The Role of Neural Tension in Stretch-Induced Strength Loss

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Abstract

McHugh, MP, Tallent, J, and Johnson, CD. The role of neural tension in stretch-induced strength loss. *J Strength Cond Res* 27(5): 1327–1332, 2013–The purpose of this study was to determine if neural tension during passive stretching affected subsequent strength loss. Eleven healthy subjects (10 men, 1 woman; age 34 ± 12 years) performed maximal isometric hamstring contractions at 100°, 80°, 60°, and 40° knee flexion before and after five 1-minute hamstring stretches performed in either a spinal neutral position or a neural tension position. One leg was stretched in the neutral position and the other in the neural tension position. Hamstring electromyography (EMG) activity was recorded during all contractions and stretches. Passive resistance to stretch was reduced by 11% after stretching ($p < 0.01$; no difference between neutral or neural tension stretches $p = 0.41$). Stretch-induced strength loss was apparent after neural tension stretches (12%, $p < 0.01$) but not after neutral stretches (5%, $p = 0.09$). There was a rightward shift in the angle-torque curve after neutral stretches (strength loss on ascending limb, strength gain on descending limb, $p < 0.01$). This effect was not apparent after neural tension stretches ($p = 0.43$). Stretching did not affect EMG activity during isometric contractions (<2% decline $p = 0.58$; no difference between neutral and neural tension, $p = 0.86$). Hamstring stretching with the spine in a neutral position did not result in a significant strength loss but shifted the length-tension relationship such that strength was decreased at short muscle lengths and increased at long muscle lengths. Hamstring stretching with increased neural tension resulted in strength loss with no associated shift in the length-tension relationship.

Key Words slump test, length tension, passive stretch, hamstrings

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Introduction

It is well established that maximum voluntary strength is depressed after a series of passive stretches (for review see [3,8]). This effect is referred to as stretch-induced strength loss and has been attributed to neural inhibition of the stretched muscle group (1,2,4). However, it is apparent that static stretching also changes the angle-torque relationship such that there is strength loss at short muscle lengths but not at long muscle lengths (6,10,13). This effect has been attributed to decreased passive muscle stiffness after stretching and its effect on the length-tension relationship (6,13). Previous studies have shown that after repeated stretches, the extensibility of the muscle-tendon junction is increased, whereas the extensibility of the tendon and muscle fascicles is unaffected (11,12). Such an effect would allow greater sarcomere overlap during poststretching isometric contractions.

The slump test has been used to assess adverse neural tension in patients with low back and hamstring injuries, and involves tensioning the neural tissues without additional hamstring stretch (14,15). However, there is limited research on the clinical utility, validity, and reliability of this test. One recent study examined the effect of increased neural tension via the slump test position on passive resistance to stretch (9). Interestingly, increased neural tension via the slump test position resulted in increased passive resistance to stretch of the hamstring muscle group (9). This effect was attributed to passive tension in the neural structures with no apparent contribution from a contractile response to stretch.

Although previous studies have indicated a neural component to stretch-induced strength loss (1,2,4), the exact mechanism of that effect has not been established. It is possible that passive tension on the neural structures during stretch might contribute to stretch-induced strength loss. Therefore, the purpose of this study was to examine the effect of neural tension during hamstring stretching via the slump test position on subsequent stretch-induced strength loss. The hypothesis was that stretch-induced strength loss would be greater after stretches with excessive neural tension.

Methods

Experimental Approach to the Problem

This study used a repeated measures design to assess the effect of neural tension during passive hamstring stretches on

stretch-induced strength loss. Isometric knee flexion strength was measured before and after a series of passive static stretches in either the slump test position (neural tension stretch; Figure 1) or in the neutral spine position (neutral stretch) One leg was stretched in the neutral position, and the contralateral leg was stretched in the neural tension position. Test order (neural tension or neutral performed first) was alternated between subjects.

Subjects

Ten men and 1 woman (age 34 ± 12 years, height 1.77 ± 0.99 m, weight 74.8 ± 12.5 kg, body mass index 23.7 ± 2.5; mean ± *SD*) volunteered to participate in the study. All the subjects were recreationally active, and none had a hamstring or low back injury in the preceding 5 years. Four subjects were runners, and the primary activities of the other subjects were soccer, Gaelic football, ice hockey, triathlon, rowing, baseball, and tennis. All the subjects participated in these activities at least on a weekly basis, and no subject had participated in sports or strenuous physical activity in the previous 48 hours. The study was approved by the institutional review board, and the subjects provided written informed consent.

Procedures

Isometric strength tests were performed on an isokinetic dynamometer (Biodex System 2, Shirley, NY, USA). The subjects were seated with the test thigh flexed 45° above the horizontal, and the seat back at 90° to the horizontal.

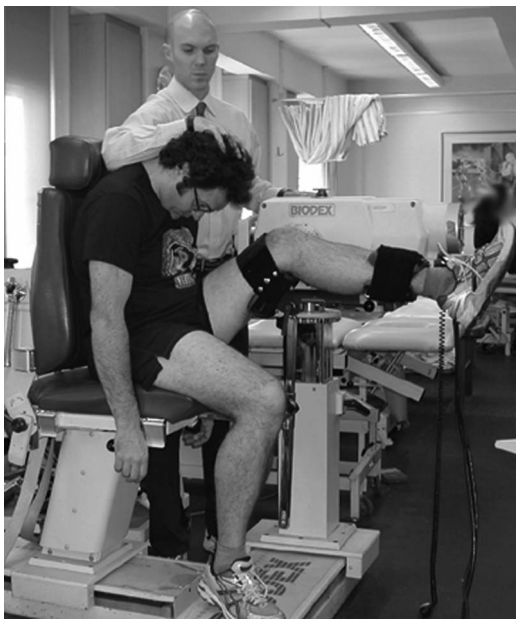

Figure 1. Neural tension stretch position for passive stretches. The cervical and upper thoracic spine was maintained in flexion during passive stretches.

The subjects performed 2 maximal voluntary contractions (MVCs) at 100°, 80°, 60°, and 40° knee flexion. The subjects were provided consistent verbal encouragement during all maximal efforts. In the test position used, the total torque measured during maximum isometric contractions represents the torque resulting from active muscle contraction, torque resulting from the mass of the limb at the respective knee flexion angle, and the torque resulting from the passive tension in the hamstring muscles at the respective knee flexion angle. The limb mass component is maximum at the knee flexion angle at which the limb is horizontal to the ground (gravity effect at its maximum). This typically occurred at a knee flexion angle of 50°. The passive tension component increases as the knee is extended and the hamstring muscle group is elongated. The torque contribution from the combination of limb mass and passive hamstring tension was documented by recording the baseline torque at each knee flexion angle with the subject relaxed. This baseline torque value was subtracted from the torque measured during MVCs at each test angle to provide a measure of contractile torque production. The average of the 2 contractions at each angle is reported. After the baseline isometric strength testing five 1-minute static stretches were applied (in either the neutral position or the neural tension position) after which isometric strength was retested. When the poststretching isometric test was completed on the first test leg, the protocol was repeated on the contralateral leg with the stretch technique (neutral position or neural tension position) alternated. For each subject, the test order was alternated between 4 possible combinations of starting conditions: (a) dominant limb-neutral position, (b) nondominant limb-neutral position, (c) dominant limb-neural tension position, (d) nondominant limb-neural tension position. Thus, with 11 subjects, the first 3 combinations were repeated 3 times, whereas the fourth combination was repeated twice.

Passive stretches were performed in the same seated position as the isometric contraction. The knee was passively extended at $5°\cdot s^{-1}$ from a starting position of 100° knee flexion to the point of maximal tolerable stretch. The subject pressed a switch to stop the dynamometer at the maximum tolerable range of motion (ROM). The stretches were held for 1 minute at the maximum ROM and returned to that same maximum angle for each of the 4 subsequent 1-minute stretches. Resistance to stretch was recorded during the first and fifth stretches and stretch torque at the beginning and end of the 1-minute static stretch was documented. The subjects were asked to grade their stretch discomfort for the first stretch on each leg on a scale of 0–10 where 0 = "no stretch discomfort at all" and 10 = "the maximum imaginable stretch discomfort." For stretches in the neutral position, the subjects kept their head against the headrest for the duration of the stretch to ensure that they maintained a neutral spine position. For neural tension stretches, a physical therapist positioned himself behind the subject and before the commencement of the stretch instructed the subject to relax and let his or her head, neck and trunk slump down,

and allow his or her arms to dangle. Then, the physical therapist manually maintained the cervical and upper thoracic spine flexion for the duration of the stretch (Figure 1).

Surface electrodes were placed over the medial and lateral hamstrings after standard skin preparation, and signals were recorded telemetrically during all stretches and isometric contractions (Noraxon TeleMyo, Scottsdale, AZ, USA). The skin was abraded and cleaned with alcohol before attachment of adhesive electrode pairs with a 1-cm interelectrode distance. Standard electrode placement is midway between the fibular head and the ischial tuberosity for the lateral hamstrings and midway between the medial femoral condyle and the ischial tuberosity for the medial hamstrings. However, the dynamometer thigh support impeded with the electrodes for most subjects in which case they were placed more proximally such that the thigh support did not interfere with the electrodes. The electromyography (EMG) signals were filtered (10–500 Hz) and amplified (60 dB; Noraxon TeleMyo) before sampling at 1 kHz (BioPac Systems AcqKnowledge, Goleta, CA, USA). The amplitude of the raw EMG signal was quantified by using root mean square (RMS) smoothing with a 100-millisecond window. Stretch-induced EMG signals (RMS values) were normalized to signals recorded during the isometric strength tests performed before stretches. The median frequency during isometric contractions was computed by applying Fast Fourier Transforms to demeaned 1,024-point Hanning-windowed sections of the raw EMG. Median frequency was calculated for isometric contractions at 100° and 40° of knee flexion, shortest and longest muscle lengths, respectively.

Resistance to stretch, EMG response to stretch, pain response during stretch, and maximum knee joint ROM during hamstring stretches with and without neural tension were recorded.

Resistance to stretch during passive stretches was recorded as the torque output at maximum ROM at the point when the dynamometer was stopped and after 1 minute of sustained stretch at that ROM. The EMG response to stretch was quantified as the peak EMG amplitude (RMS value) during the stretch. In cases where an EMG response was apparent, peak activity always occurred at the maximum ROM as the dynamometer was stopped. The maximum ROM was the change in joint angle from the starting position (100°) to the knee flexion angle at which the subject stopped the dynamometer. This was recorded for the first stretch on each respective leg. The pain response to stretch was reported after the first stretch on each leg (0 = "no stretch discomfort at all" and 10 = "the maximum imaginable stretch discomfort").

Angle-torque relationships were fitted to second-order polynomials to provide a representation of the length-tension relationship. Based on the individual polynomial equations, torque data were computed for 120°, 100°, 80°, 60°, 40°, and 20° knee flexion to get a better representation of the extremes of motion (the dynamometer setup did not allow for measurements at 120° and flexibility in some subjects prevented measures at 20°).

Statistical Analyses

Effect of stretch technique (neutral vs. neural tension) on passive resistance to stretch, stretch-induced strength loss, and EMG activity was assessed with repeated measures analysis of variance (ANOVA). Stretch-induced strength loss was computed as a percentage of the prestretching value at each angle using the measured torque values. Percent strength loss was compared between stretch techniques and across joint angles using a 2 × 4 (stretch technique × angle) repeated measures ANOVA with Bonferroni corrections for pairwise comparisons. Changes in the angle-torque relationship were assessed using the torque values calculated from the polynomial equations. Based on intrasubject variability in stretch-induced strength loss in the hamstrings (10), it was estimated that with 11 subjects a 10% difference in stretch-induced strength loss between stretching techniques

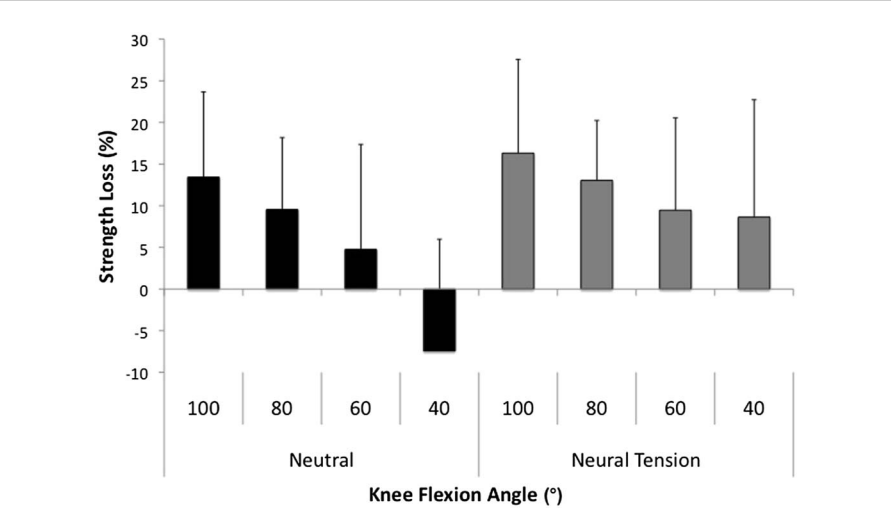

Figure 2. Stretch-induced strength loss after stretches in the neutral position and stretches in the neural tension position. Stretch technique (neutral vs. neural tension) × knee flexion angle (100°, 80°, 60°, 40°) $p < 0.05$. Main effect of stretching on strength loss: neutral stretches $p = 0.09$; neural tension stretches $p < 0.01$. Main effect of angle on strength loss: neutral stretches $p < 0.01$; neural tension stretches $p = 0.2$. For neutral stretches values at 100° greater than at 40° $p < 0.01$. Mean ± *SD* displayed, $n = 11$.

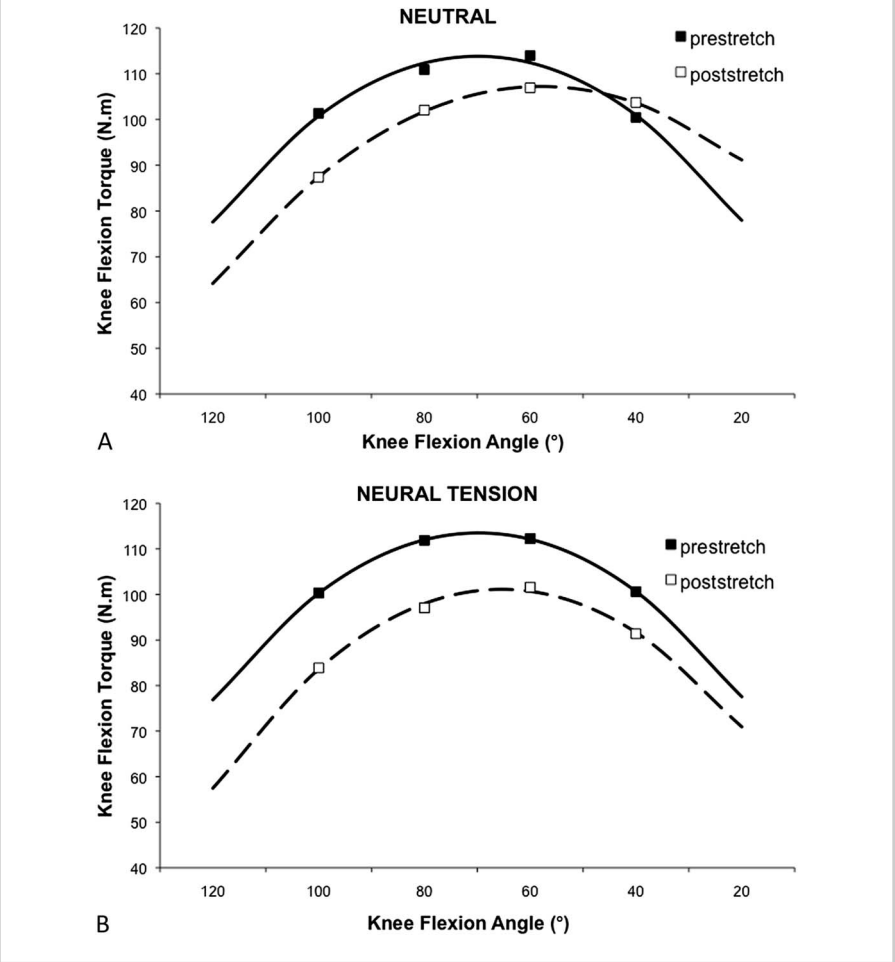

Figure 3. Isometric knee flexion torque before and after stretching in the neutral position (A) and the neural tension position (B). Square symbols are the mean of the measured torque ($n = 11$). Lines are based on the mean torque derived from the individual second-order polynomial equations. Analyses were performed on the torques derived from the polynomial equations at 6 joint angles (120°, 100°, 80°, 60°, 40°, 20°). There was a significant rightward shift in the angle-torque curve (A: $p < 0.01$). By contrast, there was strength loss after stretching in the neural tension position (B; $p < 0.01$) but no shift in the angle-torque curve ($p = 0.43$).

could be detected at $p < 0.05$ with 80% power. This estimate was based on the *SD* of the stretch-induced knee flexion strength loss for measurements made at 6 different joint angles (10). The mean of the *SD*s at each joint angle was used for estimating the effect size here using a standard computation (StatMate Version 2; GraphPad Software Inc.). The level of significance was set at $p \leq 0.05$.

Results

The maximum ROM was 86 ± 8° for neutral stretches and 80 ± 9° for neural tension stretches ($p = 0.13$; stretches started at 100° of knee flexion, so 86° represents a final anatomic knee flexion angle of 14° and 80° represents 20° knee flexion). Stretch intensity, as measured by stretch discomfort, was similar between neutral stretches and neural tension stretches (7.4 ± 1.1 vs. 7.4 ± 1.2, $p = 0.81$), and values were well correlated ($r = 0.86$, $p < 0.01$). Passive resistance to stretch (peak force at maximum ROM at time zero of the 60-second stretch) was reduced by 11% from the first to the fifth stretch ($p < 0.01$) with no difference between neutral (11%) or neural tension (11%) stretches ($p = 0.72$). Resistance to stretch declined during each 60 seconds of static stretch (stress relaxation). The stress relaxation response was progressively less for each stretch (15% for the first stretch, 11% for the fifth stretch; $p < 0.01$) with no difference between neutral or neural tension stretches ($p = 0.69$). There was negligible EMG activity during passive stretches with peak values at maximum ROM for the first stretch of 1.0 ± 0.7% MVC, with no difference between neutral or neural tension stretches ($p = 0.81$). Stretch-induced EMG response was greater for the lateral vs. medial hamstring (1.7 vs. 0.3%; $p < 0.01$).

There was a significant stretch-induced strength loss after stretches in the neural tension position (11.9 ± 7.9%, $p < 0.01$) but no significant stretch-induced strength loss after stretches in the neutral position (5.1 ± 9.0%, $p = 0.09$; difference between stretch techniques $p = 0.08$). Furthermore, stretch-induced strength loss was not different across knee flexion angles after neural tension stretches (angle effect $p = 0.20$). However, after stretches in the neutral position, there was a decline in strength at high knee flexion angles (short muscle length) and an increase in strength at low knee flexion angles (long muscle length; angle effect $p < 0.01$; Figure 2).

There was a rightward shift in the angle-torque (length-tension) curve after neutral stretches such that torque was decreased on the ascending limb and increased on descending limb (stretch effect × knee flexion angle $p < 0.01$; Figure 3A). This effect was not apparent after neural tension stretches (stretch effect × knee flexion angle $p = 0.43$; Figure 3B).

Stretching did not affect EMG amplitude during isometric contractions (<2% decline, $p = 0.58$; no difference between neutral and neural tension, $p = 0.86$). Similarly, stretching did not affect EMG median frequency ($p = 0.42$) with no difference between stretch techniques ($p = 0.92$). Median frequency was higher at short (100°) vs. long (40°) muscle lengths (91 vs. 80 Hz, diff 12 ± 4 Hz, $p < 0.01$).

Discussion

As hypothesized, the addition of neural tension during hamstring stretching, via the slump test position, increased stretch-induced strength loss. Strength loss after stretching with added neural tension was 12% (averaged across the 4 joint angles tested), which was significantly greater than the nonsignificant 5% strength loss seen after stretching without added neural tension. However, this greater stretch-induced strength loss with neural tension was not associated with observable changes in EMG activity, so there was no direct evidence of neural inhibition. Neither the amplitude (RMS) nor the frequency content (median frequency) of the EMG signals was changed after stretching. It is unlikely that this negative finding was because of a type 2 error. The observed changes in EMG amplitude from prestretching to poststretching were <2% for either stretching technique. Based on the *SD* of the difference between prestretching and poststretching EMG amplitude (average difference in voltage from prestretching to poststretching for the medial and lateral hamstrings for each stretching condition), it was determined that a 9% decrease in EMG amplitude could have been detected at $p < 0.05$, with 80% power. With respect to median frequency, the largest observed poststretching decrease was 5 Hz for the medial hamstring at 40° and the average poststretching decrease across joint angles was 1 Hz. Based on the *SD* of the difference between prestretching and poststretching median frequency, it was determined that a 7-Hz decrease in median frequency could have been detected at $p < 0.05$, with 80% power. Thus, there was sufficient power to detect meaningful changes in EMG amplitude (9%) or frequency (7 Hz) after stretching. The difference in median frequency during isometric contractions at 100° vs. 40° knee flexion was 12 ± 4 Hz ($p < 0.01$). This is consistent with the known effect of muscle length on the frequency content of the EMG signal and further indicates that there was sufficient measurement sensitivity to detect a meaningful effect of stretching on median frequency.

The small change in EMG amplitude after stretching, observed here, is in contrast with the results of Avela et al. (2) who reported a 20–29% reduction in the EMG amplitude after repeated passive stretching of the plantar flexors. However, that study involved 60 minutes of repeated passive stretching. More in line with this study, Cramer et al. (4) used a quadriceps stretching protocol consisting of an 8-minute total stretch time for each leg, and demonstrated a 5% reduction in the EMG amplitude. This effect was apparent in both the stretched and nonstretched limbs. In this study, the total stretch duration was 5 minutes for each leg, and no significant decrease in EMG activity was apparent during subsequent MVCs. It is possible that a longer total stretch time might have resulted in decreased EMG activity.

Stretching in the neutral spine position resulted in a shift in the angle-torque curve for isometric contractions such that there was decreased torque at short muscle lengths and increased torque at long muscle lengths. This represents a rightward shift in the length-tension curve and is consistent with previous findings (6,10,13). During isometric contractions, muscle fibers shorten, and the tendon and aponeuroses lengthen to the point at which the contractile force is insufficient to further elongate the tendon and aponeurosis (5,7). The rightward shift in the length-tension curve can be explained by greater tendon and aponeurosis elongation during poststretching isometric contractions thereby allowing greater muscle fiber shortening at a given joint angle. Such an affect implies that the stretching protocol increased tendon and aponeurosis compliance. Interestingly Morse et al. (12) demonstrated that the increase in compliance of the medial gastroc muscle-tendon unit after a series of passive stretches was primarily because of increased compliance at the myotendinous junction with no change in tendon or fascicle compliance. It was concluded that stretching increased aponeurosis compliance. Similar effects in the medial gastroc were recently demonstrated by Mizuno et al. (11). It remains to be determined if similar effects of stretching are seen in the hamstring muscles.

Passive resistance to stretch was decreased by 11% after stretching in the neural tension position and after stretching in the neutral position. However, there was no apparent rightward shift in the length-tension curve after stretching in the neural tension position. The absence of this effect after neural tension stretches suggests that the muscle fibers did not shorten more during poststretching isometric contractions vs. prestretching contractions. Because the neural structures are in parallel to the muscle-tendon units, with branches terminating within muscle tissue, it is possible that tension in the neural structures may have stress shielded the tendons and aponeuroses of the hamstring muscles. However, maximum ROM was not significantly different between neural tension (80°) and neutral (86°) stretches, so actual elongation of the hamstring muscles may not have differed between stretching techniques. This lack of difference in maximum ROM differs from previous work showing that the maximum ROM was reduced by 8° with neural tension stretches vs. neutral stretches of the hamstrings (9). However, in that study, neutral and neural tension stretches were applied alternately to the same leg, whereas in this study, 1 leg received neural tension stretches while the other leg received neutral stretches. Differences in hamstring flexibility between legs may have confounded the comparison of maximum ROM between stretch techniques in this study.

The fact that passive resistance to stretch was reduced by a similar amount after neutral and neural tension stretches indicates that the viscoelastic adaptations to stretch were

similar. Nevertheless, it is possible that tissue elongation across the different load bearing tissues (muscle, tendon, aponeurosis, neural structures) differed between neural tension and neutral stretches thus affecting subsequent isometric contractions differently. Ultrasonic imaging of fascicle length changes would be required to confirm such an effect but such techniques have not been reported for the hamstring muscles.

This study used a sample of convenience, and as such, the data may not be generalizable to populations with specific training or flexibility patterns. Whether these findings hold true for individuals that regularly stretch remains to be determined. Similarly, this study did not examine possible gender differences in effects of neural tension on stretch-induced strength loss. The scope of the study was limited to assessing the effect of neural tension on stretch-induced strength loss in a sample sufficiently large enough to detect meaningful differences in response to a single bout of stretching. It is not known if individuals with greater passive muscle stiffness experience greater neural tension during stretching and thus sustain greater stretch-induced strength loss or show a greater shift in the length-tension relationship after stretching.

In conclusion, hamstring stretching with the spine in a neutral position did not result in significant strength loss but did result in a shift in the length-tension relationship such that strength was decreased at short muscle lengths and increased at long muscle lengths. Hamstring stretching with increased neural tension resulted in marked strength loss (12%) with no associated shift in the length-tension relationship.

Practical Applications

Hamstring stretching with the spine in a neutral position is recommended to effectively decrease passive muscle stiffness with no associated strength loss. Hamstring stretching with a flexed spine should be avoided to prevent stretch-induced strength loss.

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