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Muscle Stiffness and Spinal Stretch Reflex Sensitivity in the Triceps Surae

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Context: Greater musculotendinous stiffness may enhance spinal stretch reflex sensitivity by improving mechanical coupling of the muscle spindle and the stretch stimulus. This heightened sensitivity would correspond with a shorter latency and higher-amplitude reflex response, potentially enhancing joint stability.

Objective: To compare spinal stretch reflex latency and amplitude across groups that differed in musculotendinous stiffness.

Design: Static group comparisons.

Setting: Research laboratory.

Patients or Other Participants: Forty physically active individuals (20 men, 20 women).

Intervention(s): We verified a sex difference in musculotendinous stiffness and compared spinal stretch reflex latency and amplitude in high-stiffness (men) and low-stiffness (women) groups. We also evaluated relationships between musculotendinous stiffness and spinal stretch reflex latency and amplitude, respectively.

Main Outcome Measure(s): Triceps surae musculotendinous stiffness and soleus spinal stretch reflex latency and amplitude were assessed at 30% of a maximal voluntary isometric plantar-flexion contraction.

Results: The high-stiffness group demonstrated significantly greater stiffness (137.41 ± 26.99 N/cm) than the low-stiffness group did (91.06 ± 20.10 N/cm). However, reflex latency (high stiffness = 50.11 ± 2.07 milliseconds, low stiffness = 48.26 ± 2.40 milliseconds) and amplitude (high stiffness = 0.28% ± 0.12% maximum motor response, low stiffness = 0.31% ± 0.16% maximum motor response) did not differ significantly across stiffness groups. Neither reflex latency ($r = .053$, $P = .746$) nor amplitude ($r = .073$, $P = .653$) was related significantly to musculotendinous stiffness.

Conclusions: A moderate level of pretension (eg, 30%) likely eliminates series elastic slack; thus, a greater change in force per unit-of-length change (ie, heightened stiffness) would have minimal effects on coupling of the muscle spindle and the stretch stimulus and, therefore, on spinal stretch reflex sensitivity. It appears unlikely that differences in musculotendinous stiffness influenced spinal stretch reflex sensitivity when initiated from a moderate level of pretension. Consequently, differences in musculotendinous stiffness did not appear to influence dynamic joint stability with respect to reflexive neuromuscular control.

Key Words: latency, amplitude, material modulus, compliance, neuromuscular control

Key Points

- Soleus spinal stretch reflex latency and amplitude did not differ among individuals with high or low triceps surae muscle stiffness.
- Differences in musculotendinous stiffness had a minimal influence on spinal stretch reflex sensitivity when initiated from a moderate level of pretension.
- Differences in musculotendinous stiffness did not appear to influence dynamic joint stability with respect to reflexive neuromuscular control.

Musculotendinous stiffness (MTS) has been proposed to contribute to joint stability through both mechanical and neural aspects.[1–6] The neural influence potentially is evidenced in the spinal stretch reflex (SSR). Higher levels of MTS have been associated with shorter SSR latency[7,8] and greater SSR amplitude.[9–11] A reflexive response to joint perturbation that is of greater magnitude and shorter latency may allow for a more dynamically stable joint.[12,13]

The SSR is elicited by a rapid increase in musculotendinous length, exciting Ia afferent neurons housed within the muscle spindle. These neurons project monosynaptically onto homonymous α-motor neurons, thus producing a vigorous contraction, which resists the imposed lengthening. The musculotendinous unit possesses inherent series elastic slack. Consequently, Rack et al[14] suggested that sufficient MTS must be present to compensate for this slack, allowing the muscle spindle to ''see'' an imposed mechanical load, and that greater compliance (inverse of stiffness) delays spindle excitation. Greater musculotendinous tension enhances mechanical transduction to the muscle spindle, effectively improving mechanical coupling of the spindle and the stretch stimulus.[10] With this concept in mind, we hypothesized that higher levels of MTS, defined as the ratio of change in force (or tension) to change in length (Δforce/Δlength), would be associated with heightened SSR sensitivity, because a stiffer muscle displays a greater increase in tension per unit of lengthening than a more compliant muscle.

Numerous investigators have noted relationships between MTS and SSR sensitivity. However, various methodologic issues hinder interpretations of these relationships. For example,

Avela and Komi[9] identified decreases in triceps surae MTS and SSR amplitude after long-term stretch-shorten cycle exercise, yet it is unclear if a causal relationship exists or if the diminished reflex amplitude was a manifestation of fatigue. Similarly, He[11] compared rectus femoris SSR amplitude between upright seated (shortened) and supine (lengthened) conditions, reporting that the SSR could be elicited only in the lengthened condition. Although it appears as though the increased muscle tension and MTS associated with the supine condition increased SSR sensitivity, differences in descending inhibition due to postural changes between the conditions cannot be ruled out.[15] Furthermore, the sample consisted of patients with spastic multiple sclerosis; thus, the potential neural implications of the disease cannot be discerned. Finally, Fellows and Thilmann[7] demonstrated that under passive conditions, as the ankle angle before perturbation moved progressively toward dorsiflexion, thereby increasing tension in the triceps surae, the latency of the SSR was reduced substantially. The heightened tension in the triceps surae created by progressive dorsiflexion likely increased passive MTS, but this property was not measured directly; therefore, the relationship between MTS and SSR sensitivity cannot be assessed directly.

The relationship between MTS and reflexive neuromuscular control is not fully understood. In particular, the effects of MTS on SSR sensitivity have yet to be identified. Stiffness is an inherent property of musculotendinous tissue that can be modified via various training and rehabilitation schemes.[16–20] As such, identifying its influence on factors that contribute to dynamic joint stability would allow for an evaluation of its viability as a factor to be targeted via injury prevention programs. The purpose of our study was to investigate the relationship between MTS and SSR sensitivity in the triceps surae by comparing the latency and amplitude of the soleus SSR across 2 groups with different MTS (men: high-stiffness group, women: low-stiffness group).

METHODS

Participants

Forty individuals (20 men, 20 women) who met the following criteria volunteered as participants: no history of (1) chronic or acute (ie, within 6 months of data collection) lower extremity musculoskeletal injury, (2) lower extremity surgery, or (3) neurologic disorder. Participants were also physically active at least 3 times per week for 20 minutes. Before participating, all volunteers read and signed informed consent approved by the Institutional Review Board, which also approved the study. Participant descriptive statistics are presented in Table 1.

Table 1. Participants' Descriptive Statistics

	Mean ± SD
Height, m	
Men	1.81 ± 0.06[a]
Women	1.67 ± 0.07
Mass, kg	
Men	81.83 ± 12.21[a]
Women	63.55 ± 9.84
Age, y	
Men	22.10 ± 2.90
Women	22.40 ± 2.96
Leg length, m	
Men	0.83 ± 0.04[a]
Women	0.77 ± 0.03

[a]Indicates significant difference between groups ($P < .001$).

Experimental Procedures

Altering MTS experimentally without potentially altering SSR sensitivity is difficult, because the effects of the treatment (eg, increasing background electromyographic [EMG] activity, heat application, or stretching) on mechanical and neurophysiologic properties cannot be distinguished. However, previous researchers[2,21–23] have demonstrated that MTS is greater in men than in women. For this reason, we chose to perform static group comparisons across groups known to differ in MTS rather than implementing an experimental design. We verified a sex difference in triceps surae MTS experimentally and subsequently assessed the latency and amplitude of the SSR to determine whether a group with high stiffness (men) demonstrated different SSR characteristics than a group with low stiffness (women).

We measured leg length from the greater trochanter to the lateral malleolus and used this value as a covariate to account for variance in SSR latencies related to the distance the neural impulse traveled to and from the spinal cord.[24] We assessed EMG activity of the soleus using Ag/AgCl surface electrodes (interelectrode distance = 2 cm). Because MTS has been demonstrated to not differ between limbs,[19] we used the right leg for all assessments. For each participant, we assessed (1) maximal voluntary isometric plantar-flexion contractions (MVIC), (2) triceps surae MTS, and (3) soleus SSR latency and amplitude. The MVICs were tested first because these reference values were necessary to determine loading conditions for the remaining assessments. We then counterbalanced the order of MTS and SSR assessments. A rest period of 5 minutes was provided between assessments to reduce the likelihood of fatigue.

The MVICs were performed using a custom loading device (Figure 1) with the participants seated; the hip, knee, and ankle joints positioned at 90° of flexion; and the metatarsal heads placed on a wooden block. We fixed the block to a force plate (model 4060; Bertec Corp, Columbus, OH), allowing plantar-flexion force to be captured in the vertical ground reaction force (GRFv). A block of equal height was placed under the calcaneus, maintaining the ankle at 90° of flexion. We secured the loading device on the distal anterior thigh using a winch, which restricted plantar-flexion motion, making the contraction isometric. Participants plantar flexed maximally against the device as GRFv data were sampled. One practice trial and 1 collection trial (approximately 3 seconds) were performed. Mean GRFv was calculated over each 25-millisecond interval, with the largest mean value serving as the MVIC ($GRFv_{max}$).

We standardized background plantar-flexion effort across assessments and participants. To determine the appropriate applied load, participants were positioned as in the MVIC testing, except that we removed the block under the calcaneus and the winch that restricted plantar-flexion motion. An arbitrary load was placed on the loading device initially, and participants maintained the ankle at 90° of flexion via isometric plantar flexion (Figure 2). We adjusted the magnitude of the applied load to produce 30% ± 5% $GRFv_{max}$. A previous report on the efficacy of this device to isolate plantar-flexion effort

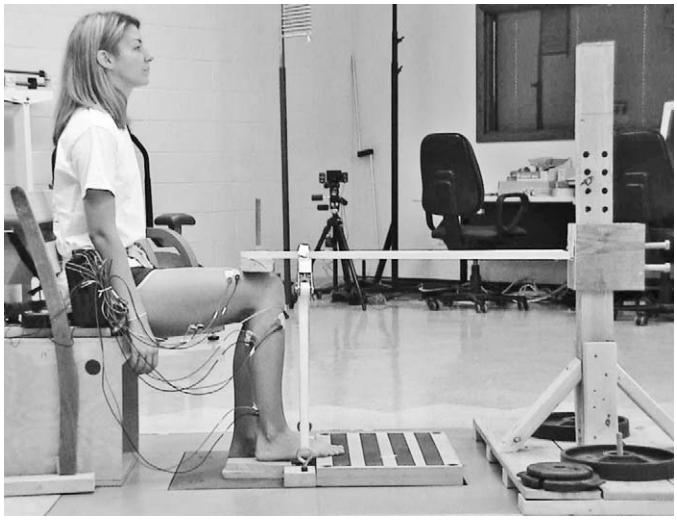

Figure 1. Triceps surae maximal voluntary isometric contraction assessment. The wooden plank was fixed rigidly to a force plate. The hip, knee, and ankle joints were oriented at 90° of flexion, and the metatarsal heads were placed on the top surface of the wooden plank such that plantar-flexion force was captured in the vertical ground reaction force. The position of the triceps surae loading device was held constant using a winch so that isometric triceps surae contraction could be produced. Reprinted from *Clinical Biomechanics (Bristol, Avon)* with permission from Elsevier.[22]

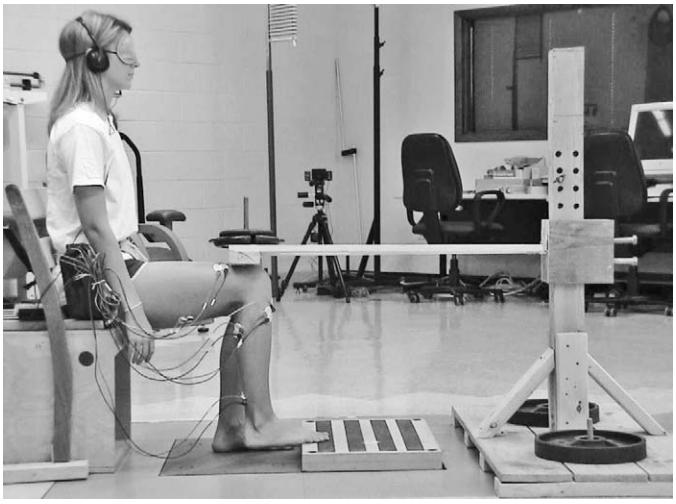

Figure 2. Participant positioning for stiffness assessments and determination of applied load. Rigid fixation of the wooden plank to the force plate allowed oscillatory motion of the system to be characterized in the vertical ground reaction force (GRFv) during stiffness assessments. The applied load was determined by adding mass until the GRFv equaled 30% ± 5% $GRFv_{max}$ obtained during triceps surae maximal voluntary isometric contraction assessments. The vertical arrow indicates the application point for the downward manual perturbation force used to initiate oscillatory motion during stiffness assessments.

indicated that EMG activity of the knee flexors and extensors and the tibialis anterior was negligible (approximately 1% MVIC).[25]

Musculotendinous Stiffness Assessments

We estimated triceps surae MTS from the damped frequency of oscillatory motion about the ankle. Participant positioning was identical to that used to determine the applied load (Figure 2). After plantar flexion against the applied load to the testing position, we applied a downward manual perturbation to the system at the applied load, initiating the damped oscillatory motion.[26,27] Participants were blindfolded and wearing headphones playing white noise, and we applied perturbations randomly within 10 seconds to reduce the likelihood of anticipation of the perturbation. Participants were instructed to attempt to maintain a constant level of plantar-flexion force and not to intervene once the perturbation began.

The damped oscillatory motion about the ankle was characterized in the GRFv (Figure 3). We calculated stiffness (k) from the damped frequency of oscillation (f) and the mass of the system (m) using the equation $k = 4\pi^2 mf^2$. System mass equaled the sum of the shank and foot segment mass[28] and the mass of the applied load. Participants performed 3 practice trials and 7 assessment trials, with a minimum of 30 seconds of rest between trials.

Anthropometrics influence MTS. To account for between-subjects discrepancies in height and mass, we derived triceps surae stress and strain, allowing for an estimate of material modulus (ie, MTS standardized to anthropometric factors). We secured an electrogoniometer (model XM65; Biometrics, Ltd, Cwmfelinfach, Gwent, UK) to the calcaneus and posterior shank to measure sagittal-plane ankle joint displacement. Change in triceps surae length was derived from ankle joint displacement via a second-order polynomial function[29,30] and was expressed relative to preperturbation length, estimating triceps surae strain. We then multiplied MTS by the change in triceps surae length, producing the change in plantar-flexion force (Δforce/Δlength $\times$ Δlength = Δforce). Because the plantar flexors provide 70% to 80% of the total plantar-flexion force,[31–33] this value was multiplied by 0.80 to derive the change in triceps surae force. Triceps surae force then was standardized to sex-specific estimates of physiologic cross-sectional area derived from previous literature,[33,34] estimating triceps surae stress. We then calculated material modulus as the ratio of stress to strain. For more detail on MTS and material modulus assessments, see Blackburn et al.[22]

Spinal Stretch Reflex Assessments

We assessed soleus SSR latency and amplitude via EMG analysis during rapid dorsiflexion perturbation. A spring-loaded platform provided dorsiflexion perturbations, stretching the triceps surae and initiating the SSR. Means and SDs for the perturbation mechanics are provided in Table 2. Participant positioning was as in the MTS assessments (Figure 4). Participants were again blindfolded and were wearing headphones playing white noise, and we released the platform at random within 10 seconds.

We defined SSR latency as the time interval between the onsets of perturbation and the SSR. Perturbation onset was determined via computer algorithm from perturbation-platform tangential acceleration provided by an accelerometer (model 356A22; PCB Piezotronics, Depew, NY). We identified SSR onset by visual inspection[35,36] of the soleus EMG waveform between 30 and 70 milliseconds postperturbation for the first point of inflection with a slope greater than that associated with background EMG activity (Figure 5). Additionally, we used a square-pulse stimulator (Grass Telefactor; Astro-Med, Inc, West Warwick, RI) to stimulate the tibial nerve maximally

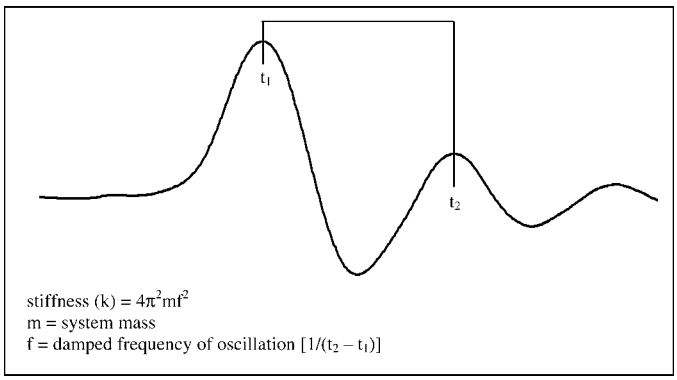

Figure 3. Musculotendinous stiffness assessment data. Damped oscillatory motion about the ankle was induced via a brief, downward manual perturbation and was captured in the vertical ground reaction force. With knowledge of the time stamps for consecutive oscillatory peaks (t_1 and t_2), the damped frequency of oscillation and musculotendinous stiffness could be calculated.

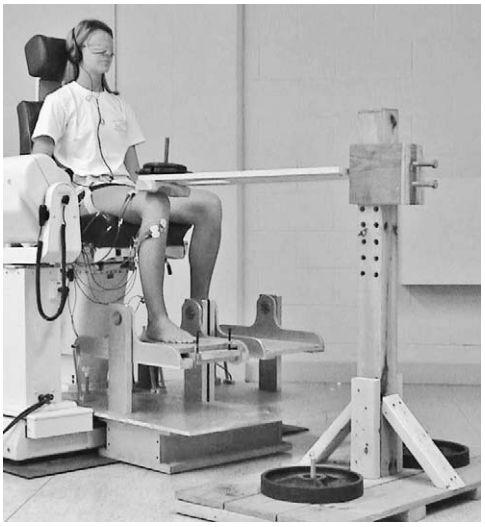

Figure 4. Spinal stretch reflex assessment. The applied load and lower extremity kinematics were identical between musculotendinous and spinal stretch reflex assessments. Reprinted from the *Journal of Electromyography and Kinesiology* with permission from Elsevier.[22]

via the popliteal space, eliciting the maximum motor response (M_{max}). The SSR amplitude was calculated as the peak-to-peak voltage after perturbation and was standardized to M_{max} (S/M ratio) to allow between-subjects comparisons.[37–39] Participants performed 3 practice trials and 7 assessment trials, with a minimum of 30 seconds of rest between trials.

Data Collection, Reduction, and Analysis

All data were sampled at 1000 Hz and were reduced using custom software (LabVIEW; National Instruments, Austin, TX). The EMG data were collected via telemetry (model T42L-8TO; Konigsberg Instruments Inc, Pasadena, CA; differential amplification, input impedance = 200 kΩ, common mode rejection ratio >70 dB, signal-to-noise ratio >40 dB), and amplified by a factor of 5000 (0.01 to 500 Hz). The EMGs were corrected for DC bias and were band-pass (10 to 350 Hz) and notch (59.5 to 60.5 Hz) filtered (fourth-order Butterworth). The GRFv was low-pass filtered at 10 Hz (fourth-order Butterworth). Electrogoniometer data were smoothed using a 25-millisecond root-mean-square sliding window function, and raw accelerometer data were analyzed.

We compared MTS, material modulus, SSR latency and amplitude, height, and mass across groups using independent-samples *t* tests. In an effort to account for differences in the distance that the neural impulse traveled to and from the spinal cord as a function of leg length,[24] we also assessed SSR latencies using 1-way analysis of covariance (covariate = leg length). For each dependent variable, we eliminated the trials with the highest and lowest values, leaving 5 trials for mean calculations. In the case of SSR trials, amplitude displayed greater variability than did latency and thus was used as the rejection criterion. We also used simple linear regression to evaluate the relationships between material modulus and SSR latency and amplitude, respectively. The relationship between material modulus and SSR latency was assessed first by regressing latency on material modulus, followed by regression of latency standardized to leg length on material modulus to account for its potential between-subjects bias. Finally, we used independent-samples *t* tests to evaluate group differences in background plantar-flexion effort (% $GRFv_{max}$) and SSR perturbation amplitude and velocity. Statistical significance was established a priori at $\alpha = .05$.

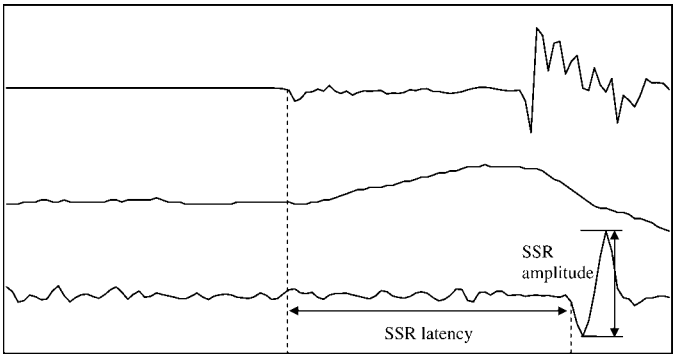

Figure 5. Spinal stretch reflex (SSR) assessment data. Waveforms represent a single trial (upper tracing = tangential platform acceleration, middle tracing = ankle joint displacement, lower tracing = soleus electromyographic activity). Dotted lines represent the onsets of dorsiflexion perturbation (tangential platform acceleration) and the soleus SSR.

We assessed intrasession reliability for MTS, SSR latency,

Table 2. Perturbation Amplitude and Velocity and Background Effort by High-Stiffness and Low-Stiffness Groups (Mean ± SD)

Dependent Variable	High-Stiffness Group	Low-Stiffness Group	*P* Value
Perturbation amplitude, °	3.50 ± 1.33	4.19 ± 1.36	.107
Perturbation velocity, °/s	84.18 ± 32.24	107.63 ± 32.90	.022[a]
Background effort, % maximal vertical ground reaction force	28.55 ± 2.59	30.40 ± 3.70	.074

[a]Indicates significant difference.

Table 3. Musculotendinous Stiffness, Material Modulus, Stretch Reflex Latency, and S/M Ratio by High-Stiffness and Low-Stiffness Groups (Mean ± SD)[a]

Dependent Variable	High-Stiffness Group	Low-Stiffness Group	*P* Value
Musculotendinous stiffness, N/cm	137.41 ± 26.99	91.06 ± 20.10	<.001[b]
Material modulus, Pa	2778.51 ± 549.95	1968.58 ± 439.61	<.001[b]
Stretch reflex latency, ms	50.11 ± 2.07	48.26 ± 2.40	.0013[b,c]
S/M ratio	0.28 ± 0.12	0.31 ± 0.16	.455

[a]S/M ratio indicates the soleus spinal stretch reflex amplitude standardized to the maximal motor response via electric stimulation.
[b]Indicates significant difference.
[c]Indicates nonsignificant difference based on 1-way analysis of covariance between groups (covariate = leg length).

and the S/M ratio (the soleus spinal stretch reflex amplitude standardized to the maximal motor response via electric stimulation) by calculating intraclass correlation coefficients (ICCs [2,1]) and the associated standard error of measurement (SEM). As visual identification of reflex onsets is subjective in nature, we performed similar procedures to determine reliability of the visually identified SSR onsets. We randomly selected 20 SSR trials, copied each trial twice, and encoded these trials such that the primary investigator was blind to the trial identities. Each trial was assessed to determine SSR latency. Because perturbation onset was determined via computer algorithm, it was identical for each assessment of a given trial. Therefore, variability in latency between assessments of a given trial was due only to variation in the visually identified reflex onset. Thus, reliability of latency across these reproduced trials provided an indication of the reliability of the primary investigator's ability to visually determine reflex onset. The identity of each trial was revealed after analysis, and the associated ICC (2,1) and SEM were calculated.

RESULTS

Men were taller ($P < .001$) and had longer legs ($P < .001$) and greater mass ($P < .001$) than women. The MTS ($P < .001$) and material modulus ($P < .001$) were also greater in men. Therefore, we categorized men as the high-stiffness group and women as the low-stiffness group. The SSR latency was significantly shorter in the low-stiffness group ($P = .013$), but 1-way analysis of covariance (covariate = leg length) indicated that this difference was not significant ($P = .872$, observed power = .80) when between-subjects differences in leg length were accounted for. Group differences in the S/M ratio were also nonsignificant ($P = .455$, observed power = .15). Means and SDs for each dependent variable are listed in Table 3. Regression analysis indicated that material modulus was significantly and positively related to SSR latency ($r = .420$, $P = .007$). However, when SSR latency was standardized to leg length, this relationship was not significant ($r = .053$, $P = .746$). The relationship between material modulus and SSR amplitude was also nonsignificant ($r = .073$, $P = .653$).

The SSR perturbation amplitude was not significantly different between stiffness groups ($P = .107$). Conversely, perturbation velocity was significantly greater in the low-stiffness group ($P = .022$), presumably due to less foot-segment inertia and triceps surae MTS, thus providing less resistance to the spring-driven perturbation. The applied load effectively produced 30% ± 5% $GRFv_{max}$, and no differences were noted between stiffness groups ($P = .074$). Means and SDs for each of these variables are presented in Table 2.

When considering the combination of ICC (reliability) and SEM (precision) values, the dependent variables demonstrated moderate to high intrasession reliability as indicated by the following combinations (ICC, SEM): MTS (0.89, 11.29 N/cm), S/M ratio (0.70, 9% M_{max}), SSR latency (0.51, 2.08 milliseconds). The primary investigator (J.T.B.) also demonstrated high intrasession reliability in visually identifying the onset of the SSR (ICC = 0.99, SEM = 0.18 milliseconds).

DISCUSSION

Our primary findings were that soleus SSR latency and amplitude were not different between high- (men) and low- (women) stiffness groups, and neither SSR latency nor amplitude was related significantly to material modulus. Our hypothesis that greater stiffness would correspond with shorter-latency, higher-amplitude SSR responses was not demonstrated to be tenable. We suggest that the moderate level of pretension from which the SSR assessments were made (ie, 30% MVIC) removed sufficient series elastic slack in each group such that differences in the mechanical coupling of the muscle spindle and the stretch stimulus were minimal.

Men displayed greater MTS than women, a finding that has been demonstrated previously in both the upper[40] and lower[2,21–23] extremities. Each of these authors suggested that anthropometric variations contributed to the observed MTS differences. In general, greater limb mass and length in men contribute to higher MTS. Blackburn et al found that when MTS was standardized to segment mass[41] or applied moment (the product of segment mass and length),[2] sex differences were negligible and nonsignificant. If MTS differences in the current investigation were associated purely with anthropometric discrepancies, our ability to assess the influences of MTS on SSR characteristics could have been compromised. However, men also demonstrated significantly greater material modulus. Stiffness is a ratio (Δforce/Δlength); thus, it is likely that standardization to a single value (eg, segment mass or applied moment) does not adequately account for the effects of anthropometric factors on the Δforce and Δlength, respectively. By estimating triceps surae stress and strain, we effectively calculated stiffness independent of anthropometric factors (ie, material modulus). This finding is in agreement with results from the previous literature[21] and is likely a function of differences in tendon stiffness and muscle architecture.[21,42] Greater material modulus in men suggests that we did, in fact, compare characteristics of the SSR between high and low MTS groups.

The literature suggests that timely recognition of the stretch stimulus by the muscle spindle is related to the stiffness of the musculotendinous unit.[7,10,11,14] However, SSR latency differed between MTS groups in our investigation by only 2 milliseconds, whereas the standardized amplitude differed by only 3% M_{max}. We are aware of only 1 additional investigation in

which SSR responses across groups that differed on MTS were directly compared. Pisano et al[43] noted greater wrist flexor stiffness in men compared with women, yet flexor carpi radialis SSR sensitivity (as measured by the perturbation velocity required to elicit a reflex response) did not differ between these groups. In related research, Huston and Wojtys[44] demonstrated statistically similar hamstring latencies between the sexes in response to anterior knee joint perturbations. Although these authors did not measure MTS, the previous literature indicates that men posses greater hamstring stiffness than women do.[2,23]

Numerous experimental variables, such as perturbation velocity and amplitude and agonist and antagonist background effort, influence SSR characteristics, but Blackburn et al[25] demonstrated in a previous report that the small-magnitude differences in these values between stiffness groups had negligible effects on our data. Similarly, Diener et al[45] showed that changes in perturbation velocity from 20° to 100°/s and changes in perturbation amplitude from 1° to 8° had no effects on SSR latency or amplitude. These small-magnitude differences are similar to those noted between groups in our investigation, suggesting that the between-groups differences in these experimental variables had little influence on our dependent variables. Additionally, our SSR latency values are similar to those reported in the literature.[7,46] Comparison of our SSR amplitude data with previous findings is limited, however, because the methods used by previous authors[37–39,47] included standardized reflex amplitudes to M_{max} which differed substantially from our procedures.

Our hypothesis that between-subjects differences in MTS account for differences is SSR sensitivity was related to the presence of inherent series elastic slack within skeletal muscle and the notion that a stiffer muscle displays a greater change in tension per unit of lengthening relative to a more compliant muscle. Several investigators[5,48] have demonstrated a quasi-linear increase in MTS with increasing pretension/background effort. Interestingly, Kasai and Komiyama[49] and Hutton et al[50] reported significant increases in SSR sensitivity with increasing pretension (and consequently MTS) from low levels to moderate and high levels (ie, 10% to 50% MVIC and 25% to 100% MVIC, respectively). However, Enoka et al[51] and Gollhofer et al[10] noted that SSR sensitivity was independent of pretension when increased from 50% to 100% MVIC and 30% to 60% MVIC, respectively. These findings suggest that enhancement of SSR sensitivity with increasing pretension and MTS is limited to the lower end of the activation continuum, with the critical value likely between 20% and 30% MVIC.[10,51,52]

Other investigators have demonstrated a similar trend for differences in electromechanical delay between loading conditions at the lower end of the activation continuum and a lack of differences at moderate and high levels.[53,54] *Electromechanical delay* is defined as the temporal lag between the onsets of neural activity and force production during skeletal muscle contraction, and a substantial portion of this delay is attributable to series elastic slack.[55] Series elastic slack must be minimized for optimal recognition of the stretch stimulus by the muscle spindle.[14] Vint et al[54] demonstrated that although stepwise changes in the level of pretension altered electromechanical delay at the lower end of the activation continuum (0% to 25% MVIC), this effect was negligible at higher levels of background effort (20% to 50% and 50% to 75% MVIC). In a similar investigation, Zhou et al[53] reported that there was a significantly longer electromechanical delay at 30% MVIC than at 60% and 80% MVIC but that the delay at 60% and 80% MVIC was not significantly different.

In combination, these findings suggest that a threshold exists beyond which increases in pretension and MTS do not have substantial effects on series elastic slack; thus, a greater change in force per unit-of-length change (ie, heightened MTS) would have minimal effects on SSR sensitivity beyond this threshold. Therefore, it is likely that plantar-flexion effort of 30% MVIC reduced series elastic slack to similar levels in both MTS groups in our investigation, such that no appreciable differences in mechanical coupling of the muscle spindle and the stretch stimulus were present. We suggest this as the most likely explanation for the lack of significant differences in SSR characteristics across stiffness groups.

It is important to note that the functional significance of the SSR relative to dynamic joint stability is a topic of debate, the details of which are beyond the scope of this investigation. In short, it has been suggested that regardless of its magnitude, the latency of this reflex response is likely too long to prevent it from making substantial contributions to resisting joint perturbations. However, others have proposed that this reflex response and others like it, such as the ligament-muscle protective reflex, play important roles in maintaining joint stability.[12,13] These latter authors postulated that although the inherent latencies of these peripheral responses may be too long to prevent tissue injury, they are superimposed on preparatory muscle activity and may prevent complete tissue disruption, preserving the limit between sprain and rupture of static restraints. These responses also help regulate MTS, potentially contributing to load compensation and mechanical joint stability.[6,56,57] Furthermore, musculoskeletal injury alters the time course of spinal-based reflexes by prolonging their latencies.[58] Although the direct implications are not readily apparent, these data suggest that the SSR plays a role in dynamic joint stability and neuromuscular control that has yet to be clearly defined.

A limitation to our methods is that the relative contributions of the individual components of the triceps surae (ie, soleus, medial gastrocnemius, and lateral gastrocnemius) to total plantar-flexion force during our experiments are not known. Knee flexion shortens the gastrocnemii due to their biarticular nature, whereas soleus length is unaltered. The literature suggests that with the knee flexed to 90°, as in our investigation, the contributions of the gastrocnemii to triceps surae force are negligible. Gastrocnemius force production is restricted in this shortened position due to both mechanical and architectural limitations[59] and neural inhibition[60,61] and is partially accounted for by an increase in soleus activity. Therefore, our stiffness data primarily describe the soleus, but minimal contributions of the gastrocnemii cannot be ruled out. A second limitation of our methods is that our estimate of MTS did not isolate the triceps surae exclusively but rather measured ankle joint stiffness. However, previous researchers,[62] using an animal model, suggested that under passive conditions, the tendon contributes 10% to the total passive stiffness, whereas the musculature contributes 41%. Because MTS increases quasi-linearly as a function of muscle activation,[5,48] the musculotendinous contribution to total joint stiffness undoubtedly dominates. Furthermore, although the triceps surae provides the largest contribution to total plantar-flexion force (70% to 80%),[31–33] numerous synergists (eg, tibialis posterior) also contribute to the net force. We attempted to account for these discrepancies,

yet the exact contributions of the triceps surae, as well as each of its components, to the stiffness measures presented here cannot be determined. Finally, the estimates of physiologic cross-sectional area used to derive triceps surae stress were derived from the previous literature and were not measured directly from our sample. These data are representative of comparable samples; however, potential differences in the various samples and subsequent errors in estimation of triceps surae stress cannot be ruled out. Additionally, these physiologic cross-sectional area values included all components of the triceps surae. Given that the relative contributions of each component to total plantar-flexion force are unknown, these values may have underestimated triceps surae stress.

CONCLUSIONS

Soleus SSR characteristics were compared between a group with high triceps surae MTS and a group with low triceps surae MTS. We hypothesized that greater MTS would correspond with shorter-latency, higher-amplitude reflex responses by enhancing mechanical coupling of the muscle spindle and the stretch stimulus. Although the groups differed significantly in MTS, no group differences were observed for SSR characteristics. Our data suggest that moderate levels of pretension effectively reduced inherent series elastic slack, such that group differences in the mechanical coupling of the muscle spindle and the stretch stimulus were minimal, thus limiting the potential influence of MTS on SSR sensitivity. As such, it appears unlikely that population differences in MTS influence SSR sensitivity with moderate levels of pretension. Consequently, population differences in MTS do not appear to influence dynamic joint stability with respect to reflexive neuromuscular control. Future research is necessary to evaluate the influences of MTS on neural and mechanical contributions to joint stability, as well as the functional implications of peripheral reflex responses.

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