Mechanosensitivity of the Lower Extremity Nervous System During Straight-Leg Raise Neurodynamic Testing in Healthy Individuals

Clinical neurological examinations are an integral part of clinical decision making for determining neural involvement in individuals with altered physical function and activity participation. One aspect of a standard neurological examination involves assessing the sensitivity of peripheral nerves to limb movement, termed mechanosensitivity. Mechanosensitivity is thought to be a normal protective mechanism that allows the nerves to respond to the mechanical stresses imposed upon them during movement.25

Neurodynamic tests are used to assess the nervous system’s mechanosensitivity through monitoring the response to movements that are known to alter the mechanical stresses acting on the nervous system. The most common lower quarter neurodynamic test is the passive straight-leg raise (SLR) test.5,26 The basic SLR test consists of the tester performing passive hip flexion, with the patient in a supine position and the knee held in full extension.9

A recent systematic review of SLR testing indicated a lack of standardization, including the use of various criteria for determining the test end point.4 The authors of this review reintroduced standardized methodology proposed by Breig and Troup in 1979, including the use of the first onset of pain as the end point during the SLR test.31 Despite these recommendations, alternative end points, such as maximally tolerated symptom, are still utilized.7 Because SLR testing is performed in both symptomatic and asymptomatic populations, differing interpretations of test results are possible.7,8

STUDY DESIGN: Cross-sectional, observational study.

OBJECTIVES: To explore how ankle position affects lower extremity neurodynamic testing.

BACKGROUND: Upper extremity limb movements that increase neural loading create a protective muscle action of the upper trapezius, resulting in shoulder girdle elevation during neurodynamic testing. A similar mechanism has been suggested in the lower extremities.

METHODS: Twenty healthy subjects without low back pain participated in this study. Hip flexion and surface electromyographic measures were taken and compared at the onset of symptoms (P1) and at the point of maximally tolerated symptoms (P2) during straight-leg raise tests performed with ankle dorsiflexion (DF-SLR) and plantar flexion (PF-SLR).

RESULTS: Hip flexion was reduced during DF-SLR by a mean ± SD of 5.5° ± 6.6° at P1 (P = .001) and 10.1° ± 9.7° at P2 (P < .001), compared to PF-SLR. DF-SLR induced distal muscle activation and broader proximal muscle contractions at P1 compared to PF-SLR.

CONCLUSION: These findings support the hypothesis that addition of ankle dorsiflexion during straight-leg raise testing induces earlier distal muscle activation and reduces hip flexion motion. The straight-leg test, performed to the onset of symptoms (P1) and with sensitizing maneuvers, allows for identification of meaningful differences in test outcomes and is an appropriate end point for lower extremity neurodynamic testing. J Orthop Sports Phys Ther 2009;39(11):780-790. doi:10.2519/jospt.2009.3002

KEY WORDS: neural provocation test, neural tension, sciatic nerve, sensitizing maneuvers
asymptomatic limbs, it is important to know the normal healthy response of the nervous system at both end points to support this recommendation.

Interpretations of neurodynamic examination findings are based primarily on expert consensus. The proposed interpretations of a “positive” test include considerations for whether the test (1) reproduces the patient’s symptoms, (2) identifies asymmetry between limbs or significant deviation from norm, and (3) induces changes in symptoms by distant joint movement, also called “sensitizing movements.” The third consideration is critical to identify the nervous system as the source of limitations to movement and is termed “structural differentiation.” Sensitizing movements involve adding a limb movement distant to the location of symptoms that would affect the neural structures in the limb without affecting the nonneural tissue local to the area of symptoms.

Ankle dorsiflexion is a common sensitizing maneuver for SLR testing. Studies in rats and dogs have demonstrated increased strain (elongation) in the sciatic nerve at the proximal thigh when ankle dorsiflexion was added to SLR testing. Further support for the use of ankle dorsiflexion as a sensitizing maneuver is provided by findings from a cadaveric study, in which prepositioning the ankle in dorsiflexion created distal movement in the tibial nerve at the knee and ankle. Clinically, prepositioning the ankle in dorsiflexion leads to a reduction of hip range of motion during SLR testing, when taken to maximal resistance to hip flexion in people with low back pain and healthy individuals.

Neurodynamic testing can also produce increases in local muscle tone. SLR testing without ankle dorsiflexion has been shown to induce hamstring and gluteal muscle activity when the hip flexion is held at the maximally tolerated position. However, this study was performed on a small number of subjects and statistical analysis was not performed. In another study, an increase in hamstring muscle activity was shown at the maximum hip flexion range (determined by the tester) in contrast to relative electrical silence through the rest of the range in healthy individuals. This mean ± SD increased activation was only 3% ± 1% of maximal voluntary contraction and was not a statistically significant increase. Prepositioning in ankle dorsiflexion induces hamstring muscle activation earlier in hip flexion range during SLR testing in healthy individuals.

This study also did not include statistical analysis. Muscle activity provoked during the sensitized SLR test is thought to provide a protective mechanism to restrict further movement and to help prevent overstretch nerve injuries. This is consistent with findings in the upper limb, where passive neurodynamic testing has been shown to induce muscle activity from adjacent musculature.

No study to date has simultaneously explored the differences in range of motion, symptoms, and muscle responses for SLR neurodynamic testing at both the onset and maximally tolerated symptoms in healthy individuals. In addition, no study has provided statistical analysis of both proximal/distal and flexor/extensor muscle activity during SLR neurodynamic testing. It is important to understand the specific effects of sensitizing maneuvers at each of these testing end points in normal asymptomatic individuals to guide clinical decision making and to help establish standardized testing methodology in symptomatic populations. The same test end point should be utilized in the uninvolved and involved limbs in people with nerve injuries, which necessitates understanding the normal response of the nervous system on the uninvolved limb.

In this study we attempted to elucidate the specific effects of the ankle dorsiflexion sensitizing maneuvers on the mechanism of lower extremity posterior neural structures in healthy individuals. The aims were to determine the amount of hip motion and muscle activity during 2 versions of the SLR (including ankle dorsiflexion sensitization) at 2 end-points predefined as the onset of symptoms (P1) and maximally tolerated symptoms (P2). Additionally, we analyzed the reliability of repeated SLR testing.

**METHODS**

This cross-sectional study included 20 healthy participants recruited from local medical and academic communities. Exclusion criteria included low back or lower extremity pain lasting longer than 3 consecutive days in the past 6 months, peripheral neuropathy, diabetes mellitus, complex regional pain syndrome, lumbar spine surgeries, chemical dependence or alcohol abuse, a history of lower extremity nerve trauma, or chemotherapy in the past year. Participants had to meet flexibility requirements of hip flexion of 90° or more with the knee flexed, full knee extension, ankle dorsiflexion of 0° or more, and plantar flexion of at least 30°. The Institutional Review Boards at University of California, San Francisco, and San Francisco State University, and the Clinical Research Center’s Advisory Committee at University of California, San Francisco approved this study. Written, informed consent was obtained from the participants prior to testing. All participants attended a single clinical assessment session. A subset of subjects (n = 5) returned within 1 to 2 weeks for an identical clinical assessment session for reliability testing. One examiner (B.B.) performed all physical examinations.

**Clinical Assessment Session**

Participants completed a medical history questionnaire. In addition, the subjects were instructed in the use of a visual symptom-reporting card, which included a body chart, an 11-point pain scale, and a list of qualitative descriptors adapted from the McGill Pain Questionnaire. The 11-point numeric pain rating scale had the anchors of 0 (“no pain”) and 10 (“worst pain possible”). This type of scale has good reliability and validity across multiple ages and races.
SLR Testing
The subject was positioned in supine, with a 2.5-cm-thick foam head support as the standardized position for neurodynamic SLR testing (FIGURE 1). Additional pillows were provided if requested. A blood pressure cuff bladder was centered under the subject’s low back and was inflated to 40 mmHg, just prior to SLR testing. Changes in cuff pressure were documented at end of movement, during SLR testing, as a gross assessment of change in lumbar spine lordosis. Comparisons were made between the SLR tests performed with the ankle in either dorsiflexion or plantar flexion. The subject’s right ankle was placed in an APU PRAFO ankle brace, with outrigger bar and extra straps (Anatomical Concepts, Inc, Poland, OH) to maintain a fixed ankle position in either plantar flexion (30°) or in neutral (0°) dorsiflexion. The SLR performed with the ankle in 30° of plantar flexion (PF-SLR) was considered the base or reference test, and the SLR performed with the ankle in neutral position (DF-SLR) was considered the sensitized SLR test (FIGURE 1A).

Electromyography (EMG) Setup
Standard 1-cm circular bipolar Ag/AgCl surface EMG electrodes (Noraxon USA, Inc, Scottsdale, AZ), with an interelectrode distance of 2 cm, were placed over the gluteus maximus, semitendinosus, biceps femoris, medial gastrocnemius, soleus, rectus femoris, vastus medialis, and tibialis anterior muscles of the right lower extremity (FIGURE 1B). Electrode placement was in accordance with surface EMG for noninvasive assessment of muscles (SENIAM) guidelines. A single reference electrode was placed over the right patella. Skin preparation included cleaning and vigorous rubbing with an alcohol-soaked gauze pad. Three repetitions of 5-second maximal voluntary isometric muscle contractions (MVC) were performed against manually provided resistance, with the subject in supine, for purposes of EMG signal normalization. During MVC testing, the limb was supported on pillows, if appropriate, and stabilized manually immediately proximal to the joint being tested. Similar to other studies, the calf musculature was tested in a neutral ankle position, the quadriceps and hamstrings were tested with the knee in approximately 30° flexion, and the gluteal musculature was tested in approximately neutral hip flexion. MVC procedures included instructions to either push or pull against the examiner’s resistance and to not let the examiner move the limb. EMG signals were amplified (×2000) and acquired with a bandwidth frequency of 50 to 500 Hz, and a sampling rate of 2000 Hz, using a TeleMyo 900 System, NorBNC and A/D USB converter using MRXP Master Package software, Version 1.06.21 (Noraxon USA, Inc).

Goniometer Setup
Twin-axis electromyogoniometers (Noraxon USA, Inc) were placed laterally across the hip and knee joints to measure sagittal and coronal plane motion (FIGURE 1B). Coronal plane motions were used to evaluate that neutral hip abduction and adduction were maintained during testing. The hip goniometer was placed with the proximal end parallel to the subject’s torso adjacent to the iliac crest and the distal end on the lateral thigh, in line with the lateral femoral condyle. The knee goniometer was placed with the proximal end aligned with the greater trochanter of the femur and the distal end aligned with the lateral malleolus. Care was taken to ensure that the middle of the goniometer coil was centered over the axis of rotation for each joint. Goniometers were held in place with double-sided tape and custom-made neoprene straps (FIGURE 1B). A wall placard provided the tester with visual input of 10° increments and was placed so that the origin was aligned with the subject’s right greater trochanter. The participants were given a custom-built handheld electronic button (trigger), which was held in the dominant hand with both hands resting on the abdomen (FIGURE 1B). Goniometer and trigger data were acquired at 2000 Hz and synchronized with the EMG data, using the NorBNC and A/D USB converter (Noraxon USA, Inc).

Testing Procedure
One instructional trial was performed on the left lower extremity prior to formal testing of the right lower extremity. For the right limb, a total of 4 SLR tests were performed, with 2 trials assigned in a random order for each ankle position. The order was randomized to minimize the effects of test order on the SLR outcomes. A metronome and wall placard were used to facilitate consistent SLR testing speed of approximately 5°/s (FIGURE 1B). The tester placed the subject’s knee in full extension (defined as end range resistance) without lifting the
thigh off of the mat, and the subject was instructed to indicate this start position ("start") by pressing the trigger 3 times. While holding the knee in full extension, the subject’s hip was moved passively into hip flexion, while manually avoiding rotation, abduction, or adduction of the femur. The subject indicated the onset of symptoms (P1) and the symptom limit (P2) during the SLR by pressing the handheld trigger. Specifically, the subject was instructed to indicate "the moment you feel the first onset of any symptoms" (P1) and when "your symptoms become too intense to continue and feel you cannot tolerate any further movement" (P2). The motion was stopped at P2, and this position was held for 5 seconds, before the limb was returned to a resting position on the plinth. Two-minute rests were given between each SLR trial. Subjects were asked to report symptom location, intensity, and quality at the start position, at P1 (delayed reporting until immediately after P2 because motion was not stopped at this position), at P2, and then after a 2-minute rest.

**Data Processing**

Surface EMG signals were converted using a root-mean-squared (RMS) formula, with a 50-millisecond interval. Mean voltage for EMG and degrees for hip range of motion were obtained for a 100-millisecond window centered on each of the following 3 time points: start, P1, and P2. For each muscle, MVC measurements were averaged from the center 3-second window of each of 3 repeated MVC tests. SLR testing surface EMG values were converted into percent MVC for each muscle. A "triggered muscle response" was defined as an increase in EMG activity (expressed as percent MVC) of at least a 1.5-fold above the supine-lying, resting levels (taken lying supine prior to establishing the start position). For example, if there was 3.0% MVC activity of the hamstring muscle in resting, the muscle was considered activated (triggered muscle response) at 4.5% MVC during SLR testing.

### Goniometer Reliability Testing

The goniometers were attached to a rigid, wood-hinged model to test the reliability and validity of measurements compared to fixed metal angles of 0°, 30°, 45°, 60°, and 90°. Further reliability testing was performed on a subset of 5 participants, by performing 10 repeated SLR tests to arbitrarily, but consistently, predetermined hip flexion positions. Specifically, the beam from a laser level, placed on a fixed wooden surface, was aimed horizontally across the room at an angle perpendicular to the subject’s limb and at an arbitrary height within the subject’s symptom-free hip flexion range of motion. A second tester pressed the trigger when the subject’s limb blocked the laser beam, and the hip flexion angle was then measured.

### Statistical Analysis

All statistical analyses were performed using SPSS software, Version 14.0 (SPSS Inc, Chicago, IL). Descriptive statistics were used to describe the mean ± SD for all variables except frequency descriptive statistics for symptom quality and loca-
tion, which are reported as percentages. Repeated-measures, general linear models were used for within-condition differences between the rest, start, P1, and P2 positions for EMG, range-of-motion, and symptom intensity data. Between-test comparisons (DF-SLR to PF-SLR) were made using paired t tests. The general linear model calculations were adjusted due to nonsphericity using a Greenhouse-Geisser correction. Pearson correlation coefficients were calculated to assess the relationship between the lumbar pressure cuff measure and hip flexion range of motion at P2. An intraclass correlation coefficient (ICC) was used for repeated-measures reliability analysis and reported with the 95% confidence interval (CI). The minimal detectable change for hip flexion range of motion was calculated using the standard error of the measurement.13 Alpha was set at .05. Significance was set at P<.05.

RESULTS

The average ± SD age of the 20 participants was 50.4 ± 12.0 years (range, 25-63 years) and included 14 women and 6 men. Height was 1.7 ± 0.1 m, body mass was 71.2 ± 24.8 kg, and body mass index (BMI) was 25.9 ± 8.8 kg/m².

SLR Neurodynamic Testing

The average ± SD for angular velocity of the PF-SLR was 3.0°/s ± 1.0°/s and of the DF-SLR was 2.8°/s ± 0.9°/s (P = .045).

Symptom Intensity

As expected, the mean ± SD symptom intensity at P1 and P2 was increased above resting levels for both versions of the SLR (P<.001) (FIGURE 2A). There was also an increased symptom intensity from P1 to P2 for both versions of the SLR (P<.001). During PF-SLR the mean ± SD symptom intensity went from 0.1 ± 0.3 at the start position to 2.5 ± 1.6 at P1 and to 6.6 ± 2.1 at P2. In contrast, during DF-SLR, the mean intensity went from 0.4 ± 0.9 at the start position to 3.2 ± 1.9 at P1 and to 7.0 ± 1.8 at P2. The mean intensity at P1 was significantly higher by 0.7 ± 0.9 points during the DF-SLR compared to PF-SLR (P = .002). There was no difference in mean intensity between PF-SLR and DF-SLR at the start position or at P2. In general, symptom intensity was not correlated with muscle activity (percent MVC), except at the start position. Muscle activity (percent MVC) and symptom intensity were significantly correlated at the start position during the PF-SLR for the semitendinosus (r = 0.56, P = .013), anterior tibialis (r = 0.53, P = .021), and vastus medialis (r = 0.71, P = .001), and during DF-SLR for the semitendinosus (r = 0.49, P = .032). At P1 muscle activity was significantly correlated with symptoms during PF-SLR for the gluteus maximus (r = 0.48, P = .039). There were no other significant correlations between muscle activity and symptom intensity at either predefined point in either SLR test.

Goniometric Validity and Reliability Testing

Repeated goniometric measures on the wooden hinged model were a mean ± SD of 0.3° ± 0.2° for the known 0°
angle, 31.3° ± 0.5° for the known 30° angle, 47.8° ± 0.7° for the known 45° angle, 64.1° ± 0.8° for the known 60° angle, and 95.9° ± 1.3° for the known 90° angle. Reliability (ICC) of repeated goniometric measures in the sagittal and coronal plane on the wooden hinged model was 1.00 (95% CI: 1.00, 1.00). Using a subset of 5 participants, the range of variability with repeated goniometric testing of hip flexion to arbitrary but consistent positions in the symptom-free range (up to a maximum of 40°) was from 1.0° ± 0.3° to 2.4° ± 0.7°, with an ICC of 1.00 (95% CI: 0.99, 1.00). The minimal detectable change for hip flexion range of motion was 0.4° using this methodology.

Range of Motion

ICC, for hip flexion range of motion between trials were 0.87 (95% CI: 0.69, 0.95) for PF-SLR at P1, 0.96 (95% CI: 0.91, 0.99) for PF-SLR at P2, 0.78 (95% CI: 0.50, 0.91) for DF-SLR at P1, and 0.88 (95% CI: 0.73, 0.95) for DF-SLR at P2. The hip range of motion to P1 and to P2 during the SLR test was greater than the start position for both DF-SLR and PF-SLR (P<.001) (FIGURE 2B). In addition, hip range of motion was significantly greater at P2 than P1 for both PF-SLR and DF-SLR (P<.001). There was 13.9% less hip flexion ROM at P1 during DF-SLR compared to PF-SLR, with a 95% CI from 2.4° to 8.6° (P = .001) (FIGURE 2B). At P2 there was 14.9% less hip flexion ROM in DF-SLR compared to PF-SLR, with a 95% CI from 2.4° to 8.6° (P<.001). There was no difference in hip abduction/adduction between PF-SLR and DF-SLR at P1 (P = .318) or at P2 (P = .572). There was no difference in knee flexion/extension between PF-SLR and DF-SLR at P1 (P = .124) or at P2 (P = .260). There was no difference in knee coronal plane positioning between PF-SLR and DF-SLR at P1 (P = .648) or at P2 (P = .498). Repeated testing between multiple testing sessions (mean ± SD interval of 10.4 ± 4.3 days) performed on a subset of 5 subjects had an ICC of 0.87 (95% CI: 0.68, 0.95) for hip flexion ROM measurement.

Muscle Activation

The coefficient of variation for repeated MVC trials was 14.23%, which supported use of averaging of the 3 trials. There was relative EMG silence of the muscles until muscle activation was triggered late in the hip range of motion (FIGURE 3). During PF-SLR, rectus femoris became activated at P1 (P = .021) (TABLE). When the PF-SLR was taken to P2, a slightly different pattern of muscle activation was seen. The rectus femoris remained activated (P = .025), while additional muscle activation was seen in glutaeus maximus (P = .045), vastus medialis (P = .010), soleus (P = .013), medial gastrocnemius (P = .018), biceps femoris (P = .049), and tibialis anterior (P = .037).

The addition of ankle dorsiflexion created a different pattern of muscle activation (TABLE). During DF-SLR, muscle activation criteria was met for the soleus (P = .015), semitendinosus (P = .005), tibialis anterior (P = .003), and vastus medialis (P = .027) at P1. When taken to P2 during DF-SLR, these 4 muscles remained activated (P = .010, P = .021, P = .001, P = .014), and the medial gastrocnemius (P = .003) and rectus femoris (P = .024) were triggered. Between-test comparisons identified a significantly greater soleus and tibialis anterior muscle activation at P1 during DF-SLR compared to PF-SLR (P = .042 and P = .008). At P2, there was a significantly higher activation of the tibialis anterior and the vastus medialis during DF-SLR compared to PF-SLR (P = .008 and P = .028).

Symptom Location

Eighty-five percent of the subjects had no symptoms at the start position in PF-SLR and 75% in DF-SLR (FIGURE 4). For those subjects who reported symptoms in

<table>
<thead>
<tr>
<th>TABLE</th>
<th>Muscle Activation Pattern*</th>
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<tbody>
<tr>
<td>Muscle</td>
<td>Resting</td>
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<td>Soleus</td>
<td>6.8 ± 2.8</td>
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<tr>
<td>Medial gastrocnemius</td>
<td>5.2 ± 1.5</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>2.7 ± 1.1</td>
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<tr>
<td>Vastus medialis</td>
<td>7.2 ± 4.1</td>
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<tr>
<td>Rectus femoris</td>
<td>6.8 ± 3.6</td>
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<tr>
<td>Semitendinosus</td>
<td>3.8 ± 1.3</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>4.5 ± 1.5</td>
</tr>
<tr>
<td>Gluteus maximus</td>
<td>12.1 ± 4.5</td>
</tr>
</tbody>
</table>

Abbreviations: DF, dorsiflexion; MVC, maximal voluntary isometric muscle contraction; P1, onset of symptoms; P2, point of maximally tolerated symptoms; PF, plantar flexion; SLR, straight-leg raise.

* Values are mean ± SD percent MVC.

† Statistically significant increase (P<.05) above resting levels for general linear model of repeated measures for within-test differences.

‡ Statistically significant difference (P<.05) between PF-SLR and DF-SLR tests, using paired t test comparison for start and P1 and P2.
During PF-SLR, the right posterior leg was more frequent, followed by tight/tension (50% at P1 and 40% at P2), and third most common was ache (10% at P1 and 15% at P2). Pain and numbness were reported infrequently during SLR, and no subjects reported tingling or pins/needles. After 2 minutes of rest following the SLR test, 90% of the subjects reported no symptoms following PF-SLR and 70% reported no symptoms following DF-SLR. The symptoms that remained after PF-SLR were most commonly ache (15%) and stretch (10%), and after DF-SLR were most commonly ache (15%) and stretch (10%).

**Lumbar Spine Pressure Cuff Measure**
Repeated-measures reliability (ICC) of the lumbar pressure cuff measurements taken at P2 was 0.87 (95% CI: 0.69, 0.95) for PF-SLR and 0.91 (95% CI: 0.78, 0.96) for DF-SLR. Lumbar pressure cuff measurements increased from 40 mmHg at start position to a mean ± SD of 67.6 ± 11.5 mmHg at P2 during PF-SLR and 66.5 ± 12.6 mmHg at P2 during DF-SLR. The pressure in the cuff at P2 was not significantly different between PF-SLR and DF-SLR (P = .298). Pearson correlations between the lumbar pressure cuff measurement and hip flexion range of motion at P2 were 0.77 (P < .001) for the PF-SLR and 0.79 (P < .001) for the DF-SLR.

**DISCUSSION**

This study further supports the concept that ankle positions may be used as sensitizing maneuvers to the base SLR test. The quality and location of symptoms were altered and a broader muscular response was triggered with the addition of the sensitizing maneuver of ankle dorsiflexion. The higher symptom intensity that we observed in healthy subjects at P1 during DF-SLR compared to PF-SLR was statistically significant but did not meet the 2-point threshold for clinical significance and is therefore not a meaningful difference. Hip flexion range of motion was reduced during the dorsiflexion version of the SLR test at both the
onset and maximally tolerated symptoms. The lower bound of the CI exceeded the minimal detectable change, indicating that this difference was a real difference in range. Our results are consistent with a previous study that identified a significant 9° reduction in hip range of motion by the addition of ankle dorsiflexion. We hypothesize that the SLR with ankle plantar flexion does not preload the sciatic, tibial, and plantar nerves, thus allowing the hip greater range of flexion before the nerve complex undergoes sufficient mechanical stress to trigger a symptomatic or motor response. Furthermore, we hypothesize that the SLR with ankle dorsiflexion triggers an earlier restriction to movement during SLR testing through preloading of these neural structures. This is supported by previous findings of increased mechanical stress and strain on the sciatic, tibial, and plantar nerves during ankle dorsiflexion.

Changes in muscle tone were expected to be small during SLR testing, and an appropriate threshold was necessary to determine meaningful differences. A previous study of an upper limb neural provocation test had documented a statistically significant increase of approximately 1.5 times the muscle activity compared to resting levels in upper trapezius muscle. We used this 50% increase in muscle electrical activity over resting muscle tone as a conservative threshold to define “muscle activation.” This criterion was more stringent than previously utilized thresholds of greater than 1, 2, or 3 standard deviations above the resting mean electrical activity. In fact, the criterion used in our study led to a higher threshold for activation by an average of 1.5% MVC compared to the previously utilized methodology. It was expected that stretch-induced increases in muscle tone would be no greater than 25% MVC. This was indeed the case during the passive SLR test for all muscles measured in this study.

As expected, progression of the end point of the SLR from P1 to P2 triggered EMG activity in more muscles than had been activated at P1. In the PF-SLR, moving to P2 triggered activity in gluteus maximus, vastus medialis, biceps femoris, tibialis anterior, soleus, and gastrocnemius, in addition to the rectus femoris, which was active at P1. Progression of the SLR from P1 to P2 triggered cocontractions of antagonist muscle groups, as has been documented in an upper limb neurodynamic test. Additionally, although the intensity of symptoms increased from P1 to P2, we did not observe a correlation between the increase in symptom intensity and the increase in muscle activation. This is in agreement with the work of Balster and Jull in an upper limb neurodynamic test of healthy subjects. In contrast, van der Heide et al documented a correlation between the onset of pain and muscle activity in an upper limb neurodynamic test in healthy subjects. In this latter study, however, the correlation was determined using only the subjects who experienced pain consistently. It is possible that progression of the SLR from the first onset of symptoms to maximally tolerated symptoms results in a global attempt to stop the movement by stabilizing the joints with cocontractions, as hypothesized by van der Heide and colleagues in their study of the response of biceps brachii, triceps brachii, and trapezius in an upper limb neurodynamic test.

The addition of dorsiflexion to the
base SLR induced muscle activation in both the soleus and the tibialis anterior at P1. The distal muscle activation was not seen at P1 in the PF-SLR. This muscle response was not likely due to volitional changes in muscle activation, as the subjects were instructed to remain relaxed throughout the SLR testing and were masked from viewing the EMG recordings. Distal muscle activity at the first onset of symptoms in the DF-SLR leads us to hypothesize that this is a protective reflexive mechanism of the local muscle to stop further stress and strain of the nerves by limiting further motion. Such a local protective response has been demonstrated in the upper limb, where-in neurodynamic tests that elongate the brachial plexus result in increased surface EMG activity of the upper trapezius muscle and increased contractile force of muscles that elevate the shoulder.3-6 Our study demonstrated that the mass muscle activation pattern presents earlier in the SLR if the limb is in ankle dorsiflexion.

As expected, during the SLR, symptoms reported by healthy subjects differed from those reported previously by people with lower limb radicular pain.6 In our study, a few subjects described minimal dull, ache, sore, or tenderness in the posterior hip, thigh, or leg at the start position, in which the knee was moved into full extension. It is likely that elongation of the soft tissue in the posterior limb provoked the symptoms. During the SLR testing, the most frequent symptoms reported were stretch or tension in the posterior thigh or leg. The addition of ankle dorsiflexion to the base SLR provoked more tension, tightness, and burning, and more distal location of symptoms. In these healthy subjects, pain and numbness were reported infrequently (≤10%). In contrast, SLR testing in people with lower limb radicular pain has been found to provoke reports of “pain” in 83% of the symptomatic limbs at a mean of only 58° of hip flexion.6 This study also identified the frequent report of deep symptoms that may follow a myotomal or sclerotomal pattern.6

Some researchers have proposed the first onset of pelvic movement as an end point for SLR testing when used as a lower extremity flexibility assessment,6,5,10,37 but it is unclear if this is an appropriate end point for SLR neurodynamic testing. One research study indicated that pelvic movement occurred simultaneously with hip flexion during the SLR test even when the pelvis was strapped to the table.4 Another study found that pelvic motion began after the first 10° and that lumbar lordosis began to decrease after 30° of hip flexion motion during the SLR.5 In our study suggests that, as hip range of motion increases during SLR, the pressure under the lumbar spine also increases. We found excellent reliability of this measurement during SLR testing and a strong relationship between hip range of motion and the amount of pressure measured under the low back at P2 (Pearson r = 0.77-0.79). Further research is necessary to determine whether the increase in pressure in the SLR is due to movement of the lumbar spine and pelvis, or to changes in the muscle activity of the erector spinae in the region of the blood pressure cuff. Regardless of the mechanism, it appears that movement of the pelvis or lumbar spine would not be an appropriate end point for the SLR test when used as a neurodynamic test.

What end point should be used for stopping neurodynamic tests of asymptomatic limbs that allows for both sufficient information gathering and protection of the person being tested? Our study has shown excellent reliability of hip flexion measurements at the onset of symptoms (P1) on the same day (ICC = 0.78-0.96) and repeated testing in subsequent weeks in subjects with healthy nervous systems (ICC = 0.87). We found that the altered ankle position of only 30° between the PF-SLR and DF-SLR created differences in hip ROM, symptom intensity, and muscle activation that were measurable at P1. In our study, taking the test to the maximally tolerated position (P2) did not provide additional clinically relevant information. For example, the muscle response was widespread and not specific to ankle position, symptom intensity was not discriminatory for ankle position, and P1 had already allowed for identifying reductions in range of hip flexion motion with ankle dorsiflexion. Although testing to P2 had excellent repeatability, it carries with it risks, such as overstretch and further irritation of the nervous system, particularly when used with people in pain or with suspected nerve injuries.

One of the limitations to our study is extrapolating this information to people who have pain. Our findings are from people with healthy nervous systems and provide guidelines for expectations in the asymptomatic limb of those patients with pain down 1 lower extremity. The presence of pain or injury in the injured limb may induce a different response in the asymptomatic limb. Therefore, care should be taken in extrapolating the outcomes from this study to individuals who have pain, even when testing their asymptomatic limb. Future research should consider the influence of neuropathic and nonneuropathic pain on the outcome of the SLR in the asymptomatic limb.

Limitations of application of our findings to the clinical setting also include the precise measurement tools and standardized protocols required to determine small range-of-motion differences between PF-SLR and DF-SLR. The equipment used in this study is not readily available to the clinician, and the procedures are too time consuming to be feasible in patient care. It is possible that clinicians can detect this 5° difference in hip flexion range of motion between PF-SLR and DF-SLR, as this is slightly greater than the intraobserver variability for standard hip goniometer of 2° and inclinometry of 2.7°.3 It is possible that hip rotation occurred during this SLR testing, which could have influenced our outcomes (we did not measure this axis of motion in this study). Nevertheless, standardized procedures and precision measurements can be used clinically to minimize the risks of confounding variables such as poorly controlled limb movement, different patient instructions,
and different range-of-motion measurement tools. Clinically, full ankle dorsiflexion range of motion can be used during SLR (compared to dorsiflexion to 0° used in our study) to increase the impact of sensitizing maneuvers by, theoretically, increasing the stress to the posterior elements of the lower extremity nervous system. In addition, a conceptual understanding of the impacts of sensitizing maneuvers on symptoms, nerve mobility, and muscle activity will assist with interpretation of SLR outcome measures.  

There is a limitation in making definitive conclusions based on the variability in the EMG data found in our study. Possible cross talk between muscles could have influenced the EMG findings. Because not all subjects respond with the same muscle activation pattern, extrapolation of our findings to clinical settings could be unwarranted at this phase. A better understanding of all of the possible response patterns and why individuals respond differently is necessary to ensure that the assessment of muscle response is not misinterpreted in a clinical setting. Further exploration of muscle responses in the lower extremity in various populations of people with pain during neurodynamic testing is warranted. Finally, it is quite possible that structures other than nerves and their associated connective tissue, such as blood vessels and fascia that span multiple joints in the limbs, could be contributing to the alteration in range of motion, muscle activity, and symptoms that were found in this study.

CONCLUSION

MECHANOSENSITIVITY OF THE NERVIOUS SYSTEM IS A NORMAL PROTECTIVE MECHANISM THAT INCLUDES SYMPTOM PRODUCTION, INCREASES IN MUSCLE TONE, AND SUBSEQUENT REDUCTIONS IN RANGE OF MOTION IN THE LOWER LIMB DURING NEURODYNAMIC TESTING. PERFORMING THE SLR TO THE FIRST ONSET OF SYMPTOMS IS AN ASSESSMENT TOOL THAT IS HIGHLY RELIABLE IN ASYMPTOMATIC LIMBS OF HEALTHY INDIVIDUALS, ALLOWING FOR IDENTIFICATION OF MEANINGFUL DIFFERENCES IN TEST OUTCOMES THROUGH THE USE OF SENSITIZING MANEUVERS, AND MAY BE OF USE IN PATIENTS WITH IRITABLE CONDITIONS. NORMAL PROTECTIVE MUSCLE GUARDING INDUCED BY THE NERVOUS SYSTEM TO AVOID OVERSTRETCH IN HEALTHY INDIVIDUALS SHOULD BE CONSIDERED WHEN ASSESSING RESISTANCE FELT DURING SLR TESTING AND CONSIDERED WHEN PRESCRIBING MUSCLE AND SOFT TISSUE STRETCHES.

KEY POINTS

FINDINGS: Ankle dorsiflexion, when used as a sensitizing maneuver for SLR neurodynamic testing, increases the frequency of distal symptoms, triggers a broader muscular response, and subsequently reduces the amount of hip flexion range of motion when tested to the first onset of symptoms.

IMPLICATIONS: The use of ankle dorsiflexion is an appropriate sensitizing maneuver for SLR neurodynamic testing, and performing the test to the first onset of symptoms provides sufficient information to assist structural differentiation.

CAUTION: This study was limited to individuals with no history of nerve injury and the use of precise instrumentation for assessing range of motion and muscle activity. Caution should be exercised in extrapolating these findings to clinical measurement tools and to populations with nerve injury.

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