DURATION-DEPENDENT EFFECTS OF PASSIVE STATIC STRETCHING ON MUSCULOTENDINOUS STIFFNESS AND MAXIMAL AND RAPID TORQUE AND SURFACE ELECTROMYOGRAPHY CHARACTERISTICS OF THE HAMSTRINGS

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ABSTRACT
Palmer, TB, Pineda, JG, Cruz, MR, and Agu-Udemba, CC. Duration-dependent effects of passive static stretching on musculotendinous stiffness and maximal and rapid torque and surface electromyography (EMG) characteristics of the hamstrings. J Strength Cond Res 33(3): 717–726, 2019—This study aimed to examine the effects of stretching duration on passive musculotendinous stiffness and maximal and rapid torque and surface electromyography (EMG) characteristics of the hamstrings. Thirteen young females (age = 21 ± 2 years) underwent 2 passive straight-leg raise (SLR) assessments and 2 isometric maximal voluntary contractions (MVCs) of the hamstrings before and after 4 randomized conditions that included a control treatment and 3 experimental treatments of passive static stretching for 30-, 60-, and 120-second durations. Passive stiffness was calculated during each SLR as the slope of the final 10% of the angle-torque curve. Isometric peak torque (PT), rate of torque development (RTD), peak EMG amplitude (PEMG), and rate of EMG rise (RER) were extracted from each MVC. Results indicated that PT and PEMG were not affected (p = 0.993 and 0.422, respectively) by any of the experimental treatments. Rate of torque development and RER decreased from pre- to post-treatment for 120 seconds (p = 0.001 and 0.001) but not for the control (p = 0.616 and 0.466), 30- (p = 0.628 and 0.612), and 60-second (p = 0.396 and 0.815) interventions. The slope coefficient decreased from pre- to post-treatment for the 30- (p = 0.001), 60- (p = 0.002), and 120-second (p = 0.001) stretching interventions but not for the control (p = 0.649). Given the significant stiffness reductions and lack of changes in PT and RTD for the 30- and 60-second interventions, it may be advantageous for practitioners who are using hamstring passive stretching as part of a warm-up routine, to perform such stretching on their clients for short (30–60 seconds) rather than moderate (120-second) stretching durations.

KEY WORDS straight-leg raise, range of motion, pre-exercise, neuromuscular performance, rate of torque development

INTRODUCTION
Passive stretching is often performed before exercise or competition to increase range of motion (ROM) and reduce passive stiffness of the muscle-tendon unit (MTU) (34). A decrease in passive stiffness after stretching is believed to reduce the risk of injury (35) and improve athletic performance (37). Although some research has suggested that a stretch-induced decrease in-stiffness may mitigate the risk of muscle strains (10), most studies agree that there is not enough evidence to support these claims (7,15,32). There is also little evidence to suggest that stretching improves performance (26). In fact, numerous studies in young adults have found reductions in muscle strength after an acute bout of passive stretching (4,12,16). Not all stretching studies have reported this performance deficit (7,38), thus warranting further examination.

Previous studies investigating the acute effects of stretching on maximal isometric strength have reported conflicting results. For example, some studies reported no changes in isometric peak torque (PT) or force characteristics of the hamstrings after acute stretching interventions (38,40), whereas other studies demonstrated significant stretch-induced decreases in maximal isometric strength for these muscles (16,17,24). Because the stretching protocols used in several of these studies were longer in duration (≥480 seconds) (16,17), it remains unclear whether isometric PT of the hamstrings is influenced by other, more practical bouts of passive stretch. Short (30–60 seconds) and moderate...
(120 seconds) bouts of passive stretching are often used in clinical and athletic settings (19); thus, it may be important to elucidate the stretching effects for these durations. Greater deficits in muscle strength have been suggested to occur after long and moderate compared with shorter bouts of passive stretching (36). Therefore, it is possible that passive stretching for 120 seconds may elicit greater decreases in muscle strength than 30- and 60-second stretching interventions; however, further research is needed to test this hypothesis.

Similar to maximal strength, conflicting findings have also been reported regarding the acute effects of stretching on rapid strength (rate of torque development [RTD]), with some authors reporting significant decreases in rapid strength after stretching (20,25,43) and others demonstrating no stretch-induced changes in this variable (2,44). Such discrepancies may be due in part to the different durations of passive stretching performed in each study (2). However, the effects of different stretching durations on isometric RTD have not yet been determined. Because the ability to generate torque rapidly is critical to the performance of many locomotor-related tasks (i.e., accelerating, running, and jumping) (5,28,42), it is possible that decreases in RTD after stretching may have a negative effect on these functions. Thus, to help identify stretching durations that minimize the stretch-induced decreases in performance, it may be of great value to examine the influence of stretching duration on RTD. McBride et al. (20) found that a group of young males who underwent an acute bout (99 seconds) of quadriceps passive stretching exhibited reduced isometric squat rapid but not maximal strength characteristics. Although these findings highlight the potential for greater stretch-induced decreases in RTD than PT of the lower-body musculature in young males (20), it remains to be determined whether a similar bout of passive stretching may also elicit significant decreases in rapid but not maximal strength characteristics. Although these findings highlight the potential for greater stretch-induced decreases in RTD than PT of the lower-body musculature in young males (20), it remains to be determined whether a similar bout of passive stretching may also elicit significant decreases in rapid but not maximal strength characteristics. Although these findings highlight the potential for greater stretch-induced decreases in RTD than PT of the lower-body musculature in young males (20), it remains to be determined whether a similar bout of passive stretching may also elicit significant decreases in rapid but not maximal strength characteristics.

Several mechanisms have been proposed to explain the stretch-induced deficits in muscle strength (20). One such mechanism is a decrease in muscle activation as indicated by surface electromyography (EMG). Previous studies have reported acute stretch-induced decreases in maximal muscle activation using peak EMG amplitude (PEMG) (4,12,14), whereas measurements of rapid muscle
activation, such as rate of EMG rise (RER), may also be
affected by acute stretching interventions. Acute deficits in
isometric RTD have been attributed to decreases in RER
(11); however, we are aware of no studies that have eval-
uated the changes in RER as a result of an acute bout of
passive stretching. In addition to decreases in muscle acti-
vation, alterations in passive stiffness may also be a contrib-
uting factor to the stretch-induced decreases in muscle
strength (33). It has been suggested that stiffness reductions
after stretching may decrease the rate at which forces are
transmitted to the skeletal structures, which could nega-
tively impact maximal and rapid strength capacities (9).
Herda et al. (15) reported a significant decrease in ham-
string passive stiffness after a single 30-second passive
stretch. Thus, a group of young, healthy females under-
went passive SLR and isometric hip extension strength
assessments before and after 4 randomized conditions that
went passive SLR and isometric hip extension strength
deficits in maximal strength have been reported for the
hamstrings after long-duration (480 seconds) passive
stretching protocols (16,17); however, we are aware of no
studies to date that have examined the passive mechanical
and neuromuscular responses of the hamstrings to short
(30–60 seconds) and moderate (120 seconds) bouts of pas-
see stretching. Therefore, the purpose of this study was to
examine the influence of stretching duration on passive stiffness and maximal and rapid torque production and EMG characteristics of the hamstrings in a group of young, healthy females.

**METHODS**

**Experimental Approach to the Problem**

This study used a randomized, cross-over design with
repeated measures to investigate the acute effects of
straight-leg raise (SLR) stretching on passive stiffness and
maximal and rapid torque and muscle activation character-
istics of the hamstrings in healthy, young females. Acute
deficits in maximal strength have been reported for the
hamstrings after long-duration (≥480 seconds) passive
stretching protocols (16,17); however, we are aware of no
studies to date that have examined the passive mechanical
and neuromuscular responses of the hamstrings to short
(30–60 seconds) and moderate (120 seconds) bouts of pas-
see stretch. Thus, a group of young, healthy females under-
went passive SLR and isometric hip extension strength
assessments before and after 4 randomized conditions that
included a control treatment and 3 experimental treatments
of passive static stretching for 30-, 60-, and 120-second
durations.

**Subjects**

A convenience sample of 13 young healthy females ranging
in age from 18 to 25 years (mean ± SD; age = 21 ± 2 years;
height = 161 ± 6 cm; body mass = 60 ± 8 kg; and body mass
index = 23 ± 3 kg·m⁻²) with normal flexibility (SLR ≥ 65°)
(6) volunteered to participate in this study. Each participant
completed a self-administered questionnaire before testing to
assess their health history and volume of physical activity.
None of the participants reported any current or ongoing
neuromuscular diseases or musculoskeletal injuries specific
to the ankle, knee, or hip joints. Participants were considered

### Table 1. Mean (SD) pre- and post-treatment peak torque (PT), peak EMG amplitude (PEMG), rate of torque
development (RTD), rate of EMG rise (RER), slope coefficient, and range of motion (ROM) values for the control, 30-, 60-, and 120-second passive stretching interventions.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time</th>
<th>Control</th>
<th>30 s</th>
<th>60 s</th>
<th>120 s</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT (Nm)</td>
<td>Pre</td>
<td>141.82 (38.16)</td>
<td>145.11 (35.80)</td>
<td>137.57 (34.73)</td>
<td>141.00 (35.18)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>144.60 (31.17)</td>
<td>138.89 (34.86)</td>
<td>139.50 (34.77)</td>
<td>142.41 (29.17)</td>
</tr>
<tr>
<td>PEMG (%MVC)</td>
<td>Pre</td>
<td>100.00</td>
<td>100.00</td>
<td>100.00</td>
<td>100.00</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>96.59 (19.44)</td>
<td>97.26 (16.51)</td>
<td>92.58 (18.35)</td>
<td>103.44 (17.04)</td>
</tr>
<tr>
<td>RTD (Nm·s⁻¹)</td>
<td>Pre</td>
<td>827.77 (270.24)</td>
<td>864.56 (342.80)</td>
<td>808.12 (344.32)</td>
<td>864.31 (337.55)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>852.92 (258.65)</td>
<td>847.62 (337.84)</td>
<td>838.81 (347.42)</td>
<td>730.62 (292.03)</td>
</tr>
<tr>
<td>RER (%PEMG·s⁻¹)</td>
<td>Pre</td>
<td>733.92 (281.14)</td>
<td>737.36 (238.79)</td>
<td>791.03 (263.86)</td>
<td>813.94 (241.94)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>768.22 (295.71)</td>
<td>749.93 (259.51)</td>
<td>800.79 (270.62)</td>
<td>663.26 (233.42)</td>
</tr>
<tr>
<td>Slope (deg⁻¹)</td>
<td>Pre</td>
<td>0.80 (0.25)</td>
<td>0.81 (0.35)</td>
<td>0.82 (0.34)</td>
<td>0.79 (0.32)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>0.81 (0.25)</td>
<td>0.71 (0.31)</td>
<td>0.70 (0.27)</td>
<td>0.66 (0.27)</td>
</tr>
<tr>
<td>ROM (deg)</td>
<td>Pre</td>
<td>100.38 (21.85)</td>
<td>98.77 (16.27)</td>
<td>97.42 (20.87)</td>
<td>99.31 (20.37)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>100.23 (22.58)</td>
<td>107.73 (15.35)</td>
<td>105.08 (18.15)</td>
<td>109.15 (19.77)</td>
</tr>
</tbody>
</table>

*EMG = electromyography; MVC = maximal voluntary contraction.
†Indicates a significant change from pre- to post-treatment (p < 0.050).
as being recreationally active based on their self-reported levels of aerobic (3.9 ± 2.3 h·wk⁻¹) and resistance (5.4 ± 4.2 h·wk⁻¹) exercise behaviors (29). This study was approved by the Texas Tech University institutional review board for human subject’s research, and each participant was informed of the benefits and risks of the investigation before signing an informed consent document.

**Procedures**

Each participant visited the laboratory 5 times, separated by 2–7 days at approximately the same time of the day (±2 hours). The first visit was a familiarization trial, and the remaining 4 visits were experimental trials in a randomized order: (a) control treatment, (b) passive stretching for 30 seconds, (c) passive stretching for 60 seconds, and (d) passive stretching for 120 seconds. During the familiarization trial, participants practiced the passive SLR and isometric strength assessments. In addition, the maximum tolerable torque threshold was determined for each individual as the point of discomfort but not pain as verbally acknowledged by the participant during a series of passive stretches of the hamstrings (16). This predetermined torque threshold was used for the passive stretching during the experimental trials. For each experimental trial, participants underwent the pre-treatment assessments (2 passive SLR assessments and 2 isometric strength tests), the treatment intervention, and the post-treatment assessments that occurred immediately after the intervention. All treatments and assessments were performed on the right leg (26,30). The stretching treatments consisted of varied repetitions of 30-second passive stretches (i.e., the 30-, 60-, and 120-second treatments involved one, two, and four 30-second passive stretches, respectively). The control treatment consisted of quiet resting for 5 minutes, which was approximately the same total duration as the 120-second passive stretching treatment (including rest between stretches).

**Passive Stiffness and Range of Motion.** Passive stiffness and ROM of the hamstrings were quantified during each pre- and post-treatment SLR assessment using a calibrated Biodex System 3 isokinetic dynamometer (Biodex Medical Systems Inc., Shirley, NY, USA) programmed in passive mode to move the leg toward the head at 5°·s⁻¹ (26). For each SLR, participants laid in a supine position, with the knee braced in full extension and the ankle immobilized in a neutral 90° position (between the foot and leg) with a custom-made cast that was fixed around the foot and held with straps above the ankle and over the toes and metatarsals. During the SLR, the input axis of the dynamometer was aligned slightly superior and anterior to the greater trochanter of the femur to account for movement of the greater trochanter, and restraining straps were placed over the participants’ left unstretched thigh and ankle. All SLR assessments were performed on the right leg to the point of discomfort but not pain as indicated by the participant, which was regarded as the maximum ROM (26). Once maximum ROM was reached, the leg was then immediately returned to the baseline position, which was a hip joint angle of 20° above the horizontal plane.

**Surface Electromyography.** Surface EMG was recorded for the biceps femoris from bipolar preamplified electrodes (TSD150B; Biopac Systems, Santa Barbara, CA, USA) with a fixed center-to-center interelectrode distance of 20 mm and a gain of 350 (nominal). The electrodes were taped directly to the skin and were placed at 50% of the distance between the ischial tuberosity and the lateral epicondyle of the tibia. The electrode placements were based on the recommendations of Hermens et al. (18). To decrease the interelectrode impedance, the skin was cleansed with isopropyl alcohol before electrode placement. A single pregelled, disposable electrode (EL502; Biopac Systems) was placed on the palmar side of the right wrist to serve as a reference electrode (26).

**Maximal and Rapid Torque and Muscle Activation Characteristics.** To determine the treatment-induced changes in maximal and rapid torque and muscle activation characteristics, each participant performed 2 isometric maximal voluntary contractions (MVCs) of the hamstrings before and after each treatment intervention. All MVCs were performed using the isokinetic dynamometer on the right leg at a hip joint angle of 20° above the horizontal plane, which was the same hip joint angle as the starting point of the passive SLR assessments (30). Participants laid supine during each MVC with restraining straps placed over the waist, left thigh, and ankle. One-minute rests were allotted between each MVC assessment. For all MVCs, participants were asked to extend the thigh “as hard and fast as possible” for a total of 3–4 seconds, and strong verbal encouragement was given throughout the duration of the contraction (30).

**Signal Processing.** During each SLR and MVC assessment, the torque (Nm), position (degrees), and EMG (µV) signals were sampled simultaneously at 1 kHz (MP150WSW; Biopac Systems), stored on a personal computer (Dell Inspiron 8200; Dell, Inc., Round Rock, TX, USA), and processed offline using custom-written software (LabVIEW, Version 11.0; National Instruments, Austin, TX, USA). Torque and position signals were low-pass filtered, with a 10-Hz cutoff (zero-phase lag, fourth-order Butterworth filter). The EMG signal was scaled and bandpass filtered (zero-phase lag, fourth-order Butterworth filter) from 20 to 400 Hz. In addition, for each MVC, the passive baseline torque value was subtracted from the signal so that the new baseline value was 0 Nm, and the EMG signal was rectified and low-pass filtered using a 10-Hz linear EMG envelope (39). All subsequent analyses were conducted on the scaled and filtered signals.
Maximal and rapid torque and muscle activation characteristics were calculated during each MVC according to the procedures of Andersen et al. (3). For maximal capacity, isometric MVC PT (Nm) and PEMG amplitude (µV) were determined as the highest value from the filtered torque- and EMG-time curves, respectively (Figure 1). In addition, PEMG amplitude values were normalized to the pretreatment MVC values for all post-treatment conditions (33). For rapid capacity, isometric RTD (Nm·s⁻¹) was determined as the steepest slope over 100 ms of the initial rising portion of the filtered torque-time curve. Rate of EMG rise was determined as the steepest slope over 100 ms of the initial rising portion of the filtered EMG-time curve normalized to PEMG amplitude (%PEMG·s⁻¹) (Figure 1).

To ensure all SLR assessments were passive, EMG amplitude was also calculated with a root-mean-square function for 200-ms epochs corresponding to each whole-number degree during the ROM. According to the procedures of Herda et al. (16), EMG amplitude baseline noise values were subtracted from the EMG amplitude values recorded during the passive SLR assessments. Furthermore, the corrected EMG amplitude values were normalized to the corresponding prestretch isometric MVCs and expressed as a percentage of the MVC EMG amplitude.

For passive stiffness, gravity correction was performed during each SLR using a cosine function in which the limb mass was subtracted from the torque signal across the ROM. The scaled and gravity-corrected torque and joint angle signals were plotted as passive angle-torque curves and fitted with a fourth-order polynomial regression model based on the equation reported by Nordez et al. (23). Passive stiffness was calculated as the slope of the last 10% ROM of the angle-torque curve using a previously described procedure (Figure 2) (26). The slope of the last 10% ROM was defined as the change in passive torque from 90% to the end ROM. Mean passive torque at the beginning and end of the last 10% ROM was quantified at common joint angles for all SLR assessments performed on each participant. Consequently, the same absolute joint angles could be used to calculate the slope coefficient (Nm·deg⁻¹) for each SLR assessment. The slope coefficient of the last 10% ROM was calculated using the following equation:

\[
\text{Slope Coefficient} = \frac{\text{Passive Torque}_{\text{End ROM}} - \text{Passive Torque}_{90\% \text{ ROM}}}{\text{Absolute Joint Angle}_{\text{End ROM}} - \text{Absolute Joint Angle}_{90\% \text{ ROM}}}
\]

(33). For rapid capacity, isometric RTD (Nm·s⁻¹) was determined as the steepest slope over 100 ms of the initial rising portion of the filtered torque-time curve. Rate of EMG rise was determined as the steepest slope over 100 ms of the initial rising portion of the filtered EMG-time curve normalized to PEMG amplitude (%PEMG·s⁻¹) (Figure 1).

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Reliability. Based on the procedures described by Weir (41), test-retest reliability from our laboratory was examined during passive SLR and isometric MVC assessments from 10 participants measured 48–72 hours apart. The intraclass correlation coefficient and SE expressed as a percentage of the mean were 0.94 and 15.65% for the slope coefficient, 0.92 and 5.62% for PT, 0.84 and 18.53% for PEMG, 0.74 and 11.42% for RTD, and 0.81 and 11.68% for RER, respectively. In addition, there were no systematic differences (p > 0.050) between testing sessions for any of the variables.

Passive Stretching. The repeated passive stretching of the right hamstring muscles was performed on the isokinetic dynamometer using a passive SLR. The dynamometer passively moved the leg toward the head at 5°·s⁻¹ until the maximal
A tolerable torque threshold was met. The dynamometer maintained this constant passive torque for 30 seconds. Stretches were performed for 30-second bouts with a 20-second rest period between bouts, in which the leg was returned to the baseline position (26). Each stretch was repeated until the specific time under stretch was completed for each treatment (33).

Statistical Analyses

Seven separate 2-way repeated-measures analyses of variance (ANOvas) (treatment [control vs. 30 seconds vs. 60 seconds vs. 120 seconds] × time [pre- vs. post-treatment]) were used to analyze the PT, PEMG, RTD, RER, slope coefficient, maximum ROM, and SLR EMG amplitude data. A separate 1-way repeated-measures ANOVA was used to examine the differences in stretch-induced changes for the slope coefficient between treatments. When appropriate, follow-up analyses included dependent samples t-tests.

F-ratios (F) and type I error rates (p) were reported for each ANOVA. Partial eta-squared (η²p) values were also reported to estimate effect sizes. Pearson product-moment correlation coefficients (r) were calculated for the 120-second stretching intervention to examine the relationships between RTD and the slope coefficient at baseline and the percent changes (from pre- to post-treatment) in RTD and the slope coefficient and RER. Statistical analyses were performed using IBM SPSS Statistics v. 25.0 (SPSS Inc., Chicago, IL, USA), and an alpha level of p ≤ 0.050 was used to determine statistical significance.

RESULTS

Table 1 shows the pre- and post-treatment PT, PEMG, RTD, RER, slope coefficient, and ROM values (mean ± SD) for the control, 30-, 60-, and 120-second passive stretching interventions. For PT and PEMG, there were no interactions (F3,36 = 0.918 and 0.974, respectively; p = 0.442 and 0.416; η²p = 0.071 and 0.075) and no main effects for treatment (F3,36 = 0.338 and 0.974; p = 0.798 and 0.416; η²p = 0.027 and 0.075) or time (F1,12 ≤ 0.001 and 0.692; p = 0.993 and 0.422; η²p ≤ 0.001 and 0.054). For RTD and RER, there were significant interactions (F3,36 = 4.816 and 6.404; p = 0.006 and 0.001; η²p = 0.286 and 0.348). Rate of torque development and RER decreased from pre- to post-treatment for 120 seconds (p = 0.001 and 0.001); however, there were no changes for the control (p = 0.616 and 0.466), 30- (p = 0.628 and 0.612), and 60-second (p = 0.396 and 0.815) stretching interventions (Figure 3). For the slope coefficient and ROM, there were significant interactions (F3,36 = 7.708 and 9.429; p < 0.001 and 0.001; η²p = 0.391 and 0.440). The slope coefficient decreased (Figure 3), and ROM increased from pre- to post-treatment for 120 seconds (p = 0.001 and 0.001); however, there were no changes for the control (p = 0.649 and 0.913). In addition, the decreases in the slope coefficient for all stretching interventions (30, 60, and 120 seconds) were greater than the control (p = 0.020, 0.010, and 0.018), but they were not different from each other (p > 0.999). For SLR
EMG amplitude, there was no interaction ($F_{3,36} = 0.708; \rho = 0.554; \eta_p^2 = 0.056$) and no main effects for treatment ($F_{3,36} = 0.052; \rho = 0.984; \eta_p^2 = 0.004$) or time ($F_{1,12} = 0.166; \rho = 0.691; \eta_p^2 = 0.014$). The SLR EMG amplitude values were 2.22, 2.13, 2.12, and 2.17% of MVC for the control, 30-, 60-, and 120-second passive stretching interventions, respectively. For the 120-second stretching intervention, a significant positive relationship was observed between the slope coefficient and RTD at baseline ($r = 0.619; \rho = 0.024$) and the changes in RTD and RER ($r = 0.701; \rho = 0.008$; Figure 4); however, no relationship was observed between the changes in RTD and the slope coefficient ($r = 0.067; \rho = 0.829$; Figure 4).

**DISCUSSION**

The primary findings of this study revealed no changes in maximal torque production or muscle activation (i.e., PT or PEMG) from pre- to post-treatment for any of the stretching interventions (Table 1). Significant decreases in rapid torque production and muscle activation (i.e., RTD and RER) were observed from pre- to post-stretching for 120 seconds but not for the control, 30-, or 60-second durations (Figure 3). There were significant reductions in the slope coefficient (Figure 3) and increases in ROM from pre- to post-treatment for all stretching interventions but not for the control. For the 120-second stretching intervention, there was a significant positive relationship between changes in RTD and RER; however, there was no relationship between changes in RTD and the slope coefficient (Figure 4).

Our findings indicated that the acute effects of 30-, 60-, and 120-second passive stretching interventions did not significantly alter hamstring maximal torque production. These findings are consistent with those of previous studies, which have showed no changes in isometric peak force or torque characteristics of the hamstrings after acute stretching interventions (38,40). Alternatively, however, other authors examining the hamstrings have reported significant stretch-induced decreases in maximal isometric strength for these muscles (16,17). Nevertheless, it should be noted that these authors used longer-term passive stretching protocols, which may have influenced the results and contributed to the greater stretch-induced declines in maximal strength that were found in their studies compared with the present study. For example, the present study investigated the effects of short (30–60 seconds) and moderate (120 seconds) bouts of passive stretching on hamstring PT values, whereas the previous studies examined these strength effects after stretching protocols that were longer in duration (≥480 seconds) (16,17). Previous authors have proposed that a dose-response relationship may exist between the duration of stretching and the maximal strength deficit (33,40). Ryan et al. (33) reported no changes in maximal strength of the plantarflexors after 120, 240, and 480 seconds of passive stretching. However, when the authors compared these results with other studies using prolonged stretching protocols (≥600 seconds), they found evidence to support a threshold stretching duration between 480 and 600 seconds that may distinguish between significant and nonsignificant decreases in plantarflexion strength (33). It is possible that the hamstring muscles may also exhibit a similar dose-response relationship (40), with long-term passive stretching (≥480 seconds) eliciting greater maximal strength declines than short- and moderate-term stretching interventions (≤120 seconds); however, future studies are needed to test this hypothesis.

Previous authors examining the effects of acute stretching interventions on rapid strength characteristics of the lower-body musculature have reported conflicting results, with some authors reporting no changes in rapid strength from pre- to post-stretching (2,44) and others demonstrating a significant stretch-induced decrease in this variable (20,25,43). Such discrepancies may be due to differences in muscle groups and RTD time intervals examined, testing procedures, and the duration of stretching (2,20,25). Collectively, the majority of stretching studies that have reported no changes in RTD characteristics examined rapid strength after shorter bouts of stretching (45 seconds) (2,44), whereas other studies that have reported significant stretch-induced decreases examined rapid strength after more moderate stretching periods (≥99 seconds) (20,25,43). The results of our study support these findings by demonstrating a significant stretch-induced decrease in hamstring RTD from pre- to post-treatment for 120 seconds, but not for the control, 30-, or 60-second interventions. These findings suggest that if there is a threshold necessary for causing a decrease in rapid strength, it may be approximately 120 seconds of passive stretching. It is interesting to note that although the 120-second stretching intervention reduced RTD in this study, it had no effect on PT. Similarly, McBride et al. (20) found a significant decrease in isometric squat rapid strength but not maximal strength characteristics in a group of young males after a moderate bout (99 seconds) of quadriceps passive stretching. Taken together, these findings suggest that passive stretching protocols lasting 99–120 seconds in duration may cause greater reductions in rapid compared with maximal strength capacities of the lower-body musculature in young male and female populations. Because the ability to generate torque rapidly plays an important role in many locomotor-related movement tasks, including accelerating, running, and jumping (5,28,42), it is possible that the decreased RTD observed after 120 seconds of stretching may have an adverse effect on these functions. Although further research is still needed to test these hypotheses regarding the influence of stretching on RTD and locomotor-related performances, the fact that our findings showed a lack of changes in PT and RTD for the 30- and 60-second stretching interventions provides support that shorter bouts of stretching (30–60 seconds) may not be detrimental to maximal and rapid strength.

Decreases in hamstring stiffness after acute bouts of passive stretching have been well documented in the literature (15,16,26,27). In this study, we also revealed that
hamstring passive stiffness, as indicated by the slope coefficient, was significantly reduced after passive stretching interventions of 30-, 60-, and 120-second durations. However, an interesting finding of this study was that the 30-second stretching duration elicited similar stiffness reductions (12%) as the 60- and 120-second stretching periods (15–16%). This finding indicates that stretching durations beyond 30 seconds may not elicit further decreases in passive stiffness of the hamstrings. By contrast, Herda et al. (15) showed a significant decrease in hamstring stiffness after 30 seconds of passive stretching with subsequent decreases after longer stretching durations. However, it is important to note that these additional decreases in stiffness were not evident until after 240 seconds of stretching (15). Thus, taking these findings together, it is possible that 30 seconds may be just as effective as 60 or 120 seconds of stretching at reducing stiffness in the hamstrings; however, if stretching is performed for longer durations (≥240 seconds), additional decreases in passive tissue resistance may occur, which could result in greater stiffness reductions (15). Although the previous study’s findings provide support for long-term passive stretching protocols greater than 240 seconds (15), stretching for such a duration has been shown to adversely influence numerous performance-based outcomes, including muscle power and rapid strength characteristics (43). In this study, we observed a significant stretch-induced decrease in rapid strength for 120 seconds but not for the 30- or 60-second durations. We also found significant reductions in the slope coefficient and increases in ROM from pre- to post-treatment for all stretching interventions (30, 60, and 120 seconds) but not for the control. Therefore, if the goal of stretching is to reduce stiffness and increase ROM without negatively influencing muscle strength, our findings suggest that stretching for short (30–60 seconds) rather than moderate (120 seconds) durations may be a more reasonable strategy.

Acute deficits in maximal and rapid strength after stretching have been attributed to several different mechanistic mechanisms (20). One such mechanism is a decrease in muscle activation. Maximal muscle activation as indicated by PEMG was not significantly altered in this study from pre- to post-treatment for the 30-, 60-, and 120-second stretching interventions. This finding of no changes in PEMG amplitude was consistent with the lack of changes observed for PT. Significant stretch-induced decreases in maximal EMG amplitude have been reported after long (>480 seconds) (4,12,14) but not short or moderate bouts (≤120 seconds) (7,33) of passive stretching. Therefore, in addition to explaining PT, the stretching durations (30, 60, and 120 seconds) used in this study may also explain why no changes in PEMG were observed for the hamstrings.

Previous studies investigating the acute effects of stretching on muscle activation characteristics have almost exclusively made such assessments using maximal muscle activation as the outcome measure (13,14,33). A novel aspect of our study was the assessment of RER, which to the best of our knowledge, was the first to examine the influence of stretching on this variable. Similar to RTD, we found a significant decrease in RER after stretching for 120 seconds but not for the 30- or 60-second interventions. Given these findings, it is possible that the decrease in RTD we observed after the 120-second stretching intervention was at least partially influenced by the changes in rapid muscle activation. It has been suggested that rapid muscle activation influences RTD (1) and that a decrease in RER may reduce the ability to generate torque rapidly (11). Our findings of a significant positive relationship (r = 0.701; Figure 4) between changes in RTD and RER for the 120-second stretching intervention support this hypothesis. Because RER is believed to be influenced by factors that include early motor unit recruitment, discharge rates, and rates of doublet discharge (21), the stretch-induced decrease in RER we observed may be a result of impairments in these physiological mechanisms. Such impairments have been suggested to be detrimental to muscle strength, and in particular rapid torque production (22), which could further explain why our study showed a significant decrease in RTD after the 120-second stretching intervention. Although future research is still needed to further elucidate the causes (and validity) of any potential stretch-induced changes in RTD, the present findings of a significant positive relationship between changes in RTD and RER for the 120-second stretching intervention provide support that a stretch-induced decrease in rapid strength as assessed during a supine hip extension test may be linked to reductions in rapid muscle activation of the hamstrings.

Another mechanism that may contribute to the stretch-induced strength deficit is a change in passive stiffness (33). Previous research has suggested that passive stiffness, as indicated by the slope coefficient, may influence the rate at which forces are transmitted to the skeletal structures (30), and because decreases in the slope coefficient have been reported to occur after stretching (26), it is possible that these changes may decrease force transmission characteristics, causing a reduction in the strength capacities of the muscles being stretched (9). In this study, we found significant decreases in the slope coefficient but no changes in maximal and rapid strength of the hamstrings for the 30- and 60-second interventions. These findings provide support that shorter bouts of stretching may elicit decreases in stiffness that are not detrimental to the force-producing capabilities of the muscle. For the 120-second stretching intervention, we observed significant decreases in the slope coefficient and RTD; however, interestingly, our findings did not support the relationship between the stretch-induced changes in these variables (r = 0.067; Figure 4). Collectively, these findings indicate that 120 seconds of stretching may elicit reductions in passive stiffness that are accompanied by, but not related to, decreases in rapid strength. Although the
reason for this is unclear, we do acknowledge the possibility that longer-term stretching (≥240 seconds), because of its greater effects on the slope coefficient, may yield a greater relationship between the stretch-induced changes in stiffness and RTD. Future studies examining the effects of longer-duration passive stretching protocols on the relationship between hamstring stiffness and RTD are needed to test this hypothesis. Finally, previous research has suggested that a decrease in lower-body stiffness may reduce the risk of muscle strains and other injuries to the MTU (10). Because we found significant stretch-induced reductions in the slope coefficient, it is possible that the stretching protocols used in this study may be beneficial for mitigating subsequent injury events in younger adults. Future research investigating the influence of preactivity stretching on passive stiffness and the incidence of hamstring-related injuries is needed to further examine these findings.

PRACTICAL APPLICATIONS

This investigation showed that passive stretching for 120 seconds elicited significant decreases in RTD and RER but not PT and PEMG of the hamstrings. These findings indicate that rapid torque and muscle activation capacity may be more severely impaired than maximal capacity after 120 seconds of hamstring passive stretching. For the 120-second stretching intervention, a significant positive relationship was observed between the changes in RTD and RER, suggesting that decreases in RTD after stretching may be linked to reductions in rapid muscle activation. A key finding of this study was that the 30- and 60-second stretching interventions did not significantly alter any of the maximal and rapid torque or EMG activity characteristics. Moreover, significant reductions in the slope coefficient and increases in ROM were observed from pre- to post-stretching for the 30-, 60-, and 120-second interventions. Taken together, these findings provide support that a short bout (30 or 60 seconds) of passive stretching may be an effective intervention for reducing passive stiffness and increasing ROM without negatively influencing maximal and rapid strength of the hamstrings. Because the ability to generate torque rapidly is important for the performance of many locomotor-related movement tasks including accelerating, running, and jumping, it is possible that the decreased RTD we observed after 120 seconds of stretching may have a deleterious effect on these functions. Thus, in light of these findings and given the lack of changes in RTD for the 30- and 60-second interventions, it may be advantageous for practitioners who are using hamstring passive stretching as part of a warm-up routine (before exercise or competition), to perform such stretching on their clients for short (30–60 seconds) rather than moderate (120 seconds) stretching durations.

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REFERENCES

Stretching, Stiffness, and Strength


