Treatment Effects of Spinal Manipulation on Proprioception in Subjects with Chronic Low Back Pain

by

Kenneth Edward Learman

BS, State University of New York at Buffalo, 1989

M.Ed., The Pennsylvania State University, 1992

Submitted to the Graduate Faculty of

School of Health and Rehabilitation Science in partial fulfillment

of the requirements for the degree of

Doctor of Philosophy

University of Pittsburgh

2007
UNIVERSITY OF PITTSBURGH
SCHOOL OF HEALTH AND REHABILITATION SCIENCE

This dissertation was presented

by

Kenneth Edward Learman

It was defended on

May 31, 2007

and approved by

Scott M. Lephart, PhD, ATC, Associate Professor and Chair,
Department of Sports Medicine and Nutrition, Univeristy of Pittsburgh

Chad Cook, PhD, PT, MBA, OCS, FAAOMPT, Assistant Professor,
Department of Community and Family Medicine, Director of Outcomes Research, Center of
Excellence in Surgical Outcomes, Duke University

Timothy Sell, PhD, PT, Assistant Professor,
Department of Sports Medicine and Nutrition, University of Pittsburgh

Joseph B. Myers, PhD, ATC, Assistant Professor,
Department of Sports Medicine and Nutrition, University of Pittsburgh
Dissertation Director
Treatment Effects of Spinal Manipulation on Proprioception in Subjects with Chronic Low Back Pain

Kenneth Edward Learman, PhD

University of Pittsburgh, 2007

Low back pain is a prevalent problem afflicting approximately 80% of the population during their lives. Subjects with back pain demonstrate deficits in trunk proprioception. Spinal manipulation is a treatment with known effects in pain control, increased motion and other neurophysiological effects. The purpose of this study was to examine the treatment effects of spinal manipulation on trunk proprioception in subjects with chronic low back pain (CLBP) and to determine if those effects lasted one week.

Thirty-three subjects with CLBP, aged 24-54 years participated in this unbalanced, randomized controlled crossover design. Subjects presented for two or three testing sessions and agreed to a general physical examination followed by proprioception testing with joint position sense (JPS), threshold to detect passive motion (TTDPM), direction of motion (DM) and force reproduction (FR). After proprioception testing, each subject received either a lumbar manipulation or a sham procedure followed by retesting of proprioception. This procedure was repeated the following week using the opposite treatment. Those subjects receiving spinal manipulation in the second session returned for a third session and received the sham procedure a second time.

Spinal manipulation produced a significant effect for TTDPM in the Manip 1st Group, the sham procedure produced a significant immediate effect for JPS in the Sham 1st Group, and manipulation resulted in a significant one-week residual effect for the Manip 1st Group. All other
time comparisons were not significant. The results of this study minimally support the proposed hypotheses.

The results of this study suggest that spinal manipulation has minimal effect on trunk proprioception in subjects with CLBP who are painfree at the time of testing. Subjects in this study did not demonstrate as large a deficit in proprioception as previously reported. This study might suggest that a lack of demonstrable deficit in painfree subjects at the time of testing is the result of diminished pain level.

Strong conclusions cannot be made from these results but suggest further testing comparing manipulation with no intervention or other interventions while controlling for pain level, may be important for understanding the functional implications of the neurophysiological effect of spinal manipulation.
# TABLE OF CONTENTS

**PREFACE** ...................................................................................................................................... xi

1. **INTRODUCTION** ................................................................................................................ .. 1
   1.1. Societal Impact of Low Back Pain .................................................................................. 1
   1.2. Problem of Low Back Pain ............................................................................................. 2
       1.2.1. Mechanisms of Low Back Pain .............................................................................. 3
       1.2.2. Predictors of Low back Pain ................................................................................... 3
       1.2.3. Deficits in Low Back Pain ...................................................................................... 4
       1.2.4. Passive structures .................................................................................................... 6
       1.2.5. Muscular ................................................................................................................ . 6
       1.2.6. Neuromuscular Control ........................................................................................... 7
       1.2.7. Proprioception......................................................................................................... 8
   1.3. Treatment for Low Back Pain......................................................................................... 9
   1.4. Effects of Spinal Manipulation ..................................................................................... 10
   1.5. Definition of the Problem ............................................................................................. 12
   1.6. Purpose ......................................................................................................................... 12
   1.7. Specific Aims and Hypotheses ..................................................................................... 13

2. **REVIEW OF LITERATURE** ............................................................................................... 15
   2.1. Societal Impact of Low Back Pain................................................................................ 15
       2.1.1. Incidence and Prevalence...................................................................................... 15
       2.1.2. Recurrence Rates .................................................................................................. 16
       2.1.3. Cost .................................................................................................................... ... 17
   2.2. Problem of Low Back Pain ........................................................................................... 18
       2.2.1. Mechanisms of Low Back Pain ............................................................................ 18
       2.2.2. Risk Factors and Predictors .................................................................................. 19
       2.2.3. Natural Course of Low Back Pain ........................................................................ 20
   2.3. Deficits with Low Back Pain ........................................................................................ 21
       2.3.1. Structural.............................................................................................................. . 21
       2.3.2. Muscular ............................................................................................................... 22
           2.3.2.1. Decreased Cross-Sectional Area of Select Muscles ..................................... 23
           2.3.2.2. Decreased Multifidus and Erector Spinae Strength...................................... 24
           2.3.2.3. Decreased Erector Spinae and Quadratus Lumborum Endurance................ 24
       2.3.3. Neuromuscular Control......................................................................................... 25
       2.3.4. Proprioception....................................................................................................... 26
       2.3.5. Proprioception and Trunk Position ....................................................................... 34
       2.3.6. Weight Bearing Asymmetry ................................................................................. 35
   2.4. Treatment for Low Back Pain....................................................................................... 35
       2.4.1. Mobilization and Manipulation............................................................................. 38
   2.5. Effects of Manipulation ................................................................................................ 39
   2.6. Methodological Considerations .................................................................................... 43
       2.6.1. Assessment of Proprioception............................................................................... 43
2.6.1. Joint Reposition Sense ......................................................................................... 44
2.6.2. Passive Kinesthesia .............................................................................................. 45
2.6.3. Sense of Tension .................................................................................................. 46
2.6.4. Duration of Testing .............................................................................................. 46
2.6.5. Cavitation ............................................................................................................. 46
2.6.6. Muscle Thixotropy ............................................................................................... 47
2.7. Summary ................................................................................................................... 47
3. METHODOLOGY ............................................................................................................ 49
3.1. Experimental Design ............................................................................................... 49
3.1.1. Dependent Variables ......................................................................................... 49
3.1.2. Independent Variables ...................................................................................... 49
3.2. Subject Characteristics ......................................................................................... 49
3.2.1. Inclusion Criteria ............................................................................................... 50
3.2.2. Exclusion Criteria ............................................................................................. 51
3.3. Power Analysis ....................................................................................................... 51
3.4. Subject Recruitment ............................................................................................... 52
3.5. Instrumentation ...................................................................................................... 52
3.5.1. Biodex Isokinetic Dynamometer ......................................................................... 52
3.5.2. Oswestry Disability Index .................................................................................. 54
3.5.3. Visual Analog Scale – 24 .................................................................................. 55
3.5.4. Numeric Rating Scale ....................................................................................... 56
3.5.5. Fear and Avoidance Behavior Questionnaire .................................................... 56
3.6. Testing Procedures ................................................................................................ 57
3.6.1. Pain and Disability Questionnaires .................................................................... 57
3.6.2. Subject Preparation ............................................................................................ 57
3.6.3. Order of Testing ................................................................................................ 58
3.6.4. Trunk Proprioception ....................................................................................... 58
3.7. Treatment ................................................................................................................ 62
3.8. Data Analysis ......................................................................................................... 65
3.8.1. Data Reduction .................................................................................................. 65
3.8.1.1. Proprioception ............................................................................................ 65
3.8.1.2. Oswestry Disability Index ........................................................................... 66
3.8.1.3. Visual Analog Scale – 24 ........................................................................... 66
3.8.1.4. Numeric Rating Scale ................................................................................ 66
3.8.2. Statistical Analysis ............................................................................................. 66
3.8.2.1. General Characteristics ............................................................................ 66
3.8.2.2. Proprioception ........................................................................................... 67
4. RESULTS ..................................................................................................................... 69
4.1. Instrumentation ....................................................................................................... 70
4.2. Group Characteristics ............................................................................................ 70
4.2.1. General Demographic Characteristics ................................................................ 70
4.2.2. Pain and Disability Measures ........................................................................... 71
4.2.2.1. Oswestry Disability Index ......................................................................... 72
4.2.2.2. Visual Analog Scale – 24 ........................................................................ 72
4.2.2.3. Numeric Rating Scale ................................................................................ 73
4.2.2.4. Fear and Avoidance Behavior Questionnaire ............................................ 73
LIST OF TABLES

Table 1.1: Project Research Design................................................................. 13
Table 3.1: ICC and SEM Summary for Research Instrumentation .................. 53
Table 4.1: Research Project Design............................................................... 69
Table 4.2: Reliability of Instrumentation for Proprioception Measures ............. 70
Table 4.3: Descriptive Statistics for Subject General Characteristics .................. 71
Table 4.4: Descriptive Statistics for Pain & Disability Level ......................... 72
Table 4.5: Descriptive Statistics for Physical Characteristics ......................... 74
Table 4.6: Joint Position Sense: Mixed-Model Analysis .................................... 75
Table 4.7: Joint Position Sense: Multiple Comparisons ................................. 76
Table 4.8: Threshold to Detect Passive Motion: Mixed-Model Analysis ............. 77
Table 4.9: Threshold to Detect Passive Motion: Multiple Comparisons ............. 77
Table 4.10: Direction of Movement: Mixed-Model Analysis ............................ 79
Table 4.11: Force Reproduction: Mixed-Model Analysis ............................... 80
LIST OF FIGURES

Figure 1.1: Functional Joint Stability\textsuperscript{92}  ................................................................. 5
Figure 2.1: Sensorimotor System\textsuperscript{87}  ........................................................................ 27
Figure 3.1: Biodex Systems III Isokinetic Device used for Proprioception Testing .............. 53
Figure 3.2: Joint Reposition Sense  ............................................................................................ 59
Figure 3.3: Threshold to Detect Passive Motion Sense (Kinesthesia) .................................. 60
Figure 3.4: Force Reproduction ................................................................................................. 61
Figure 3.5: Neutral Gapping Manipulation Procedure ............................................................ 63
Figure 3.6: Sham Procedure ..................................................................................................... 64
Figure 3.7: Schema Employed for Multiple Comparisons ..................................................... 68
Figure 4.1: Results for Joint Position Sense ........................................................................... 76
Figure 4.2: Results for Threshold to Detect Passive Motion ................................................. 78
Figure 4.3: Results for Direction of Passive Movement ......................................................... 79
Figure 4.4: Results for Force Reproduction ........................................................................... 80
PREFACE

I would like to thank my research committee, Dr Joseph Myers, Dr. Chad Cook, Dr. Timothy Sell and Dr. Scott Lephart for their considerable support and guidance throughout the entire process of bringing this project to fruition. You have all demonstrated exemplary professionalism in your efforts to assist me through reading endless manuscripts.

I would also like to thank my classmates and colleagues at the Neuromuscular Research Laboratory for their support and assistance. Having friends and colleagues assist by giving feedback over every aspect of the project as well as willingness to help during reliability testing and data collection will always be remembered and appreciated.

I would like to acknowledge my colleagues in the Department of Physical Therapy at Youngstown State University, Youngstown, Ohio, for their moral support and willingness to rework schedules during the four years of my doctoral work.

I want to thank Dr. Freddie Fu for The Freddie H. Fu Graduate Research Award which provided funding for the completion of this project.

This project simply would not be possible without the support of the subjects who participated in the data collection. I appreciate the men and women who willingly gave their time; most of them traveled from Ohio to be tested. Their efforts are most appreciated.

I would like to acknowledge my wife Mary and my sons, Stephen and Shane for their unconditional love and tireless support throughout my graduate career. The time required to complete a doctorate is taken away from time spent with the ones you love the most. Their acceptance and encouragement through the process has been unbelievable.
I especially want to thank my parents, Gene and Arline Learman for their endless encouragement and support throughout my entire life, acting as role models both personally and professionally. This manuscript is dedicated to my father’s memory.
1. INTRODUCTION

1.1. Societal Impact of Low Back Pain

The incidence of low back pain (LBP) has been estimated between 4%-56% of the general population per year.\textsuperscript{1-6} Between 60% and 80% of the population will experience LBP during their lives\textsuperscript{7-10} and up to 15% become chronic.\textsuperscript{11} LBP is the most frequent cause of disability in individuals less than 45 years and the third leading cause in those 45 years and older.\textsuperscript{8} LBP is most common between 35-55 years of age,\textsuperscript{3, 12} but affects people of all ages. In jobs that require an extensive amount of physical effort, 2-5% of the working population is compensated each year for work-related LBP.\textsuperscript{13, 14} LBP is second only to the common cold in missed work days in the United States\textsuperscript{14-16} affecting as much as 20% of the work force annually.\textsuperscript{17-19}

Annual prevalence rate estimates for LBP range from 41\%\textsuperscript{6} to 65\%,\textsuperscript{20, 21} while point prevalence rates approximate 30\%.\textsuperscript{22} Variability in the statistics reported reflect the challenge of performing epidemiology studies with consistent design or variability in the definition of terms.

Healthcare economists estimate that 15\% of the cases generate up to 80\% of the healthcare costs associated with LBP.\textsuperscript{23-25} In the United States during the late 1980’s, treatment for LBP was estimated at $25 billion annually with indirect costs increasing the total expenditure to $75 billion annually.\textsuperscript{19} Recent costs have not been examined within the literature.
LBP has been described as benign and self-limiting since 90% spontaneously heal in 6-8 weeks,\textsuperscript{26} yet other authors contend this view is inaccurate since many people with LBP do not fully recover\textsuperscript{26, 27} demonstrated by 1 year recurrence rates ranging from 36\% to 76\%.\textsuperscript{28-35} A review article of natural progression of low back pain concluded that there is no evidence to support the theory of spontaneous recovery within one month\textsuperscript{29} suggesting that chronic LBP (CLBP) can be operationally defined as recurrent acute episodes over a period of time. This hypothesis may imply that deficits remain following acute symptom reduction leaving the individual susceptible to further episodes of pain.

1.2. Problem of Low Back Pain

The reporting of clinical outcomes for the management of LBP has been inconsistent and unpredictable\textsuperscript{26, 36, 37} in part because best practice has not been clearly identified.\textsuperscript{38-40} Clinical evidence may be difficult to ascertain\textsuperscript{39} as most studies compare a treatment with placebo rather than compare competing treatment strategies.\textsuperscript{40} Even though some interventions appear to be more efficacious than others, the implementation of those interventions in physiotherapy clinics appears to be inconsistent or lacking.\textsuperscript{41-43} A portion of the inconsistency in management and therefore guidelines of best practice may be attributable to a lack of consensus of causation and methods of treatment for those causes in LBP.\textsuperscript{44}

Therapist training and experience level has been speculated to affect therapeutic outcomes\textsuperscript{45} but there is evidence that younger, lesser experienced clinicians can learn and implement techniques in an efficient\textsuperscript{46} and efficacious manner.\textsuperscript{47} Despite the long term problem and the efforts made to better understand and thereby better treat spinal problems; LBP remains enigmatic.\textsuperscript{40, 48}
1.2.1. Mechanisms of Low Back Pain

Most LBP appears to be of unknown etiology. Reasons for this statement include a lack of sensitivity of special testing used to assess LBP, a high rate of anatomical anomalies noted on diagnostic imaging, a failure to demonstrate a high correlation between anatomic abnormality with clinical symptomatology, and the failure of clinical examination to predict symptom and disability rates. Proposed causes for both acute and chronic LBP include: muscular injury, imbalance and hypertonicity, facet joint dysfunction, intervertebral discs (IVD), scoliosis, sacroiliac dysfunction, instability, and psychological causes; yet reliable identification of specific pathology remains illusive. Identification of pathoanatomic causation is hampered by complex innervation of pain generating structures and their close proximity to structures sharing innervation. Considering inflammation or localized trauma, multiple pain generating structures may be involved creating a multifactorial condition.

Pathological conditions have been identified by proportion. Bogduk reports that 39% of back pain is from the IVD, 33% is unidentified, 15% from the zygapophyseal joint and 13% from the sacroiliac joint. Laslett et al. reported that from 15-40% of LBP may be from the zygapophyseal joint. Bernard’s estimated SIJ contribution at 22.6%. Despite having proportional guidelines for the identification of the pathoanatomic cause of LBP, consistently selecting the appropriate cause in any individual case remains a clinical challenge.

1.2.2. Predictors of Low back Pain

Numerous studies have determined which factors may predict LBP but many of these studies have been cross sectional design and lack sufficient statistical power to draw strong conclusions. With regard to age, a range from approximately 40-60 have the highest prevalence rates but the relationship to incidence isn’t as clear. Psychosocial factors including depression,
self-esteem, job satisfaction and feelings of distress are more strongly related to the development of LBP.\textsuperscript{56-67} Physical demands of the job could also be involved with positive odds ratios for injury including: peak sagittal trunk velocity, maximum low back moment, peak lumbar shear forces, lumbar disc compression,\textsuperscript{68} and work related twisting.\textsuperscript{66} Personal characteristics including cigarette smoking,\textsuperscript{66, 69} obesity,\textsuperscript{70, 71} trunk strength and flexibility, exercise history, familial history and general health have all been implicated.\textsuperscript{55, 70, 72-78} Moshe found the only reliable predictor of occupational LBP was a previous history of LBP ascertained from pre-employment screenings.\textsuperscript{79} This evidence further supports the problem with high recurrence rates in chronic episodic LBP.\textsuperscript{79} Radiographic evidence of spondylolysis was not predictive for developing LBP in Israeli policeman\textsuperscript{80} but was predictive in football and rugby players.\textsuperscript{81, 82} The interaction of pathoanatomical and psychosocial causation is featured in the biopsychosocial model\textsuperscript{83} and may explain a portion of the unpredictability of LBP exclusively on anatomical or physiological factors and validates the results of numerous studies performed on psychosocial factors reported in the literature.

1.2.3. **Deficits in Low Back Pain**

For the purpose of this paper, the segmental dysfunction model\textsuperscript{84} will be used to describe the deficits associated with LBP and will be centered around Panjabi’s model of stabilization.\textsuperscript{85, 86} Stability has been described as a balance between passive structures, active structures and their neuromuscular control.\textsuperscript{85, 87} Panjabi described the subsystems of stability as interdependent meaning that deficits in one component could be compensated for by enhanced activity in another component. If a passive structure was damaged or a muscle lacked strength or endurance, greater muscular effort could maintain the needed level of stabilization through altered neural control to prevent further damage.\textsuperscript{88} The actual ability of the system to compensate
for structural changes should be questioned since damage to passive structures seems to be quickly followed by detrimental changes in the muscular component further challenging or because of neural control changes (figure 1.1).\textsuperscript{84, 89-91}

**Functional Joint Stability Paradigm**

![Functional Joint Stability Paradigm Diagram](image)

*Lephart et al. (1994)*

**Figure 1.1: Functional Joint Stability**\textsuperscript{92}

It is apparent that at least one of these subsystems fails to heal effectively following an acute episode of LBP leaving the individual susceptible to subsequent episodes. Ultimately, it appears that neuromuscular control is primarily responsible for prolonged pain and loss of function and this finding is consistent with conclusions drawn from peripheral joints.\textsuperscript{91}

Segmental instability refers to the state where normal control of a particular movement is temporarily lost during function. This particular condition can be due to losses in the integrity of passive structures that assist in stability, a loss of muscular stiffness due to atrophy or injury, or a loss of neuromuscular control because of compromised proprioceptive input. Using this model of
instability, it is easier to appreciate how abnormal movements at a single segment can occur at any particular range of motion resulting in abnormal force to be placed on the segment or structures surrounding the segment leading to nociceptive afferent input.

1.2.4. Passive structures

When the passive structures become pathological, a number of changes occur that limit their stabilizing capacity. Damage to ligaments, the facet joint and capsule, the vertebral endplate and/or the annulus fibrosis result in increased range and altered quality of motion at the segment. There is an increase in the total area of displacement and path length of the instantaneous axis of rotation which likely results from a larger neutral zone and there is an alteration in the acceleration and deceleration of movement with disc or facet lesions which may pose additional challenges to neuromuscular control. These mechanical changes result in functional instability of the segment which can lead to further injury perpetuating the vicious cycle demonstrated in Figure 1.1.

1.2.5. Muscular

In CLBP, the multifidus muscle demonstrates a smaller cross sectional area and a moth eaten appearance that can be attributed to atrophy of type II muscle fibers, structural changes in type I fibers, and increased intramuscular fat. These deficits result in a loss of strength and endurance. Muscular dysfunction shifts force transfer from the facet joints to the IVD and ligaments in the forward flexed posture further suggesting interdependence of stabilizing subsystems. Subjects with a history of LBP demonstrate muscle composition and functional capacity deficits in the 8th decade implicating the permanence of muscular and/or neural control mechanism changes for dynamic stabilization in chronic conditions.
1.2.6. Neuromuscular Control

The third component involves the neural control mechanism responsible for coordinating the efforts of each muscle in the active system. Spinal stiffness is a balance of activity of each muscle and specific firing patterns are utilized to provide this stiffness. There is evidence that these firing patterns are variable among subjects, depending on the activity and loading of the spine, and may demonstrate deficient capacity in low back pain. Neural control of the active subsystem may be exhibited through feedforward, and reflex mechanisms.

Any subsystem deficit results in increased muscular activity to compensate, resulting in muscular pain from prolonged contraction intensity that exceeds the threshold demonstrated in fatigue pain. The active component is the subsystem primarily responsible for providing stability in the neutral range since the passive subsystem is inefficient in this range. As segmental stabilization is limited by local muscular changes, these deficits can be overcome through greater global muscle activation; however, the global muscles fail to have the local segmental attachments necessary to create stability in a normal manner. Overactivity of the global muscles in the form of an inappropriate co-contraction may result in excessive compressive forces and ultimately result in fatigue. A greater reliance on multi-segmental trunk muscles may occur to provide stability in non-specific CLBP and even more so in those with clinical instability. Muscular coordination also appears to be compromised for lumbar erector spinae during a variety of functional activities such as gait at various speeds in subjects with LBP.

The feedforward mechanism of segmental stabilization, a co-contraction of multifidus and transversus abdominis, does not fire before the prime movers in their customary fashion in upper extremity, or lower extremity movement at intermediate and fast speeds in subjects
with LBP. Neural changes occur in LBP including an abatement of the flexion-relaxation phenomenon in subjects with a herniated nucleus pulposis thereby reducing the protective mechanism of feedforward gamma bias in the extensors prior to lifting a load.

1.2.7. Proprioception

Proprioception is an afferent component of the sensorimotor system which is essential for providing feedback in static and dynamic stabilization of each segment and the body’s posture as a whole making it an appropriate avenue for clinical study. Numerous studies have demonstrated deficits in components of proprioception in subjects that present with low back pain. Most studies have measured proprioception with joint reposition sense and kinesthetic awareness even though proprioception has been defined as having other components such as direction of motion, force appreciation, velocity and acceleration awareness. These other components have not been adequately explored in LBP literature presently.

Deficits are noted in subjects with a wide variety of pathoanatomical diagnoses including disk herniation, lumbar spinal stenosis, and segmental instability. Not all diagnoses result in joint repositioning deficits since one study found no significant difference between controls and subjects with ankylosing spondylitis. Even as the disease process progressed, position sense remained intact. Subjects with CLBP have shown deficits in balance through response time measurements when compared with control subjects. Duration of LBP does not appear to be a factor in the sensorimotor test results since deficits have been demonstrated in subjects with LBP for greater than one year, three months or greater, or acute episodic LBP.

Since wider age ranges tended to be represented in the back pain studies when compared with asymptomatic position sense studies, the effect of age should be considered. Parkhurst and Burnett’s study supported an age difference for proprioception in the subsystem of passive
movement sense in magnitude and direction; however, differences in joint reposition sense did not achieve statistical significance.\textsuperscript{120} These conclusions reinforced previous literature suggesting age related differences for passive motion sense\textsuperscript{132, 133} and have since been validated for a lack of significance in reposition sense.\textsuperscript{134}

The effect of external stimuli on joint reposition sense in the lumbar spine has demonstrated results consistent with that found in other peripheral joints of the body. Studies have found that repositioning error is lessened by the use of a lumbar support which may enhance cutaneous input to the sensorimotor system. This result has been demonstrated in healthy subjects,\textsuperscript{135} as well as in subjects with low back pain.\textsuperscript{136}

The importance of muscle spindle activity in spine proprioception has been validated as it has been in peripheral joints. Challenging the local muscles resulted in increased spinal repositioning error,\textsuperscript{137} and time to sense lumbar passive motion.\textsuperscript{138} This has been studied by using muscle fatigue,\textsuperscript{138, 139} and by using vibration.\textsuperscript{122, 137} Ironically, lumbar position sense in subjects with low back pain significantly improves with the same vibratory stimulus over the multifidus.\textsuperscript{122} In LBP, muscle spindles may be underactive and are enhanced to a more normal level \textsuperscript{140, 141} with vibration whereas in normal subjects, the vibration distorts the spindle activity making the spindle behave as if in the lengthened state\textsuperscript{122, 142, 143} resulting in undershooting of the target position.\textsuperscript{122, 137}

1.3. **Treatment for Low Back Pain**

As previously stated, multiple treatments for LBP have been attempted with varying levels of success challenging the clinician’s understanding of the most appropriate method of managing CLBP. Treatment options include stretching, strengthening, physical agents, traction,
general exercise, aerobic exercise, stabilization exercise, various forms of mobilization and manipulation. Numerous studies have been performed in recent years to examine the efficacy of these interventions.\textsuperscript{40} Many recently published RCTs fail to identify specific subpopulations with LBP and this may compromise the ability of these studies to identify efficacious interventions.\textsuperscript{40} In an attempt to deal with the many ambiguities surrounding the evaluation and treatment of LBP, researchers have created and tested clinical prediction rules to identify subgroups of LBP patients that may respond favorably to specific interventions. To date, a number of classification systems and clinical prediction rules have been developed with varying levels of success. A clinical prediction rule was developed\textsuperscript{144} and validated\textsuperscript{145} for determining a subpopulation that would respond favorably to a general sacroiliac manipulation. A preliminary clinical prediction rule for instability has also been developed but has not yet been validated prospectively.\textsuperscript{146} Classification systems have been used for 40 years to assist in identifying subgroups that would respond favorably to specific interventions\textsuperscript{147-149} and these classification systems have varying levels of evidence to support validity and reliability.\textsuperscript{1} There is a role for each intervention to play in nearly all of the clinical reasoning systems and one intervention that can be implemented within each system and has been demonstrated to be efficacious with treatment is manipulation.

1.4. Effects of Spinal Manipulation

Spinal manipulation has been demonstrated to have effects on numerous systems of the body producing therapeutic results. Manipulation has been noted to cause movement of the adjacent segments in all three motion planes\textsuperscript{150, 151} resulting in enhanced spinal range of motion over the course of intervention.\textsuperscript{152, 155} The effects have been shown to be local to the segment of intervention\textsuperscript{152} and to adjacent segments\textsuperscript{144, 145} demonstrating that it is not required to be
specifically on the hypomobile segment to achieve the desired result.\textsuperscript{154} It has not been clearly elucidated by what mechanism the change in ROM occurs.

There is a plethora of evidence supporting the hypothesis that spinal manipulation reduces pain\textsuperscript{144, 145, 155-159} in the spine\textsuperscript{159} and extremities.\textsuperscript{157} The mechanism of action has only been speculated upon. Manipulation reduces inflammatory cytokines\textsuperscript{160} and creates a barrage of afferent input to the CNS causing stimulation of the substantia gelatinosa in the dorsal horn directly through $\alpha$-$\beta$ stimulation and indirectly through descending control from the dorsal periaqueductal grey (dPAG) area.\textsuperscript{155}

Stimulation of compound action potentials (CAP) has been found in subjects with lumbar radiculopathy.\textsuperscript{151} The authors believe that the response is the result of afferent fibers reacting to the manipulative input which created small but distinct vertebral motions.\textsuperscript{151} The delay between the stimulus and the response averaged 12ms which is similar to studies on animal preparations.\textsuperscript{161-163} Herzog et al. demonstrated that manipulation in each region of the spine resulted in an increased EMG response between 50–200ms after intervention that lasted between 100–400ms in numerous muscles throughout the body suggesting that there is a more systemic EMG response to the intervention.\textsuperscript{164}

Mechanoreceptors in the lumbar spines of feline preparations revealed that the duration of the stimulating impulse resulted in differing neurophysiological responses. The shorter impulses generated larger responses in the mechanoreceptors.\textsuperscript{165} This supports the hypothesis that manipulation may have different and in some cases superior impact on clinical conditions when compared with joint mobilizations.
1.5. **Definition of the Problem**

Most diagnoses of CLBP have demonstrated signs and symptoms of segmental dysfunction or instability. A comprehensive review of the literature reveals that instability is characterized by a loss of neuromuscular control that causes structural changes to occur in the muscles responsible for stabilization; therefore, neuromuscular control appears to be largely responsible for the chronic component of CLBP. Proprioception, being a key component of the sensorimotor system, needs to be functionally intact to provide the CNS with appropriate somatosensory input for stabilization; however, little is known about the therapeutic value of most interventions for spinal dysfunction on proprioception. Since spinal manipulation has been demonstrated to be clinically effective in the proper patient population, it would be prudent to determine if manipulation were effective by means of altering spinal proprioception. To date, no clinical trials have been performed measuring the effects of spinal manipulation on proprioception in normal subjects or subjects with CLBP; therefore, manipulation’s effect on proprioception is essentially unknown.

1.6. **Purpose**

The purpose of this study was to investigate the effects of spinal manipulation on clinically measured proprioception in subjects with a history of CLBP who were pain-free at the time of testing. Using a randomized controlled crossover design (see table 1.1), subjects were assessed before and after spinal manipulation and a sham manipulation (non-thrust technique) to determine if manipulation improved proprioception in subjects with CLBP and to determine if that effect lasted one week.
1.7. Specific Aims and Hypotheses

**Specific Aim 1:** To use a randomized controlled crossover design study to measure proprioception in the lumbar spine before and after a localized spinal manipulation in subjects with CLBP and compare the clinical effects of spinal manipulation with a non-thrust, sham procedure. Proprioception was measured as joint reposition sense (JPS), threshold to detect passive motion (TTDPM), direction of movement sensation (DM), and force reproduction (FR).

**Hypothesis 1.1:** Joint reposition sense would improve in the lumbar spine immediately following a localized spinal manipulation as compared with a sham procedure in subjects with CLBP.

**Hypothesis 1.2:** Passive kinesthesia of the lumbar spine would improve following a localized spinal manipulation as compared with a sham procedure in subjects with CLBP.

**Hypothesis 1.3:** Detection of passive motion direction would improve following a localized spinal manipulation in subjects with CLBP.

**Hypothesis 1.4:** Force reproduction accuracy of the lumbar spine would improve immediately following a localized spinal manipulation as compared with a sham procedure in subjects with CLBP.

<table>
<thead>
<tr>
<th></th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Manip 1st</strong></td>
<td>Manipulation</td>
<td>Sham</td>
<td></td>
</tr>
<tr>
<td><strong>Sham 1st</strong></td>
<td>Sham</td>
<td>Manipulation</td>
<td>Sham</td>
</tr>
</tbody>
</table>

Table 1.1: Project Research Design
Specific Aim 2: To use a randomized, controlled crossover design to assess the residual effect of spinal manipulation compared with a sham procedure on subjects with CLBP one week after the manipulative intervention.

Hypothesis 2.0: Proprioception measured one week after the manipulation procedure would demonstrate residual improvement as compared with:

1. Proprioception measured pre-intervention the week before.
2. Proprioception measured one week after the sham procedure.
2. REVIEW OF LITERATURE

2.1. Societal Impact of Low Back Pain

2.1.1. Incidence and Prevalence

Incidence of low back pain (LBP) has been estimated between 4%-56% of the general population for any given year\textsuperscript{1-6} with a two year incidence rate established at 8.55\%.\textsuperscript{3} Gender does not appear to influence the results.\textsuperscript{3} Approximately 80% of the population will experience LBP at some point during their lives.\textsuperscript{7-10, 77} Subpopulations have demonstrated similar lifetime incidence ranges: 539 runners and walkers were examined revealing a lifetime incidence rate of 74\%,\textsuperscript{71} and a study of 784 Danish military recruits showed that 73\% developed back pain within 12 years of enlistment.\textsuperscript{166} Of those who experience LBP, 15\% become chronic.\textsuperscript{11} LBP frequently results in disability with a 1.8\% incidence rate of new claims in industry for 1995,\textsuperscript{167} 8 million new cases were estimated for 2004,\textsuperscript{8} and the fact that LBP accounts for 13\% of all documented disability.\textsuperscript{9} LBP is the most frequent cause of disability in individuals less than 45 years and the third leading cause in those 45 years and older.\textsuperscript{8} LBP tends to be most common in individuals between 35-55 years of age,\textsuperscript{3, 12} but is an acknowledged problem through a much greater age range with studies of preadolescents\textsuperscript{9, 34, 168} to the elderly.\textsuperscript{146, 169} Approximately one-third of school children experience intervertebral disc degeneration\textsuperscript{168} and 1.9\% demonstrate disability from LBP.\textsuperscript{170}
In a review of 56 studies performed between 1966 and 1998, annual prevalence rate estimates for LBP range from 22% to 65%. Point prevalence for LBP approximate 30% but range from 12% to 33% A cross sectional survey of occupational health showed that among 113,323 Canadians from 2000-2001, a point prevalence rate of 20.6% of self-reported chronic LBP in those over 20 years of age was demonstrated. The survey also found that functional impairment (67.8%) was more prevalent than pain (24.5%). A prevalence survey study for Australian citizens found that point prevalence was 25.6%, one year prevalence rate of 67.6% and a lifetime prevalence rate of 79.2%. A prospective cohort design of 288 British scaffolders, found a lifetime prevalence rate of 60% and an examination of over 600 students between the ages of 12-17 in two separate secondary schools found a lifetime prevalence rate of 74% for back pain with 69% being for LBP. Disability rates from LBP were lower with 53.5% and 10.5% for low disability, regardless of low or high pain intensity, and high disability, associated with high intensity pain respectively. These variable, almost conflicting results demonstrate variability in the statistics reported which may reflect the challenge of performing incidence and prevalence studies with consistent design or may demonstrate variability of the condition itself. Regardless of the source for variability, LBP is a significant health problem facing society today.

2.1.2. Recurrence Rates

LBP has been described as benign and self-limiting since 90% spontaneously heal in 6-8 weeks, while other researchers claim that this belief is inaccurate since recurrence rates range from 36% to 76%. A review of studies often quoted for spontaneous recovery refer to patients no longer seeking medical care or their return to work; not necessarily the elimination of symptoms. A recent study revealed that 3-year recurrence rates for work-related LBP in
1867 workers in New Hampshire was 33.9% for care and 17.2% for disability\textsuperscript{175} indicating that the condition continues to interfere with normal function as well as create pain.

2.1.3. Cost

Healthcare economists estimates that 15% of the cases generate up to 80% of the healthcare costs associated with LBP.\textsuperscript{23-25} In jobs that require an extensive amount of physical effort, 2-5% of the working population is compensated each year for work-related low back pain.\textsuperscript{13, 14} In Great Britain, 12.5% of all sick days were the result of LBP in 1988-1989\textsuperscript{176} and Sweden has demonstrated similar statistics with 11-19% of sick days annually from 1961 to 1987\textsuperscript{177} showing that the problem is not peculiar to the United States. LBP is second only to the common cold in missed work days in the United States\textsuperscript{14-16} affecting as much as 20% of the workforce annually.\textsuperscript{17-19} Workers compensation claims for LBP result in 40% of the total physical therapy visits annually.\textsuperscript{178, 179}

In the United States during the 1980’s, treatment for LBP was estimated at $25 billion annually with $50 billion more for indirect costs,\textsuperscript{19} and costs may have escalated through the 1990’s and 2000’s but current costs to either industry or society in general have not been easily found. In industry, a study of claims paid to workers for occupational LBP in 1995 indicated that $8.8 billion were spent but this figure does not take the indirect costs of productivity into consideration.\textsuperscript{167} Indirect costs are at least as high as direct costs\textsuperscript{180} and in some economies are significantly higher\textsuperscript{181} suggesting that curtailment of indirect costs could be the best strategy for reducing the burden on society.\textsuperscript{181}

First episodes of LBP and disability are less costly and of shorter duration than recurrent episodes.\textsuperscript{175} The progression of LBP and disability has made it imperative that researchers and
the medical community seek out interventions that will reduce the recidivism rates and the percentage of patients that progress on to chronicity and disability.\textsuperscript{13, 182}

A recently review of 17 articles attempting to identify the most cost effective intervention for non-specific LBP and was unable to identify a superior intervention from cost analysis because of the heterogeneity of the interventions, subject populations and controls of the studies.\textsuperscript{183} This is further evidence that greater consistency in study design parameters is needed in order to make generalizations to the public concerning the evaluation and treatment methodologies for LBP.

2.2. Problem of Low Back Pain

2.2.1. Mechanisms of Low Back Pain

It has been stated that most low back pain is of unknown etiology.\textsuperscript{49} There are a multitude of reasons for this statement including a lack of sensitivity of special testing used to assess low back pain, a high rate of anatomical anomalies noted on diagnostic imaging, a failure to demonstrate a high correlation between anatomic abnormality with clinical symptomatology, and a failure of clinical examination to predict symptom reports and disability rates.\textsuperscript{13, 50} Even so, it can be stated that a considerable number of cases of low back pain are the result of mechanical factors since the symptoms can be altered either positively or negatively with movement of the spine during a mechanical examination.

There are multiple proposed causes for both acute and chronic low back pain. Authors have proposed muscular involvement, facet joint, intervertebral discs (IVD), scoliosis, sacroiliac involvement, muscular imbalances, and instability; yet reliable identification of that specific pathological agent remains illusive. The ability to identify a specific painful tissue is hampered
by the innervation of potential pain generating structures. It has been identified that the dura, outer annulus and the posterior longitudinal ligament are all innervated by the sinuvertebral nerve, the zygapophyseal joint and the local muscles are innervated by the medial branch of the posterior primary rami. Since multiple structures are located in a small area and are innervated by nociceptors from the same nerves, pain referencing patterns from various structures are similar making accurate identification of a specific structure unlikely. It must also be considered that in inflammatory conditions or in circumstances where a localized trauma has occurred, multiple pain generating structures may be involved so the problem is multifactorial in nature.

Authors have identified pathological conditions by proportion. Bogduk reports that 39% of back pain is from the IVD, 33% is unidentified, 15% from the zygapophyseal joint and 13% from the sacroiliac joint. Laslett et al. has reported that from 15-40% of back pain may be from the zygapophyseal joint. Dontigny has stated that up to 95% of all low back pain may originate from a subluxed S3 facet of the SI joint; however, no scientific based study using a diagnostic gold standard was used to validate his claim. Bernard’s estimation of SIJ contribution is a more modest 22.6% retrospectively examining 1293 cases of LBP over a 12 year period. These varying percentages of pathoanatomic causation for LBP may suggest that the populations studied are not homogenous, causation may be unpredictable, or diagnostic standards may be inherently unreliable.

2.2.2. Risk Factors and Predictors

Numerous studies have been performed to determine different factors that may be predictive of low back pain. Many of these studies have been of cross sectional design and lacking in sufficient statistical power to draw strong conclusions. With regard to age, a range from approximately 40-60 seems to incur the highest prevalence rates but the relationship to
incidence isn’t as clear.\textsuperscript{55} Psychosocial factors including depression, self-esteem, job satisfaction and feelings of distress are more strongly related.\textsuperscript{56-66} Studies have also demonstrated that job related factors other than psychosocial factors could also be involved. The physical demands of the job were also predictors with positive odds ratios for injury including: peak sagittal trunk velocity, maximum low back moment, peak lumbar shear forces, lumbar disc compression,\textsuperscript{68} and work related twisting.\textsuperscript{66} Personal characteristics including cigarette smoking,\textsuperscript{66, 69} obesity,\textsuperscript{70} trunk strength and flexibility, exercise history, familial history and general health have all been implicated.\textsuperscript{55, 70, 73-75, 77, 78, 187, 188} Factors related to body build, nutritional status and general constitution cannot reliably predict incidence of back pain.\textsuperscript{189}

In addition to pathoanatomical causation, one must consider the interaction of risk factors with anatomical causation. This approach is featured in the biopsychosocial model of causation.\textsuperscript{83} This model explains a portion of the unpredictability of low back pain exclusively on anatomical or physiological factors and validates the results of numerous studies performed on psychosocial factors reported in the literature.

\textbf{2.2.3. Natural Course of Low Back Pain}

It is imperative to identify the natural progression of LBP to better understand the effects of treatment.\textsuperscript{29} False conclusions about interventions will result if we cannot identify how the disorder behaves naturally.\textsuperscript{190} Hestbaek’s systematic review of 36 studies published between 1981 and 1999 revealed that LBP does not evolve in a predictable pattern. Studies use differing methods of determining progression of the disorder making comparisons difficult. Cross sectional and longitudinal studies use cohorts of those with LBP at different stages of the disorder creating inequities during comparisons.\textsuperscript{29}
LBP does not feature true recovery but changes over time with the progression of the disorder and the progression is nonlinear in fashion so basing classification of LBP as acute, subacute or chronic on duration of symptoms alone is invalid. There may be significant variations between the short-term and long-term prognoses altering the results of a given study though it does appear that in CLBP, as age increases so does the disability. Short-term effects may view a problem as being cured; however, LBP is frequently characterized by recurrent bouts of pain and disability suggesting that chronicity can be determined by either duration or recurrence of symptoms. A previous episode of LBP is still the strongest predictor of future LBP.

It can be concluded from the literature that no specific natural course of LBP is known at this time but spontaneous recovery does not seem to occur. Any study aiming to result in a cure must address long-term follow-up since the condition is frequently characterized by pain and disability that comes and goes in the short-term over a period of years. To have an impact on the long term course of LBP, research needs to elucidate the component of the condition that remains deficient and address that component accordingly.

2.3. Deficits with Low Back Pain

2.3.1. Structural

The osseoligamentous (passive) structures that contribute to static stability include the vertebrae, joint capsules, intervertebral disks, ligaments and fascia. Structural components of stability provide most of their control at or near the end of range. Increased passive structural contribution decreased muscular requirement for stability can be demonstrated by the flexion-relaxation phenomenon which can be defined as the EMG silence that occurs in the spinal
extensors when the trunk reaches end range flexion.\textsuperscript{117} This reduction in EMG activity has also been identified during lifting tasks from full flexion.\textsuperscript{196} Mechanical modeling has supported the notion that approximately 90N of force will cause an unsupported spinal column to collapse\textsuperscript{197} and this value may be reduced in circumstances where damage to passive structures exists. Examples of passive structure deficits can include but are not limited to, spondylosis, spondylolysis, spondylolisthesis, degenerative disk disease, disk herniation, ligamentous and capsular damage. Damage to passive structures increases the size of the neutral zone that must be stabilized by active structures.\textsuperscript{86} Even though the passive structures are incapable of tolerating loads approaching functional activities, their importance to dynamic stability are apparent through the mechanoreceptors providing afferent input to the sensorimotor system which will be discussed further in this chapter.

2.3.2. \textbf{Muscular}

The second component of stability is the muscular (active) subsystem surrounding the spine which has the ability to stabilize the spine through the force of contractions.\textsuperscript{85} The amount of muscular activity influences the amount of stability at each segmental level it crosses and this relationship has a positive correlation. It should be noted that relatively small amounts of activity will provide the spine with sufficient stabilization as long as each component is functioning properly. This amount of activity, when normalized to a maximum voluntary contraction, is approximately 1.5-2\% for normal, unresisted movements.\textsuperscript{198}

The active subsystem has been subdivided into two separate components, global and local muscles.\textsuperscript{112} In Bergmark’s model, global muscles are primarily responsible for maintaining postural stability and are prime movers whereas the local muscles are primarily responsible for maintaining segmental stability during all movement or static functional activities.\textsuperscript{112} To further
illustrate this point, it has been stated that the deep medial fibers of the multifidus and rotators brevis are rich in muscle spindles and have minimal mechanical advantage for creating movement of the spine supporting the hypothesis that their primary action is to function as proprioceptive movement transducers. 199, 200

Abdominal muscles are also important in creating dynamic stability in the spine. The transversus abdominis provides tension through the thoracolumbar fascia because of its insertion on the fascia at the lateral raphe which creates a compressive stabilizing force through the entire lumbar region. The horizontal fibers of the transversus abdominis were effective in creating a closing force at the sacroiliac joint as found by Doppler study measuring vibration provided by a tuning fork; 201 therefore, the muscle is capable of increasing intra-abdominal pressure and stabilization throughout the lumbopelvic region.

2.3.2.1. Decreased Cross-Sectional Area of Select Muscles

In CLBP, the multifidus muscle demonstrates a smaller cross sectional area, 101, 102 and a moth eaten appearance that can be attributed to atrophy of type II muscle fibers, structural changes in type I fibers, 103 and increased intramuscular fat. 104, 105 It has been noted through diagnostic ultrasonography and magnetic resonance imaging (MRI) 102 that a significant loss of multifidus cross sectional area occurs on the ipsilateral side of the spine at or within one level of the segment experiencing pain. 102, 202, 203 This occurs in subjects with acute low back pain, 101, 202 subacute low back pain, 101 and chronic low back pain. 102 It has been further noted that multifidus recovery does not occur spontaneously in subjects as their pain resolves naturally. 202 The amount of reduction in the cross sectional area of multifidus is related to the duration of symptoms. 102 The cross sectional area in the ipsilateral psoas muscle is also reduced and this reduction correlates positively with the intensity of pain rating. 102 If subjects with back pain train
multifidus adequately, recovery of cross sectional area does occur. Other studies have found that while strength and endurance improve with training, cross sectional area and muscle density do not.

2.3.2.2. Decreased Multifidus and Erector Spinae Strength

The histologic changes outlined in the above section, result in a loss of strength and endurance. Muscular dysfunction shifts force transfer from the facet joints to the IVD and ligaments in the forward flexed posture further suggesting interdependence of stabilizing subsystems.

A recent study suggested that subjects with a history of LBP continue to demonstrate muscle composition and functional capacity deficits in the 8th decade implicating the permanence of muscular and/or neural control mechanism changes for dynamic stabilization in chronic conditions.

2.3.2.3. Decreased Erector Spinae and Quadratus Lumborum Endurance

Biering-Sorensen developed an endurance test, Biering-Sorensen test (BST) where the subject lies prone on a table with the upper body is hanging off to the anterior superior iliac spine is hanging off and the lower body is strapped to the table for stability. The BST measures the capacity of the erector spinae to maintain a contraction of around 45% MVIC for time. The test can be used to identify patients with LBP and may predict people likely to develop LBP. Fatigability significantly differed between a group of golfers with and without CLBP for median shift of erector spinae during the BST and this difference had a significant impact on the subject’s ability to develop an MVIC in the quadriceps muscles. These findings implicate a
wider region of muscular effect beyond local musculature; a concept referred to as regional interdependence.

2.3.3. **Neuromuscular Control**

The third component is the neural control mechanism responsible for coordinating the efforts of each muscle in the active system. Spinal stiffness is a balance of activity of each muscle and specific firing patterns are utilized to provide this stiffness. There is evidence that these firing patterns are variable among subjects, depending on the activity and loading of the spine,\(^{108}\) and may demonstrate deficient capacity in low back pain.\(^ {109, 110}\) Neural control of the active subsystem may be exhibited through feedforward, and reflex mechanisms.\(^ {86}\)

Segmental instability can be defined as the momentary loss of neuromuscular control during any functional activity.\(^ {89}\) Evidence of segmentally specific loss of neuromuscular control may be found in much of the recent research on local muscle cross sectional area and firing order of trunk muscles in healthy vs. low back pain.\(^ {101, 102}\) These differences have been noted in subjects with acute\(^ {101}\) and chronic low back pain.\(^ {102}\)

Recovery of the multifidus muscle appears critical since it is very important in the normal functioning spine. It provides segmental stiffness thereby being a prime component of neuromuscular control in the functional neutral zone.\(^ {99, 209-211}\) Multifidus has been demonstrated to be responsible for up to two-thirds of the muscle stiffness contributing to segmental stability of the L4-5 segment.\(^ {211}\) Atrophy and the presence of multifidus dysfunction has been associated with poor outcomes following lumbar disc surgery.\(^ {103}\) Functional recovery following surgery was associated with attenuation of multifidus dysfunction.\(^ {212}\)

Spinal stability is not a new concept in evaluation and treatment of low back pain. Preuss and Fung reviewed current literature on the concept of spinal buckling under submaximal loads
and concluded that subjects may display certain characteristics that predispose them to this phenomenon. Spinal buckling may be an ongoing anatomical anomaly or issues with processing of input by the CNS but may result from untreated or inadequately treated segmental injuries.

2.3.4. Proprioception

Proprioception is defined as the awareness of body position, orientation, movement and sensation of force. Proprioception is the afferent input of internal stimuli from proprioceptive fibers within the body screened from and responding to the external environment responsible for the challenges to the body’s equilibrium. There is much variability as to what constitutes the extent of proprioception in the human body and for the purpose of this study, proprioception will end with the afferent input being delivered to the CNS via the appropriate neural pathways.

To expand upon Sherrington’s definition and the above stated interpretation of proprioception, it becomes necessary to define the motor response to the proprioceptive input. Without an appropriate motor response, the afferent input would be pointless. The terminology used to define the afferent input with the motor response is the sensorimotor system. The sensorimotor system greatly expands the neural implication of proprioception since we are now linking the unconscious reception of neural input of proprioception to somatosensory, visual, and vestibular input. This afferent input must then be integrated and interpreted at the level of the cerebral cortex, brain stem, basal ganglia, cerebellum and spinal cord levels. Finally the complex efferent response must be made through the fusimotor system. see figure 2.1
The purpose of the sensorimotor system is to allow the body to integrate information to adjust posture and to refine neuromuscular responses to the environment for safe, balanced and appropriate movement during function. Proper integration of neural input is necessary for coordination of movement and position. Without proprioceptive control, appropriate dynamic stabilization would not be possible.

Proprioception is necessary to establish an accurate, efficient and coordinated response of the efferent system to the demands of the environment. Each processing center receives proprioceptive information and processes the information in its own unique way. At the cortical level, proprioceptive information is used to establish conscious awareness of posture, body position, and movement sense. At the spinal cord level, proprioception is used to grade a
reflexive response through mono and polysynaptic pathways. The reflexes are however subject to descending pathways of motor control.87,215,216

Having defined terminology relevant to proprioception and sensorimotor control, it must be stated that direct measurement of proprioception is very difficult, if not impossible, to clinically perform since proprioception is exclusively an afferent phenomenon occurring both consciously and unconsciously within the body.217 Proprioception, being a necessary component of the sensorimotor system, affords the researcher the opportunity to indirectly measure it through multiple sensorimotor pathways. While the number and types of method for assessing the sensorimotor system are extensive,217 this study will focus on methodology that has been documented previously in spine literature.

While many studies have focused entirely on either joint position sense or kinesthesia as measures of proprioception, this study examined four measures of proprioception (including force appreciation and direction of movement). It has been suggested that JPS and kinesthesia are not highly correlated modalities suggesting that a single test to quantify proprioception is lacking.218 Lack of correlation has been further verified by studies demonstrating that in acute knee ligament injuries, JPS was spared while kinesthesia was involved219 and that rehabilitation or surgery may not result in improvement in both modalities. Parkhurst et al. found that in the spine, age related changes could be noted depending on whether or not JPS or kinesthesia was examined.120

The afferent nerve endings responsible for providing proprioceptive input are extensive throughout the body. Mechanoreceptors have been identified in lumbar facet joints220 intervertebral discs,221 and other spinal connective tissues.221 Mechanoreceptors in the facet joints are not particularly dense with only five type 1, six type 2, and one type 3 receptor
identified in 13 facet capsules.\textsuperscript{220} Although there was less than 1 per joint capsule, it must be noted that numerous, and uncounted, free nerve endings (FNE) were also found. They remained uncounted because they were not described as one of the types of encapsulated nerve endings responsible for proprioception by Freeman and Wyke.\textsuperscript{222} The implication from their work is that the receptors possess a large receptor field that may display significant deficits with damage or possibly that the FNE (type 4 receptors), are not only nociceptive receptors but also mechanical in nature playing a greater role in proprioception than previously believed.\textsuperscript{222, 223}

Mechanoreceptors were identified in the outer 2-3 lamellae of the annulus fibrosis and the anterior longitudinal ligament (ALL) of 67 human IVDs and ALLs.\textsuperscript{221} Coccygeal discs of bovine specimens also have mechanoreceptors but the younger specimens had more receptors than the older specimens.\textsuperscript{221} The study found that the receptors were relatively sparse with only 50\% of the discs in LBP subjects having receptors and only 15\% of the scoliosis subjects having receptors. The largest proportion of receptors resembled GTO type receptors followed by endings resembling Pacinian corpuscles and Ruffini endings.\textsuperscript{221} The proportion of receptors found suggest that these mechanoreceptors do not appear to be a well developed system within the IVD and ALL of human specimens studied. Hypotheses for these findings may include the presence of deficits due to a lack of receptors providing input or a decline in the receptors due to the injury. Conclusions on these questions cannot be drawn from these available data.

Deficits were noted in subjects with a wide variety of pathoanatomical diagnoses. A study of 20 subjects with CLBP from disk herniation compared with 15 healthy control subjects that those with CLBP required 2.5 times more movement before detection (2.5° to 1°) of passive kinesthesia in rotation, compared with controls but this deficit reduced to a non-significant differential 3 months post surgical intervention.\textsuperscript{127} Another study assessed the passive rotational
kinesthesia of 26 subjects with lumbar spinal stenosis and found that 76.9% of subjects reported movement in the wrong direction and localized the movement to the wrong region of the body. A study compared 15 subjects demonstrating clinical segmental instability with age and gender matched controls and assessed spinal repositioning error with an electromagnetic tracking device and found that the experimental group had a 54.5% increase in repositioning error (1.7° to 1.1°). Not all diagnoses result in joint repositioning deficits since no significant difference was found in joint reposition sense in 50 subjects with mild ankylosing spondylitis and 50 controls with reposition sense being measured by an electromagnetic position tracking device. A follow-up suggested that the insignificant difference noted in the previous study was maintained even as the disease process progressed. In contrast to the above studies, Newcomer et al. studied 20 subjects with CLBP and 20 age matched controls for spine reposition error using an electromagnetic tracking system. In this study, the researchers asked the subjects to move through their full range of motion then repeat the movement and when the subject reached an approximate 50% of the original range they were asked to hold the position for 2 seconds. The subject was asked to return to neutral then repeat the position from memory and hold for 2 seconds. This was repeated 3 times without revisiting the reference position. Major challenges to the authors’ results include the lack of return to the criterion reference between each trial. It is possible that all subjects, regardless of back pain condition will forget a reference position after repeatedly moving toward and away from that position due to thixotropic alterations in afferent input through the muscle spindles. The authors’ inclusion criteria for controls allowed subjects with a history of LBP to participate as long as they had not experienced LBP in the past year. As previously stated, CLBP can be characterized by recurrent episodic pain and that neuromuscular control may be compromised in these subjects without presenting with symptoms. It must also be
noted that only three trials in each direction were performed which has been shown to lack sufficient power.\textsuperscript{225}

Duration of LBP does not appear to be a factor in the proprioception test results reported. Deficits have been noted in subjects with chronic low back pain, defined as greater than one year,\textsuperscript{121} or of three months to one year,\textsuperscript{119, 123, 124} and acute episodes of LBP shorter than 3 months.\textsuperscript{125}

Since wider age ranges may be represented in back pain studies when compared with asymptomatic position sense studies, the effect of age on proprioception should be considered. Parkhurst and Burnett’s study of 88 firefighters supported an age difference for proprioception in passive movement sense in both magnitude and direction; however, differences in joint reposition sense did not achieve statistical significance.\textsuperscript{120} This study used a homemade device to assess proprioception that was based on a continuous passive motion machine to generate the movement in the spine. The lower body was moved while the upper body remained fixed to reduce the likelihood of vestibular and upper trunk input. It should be noted that this study examined a cohort of individuals who do a lot of lifting and carrying in less than ideal conditions which may influence the age factor since age and years of experience on the job are correlated as well; however, this study supports other literature that has demonstrated age related differences.\textsuperscript{132, 133} In contrast, no statistically significant differences were found in reposition sense for 21 subjects grouped in over/under 40 years of age categories.\textsuperscript{134} In this study, reposition error was assessed using an instrumented spatial linkage for three trials which may not have sufficient power to detect a meaningful change\textsuperscript{225} yet the findings are consistent with Parkhurst and Burnett for reposition sense.\textsuperscript{120}
The effect of external stimuli on joint reposition sense in the lumbar spine has demonstrated results consistent with those found in other peripheral joint of the body. A study of 40 subjects, 20 with CLBP and 20 controls, measured spinal reposition error with an electromagnetic tracking system before and after the application of a lumbar support and then again after wearing the support for 2 hours. Both groups demonstrated significant reduction in reposition error after applying the brace but the effect of the brace was reduced after 2 hours of use to approximate the initial condition. It is interesting to note that the authors employed a different methodology compared with their previously referenced study in that 4 trials of differing spinal angles were used with a criterion reference positioning between each trial. Mixed results were found when examining 40 healthy subjects using a Latin square crossover design measuring spinal reposition with a Lumbar Motion Monitor and six trials at various angles of flexion. In this study, when the healthy subjects were divided up into high error and low error groups, the high error group experienced a significant reduction in absolute error whereas the low error group remained unchanged. It is reasonable to conclude that enhanced cutaneous input contributed to proprioceptive feedback in subjects while wearing a brace and that prolonged exposure causes the system to accommodate to the enhanced neural input.

The importance of muscle spindle activity in spine proprioception has been examined as it has been in peripheral joints. Brumagne et al. assessed joint reposition accuracy in 25 asymptomatic individuals randomly assigned to an experimental (n=16) or control (n=9) group. Reposition sense was assessed by application of a piezoresistive electrogoniometer positioned over S2. Pelvic tilts were performed through full range and the examiner asked each subject to hold a position in early to mid range for 5 seconds. Two full repetitions of pelvic tilt were performed and the subject was asked to return to the criterion position. Error was then
measured for reposition accuracy in each of six trials. A second set of six trials was performed while the experimental group received vibration of 70 Hz to the multifidus muscle as the same protocol was followed. Absolute and constant reposition error were both statistically significant in the experimental group (F[1,15] = 30.77, p = 0.0001). A similar protocol was used to assess the affect of vibration on 44 subjects, 23 LBP patients and 21 controls. In this protocol, reposition sense was measured before during and after vibration was applied. Control subjects experienced increased error rate during vibration and reduced error rate in the trials after vibration whereas the LBP group experienced improvement of reposition sense during and after the application of vibration. The implication of this result may support the notion that underactive muscle spindles are enhanced to a more normal level with vibration whereas in normals, the vibration distorts the spindle activity making them perceive greater muscle length causing them to undershoot the position target.

Similarly, proprioception has been studied before and after fatigue of the Erector Spinae group. It was found by Taimela et al. in 106 subjects, 57 with CLBP and 49 controls, that following a fatiguing bout of back extensions with a submaximal load, spinal kinesthesia, measured by passive lower body rotation at 1°/sec−1, was significantly slower than before the fatigue protocol. While significant changes occurred for both back pain conditions, the effect of fatigue was more pronounced on those with CLBP and correlated with those with higher self-reported pain intensity and frequency as well as with reported functional impairment. This fact is important because it suggests that because erector spinae muscles demonstrate deficits in endurance, they are more likely to fatigue from lower levels of submaximal activity thereby leaving them more likely to fatigue and show proprioceptive deficits from fatigue leaving them more susceptible to future damage during normal activities of daily living.
2.3.5. Proprioception and Trunk Position

Wilson and Granata conducted a study where joint reposition sense was tested on 11 subjects without a history of CLBP, defined as pain of greater than 1 month duration or no LBP in the past year, in neutral, 30°, and 60° of flexion; all three sagittal positions were also tested in 30° of right and left rotation. The reposition test was accomplished in an active-active paradigm where a target position was achieved with visual feedback and held for 5 seconds then the feedback was removed and the subject had to reposition from proprioceptive feedback and again hold for 5 seconds. The results showed that lumbar reposition sense for neutral curvature with visual feedback was 1.01° +/- 0.73° compared with 3.02° +/- 2.85° without visual feedback. Assessing was compromised in both flexion positions but the rotations did not significantly affect reposition sense.228

Allison and Fukoshima tested accuracy, precision and power of trunk repositioning at 20%, 50% and 80% of range tested in 3 postures: knees extended, knees flexed, and trunk rotated 45° for 10 trials in each condition. The study included 23 subjects without a recent history of LBP and reposition sense was assessed with electromagnetic sensors on C7 and S1. They found that trunk repositioning was significantly worse at 20% than 80% range in all three postures, knees extended, knees flexed and flexion rotation with p = 0.0002, 0.0499, and 0.0241 respectively. It was also determined that statistical power of the results increased as the number of trials increased; however, the increase may not warrant the logistical inconvenience of increasing trials beyond 6 per condition. The power increase from 3 trials to 6 was felt to be worth the increased effort. The authors concluded that the increase in power may explain why their results varied from those found by other researchers.225 In reviewing Allison and Fukushima’s results, it does not appear that performing more trials than 5 would be warranted in
this current study since the increase in power beyond 5 trials does not occur until the 10th trial and the current study will attempt to investigate proprioceptive changes that may be transient and the trials will be performed on subjects who may likely be experiencing modest levels of LBP during testing.

2.3.6. **Weight Bearing Asymmetry**

Weight bearing characteristics were examined in subjects with (n = 35) and without (n = 31) LBP to determine if asymmetry existed. Side to side variation was statistically larger in the CLBP group than the healthy control group with the differential being 8.8% to 3.6% with p < 0.001. Subjective pain level altered the amount of deviation with a correlation of r = 0.39 and p = 0.021. It is not possible to determine from the study whether the weight bearing asymmetry resulted from altered neuromuscular control or exclusively from an attempt to reduce nociceptive input. A follow-up investigation found that spinal manipulation significantly reduced the weight bearing asymmetry along with reduction of subjects reports of pain.

2.4. **Treatment for Low Back Pain**

Multiple interventions for low back pain have been attempted with varying levels of success, challenging the clinician’s understanding of the most appropriate method of managing CLBP. Potential treatment options include stretching, strengthening, physical agents, traction, general exercise, aerobic exercise, stabilization exercise, various forms of mobilization and manipulation. When reviewing the previously mentioned deficits associated with acute, subacute, and chronic LBP, it would appear that some form of exercise would be required in nearly every case; however, the efficacy of therapeutic exercise has been equivocal. Therapeutic exercise for the treatment of LBP has demonstrated variable outcomes depending on the strategy
and the type of exercise employed. In a comprehensive review of the literature, van Tulder et al. found only weak evidence to support the use of a general program to increase activity as a treatment for low back pain. An updated review further supported the conclusions found previously but additional support was reported for the exercise being at least as effective as other forms of conservative treatment for chronic low back pain. These reviews are not without their critics who feel that some studies have demonstrated outcomes showing significant improvement of functional disability scores compared with controls. It has been demonstrated that aerobic exercise provides some functional improvement for people with chronic pain but only mild pain reduction. Another study supported the use of erector spinae strengthening program through bilateral leg lifts to horizontal over the edge of a plinth showing a correlation between continued pain control and reduced disability among subjects who continued routine use of the exercise following completion of the study. Studies have demonstrated that stabilization alone does not result in significant increases in cross sectional area of the paravertebral muscles in general or the multifidus muscle specifically. If resistance training is added to the stabilization program either with or without a static 5 second hold at end range, cross sectional area is significantly increased in the paravertebrals; however, multifidus only demonstrates a significant increase when the static 5 second hold is included. These results are consistent with a study by Verna who used a varying angle roman chair apparatus with trunk hyperextension exercises over an eight week period to significantly increase both strength and endurance of the trunk extensors. The improvement was also significant at the 4 week midpoint evaluation as well.

The effects of therapeutic exercise have been included in this paper to remind the reader of the complexity of treating LBP. The equivocal results of therapeutic exercise intervention may
indicate a continued deficit that is not being adequately addressed through exercise alone. Manual therapy enhanced the therapeutic effect of exercise over exercise alone in a study of 52 subjects presenting with subacromial impingement syndrome. Subjects receiving manual therapy with exercise demonstrated significantly better outcomes defined by pain reduction, strength gains and functional assessment questionnaire. The authors suggest that manual therapy reduces pain through afferent input and may restore movement by mechanical stretching of collagen thereby allowing therapeutic exercise to be more effective. It also should be considered that manual therapy stimulates enhanced proprioception and improved gamma bias through feedforward mechanisms allowing the exercise to stimulate the muscle under enhanced neuromuscular control thereby increasing effectiveness. Considering this information, it may be possible to conclude that manual therapy can be used to enhance neuromuscular control prior to the performance of therapeutic exercise in the spine thereby increasing the effectiveness of the exercise and reducing the residual deficits noted from previous studies.

Numerous studies have been performed in recent years to examine the efficacy of these interventions. Many recently published RCTs fail to identify specific subpopulations with LBP and this may compromise the ability of these studies to identify efficacious interventions. In an attempt to deal with the many ambiguities surrounding the evaluation and treatment of LBP, researchers have created and tested clinical prediction rules to identify subgroups of LBP patients that may respond favorably to specific interventions. To date, a number of classification systems and clinical prediction rules have been developed with varying levels of success. A clinical prediction rule was developed and validated for determining a subpopulation that would respond favorably to a general sacroiliac manipulation. A preliminary clinical prediction rule for instability has also been developed but has not yet been validated prospectively.
long-term, follow-up assessments have not been performed on these clinical prediction rules making it impossible to determine whether or not patients treated using them have similar recurrence rates to previously studied treatments. Classification systems have been used for 40 years to assist in identifying subgroups that would respond favorably to specific interventions\textsuperscript{147-149} and these classification systems have varying levels of evidence to support their use. There is a role for each intervention to play in nearly all of the clinical reasoning systems mentioned and one intervention that can be implemented within each system and has been demonstrated to be efficacious with treatment is manipulation.

2.4.1. Mobilization and Manipulation

Mobilization and manipulation have been well documented in the literature. Manipulation has been demonstrated to be an effective intervention for patients with low back pain of at least four weeks duration as compared with minimal intervention except for advice to continue with normal activities and avoid undue rest. The effect of manipulation could be enhanced by including therapeutic exercise intervention.\textsuperscript{241} Other studies have demonstrated similar findings of mobilization enhancing the effects of exercise.\textsuperscript{239} One study found that manual therapy provided better outcomes than exercise alone in pain and disability outcomes up to a one-year follow-up for low back pain patients with symptom duration between 3 weeks and 6 months.\textsuperscript{242} The difference between the two treatment groups was statistically significant when compared with each other and to pretreatment baselines.\textsuperscript{242} In contrast, a study supported the opposite conclusions having both the manual therapy group and stabilization group having similar results initially but the stabilization group demonstrated superior outcomes at the one year follow-up.\textsuperscript{243} Still another study demonstrated that a combination of individualized exercise (derived to treat each subject’s specific deficits) combined with manual therapy resulted in
statistically significant reduction in pain when compared with manual therapy and nonspecific exercise or specific exercise without manual therapy in the treatment of chronic low back pain.\textsuperscript{244}

Many studies do not differentiate between mobilization and manipulative interventions even though it has been noted that differences in effect may exist. Mechanoreceptors in the lumbar spines of feline preparations revealed that the duration of the stimulating impulse resulted in differing neurophysiological responses. The shorter impulses generated larger responses in the mechanoreceptors.\textsuperscript{165} This supports the hypothesis that manipulation may have different and in some cases superior impact on clinical conditions when compared with joint mobilizations.

2.5. Effects of Manipulation

There is a plethora of evidence supporting the hypothesis that spinal manipulation reduces pain.\textsuperscript{144, 145, 155-159} The pain reduction has been noted in the spine\textsuperscript{159} and the extremities.\textsuperscript{157} The mechanism of action has only been speculated upon and evidence suggests that manipulation reduces inflammatory cytokines.\textsuperscript{160} It has been hypothesized that manipulation creates a barrage of afferent input to the CNS causing stimulation of the dorsal periaqueductal grey (dPAG) resulting in an immediate descending control of pain stimulus at the substantia gelatinosa in the second laminar layer of the dorsal horn.\textsuperscript{155} Gate control theory has also been considered in explaining the pain reduction phenomenon noted with spinal manipulation.

An in vivo study reported that small amounts of movement do occur between adjacent segments with spinal manipulation in all three planes and the amount of movement is relative to the manipulative force but does not seem to be very sensitive to the direction of force.\textsuperscript{150, 151} The effects have been shown to be local to the segment of intervention\textsuperscript{152} and to adjacent segments\textsuperscript{144, 145} demonstrating that it is not required to be specifically on the hypomobile segment to achieve
the desired result. Spinal manipulation results in enhanced spinal range of motion over the course of intervention. It has not been clearly elucidated by what mechanism the change in ROM occurs; it may be due to mechanical effects or neurophysiological changes allowing greater movement by reducing muscular guarding.

There are local neurophysiologic responses to spinal manipulative therapy. These neurophysiologic responses may enhance proprioceptive input to the spine allowing the local muscles to better respond to exercises that are designed to provide local stability. These neurophysiologic responses may serve to enhance the muscles’ ability to demonstrate functional stability and endurance. Afferents have been observed in various structures in the posterior compartment of the trunk including: the stabilizing ligaments of the intervertebral segments, the intervertebral disks, the zygopophyseal joints, the intrinsic postvertebral muscles, paraspinal muscles, and the thoracolumbar fascia.

Stimulation of compound action potentials (CAP) has been demonstrated in subjects with lumbar radiculopathy. The authors believe that the response is the result of afferent fibers reacting to the manipulative input which created small but distinct vertebral motions. The delay between the stimulus and the response averaged 12ms which is similar to similar studies on animal preparations.

A reflexive response was also noted in vivo as evidenced by EMG response to spinal manipulation and CAP response to the same manipulation. The response, however may have been muted by the distance the EMG electrodes were away from the segment being manipulated. The EMG duration of response was consistent with previous findings from Indahl. Manipulation in each region of the spine resulted in an increased EMG response between 50–200ms after intervention that lasted between 100–400ms in numerous muscles.
throughout the body suggesting that there is a more systemic EMG response to the intervention.\textsuperscript{164} EMG activity increased in the multifidus muscle of porcine specimens following stimulation of either the facet joint or the annulus fibrosis. Even though the patterns of firing were different for each structure stimulated, the effect supports the hypothesis that the multifidus may play a key role in stabilization of the segment.\textsuperscript{251} Another study replicated the EMG response to annulus stimulation and then injected the zygapophyseal joint with saline which resulted in an attenuation of the EMG response indicating a close neural connection between these structures of the lumbar spine suggesting that the facet joint may assist in regulating neuromuscular activity in the functional spinal unit.\textsuperscript{161} Perhaps the mechanical distention of the facet joint capsule replicates the mechanical effects of manipulation causing attenuation of spasm of the spinal unit reducing pain and increasing ROM.

Childs et al. studied the effects of LBP on symmetry of weight bearing in 66 subjects, 35 with LBP and 31 healthy controls to determine if LBP correlated with asymmetry of weight bearing.\textsuperscript{252} Pain was assessed verbally by an 11-point numeric rating scale and weight bearing symmetry was assessed using 2 digital scales. The authors found that subjects with LBP displayed significantly greater asymmetrical weight bearing characteristics (p < 0.001) with a mean differential of 8.8\% (8.0) of body weight compared with 3.6\% (2.6) of bodyweight for controls.\textsuperscript{252} In addition, as pain rating increased, asymmetry of weight bearing increased with an r = 0.39 and p = 0.021 but the correlation was not significant for the ODI at r = 0.26 and p = 0.098.\textsuperscript{252} A subsequent prospective study which included 30 patients with either acute or chronic LBP receiving spinal manipulation revealed that, their subjective reports of pain diminished (r = 0.5, p = 0.007), asymmetrical weight bearing characteristics, and iliac crest asymmetry significantly improved (p = 0.001).\textsuperscript{229} It has been hypothesized that the asymmetry of iliac crest
height and weight bearing characteristics are the result of local soft tissue abnormalities which would require further research to validate.\textsuperscript{229,253}

Since the preceding studies were published, Knutson et al. authored an article that supported the soft tissue hypothesis by demonstrating that asymmetries in apparent leg length of 47 subjects with LBP were associated with reduced endurance times for the erector spinae measuring the BST (F2,44 = 13.909, \( p = 0.001 \)) and quadratus lumborum using the side-support test (left side: F2,64 = 7.59, \( p = 0.001 \); right side: F2,4 = 7.97, \( p = 0.001 \)).\textsuperscript{254} The authors suggest that the results provide evidence for a hypothesis that fatigued ipsilateral muscles become hypertonic and the resultant spasm draws the iliac crest superiorly giving the illusion of a pelvic obliquity and shortened leg length. If the manipulative technique corrects the soft tissue abnormality, one can only speculate upon the mechanism of the action. Studying a component of the sensorimotor system through proprioception may serve to elucidate a possible mechanism for the action of the manipulation in subjects presenting with apparent leg length discrepancies and asymmetrical weight bearing. These data serve as the foundation for this study’s attempt to determine if proprioceptive changes occur concomitantly with weight bearing asymmetry changes in subjects with CLBP.

In addition to the positive effects of spinal manipulation, one must also consider the potential risks of implementing manipulation. There is clinical concern regarding the potential harmful effects of spinal manipulation.\textsuperscript{255,256} The primary concern is the devastating outcome of producing cauda equina syndrome with lumbar manipulation. A systematic review of the literature revealed 11 reported cases of cauda equine in over 77 years of literature reporting. These data have been used to roughly estimate that that cauda equine occurs in 1 in 100,000,000 manipulations.\textsuperscript{257} Less serious side effects from spinal manipulation have been reported and do
occur with greater frequency. The predominant side effect can be described as a transient increase in the subject’s symptoms. In 64% of the cases, an increase in symptoms occurred within 4 hours of treatment and 74% of cases resolved within 24 hours of treatment, with less than 20% lasting greater than 48 hours.

2.6. Methodological Considerations

The dependent variables outlined in this study may be assessed in numerous ways. There are strengths and weaknesses associated with each of these methodologies. This section of the chapter will review what the pertinent literature reports on the difficulties associated with the methodology and will provide a theoretical basis for the investigation techniques selected in the following chapter.

2.6.1. Assessment of Proprioception

The Biodex Systems Three (Biodex Inc., Shirley, New York) has been widely used for the measurement of strength and endurance for numerous joints in a plethora of research studies. The test-retest reliability has been examined for a number of joints and it has been found to be reliable and valid for measurement. Symons found that the Biodex System 3 was reliable for isokinetic and isometric testing of older men with ICC ranging from .84-.94. Lund found that the Biodex was reliable and displayed no learning effect during testing and Leggin found that Interrater ICC of .93 - .96 and intrarater of .97 - .99. The use of the Biodex for the assessment of proprioception generally and spine proprioception specifically has not been as widely described in the literature.

It is also imperative to note that in order to externally assess proprioception, conscious control must be examined. This is likely an incomplete picture since proprioception is used in an
unconscious manner during function. As has been demonstrated previously, high correlations between submodalities of proprioception is lacking in the literature suggesting the difficulty that may be encountered by drawing strong conclusions from the data collected.

2.6.1.1. Joint Reposition Sense

A variety of methods have been used to assess spine reposition sense with differing reliability data and differing gross values of accuracy yet they are close enough to demonstrate a trend of assessment that may be implemented in other studies.

Swinkels and Dolan examined 20 healthy subjects’ reposition sense for each section of the trunk at 1/3rd, ½ and 2/3rd range of motion, as well as upon their return to upright. Three trials that were averaged at each position using an electromagnetic inclinometer with a sensor at each spinal section. Results demonstrated a trend toward a difference between the inner and outer ranges with outer range being less accurate, but it did not reach statistical significance for each sensor except the S2 sensor. Statistical significance at the S2 level may implicate reduced proprioception at the hip joint rather than the spine. It may be possible that significance was not achieved in more regional comparisons due to a lack of power since three trials were used by comparison with ten trials in the Allison study. It is also interesting to note that the most accurate sensor was the T1 sensor indicating that people likely use head position to determine position sense. This fact may indicate that it would be better to eliminate repositioning sense from the head and the vestibular system from studies intending to examine trunk reposition sense.

Preuss, Grenier and McGill examined the affect of body position on lumbar reposition sense by testing 70 asymptomatic male subjects in standing, sitting, and four point kneeling (FPK) under two conditions: eyes open and eyes closed. The sensors were placed on T12 and
S1 and the subjects were asked to move through a specified range and return to the neutral posture which was measured for accuracy. The subjects performed 4 trials of 5 positions, flexion, left and right side flexion, and left and right rotation for sitting and standing and flexion for FPK. The results showed that the standing condition was significantly more accurate (mean repositioning error) than either the sitting or FPK positions in both visual conditions (p<0.0001) except blindfolded sitting vs. standing (p = 0.0993). Precision of repositioning (reposition error variance) was also significantly different with FPK being less precise than standing (p<0.0001) but standing and sitting difference did not achieve significance. It is also important to note that 4 trials were used which Allison and Fukushima’s study found to be the minimum for statistical power at 0.60. The authors conclude that weightbearing may alter proprioceptive feedback through differing stimulation of mechanoreceptors or altered muscle spindle and GTO input. The protocol used in this study was excessively long at 2-3 hours and fatigue may have played a role in the results of these data; however, the findings were not substantially different from other, similar studies. Pilot testing data for this study revealed an ICC for JPS testing of 0.473 with a standard error of 0.2595 which is a fair result.

2.6.1.2. Passive Kinesthesia

The Biodex has also been used in numerous studies for sensorimotor assessment. Traditionally, speeds of ranging between 0.5 – 2°/s have been used to target slow adapting mechanoreceptors in threshold to detection of passive motion (TTDPM) studies and the Biodex System 3 currently has been equipped with software allowing the passive mode of assessment to be slowed to 0.25°/s increasing the sensitivity of the instrument to make it easier for researchers to identify and quantify deficits of proprioception in injured states and more accurate in determining improvement in rehabilitation. Pilot data for this study revealed an
ICC for TTDPM of 0.792 with a standard error of 0.1025 which is a moderate bordering on significant result.

2.6.1.3. Sense of Tension

The Biodex Systems 3 has the capacity to be used for force appreciation. This subcomponent of proprioception is the least frequently tested in the spine literature. While the software has not been specifically designed for this purpose, modifications of standard protocols allow researchers to perform multiple repetition trials and with alteration of visual cues (turning the monitor off during the force replication trial) accuracy of force replication can be assessed in data output. This specific submodality of proprioception has not been as widely studied in the spinal literature, therefore, its response to injury and treatment has received less critical inquiry than JPS and kinesthesia. The correlation between force appreciation and either JPS or kinesthesia is equally lacking. 218 Pilot data for this study revealed an ICC for force appreciation of 0.584 with a standard error of 0.2053 which is a fair result.

2.6.2. Duration of Testing

The length of the assessment process for proprioception is a major concern since a number of the studies reviewed demonstrated transient neurophysiologic changes following spinal manipulation. The proprioception protocol needs to be of sufficient length (containing at least 4 trials) to enhance statistical power 225 yet be short enough to maximize the recognition of the transient neurophysiologic effects of the intervention as well as to reduce the likelihood of irritating the subject’s LBP.

2.6.3. Cavitation

The question of whether or not cavitation (the audible or palpatory pop during treatment) is required in order for manipulation to be clinically successful has been debated in the literature.
Flynn et al. found in their study on a clinical prediction rule for subjects who would demonstrate improvement with lumbar manipulation clearly found that cavitation was not required for temporary improvement of acute low back pain. Whether or not cavitation might alter the results of this study’s results is not known so methodology will be altered to minimize the effects of lack of cavitation by repeated manipulation in cases where cavitation does not occur.

### 2.6.4. Muscle Thixotropy

Muscle thixotropy describes the concept of recent muscular activity history affecting the outcome of testing. It has been demonstrated that the position of a joint or the amount of muscular activity directly preceding the active test described has affected the end result of that test. Specifically, end range positioning or submaximal muscular forces have been known to alter EMG activity and joint position sense. In order to minimize the affects of muscular thixotropy, testing procedure will include several repetitions of active range of motion through the full range to neutralize the joint position and muscular history. Submaximal extension practice trials will also be used to prevent the subject from increasing the error rate on the first repetition of force appreciation sense following a submaximal voluntary contraction.

### 2.7. Summary

Many deficits can be attributed to loss of neuromuscular control and the interrelationship of these factors is the theoretical basis of this study. Regardless of whether the deficits and damage have occurred to the passive, active, or neuromuscular control structures, the results are clinically the same; challenging the dynamic stability of the system during function thereby setting the system up for future damage through a vicious cycle of pain and pathoanatomical
Numerous neurophysiologic effects of spinal manipulation have been demonstrated in the literature and their affect on spinal proprioception has not been investigated to determine whether or not functional dynamic stabilization may be enhanced by spinal manipulative therapy. Investigating proprioceptive changes from manipulation may further enhance the clinician’s understanding of the impact the intervention may have on other therapeutic modalities frequently used for treatment of CLBP.
3. METHODOLOGY

3.1. Experimental Design

This study was a randomized, controlled unbalanced crossover design comparing the effect of spinal manipulation versus sham manipulation on spinal proprioception in subjects with CLBP.

3.1.1. Dependent Variables

Dependent variables included lumbar proprioception measured by trunk reposition error (JPS), threshold to detect passive motion (TTDPM), accuracy percentage for direction of movement perception (DM), and error for extensor force reproduction sense (FR).

3.1.2. Independent Variables

The independent variables were the treatment group, manipulation versus sham manipulation, and time.

3.2. Subject Characteristics

Thirty-three subjects participated in this research study. Seventeen subjects were randomly assigned to the group receiving manipulation first (Manip 1st) and 16 subjects to the group receiving sham procedure first (Sham 1st) group. Subjects gave informed consent to participate in the study as required by the University of Pittsburgh Institutional Review Board.
3.2.1. **Inclusion Criteria**

Subjects demonstrated the following:

1. CLBP operationally defined as symptoms lasting at least 3 months or recurrent episodic LBP that compromised their normal daily function at least twice during the previous year.

2. Subjects reported a history of signs and symptoms consistent with mechanical low back pain which included:
   
   A. Pain that is aggravated by specific activities
   
   B. Pain that may be position dependent
   
   C. Pain that is alleviated by specific positions or activities

3. Subjects did not demonstrate evidence of acute nerve root irritation or pain from systemic disease processes.

4. At the time of testing, subjects reported a current pain level of less than 1 on the numeric rating scale and an average pain level during the previous 24 hours (VAS – 24) of 3 or less.

5. Subjects reported a current disability level between 0 – 20% on the Oswestry Disability Index.

6. Subjects reported a Fear and Avoidance Behavior Questionnaire (FABQ) work scale subset of less than 19.

7. Subjects were between the ages of 18 – 65.
3.2.2. **Exclusion Criteria**

Subjects did not demonstrate the following conditions to prevent potential exacerbation of their CLBP condition.

1. Contraindications for the use of spinal manipulation.
2. Pain from acute nerve root irritation.
3. Acute disk herniation with foraminal stenosis determined by the presence of acute nerve root signs.
4. Advanced central stenosis determined by subjective history and pattern of presentation in general physical examination.
5. Subjects displaying systemic pathology were excluded from this study.

3.3. **Power Analysis**

An a priori power analysis determined that 13 subjects were required for each treatment arm in order to obtain a power of 0.798 for a period effect and 0.88 for a treatment effect at \( \alpha < 0.05 \). The power analysis was based on previous studies examining proprioceptive differences between healthy subjects and those with LBP. Joint position sense data from Koumantakis et al. yielded conservative estimates with healthy subjects demonstrating an error rate of 3.67° +/- 1.82° and those with low back pain 5.46° +/- 3.54°. Since there was a lack of data examining proprioception changes with treatment, the current estimate may have resulted in an inflated effect size; therefore, 15 subjects were included per treatment arm.
3.4. Subject Recruitment

Subjects were recruited for this study utilizing a number of specific strategies including contacting physical therapy and chiropractic clinics for subjects who may satisfy the inclusion criteria established for the study. Flyers (Appendix A) were posted around campus since CLBP is a relatively common problem and at local country clubs since amateur golfers tend to demonstrate CLBP. No subject attrition occurred; therefore, all 33 subjects who started the testing procedures completed data collection.

3.5. Instrumentation

3.5.1. Biodex Isokinetic Dynamometer

Trunk reposition sense (JPS), passive trunk kinesthesia (TTDPM), direction of movement perception (DM) and trunk extensor force replication (FR) were assessed using the Biodex System III Multi-Joint testing and Rehabilitation System (Biodex Medical Inc., Shirley, NY). Calibration of the Biodex dynamometer had been performed in accordance with the specifications outlined in the manufacturer’s service manual. The trial-to-trial and day-to-day reliability and validity of torque measurement of the Biodex System III were all previously established with intraclass correlation coefficients (ICC) reported to be 0.99-1.268
Figure 3.1: Biodex Systems III Isokinetic Device used for Proprioception Testing

For proprioception application, ICCs were determined during pre-study pilot testing and were found to be fair for FR and JPS and moderate for TTDPM according to the standards identified by Shrout and Fleiss. The results of pilot testing for this study can be found in Table 3.1. Furthermore, a pattern of a significant learning effect was not apparent for either TTDPM or FR; however, learning may have been apparent with JPS.

<table>
<thead>
<tr>
<th>Measure</th>
<th>ICC</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>TTDPM</td>
<td>0.792</td>
<td>0.1025</td>
</tr>
<tr>
<td>JPS</td>
<td>0.473</td>
<td>0.2595</td>
</tr>
<tr>
<td>FR</td>
<td>0.584</td>
<td>0.2053</td>
</tr>
</tbody>
</table>

Table 3.1: ICC and SEM Summary for Research Instrumentation
The Biodex Systems Three (Biodex Inc., Shirley, New York) has been widely used for the measurement of strength and endurance for numerous joints in a plethora of research studies. The test-retest reliability has been examined for a number of joints and it has been found to be reliable and valid for measurement. Symons found that the Biodex System 3 was reliable for isokinetic and isometric testing of older men with ICC ranging from 0.84-0.94. Lund found that the Biodex was reliable and displayed no learning effect during testing and Leggin found that Interrater ICC of 0.93 - 0.96 and intrarater of 0.97 - 0.99.

The Biodex has also been used in numerous studies for sensorimotor assessment. The Systems 3 has been equipped with software allowing the passive mode of assessment to be slowed to 0.25° sec\(^{-1}\) making it more appropriate for assessing TTDPM more precisely and accurately. Increasing the sensitivity of the instrument in this manner could make it easier for researchers to identify and quantify deficits of proprioception in injured states and more accurately determine measures of improvement in rehabilitation.

The Biodex Systems 3 has the capacity to be used for FR. This subcomponent of proprioception is the least frequently examined. While the software was not specifically designed for this purpose, modifications of standard protocols allowed investigators to perform multiple repetition trials while altering visual cues (obsuring the monitor during the force replication trial) to assess accuracy of FR through data output.

3.5.2. Oswestry Disability Index

The Oswestry Disability Index (ODI) was developed to increase objective measurements of disability related to low back pain. The ODI is a self-report questionnaire of the subject’s perceived disability related to LBP and consists of physical and social components. The ODI was specifically designed to measure physical activity levels and changes within those
levels. The Questionnaire consists of ten specific functional dimensions: pain intensity, pain variability, personal hygiene, lifting, walking, sitting, standing, sleeping, social activity and travel. This list encompasses a fairly comprehensive list of functional activity relevant to daily life. Each item is scored on a six point cardinal scale (0-5) and the sum of the responses is multiplied by 2 to create a percentage from 0-100%. Higher percentage scores indicate greater perceived disability with normal everyday functions. The minimum clinically important difference (MCID) has been established at 6% and is used to distinguish a difference that can be interpreted as a true change from a stable condition.

The ODI has a sufficient width scale to detect positive or negative changes with validity. The tool’s test-retest reliability (r= 0.94 – 0.99), construct validity, and responsiveness properties have been repeatedly evaluated as clinically effective. The ODI exhibits good overall responsiveness and demonstrates superior measurement properties when compared with other self-report questionnaires and is considered a gold-standard for measuring disability related to LBP. The ODI has been used as the reference standard for criterion validity for other disability questionnaires. The ODI has been translated into a number of different languages and has been validated in those languages making it an appropriate tool to use in LBP research because of widespread usage.

3.5.3. Visual Analog Scale – 24

The visual analog scale (VAS) is a self-reported pain assessment tool that requires the subject to place an X on a 10cm long straight line with stops on each end. The left stop equaling no pain and the right stop equaling the worst pain imaginable. The scale can then be broken down into a length in millimeters and expressed numerically from 0-100mm. The VAS has demonstrated test-retest stability of 0.82. Scrimshaw and Maher compared three pain scales
(VAS-current, VAS-24, and MPQ-24) for responsiveness and found that the VAS-24 was statistically more responsive to change. Responsiveness was measured using 3 tools including the receiver operating characteristics, the t-value for independent change and Spearman’s rank correlation coefficient, to ensure confidence in the results. All 3 tools demonstrated similar results. The authors concluded that the VAS-24 was the preferable pain tool for use in clinical trials and practice.279

3.5.4. **Numeric Rating Scale**

The numeric rating scale (NRS) is a self-reported pain scale that requires the subject to verbally rate their pain on a 0-10 scale with 0 being no pain and 10 being the worst pain imaginable.280 This scale is the most frequently used scale in clinical practice.281 The test-retest reliability and validity have been found to be sufficient for use in subjects with CLBP.282

The NRS score can be compared with the VAS and should result in a similar score as the VAS converted from millimeters to centimeters. Ohnhaus and Adler found that when the NRS and VAS are used in tandem, the scores can be arithmetically manipulated and the numeric values correlate well for pain rating (r = 0.84, p < 0.01) and for pain relief (r = 0.81, p < 0.001).283

3.5.5. **Fear and Avoidance Behavior Questionnaire**

The Fear and Avoidance Behavior Questionnaire (FABQ) is an instrument used to assess the subject’s perception of how their normal activity affects their back pain. It is divided into two sections, normal physical activity and work related activity. The FABQ may have some validity exploring potential psychosocial involvement that may limit the subject’s ability to participate in the physical testing of the study.
3.6. Testing Procedures

3.6.1. Pain and Disability Questionnaires

Each subject was asked to fill out several self-reporting instruments regarding their pain level and perceived level of disability including the ODI (Appendix B), NRS (Appendix C), VAS – 24 (Appendix D) and the FABQ (Appendix E) prior to performance of any testing procedure.

3.6.2. Subject Preparation

The subjects began their participation in this research project by signing the informed consent prior to any examination or intervention. Prior to testing, each subject received a general physical examination to ensure clinical presentation consistent with the inclusion criteria set forth in the research proposal and to obtain physical characteristics used for determining potential follow-up studies. The general physical examination included a comprehensive history, questioning the subject about their back pain, past treatments and general medical questions to be sure that they did not have any condition that may preclude the use of spinal manipulation for safety reasons (Physical exam, Appendix F). Range of motion and strength testing of the spine and hips to determine if any neurological involvement existed or if pain would limit strength demonstration. Neurological testing was performed as appropriate if the subject’s history was consistent with neurological involvement or if any myotome weakness may be suggested during strength testing. Neurological testing may have included dermatome sensory testing for light touch, deep tendon reflexes, straight leg raises and/or seated slump testing to assess nerve mobility. Neurological testing proved to be largely unnecessary since most of the subjects’ histories did not suggest neurological involvement. Special tests for segmental instability
including the prone segmental stability test and general spring testing through the lumbar spine and pelvis were performed as needed.

Between the pain questionnaires and the physical examination, all 5 factors identified by Flynn et al. as predictors of success in the clinical prediction rule for effectiveness of spinal manipulation were assessed. It was the intent of the investigators to do so in order to have the option to explore potential relationships in data analysis.

### 3.6.3. Order of Testing

The specific order of proprioception testing was randomized prior to the commencement of any testing procedure for each of the two or three testing sessions to avoid testing bias secondary to consistent testing order.

### 3.6.4. Trunk Proprioception

Trunk proprioception was assessed by first explaining the testing procedure to the subject followed by instruction to be seated in the semi-standing lumbar sagittal plane attachment for the Biodex. The subject was then strapped to the attachment using two velcro straps across the thighs and pelvis to minimize hip involvement, and the H-harness across the chest very firmly to ensure the trunk moved consistently with the dynamometer. The trunk attachment has a solid pad behind the sacrum to provide support to the sacral base maintaining a more upright seated posture during testing.

JPS was assessed starting from a semi-seated spine position comfortably placed in neutral. The limits of movement were placed within the subject’s comfortable range to prevent any movement from occurring that may increase their pain level significantly. The subject was blindfolded to limit visual cueing of position and the subject actively moved forward to the 30° target position, they were instructed to remember the position while it was held for 5 seconds.
The trunk was actively returned to the neutral position then the subject was instructed to move back to the target position and when the target position was achieved, they marked the position by depressing the hold button. The target and the reposition angles were recorded in a laptop computer. This procedure was repeated until 6 trials had been collected. The differences between the target position and the measured reposition effort (reposition error) were averaged for the 6 trials for data analysis.

TTDPM and the direction of movement was tested by placing the subject in the same neutral starting position, blindfolded and wearing headphones with white noise to reduce external visual and auditory input. The Biodex was started in passive mode at 0.25°/s. The investigator instructed the subject to press the hold button upon first perception of trunk motion.
and identify in which direction the movement occurred; therefore, the subject would have to let
the trunk move enough to detect the direction. The time from first instruction to commencement
of motion was randomly selected by the investigator between 3 and 20 seconds after the
investigator initiated the white noise. Six trials were performed, 3 toward flexion and 3 toward
extension in random fashion to establish accuracy of direction as well as sensitivity to
movement. The threshold angle of movement required (angle of detection – starting angle) was
averaged and the percentage of correct directional responses was recorded for statistical analysis.

Figure 3.3: Threshold to Detect Passive Motion Sense (Kinesthesia)

To measure FR, a 7-second maximal voluntary isometric contraction toward extension
was performed from neutral to establish a force value for testing. Fifty percent of this value was
calculated and used as the target force for replication. The 50% value has been found to be an
appropriate submaximal value for force replication studies in peripheral joints reducing demonstrable error rates.\textsuperscript{284, 285} This value was located on the computer monitor as a target line. The subject then performed an isometric extension contraction against the dynamometer to the target value. The force was held for 5 seconds. After a 5 second pause, the monitor was covered to eliminate visual feedback and the subject attempted to replicate the force for 5 seconds. This procedure was performed for 5 total pairs of trials. The force value for the last four seconds of each repetition of each pair of trials was averaged and the error rate determined by subtracting the mean value of the replication repetition (no visual reference) from the mean value of the reference (visual target driven) repetition. The 5 differences were averaged for statistical analysis.

Figure 3.4: Force Reproduction
3.7. Treatment

Following initial testing, the subject either received a spinal manipulation or a sham procedure (non-thrust) in the manipulative position to simulate a manual technique and blind the subject to treatment group. To perform the manipulation, the subject was positioned on their painful side, if clinical presentation indicated possible discogenic pathology or on the opposite side for all other pathology. The investigator attempted to isolate the involved dysfunctional segment by flexing the subject’s lower body from below until gapping was felt at the inferior spinous process. The subject’s trunk was then passively rotated toward the ceiling until the spinous process at the superior side of the spinal segment began to move, indicating any further movement would be biased to the segment in question. The investigator placed the inferior forearm against the posterior hip and the superior forearm across the anterior shoulder to increase rotation. The subject was taken to the end of available trunk rotation and was asked to take a deep breath. During the exhalation, the therapist engaged the end of range and applied a high velocity low amplitude thrust into the barrier of movement (see figure 3.4).
If no cavitation (audible pop) was perceived, a second thrust was performed. If cavitation was still not perceived, the subject was placed on the opposite side and the manipulation was repeated up to two more times.

In the sham procedure, the subject was placed on their side as in the manipulation. The lower body was flexed slightly to simulate manipulative procedure but not far enough to stimulate the flexion-relaxation phenomenon. The investigator’s inferior hand was placed on the upper lumbar spine to shield the region from movement while the forearm of the same hand was placed along the posterior hip. The superior forearm was placed on the shoulder to rotate the thoracic spine into midrange. This position of midrange thoracic rotation was held for 15 seconds before being returned to neutral (see figure 3.6). The subjects were told two interventions were being compared to blind the subjects to study goals.
After the intervention, all testing was repeated as described above following a randomized order. The subject also filled out the NRS to determine if testing or treatment impacted their CLBP.

Approximately one week later, each subject returned to the neuromuscular research lab for the next session. Prior to testing, each subject filled out an activity log questionnaire (Appendix: G) to determine if any substantive change occurred in their back condition during the period between testing sessions. In this testing session, each subject received the opposite treatment to act as their own controls by being subjected to the same testing procedures but with the opposite treatment. One week after the second testing session, the subjects from the sham 1st group returned for a third testing session. This final session was used to increase the statistical power of the 1-week residual effect of manipulation by having all manipulation sessions followed by a sham session one week later. This process was used to examine the second specific aim.
3.8. Data Analysis

3.8.1. Data Reduction

3.8.1.1. Proprioception

JPS was analyzed by taking the absolute value of the difference between the target angle and subject’s reposition angle. The mean difference for the six trails in each of the pre and post intervention testing sessions was entered in SPSS 15.0 for statistical analysis of Hypothesis 1.1.

TTDPM was analyzed by taking the absolute value of the difference between the starting angle and the test termination angle (angle of detection – starting angle). The mean difference for the six trials in each pre and post intervention testing sessions was entered in SPSS 15.0 for statistical analysis of Hypothesis 1.2.

Correct directional responses were assessed by taking the total number of correct responses and dividing it by the total possible responses (6) and multiplying it by 100 to get a percentage score. The pre and post intervention testing session scores were entered into SPSS 15.0 for statistical analysis of Hypothesis 1.3.

FR was analyzed by averaging the final 4 seconds of data from all ten of the 5-second repetitions of force production. Error rate was determined by subtracting the replication trial (eyes closed) mean from the reference (eyes open) trial. These data were collected at 100Hz and each trial was 5 seconds in length making the data set of each trial 500 data points. The first 100 points were deleted to leave the remaining 400 points for analysis. The average force values of these 400 points were used for comparison with the force replication trial. The absolute value of the difference between the trials was considered the error rate. The 5 differences were averaged and entered in to SPSS 15.0 for statistical analysis of Hypothesis 1.4.
3.8.1.2. Oswestry Disability Index

Each ODI item score was totaled and multiplied by 2 to obtain the ODI percentage score. Each percentage score was recorded and entered into SPSS 15.0 for statistical analysis of the homogeneity of the treatment groups.

3.8.1.3. Visual Analog Scale – 24

The mark entered on the sheet corresponding to the 24 hour average pain levels was measured from the left end-stop to obtain a measurement in mm which was divided by 10 to result in a length measurement in cm. These values were entered into SPSS 15.0 for statistical analysis of the homogeneity of the treatment groups.

3.8.1.4. Numeric Rating Scale

The reported values corresponding to current pain levels were recorded to ensure that each subject met the inclusion criteria of current pain level of 1 or less for the testing sessions. These values were entered into SPSS 15.0 for statistical analysis of the homogeneity of the treatment groups.

3.8.2. Statistical Analysis

3.8.2.1. General Characteristics

Randomizing subjects to treatment groups should ensure the groups represent the same general population of CLBP. This does not assume that all subjects with CLBP are homogeneous with respect to pathoanatomical causation or clinical presentation but the mean of the treatment groups would be generally similar. To test this assumption, group characteristics were entered into SPSS 15.0 and were examined using independent t-tests for parametric variables, Mann-
Whitney U tests for non-parametric variables and chi-square for non-parametric dichotomous variables with significance set at $\alpha = 0.05$.

### 3.8.2.2. Proprioception

A doubly-multivariate analysis with repeated measures was proposed to analyze the DVs for statistical significance. In an effort to use the most appropriate analysis, Pearson product-moment coefficients of correlation were performed to determine the strength of relationship between the DVs. The relationship between the 4 DVs was not very strong and did not demonstrate many statistically significant pairs of contrasts; therefore, it was decided to analyze these data using a univariate analysis. Tabachnick and Fidell reported that unless the DVs are either highly negatively correlated or moderately correlated, either positively or negatively, the ANOVAs are appropriate.

A mixed-model univariate analysis does not allow assessment of Sphericity. To account for this fact, a variance-covariance structure had to be theoretically assessed for goodness of fit. An autoregressive heterogeneous structure made theoretical sense and was used for this analysis.

The mean trial data from each of the testing sessions was entered into SPSS 15.0 for statistical analyses. These data were analyzed using a mixed-model univariate analysis with repeated measures for each of the four dependent variables of proprioception. If statistical significance was found, multiple comparisons were performed using paired t-tests with the appropriate Bonferroni adjustment. Paired t-tests were restricted to those comparisons that would directly measure pairs corresponding to the study hypotheses. For the Manip 1 $^{st}$ group, there were 4 time periods compared with pre and post intervention testing periods for each of two testing sessions. Rather than compare the 6 potential pairs of 4 time periods, the analyses compared time periods 1-2 and 3-4 for specific aim 1 and time periods 1-3 for specific aim 2.
Three paired t-tests results in a Bonferroni adjustment of $\alpha = 0.017$. For the Sham 1st group, having 3 testing sessions and 6 time periods, 15 total potential pairs existed. Five pairs were analyzed including 1-2, 3-4, and 5-6 for specific aim 1 and 1-3 and 3-5 for specific aim 2 (see figure 3.5). Five paired t-tests results in a Bonferroni adjustment of $\alpha = 0.01$.

**Figure 3.7: Schema Employed for Multiple Comparisons**

<table>
<thead>
<tr>
<th>Residual Effect</th>
<th>Rx Effect</th>
<th>Residual Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Period 1</td>
<td>Period 2</td>
</tr>
<tr>
<td>Pre Sham</td>
<td>Post Manip</td>
<td>Pre Sham</td>
</tr>
<tr>
<td></td>
<td>Period 3</td>
<td>Period 4</td>
</tr>
<tr>
<td>Pre Manip</td>
<td>Post Sham</td>
<td></td>
</tr>
</tbody>
</table>

Rx Effect = treatment effect for specific aim 1
Residual Effect = 1 week effect for specific aim 2
4. RESULTS

The purpose of this study was to compare the treatment effects of spinal manipulation with a sham procedure on proprioception of the trunk. The one week residual effect of manipulation was also analyzed. A randomized, controlled, crossover design was employed for this study (see Table 4.1). Dependent variables included joint Position sense (JPS), threshold to detect passive motion (TTDPM), direction of passive motion (DM), and force reproduction (FR). The independent variables included treatment group and time. The treatment implemented was a rotational, neutral gapping manipulation at the segmental level provocative for the subject’s symptoms. The non-manipulative sham procedure involved setting the provocative segmental level but sustaining a mid-range position for 15 seconds rather than stretching or thrusting into end-range. A repeated measures MANOVA was proposed for all dependent variables across each point in time, 4 points for the group who received the manipulation first (Manip 1st) and 6 points for group who received the sham procedure first (Sham 1st). The significance level was set at $\alpha = 0.05$ a priori. The significant level within each dependent variable was ascertained via post hoc paired t-tests employing Bonferroni corrections.

<table>
<thead>
<tr>
<th></th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manip 1st</td>
<td>Manipulation</td>
<td>Sham</td>
<td></td>
</tr>
<tr>
<td>Sham 1st</td>
<td>Sham</td>
<td>Manipulation</td>
<td>Sham</td>
</tr>
</tbody>
</table>

Table 4.1: Research Project Design
4.1. **Instrumentation**

Consistency of the instrumentation was examined using a Model 3 ICC (3,k) as described by Shrout and Fleiss.\textsuperscript{269} The purpose of this analysis was to determine if the instrumentation implemented was reliable for use with subjects reporting CLBP. The statistical analysis was performed by the software package R 2.4.1 (The R Foundation for Statistical Computing, 2006) and table 4.2 contains a summary of ICCs and SEMs for each dependent variable assessed in this study.

<table>
<thead>
<tr>
<th>Measure</th>
<th>ICC</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>TTDPM</td>
<td>0.904</td>
<td>0.0471°</td>
</tr>
<tr>
<td>JPS</td>
<td>0.785</td>
<td>0.1058°</td>
</tr>
<tr>
<td>FR</td>
<td>0.607</td>
<td>0.1937N*m</td>
</tr>
</tbody>
</table>

Table 4.2: Reliability of Instrumentation for Proprioception Measures

4.2. **Group Characteristics**

Group characteristics were examined using independent samples t-tests for parametric variables and Mann-Whitney U tests for non-parametric variables with significance set at $\alpha = 0.05$.

4.2.1. **General Demographic Characteristics**

Subjects for this study were otherwise healthy males and females between the ages of 24 -54 presenting with CLBP. Descriptive demographic data with comparisons for each subject group can be found in Table 4.3. Independent samples t-tests for parametric variables, and a Pearson chi-square test for dichotomous, non-parametric variable (gender) determined that there
were no statistically significant differences between the groups for general demographic characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Manip 1st Mean ± (SD)</th>
<th>Sham 1st Mean ± (SD)</th>
<th>T</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>37.4 (9.21)</td>
<td>37.25 (8.65)</td>
<td>0.052</td>
<td>31</td>
<td>0.959</td>
</tr>
<tr>
<td>Height</td>
<td>1.762m (0.092m)</td>
<td>1.759m (0.087m)</td>
<td>0.113</td>
<td>31</td>
<td>0.911</td>
</tr>
<tr>
<td>Mass</td>
<td>85.53kg (11.48kg)</td>
<td>85.43kg (17.77kg)</td>
<td>0.019</td>
<td>31</td>
<td>0.985</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Pearson $\chi^2$</th>
<th>Asymp. Sig (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>12/17</td>
<td>12/16</td>
</tr>
<tr>
<td>Women</td>
<td>5/17</td>
<td>4/16</td>
</tr>
</tbody>
</table>

Table 4.3: Descriptive Statistics for Subject General Characteristics

4.2.2. Pain and Disability Measures

The subjects for this study demonstrated signs and symptoms of CLBP, operationally defined as LBP of greater than 3 months duration or recurrent episodic LBP with at least 2 episodes during the previous 12 months. All subjects were relatively pain-free at the time of testing with 30 subjects reporting a pain level of 0/10 and a group mean of 0.12. Multiple tools were used to examine the subjects’ activity level, signs, and symptoms throughout the testing procedure. Descriptive statistics with statistical analysis can be found in Table 4.4. Independent samples t-tests determined that there were no statistically significant differences between the groups for pain and disability characteristics.
<table>
<thead>
<tr>
<th></th>
<th><strong>Manip 1st Mean ± (SD)</strong></th>
<th><strong>Sham 1st Mean ± (SD)</strong></th>
<th><strong>T</strong></th>
<th><strong>df</strong></th>
<th><strong>Sig. (2-tailed)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ODI</strong></td>
<td>22.12% (16.7%)</td>
<td>25.33% (19.68%)</td>
<td>-0.772</td>
<td>31</td>
<td>0.446</td>
</tr>
<tr>
<td><strong>NRS</strong></td>
<td>0.12 (.49)</td>
<td>0.25 (.68)</td>
<td>-0.645</td>
<td>31</td>
<td>0.524</td>
</tr>
<tr>
<td><strong>VAS-24</strong></td>
<td>0.759cm (.788cm)</td>
<td>1.0cm (1.05cm)</td>
<td>-0.489</td>
<td>31</td>
<td>0.628</td>
</tr>
<tr>
<td><strong>FABQ – PA</strong></td>
<td>16.0 (6.02)</td>
<td>16.57 (4.20)</td>
<td>0.059</td>
<td>30</td>
<td>0.955</td>
</tr>
<tr>
<td><strong>FABQ – W</strong></td>
<td>9.65 (7.51)</td>
<td>9.62 (9.09)</td>
<td>-0.478</td>
<td>31</td>
<td>0.636</td>
</tr>
</tbody>
</table>

ODI = Oswestry Disability Index, NRS = numeric rating scale, VAS-24 = 24 hr. visual analog scale, FABQ – PA = Fear and Avoidance Behavior Questionnaire – physical activity subscale, and FABQ – W = Fear and Avoidance Behavior Questionnaire – work subscale

Table 4.4: Descriptive Statistics for Pain & Disability Level

4.2.2.1. Oswestry Disability Index

The ODI was used to examine how severe the subjects’ worst episode of LBP during the previous 12 months differed. Each subject was instructed to score the instrument using their most severe pain level as a reference. There were no statistically significant differences between groups for the ODI. Variance levels in ODI were low enough to use independent sample t-tests comparisons.

4.2.2.2. Visual Analog Scale – 24

The VAS-24 was measured at the beginning of each testing session. The initial measures were included in the group comparison analysis to determine if the treatment groups were significantly different.

Follow-up measures were examined to determine if the subjects’ symptoms changed during the course of testing. While subjects may have been pain-free at the time of testing, many subjects reported that they had fairly consistent (predictable) symptoms during the course of a day or week. Most subjects reported little change in their usual pain behavior but one subject notably demonstrated a reduction in pain over the course of the testing sessions. Examining this subject’s data revealed that it behaved similarly to the group means implying that the symptom change did not impact the data observed.
4.2.2.3. Numeric Rating Scale

All subjects began testing in a relative pain-free state. Two subjects reported that during the manipulative treatment session, pain increased temporarily but subsided to a pain-free level by the end of the post intervention session. One subject reported significant pain at the beginning of the second testing session. Gentle stretching and ambulation was prescribed and adequately reduced symptoms prior to initiation of the testing session. There were no statistically significant differences between groups for the NRS scores taken at the initial testing session.

4.2.2.4. Fear and Avoidance Behavior Questionnaire

Fear and Avoidance Behavior Questionnaire scores were taken at the beginning of the initial testing session and examined for similarity. The work and physical activity subsets were analyzed separately since they may have different implications in function. FABQ-PA and FABQ-W scores taken at the initial testing session were found to lack statistical significance.

4.2.2.5. Activity Log

An activity log was filled out by subjects at each follow-up testing session to determine if they had changed activity levels and how that change may have impacted their self-reported symptoms. Without exception, the activity logs demonstrated that the subjects performed only their normal activity levels throughout the time of testing. In a few instances, subjects reported either mildly increased or decreased symptoms during or between session intervals but in no instance did their activity or symptom change demonstrate a scenario that might impact study results.
4.2.3. General Physical Characteristics

Physical characteristics were measured before the initiation of testing to ensure that inclusion criteria were met while exclusion criteria were avoided. Mobility and stability of the spine have been associated with clinical effectiveness of spinal manipulation therapy (SMT) and were recorded prior to testing. The lumbar spine was spring tested for hypomobility. Functional instability was examined using the prone segmental instability test (PSI).

Since clinical effectiveness of an audible pop during SMT has been examined and reported on in the literature, it was recorded during treatment sessions in this study. All physical characteristics examined did not prove to be significantly different for the treatment group during this study. Descriptive statistics with statistical analyses can be found in Table 4.5. Mann-Whitney U tests for the SMT clinical prediction rule and Pearson chi-square tests for mobility and stability determined that there were no statistically significant differences between the groups for physical characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Manip 1st</th>
<th>Sham 1st</th>
<th>Mann-Whitney U</th>
<th>Asymp. Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPR*</td>
<td>12/17 total</td>
<td>13/16 total</td>
<td>88.000</td>
<td>0.063</td>
</tr>
<tr>
<td>3</td>
<td>10</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Pearson $\chi^2$</th>
<th>Asymp. Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Audible</td>
<td>0.308</td>
<td>0.579</td>
</tr>
<tr>
<td>Hypomobility</td>
<td>1.588</td>
<td>0.208</td>
</tr>
<tr>
<td>(+) PSI</td>
<td>0.013</td>
<td>0.909</td>
</tr>
</tbody>
</table>

CPR = Clinical Prediction Rule *At least 3 of 5 predictors
PSI = Prone segmental instability test

Table 4.5: Descriptive Statistics for Physical Characteristics
4.3. **Trunk Proprioception**

Correlation analysis of the data demonstrated a weak relationship between the means of dependent variables with JPS and DM having a relationship of $r = -0.347$ with $p = 0.048$. All other DV relationships were non-significant. Comparing individual trials of data, 15 of 216 pairs showed significance yielding a significance rate of $p = 0.067$ or 4 pairs greater than chance alone suggesting a weak relationship and supporting the use of a univariate analysis of the DVs.

4.3.1. **Joint Position Sense**

Mixed-model repeated measures univariate analysis demonstrated a simple main period effect for JPS ($p = 0.016$). It was further determined through post hoc analysis that the significance was found between time periods 1 and 3 with $p = 0.006$ for Manip 1st and between time periods 1 and 2 with $p = 0.005$ for Sham 1st. These results suggest the sham procedure produced greater reduction in error for JPS than manipulation but that the improvement in JPS from manipulation was maintained, in fact enhanced, one week post intervention. Further improvement shown one week later was statistically significant. The significant improvement noted following the sham procedure provided during session one was partially lost at the one week follow-up for Sham 1st group. Please see tables 4.6 – 4.7 and figure 4.1 for details.

<table>
<thead>
<tr>
<th>Source</th>
<th>Numerator df</th>
<th>Denominator df</th>
<th>F</th>
<th>Sig.</th>
<th>Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>1</td>
<td>35.364</td>
<td>0.419</td>
<td>0.521</td>
<td></td>
</tr>
<tr>
<td>Period</td>
<td>5</td>
<td>70.552</td>
<td>3.026</td>
<td>0.016</td>
<td>0.91</td>
</tr>
<tr>
<td>Group * Period</td>
<td>3</td>
<td>61.526</td>
<td>0.776</td>
<td>0.512</td>
<td></td>
</tr>
</tbody>
</table>

*Table 4.6: Joint Position Sense: Mixed-Model Analysis*
### Paired Differences

<table>
<thead>
<tr>
<th>Time Periods compared</th>
<th>Mean</th>
<th>SD</th>
<th>98.33% CI Manip 1st</th>
<th>99% CI Sham 1st</th>
<th>t</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
<th>Power</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Manip 1st</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-2</td>
<td>0.694</td>
<td>1.976</td>
<td>-0.587</td>
<td>1.974</td>
<td>1.447</td>
<td>16</td>
<td>0.167</td>
<td>0.85</td>
</tr>
<tr>
<td>1-3</td>
<td>1.049</td>
<td>1.372</td>
<td>0.160</td>
<td>1.938</td>
<td>3.151</td>
<td>16</td>
<td>0.006</td>
<td>0.17</td>
</tr>
<tr>
<td>3-4</td>
<td>-0.387</td>
<td>1.039</td>
<td>-1.060</td>
<td>0.286</td>
<td>-1.536</td>
<td>16</td>
<td>0.144</td>
<td>0.75</td>
</tr>
<tr>
<td><strong>Sham 1st</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-2</td>
<td>0.816</td>
<td>1.005</td>
<td>0.075</td>
<td>1.556</td>
<td>3.247</td>
<td>15</td>
<td>0.005</td>
<td>0.94</td>
</tr>
<tr>
<td>1-3</td>
<td>0.528</td>
<td>1.437</td>
<td>-0.531</td>
<td>1.586</td>
<td>1.468</td>
<td>15</td>
<td>0.163</td>
<td>0.30</td>
</tr>
<tr>
<td>3-4</td>
<td>0.208</td>
<td>1.534</td>
<td>-0.922</td>
<td>1.337</td>
<td>0.541</td>
<td>15</td>
<td>0.596</td>
<td>0.08</td>
</tr>
<tr>
<td>3-5</td>
<td>0.603</td>
<td>1.599</td>
<td>-0.575</td>
<td>1.781</td>
<td>1.508</td>
<td>15</td>
<td>0.152</td>
<td>0.31</td>
</tr>
<tr>
<td>5-6</td>
<td>-0.315</td>
<td>0.700</td>
<td>-0.831</td>
<td>0.201</td>
<td>-1.801</td>
<td>15</td>
<td>0.092</td>
<td>0.54</td>
</tr>
</tbody>
</table>

Table 4.7: Joint Position Sense: Multiple Comparisons

![Figure 4.1: Results for Joint Position Sense](image-url)

Figure 4.1: Results for Joint Position Sense
4.3.2. **Threshold to Detect Passive Movement**

Mixed-model repeated measures univariate analysis demonstrated a simple main period effect for TTDPM with \( p = 0.044 \). It was also determined that a group * period interaction with \( p = 0.013 \). It was further determined through post hoc analysis that the significance was found between time periods 1 and 2 with \( p = 0.008 \) for Group A. See tables 4.8 – 4.9 and figure 4.2 for details.

<table>
<thead>
<tr>
<th>Source</th>
<th>Numerator df</th>
<th>Denominator df</th>
<th>F</th>
<th>Sig.</th>
<th>Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>1</td>
<td>28.897</td>
<td>3.532</td>
<td>0.070</td>
<td></td>
</tr>
<tr>
<td>Period</td>
<td>5</td>
<td>43.338</td>
<td>2.514</td>
<td>0.044</td>
<td>0.95</td>
</tr>
<tr>
<td>Group * Period</td>
<td>3</td>
<td>41.322</td>
<td>4.048</td>
<td>0.013</td>
<td></td>
</tr>
</tbody>
</table>

Table 4.8: Threshold to Detect Passive Motion: Mixed-Model Analysis

<table>
<thead>
<tr>
<th>Time Periods compared</th>
<th>Mean</th>
<th>SD</th>
<th>98.33% CI Manip 1st</th>
<th>99% CI Sham 1st</th>
<th>t</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
<th>Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manip 1st</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-2</td>
<td>0.317</td>
<td>0.432</td>
<td>0.037</td>
<td>0.597</td>
<td>3.026</td>
<td>16</td>
<td>0.008</td>
<td>0.81</td>
</tr>
<tr>
<td>1-3</td>
<td>0.221</td>
<td>0.538</td>
<td>-0.128</td>
<td>0.570</td>
<td>1.693</td>
<td>16</td>
<td>0.110</td>
<td>0.36</td>
</tr>
<tr>
<td>3-4</td>
<td>-0.108</td>
<td>0.381</td>
<td>-0.355</td>
<td>0.138</td>
<td>-1.175</td>
<td>16</td>
<td>0.257</td>
<td>0.27</td>
</tr>
</tbody>
</table>

| Sham 1st              |      |     |                     |                 |      |     |                 |       |
| 1-2                   | 0.002| 0.176| -0.128             | 0.131           | 0.043| 15  | 0.967           | 0.01  |
| 1-3                   | -0.041| 0.253| -0.227             | 0.145           | -0.653| 15  | 0.524           | 0.10  |
| 3-4                   | 0.073| 0.159| -0.044             | 0.190           | 1.841| 15  | 0.086           | 0.44  |
| 3-5                   | -0.026| 0.194| -0.170             | 0.117           | -0.540| 15  | 0.597           | 0.10  |
| 5-6                   | 0.031| 0.152| -0.081             | 0.144           | 0.820| 15  | 0.425           | 0.12  |

Table 4.9: Threshold to Detect Passive Motion: Multiple Comparisons
Figure 4.2: Results for Threshold to Detect Passive Motion

4.3.3. Direction of Passive Movement

Mixed-model repeated measures univariate analysis demonstrated no significant simple main effect for period with $p = 0.791$. It was found that the data with multiple time periods with a variance of zero would not run using the autoregressive heterogeneous model so this dependent variable was analyzed using an autoregressive model.

Subjects reported very few trials inaccurately either before or after intervention. The error rate was approximately 0.04% with only 4 errors in 996 trials. The means and standard deviations for each treatment group can be found in the figure 4.3. The results of the statistical analyses can be found in tables 4.10.
Table 4.10: Direction of Movement: Mixed-Model Analysis

<table>
<thead>
<tr>
<th>Source</th>
<th>Numerator df</th>
<th>Denominator df</th>
<th>F</th>
<th>Sig.</th>
<th>Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>1</td>
<td>60.635</td>
<td>0.266</td>
<td>0.608</td>
<td></td>
</tr>
<tr>
<td>Period</td>
<td>5</td>
<td>104.177</td>
<td>0.480</td>
<td>0.791</td>
<td>0.51</td>
</tr>
<tr>
<td>Group * Period</td>
<td>3</td>
<td>107.715</td>
<td>0.991</td>
<td>0.339</td>
<td></td>
</tr>
</tbody>
</table>

Figure 4.3: Results for Direction of Passive Movement

4.3.4. Force Reproduction

Mixed-model repeated measures univariate analysis demonstrated no statistically significant effect for FR with p = 0.063. See tables 4.11 and figure 4.4 for details.
<table>
<thead>
<tr>
<th>Source</th>
<th>Numerator df</th>
<th>Denominator df</th>
<th>F</th>
<th>Sig.</th>
<th>Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>1</td>
<td>35.316</td>
<td>0.179</td>
<td>0.675</td>
<td></td>
</tr>
<tr>
<td>Period</td>
<td>5</td>
<td>50.801</td>
<td>2.258</td>
<td>0.063</td>
<td>0.82</td>
</tr>
<tr>
<td>Group * Period</td>
<td>3</td>
<td>52.108</td>
<td>0.752</td>
<td>0.526</td>
<td></td>
</tr>
</tbody>
</table>

Table 4.11: Force Reproduction: Mixed-Model Analysis

![Force Reproduction Graph]

Figure 4.4: Results for Force Reproduction

4.4. Summary

The results of the study do not consistently support the proposed hypotheses of specific aims 1 and 2. For specific aim 1, hypothesis 1.2 was supported for KIN. Hypotheses 1.1, 1.3, and 1.4 were not supported. For specific aim 2, hypothesis 2.0 was supported for JPS while KIN, DM and FR were not supported.
5. DISCUSSION

5.1. Effects of Spinal Manipulation

The purpose of this study was to examine the effects of spinal manipulation on trunk proprioception in subjects with chronic low back pain. The first specific aim was to measure proprioception in the lumbar spine before and after a localized spinal manipulation in subjects with CLBP and compare the clinical effects of spinal manipulation with a non-thrust, sham procedure. The second specific aim was to assess the residual effect of spinal manipulation compared with a sham procedure on subjects with CLBP one week after the manipulative intervention.

With respect to Specific Aim 1, Hypotheses 1.1 – 1.4, spinal manipulation had a statistically significant effect on TTDPM in Manip 1st group subjects only. For the second Specific Aim, Hypothesis 2.1, spinal manipulation had a statistically significant one week residual effect on JPS alone. No statistically significant effects were observed for either DM or FR. In general, there was also a tendency to maintain an improvement in proprioception during the pre-intervention testing period one week following the intervention but this tendency was only significant for JPS.

For joint position sense, these data supported the null hypothesis as time periods 1 and 2 for the Manip 1st group and time periods 3 and 4 for the Sham 1st group failed to achieve significance with p = 0.167 and p = 0.596 respectively. For threshold to detect passive
movement, the null hypothesis was rejected for the Manip 1st group since time periods 1 and 2 demonstrated \( p = 0.008 \) but the null hypothesis was not rejected for the Sham 1st group because time periods 3 and 4 failed to achieve significance with \( p = 0.086 \). For Direction of Movement, these data supported the null hypothesis as both groups failed to achieve significance. For Force Reproduction, the null hypothesis was not rejected since significance was not demonstrated for either group. For the 1 week residual effect, the null hypothesis was largely upheld since the Manip 1st group time periods 1 and 3 only achieved statistical significance for JPS with \( p = 0.006 \) but failed to demonstrate significance for threshold to detect passive movement (\( p = 0.110 \)), force reproduction (\( p = 0.160 \)), and direction of movement (\( p = 0.333 \)). The Sham 1st group time periods 3 and 5 failed to achieve significance with joint position sense (\( p = 0.152 \)), threshold to detect passive movement (\( p = 0.597 \)), force reproduction (\( p = 0.317 \)), and direction of motion (\( p = 0.333 \)).

Reviewing the results outlined above, it can be suggested that each subsystem of proprioception did not respond the same over the course of this study. These results are consistent with other reports of discontinuity between proprioceptive subsystems within the literature.\(^{120,218}\)

### 5.1.1. Joint Position Sense

Joint position sense showed a non-significant improvement trend for both treatment groups following spinal manipulation. Neither group demonstrated enough improvement to achieve statistical significance; however, the Manip 1st group improved more than the Sham 1st group which approached significance. It is interesting to note that even though both groups showed a general trend toward improvement over the course of each data collection session, during the testing session following manipulation, pre-intervention testing showed improvement.
but this improvement was lost in the post-intervention trials as error rates increased. Comparing the second sham testing session with the first sham session for the Sham 1\textsuperscript{st} group, it is clear that the statistically significant improvement demonstrated in the first session was not repeated in the post manipulation session. This difference might suggest that any cutaneous input would result in an enhanced proprioceptive response in CLBP. In the sham procedure, cutaneous input would be provided by the investigator simply by placing hands on the lumbar spine during the sham procedure set-up. This finding is consistent with conclusions reported by Newcomer.\textsuperscript{136} Once initial improvements had been made, either by treatment or by learning, the cutaneous input was no longer sufficient to replicate the improvement in the follow-up session. Since cutaneous input was not as effective in improving error rate in normal subjects as those with LBP, the improved level may not have demonstrated a large enough deficit to show improvement with additional cutaneous input or the improvement brought the subject’s performance to a level where further cutaneous input could not increase physiologic performance.

5.1.2. \textbf{Threshold to Detect Passive Motion}

TTDPM demonstrated different tendencies from JPS. Sham 1\textsuperscript{st} group test results revealed little change throughout testing irrespective of treatment condition. There was minor improvement in error following the manipulation intervention with no residual effect noted a week later. The Manip 1\textsuperscript{st} group showed statistically significant improvement following the manipulative intervention, but the improvement was not maintained during the following testing session. Like JPS, TTDPM deteriorated in the post-intervention testing trials the week after manipulation. Again, this post-intervention deterioration is inexplicable. The sham intervention should have no deleterious effect on proprioception. By definition, a sham intervention should have no effect at all, yet it appears to have either an improving or deleterious effect depending on
the timing of that treatment. Looking at the means of the errors (figure 4.2), it is apparent that a
difference exists between the treatment groups during their initial testing session for TTDPM.
This statistically significant difference may likely explain a large portion of the difference noted
in response to the interventions. If you compare the error rates throughout testing, there is a
difference between the groups but that difference is magnified during the initial testing session. It
is also worth noting that for the Sham 1st group, pre-intervention error rates progressively
increase throughout the study. This observation is specific to TTDPM since it was not observed
in any of the other dependent variables.

5.1.3. Direction of Movement

In this study, direction of movement demonstrated error rates considerably lower (0.04%)
than those reported in other literature examining this variable. Leinonen et al. found that
76.9% of subjects reported the wrong direction of trunk rotation in at least 20% of trials
suggesting a significantly higher error rate than demonstrated in the current study. It should be
pointed out that Leinonen’s study examined subjects with a specific pathoanatomic causation
(spinal stenosis) which may result in differences from the current study since the subjects who
develop spinal stenosis tend to be older than in the current study. The average age of subjects in
Leinonen’s study was 56.37 years compared with 37.33 years in the current study. Age related
variation in TTDPM has been reported on in the literature. Even though direction of
movement differs from TTDPM, DM may be fundamentally linked to TTDPM since both
proprioception subsystems depend on detection of passive motion and the two subsystems
demonstrated deficits when studied concomitantly. During the course of this study, too few
errors were made in the direction of movement to lend credence to a meaningful analysis. The
univariate analysis demonstrated no significant period effect for the dependent variable.
There are reasons why these results may have been observed. First, subjects may have stopped the Biodex not when they felt movement but when they knew which direction the Biodex was moving them which would alter the results of both dependent variables. They would improve their performance in the DM but reduce their performance in TTDPM. Comparing the error rates observed in this study with error rates reported in previously published literature\textsuperscript{128, 138, 218} suggests that the observed error rate was comparatively low. Since the instrumentation used in this study is not the same as reported in the previously mentioned studies, conclusions about comparisons should be made with caution. It is entirely possible that results of the current study are dependent on the methodology employed since it incorporated different instrumentation as well as multiple components of proprioception.

Second, the lack of errors in DM may result from the instrumentation used to measure the dependent variable. The short delay that inevitably occurs between the realization that movement has occurred and depressing the switch to stop the Biodex may be enough to allow the subject to better appreciate the direction of movement by the time the investigator asked for the direction of movement. Even though every type of instrumentation will have some delay, there may be some variability in the devices used that may have increased response times in this particular study. Incorporating this logic into the discussion suggests that the instrument is really testing the hand response to the perception of movement more so than the perception of movement itself\textsuperscript{138}. Future studies may benefit from an improvement in the mechanism used to stop the instrument from passively moving the trunk but until a consistent mechanism is devised for all instrumentation, this limitation will continue to be apparent.
5.1.4. Force Reproduction

It is apparent that FR may not have demonstrated any meaningful change over the course of the study. The univariate analysis did not reveal a statistically significant difference but approached significance for a period effect with $p = 0.063$. A non-significant improvement was noted for the Manip 1st group following manipulative intervention. The one-week follow-up session also showed improvement in both pre and post sham intervention sessions. The Sham 1st group subjects showed a consistent pattern of reduction in error over the course of testing. Analyzing the improvement trend revealed a statistically significant within-subjects linear pattern in the Sham 1st group with $p = 0.023$. The within-subjects linear pattern was not significant for the Manip 1st Group with $p = 0.067$. This linear pattern suggests that the improvement observed may be due to learning rather than treatment effects since the means of the error rates progressively reduced throughout the course of the study. Learning through multiple repetitions of force reproduction has been previously demonstrated in a study examining force production characteristics in subjects with CLBP. However, the number of repetitions required to produce multiple consecutive trials within the 10% variance range were not reported to determine how this study’s results may have compared to those of Descarreaux et al.

The impact of learning in conscious proprioception warrants further investigation. When first considered, learning appears to be a negative consequence of the methodological design, but the possible clinical impact of learning must be addressed. It is possible that the learning effect through the high number of repetitions performed over a two week period is an acceptable way to treat deficits noted in this population. At least two randomized controlled trials have used repetition to train subjects with CLBP to perform two separate subsystems of proprioception at a level of variance and error rate consistent with healthy control subjects. It is possible to
design a future study that not only looks at proprioception (demonstrable error rates) but also assesses neuromuscular control by other means (strategies to obtain these error rates) to explore the clinical implications of learning on neuromuscular control and function in subjects with CLBP. Examining strategies to perform neuromuscular testing could incorporate a qualitative research component with videotaping to investigate differences in strategy or randomized controlled trials using different strategies could be compared for relative effectiveness. Studying both the number of trials used and the subjects’ strategies for performing those trials may further validate the conclusions described by Descarreaux et al.\textsuperscript{287} in their study of the number of trials required to obtain a standardized performance level in subjects with trunk proprioception deficits and strategies in neuromuscular control.

Considering the potential sources of variability in the performance of motor tasks could explain some of the results revealed in this study. Joint position sense and force reproduction require memory of position and force to reproduce without visual feedback. The sensory system should explain a significant amount of that variability of performance due to uncertainty in target position and force sense.\textsuperscript{289} In tasks that require complex movement or memory, a sizeable amount of variation could be explained by preparatory planning in addition to sensory uncertainty.\textsuperscript{290} One might assume that many repetitions of a given task would slowly reduce this central source of variability leaving only sensory system variability. It would not be readily presumed that central control in premotor planning would explain variability of performance in a well learned task yet Churchland et al.\textsuperscript{290} suggested that approximately 50% of the variability in velocity control in a simple task resulted from preparatory planning. These findings suggest that no matter how well learned the task; premotor planning explains variability in task performance. Perhaps there is a central factor involved in subjects with musculoskeletal injuries in addition to
the sensory system deficits that are often implicated. One spine study included a peripheral joint reposition test to theoretically rule out central processing from having an impact of spine proprioceptive deficits. It is possible that these central factors are influenced by the sensory system.

5.1.5. One-Week Residual Effect

The results of this study are inconclusive for an improvement in the subjects’ performance one week after spinal manipulation. In the case of the Manip1st group, a statistically significant improvement was noted between the pre-manipulation intervention test session with the pre-intervention test session one week later even though deterioration was noted in the post-intervention session. Force Reproduction demonstrated a non-significant yet similar pattern of improvement one week later but TTDPM showed a one week follow-up session that was better than the pre-manipulation test session but worse than the post-manipulation testing session showing that some of the gains noted through manipulation were lost during the ensuing week.

The Sham 1st group demonstrated different results. Force reproduction and JPS revealed steady improvement over all three testing sessions while TTDPM showed slight deterioration over all three testing sessions. These results suggest that learning played a primary role in determining the outcomes. Only the sham 1st initial sham session showed a statistically significant period effect, all other sessions were non-significant variations. The residual analysis further supports the idea that using proprioception testing as an intervention to improve proprioception may be a viable clinical option; however, further testing is required to determine how much of the variation noted in this study is attributable to treatment vs. learning.
5.2. Subjects with CLBP may not Demonstrate Proprioception Deficits

By implementing the operational definition of CLBP used in this study, the subjects did not demonstrate a significant amount of LBP at the time of testing. Motor control literature suggests that this may not be a limitation since periods of reduced pain may not be associated with normal levels of neuromuscular control; however, it is not currently known how variable trunk proprioception may be based on symptom level.

Previous studies examining proprioception in subjects with CLBP have not identified that they used pain-free subjects during testing. One recent study did use pain-free subjects during testing who previously had significant episodes of LBP within the past year. This study found deficits in JPS for flexion in subjects with previous LBP compared with subjects without LBP. It should also be noted that Tsai et al. did not find significant differences for any other direction of movement for JPS besides flexion. The findings of Tsai et al. are somewhat consistent with other findings noted in the literature. Newcomer et al. found that there were no significant differences between subjects with CLBP and healthy controls for any plane of lumbar motion but did find a significant difference in error rates between all subjects tested for flexion, compared with all other planes of movement. This suggests that flexion is inherently different than other planes of movement because mobility is increased through a reduction of segmental resistance during mid-range movement as compared with other planes of motion. Neither Tsai et al. nor Newcomer et al. examined any other subsystem of proprioception so it cannot be speculated whether or not their results may be comparable to TTDPM or FR.

A possible explanation for the results demonstrated in this study is that subjects with CLBP who are currently pain-free may not demonstrate the same level of deficit noted in subjects experiencing LBP at the time of testing. It is not possible to fully examine this potential
difference since this study did not include subjects who experienced pain at the time of testing. It is however, possible to examine the relationship between healthy subjects with those demonstrating CLBP who were pain-free at the time of testing. During reliability testing of the isokinetic instrumentation used in the current study, 17 healthy subjects were examined for proprioception. The results of the reliability testing were reported previously in this paper. To examine the question of level of deficit, the pre-intervention testing scores of the first testing session of the healthy subjects was compared to the same pre-intervention session of this study’s test subjects (Manip 1st group and Sham 1st group) through one-way ANOVAs. The results demonstrated that the differences in error rates between the 3 groups were minimal. These data suggest that the only statistically significant difference lie between Manip 1st group and Sham 1st group for TTDPM with p = 0.030 and Manip 1st group approximating the healthy group and the Sham 1st Group being significantly lower. All other comparisons failed to achieve significance, suggesting that the potential effect size estimated in the a priori power analysis was too large to demonstrate statistical significance through the intervention proposed at the sample size selected. If the groups were broken down by healthy vs. CLBP and independent samples t-tests are used to compare the means of the first trials, no statistically significant differences were noted in the analysis.

These analyses suggest that subjects with CLBP who are currently pain-free do not consistently differ from normal test subjects in any substantive way with reference to conscious proprioception as determined by this particular methodology. If the study subjects did not differ from normal, then it is entirely possible that the results of the study are altered by this lack of difference. A correlation between level of proprioception deficit and subject back pain would imply that the level of pain may be an indicator of proprioception and neuromuscular
performance. Reviewing other studies\textsuperscript{224, 225} performed on normal subjects and those with LBP, pain-free status for one-year is often used as an inclusion criterion for the healthy control group. This definition does not exclude subjects with CLBP from being control subjects as long as their conditions have been under control for more than one year.

This finding is not consistent with other studies\textsuperscript{138, 224} that examined conscious proprioception in subjects with CLBP suggesting that the differences found here may result from the subjects’ pain status at the time of testing. It may also suggest that the instrumentation was not sensitive enough to ascertain differences present in this population of subjects with CLBP suggesting that this instrumentation is not a valid measure of proprioception.

These data suggest that test subjects in the study statistically resemble normal subjects and may imply that decreased neuromuscular control of the trunk is not involved in subjects with CLBP, challenging the argument that NMC is the subsystem responsible for high recurrence rates of LBP. A potential reason for this observation may lie in the method of testing proprioception in this study. The testing procedures examined in this study represent conscious proprioception since the subject was either actively reproducing a position or force or the conscious perception of passive movement and direction. Another aspect of proprioception involves the unconscious control and perception of movement. This aspect is more likely involved in injury since it is responsible for the immediate response to the unpredicted perturbation that can happen to the athlete during sport and the non-athlete during function. The methodology employed in this study cannot make any judgment related to unconscious control making further research necessary to address this issue. Other studies have attempted to measure response rates to unexpected perturbation and thereby examine unconscious mechanisms of functional stability and have shown that significant differences exist between subjects with LBP.
and healthy controls. Exploring the unconscious mechanisms of proprioception and dynamic joint stability may be warranted since injuries often occur when unexpected loads are imposed upon the human spine and this may be an avenue for further research.

5.3. Homogeneity of Subjects

The statistical analysis of the subject characteristics would suggest that the randomization technique employed adequately nullified any potential differences that may have been apparent between the groups tested; however, this cannot be stated with complete confidence. Randomization minimized the differences noted between the groups but does not minimize differences that may be observable within the groups. There may be subgroups of individuals within the groups that may behave differently than the group as a whole resulting in skewed study results. The total number of subjects incorporated in this study is simply not adequate to perform a satisfactory secondary analysis of these potential subgroups; however, this study may be used as pilot data to explore these potential differences in subsequent studies.

Characteristics identified during a comprehensive subjective and physical examination may be able to identify subjects who may benefit more from certain interventions. The theoretical basis for classification systems and clinical prediction rules involves the concept that individuals with LBP are a heterogeneous group and that subcategories exist that may alter response to treatment. Heterogeneity may play a significant role in the inability of clinicians to identify a best practice for the treatment of LBP. Since the results of this study do not appear to suggest that spinal manipulation will improve proprioception in all subjects who have CLBP and are currently pain-free, it may be possible to identify subgroups of subjects who will benefit from spinal manipulation.
5.4. Study Limitations

5.4.1. Instrumentation

The methodology employed in this study was novel compared with other research studies and involved a very large apparatus intimately attached to the subject which may have provided enough external stimuli to alter the subject’s natural internal proprioception through cutaneous mechanoreceptor stimulation. This particular issue has been well documented within the literature.\(^{293,294}\) Despite the knowledge of this methodological limitation, the Biodex Systems 3 was employed for the ease and speed of use. Previous literature on the neurophysiological effects of spinal manipulation has reported the transient nature of the changes examined making a rapid method of assessment imperative to examine these transient effects.\(^{295-297}\) The trade-off of additional external stimuli for speed of use was determined to be acceptable under the clinical circumstances. This limitation would not impact FR since cutaneous input would not necessarily alter the subjects’ perception of voluntary force but could impact JPS, TTDPM, and DM. Development of a different type of attachment system from dynamometer to the trunk may eliminate or at least diminish the external cutaneous input thereby, improving the validity of data collection for DM and TTDPM. Ideally, the attachment would make contact with the majority of the trunk and would employ pneumatic pressure\(^{217}\) or gel to keep contact pressure very similar over the entire system. The caveat of such an attachment may be that large areas of contact in general may enhance proprioception consistent with the use of bracing\(^{136}\) Other instrumentation described in the literature,\(^{128,138}\) may employ a superior attachment system however the plane of motion tested was not sagittal.
An additional limitation of using the apparatus chosen involves the lack of ability to completely eliminate vestibular input to the sensorimotor system. Because the entire trunk moved with the fulcrum about the coronal axis at approximately L4-L5, movement of the head was not eliminated allowing the subjects to use head position to assist in repositioning of the trunk in JPS and with the detection of trunk movement during TTDPM. The use of a blindfold to eliminate vision partially attenuates this ability by eliminating the sensorimotor system’s ability to triangulate input with the use of vision but cannot completely eliminate the effects of the additional vestibular input. This limitation could have been minimized by using an electromagnetic goniometer and having the subject move the lumbar spine in the sagittal plane while maintaining a more constant head position\textsuperscript{119, 298} but the apparatus required for such a methodology would require an extensive calibration time that may have nullified the transient neurophysiological effects previously mentioned. Using a pelvic tilt method for trunk reposition sense would minimize the forward-backward head movement but would allow an up and down movement of the head which also could be used to enhance perception of trunk position. In addition, the use of a pelvic tilt with the electromagnetic systems brings proprioception of the hip more prominently because the subject would have to alter sagittal plane hip angle during the pelvic tilt in order to maintain steady state weight bearing on the supportive surface. Since the acuity of proprioception of the hip has not been reported extensively in the literature, the impact of allowing the hip to contribute to spinal proprioception cannot be estimated. Any attempt to minimize additional proprioceptive input from either above or below the lumbar spine will result in additional input from the opposite (distant) segment; attempting to completely isolate the lumbar spine in proprioceptive measures is not functionally possible with current methodology.
5.4.2. Subjects Strategy during Testing

Subjects within the study may use differing strategies to attempt to reposition the trunk with greater accuracy. The strategy employed may account for significant variability in results for proprioceptive tests. Subjects could intentionally enhance gamma bias through a deliberate and significant co-contraction of the local stabilization muscles of the trunk in an effort to protect the trunk from irritation during testing. Using a co-contraction during task performance results in increased EMG variability but reduced movement variability. This phenomenon may not be as readily employed by subjects with no history of back pain since they are not attempting to protect themselves from pain but any individual may employ this strategy in an attempt to increase accuracy.

It was noted during testing that some subjects demonstrated inconsistent performance that would not likely represent normal variability. This unfortunately is the by-product of performing a high number of repetitions to enhance statistical power in testing. Subjects may have become bored or distracted during testing, it is also possible that they were inconsistent in their attempts to enhance gamma bias through co-contractions causing outlier trials during testing sessions. It was also apparent that subjects demonstrated different strategies for JPS that could easily affect the results. Some subjects would perform a generally quick repositioning and would depress the marker switch while still moving into sagittal plane flexion while others would stop near their final target and would wiggle back and forth to fine tune their position prior to depressing the marker switch. Since the exact strategy to be performed was not outlined during pre-test instructions, the dramatic variations in strategy were tolerated.
5.4.3. Sham Procedure may have Therapeutic Benefit

A potential problem with this study might lie in the interpretation of the sham procedure. In an attempt to reduce bias, subjects were told two differing treatment procedures were being evaluated. In order for this strategy to have scientific merit, it is necessary for the sham procedure to appear to have clinical importance. When hands are placed on a subject to give the illusion of therapeutic effect, it becomes possible that a therapeutic benefit may result. In a small number of cases, an audible pop was discerned during the sham procedure but the audible was not necessarily in the lumbar spine. While there is little evidence suggesting that the audible pop has therapeutic benefit from a pain and disability perspective, its effect on neuromuscular control has not been evaluated in the literature. There is the possibility that the audible pop is acting as a confounding variable in this study however the number of subjects demonstrating this phenomenon was not sufficient to allow an analysis of the results of its effect.

5.5. Future Research

It is apparent that learning may have played a role in forming the results observed in this study. Future studies designed specifically to test the relative contribution of learning vs. the intervention in determining the variation observed in conscious proprioception are needed to ascertain the true effect of spinal manipulation. This study could involve training sessions prior to testing to stabilize performance before using an intervention. If learning is a viable treatment option as existing literature suggests. The training program may nullify the deficit that manipulation seeks to resolve. It may be necessary to use both interventions, repetition vs manipulation in a pragmatically controlled clinical trial to further explore this relationship.
Future studies are warranted exploring other methodology for assessment of trunk proprioception and the effects of spinal manipulation on them. These other methodologies may be employed to determine if these results can be replicated using other equipment more readily found in clinical environments. This is an important factor clinically since specialized neuromuscular assessment tools are not found in the average clinic yet the majority of patients treated are not found in large research centers. Clinical practicality must be a consideration in research.

It is evident that the effects of manipulation on proprioception may last longer than 10 minutes secondary to the results demonstrated in this study but one week residual effects are less certain. Studies are needed to better control variables that may contribute to 1-week residual effects. Studies using larger subject groups will also assist by allowing statistical analyses that require larger sample sizes to provide adequate power when controlling for confounding variables.

Further studies examining the effects of other treatment modalities on clinically measurable proprioception are also warranted to determine their neurophysiological effects in the treatment of LBP. Many methods of treating LBP have shown variable levels of treatment success. It is not currently known which treatments are most effective or even if best practice can be ascertained. A first step in determining best practice with regards to effect on proprioception is to show which treatments have a clinically measurable impact on proprioception. There is an added benefit in studying different treatment strategies. Since studies have suggested that subgroups of patients may respond clinically to different treatment strategies, none of these classification systems or clinical prediction rules has explored the effect of the proposed treatments on neuromuscular control of the trunk. Since neuromuscular control may be the
subsystem responsible for high recurrence rates, taking this variable into consideration is warranted.

Once a greater expanse of literature is available regarding the treatment effects of various modalities on trunk proprioception, comparison studies can be initiated to determine which treatments or combinations of treatments may be best for the resolution of LBP and possibly reduce the currently high recidivism rates demonstrated in the chronic state of LBP.

5.6. Conclusions

Spinal manipulation had a statistically significant effect on TTDPM in Manip 1st group subjects only. Spinal manipulation had a statistically significant one week residual effect on JPS. Spinal manipulation did not have a statistically significant effect on either DM or FR. There was also a tendency to maintain the improvement in proprioception one week following the intervention but this tendency was only significant for JPS. The results of this study may be explained at least partly by learning rather than by the treatment effect alone. Strong conclusions cannot be drawn from these data presented in this study but further investigation is warranted to better determine the effects of manipulation on conscious proprioception of the trunk in subjects presenting with CLBP.
SUBJECTS NEEDED FOR A LOW BACK PAIN RESEARCH STUDY

What: Study measuring the effects of spinal manipulation on back position sense and balance in people with chronic low back pain

Where: Neuromuscular Research Laboratory
University of Pittsburgh
3200 South Water Street

Who: Individuals with low back pain that has either lasted continuously for at least three months or has had recurrent back pain for longer than one year.

Ages between 18-65 years old

If interested, please contact:

Ken Learman, MEd, PT
Kel15@pitt.edu
(412) 432-3800
Appendix B: ODI

This Questionnaire is designed to enable us to understand how much your Low Back Pain had affected your ability to manage your everyday activities. Please answer each section by marking in each section the ONE BOX that most applies to you. We realize that you may feel that more than one statement may relate to you, but PLEASE ONLY MARK THE ONE BOX WHICH MOST CLOSELY DESCRIBES YOUR PROBLEM.

Section 1 - Pain Intensity
- I can tolerate the pain I have without having to use pain medication
- The pain is bad, but I can manage without having to take pain medication
- Pain medication provides me with complete relief from pain
- Pain medication provides me with moderate relief from pain
- Pain medication provides me with little relief from pain
- Pain medication has no effect on my pain

Section 2 - Personal Care (washing, dressing etc.)
- I can take care of myself normally without causing increased pain
- I can take care of myself normally, but it increases my pain
- It is painful to take care of myself, and I am slow and careful
- I need help, but I am able to manage most of my personal care
- I need help every day in most aspects of my care
- I do not get dressed, I wash with difficulty, and I stay in bed

Section 3 – Lifting
- I can lift heavy weights without increased pain
- I can lift heavy weights, but it causes increased pain
- Pain prevents me from lifting heavy weights off the floor, but I can manage if the weights are conveniently positioned (e.g., on a table)
- Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned
- I can lift only very light weights
- I cannot lift or carry anything at all

Section 4 – Walking
- Pain does not prevent me from walking any distance
- Pain prevents me from walking more than 1 mile (1 mile = 1.6 km)
- Pain prevents me from walking more than 1/2 mile
- Pain prevents me from walking more than 1/4 mile
- I can walk only with crutches or a cane
- I am in bed most of the time and have to crawl to the toilet

Section 5 – Sitting
- I can sit in any chair as long as I like
- I can only sit in my favorite chair as long as I like
- Pain prevents me from sitting for more than 1 hour
- Pain prevents me from sitting for more than 1/2 hour
- Pain prevents me from sitting for more than 10 minutes
- Pain prevents me from sitting at all

Section 6 – Standing
- I can stand as long as I want without increased pain
- I can stand as long as I want, but it increases my pain
- Pain prevents me from standing for more than 1 hour
- Pain prevents me from standing for more than 1/2 hour
- Pain prevents me from standing for more than 10 minutes
- Pain prevents me from standing at all

Section 7 – Sleeping
- Pain does not prevent me from sleeping well
- I can sleep well only by using pain medication
- Even when I take medication, I sleep less than 6 hours
- Even when I take medication, I sleep less than 4 hours
- Even when I take medication, I sleep less than 2 hours
- Pain prevents me from sleeping at all

Section 8 – Social Life
- My social life is normal and does not increase my pain
- My social life is normal, but it increases my level of pain
- Pain prevents me from participating in more energetic activities (e.g., sports, dancing)
- Pain prevents me from going out very often
- Pain has restricted my social life to my home
- I have hardly any social life because of my pain

Section 9 – Traveling
- I can travel anywhere without increased pain
- I can travel anywhere, but it increases my pain
- My pain restricts my travel over 2 hours
- My pain restricts my travel over 1 hour
- My pain restricts my travel to short necessary journeys under 1/2 hour
- My pain prevents all travel except for visits to the physician / therapist or hospital

Section 10 – Employment/Homemaking
- My normal homemaking/job activities do not cause pain
- My normal homemaking/job activities increase my pain, but I can still perform all that is required of me
- I can perform most of my homemaking/job duties, but pain prevents me from performing more physically stressful activities (e.g., lifting, vacuuming)
- Pain prevents me from doing anything but light duties
- Pain prevents me from doing even light duties
- Pain prevents me from performing any job or homemaking chores
Appendix C: NRS

This test would be performed in a verbal manner but the following figure is a visual representation.

Please use the scale below to rate your average pain during the past 24 hours and your pain currently.

**Current Pain**

0 = NO PAIN  10 = EXTREMELY INTENSE

0 1 2 3 4 5 6 7 8 9 10

**Average Pain Last 24 Hours**

0 = NO PAIN  10 = EXTREMELY INTENSE

0 1 2 3 4 5 6 7 8 9 10
Appendix D: VAS - 24

The VAS-24 is similar but is a 10 cm straight line with end lines and the subject is asked to place an X on the line between the left hash “no pain” and the right hash “worst pain you could imagine” and the reference is 24 hour average pain level.

No pain | most severe pain
---|---
Appendix E: FABQ

Name:______________________________   Date:_____________

Here are some of the things other patients have told us about their pain. For each statement, please mark the number from 0-6 to indicate how much physical activity such as bending, lifting, walking, or driving affect or would affect your back pain. Please circle the number that is associated with your pain.

<table>
<thead>
<tr>
<th>Statement</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) My pain was caused by physical activity</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>2) Physical activity makes my pain worse</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>3) Physical activity might harm my back</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>4) I should not do physical activities which (might) make my pain worse</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>5) I cannot do physical activities which (might) make my pain worse</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>
The following statements are about how your normal work affects or would affect your back.

<table>
<thead>
<tr>
<th>Statement</th>
<th>Completely</th>
<th>Unsure</th>
<th>Completely</th>
</tr>
</thead>
<tbody>
<tr>
<td>6) My pain was caused by my work or by an accident at work</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7) My work aggravated my pain</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8) I have a claim for compensation for my pain</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9) My work is too heavy for me</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10) My work makes or would make my pain worse</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11) My work might harm my back</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12) I should not do my regular work with my present pain</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13) I cannot do my normal work with my present pain</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14) I cannot do my normal work until my pain is treated</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15) I do not think that I will be back to my normal work within 3 months</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16) I do not think that I will ever be able to go back to work</td>
<td>0 1 2 3 4 5 6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix F: Physical Examination

Subject ID Number: ______________

Description of symptoms: ________________________________

Location of symptoms: ________________________________

Duration of symptoms: ________________________________

Number of episodes total: ___________ Past year: ______________

Associated symptoms: ________________________________

Aggravating factors: ________________________________

Relieving factors: ________________________________

Prior Treatment: __________________ Effects: __________________

Medication use: __________________ FABQ score: __________________

Past Medical History: ________________________________

____________________________________________________________________

Posture: _____________________________________________

____________________________________________________________________

<table>
<thead>
<tr>
<th>Trunk</th>
<th>ROM</th>
<th>Strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Side Bending</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right Side Bending</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Rotation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right Rotation</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Repeated Movements: ________________________________

<table>
<thead>
<tr>
<th>Hip</th>
<th>ROM</th>
<th>ROM</th>
<th>Strength</th>
<th>Strength</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left</td>
<td>Right</td>
<td>Left</td>
<td>Right</td>
</tr>
<tr>
<td>Flexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extension</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abduction</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Special Tests:

#### Instability testing:
- **Central PA glides:**
- **Unilateral PA glides:**

Prone Segmental Instability Test: ________________

#### Neuro Testing:
- **SLR:** ________________  **Slump Test:** ________________
- **Light Touch:** ________________

#### SIJ Testing: (to be clinically relevant, 3 of 5 must be +)
- **Compression:** ________________  **Distraction:** ________________
- **Sacral Thrust:** ________________  **Thigh Thrust:** ________________
- **Gaenslen’s Test:** ________________

#### Other physical tests as appropriate:
Appendix G: ACTIVITY LOG

Subject: ___________________________   Date: __________________

Please answer the following questions as they relate to the time period from the last testing session to today.

1) Have you experienced any variation in your normal pain or symptom levels?
   Comments: _______________________________________________________

2) Have you changed your use of medication for your low back problem since the last testing session?
   Comments: _______________________________________________________

3) Have you changed your work habits since the last testing session?
   Comments: _______________________________________________________

4) Have you changed your workouts or leisure activities since the last testing session?
   Comments: _______________________________________________________

5) Have you done anything out of the ordinary, like drive a long trip or do some unusual work, since your last testing session?
   Comments: _______________________________________________________

6) Has anything changed significantly since your last testing session that you think I should know about?
   Comments: _______________________________________________________
BIBLIOGRAPHY


111. Keller A, Brox JI, Gunderson R, Holm I, Friis A, Reikeras O. Trunk muscle strength, cross-sectional area, and density inpatients with chronic low back pain randomized to lumbar fusion or cognitive intervention and exercises. Spine. 2004;29:3-8


138. Taimela S, Kankaanpää M, Luoto S. The effect of lumbar fatigue on the ability to sense a change in lumbar position: A controlled study. Spine. 1999;24:1322-


142. Goodwin GM, McCloskey DI, Matthews PBC. The contribution of muscle afferents to kinesthesia shown by vibration induced illusions of movement and by the effects of paralysing joint afferents. Brain. 1972;95:705-748


145. Childs JD, Fritz JM, Flynn TW, Irrgang JJ, Johnson KK, Majkowski GR, Delitto A. A clinical prediction rule to identify patients with low back pain most likely to benefit from spinal manipulation: A validation study. Ann Intern Med. 2004;141:920-928


185. Dontigny RL. Pelvic dynamics & the SP3 subluxation of the sacroiliac joint. *AAOMPT 9th Annual Conference*. 2003


Manniche C, Jordan A. Re: We have misunderstood the purpose of a Cochrane Database. *Spine*. 2001;26:994


Frost H, Lamb SE, Moffett JA, Klaber, Moser JS, Fairbanks JCT. Randomised controlled trial for evaluation of fitness programme for patients with chronic low back pain. 1995


258. Childs JD. Validation of a clinical prediction rule to identify patients likely to benefit from spinal manipulation: A randomized clinical trial. *School of Health and Rehabilitation Science.* 2003:246


   Brain Res. 1982;244:186-189


   2007.

287. Descarreaux M, Blouin JS, Teasdale N. Force production parameters in patients with low 
   back pain and healthy control study participants. Spine. 2004;29:311-317

288. Descarreaux M, Blouin JS, Teasdale N. Repositioning accuracy and movement 
   parameters in low back pain subjects and healthy control subjects. Eur Spine J. 
   2005;14:185-191

   2005;437:412-416

   Neuron. 2006;52:1085-1096

   trunk and bilateral knees in young subjects with lumbar disc herniation. Spine. 
   2005;30:E528-E533

292. Radebold A, Cholewicki J, Panjabi MM, Patel TC. Muscle response pattern to sudden 
   trunk loading in healthy individuals and in patients with chronic low back pain. Spine. 
   2000;25:947-954

293. Barrett DS, Cobb AG, Bentley G. Joint proprioception in normal, osteoarthritic and 

   1998;23:590-597

295. Terrett AC, Vernon H. Manipulation and pain tolerance. A controlled study of the effect 
   of spinal manipulation on paraspinal cutaneous pain tolerance levels. Am J Phys Med. 
   1984;63:217-225

296. Vernon HT, Aker P, Burns S, Viljakaanen S, Short L. Pressure pain threshold evaluation 
   J Manipulative Physiol Ther. 1990;13:13-16

