200} and changes in normalized rate of EMG rise after both stretching conditions. Our data showing reduction in involuntary rate of torque development and increase in voluntary and involuntary electromechanical delay also suggest that the mechanical transmission of force along the muscle–tendon unit might have been affected by stretching. The longer voluntary electromechanical delay was also related to the changes in voluntary rate of torque development. Also, changes at the muscular level, indicated by decrease in electrically evoked rates of torque development were observed, but these were small and not associated with changes in voluntary rate of torque development. Furthermore, there was no clear evidence that calcium-related mechanisms were specifically affected by stretching protocols or had a meaningful effect on rate of torque development in the current study. Thus, both mechanical alterations in force transmission within the muscle and the loss of rapid muscle activation capacity resulting from a decreased central drive emerged as mechanisms related to the loss of rapid force production. From a practical point of view, it is notable that the reductions in voluntary rate of torque development were fully recovered within 15 min, thus practitioners might consider imposing a short time delay between stretching exercises and activities that require high rates of force/torque development when moderate durations (e.g., 5 min) of stretching are imposed on the muscles.

Author contributions GST and AJB conceived and designed the study. GST and LS conducted the experiments. GST analyzed the data, and drafted the first version of the manuscript. GST, LB, KN, and AJB critically revised the manuscript. All authors read and approved the manuscript.

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Compliance with ethical standards

Conflict of interest No conflicts of interest, financial or otherwise, are declared by the author(s).

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