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Article Title: Effects of a Novel Neurodynamic Tension Technique on muscle extensibility And Stretch Tolerance: A Counterbalanced Cross-Over Study

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Effects of a novel neurodynamic tension technique on muscle extensibility and stretch tolerance: a counterbalanced cross-over study.

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Abstract.

Context: Neurodynamic tension affects hamstring extensibility and stretch tolerance, and is considered important in hamstring injury management. Neurodynamic tension was postulated to affect segmental muscle extensibility and stretch tolerance, and potentially also demonstrate extra-segmental and contralateral effects. **Objectives:** Assess the effects of a novel sciatic-tibial neurodynamic tension technique, the modified long sit slump (MLSS), on segmental, extra-segmental and contralateral muscle extensibility and stretch tolerance. . **Study design:** Counterbalanced cross-over study. **Setting:** University research laboratory. **Participants:** Thirteen healthy and active subjects (mean±SD age 24±8 y, BMI 23.1±2.8 $kg \cdot m^{-2}$). **Intervention:** MLSS application (5 seconds, 5 repetitions, 3 sets) on two occasions with a three-week washout period, and either stance or skill leg treated in a counterbalanced manner. **Main outcome measures:** Segmental and extra-segmental muscle extensibility were measured utilising passive straight leg raise (PSLR) and prone knee bend (PKB) at pre-, immediately post- and one hour post-intervention. Stretch intensity ratings were measured utilising a simple numerical rating scale (SNRS). **Results:** MLSS significantly increased PSLR and PKB bilaterally ($p<0.001$). The effect for PSLR was greater in the ipsilateral leg compared to the contralateral leg (baseline to one hour post: +9±6°and +5±5° respectively, $p<0.001$), but not for PKB (baseline to one hour post: ipsilateral leg +5±5°, contralateral leg +5±4°). For both PSLR and PKB the effect of the first session was retained at the start of the second session 3 weeks later. SNRS data were consistent with increased stretch tolerance. **Conclusions:** Application of a novel sciatic-tibial neurodynamic tension technique, the MLSS, increases muscle extensibility and stretch tolerance segmentally, extra-segmentally and contra-laterally. **Level of evidence:** 2C Outcomes research.

Key words: flexibility, hamstrings, muscle extensibility, neurodynamics, stretching, neuronal desensitisation.

INTRODUCTION

Hamstring strain injury (HSI) is one of the most common non-contact injuries in athletes,[1-3] with high rates of recurrence,[4] despite considerable research efforts.[5] The role of hamstring flexibility, also termed extensibility herein, in HSI, [4,6-7,11] re-injury and rehabilitation, [2,8,12,13] has not been fully elucidated to date. [8-10] Neurodynamics is a term describing mobilisation of the nervous system and its surrounding structures.[14-15] Neurodynamic tension techniques elongate the neural tissue and are considered to increase nerve tension and strain, whereas neural sliding techniques aim to maximise nerve excursion.[16] Neurodynamic tension has been demonstrated to significantly influence hamstring extensibility[17-18] and is considered important in HSI, re-injury and rehabilitation.[19-20] For example, Turl & George[20] demonstrated 57% of elite rugby players with recurring grade one HSI demonstrated positive slump test[21] after returning to play, suggesting suboptimal neurodynamics may contribute to known high rates of re-injury.[4,22] Similarly, Kornberg & Lew[19] demonstrated inclusion of a neurodynamic tension technique to rehabilitation of Australian Football League players with HSI resulted in significantly faster return to play.

Human in-vivo hamstring stretching studies in non-injured subjects strongly supports stretch tolerance as a primary mechanism responsible for lasting increases in hamstring extensibility utilising intervention protocols of up to eight weeks duration, with longer term stretching postulated to potentially induce structural alterations in hamstring muscle length and passive stiffness.[23-25] Immediate stretch-induced changes in hamstring passive stiffness are considered to be due to viscoelastic stress relaxation, with effects typically potentiated within five loading cycles and attenuated within an hour.[26] Previous research has demonstrated lasting increases in hamstring extensibility are of similar magnitude irrespective of the stretching protocol utilised, citing total weekly stretch time as the most important variable.[27-29] However, there is some evidence that more intense stretching may effect greater changes in extensibility, or at the very least, saves time and is therefore considered more efficient.[28,30] As neurodynamic tension is associated with relative increased levels of reported stretch intensity during hamstring stretch for a common ROM,[17,31] it was postulated that it may have a significant role in afferent modulation of stretch tolerance.[18,25]

Compared to muscle stretching protocols, there has been relatively little research investigating utilisation of neurodynamic techniques on lasting changes in hamstring extensibility and stretch tolerance.[18,32-33] For example, Castellote-Caballero and colleagues[32] demonstrated a significant increase in passive straight leg raise (PSLR) of nine degrees following three sessions of a neurodynamic slider over one week. Although comparatively this is an average PSLR gain for a hamstring extensibility study, it was achieved in a relatively short period of time.[34-35] More recently, Sharma and co-workers[18] reported significantly greater hamstring extensibility gains when neurodynamic techniques and muscle stretching were utilised compared to muscle stretching alone, but the intervention dosing between the groups was inconsistent which lessens the strength of conclusions drawn from this randomised controlled trial (RCT).

The specific groups of afferent neurones primarily affected during stretching and modulation of stretch tolerance are yet to be fully elucidated.[25,36] Small and large diameter proprioceptors are fundamentally implicated in stretch sensation, but a significant role of mechanosensitive nociceptors has also been suggested and warrants more detailed consideration.[24,36-39] As initiation of stretch discomfort has been reported to occur at 85% of muscle passive torque values recorded for maximal stretch tolerance,[40] direct activation of mechanosensitive nociceptors resulting from stretch-induced tensile strain, secondary compression, or a combination of the two, is probable.[37-38,41]

Notwithstanding likely short term modulation of stretch tolerance through an inhibitory nociceptive 'gating' mechanism at the spinal dorsal horn through activation of non-nociceptive afferent fibres,[36,42-44] proprioceptor and mechanoreceptor discharge in the early stage of muscle stretch could sensitise mechanosensitive nociceptor discharge towards activation thresholds,[38,41,46] particularly as peripheral afferent neuropeptides are largely unspecific to fibre type.[38,46-47] This is likely accentuated by mechanisms such as the axon reflex and afferent convergence.[38,45] Furthermore, the same afferent neuropeptides which are utilised distally are produced in dorsal root ganglia,[46-47] the neuropeptides having both peripheral and central neuromodulatory effects that may outlast the duration of stretch.[25,36] Moreover, the parameters and context of stretching likely affect spinal and supraspinal processing, which may also alter the diffuse noxious inhibitory system (DNIS), and has also been implicated in modulation of stretch tolerance through conditioned learning.[36,44]

Inter-neuronal activation and recruitment of latent nociceptive circuits is considered a primary mechanism by which pain spreads segmentally, extra-segmentally and contralaterally.[48-52] Given such central pain sensitisation has been considered a form of neuronal long term potentiation (LTP) and learning,[42,44,53-54] it was postulated herein that the increased stretch tolerance from stretching could be a form of neuronal long term depression (LTD),[43,55] and stretch tolerance may also demonstrate a similar course of segmental, extra-segmental and/or contralateral effect, given the appropriate stimulus.[51,56]

Therefore the study hypothesis was that application of a novel sciatic/tibial nerve neurodynamic tension technique, the modified long sit slump (MLSS), would increase muscle extensibility and stretch tolerance segmentally, extra-segmentally, and contra-laterally.

METHODOLOGY

Study design

A counterbalanced crossover experiment over two intervention sessions was utilised, with each intervention session utilising a single limb from each subject (**Figure 1**). In order to avoid effects of intervention order and/or limb dominance, the treatment order was counterbalanced with 7 subjects having the stance leg treated first and the remaining 6 subjects receiving treatment on the skill leg first, the skill leg defined as that which the subject reported to preferentially use to kick a ball. Previous research has not demonstrated any contralateral effects from unilateral stretching[24,32,36] and a three week 'wash out' period was deemed sufficient for any treatment effects to wear off.[28,57] The independent variables were unilateral neurodynamic intervention (MLSS) over two sessions, the dependent variables being ipsilateral and contralateral hamstring and rectus-femoris extensibility and stretch tolerance. The dependent variables were measured pre-, immediately post- and one hour post-intervention. Subjects were requested not to partake in unfamiliar physical activity for three days prior to testing and strenuous physical activity on the day of testing, and not to stretch the lower limbs between intervention sessions. All testing was performed in a university laboratory. Recruitment and data collection occurred between March and April 2016.

Participants

A healthy and active sample of convenience was recruited from a university population. Assuming alpha = 0.05 with 80% power and utilising one degree standard error of measurement and four degree minimum detectable difference for a hand held inclinometer, *a priori* sample calculation was 12.[58] Subjects were recruited via print poster, electronic university noticeboard, and limited e-mail recruitment. One extra subject was recruited in case of drop out, with a final sample size of 13 (9 male, 4 female, mean ± SD age 24±8 years, Body Mass Index 23.1±2.8 kg·m^{-2}). Healthy and active was defined as no history of significant medical conditions and a minimum Tegner Activity Scale[59] rating of five, respectively. Further exclusion criteria were significant neurological or orthopaedic conditions, past history of HSI, significant low back pain, and participation in a formal hamstring lengthening or strengthening program in the previous six months. Subjects with clinically 'tight' hamstrings were recruited, adopting values equal or lower than 75º for men and 80º for women, with potential participants with PSLR above these values excluded from the study.[34,60-61] Ethics approval was obtained through the University of Bath Research and Ethics Approval Committee for Health (REACH; EP 14/15 201) and suitable subjects were required to provide signed, informed consent. The rights of all subjects was protected.

Procedures

Subjects were screened for clinically 'tight' hamstrings by PSLR utilising a hand held inclinometer (Isomed AcuAngle).[58,62] The subject lay supine with the non-tested thigh secured to the plinth with a firm adjustable strap. The base of the inclinometer was marked on the anterior distal tibia of the tested leg, corresponding to the zero value. The inclinometer was secured with Velcro straps and the subject was instructed to fully relax during testing. The examiner raised the leg slowly until the subject expressed maximal stretch tolerance was reached or firm resistance to further elevation was encountered. The subjects were given a standard set of scripted instructions for the PSLR procedure, with only one measure utilised for screening, consistent with clinical practice.

Assessment

PSLR was utilised as the ipsilateral and contralateral segmental muscle extensibility measure, as described above. A simple numerical rating scale (SNRS), with zero representing 'no muscle stretch' and ten representing 'the worst muscle stretch imaginable' was utilised as a subjective measure of stretch intensity.[36] SNRS measures were taken at maximal PSLR ROM for pre and post intervention time points (SNRS Max), and at the pre intervention maximal PSLR ROM for the post intervention time points (SNRS Com). If post intervention PSLR was less than pre intervention, SNRS Com was not assessed. Ipsilateral and contralateral extra-segmental extensibility of the rectus-femoris was measured utilising a prone knee bend (PKB) procedure. Subjects lay prone with a strap stabilising the pelvis applied at the level of the lower half of the sacrum. The subject's tested hip was positioned in approximately 10º extension by placing a high density foam roll between the thigh and the plinth, immediately proximal to the superior patella. The examiner slowly flexed the knee until the subject expressed maximal stretch tolerance was reached or further ROM was blocked by the posterior thigh. The examiner then placed the inclinometer on the previously marked points on the tibia to measure ROM. PKB SNRS stretch intensity measurement procedures were as for PSLR. All measurements were repeated 5 times, the fifth of which was recorded. Subjects remained in the laboratory resting room between immediate and one hour post-intervention assessments.

Warm-up

A light warm-up of 10 minutes of cycling on a stationary bicycle at a minimal resistance was adopted immediately prior to intervention, with subjects instructed to maintain an intensity whereby they were not short of breath.

Intervention

The MLSS intervention is shown in (**Figure 2**): In the starting position, subjects were positioned hemi-sitting on a plinth (adjusted to height approximately 15 cm below greater trochanter), with the stretched limb resting on the plinth while the other limb rested parallel on the floor. With the knee on the plinth flexed in the starting position, the subject used their opposite hand to reach forward to hold the lateral border of the opposite foot, placing it in dorsiflexion and eversion. This action maintains trunk flexion and relative internal rotation of the tensioned leg. The subject was then instructed to straighten the knee and internally rotate the femur with overpressure on the anterolateral distal thigh with the ipsilateral hand. The therapist assisted to facilitate sciatic/tibial tract tension positions and if full neurodynamic elongation was well tolerated the patient was asked to add further trunk and cervical flexion, but only two subjects tolerated the additional trunk and cervical MLSS component in this sample with clinically tight hamstrings. Stretch duration was 5 seconds, 5 repetitions and 3 sets, paced with a mobile metronome set at 1 Hz (Android 1.2.4; 2012). Subjects were given 10 seconds rest between repetitions and two to three minutes between sets. Subjects were clearly instructed before and during the intervention sessions that the stretch procedure aimed to achieve maximal stretch tolerance and may involve some discomfort, however, if the stretch became too uncomfortable they should notify the tester immediately to reduce stretch intensity. Similarly, subjects were also instructed to report symptoms such as pins and needles, numbness or discomfort proximal to the ischial tuberosity.

Data analysis

Data analysis was performed using SPSS for windows. Exploratory data analysis and significance testing utilising the Shapiro-Wilk test suggested the pre-intervention data was normally distributed. Comparison of mean pre- to post-intervention PSLR and PKB ROM and SNRS ratings was carried out utilising 3-way repeated measures analysis of variance (ANOVA) with the factors session (1 / 2), side (ipsilateral / contralateral) and time (pre / post / post 1 hour). *Post hoc* analysis using Bonferroni correction was performed to determine differences between time points for analyses with a significant main effect of time. If assumption of sphericity was violated utilising Mauchley's test, the data was corrected with the Greenhous-Geisser equation. *Post hoc* correlation analysis was also performed utilising Pearson's correlation coefficient. Significance was set at alpha = 0.05 for all statistical tests.

RESULTS

Figure 3A shows the changes in PSLR following MLSS. MLSS significantly increased PSLR directly after the intervention, with no further increase 1 hr later (main effect of time: $p<0.001$). The effect of the unilateral MLSS intervention was evident in both legs, but greater in the ipsilateral leg compared to the contralateral leg (baseline to one hour post: +9±6°and +5±5° respectively, main effect of side: $p<0.001$). PSLR increased to a similar extent in both sessions (no significant session x time interaction effect), despite the fact that the effect of the first session was retained at the start of the second session 3 weeks later (main effect of session: $p<0.001$).

The effects of the MLSS intervention on PKB were mostly similar (**Figure 3B**), with significant main effects of time ($p<0.001$) and session ($p<0.001$). PKB increased from baseline to directly post ($p<0.001$), but there was no further significant increase one hour following the intervention. There was no significant effect of side, with similar effects on the ipsilateral leg and the contralateral leg (baseline to one hour post: +5±5° and +5±4° respectively). Post-hoc analysis also revealed moderate to strong negative correlation between pre-intervention ROM and the size of the ROM treatment effect for both PSLR ($r=-0.32$; $p<0.05$) and PKB immediately ($r=-0.56$; $p<0.001$), and one hour post intervention ($r=-0.53$; $p<0.001$; $r=-0.68$, $p<0.001$).

Subjective stretch intensity ratings were consistent with increased stretch tolerance following the MLSS intervention (**Table 1**). Post-intervention ratings taken at the pre-intervention maximal joint angle decreased for the PSLR (main effect of time: $p<0.001$), with a greater decrease in the ipsilateral side (main effect of side: $p<0.001$; time x side interaction effect: $p<0.05$). Conversely, ratings at the maximal joint angle achieved at each time point increased (main effect of time: $p<0.01$), again with a greater change in the ipsilateral side (main effect of side: NS; time x side interaction effect: $p<0.001$). PSLR stretch intensity ratings were higher in the second session compared to the first session (main effect of session: $p<0.001$).

PKB stretch intensity ratings at the pre-intervention joint angle followed a pattern similar to the PSLR ratings, with a significant decrease following the intervention (main effect of time: $p<0.001$), and higher ratings during the second session (main effect of session: $p<0.05$), but no significant main effect of side or time x side interaction effect (**Table 1**). No significant main effects of time, session, or side, and no interaction effects were observed for PKB stretch intensity ratings at the maximal joint angle achieved at each time point. No differences were observed in the responses for any parameters between participants who received the initial treatment on their skill leg or stance leg.

DISCUSSION

The purpose of the study was to assess potential segmental, extra-segmental and contra-lateral effects of applying a novel sciatic nerve neurodynamic tension technique, the MLSS, in healthy and active adults. We observed significant mean increases in ipsilateral and contralateral PSLR and PKB immediately and one hour post intervention, which is consistent with neurodynamic tension being an important neuro-modulator of muscle extensibility, and is further supported by the finding that these effects were significant after the first intervention session and maintained for three weeks. As to the authors' knowledge lasting extra-segmental and contralateral muscle extensibility gains from unilateral intervention have not previously been reported,[24,32,36] these results require verification through additional studies.

The pooled mean increase in PSLR from pre first intervention to one hour post second intervention of 15±6º represents a relative increase of 19±8%, utilising a total stretch time of 75 seconds per leg. This may be considered above average for PSLR gain in a hamstring extensibility study,[35] but achieved with considerably less total stretch time than previously reported.[28,34] For example, Ayala and colleagues[34] demonstrated a mean increase of 14º in PSLR utilising 540 seconds total weekly stretching over 12 weeks. Therefore the results of the current study provide a novel finding in that neurodynamic tension and stretch intensity appear to have a highly significant role in muscle extensibility,[18,30] compared to previous research which has purported total weekly stretch time as the most important parameter.[27-29] Thus MLSS intervention could potentially be utilised to make stretching practices more efficient in increasing hamstring extensibility by reducing total stretch time. However, further research is required as the current study utilised a narrow sample of young and healthy adults, whereas less robust populations, such as the elderly or those with irritable musculoskeletal conditions, may not tolerate application of higher levels of stretch intensity and neurodynamic tension, and thus be inappropriate for MLSS intervention.[26,36] Moreover, given the lack of blinding and cross-over design of the current study, a follow-up investigation to verify and compare the effects of MLSS intervention utilising single blinded RCT design is indicated.

Increased stretch tolerance from stretching is considered to occur through decreases in perception of stretch intensity for a common joint angle (SNRS Com) and potentially through increased tolerance to higher intensity stretch sensation (SNRS Max).[25,36] Consonant with the post intervention ROM changes, significant mean decreases in SNRS Com for ipsilateral and contralateral PSLR and PKB are consistent with modulation of stretch tolerance through neuronal desensitisation. Interestingly, PSLR but not PKB outcome measures demonstrated small but significant concomitant increase in SNRS Max, suggesting modulation of muscle extensibility by both neuronal desensitisation and increased tolerance of higher stretch intensity segmentally, but not extra-segmentally. This may also be a novel finding, as previous research has largely demonstrated constant maximal stretch intensity ratings pre-post stretching intervention.[31,36,57] The contrasting result of the present study may be due to the MLSS being a therapist-assisted technique eliciting greater amounts of neurodynamic elongation and stretch intensity.[16,17,31,63]

Previous investigations of neurodynamics and muscle extensibility have reported varying results. For example, Sullivan and colleagues[64] demonstrated focused hamstring muscle stretches were more effective than hamstring stretches in a stooped position that was consistent with elongation of the neuraxis.[16,63] However, the study by Sullivan and colleagues[64] reported maintenance of ankle plantar flexion and adoption of a low to moderate stretch intensity protocol, which may have elicited only neural unfolding, rather than nerve excursion, tension or strain,[16,63] with the stooped stretch, and subsequently provided relatively less stimulus to modulate stretch tolerance.[18,32] Nevertheless, the current study adds to more recent reports demonstrating efficacy of neurodynamic interventions in producing lasting increases of hamstring extensibility and stretch tolerance.[18,32-33]

The MLSS produces elongation of the sciatic/tibial nerve tract through a combination of ankle dorsiflexion and eversion, knee extension, hip internal rotation and trunk flexion, with likely resultant increases in nerve tension and strain.[16-17,63,65] Its potential advantage over other sciatic/tibial neurodynamic tension techniques, such as the slump[21] and long sit slump,[14,19] is that it is postulated to produce maximal tolerated sciatic/tibial nerve tract elongation, with relatively less flexion stress on lower lumbar spinal segments[66] through antagonistic rotation of the ilia around the sacrum in the hemi-sitting position.[67] Given unilateral sciatic-tibial sliding has previously demonstrated not to produce contralateral hamstring extensibility effects,[32] while comparison between a bilateral glider and unilateral tensioner was statistically non-significant,[18] further comparative studies of neurodynamic techniques, including the MLSS, on muscle extensibility and stretch tolerance is indicated.[33]

An interesting *post-hoc* finding of the current study was the significant moderate to strong inverse correlation between pre-intervention PSLR ROM and the magnitude of the ROM increase immediately ($r = -0.318$; $p < 0.05$) and one hour ($r = -0.526$; $p < 0.001$) post intervention, suggesting a potential 'diminishing returns' effect of the MLSS with respect to muscle extensibility. This is in contrast to the findings by Ayala and colleagues[34] who demonstrated no significant difference between subjects with and without tight hamstring tightness in response to 12 weeks of muscle stretching. Notwithstanding the large difference in total stretch time, a possible explanation of these seemingly differing results, is that the stretching protocol utilised by Ayala and colleagues,[34] through adoption of ankle dorsiflexion in two out of the four techniques, appear a combination of stretches which preferentially target muscle and neural tissue at moderate levels of stretch intensity whereas the MLSS preferentially targets the neural tissue at high stretch intensity.[16,28,30,63] Although the PKB measures in the current study were also significantly inversely correlated to pre-intervention ROM, tight rectus-femoris was not an inclusion criterion so this effect may have been due some subjects achieving full PKB ROM.

The specific neuronal mechanisms responsible for modulating stretch tolerance are yet to be fully elucidated. Large diameter proprioceptors have been implicated in modulating stretch tolerance through spinal gating,[24,36] but this mechanism may not have a significant lasting effect.[42-43] Furthermore, as muscle spindle and golgi organ receptors are considered absent outside the musculotendinous tissues,[38] and muscle stretching protocols have previously not demonstrated lasting extra-segmental nor contralateral effects, [24,32,36] this suggests the effects of the MLSS were probably not modulated primarily by proprioceptors.[25,68,69] However, this postulation is not inconsistent with the possibility that during stretching, low threshold proprioceptors and mechanoreceptors may sensitise high threshold receptors, such as mechanosensitive nociceptors, towards activation thresholds[38,41,46] through mechanisms such as the axon reflex and afferent convergence, as well as non-specificity of peripheral afferent neuropeptides to fibre type.[45,47] Conditioned learning and increased activation of the DNIS have also previously been implicated in increases of muscle stretch tolerance,[36] and is not inconsistent with the results the current study. Compared to previous muscle stretching research, the relatively higher levels of neurodynamic tension and stretch intensity with MLSS intervention may have acted as a stronger neural stimulus for subjects' learning to tolerate muscle stretch, which could explain the novel extra-segmental and contralateral effects. A future study utilising the MLSS which includes a muscle extensibility and stretch tolerance outcome measure proximal to the lumbar and lumbosacral plexus may provide further insights into the role of conditioned learning and DNIS activation, versus more local neuronal signalling at the spinal level, but fully elucidating these mechanisms may require corroboration with direct neurophysiological measures.

Desensitisation of mechanosensitive nociceptors has previously been implicated in modulation of muscle stretch tolerance and is also consistent with the results of the current study.[24,36] The extra-segmental and contralateral effects induced by the MLSS are also consonant with the proposition that increased stretch tolerance may be a form of nociceptive LTD,[43,55] akin to sensitisation as a form of LTP,[42,44,53] through recruitment of latent neuronal circuits.[48,51,54] Interestingly, A-delta but not A-beta afferent stimulation has been demonstrated to induce C-fibre LTD and de-potentiate LTP in the rat spinal dorsal horn, which provides a plausible mechanism for future investigations of stretch tolerance modulation in humans.[43]

Additionally, the sympathetic nervous system (SNS) and autonomic balance may also have a significant role in modulating stretch tolerance as sympathetic efferent and afferent fibres are considered to constitute a substantial proportion of lower limb peripheral nerve[70-72] and co-utilise noradrenaline and substance P, which are strongly implicated in nociceptor sensitivity and neuronal recruitment.[38,42,48,53,73] Moreover, SNS tracts possess complex anatomical and physiological configurations including multiple segments and bilateral midline crossing spinally. multi-segmental serial and parallel processing supra-spinally, and likely rapid autocrine and paracrine autonomic signalling.[74-77] Notwithstanding the aforementioned potential role of the SNS modulating stretch tolerance through neuronal desensitisation, significantly higher SNRS ratings in session two compared to session one for most of the outcome measures could be due to autonomic modulation of stretch tolerance through attenuation of 'threat' perception during stretch.[78] However, some contrasting findings, predominantly for the PKB data, further supports a difference between segmental and extra-segmental stretch tolerance modulation, but the potential of type 2 error, due to small sample sizes, should also be considered. Moreover, given modulation of autonomic balance is a primary mechanism proposed to underlie yoga efficacy[79] and the likely overlap between yoga postures and neurodynamic tension positions,[80] further investigation of the role of the autonomic nervous system and its role in muscle extensibility, neurodynamics and HSI, is warranted.[81]

There were several limitations to the current study. Although there is in-vivo evidence demonstrating validity in administering targeted nerve excursion and strain through neurodynamics,[16,82] there is an absence of studies which demonstrate differentiation between muscle and nerve biomechanics with neurodynamic intervention, obviating a need for further research to improve content and construct validity.[83] Another major limitation of the current study, due to resource limitations at MSc study level, was that all measurements and intervention were performed by the same experienced musculoskeletal physiotherapist, raising the internal bias of the study.[84] Therefore verification of the study's results in a single blinded RCT is indicated. Another limitation was that the PKB procedure utilised has not been validated for rectus-femoris muscle extensibility, despite common clinical utilisation. Nevertheless, the high consonance between mean PKB ROM and SNRS changes suggests high measurement error was probably not a significant factor. Given the PKB procedure is simple and efficient for a single examiner, future investigation of its validity is warranted. An additional potential source of bias was not testing SNRS Com measures when post intervention ROM was less than pre-intervention, which avoided moving the limb beyond the maximally tolerated point. However, this only occurred with PSLR measures in one subject in the first intervention session, and with several PKB measures in subjects who had full PKB ROM, and is not considered to have significantly affected the results. Lastly, the study was limited to healthy and active adults with clinically tight hamstrings recruited from a university population, resulting in a relatively young and robust sample. Notwithstanding due care required in applying neurodynamic tension techniques in less robust populations, investigation of the effects of the MLSS in a slightly older sample, or those with past HSI, is indicated.[16]

CONCLUSIONS

Application of a novel sciatic-tibial neurodynamic tension technique, the MLSS, produced significant and lasting segmental, extra-segmental and contralateral increases of muscle extensibility and stretch tolerance in a healthy, active sample with clinically tight hamstrings. Additional studies are indicated to verify the findings and further investigate potential MLSS effects in different samples.

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DISCLOSURE

The authors have nothing to disclose.

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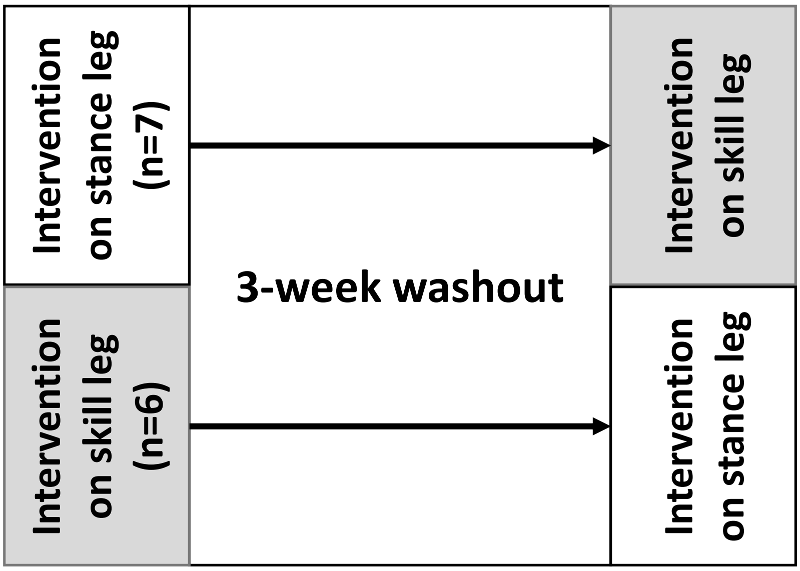

Figure 1. During session 1, half the subjects received the MLSS intervention on the stance leg and the other half of the subjects received the intervention on the skill leg. Measurements were taken pre-, directly post, and one hour post-intervention. Following a 3-week washout period the intervention was repeated on the other leg.

"Effects of a Novel Neurodynamic Tension Technique on muscle extensibility And Stretch Tolerance: A Counterbalanced Cross-Over Study" by Pietrzak M, Vollaard N BJ
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2A

2B.

2C

2D

Figure 2. Modified long sit slump (MLSS). Start position (top row; **2A &2B**) and end position (bottom row; **2C & 2D**). The subject starts hemi-sitting with the stretched limb on the plinth and the knee flexed. The subject uses their opposite hand to reach forward and hold the lateral border of the foot, placing it in dorsiflexion and eversion. They are then instructed to extend the knee and internally rotate the femur. The therapist assists to facilitate neurodynamic tension positions, and if the position is well tolerated, the subject is facilitated to add further trunk and cervical flexion.

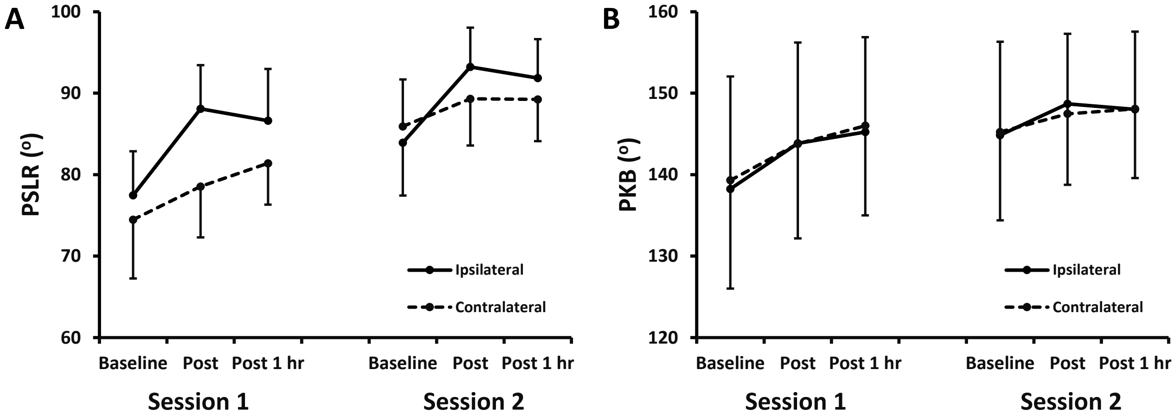

Figure 3: Effect of the MLSS intervention on: A) passive straight leg raise (PSLR), and B) prone knee bend (PKB). The intervention was performed on either the stance leg (n=6) or skill leg (n=7) in session 1, and on the other leg 3 weeks later in a counterbalanced manner. Main effects for PSLR: time p<0.001, side p<0.001, session p<0.001. Main effects for PKB: time p<0.001, side NS, session p<0.001.

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TABLE 1. Mean stretch intensity ratings on a simple numerical rating scale (SNRS) from 0 (‘no muscle stretch’) to 10 (‘the worst muscle stretch imaginable’). ‘Com’ represents the score taken at the pre-intervention joint angle for that session, whereas ‘Max’ represents the score at maximal stretch tolerance for each time-point. Effect of time: * p<0.05, ** p<0.01, *** p<0.001 compared to pre within the session; effect of side: †† p<0.01 compared to ipsilateral side; effect of session: # p<0.05, ### p<0.001 compared to session 1. Values shown are mean±SD.

		Session 1			**Session 2**		
		Pre	**Post**	**Post 1 hour**	**Pre**	**Post**	**Post 1 hour**
Ipsilateral PSLR	Com	7.4±0.8	5.1±1.4***	5.4±1.5***	8.1±0.9###	6.2±1.0***###	6.9±1.3***###
	Max		7.9±1.0**	8.0±1.2**		8.7±0.6**###	9.0±0.8**###
Contralateral PSLR	Com	7.8±0.8†	6.3±0.9**††	5.4±1.4**††	8.4±1.1†###	7.1±0.9**††	7.3±1.1**††
	Max		7.5±0.7	8.0±0.9		8.6±0.7###	8.7±0.9###
Ipsilateral PKB	Com	7.2±1.1	5.8±1.8***	5.6±1.7***	7.6±1.2	5.6±1.8***#	6.4±1.6***#
	Max		7.2±1.4	7.4±1.4		7.2±1.5	7.6±1.3
Contralateral PKB	Com	7.1±1.6	6.0±1.7***	5.4±1.6***	7.8±1.0	6.6±1.4***#	6.5±1.7***#
	Max		7.3±1.4	7.2±1.6		7.7±1.4	7.6±1.7