Do Stretch Durations Affect Muscle Mechanical and Neurophysiological Properties?

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Key words
- H reflex
- M wave
- soleus
- stiffness
- dose-response

Abstract

The aim of the study was to determine whether stretching durations influence acute changes of mechanical and neurophysiological properties of plantar flexor muscles. Plantar flexors of 10 active males were stretched in passive conditions on an isokinetic dynamometer. Different durations of static stretching were tested in 5 randomly ordered experimental trials (1, 2, 3, 4 and 10×30-s). Fascicle stiffness index, evoked contractile properties and spinal excitability (Hmax/Mmax) were examined before (PRE), immediately after (POST0) and 5 min after (POST5) stretching. No stretch duration effect was recorded for any variable. Moreover, whatever the stretching duration, stiffness index, peak twitch torque and rate of force development were significantly lower at POST0 and POST5 as compared to PRE ($P<0.05$). Electromechanical delay was longer at POST0 and POST5 as compared to PRE ($P<0.05$). Whatever the stretch duration, no significant changes of Hmax/Mmax ratio were recorded. In conclusion, 30 s of static stretching to maximum tolerated discomfort is sufficient enough to alter mechanical properties of plantar flexor muscles, but 10×30 s does not significantly affect these properties further. Stretching does not impair spinal excitability.

Introduction

Stretching has been used for a long time in physical activity to increase range of motion around a joint. Stretching also demonstrated some acute detrimental effects. Recently, significant reductions in maximal voluntary strength, muscle power or evoked contractile properties were recorded immediately after a single bout of static stretching, underpinned by various mechanisms [1,3–5, 8,9,18,19,21,24,38,41]. Mechanical impairments may originate from musculotendinous stiffness reductions [34] and a shift of the optimal length toward longer muscle lengths [38]. This could be attributed to passive torque reductions [18,19, 29] and/or to changes of flexibility and displacement of the connective tissue and/or muscle fascicles [29,30]. Neural factors, witnessed by a reduction in muscle activity [3], may also play a key role more particularly close to stretch interventions, since they are quickly restored [9]. This decrease is generally ascribed to reductions in motoneuron excitation due to sensory afferents. Pre and post-synaptic inhibitions have been suggested [12]. It seems that alteration in the discharge rate of muscle spindles at rest, sensitive to changes in muscle length or stiffness, is one potential origin [1].

The magnitude of these deleterious effects could be attributed to several factors such as the studied population [2] or characteristics of stretching interventions [5]. For example, it is well established that stretching-induced force decreases are dependent on stretch durations; the longer the stretch duration, the greater the force reductions [5,21]. While static stretching shorter than 30 s produces trivial strength decreases, solid evidence is present in the literature with longer durations (>90 s) [5]. However, the effects of different static stretching durations on neuromuscular and neurophysiological parameters are not clearly determined. Indeed, just a few studies have examined the duration influence on MTU stiffness [14,26,32,34], on evoked contractile properties [33] and none on spinal excitability. Therefore, the aim of the present study was to investigate the acute effects of different stretching durations on neuromuscular and neurophysiological properties of plantar flexor muscles. Based on previous studies, it was hypothesized

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that the magnitude of stretch-induced stiffness and evoked contractile properties changes would be dependent on static stretching duration.

Materials & Methods

Participants

10 active men, recruited from the sport science faculty, with no recent history of lower limb injury or illness volunteered for the study. Their mean ± standard deviation (SD) age, height and body mass were 24.0 ± 1.5 years, 177.5 ± 5.5 cm and 72.0 ± 5.5 kg, respectively. All were recreationally active with ~7 h training per week. Participants were requested to refrain from intense exercise and flexibility training for 24 h before testing sessions. Prior to participation, they were fully informed about the purpose of the study and experimental procedure. All signed an informed consent form. The study was conducted according to the Declaration of Helsinki, to the ethical standards of the journal [13] and approval was obtained from the local committee on human research.

Experimental procedure

Participants attended the laboratory on 5 separate occasions to determine the effects of 5 static stretching durations on an index of fascicle stiffness, contractile properties and spinal excitability. As the experiment involved an acute intervention, limb dominance was ignored and all static stretching procedures were conducted on the right plantar flexor muscles. Static stretching durations were randomly presented and were 1 × 30 s, 2 × 30 s, 3 × 30 s, 4 × 30 s and 10 × 30 s. Randomization was determined by participants at the beginning of the first session. Sessions were separated by at least 48 h. No warm-up was performed prior to experiments. Once the equipment was installed, participants conducted one of the 5 stretching durations.

Stretching was performed on an isokinetic dynamometer (Biodex System 4, BIODEX Corporation, Shirley NY, USA). Subjects were lying prone on the dynamometer with the knee fully extended (180°) to ensure that the triceps surae was placed under significant stretch and contributed significantly to plantar flexor joint moment. The ankle joint angle was measured using an electro-goniometer (TSD 130B, Biopac systems Inc., Santa Barbara, CA, USA) attached laterally at the ankle. The foot was securely attached to the footplate of the dynamometer. To minimize heel displacements, the foot was first positioned and fastened inside a shoe (adapted to participant's size and fixed by the sole to the dynamometer footplate) and second firmly attached to the footplate with straps. The lateral malleolus was aligned to the center of rotation of the dynamometer. From here, participants were in a fixed position attached to the dynamometer for ~20 min when stretching was performed only once and for ~35 min with 10 repetitions. The passive range of motion was first determined starting from a maximal plantar flexion, then slowly stretching plantar flexor muscles until the maximal tolerated discomfort and returned immediately to an neutral position (0° = sole of the foot perpendicular to the leg). This angle was used as the end range of motion (ROM) for every stretch [4, 18]. During the stretching procedure (immediately performed after the initial tests), subjects' ankle was passively rotated though the thus-determined ROM at 2 °.s^{-1}, a slow angular velocity to avoid myotatic reflex [27], which ensured that full ROM was achieved. This maximal stretched position was held for 30 s. The ankle was then released to return back to maximal plantar flexion at 5 °.s^{-1}. This procedure was repeated 1, 2, 3, 4 and 10 times according to the stretch duration tested. No rest was permitted between stretches. Therefore, the stretch procedure used here corresponded to cyclic stretches followed by a 30-s static hold in the maximal dorsiflexion position. This stretching procedure was performed 5 min after initial tests and maximal dorsiflexion angle determination. Participants were instructed to relax while stretching, in order not to offer any resistance to the passive motion of the dynamometer.

Measurements

Tests (total duration = 2 min) were conducted before (PRE), immediately and 5 min after the stretching procedure (POST0 and POST5, respectively). They consisted in measurements of the fascicle stiffness index via passive ankle torque and fascicle length variations, followed by spinal excitability via maximal H reflex (Hmax) and M wave (Mmax) amplitudes, and finally by plantar flexors' contractile properties via peak twitch torque (PTT), rate of force development (RFD) and electromechanical delay (EMD). During tests and the 5 min of rest after the stretching procedure, the ankle joint was positioned in neutral position for spinal excitability and contractile properties assessments.

To determine the fascicle stiffness index, passive ankle torque and fascicle length were measured. Passive torque was measured via the Biodex dynamometer throughout the complete dorsiflexion ROM that was determined before the stretching procedure. Torque was recorded using TIDA software (Heka Elektronik, Lambrecht, Germany). Fascicle length changes were measured using a real-time B-mode ultrasound video imaging (AU5; Esaote Biomedica, Florence, Italy). A 7.5-MHz linear-array probe was oriented along the longitudinal axis of the MTU and held at 50 % of gastrocnemius medialis muscle length in the midsagittal line. The movement of the muscle fascicles from the deep to superficial aponeuroses was measured. With ultrasonography, when fascicles extended off the acquired ultrasound image, the length of the missing portion of the fascicle was estimated by extrapolating linearly both the fascicular path, visible in the image, and the aponeurosis. Ultrasound images, goniometer and passive torque data were synchronized using a custom-made trigger. Images were then extracted every 5° of dorsiflexion, except for the last 5°, for which they were extracted every 1° [29]. The passive fascicle stiffness index was quantified by expressing variations of passive torque to variations of fascicle length. Passive torque and fascicle length variations were quantified as the difference between maximal plantar flexion and maximal dorsiflexion positions. Length variations of 2 fascicles were considered and averaged, using an open source digital measurement software (Image J, NIH, USA). The angle of insertion of the fascicles with the deep aponeurosis (pennation angle) was also measured.

Posterior tibial nerve stimulation was used (DS7A, Digitimer, Hertfordshire, UK) for neuromuscular properties. As indicated above, a 0° ankle joint angle was used. The electrogoniometer was used to control this ankle angle for PRE and POST measurements. The cathode (10 mm diameter) was pasted in the popliteal fossa and the anode (5 × 10 cm) was pasted to the patella on the anterior surface of the knee. Electrical stimuli were rectangular pulses (1 ms duration) delivered with increasing current intensities. First, 2-mA increments were used from 0 until the soleus Hmax was obtained. Second, 5-mA increments were used until the soleus Mmax. 3 stimulations were performed at each

intensity with a 5-s interval between stimuli. The thus-adjusted Hmax and Mmax stimulation intensities were applied for PRE, POST0 and POST5 measurements with 6 successive stimuli, separated by 10 s and alternating Hmax and Mmax intensities. Hmax and Mmax peak-to-peak amplitudes were measured from the soleus electromyographic traces (EMG). A Hmax/Mmax ratio was subsequently calculated, from the average peak-to-peak amplitudes of Hmax expressed in relation to the average peak to peak amplitudes of Mmax [1, 15, 38]. 10 s after Hmax and Mmax measurements, a triplet was delivered (3 stimuli at Mmax intensity, 100 Hz frequency). The mechanical responses were used to calculate the peak torque of the triplet (PTT), the maximal rate of force development (RFD, maximal value measured from the torque first derivate), and electromechanical delay (EMD, delay between the M-wave of the first stimulus and the onset of torque rise).

Mechanical and EMG signals were digitized online using a Tida system with a 5000 Hz sampling frequency. Surface EMG was collected with one pair of silver-chloride electrodes applied over the soleus along the mid-dorsal line of the leg, i.e., ~5 cm distal from where the 2 heads of the gastrocnemius join the Achilles tendon. Electrodes were 10 mm diameter with 20 mm inter-electrode distance. The reference electrode was fixed halfway of gastrocnemii bellies. Low impedance (< 2000 Ω) of the skin-electrode interface was obtained by shaving, abrading with sandpaper and cleansing with alcohol. EMG signals were amplified with a bandwidth frequency ranging from 10 to 5000 Hz (gain = 500).

Statistical analysis

Quantitative variables were presented as mean values and standard deviations (SD). A 2-way analysis of variance (ANOVA) with repeated measures was used to analyze the fascicle stiffness index, variations of passive torque, fascicles length and pennation angle, Hmax and Mmax amplitudes, Hmax/Mmax ratio, PTT, RFD and EMD. Time (PRE vs. POST0 vs. POST5) and stretch duration (1 × 30 s, 2 × 30 s, 3 × 30 s, 4 × 30 s and 10 × 30 s) were used as repeated measures. When significant main effects or interactions were present, a Student Newman-Keuls post hoc test was subsequently conducted. Statistical significance was accepted at an alpha level of 0.05. Statistics were performed using Statistica software (version 8.0, Statsoft, Tulsa, USA). Statistical power was generally high, being 1.00 for stiffness and passive torque, 0.99 for PTT, 0.95 for RFD and 0.96 for EMD.

Results

Statistical analyses did not reveal any significant interaction (time × stretch duration) for all variables studied. Moreover, no stretch duration effect was obtained. Our data only revealed significant main time effects (PRE vs. POST0 vs. POST5) for most variables.

Static stretching significantly affected the passive fascicle stiffness index. Whatever the stretching duration, the stiffness index was significantly lower at POST0 when compared to PRE (mean decrease: −14.5 ± 10.4 %; $P < 0.05$). Values did not return to baseline at POST5 and were not significantly different when compared to POST0 (**Fig. 1a**). Stiffness alterations were mainly attributed to passive torque and not to architectural changes. Indeed, no main effects and interactions were obtained for fascicles length and pennation angle (**Table 1**). As for passive ankle torque, a significant reduction was measured POST0 as compared to PRE (mean decrease: −15.2 ± 12.9 %; $P < 0.05$). Values did not return to baseline at POST5 and values were not different between POST0 and POST5 (**Fig. 1b**).

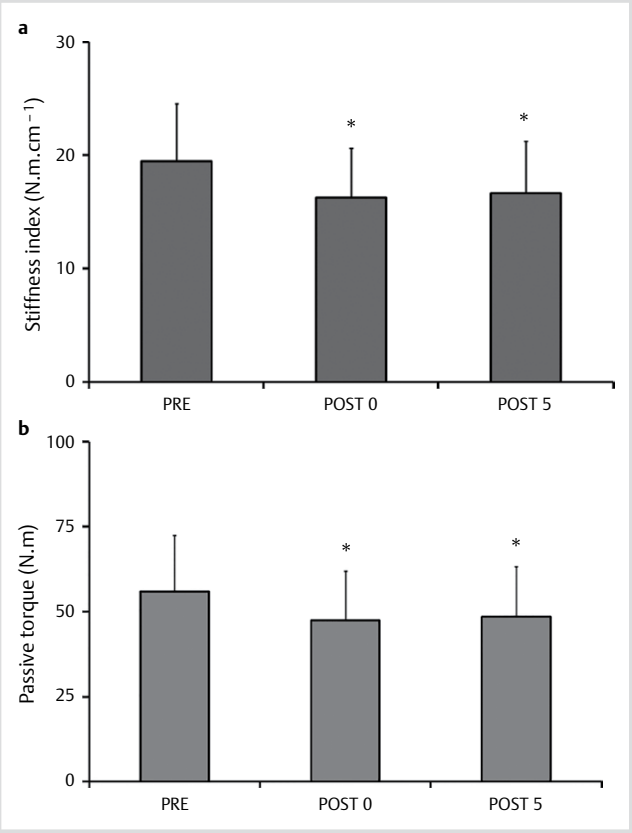

Fig. 1 Effects of stretching on fascicle stiffness index **a** and passive torque **b** assessed before (PRE), immediately (POST0) and 5 min (POST5) after stretching. The 5 time condition values are averaged (mean values ± SD). *: significant differences with PRE ($P < 0.05$).

Table 1 Variations of fascicle length, pennation angle and passive torque assessed before (PRE), immediately (POST0) and 5 min (POST5) after stretching.

		Fascicle length variation (mm)	Pennation angle variation (deg)	Passive torque variation (N.m)
1 × 30 s	PRE	27.8 ± 5.5	12.5 ± 3.3	53.0 ± 17.2
	POST 0	28.5 ± 4.5	12.4 ± 3.3	45.2 ± 16.4 *
	POST 5	28.5 ± 4.9	11.7 ± 2.4	47.3 ± 15.9 *
2 × 30 s	PRE	28.6 ± 5.4	11.9 ± 2.8	58.6 ± 14.1
	POST 0	28.8 ± 5.2	11.7 ± 2.9	49.8 ± 12.4 *
	POST 5	29.0 ± 4.5	12.6 ± 2.3	51.8 ± 14.3 *
3 × 30 s	PRE	29.8 ± 7.6	12.7 ± 2.7	54.8 ± 16.4
	POST 0	30.5 ± 6.4	12.2 ± 2.5	48.7 ± 16.7 *
	POST 5	30.2 ± 6.6	11.9 ± 2.6	48.7 ± 15.7 *
4 × 30 s	PRE	30.3 ± 7.3	12.1 ± 2.7	57.4 ± 18.8
	POST 0	30.4 ± 7.2	11.7 ± 2.4	49.4 ± 15.7 *
	POST 5	30.5 ± 6.2	11.7 ± 2.3	49.7 ± 17.5 *
10 × 30 s	PRE	28.5 ± 6.0	12.1 ± 3.1	55.3 ± 17.0
	POST 0	29.2 ± 6.0	11.4 ± 3.1	43.6 ± 12.2 *
	POST 5	28.8 ± 6.0	11.5 ± 2.8	45.0 ± 10.6 *

Values represent changes between maximal plantarflexion and maximal dorsiflexion and are expressed as mean values ± SD

* Significant differences with PRE ($P < 0.05$)

Table 2 Hmax, Mmax amplitudes and Hmax/Mmax ratio assessed before (PRE), immediately (POST0) and 5 min (POST5) after stretching.

		Hmax (mV)	Mmax (mV)	Hmax/ Mmax ratio
1×30 s	PRE	2.9±2.5	5.4±3.4	0.51±0.22
	POST0	2.9±2.6	5.6±3.3	0.49±0.20
	POST5	2.8±2.8	5.5±3.4	0.46±0.24
2×30 s	PRE	2.8±3.3	5.4±4.3	0.53±0.22
	POST0	2.9±3.4	5.6±4.4	0.51±0.23
	POST5	2.9±3.7	5.6±4.5	0.55±0.22
3×30 s	PRE	3.6±2.0	6.0±3.6	0.68±0.35
	POST0	3.6±2.1	6.1±3.6	0.66±0.36
	POST5	3.6±2.1	6.1±3.6	0.62±0.31
4×30 s	PRE	2.3±1.7	5.0±1.5	0.44±0.20
	POST0	2.4±1.9	5.1±1.7	0.43±0.23
	POST5	2.4±2.0	5.2±1.7	0.45±0.25
10×30 s	PRE	2.5±1.5	4.5±2.3	0.56±0.16
	POST0	2.5±1.8	4.5±2.2	0.54±0.18
	POST5	2.5±1.4	4.6±2.4	0.53±0.15

Values are expressed as mean values ± SD

No stretch duration effect was recorded for Hmax, Mmax and Hmax/Mmax ratio (**Table 2**). Moreover, no significant alteration of these parameters was obtained at POST0 and POST5, as compared to PRE.

PTT, RFD and EMD modifications were independent of the stretch duration. Whatever the stretch duration, PTT and RFD were significantly lower ($P<0.05$) at POST0 as compared to PRE (mean decrease: −5.4±6.5% and −2.8±4%, for PTT and RFD, respectively) (**Fig. 2**). EMD was altered by stretch interventions with a slight but significant increase in time (+8.8±1.5%; $P<0.05$). PTT, RFD and EMD did not return to baseline 5 min after stretch interventions and values were not different between POST0 and POST5.

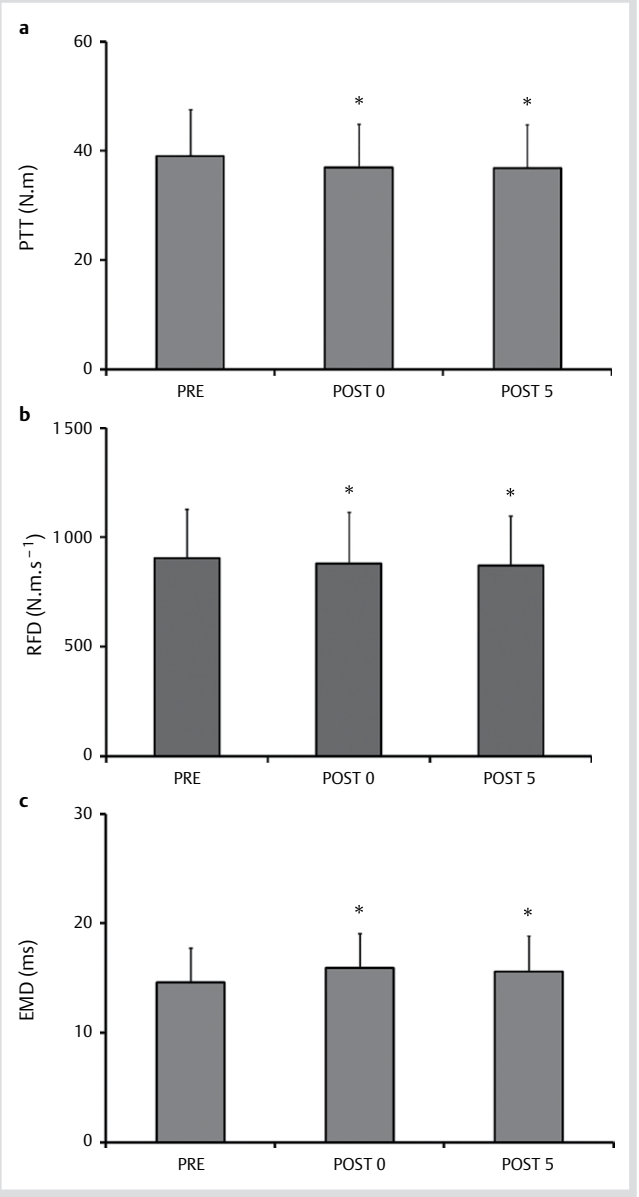

Fig. 2 Effects of stretching on PTT **a**, RFD **b** and EMD **c** assessed before (PRE), immediately (POST0) and 5 min (POST5) after stretching. The 5 time condition values are averaged (mean values ± SD). *: significant differences with PRE ($P<0.05$).

Discussion

The aim of the present study was to determine the influence of stretching duration on neuromuscular properties such as the fascicle stiffness index, contractile properties and the spinal excitability of plantar flexor muscles. Our results did not demonstrate any static stretching duration effect. However, static stretching produced a reduction of passive fascicle stiffness index and contractile properties. No alteration of spinal excitability was observed.

Stretching duration

The present study demonstrated that changes in mechanical properties (i.e., passive fascicle stiffness and contractile properties) occurred immediately after a single 30-s static stretching bout. Previous studies using short static stretching durations also recorded such impairments [14, 32, 34, 36, 41]. For example, Ryan et al. [34], reported impairments of musculotendinous stiffness after two 30-s bouts of stretching. Also, Sekir et al. [36], using 2×20 s static stretches, registered concentric and eccentric torque reductions.

However, our main results indicated that passive fascicle stiffness, twitch contractile properties and spinal excitability of plantar flexor muscles were not influenced by static stretching duration. Indeed, the repetition of 30-s static stretching resulted in similar alterations compared to only one 30-s stretch. However, regarding the literature, evidence exists which indicates that the likelihood and magnitude of force decreases are dependent on stretch durations [5, 21]; the longer the stretch duration, the greater the force reductions [5, 21]. Nevertheless, stretching duration influences on mechanical and neurophysiological mechanisms underpinning force losses are not clearly determined. Indeed, conflicting results were observed, especially concerning visco-elastic properties. In agreement with our study, Ryan et al. [32] reported that 2-min static stretching resulted in similar MTU stiffness decrements than 4-min and 8-min. Similarly, Ryan et al. [34] showed a significant stiffness index reduction after 2×30 s static stretching (a single stretch was not sufficient enough to induce stiffness changes) and 2 additional 30-s stretches did not decrease stiffness further. In contrast, Herda et al. [14] reported that stiffness decreased after 30-s stretching, with subsequent decreases up to 6-min after stretching. Moreover, for Matsuo et al. [26], stiffness was not sig-

nificantly depressed after 20-s and 60-s stretching bouts, but was after 180-s and 300-s stretching bouts.

These conflicting results could originate from discrepancies in protocols, such as muscle group. For example, Matsuo et al. [26] stretched hamstrings whereas others such as Ryan et al. [32,34] and our study considered plantar flexor muscles. Each muscle is unique in terms of its structural organization (e.g., muscle-tendon interaction), which could have significant functional influence. For instance, hamstrings (long fascicle length and intermediate PCSA) are designed for low force production with large excursions, whereas plantarflexors (short fascicle length with large PCSA) are designed toward force production with short excursions [23]. Also, the muscle-tendon structural characteristics may influence force production capacity. During running, for example, fascicle and tendon length have been shown to have major influence on stiffness [35]. Little is known with respect to this muscle-tendon interaction following stretching, but we can suggest that it could have an influence on the magnitude of the stretch-induced passive stiffness alterations.

The muscle group studied could not solely explain the discrepancies obtained with some studies [33]. Differences in stretching and experimental procedures might also explain these conflicting results. Indeed, no warm-up was used in the present study while others [33] requested participants to perform two 5-s isometric maximal voluntary contractions before stretching. Some authors revealed that the negative effects of stretching intervention could be eliminated, if stretching was performed after isometric contractions [20]. Moreover, some authors such as Strickler et al. [37] suggested that tissue temperature could have a significant effect on a muscle's responses to stretching. As a result, maximal muscle contractions included in warm-up might likely have altered the stretch-induced stiffness and force production alterations, at least for short stretching durations.

In addition, Herda et al. [14] used a constant-torque passive stretching protocol, whereas we used a constant-angle static stretching. Some authors have recently reported differences between these 2 stretching procedures on the visco-elastic properties [14,42]. Indeed, constant-torque stretching protocols could provide more tension on the muscle and could result in greater changes in visco-elastic properties compared to a constant-angle protocol. In fact, while holding a stretch at a constant angle, a stress/relaxation response occurs, which may produce less tension on the muscle. Thus, it appeared that constant-angle and constant-torque stretching affect the muscle-tendon visco-elastic properties differently. It could mostly explain the lack of further stiffness decreases with longer stretching durations. Against this hypothesis, Ryan et al. [32,34], using a constant-torque protocol, did not report any stretching duration effect. However, maximal voluntary contraction was conducted prior to the first pretreatment stiffness assessment, which could minimize stretch-induced stiffness decrements and the magnitude of stretching duration effects.

Twitch contractile properties modifications were also independent of the stretch duration. To our knowledge, only one experimental study has compared the effect of different durations of static stretching on these parameters [33]. These authors showed that 2 min of static stretching did not affect plantar flexors contractile properties, whereas 4 and 8 min did. This study supported a stretching duration effect that contradicted our present findings.

The time course of the effect of static stretching has also been investigated. The results showed that stretch-induced fascicle stiffness and twitch contractile properties changes did not disappear within 5 min after stretching. Indeed, these parameters remained depressed and did not change further. Mizuno et al. [28] presented that decreased stiffness returned to baseline within 15 min. Moreover, they assessed a lower diminution compared to the present study. Ryan et al. [32] have also showed that stiffness decreased immediately after stretching and returned to baseline within 10 min following 2 min of stretching, and within 20 min after 4–8 min of stretching. These 2 studies confirmed that alterations of these mechanical parameters do not disappear immediately after stretching. However, some other studies revealed conflicting results. Indeed, it has already been shown that with some types of stretching applications, mechanical effects may disappear immediately after stretching [25,27]. Magnusson et al. have shown that 3 repeated static stretches of 45 s separated by 30 s resulted in no change in torque/angle curves. In McNair's study (with 10-s rest periods between 4×15 s and 2×30 s static stretches), there was no change in stiffness after stretching. These conflicting results may be explained in part by the discrepancies in stretching procedure. Indeed, in Magnusson's study, care was taken to avoid a painful response during determination of the final angle that consequently induced a lower stretch load. Moreover, during the stretch maneuver, 5 °.s^{-1} was used (a greater velocity than in our study), allowing muscles to be stretched during a shorter period. In McNair and Magnusson's studies, short rest periods were presented between stretching phases, providing an additional recovery time. These conflicting results show that the biomechanical effects of stretching may vary depending on the stretch characteristics (i.e., intensity, duration, type of stretch application) and that there might be a threshold at which these effects become more long lasting.

It should finally be noted that no control condition (i.e., with no stretch) was tested here. Therefore, the testing procedure used in the present study could have influenced our results. Additional measurements have been performed and conducted on 3 participants before the onset of the study to make sure that results were not influenced by the experimental procedure. Fascicle stiffness index measurements have been performed before (PRE), immediately (POST0) and 5-min (POST5) after a rest duration corresponding to our 10-stretch condition. Except during fascicule stiffness index measurements, the ankle was in neutral dorsiflexion position and attached to the dynamometer continuously for ~35 min. These additional measurements confirmed that the stiffness evaluations procedure did not affect stiffness changes.

Acute changes in mechanical properties

We reported that passive fascicle stiffness was significantly reduced after stretching. A number of studies have also reported such a decrease [7,14,20,26,28–30,32,34]. In the present study, these impairments are not architectural in nature because of the lack of changes in fascicles length and pennation angle variations. Similar findings were recently obtained [7]. Nevertheless, authors suggested that modifications of the aponeurosis and connective tissues, such as endomysium, perimysium and epimysium, considered as a major extracellular contributor to passive tension, could lead to a change in overall stiffness [10,29,30]. However, the nature of these elements and how they are affected by passive stretching is still partly unknown and a future topic of interest.

Considering decreases in passive ankle torque, mechanical modifications are more likely involved in fascicle stiffness decrements. For similar stretching durations, literature reported stiffness and passive torque reductions, confirming our findings [14,26,28,29]. When MTU is passively stretched, the resistance produced could originate from several structures and mechanisms, including non-contractile protein elongation (especially titin) [10,31,34]. For these authors, titin is thought to be one important source of passive tension and to resist against lengthening in relaxed muscles. Being very solicited during stretching [39,40], alterations of titin could partly explain stiffness decrements. Alterations of titin could result in modifications of filament overlapping [16]. This could lead to a decrease in the number of attached cross bridges and thus to decrease passive tension and passive stiffness of the sarcomere. However, the lack of any fascicle length alteration would partly exclude this mechanism.

Another potential mechanism is related to altered stable cross-bridges between actin and myosin filaments [10]. Authors proposed that these residual cross-bridges could be a significant origin of the passive tension during stretching [10,31].

Other mechanisms such as altered length-tension characteristics [8,9] and changes in length of tendons and aponeuroses [22] may influence force-generating abilities after stretching. Tendons, not investigated here, could not be excluded from potential mechanisms. However, the contribution of this structure to stiffness changes remains debated. Indeed, while some authors registered tendon stiffness decreases [17,22], others were unable to measure any tendon stiffness modifications after stretching [6,19].

Acute changes in neurophysiological properties

While changes in structural and mechanical properties of the MTU are well documented, changes in neural factors are less explored. In the present study, no alteration of soleus spinal excitability was observed. Indeed, H/M ratio was unchanged after stretching. Other authors also reported such a result [38]. Nevertheless, stretch-induced force losses have been ascribed in part to neural factors, including changes in spinal excitability [1]. However, their stretching protocol, involving repeated stretches at a high speed for an hour, was different from that of Weir et al. [38] and the present study, which could induce different neurophysiological changes [38]. Guissard et al. [11] supposed also that static stretching could decrease spinal excitability. However, in their study, changes in H/M ratio occurred during stretching. It is suggested that spinal excitability could be affected during stretching and recovered immediately after stretching. Therefore, we can speculate these inhibitions would last only as long as the stretching maneuver is maintained, even in the case of 10×30-s static stretching. To explain spinal excitability changes, Avela et al. [1] have suggested a reduced resting discharge of muscle spindles induced by a reduction in muscle stiffness. Nevertheless, the present modifications of stiffness occurred without any change in H/M ratio, therefore without any change in muscle spindles afferents sensibility. As a consequence, it does not seem so obvious that the decreased stiffness of muscles plays a part in altering the spinal excitability.

Conclusion

While spinal excitability was unaffected by stretching, 30 s of static stretching to maximum tolerated discomfort reduced fascicle stiffness and twitch contractile properties. However, 10 repetitions of 30-s stretching did not significantly affect more. This suggests that the dose-response effect between stretching duration and stretch-induced changes is not as evident. There are suggestions from our findings that the changes within the MTU may be due, in part, to altered properties of connective tissue elements and non-contractile protein. Nevertheless, the nature and the contribution of these elements to stretching is a topic of future interest. Regardless of stretch duration, static stretching should be avoided in athletes prior to strength, speed or explosive activities. However, in athletes conducting activities with high degree of flexibility or in patients, the present study demonstrated that stiffness reductions are obtained even after a single short duration stretching. Repeating stretching does not appear more effective.

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