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Mechanical Effects of a Specific Neurodynamic Mobilization of the Superficial Fibular Nerve: A Cadaveric Study

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Context: A specific neurodynamic mobilization for the superficial fibular nerve (SFN) has been suggested in the reference literature for manual therapists to evaluate nerve mechanosensitivity in patients. However, no authors of biomechanical studies have examined the ability of this technique to produce nerve strain. Therefore, the mechanical specificity of this technique is not yet established.

Objectives: To test whether this examination and treatment technique produced nerve strain in the fresh frozen cadaver and the contribution of each motion to total longitudinal strain.

Design: Controlled laboratory study.

Setting: Laboratory.

Main Outcome Measure(s): A differential variable reluctance transducer was inserted in 10 SFNs from 6 fresh cadavers to measure strain during the mobilization. A specific sequence of plantar flexion, ankle inversion, straight-leg raise position, and 30° of hip adduction was applied to the lower limb. The mobilization was repeated at 0°, 30°, 60°, and 90° of the straight-leg raise position to measure the effect of hip-flexion position.

Results: Compared with a resting position, this neurodynamic mobilization produced a significant amount of strain in the SFN (7.93% ± 0.51%, $P < .001$). Plantar flexion (59.34% ± 25.82%) and ankle inversion (32.80% ± 21.41%) accounted for the biggest proportions of total strain during the mobilization. No difference was noted among different hip-flexion positions. Hip adduction did not significantly contribute to final strain (0.39% ± 10.42%, $P > .05$), although high variability among limbs existed.

Conclusions: Ankle motion should be considered the most important factor during neurodynamic assessment of the SFN for distal entrapment. These results suggest that this technique produces sufficient strain in the SFN and could therefore be evaluated in vivo for correlation with mechanosensitivity.

Key Words: manual therapy, neurodynamics, straight-leg raise, mechanosensitivity, nerve strain

Key Points

- Ankle motions should be considered clinically more important than the straight-leg raise during superficial fibular nerve entrapment evaluation.
- The straight-leg raise should be used as a differentiation maneuver during superficial fibular nerve strain testing.
- Hip adduction does not contribute significantly to superficial fibular nerve strain during straight-leg raise testing.

Neurodynamic mobilizations (NDMs) are a range of techniques used clinically to test the mechanical and symptomatic responses to movement of a patient's nerves.[1] Passive mobilizations and sensitizing movements[2,3] are applied to induce nerve strain, enabling the clinician to assess the relevant nerve mechanosensitivity to these induced forces. *Mechanosensitivity* refers to the relative sensitivity of a nerve when exposed to external force or loads and is thought to be a protective mechanism against mechanical stress,[2] which may result in pathological changes.[4] Heightened mechanosensitivity is considered an abnormal response during neurodynamic evaluation.[5,6]

Superficial fibular nerve (SFN) entrapment neuropathy is a condition in which the SFN experiences prolonged mechanical compression at the subcutaneous exit point by the crural fascia.[7] Emerging from L4 through S3, the sciatic nerve courses along the posterior aspect of the thigh and splits at the popliteal level to form the tibial (medial) and common fibular nerve (lateral). The SFN (roots L4–S1) originates from the common fibular nerve along the proximal insertions of the fibularis longus muscle and exits the crural fascia at the distal one-third of the lower leg. Symptoms of SFN entrapment include pain, paresthesia, or both on the anterolateral aspect of the leg and the laterodorsal aspect of the foot[8,9] except between toes 1 and 2. A prevalence of 3.5% of SFN entrapment neuropathy at the exit from the crural fascia in patients with chronic leg pain has been reported.[10] Additionally, Falciglia et al[11] observed SFN entrapment neuropathy in 4.1% of severe ankle sprains in

children and adolescents. In the management of peripheral neuropathies, conservative options such as physical rehabilitation are often recommended before referral to physicians who specialize in pain managment.[12,13] Among the modalities used by manual therapists, NDMs were described as effective in the management of peripheral neuropathies,[14–16] cervical radiculopathies, and low back pain, although more robust evidence is yet to be published.[17]

Earlier authors[2,5] noted that neural tissue responds to movement by strain and excursion. Changes in nerve strain are influenced by joint position,[17,18] surgery,[19] and injury.[20] Moreover, many researchers have demonstrated the contribution of lower limb movement to tibial and sciatic nerve strain during the straight-leg raise (SLR) test combined with ankle dorsiflexion.[18,21,22] Çelebi et al[23] conducted a sonoelastographic investigation and found that sciatic nerve stiffness at the gluteal region increased in patients with lumbar disc herniation. Furthermore, Neto et al[24] showed a reduction in nerve stiffness immediately after NDM in patients affected by sciatica who assumed a static slump position. However, these results contradict those of a previous study[25] using a long-sitting slump position. This suggests that clinicians must investigate neuropathic pain with various techniques to find the most appropriate type of mobilization for each patient.[2]

Also, because of the poor efficiency of the lymphatic system for drainage, chronic local edema and intraneural fluid accumulation within the nerve may lead to fibrosis, impairing the ability of the nerve to glide freely[26,27] and thereby impairing the stretch response of the nerve and its normal physiological functions.[28,29] Strain can play a role in nerve physiology: strain of $\geq$15.7% applied to the rabbit sciatic nerve interrupted neural vascularization.[30] In an in vitro study of the tibial nerve, Brown et al[31] determined that a mechanical influence in the form of passive mobilization of the ankle caused dispersion of the intraneural fluid. Moreover, Boudier-Revéret et al[32] stated that strain and fluid dispersion may not strongly correlate because no differences were noted between sliding and tensioning neural mobilization techniques on fluid dispersion. This finding could indicate the importance of general movement and mobilization techniques for fluid dispersion.

Although NDMs are commonly used by manual therapists, a lack of standardization in the application of neurodynamic tests makes the evaluation of their clinical effects difficult.[33] A specific NDM with SFN bias is, for the moment, based on neurodynamics reference books[34] and anecdotal evidence.[35] Whether these proposed techniques produce nerve elongation or the magnitude of such elongation is unknown. This could have a significant clinical effect, as the sequence used to evaluate neural mechanosensitivity may not be the most efficient in eliciting or reproducing a patient's symptoms, thereby resulting in inconclusive findings. Previous authors[18] have identified hip flexion as an important influencer of strain measured at the tibial nerve. This could suggest the importance of hip position during NDM with SFN bias as a sensitizing motion. To our knowledge, no investigators have studied the biomechanical influence of hip position in the frontal plane on lower limb neurodynamics for the SFN.

Therefore, the purposes of our study were to examine if NDM with SFN bias[34] produced longitudinal strain at the exit of the SFN from the crural fascia and to quantify the strain behavior of the SFN throughout the mobilization. The first objective was to compare the effect of 4 hip positions (as used during an SLR test) on total strain after a complete mobilization. We hypothesized that applying a neurodynamic test at 90° of hip flexion during the SLR would produce the most strain at the SFN. The second objective was to describe the contribution of each motion in the mobilization sequence to the total strain. We hypothesized that the hip-adduction (ADD) component of the NDM might lower the strain experienced by the SFN because of the medial route of the lumbar plexus in regard to the abduction-ADD axis of the hip. Because it has been reported[36] that neighboring joints seem to have a great influence on nerve strain during NDM, we hypothesized that ankle motions might be the biggest contributors to total nerve strain. These results may help researchers and clinicians better understand the mechanical behavior of the SFN during NDM and could support the use of NDM for further in vivo studies.

METHODS

Specimens

Six fresh frozen cadavers from the Université du Québec à Trois-Rivière's anatomy laboratory were selected for this study: 4 female and 2 male (mean age = 84 $\pm$ 4.33 years, body mass index = 21.6 $\pm$ 1.67 kg/m^2). Because of acquired local lesions, 2 lower limbs from 2 cadavers were not included, so 10 lower limbs were tested. The project received approval from the Anatomy Department Subcommittee's Ethics Board at the Université du Québec à Trois-Rivières period.

Specimen Preparation

The cadavers were positioned lying supine on an experimental frame. Before data collection, the specimens were thawed for 48 hours. We palpated each abdomen looking for a soft end feel and controlled the temperature to confirm that the bodies were fully thawed. All joints of the lower limbs were mobilized to ensure maximal range of motion in their anatomical planes.

The skin was incised longitudinally over 8 cm at the anterolateral aspect of the distal third of the leg, allowing us to reach the SFN (Figure 1). Care was taken to maintain the integrity of the crural fascia where the SFN exits, preserving the moving plane of the nerve. No crural fascia were incised during the dissection. The surrounding superficial adipose tissue was cleaned using a 23-blade scalpel to obtain adequate nerve visualization.

Segmental SFN linear elongation was measured using a differential variable reluctance transducer (DVRT) with 6-mm stroke length (Parker LORD MicroStrain Sensing System). The DVRT was inserted in the nerve via 2 barbed pegs 2 cm inferior to the exit of the SFN. The nonmoving part of the sensor was sutured around the nerve's axis (Figure 2) by an anatomist with more than 15 years of cadaveric research experience to ensure DVRT stability.

The communicating wire and wireless transmitter were secured to the proximal lateral aspect of the leg using zinc-oxide tape to avoid interference with any soft tissue of the lower leg. Node Commander software (Parker LORD MicroStrain Sensing System) was used for data collection. The cadaver's pelvis and thorax were then secured to the experimental frame using a ratchet tie-down strap to stabilize the specimen throughout the mobilizations.

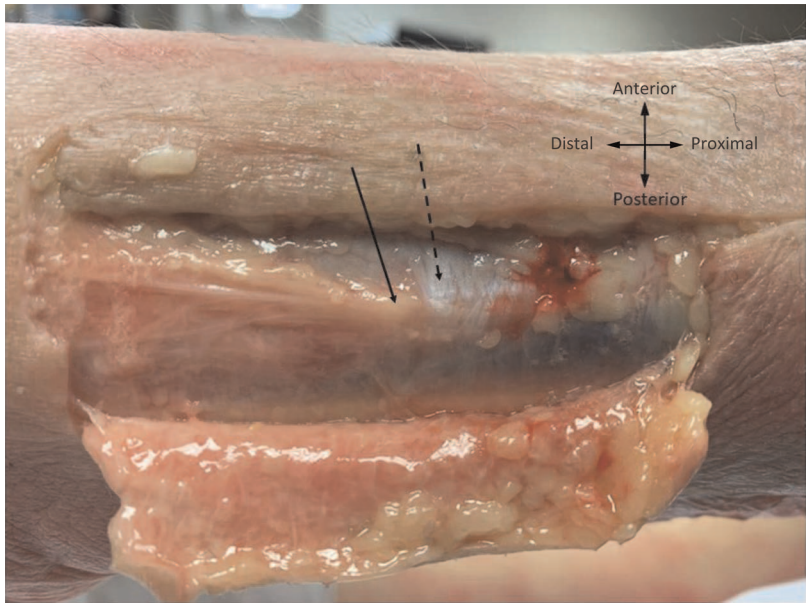

Figure 1. Dissection window of the superficial fibular nerve. Lateral view. The superficial fibular nerve is indicated with a continuous arrow; the crural fascia is indicated with a dotted arrow.

Experimental Setup and Data Collection

To ensure reliability and accuracy of hip movements during testing, we used an optoelectronic motion-capture system (Prime^{X22}, Optitrack, NaturalPoint Inc). Two intracortical pins, mounted on top by 1 cluster of 4 reflective markers, were introduced in the diaphysis of the femur and the superior aspect of the anterior-superior iliac spine.

Mobilization was performed by a physical therapy technologist licensed in Québec, Canada. The motion sequence followed the specific order described in the reference literature[34]: (1) maximal available ankle plantar flexion (PF), (2) maximal available ankle inversion (INV), (3) hip flexion (part of the SLR mobilization), and (4) 30° of ADD.

The hip-flexion position of the mobilization was randomized for every limb using MATLAB (version R2020b; MathWorks) to make sure nerve creep was not a confounding factor. Ankle inversion was considered a motion in the frontal plane as described by Brockett et al.[37] Each mobilization was repeated 3 times and replicated at different randomized hip-flexion positions (0°, 30°, 60°, 90°) of the SLR. Each position was maintained for 2 seconds to ensure that stable measurements were obtained. Between trials, the limbs were brought back to the resting position and maintained for 1 minute to limit the possible effect of creep on the nerve. The examiner was blinded to the strain data during the NDM. During all procedures, the

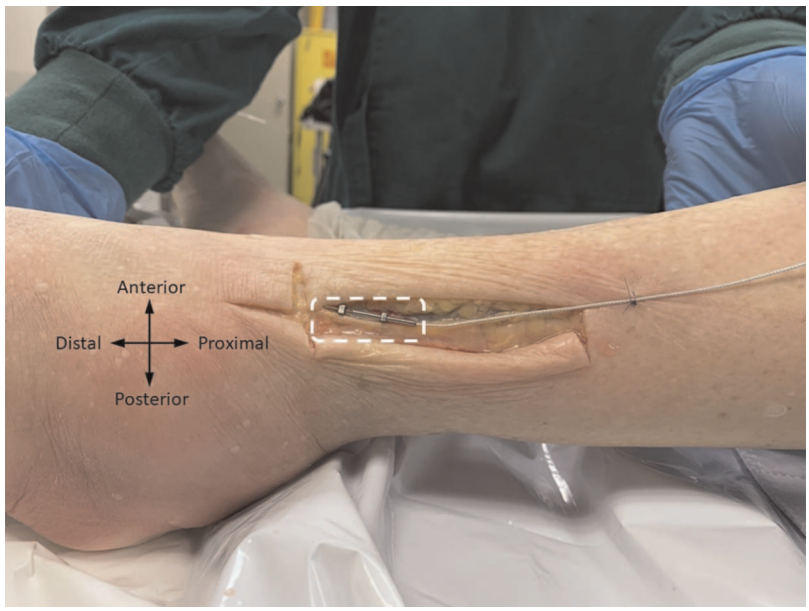

Figure 2. Electromechanical strain gauge (differential variable reluctance transducer [DVRT]) inserted in the left superficial fibular nerve. The DVRT is indicated by the dotted rectangle.

Table 1. Intraclass Correlation Coefficients With Absolute Agreement and SEM

Hip flexion, °	Between Sessions	Within Sessions	Standard Error of Measurement
0	0.80	0.95	2.07
30	0.71	0.90	0.86
60	0.98	0.79	1.14
90	0.97	0.84	0.82

nerve and surrounding tissues were kept hydrated using physiological saline solution (water and NaCl at 0.9%).

Continuous electromechanical measures were obtained in volts, and we applied the manufacturer's conversion curve to calculate displacement in millimeters. Elongation was then used to calculate strain of the nerve tissue. Strain (ε) was expressed as the deformation of the length variation from the initial length of the nerve tissue according to the following equation:

$$\varepsilon = \Delta L / L_0.$$

The resulting strain was expressed as a percentage of elongation (positive value) or shrinkage (negative value). We considered the anatomical reference position as the initial measure (L_0) of length with cadavers lying supine.

Statistical Analysis

Descriptive statistics of the strain were collected at each position of the mobilization sequence. Normal distribution of the data was confirmed using the Shapiro-Wilk test of normality (significance = 0.330). We then conducted a 1-way analysis of variance (ANOVA) on the strain (%). A post hoc Tukey test was applied for multiple comparisons. Statistical tests were calculated using SPSS (version 24; IBM Corp), and the data were extracted using MATLAB. Independent variables were the technique sequence and hip-flexion range of motion, and the dependent factor was the strain measured in the nerve tissue. A test-retest intrarater reliability analysis of strain was performed on 2 cadavers with a 1-hour interval between mobilizations, repeated twice after a randomization protocol. Intrarater reliability was measured with a 2-way random-effects absolute agreement intraclass correlation coefficient.

RESULTS

Reliability

The mean intraclass correlation coefficient (Table 1) with absolute agreement was 0.86 for the strain measure at the end of the mobilization.

Strain

Final strain measured in the SFN at the end of the mobilization with all motions combined is presented in Table 2. Compared with the anatomical resting position, differences in strain were produced at the nerve (7.93% ± 0.51%, $P < .001$).

With all motions combined at the end of mobilization, we did not observe any difference in strain among the different SLR hip-flexion positions ($P = .851$; Table 2).

A general view of the strain behaviors throughout the entire mobilization is shown in Figure 3. The peak strain percentage was reached after the hip-flexion position during every mobilization (after INV at 0° of hip flexion). Plantar flexion and INV were the main motions inducing nerve elongation, at 4.66% ± 0.53% and 2.54% ± 0.18%, respectively.

Motion Contribution to Total Strain

Motion contribution to total strain was defined as the percentage a specific motion contributed to a scale of 100%, which represents the total strain attained at the end of the mobilization (Figures 3 and 4). The mean contributions (%) of motions at every hip-flexion level are provided in Figure 3. Globally, during mobilization, PF (59.34% ± 25.82%) and INV (32.80% ± 21.41%) were consistently the largest contributors to strain. Nevertheless, their contributions steadily decreased as hip flexion became increasingly important as a contributor (Figure 4).

These data by motion at different hip-flexion positions are provided in Figure 4. No difference was found for PF ($P = .695$), INV ($P = .643$), or ADD ($P = .202$). Therefore, the contributions of these motions did not differ, whether they were performed at 0°, 30°, 60°, or 90°. As seen in Figure 4, the hip-flexion positions' contributions to total strain differed (0°/30°/60°/90°; $P = .003$).

We averaged contribution values from each position (Table 3). A 1-way ANOVA was conducted to compare each motion against the others to determine whether a statistical difference was present. The ANOVA showed a difference among global motions ($F = 84.104$, $P < .001$). A post hoc Tukey analysis indicated that PF contributed more to strain than INV ($P < .001$), hip flexion ($P < .001$), or ADD ($P < .001$). Ankle inversion also contributed more than hip flexion ($P < .001$) and ADD ($P < .001$). However, no difference was found between hip flexion (6.96% ± 10.56%) and ADD (0.39% ± 10.42%; $P = .381$).

DISCUSSION

To our knowledge, this is the first study investigating the mechanical effect of a specific neurodynamic test of the SFN composed of hip and ankle movements in fresh cadavers. Our aim was to assess the ability of a specific neurodynamic test to produce strain of the SFN at the exit from the crural fascia. We observed that strain was indeed produced during an NDM with SFN bias (7.12%–8.23%). This finding is unsurprising, as other authors have described the effect of SLR mobilizations on the sciatic, tibial, and plantar nerves[21] and at lumbar roots L4 through S1.[22]

Although testing of SFN mechanosensitivity using neurodynamics has been described previously,[38] no biomechanical studies have addressed nerve strain. Even though

Table 2. Final Strain With All Motions Combined at Different Hip-Flexion Positions[a]

Hip-Flexion Position, °	Final Strain, Mean ± SD, %	95% CI
0	7.12 ± 4.14	4.15, 10.08
30	8.17 ± 2.72	6.22, 10.12
60	8.23 ± 3.59	5.66, 10.80
90	8.19 ± 2.59	6.34, 10.04

[a] Analysis of variance $P = .851$.

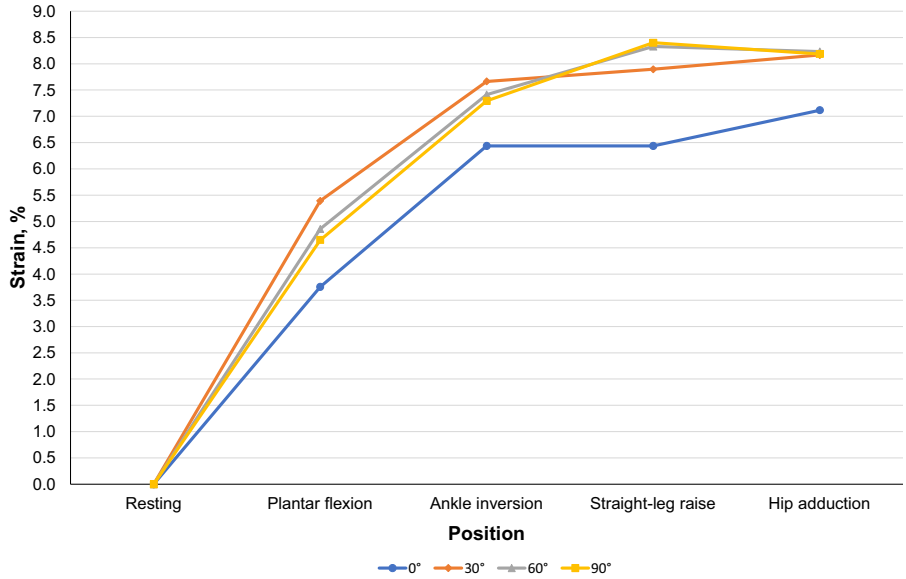

Figure 3. Strain behavior during mobilization. x-Axis, motion; y-axis, strain percentage.

the specific order of mobilization produced a significant amount of strain on the SFN, we did not find a difference in final absolute strain from the distinct levels of hip flexion used during the SLR component (Table 2). Earlier researchers[39] observed that the order of mobilization may not have influenced final strain on the tibial and sciatic nerves during SLR testing on cadavers. This implies that another order might have yielded similar results. Our results suggested that hip-flexion positions during SLR might not influence the final strain.

Additionally, we noted that hip flexion and ADD seemed to have less influence on total strain and that ankle movements (PF and INV) were the main relative contributors to SFN strain (59.34% to 32.80% of total strain). This is consistent with a previous study[40] in which neighboring joints to the tested nerve elicited a greater mechanical influence. Plantar flexion was consistently the highest contributor to total strain of the SFN. This finding is supported when we consider the normal anatomy of the SFN as it traverses the dorsal aspect of the foot anterior to the transverse axis of the ankle.

Our results could also demonstrate the critical implications of ankle motions on the testing of mechanosensitivity in the SFN and reinforce their effect on SFN deformation. Although the hip-flexion position did not cause the most strain in the evaluated segment of the SFN, it is generally used in a clinical setting as a differentiation maneuver. Its increasing contribution to strain during the mobilization is why it should be used clinically for pain differentiation. However, the amount of strain that is clinically significant in the living population has yet to be examined. Therefore, we cannot confirm the meaningfulness of various hip-flexion levels as a differentiation maneuver because the lack

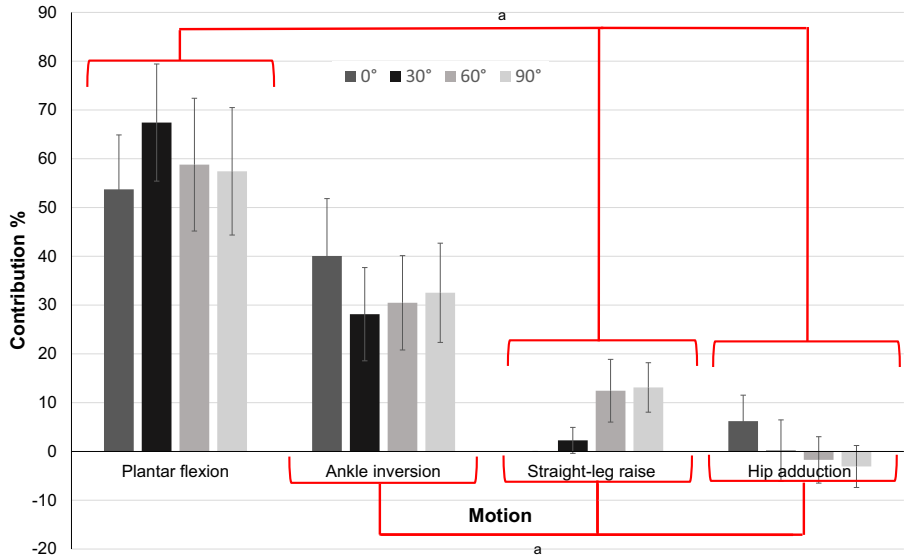

Figure 4. Relative contribution percentage by motion at different hip-flexion positions (0°/30°/60°/90°). [a] Indicates a significant difference.

Table 3. Global Motion Contribution (%) After Grouping Hip-Flexion Positions

Motion	Relative Contribution ± SD	95% CI
Plantar flexion	59.34 ± 25.82	51.09, 67.60
Ankle inversion	32.80 ± 21.41	25.96, 39.65
Hip flexion[a]	6.96 ± 10.56	3.58, 10.34
Hip adduction[a]	0.39 ± 10.42	−2.94, 3.73

[a] No statistical difference was evident among these motions ($P = .381$).

of statistical difference may or may not be clinically significant. As the SLR is usually performed with the ankle dorsiflexed, the initial motions could lower the innate neural tension at the proximal sciatic nerve, explaining why hip flexion may have less influence on SFN elongation.

Earlier authors[41] have studied the effect of INV as a single motion on SFN strain and excursion with a simulated talofibular ligament tear in a cadaveric setting. They measured a comparable amount of strain with an in vitro simulated ankle sprain (3.0%–11.6%) relative to our work (4.15%–10.80%). Interestingly, we did not identify strain >10.80%, which is far lower than the 15.7% cited as detrimental to neural vascularization.[32] This SFN mobilization technique could then be considered safe to execute in vivo.

Our hypothesis that ADD would lower the amount of strain was not confirmed across all conditions, as it did not change the elongation of the SFN across all mobilizations. We noted a slight reduction at 60° and 90° of SLR and no effect at 0° and 30°. This reduction could indicate that a more proximal phenomenon may be happening at the gluteal region, where an anchor of the sciatic nerve would change its response when mobilizing >30° of ADD. We propose that the lumbar plexus, passing medially to the coronal axis of the hip, may explain why strain seemed mainly unaffected at the SFN in an adducted position of the hip. Additionally, the stress response of cadaveric tissue may differ from that of living tissue. Comparative studies in the living should be conducted to compare stress responses using shear-wave elastography.

LIMITATIONS

Although our investigation provides new insights on NDMs in the lower extremity, certain limitations arose. First, we considered only longitudinal stresses. Other biomechanical forces, such as shear and compressive forces, were not addressed. Second, the tester tried to maintain movements in the perfect anatomical planes with infrared tracking but could have induced a small amount of hip internal rotation during the ADD part of the mobilization. This might have had a minimal influence on the strain in the SFN. A cadaveric study such as ours, involving a minimal but certain amount of dissection, could have modified the moving plane of the nerve. The age group of the cadavers did not represent the typical athletic trainer's patient population. As peripheral nerve tissue ages, stiffness increases, which could change strain values compared with a younger and more active population. This might also affect variability in strain: a younger population may present more variability, emphasizing the need for the clinician to apply different movement combinations. The results were obtained in a cadaveric setting; thus, applicability could differ in a clinical population. Our findings can, however, be a starting point for in vivo studies using noninvasive measurement techniques such as shear-wave elastography.

CONCLUSIONS

A specific NDM increased the longitudinal strain of the SFN. Different hip-flexion positions during the SLR maneuver did not seem to affect final longitudinal strain, although they became more significant contributors as the range of motion increased. Clinicians should consider ankle motions crucial to producing strain in the SFN in order to evaluate mechanosensitivity in patients. Our results also showed that clinicians must evaluate different positions of the hip to characterize SFN mechanosensitivity, as ADD showed a significant interlimb variable effect on strain. It is interesting to note that the "optimal" amount of strain during mobilization for clinical results has yet to be established. Future clinical studies are recommended to determine the effect of ankle and hip movements on the symptoms expected to originate from the SFN.

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