

EFFECT OF NEURODYNAMIC TREATMENT ON NERVE CONDUCTION

THE EFFECT OF A NEURODYNAMIC TREATMENT ON NERVE
CONDUCTION IN CLIENTS WITH LOW BACK PAIN

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A Thesis Submitted to the School of Graduate Studies in Partial Fulfillment of the
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Abstract

Neurodynamics refers to the mechanical and physiological components of the nervous system and the interconnections between them (Shacklock, 1995). This is a phase 1 pilot trial investigating the immediate effect of a neurodynamic treatment as compared to a sham treatment in eight participants with low back pain. Primary outcome measures included: H-reflex latency and nerve conduction velocity. Secondary outcome measures included: the sitting slump test and visual analog scale for pain following a neurodynamic treatment compared to a sham treatment on eight participants with low back pain. T-tests were used to analyze any differences between the groups at baseline and post-intervention. No statistically significant differences were observed between the groups at baseline. Statistically significant differences were noted post-intervention between the treatment groups for H-reflex latency ($t(5)=4.323$, $p=0.008$) and the unaffected leg sitting slump test ($t(5)=3.402$, $p=0.019$). The H-reflex latency increased for the group following the neurodynamic treatment and decreased following the sham treatment. This was not expected and is of interest due to the possible mechanisms that may be underlying these phenomena. Despite the small sample size used in this study, differences were observed and displayed trends that were unanticipated. These between-group differences are of interest but require further investigation using a larger sample population. Sample size calculations for future studies based on the primary outcome measures yielded a sample of 2008 participants. This accounted for

both a 20% difference between the two groups and a 20% dropout rate. Future studies need to investigate the most beneficial length of time, type and dosage of neurodynamic treatments, as well as, the most appropriate times to assess the outcome measures. Comparison to controls would be beneficial in subsequent studies.

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List of all Abbreviations and Symbols

α - alpha

β - Beta

1a afferents – a type of nerve fiber that carries proprioceptive messages to the central nervous system

BMI – body mass index

C-fibers – a type of nerve fiber usually carrying nociceptive signals

D/c - discontinued

H:M ratio – Hoffman reflex amplitude compared to the M-wave amplitude (Palmieri et al, 2004)

H-reflex – Hoffman reflex

L5/S1 – the zygoapophyseal joint consisting of the 5th lumbar vertebra and the 1st sacral vertebra

L-spine – lumbar spine

MCID – minimal clinically important difference

MDC – minimal detectable change

M-wave/M-response – muscle response caused by electrical stimulation of peripheral nerve (Palmieri et al, 2004)

mm – millimeters

ms – milliseconds

m/sec – meters per second

n – number of people in a group

NC – nerve conduction

NCV – nerve conduction velocity

NDT – neurodynamic treatment

P4 – name of a pain measurement tool

RCT – randomized control trial

SEM – standard error of measurement

SI joint – sacroiliac joint

SLR – straight leg raise

SPSS – Statistical Package for Social Science

T12/L1 – the zygoapophyseal joint consisting of the 12th thoracic vertebra and 1st lumbar vertebra

VAS – Visual Analog Scale

Declaration of Academic Achievement

I, Diana Dawson, developed the idea for the research project, wrote the research ethics proposal, applied unsuccessfully for a grant, advertised, screened and assessed for safety and appropriateness of candidates for the study, as well as, performed two outcome measures for the study. I analyzed the data and wrote the final report.

Professor Dr. Linda Woodhouse assisted in the formulation of the research project, assisted with the research ethics proposal and grant application, and provided support and guidance during the research project and analysis as well as, editorial suggestions for the final report.

Professor Dr. Victoria Galea conducted the electrophysiological testing for the study. Additionally, she provided guidance and support during the research project and analysis, as well as, editorial suggestions for the final report and manuscript.

Professor Dr. Joy MacDermid provided guidance and support during the research project and analysis, especially statistical analysis, as well as, editorial suggestions for the final report.

Associate clinical professor Anita Gross provided guidance and support during the research project and analysis, submitted clients to the project, as well as, editorial suggestions for the final report and manuscript.

Physiotherapist, Heidi Gerber provided the treatments to the participants during the study.

Janice Cheung, Margaret Lomotam and Derek Dawson assisted with range of motion measurements during the study.

Introduction

Low back pain is a prevalent condition in society. Low back pain can refer to pain, which remains centered in the low back region, as well as, pain, which radiates from the low back to the buttocks and/or legs. Cassidy et al (1998) reported that 84.1% of adults in their study had been afflicted with low back pain at some time in their life. The point prevalence of low back pain in this population was reported as 28.4%. WorkSafeBC (2009) reported that excluding claims that did not result in loss of work time, between 2000 and 2009, back strain claims made up 23.9% of all claims submitted. Dagenais et al (2008) conducted a systematic review investigating the costs of low back pain. Direct costs of low back pain in the United States ranged from over 12 to over 90 billion dollars per year. Indirect costs in the United States ranged from over 7 to over 28 billion dollars per year. This data collection is based on 1996, 1998 and 2004 data, respectively.

As low back pain greatly impacts both individuals and society, as a whole, it is essential that physiotherapists understand the underlying mechanisms and efficacy of treatments for low back pain. Physiotherapists commonly use neurodynamic techniques to treat individuals who present with low back pain. Despite frequent use and anecdotal suggestions of clinical benefits, there is a dearth of research investigating whether the underlying physiological assumptions upon which these techniques are based are valid. The concept of neurodynamics was originally introduced by Shacklock in 1995. It refers to both

the mechanical and physiological components of the nervous system as a whole, and the interconnections between them (Shacklock, 1995). Terms such as 'adverse mechanical tension' and 'adverse neural tension' have been used in the past and continue to be used by some clinicians today. Shacklock expressed concern that these terms are too narrow in scope as they focus only on the mechanical aspects of the nervous system. In a 2005 publication, Shacklock (2005b) explained that these terms lead practitioners to believe there is a need to stretch the nerve and cited a lecture by Dr. John Marshall (1883), spoken to the Royal College of Surgeons of England, in which Dr. Marshall suggested a treatment that involved stretching a nerve for five minutes. Considering more recent findings related to the physical properties of nerve tissue and the amount of tension that a nerve can tolerate, the risk of permanent damage to the nerve with this technique is apparent (Sunderland and Bradley, 1961; Lundborg and Rydevik, 1973; Ogata and Naito, 1986).

In his explanation of the theory of neurodynamics, Shacklock (2005a) divides the body into three mechanical parts: neural structures, innervated tissues and a mechanical interface. The mechanical interface is defined as the structure(s) that surround the nervous system. The different components of the nervous system are referred to as the neural structures and the innervated tissues (defined as tissues that have a nerve supply).

The theory of neurodynamics gives consideration to both the mechanical and physiological state of the nervous system (Shacklock, 1995). As humans

survive by moving, the mechanical components of the nervous system must be resilient enough to endure tension and compression, as well as, allow movement of the nerves (i.e. sliding) (Shacklock, 2005a). Many tissues protect the peripheral nerve from damage when it is undergoing tension (Sunderland and Bradley, 1961). Sunderland and Bradley (1961) stated the importance of both the perineum and funiculi in this role. They found that a nerve that was elongated by 20% of its length, no longer had any elastic reserve. If lengthening occurred beyond this point, permanent damage could occur. As the nervous system must also be able to tolerate compressive forces, nerves may change shape as a method of enduring increased pressure and possibly allowing it to slide into a position of less compression (Nakamichi and Tachibana, 1995; Shacklock, 2005a).

Finally, McLellan and Swash (1976) performed a study in which needle electrode placement demonstrated that nerves move separately from the tissues surrounding them. A needle electrode was inserted into the median nerve of the participant's upper arm. The amount of nerve movement was calculated by measuring the length of the entire needle, as well as, both the distance moved and the length of the portion of the needle sticking out of the skin during movement of the participants' wrist, fingers, forearm, elbow, neck and thorax.

Shacklock (2005a) explains that the nervous system's state of equilibrium occurs when tension is balanced throughout the system. As such, nerves will

slide towards the area of greatest tension and must have the ability to slide both longitudinally and across their width (Shacklock, 2005a).

The physiological components of the theory of neurodynamics that Shacklock (2005a) referred to included: blood flow/oxygen supply to the nerve, nerve conduction and axonal transport. It is important to understand how intraneural blood flow is controlled to appreciate its central importance within the theory. Crucial to the comprehension of the following studies is the recognition that unmyelinated nerve fibers (C fibers) transmit nociceptive messages that are the result of mechanical input or heat, as well as, nociceptive messages that are difficult to pinpoint to a specific site (DeLeo, 2006). A study by Zochodne and Ho (1991) demonstrated that electrical stimulation of rat sciatic nerve trunks increased blood flow within the nerve. They suggested that this may have occurred secondary to the stimulation of the local unmyelinated nerve fibers causing a vasodilation of the blood vessels supplying the nerve (the vasa nervorum). Additionally, studies performed on rats by Zochodne and Low (1990) and Zochodne et al (1990) support the theory that sympathetic nerve receptors on the vasa nervorum cause vasoconstriction of these vessels. Shacklock (2005a) explained that if nociceptors of unmyelinated nerve fibers were stimulated frequently, intraneural blood flow would probably increase. Subsequently, this could result in an imbalance of vasodilation and vasoconstriction in the vessels supplying the nerve, leading to inflammation of the nerve.

Shacklock (2005a) explains if improper neural mechanics (pathomechanics) or improper neural physiology (pathophysiology) is present, then improper dynamics may ensue. This concept has been termed pathodynamics. Shacklock (2005a) states an example of this is the alteration in neural physiology that occurs in patients with diabetes. Dellon et al (1988) performed a study on rats in which they banded the rats' sciatic nerve (creating compression). The diabetic rats had reduced conduction velocity and amplitude compared to the rats without diabetes. Altered pathodynamics in individuals with diabetes results in increased susceptibility to the negative results of nerve compression (pathomechanics) and the development of peripheral neuropathies compared to healthy individuals with normal blood glucose regulation (Dellon et al., 1988; (MacKinnon, 1992; reviewed in MacKinnon and Dellon, 1988 both cited by Shacklock, 2005a)).

Pathomechanics will also affect nerve physiology (Shacklock, 2005a). Lundborg and Rydevik (1973), and Ogata and Naito (1986) performed studies to examine the response of the peripheral nerves of rabbits to tensile force and reported that the nerve became void of circulation at a mean of 15% and greater than 15.7% of increased tension (lengthening of the nerve), respectively. In addition to tolerating tensile forces, the nerve must also be able to tolerate compression. Gelberman et al. (1983) examined the effect of compression on nerve function in humans. They found that after 25 to 40 minutes of 50mmHg of compression applied to the wrists of study participants, they experienced a

complete loss of sensory function of their median nerve. An additional 15 to 20 minutes of compression following the loss of sensory function resulted in decreased motor function. Ogata and Naito (1986) also studied the effect of compression on nerves. They found that 50 to 70 mmHg of compression applied to the sciatic nerve of rabbits resulted in a total loss of circulation to that nerve.

In summary, studies have clearly demonstrated that physiological changes in nerves impact nerve mechanics, and mechanical forces sustained by a nerve affect their physiology (Shacklock, 2005a).

Literature Review

Overview of Neurodynamic Studies

There have been a number of studies that have investigated the effect of different neurodynamic techniques on a variety of outcome measures. Ellis and Hing (2008) performed a systematic review of the literature regarding the effectiveness of neural mobilization. They used the PEDro scale to evaluate the randomized control trials retrieved. Ellis and Hing (2008) reported that the few studies that met the inclusion criteria of their review did not provide strong support of neurodynamic techniques and the studies themselves were only of limited-moderate methodological quality.

Schäfer et al (2010) investigated the response to a neurodynamic treatment by seventy-seven participants who had been categorized into four

different groups by the authors. The four groups were: neuropathic sensitization, denervation, peripheral nerve sensitization and musculoskeletal. Primary outcome measures included: the Roland Morris disability questionnaire, an eleven point numerical rating scale for pain and a global perceived change scale. The peripheral nerve sensitization group showed the greatest response to treatment when compared to the other groups. The authors suggest that in research, classifying patients allows more precise recommendations regarding treatment effectiveness.

Scrimshaw and Maher (2001) investigated the effect of neurodynamic exercises as part of a post-operative treatment for participants who had had L-spine surgery. The study consisted of 2 groups, both of which performed a standard set of exercises. One of the groups' programs also included a series of neurodynamic techniques (both passive and active). No significant difference was apparent for the outcome measures investigated. It should be noted that both the participant population and the neurodynamic technique differed from that used in the present study.

Studies Investigating the Effect of a Neurodynamic Technique on

Electrophysiological Measures

If one considers the theory of neurodynamics, they would expect a mechanical input affecting the neurodynamic system to affect the physiological system (Shacklock, 1995). One of the physiological functions discussed by

Shacklock (2005a) is nerve conduction. This is commonly measured by electrophysiological testing. Bialosky et al (2009) investigated the effect of a neurodynamic technique compared to a sham treatment in forty females with carpal tunnel syndrome. One of the outcome measures was electrophysiological testing. No statistically significant difference was found. Alternately, Mahmud et al (2006) demonstrated an association between a clinical test designed to examine the mobility of the nerves and electrophysiological testing of that nerve. It is obvious there is conflicting data regarding the effect of neurodynamic techniques and electrophysiological testing. The effects of a neurodynamic technique will be further investigated in the present study.

Study Investigating the Effect of a Neurodynamic Technique on Neurodynamic Testing

Coppieters et al (2003) investigated the response to cervical mobilizations (lateral translations) in participants with “cervicobrachial pain” (nerve involvement). The group of participants who received lateral translations of the cervical spine demonstrated a statistically significant improvement for: elbow extension range of motion, intensity of pain and the area of symptoms during a median nerve test. The comparative group received ultrasound. No statistically significant improvements were observed in this group. This study examines the effects of a technique affecting the tissues surrounding the nerves on a neurodynamic test in the upper quadrant. The present study will compare a neurodynamic mobilization on a neurodynamic test in the lower quadrant.

Studies Investigating the Effect of a Neurodynamic Technique on Pain

Cleland et al (2006) investigated the effect of a 3 week program of slump stretching (30 seconds/session) combined with L-spine mobilizations and exercise in participants with non-radicular low back pain compared to a group of participants receiving only L-spine mobilizations and exercise. They found statistically significant improvements for the Oswestry Disability Index, symptom centralization and pain as scored on the numeric pain rating scale (NPRS).

Similarly, Nagrale et al (2011) performed a larger randomized control trial investigating the effect of a 3 week treatment program of L-spine mobilizations and exercise versus L-spine mobilizations, exercises and slump stretching in participants with non-radicular low back pain. The study participants were evaluated using the Oswestry disability index, numeric pain rating scale and the fear-avoidance belief questionnaire at 1,2,3 and 6 weeks after the start of treatment. All participants showed a statistically significant improvement for all the measures. The group that performed the slump stretch in addition to the standard treatment showed greater improvement than the group who received the standard treatment alone. Although the amount of improvement decreased between the third week and sixth week, all participants still showed significant improvement compared to the baseline measurements.

Both of these studies showed a greater improvement on pain scales following a neurodynamic technique in combination with a set treatment protocol

compared to the set treatment protocol, alone. Immediate pain levels will be assessed following neurodynamic treatment compared to sham treatment in the present study.

Although there are a number of studies investigating the effect of neurodynamic techniques on different outcome measures, there are few studies investigating the electrophysiological effects of neurodynamic techniques (in the low back). Neurodynamics refers to the mechanical and physiologic function of the nerve (Shacklock, 1995). “Neurodynamic techniques” used in physiotherapy are believed to affect both of these components of nerve function. The objective of the present study was to conduct a phase 1 pilot trial that would determine the short-term impact on nerve function (as indicated by H-reflex latency and nerve conduction velocity), performance of a clinical slump test and reported pain following a neurodynamic treatment technique in comparison to a sham treatment in clients with low back pain. Specifically, this study compared the physiological effects of a neurodynamic treatment technique commonly used by physiotherapists to treat patients with low back pain to a sham technique. The electrophysiological assessments for this study included H-reflexes, as well as, tibial and sural nerve conduction velocity in clients with low back pain. Stretanski (2004) clearly explained the concept on which H-reflexes are based. He stated that when the tibial nerve (at the popliteal fossa) is stimulated electrically, excitation of the afferent portion of a reflex arc that travels to the dorsal root ganglion of S1 occurs. There is a

subsequent excitation of the alpha motoneuron that causes a contraction of the soleus/gastrocs. If there is a problem with S1 nerve conduction, then the motor response will be slow or non-existent.

The results of this pilot study were used to estimate the sample size required to conduct a full RCT.

In the present study, three research questions were examined for any preliminary trends. The research questions are listed. Is there an immediate difference in H-reflex latency or tibial or sural nerve conduction following a neurodynamic technique compared to a sham treatment in clients with low back pain (males and females, aged 18 years and older)? Is nerve mobility, as measured by knee extension range of motion during a sitting-slump test, immediately altered following a neurodynamic technique compared to a sham treatment in clients with low back pain (males and females, aged 18 years and older)? Is there an immediate alteration in pain (i.e. change of Visual Analogue Pain scores) following a neurodynamic technique compared to a sham treatment in clients with low back pain (males and females, aged 18 years and older)?

The null primary hypothesis was: the application of a neurodynamic technique will not immediately alter H-reflex latency, tibial or sural nerve conduction more than a sham treatment in clients with low back pain (males and females, aged 18 years and older), as measured by H-reflexes, motor and sensory nerve conduction.

The alternate primary hypothesis was: a neurodynamic treatment technique will immediately alter H-reflex latency, tibial or sural nerve conduction more than a sham treatment in clients with low back pain (males and females, aged 18 years and older), as measured by H-reflexes, motor and sensory nerve conduction.

Methods

Ethics

The Research Ethics Board of Hamilton Health Sciences/McMaster approved this study. All participants were provided with a letter of information, and given an opportunity to ask questions. All participants signed an informed consent form agreeing to their participation and storage of data.

Study Design

The study design is a randomized, double blind controlled pilot study of participants with low back pain. There are two intervention groups, a neurodynamic treatment group and a sham treatment group.

Sample Description

This study is a pilot study and therefore had a smaller sample size than would be required for a large, confirmative, randomized control trial. The sample consisted of 8 participants. The goal was to generate data to determine the mean difference and standard deviation between the two groups. This

information can be used to calculate the sample size for a larger randomized controlled trial.

Participants

The following inclusion criteria were considered during participant selection. Participants included both males and females aged 18 years and older. They needed to have the ability to speak and read English, as well as, understand, and fill out the consent form and questionnaires. Participants had low back pain graded verbally as 4/10 or higher, on a 0-10 scale at entrance into the study, with or without radiation to the leg. A clinical decision that it was safe for the participant to perform all of the study components was determined by the same experienced clinician. This decision was based on the initial history and objective physical examination

Exclusion criteria included: the inability to tolerate sitting for 10 minutes, nerve conduction disorders diagnosed by a physician such as, diabetes or multiple sclerosis and if the individual had been a participant in an investigational drug study within the past three months.

Intervention

The neurodynamic technique involved mobilizations (small movements) to the joints of the L-spine and SI joints while the participant sat in a slumped position with both knees straight (supported), ankles dorsiflexed and neck flexed.

This type of technique is routinely performed by physiotherapists in everyday clinical practice. The sham treatment involved teaching the participant how to tighten the transversus abdominus muscle. The transversus abdominus muscle helps to support the abdominal contents (Martin, A.H., 1985) and is considered to be one of the “core” muscles that help to support the trunk. The participant was then asked to tighten the transversus abdominus muscle ten times, holding the contraction for ten seconds each time. This one-time treatment was not expected to change the outcome measures (nerve conduction tests, sitting slump test).

Outcome Measures

Primary outcome measures

Our primary outcome measures were H-reflex latency, motor and sensory nerve conduction tests pre- and post-intervention. Motor nerve conduction tests were performed on the tibial (mixed sensory/motor) nerve and sensory nerve conduction tests were performed on the sural (cutaneous sensory) nerve. The H-reflex latency tests were conducted on the tibial nerve. The peak to peak amplitude measured as a ratio of the maximum H- to the maximum M-response was also compared pre and post-intervention but it was not a primary outcome.

Secondary outcome measures

Secondary outcome measures included a neurodynamic test (the sitting slump test) and the Visual Analog Scale for pain (Huskisson, 1974; see Appendix C).

The neurodynamic test used is called the sitting slump test. Various researchers and clinicians have posited theories as to the underlying physiology responsible for the clinical changes reported with these maneuvers, for example, Petran in 1909, as cited by Woodhall and Hayes(1950), Cyriax (1942), Inman and Saunders(1942), (as cited by Butler(1991)) and Maitland(1979). The measurement of knee extension range of motion during the slump test was performed both pre- and post- intervention. The Visual Analog Scale for pain was an additional secondary measure used. The participant completed this scale both pre- and post-intervention.

Procedures

The present study was conducted at the Institute of Applied Health Science, McMaster University, Hamilton, Ontario. Recruitment was achieved through posters located at McMaster Hospital and University, a distribution of posters to a pre-established group of clinics interested in research, a family doctor, an e-mail to rehabilitation science graduate students at McMaster University, and from the study registration website (ClinicalTrials.gov).

Treatment allocation was concealed from participants, as well as, the research team except the clinician who provided the treatment. The random allocation sequence was computer-generated externally to the primary and clinical investigators, and was then e-mailed to the treating clinician. A file folder enclosing the hard copy was kept in a locked cabinet within the laboratory in the

Institute of Applied Health Sciences building. Participants were screened and if appropriate, enrolled in the study by the experienced clinician who also performed the subjective/objective examination and the secondary outcome measures. The treating clinician assigned treatment to participants by the random allocation sequence.

The investigator and experienced clinician were blinded to all outcome measure scores other than the ones they were performing at that time. Participants were blinded to all outcome measure scores other than the VAS. Participants became an assessor for an outcome measure when they completed the VAS. Pre-intervention scores were kept on a separate piece of paper from post-intervention scores or for the VAS, the pre-intervention scale was folded so it was not visible to the participant when they completed the post-intervention VAS. The treating clinician was blinded to all the pre-intervention outcome measure scores. All procedures were performed in a separate closed room with either one investigator or clinician present with the participant to ensure blinding of the other members of the research team. The participant was asked not to discuss the treatment intervention with anyone but the treating clinician, as this could affect the results of the study.

Participants were asked to avoid exercise, stimulants/depressants, pain medications and food for four hours prior to participation in the study.

Upon arrival, the consent form was reviewed and signed by the participants. Participants then completed two pain scales, the P4 (Spadoni et al, 2004; see Appendix B) and the visual analog scale. The P4 pain scale (0-40 scale) provided information regarding the pain throughout the day while the visual analog scale (0-100 mm scale) provided information on the pain at that moment in time. The subjective history was reviewed by the experienced clinician to ensure the safety of the participant.

The investigator performed baseline nerve conduction studies of the tibial and sural nerve of the participant's affected limb, using motor and sensory nerve conduction tests. This investigator performed all of the electrophysiological nerve assessments for the study. The participant's affected limb was tested. If there was not one limb more affected than the other, the dominant lower limb was tested. A Neuro-Max 1004 (XLTEK) machine was used to perform all of the electrophysiological nerve tests. The room temperature remained constant during each participant's procedure.

Procedures for the physiological nerve tests were performed as described by Kimura (1983). Electrode placement for measurement of tibial nerve motor conduction, via the medial plantar nerve, was as follows (see Figure 1, 2): two electrodes were placed on the abductor hallucis muscle belly and tendon, respectively. A ground electrode was placed posterior and distal to the medial malleolus. The tibial nerve was stimulated 1.) medial to the Achilles tendon, just superior to the top of the medial malleolus (approximately 10 cm above the

electrodes placed on the abductor hallucis) and 2.) In the popliteal space. The distance between the two sites was measured in millimeters (mm) yielding a tibial nerve conduction value in meters per second (m/sec).

For the sural sensory nerve conduction (see Figure 3), one electrode was placed distal to the lateral malleolus and a second electrode was placed along the dorsal surface of the fifth metatarsal/tarsal joint. A ground electrode was placed posterior to the lateral malleolus. The nerve was stimulated just lateral to the midline of the distal one third of the calf. Nerve conduction was measured in m/sec.

H-reflex testing was also performed. The H-reflex/M-reflex was measured on the tibial nerve (see Figure 4). Two electrodes were placed on the soleus muscle. The ground electrode was placed over the tibial tuberosity on the front of the leg just below the knee. The stimulation was applied over the tibial nerve in the popliteal fossa. The latency of the maximum H-reflex was measured in milliseconds (ms). Additionally, the H-wave amplitude was normalized using the H:M ratio, measuring peak-to-peak amplitude, in millivolts.

Following electrophysiological testing, the experienced clinician performed a neurological scan and then, if the participant was deemed safe to continue, assessed the lumbar spine (L-spine) and sacroiliac (SI) joints. Additionally, the same clinician performed a neurodynamic test to determine if the ability of the nerves to move and respond to both tensile and compressive forces had been

affected. The test used is called a straight leg raise (SLR) (See Figure 5). The initial ideas regarding specific test procedures, underlying mechanisms responsible for the clinical symptoms, and interpretations of the test findings for the SLR have been described by many different groups (Lasegue(1864), Forst(1881), De Beurmann(1884), Fajersztajn(1901), and Moutard-Martin and Parturier(1907), all cited by Woodhall and Hayes, 1950). In this test, the participant lies on his or her back and the clinician passively lifts the participant's leg with the knee straight until the response is deemed to be positive or the a priori determined limit of range of motion is reached (Breig and Troup 1979, as cited by Butler, 1991; Butler, 1991). In our study, we determined the end point to be when there was symptom reproduction or participant tolerance (i.e. pain) was reached. A positive test indicating neurodynamic involvement included: symptom reproduction and/or a non-negligible difference in hip flexion range of motion between a participant's lower limbs. In the present study, an ankle brace was used to maintain a constant position of ankle dorsiflexion. The brace was set to 110 degrees of dorsiflexion. Butler (1991) describes a number of different movements that can be used to increase the sensitivity of the test. In this study, neck flexion was used, for which Butler also referenced Cyriax (1978). The participant was asked to bend their neck to bring their chin to their chest and then bring the head back down to the bed. The purpose of this test was to determine if this maneuver affected (i.e. increased or decreased) the symptoms. Additionally, if required, hip internal rotation, applied prior to the SLR, was used

as a sensitizing movement, and neck extension was assessed to see if this changed the symptoms. An assistant observed the range of motion for hip flexion in the SLR position. Measurements were taken three times and averaged to obtain a value.

The sitting slump test was also used to determine if there was a neurodynamic issue (i.e. with the ability of the nerve to move and respond to tensile and compressive forces) (See Figure 6). This test was also used as an outcome measure and thus was performed both pre- and post-treatment. As described by Butler (1991), in the initial part of the slump test, the participant sits in a position that keeps the sacrum vertical while the upper and lower back, and then the neck are flexed forward sequentially. Each of these respective components is followed by the application of overpressure from the clinician. The participant then straightens the knee and points their foot toward their face (ankle dorsiflexion). At this point, the participant's neck is allowed to return to a neutral position. The clinician assesses the response to each additional movement. In the present study, ankle dorsiflexion was maintained throughout the entire test using an ankle brace (set at 110 degrees of dorsiflexion). Overpressure was only applied gently if at all, due to the nature of the participants' symptoms. In addition, when used as an outcome measure (knee extension range of motion was measured in this position), neck flexion was maintained. An assistant observed the range of motion for knee extension. Measurements were taken

three times and averaged to obtain a score. This test was performed on each leg.

If the ability of the nervous system to respond to these mechanical tensile forces was affected, the participant was randomly allocated to one of the two treatment groups by the treating clinician. As previously mentioned, the random allocation sequence was computer generated. If the ability of the nervous system to respond to mechanical tensile forces was not affected, the participant was not randomized to a group and their involvement in the study was complete. It should be noted that the neurodynamic testing was performed after the nerve conduction tests to ensure that the neurodynamic clinical tests performed would not alter the baseline electrophysiological test results.

Participants with altered neurodynamic testing who were continuing in the study were seen by the treating clinician who performed either the active intervention (neurodynamic technique) or sham treatment according to the randomization allocation of the participant.

Immediately following treatment, the investigator repeated the H-reflex testing and tibial and sural nerve conduction studies. Following the nerve conduction studies, the experienced clinician repeated the neurodynamic outcome measure previously described (sitting slump test). Finally, the participant completed the visual analogue pain scale again, to evaluate their pain at that moment.

Harms

Frequently during the study, participants were asked if they were tolerating the protocol. At the end of the study, only one participant's VAS score had increased (by 2 mm which is not considered a statistically significant change score). The participant reported mild soreness related to movements they did not regularly perform. No participants contacted the investigator or experienced clinician following study completion due to untoward effects.

Data Analysis

The Statistical Package for Social Sciences (SPSS) version 19 (SPSS Inc., Chicago, IL) statistical software was used for all data analyses. Descriptive characteristics of the groups at baseline were examined using univariate analyses. The means and standard deviation were calculated. The group mean for each item was compared using a two-tailed Student's independent t-test (Table 1). Additionally, two-tailed Student's independent t-tests were used to compare the effects (i.e. mean difference (change scores: pre-treatment score minus post-treatment score)) of the neurodynamic treatment versus those of the sham treatment (Table 2). The null hypothesis was rejected when $p < 0.05$.

Sample size calculations were performed using the web calculator at <http://www.stat.ubc.ca/%7Erollin/stats/ssize/n2.html> for all outcome measures. The values used for alpha and Beta were 0.05 and 0.2, respectively. The sigma

value used was the pooled standard deviation of the change scores of the two groups.

Results

Thirteen volunteers were assessed for eligibility (Figure 7). Five of these volunteers were excluded, two because they did not meet the inclusion criteria, two due to schedule conflicts and one did not attend his scheduled appointment. Eight volunteers were enrolled in the study. Four participants were allocated to each treatment group (neurodynamic treatment group or sham treatment group). All participants received their allocated treatment. The intervention was not discontinued for any of the participants and no losses due to follow-up occurred (no follow-up appointment/contact was required after the initial treatment session). All participants in both treatment groups were included in the analysis.

Participants were recruited between March 2011 and October 2011. Participants were enrolled in the study between July 2011 and October 2011.

There were no statistically significant differences found for any of the following baseline measures or demographic characteristics: SLR (affected, unaffected), P4, age, sex or leg that electrophysiological testing was performed on (Table 1). It should be noted that the female:male ratio was 7:1. Six participants had the electrophysiological testing performed on their right leg, 1 participant had it performed on the left leg and the leg was not recorded for one participant.

Analysis of the baseline data did not show a statistically significant difference between individuals in the neurodynamic treatment group and sham group for any of the outcome measures: motor nerve conduction ($t(2.19)=0.572$, $p=0.620$), sensory nerve conduction ($t(2.09)=1.090$, $p=0.385$), H-reflex latency ($t(5)=2.128$, $p=0.087$), H:M ratio($t(5)=0.924$, $p=0.398$), sitting slump test(unaffected leg knee extension)($t(5)=0.358$, $p=0.735$), sitting slump test (affected leg knee extension)($t(5)=0.598$, $p=0.576$), and visual analog scale($t(6)=0.591$, $p=0.576$) (Table 1).

Student's t-tests were used to compare change scores (pre- minus post-intervention measurements) between the neurodynamic treatment group and sham treatment group for the following items: motor nerve conduction, sensory nerve conduction, H-reflex latency, H:M ratio, knee extension in the sitting slump test, and the VAS. The data are listed in Table 2. No statistically significant difference was found for the change scores of tibial motor conduction ($t(2.01)=0.623$, $p=0.597$), or sural sensory nerve conduction ($t(5)=1.488$, $p=0.197$). A statistically significant change was observed for H-reflex latency ($t(5)=4.323$, $p=0.008$). The mean change (pre-post) for H-reflex latency was -0.92 ms for the neurodynamic group and 0.28 ms for the sham group. This indicates that the sham group's mean H-reflex latency decreased whereas the neurodynamic group's mean H-reflex latency increased (became slower). It should be noted that there was not a statistically significant difference for the H:M ratio ($t(5)=0.714$, $p=0.507$) between the two groups .

Knee extension range of motion in the sitting slump test was measured pre- and post-intervention (Table 2). The Student's t-test comparing the change scores for the neurodynamic treatment group and sham treatment group for the affected and unaffected leg were $t(5)=0.616$, $p=0.565$ and $t(5)=3.402$, $p=0.019$, respectively. A statistically significant difference (2.5 +/- 1.73 degree increase knee extension for neurodynamic group, 2.0 +/- 1.73 degree decrease knee extension for sham group) was noted for the unaffected leg.

Finally, the VAS was measured pre- and post-intervention for the two groups (Table 2). Using the Student's t-test, no statistically significant change was observed between the two groups ($t(5)=0.167$, $p=0.874$).

Sample size requirements for all outcome measures are listed in table 3. For the primary outcome measures, sample sizes were calculated using a 20% difference between the neurodynamic treatment and sham groups. The outcome measure requiring the greatest sample size was the sural nerve conduction velocity. Allowing for a 20% dropout rate, a future study powered at 0.8 would require 2008 participants. If both primary and secondary outcome measures are considered, a sample size of 2258 participants (accounting for a 20% drop-out) would be required.

Discussion

Synopsis

A statistically significant difference in H-reflex latency was found when comparing the change score (pre- minus post-intervention scores) of the neurodynamic treatment group to the sham treatment group. No statistically significant difference was found for tibial or sural nerve conduction or H:M ratio. The null hypothesis was rejected as the H-reflex latency displayed a statistically significant difference between the groups.

Analysis of secondary outcome measures, specifically, the sitting slump test for the unaffected leg demonstrated a statistically significant change when comparing the neurodynamic treatment group to the sham treatment group. The sitting slump test for the affected leg knee extension did not demonstrate a statistically significant change. The difference in VAS scores post-treatment compared to pre-treatment was not statistically significant following a neurodynamic treatment technique compared to a sham treatment. The lack of statistically significant data indicating a difference between the two treatment groups for most measures may be partially attributed to the sample size of the study. As the group of individuals studied was small, it would require a greater difference to create a statistically significant change.

The outcome measures chosen included: nerve conduction testing and H-reflexes, the sitting slump test and the VAS. Nerve conduction testing and H-

reflexes were chosen as a measure of the nerve's physiological function. The theory of neurodynamics shows the interconnection between nerve mechanics and physiology (Shacklock, 1995). In this study we attempted to measure the effect on nerve physiology, following a technique theorized to affect nerve mechanics and subsequently, nerve physiology. If a change in nerve physiological function (as measured by the electrophysiological testing) occurred following this technique compared to the sham technique, the null hypothesis would be rejected and the evidence would support the theory of neurodynamics.

Possible Mechanisms Underlying The Results

Electrophysiological Testing

The majority of change scores for the outcome measures did not show a statistically significant difference. The change score for the H-reflex latency did demonstrate a statistically significant difference between the two groups. The change score mean showed an increase in the H-reflex latency for the neurodynamic treatment group and a decrease for the sham group. This difference although statistically significant, should be accepted with caution. This was a pilot study and the sample size was small. If these results occurred in a large randomized control trial, then a possible explanation could be the tension created in the neurodynamic technique temporarily reduced blood flow causing a temporary slowing of the H-reflex (physiological function). Herrington (2006) discussed the possibility that a tensioner technique could cause nerve damage

secondary to lack of oxygen. Alternate explanations are: the neurodynamic technique used in this study reduced the sensitivity of the nervous system, was ineffective, created the opposite effect than desired or the theory that is currently accepted is either incomplete or inaccurate.

It should be noted that although the response to the neurodynamic treatment was a slowing of the H-reflex, this group experienced a greater change than the sham group. It is possible the neurodynamic treatment changed the reactivity or sensitivity of the nervous system.

The youngest participant (who was in the neurodynamic group) in the study had an increase in H-reflex latency of 0.50. Interestingly, this participant had a reduction of the M-wave latency of 2.33. This is approximately 3.5 times the next greatest decrease (from the placebo group). All other participants had an increased M-wave latency following treatment. The M-wave is not expected to change substantially between consecutive measurements. A change in M-wave latency can result if electrode placement is altered or the stimulation location is changed. In the present study, electrodes were not removed between measurements to ensure consistency and the pre-intervention stimulus location was marked with indelible ink. One questions if the greater change is related to the participant's age and nervous system responsivity or if they are an outlier and not a true representative of the population.

Although not statistically significant, the trends seen by the motor nerve conduction versus sensory nerve conduction are of interest. All motor nerve conduction speeds slowed except for one participant in the placebo group. Sensory nerve conduction increased for two of the three participants in the neurodynamic group whereas all the participants in the placebo group demonstrated decreases in sensory nerve conduction. This is interesting because as previously mentioned, the technique used in the present study is a tensioner technique and there is a possibility it temporarily reduced blood flow, explaining the slowing of the motor nerve conduction. Gelberman et al (1983) found compression to the median nerve in humans created sensation loss prior to a reduction in motor function. It is possible that the sensory nerve in our study responded more quickly to the neurodynamic technique and was beginning to show signs of recovery at the time of testing, provided that ischemia had in fact occurred.

In the present study, electrode position remained constant during both electrophysiological testing sessions. Horowitz and Krarup (1992) conducted a study examining the response of the sural nerve during electrophysiological testing. They found that the conduction of the sural nerve decreased below the knee compared to above the knee. They also found different conduction velocities between the lateral malleolus and midcalf (51.0 +/- 0.4 m/s for females, 50.6 +/- 0.4 m/s for males) and between the dorsum of the foot and the lateral malleolus (46.2 +/- 0.4 m/s). For both areas, the nerve conduction speed

decreased by 0.5 m/s for every 10 years of increased age. It is apparent that electrode placement must remain constant in test-retest experiments; and that age has an impact on sural nerve conduction velocity.

Rivner et al (1990) studied the effect of different variables, such as height and age on nerve conduction velocity and distal latency. They found that age was inversely correlated with tibial nerve conduction speed. In this study, as opposed to Horowitz and Krarup (1992), sural nerve conduction speed was not correlated with age. Additionally, age was not correlated with distal latency. In the study, height was inversely correlated with nerve conduction velocity in the leg and had a greater influence than age. This study demonstrates that the sural and tibial nerves do not react similarly to all variables. Therefore, we cannot assume they will necessarily react the same way to a neurodynamic treatment. Subsequently, a number of indications of nerve conductivity are necessary to accurately evaluate the effect of the neurodynamic technique. In the present study, we chose four different measures to increase the chance of detecting any differences between the groups.

Buschbacher (1999) found that nerve conduction velocity and amplitude (base to peak) of the tibial motor nerve to the abductor hallucis decreased with height and age. Since pre- and post-intervention measurements in the present study were performed on the same person, these variations were accounted for.

Kimura (1984) reported some areas researchers must be aware of so that inaccurate data is not obtained. Some areas that were included were: variation in temperature (as nerve conduction velocity decreases with decreasing temperature), the position of the nerve in the body (distal versus proximal) and age (nerve conduction velocity decreases as age increases). The temperature remained unchanged during the time each participant was involved in our study, electrode positioning remained the same and age was increased by approximately an hour between the two measurements of nerve conduction velocity.

Clinical Neurodynamic Test (Sitting Slump Test)

The sitting slump test was used as the measure of nerve mechanical function. It was chosen over other neurodynamic techniques, such as the SLR, because of the ease with which this technique could be applied, as well as, the fact that the measurement could be taken when the nervous system as whole, was more engaged (slump, neck flexion, knee extension, ankle dorsiflexion) (Butler, 1991). A comparison of the SLR and slump tests was performed by Walsh and Hall (2009). They conducted a study examining the correlation and agreement between the two tests. The assessment of range of motion for both the leg with and without symptoms, showed a correlation of $r = 0.64$, $p < 0.001$ and $r = 0.30$, $p = 0.05$, respectively. The correlation was stronger for the symptomatic leg compared to the asymptomatic leg.

Youdas et al (2010) conducted a study investigating the difference in hamstring length and EMG activity following hamstring stretching via two different techniques (hold-relax; hold-relax with antagonist contraction). Knee extension during both technique application and outcome measurements was performed with the participant in supine with the hip flexed to 90 degrees. The investigators reported a minimal detectable change (with a confidence interval of 95%) of 7 degrees for a knee extension change related to a change in hamstring length. The authors did not discuss the potential effect of neurodynamics. This could have contributed to the increased knee extension that was observed.

From the limited data that was obtained in the present study, it was noted that all but two measurements obtained from the neurodynamic group demonstrated an increase in knee extension during the sitting slump test. These two measurements were the same pre- and post-treatment. The placebo group demonstrated increases, decreases and no difference for knee extension range of motion during the sitting slump test. For all of the measurements, the range of values varied from an increase of 9 degrees to a decrease of 3 degrees. In Youdas et al's (2010) study, a standard error of measurement of 3 degrees was reported for knee extension range of motion measured by one investigator, using a goniometer. In the neurodynamic treatment group, three of the six measurements that changed, showed a change that was greater than 3 degrees. In the sham group, only one of the six measurements that changed, showed a change that was greater than 3 degrees.

The neurodynamic treatment group demonstrated a statistically significant difference for the unaffected leg but not the affected when compared to the sham group. The unaffected leg knee extension range of motion improved or stayed the same for the neurodynamic group while the knee extension range of motion worsened for the sham group. A small sample size and the possibility of measurement error must be considered with regards to the results. Additionally, mobility gains by the neurodynamic system are not exclusive to knee extension range of motion and the potential slack that may be gained could be absorbed by other joints such as the neck or thorax during maximum slump/flexion rather than the “affected” leg. Thus, a change in knee extension range of motion may not have been apparent. It is interesting that these findings were apparent in the unaffected leg versus the affected leg but it should be noted that the “affected” leg was the most affected leg or in the absence of symptoms, the dominant leg. In the neurodynamic group, two of the four participants’ affected leg was their dominant leg. In the sham group, one participant’s affected leg was truly affected, the other two participants’ affected leg was their dominant leg. Due to both time constraints and consideration of the participants’ pain tolerance, only the “affected” leg was tested electrophysiologically. It would be of interest to know if there were electrophysiological changes post-treatment in the unaffected leg.

Pain Scale (Visual Analog Scale)

The VAS was used as a measure of pain. It was chosen over other pain scales, such as, the P4 because the participant could rate their pain at that moment in time. Therefore, if a change occurred post-treatment compared to pre-treatment, it would be noted by the change in the VAS score. Hägg et al (2003) conducted a study aimed at estimating the minimal clinically important difference (MCID), the error of measurement and clinical meaning of the change scores of 4 different outcome measures for clients with chronic low back pain. One of these measures was the VAS. The VAS was estimated to have a MCID of 18-19 mm, but the researchers suggested using a value of about 20 mm in future studies. The researchers estimated the standard error of measurement (SEM) of the VAS to be 6 mm (95% “tolerance interval” 15 mm), meaning the MCID of 18-19 mm falls outside of the error of measurement associated with the VAS. This makes the VAS an effective tool for evaluating change in clients with low back pain. The other three tools evaluated did not achieve a substantial separation, if any, between the MCID and the 95% tolerance interval for the standard error of measurement (SEM). In the present study, it should be noted that although not statistically significant, six of the seven completed pre- and post VAS had lower scores following treatment. Despite this, only one participant in the sham group experienced a clinically important difference as measured by the VAS (27mm change). The lack of statistically significant change may be partially attributed to the fact that participants underwent a through assessment prior to

treatment. Directly after assessment and treatment, clients can experience a temporary increase in pain related to treatment soreness. Overall, this did not seem to be the trend.

Sample Size Calculations

Sample size calculations for future studies yielded large sample sizes. This is due in part, to the greater variability in the values from electrophysiological testing resulting in large standard deviations around the means. Subsequently, to ensure the difference between the groups is not due to the variability of the measure, itself, either the sample size or the difference between the groups has to be large. For example, if sample size for the primary outcome measures was calculated at a 40% difference between the groups, rather than a 20% difference, the sample size required would decrease from 2008 participants to 503 participants.

The underlying mechanism of nerve function is important in the practice of physiotherapy. It is important we have knowledge of the theories on which nerve function is based and that these theories are supported by evidence from research. Part of the intent of this study was to gain evidence that would either support our current understanding of nerve function or encourage the exploration of new ideas.

Clinically, it is important for a physiotherapist to understand the underlying concepts of the theory of neurodynamics, so that they are fully aware of the

impact of their techniques on the nervous system, mechanically and physiologically (Shacklock 1995, 2005a). The awareness that the physiology of the nerve will affect its mechanics, is an important concept when gauging a client's response to treatment and evaluating its effectiveness (Shacklock, 1995, 2005a). In broader terms, the realization that a neurodynamic technique affects the nervous system as a whole, rather than simply the local structures, allows one to appreciate the global impact of these techniques (Shacklock, 2005a). As explained by Shacklock (2005a), an understanding of these concepts during assessment, allows the recognition that the presenting problem may very much be affected by neurodynamic input from a previous injury located elsewhere in the body. Awareness of these interconnections and subsequent impact on the nervous system allows one to assess and treat injuries more holistically.

Comparative Literature

As previously mentioned, there are few studies that investigate the effect of a neurodynamic treatment on electrophysiological testing. Research in this area would help to broaden our understanding and knowledge of the relationship between nerve mechanics and physiological function.

Electrophysiological Studies

Mahmud et al (2006) reported a correlation between a median nerve bias neurodynamic test and nerve conduction studies. The sample size in this study consisted of 38 participants.

Kerr et al (2002) studied the response in α -motoneuron excitability of both subjects with and without altered neural tension. They found the group without altered neural tension demonstrated a decrease in α -motoneuronal excitability when in slump and slump with neck flexion. The group with altered neural tension did not demonstrate statistically significant changes in α -motoneuronal excitability but the change noted was actually an increase in α -motoneuronal excitability. This impacts the choice of control subjects in future research as based on these study results, those with and without altered neural tension may react differently.

Dishman and Bulbulian (2000) investigated the effect of lumbosacral mobilizations and manipulations on the tibial H-reflex in participants without low back pain. They found that following both spinal mobilizations and manipulations, there was a temporary decrease (30 seconds) in H-reflex amplitude of the gastrocnemius. In the present study, both groups had spinal mobilizations performed during the assessment. Therefore, the effect of this would be standardized across all participants but it may somewhat affect the ability to find differences between the two groups. In addition, the neurodynamic treatment group had spinal mobilizations (in a position of neural tension) immediately prior to electrophysiological testing. One question is if this may have contributed to the increased latency of the H-reflex. Amplitude of the H-reflex measures the “excitability” of the tibial motoneurons (Schieppati, 1987). As this has been

shown to temporarily decrease with spinal mobilizations (Dishman and Bulbulian, 2000), it is possible that the speed of the H-reflex would decrease, as well.

As previously mentioned, Bialosky et al (2009) conducted a randomized control trial investigating the effect of a neurodynamic technique compared to a sham neurodynamic technique in forty female patients with carpal tunnel syndrome. The sham technique was thought to decrease median nerve tension. All patients were given a splint to be worn at nighttime and during aggravating activities. Outcome measures included pain (on a numeric rating scale), the Disability of the Arm, Shoulder, and Hand Questionnaire, grip strength, sensation (pain, temperature), electrophysiological testing including distal onset latency and peak amplitude of the abductor pollicis brevis muscle (motor) and the combined sensory index. After 3 weeks of treatment, the only significant difference between the two groups was a mean decrease of temporal summation (for thermal-induced pain) in the neurodynamic treatment group whereas the sham group experienced a mean increase of temporal summation (thermal-induced pain). The investigators stated a number of study limitations. One of the limitations is that the brace that was provided may have somewhat limited the differences that could be attributed to the neurodynamic and sham treatments. Additionally, I question if the sham technique is truly a sham, as not all neurodynamic techniques are designed to increase tension in the nerve (i.e. slider techniques described by Shacklock (2005a) do not create significant increases in nerve tension).

Neurodynamic Testing Studies

Herrington (2006) studied the effects of two techniques (tensioner, slider) on knee extension in the slump position in females aged 19-24. Shacklock (2005a) defines a tensioner as a technique, which causes an increase in tension along the nervous tissue within the nervous tissues' elastic limit. He defines a slider as a technique that causes the nervous tissue to slide in relation to the surrounding tissues. Herrington (2006) found the average improvement of knee extension in the slump of 3.4 degrees +/- 2.5 degrees for the tensioner technique and 4.3 degrees +/- 2.6 degrees for the slider technique. One researcher performed all the measurements. The technique used in the present study would be considered a tensioner technique.

Szlezak et al (2011) investigated the effect of unilateral posterior-anterior mobilizations of T12/L1-L5/S1 facet joints on a same-sided unilateral SLR in participants without significant low back problems. The mobilization treatment group was compared to two other groups: one receiving no treatment and the other, undergoing stretching of the posterior muscles of the leg (as per their SLR testing protocol). A statistically significant increase in range of motion on the unilateral SLR was found for the mobilization treatment group. No statistically significant difference was observed in the other groups. Szlezak et al hypothesized this difference was related to neurodynamic changes. As all participants in the present study had mobilizations of their L-spine and sacrum

during assessment, this may have affected the results of those in the sham treatment group and made differences between the two groups less apparent.

Pain Studies

Cleland et al (2006) and Nagrale et al (2011) both found greater improvements on a pain scale following a program that utilized neurodynamic techniques compared to one which did not. In the present study, we did not find statistically significant improvements in post-treatment pain levels. A different pain scale was used in the former two studies compared to the present study. In addition, the former studies investigated pain relief after a longer period of treatment whereas the present study investigated immediate pain relief. Treatment soreness must be considered when evaluating immediate pain relief.

As previously mentioned, Schäfer et al (2010) categorized participants into different groups and assessed their response to neurodynamic treatment. Based on the algorithm and description provided by Schäfer et al (2010), and the tests we performed during the lower quadrant scan, all but one participant in the present study would be classified in the peripheral sensitization group. Only one participant did not meet the criteria for the peripheral sensitization group but this may have been a result of the participant's inability to relax during testing. Pinprick sensation and nerve sensitivity to palpation were not tested in the present study as they were in Schäfer et al (2010). According to the findings of Schäfer et al (2010), since the majority of participants in the present study would

be classified in the peripheral sensitization group, they may have had a greater chance to respond to treatment. In the present study, the VAS was used to assess pain. Similarly, Schäfer et al (2010) used a numeric rating scale to assess pain. No statistical significant difference was found for pain following the neurodynamic treatment in the present study. Conversely, in Schäfer et al (2010), there was a significant difference in pain between the peripheral sensitization group and two of the other three groups once baseline differences had been accounted for.

No other studies were found to investigate the effect of a neurodynamic treatment to the low back compared to a sham treatment on the combination of these three measures: electrophysiological testing, a neurodynamic outcome measure (sitting slump test) and the visual analog scale.

Limitations

As with all scientific research, the present study has limitations involving the design as well as the extrapolation of the results. Limitations involving the study methodology include: the fact that the individuals in the sham group received some mobilizations to their nervous system solely by the testing of neurodynamic involvement and the sitting slump outcome measure. Additionally, the way that a clinician in practice may test for neurodynamic involvement may include techniques similar to the treatment the neurodynamic group received. This could not be performed as it could have altered the results of the sham

group. Thus, it is possible an individual with neurodynamic involvement may have tested as a false negative and been unnecessarily excluded from the study.

As previously mentioned, the sample size for the present study was quite small. As a result, for a statistically significant difference to be observed, a greater change would have had to occur. In other words, more statistically significant differences may have been found, if the sample size had been larger. An additional limitation related to our sample is that our sample population did not equally represent both males and females (1:7, male:female ratio).

The sample population included individuals 18 years or older with low back pain (with or without radiation to the leg(s)). The chronicity of the participants' back pain varied from 6 months to greater than 24 years. Flor et al (1997) found that patients with chronic back pain demonstrated greater changes in both cortical representation and responsiveness to back-specific stimuli related to the length of time the condition had been present. Therefore, it may be easier to detect overall change in a more homogenous sample of individuals with shorter pain chronicity.

A potential methodological limitation is the ankle brace. The ankle brace was used to maintain the ankle in 110 degrees of dorsiflexion. This could create a problem if an individual does not have this amount of dorsiflexion or if they had excess of this amount and the neurodynamic system was not tensioned to its elastic limit. Potentially, this could have altered our ability to detect a change in

knee extension range of motion. An effort was made by the experienced clinician using the ankle brace to create the best possible fit of the brace using the adjustable straps, while maintaining the pre-set dorsiflexion.

Finally, electrophysiological testing was performed on the tibial and sural nerves. If there was a compromise of a different nerve or contributing nerve root, the effect of the neurodynamic technique may not have been apparent in the electrophysiological testing that was completed.

In terms of consistency, two participants did not have their symptoms altered by the change of neurodynamic positions (i.e. neck flexion on/off) but as the original technique produced pain, they were counted as having a neurodynamic component to their pain. After the enrolment of these participants, hip internal rotation and neck extension were used if necessary to further sensitize the system. The subsequent participants' pain was changed by using these additional techniques.

For outcome measures, not all measures could be obtained for the electrodiagnostic testing for all participants. For the sitting slump test, the data for one participant could not be analyzed. This was a result of missing data, in terms of which leg (right/left) was the affected side. Finally, one post-treatment VAS was not obtained.

Measurement error is a limitation of all the outcome measures. In an attempt to control this, the investigator chosen to perform the electrophysiological

testing had approximately twenty years of experience testing electrophysiological measures. Additionally, electrodes were not removed between electrophysiological testing to ensure consistency and permanent marker was used to delineate the area that was electrically stimulated. Participant tolerance of the electrical current, as well as, fear of pain may also have affected measurement accuracy. Additionally, the depth at which the nerve is positioned, specifically, the amount of adipose tissue present will affect the ease with which electrophysiological measures can be detected (Poinier and Chalk, 1995-2011). Finally, the machine itself, could create measurement error but no indication of malfunction was apparent.

Knee extension and hip flexion range of motion measurements were performed by an assistant to increase accuracy with proper positioning of both the participant and the individual performing the measurements. The experienced clinician trained the assistants and supervised the range of motion measurements throughout the study. For each measure, the mean of three measurements was used for analysis. An exception to this was, a single measurement and the mean of two measurements for hip flexion during the SLR was used for one participant (right and left leg, respectively). All other participants' range of motion measurements used in analysis were a mean of three measurements. Additionally the order of movements to assume the sitting slump position was performed the same way, each time.

The ability of the participant to evaluate pain via the VAS may have resulted in measurement error but as the VAS was performed both pre-and post-intervention, the potential for error would have been present during both measures.

There were some limitations with regards to risk of bias. Firstly, the participant was not blinded to the intervention, although they were blinded to whether it was the neurodynamic treatment or sham treatment. Additionally, the clinician providing the treatment was not blinded to which intervention they were administering. In terms of outcome assessors, all were blinded to the intervention except the participant, (who became an assessor when they completed the VAS). Finally, although the investigator, experienced clinician and participant were blinded to the results of the pre-treatment outcome measures when performing post-treatment outcome measures, it is possible they may have known the previous results from memory alone.

Only one of the electrophysiological measures tested showed a statistically significant difference following a neurodynamic technique compared to a sham treatment. Based on the theory, it was hypothesized that addressing the nerve mechanics would improve nerve conduction (physiological function). During this study, we investigated the immediate effects of a neurodynamic treatment, therefore, it was performed at only one session. Clinically, this technique would normally be applied over a number of sessions. Change may not be detectable until the technique has been performed a number of times.

Future Research

There are many considerations for future research. In the future, I would suggest initially using a small sample size of approximately 12 participants (accounting for a 20% drop-out rate) with all outcome measures being repeated over time. This would allow for an increased number of measures with decreased biological variability, as the same participants would be repeatedly tested. The study would investigate the effect of six-eight weeks of neurodynamic treatment techniques in participants with a neurodynamic component to their low back pain. The longer study design is important because in clinical practice, a change is not always apparent after a single treatment. As part of the longer study design, home neurodynamic mobility exercises would be incorporated to encourage continued gliding/movement of the nervous tissue. This is typical of clinical practice. This study would help to estimate the length of time that neurodynamic treatments may be required to observe a change, as well as, the appropriate time points at which to assess outcome measures. Subsequently, studies could be performed investigating the type (tensioner versus slider) and dosage of neurodynamic treatments required to create change. Once these criteria are established, a study with a larger sample size (2258 participants, from our calculations) could be conducted to investigate the effect on a neurodynamic treatment group compared to a control group with low back pain. The control group would not receive treatment but would complete

outcome measures at the respective time periods. This would allow a comparison to the natural progression of the condition.

Once these baseline studies were completed, it would be important to consider comparison to healthy controls. It is important to compare the effects of these treatments in those with and without the symptoms to distinguish if a change in nerve conduction, for example, is a normal response for all or just in those in whom the nerve function was previously limited. Furthermore, investigation is required to determine if any potential changes in nerve mechanics or physiology is truly a result of the treatment technique improving nerve function.

Methodologically, I suggest using a fixation device to maintain pelvis/hip position as used in Herrington (2006) to further ensure standardized positioning during the sitting slump test pre- and post-intervention.

The use of a functional scale in addition to the VAS (pain scale), would provide useful information regarding the impact of the different treatments on daily activities. I would suggest the use of all the electrophysiological measures used in the present study as one cannot assume that any of the measures will respond in a similar manner. Potentially, a younger and more homogenous sample population may allow for group differences to be more easily detected secondary to both the speed of nervous system conduction (Rivner, 1990) and less within group differences.

Conclusion

It was hypothesized that altering nerve mechanics via a neurodynamic treatment would have a direct effect on nerve physiology, as measured by electrophysiological testing. This was observed in the H-reflex measurements at a statistically significant level. However, the response was the opposite of what was initially expected. Furthermore, although not statistically significant, the motor and sensory nerves did not respond in a similar fashion to the respective interventions. A statistically significant difference was observed for the unaffected leg knee extension range of motion during the sitting slump test. These statistically significant differences should be accepted with caution. This was a pilot study, and accordingly, the sample size was small. Sample size estimates were calculated for a full-randomized control trial of similar design. Additional suggestions for other preliminary studies were also made. Possible explanations for the present study's findings have been discussed, but ultimately further research is required. The question of short-term versus long-term results of neurodynamic treatment, the appropriate type and dosage of treatment as well as timelines of treatment and re-assessment all require further investigation. The observation of trends in the data as well as the estimates for sample sizes for a larger randomized control trial can assist researchers in the planning of future studies.

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Appendix A: Glossary of Terms

Mobility of nerves: ability of nerves and nervous tissue to slide/move

Tensile strength: Maximum stress a nerve can withstand while being stretched or pulled before breakdown occurs (opposite of compressive strength)

Compressive strength: Maximum stress a nerve can withstand while being squeezed or squished before breakdown occurs

Appendix B: P4 Pain Instrument (Spadoni et al, 2004)

Title of Study: The Effect Of A Neurodynamic Treatment On Nerve Conduction In Clients With Low Back Pain

Principal Investigator: Diana Dawson, Rehabilitation Science Master's Student

Co-Investigator: Dr. Linda Woodhouse/Dr. Vickie Galea, McMaster University

P4 Instrument

When answering these questions, think only of the pain you are experiencing in relation to the problem for which you are having treatment.

Circle 1 number for each of the 4 questions.

On average, how bad has your pain been:

	No Pain										Pain as Bad as it Can Be
In the morning over the past 2 days?	0	1	2	3	4	5	6	7	8	9	10
In the afternoon over the past 2 days?	0	1	2	3	4	5	6	7	8	9	10
In the evening over the past 2 days?	0	1	2	3	4	5	6	7	8	9	10
With activity over the past 2 days?	0	1	2	3	4	5	6	7	8	9	10

(Spadoni et al, 2004)

Included with permission from Gregory Spadoni.

Appendix C: Visual Analog Scale

Participant ID: _____

Visual Analog Scale (question modified from Huskisson, 1974)

Title of Study: The Effect Of A Neurodynamic Treatment On Nerve Conduction In Clients With Low Back Pain

Principal Investigator: Diana Dawson, Rehabilitation Science Master's Student

Co-Investigator: Dr. Linda Woodhouse/Dr. Vickie Galea, McMaster University

Post-Intervention

How severe is your pain right now? Place a vertical mark on the line below to indicate how bad you feel your pain is right now.



Pre-Intervention

How severe is your pain right now? Place a vertical mark on the line below to indicate how bad you feel your pain is right now.



Figure One: Electrode Placement for the Measurement of the Tibial Motor Nerve Conduction



Figure Two: Electrode Placement for the Measurement of the Tibial Motor Nerve Conduction (II)



Figure Three: Electrode Placement for the Measurement of the Sural Sensory Nerve Conduction



Figure Four: Electrode Placement for the Measurement of the H-reflex



Figure Five: The Straight Leg Raise Test



Figure Six: The Sitting Slump Test



Figure Seven: Flow Diagram of a Phase 1 Pilot Trial Comparing a Neurodynamic Treatment to a Sham Treatment

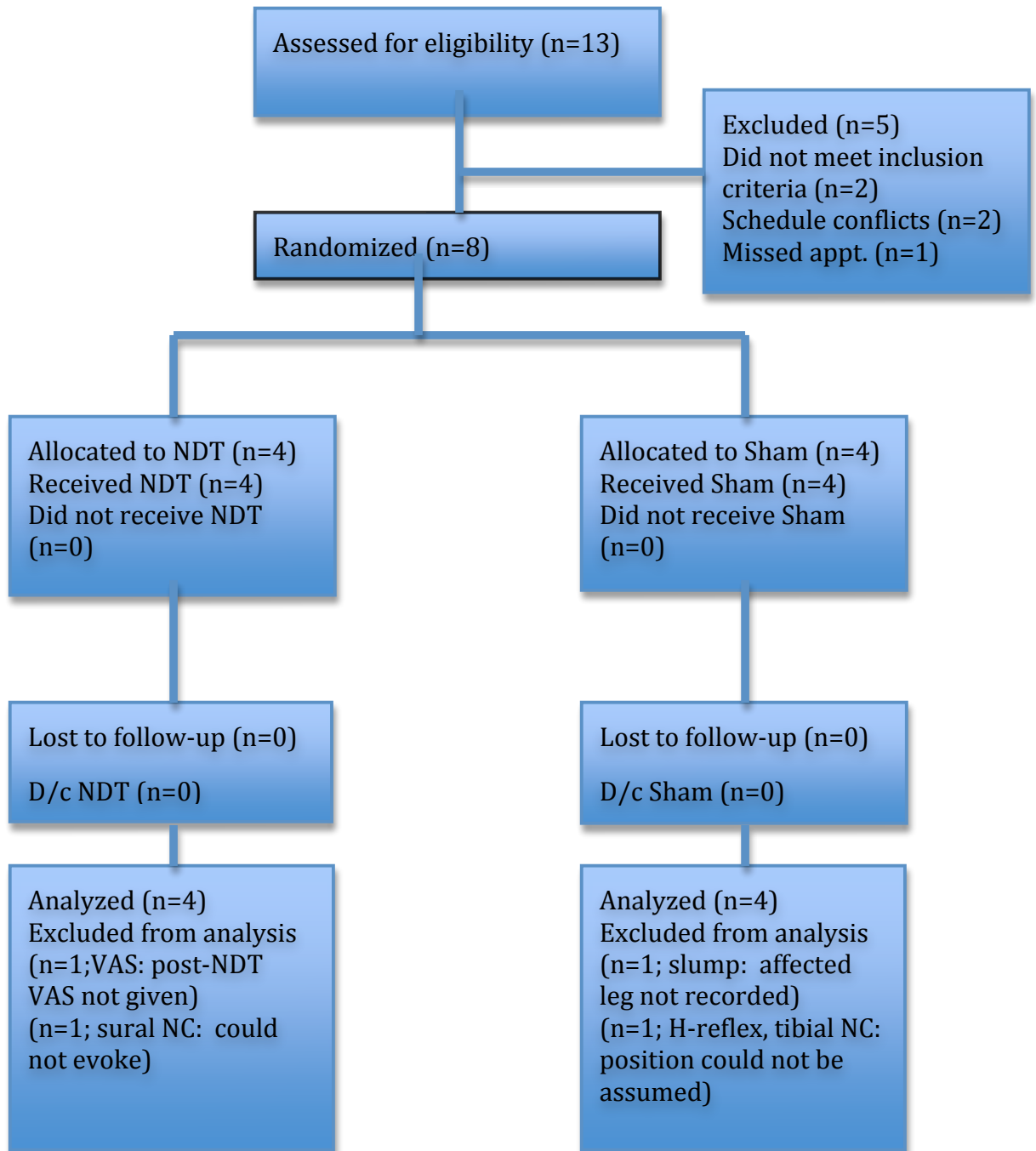


Table One: Baseline Demographics and Outcome Measures

<u>Characteristic</u>	<u>NDT Baseline</u> Mean; n=4 or 3*	<u>Sham Baseline</u> Mean; n=4 or 3*	<u>t-test; p-value</u>
Age	39.50 (17.46)	54.50 (13.96)	t(6)=1.342; 0.228
Sex	4 females	3 females; 1 male	t(3)=1.000; 0.391
Leg Tested	4 right	2 right; 1 left; 1 NA*	t(2)=1.000; 0.423
P4 Scale	25.25 (5.91)	17.00 (8.52)	t(6)=1.591; 0.163
SLR (Affected)	62.75 (20.84)	69.33 (23.97)*	t(5)=0.389; 0.713
SLR (Unaffected)	63.50 (19.49)	71.67 (23.97)*	t(5)=0.500; 0.638
H-reflex latency	29.30 (1.09)	32.17 (2.46)*	t(5)=2.128; 0.087
H:M Ratio	0.73 (0.26)	0.48 (0.46)*	t(5)=0.924; 0.398
Tibial motor NCV	49.70 (3.24)	54.03 (12.82)*	t(2.19)=0.572; 0.620
Sural sensory NCV	51.17 (12.36)*	43.30 (2.17)	t(2.09)=1.090; 0.385
Slump (Affected)	172.00 (5.10)	168.67 (9.71)*	t(5)=0.598; 0.576
Slump (Unaffected)	171.75 (5.25)	169.33 (12.42)*	t(5)=0.358; 0.735
Visual Analog Scale	4.10 (2.01)	3.13 (2.62)	t(6)=0.591; 0.576

Table Two: Outcome Measures and H:M Ratio Change Score Data

Outcome	NDT Post Mean (SD) n=4 or 3*	Sham Post Mean (SD) n=4 or 3*	NDT Mean Diff (SD) n=4 or 3*	Sham Mean Diff (SD) n=4 or 3*	T-test p value
H-reflex latency	30.21 (1.33)	31.89* (2.28)	-0.92 (0.35)	0.28* (0.38)	t(5)=4.323 p=0.008
H:M ratio	0.67 (0.18)	0.31* (0.20)	0.06 (0.13)	0.17* (0.26)	t(5)=0.714 p=0.507
Tibial motor NCV	47.15 (3.72)	47.13* (1.72)	2.55 (0.79)	6.90* (12.08)	t(2.01)=0.623 p=0.597
Sural sensory NCV	47.43* (1.36)	49.28 (6.04)	3.73* (11.26)	-5.98 (6.10)	t(5)=1.488 p=0.197
Slump (Affected)	176.00 (2.31)	171.00* (8.72)	-4.00 (4.24)	-2.33* (2.08)	t(5)=0.616 p=0.565
Slump (Unaffected)	174.25 (4.35)	167.33* (13.43)	-2.50 (1.73)	2.00* (1.73)	t(5)=3.402 p=0.019
Visual Analog Scale	3.83* (2.14)	2.23 (2.89)	1.03* (0.57)	0.90 (1.27)	t(5)=0.167 p=0.874

Table Three: Sample Size Calculations

Outcome Measure	Participants/group	Total (2 groups)	Total (accounting for 20% drop-out)
H-reflex latency	660	1320	1650
Tibial motor nerve conduction	485	970	1213
Sural sensory nerve conduction	803	1606	2008
VAS	5	10	13
Sitting slump (unaffected leg)	295	590	738
Sitting slump (affected leg)	903	1806	2258