The effects of neurodynamic straight leg raise treatment duration on range of hip flexion and protective muscle activity at P1

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Study design: Randomized, single blind, same subject crossover trial.
Objectives: To compare the effects of two neurodynamic treatment doses on range of hip flexion (ROM HF) and electromyographic (EMG) activity of semitendinosus, at first onset of pain (P1).
Methods: A total of 26 healthy participants without low back or leg pain received each treatment in a random order with at least 48 hours between sessions. Baseline ROM HF and EMG magnitude of semitendinosus at P1 were collected. Subjects then received either 3×1 or 3×2 minutes of oscillating end of range (grade IV+) straight leg raise (SLR) neurodynamic treatment and were re-assessed for baseline measures.
Results: There was no significant difference between groups in EMG magnitude ($P=0.190$) and ROM HF ($P=0.739$) at P1. There was also no significant difference within groups in EMG magnitude at P1 ($P=0.182$); however, there was a significant improvement in ROM HF at P1 in both groups compared to baseline readings ($P=0.000$), with increases of 6.7° and 5.1° for the 3×1- and 3×2-minute groups, respectively.
Conclusion: Findings indicate that 3×2 minutes of oscillating grade IV+ SLR neurodynamic treatment has no additional benefit over 3×1 minute, on ROM HF or EMG magnitude of semitendinosus at P1. Using an oscillating SLR treatment may, however, help to increase pain-free ROM HF, although further studies are necessary to confirm this.

Keywords: Neural mobilization, Neurodynamics, Straight leg raise, Treatment duration, Treatment dose

Introduction

The Straight Leg Raise (SLR) test is widely used in the assessment of altered nerve mechanosensitivity.[1,2] In a pathological state, increased mechanosensitivity may be apparent through the nociceptive mediated flexor withdrawal, a protective muscular contraction postulated to prevent the occurrence of nerve dysfunction.[1,2] Studies indicate that medial hamstrings are the first indicator of muscle activity in response to the passive SLR test[2] and that semitendinosus activity significantly increases ($P=0.005$) at the first onset of pain (P1).[1]

The passive SLR test has previously been reported to have excellent reliability in detecting P1, with a reported intra-class correlation coefficient (ICC) of 0.89[3] and an intra-rater correlation coefficient of 0.91.[4] Furthermore, Coppieters *et al.*[5] found that there was no significant difference ($P \geq 0.11$) in pain intensity between participants with induced muscle pain and those without, when undertaking the SLR test. This led the authors to suggest that when pain is of a non-neural origin, it would result in a normal SLR test response, contributing toward its validity (specificity) in detecting altered nerve mechanosensitivty.[5]

To date, there are only a limited number of studies that have investigated EMG activity during the passive SLR test.[1,6,7] Boyd *et al.*[1] reported that EMG activity in soleus, semitendinosus, tibialis anterior, and vastus medialis all increased at P1, when undertaking the SLR with ankle dorsiflexion. These findings led the authors to suggest that co-contractions were responsible for helping to stabilize the joints and limit movement.[1] A further study supports the protective muscle activity hypothesis by reporting that EMG activity of semimembranosus increased in a proportional manner after 40° SLR,

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although the authors did not investigate subjects' pain responses.[6]

Similar findings have also been documented in upper limb neurodynamic studies.[8,9] It has previously been reported that less extensible participants have significantly higher ($P<0.05$) EMG activity of upper trapezius during the Upper Limb Neurodynamic Test One (ULNT1).[8] Balster and Jull[8] suggest that EMG activity increased to enable shoulder girdle elevation, especially in less extensible participants, and therefore protect the nervous system from excessive tensile forces. This hypothesis appears plausible as shoulder girdle elevation is a desensitizing maneuver[10] that may reduce strain and decrease pressure around the median nerve.

Pain is also an important parameter and has been measured extensively during neurodynamic testing.[1,11,12] Some authors debate the significance of pain alone in mediating the flexor withdrawal, with one study reporting that, in some cases, EMG activity of upper trapezius and brachialis increased before P1;[11] however, despite the academic debate there appears to be a majority consensus that pain is involved in a complex protective interaction whereby muscle tone is increased and eventually limits movement during neurodynamic testing.[2,6,7,11,12]

Straight Leg Raise Treatment

A number of treatment effects from neurodynamic treatment have been proposed. Tal-Akabi and Rushton[13] documented that the rationale behind neurodynamic treatment is to improve axoplasmic flow and through this mechanism improve nerve conduction. It has also been suggested that mobilizing a nerve may disperse edema and reduce pressure resulting in improved microcirculation,[13–15] which might facilitate healing of an injured nerve.[10] Other plausible hypotheses include reducing the nerve's sensitivity to abnormal impulse-generating sites and restoring restricted movement through breaking down scar tissue adhesions with nerve gliding,[14,15] and therefore improving neural tissue viscoelasticity.[10,16] Furthermore, it has also been proposed that neurodynamic treatment may induce hypoalgesia through modulating descending inhibitory pain mechanisms.[17] Despite the range of hypotheses, it is widely reported that there is a lack of research about the therapeutic effects of neurodynamic treatment, despite the fact it is commonly used in clinical practice.[10,16,18]

Only a limited number of studies were found investigating the efficacy of lower limb neurodynamic treatment.[15,19–21] Studies by Adel[19] and Cleland *et al.*[15] report the favorable effects for patients with low back dysfunction and non-radicular low back pain who receive neurodynamic treatment. Adel[19] investigated the use of neurodynamic SLR treatment, and Cleland *et al.*[15] investigated slump stretching, both in addition to a normal treatment program of lumbar spine mobilization and exercise. The studies reported significant reductions ($P<0.05$) in pain,[15,19] disability,[15,19] centralization of symptoms,[15] and decreased nerve root compression on magnetic resonance imaging.[19] Further research conducted in upper limb studies appears to support the efficacy of using neurodynamic treatment for improving range of motion (ROM) and decreasing pain.[9,21–23]

Current research has identified treatment interventions to often have small effect sizes. A research priority has been to attempt to subgroup patients based on their symptoms in order to investigate who will likely respond to treatment. One rationale for this approach is the potential heterogeneity present in individuals going for physical therapy. A recent article by Schafer *et al.*[21] used two passive neurodynamic treatments: lumbar lateral flexion in side lying contralateral to the painful side for 60 seconds, and hip and knee flexion followed by hip and knee extension for 30 seconds. The results indicated that over half of those with peripheral nerve sensitization arising from suspected nerve inflammation responded favorably to neurodynamic treatment with improvements in pain, disability, and global perceived change ($P=<0.05$). The results published by Schafer *et al.*[21] provide insight into our understanding of who will likely respond to neurodynamic treatment and suggest that if patients are sub-grouped prior to treatment, the efficacy is greatly enhanced.

Published research about neurodynamic treatment appears to support its use in the treatment of altered nerve mechanosensitivity; however, an optimum treatment dose has not yet been established.

Treatment duration

Current neurodynamic research has administered a wide range of treatment durations with a lack of standardization.[13,15,19–22,24] Some investigations have used up to seven different neurodynamic techniques consisting of one to seven sets lasting 5–60 seconds,[9,15,19–24] with one study adapting treatment dose based on patients' responses.[13] To the authors' knowledge, there have been no published studies that have compared different treatment durations; therefore, all treatment doses selected in clinical practice are based on anecdotal evidence and empirical evidence is needed to validate their efficacy.

Limited research has been performed investigating neurodynamic treatment duration; therefore, studies on the duration of joint mobilizations might be extrapolated. It could be hypothesized that some of the mechanisms associated with the benefits of joint mobilizations may be similar to those of neurodynamic treatment.[25] In one study using participants

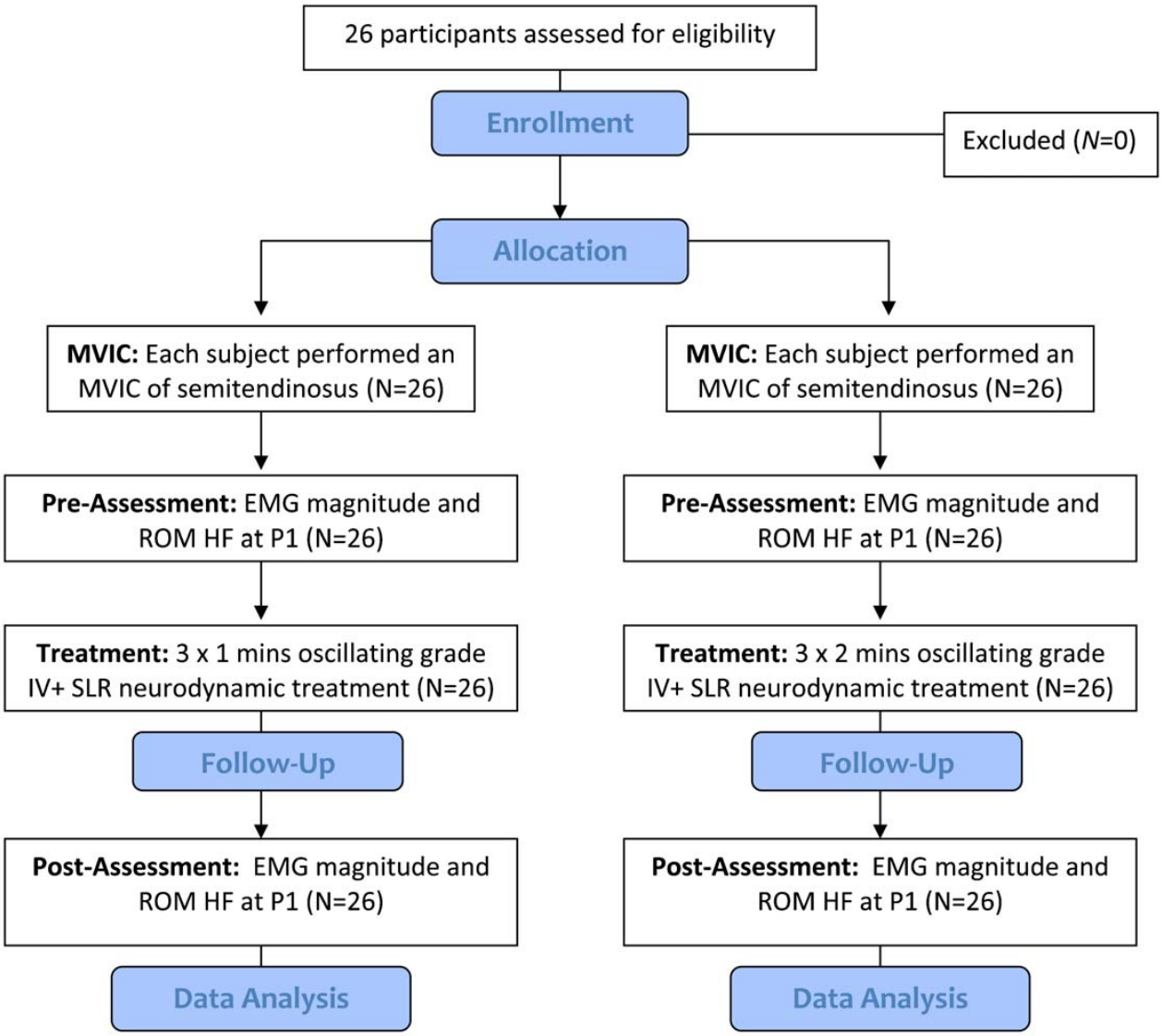

Figure 1 Flow chart of the methodological procedure. Abbreviations: N, numbers; MVIC, maximum voluntary isometric contraction; EMG, electromyographic; ROM HF, range of hip flexion; P1, first onset of pain/discomfort; SLR, straight leg raise.

with mild to moderate knee pain from osteoarthritis, 3×3 minutes of anterior–posterior (AP) mobilizations to the tibio-femoral joint resulted in a significantly decreased ($P<0.05$) pressure pain threshold (PPT) both locally (knee) and more widespread (heel) when compared with manual contact or control.[17] Furthermore, in a study using rats, Sluka and Wright[26] experimentally produced pain by injecting capsaicin into the ankle joint and compared paw withdrawal thresholds before and after different durations (3×1, 3×3, 3×5 minutes) of AP knee joint mobilizations, with hand contact or control. The results outlined that only 3×3 and 3×5 minutes of knee mobilizations produced a significant hypoalgesic response ($P<0.05$), which lasted for more than 30 minutes post-mobilization. Although these findings appear to support the use of longer treatment durations, studies are required to assess whether such times are necessary in humans. The current investigation aimed to determine whether different durations of passive SLR neurodynamic treatment affected ROM HF and EMG magnitude of medial hamstrings at P1.

Method

The study was retrospectively registered as a clinical trial in the Australian New Zealand Clinical Trials Registry (ANZCTR) and provided with the trial identification ACTRN12613000956707. The study adopted a single blind, randomized, same subject crossover design. Each participant attended The Human Movement Laboratory on two occasions, receiving each experimental condition in a random order (Fig. 1).

Participants

A total of 26 asymptomatic volunteers (14 male and 12 female) aged 20–24 years (SD $\pm$ 1.4) participated in the study (Table 1). Participants were selected from sealed envelopes by a blinded researcher and assigned to experimental condition A or B in a random

Table 1 Anthropometric measurements of the participants: height, weight, and BMI (*N*=26: 14 males and 12 females)

	Height (cm)	Weight (kg)	BMI (kg/m^2)
Mean	172	72.3	24
SD	±9	±11.3	±2.6
Range	161–188	48–96	17.8–27.8

Abbreviations: cm, centimeters; kg, kilograms; m^2, meters squared; *N*, number; SD, standard deviation; BMI, body mass index.

order. All participants were screened prior to testing. Participants were excluded if they had any current or recent (<12 months) lower back or leg pain, were unable to maintain a passive SLR position, had a positive neurological integrity test (muscle weakness/motor loss, altered sensation, or abnormal reflexes), or had any red flags to manual therapy. Participants with a body mass index (BMI)>28 kg/m^2 were excluded to allow a comparable thickness of subcutaneous tissue,[27,28] and volunteers more than 48 years were excluded as age can cause large variations in EMG magnitude recordings.[29] All participants signed written informed consent forms, and the rights of all participants were protected. Ethical approval was gained from the University of Brighton's research ethics and governance committee.

Equipment, procedure, and measurements

In prone lying, the mid-belly of semitendinosus was identified (dominant limb) and the local area was cleaned with isopropyl alcohol wipes and shaved. An active, pre-amplified bipolar electrode (Biometrics [SX-230], Biometrics Ltd, Gwent, UK) was then placed longitudinally over the mid-belly of semitendinosus and secured using Biometrics' adhesive pads (T350). In order to improve repeatability for the second session, the electrodes' outline was marked and secured by the same researcher as on the first occasion. Electromyographic data were collected by synchronizing EMG electrodes with a data acquisition unit (DataLINK [DLK800], Biometrics Ltd, Gwent, UK), and a ground electrode (R200) was placed over the ulna styloid.

Participants were then asked to turn supine. A 12-cm wooden box was placed under the dominant heel, and a maximum voluntary isometric contraction (MVIC) was performed by each participant. Participants were given instructions, 'When I say go, push your heel into the box as hard as you can for 5 seconds'. When EMG data were obtained, the box was removed. Each participants had an ankle–foot orthotic applied to maintain the ankle in plantargrade, and a dual-axis flexible electrogoniometer (Biometrics [SG150], Biometrics Ltd, Gwent, UK) was fitted around the hip joint to measure sagittal plane motion. The proximal end of the electrogoniometer was placed parallel to the subject's torso (adjacent to the iliac crest) with the distal end on the lateral thigh.[8] The electrogoniometer's sampling frequency was set to 1000 Hz. A trigger (consisting of a button) was then placed on the patient's hand in the contralateral side with a second EMG electrode placed on the thenar eminence. Participants were instructed to press the trigger when they felt the first onset of pain and/or discomfort. (Squeezing the trigger resulted in a spike of EMG activity from the thumb muscles, which was used to indicate P1.)

The passive SLR was then performed that consisted of progressive hip flexion with the knee held in full extension. It was explained to the participants that they would likely feel a mild stretching sensation on the back of their thigh during the test; however, they should only squeeze the trigger when they first feel a sensation of pain and/or discomfort (P1). At this point, the limb was gently lowered to the plinth. Electromyographic magnitude of semitendinosus and ROM HF were recorded from this point.

Treatment

Once the baseline testing had been completed, the treatment procedure commenced. The AFO (ankle-foot orthotic) was left *in situ*, and the hip and pelvis were unsupported. The leg was raised in the passive SLR position until P1 was reached, and at this point, the hip was oscillated using a small amplitude at the end of range (Grade IV+).[30] Treatments lasted for either 3×1 or 3×2 minutes, with each oscillation standardized using a metronome (1.5 Hz) and each set separated by 1-minute rest intervals. After treatment had finished, the limb was gently lowered to the plinth and post-assessment passive SLR testing was carried out recording ROM HF and EMG magnitude of semitendinosus at P1, as previously described.

Participants were then required to attend the laboratory for a second session in order to receive the second experimental condition. Laboratory sessions were separated by at least 48 hours to control for carryover effects.[31] Reliability of the electrogoniometer to measure ROM HF was carried out by comparing each participant's ROM HF at P1 on two separate occasions, prior to each treatment session.

Data management

Range of hip flexion and EMG magnitude of semitendinosus at P1 were calculated using Matlab (The MathWorks, USA). Surface EMG signals were full wave rectified, converted to root mean square (RMS), and normalized to a percentage of MVIC,[32] to give EMG magnitude at P1. Intra-class correlation coefficient was calculated to determine the examiner's reliability in detecting ROM HF at P1. Shapiro–Wilk's normality testing was undertaken prior to analyzing the data. A two-way analysis of variance (ANOVA)

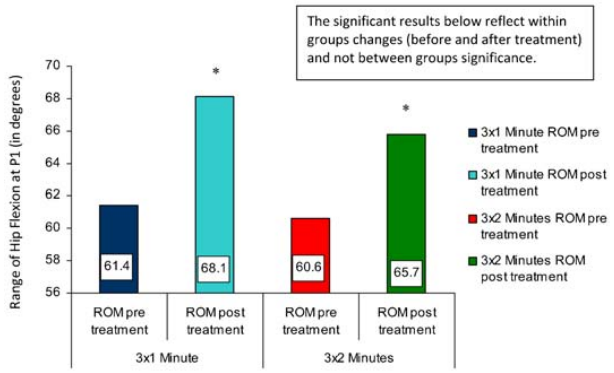

Figure 2 Changes in range of hip flexion at P1, pre- to post-treatment. Significant changes indicated as $P<0.05$*. Abbreviations: ROM, range of motion; P1, first onset of pain/discomfort.

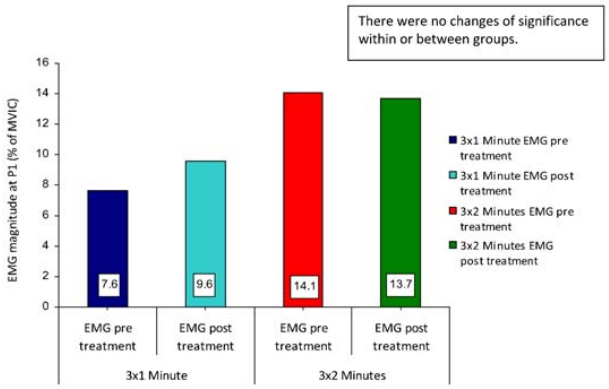

Figure 3 Changes in normalized EMG magnitude (% MVIC) at P1. Significant changes indicated as $P<0.05$*. Abbreviations: EMG, electromyography; MVIC, maximum voluntary isometric contraction; P1, first onset of pain/discomfort.

was then used to test each hypothesis (correcting for violation of sphericity using Greenhouse–Geiser). The dependent variables were EMG and ROM HF (before and after treatment), and the independent variable was duration (3×1 and 3×2 minutes). A probability level of $P \leq 0.05$ was used to test statistically significant differences.[33]

Results

All 26 subjects completed the study with no adverse effects.

Reliability of measuring ROM HF at P1

The outcome of the reliability testing for the ROM HF was an ICC of 0.73 (mean ROM HF at P1 was 62.4° [±17°] on the first occasion and 59.6° [±15.2°] on the second).

Range of hip flexion at P1

Normality testing using Shapiro–Wilk indicated that the data were normally distributed. A two-way ANOVA (correcting for violation of sphericity using Greenhouse–Geiser) demonstrated no significant difference between 3×1 and 3×2 minutes of oscillating grade IV+ SLR neurodynamic treatment on ROM HF achieved at P1 ($P=0.739$, $F=0.114$). There was, however, a significant difference within groups in ROM HF at P1 ($p<0.01$, $F=38.002$), with a mean increase of 6.7° in the 3×1-minute group and 5.1° in the 3×2-minute group (Fig. 2).

Electromyographic magnitude of semitendinosus at P1

Normality testing using Shapiro–Wilk indicated that the data were normally distributed. A two-way ANOVA (correcting for violation of sphericity using Greenhouse–Geiser) demonstrated that there was no significant difference between 3×1 and 3×2 minutes of passive oscillating grade IV+ neurodynamic SLR treatment on EMG magnitude of semitendinosus at P1 ($P=0.190$, $F=1.819$). Results also indicated that there was no significant difference within groups pre- to post-treatment ($P=0.182$, $F=1.886$), detailed in Fig. 3.

Discussion

The primary findings of the study were that there were no statistically significant differences between 3×1 or 3×2 minutes of passive oscillating grade IV+ neurodynamic SLR treatment on EMG magnitude of semitendinosus or ROM HF at P1. This may suggest that using a longer treatment duration (2 minutes) is not different than using a shorter duration (1 minute) on these measures.

Reliability of measuring ROM HF at P1

According to Hass,[34] reliability of the electrogoniometer was shown to be acceptable[33] (ICC=0.73) with results similar to those reported in Hall *et al.*[2] This ensures that the passive SLR test has an acceptable ability to reproduce consistent results in detecting P1; and therefore, any potential changes can largely be attributed to a treatment effect.

Range of hip flexion at P1

Current research in neurodynamic treatment has adopted varying treatment durations,[9,13,19–23] some of which have not been standardized,[9,13,20,24] which prevents conclusions about optimum treatment duration. The only study we found in the literature that compares different treatment durations of manual therapy interventions was a study by Sluka and Wright,[26] which highlights the paucity of published research.

Comparing the findings of Sluka and Wright[26] to the current study is dubious as the treatment duration used in the present study (3×2 minutes) was less than those used by Sluka (3×3 and 3×5 minutes).[26] This may be reflected in the contrasting results as Sluka and Wright[26] found that a longer duration of knee mobilizations (3×3 and 3×5 minutes) had a significantly increased hypoalgesic effect, when compared to mobilizations of a shorter duration (3×1 minutes), on measures of paw withdrawal. The current study does not support the use of longer

neurodynamic treatment durations (3×2 minutes), and results appear to indicate that if a clinician is aiming to increase pain-restricted movement, then using a shorter treatment duration (3×1 minutes) would have a similar effect.

Although it was not the current study's primary aim to compare differences within groups (before and after), it is interesting to highlight that both treatment groups had statistically significant increases in ROM HF post-treatment. Although no previous study has investigated the increase in ROM HF that is required to be meaningful, the large increases found in the current study (3×1-minute group improving by a mean of 6.7° and the 3×2-minute group improving by a mean of 5.1°) might be suggested to be clinically significant. Current research demonstrates that neurodynamic mobilization can have a significant hypoalgesic effect[9,13,15,19–23] that might explain the increases in ROM HF found in the present study; however, it might also be the product of elastic deformation, in the form of creep and hysteresis. The current study's results are in agreement with Tal-Akabi and Rushton[13] who investigated the effects of neurodynamic treatment (ULTT2a) on active range of wrist flexion and extension in patients with carpal tunnel syndrome (CTS). The authors[13] reported that wrist flexion increased by 8° and wrist extension by 23°, which were both significantly higher than baseline measurements and when compared with a control group. The reasons for the improvement in ROM documented by Tal-Akabi and Rushton,[13] as well as in the present study, might be multifaceted; however, both neural and non-neural structures would have been affected by these techniques and, in addition, descending inhibitory pathways may have been stimulated. The improvements in ROM documented by Tal-Akabi and Rushton[13] appear to be of a greater magnitude when compared to the present study; however, this may be due to the potential differences in treatment duration, which were varied depending on the patients' symptoms (severity and irritability).[13]

It is important to note, however, that no placebo group was employed in the current study, as the primary aim was to compare different treatment durations. Although a statistically significant improvement was demonstrated, it remains unclear if this was a genuine treatment effect, and future research should remedy this by utilizing a control group.

Electromyographic magnitude of semitendinosus at P1

The results indicate that there was no significant difference in EMG magnitude of semitendinosus at P1, within or between groups. A wide range of published literature supports the hypothesis that protective antagonistic muscle activity is a normal response to neurodynamic testing[1,2,6,7,12] with those studies' results appearing to indicate that the increased activity is a result of increased pain.[11] The current findings might suggest that passive oscillating grade IV+ neurodynamic SLR treatment does not decrease EMG magnitude (facilitate muscle relaxation). However, it is important to note that ROM HF at P1 increased pre- to post-treatment in both treatment groups, and therefore, EMG magnitude recordings (taken at P1) occurred later in ROM HF. Despite this increase in ROM HF at P1, EMG magnitude data post-treatment were not significantly different from pre-treatment. This might be argued to support the efficacy of neurodynamic treatment, as EMG magnitude remained the same despite the within groups increase in ROM HF at P1. These results might also suggest that there is a normal and unchanging EMG magnitude response from semitendinosus at P1, and may support suggestions that the EMG response to pain is proportional to the magnitude of pain felt.[11]

Using the results of the present study will enable manual therapists to better understand the effects of using neurodynamic treatment. It would appear that if a clinician wants to improve pain-restricted ROM, then using a shorter duration (1 minute) of neurodynamic treatment would have a similar effect when compared to a longer treatment duration (2 minutes); however, neurodynamic treatment, irrespective of treatment duration, may help to increase pain-restricted ROM. Potential reasons for the lack of significance between different treatment durations found in the present study may be that the durations were too similar (3×1 and 3×2 minutes), with larger treatment durations needed (3×3 or 3×5 minutes) in order to reach significance. Furthermore, if neurodynamic mobilizations exert their effects through a hypoalgesic or viscoelastic response, then it may be that only a short period of time (3×1 minutes) is required for them to be effective, with little or no additional benefit thereafter (3×2 minutes). The current study's results also appear to suggest that neurodynamic treatment does not decrease protective antagonistic muscle activity, which seems to be a normal protective mechanism that remains unchanged following neurodynamic treatment.

Although the study does enhance our understanding of the effects of neurodynamic treatment, it does have a number of limitations. First, due to the study's primary aim to compare treatment durations, no control group was utilized, and therefore, it is uncertain whether the improvement in ROM HF was a real treatment effect or a measurement error. The relatively small sample size might also be suggested as a limitation, potentially resulting in false-negative results. Therefore, the non-significant findings between groups (3×1 and 3×2 minutes) on

measures of ROM HF and EMG magnitude of semitendinosus at P1, and within groups (before and after) on EMG magnitude of semitendinosus at P1 may have produced false-negative results due to the small sample size. Finally, the current study used a narrow cross-section (in terms of age) of the healthy/asymptomatic population and thus extrapolating the present findings to those with pathology must be undertaken with caution. Future research should therefore use a control group to investigate whether the reported increase in ROM HF was an effect of treatment, be adequately powered to limit the possibility of false-negative results, and use a larger cross-section of the symptomatic population.

Conclusion

The results of this study suggest that there is no additional benefit of increasing the duration of a passive neurodynamic SLR treatment on measures of ROM HF or EMG magnitude of semitendinosus at P1. It is possible that the first onset of pain may occur later in range after a passive SLR treatment, but due to the lack of a control group in the current study, this result must be interpreted with caution. Finally, the current study's results suggest that there is no difference in EMG activity of semitendinosus at P1, following either 3×1 or 3×2 minutes of passive neurodynamic SLR treatment.

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References

1 Boyd BS, Wanek L, Gray AT, Topp KS. Mechanosensitivity of the lower extremity nervous system during straight-leg raise neurodynamic testing in healthy individuals. J Orthop Sports Phys Ther. 2009;39:781–90.
2 Hall T, Zusman M, Elvey R. Adverse mechanical tension in the nervous system? Analysis of straight leg raise. Man Ther. 1998;3:140–6.
3 Boland RA, Adams RD. Effects of ankle dorsiflexion on range and reliability of straight leg raising. Aust J Physiother. 2000;46:191–200.
4 Gabbe BJ, Bennell KL, Wajwelner H, Finch CF. Reliability of common lower extremity musculoskeletal screening tests. Phys Ther Sport. 2004;5:90–7.
5 Coppieters MW, Kurz K, Mortensen TE, Richards NL, Skaret IA, McLaughlin LM, *et al*. The impact of neurodynamic testing on the perception of experimentally induced muscle pain. Man Ther. 2005;10:52–60.
6 Goeken LN, Hof L. Instrumental straight-leg raising: results in healthy subjects. Arch Phys Med Rehabil. 1993;74:194–203.
7 Goeken LN, Hof L. Instrumental straight-leg raising: results in patients. Arch Phys Med Rehabil. 1994;75:470–7.
8 Balster SM, Jull GA. Upper trapezius muscle activity during the brachial plexus tension test in asymptomatic subjects. Man Ther. 1997;2:144–9.
9 Coppieters MW, Stappaerts KH, Wouters LL, Janssens K. Abberant protective force generation during neural provocation testing and the effect of treatment in patients with neurogenic cervicobrachial pain. J Manipulative Physiol Ther. 2003;26:99–106.
10 Butler DS. The sensitive nervous system. Unley, Australia: NOI Group Publications; 2000.
11 Jaberzadeh S, Scutter S, Nazeran H. Mechanosensitivity of the median nerve and mechanically produced motor responses during upper limb neurodynamic test 1. Physiotherapy. 2005;91:94–100.
12 Van der Heide B, Allison GT, Zusman M. Pain and muscular responses to a neural tissue provocation test in the upper limb. Man Ther. 2001;6:154–62.
13 Tal-Akabi A, Rushton A. An investigation into the effectiveness of carpal bone mobilisation and neurodynamic mobilisation as methods of treatment for carpal tunnel syndrome. Man Ther. 2000;5:214–22.
14 Butler DS, Coppieters MW. Letter to the editor. Man Ther. 2007;12:e7–8.
15 Cleland JA, Childs JD, Palmer JA, Eberhart S. Slump stretching in the management of non-radicular low back pain: a pilot clinical trial. Man Ther. 2006;11:279–86.
16 Shacklock M. Improving application of neurodynamic (neural tension) testing and treatments: a message to researchers and clinicians. Man Ther. 2005;10:175–9.
17 Moss P, Sluka K, Wright A. The initial effects of knee joint mobilisation on osteoarthritic hyperalgesia. Man Ther. 2007;12:109–18.
18 Ellis RF, Hing WA. Neural mobilisation: a systematic review of randomised controlled trials with an analysis of therapeutic efficacy. J Manual Manipulative Ther. 2008;16:8–22.
19 Adel SM. Efficacy of neural mobilisation in treatment of low back dysfunctions. J Am Sci. 2011;7:566–73.
20 George SZ. Characteristics of patients with lower extremity symptoms treated with slump stretching: a case series. J Orthop Sports Phys Ther. 2002;32:391–8.
21 Schafer A, Hall A, Muller G, Briffa K. Outcomes differ between subgroups of patients with low back pain following neural manual therapy: a prospective cohort study. Eur J Spine. 2011;11:482–90.
22 Allison GT, Nagy BM, Hall T. A randomised clinical trial of manual therapy for cervico-brachial pain syndrome—a pilot study. Man Ther. 2002;7:95–102.
23 Baysal O, Altay Z, Ozcan C, Ertem K, Yologlu S, Kayhan A. Comparison of three conservative treatment protocols in carpal tunnel syndrome. Int J Clin Pract. 2006;60:820–8.
24 Bialosky JE, Bishop MD, Price DD, Robinson ME, Vincent KR, George SZ. A randomised sham-controlled trial of a neurodynamic technique in the treatment of carpal tunnel syndrome. J Orthop Sports Phys Ther. 2009;39:709–23.
25 Bialosky JE, Bishop MD, Price DD, Robinson, ME, George SZ. The mechanisms of manual therapy in the treatment of musculoskeletal pain: a comprehensive model. Man Ther. 2009;14:531–8.
26 Sluka KA, Wright A. Knee mobilisation reduces secondary mechanical hyperalgesia induced by capsaicin injection into the ankle joint. Eur J Pain. 2001;5:81–7.
27 Krekoukias G, Petty NJ, Cheek L. Comparison of surface electromyograpic activity of erector spinae before and after the application of central poteroanterior mobilisation on the lumbar spine. J Electromyogr Kinesiol. 2009;1939–45.
28 Lariviere C, Gagnon D, Loisel P. The comparison of trunk muscles EMG activation between subjects with and without chronic low back pain during flexion-extension and lateral bending tasks. J Electromyogr Kinesiol. 2000;10:79–91.
29 Hodges PW, Bui BH. A comparison of computer-based methods for the determination of the onset of muscle contraction using electromyography. Electroencephalogr Clin Neurophysiol. 1996;101:511–9.
30 Maitland G, Hengeveld E, Banks K, England K. Maitland's vertebral manipulation, 7th edn. London: Elsevier; 2009.
31 Krouwel O, Hebron C, Willett E. An investigation into the potential hypoalgesic effects of different amplitudes of PA mobilisations on the lumbar spine as measured by pressure pain thresholds (PPT). Man Ther. 2010;15:7–12.
32 Burden A. How should we normalize electromyograms obtained from healthy participants? What have we learned from over 25 years of research. J Electromyogr Kinesiol. 2010;20:1023–35.
33 Altman DG. Practical statistics for medical research. London: Chapman and Hall; 1991.
34 Hass M. statistical methodology for reliability studies. J Manipulative Physiol Ther. 1991;14:119–32.