

The role of neural tension in hamstring flexibility

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Resistance to stretch, electromyographic (EMG) response to stretch, stretch discomfort and maximum range of motion (ROM) were measured during passive hamstring stretches performed in the slump test position (neural tension stretch) and in the upright position (neutral stretch) in eight healthy subjects. Stretches were performed on an isokinetic dynamometer at 5°/s with the test thigh flexed 40° above the horizontal, and the seat back at 90° to the horizontal. Surface EMG signals were recorded from the medial and lateral hamstrings during stretches. Knees were passively extended to maximum stretch tolerance with test order (neural tension vs neutral) alternated between legs. For

neural tension stretches, the cervical and thoracic spine were manually flexed. Maximum ROM was 8° less for the neural tension stretch vs the neutral stretch ($P < 0.01$). Resistance to stretch was 14–15% higher for the neural tension stretch vs the neutral stretch ($P < 0.001$) at common joint angles in the final third of ROM. Stretch discomfort and EMG response were unaffected by neural tension. In conclusion, an increased passive resistance to stretch with the addition of neural tension during passive hamstring stretch despite no change in the EMG response indicates that passive extensibility of neural tissues can limit hamstring flexibility.

Resistance to passive stretch of skeletal muscle in healthy human subjects has been attributed to the extensibility of the non-contractile connective tissue components of muscle tendon units (McHugh et al., 1992), the stretch-induced contractile response to stretch (Etnyre & Abraham, 1988) or resting tension in myofibrils (Magid & Law, 1985). However, studies that have simultaneously examined range of motion (ROM), resistance to stretch and electromyographic (EMG) responses from the stretched muscle indicate that resistance to stretch is primarily due to the viscoelasticity of the non-contractile component (Magnusson et al., 1996, 1997; McHugh et al., 1998).

The slump test is used to assess adverse neural tension in patients with low-back and hamstring injuries, and involves tensioning the neural tissues without additional hamstring stretch (Kornberg & Lew, 1989; Lew & Briggs, 1997; Webright et al., 1997; Turl & George, 1998). This is achieved by flexing the cervical and thoracic spine during hamstring stretch (Fig. 1). Additional neural tension can be provided by simultaneously dorsiflexing the ankle. The goal of the test is to differentiate nerve root pain from muscle pain. A positive test is defined as one in which the symptoms are produced during hamstring stretch in the slump position and alleviated with cervical extension (Kornberg & Lew, 1989). The pain elicited by the slump test is thought to be due

to excessive nerve stretch (intra-neural), or reduced neural mobility at the interface with the surrounding muscle tissue (extra-neural) (Turl & George, 1998). The evidence that the slump test actually tensions neural structures is based on the clinical signs (eliciting pain) rather than any direct evidence of an increased tension in the neural tissues. Lew and Briggs (1997) recorded the hamstring EMG response and tension in the biceps tendon in the slump position with and without cervical flexion and concluded that this maneuver did not further elongate the hamstring muscle group and did not elicit a hamstring EMG response. Therefore, pain elicited during the test was attributed to neural tension as opposed to an increased stretch on the hamstrings. More recently, Laessøe and Voigt (2004) demonstrated that the slump test decreased the maximum ROM and the maximum tolerated resistance to stretch during hamstring stretches. However, the relationship between resistance to stretch and ROM was not examined throughout the common ROM for the stretch. Therefore, it could not be determined if the additional neural tension increased passive resistance to stretch at submaximal ROM (e.g. in the mid-range of the available joint motion). The possibility that ROM during passive stretch of skeletal muscle in healthy subjects is limited by the extensibility of the neural tissues has not been examined previously.

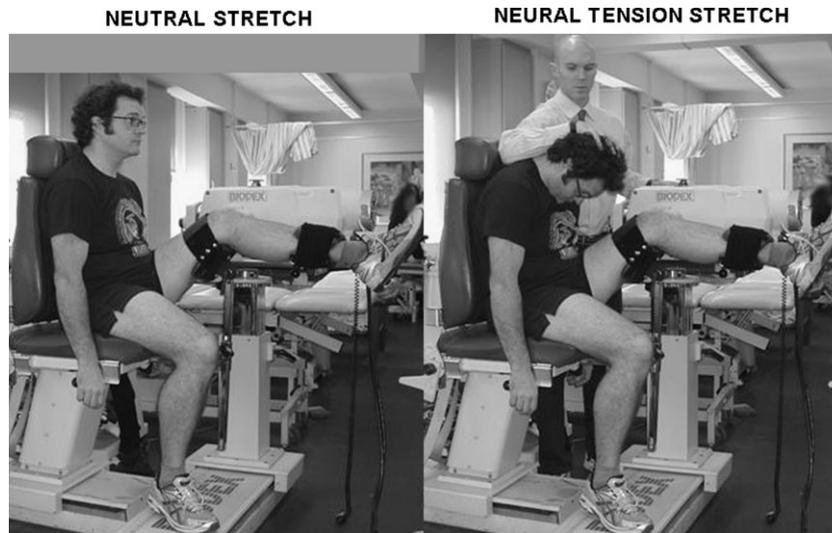


Fig. 1. Test position for the neutral stretch (left) and the neural tension stretch (right).

Therefore, the purpose of this study was to determine if neural tension, via the slump test, during passive hamstring stretch, affected maximum ROM, stretch discomfort, resistance to stretch, or the EMG response to stretch in healthy subjects. It was hypothesized that neural tension would decrease maximum ROM and increase stretch discomfort without affecting the EMG response or resistance to stretch.

Methods

Resistance to stretch, knee flexion ROM, hamstring EMG activity and stretch discomfort were measured during passive hamstring stretches in eight subjects (six men, two women), mean (SD) age 39 (13) years, weight 70 (11) kg. Subjects had no significant history of low-back or hamstring injury, were in good general health and were active (all reported being able to run 1 mile in <10 min). All subjects gave written informed consent and the study protocol was approved by institutional review board. Stretches were performed with the thoracic and cervical spine in neutral position (neutral stretch) and in the slump test position (neural tension stretch). For the neural tension stretch, manual pressure was provided to maintain cervical and thoracic flexion during the test. Starting leg (right or left) and test sequence (stretch with or without neural tension) were alternated between subjects. The test sequence for a subject beginning with their right leg being stretched in the neutral position was as follows: right leg neutral stretch, right leg neural tension stretch, left leg neural tension stretch and left leg neutral stretch. Thus, there were four possible test sequences and with eight subjects each sequence was performed twice. Each stretch on the same leg was separated by 4 min, with approximately 10 min between stretches on different legs (additional time to switch the dynamometer to the opposite leg).

Hamstring stretching protocol

Stretches were performed using an isokinetic dynamometer (5°/s) with subjects seated, the test thigh flexed 40° above the horizontal and the seat back at 90° to the horizontal (Fig. 1). Knees were passively extended from 90° flexion to maximum

stretch tolerance. Subjects held a switch in their hand during stretches that automatically stopped the stretch at self-determined maximum ROM. The stretch was then released. For neural tension stretches, the cervical and upper thoracic spine were manually flexed by a physical therapist (Fig. 1, right). Subjects were asked to grade their stretch discomfort on a scale of 0–10 where 0 = “no stretch discomfort at all” and 10 = “the maximum imaginable stretch discomfort”. While the slump test is usually performed in sitting without additional thigh flexion of the test limb, the experimental setup in the present study was used to ensure that maximum ROM was achieved before the knee was fully extended for both neural tension and neutral stretches.

EMG measurements

Surface EMG electrodes were applied to the medial and lateral aspects of the hamstring muscle group. Standard electrode placement for the biceps femoris (lateral) and semitendinosus (medial) was not possible due to the placement of the stabilizing pad used to position the thigh in 40° flexion (see Fig. 1). Therefore, electrodes were placed proximally after abrading and cleaning the skin. Two maximal voluntary isometric hamstring contractions (MVC) were performed at the stretch starting position (90° knee flexion) before the first stretch for subsequent normalization. EMG signals were recorded by telemetry (Noraxon TeleMyo, Scottsdale, Arizona, USA) during the isometric contractions and the stretches. EMG signals were filtered (10–500 Hz) and amplified (60 dB; Noraxon TeleMyo) before sampling at 1 kHz (BioPac Systems AcqKnowledge, Goleta, California, USA). The signals were then rectified and smoothed (40-ms RMS). A 1 s average covering the visual peak EMG amplitude was recorded for the two MVCs and then averaged across contractions and muscles. The 1 s average EMG amplitude at maximum ROM for each stretch was recorded and averaged across muscles. Stretch-induced EMG signals are expressed as a percentage of MVC.

Normalization of resistance to stretch

Because maximum ROM varied between subjects, knee flexion angle for each leg was expressed as a percentage of the maximum ROM achieved for the stretch (neutral vs neural

tension) that yielded the lowest maximum ROM. The starting angle was defined as 0° and the maximum ROM for the stretch yielding the lower maximum ROM (neutral vs neural tension) on a given leg was defined as 100%. Thus, the maximum ROM for the stretch yielding the higher ROM was given a value >100% depending on the relative difference between the maximum ROM for the two stretches. Resistance to stretch at 10% increments in ROM was recorded for each stretch providing 11 paired values (neutral and neural tension trials) from 0 to 100% for each leg.

Statistics

Normalized resistance to stretch (10% increments from 0 to 100%) was compared between the neural tension stretch and the neutral stretch with an 11 × 2 repeated measures ANOVA. Bonferroni corrections were used for planned pairwise comparisons (11 comparisons between neural tension and neutral stretches from 0 to 100% of maximum ROM, $P < 0.05/11 = P < 0.0045$). Based on the reliability of measures of passive resistance to hamstring stretch (Magnusson et al., 1995), it was estimated that with eight subjects testing both legs in a fully repeated measures design, a difference of 12% in passive resistance could be detected between stretching techniques (neutral vs neural tension) at $P < 0.05$ with 80% power.

Results

Maximum ROM was 8(5)° (mean and SD) less (9%) for the neural tension stretch vs the neutral stretch ($P < 0.01$), while stretch discomfort, EMG response, and maximum resistance to stretch did not differ between stretches (Table 1). The increase in resistance to stretch with the increasing stretch was progressively greater for the neural tension stretch vs the neutral stretch (stretch position by joint angle $P < 0.001$); resistance to stretch at common angles was 14–15% higher for the neural tension stretch vs the neutral stretch during the last third of the ROM (Fig. 2).

Discussion

The primary finding in this study was that adding neural tension during a hamstring stretch (slump position) increased the resistance to stretch compared with a hamstring stretch with the spine in a neutral position. This increased resistance was not due to a change in the contractile response to stretch.

The contractile response to hamstring stretch was low regardless of stretch position (4.6% MVC in neutral position and 3.2% MVC in neural tension position). Importantly, there was no observable contractile response to stretch in any subjects until within the last 10° of ROM and differences in resistance to stretch between neural tension and neutral stretches were apparent approximately 25° before maximum ROM (final third of ROM; Fig. 2). Additionally, 40% of stretches showed no observable EMG response on examination of the raw EMG signals. A secondary finding was that an increased neural tension during a hamstring stretch resulted in an 8° decrease in the maximum ROM that the subject could achieve (9% decrease), while maximum resistance to stretch and stretch discomfort were not different between the neural tension and neutral stretches. Because subjects were instructed to terminate the stretches at the maximum tolerable ROM, the lack of difference in stretch discomfort and maximum resistance to stretch indicate that there was no systematic difference in the subjective inten-

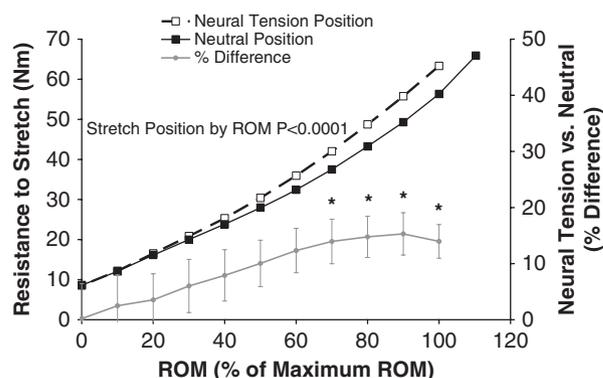


Fig. 2. Resistance to stretch throughout the range of motion (ROM) for the neutral and neural tension stretches. ROM is expressed as a percentage of the maximum ROM achieved in the stretch (neural tension or neutral) with the lower maximum ROM (the neural tension stretch had a lower maximum ROM than the neutral stretch in 13 of 16 stretches with the other three having the same maximum ROM for both types of stretches). The percentage difference in resistance to stretch between neural tension and neutral stretches is shown for each 10th percentile in ROM (*resistance to stretch significantly greater for neural tension vs neutral $P < 0.05$).

Table 1. Maximum values for resistance to stretch, ROM, stretch discomfort (VAS) and EMG response

	Neutral position	Neural tension position	P-value
Maximum ROM (higher value = more flexible/more extended knee)	83 ± 7°	76 ± 9°	0.002
VAS at maximum ROM	6.3 ± 2.1	6.9 ± 2.3	0.29
EMG at maximum ROM (%MVC)	4.6 ± 5.4	3.2 ± 4.8	0.1
Maximum resistance to stretch (Nm)	65.9 ± 22.0	62.9 ± 21.1	0.19

Values are given as mean ± SD.

ROM, range of motion; EMG, electromyograph; MVC, maximal voluntary isometric hamstring contractions.

sity of stretch between the neural tension and neutral stretches.

Tensioning of the neural structures during passive stretch is the basis of the slump test but the possibility that this neural tension might increase passive or active resistance to stretch has not been extensively examined. In the absence of significant contractile activity, an increased resistance to hamstring stretch in the neural tension position (Fig. 2) can be attributed to tensile force in the neural structures. Such a finding has not previously been demonstrated *in vivo* during passive stretch of human skeletal muscle. This increased tension may be due to resistance to elongation of the neural tissues (intra-neural) or adhesions between the neural tissues and the surrounding tissues preventing the neural tissues from gliding freely (extra-neural) (Turl & George, 1998). The increased resistance to stretch during the neural tension stretch vs the neutral stretch can also explain the lower maximum ROM. Maximum ROM is a function of the subject's ability to tolerate the discomfort associated with the increasing resistance to stretch. Because resistance to stretch with an increasing ROM during the stretch was greater for the neural tension stretch vs the neutral stretch maximum ROM was achieved at a lesser ROM.

A difference in sagittal plane pelvic rotation between the two stretch techniques is an important potential confounding factor in the experimental intervention used in this study. If the pelvis rotated anteriorly in the neural tension position vs the neutral position, this would increase the stretch on the hamstring muscle group at any given knee flexion angle. This anterior pelvic rotation would increase resistance to stretch. Accurate measurement of subtle changes in pelvic rotation in the seated posture used here would probably require radiographic imaging, which would not have been practical in this setting. In any case, cervical and thoracic flexion in the slump position are more likely to posteriorly tilt the pelvis thereby decreasing the stretch on the hamstrings. Posterior pelvic tilt has been documented with the slump position (O'Sullivan et al., 2006). While care was taken to maintain pelvic and lumbar spine position between stretches, some posterior pelvic tilt with the slump position may have occurred. Such an effect would have decreased the ability to detect differences in resistance to stretch between the neural tension and neutral stretches. Because marked differences in resistance to stretch were apparent between stretch techniques, the confounding effect of possible pelvic rotation was probably not a limiting factor.

Potential differences in ankle angle during the neural tension and neutral position stretches are another confounding factor. Dorsiflexion limits maximum active knee extension ROM in the slump test

(Johnson & Chirello, 1997). This effect is similar in magnitude to the effect of cervical flexion; dorsiflexion combined with cervical flexion has a compounding effect (Johnson & Chirello, 1997). Therefore, while the addition of dorsiflexion might have further limited ROM in this study, the existing literature indicates that cervical and thoracic flexion without dorsiflexion would be sufficient to limit ROM. Furthermore, because the gastrocnemius muscles cross the knee and ankle joint, passive stretch of those muscles (with ankle dorsiflexion) would provide additional passive stretch to the hamstrings; tension in the gastrocnemius muscles would tend to pull the knee into extension. It was important to avoid such an effect so that the primary difference between the two stretch conditions was the addition of neural tension with no additional stretch on the hamstring muscles.

Positive slump tests, indicating adverse neural tension, have been documented in athletes who have sustained recurrent hamstring strains (Turl & George, 1998). It was unclear if this effect was due to intra-neural factors (stretch injury of the neural tissues) or extra-neural factors (reduced mobility of the neural tissues due to scarring and adhesions in the muscle tissue). The extent to which excessive neural tension contributed to resistance to stretch was not examined but it was noted that flexibility (maximum ROM) was not different between the previously injured players and controls, or between the players with and without positive neural tension tests. However, this does not preclude a greater contribution to resistance to stretch from the neural tissues in the previously injured athletes.

Numerous studies have demonstrated acute losses of maximal voluntary strength following passive muscle stretching (see McHugh & Cosgrave, 2010 for review). These effects have been attributed to neural inhibition as evidenced by the reduced amplitude of the surface EMG after rapid cyclic stretches (Avela et al., 1999; Avela et al., 2004) or strength loss in the contralateral non-stretched limb after static stretches (Cramer et al., 2005). The mechanism for this stretch-induced neural effect is currently unknown but it may be related to adverse neural tension during passive stretch. In an animal model, sustained stretch to peripheral nerves was shown to result in a depression in nerve conduction velocity that was apparent 30 min after release of the stretch. The hypothesis that stretch-induced strength loss is due to neural tension could be tested by comparing strength loss following stretching with and without neural tension.

The small EMG response to hamstring stretch with or without neural tension is consistent with previous work (Magnusson et al., 1996, 1997; McHugh et al., 1998) and emphasizes that resistance

is primarily due to the passive extensibility of the tissues under stretch. Within the passive structures, it appears that there is an elongation in the tendon–aponeurosis complex and muscle fascicles during passive stretch (Herbert et al., 2002). However, passive muscle stiffness measured during the stretch of relaxed muscles was shown to be unrelated to tendon–aponeurosis stiffness measured during isometric contractions (Kubo et al., 2001). Therefore, the extensibility of the muscle fibers, and more importantly, the surrounding fascial components (endomysium, perimysium and epimysium) are thought to be the primary determinants of passive resistance to stretch. In this regard, it has been suggested that the extensive connective tissue network of the perimysium is the major contributor to passive resistance to stretch (Purslow, 1989). The possibility that tension in neural tissues can contribute to passive resistance to stretch was not considered in these studies (Purslow, 1989; Magnusson et al., 1996, 1997; McHugh et al., 1998; Kubo et al., 2001) but appears to be a possibility based on the present results.

It was estimated that a 12% difference in resistance to stretch would be detectable at $P < 0.05$ with 80% power using a sample of eight subjects. Thus, the observed effects of 14–15% higher resistance with the neural tension stretch fitted well with the prediction for this sample size. However, a priori effect sizes were not estimated for the other dependent variables. Of relevance is the magnitude of difference in stretch discomfort and stretch-induced EMG response between stretch techniques that could have been detected. Based on the results, it is estimated that there was 80% power to detect a difference in stretch-

induced EMG of 3.5% MVC and a difference in stretch-induced discomfort of 2.5 points between neural tension and neutral stretches at $P < 0.05$.

In conclusion, the addition of neural tension during hamstring stretches, by way of the slump test position, significantly the increased passive resistance to stretch without affecting the contractile response to stretch. These findings indicate that extensibility in the neural tissues can contribute to passive resistance to stretch.

Perspectives

The possibility that passive tension in the neural tissues might contribute to resistance to stretch has not been extensively studied. In this study, the increase in passive resistance to hamstring stretch with cervical and thoracic flexion (slump test position), in the absence of meaningful EMG activity, indicates that extensibility of neural structures can contribute to musculoskeletal flexibility. The clinical and practical relevance of these findings are twofold: (1) adverse neural tension may be a contributing factor in the high recurrence of hamstring strains (as an indicator of adhesions within the muscle or as a source of recurrent pain); (2) neural tension during passive stretching might be the neural mechanism for stretch-induced strength loss. The role of neural tension in hamstring strain recurrence and stretch-induced strength loss can be examined in future studies using this methodology.

Key words: slump test, viscoelasticity, stretching, muscle extensibility.

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