Validation of Clinical Tests Used to Identify Patients Who Would Benefit From Trunk Stabilization Exercises: Preliminary Steps to Refine Test Interpretation and Improve Intervention Prescription

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Dedication

This work is dedicated to the loving memory of my uncle, Lee Ki Sung. All the radios you encouraged me to take apart to learn how they work…never granted me any of your engineering and mathematical skills.

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Abstract

Validation of Clinical Tests Used to Identify Patients Who Would Benefit From Trunk Stabilization Exercises: Preliminary Steps to Refine Test Interpretation and Improve Intervention Prescription

Won Sung

Low back pain (LBP) presents a challenge in rehabilitation due to its heterogeneous presentation across patients. However, trunk stabilization exercises have been identified to be successful in patients that meet specific clinical prediction rules. Identifying mechanisms that underlie the tests used in the clinical prediction rules may aid in better understanding impairments in these patients. This may aid in refining intervention selection and prescription. The purpose of this dissertation was to identify mechanisms underlying clinical tests that are used to predict a patient's success with trunk stabilization exercises: aberrant movements observed during forward bending and the prone instability test. The aims were to: 1) characterize lumbar extensor muscle neuromuscular control during active forward bending and the prone instability test (PIT); 2) validate clinical assumptions of the role that impaired lumbar multifidus muscle activity has in aberrant movements patterns during a forward bend task and a positive prone instability test.

Aim 1 results revealed that all trunk extensors are activated to a greater extent in those with aberrant forward bending. However, the lumbar multifidus provided the greatest contribution. In the prone instability test, muscle activity during the leg raising portion of the test resulted in a significant increase in spinal stiffness and reduction in pain. However, participants with LBP had greater reliance on fewer muscle synergies that involved dominance of extrinsic muscles compared to participants without LBP.

Aim 2 results revealed that a positive prone instability test with pain reduction and spinal stiffness increase could be yielded in participants with LBP through electrical stimulation of the lumbar multifidus. However, electrical stimulation driven fatigue to the muscle was not able to produce aberrant movement in individuals without LBP.

Adaptations in neuromuscular control during forward bending and the prone instability test in individuals with LBP suggest that exercises that include movement control and coordination may be necessary within the intervention.

Chapter 1: Proposal

1.1 Abstract

Background: Clinical tests that can predict successful outcomes with trunk stabilization exercises have been identified. However, there are still approximately 30% of patients who meet these criteria and still fail with these exercises. Understanding the mechanisms behind these criteria that allow prediction for success may give better insight to the pathologic process. This information would be helpful in improving focus of the intervention and improving efficacy for treatment.

Purpose: The purposes of this study are to 1) characterize (describe and quantify) lumbar extensor muscle neuromuscular control during forward bending and the prone instability test, and 2) validate clinical assumptions of the role that lumbar multifidus has in aberrant movement patterns during a forward bend task and a positive prone instability test.

Methods: In the first aim, EMG data captured during the forward bend and the prone instability test will be used to describe and quantify the contribution of lumbar extensors during these tasks. For aim 2, neuromuscular electrical stimulation will be utilized to selectively fatigue the lumbar multifidus to determine if inhibiting its contribution during forward bend produces aberrant movements. Electrical stimulation will also be used to isolate activation of the lumbar multifidus to determine if this muscle group can produce a positive prone instability test in healthy controls versus patients with low back pain.

Data Analysis: For aim 1, cross correlation coeffecients and time lags of EMG signals will be used to determine muscle patterns during the forward bend. EMG amplitudes will also be used to assess motor unit synchronization during the forward bend. Repeated

measures ANOVA will be used to analyze spinal stiffness changes and muscle activation during the prone instability test. For aim 2, repeated measures ANOVA will be used to compare kinematic variables pre and post NMES to determine if fatigue is able to reproduce aberrant movements. Mixed measures ANOVA will also be used to analyze spinal stiffness changes of the spine with NMES and compare those changes between healthy controls and patients with low back pain.

Significance: The study's significance lies in the study of underlying mechanisms behind predictive tests to determine if there are potential differences between responders and non-responders to trunk stabilization exercises. The information gained from this study can potentially enhance intervention selection and prescription for patients as well as decreasing the percentage of non-responders.

Innovation: The study's innovations lie in being one of the first to address the mechanisms behind the predictive tests to determine success with trunk stabilization exercises. It is also unique in its aim to utilize electrical stimulation to selectively recruit and impair a muscle group that is difficult to test in isolation for the purpose of validating the tests' mechanisms.

1.2 Specific Aims

Low back pain (LBP) results in socioeconomic costs over $100 billion per year[1,2], is responsible for 26% of disability claims in the US[3], and with a 162% increase in prevalence[4], costs and disability is likely to continue to increase. Disability from LBP stems from a cascade of pain that start with recurring episodes, progressing to chronic LBP.[5,6] This recurrence of pain is theorized to be a result of progressively increasing reduction in spinal structural integrity causing faulty sensory information to the neuromuscular system, which eventually leads to muscle inhibition and further spinal damage[7]. However, rehabilitation utilizing trunk stabilization exercises (TSE) to improve the spine's ability to attenuate harmful stress can potentially decrease the recurrence of LBP. Characteristics identifying individuals with LBP who would benefit from a TSE rehabilitation approach is currently driven by a clinical prediction rule (TSECPR) that attempts to optimize patient outcomes.

Initially, 4 patient characteristics were identified under the TSECPR, with the presence of 3 or more of these characteristics increasing post examination likelihood for treatment success to 67%.[8] A recent validation study of the TSECPR has found that presence of 2 of the 4 aforementioned exam findings: aberrant movement during forward bend and a positive prone instability test (PIT), improve diagnostic accuracy of the TSECPR[9]. However, even with this modified TSECPR, 28% of patients who met the criteria did not respond to treatment. Therefore, there is roughly a 30% chance of non-response, either in individual's post-exam probability or group probability for successful outcomes. Panjabi's theory suggesting symptom recurrence (2006) progresses to chronic pain centers on reduced ability to improve spinal stability through regulation and output

of muscular forces and movement control. Examination for aberrant movement patterns and the PIT are believed to be appropriate tools to assess movement control and muscle function as well as guide intervention selection. However, very little to date is known about the neuromuscular mechanisms responsible for the presence of aberrant movement or an individual's PIT results. Understanding the mechanisms or impairments underlying these tests would add to their diagnostic validity, yielding more accurate interpretation of clinical findings, and potentially lead to identifying improved interventions for those who have failed with TSE in the past.

Kinematic assessment and quantification of aberrant movement during forward bend has been studied, with instability catch/judder (JUD) and altered lumbopelvic rhythm (aLPR) being associated with patients who have a history of or current episode of LBP compared to those with no LBP.[10] In forward bend, the paraspinal extensor muscles perform an eccentric contraction to control the trunk during the forward bend phase, and then perform a concentric contraction during the return to the upright phase with assistance from the gluteus maximus and hamstrings.[11-14] It is theorized that aberrant and/or unsmooth motion is representative of the neuromuscular system's unsuccessful attempt to stabilize a joint.[15] Therefore, aberrant movements during the forward bend suggest dysfunction of the lumbar extensor muscles resulting in inadequate control and protection of the joint during movement in patients with LBP. However, motor control and muscle activity associated with clinically observed aberrant trunk motion during forward bending has not been studied.

A positive PIT is defined as a reduction in LBP during an examiner-applied posterior to anterior force to the lumbar spine during prone bilateral hip extension. The

clinical interpretation of a positive test is that the lumbar extensor muscles, particularly the lumbar mutifidus (LM) group based on its anatomical structure, adequately contract and stiffen the lumbar spine thereby reducing the subject's LBP during the examiner-applied force. A negative test, where pain is not altered by active hip extension, is implied as inadequate muscle activity to stabilize the spine against the examiner's application of an external load. However, these clinical assumptions have not been validated. It is not known if stiffening of the spine occurs during this test nor what muscles are active to drive this potential stiffening. Therefore, observations of aberrant motion during forward bending and PIT assessment outcomes may be signs of lumbar extensor muscle dysfunction. However, this has not been systematically evaluated or validated. The ability to identify impaired performance of the lumbar extensor muscles, specifically the lumbar multifidus (LM) is important. A greater number of prognostic patient characteristics in the TSECPR have been associated with inactivity of the LM[16]. However, no direct link has yet to be made with LM activity and an individual's result with PIT, nor the presence of aberrant movement. The work of Hebert, et al. (2010) suggests that LM function plays a key role in smoothly controlling forward bending and in stiffening the spine, and perhaps adequate LM activation is the key to successful outcomes following TSE. Those patients who demonstrate likelihood for success with TSE, but who fail to respond to the intervention may have a deficiency in activation and/or significant morphologic changes (cross sectional area, muscle to intramuscular fat ratio) of the LM that requires additional intervention. However, this is not yet substantiated in the current literature.

The *long-term goal* of this research is to identify primary mechanisms associated with impaired neuromuscular control in LBP patients in order to improve diagnostic criteria and intervention efficacy. The aims of this proposal contribute to the long-term goal by characterizing muscle activation patterns during two important clinical tests that predict success with TSE beginning the process of validating clinical assumptions. In addition, this investigation will determine the contribution of LM to the finding during these tests by assessing the effect of 1) enhanced activity of the LM on clinical interpretation of the PIT, and 2) fatigue or diminished LM activation on movement control during active forward bending. Since interventions are guided by these tests, improved understanding of the underlying neuromuscular mechanisms associated with results of these clinical tests will improve diagnostic accuracy, and elucidate impairments associated with responders and non-responders to TSE. Ultimately this work will improve the delivery of care for patients who previously may not have had successful outcome with rehabilitation.

Aim 1: Characterize lumbar extensor muscle neuromuscular control strategies through trunk muscle activation patterns during active forward bending and the prone instability test.

1a. Describe and quantify trunk muscle activity during a forward bend task.

Hypothesis: Patients with low back pain will have higher LM and lumbar erector spinae EMG amplitudes compared to healthy control subjects.

Expected Outcome: *Identification of typical and aberrant trunk muscle activation patterns* in individuals classified with normal and aberrant (JUD, aLPR) forward bend movement patterns.

1b. Describe and quantify trunk and hip muscle activity as well as changes in spine stiffness during the PIT test.

Hypothesis: Lumbar spine stiffness increases during the PIT and is associated with muscle activity of the lumbar extensors.

Expected outcome: Determine if spinal stiffness changes *do* occur during the PIT. Identify the role of the lumbar extensor muscles, particularly the lumbar multifidus, during the test. Determine if there is an association between spinal stiffness changes and muscle activation patterns during the PIT.

Aim 2: Validate clinical assumptions of the role that lumbar multifidus muscle activity has in aberrant movements patterns during a forward bend task and a positive prone instability test.

2a. *Characterize the effects of isolated* lumbar multifidus *muscle fatigue,* achieved by a neuromuscular electrical stimulation (NMES) fatigue protocol, on movement quality during a forward bend task in healthy controls.

Hypothesis: Subjects with a typical forward bend movement pattern will demonstrate aberrant movement pattern following fatigue of the lumbar multifidi muscles.

Expected outcomes: Production of aberrant movement through attenuation of LM activity via fatigue will support that theory that impaired activation of the LM impacts movement quality during the forward bend test.

2b. Determine if *electrically induced LM contraction using NMES* can yield:

i. Increased stiffness of the lumbar spine during examiner applied posterior to anterior force on the lumbar spinous process.

ii. Positive prone instability test result.

Working Hypothesis: Stiffening of the spine is responsible for the pain reduction during the PIT and this is primarily achieved by strong activation of the LM muscle group.

Expected Outcomes: Identification of the role that the LM plays in the PIT.

Aim 1 will allow for improved understanding of muscle activation patterns during clinical tests that are used to prescribe TSE. Aim 2 provides an approach to investigate the role of the LM during a forward bend task by selectively fatiguing the muscle with NMES. As LM has been suggested to be a key muscle in successful outcomes with TSE [16], enhanced understanding of this muscle's role in clinical tests that can predict both success and failure of intervention in patients is crucial. The results of this work will validate underlying mechanisms associated with the forward bending and prone instability tests, and improve a clinician's ability to interpret test findings with respect to intervention selection.

1.3 Significance

During 2002, over 250,000 people experienced low back pain in the United States (LBP) with medical costs and lost wages exceeding $100 billion.[1,2] There had been an estimated 162% increase in the prevalence of LBP and by 2005, overall yearly medical costs for patients with LBP were near double that of patients without LBP.[4] Up to 26% of disability claims in the US are attributed to LBP.[3] These statistics may be attributable, in

part, to the recurrent nature of LBP which has been reported to range from 47-84 % [17] in a 1 year period. Musculoskeletal changes effecting spinal function may play a role in both the recurrence [18-20] and chronicity of LBP [21], with many of these structural changes being associated with the lumbar multifidus (LM). Beside structural changes, reduced muscle activity of the LM has been associated with tests used to identify patients who would benefit from trunk stabilization exercises (TSE).[16] Improvements in muscle structure have been reported following implementation of a TSE that focuses on isolated activity of the LM[22,23] while general back strengthening exercises have revealed no improvements in LM activation.[12] This suggests that certain types of exercise may have a positive effect on the structures of the spine that play a role in recurrence and chronicity, making the role of rehabilitation exercises an important aspect of reducing risk for recurrence and chronicity of LBP.

Trunk stabilization exercises focusing on the coordination, endurance, and strength of the trunk are one of 3 rehabilitation interventions subgroups with strong evidence of efficacy.[24] Intervention provision by subgrouping into a treatment categories based on patient characteristics and examination findings have been shown to lead to better outcomes[25] and are more cost effective. [26] Prognostically, the TSECPR group, has a positive likelihood ratio (+LR) of 4.0 and post-test chance for success of 67% if 3/4 characteristics are met.[8] The subgroup identified for manipulation has a much higher likelihood for success (+LR=13.2 if 4/5 characteristics present) and a 92% post-test chance for success.[27] The lower percentage of post treatment success in the TSE group may be tied to impaired LM function. In patients who do not respond to TSE and do not fit other treatment subgroups, failure to improve LM activity or function may be the

driving factor. Impairments of this muscle happen quickly after the initial onset of LBP [28], and it's recovery relies on specific localized intervention versus generalized exercise. [23,29] If these impairments are not addressed, patients will present with alterations in muscle physiology and deficits in muscle function after cessation of pain , as this muscle group does not recover spontaneously.[19] The ability to properly rehabilitate this muscle may be the true predictor for recovery in these patients. However, the role that the LM plays in development of LBP in these patients, and it's involvement with a positive or negative response to TSE is unknown.

Based on a 1 year prevalence estimate of LBP (36%), 112 million Americans may develop low back pain that limits activity.[30] Patients that could potentially be categorized as responders to TSE could range from 12%-38% of this number.[9,31] Taking a conservative estimate of 25% of these patients (midpoint of the above range), yields over 50 million patients identified to benefit from TSE in the US alone. A 30% non-response rate yields, almost 17 million patients that could meet criteria for TSE, but do not fully benefit from it. Based on the potentially large number of patients with non-response to TSE, improving the percentage of responders to TSE could have a significant impact on the cases of recurrence and chronicity of LBP.

Two of the 4 tests used to categorize patients into the TSE group have been identified to be better at predicting patients who would respond to TSE: prone instability test (PIT) and the presence of aberrant movements during forward bend. [9] Patients with these 2 clinical characteristics have been shown to have disruption of movement during the midrange forward bending, typically identified as the neutral zone, where muscle activity should be the dominant stabilizer.[32] The LM has been identified to provide up to

2/3 of the stabilizing force in the spine[33,34] and these findings may suggest a reduction in LM activity during movement. Decreased LM activity has been shown to be an important prognostic factor for patients who benefit from TSE[16], and the presence of aberrant movement increases likelihood for success outcomes twofold. This suggests that, during forward bend, observation of aberrant movements may be signs of LM impairment, with presence of the sign suggesting the need to improve muscle function (either activation and/or strength).

However, there is a different scenario with the PIT. The underlying assumption of the PIT is that the reduction of examiner induced pain to the spine that occurs during leg elevation is a result of increased trunk extensor and hip muscle activity resulting in increased spinal stiffness.[35] A positive finding on the PIT results in a twofold increase of success with stabilization exercises. Muscle activity of the trunk's anterior wall during abdominal hallowing and bracing have demonstrated increase in spinal stiffness in healthy subjects.[36] Similar mechanisms may exist in the posterior wall. While the physiologic mechanism of this test has not been established, exercises involving unilateral prone hip extension have demonstrated that the LM, gluteus maximus, and erector spinae are the predominant muscles active during similar tasks.[37,38]

While LM activity attenuation has been associated with prognostic factors for success with TSE[16], in certain individuals, greater or more significant impairment in LM muscle activity may actually afford insufficient stabilizing ability and thus not change pain presentation during leg elevation. A negative PIT increases likelihood of failure 6 fold. In patients with a positive PIT, while activity of LM may be decreased during function, they may still possess some ability to increase recruitment of that muscle during

active hip extension, suggesting the muscle group has the ability to activate; there is some reserve muscle capacity that can be augmented with rehabilitation Patients with a negative PIT may not have this reserve capacity, and general stabilization exercises may not be adequate for successful reduction in pain and improvement in function. This subgroup of patients potentially requires more specific interventions targeted at improving LM muscle function.

Borrowing from knee rehabilitation literature, the inability to activate muscle to some requisite amount has prevented patients from achieving muscle hypertrophy[39,40], and this may be similar in this subgroup of LBP patients. The ability to identify if similar mechanisms are present in this subgroup would not only aide recovery of those patient who may fail with this intervention, but also improve the efficacy of treatment for patients who would typically be identified as having successful outcomes with this intervention. In order to do so, we must have an understanding the neuromuscular control and coordination is present in a healthy sample devoid of symptoms. This allows comparison of movement patterns of those who have pain due to altered or impaired movement coordination to provide better informed therapeutic prescription.

There may be types of characteristics noted in aberrancies, or characteristics in muscle activity, response associated with non-responders. But before we can interpret these potential differences, we need to validate the mechanism underlying the specific tests results. Validating clinical assumptions of diagnostic test may be the first step in improving the diagnostic properties or predictive validity of clinical prediction rules associated with responders to trunk stabilization exercises. Improvement in diagnostic tests may in turn, assist in improving intervention selection and implementation.

1.4 Innovation

To date, no studies have directly investigated the construct validity or neuromuscular mechanisms underlying standing forward bend aberrant movement testing patterns and the PIT. Aim one allows for this by characterizing the muscle activation patterns during these clinical tests. Based on the predictive ability of these tests, [9,41,42] characterization of muscle activity during these tests is important because it leads to prescription of specific exercises focused on improving muscle function. Understanding the muscle activity that occurs during the presence or absence of these specific clinical findings would allow investigators to understand typical versus atypical neuro-motor responses. This information would translate directly to clinical care through improvement of exercise prescription.

Validating clinical assumptions related to LM performance is difficult because of this muscle groups morphology and synergistic activity to other muscle groups in the thoraco-lumbar spine. This makes isolating the LM for study in humans challenging. By utilizing electrical stimulation it may be possible to selectively recruit the LM muscles and test the assumptions of the prone instability test. Through this method, investigators can determine if specific activation of this muscle group leads to stiffening of the spinal column, compare that stiffening to that achieved during a PIT and better understand the role of the LM in symptom reduction during the PIT. By utilizing the same electrical stimulation of the LM to cause local muscle fatigue, investigators can selectively impair the role of the LM in healthy subjects with no identified aberrant motion during forward bend. This would help determine if altered function of the LM muscle group has a significant impact on movement patterns during forward bending. This paradigm would

allow investigators to determine the criterion validity of these tests. Understanding the role of the LM would provide a significant leap forward in interpretation of these tests. Since these tests predict both responders and non-responders to TSE treatment the findings will result in improved outcomes for patients with LBP.

1.5 Background

1.5.1 Rehabilitation of Low Back Pain

Low back pain (LBP) is a major source of activity limitations in the industrialized world with medical costs and lost work day/wage costs ranging from $5 to $100 billion dollars.[2,43,44] Over 250,000 people experienced low back pain in the United States in 2002. [1] With a prevalence of 36% in the population and adjustment to the current US population, that number exceeds 100 million people having experienced low back pain.[30] Of these patients, 33-73% will have at least 1 recurrence within 12 months, while the number of prior episodes of LBP increases the likelihood of future episodes.[17,45] In patients with acute LBP, 10-33% of them will go on to develop chronic pain.[46,47] Based on the incidence and prevalence of low back pain, the number of patients who are at risk of experiencing recurrent LBP with progressing to chronic LBP can come to represent a large number of the population, with profound impacts on societal productivity, health care costs, and quality of living. Improved methods to prevent future episodes and intervene in current episodes would have a great impact but requires identification of risk factors that drive recurrence and chronicity.

Physical factors may not always be considered the primary risk factor for development of chronic LBP. Presence of psychosocial factors, clinically referred to as yellow warning flags, have been thought to be most predictive of chronic LBP. [48,49]

However, there is emerging evidence that the intensity of pain and impairments at symptom's first onset is a significant prognostic factor for chronicity of LBP as well.[50] It has also been identified that injury and pain can *lead* to the depression, catastrophizing thoughts, and fear avoidance in people typically not considered at risk for chronic LBP.[51] Therefore, pre-existing psychosocial risk factors may not be sole contributor to chronicity in some patients. Rather, there may be instances where physical characteristics or conditions drive symptoms to recurrence and eventual chronicity. Addressing patients' experience with pain, impairments, and recurrence as early as possible could reduce this progression into chronicity in certain subgroups of patients. Improving current methods for intervention selection and provision of care has great potential for allowing clinicians to do just that.

The current trend in rehabilitation of patients with LBP is to match interventions by subgrouping patients into treatment groups based on clinical findings.[24] There are 4 treatment groups considered in this treatment based classification system (TBC): Manipulation, specific exercise, traction, and stabilization exercise groups.[52] Three of the 4 groups are focused primarily on reducing acute pain/ symptoms. Manipulation group has excellent odds of reducing acute pain in patients that meet the clinical exam criteria[27] through neural modulation of pain and changes in disc hydration.[53-55] Specific exercise and lumbar traction are aimed at patients with referred leg pain with support for use of centralization or directional preference in reducing symptoms through changes in disc hydration[56], while traction continues to be equivocal in these patients.[24] These groups can typically reduce pain within 3-7 sessions[27,57,58], which may be beneficial for dealing with the risk of chronicity resulting from the experience of pain at initial onset. Based on their

mechanism they may be best utilized in more acute exacerbations of pain, as discal mechanisms are theoretically believed to occur in the earlier stages of LBP.[59,60]

The TSE subgroup can potentially address problems with recurrence and chronicity of LBP through improving motor control and movement coordination impairments (MCI) in patients. Neural and mechanical faults progressing from initial discal involvement leading to increased spinal instability have been theorized to be responsible for certain types of LBP.[61,62] These faults include changes in muscle physiology, reduction of morphologic quality[18,21,63,64] and inhibition of neural control in muscles protecting the spine.[63,65-67] It has been theorized that failure to correct these faults leads to recurrence and chronicity of LBP.[7] Interventions associated with the TSE group have been identified to be beneficial in patients with MCI [24] and based on theorized mechanism associated with LBP and spine mechanics, have the potential to make a great impact on recurrence and chronicity.

1.5.2 Theoretical Basis of Spinal Instability and Trunk Stabilization Exercises

The conceptual framework for TSE is grounded by the theoretical mechanism of spinal degeneration. The spine is an inherently unstable system[68] that requires external support through muscle activity for structural integrity.[59,69] The sequelae of spinal degeneration associated with development of LBP was characterized as a stepwise mechanism beginning with an injury that causes instability of the lumbar spine. This segmental instability is characterized by greater mobility in the segments of the lumbar spine.[59] The stabilization mechanism of the spine has been defined by 3 separate systems that work in unison.[61,62] Panjabi (1992A, 1992b) refers to the presence a *passive subsystem* of osseous and ligamentous structures that give increase integrity to the spine.

Injury to the spine, typically at the intervertebral level have been reported to increase segmental mobility and joint laxity.[70] The system is able to compensate for this insult through increased muscle activity of the *active subsystem*. Increased muscle activity is modulated by neural input from the *neural subsystem* [62] through feedback from the ligaments and muscles.[71] Failure of one or more of these systems is associated with LBP[72] with failure to correct the faults potentially resulting in recurrence and chronicity of LBP [7]. When approaching the problem from this perspective, active or physical rehabilitation is best aligned to address the faults in the muscular and neural subsystems, with a goal of reducing the progression of recurrence to chronicity.

1.5.3 Evolution of Trunk Stabilization Exercises:

1.5.3.1 Moving from theory into practice: Role of abdominal muscle activity in trunk stabilization exercises

Muscles of the trunk that play a role in the stabilization of the spine are an area of interest to rehabilitation sciences, as there is evidence to support their role in spinal stability. Much of this evidence centers on biomechanical concepts of spinal stiffening. In a typical spine the inherent instability is addressed with muscle activity in the beginning of the motion until passive tension across the muscles and ligaments engage contributing to stability in the end range.[62] Muscle co-contractions have demonstrated an ability to stiffen the spine with increasing levels of co-contraction leading to increasing levels of stiffness.[36,73,74] Findings on spinal stiffening with muscle co-contractions have gone on to influence rehabilitation through spinal stabilization exercises to address patients with LBP.

There are many philosophies on spinal stabilization exercises. One approach suggests all muscles around the spine act in unison, contributing to some portion of spinal

stability and should be strengthened as a unit.[75] Other approaches are more selective, based on a theory of local intrinsic spinal muscles (transverse abdominus, internal oblique, and lumbar multifidus) and global extrinsic muscles(quadratus lumborum, erector spinae, latimus dorsi, and gluteal), with local muscles responsible for stabilization of the spine.[76] Based on the latter approach, isolated training of the intrinsic muscles are fostered first, and then progressed to incorporate global muscles, and finally functional tasks.[77] The concept of intrinsic muscle dysfunction came to the forefront of rehabilitation with the findings of delayed onset of the transverse abdominus in patients with chronic low back pain compared to healthy controls during rapid arm and leg movements.[67,78,79] This led the authors to conclude that there were postural control impairments in patients with LBP. Translation of these findings is difficult to in-vivo situations. These studies were performed in static positions, while postural control involves coordination of body orientation in space during movement.[80] The findings do raise questions of the neural subsystem's involvement in LBP, at least in relation to the impairments associated with the transverse abdominus.

Muscle onset timing is based on delays seen with EMGs, with this technology in general, providing some information on the central nervous system's control over a motor unit. Therefore, changes in activation timing or any other EMG characteristic of a muscle may be considered to provide, at least some indirect information on the neural subsystem's control over muscles. Further work in muscle onset delays of the transverse abdominus in patients with chronic LBP would lead to findings or re-organization of motor cortices, suggesting some central nervous system changes are associated with patients who experience LBP [66] There is some evidence that with isolated motor control

training, improvements in onset times occur with lasting effect in patients reporting chronic LBP.[65,81] However, there are methodological issues with these findings: repeatability with these measures suggest the onset time differences do not exceed standard errors of measure, and use of real time ultrasound imaging is not able to detect muscle onset delays[82] raising a question about the clinical relevance of transverse abdominus onset times. Other findings suggest that general exercise and isolated motor control exercises result in similar improvements on muscle onset timing[83] in patients with chronic LBP, suggesting that the impairments may have other causes such as pain inhibition or deconditioning. In a sample of patients with chronic LBP, similar to the patients in these prior studies identifying onset times and cortical changes, improvement in transverse abdominus function was not associated with successful outcomes.[84]

While the evidence surrounding abdominal muscle activation may be equivocal, increase in intra-abdominal pressure through abdominal contractions has also been considered a mechanism of improving spinal stability. Increase in intra-abdominal pressure modeling suggests it aids in decompressing spinal compression[85], with increase in intra-abdominal pressure independent of abdominal contraction demonstrating increase in spinal stiffness.[86] Spine modeling has demonstrated that this increase in intra-abdominal pressure can provide approximately 10% of extensor torque required to unload spinal compression[87], allowing for stabilization of the spine with decreased trunk extensor demand.[88] In conditions where the trunk extensors may not be functioning properly, the abdominal muscles' role in increasing intra-abdominal pressure may add additional stability to the spine. However, selective activation of the transverse abdominus or the oblique abdominals may not provide significant stiffening to the spine

[89] while increases in stiffness from intra-abdominal pressure may include contributions from other muscles of the trunk and other mechanisms such as tension on the thoracolumbar fasciae.[90] This is supported by EMG comparisons during abdominal bracing task and an abdominal hallowing task in healthy control subjects. Abdominal hallowing resulted in a 26.8% increase in spinal stiffness from resting condition, while abdominal bracing yielded a 47% stiffness increase.[36] Abdominal hallowing was characterized by internal oblique/transverse abdominus activity while abdominal bracing involved lumbar erector spinae activation of 30% MVIC among subjects. While abdominal contractions alone can increase stiffness of the spine above resting level we do not know from this study alone how much stiffness the spine requires above baseline to decrease pain and also see that spinal stiffness is enhanced with contribution from the lumbar extensors. These findings challenge prior interpretations of transverse abdominus' role and importance in rehabilitation. They also lead us to consider the role of the paraspinals and extensors in spinal stabilization as, strategies to increase intra-abdominal pressure may be attempts to compensate for decreased extensor function based on the works above.

1.5.3.2 Developing a theory into practice: establishing the role of the lumbar extensors and their contribution to spinal stability

The primary lumbar extensor muscle group identified as the multifidus muscles has potential to be the most significant of the stabilizers of the lumbar spine. They represent the largest muscle group with direct attachment to the lumbar spine whose fibers have been associated with stabilization of the vertebral segments during motion. Their physiologic cross sectional area provides more force generation to stabilize the

spine than the quadratus lumborus, illiocostalis thoracis and lumborum, or rectus abdominus[91], supporting their role in stabilization. Their morphology and histology allow for this muscle group to generate more force as it is lengthened[91,92] aligning them to stabilize the spine as trunk flexion angles increase. The flexors and extensors are reported to work in a co-activation pattern to promote stability of the spine[74], however, lumbar multifidus models have been shown to provide up to 2/3 of that stabilizing force about the spine[93]. Therefore, the lumbar multifidus would, anatomically and morphologically, be at an advantage to be the key player in spinal stiffness associated with stabilization of the spine.

While the support for the role of transverse abdominus is primarily based on delayed onset, several properties of lumbar multifidus have been associated with the presence of LBP. Reduced cross sectional area of the lumbar multifidus has been present in patients with acute to subacute low back pain, with the atrophy located ipsilateral to the pain and typically isolated to one level that has been identified to be the pain generating level through physical examination.[28,94] The specific localization of the atrophy to side and vertebral level would suggest that some direct relationship exists between the pain and muscle atrophy. The time from pain onset to atrophy has been noted to be too short to be attributed to disuse atrophy[28] which suggests there may be some more direct link between LBP onset and LM atrophy. This type of rapid atrophy to muscles can often be from loss of neural input[95] and can be a result of nerve root lesion in the lumbar spine.[96] Experimental injury to the annulus, as well as nerve root injury, has demonstrated atrophy in the lumbar multifidus, in as little as three days in porcine models[97] potentially supporting the presence of a more direct link between pain and

muscle changes in the LM. The atrophy noted by Hides et al. (1994) does not improve unless specifically targeted therapy is provided. Failure to regain function of the LM in the absence of automatic recovery of the LM may be directly associated with prevalence of recurrent pain.[23] It can be speculated that failure to regain this muscle's function leads to chronicity further degradation of the spinal column as patients who require surgery for their LBP demonstrate this asymmetric atrophy[94,98] that is localized and likely not associated with disuse.

Aside from atrophy, and perhaps as symptoms become more chronic, increased fatty infiltrate to the muscle also become apparent.[18,99] D'Hooge, et al. (2012) reported that while overall cross sectional area was not changed in patients with low back pain, the increase in fatty infiltrate had more effect on the quality of the muscle. Morphologic changes to the LM have associated with increase in inflammatory cytokines related to spinal injury on animal models[100]. The changes likely affect their ability to stabilize the spine as the muscle loses the ability to generate force, as force generating tissue is replaced by non-contractile adipose tissue. Experimental injury has also demonstrated an increase in muscle stiffness within 12 weeks from a simulated disc degeneration puncture in rabbits.[101] Considering the LM's ability to generate force increases as the muscle is lengthened into a trunk flexed position [91,92], histological changes leading to increased stiffness are likely to decrease the extensibility of the LM. This reduction in muscle extensibility can lead to reduction in the muscle's ability to lengthen during forward bend thus inhibiting force generation to stabilize the spine.

There are some questions that are raised to the relationship of LM and LBP. The association of fatty infiltrate's to LBP is strong in adults in their 40's independent of BMI

and activity levels with greater amount of fatty infiltrate increasing odds of having experienced low back pain, within the last year. [99] This is not true of all patients with LBP as the association of fatty infiltrate and occurrence of LBP decrease in a 10-year follow up of these patients at 49-50 years of age.[102] These increased odds of low back pain occurrence highlight the importance of the lumbar multifidus. However the decreasing odds a decade later may not suggest the initial study results were spurious relationships, but instead be could suggestive that the patients have regained some stability from compensation by the passive subsystem.[59,60]

Aside from structural changes in the muscles, there also appear to be metabolic changes that may be related to their morphologic changes. LM of patients with chronic low back pain demonstrate imaging shifts on fMRI compared to healthy controls that suggest a change to higher glycolytic muscle fibers.[19] According to the authors, increase to a more anaerobic metabolism with higher rates of metabolic activity could represent muscles that have to perform higher intensity contractions with the side effect of more rapid fatigue. Loss of physiologic cross sectional area through atrophy and muscle fatty infiltrate could result in more demand placed on the remaining muscle. Morphologic and metabolic changes in the lumbar multifidus that do not improve may factor into recurrence of pain or chronicity in some patients.[18] More importantly, in a study investigating predictors for success with TSE, greater impairment in LM activation levels were responsible for greater number of exam findings placing people into a stabilization exercise category, with 80% of these patients having had prior LBP.[16] This draws one to consider the impact that LM may have on movement control that TSE may address as well as factoring into recurrence. Overall, however, these findings substantiate the role of

LM in LBP and rehabilitation. Given these results and association to changes in LM with LBP, TSE should be one of the most effective intervention methods in patients with low back pain. However, that has not always been the case, and methods to identify patients who would benefit from TSE have been studied with mixed results.

1.5.4 Identifying patients with low back pain that would benefit from trunk stabilization exercises

Armed with a strong theoretical background supported by evidence, specific stabilization exercises should have potential to improve outcome and function in patients with LBP. One of the first randomized control trials on stabilization exercises in patients with LBP demonstrated successful outcomes compared to a control group with continued lasting effects at follow up.[103] However, other randomized control trials using trunk stabilization exercises have shown no additional effect from stabilizing exercises in patients with LBP.[104-106] A Cochrane review in 2000 suggested that in some cases no treatment for LBP was equally effective as exercise.[107] The difference in outcomes between O'Sullivan et al. (1997) and other randomized trials could be due in part, to patient selection. O'Sullivan randomized the intervention to a specific group of patients with spondylolysis or spondylolisthesis of the spine, likely with instability from those osseous faults. Other investigators had patients with non-specific LBP. This highlights the importance of matching proper interventions to patients, which is the aim of treatment-based classifications. Since then, there is evidence that specific classification based rehabilitation for LBP may be more successful than a general rehabilitation program[26] and support that matching interventions to exam criteria is crucial for good outcomes.[52]

As a result, there has been the development of a clinical prediction rule for determining the type of patients who that would benefit from trunk stabilization exercises.[8] The TSECPR aims to identify patients who would benefit from trunk stabilization exercises. Initially, 4 patient characteristics were identified, with the presence of 3 or more of these characteristics increasing post examination likelihood for success to 67%.[8] More importantly, there are still other patients, who are identified in this prediction rule to fail with trunk stabilization exercises. Some may be identified as failing with stabilization exercises because they may belong to another group. However, other's may be failing because of a current inability of the TSECPR to detect the need for adjunctive treatment, perhaps introducing methods to selectively recruit key specific muscle stabilizers that are not functioning well due to inhibition or morphological changes. Better understanding of LM inhibition could help in improving diagnostic tests and interventional efficacy.

Up to now, the proposal has only mentioned morphologic and metabolic changes to the LM. However, neural control of the LM also appears to be affected in patients with LBP. Investigators using real time ultrasound imaging have detected cross sectional area differences as well as impairments in volitional contraction of the lumbar multifidus[63] leading the authors to suggest changes in neural control of the muscle. Similar findings are noted in healthy control subjects with induced pain through saline injection[108], demonstrating a rapid inhibition of the muscle. While the mechanism is not well elucidated, pain related inhibition is a plausible explanation. The lumbar multifidus would not be the only muscle group that has shown inhibition with injury, as the

quadriceps have demonstrated similar findings following surgery unrelated to the quadriceps, that appears to be influenced by central nervous system impairments.[109,110]

Mechanism for impairment in the LM may have a very similar neural component. In these patients with quadriceps activation failure, development of a method to detect the underlying mechanism of neural inhibition has resulted in the use of electrical stimulation as an important supplemental intervention in these patients.[40,111,112] In fact, not providing supplemental intervention has shown to inhibit recovery of the quadriceps in these patients.[40,110] In patients with LBP, a review of trunk extensor exercises reports that general exercises are not always able to have lasting training effect unless care is taken to isolate specific muscle activity.[29] However, there is no further information currently available in patients with LBP to determine if 1) inhibition of LM is truly responsible for recurrent LBP 2) if there is a neural component to potential inhibition and 3) is supplemental interventions to the LM would be beneficial. We first need to determine if LM is truly a major factor in recurrence and to what extent its impairments play in LBP. This may lie in the two tests that identify patients who would benefit from TSE: presence of aberrant movement and the PIT.

1.5.4.1 What does aberrant movement during the forward bend tell us?

A recent validation study has found that the presence of 2 of the 4 aforementioned exam findings: aberrant movement during forward bend and prone instability test (PIT), improve diagnostic accuracy of TSECPR.[9] Patients with these 2 characteristics have been shown to have disruption in linear and angular displacement during the midrange forward bending, where muscle activity should be the dominant stabilizer.[32] Altered posture and lumbopelvic rhythm during forward bend have been speculated to be resulting from

decreased LM activity and overuse of the erector spinae[113] based on decreased paraspinal muscle activity during forward bend in patients with chronic LPB[11], but these works have been on a heterogenous population of patients with LBP without an isolated study into the lumbar multifidus. Decreased LM activity however, has been shown to be an important prognostic factor for patients to benefit from TSE[16]. The presence of aberrant movement increases likelihood for success twofold. However, even with this modified TSECPR, 28% of patients who met the criteria did not fully respond to treatment. Based on 1) movement disruptions identified by Teyhen et al. (2007) and theoretical basis of muscle impairments speculated by van Wingerden et al. (2008), 2) the impaired return of LM activity after a low back injury[23], and 3) need for specific training of the muscles to regain function[29], there is a possibility that there may be intervention selection issues associated with lack of response to TSE. However, before that can be determined, these underlying assumptions about the mechanism responsible for aberrant movements must be validated. Aberrant patterns of motion have been associated with LBP by many investigators.[8,114-116] The common aberrant movements are clinically defined are an instability catch/judder (JUD), deviation way from the sagittal plane (DEV), and altered lumbopelvic rhythm (aLPR). The operational definition of these are listed in table 1.1 as defined by Biely, et al. (2014).

Table 1.1. Operational definition of aberrant movements

Aberrant Movement	Operational Definition
Judder (JUD)	Sudden acceleration or deceleration of the trunk or quick momentary deviations away from the sagittal plane during forward bend
Deviation from the sagittal plane (DEV)	Movement away from the sagittal plane during forward bend, occurring in the transverse and/or frontal plane
Altered Lumbo-pelvic rhythm (aLPR)	Hip motion dominates first 1/3 of trunk motion on forward bend and/or lumbar segment movement dominate in the last 1/3 of forward bend; lumbar motion dominates in the first 1/3 of return from forward bend and/or hip motion dominates lumbar movement in the last 1/3 of return from forward bend.

The criterion validity of aberrant movement during forward bend has been studied, with clinical observation of JUD and deviation from the sagittal plane DEV being significantly associated with active LBP, separating subjects with aberrant movements with no history of low back pain[42]. This supports that while aberrant movements are present across populations, certain types of aberrant movement may have greater significance in the study of movement control in patients with LBP. While methods to quantify the 3 dimensional kinematics of these aberrant movements using secondary analysis from Biely et al (2014) yielded similar findings of unique aberrant movements' ability to identify those with LBP, these movements were aLPR and JUD[10],

not DEV. More importantly, the frequency of aberrant movements during the forward bend is highly associated with low back pain[42], suggesting that these are a consistent motor pattern within the individual and not spurious findings based solely on prevalence of aberrant movement across individuals.

Both Biely, et al. (2014) and Wattananon (2014) identify JUD's association with the presence of LBP. However, the discrepancy between DEV vs aLPR between the two studies is unclear. In regards to DEV's association with LBP, the chi-square analysis only identifies an association, but not a strength of association and can be largely effected by frequency of a condition.[117] DEV was present in a large number of the sample, including 43% of the forward bend observation of healthy individuals with no LBP.[42] It is likely that once analyzed using kinematic variables to more specifically identify those with DEV, this aberrant movement became a more pervasive pattern seen across populations and was not significantly associated with LBP.[10] Biely, et al. (2014) also mention that aLPR tended to present in unison with other aberrant movements; it is possible that more subtle aLPR in the observational study were overshadowed by other more prominent aberrant movements, decreasing the number of times aLPR was detected effecting analysis. However, despite the discrepancies, there are several neuromuscular factors that may play a role behind the mechanism of these aberrant movements.

During forward bending, the extensors must act to control the trunk mass, eccentrically during the lowering phase and concentrically during the return phase. It is theorized that atypical and unsmooth motion can be representative of the neural system's attempt to stabilize a joint via motor unit synchronization during a challenging task and represented on EMG with increase in peak amplitude and power band spectrum.[15] The

increase in EMG amplitude during synchronization is likely due to increasing motor unit recruitment simultaneously to generate more force. Fluctuations in force production during contraction have been noted with increasing motor unit recruitment[118] and this may be occurring during aberrant movement patterns. In hand muscles with similar multi-pennate and multi-segmental morphology to the LM, these types of motor unit synchronizations have been found to occur more during eccentric contractions[119], similar to the lowering phase of forward bend where aberrant motions kinematics were quantified in patients with LBP.[10] The early hip motion associated with aLPR may be due to the neural system's attempt to increase motor unit contribution during forward bend by recruiting other motor groups. This strategy of recruiting other muscle groups are present with extensor function in healthy subjects: as demand on the extensors increase, lumbar multifidus activity increases along with a shift from medial erector spinae to the lateral erector spinae along with recruitment of quadratus lumborum, likely to increase the physiologic CSA of muscles participating in the activity.[120] Fatiguing exercise of the trunk in healthy subjects have demonstrated increases in peak EMG initially of the lumbar extensors, up to a threshold, but once the threshold is exceeded, there is reduction in lumbar extensor EMG, with increased recruitment of hip extensors.[121] The early onset of hip motion or domination of hip motion, from a motor recruitment perspective may be similar in mechanism: recruitment of other muscles to facilitate a goal of forward bending.

Along with the possible neural drive mechanism for aberrant movements that can be assessed using sEMG, physiologic muscle properties potentially lend evidence for a muscular mechanism behind aberrant movements. The multipennate fiber orientation of

the LM [64], lends itself for coordinated activation and deactivation of motor units during motion to produce smooth movements, with irregular motion likely occurring with motor unit synchronization. Aberrant movement during forward bend may represent changes in motor control strategy during the forward bend to deal with increasing load and lever arm of the trunk through motor unit synchronization. However, this is speculation and analysis of EMG characteristics during the movement may provide more insight to this theory by providing more detailed knowledge of the neuromuscular mechanisms underlying aberrant movement during the forward bend.

1.5.4.2 What might the prone instability test tell us?

The PIT is performed with the patient prone, trunk supported on the examining table, and the legs over the end of the table with the feet on the floor. Passive posterior to anterior (PA) forces are applied to the intervertebral segments. PA loads are able to cause deformation of the spine with translation of the vertebral bodies in the sagittal plane.[122,123] PA loads are applied to the spine for provocative testing for various diagnostic purposes including identification of painful segments[23,28] and assessing for segmental mobility.[35,124] If pain provocation occurs with this load, the patient is asked to lift the legs off the floor, holding on to the table as necessary to maintain position. A positive PIT is represented by an elimination of pain when the posterior to anterior load is reapplied to the pain provoking segment.[35,125] The extensors are assumed to contract to stiffen the spine, resulting in symptom reduction. A negative test: symptoms not abolished or diminished, is not as well elucidated. A negative result may suggest inadequate muscle activity to stabilize the spine to eliminate symptoms.

Similar tests exist to test the ability of muscular activity to stabilize the shoulder during provocative testing, but in these tests muscle activation to eliminate or decrease pain are both considered favorable response.[126,127] However, it is uncertain with the PIT, what impact elimination of symptoms versus reduction of symptoms may have on overall prognosis for benefit from TSE. In both cases, the lumbar extensors may be working to stabilize the spine while the latter may suggest insufficient activation for complete stabilization. Failure to eliminate or reduce symptoms may be more indicative of failure to stabilize the spine with extensor activity. However, the validity of these test assumptions has not been systematically investigated. Muscle activity is also uncertain with the PIT. EMG analysis has found lumbar erector spinae (LES) and LM activity to be similar during a bilateral hip extension task.[128] However, in their test the subjects brought their hips from a neutral position to a hip extended position while the upper trunk was stabilized with straps. The difference in movement positions from this task to the PIT makes generalization of EMG activity questionable, but it does suggest that the trunk extensors have a large role during this type of movement.

Dysfunction of the lumbar extensors has been *clinically* assumed to be responsible for aberrant movement and the results of the PIT, but this has not been validated. The ability to identify impaired performance of the extensors is important as the function of these muscles, specifically the lumbar multifidus (LM) has been identified as a key muscle related to predicting success with TSE with higher numbers of predictor variables being present with lower LM activation levels.[16] PIT had the highest univariate point biserial correlation (r= .38) of the predictor variables followed by presence of hypermobile segments (r=.36) in relation to reduced LM activity levels. What is unclear

from this data is what role decreased LM activation has with a positive PIT, considering that LM activity is what is *assumed* to give a positive response. Intuitively, a positive PIT would be *negatively* correlated with LM function.

Based on similarity of r values between the PIT and rating of hypermobile segments, the results may be influenced by the number of people who had hypermobile segments in relation to decreased LM activity. The patient must first have pain with a PA load to be considered for positive for the test. Perhaps with decreased levels of LM activity associating with greater hypermobility, patients with good LM activation did not demonstrate pain with a PA load, leading to negative test results. Therefore, the first step in detecting a positive PIT: pain with PA load, may be a reflection of changes in LM activation. However, the patients in this study may have had adequate LM activation with hip extension to stabilize the spine. These results however, are the only link to the PIT and LM activity and do not clarify a relationship between LM and PIT findings, making interpretation of the test difficult. While the PIT helps to identify patients who would benefit from TSE, better understanding *why* could favorably impact clinical decision making. One such possibility is through selection of interventions that may preferentially target specific muscle related impairments, such as LM activation and/or strength deficits. Delineation and improved appreciation of the mechanisms responsible for the presence of aberrant movements and PIT findings, allows for better interpretation of test findings, so clinicians understand movement control impairments that are driving these test results, which is the goal of this proposal.

1.6 Preliminary Studies

The purposes of the preliminary studies were to pilot and verify the different methodological approaches needed to address the aims of the proposal. For aim 1, data reduction and analysis methods were tested to determine the feasibility of identifying muscle activation patterns during the forward bend. In addition, a pilot study was also performed to determine if the linear elastic beam stiffness model chosen to study spine stiffness met assumptions of linearity. For aim 2, several methods were used to determine the ability of NMES to isolate and fatigue the LM. EMG data was collected in a small sample to ensure the feasibility of collecting trunk muscles activity during the test. Testing was also performed to determine if the NMES created significant disturbance within the electromagnetic field during collection of spine stiffness data.

1.6.1 Approach to sEMG analysis during the forward bend: Example in several subjects

The purpose of aim 1a is to characterize surface EMG (sEMG) signals of patients with and without aberrant movements associated with low back pain. The working hypothesis of aim 1a is that the lack of a smooth movement pattern is due to increased demand on the neuromuscular system during forward bend that requires altered trunk muscle activation or that is not adequately met. It is theorized that, as challenge on the musculoskeletal system increases, synchronization occurs within motor unit to produce more force, resulting in increased EMG amplitude with reduction in smooth movement [15,118]. Therefore, increases in muscle EMG amplitude in the time domain that aberrant movement occurs, may indicate this. However, there may also be *synchronization between muscles of the trunk* during the task, which may be reflected in the sEMG

activation patterns between muscles. This type of muscle synchronization has been seen in muscles of the knee, with synchronization increasing as demand on the muscles increase.[129] Analysis through cross correlation of trunk muscles is one approach to investigation of this theory. Cross correlation involves correlation of 2 time series signals against each other, shifting 1 signal back and forth against the other, with correlation values determined at each shift. This function gives the point in time (phase lag, T) that the signals are most correlated with the other and the correlation coefficient (r).[130] T provides information on muscle timing, positive r indicates muscles in phase, and negative r indicates muscles out of phase.[131] Therefore, a positive r with small T would indicate muscles activating in phase within a short time of each other, with increasing T values indicating further temporal width between contractions. A negative r would indicate one muscle activating while another muscle deactivates, with T representing the time between those two events. The magnitude of r values would determine how in phase or out of phase muscles are in relation to other muscle pairings and their respective r values.

The purpose of this preliminary analysis was to investigate the feasibility of this approach to characterizing motor behavior by analyzing sEMG of primary muscles during forward bending. Chang et al. (2012) found increasing cross correlation values and decreasing time lags as muscle fatigue increased, likely from the need to increase synchronized muscle activity to complete the task. This may be the same in patients with LBP performing a forward bend with a muscular system that is impaired and not adequate to complete the task and need more muscles to synchronize for task completion.

Based on association of types of aberrant motion to the presence and absence of low back pain.[10] Investigating the mechanism behind these specific aberrant movements could possibly yield information about the faults in the system that these observations are detecting, and potentially bring investigators closer to optimal interventions. Biely et al (2014) found that the clinical observation of JUD and DEV were strongly associated with patients with LBP. Secondary analysis of this data was performed to quantify and validate kinematic variables that identify these aberrant movements [10]. This secondary analysis found significant associations between kinematically defined JUD, aLPR and LBP. The current proposal will be investigating sEMG data synchronized with the kinematic variables that were significantly associated with LBP, so will focus on subjects whose kinematic data identifies them as having JUD and/or aLPR. Cross correlation values and time lags will be studied to look at synchronization of muscles. Percent contribution of muscle activation during the forward bend will be studied to identify contribution of specific muscles during the forward bend to characterize contribution of muscles during the task. sEMG amplitude during forward bend will be studied to determine if amplitude differences exist in patients with aberrant movement versus healthy controls.

Methods

For this preliminary analysis, 4 patients with low back pain and 4 healthy subjects were selected from this sample for preliminary data analysis. This sample of 8 subjects was taken from 32 patients (62% female, age 44±9.8) with LBP identified through clinical examination to benefit from TSE [8,9] and 37 healthy subjects (64% female; age 42±10.5) that participated in a prior LBP study and were part of the sample used by

Wattananon (2014). These subjects all had simultaneous recording of sEMG and kinematic variables recorded during the forward bend. The current LBP group had pain present for 12 weeks or less with no course of physical therapy for their current symptoms. Exclusion criteria were: history of spinal surgery, peripheral or central neurologic signs, lower extremity surgery or injury that would affect testing, systemic symptom or pregnant.

An electromagnetic tracking system (Liberty, Polhemus Inc., Colchester VT) was used to capture kinematic data. Electromagnetic sensors were mounted on thermoplast molded to fit the contours of anatomic landmarks. These sensors were placed on the spinous process of T3, L1,and S2 to model segments of the trunk, and a sensor on the lateral condyle of the femur to model the movement of the pelvis on the femur (see complete details of subject preparation in Appendix E). SEMG (SA Instrumentations, San Diego, CA) data for 16 trunk muscles were also collected (gain 500; band pass filtered 20-500Hz). Skin surface was prepped by cleaning with alcohol and abrading with sand paper. Pairs of Ag-AgCl electrodes were placed with 2cm inter-electrode distance along landmarks for muscles as described bilaterally in Table 1.2. A reference electrode was placed on the lateral malleolus. A custom program (Labview 8.6, National Instuments, Austin Tx) was used to collect sEMG (2400 Hz) and kinematic (120Hz) data simultaneously.

Subjects underwent collection of quiet resting sEMG for 2, 30 second trials followed by maximal and submaximal strength testing for trunk flexion, extension, and bilateral side bending for the purpose of sEMG normalization. They were seated in a custom device designed to secure the lower extremities to minimize their contribution to

the force, and a tension load cell was utilized to measure their strength. Visual feedback was given to the subjects at 15% of their body weight for submaximal contraction while no visual feedback was provided for maximal contraction (sMVIC). They then performed 2 sets of 3 forward bend trials with return to standing (6 forward bends). Subjects were asked to bend forward as far as they comfortably could and then return to standing.

Table 1.2Bilateral surface sEMG electrode placement

Muscle	**Location**	**Muscle**	**Location**
Rectus Abdominus (RA)	3cm lateral to umbilicus	**Latissmus Dorsi (LD)**	Midline between spinous process of T9 and axillary line
External Oblique1 (EO1)	15 cm lateral to umbilicus	**Thoracic Erector Spinae (TES)**	5cm lateral to T9 spinous process
External Oblique2 (EO2)	5cm above and 5cm medial to EO1	**Lumbar Erector Spinae (LES)**	3cm lateral to L2 spinous process
Transverse Abdominus/Internal Oblique (IO)	2cm below and medial to ASIS and above the inguinal ligament	**Lumbar Multifidus (LM)**	2cm lateral to L5 spinous process

Kinematic variable were used to quantify and develop an algorithm to detect JUD and aLPR[10] using the following kinematic definitions established from this study: JUD: defined as sudden changes in instantaneous velocity or fluctuations in lumbar or pelvis segment angular velocity ; aLPR: reversal of the lumbopelvic rhythm in which hip motion is greater than lumbar spine motion in the first 1/3 of forward bend, and/or lumbar motion is greater than hip motion in the last 1/3 of movement. Kinematic data were analyzed using the algorithm developed by Wattananon (2014) and each forward bend

performed by the subject was rated for presence/absence of JUD and/or LPR using a custom program (Labview 8.6). Calculation of kinematic variables and the methods used to detect aberrant movement are presented in Appendix F and G, respectively. For the purpose of this preliminary study, a sample of 2 patients with LBP identified as having only JUD , 2 patients with only aLPR (4 LBP patients with aberrant movements), and 4 healthy controls with no JUD and aLPR were selected and their 6 forward bend movements were analyzed. Subjects were DEV were not analyzed in this preliminary study, as the presence of DEV appears to be a common characteristic even in healthy subjects as 43% of subjects with no low back pain were observed to have DEV (Biely et al. 2014), and the kinematics of DEV did not identify those patients with LBP (Wattananon 2014). Therefore, ignoring the presence of DEV and analyzing the sEMG patterns associated with LPR and JUD may be the most meaningful.

Data Reduction

Resting kinematic plot of the lumbar segment on the pelvis was measured and plotted against time to determine angular velocity. For subjects standing at rest, many forward bends started at a negative velocity. Therefore the first 0 crossing for *angular velocity* was used as a reference point, and the first data point that exceeded 1 deg/sec was considered the start of the forward bending phase. The forward bending phase is represented by a positive velocity based on the kinematics point of reference. As the segments begin to return from forward bend, the angular velocity assumes a negative value. Therefore, forward bending phase was considered to stop at the second 0 crossing, where the velocity transitioned from a positive to a negative value. Remaining data points from that point were considered to be return form forward bend and not currently

analyzed (Figure 1.1). Time stamps from the first and second crossing were used to obtain EMG signals for the forward bend.

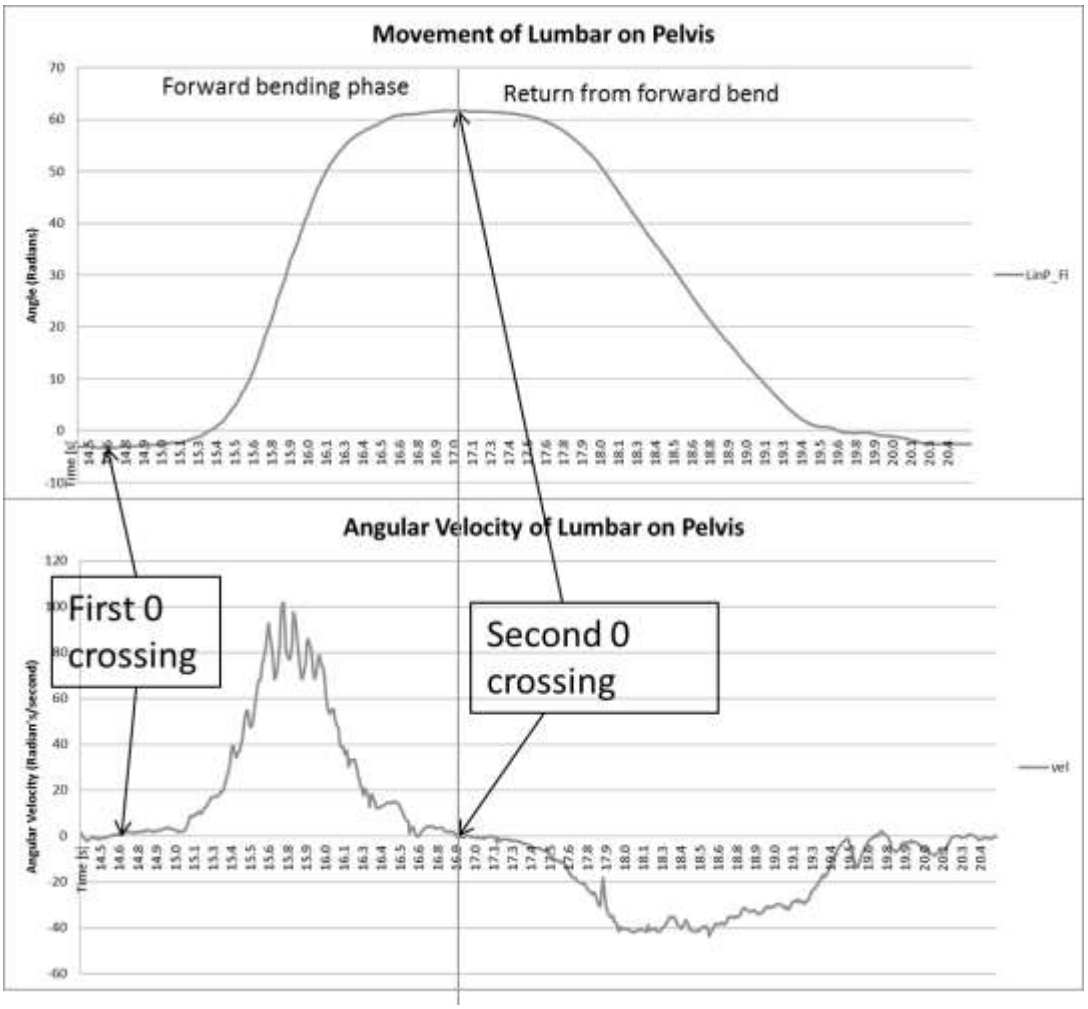


Figure 1.1 Determining initiation of the forward bend. Plot of angular displacement of the lumbar segment on the pelvis, with angular velocity plots in the forward bend and return from forward bend.
The first 0 crossing of the angular velocity was considered the initiation of forward bend and the second 0 crossing was considered the initiation of return from forward bend. SEMG from the forward bend portion was analyzed in this preliminary study.

Raw sEMG data had heart rate artifact removed using a custom fast independent component analysis (ICA) program (Labview 8.6). Cross correlations were performed on the forward bend portion task, using non normalized EMG signals with a 30ms RMS

window and a correlation timing window of 1.75 second to examine synchronicity between muscle groups.

A custom data synchronization program was used to RMS (Tc = 30ms) and down sample the sEMG to 120Hz (data collection frequency of the electromagnetic system), and synchronizes the signal to the kinematic data. Four subjects with aberrant movements were randomly selected along with 4 healthy subjects, reduced through this process. EMG amplitudes of the right and left lumbar multifidus (RLM, LLM), right and left lumbar erector spinae (RLES, LLES), and right and left thoracic erector spinae (RTES, LTES) were each averaged over the first 2 seconds of the initiation of the forward bend. The heart rate removed EMG signals were then RMS filtered without down sampling (2400 Hz) and the same 2 seconds of data were averaged for the same muscles. A paired t test ($P<0.05$) and coefficient of variation was performed on the signals of these 6 individual muscles at 120Hz and 2400 Hz, to determine if there was any change in signal amplitude as a result of downsampling from 2400 Hz to 120 Hz. There was no significant difference between the sEGM signals at 120Hz and 2400 Hz, with CV at less than 1%. Based on this, it was decided to perform exploratory analysis of the data at the down sampled rate (120Hz) synchronized to the kinematics.

Data Analysis

Side-to-side symmetry of the extensor muscle groups was investigated first, to determine if sides should be analyzed as individual muscles, or if they should be collapsed using a 2 x 2 mixed model ANOVA as well as an Intraclass Correlation Coefficient ($ICC_{2,1}$). Mean sEMG amplitude during the forward bend was determined for

each forward bend trial per muscle, per side and used in both ANOVA and $ICC_{2,1}$. Coefficient of variation (CV) between trials of the mean forward bend sEMG amplitude was calculated for each subject by muscle to determine variation between forward bends (Table 1.3). Due to high CV's, each forward bend was handled as a separate case, rather than collapsing by trials for side-to-side symmetry. For mixed model ANOVA, left and right Factors of the muscles: LM, LES, and TES each with 2 levels were considered within groups variable and the factors of LBP with 2 levels, was used as between subjects measure. $ICC_{2,1}$ coefficients were calculated separately for LBP and healthy control subjects per muscle group, with side as the testing variable.

To determine synchronicity of muscle groups, cross-correlation values (r) and lag times (T) were collected for 6 forward bend trials for all subjects. Correlations (r) and T values were averaged across trials per subject and compared across groups. Following cross correlation of the non-normalized signal, sEMG from strength testing was used to normalize the EMG. Submaximal contractions tended to level sooner than maximal contractions, allowing for longer time durations to obtain sEMG amplitudes. A 2 second mean around the peak amplitude was obtained for each submaximal test and two trials were averaged, as this method provided a stable value for normalization with lower standard deviations compared to the same values for maximum contraction. The highest mean peak average sEMG amplitude from flexion, extension, or side bending to either side was used to normalize sEMG data to obtain a percentage of the submaximal volitional contraction (sMVIC) during the forward bend. After sEMG normalization each forward bend which ranged from 2-4 seconds in duration, was time normalized by dividing into 10 epoch bins and sEMG for each muscle was analyzed within the bins.

Table 1.3Coefficient of variation of the mean SEMG amplitude during forward bend, between trials, per muscle.

Subject	Group	RLM	LLM	RLES	LLES	RTES	LTES
1	LBP	0.40	0.60	0.46	0.60	0.76	0.59
2	LBP	0.46	0.63	0.41	0.50	0.67	0.40
3	LBP	0.94	0.96	0.85	1.13	0.48	0.67
4	LBP	0.77	0.86	0.53	0.82	0.92	0.87
5	Control	0.53	0.57	0.30	0.59	0.31	0.43
6	Control	0.73	0.74	0.58	0.76	0.55	0.47
7	Control	0.47	0.64	0.45	0.57	0.46	0.62
8	Control	0.62	0.58	0.52	0.51	0.55	0.62

Results

Muscle Activation Symmetry

Table 1.4 contains descriptive statistics for normalized mean sEMG amplitudes between groups, for left and right muscles. There was a significant main effect of muscle side ($F_{3,44}$)=29.8, p<.001. There was an effect within groups for left to right difference for LM ($F_{1,46}$)=44.5, p<.001 and LES($F_{1,46}$)=10.2, p<.001, but not for TES ($F_{1,46}$)=1.2, p=.281. There was a between groups effect for left to right sided difference between patients with LBP vs control subjects for LM ($F_{1,46}$)=4.9, p<.05, LES($F_{1,46}$)=5.4, p<.05, and TES ($F_{1,46}$)=12.3, p=.001.

Due to between groups effect on side-to-side symmetry of sEMG amplitudes during the forward bend, $ICC_{(2,1)}$ was calculated separately for patients and control subjects. Table 1.5 contains the correlation coefficients, which were overall, fairly high.

Table 1.4 Descriptive Statistics of sEMG amplitude. Side-to-side means and standard deviations of average SEMG amplitudes (mv) during the forward bend, per muscle group between patients with low back pain and healthy control subjects.

	LM				**LES**				**TES**			
	Right		**Left**		**Right**		**Left**		**Right**		**Left**	
Groups	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Controls	0.82	0.50	0.61	0.38	0.50	0.22	0.47	0.29	0.18	0.09	0.26	0.13
LBP	0.54	0.35	0.40	0.31	0.39	0.22	0.27	0.22	0.51	0.39	0.36	0.24

Table 1.5 ICC coefficients for side-to-side difference. $ICC_{(2,1)}$ coefficients for average sEMG amplitudes during the forward bend, per muscle group between patients with low back pain and healthy control subjects.

	LM	LES	TES
Controls	0.77	0.90	0.77
LBP	0.84	0.77	0.87

Muscle group synchronization:

Results between ICC and mixed ANOVA were conflicting for side-to-side muscle symmetry. As this was a preliminary study, it was decided to treat muscle sides as individual muscles to explore muscle group synchronization. Table 1.6 contains cross correlation coefficients (CCC) (r) and lag between muscle onsets (T). Normality of CCC was performed using Kolmagrov-Smirnoff test using individual forward bend CCC trials for each subject. Assumptions were met for normal distribution, as a Fisher's transformation was not performed prior to averaging r-values. There are overall lower r values in healthy controls across the lumbar extensors, with higher T between muscle. CCC tended to be higher across bilateral LM and LES in patients with LBP, but not so for relationships among TES or between TES and the LM/LES groups.

Table 1.6. Mean cross correlation coefficient (r) and time lag (T) between muscles during the forward bend. Patients with LBP with JUD and aLPR demonstrate higher r-values and shorter T duration compared to healthy control subjects.

	Healthy Controls				**LBD JUD and aLPR**			
	Mean r	SD r	Mean T (s)	SD T	Mean r	SD r	Mean T (s)	SD T
RLM-LLM	**0.36**	**0.18**	**1.00**	**0.63**	**0.74**	**0.20**	**0.31**	**0.39**
RLM-RLES	**0.32**	**0.19**	**1.42**	**0.69**	**0.68**	**0.23**	**0.32**	**0.38**
RLM-LLES	**0.27**	**0.22**	**1.24**	**0.68**	**0.72**	**0.25**	**0.30**	**0.40**
RLM-RTES	0.17	0.17	1.44	0.88	0.32	0.21	0.64	0.06
RLM-LTES	0.18	0.21	1.34	0.61	0.48	0.19	0.47	0.23
LLM-RLES	**0.19**	**0.22**	**1.11**	**0.64**	**0.74**	**0.20**	**0.37**	**0.33**
LLM-LLES	**0.54**	**0.15**	**1.22**	**0.50**	**0.77**	**0.24**	**0.28**	**0.42**
LLM-RTES	0.19	0.14	1.14	0.83	0.26	0.25	0.57	0.13
LLM-LTES	0.23	0.15	1.43	0.62	0.48	0.25	0.52	0.18
RLES-LLES	**0.21**	**0.21**	**1.23**	**0.77**	**0.75**	**0.23**	**0.29**	**0.41**
RLES-RTES	0.22	0.15	1.46	0.94	0.24	0.20	0.57	0.13
RLES-LTES	0.19	0.20	0.26	0.56	0.44	0.19	0.39	0.31
LLES-RTES	0.20	0.17	2.44	1.05	0.25	0.26	0.66	0.04
LLES-LTES	0.17	0.21	1.23	0.68	0.49	0.21	0.55	0.15
RTES-LTES	0.22	0.12	0.83	0.54	0.32	0.22	0.25	0.45

EMG amplitude during forward bend

Table 1.7 contains mean normalized sEMG amplitudes during the forward bend, represented as a percentage of the sMVIC time normalized into 10 bins, each representing 10% of the forward bend. Patients with aberrant movements tended to

demonstrate a higher activation of the trunk extensors during the forward bend especially the lumbar multifidus compared to healthy controls with no aberrant patterns. These patients maintain a higher level of LM activation throughout the movement, but the activity of LM peaks in the beginning of the motion, with LES peaks happening within the same bins. However, there appears to be a change in movement strategy that occurs at the halfway mark of the forward bend in these patients: LM activation while remaining high begins to decrease while TES activity begins to increase. In healthy control subjects, TES activity levels stay fairly steady while both LM and LES groups begin to increase activation towards 50% of the motion and then decrease fairly sharply.

Movement control during forward bend:

Figure 1.2, Figure 1.3, and Figure 1.4 depict the percent contribution of extensor muscle activation during the forward bend along with the angular velocity of the lumbar segment relative to pelvic segment. Based on muscle symmetry among healthy controls without DEV, it was decided to average the forward bends of these subjects together for analysis. Healthy control subjects, there is an early rise in angular velocity and a gradual reduction in velocity towards the end of the forward bending phase. There is no large fluctuation in the muscle activity through the 10 bins. In the patients with aLPR, lumbar velocity starts lower and gradually increases by the third bin. This is likely due to the pelvic dominant movement in the beginning of motion. As velocity increases, LM and LES are the dominant muscles with no change in velocity. By midpoint of the 6^{th} bin, there is a dramatic increase in TES activation along with a sharp reduction in velocity. There is a similar pattern in patients with LBP presenting with JUD, as they begin to

demonstrate a shift in muscle activation strategy followed by a reduction in forward bend velocity.

Table 1.7. Percentage of the sMVIC trunk extensors during the forward bend, time normalized to 10 bins each representing 10% of the forwarding bending motion. Shading separates the forward bend by halves.

LBP (JUD and aLPR Combined)

	RLM		**LLM**		**RLES**		**LLES**		**RTES**		**LTES**	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Bin 1	**75%**	55%	**64%**	38%	49%	22%	42%	26%	25%	9%	10%	4%
Bin 2	**127%**	64%	**97%**	36%	**62%**	23%	**68%**	37%	24%	14%	10%	6%
Bin 3	**141%**	53%	**114%**	34%	**74%**	24%	**84%**	42%	26%	11%	10%	3%
Bin 4	**140%**	53%	**97%**	40%	**78%**	26%	**76%**	33%	32%	11%	15%	8%
Bin 5	**107%**	38%	**80%**	41%	**72%**	24%	**61%**	32%	45%	19%	30%	12%
Bin 6	61%	20%	52%	30%	**54%**	18%	**40%**	20%	**78%**	23%	**40%**	15%
Bin 7	50%	20%	30%	12%	44%	14%	21%	8%	**85%**	18%	**40%**	9%
Bin 8	48%	23%	30%	8%	38%	18%	19%	7%	**49%**	23%	**24%**	12%
Bin 9	61%	34%	43%	21%	41%	23%	27%	14%	**36%**	29%	**11%**	12%
Bin 10	85%	53%	61%	36%	47%	31%	39%	26%	**61%**	55%	**23%**	36%

Healthy Control Subjects

	RLM		**LLM**		**RLES**		**LLES**		**RTES**		**LTES**	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Bin 1	21%	13%	18%	14%	29%	16%	15%	12%	15%	5%	21%	7%
Bin 2	30%	6%	24%	11%	36%	11%	18%	13%	21%	6%	27%	6%
Bin 3	46%	12%	46%	7%	**47%**	13%	**39%**	7%	24%	6%	31%	8%
Bin 4	**71%**	14%	**63%**	15%	**64%**	19%	**57%**	19%	28%	5%	34%	5%
Bin 5	**86%**	9%	**80%**	13%	**62%**	17%	**65%**	18%	26%	5%	37%	6%
Bin 6	**87%**	31%	**65%**	28%	**59%**	24%	**66%**	26%	23%	4%	35%	11%
Bin 7	**49%**	31%	**39%**	28%	27%	14%	29%	16%	20%	6%	31%	18%
Bin 8	8%	4%	7%	2%	11%	4%	7%	3%	14%	4%	31%	19%
Bin 9	9%	1%	7%	1%	15%	6%	6%	2%	12%	3%	28%	13%
Bin 10	9%	2%	7%	1%	9%	2%	5%	1%	15%	3%	25%	11%

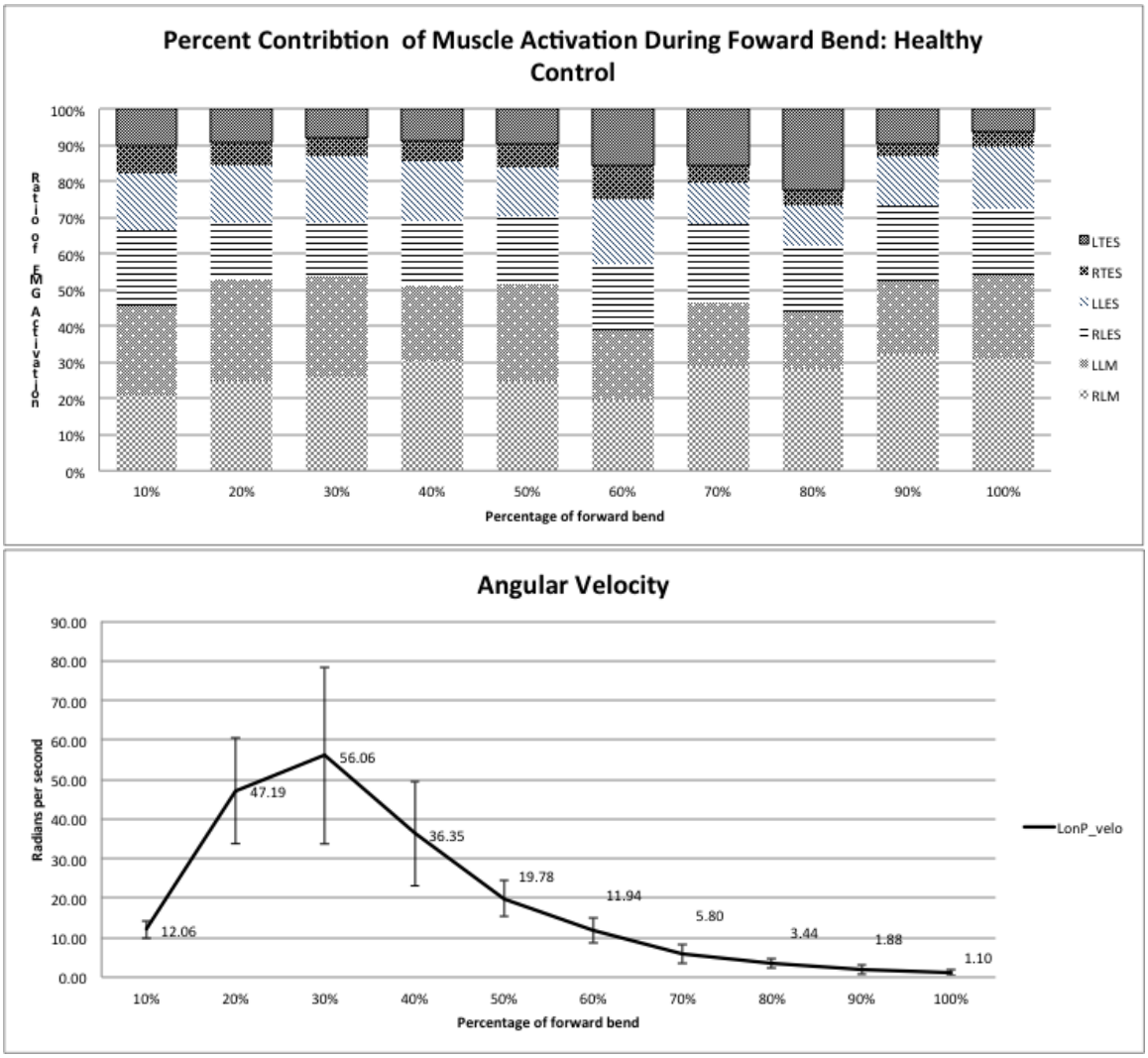


Figure 1.2. Average muscle activation pattern and angular velocity of 6 forward bends, in 4 control subjects with no aberrant movement over 10 epochs of movement. There is a sharp increase in velocity in the first bin that reaches maximum velocity by the third bin (roughly 30% of motion). Percent activation of sEMG muscle activation of the trunk extensors are fairly stable throughout the motion.

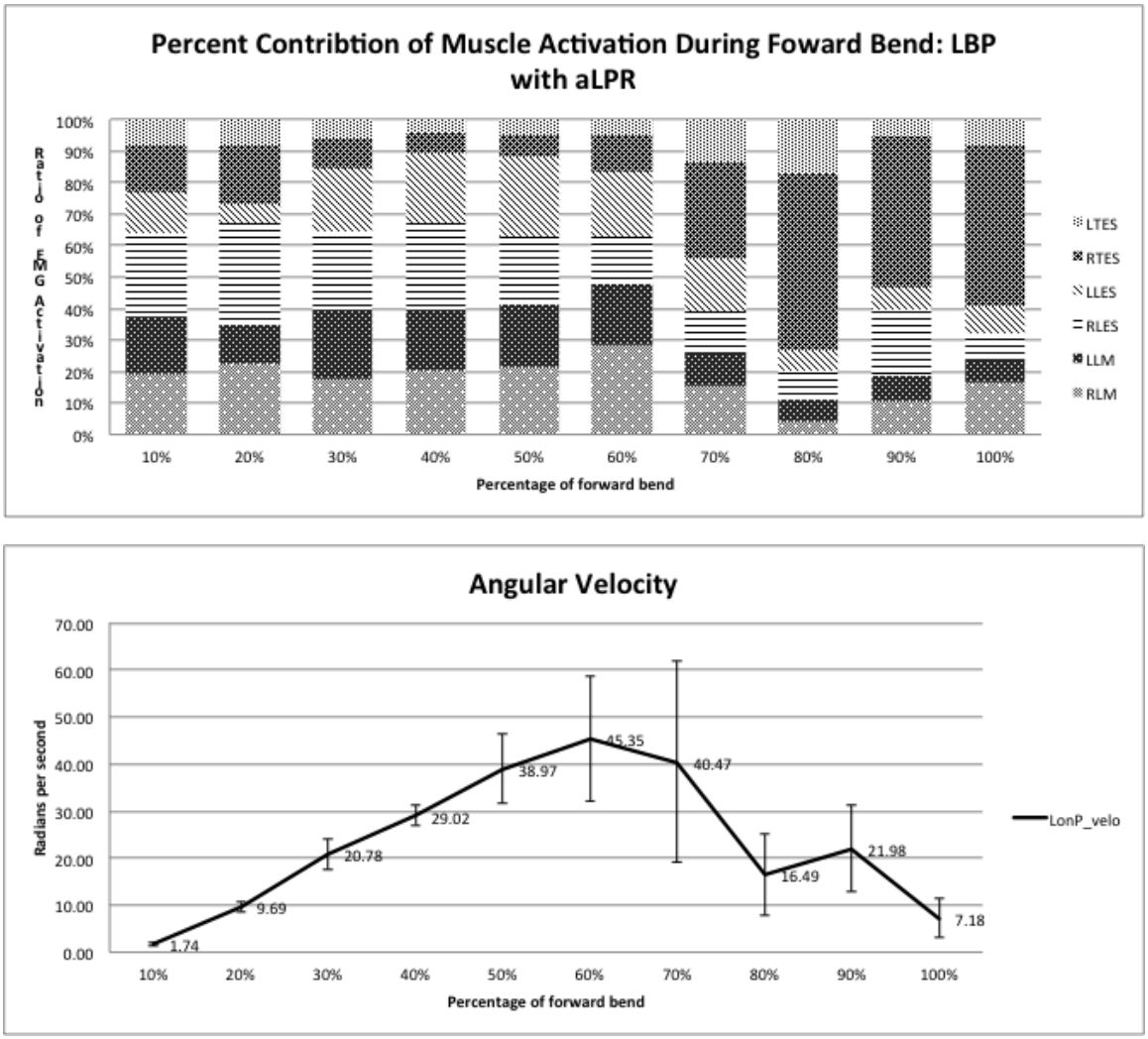


Figure 1.3. Average muscle activation pattern and angular velocity of 6 forward bends, in 2 subjects with aLPR, in radians per second over 10 epochs of movement. Lumbar segment velocity slowly increases and reaches maximum by the 60% of the motion. At the 70% point, there is a change into a thoracic erector spinae dominant pattern, with a sharp reduction in angular velocity.

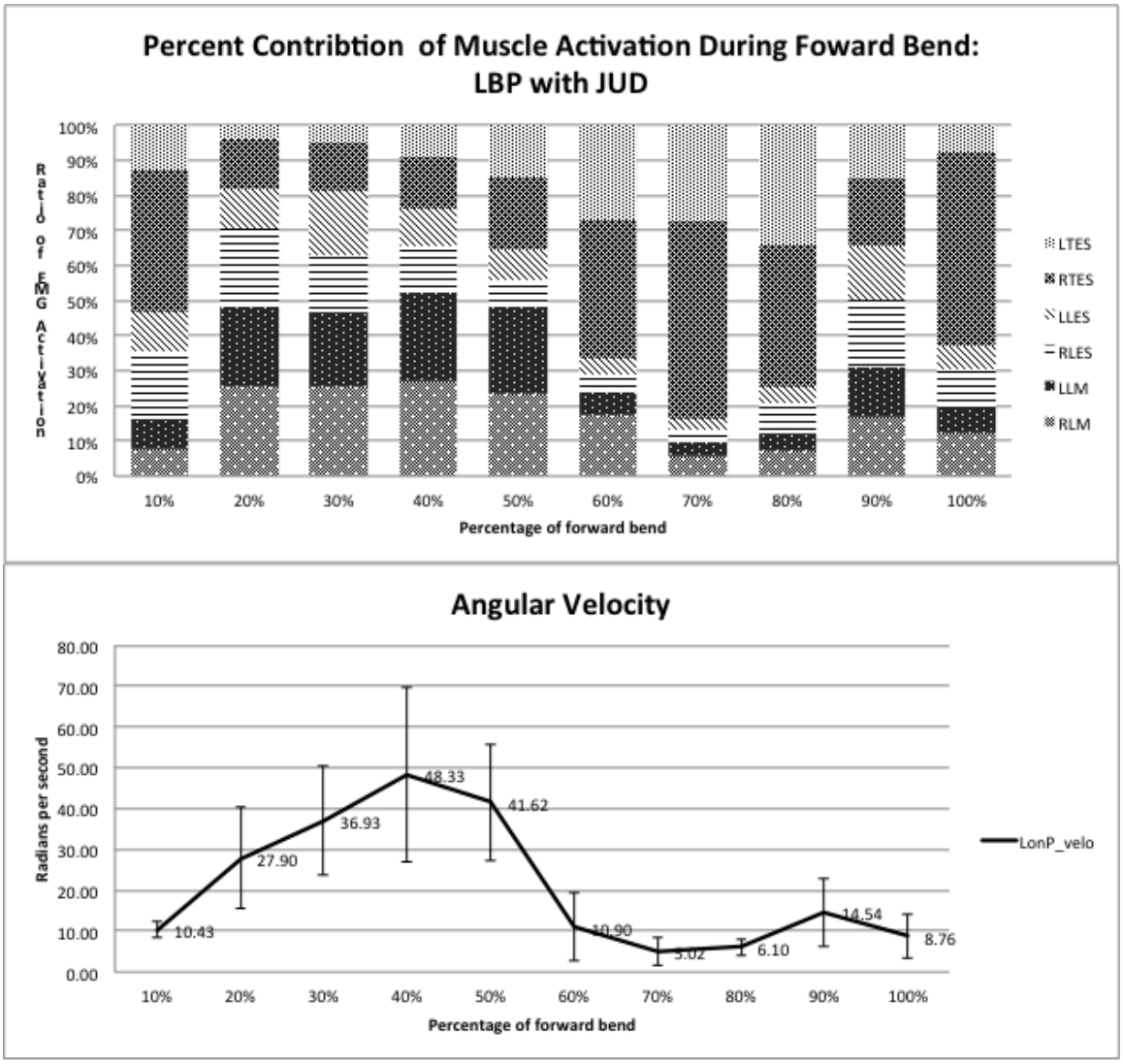


Figure 1.4. Average muscle activation pattern and angular velocity of 6 forward bends, in 2 subjects with JUD, in radians per second over 10 epochs of movement. Lumbar segment velocity increase is more gradual but similar to healthy control subjects, with peak velocity by the 40% of the forward bend. At 60% of the motion, there is a change over into a thoracic erector spinae dominant pattern, with a sharp reduction in angular velocity.

Conclusion

All subjects appear to demonstrate statistically significant side-to-side asymmetry of muscle activation during the forward bend. The operational definition of DEV would suggest that the overriding muscle activation characteristic of this movement pattern would be asymmetric muscle activity. However, based on the current results of mixed model ANOVA, asymmetry of muscle activity appears to be present across all groups. And therefore, may support the decision to ignore DEV and focus on aLPR and JUD. There are conflicting results from the ICC and the ANOVA. ICC was utilized for it's properties in determining how well groups resemble one another, as in the case of test-retest reliability. However, the differences may be occurring since ICC uses a random effects model versus a fixed model used in the ANOVA. In this case, where for the purpose of this proposal, the muscles of interest will be fixed, the mixed model ANOVA gives a better interpretation of symmetry. The asymmetry of the muscle groups would also suggest that the sEMG should be analyzed with independent sides, rather than collapsing sides to analyze as one muscle group. Prior to analysis of the data for the proposed study, side-to-side differences will be analyzed as was done in this preliminary study, to determine if muscles should be analyzed collapsed or as individual right vs. left side. This preliminary analysis was based solely on statistical significance of symmetry. Further work with larger sample size will include establishing minimal detectable change to determine if side to side differences exceed error, as well as calculation of effect size between sides to aid in the decision of collapsing sides or analyzing sides separately.

On inspection, the cross correlation values appear to demonstrate higher CCC values and shorter lag in patients with low back pain that present with JUD and aLPR,

compared to healthy controls. While not tested statistically due to low number of subjects, the higher r suggests more muscles acting in phase with each other in these patients. For many of the extensor muscles that had CCC above .5 in patients, the lag times were below 500ms. In prior studies, muscles with lag times below 500ms were considered to be acting simultaneously [131,132] , possibly supporting the theory that muscle synchronization is necessary to complete a challenging task. In healthy controls, these muscles had low CCC, with lag greater than 1 second. This may be more representative of synergistic activation of muscles, with certain muscle groups dominating during different portions of the forward bend to allow for smooth motions. This may be the case of the data in Table 1.7.1.6 representing the percentage of muscle sMVIC during the forward bend. In healthy control subjects, the LES begins to peak in bin 3 through bin 5 (30-50% of forward bend), with LM peaking in bins 4-6 (40-60% of the forward bend). Interestingly, in the later stages of forward bend, subjects with LBP demonstrating aberrant movement continue to maintain high levels of muscle activity, whereas healthy controls demonstrate a drop in muscle activity. This may represent a difference in passive structures between the two groups, with healthy control subjects being able to rely on passive structure tension to provide for spinal stability. The stability of muscle percent contributions through the 10 bins in the healthy controls may support this, and warrants further study. Preliminary data suggests that group comparisons of CCC and lag values should be able to determine differences in muscle synchronization.

While overall correlations in the LBP patients with aberrant movements tend to be higher between LM and LES, CCC values are lower in relation to the TES, suggesting this muscle group may be more out of phase during the forward bending phase of

movement. In fact, the muscle activation contributions of the forward bending phase demonstrates a higher LM and LES in the first ½ of the motion with a switch to a TES dominant activation pattern, suggesting a change in muscle activation strategy. In both the aLPR and JUD, the central nervous system may be trying to control the decent of trunk but the muscle activity may not be adequate, and a change in strategy has to occur: recruitment of the TES to abruptly decrease angular velocity. This may be represented by the out of phase correlation coefficients between LM, LES, and TES. Lastly, the patients with low back pain tended to possess a larger percentage of the sMVIC during the forward bend compared to healthy controls, which may suggest a larger percentage of motor unit activation during the task, and could also represent synchronization of motor units during the forward bend. Based on preliminary results, the current method of binning sEMG data and cross correlation appear to offer an appropriate approach to characterize sEMG patterns during the forward bend.

1.6.2 Bending Stiffness of an Elastic Beam: Validating Linearity Assumptions of Stiffness.

Completion of Aim 1b and 2A will require the ability to identify stiffness changes of the spine. To accomplish this, an elastic beam model of the spine will be used. In this model, the lumbar spine represents an elastic beam that is supported by the thoracic cage and the pelvis as cantilevers, with a force applied to the spine to produce angular and linear displacement [133]. A formula has been derived to model this during posterior to anterior stiffness during a manipulative force, using the formula below:

$$EI = \frac{\frac{Pb}{2L}[(e+a)d + ab]}{\theta_{L1S}}$$

where stiffness (EI) is defined by pressure applied to the spine (P), the distance between the rib cage cantilever and the sacrum cantilever (L), the horizontal distance between the pressure applied to the spine and the sacrum (b), the horizontal distance from the rib cage cantilever to the pressure applied (a), and the maximum angular displacement of the spine (θ_{L1S})[134].

The elastic beam model has been utilized by Shum et al, (2013) to measure changes in bending stiffness of the lumbar spine during posterior to anterior mobilizations. In their study, electromagnetic sensors were placed on the first lumbar spinous process (L1, representing the thoracic cantilever) and on the sacrum at the first spinous tubercle (S1 representing the pelvic cantilever) to measure bending stiffness of the spine using displacement of the sensors. However, the equation operates on an assumption that structural bending can be represented by linear relationship. Work was performed in the lab to check this assumption of a linear relationship to force and bending stiffness. In this study, two cantilevers of known distance supported a PVC beam. The beam stiffness was tested under several conditions to determine if the stiffness measures meet the assumptions of linearity. Verifying this assumption is important when comparing load delivered to the spine and stiffness changes during the planned experiment for aim2b.

Methods

A 94 cm long PVC beam, 3mm in thickness, with an inner diameter of 2cm was supported at its ends by wooden beams and is depicted in Figure 1.5. Wood was used to limit distortion of the electromagnetic sensors. Two electromagnetic sensors (sensors 1 and 2) (Liberty, Polhemus Inc., Vermont) were attached to the ends of the PVC beam, 31

cm apart. Sand weights held in a plastic container were suspended from a nylon cord 21 cm from sensor 1 and 11 cm from sensor 2 to allow for testing vertical displacement of the beam with varying loads. The load was offset between the two sensors, as this may be the case when testing aim 2b. A third sensor (sensor a) was initially placed directly above the load as an additional sensor to detect displacement directly at the load and as an additional sensor in determining linearity of the formula. Data were collected at 120 Hz. A plastic container was filled with sand of gradually increasing weights (7.5lb, 12 lbs, 16.5 lbs, 24 lbs) and vertical displacement measured at each weight increase.

Sensors were moved to various locations as depicted in Figure 1.6 to determine if position of the sensor along the beam would affect linearity. The experiment was repeated with a compression load cell with a ferrous metal casing that will be used in the planned validity experiment. The ferrous metal was introduced in to the field to determine if the presence of a small amount of metal would significantly distort the electromagnetic field and therefore alter stiffness measures.

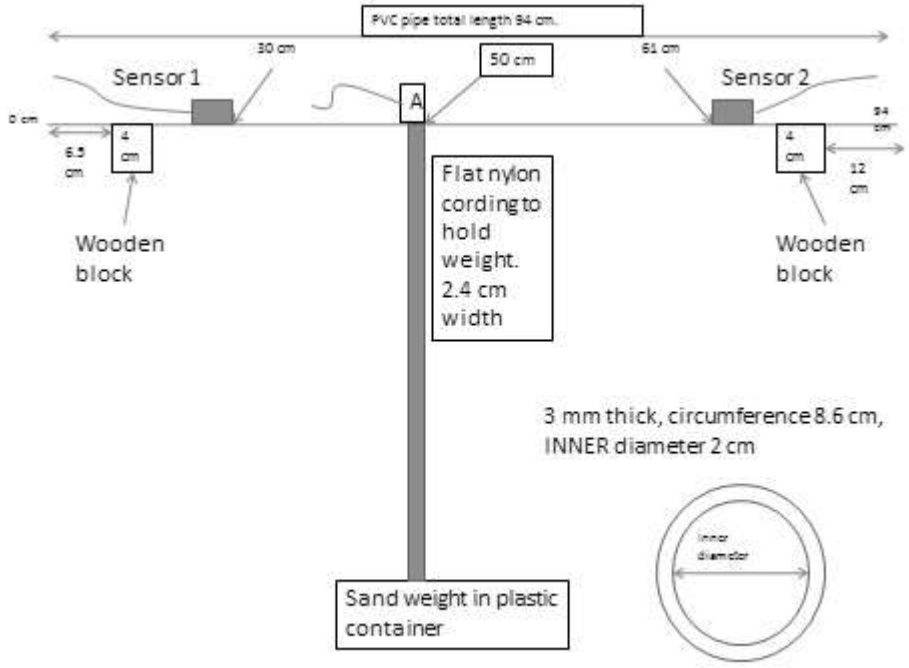


Figure 1.5. Setup to test linearity of the elastic beam formula with distances between sensors and the load along the PVC pipe. Sensors 1 and 2 represent two electromagnetic sensors used by Shum et al (2014). Sensor A was initially placed directly over the load.

This procedure was repeated with sensors 1 and 2 as well as the load remaining in the same position, while sensor A was placed 10 cm away from sensor 1, and then again with sensor A 23 cm away from sensor 1, as illustrated in Figure 1.6, to have additional data points for vertical displacement data.

Data analysis

A dual quaternion algorithm was used to represent displacement of a rigid body was used to plot the rotation in relation to the sensors, using the formula by Shum et al.(2013).

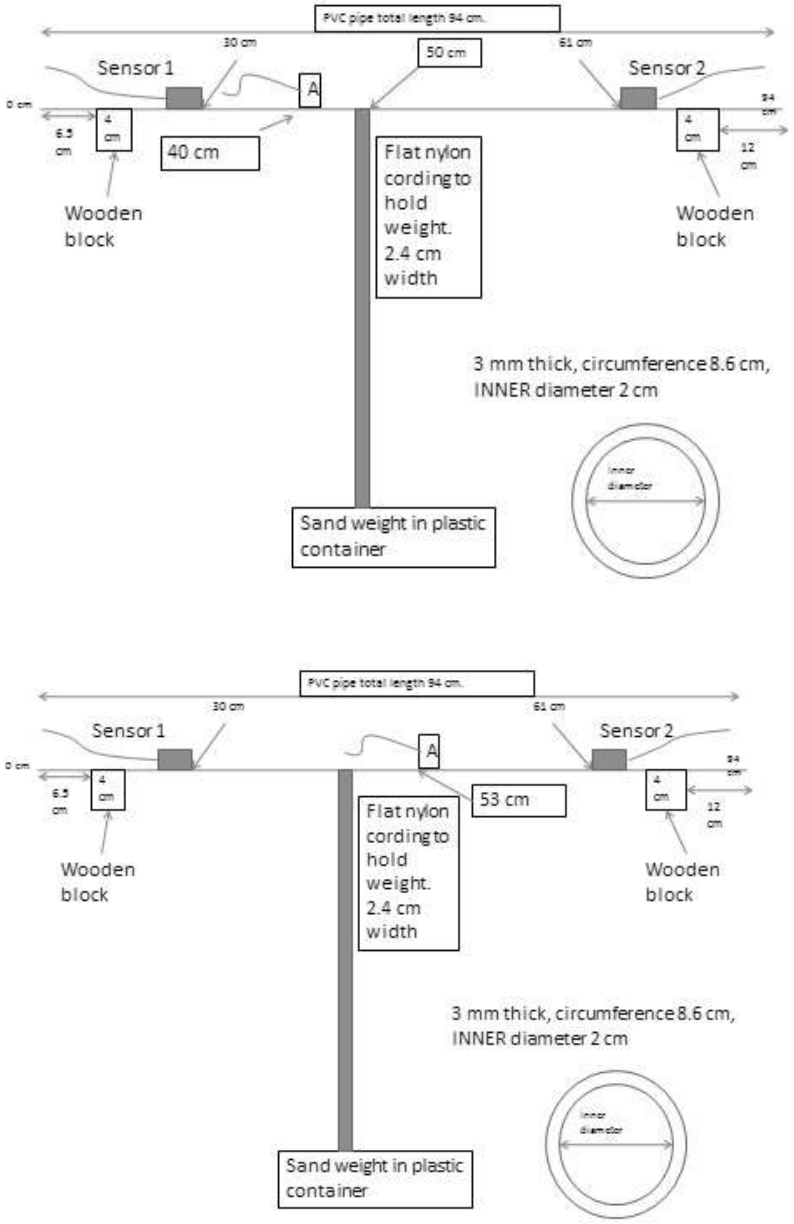


Figure 1.6. Depicts the movement of the middle sensor to varying distances from the load and 2 end sensors to determine linearity of the model and equation.

Results

There is a linear relationship between load and sensor rotation change, including with introduction of metallic compression load cell.

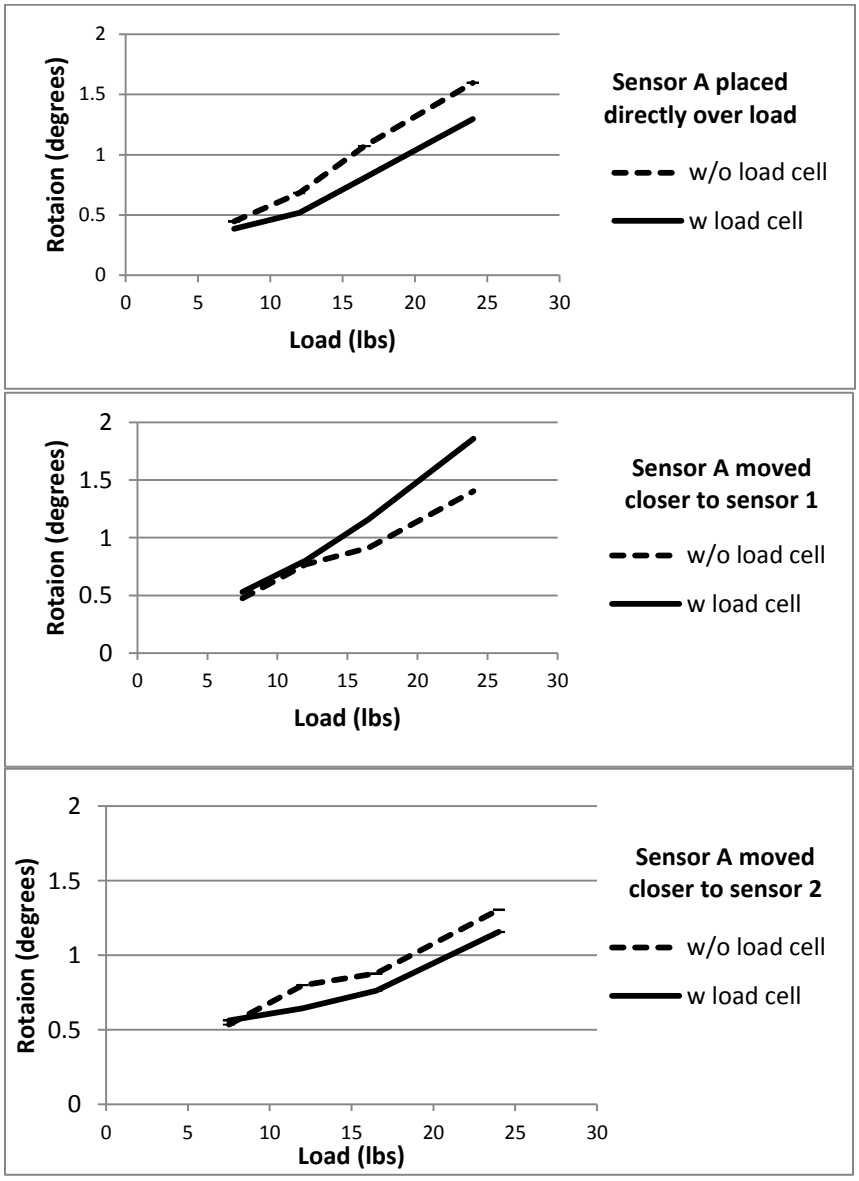


Figure 1.7. Plot of sensor rotation change over load.

Conclusions

The equations used by Shum et al (2013) were determined to satisfy the assumptions of linearity in an elastic beam, with minimal distortion (within measurement error of the instrument) introduced by the metal compression load cell. This allows for use of the equation in aim 2b for determining spine stiffness.

1.6.3 Determining the Ability to Measure Spinal Stiffness In vivo Using Currently Available Kinematic Equipment

In validating the clinical assumption of spinal stiffness during the PIT, as well as determining if electrically elicited muscle contractions can yield spinal stiffness changes, electromagnetic sensors will be used to track spinal segment displacement, and thus used to calculate spinal stiffness. The purpose of this preliminary study was to examine load to spine deformation relationship using current kinematic equipment in the lab and refine methodology in collecting and analyze this data.

Methods

A 38-year-old female subject with a history of recurring low back pain who was currently asymptomatic participated. She was placed in prone with the spine in less than 5 degrees of extension. Two electromagnetic sensors were placed at L1 and S2 as described by Shum et al 2013. A compression load cell (Transduce Techniques, Temecula, CA) was attached to a custom apparatus to measure the force applied during a posterior to anterior load applied to the spine (Figure 1.8). The subject was asked to exhale and hold her breath as a posterior to anterior (PA) load was applied to L3 at 22 Newtons (N) (5lbs). Two trials were administered 120 seconds apart. Real time visual feedback from the compressive load cell was provided on a computer screen to allow the

examiner to keep forces consistent between trials at ± 2.5% of the target load. This was repeated at 44 N (10lbs) of PA load with a 5-minute rest period between incremental load increases. Prior studies investigating posterior to anterior loads on the spine have utilized forces ranging from 50 to 200 N, equaling 11 to 45 lbs of force [86,135,136]. Twenty-two N and and 44 N were utilized in this study as loads were administered manually rather than a mechanical loading device, and 44N was the maximum load that could be manually applied consistently with visual feedback.

Figure 1.8. Apparatus attached to a compression load cell to apply anterior loads to the spine.

The subject was then placed in the testing position for the PIT. A wooden, non-height adjustable plinth was utilized to avoid electromagnetic field distortion. A set of stepping blocks were placed under subjects as necessary to place their ASIS at the height of the table. She was then asked to lie on the table such that the table would support the upper

body with legs extended off the table, supported by the stepping blocks (if necessary). Two trials of each load (22, 44N) were applied again to the spine at L3 using the same trials and rests as described above in prone. Once testing was performed in the resting position of the PIT, the subject was placed in the PIT testing position again, and a 24-inch high beam attached to a gate was placed above the calf. She was asked to raise the legs to the 24-inch height to standardize leg raising position. Prior to testing, pilot subjects were given the opportunity to be in the PIT testing position and perform the leg raise (hip extension. Several heights were trialed for the gate, but subjects tended to overshoot the target, which may impact sEMG analysis of the PIT in future aims of the study. Subjects were more consistent with reaching the 24-inch high gate without overshooting and so was used for this study. This was performed following an audible signal, and a PA load was applied at 22 N to L3. This was repeated for 2 trials with 2 minutes rest between trials, followed by 2, 44 N trials as in the above sessions. Complete protocol sequence is presented in Appendix C.

Data Analysis

Rotation of the L1 sensor about the S2 sensor was measured in degrees, as force was applied to the L3 segment. Bending compliance of the spine during load application was expressed as the slope of the load (y axis) against the angle change (x-axis). Stiffness of the spine was expressed as the inverse of the compliance (1/compliance slope). A regression line was also created to determine the line of best fit.

Results

Table 1.8 contains the compliance slope, stiffness index, and R^2 of the best fit curve during the 3 conditions tested under two different loads. There are strong

associations between the load and the angle change. However, the flexibility of the spine to load does not demonstrate a consistent change across the conditions. The subject did not demonstrate any increase in spinal stiffness using the current calculation of compliance and stiffness using slope values. Figure 1.9 depicts graphical plots of the load versus angle change. In the examples shown, there are 6 degrees of change to the anterior load in the prone and PIT resting position. There is a 3 degree change in the PIT leg raising position demonstrating reduction in angular motion between the L1 and S2 sensors, with leg raising. However, the stiffness index as calculated does not reflect a stiffness change.

Table 1.8. Compliance (slope of load versus angle change), Stiffness index (1/compliance) and R2 of the best fit curve. All plots fit a linear best fit curve.

	Prone				PIT Resting Position				PIT Leg Raised			
	22N		44N		22N		44N		22N		44N	
	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2	Trial 1	Trial 2
Compliance	5.7	3.9	6.8	4.7	3.5	6.2	4.6	4.7	4.8	7.6	6.8	4.7
Stiffness	0.17	0.26	0.14	0.21	0.29	0.15	0.22	0.21	0.2	0.13	0.14	0.21
R2	0.98	0.99	0.94	0.89	0.96	0.94	0.98	0.9	0.88	0.99	0.94	0.89

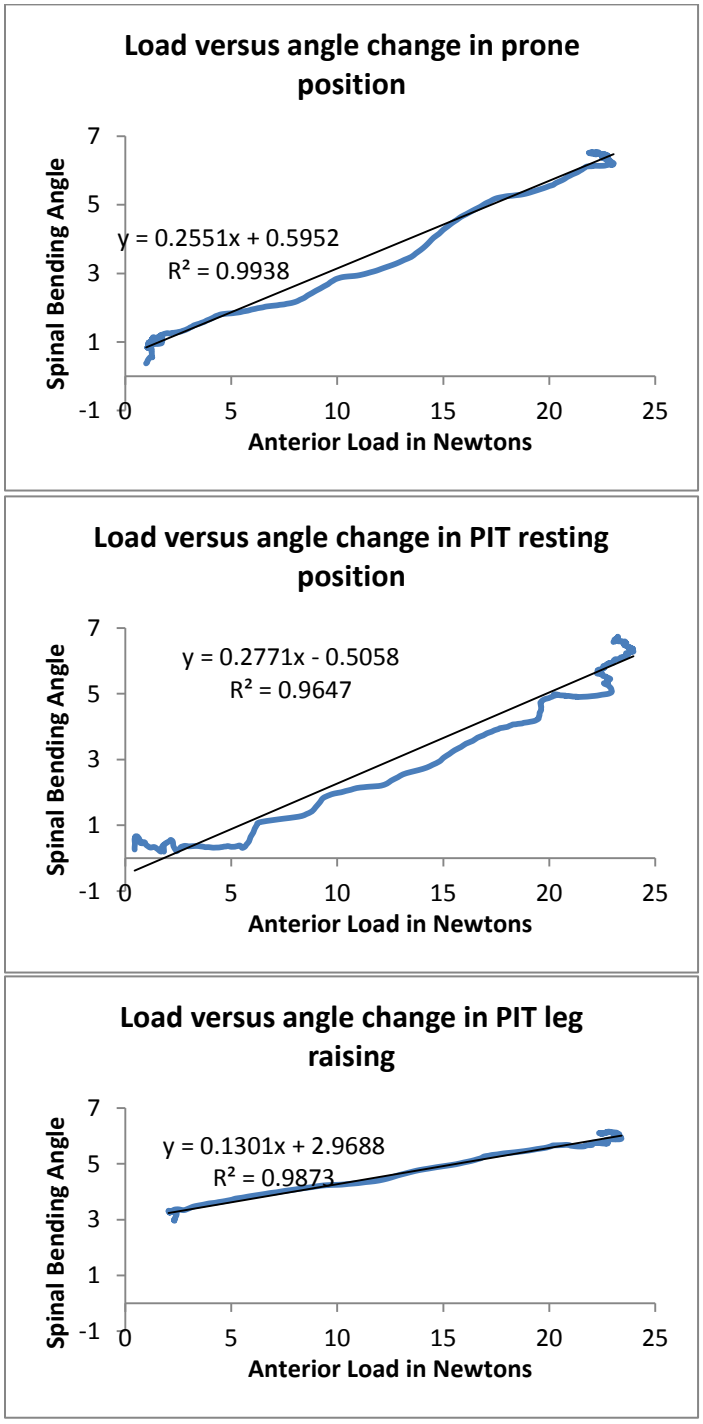


Figure 1.9.Example of scatter plot of angle change versus load in three different positions, along with associated r^2 values. Steeper slope reflects greater flexibility of the spine. In the Prone Instability Test, leg raising position, the angle starts at a higher baseline as the subject demonstrated an increased lordosis of the spine upon leg raising. The slope was calculated based on the angle of the spine and it's change, once the load was applied.

Conclusion

A subject with recurring symptoms who was currently asymptomatic was specifically chosen for this preliminary trial to provide the most variability that may be possible during the testing protocol. She would potentially offer challenges during the testing protocol that we may face in the future with patients who have low back pain, while having lower risk for symptom irritability as the protocol was piloted. She did not report any pain production during the testing but did report an overall irritation of her symptoms.

The plot of load to angle demonstrated a strong, nearly linear association between load and angle change, supporting the findings of preliminary study 5.2. It demonstrates our ability to obtain spinal compliance changes that occur during the testing. The paradoxical nature of increasing flexibility during the leg raising portion of the PIT of this subject may be due to her history of recurring pain and we are likely to have differing results with other subjects.

There is broad variability in spinal compliance throughout the testing conditions and loads applied. Scatter plots in some cases demonstrated changes in angle even as load was held fairly constant, demonstrating hysteresis. This is likely an effect of the rate of loading. In practice sessions prior to testing of this subject, greater hysteresis was seen when load was applied more abruptly, impacting the slope and stiffness values. This did decrease over time with tester practice, and will be a necessary motor skill that needs to be developed by the tester. Gradual loading appears promising for decreasing this effect of hysteresis. This may be the case as demonstrated in Figure 1.9. In the resting position, there is a 6 degree angle change from application of the load while the leg raising leads to

a reduction in the angular change once load is applied. This would appear to be a stiffness increase as there is less spinal bending. However, the slope may be impacted by the rate of loading and hysteresis. There may also be an effect of muscle guarding during the test that will affect the slope. Ensuring subject comfort will likely reduce this guarding. It may be possible to reduce this guarding through introducing the load application apparatus to the subjects while testing for painful segments. SEMG will also be collected during the PIT test. Therefore, monitoring of the sEMG signal during testing may also give the tester information of subject guarding and allow for coaching to relax.

Currently, this preliminary case study demonstrates the ability to use current equipment in the laboratory to collect and analyze the data necessary for the study. It is still necessary to run a smaller study to establish a more robust pattern of spinal mobility changes that occur with loading. We also need to establish instrumental and tester error to determine standard error of measure and minimal detectable change values necessary to interpret our findings.

1.6.4 Isolation of Lumbar Multifidus using NMES: Near Infrared Spectroscopy

The purpose of this preliminary study was to determine the ability for NMES to target the LM. Aims 2A and 2B require selected activation of the LM and the ability to fatigue the LM. Based on the surface anatomy in the lumbar region with several muscles located in a common area including LM and LES, a method was required to confirm isolated activity of the LM, with negligible activation of the LES. The use of both sEMG and indwelling EMG electrodes pose limitations, as the use of sEMG to confirm electrical activity in surrounding muscles would be tainted by skin conductance of the electrical signal from NMES and the limited sampling area of fine wire EMG cannot

adequately confirm electrical silence in surrounding areas. Near infrared spectroscopy (NIRS) offers advantages for studying muscle activation characteristics as NIRS is not distorted by NMES conductance and has been used in the past in conjunction with electrical stimulation to study muscle metabolism.[137] It has the potential to indirectly determine magnitude of muscle contraction through blood volume changes in a muscle[138] when referenced against EMG. It also provides insight into muscle physiology through comparing the ratio of oxygenated to deoxygenated blood.[139] Carefully placed sensors could provide the ability to detect and differentiate LM recruitment compared to other trunk extensors (lumbar and thoracic erector spinae), offering suggestions for electrode placement. Changes in the magnitude of blood flow volume and ratio of oxygenated to deoxygenated blood through iterative stimulations could potentially determine if NMES sufficiently overloads a muscle by using muscle fatigue parameters to determine appropriate prescription of NMES dosage.

The purpose of this preliminary study was to 1) determine how well NMES can be utilized to isolate LM activation; and 2) determine if NMES could result in fatigue of the LM via assessment of muscle fatigue and the relationship between muscle physiologic changes (NIRS) and electrical activity (EMG) across a range of activation levels.

Methods

Subjects

Subjects between the age of 18-50 were recruited. 12 healthy subjects (5 female) with average age 29 +/- 5, BMI 27.4, +/- 5.2 participated in the experiment. Subjects were a sample of convenience recruited from the University area through word of mouth and flyers. Exclusion criteria were current or a history of low back pain that limited function for greater than 3 days. This data was collected under Drexel IRB protocol number 1404002752 (see Appendix D).

Procedure

All subjects underwent skin preparation as mentioned in section 6.1. Pre-gelled (Ag-AgCl) disposable surface EMG electrodes (2cm inter-electrode distance) were applied to target muscles as listed in table 1.9. The reference electrode placed on the left lateral malleolus according to ISEK standards.

A continuous wave (CW) near infrared spectroscopy device with three separate probes was used to measure hemodynamic changes of the muscles (Drexel University Biomedical Engineering, Philadelphia, PA). Each NIRS probe had one light source and three detectors embedded into a foam molded square block. Two of the detectors were placed at 2.8 cm distance from the light source (far channels), and one detector at 1 cm from the light source (near channel). This selection leads to a penetration depth of up to 0.5 cm at the ‘near channel’ and up to 1.4 cm at the ‘far channels’ to measure the hemodynamic changes within superficial tissues -including the skin- for near channel and deeper layers –including muscle- for far channel. Probes were placed so the light source and the far channel (deep probe) would be 3 cm from the spinous process of S1-L3 for

LM, 5 cm lateral to spinous process of T12-L1 for LES, and 5 cm lateral to spinous process of T9-T7 for the TES (Figure 1.10). Real time ultrasound was used to identify superficial tissue (skin and fasciae) on 4 subjects upon completion of the study. Mean skin and fasciae depth was 5.4mm ±2.1, with muscle belly starting below that level. This would allow the far channel to have adequate penetration to the LM, LES, and TES.

Table 1.9 sEMG placements on the trunk during testing of trunk muscles

Muscle	**Location**	**Muscle**	**Location**
Gluteus Maximus (G. Max)	Midpoint between the lateral edge of the sacrum and greater trochanter	**Gluteus Medius (G.Med)**	5 cm posterior and 15 cm inferior to the midpoint of the iliac crest
Latissmus Dorsi (LD)	Midline between spinous process of T9 and axillary line	**Thoracic Erector Spinae (TES)**	5cm lateral to T9 spinous process
Hamstring (HS)	15cm from the ischial tuberosity	**Lumbar Erector Spinae (LES)**	3cm lateral to L2 spinous process
Transverse Abdominus/Internal Oblique (IO)	2cm below and medial to ASIS and above the inguinal ligament	**Lumbar Multifidus (LM)**	2cm lateral to L5 spinous process

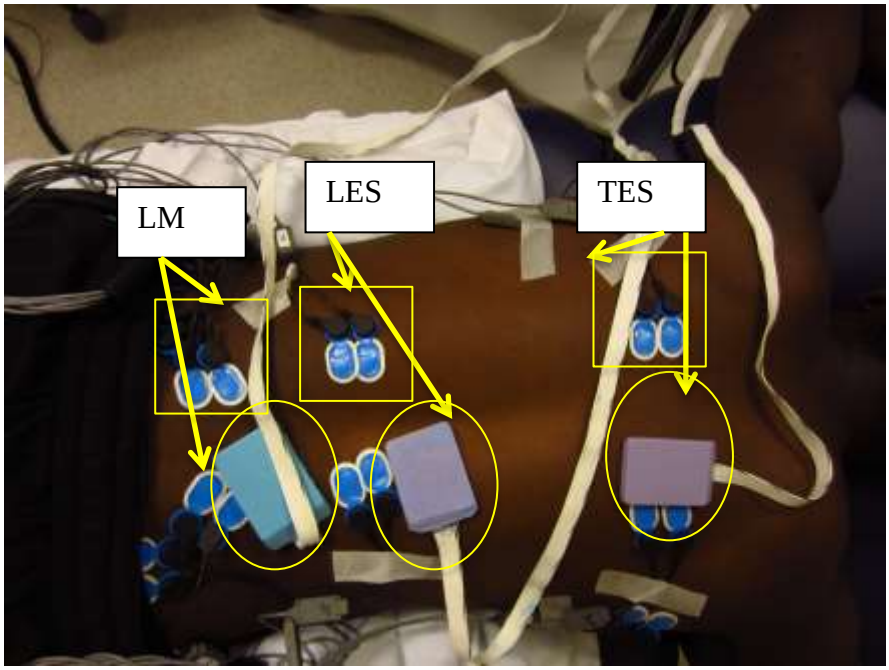


Figure 1.10. Surface EMG and fNIR setup. SEMG are surrounded by the square, while fNIR probes are highlighted by circles.

NIRS raw optical intensity data were recorded at two wavelengths (730 nm and 850 nm) with a sampling rate of 2 Hz. Similar collection rate and light source to detector distances have been used to collect blood volume in the erector spinae [140]. To remove environmental and physiological irrelevant data (subject movement artifact, respiration and heart pulsation effects), a low-pass filter with a cut-off frequency of 0.14Hz was applied to the raw optical data.

SEMG signals were normalized using a modified Biering-Sorensen test, single leg bridge bilaterally, and a curl up. Subjects performed a modified Biering-Sorensen test [141] while lying prone over the edge of a plinth. The head of the plinth was angled downward at 20 degrees with the ASIS aligned with the break of the plinth. The subjects laid with

their arms across the chest in a flexed position with the upper body resting on the angled head of the plinth with their calf and pelvis strapped to the table. They were asked to raise their trunk to neutral at an audible signal. They performed three trials for 10 seconds, to obtain a steady submaximal volitional isometric contraction (sMVIC) for the trunk extensors. Three trials for 10 seconds were performed for this test as it was also used for the preliminary study with NIRS. Two repetitions of a 5 second single leg bridge were performed bilaterally to obtain sMVIC measures for the G.max and HS. Curl up was performed with the subjects lying in a hooklying position with their backs reclined to a 45 degree angle. They were asked to lift their backs away from the support and hold for 5 seconds. This was repeated for 2 trials. These were performed for data to be used in preliminary study 6.6.

In a fully supported prone position, they then performed the multifidus lift test (MLT) with the dominant arm [142] under the no load (MLTN) and high load conditions (MLTH) [143]. With the upper body supported on a plinth they performed a bilateral prone leg lifting task adapted from the prone instability test (PIT) [35]. Each test was performed for 5 trials and held for 10 seconds with 30 seconds rest between trials. EMG and NIRS were collected simultaneously 30 seconds before the test (rest phase) and during the test maneuver. These tasks represent different levels of trunk extensor activation, which allowed for multiple comparisons of EMG and NIRS and established maximum voluntary isometrics contraction (MVIC) for EMG normalization.

After test movements were completed, EMG electrodes were removed and 5cm x 5cm carbon foam stimulating electrodes were placed 1cm from the L5-L2 spinous process bilaterally. NIRS sensors were left in place (Figure 1.11). In certain cases,

stimulating electrodes were cut to remain within the muscle borders of the LM. Subjects were placed in the prone position with less than 10 degrees of lumbar extension, a fixation belt was placed across the pelvis to limit anterior pelvic tilt, and NMES was administered until a visible anterior tilt was observed. NMES was administered using a clinical device (EMPI Continuum, Minnesota, USA) at 35Hz, 400us pulse duration, 1 second ramp, with 10 seconds on time and 50 seconds off time for 10 contractions. Subjects were instructed to relax and not contract any muscles during the stimulation.

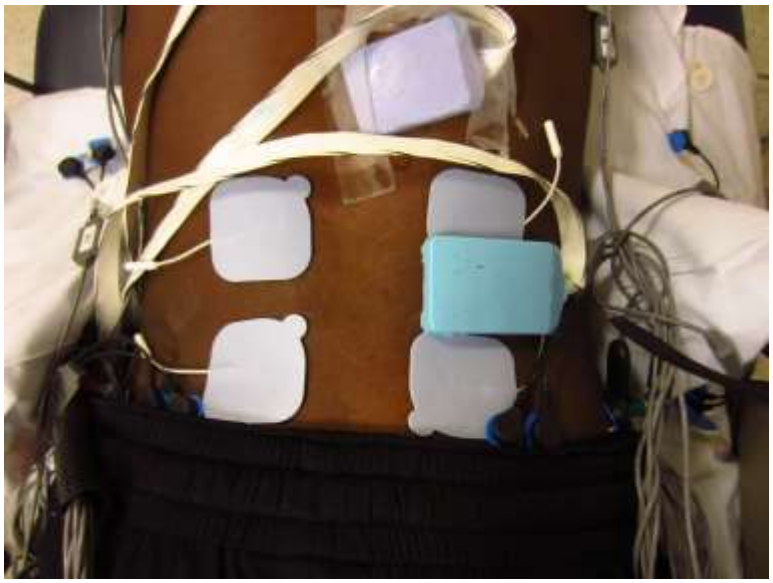


Figure 1.11. Set up of NMES and NIRs probes during stimulation of LM. Square NMES stimulating electrodes were placed bilaterally along the LM, while the rectangular fNIR probes were on the right side of the subject. SEMG electrodes have been removed from the subject.

Data Reduction

SEMG:

All sEMG signals were heart rate stripped using a fast independent component analysis (ICA). Data were then low pass filtered (20Hz; RMS filter) and downsampled to match the NIRS data collection rate using a custom LabView program (v8.6, National Instruments, Austin, TX). Average mean amplitude of 2 trials was determined over a 2-second period once steady state activation was reached. This approach provided the most stable value. Trunk extensor muscle sEMG activations were normalized to the MVIC produced during the trunk extensor test.

NIRS:

Change in absorbency of the 730 nm and 850 nm light source by hemoglobin are used to determine the level of oxygenated blood (HbO) and deoxygenated blood (Hb) using a logarithmic ratio of detected light during the resting phase, and comparing that to the activity phase. The ratio provides a unit less ratio of rest to activity that is referred to as arbitrary units (AU). The logarithmic ratio is used to account for the decay of light that occurs as it travels from its source. The AU was set to 0 at rest through subtraction of the resting levels from the time series, and represent change in HbO2 and Hb during the activity compared to the baseline. Total blood volume is the sum of HbO2 and Hb.

Data Analysis

Heart rate stripped sEMG amplitudes normalized to the Modified-Sorensen (sMVIC) and NIRS variables were time synchronized to data from all testing conditions. Each subject had Pearson correlations performed within tests between sEMG and NIRS variables using time series data stream 10 seconds before the test (rest) and the 10

seconds during the test. This was done to determine if within individuals, NIRS and sEMG variables were associated with each other: how accurately does change in sEMG amplitudes reflect changes in NIRS variable. This was also done to determine which channel's data would be used in group analysis, since there were two far channels and one channel may better represent blood volume changes at the muscle targeted by sEMG electrodes. The NIRS far channel that had the highest correlation was then used for group comparisons. Pearson correlations of group sEMG amplitudes and NIRS variable were performed and used to determine variables for linear regression. Results from the linear regression were used to predict a percentage of the sMVIC activation during NMES across 10 subjects. Two subjects' NMES data were not entered into analysis due to potential data corruption of the NMES NIRS files, detected in post processing. A paired t-test of the predicted percent sMVIC of the 1st and 5th NMES stimulations were performed to determine if NMES could provide adequate dosage to overload/fatigue the LM to achieve therapeutic benefits. ANOVA of mean LM, LES, and TES HbO2 AU over the 10 second NMES stimulations were performed to determine its ability to isolate activation of LM.

Results

Individual within subjects absolute correlation values were high, for all muscle groups' sEMG amplitudes vs HbO2, Hb, and blood volume. These ranged from .71 to .97 individually, between tests. However, for LES and TES, there was great variability in the direction of the correlations across all conditions. A negative correlation would mean a reduction in a NIRS variable in relation to sEMG increase. When negative correlations occurred in an individual, they occurred in conjunction with reduction in total blood

volume within that trial. This only happened in 8 of 100 cases in LM during the MLT. However, during the PIT and Modified Biering Sorensen tests, tests that required more movement of the lumbar on pelvis compared to the MLT, LM also demonstrated highly negative correlations. An example is provided in Table 1.10.

Table 1.10 Example of highly negative correlation coefficients between LM and LES EMG between 5 trials within a subject.

	EMG vs NIRS variables for LES during MLT, unweighted condition			EMG vs NIRS variables for LM during PIT condition		
	HbO2	Hb	Total vol.	HbO2	Hb	Total vol.
Trial 1	-0.90	-0.90	-0.90	-0.83	-0.82	-0.82
Trial 2	-0.95	-0.94	-0.95	-0.84	-0.77	-0.82
Trial 3	-0.98	-0.95	-0.97	-0.83	-0.78	-0.82
Trial 4	-0.78	-0.82	-0.95	-0.77	-0.84	-0.71
Trial 5	-0.98	-0.99	-0.99	-0.86	-0.90	-0.87

Since the change in direction of correlation was present in LM with activities that were potentially more challenging, the relationship between direction of correlation and EMG variables was further analyzed. NIRS vs sEMG correlation directions (+ vs -) entered into a point biserial correlation versus sEMG amplitudes to determine if there was an effect of sEMG amplitude, amplitude of the muscle recruitment, on differences in blood flow. There were no significant correlations, among LM, LES, nor TES for amplitude of muscle recruitment across variables, with r ranging from .08 to .24. This was not investigated further due to the lack of association between magnitude of muscle recruitment and changes in blood flow. As blood flow values did not appear to be impacted by the magnitude of the recruitment but perhaps amount of movement allowed

to happen in the area of the muscle, the impact of reduction in blood flow could not be well accounted for. Therefore, LM sEMG and NIRS variables during the MLT conditions that proved to have stable correlation directions were used to predict muscle activation during the NMES.

In group correlations of sEMG and NIRS variables, LM HbO2 vs LM sEMG yielded the highest correlations ($p<.05$, $r=.81$). This is supported by prior studies that establish HbO2 to be most reliable during muscle activity [144]. Plots of these variables against each other demonstrated a linear best fit line; therefore, HbO2 and sEMG amplitude of the LM were entered into a linear regression. Linear regression revealed a significant relationship between sEMG amplitude and HbO2 (Beta=.269, $p<.001$) with an overall model fit of $Y=0.269x-0.0000332$, adjusted $R^2=.62$. This formula was used to predict the percentage of sMVIC during NMES for trials 1-5, using HbO2 values. This is represented in Figure 1.12. Individuals produced as much as 100% of the sMVIC in the first trial of NMES and as low as 8% of the sMVIC by the 5th trial. There was a significant difference between the 1st and 5th trial of NMES to the LM, $p<.05$.

Based on high within subject correlation coefficients between LM, LES, and TES sEMG to NIRs variables, it was determined that the NIRS sensors at the LES and TES would likely give good representations of muscle activity during NMES. LM demonstrated significantly greater HbO2 levels (mean=1.15 AU ± 0.8) compared to the LES (mean=0.3 ±0.8) and the TES (mean=0.2 ±0.45), $F(1,39)=39.21$, $p<.001$. Figure 1.13 is an example of the difference between LM, LES, and TES HbO2 from rest to NMES stimulation.

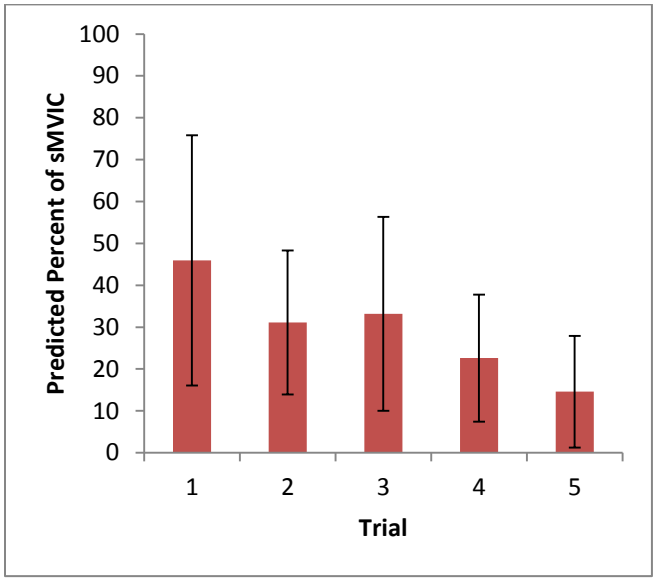


Figure 1.12 Average predicted percent of s MVIC from NMES across 5 trials.

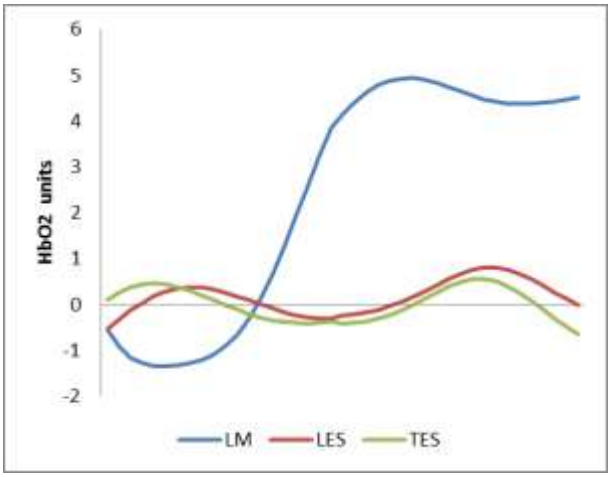


Figure 1.13 HbO2 levels of LM, LES, and TES from rest to NMES stimulation.

Conclusion

The linear relationship of HbO2 to sEMG activity allows for prediction of muscle recruitment using HbO2 measures during NMES, at least during submaximal contraction. While the NMES achieves less than 50% of sMVIC, the long on time of ten seconds likely ensures that the muscle is achieving overload and fatigue, as represented by the reduction in HbO2 by the 5^{th} NMES stimulation. This suggests that it should be able to adequately fatigue the LM to test aim 2B. In carrying on with the proposal, it may be beneficial to increase NMES on times to 15 seconds and go through completion of 10 NMES elicited impulses to assure fatigue of this muscle and limit its ability to contribute in the forward bend.

The benefits to NIR are that it appears to be able to determine if there is muscle activity occurring. There are overall strong correlations with blood levels and sEMG activity. As sEMG activity occurs, there is some change in blood physiologic characteristics. In regards to the direction of the correlations, this may have to do with movement of the muscle during the task. In the NMES condition, the pelvis was stabilized to allow an isometric anterior pelvic tilt. During the MLT conditions, there was fairly minimal movement occurring between the pelvis and the lumbar spine likely resulting in minimal shortening of the LM during the contraction. However, in several subjects, there were visible contractions of the LES and TES as well as movement in the thoracic spine during these movements. In these conditions where the muscle is allowed to shorten during the contraction, overall blood flow may be constricted due to the contraction. Even in the literature, there is discrepancy in blood flow characteristics with NIR testing with some studies demonstrating increase in blood flow along with increase

is HbO2 [140,145] but others seeing reduction in blood flow and a plateau in HbO2 [144]. High correlations, within individuals but changes in direction of the correlation would suggest that there are still several factors that need to be considered in future testing of this manner. However, it does suggest that the NIRS sensors are reporting some activity in relation to the sEMG amplitude, based on the high correlation values. What can be assumed from the current results are, that any change in the NIR value may be indicative of muscle activity, and flat line blood physiologic characteristics that vary minimally from resting values can be interpreted as muscle silence. With this in mind, the differences noted between the LM, LES, and TES in HbO2 characteristics are likely very representative of activity occurring at the LM, and not at the LES and TES with the use of NMES.

Overall, for the purpose of this proposal, it appears that NMES provides the ability to isolate activation of the LM with minimal activation of the LES and TES, and is able to obtain muscular fatigue, to provide a "knockout" model of the LM.

1.6.5 Isolation of the Lumbar Multifidus using NMES: Rehabilitative Ultrasound Imaging and pre-post extensor strength test

The purpose of this pilot study was to confirm the results of the NIRS in detecting isolated activity of the LM during NMES elicited contraction using rehabilitative ultrasound imaging (RUSI). RUSI has been found to be a reliable method to identify muscle thickness changes and activation [146-148]. The aims of this study were to investigate the ability to isolate muscle activation to the LM during NMES elicited contraction. It was also designed to determine if NMES could fatigue the LM as demonstrated by changes in RUSI characteristics of contraction time during NMES and strength reduction

following NMES due to LM fatigue. It was hypothesized that isolated contraction to the LM would yield minimal changes to the LES during NMES stimulation. It was also hypothesized that fatigue to the LM would lead to decrease in muscle thickness change during the contraction, lower contraction time as the muscle fatigues and loses contractile ability. It was also hypothesized that fatigue of this muscle group would lead to force reductions of the trunk extensors when compared to pre and post NMES.

Methods

Five healthy subjects from a sample of convenience, age=25.6 ±1.1, BMI: 22.9±3.1 with no history of low back pain participated in this study. The examiner underwent 12-hours of training with RUSI for the trunk, and has been utilizing RUSI in patient care for 2 years. He has been utilizing NMES to the lumbar spine in treatment of patients with low back pain for 13 years.

Procedure

Subjects first performed a Modified Biering-Sorensen test as described in section 5.4. They were asked to hold the position while a hand held dynamometer was used to measure trunk extensor strength with resistance applied at the T7 spinous process, using an isometric break test, for two trials (Figure 1.14). MDC_{90} of 3.7 lbs. using this procedure had been established for a prior case study on NMES. Following strength testing, subjects underwent measurement of LM and LES using RUSI (Mindray, MSK50 Shenzhen, China) with a 2-6Hz curvilinear array probe during administration of NMES to the LM.

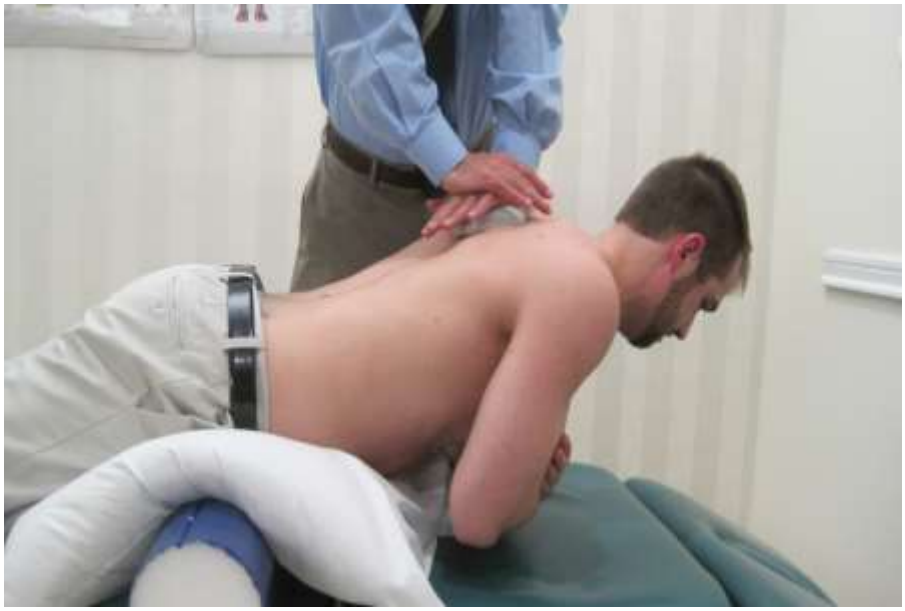


Figure 1.14 Trunk extensor strength testing with hand held dynamometer

Motion mode (m-mode) ultrasound was used to detect both onset and offset of contractions while also measuring thickness change of the muscle. M-Mode presents ultrasound data as a time series. As changes occur in the muscle fascicles, they are presented as grey scale disturbances along the time series. It also presents a brightness mode (b-mode) image above the time series typically associated with RUSI that can be used to measure thickness changes of the muscle (Figure 1.15). Muscle onset and offset has been found to be detectable using visual inspection in m-mode [149], and is useful in determining muscle onset when thickness change in b-mode may not be obvious [150]. Although m-mode has not been found to be a reliable method for detecting onset times [151], these errors ranged from 16 to 22ms. When analyzing muscle activity that takes multiple seconds, such as the duration of the contraction, this type of error may be a negligible factor.

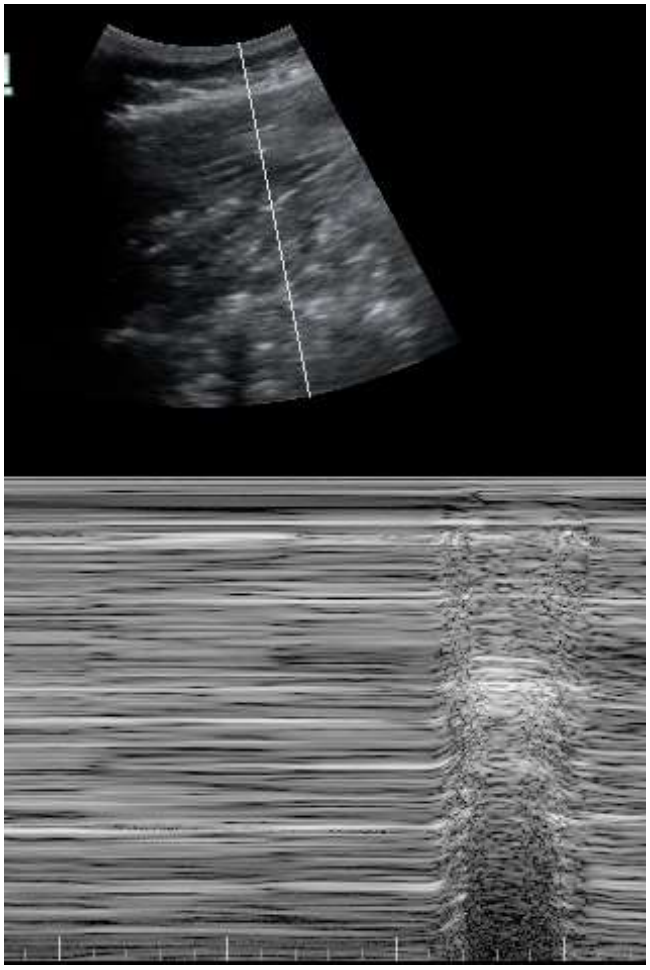


Figure 1.15. M-Mode image of lumbar multifidus during NMES.

Subjects were placed in a prone position with setup of electrodes as described in section 6.4 with electrodes cut to size to stay within the boundaries when necessary. To determine if NMES could isolate contraction to LM, subjects initially received 6 short length contractions of 20Hz, 400us, 3 seconds in duration which was triggered manually. Three seconds was chosen to minimize fatigue of the muscle during this phase as it was the shortest stimulation time that could be used with this stimulation unit, while still ramping stimulation. Ramping electrical stimulation was necessary to avoid gross

contractions of the trunk from subjects' guarding response resulting from a sudden onset of electrical stimulation. LES was measured using a longitudinal approach as described by Watanabe, et al. (2004) with the probe positioned within 3-5 cm of the spinous process. Location of the probe was determined for each subject using a transverse oriented view of the muscle, and looking for the fasciae boundary. Once the LES was found, the probe was turned to a longitudinal orientation so that the probe was between L2 and T1. NMES was triggered manually and m-mode video was captured for that time sequence. This was repeated for three trials to obtain 3 separate m-mode clips. This process was then repeated to capture LM with the probe positioned just above S1, lateral to the spinous process to capture the facet joints and the LM. RUSI image capture of the LM was done in a manner to first identify L5 and S1 based on the location of the sacrum and then the ultrasound head was moved caudally so that the L5 facet joint was in view without the sacrum. This was done to *minimize* examiner bias of the LM vs LES during image processing post collection, through identification of the sacral border. Bias could not be *eliminated*, as the transverse process captured in LES is visually different from the facet joints captured with LM. The RUSI beam was pointed directly downward, in the area of L4 for LM and L1 for LES.

To measure the ability of NMES to fatigue the LM, subjects received 2 manually controlled, 15 second, 400us, 50Hz NMES stimulations with concurrent measurement of m-mode RUSI at LM, with 50 seconds rest between. They then received 6 automated stimulations using the parameters above, with 15 second on time and 50 second off time. After 6 automated cycles, subjects received 2 manually controlled stimulations with concurrent measurement of m-mode RUSI. The sequence of manual versus automated

delivery methods was to allow the examiner control over the delivery of the stimulus for image capture. Following NMES, subjects were placed back into the Modified Biering-Sorensen position and re-tested with two trials.

Measurements

Images were saved as video files and converted to 24bit grey scale for analysis offline through imageJ (Wayne Rasband, National Institutes of Health, USA). Video files were assigned an 18 number, 3-letter identifier by the RUSI that made distinguishing files difficult. Determining contraction durations during NMES required knowledge of when a true contraction occurred. Studies to date have been concerned with onset times using computer analysis of the frequency components of the image [150,151,153]. Therefore different methods needed to be developed for this pilot study. Full descriptions of the methods to determine contraction duration are outlined in Appendix E. There were subtle differences that were noticeable between the LES and LM due images due to the different appearance of facet joints versus transverse processes. Therefore, the top of the screen was obscured at the time of the rating (with exception to the menu bar at top of the screen) using paper and tape. The first measurement that occurred was with m-mode grey scale histogram analysis as defined in Appendix E for contraction duration. This was done to block the b-mode view. Once this was completed, the blinder was flipped to confirm contraction with b-mode. There were two ratings, 1 week apart, to establish test-retest reliability.

Muscle thickness was only measured if a contraction was confirmed and there was a contraction duration. Muscle thickness change was performed as described by others: from the tip of the facet joint to the inner edge of the fasciae over the muscle for

LM [143,154] and from the tip of the transverse process to the inner fasciae edge for LES [152]. Bias was unavoidable with this process. However, only 1 subject had a detectable contraction in the LES that required thickness limiting this source of bias. For the other subjects, there was 100% agreement between the first and second blinded rating of the LES in m-mode for the absence of contraction. As the purpose of this pilot study was to determine if LES contraction occurred, the first step of identifying a contraction through m-mode grey scale analysis that could be blinded, was of more importance. LES measure was planned mainly for descriptive purposes. Statistical comparison was initially planned to compare contraction durations of the LM and LES during the 3-second contraction. However, only one subject had detectable contraction at LES, so descriptive statistics (Mean, SD) were performed.

To determine the ability of NMES to fatigue the LM, paired t-test with bonferroni correction of LM contraction duration and muscle thickness change during the first and second, ninth and tenth contractions were planned. The first and second trial values were to be averaged and compared against the values for the ninth and tenth trial.

Test-retest reliability

Test retest reliability was determined for muscle thickness change during the short contraction duration (short impulse=3 second impulse) and the long contraction duration (long impulse=15 second impulse) using $ICC_{(2,1)}$. ICC was then used to calculate standard error of measure (SEM) and minimal detectable change with 90% confidence (MDC_{90}). The two contraction durations were calculated separately due to the large difference in contraction times that would skew the SEM and MDC. These were used to determine if

measurement differences across conditions exceeded rater error and if changes were meaningful.

Results

Reliability

ICC, SEM and MDC_{90} values are presented in Table 1.11. ICC value for muscle thickness was negative; therefore other variables could not be calculated. There were large differences in muscle thickness change ratings between the first and second readings. Percent difference between the first and second ratings was performed, to determine the differences. Average percent difference across subjects for rating 1 and 2 were 74%. Based on this, further comparisons of muscle thickness change were not made as planned, due to the likelihood of large error in measuring this variable.

Table 1.11. ICC coefficients, SEM, and MDC. Time in seconds (s)

	$ICC_{2,1}$	SEM	MDC
Contraction duration (short)	0.88	0.4 s	0.9 s
Contraction duration (long)	0.92	0.1 s	1.9 s

Isolation of LM with NMES

Based on m-mode ratings, only 1 subject was identified as having an LES contraction. Across 3 trials, this subject's mean contraction duration with the short impulse was 0.9 seconds (sd=0.2). The mean contraction duration of the LM during this phase for subjects across 3 trials was 3.6 (sd=0.4). While statistical inference cannot be

made, the contraction duration difference between LES and LM exceed the MDC established for the short duration impulse.

Fatigue of the LM with NMES

There was a significant difference in contraction duration between the first and second contraction (mean=15.5 seconds, ± 0.4) compared to the ninth and tenth contraction (mean=11.1 seconds ± 2.3), p=.018. The mean difference of 4.4 seconds exceeds the MDC_{90} for contraction duration. There was also a significant reduction in trunk extensor strength before the NMES (mean=40.9lbs ± 10.1) and following the stimulation protocol (mean=30.8lbs± 7.6), p<.001.

Conclusion

The method appears to support the ability of NMES to isolate contraction of the LM with minimal involvement of the LES. The method above using a combination of m-mode and b-mode ultrasound potentially gives investigators a method to determine contraction onset and contraction times during NMES while also measuring LM thickness changes with NMES. However, this was a sample of convenience using young, healthy individuals with good muscle quality. The good echodensity of the muscles likely played a large role in obtaining many of the measures above. Determining muscle onset and offset times was a function of pixel grey scale. Therefore, it is uncertain if this method will be as reliable or valid when applied to individuals with poor muscle quality where echodensity may be an issue.

The inability to detect thickness change may be that the NMES does not provide stimulation to a recruitment threshold where cross sectional area change is observable. It may also have to do with the method the images were obtained. Within m-mode, the b-

mode image is smaller therefore the b-mode image is compressed with resulting loss in resolution. To obtain measurable thickness using imageJ, the image had to be expanded leading to pixilation of the image that made distinguishing landmarks difficult. This increased the source of error for measurement. From the current data, it appears that NMES can isolate the LM and generate fatigue as seen by reduction in contraction times and trunk extensor strength. It appears to be able to isolate LM based on m-mode characteristics comparison between the LM and LES. If thickness change associated with NMES is to be studied in greater detail, it should be done separate of m-mode. It should also be performed on separate sessions with adequate rest between sessions so that true indicator of what happens between the first and last NMES elicitations can be gathered.

1.6.6 Preliminary Analysis of Muscle Activity During the Prone Instability Test

The purpose of this pilot study was to obtain descriptive information about trunk extensor activity during the prone instability test. This was part of a larger pilot study to determine the ability of NMES to isolate lumbar multifidus activity (preliminary study section 6.4), and was collected to aid in approximating the percentage of voluntary isometric contraction that NMES is able to elicit. The current sEMG setup used in the forward bend listed in table 1.7.1.1 may over represent the abdominal muscles with inadequate representation of the lower quarter, based on movements during the PIT. This secondary analysis of data collected during study 5.4 would aid in identifying other muscles that may be recruited during the PIT and help determine muscle selection for sEMG in the proposed study.

Methods

Subjects

Subjects between the age of 18-50 were recruited. 12 healthy subjects (5 female) with average age 29 +/- 5, BMI 27.4, +/- 5.2 participated in the experiment. Subjects were a sample of convenience recruited from the University area through word of mouth and flyers. Exclusion criteria were current or a history of low back pain that limited function for greater than 3 days. This data was collected under Drexel IRB protocol number 1404002752 (see Appendix D).

Procedure

All subjects underwent skin preparation as mentioned in section 6.1. Pre-gelled (Ag-AgCl) disposable surface EMG electrodes (2cm inter-electrode distance) were applied to target muscles as listed in table 1.7.4.1 with the internal oblique/transverse abdominus muscles representing the abdominal group. The reference electrode placed on the left lateral malleolus according to ISEK standards.

SEMG signals were normalized using a modified Biering-Sorensen test, single leg bridge bilaterally, and a curl up as described in section 6.4 and the PIT leg raising task was administered prior to the delivery of NMES. Unlike the preliminary study described in section 6.3 (which was performed prior to study 6.4) there was no standardized height for leg raising during this test.

Data Reduction and Analysis

Two subject's PIT data were not analyzed due to equipment malfunction, one detected prior to testing and one detected in post processing of the PIT data. Data were

sampled at 2400 Hz, passed through a pre-amplifier at a gain of 500, and band pass filtered at 20-500Hz. Two additional subjects had a large noise to signal ratio of bilateral G.Med that could not be resolved in post processing, therefore, G.Med was not included for these two subjects. Raw EMG signals were heart rate stripped using fast ICA algorithm. Mean maximum amplitude of each muscle was calculated over 2 seconds of data starting when the subjects reached a steady state of muscle activity during the modified Biering-Sorensen test , single leg bridge, and the curl up. Average mean amplitude of 2 trials was determined over a 2-second period once steady state activation was reached. This approach provided the most stable value. For reduction of the PIT, an 8 second average (2 seconds to achieve steady state) of muscle activity was calculated for all muscles, using an RMS smoothing filter with a 30ms time constant. Once completed, all muscles were normalized to the sMVIC. Muscle activity was expressed as a percentage of the sMVIC. Univariate repeated measures ANOVA were performed for muscle groups, at an alpha of .05 for the 1^{st} and 3^{rd}, 1^{st} and 4^{th}, and 1^{st} and 5^{th} trials to screen if there was an impact of fatigue based on the response of the 3 subjects that did not complete 5 trials. There were significant differences for most muscles at the 4^{th} and 5^{th} trials. Therefore, the first 3 trials of the PIT were used for analysis.

Left to right muscle comparisons were made for each muscle group using a within subjects ANOVA with 8 factors (8 muscle groups) and 2 levels (left vs right) with each trial entered as a separate case per subject. Before analysis, data were screened for normality. One subject had a right G.Med that was 3 times the sMVIC and was an extreme outlier, therefore, this muscle group (left and right) was excluded from side-to-side symmetry analysis for this subject. Once excluded, the data met normality

distribution using Kolmogorov-Smirnoff tests. There was a significant effect for side-to-side difference between muscle groups ($F_{8,6}$)=32.13, p<.001. Univariate comparison following this analysis revealed that G.Max was the only significant factor, ($F_{1,13}$)=19.1 p<.001 with no other factors were significant for side-to-side differences. F-values ranged from 1.2 to 2.8 for all other groups. Because no other groups had a side-to-side difference with the omnibus being driven by 1 muscle group and the main purpose to gain an understanding of muscle contributions during the test for electrode placement selection, it was decided to average sides together for final analysis.

Left and right sides of the muscles were averaged, and trials collapsed. Descriptive statistics (mean, SD) for the percentage of sMVIC are presented in Table 1.12. SEMG percent contributions during the task, derived from % sMVIC were then compiled for each subject where the muscle of interest's contribution (MM) was calculated as MM= (MM%sMVIC/∑MM%sMVIC)x100 where ∑MM%sMVIC was the sum of all muscles percent activation of the sMVIC, and MM%sMVIC was the muscle's percent activation of sMVIC. These results are presented in Table 1.7.6.2

Results

Table 1.7.6.2 demonstrates the range of variability across muscle activation by subjects during the PIT. Subjects 2, 3, and 4 have fairly even distribution of muscle activity across the muscles. However, other subjects demonstrate a preference to the TES or HS during the test. No subject demonstrated LM as the highest activating muscle group. The IO appears to have negligible activation during this test. The TES, LD, and HS appear to activate at a higher percentage of the sMVIC than other muscle groups with

larger standard deviations (Table 1.13), which suggest that healthy control subjects have a varied strategy of activating these muscles during the test.

Table 1.12. Group mean and standard deviation of the percent sMVIC for muscles during the prone instability test

	Mean	SD		Mean	SD
IO	9.2%	10.9%	LM	63.3%	22.9%
TES	110.8%	62.7%	G.MAX	27.8%	11.6%
LD	87.6%	93.1%	G.MED	56.0%	54.3%
LES	70.9%	25.0%	HS	90.3%	76.1%

.

Table 1.13. Percent contribution of muscles during the PIT. Muscles with the highest activation ratio per subject are bolded. * Subjects with high noise to signal ratio of G.Med, with muscle excluded from analysis.

Subject	**IO**	**TES**	**LD**	**LES**	**LM**	**G.MAX**	**G.MED**	**HS**
1	0.19	11.38	10.26	16.23	16.60	2.43	9.89	**33.02**
2	1.30	**31.99**	7.51	13.73	24.48	9.07	*	11.92
3	0.81	19.06	1.34	15.44	21.07	11.68	**20.54**	10.07
4	0.13	18.92	7.94	**27.74**	18.92	4.54	3.15	18.66
5	1.06	**44.29**	9.57	12.35	11.88	6.17	9.88	6.94
6	0.47	10.43	15.88	**25.36**	14.34	9.12	7.82	16.82
7	1.34	6.60	18.14	10.32	9.29	2.56	23.27	**28.59**
8	4.34	14.70	**42.16**	5.51	3.63	2.01	3.17	24.48
9	4.33	**38.48**	15.43	13.83	6.68	6.02	6.59	8.65
10	1.94	**42.77**	20.95	11.15	11.74	8.15	*	3.30

Conclusion

Based on the TES and LES contribution to the common extensor and thoraco-lumbar fasciae, it is possible that their contribution to the PIT is meaningful and helps with supporting the trunk. The role of the HS is evident in the hip extension portion of the test, but it's contribution to a positive test response in unclear. Their attachment to the pelvis may help with stabilizing the spine from the distal end, but it is difficult to determine if the large activation amounts seen in some subjects have to do with the hip extension required or if this activity would help to protect the spine against a PA load. The relationship between the TES and HS seen in some of the subjects may suggest there is some role the HS has in protecting the spine but these are associations at best.

From this dataset, while it was not statistically analyzed, if we consider all muscles involved, the LM plays a smaller role during this test. However, these are a combination of local and global trunk stabilizing muscles and we do not know what contribution all of these muscles acting on the spine have for predicting success with stabilization exercises. If the muscles of the lower extremity involved in hip extension are ignored(Table 1.13) the LM appears to have a larger proportion of activation during the test. The ability to isolate LM and determine if this alone can produce positive responses during the PIT will help shed light on what the major muscle contributors may be during this test. A positive response solely from LM NMES could suggest that the other muscles do not play a major role. The limitation to this preliminary study however, is that there was no standardization of how high the leg was raised in this protocol and the differences in HS and TES could be simply from varying leg raising heights across subjects: HS from hip extension and TES from the amount of flexor moment created during the test based

on leg raising height. In addition, these were healthy subjects with no force applied to the spine to determine painful response was present. Therefore, no one had a negative test (as clinically defined). The role of the LM during the test may be better defined by being able to recruit individuals with negative test findings. Overall, there is great amount of variability in muscle activity during this test as it was performed. In order to study the test and muscle contribution during this test, greater standardization of this test needs to be considered for the final proposal.

For the purpose of this proposal it would be beneficial to collect information on LD, TES, gluteal, and HS along with LES and LM during the PIT to understand the contribution from upper and lower quarter muscles. The proposed study will have a limit on sEMG leads available. Based on observation above, G.Max provided less contribution during the PIT than G.Med, therefore, G.Max will not be assessed bilaterally during the test. Prior work on G.Max activity has revealed that muscle activity increases as the hip joint reaches extension.[155] During this preliminary analysis, hip extension was not controlled, but subjects did not bring their hips to full extension. This could explain the lower percent activation of G.Max during this test. IO/TA had minimal contribution to the test, but it may still be beneficial to have abdominals represented during the test, therefore, external oblique may offer a different perspective on the role of trunk flexors during the test. It would also be beneficial to standardize leg raising height across subjects so that muscle activity from height of the leg raise influencing interpretation of muscle activity during the test is minimized.

1.7 Research Methods

Aim 1: Characterize lumbar extensor muscle neuromuscular control strategies through trunk muscle activation patterns during active forward bending and the prone instability test.

1.7.1 Methods for Aim 1a: Describe and quantify trunk muscle activity during a forward bend task.

The objective of aim 1a is to characterize muscle activation patterns in healthy individuals devoid of aberrant movement patterns (altered lumbopelvic rhythm (aLPR) and judder (JUD)) and the muscle activation patterns of patients with LBP who have JUD and aLPR. This aim will be accomplished through a descriptive study utilizing data from our previously conducted cross-sectional designed study. Expected Outcome: Identification of typical and aberrant trunk muscle activation patterns in individuals classified with normal and aberrant (JUD, aLPR) forward bend movement patterns

Subjects

The pool of 69 subjects (52% female; age 43± 11.2) noted under preliminary study 5.1 and used for the development of the approach to analyzing the sEMG activity will be used for this aim. Subject demographics, inclusion, and exclusion criteria are listed in section 5.1.

Instrumentation and Data Collection

Kinematic and sEMG data collection procedures have been described in the preliminary study section 1.6.

Procedures

Kinematic data will be analyzed using the algorithm developed by Wattananon (2014) where each forward bend motion is rated for presence/absence of JUD and/or aLPR(Appendix G). Operational definitions for aberrant motion during the forward bend are as follows: 1) Instability catch and judder (JUD): sudden changes in instantaneous velocity or fluctuations in lumbar or pelvis segment angular velocity, and 2) altered lumbopelvic rhythm (aLPR): hip motion is greater than lumbar spine motion in the first 1/3 of forward bend, and/or lumbar motion is greater than hip motion in the last 1/3 of movement[10].

Data Reduction and Analysis

Forward bend motions that demonstrate aberrant patterns of JUD and aLPR will be used to investigate muscle activation patterns associated with these aberrant movements. These muscle activation patterns will then be compared to activation patterns from healthy control subjects with typical movement patterns. In order to investigate muscle activity patterns during forward bend for aim 1a, sagittal plane kinematics of the lumbar segment with respect to the pelvic segment will be used to determine the forward bending phase of the motion. The first zero crossing of the lumbo-pelvic angular velocity that exceeds 1 radian/sec will be considered the initiation of the forward bend movement. If there is no negative velocity, then, the point where angular velocity exceeds 1radian per second will be used. The point where angular velocity crosses from a positive value to a negative value (second 0 crossing) will be considered the end of the forward bending phase. Heart rate will be removed from all EMG signals with a fast independent component analysis (ICA) Labview program. The EMG signals will be further processed

with a 10Hz Bartlett RMS filter and all trunk muscles will be cross correlated against each other. In order to investigate synchronization of muscles, cross correlation coefficients (CCC) will be calculated for all trunk muscle combinations (LM, LES, TES) as described in the preliminary study. Positive correlations will indicate that muscle pairs are acting in phase while negative CCC will indicate that muscle pairs are acting out of phase. The magnitude of CCC between muscle pairs determines how *in phase* or *out of phase* muscles are in relation to other muscle pairings. Cross correlation phase lags (T) between muscles will also be used to assess muscle synchronization. Small T (<500ms) values will be considered to be indicative of simultaneous muscle activation[131,156]. CCC will be checked for normality assumptions, with Fisher's Z transformation[157] performed if normality distributions are not met. A MANOVA of the CCC and T of the LES, TES, and LM muscles will be performed between LBP patients with aberrant movements and healthy control subjects with typical movements to determine synchronization of muscle activation during the forward bend. Muscles that are acting more synchronously would yield higher CCC and lower T compared to other muscle pairings.

Exploration of motor unit synchronization within muscles during the forward bend will be performed by analysis of sEMG amplitude data. Kinematic and sEMG normalized to the submaximal volitional contraction (sMVIC) data will be time normalized into 10 bins during the forward bending phase (as defined in the preliminary study 5.1) and compared between groups using a mixed model ANOVA. Muscle and bins will be held as within group factors with low back pain as the between groups factor. The underlying theory here will be that motor unit synchronization will lead to higher EMG amplitudes within individual muscles[15,118] in patients with LBP that possess aberrant

movements compared to healthy controls with typical movement. Percent contribution of muscle activation during the forward bend will also be calculated for each bin as performed in the preliminary study to identify contribution of the trunk muscles during the forward bend. A mixed model ANOVA will be used to compare the bins during the forward bend between groups.

1.7.2 Common Instrumentation, Data Collection Procedures and Measures for prospective Aims 1B, 2A, and 2B.

Kinematics

Stiffness modeling:

In order to measure spinal stiffness an electromagnetic tracking system (Liberty, Polhemus Inc.) will be used to track trunk position data during the PIT. This data will be used to calculate a change in the angle between the two sensors and then determine stiffness of the spine by the change in rotation of sensor 1 and sensor 2. One sensor will be placed on S2 and another on L1, with a data sampling frequency of 120Hz[123]. Data reduction methods utilized in the preliminary study for beam bending stiffness and instrument error associated with our methods will be used to determine bending stiffness during this protocol.

Compression Load Cell

A custom apparatus will be used to apply a PA load over the transverse processes of (L1-L5) of the subject's lumbar spine. A compression load cell will be attached to the apparatus (Figure 1.8) and data streamed in real-time to a computer monitor that will provide real time visual feedback of the compression load applied to the spine by the examiner. The target force will be displayed on the screen with a ±2.5% window to ensure the tester achieves similar loads to the spine during the test. An event marker will

be made available to all subjects to indicate if there is a painful response to the load. The event marker will notate the amount of load that was applied at the time of the marker, so that same load can be re-applied during subsequent trials.

Forward bending kinematics:

Spinal kinematic data during the forward bending task will be collected as described in preliminary study section 1.6.1. Subject preparation and calculation of kinematic variables are described in Appendix F and Appendix G respectively.

Surface Electromyography

For aim 1B, a pre-amplified 14 channel sEMG unit (SA Instrumentation, San Diego, CA) will be used to collect muscle activity during the PIT. Pre-gelled Ag-AgCl electrodes will be placed at anatomical landmarks (Table 1.14) with a 2cm inter-electrode distance and a reference electrode placed on the lateral malleolus. One channel on the data collection board will be utilized for a signaling trigger that allows the subject to identify any painful event. All EMG data will be collected at 2400 Hz. with a gain of 500 and band pass filtered at 20-500Hz. Based on preliminary findings in section 1.6.6, gluteus maximus will only be recorded on one side, as it had a smaller percent activation during the PIT compared to the gluteus medius.

Table 1.14. Anatomic landmarks for placement of sEMG electrodes for aim 1B.

Muscle	Location	Muscle	Location
Gluteus Maximus (G.Max)	Midpoint between the lateral edge of the sacrum and greater trochanter	**Gluteus Medius (G.Med)**	5 cm posterior and 15 cm inferior to the midpoint of the iliac crest
Hamstring (HS)	15cm from the ischial tuberosity	**Thoracic Erector Spinae (TES)**	5cm lateral to T9 spinous process
Lumbar Multifidus (LM)	2cm lateral to L5 spinous process	**Lumbar Erector Spinae (LES)**	3cm lateral to L2 spinous process
External Oblique	15 cm lateral to umbilicus	**Latissmus Dorsi (LD)**	Midline between spinous process of T9 and axillary line

Neuromuscular Electrical Stimulation

For Aim 2A and 2B a clinical NMES device will be used to provide electrical stimulation to the LM. Stimulating electrode placement will be identical to those in the preliminary study as described in section 1.7.4 (Figure 1.16). NMES will be delivered at 50pps of 400us phase duration, with an on time of 15 seconds. The stimulation duration will be extended to 15 seconds to allow the tester ample reaction time to deliver compression loads to the spine. This also mimics the stimulation parameters used in the RUSI pilot study in section 5.5. Longer contraction duration times should aide in promoting fatigue of the target muscle. The stimulation will be manually triggered during the prone instability test. For the fatigue protocol, 15 seconds off time will be utilized to decrease rest period and increase the likelihood for fatigue.

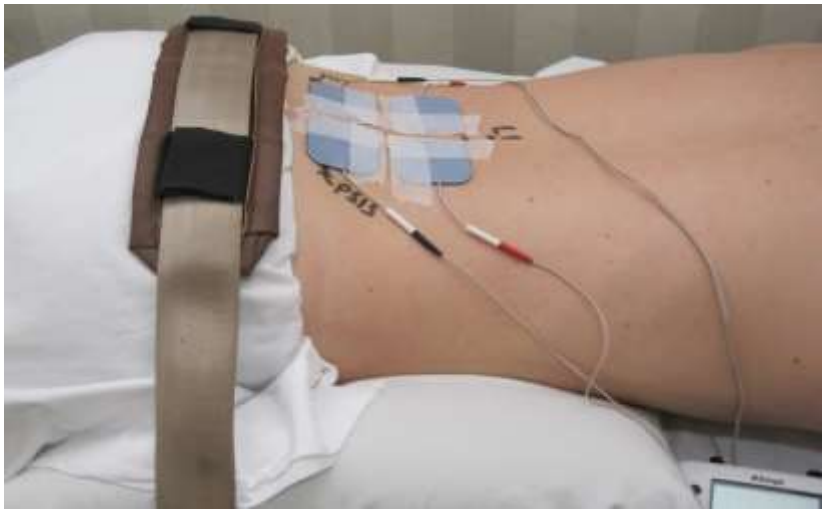


Figure 1.16.NMES setup for targeting lumbar multifidus for aims 1B, 2A, and 2B.

Pain Intensity Scale

Numerical pain rating scale (NPRS) (MDC=2;MICD=2.2;[158] will be used to measure pain intensity for aim 1B and 2B. See Appendix J for example of scale and anchor terms.

Disability Index

Oswestry Disability Index (ODI) (MDC=10;[159] will be used to measure self-perception of disability from patients with LBP. See Appendix J for example of the outcome scale.

Kinesiphobia

Fear avoidance beliefs questionnaire (FABQ) [49] will be collected on patients with LBP and will be used for descriptive purposes. See Appendix J for example of the outcome scale.

Fatigue

The Borg scale of perceived exertion [160] will be used to measure fatigue levels before and after testing. When a subject has an increase greater than 1 on the Borg following an activity, they will rest until it returns to baseline levels.

1.7.3 Methods for Aim 1b: Describe and quantify trunk and hip muscle activity as well as changes in spine stiffness during the PIT test.

The objective of aim 1b is to characterize muscle activation patterns of the trunk extensors during the PIT. Hypothesis: Lumbar spine stiffness increases during the PIT and are associated with muscle activity of the lumbar extensors. This study will employ a cross-sectional design using patients with LBP and healthy controls to characterize muscle activation patterns during the PIT. The result of a patient's PIT will be difficult to predict prior to the test. Therefore, all subjects with low back pain will be tested, and positive/negative test responses will be handled as a covariate in the analysis. Spine stiffness changes will be compared within subjects in each phase of the test (resting and active leg raise). Expected outcome: Determine if spinal stiffness changes do occur during the PIT. Identify the role of the trunk lumbar extensor muscles, particularly the lumbar multifidus, during the test. Determine if there is an association between spinal stiffness changes and muscle activation patterns during the PIT in increasing spinal stiffness and reducing pain during the PIT.

Subjects

Ten healthy subjects between the ages of 18-45 will be recruited. Exclusion criteria *specific to healthy subjects* will be a history of LBP that lasted longer than 3 days or required a visit to a health professional, BMI greater than 30, presence of aberrant motion during forward bend, and history of abdominal, back, or lower extremity surgery. Ten patients between the ages of 18-45 with current low back pain or a history of low back pain that lasted more than 3 days which is currently in remission will also be recruited. Exclusion criteria *common to both healthy subjects and patients with low back pain* are listed in Table 1.15.

Table 1.15. Exclusion criteria for aim 1B, 2A, 2B.

Common Exclusion Criteria for Patients and Healthy Controls	
Permanent structural spinal deformity (e.g., scoliosis),	Spinal fracture or history of spinal fracture
Osteoporosis	Active inflammatory joint disease
Signs of systemic illness or suspected non-mechanical LBP (spinal tumor , cancer or infection)	Active treatment of another medical illness that would preclude participation in any aspect of the study
Previous spinal or hip surgery	Frank neurological loss, i.e., weakness and sensory loss in a NR distribution
history of neurologic disease that required hospitalization	Pain or paresthesia below the knee
Body mass index greater than 30 kg/m2: The body mass index will be calculated from measured weight and height	Has performed rehabilitative exercises in the past with return to full function and no recurrence
Leg length discrepancy of greater than 2.5 cm	Current pregnancy
Allergies to medical tape or adhesives	

Procedures

Prior to testing, healthy subjects will be screened for the presence of aberrant movement during forward bending. Following the movement screen, sEMG electrodes will be placed on the LM, lumbar erector spinae, thoracic erector spinae, latissimus dorsi, gluteus medius, hamstring, and external oblique as described previously. All subjects will perform 2 trials of the modified Biering-Sorensen test, unilateral bridge (both sides), trunk flexion with tester applied resistance, along with bilateral resisted shoulder extension at 90 degrees, and bilaterally resisted shoulder flexion. The EMG data collected from these trials will be used for normalization purposes. Two electromagnetic sensors will be then be placed on the spinous processes of L1 and S2. All LBP and healthy subjects will be placed prone on a plinth. A posterior to anterior force will be applied by the examiner to spinous levels L1-S1 to determine any painful segments, using the compression load cell with a load of 22 N. Kinematic data will be collected to determine baseline spinal segmental mobility. When a painful segment is identified it will be marked with a skin marker, and the subject will be asked to assume the resting position of the PIT. Pressure will be applied to the segment again to confirm the presence of pain. If more than 1 painful segment exists, the subject will be asked to identify the most painful segment as outlined in Appendix I.

Testing in the resting phase:

Table 1.16 outlines the testing conditions, trials, and loads that will be used for the PIT. Healthy subjects will be placed in the resting PIT test position as described in preliminary study 6.3. Once properly positioned, 5 seconds of resting EMG and kinematic data will be collected via an examiner triggered button. After 5 seconds, the

data collection program will trigger an audible alarm and a PA load of 22 N will be applied to the pre-determined spinal segment via the apparatus attached to the compression load cell. The examiner will ramp up the force to get to the target load within 3-5 seconds, and total data collection time (including 5 second resting data collection) will last for 20 seconds. Visual force feedback will be provided in real time to achieve the target load (tolerance ± 2.5%). An event marker will be placed next to the subjects hand to tap in the event that there is pain provocation. This procedure will be performed three times with 2 minutes rest between trials. If the subject reports pain provocation, the record from the event trigger will be plotted and the load that reproduced pain will be utilized for the remainder of testing. If there was no pain provocation, the same 22 N ± 2.5% load will be used for remainder of testing.

LBP subjects will be placed in the resting PIT testing position and an anterior load will be applied until pain is produced. Data collection will be similar to that of healthy subjects. Once positioned in the resting position, data collection will begin via an examiner triggered button and 5 seconds of resting EMG and kinematic data will be collected. After 5 seconds, the data collection program will trigger an audible alarm and anterior force will be applied to the marked painful spinal segment via the apparatus attached to the compression load cell. The applied load will be slowly increased until pain provocation occurs. The patient will verbally report pain provocation to the examiner of, while simultaneously pressing the event marker placed by their hand. NPRS will be collected for each pain provocation point for healthy controls and patients. Visual feedback will be set at 100% ± 2.5% of the load noted at the time of the painful event

marker and that load will be used for the subsequent 2 trials. Two minutes of rest will be allowed in between trials.

Testing in the leg raise position

Once resting condition data have been collected, all subjects will perform the active leg raising portion of the PIT. For this test, subjects will be asked to position themselves in the resting position , and a beam placed over a 24-inch high gate will be placed over their calves in order to standardize leg raising. Once positioned in the resting position, data collection will being via the examiner triggered button and 5 seconds of resting EMG and kinematic data will be collected. After 5 seconds, the data collection program will trigger an audible alarm. Subjects will be asked to raise their legs to the gate. The examiner will apply the same amount of an anterior load, to the same spinal segment as was done in the resting condition. Visual feedback will be set to 22N (± 2.5%) for healthy subjects without pain provocation or the pain provocation load (± 2.5%) that was established in the resting condition. Subjects will undergo 3 trials of the leg raising test. Subjects will be asked to trigger the event marker and provide a numerical pain rating during the test if there is pain with compression. A positive PIT is defined as elimination of pain during the leg raising portion. If a subject experiences a pain reduction of 2 or more points, during the leg raising portion, the test will be considered a negative test but with symptom improvement . Three trials of the standardized load will be completed. All subjects with LBP, who had a positive test, will undergo 1 trial of the maximal load the examiner can produce during the leg raise to determine the amount of load the subject can tolerate under the leg raising condition and the effects of that load on symptoms. This is being done to mimic the clinical testing

situation, since clinically the load is not held constant during PIT testing conditions. To compare stiffness values across the conditions (prone, PIT position, PIT leg raise) we are holding the applied force constant during our experiment.

Table 1.16. Outline of load application by position during PIT

	PIT Resting Position			PIT Leg Raising	
	Load			Load	
	Segment Level	Trial 1	Trial 2 and 3	Trial 1-3	Trial 4 (only if test is positive)
Healthy Control	L3 or pain provoking segment if any is identified	22 N, and possibly identify pain provoking load	22 N or pain provoking load from trial 1	22 N or pain provoking load from PIT resting position	Healthy Control with pain provocation has a positive test response: Apply maximal load
LBP	Pain provoking segment	Gradually apply load until pain is provoked	Pain provoking load from trial 1	Pain provoking load from PIT resting position	LBP patient with positive test response: Apply maximal load

Data Reduction and Analysis

To characterize muscle activity during the PIT, EMG data from the 14 muscles will be heart rate stripped and normalized to sMVIC. The mean sEMG amplitude during steady state leg elevation (over 2 seconds) will be used to create a percent of muscle contribution during the test, along with descriptive statistics (mean, standard deviation) of muscle activity. Test of multiple proportions using Chi Square analysis will be used to determine differences in muscle contribution between patients with low back pain patients and healthy subjects.

Spinal compliance and stiffness will be calculated as described in preliminary study 5.3. Repeated measures ANOVA will be used to determine if there are differences in spinal stiffness changes within patients with low back pain and healthy subjects during the resting and leg raising portions of the test. An alpha of .10 will be set for the omnibus, and effect size calculated for the change in spinal stiffness. In the event that there are patients with a negative test, or healthy control subjects with pain production that does not abolish with leg raising, the result of the test (positive/negative) will be used as a covariate in determining spinal stiffness changes.

Aim 2: Validate clinical assumptions of the role that lumbar multifidus muscle activity has in aberrant movements patterns during a forward bend task and a positive prone instability test.

1.7.4 Methods for Aim 2a: Characterize the effects of isolated lumbar multifidus muscle fatigue, achieved by a neuromuscular electrical stimulation (NMES) fatigue protocol, on movement quality during a forward bend task in healthy controls.

Hypothesis: Subjects with a typical forward bend movement pattern will demonstrate aberrant movement pattern following fatigue of the lumbar multifidi muscles. The objective of this aim is to determine if a temporary, isolated impairment of the LM through fatigue, can reproduce or worsen aberrant movements (detected via visual observation as well as kinematic detection) during an active forward bend task. A pre-posttest design will be used, with trunk kinematics during forward bend as the dependent variable measure before and after NMES fatigue of the LM.

Expected outcomes: Evidence to support the concept of lumbar extensor dysfunction as a primary mechanism underlying aberrant movement patterns during standing forward

bending. Production of aberrant movement through attenuation of LM activity via fatigue will support if reduced participation of this muscle during the forward bend has a major impact on movement quality.

Subjects

Healthy subjects recruited for aim 1b will be used for this aim. If subjects had pain provocation during the PIT test that persists other healthy subjects will be recruited to prevent the potential of experimental pain induced aberrant movements. See aim 1B for inclusion/exclusion criteria for healthy subjects. Care will be taken to detect aberrant movements through clinical observation. However, it must be recognized that a discrepancy between clinician observation and kinematic identification of aberrant movement exists (Wattananon, 2014). Subjects may be cleared during observation, however be categorized as having aberrant movement once forward bending is quantified using the kinematic algorithm. The purpose of pre-screening is to minimize this occurrence or only marginally exceed criteria for typical movement. By doing so, we hope to increase the likelihood of producing larger aberrant motion patterns that can exceed kinematic MDC's following the NMES fatigue protocol.

Procedures

For subjects who participated in the previously described studies an additional electromagnetic sensor will be placed on the lateral femoral condyle and spinous process of T3 as outlined in Appendix F. They will undergo a digitization trial , data will be calibrated with the digitization file, and converted to segment angular rotations using Euler angles to calculate kinematic variables as described in Appendix G. They will perform 2 trials of 3 repetitions of the forward bend task during which time segment

position will be collected. They will then be positioned to receive 10 electrical stimulation trains at 400us, 50 pps, with 15 second on time, and 60 seconds between stimulations, followed by the performance of 2 trials of 3 repetitions of the forward bend task. This number of NMES elicited contractions should be adequate to achieve fatigue based on results of preliminary study 1.6.4 and 1.6.5.

For newly recruited healthy subjects, subjects will have electromagnetic sensors placed as described in Appendix F, and then undergo a digitization process to calibrate kinematic segments. They will then receive 10 electrical stimulations as outlined in aim 2a. Following electrical stimulation, they will undergo an additional 2 trials of 3 repetitions of the forward bend task.

Kinematic criteria developed by Wattananon (2014) will be used to identify aberrant motion, as follows:

Coupling-angles of the femur on pelvis and pelvis on lumbar segments and phase-plane diagrams will be created. Coupling-angle will be used to determine the time point (T) that the coupling angle crosses the standard deviation band indicating a shift from lumbar dominant to pelvis/hip dominant motion. This will be used to determine if reversal of lumbopelvic rhythm occurs. Lumbar on pelvis coupling angle that exceeds 59 degrees, before 38% of the total forward bend will be considered as aLPR. MDC_{90} has been established by Wattananon using the same protocol as 11.9% of time normalized movement and this value will be able to determine if a shift in lumbopelvic rhythm is meaningful.

The number of local minimum and local maximum, characterizing sudden acceleration/deceleration will be quantified using the phase plane diagram. The

presence of 6 or more local minimum would indicate the presence of judder. MDC_{90} has been established as 1.6 local minimum, so forward bends with increases in minimum/maximum that exceed 2 local minimum will be considered to have worsened.

Forward bend will be analyzed using the above kinematic criteria. Subjects who do not have aberrant movement prior to NMES will be considered to demonstrate aberrant movement following NMES if they satisfy two criteria: They exceed the kinematic cutoff points for either aLPR and/or JUD and they exceed it by a value greater than the MDC_{90}. If subjects are determined to have aberrant movements through kinematic analysis pre NMES but not in the clinical observation, their movement pattern will be considered to have worsened if kinematic cut offs exceed MDC (Appendix H).

Repeated measures ANOVA using an alpha of .10 will be used to determine if there is a statistical difference between pre and post electrical stimulation of forward bend, with time crossing (T) and number of local minima as the dependent variables measured before and after NMES. This will be used to analyze overall differences in kinematics following NMES. To determine the impact of NMES to create or worsen aberrant movements, response of forward bend following NMES will be dichotomized: created/worsened aberrant vs. no change. McNemar's chi square change test will be utilized with an alpha of .10 to determine if there were significant changes following NMES.

1.7.5 Methods for Aim 2b: Determine if electrically induced LM contraction using NMES can yield increased spinal stiffness and a positive prone instability test.

This will be a pre-post design study with pain and spinal stiffness as dependent variables. NMES will be the independent variable that all subjects will receive. Pain and stiffness changes will be measured in the PIT position pre NMES and again during NMES. Response to the test and its effect on stiffness changes will be handled as a covariate as per aim 1B.

Expected outcomes: Identification of the role that the LM plays in the PIT. A large portion of the PIT test's impact on intervention is based on assumed roles of the trunk extensors, particularly the LM and associated spinal stiffening. This approach will provide a method to investigate those assumptions and directly support or change clinical practice.

Subjects

Healthy subjects and patients with low back pain recruited for aim 1b will also be used for this aim. See details of inclusion and exclusion criteria under aim 1b.

Procedures

All subjects will first undergo testing for aim 1b. Following completion of testing for aim 1b and 2a EMG electrodes over the LM will be removed. Healthy subjects who had symptom provocation during the PIT will be asked if they have residual symptoms. Healthy subjects that do not have residual symptoms will receive 10 additional NMES stimulations. Electrical stimulation electrodes will be placed 1cm from the spinous process between the spinous process of S1-L3 bilaterally as previously described.

For determining spinal stiffness changes with adjunctive NMES, all subjects will be placed in the PIT position and a fixation belt will be placed across the sacrum below the S2 recording sensor. Three anterior forces will be applied, with 120 seconds in between at the prior established segment and anterior force (aim 1b, using the compression load cell apparatus). Following application of anterior forces in the relaxed position, electrical stimulation will be applied using a clinical stimulation unit at 400 us pulse duration, 50 pps, with amplitude increased until LM muscle contraction is visualized and an isometric anterior tilt of the pelvis is achieved. Data collection will begin via an examiner triggered button and 5 seconds of resting kinematic data will be collected. After 5 seconds, the data collection program will trigger an audible alarm at which point the NMES will be triggered by the examiner and anterior force will be applied. For healthy subjects, anterior forces will be administered at the L3 level or at the segment that was found to be painful in aim 1B with previously applied forces. Subjects with low back pain will have the anterior force reapplied at the pain provoking segment using prior established pain provoking force. This will be repeated for 3 trials with 120 second rest between trials. All subjects will have access to an event trigger to activate, as in aim 1B, if there is pain provoked during the anterior force. NPRS will be collected on all subjects before and after each trial. Elimination of pain with anterior load will be considered a positive response to NMES, while reduction of pain by 2 points or more will be considered a negative response, with pain reduction.

Healthy subjects selected that do not have residual pain following this procedure will be moved to a prone position with a fixation belt positioned on the pelvis, under the S2 kinematic sensor and receive an additional 10 electrical stimulations delivered with no

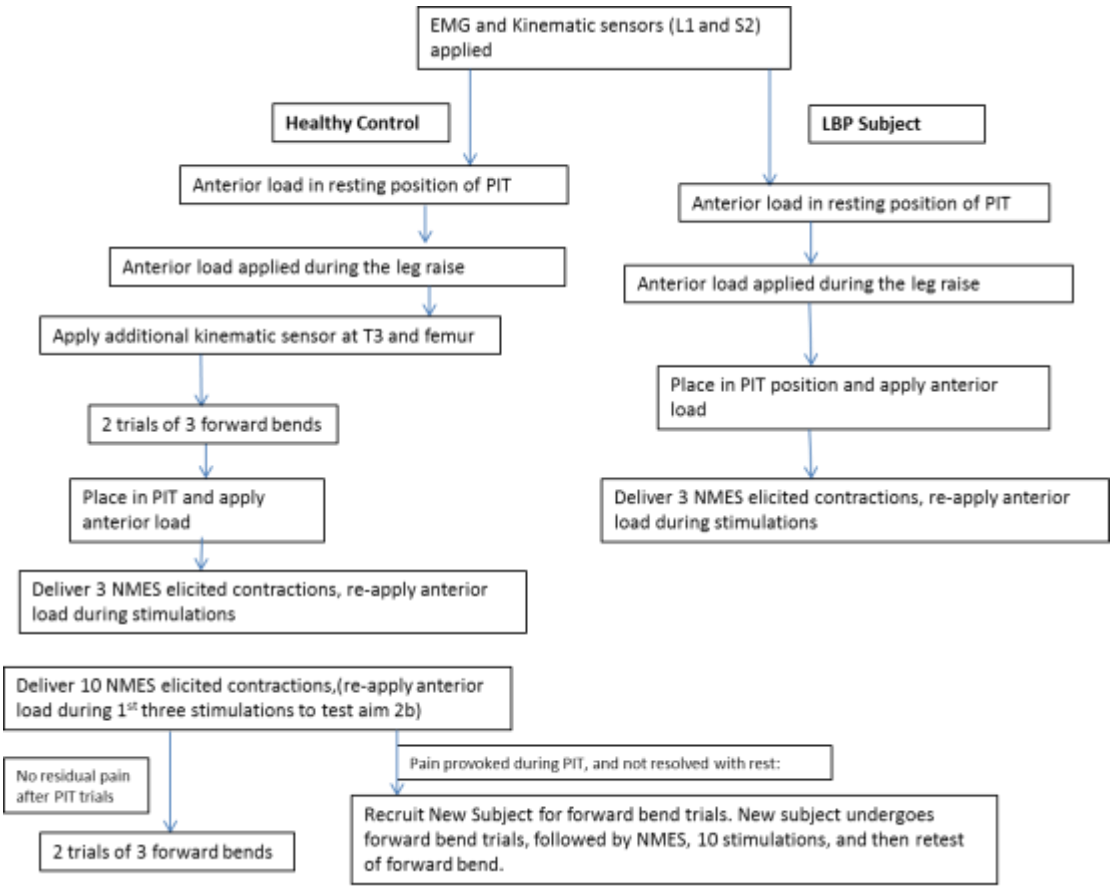
anterior load applied. This will be to achieve muscular fatigue of the LM to conclude aim 2a.

Data Reduction and Analysis

Methods used in preliminary study 5.3 will be used to measure changes in spinal stiffness. Mixed model ANOVA will be used to determine if there are differences in spinal stiffness changes between patients with low back pain and healthy subjects during the resting and NMES portion of the test. An alpha of .10 will be set for the omnibus, and effect size calculated for the change in spinal stiffness. In the event that there are patients with a negative test, or healthy subjects with pain production that does not abolish with leg raising, the result of the test (positive/negative) will be used as a covariate in determining spinal stiffness changes.

The flow of data collection for aims 1B, 2A, and 2B are illustrated in Figure 1.17.

Figure 1.17Testing protocol for aim 1B, 2A, and 2B.



1.8 Potential Problems and Alternative Strategies

Aim 1B

Low back pain patients may not have symptom elimination during the leg raising portion of the PIT. It is uncertain how many subjects will demonstrate a negative test result. In this case, as described in the data analysis segment of aim 1B, correlations between kinematic spinal stiffness changes and EMG signals may offer some information on the relationship between muscle activity, spinal stiffness, and pain reduction. Another option would be to add additional subjects with low back pain to the study protocol in an attempt to insure that 10 subjects demonstrate a positive test response. Lastly, we can also add additional subjects to increase the number that demonstrates a negative PIT to better understand the differences between a positive and negative test result. The alternatives will depend on the number of subjects enrolled that demonstrate positive versus negative response. An additional method may also be to provide an electrical stimulation superimposition during the PIT in patients with LBP with negative test results.

Subjects may demonstrate stiffness changes as a result of viscoelastic changes from repetitive compressive loads. This may impact stiffness change data during the leg raising task, as well as during aim 2B's NMES elicited contraction. To minimize this, loads will be applied gradually to the spine, and ample resting time of 2 minutes will be given before trials. All subjects will receive PA spring tests prior to the application of kinematic sensors, to determine the presence of painful segments. This may also help to precondition tissues prior to testing protocol and minimize changes resulting from viscoelastic effects. The amount of change that is a factor of test protocol error also needs to be established. In order to accomplish this, a smaller sub-study will be performed on a

mix of healthy controls subjects and patients (with a history of low back pain, but currently in remission), to determine test-retest reliability of the stiffness changes measured with kinematic sensors.

Aim 2a

Subjects may be fatigued from the leg raising task of the PIT performed for aim 1B. This may affect kinematic data collected during the pre and post electrical stimulation conditions. If already fatigued, they may develop or increase aberrant movements prior to testing of aim 2B. To deal with this potential problem, ample resting time will be used between trials, Borg scale will be used to determine fatigue levels before and after testing, and care will be taken to avoid mistakes that will require additional trials. Healthy control subjects will be screened for the presence of aberrant movements before participating. Healthy control subjects may also develop pain from testing which is unlikely based on practice sessions performed to refine the testers load application. However, if pain is produced during the testing of the PIT that does not resolve, the subject will not proceed to the forward bend protocol.

aim 2a and 2b.

These aims are proof of concept studies, and preliminary data does not yet exist to support the number of subjects that are necessary to power the study. This may lead to a type II error. To deal with this error, an alpha of .10 will be used rather than .05. Effect size will be calculated during the study to quantify the difference in spinal stiffness changes that occur as a result of hip extension during the PIT and during NMES to the LM. In the absence of statistical significance, the effect size will be able to quantify the strength of association and proportion of variance explained in spinal stiffness changes as

a result of a leg raise and the NMES. Effect size will also be used to determine changes in kinematic variables of local minima of the phase plane diagram used to detect JUD and change in time crossing T of the coupling-angle diagram used to detect aLPR [161]. A planned analysis of the data midway once 5 subjects in the healthy control and low back pain groups have been completed can be completed and a power analysis performed on stiffness changes during the PIT and NMES. This should refine the number of potential subjects that are needed for the study, and additional subjects will be recruited as necessary.

Preliminary studies 1.6.4 and 1.6.5 established the ability to isolate LM with NMES, and that fatigue with the protocol is promising. However, another fatigue study is planned using EMG pre and post NMES to study medial frequency drop following stimulation. Subjects will perform a modified Biering-Sorensen test (holding for 30 s) as identified in Appendix J, with EMG collected for the LM and LES. They will then receive NMES to the LM as described in previous sections. They will then perform another Biering-Sorensen test with EMG collection to analyze medial frequency drop as another measure of fatigue. After 5 minutes rest, they will perform the Biering-Sorensen test again to determine level of muscle fatigue. This repeated test will be used to determine if subjects will remain adequately fatigued as it takes several minutes to prep them for the post fatigue forwarding bending tests. It is anticipated that the transition from NMES through data collection will happen within a 5 minute window.

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Chapter 2: Muscle activation characteristics during forward bending in individuals with low back pain and movement coordination impairment: a planned secondary analysis.

Abstract

Background: Forward bending is used to classify individuals with movement coordination impairment related back pain (MCILBP). Identifying mechanisms involved in this impairment may help identify specific treatment options.

Objective: Characterize muscle activation differences of the trunk extensors between individuals with and without MCILBP.

Study Design: Secondary analysis of data from a cross sectional study on individuals with MCILBP.

Methods: Data from 24 individuals with MCILBP and aberrant movements during forward bend were included in this analysis. Fourteen met a-priori kinematic criteria for altered lumbopelvic rhythm and 10 met criteria for rapid acceleration and deceleration in lumbar angular velocity. These individuals were compared to 15 individuals with no history of low back pain and typical movement patterns. Surface EMG collected on the lumbar multifidus (LM), lumbar erector spinae (LES), and thoracic erector spinae (TES) muscles was used to characterize and compare muscle activity during forward bend. Mean EMG, normalized to a submaximal contraction, was used to compare muscle activity. EMG signals were divided into epochs to compare movement patterns. Cross-correlation was used to compare co-activation.

Results: Individuals with MCILBP had greater lumbar multifidus, lumbar erector spinae, and thoracic erector spinae muscle activity during forward bend, compared to individuals with no history of low back pain ($p<.05$). Individuals with aberrant movement reached peak extensor activity at a later phase in forward bending but maintained increased activity for longer duration than individuals without aberrant movement. Individuals with typical movement appear to be characterized by greater co-activation between lumbar multifidus and lumbar erectors spinae than those with aberrant movements.

Conclusions: Individuals with MCILBP who demonstrate aberrant movements are characterized by higher trunk extensor activation compared to individuals with no history of low back pain and typical movement. This finding may be an attempt of the trunk extensor muscles to increase spinal stability during a forward bend movement.

Introduction

Low back pain (LBP) effects up to 26% of the population in the United States.[1] It has been reported that upwards of $100 billion in medical costs and lost wages per year are associated with caring for individuals with LBP.[2] Rehabilitation of individuals with LPB is challenging due to little association between pathoanatomic mechanisms and symptoms .[3] However, selecting interventions by subgrouping individuals into treatment based classifications has resulted in improved outcomes in individuals with acute to subacute LBP. [4] One treatment subgroup consists of individuals identified to benefit from trunk stabilization exercises.[5,6] These individuals are considered to have movement coordination impairments (MCI) of the lumbopelvic region resulting from impairments in muscle function.[3] Unresolved impairments in muscle activation are theorized to drive recurrence and chronicity of LBP[7]. Therefore, individuals with movement coordination impairment related LBP (MCILBP) might be an important subgroup to study to determine whether impairments in neuromuscular control exist during functional tasks.

Many classification systems exist for determining whether an individual has MCILBP.[6,8-10] All of these systems assess the quality of a forward bending motion as part of their examination. Forward bending requires coordination of multiple segments including the spine and the hips[11] and may provide sensitive diagnostic information on movement control. Kinematic analysis has demonstrated that aberrant movements occur in the midrange of the forward bend movement[12] where muscle activity is believed to play a primary role in spinal stabilization.[13,14] These aberrant movements have been considered construct validation for this subgroup having impairments in muscle controlled spine stability.[12] Therefore, forward bending represents an important task by

which to assess an individual's movement coordination and control. Further study of neuromuscular control in this subgroup of individuals would provide information that could be useful for guiding interventions within these classification systems.

Previous studies have shown that higher levels of trunk muscle co-activation occur in individuals with LBP.[15] Higher levels of trunk muscle co-activation have been proposed to offer joint protection, but perhaps at the cost of movement control and precision.[16] This finding may provide one explanation for aberrant forward bending but this has yet to be established. Increased LES activation during forward bend has been reported in patients with LBP.[17] This is believed to result in aberrant movements from reduction in the stabilizing activity of the lumbar multifidus (LM) .[18] A reduction in LM contraction ability among individuals with MCI[19] may lend support to this theory. Impaired muscle function may result in synchronization of motor units within a muscle to increase motor output, represented by an increase in EMG amplitude, at the cost of smooth fluid movement.[20,21] Increasing the ratio of hip flexion to lumbar flexion angle has been reported to improve the lumbar extensors' ability to stabilize the spine.[22,23] This may cause alteration in the coordination of the multi segment spine and pelvis during forward bending, resulting in aberrant movement.

Prior research has suggested that movement disruptions occur in stages of forward bending when muscle activity provides primary stabilization to the spine.[12,24] Combining this knowledge with 1) evidence of LM activation impairments in individuals with MCILBP[19] and 2) the role that the LM is believed to have in forward bending[18] provides theoretical support for altered neuromuscular control of the lumbar extensor muscles driving the differences in forward bending movement patterns between healthy

individuals and those with MCILBP. Identifying differences in neuromuscular control between these groups during forward bending would help substantiate these theories and provide valuable information in the mechanism related to impaired movement control.

The overall aim of this secondary analysis was to identify and characterize differences in neuromuscular control during forward bending between individuals without a history of LBP and a typical forward bending movement pattern, and individuals identified to have MCILBP and aberrant forward bending movements. There were 3 study hypotheses. The first purpose was to determine whether differences in trunk extensor muscle activity, across the entire forward bend task, existed between groups. It was hypothesized that individuals with MCILBP would have *greater activation* of the LM and erector spinae muscles when compared to individuals with no LBP and typical forward bending movement. The second purpose was to compare muscle activation patterns *throughout* the forward bending task between these two groups. It was hypothesized that individuals with MCILBP would demonstrate altered patterns of muscle activation during forward bending when compared to individuals with no LBP and typical forward bending movement. The third purpose was to determine whether there were differences in co-activation of trunk extensor muscles during forward bending. It was hypothesized that there would be greater co-activation of the lumbar and thoracic erector spinae in individuals with MCILBP.

Methods

Participants

Participants for this analysis were drawn from a clinical trial that investigated the effects of trunk stabilization exercises on seated postural control in individuals with MCI related LBP. The study was approved through (blinded) university's institutional review board and data were collected from 2009 through 2013.

Participants were invited to participate if they had LBP for less than 12 weeks, average pain greater than 3/10 on a numeric pain rating scale, and Oswestry disability index of 20% or greater. Exclusion criteria were: history of spinal surgery, peripheral or central neurologic signs, lower extremity surgery or injury that would affect testing, systemic symptoms, prior physical therapy for their LBP, or pregnancy. Individuals that met inclusion criteria underwent a clinical examination by physical therapists to determine whether they met the classification of MCILBP using criteria established by Hicks, et al. (2005) and Sahrmann (2001) (see Sung, et al. (2015) for specific criteria). Thirty-three individuals with MICLBP (20 females, mean body mass index (BMI): 25.6 ± 4.4 kg/m^2, mean age: 32 ± 14 years) were selected to participate in the postural control study. They were matched by gender, age (± 5 years), and BMI (± 5 kg/m^2) to 33 individuals without a history of low back pain (mean BMI: 23.8 ± 3.8 kg/m^2, mean age: 34 ± 13 years). Participants for the current analysis were drawn from this total pool of 66 individuals with and without a history of LBP.

Instrumentation and procedures

As part of the study, participants had simultaneous recording of surface EMG and thoraco-lumbo-pelvic kinematics during a forward bending task. An electromagnetic

tracking system (Liberty, Polhemus Inc., Colchester VT) was used to capture kinematic position data from the thoracic and lumbar spine, pelvis and femur. EMG data were collected (SA Instrumentations, San Diego, CA gain 500; band pass filtered 20-500Hz) from the LM, LES, and thoracic erector spinae (TES) musculature.

Skin preparation for EMG involved cleaning with alcohol followed by light abrasion. Pairs of Ag-AgCl electrodes with 2 cm inter-electrode distance were placed bilaterally along target muscles as follows: LM- 2 cm lateral to L5 spinous process , LES - 3 cm lateral to L2 spinous process, and TES – 5 cm lateral to T9 spinous process.[26] A reference electrode was placed on the lateral malleolus.

Participants underwent collection of quiet resting EMG for 2, 30-second trials. This was followed by performance of resisted trunk flexion, extension, and bilateral side bending in a custom device designed to minimize lower extremity contribution. This was performed for EMG normalization. Submaximal contractions (sMVIC) were used for normalization due to uncertainty of patients with LBP producing maximal effort.[27] SMVIC has also been reported to have better reliability compared to MVIC in normalizing trunk EMG data.[28] In order to standardize the sMVIC across participants, a computer monitor with a force target (15% of their body weight) was provided to the participants during the sMVIC trials.

Electromagnetic sensors were then placed on the spinous process of T3, L1, and S2 along with one sensor on the lateral condyle of the femur to model the thoracic, lumbar and pelvic segments of the trunk. EMG (2400 Hz) and kinematic data (120Hz) were collected simultaneously and time synchronized through a custom program

(LabVIEW 8.6, National Instruments, Austin, TX). Participants used their preferred strategy to perform 6 maximal forward bend movements.

Rating of forward bend movements and subject selection for analysis

This analysis used previously validated kinematic algorithms to identify the presence/absence of two types of aberrant movements during the forward bend task. Aberrant movements consisted of altered lumbopelvic rhythm and judder (rapid acceleration and deceleration in lumbar angular velocity). These aberrant movements have been shown to be able to identify individuals with MCILB[24] who would benefit from trunk stabilization exercises. Abnormal lumbopelvic rhythm (aLPR) was defined as a lumbopelvic coupling angle exceeding 58 degrees within the first 38% of the forward bend motion on a graph plotting lumbar angle versus pelvic angle during forward bending. Judder was defined by the presence of 6 or more decelerations on a lumbar phase-plane graph (segment angular velocity versus angular displacement)[24]. A custom written LabView 8.6 (National Instruments, Austin, TX) program was used to apply the algorithms to each individuals forward bend trials.

Prior work has determined that not all individuals with LBP demonstrate aberrant motion, nor do all individuals without a history of LBP consistently present with typical motion during a forward bend task.[29] Therefore, to ensure that movement pattern classification was based on a *consistent* movement pattern, we established the following criteria for rating forward bend movements. Individuals without a history of LBP and a typical movement pattern (TYP) were identified and selected for inclusion when aberrant movement was detected in *2 or less* forward bend trials. Individuals with LBP and aberrant forward bending patterns were selected for inclusion when aLPR or judder was

detected in *3 or more trials*. Based on these criteria, two LBP groups with aberrant movement patterns were identified: aLPR only and, both aLPR and judder (aLPRJUD). Separation of LBP participants into two movement patterns groups was determined a priori based upon preliminary analysis of EMG data that demonstrated distinct muscle activation patterns between these groups. Table 2.1 contains demographic information of participants selected for inclusion into this analysis from the original study on postural control.

EMG data reduction

EMG data reduction included removing heart rate artifact using fast independent component analysis[30], rectification (RMS, Tc=30ms) and resting EMG signal subtraction through a custom LabView 8.6 (National Instruments, Austin, TX) program. In order to determine whether the trunk extensor muscle activation data should be treated as a group, or as separate muscles, a cross correlation analysis was performed.[31] Coefficient values were significantly different ($P<.001$), which indicated minimal cross talk between muscles. This finding supported our initial thought that each muscle group would be analyzed separately.

To investigate EMG amplitude for muscle activation differences during forward bending, signals were normalized to the sMVIC and analyzed for left-to-right differences (paired t-test, alpha=.016). No significant differences in muscle activity levels between sides existed; therefore, muscle activity levels were considered to be symmetrical and EMG data were analyzed as an average of left and right sides for LM, LES, and TES. Within session reliability was calculated using normalized EMG of the 1st and 6th forward bend trial of all participants in this analysis. $ICC_{(3,2)}$ was determined to be as follows:

LM: .91, LES: .95, TES: .95. Standard error of the measure (SEM) was determined to be 13% for LM, 9% for LES, and 14% for TES.

Statistical analysis

Kinematic rating of forward bending revealed three distinct movement groups: individuals with a history of low back pain that demonstrated either aLPR or aLPRJUD and individuals without LBP who demonstrated TYP movement pattern. Therefore analysis consisted of three groups. All data analyses were performed using SPSS 21 (IBM, Armonk, NY).

Differences in trunk extensor muscle activation between groups: mean muscle EMG amplitude

To compare differences in trunk extensor muscle activation between groups, normalized EMG amplitude was compared between groups using mixed ANOVA (within groups: muscles; between groups: typical (TYP), aLPR, and aLPRJUD, alpha=.05). Post-hoc analyses were performed using Tukey's HSD multiple comparison correction with harmonic means adjustment for unequal sample size.

Muscle activation pattern differences between groups: EMG amplitudes within movement epochs

To characterize and compare trunk extensors activation patterns between the groups *throughout* the forward bending task, normalized EMG signals for each extensor muscle group were divided into10 epochs. Each epoch represented 10% of the forward bend task. For each muscle, the average EMG signal within each epoch was determined. Epochs were then averaged across trials and compared using mixed ANOVA (within groups: muscles and epochs; between groups: TYP, aLPR, and aLPRJUD).

Kinematic data for lumbar flexion angles, lumbar angular velocity, and ratio of hip to lumbar flexion were calculated and presented as means within epochs. This information was presented to assist with interpretation of EMG data within epochs. Mixed ANOVA with Tukey HSD post hoc testing was performed to compare mean lumbar velocity, mean lumbar flexion angles, and mean hip to lumbar flexion ratio as an average of the complete forward bending phase (alpha=.05).

Comparison of muscle co-activation during the forward bend task

Co-activation of trunk extensor muscles were first quantified using the cross-correlation function. [33] LabVIEW 8.6 (National Instruments, Austin, TX) was used to calculate the cross-correlation coefficient between muscle pairs (R_{xy}) using the equation:

$$R_{xy}(\tau) = \left(\frac{1}{T}\right)\frac{\int_0^T x(t)y(t\pm\tau)dt}{\sqrt{R_{xx}\,(0)R_{yy}(0)}} \qquad (1)$$

In equation 2, τ refers to the phase shift (magnitude of the shift between x and y , T is the duration of the signal record, x(t) is the signal held stationary, and y(t) is the time shifted signal. R_{xx} (0) and R_{yy}(0) represent zero phase lag autocorrelations and serve to normalize the correlation coefficient.[34] The R_{xy} range is between +1 and -1, with highly positive numbers indicating muscles acting in phase, while highly negative values would indicate activation of one muscle while the other muscle is not active.

A 300 ms limit was placed on the maximum shift allowed to obtain a cross correlation coefficient.[31,34] Without a limit on the phase shift, highly correlated points in two signals that are separated by a large period of time may be falsely interpreted as co-activation. Side-to-side pairings in the cross correlation co-efficient revealed no significant differences ($p<.05$) so symmetry was assumed and sides were averaged. Moderate correlations between R_{xy} pairings (.3-.64) required the use of MANOVA to

compare muscle co-activation between groups using LM-LES, LM-TES, and LES-TES muscle pairing. Phase-lag (τ) between the muscle pairings at the maximal correlation was compared using MANOVA to determine onset latencies and activation order between muscle pairings (alpha=.05). When the first muscle listed in the pairing is activated prior to the second muscle, τ is positive. Negative τ values indicate activation of the second muscle prior to the first muscle listed in the pairing.

Muscle co-activation was *characterized* by calculating how much each muscle contributed to the forward bend. The contribution of individual muscles was calculated as:

$$Muscle_{percent\ contribution} = \left(\frac{Target\ Muscle\ EMG_{\ normalized}}{LM\ EMG_{normalized} + LES\ EMG_{normalized} + TES\ EMG_{normalized}}\right) = \text{x } 100 \qquad (2)$$

Muscle percent contribution was compared using MANOVA, (alpha=.05).

Results

Differences in trunk extensor muscle activation between groups: mean muscle EMG amplitude

There was a significant main effect of muscles during the forward bending task $F_{(2,35)}$=4.841, p=.014, η^2=.217. There was also a significant main effect of group: $F_{(2,36)}$=11.17, p<.001, , η^2=.383 Post hoc analyses revealed LM activation (mean=75% ± 42%) to be significantly greater than both LES (mean=58% ±41%) and TES (62% ± 43%). However there were no differences between LES and TES. There was no significant interaction $F_{(2,36)}$=2.197, p<.126, η^2=.109 between movement groups and muscles. Figure 2.1depicts muscle activity levels between the movement groups. Post hoc analyses (alpha <.05) revealed significantly lower EMG amplitude of the LM in the TYP group compared to both aLPR and aLPRJUD groups. However, there were no differences

were detected between the aLPR and aLPRJUD groups. LES demonstrated significantly higher amplitude in the aLPRJUD compared to the TYP group. However there were no differences between TYP and aLPR groups or between the aLPR and aLPRJUD groups. TES activity was significantly higher in the aLPRJUD group than TYP or the aLPR group. However, there were no differences between TYP and aLPR.

Muscle activation pattern differences between groups: EMG amplitudes within movement epochs

ALPRJUD demonstrated significantly slower velocity compared to aLPR and TYP with no differences between the latter groups. TYP demonstrated greater lumbar flexion angles compared to the aberrant groups, with no difference *between* aberrant groups. This is in agreement with prior work that found individuals with LBP typically demonstrate less lumbar motion and slower velocity during forward bending compared to individuals without LBP[11,32]. There were no statistical differences in the hip to lumbar flexion ratio across groups.

Figure 2.2 displays kinematic descriptive data for mean lumbar and hip flexion angles, lumbar flexion velocity, and the ratio of hip to lumbar angles during forward bending within the epochs. There was a significant main effect of muscles across the epochs $F_{(27,10)}=3.43$, $p=.023$ $\eta^2=.903$ during forward bending. There was also a main effect of groups across epochs. $F_{(18,19)}=2.69$, $p=.019$ $\eta^2=.719$. There was a significant interaction across the epochs between movement groups, $F_{(54,22)}=2.645$, $p=.007$, $\eta^2=.867$. Post hoc analysis revealed a significant effect of LM $F_{(9,324)}=3.65$, $p=.026$ $\eta^2=.088$, LES $F_{(9,324)}=3.719$, $p=.018$ $\eta^2=.094$, and TES $F_{(9,324)}=6.46$, $p=.000$ $\eta^2=.152$ across epochs.

However, there was no muscle x group x epoch interaction among any muscle group (Figure 2.3).

Comparison of muscle co-activation during the forward bend task

Comparison of muscle co-activation revealed a significant difference between the muscle pairings among movement pattern groups $F_{(6,72)}$=2.58, p=.025, η^2=.177. Cross-correlation coefficient values are listed in Table 2.2. Post-hoc comparisons were performed among muscle pairings between the TYP and aLPR, as well as TYP vs aLPRJUD to determine the differences in co-activation that exist within the aberrant movement groups. There were significant differences between TYP and aLPR $F_{(3,26)}$=3.49, p=.019, η^2=.313 for muscle co-activation. TYP displayed higher LM-LES in-phase activation compared to aLPR p=.016, but there were no differences between LM-TES or LES-TES (Table 2.2). This was similar in the TYP vs aLPRJUD comparison, $F_{(3,22)}$=3.69, p=.027, η^2=.335, with TYP demonstrating higher in-phase activation of LM-LES compared to aLPRJUD, p=.008, with no differences between other pairings.

There was no significant effect of lag times between movement groups among muscle pairings, $F_{(6,72)}$=0.924, p=.48, η^2=.072, suggesting that muscle pairings were activated nearly simultaneously during forward bending. Based on negative phase lag values (Table 2.2) all groups demonstrated a cephalocaudal muscle activation pattern independent of their forward bending movement pattern with the exception of the LM-TES pairing in the judder group.

There was a significant main effect of muscle percent contribution differences, $F_{(2,72)}$=5.19, Wilkes Lambda p=.011, η^2=.229, during the forward bend task. Overall, LM contributed the most (mean =37.2%, SD= 14%) followed by the TES (mean=34.4%,

SD=16.5), with the LES contributing the least (mean=28.1%, SD= 11.8%). However, the contribution of muscles was not significantly different between groups $F_{(4,70)}$=0.952, p=.77, η^2=.148.

Discussion

The purpose of this analysis was to investigate neuromuscular control between individuals without LBP and typical forward bend movement, and individuals with MCILBP who demonstrate aberrant movements during forward bending. In a heterogeneous condition such as low back pain, analysis of motor control has been able to identify differences between participants, when they are placed into subgroups for analysis, rather than pooling all individuals with LBP into 1 group[35] The strength of this study is that it investigates a specific subgroup of patients with LBP, thus reducing the effects of heterogeneity and providing more specific information that may be relevant for treatment. Through separation of different forward bending movement patterns via kinematic analysis, we were able to characterize muscle activation and recruitment patterns that may be driving clinically observed aberrant movements. We identified differences in muscle activation within movement groups. We were also able to identify that these movement groups have unique muscle activation patterns during forward bending. However, there does not seem to be greater co-activation of the LES-TES within the aberrant groups. In contrast, the co-activation within LM-LES appears to be greater in TYP compared to the aberrant groups.

Differences in trunk extensor muscle activation between groups: mean muscle EMG amplitude

Our first finding identified muscle activation differences during forward bending between TYP and aberrant movement groups (Figure 2.1). LM was activated to a greater extent across all movement groups during forward bending, while LES and TES were activated on the same degree within the groups. This supports the theory that LM provides the predominant stabilizing force in forward bending[18]. However, the theory that aberrant movement may be a result of greater LES was not supported, as neither LES nor TES activation was significantly greater than LM in the aberrant groups. Both aberrant groups demonstrated significantly higher LM activation compared to TYP. However, aLPRJUD was distinguished by greater activation of all 3 muscles, compared to TYP. There was a moderate effect size for group (η^2=.217) and muscle (η^2=.383) main effects. Muscle activation differences between groups also exceeded the SEM. Collectively this indicates that these differences are clinically meaningful.

Prior research suggests that LM activation deficits are present in patients with MCI related LBP[19]. These findings are based on ultrasound imaging of muscle contraction with inference of activation rather than a direct measurement of the muscles electrical activity. Our findings identify *increased activation* of the LM in individuals with MCILBP and aberrant movements. These findings may not contradict, but rather support each other. A reduction in contractile ability of the LM within this subgroup of patients may require recruitment of additional motor units in an attempt to provide lumbar segment stability during movement.

Muscle activation pattern differences between groups: EMG amplitudes within movement epochs

Our findings demonstrated differences between muscles across epochs among the movement groups. There was a large effect size in the group by epoch interaction (η^2=.719), and group differences for muscle activation exceeded the SEM, indicating clinically meaningful findings. Within the TYP group, LM and LES reached peak EMG amplitude by 40% of movement and began returning to baseline by the end of forward bending (Figure 2.3). However, for the aberrant movement groups, peak LM and LES amplitudes occured later in the movement and the EMG amplitudes remained elevated. TES had a similar pattern across all groups in the 1st 50% of the movement. The activation peaks within the 5th to 6th epoch. Once activity peaks, aLPR begins to reduce activity and approached baseline activation levels. However, TYP and aLPRJUD did not return to baseline levels. TES activation in the TYP group had small fluctuations after the peak but remained fairly steady. Within the aLPRJUD group, TES activity continued to increase. Impairments in muscle contraction of the extensors identified by several studies in individuals with LBP[19,36,37] may require either additional muscle activation or maintained activation to provide lumbar segmental stability during movement. Studies on muscle quality and contractile abilities within this population would help to determine if that is the case.

Muscle activation pattern and aberrant movement

Group differences in hip to lumbar flexion angle ratios were not significant through the movement epoch (Figure 2.2b). However, the aberrant groups did have a greater proportion of hip flexion within in the first 20 percent of movement. The lack of

statistical significance was likely due to the greater proportion of hip flexion that *all* groups demonstrate at the end of forward bending. The ability of lumbar extensors ability to resist anterior shear in forward bending have been found to improve with greater hip flexion, due to changes in muscle fiber orientation.[23] The aberrant groups, both of which demonstrated aLPR, may alter the coordination of the lumbar and pelvic segments in order to take advantage of this mechanical phenomenon. Future studies investigating the coupling of the hip and lumbar spine during flexion and its effect on muscle orientation and vertebral shear may help to expand on this speculation.

Flexion-relaxation or electrical silence of the lumbar extensors at higher angles of lumbar flexion is attributed to increases in spinal stability from the passive tension of the lumbar extensors[38-40]. Peak stabilization from passive tension has been found to occur around 40 degrees of flexion and greatly reduce LES EMG activity.[41] The TYP group reached 40 degrees of lumbar flexion angle by the 5th epoch along with a reduction in LM and LES around that point. However, the aberrant groups maintained their lumbar flexion angle between 40-50 degrees, with continued activation of the extensor muscles (Figure 2.2a). They may require the muscle activation to enhance stability of the spine within this range if there is injury to the passive structures of the spine. However, the aLPRJUD group had distinct, high activation of all muscle groups compared to the TYP group. The aLPRJUD group was further separated by higher TES activation. The aLPRJUD group may have required additional TES activity to maintain spinal segment stability within this 40-50 degree lumbar flexion range that aLPR group does not. The higher degree of muscle activation during forward bending and the motor unit synchronization required to maintain that activation may account for the rapid acceleration and deceleration of judder

in this group.[21] What is unknown from this secondary analysis is why the aberrant groups restrict their lumbar motion between 40-50 degrees, and why the aLPRJUD group requires greater muscle activation.

Co-activation of muscles during forward bending

The hypothesis of greater co-activation across LES and TES in the aberrant movement groups was not supported. Cross-correlation coefficient between the LM-LES muscle pairing was significantly greater in TYP compared to the aberrant groups, suggesting greater co-activation between LM-LES in TYP. While LM had the greatest activation during forward bending, it may indicate that a synergistic activation between the LM and LES is also important to control forward bending.

The phase lags derived from the cross-correlation analysis did reveal an interesting finding. Phase lags suggest a cephalo-caudal order of activation of muscles. The only muscle pairing considered to fire from inferior to superior was the LM-TES pairing within the aLPRJUD group. However, the phase lag was only 0.6 ms with a large standard deviation. Within this group, the LM-LES phase lag was negative, suggesting LES was activated prior to LM. The LES-TES phase lag was also negative, suggesting TES was activated prior to LES. Therefore, it is likely that the TES was activated prior to LES. This has interesting implications. If the assumption within this secondary analysis is that TES and LES activation was greater in the aberrant groups to compensate for some deficiency in LM function, the activation order may suggest that this increase in muscle activation was a pre-planned feed forward mechanism. However, these interpretations

must be tempered carefully given the large variance in the data, the relatively small phase lags throughout muscles between groups, and the small effect size.

Limitations and considerations

Clinically, aberrant movement were based on participants selected through clinical examinations and selected on the presence of *at least 1* type of aberrant motion during forward bending.[6] To ensure that movement patterns were a regular pattern of behavior rather than a single isolated event, we selected participants based on the consistency of kinematic movement ratings. Therefore, these findings are only generalizable to individuals with consistent aberrant movement during forward bending. However, in these individuals, the findings provide insight into their neuromuscular control and may impact future intervention selection.

Increase in LES and TES activation have been identified in patients with recurrent LBP and is theorized to enhance spinal stability.[42] The results from the current study support the theory. Increased muscle activation may be influenced by fear of pain and resulting maladaptive increase in muscle activation.[9,43] However, our participants with LBP did not have $FABQ_p$ scores that suggest heightened fear of movement. Furthermore, participants in this study did not demonstrate an association between pain and fear of movement to other trunk movement tests.[25] It is also unlikely that pain drove the differences between the aLPR and aLPRJUD groups, as there were no significant group differences in pain intensity. It is unlikely that differences noted in this analysis are due to maladaptive behavior or fear of movement.

Because the purpose of this analysis was to identify the neuromuscular control of the trunk extensors, agonist-agonist muscle relations were not investigated. There may be

differences in agonist-antagonist muscle interactions that occur in patients with MCILBP that contribute to the aberrant movement patterns. Investigating the interaction of flexors and extensors during forward bending may provide further information about changes in neuromuscular control in patients presenting with aberrant movements. Increased extensor activation during forward bending and differences in extensor activation pattern within movement epochs were identified in patients with aberrant movements. However, several conditions may require this change in neuromuscular control. Increased signaling may have been necessary to improve spinal stability during bending. However, increase in electrical activity does not indicate that greater force generation occurred in the aberrant movement groups vs TYP. It may have been an attempt to activate a larger number of motor units due to deterioration of muscle quality. Future investigation into the muscle characteristics and quality in patients with these aberrant movements may yield more information to guide intervention.

Conclusions

The subgroup of individuals with MCILBP in this study appear to have two predominant aberrant movement patterns: aLPR and aLPRJUD. During forward bending, LM appears to be the largest contributor across all movement groups. Higher levels of extensor activity overall appears to distinguish the aberrant groups from individuals without LBP and typical forward bending movement. Several modifiable factors may be contributing to aberrant movements. There may be impairments in force generating properties of muscle that require recruitment of additional motor units. If that is the case, interventions that effect muscle capacity such as strength and endurance may need to be considered in treatment of these patients. There may be impairments in movement

coordination of the lumbar spine with the hip and pelvic complex. It may be reasonable to attempt restoring the movement pattern in patients to obtain similar movement and muscle activation patterns as the noted in individuals without LBP. This may be obtained through exercises that focus on improved movement coordination between the spinal segments as proposed by Sahrmann (2001) and O'Sullivan (2000).

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Tables and Figure

Table 2.1 Demographic information of participants used for analysis (mean ± standard deviation).

Group	Subjects Identified (N)	Sex	Age	BMI	Oswestry Disability Index	Pain	$FABQ_p$
Typical Movement	15	11 female (73%)	34.4 ± 11	23.5 ± 4.4 kg/m^2	NA	NA	NA
Altered Lumbopelvic Rhythm	14	10 female (71%)	38.2 ± 15	26.9 ± 6.1 kg/m^2	25.4 ±10.7	3.4 ± 1.2	16.2 ± 6.1
Altered Lumbopelvic Rhythm with Judder	10	7 female (70%)	37.2 ± 17	26.7 ± 4.7 kg/m^2	17.8 ±9.4	4.4 ± 2.2	12.5 ± 9.9

Note: No significant group differences (alpha >.05) for age, body mass index (BMI), Oswestry Disability Index, pain intensity, and Fear Avoidance Beliefs Questionnaire-physical activity subsection ($FABQ_p$). There was similar representation of sex within all groups.

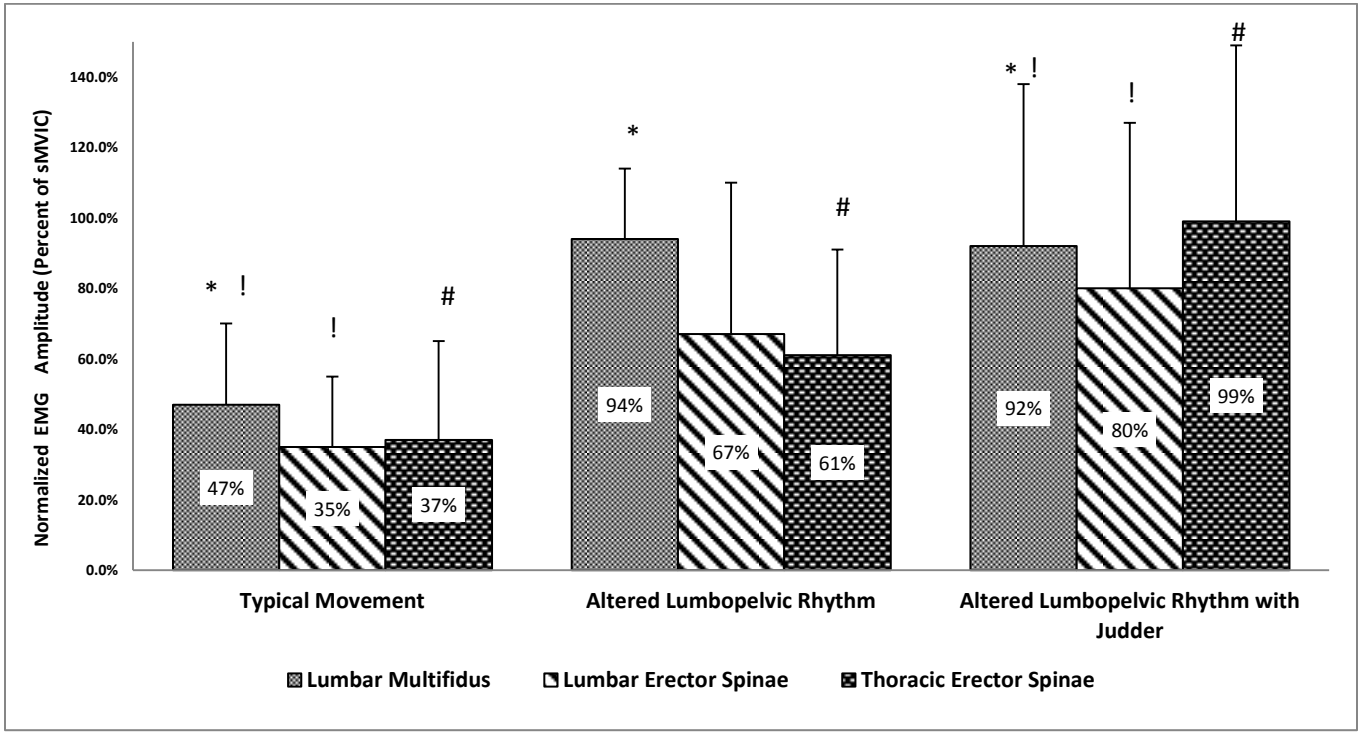


Figure 2.1. Mean normalized EMG amplitude across trunk extensor muscles (+/- SD) between individuals without LBP and typical movement, individuals with altered lumbopelvic rhythm, and individuals demonstrating altered lumbopelvic rhythm with judder during forward bending. The individuals without LBP demonstrated lower EMG amplitudes across all muscle groups in relation to the two aberrant movement groups. Lumbar multifidus recruitment was significantly higher in both aberrant groups compared to individuals without LBP (*). The group with altered lumbopelvic rhythm with judder had higher lumbar multifidus and erector spinae EMG amplitudes compared to individuals without LBP (!). This group also demonstrated higher thoracic erector spinae amplitude in comparison to individuals without LBP and the altered lumbopelvic rhythm group (#).

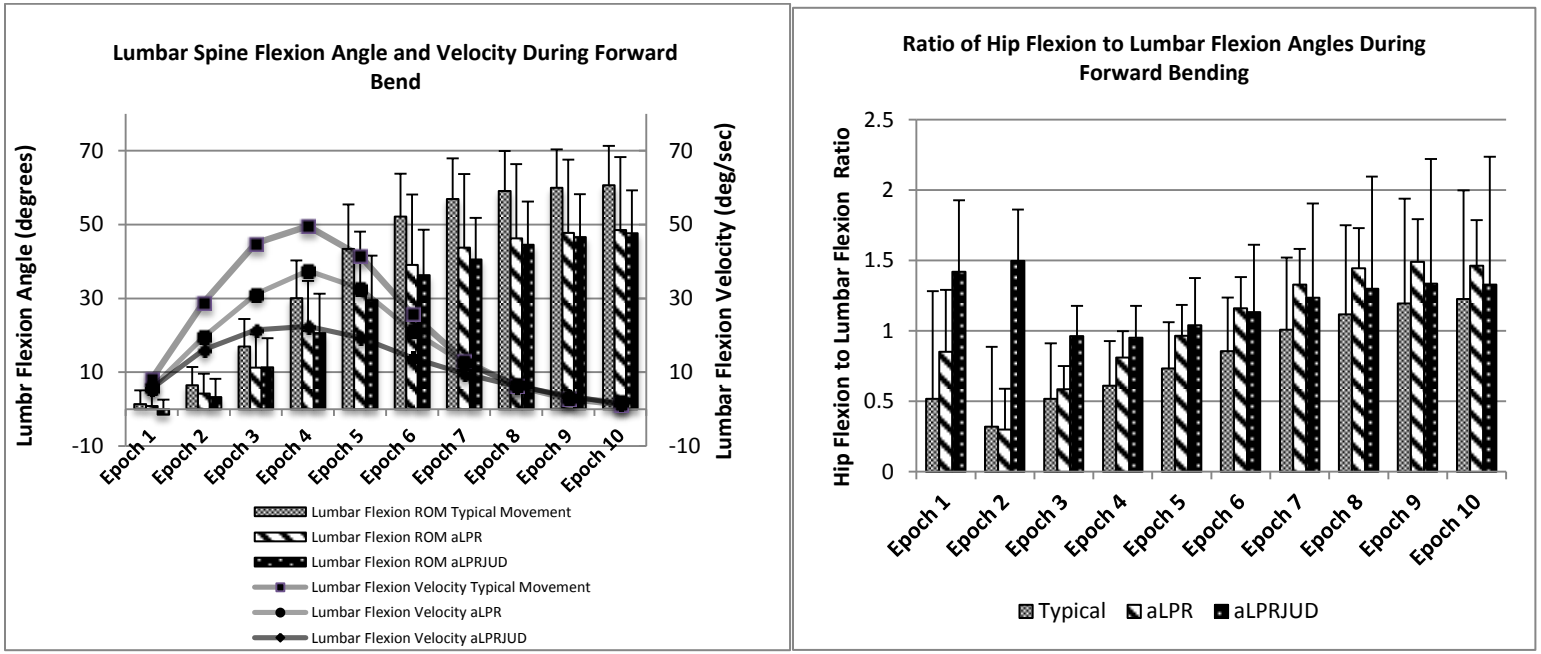
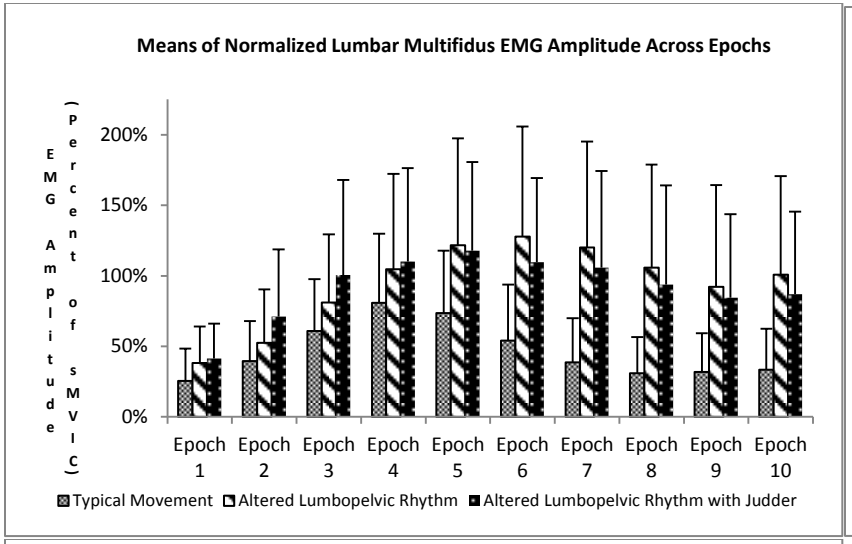
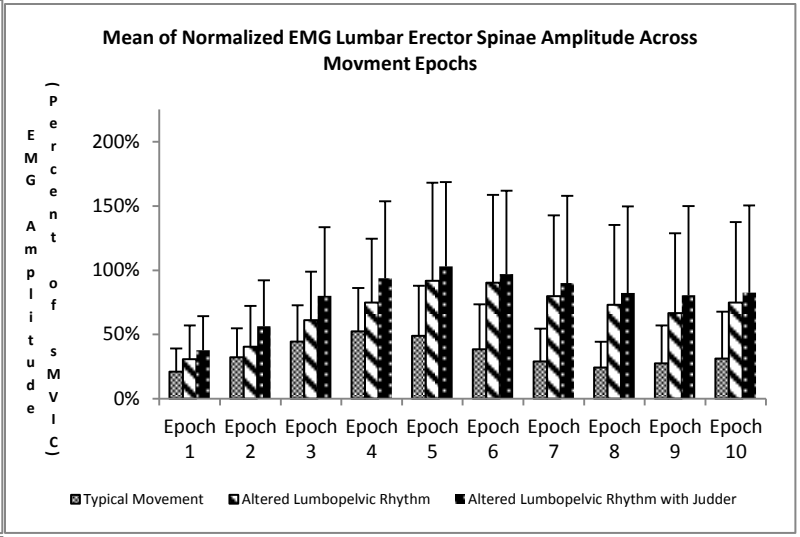
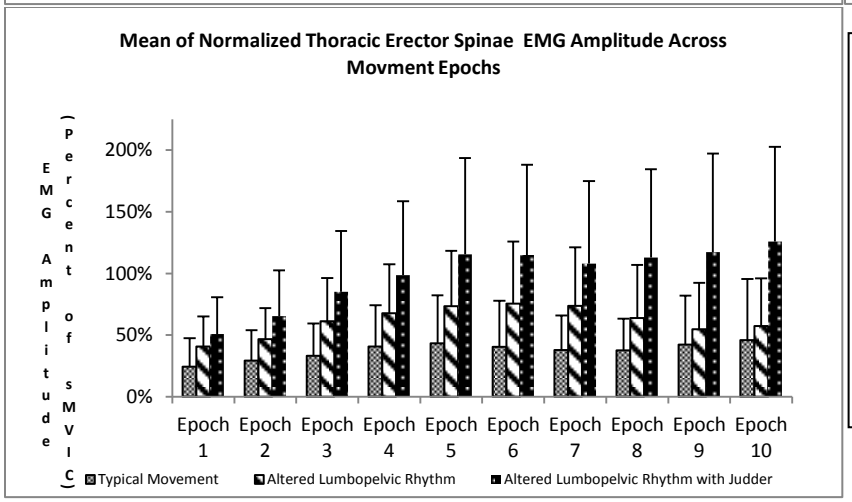


Figure 2.2 Graph of the kinematic descriptive data for lumbar flexion angles and velocity, and ratio of hip flexion to lumbar flexion. Data were time normalized with each epoch representing 10% of the forward bending. (2a) displays mean lumbar spine flexion angle and velocity within movement epochs across movement groups; (2b) displays the ratio of hip flexion to lumbar flexion angles during forward bending. Ratios greater than 1 represent greater hip flexion during movement.







Each epoch represents 10% of the forward bend task. In individuals without LBP, LM and LES demonstrated a tendency to reach peak amplitude by the 4th epoch and then reduce muscle activation levels close to baseline values as the motion progressed. However, in the aLPR and aLPRJUD groups, LM peaked between the 4th and 5th epoch and continued to remain active through the motion, not returning back to baseline levels. TES recruitment maintained stable across epochs in individuals without LBP , while the aLPR group tended to reach a peak between the 5th and 6th epoch before approaching baseline levels as movement progressed to full forward bending. In individuals with aLPRJUD the same peak in behavior was noted but there was no trend in returning to baseline values

Figure 2.3 Means muscle amplitude during epochs of forward bending.

Table 2.2 Mean, standard error of the mean (SEM), and standard deviation (SD) of cross-correlation coefficients and phase lag (τ) between trunk extensor muscle pairs during forward bending.

	Typical Movement				Altered Lumbopelvic Rhythm				Altered Lumbopelvic Rhythm with Judder			
	Cross Correlation Coefficient		Phase Lag (ms)		Cross Correlation Coefficient		Phase Lag (ms)		Cross Correlation Coefficient		Phase Lag (ms)	
	Mean	SEM	Mean	SD	Mean	SEM	Mean	SD	Mean	SEM	Mean	SD
LM-LES	**0.64**	0.02	-12.2	50.8	**0.55**	0.02	-55.4	101.3	**0.53**	0.03	-19.6	62.1
LM-TES	0.43	0.02	-15.6	100.2	0.41	0.03	-48.7	74.0	0.35	0.03	0.6	86.1
LES-TES	0.46	0.03	-14.2	79.8	0.48	0.03	-18.8	57.3	0.43	0.04	-8.8	43.2

Note: Individuals with typical movement demonstrated higher co-activation during the forward bending for the LM-LES pairing compared to the two aberrant movement groups. No differences were found between other muscle pairings. The phase lags suggest a top-down control for all movement groups during the forward bend, with exception to the LM-TES pairing in the aLPRJUD group. However, this lag time was less than 1ms.

Chapter 3: Spine stiffness changes and muscle activation of the lumbar extensors during the prone instability test

Abstract

Purpose: The prone instability test (PIT) is used to identify individuals with low back pain (LBP) that would benefit from trunk stabilization exercises. It is theorized that activity from muscles such as the lumbar multifidus (LM) during the leg raising portion of the PIT enhances spinal stiffness resulting in pain reduction. However, evidence to support this theory is lacking. The purpose of this study was to compare and contrast the following in individuals with LPB and healthy participants: 1) pain and spinal stiffness changes between testing conditions of the PIT and during electrically elicited contraction of the LM and 2) muscle activation patterns during the PIT leg raise.

Participants: Ten participants with LBP, 10 participants with no low back pain.

Materials/Methods: Three-dimensional kinematics was used to measure spinal stiffness using a bending beam model. Stiffness changes were compared across PIT conditions and compared between groups. Surface EMG was collected on trunk and limb musculature. Principle component analysis was used to extract muscle synergies.

Results: There were significant increases in stiffness during the PIT test conditions and with electrical stimulation of the LM within participants ($p=.001$). Patients with LBP also had significant reduction in their pain across conditions ($p=.017$). There were no between group differences in the percentage of stiffness change between PIT conditions ($p>.05$). Participants without LBP used three muscle synergies during the active leg raise:

1) lumbar stabilization 2) thoracic spine and pelvic stabilization, and 3) limb movement. Participants with LBP used only two muscle synergies: 1) thoracic spine stabilization and leg raising and 2) lumbar spine and pelvis stabilization.

Conclusions: Spinal stiffness changes occur during the PIT that can be reproduced with electrical stimulation of the LM muscle. Participants without LBP demonstrate a muscle synergy pattern where each synergy suggests a distinct function during the task. The muscle activation strategy of participants with LBP tended to use muscles in a more global pattern with less distinction between functions.

Introduction

Treatment of individuals with low back pain (LBP) can be challenging because the heterogeneous nature of the condition makes intervention selection difficult[1]. However, providing rehabilitation by subgrouping patients into treatment classifications has been found to have superior outcomes compared to unmatched treatments[2]. Trunk stabilization exercise (TSE) is one of these treatment classifications. The intervention is thought to address LBP resulting from impairments in muscle coordination in patients with spinal instabilities.[3,4] The interventions are thought to enhance muscle function and movement coordination to enhance stability of the spine.[5,6]

The prone instability test (PIT) is a clinical test that can be used to help predict which subgroup of individuals with LBP would benefit from trunk stabilization exercises.[3,7] The PIT is performed with the patient prone on an examining table with the trunk supported and the legs over the end of the table and feet on the floor. The test begins with assessing for painful lumbar segments through a clinician applied posterior to anterior (PA) force on the spine. If a painful spinal segment is present during PA testing, the individuals raise their feet off the floor using hip extension and the PA force is reapplied to the painful segment. The test is considered *positive* if pain is *reduced or eliminated* during PA force with active hip extension.[8,9]

Individuals with LBP who require trunk stabilization exercises are theorized to have impairments in the ligamentous or bony structures resulting in increased spinal segment mobility and reduced spinal stiffness.[10,11] Muscle recruitment modulated through neural control is believed to augment spinal stability in this condition.[12,13] Based on this theory, the PIT may be testing 2 constructs of LBP: pain from reduced spinal stiffness

and reduction in pain from muscle enhanced stability. However, mechanisms underlying pain with PA glide and reduced pain with active hip extension during this test are unknown.

Pain provocation tests, using PA force to the spine, have been determined to be more reliable than segmental mobility judgements[14] and are considered to be clinically acceptable tests.[15] Symptom provocation has also been found to have moderate to high correlations with atrophy of the lumbar multifidus (LM) at the tested segment.[16] Individuals with LBP identified to benefit from TSE, using the PIT as one predictor, have been shown to have LM activation impairments.[17] With LM reported to contribute up to 2/3 of lumbar spine stability[18], association of pain provocation with LM atrophy, and LM activation impairments in this LBP subgroup, it is plausible that the PIT may be detecting lumbar segmental impairment, as well as the ability of the LM to enhance spinal stability.

Voluntary contractions of the abdominal and lumbar extensor muscles have been found to increase spinal stiffness.[19] Isolated contraction of the lumbar extensor muscles at maximal or submaximal efforts (30-50% of the maximal volitional contraction [MVC]) have also been reported to increase spinal stiffness to external PA forces.[20,21] An exercise that is similar to the hip extension phase of the PIT has been shown to produce muscle activation levels between 60-80% of MVC of the thoracic and lumbar erector spinae musculature (TES, LES).[22] While that study offers some preliminary concept to the role of muscles during the PIT, it only details the role of the TES and LES and do not describe the role of the LM. There is also no description of other muscles of the upper and lower limbs such as the latissimus dorsi and gluteal muscles during the PIT. Both of these

muscles could have a role in spinal stabilization through anatomical attachments. There is also no information regarding spinal stiffness changes during the PIT.

Individuals with LBP have been noted to have impaired neuromuscular control, such as altered muscle activation patterns and delays in muscle onset and offset timing when compared to healthy participants.[23-26] Therefore, comparison of neuromuscular control between individuals with and without LBP may provide a better understanding of mechanisms during the test. Lastly, it is unknown whether spinal stiffness changes may be driving pain reduction during the test. The PIT relies on symptom reduction or elimination to predict success with TSE. Understanding the mechanism that drives symptom reduction during this test may translate to information that is useful in treatment planning for individuals with LBP.

Given the proposed role of the LM muscle in spinal stability, it would be valuable to understand the role of this muscle group during the PIT with regards to symptom reduction and enhanced spinal stiffness. Electrical stimulation has demonstrated the ability to produce contractions that are within 50% of the MVC of the lumbar extensors.[27] This is the same range that has been identified to increase spinal stiffness.[20,21] Therefore selective recruitment of the LM through electrical stimulation (LMES) during application of a PA force to individuals with segmental pain would allow for studying the role of this muscle during the PIT.

The first purpose of this study was to explore spinal stiffness changes as well as identify and compare lumbo-pelvic muscle activation patterns during the PIT among healthy participants and those with LBP. It was hypothesized that all participants would demonstrate increased spinal stiffness under posterior to anterior test force application

when progressing from the prone testing position to the leg raising position of the PIT. In those participants with LBP who achieved a positive PIT test, it was hypothesized that spinal stiffness changes would not be different from healthy participants. Based on the findings from Hebert, et al. (2010), it was hypothesized that individuals with LBP would have an altered pattern of muscle activation and decreased activation of the lumbar extensors during the leg raising portion of the PIT. This altered muscle activation pattern would be present even with a positive PIT finding. The second purpose of the study was to determine whether selective recruitment of LM would be able to reproduce the pain reduction and stiffness changes of the PIT in individuals with LBP who demonstrated a positive test. It was hypothesized that selective recruitment of the LM through LMES would result in pain reduction and stiffness increase in individuals with LBP and that the increased stiffness would not be different from that produced during the PIT test leg raise.

Methods

Participants

Power analysis (G-power) from a pilot study determined 4 participants were necessary to detect a difference of moderate effect size in stiffness within participants with LBP between the PIT conditions (Cohen's d=.5, beta=.95). The study was approved through (blinded) University's institutional review board. Ten individuals with recurrent or episodic LBP and 10 individuals without a history of LBP between the ages of 18-45 were recruited through flyers and word of mouth. Individuals without a history of LBP (NLBP) were included if they had no history of LBP that required medical intervention or limited their activity for longer than 3 days. Individuals with LBP were included if they

had current LBP that required medical attention or had limited their activity and function for no longer than 6 months. All participants were excluded if they had permanent structural spinal deformity (e.g., scoliosis), spinal fracture or history of spinal fracture, osteoporosis, active inflammatory joint disease, signs of systemic illness or suspected non-mechanical LBP (spinal tumor , cancer or infection), previous spinal or hip surgery, frank neurological loss, pain or paresthesia below the knee, leg length discrepancy of greater than 2.5 cm, current pregnancy, allergies to medical tape or adhesive, body mass index (BMI) greater than 30 kg/m^2, or performed rehabilitative exercises in the past with return to full function and no recurrence.

Individuals with LBP completed the Oswestry Disability Index for current functional status [28] and the Fear Avoidance Beliefs questionnaire.[29] All participants completed the Fear of Pain questionnaire-short form[30,31], which has been associated with ability to tolerate electrical stimulation.[32] All participants were also asked to rate pain using the numerical pain rating scale during PA compressions to the spine.[33] Participant demographics are detailed in Table 3.1.

Instrumentation

An electromagnetic tracking system (Liberty, Polhemus Inc., Colchester VT) was used to capture kinematic data using electromagnetic sensors placed on the spinous processes of L1 and S2(120 Hz collection frequency). EMG data were collected (SA Instrumentations, San Diego, CA gain 500; band pass filtered 20-500Hz) from the External Oblique (EO), LM, LES, TES, Latissimus Dorsi (LD), Gluteus Maximus (GM) and Hamstring (HS) muscles bilaterally using pairs of Ag-AgCl electrodes with 2cm

inter-electrode distance (Table 3.2).[34,35] Skin preparation for EMG involved cleaning with alcohol followed by light abrasion.

A custom apparatus consisting of a compression load cell (Transducer Techniques, Temecula, CA) was used to apply a PA force over the spinous processes of the participant's lumbar spine. Load cell data were streamed in real-time to a computer monitor providing visual feedback of the force applied by the examiner with a ±2.5% margin to ensure similar forces were applied to the spine during testing. An event marker was available to all participants to indicate the presence of pain during the application of the PA force. This allowed us to capture the amount of PA force applied to the spine that was associated with pain onset.

LMES was delivered through 4 bifurcated leads from 1 channel attached to 2.5cm^2 buffered electrodes using a clinical device (EMPI Continuum, Minnesota, USA) at 50 Hz, 400 μs pulse duration at minimum intensity of 20 mA. Frequency was selected at 50Hz based on the force-frequency findings of Russ et al. (2009). A study using infrared spectroscopy demonstrated that LMES can obtain preferential stimulation to the LM equivalent to 43% of the MVC with minimal recruitment of the LES.[36]

Procedure

Surface EMG electrodes were applied and 2 trials of resting EMG (30 second duration) were collected in supported quiet sitting. Following resting EMG collection, participants performed 2 trials of the modified Biering-Sorensen test, unilateral bridge (both sides), trunk flexion with tester applied resistance, along with bilateral resisted shoulder extension, and bilaterally resisted shoulder flexion (Appendix J) to obtain a

maximal volitional isometric contraction (MVIC) of the trunk and hip muscle groups. Kinematic sensors were placed on L1 and S2. Participants were placed in prone lying. Testing conditions for the PIT included 1) Full prone position: participants were prone with arms and legs on the table, 2) Resting position of the prone instability test (RPIT): participants were partially prone with the upper trunk on the table and feet on the floor, and 3) Prone instability leg raising position (PITLR): participants raised their feet off the floor via hip extension (Figure 3.1).

Stiffness testing: full prone position

A physical therapist specializing in spinal rehabilitation with 14 years of clinical experience performed all testing. Spinal stiffness and pain response in the full prone position was performed as a baseline condition with minimal to no muscle activation. This position was compared to the positions of the PIT. Participants were asked to take a deep breath then exhale, and refrain from inhaling during testing of each segment to minimize breathing artifact. A posterior to anterior force was applied by the examiner to the spinous processes of levels L1-L5. For participants with LBP the PA force was applied gradually until they reported pain production or increase in current pain. Participants indicated the presence of pain verbally and with an event trigger placed in their hand. Participants were asked to rate their pain from 0-10 on the numeric pain rating scale (0=no pain, 10= worst possible). The painful segment was marked and a visual target was set to the pain producing force (+/- 2.5%) and displayed to standardize the PA force that would be applied during testing across all PIT conditions. Next, participants underwent 2 trials of PA force application over the painful segment using the previously determined amount of force, with 2 minutes rest between trials. The force applied to the

spine was standardized in this manner within participants to compare stiffness changes between conditions. Participants with LBP that had more than 1 painful segment were asked to identify the most painful segment for testing. Within each trial, participants were asked to mark pain presence with the event trigger and rate the pain via NPRS.

NLBP participants received a standardized 22 N force at L3. L3 was chosen due to its midpoint location in the lumbar spine. The force was based on an average force needed to produce pain in pilot studies performed in the lab using a load cell and the clinic using a hand held dynamometer. EMG was collected on all individuals to ensure minimal muscle activity in the prone position during PA force application.

Stiffness testing: Resting Prone Instability Tests Position (RPIT)

Once testing was completed in the full prone position, participants were placed in the RPIT (Figure 3.1) with the edge of the table placed at ASIS level. They were instructed to have their arms overhead but not holding onto the table and their feet were resting on the ground with their knees extended. The painful segment was reassessed to confirm pain in this position using the same PA force as in full prone position. Once the painful segment was confirmed, the same PA force was reapplied while the participant refrained from breathing. Participants were asked to press the trigger when they experienced pain. Participants rated their pain at the end of the trial. Two trials were performed with 2 minutes rest between trials. EMG data were collected to ensure minimal muscle during testing in this position.

Stiffness testing: Prone Instability Test Leg Raise (PITLR)

Following 2 trials of stiffness testing in RPIT, a 24 inch high gate was placed above the participant's calf (Figure 3.1). They were asked to hold on to the table with the shoulder abducted less than 120 degrees and elbows flexed as necessary. Participants were instructed to raise their legs to the height of the gate keeping their knees extended. The same PA force and segment were tested. Participants were again asked to rate their pain (if any) and press the trigger if and when they experienced pain during the test. Two trials were performed. EMG was collected during PITLR. Participants also underwent 1 trial of the maximum force tolerated by the participant or that the tester could produce (PITLRmax). This step was included as there is no clear instruction on how much force should be applied to the spine during clinical testing of the PIT.

Stiffness testing: full prone position with LMES

Following PITLR, participants were placed in the full prone position and stimulating electrodes were placed 1.5 cm lateral to the spinous process of L5-L2 (Figure 3.2).[36] LMES was administered to 20 mA. If a tetanic contraction was not observed, the intensity was increased in 1 mA increments until a tetanic contraction was achieved. Once the stimulating dosage was set, a manual trigger was used to deliver 10 seconds of electrical stimulation. PA force was once again applied to the spinal segment 3 seconds into the LMES. Two trials were performed using the same spinal segment and force established in the full prone position.

Data Reduction and Statistical Analysis

PIT clinical results

Pain between the PIT conditions in participants with LBP were compared using paired t tests (alpha = 0.05). Differences in the PA force used during the test between healthy participants and individuals with LBP were assessed via independent t-tests (alpha =.05). No adjustments were made to alpha based on low, non-statistically significant correlations between the measures.

Spine Stiffness

An elastic beam model to measure changes in bending stiffness of the lumbar spine during posterior to anterior mobilization was used to measure spinal stiffness.[37] A formula has been derived to model spinal stiffness to PA force on the lumbar spine:

$$EI = \frac{\frac{Pb}{2L}[(e+a)d + ab]}{\theta_{L1S}}$$

where stiffness (EI) is defined by pressure applied to the spine (P), the distance between the rib cage cantilever and the sacrum cantilever (L), the horizontal distance between the pressure applied to the spine and the sacrum (b), the horizontal distance from the rib cage cantilever to the pressure applied (a), and the maximum angular displacement of the spine (θ_{L1S}).[38] Rotation of the L1 sensor about the S2 sensor was measured in degrees, as force was applied to the identified segment in patients with LBP and at L3 in healthy participants. Bending compliance of the spine during force application was expressed as a compliance slope of the force (y axis) against the angle change (x-axis) and expressed in (N·m/deg). Plots of this data while participants received PA force in the full prone position revealed a that a linear equation represented the line of best fit for the data with a

median r^2=.96, (min=.75, max=.99). Stiffness of the spine was expressed as the inverse of compliance (1/compliance slope) expressed in ($N \cdot m/deg^{-1}$). A within day reliability study of 5 participants was done to assess intra-rater reliability of obtaining spinal stiffness measures in the full prone position ($ICC_{(2,1)}$, =.90; standard error of the measure (SEM)=2.1 $N \cdot m/deg^{-1}$), RPIT ($ICC_{(2,1)}$,=.79; SEM=4.7 $N \cdot m/deg^{-1}$), and during PITLR ($ICC_{(2,1)}$,=.95; SEM=1 $N \cdot m/deg^{-1}$).

Comparison of stiffness within and between participants

All comparisons were performed using SPSS 21(IBM, Armonk NY). Stiffness data were non-normally distributed (Kolmogorov-Smirnov test, p<.05). Therefore the data were transformed using an inverse transformation. Repeated measures ANOVA was conducted to assess differences in stiffness between the positions/ test conditions of prone, RPIT, PITLR, PITLRmax, and LMES (alpha=.05). This was performed separately for NLBP participants and participants with LBP. Planned comparisons were made between prone and RPIT, RPIT and PITLR, PITLR and PITLRmax, prone and LMES, RPIT and LMES, and PITLR and LMES. Alpha was set to .05 with a significant Omnibus on the repeated ANOVA.

Percent change in spinal stiffness were non-normally distributed (Kolmogorov-Smirnoff test, p<.05). Therefore data were transformed using an inverse transformation. Differences between groups were compared using the percent change in stiffness between the PIT testing positions. Percent change was calculated as [(final value – initial value/initial value x 100)] and compared using independent t-test for independent samples with Bonferroni correction for multiple comparisons (alpha=.0125)

Muscle Activation during PITLR

EMG data collected during testing had heart rate artifact removed via fast ICA[39] and resting EMG subtracted. Data were then normalized and represented as a percentage of the MVIC. EMG data were non-normally distributed (Kolmogorov-Smirnov); therefore the data were transformed using an inverse transformation (1/EMG). EMG data were averaged by side because there were no significant side to side differences ($p<.05$). Principle components analysis (PCA) was first used to extract muscle synergies of NLBP participants and participants with LBP with suppression of correlations below .5 and Bartlett's test of sphericity (alpha=.05). Muscles that loaded onto synergy components were entered into a mixed-model ANOVA (within participants: muscle; between participants: group) and individual muscle activation amplitude levels were compared during the PITLR. Post-hoc analysis using Fisher's Least Significant Difference was performed on significant interactions and effects.

Results

PIT clinical results

Clinical results of the PIT test are presented for descriptive purposes. No healthy participant demonstrated pain during any of the PIT conditions. Nine of 10 LBP participants had pain provocation in the full prone position. All 9 patients with LBP had a positive PIT. Eight of the 9 had pain elimination with PA force during LMES. Force used and pain produced between the tests conditions are detailed in Table 3.3. There were no significant differences in force used in the PIT conditions between healthy and low back

pain participants (p>.05). Pain during PA force was significantly less in the PITLR, PITLRmax, and LMES conditions compared to full prone and RPIT conditions in participants with LBP (p<.05). There was not a significant difference in pain between the full prone and RPIT conditions (p>.05).

Stiffness between conditions

EMG data from the full prone position revealed muscle activation ranging from 0% ± 1% (TES) to 9% ± 2% (EO) of the MVIC across all participants. EMG activity ranged from 4%± 12% (EO) to 13% ±19% (G.Max) of the MVIC for the resting PIT or start position of the test across all subjects. There was no significant difference (p>.05) in muscle activity between groups in full prone nor RPIT.

NLBP participants

A significant difference in spinal stiffness was found between the full prone and PIT conditions within *healthy participants* $F(4,36)=4.731$, $p=.001$, $\eta^2=.345$. Planned comparisons revealed stiffness to be significantly greater in PITLR (p=.042, d=2.11) and LMES (p=.007, d=1.01) compared to the full prone position. Stiffness was also significantly greater in PITLR (p=.039, d=.385) and LMES (p=.029, d=.46) compared to RPIT (starting position of the PIT). There were no significant differences in stiffness between full prone and RPIT (starting position of the test) (p=.33, d=.46). There was no significant difference in stiffness in LMES (p=.912, 2=.239) nor PITLRmax (p=.91, d=.21) compared to PITLR.

Participants with LBP

A significant difference in spinal stiffness was found between the full prone and PIT conditions within the LBP group $F(4,36)=7.387$, $p=.017$, $\eta^2=.831$. Planned comparisons revealed stiffness to be significantly greater in RPIT ($p<.001$, $d= 1.75$), PITLR ($p<.001$, $d=1.28$), and LMES ($p=.0135$, $d=.956$) compared to the full prone position. Stiffness was significantly greater in PITLR ($p=.021$, $d=.219$) and LMES ($p=.025$, $d=.488$) compared RPIT. There was no significant difference in stiffness in the LMES ($p=.588$, $d=.326$) nor PITLRmax ($p=.91$, $d=.038$) compared to PITLR.

Stiffness change between groups

No significant difference in the percent change of stiffness in RPIT ($p=.432$, $d=.072$), PITLR ($p=.91$, $d=.03$) nor LMES ($p=.97$, $d=.2$) were found with respect to the full prone position between NLBP participants and those with LBP. There was also no significant difference between groups for the percent of stiffness change from RPIT to PITLR ($p=.45$, $r=.38$).

Muscle Activation during PITLR

NLBP participants

PCA yielded 3 components in the synergy extracted from NLBP participants, accounting for 93.2% of the variance during PITLR (KMO=.48, Bartlett's test=.006). The first synergy accounted for 41.8% of the variance and included the LD, LES, and LM. The second synergy accounted for 31.7% of the variance (73.5% cumulative) and

included the TES and G.Max. The third synergy accounted for 19.7% of the variance (93.2% cumulative) and consisted of the HS.

Participants with LBP

The synergy extracted for participants with LBP yielded 2 components accounting for 77.3% of the variance during the PITLR (KMO=.534, Bartlett's test=.048). The first synergy accounted for 56.8% of the variance and included TES, LD, and HS. The second synergy accounted for 20.5% of the variance (cumulative 77.3%) and included LES, LM, and G.Max (Table 3.4).

Group Comparison of Individual Muscle Activations

The external oblique muscle group did not load on any synergy for either group. Therefore, it was left out of the individual muscle activation comparisons. The result of a mixed model ANOVA comparing muscle activity between groups during the PITLR demonstrated significant differences across muscles, $F_{(5,13)=}4.475$, $p=.014$, $\eta^2=.314$. There was also a significant interaction between the healthy and LBP participants across muscles $F_{(1,17)}=5.628$, $p=.03$, $\eta^2=.249$. Table 3.5 contains EMG values of muscle activation as a percentage of the MVIC. Post hoc analysis revealed no significant activation differences between the muscles in healthy participants ($p>.05$). In patients with LBP, both G.Max and HS had significantly less activation compared to TES, LES, and LM ($p<.05$). There were no significant differences between G.Max and HS. There were also no significant differences between TES, LES, and LM. There was a significant difference between LD and G.Max ($p=.034$). Between group comparison revealed

significantly greater activation of the LES (p=.043), LM (p=.016), and G.Max (p=.016) in healthy participants versus patients with LBP.

Discussion

Statistically significant increases in spinal stiffness were found between the full prone position and the PITLR testing position within participants with LBP that were similar to NLBP participants. This validates the clinical assumption of the test that associates the leg raise with stiffening of the spine. The EMG findings demonstrate differences in muscle activation synergies and the level of individual muscle activations in participants with LBP versus those without LBP. However, the participants with LBP were still able to reduce their symptoms. The change in muscle activation may provide an adequate compensation to reduce pain in patients with positive PIT test results. Interestingly, preferential recruitment of LM through LMES was also able to reproduce spinal stiffness increases and pain reduction when individuals with LBP were in the full prone position. This supports the important role of the LM in modulating lumbar spine stiffness. It also suggests spinal stiffening may play a role in the reduction of pain against PA force. This may suggest a role for LMES to enhance current rehabilitation to target this muscle.

Spinal stiffness changes during the PIT

Figure 3.3 displays stiffness changes within groups for both LBP and healthy participants. Both groups demonstrate an increase in stiffness from the full prone position to the PITLR with LBP participants demonstrating significant reduction in pain during the PITLR. The increase in stiffness from full prone to PITLR exceeded the SEM with

large effect sizes (NLBP d= 2.11; LBP d=1.78). Both groups also demonstrated a significant change in spinal stiffness from RPIT to PITLR that exceeded the SEM, while demonstrating moderate effect sizes (NLBP d=.385, LBP d=.219). Increase in spinal stiffness with PITLR that exceeded measurement error and demonstrated a moderate to large effect sizes indicate meaningful findings that were linked to *clinically relevant* results of the PIT for those with current LBP. This has important clinical implications, as it suggests muscle activity associated with the PITLR does contribute to an increase in spinal stiffness during the test, which is performed between the RPIT and PITLR conditions. These changes in spinal stiffness support the assumption that muscle activity during the PITLR is associated with an increase in spinal stiffness.

Participants with LBP had pain provocation in in the RPIT that was similar to the full prone position. However, their stiffness increased significantly from full prone to the RPIT position. In contrast, healthy participants did not have a significant change in stiffness from full prone to the RPIT position. There was some electrical activity across both groups in the RPIT position that may explain for some of the increase in stiffness from full prone to RPIT position. However, the muscle activity in the RPIT was similar in both groups. Increase in stiffness in the RPIT may be attributed to passive tension of the lumbar extensor muscles. Reduction in spinal stability could require greater use of the lumbar muscles, resulting in muscle hypertrophy in individuals with LBP that may result in resting tissue stiffness across the extensors.[40] The extensors may be on more tension within the RPIT position, resulting in an increase in RPIT stiffness. Differences in muscle length across the hips may also be a factor that was not measured in this study. Lastly,

there is evidence of an increase in muscle stiffness in patients with LBP[41,42], but it is unknown if this would alter stiffness in the RPIT position.

LMES during application of PA force was performed in the full prone position due to better experimental control and a smaller SEM in that position, which would allow for better detection of change in spinal stiffness. Both participants with and without LBP had significant increase in spinal stiffness with LMES, with a large effect size (NLBP d=1.01, LBP d=.956) that exceeded SEM, indicating clinically meaningful differences. Participants with LBP had a significant reduction in pain against PA force with LMES, along with stiffness changes with respect to the prone position that were similar to participants without LBP (Figure 3.4). The results suggest that preferential muscle activation of the LM can replicate positive PIT results along with increases in spinal stiffness in participants with LBP.

Muscle Synergies and Level of Activation during the PIT

PCA extracted muscle synergies with different characteristics for individuals with and without LBP. Participants without LBP had 3 components or muscle synergies that explained a large percentage of the variance. Items within a component are considered to be acting in unison to contribute to some aspect of the synergy.[43] The first component in healthy participants explained the majority of the variance and included the LD, LES, and LM. The LD has an attachment to the spine via the thoracolumbar fascia while the LM and LES have bone/tendon interface with the lumbar spine and are considered intrinsic stabilizers[44]. This may be a lumbar spine stabilizing synergy. LES and LM are likely stabilizing the lumbar spine while the LD may be supporting the trunk on the table, while

providing some contribution to lumbar spine stability through the fascia. The second component included TES and G.Max and may be associated with stabilizing the thoracic spine and pelvis respectively during leg raising. While the G.Max is a hip extensor and may be contributing to raising the feet off the ground, it may have a larger role in it's interaction with the thoracic spine to stabilize the pelvis due to its loading on the 2nd component. It is also possible that the posterior pelvic tilt moment created by the gluteus maximus when raising the feet off the ground[45] require the TES to counterbalance that action. The third component of the synergy may play a larger role in lifting the feet off the ground and is represented by the hamstring.

In participants with LBP, only two components were extracted by the PCA. While the same muscle groups appeared in the muscle synergies of the LBP patients as the healthy controls, they loaded in different combinations on different components, and accounted for different proportions of the total variance. The first muscle synergy accounted for a majority of the variance during PITLR consisted of TES, LD, and HS. Given the action of these muscles and their attachments, they may be working to accomplish more than 1 role within the PITLR, thoracic spine stabilization and leg raising. TES and LD may be functioning to maintain the upper trunk on the table during leg raising. The TES and LD have an extension moment on the spine and may also be working to counterbalance the flexion moment caused by posterior tilting of the pelvis created by the hamstring during leg raising. The latter action that is proposed may also be contributing to gross trunk stability during the PITLR. One possible explanation to this response in participants with LBP could be due to the lower activation of the LM and LES, which loaded onto the second muscle synergy with the G.Max and accounted for

less variance in the PITLR. Within participants with LBP, this synergy may be providing lumbopelvic stability during the leg raising. However, because muscle activation is significantly lower than participants without LBP, these muscles may not be as effective in contributing to stability of the lumbopelvic region. Despite the different strategy, the percent change in stiffness from the full prone to PITLR positions between groups was similar (Figure 3.4). There was no statistically significant difference in the percent stiffness change, and the small effect size suggests that any differences between the groups were not clinically meaningful.

Limitations

Our findings should be interpreted with consideration of the difference in sex distribution between the healthy and LBP participants. There are likely anthropometric differences that may have an impact on findings. A large factor may be in the gynecoid pelvis of women, which may impact orientation and length tension relationship of all trunk muscles that are attachments on the pelvis. This may impact how muscles contribute to the PITLR . However, hip extension is in the sagittal plane and may not be affected as greatly by different bony dimensions in the frontal plane of a gynecoid pelvis.

LMES was assumed to preferentially activate the LM based on prior studies within our lab[36] (Appendix K). However, based on anthropometric and morphologic differences across patients, this is difficult to generalize. However, even if LES was recruited partially within the participants, our PCA suggests that both LM and LES may function as lumbar stabilizers during this test. Therefore, the findings would still be applicable in demonstrating that the lumbar extensors may have a considerable effect in

the mechanism of the PIT. Pain elimination during the use of LMES (secondary to gaiting from the ES) may have contributed to the effects.[46] However, pilot studies conducted in our lab using pain pressure thresholds along the spinous process of the lumbar spine in healthy participants during LMES did not yield any significant reduction in perceived pain as a result of neuromuscular electrical stimulation (Appendix K).

Many aspects of the PIT were standardized to study the mechanism of the test, without having a clear indication of all testing variance that may exist in the clinical settings. This impacts generalizability and external validity of these findings. To study muscle activation within the test, leg raising was set to 24 inches for all participants. To study stiffness changes across the different PIT conditions from full prone to PITLR, the amount of force that was delivered was kept constant for each participant. However, even when maximal forces were applied, patients still had pain reduction during the test. Given the uncertainty of testing procedure across clinicians, further clinical standardization of this test may improve overall reliability and validity of this test.

In a commentary on the reliability of the PIT, Hicks (2011) mentioned the effect of introducing variability on the test findings. While standardizations occurred for the sake of experimental control, the investigators made the best attempt to perform the test as described in order to maximize external validity. Patient positioning, leg positioning and knee positioning, and arm positioning were all matched to the test as best as possible. Where the force applied to the spine during testing is not expressly stated, the investigators applied similar forces to limit confounding variability to the study, but did perform 1 trial with a maximal examiner possible force. Based on these measures, we feel

that testing was performed as close as possible to the clinic situation while still providing adequate controls.

We are also unable to state whether muscle activation has a direct role in spinal stiffening, but may be able to provide some supporting evidence to this possibility. There were increases in spine stiffness from the RPIT to PITLR conditions across both groups. More importantly, we were able to obtain an increase in stiffness in patients with LBP and healthy participants from prone to LMES. This was statistically significant with large effect sizes in both groups. This may support the construct that muscle activity can result in spinal stiffness along with pain reduction.

PA forces were applied manually by a clinician. Other studies of similar methods used an instrumented device to standardize the rate of PA force application. In order to maintain generalizability of findings, clinician applied forces were used. While this may be a limitation, intra-rater reliability for stiffness measures were high for all testing conditions. There was a linear relationship between the force applied and spinal bending for all participants. Lastly, fidelity tests of the investigator applying the load (WS) was performed on randomly drawn participants in this study. Rate of force application was calculated for each participant across conditions. There was less than a 10% coefficient of variation in the rate of force application during the study (Appendix D). These findings combined would suggest a fairly consistent rate of force application.

Conclusion

Spinal stiffness increases with pain reduction during the PITLR were found in patients with LBP. Similar pain reduction and spine stiffening was noted with LMES to patients with LBP. That, combined with other studies[19,21] may support the ability to

obtain spinal stiffening through muscular activation. Therefore, there may be sufficient evidence to state that muscle activation during the test increases spinal stiffness resulting in pain reduction. There also appears to be differences in muscle activation strategies between patients and healthy participants during the leg raising that may describe a preference for use of a global stabilization system rather than intrinsic muscles in individuals with LBP presenting clinically similar to our participants.

Clinical Implications

Reproduction of positive test results with LMES may have important implications in patients who are unable to reduce or eliminate pain from PA force during PITLR. These patients would be predicted to fail with TSE. This may be due to reduced LM activity or inability to develop a successful compensatory mechanism. However, performing the PIT in these patients with a negative PIT with LMES may aid in determining if the patients has adequate LM muscle cross sectional area and/or quality to have a positive result. Based on the synergies however, strengthening alone may not be adequate for improvement in function. Individuals with LBP may be relying more on a global stabilization system. If that is the case, focus on facilitating intrinsic spinal stabilizers (O'Sullivan, 2000) such as the LM may be necessary.

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Tables and Figures

Table 3.1 Participant demographics.

	Sex	Age	BMI (kg/m2)	Trunk length (cm)	Skin Thickness (mm)	Fear of Pain	$FABQ_P$	Oswestry Disability Index
NLBP	2 Female	28.5 (5.9)	22.6 (2.3)	55.1 (4.2)	6 (2)	23 (6.7)	--	--
LBP	5 Female	28.8 (3.1)	23.5 (1.4)	50.8 (3.8)	6 (2)	21.5 (5.1)	2.9 (3.9)	17.4 (17.1)

Mean (SD) Age, body mass index (BMI), trunk length, skin thickness, Fear of pain (FOP), Fear Avoidance Beliefs Questionnaire Physical subjection ($FABQ_P$) and Oswestry Disability Index.

Figure 3.1 Prone, Resting position of the prone instability test (RPIT) and leg raising (PITLR)

Table 3.2 Surface EMG electrode placement.

Muscle	Location	Muscle	Location
Gluteus Maximus (G.Max)	Midpoint between the lateral edge of the sacrum and greater trochanter	**Thoracic Erector Spinae (TES)**	5cm lateral to T9 spinous process
Hamstring (HS)	15cm from the ischial tuberosity	**Lumbar Erector Spinae (LES)**	3cm lateral to L2 spinous process
Lumbar Multifidus (LM)	2cm lateral to L5 spinous process	**Latissimus Dorsi (LD)**	Midline between spinous process of T9 and axillary line
External Oblique	15 cm lateral to umbilicus		

Table 3.3Mean (SD) load and pain during the prone instability test (PIT) conditions and with Lumbar multifidus electrical stimulation (LMES).

	Healthy	LBP
Load (N)	22 (0)	24.4 (8.8)
PITLRmax Load	41.1 (13)	42.1 (12.9)
Pain Prone	0 (0)	4.6 (2)
Pain RPIT	0 (0)	4.1 (1.7)
Pain PITLR	--	0.33 (0.7)
Pain PITLRmax	--	0.6 (1.4)
Pain LMES	0 (0)	0.5 (1.3)

Note: The same load was used in prone, resting position of the prone instability test (RPIT), and prone instability test leg raising position (PITLR).There was no difference in loads applied during the maximal loading segment of the PITLR (PITLRmax) between healthy subjects and those with low back pain

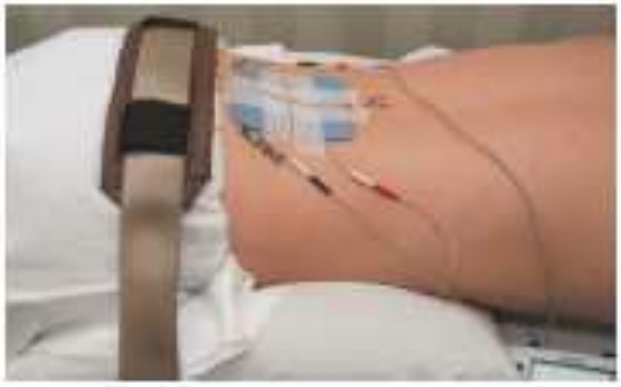

Figure 3.2 Electrode placement for electrical stimulation to the lumbar multifidus

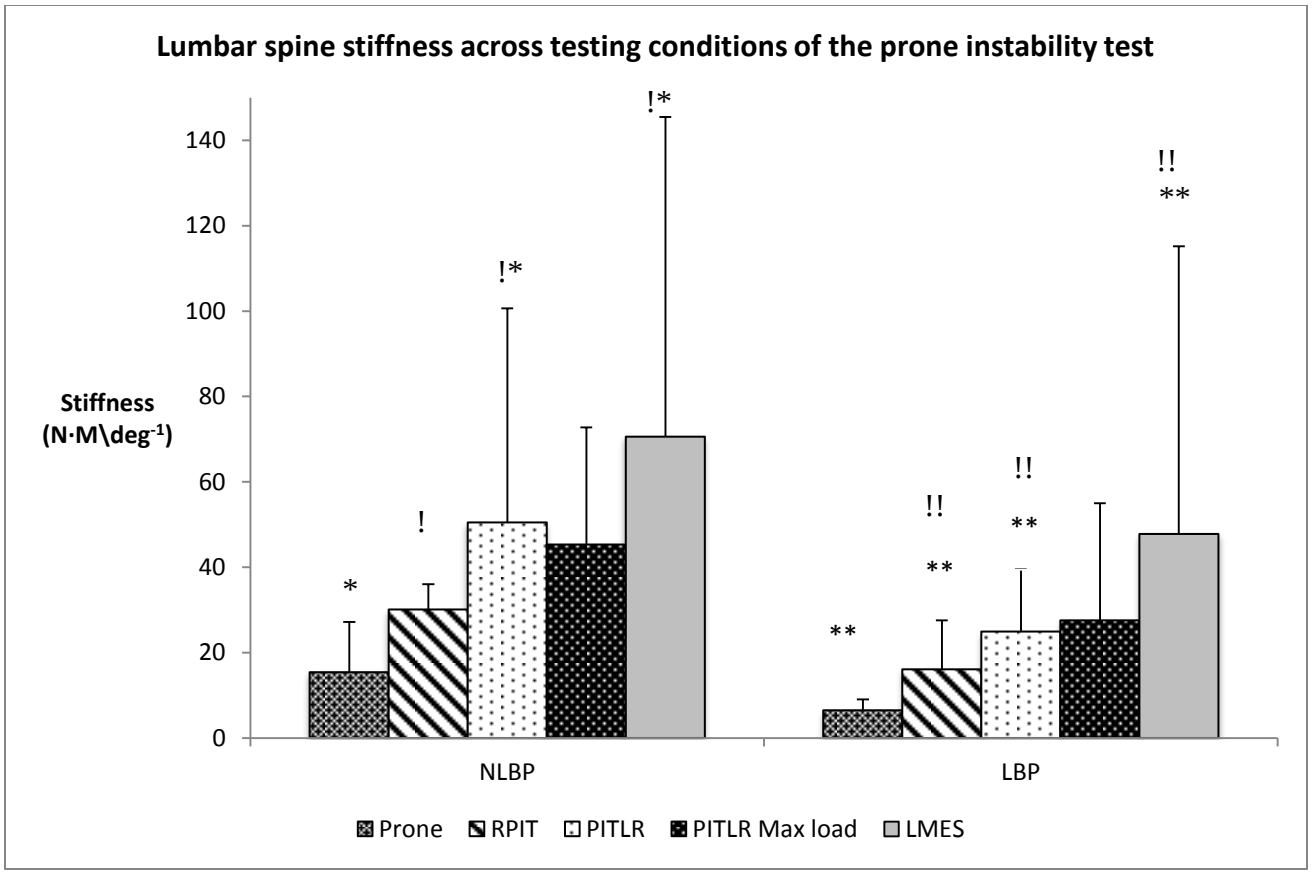

Figure 3.3 Mean spinal stiffness during testing conditions. Participants without low back pain (NLBP) had a significant increase in stiffness in the prone instability test leg raising position (PITLR) and lumbar multifidus electrical stimulation (LMES) positions compared to prone (*) and resting position of the prone instability test (RPIT) (!). Participants with low back pain (LBP) demonstrated significantly greater stiffness in RPIT, PITLR, and LMES compared to prone (**). Stiffness was also significantly greater in PITLR and LMES compared to RPIT (!!).

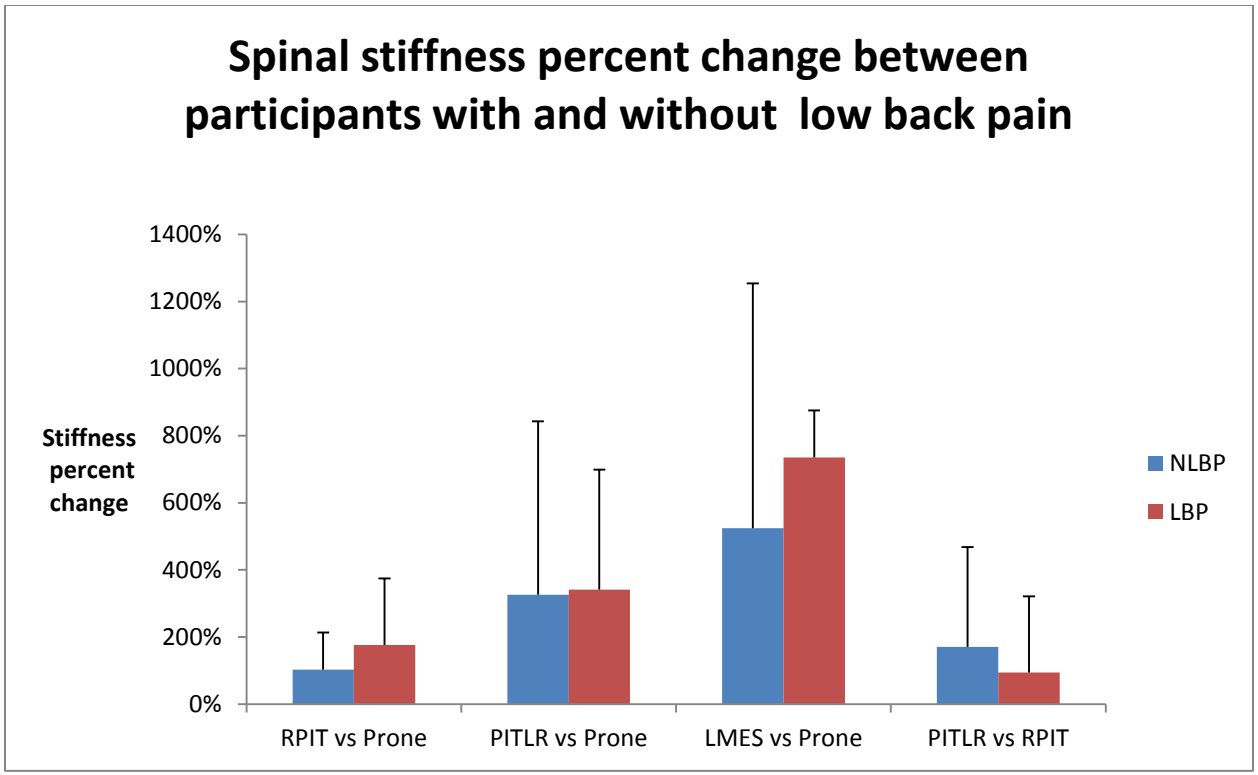

Figure 3.4. Mean percent change for group differences between testing conditions. There was no statistically significant difference in the percentage of stiffness change in testing conditions between participants without low back pain (NLBP) and participants with LBP.

Table 3.4. Muscle synergy extraction results from principle components analysis with matrix correlation values for muscles and variance accounted for (VAF) per synergy.

NLBP						LBP			
Synergy 1 (41% VAF)		Synergy 2 (31.7%VAF)		Synergy 3 (19.7% VAF)		Synergy 1 (56.8% VAF)		Synergy 2 (20.5% VAF)	
Muscle	Matrix Correlation	Muscle	Matrix Correlation	Muscle	Matrix Correlation	Muscle	Matrix Correlation	Muscle	Matrix Correlation
LD	.98	TES	.92	HS	.98	TES	.91	LES	.77
LES	.93	G.Max	.93			LD	.73	LM	.94
LM	.73					HS	.89	G.Max	.95

Note: Variance accounted for (VAF)

Table 3.5. Mean (SD) of normalized muscle activation during the prone instability test leg raising phase (percentage of the sMVIC).

Muscles	Healthy	LBP	Percent Difference
External Oblique	22% (17%)	12%(14%)	45%
Thoracic Erector Spinae	60% (28%)	62% (69%) ! #	3%
Latissimus Dorsi	84% (62%)	38% (32%) *	55%
Lumbar Erector Spinae	63% (24%)	41% (12%) ! #	35%
Lumbar Multifidus	65% (26%)	44% (17%) ! #	32%
Gluteus Maximus	56% (52%)	16% (17%) ! *	71%
Hamstring	43% (30%)	29% (19%) #	33%

Note: The third column represents percent difference in activation of patients with LBP compared to healthy subjects. External oblique is presented for descriptive information but was not included in the comparison of muscle activation due to results of the principle components analysis. There was significantly greater activation of the lumbar erector spinae, lumbar multifidus, and gluteus maximus in healthy subjects versus patients with LBP. There were no differences within muscles for the healthy subjects. Patients with LBP demonstrated no significant differences between thoracic erector spinae, lumbar erector spinae, or lumbar multifidus ($p>.05$). There were also no differences between gluteus maximus or hamstrings ($p>.05$). However, there were significant differences between the gluteus maximus and the thoracic erector spinae, lumbar erectors spinae, and lumbar multifidus (!) ($p<.05$). There were also significant differences between the hamstrings and the thoracic erector spinae, lumbar erector spinae, and lumbar multifidus (#) ($p<.05$). Lastly, there was a significant difference between the gluteus maximus and latissimus dorsi (*) ($p<.05$).

Chapter 4: Effects of neuromuscular electrical stimulation induced fatigue of the lumbar multifidus on forward bending kinematics

Abstract

Background/Purpose: Observation of aberrant movement during forward bending is used to predict individuals with low back pain (LBP) who would benefit from trunk stabilization exercises. Dysfunction of the lumbar multifidus muscle may play a role in the development of aberrant movement. The purpose of this study was to determine whether fatigue from neuromuscular electrical stimulation to the LM (LMES) would reproduce deviations from typical forward bending patterns in individuals without LBP.

Subjects: Nine individuals without a history of LBP and no observed aberrant movement on visual screening participated in this study.

Methods: Kinematics data were collected during forward bend pre and post electrical stimulation to fatigue the LM. Forward bending kinematic data were rated, pre and post LM fatigue for presence or absence of aberrant movement using predetermined criteria. Chi-Square analysis was performed to compare forward bend classification changes following LMES.

Results: There was a significant change in individual's forward bend classifications following LMES ($p<.05$). Six of 9 participants had a change in forward bending classification following electrical stimulation. Three individuals with kinematic patterns rated as typical movement pre LMES, had production of aberrant movement following LMES. However, 3 had elimination of aberrant movement that was noted in their pre fatigue kinematic patterns.

Conclusions: There were changes in forward bending classification of individuals following LMES. However, not all individuals had changes consistent with a worsening of their movement pattern as expected. Failure to demonstrate deviation from typical forward bending may have resulted from an inability to fatigue LM in some individuals, potential recovery from fatigue prior to post-test forward bending trials, or presence of robust musculoskeletal system in healthy individuals that compensated for fatigue of one muscle group. Conversely, LM impairments may not be the sole contributor to aberrant movements.

Introduction

Observational rating of forward bending is an important clinical measure in many examination pathways for low back pain (LBP). Pain behavior and movement quality during forward bending are often used for treatment classification for individuals with LBP[1-3]. The typical trunk forward bending pattern has been characterized by lumbar dominant movement in the initial phase followed by equal contribution of lumbar and hip flexion within mid phase[4]. As the trunk continues to bend, anterior displacement of the trunk's center of mass results in posterior movement of the pelvis to maintain the center of mass within the base of support which requires more hip flexion than lumbar flexion[5]. Alterations in this movement pattern have been able to reliably differentiate individuals with current LBP or a history of LBP from those without a history of LBP[6] highlighting the importance of forward bend movement assessment in diagnosis.

Individuals with LBP who demonstrate aberrant movement during forward bending are considered to have trunk movement coordination impairments (MCI) .[2,3] These individuals have demonstrated improvement in pain and function when prescribed trunk stabilization exercises (TSE) .[7-10] Kinematic analysis of individuals who were identified to benefit from TSE demonstrated disruptions between lumbar motion segments in the mid-range of movement, where muscle control is responsible for spinal stability[11]. Further kinematic study of this LBP subgroup identified that alteration in segment coordination (lumbopelvic rhythm, aLPR) and movement smoothness with sudden deceleration and acceleration in lumbar segment angular velocity (judder) during

forward bending were able to identify individuals who would benefit from trunk stabilization exercises.[12]

It has been theorized that aberrant forward bending may result from reductions in lumbar multifidus (LM) activation resulting in higher lumbar erector spinae (LES) recruitment.[13] The muscle fibers of LM have a high concentration of titin, which is thought to be responsible for the higher force generation during eccentric contractions[14-16] such as forward bending. This attribute suggests that the LM has large contributions to movement control of the lumbar spine during forward bending. Eccentrically controlled movements are also thought to require greater motor planning compared to concentric controlled movement because of greater coordination between the movement target and movement trajectory.[17] Eccentric control of movement requires a large amount of sensory feedback to coordinate agonist-antagonist force generation in order to move towards a target at controlled speeds[18], which may be provided by the LM.[19-21]

LM activation impairments have been associated with individuals with LBP who have been identified to benefit from TSE[22]. This impairment may cause a reduction in force production and sensory feedback, contributing to aberrant forward bending movement. An experimental condition that temporarily reduces the role of this muscle may be helpful in studying its impact on a forward bend movement. The ability to produce aberrant movements in individuals without LBP and typical movement through preferential fatigue of the LM may help determine the muscle's role in trunk movement control. Generalized fatigue of the lumbar extensors has been identified to reduce postural control in healthy individuals [23]. However, this study used a static postural control task rather than a movement task. Electrical stimulation (ES) presents

investigators with the ability to preferentially fatigue the LM[24], and may be an effective method of temporarily inhibiting LM function during movement. The purpose of this study was to identify the effects of ES induced fatigue to the LM on forward bending movement in individuals without LBP. It was hypothesized that LM fatigue would result in deviation from a typical forward bend movement pattern; specifically a reduction in movement smoothness (judder) and segment coordination (altered lumbopelvic rhythm).

Methods

Participants

Individuals without LBP who were between the ages of 18-45 and demonstrated typical forward bending movement upon visual screening were recruited for the study. Participants were excluded if they had LBP limiting function that lasted for greater than 3 days or required medical attention, were pregnant, had a history of abdominal or lower limb surgery, and/or body mass index (BMI) greater than 30. This study was approved through the university's institutional review board. Data was collected on ten individuals. However, loss of kinematic data was identified in post processing analysis. Therefore, data of nine individuals (2 female) with a mean age of 29 ± 5.9 years, mean body mass index of 22.6 kg/m^2 ± 2.3, and mean skin thickness of 6mm ± 2 were analyzed for this study.

Instrumentation

An electromagnetic tracking system (Liberty, Polhemus Inc., Colchester, VT) captured position and orientation of trunk segments at 120 Hz during forward bending. Kinematic sensors mounted to Orthoplast were attached to the following body landmarks: 1) right femur (15 cm. superior to the right femoral lateral epicondyle), 2) pelvis (over the spinous process of S2), 3) lumbar spine (over the spinous process of L1), and 4) thoracic

spine (over the spinous process of T3). Lumbar multifidus neuromuscular electrical stimulation (LMES) was delivered through 4 bifurcated leads from 1 channel attached to 2.5 cm^2 buffered electrodes placed 1.5 cm lateral to the spinous process of L5-L2. A clinical device (EMPI Continuum, Minnesota, USA) was used to deliver stimulation at 50 Hz, 400 μs phase duration at an intensity of 20 mA.[25]

Procedure

Each participant performed 6 forward bend movements using their typical, self-selected pattern. Following this, each individual was placed on a plinth in a prone position and 4 pre-gelled carbon foam electrical stimulating electrodes were placed over the LM muscles. LMES was administered to the muscles at 400 us phase duration, 75 Hz, 15 seconds on and off time, with minimum 20 mA intensity for 60 cycles. Prior work in the lab demonstrated EMG mean median frequency reduction of 32% ±12% in the LM and a mean median frequency reduction of 8% ± 4% in the lumbar erector spinae following 60 stimulation cycles (Appendix K). Following LMES, each individual repeated 6 forward bend movements.

Kinematic Data Reduction

Kinematic data were calibrated to a global reference frame and converted to segment angular rotations using Euler's angle in Cardan sequence (x, y, and z). Segment angular rotations included total trunk flexion (thoracic spine motion with respect to the pelvis), lumbar flexion (lumbar spine motion with respect to the hip/pelvis) and pelvic anterior rotation (pelvic motion with respect to the femur). An algorithm was used to automatically determine 5% and 95% of the total movement for each repetition. This step was performed to reduce algorithm error associated with small fluctuations in motion at

the beginning and end ranges of motion. Kinematic data were filtered with a dual pass Butterworth filter (2nd order low pass at 5 Hz) and time-normalized to 51 data points (100 % of total movement) across the forward bend movement. Coupling angles between the pelvis and lumbar spine segments were derived and plotted over the forward bend movement. Lumbar segment phase plane plots (velocity vs. angular displacement)[12,26] were also created. Custom algorithms and plotting programs created in LabView (v8.6, National Instruments, Austin, Texas) were used to classify individual forward bend movement patterns.

Peak thoracic, lumbar, and pelvic segment angular velocities were calculated along with peak lumbar, thoracic, and pelvic segment excursion angles pre and post LMES for descriptive purposes.

Rating of forward bending movement

Kinematic algorithms for identifying aLPR and judder were developed and validated in our lab.[12] To determine aLPR, lumbar flexion angle was plotted against pelvic anterior tilt angles (Figure 1a) to derive coupling angles between the lumbar and pelvic segments (Figure 1b). Altered lumbopelvic rhythm was defined by an early shift to pelvic dominated movement using the time point (T) during forwarding bending when the coupling angle between the lumbar and pelvic segments exceeded 58 degrees. T values occurring less than 38% of the way through the forward bending movement were considered to be an indicator of altered lumbopelvic rhythm (aLPR). Judder was identified through the presence of 6 or more deceleration events or local minima (Lmin) of lumbar flexion angular velocity on a lumbar phase-plane graph (Figure 4.1c). Forward bends were rated to be aberrant if a participant's kinematic variable exceeded standard

error of measure (SEM) for the respective variable (SEM for T=9.8%, SEM for Lmin=1) .[12] This resulted in a kinematic cut point of 7 Lmin for judder and T=28% for aLPR. This decision was made to address forward bend movements that were close to cutoff values for aberrant movement. Each forward bend trial of each individual was rated using this algorithm.

Classification of forward bend movement pattern

Kinematic ratings for forward bend movements were used to classify individuals into a forward bend movement category (typical or aberrant) pre LMES and again following LMES. Forward bend ratings were used to classify individuals into 1 of 4 categories: 1) typical movement, 2) aLPR only, 3) judder only, or 4) both aLPR and Judder. Individuals were classified as having an aLPR or judder pattern if they met the kinematic criteria for the respective aberrant movement for 3 or more forward bend trials. If they met the criteria for an aberrant movement for 2 or less forward bend movements, they were *not considered* to have that aberrant movement. Individuals were classified as having typical movement if they did *not meet the criteria for either* aLPR or judder. This decision was based on prior work in our lab that determined stable and consistent EMG patterns in participants when they were categorized to have aberrant movement based on this rule. Forward bend movement patterns post LMES were rated using the same algorithm. Each individual's forward bend pre LMES Lmin and T were averaged and compared to post LMES mean values. All individuals who had a change in forward bending classification following LMES had a mean difference in kinematic variables pre and post LMES that exceeded MDC_{90} for the respective aberrant motion (Lmin=1.6, T=11.6%).[12]

Data and statistical analysis

Peak thoracic, lumbar, and pelvic segment excursion and angular velocities were normally distributed. Therefore, segment angles (alpha=.016) and segment velocities (alpha=.016) were compared respectively using paired t-test pre and post LMES with Bonferroni correction.

Chi-square analysis of good fit was performed (alpha=.05) to determine if forward bending classifications changed following LMES. Lmin and T values were averaged across trials by participants pre and post LMES. They were compared with paired t-tests to determine if there was a significant change in kinematic quality of forward bending pre and post LMES (alpha=.025).

Results

Segment excursion and velocity data are presented in Table 1. There were no significant differences in peak segment velocity or excursion pre and post ES ($p>.05$). All individuals reached peak segment excursions between 70-90% of forward bending both pre and post LMES.

Prior to the delivery of ES, two individuals (participants 8 and 9) were identified to have no aberrant movement based on the *a priori* classification rules (Table 2). Two individuals (participants 1 and 3) were classified as having aLPR. Participant 2 was classified as demonstrating aLPR following LMES fatigue. Figure 2 displays lumbar-pelvis coupling angle graphs of individual forward bend trials pre and post LMES for all participants.

Five of nine individuals were classified as demonstrating judder pre LMES. Of the four individuals with no judder, two (participants 1 and 8) developed judder following ES. Three of five individuals classified as demonstrating judder pre LMES no longer demonstrated this aberrant movement post LMES. Figure 3 displays lumbar phase-plane graphs of forward bend trials for all individuals pre and post LMES.

Overall, six of nine individuals demonstrated a change in forward bend movement pattern following LMES, representing a significant relationship between ES and movement patterns X^2 (1)=9, p=.003, phi=.707 with a large effect size. All individuals who underwent a movement pattern change had a minimum of 3 of 6 forward bend trials *change* from pre ES conditions. Three individuals developed an aberrant movement pattern, and three had elimination of an aberrant movement following LMES (Table 2).

Paired t-tests revealed no significant difference in lumbar segment velocity acceleration and deceleration (Lmin) pre (mean=6.37 ± 2.4) and post (mean=5.7 ± 2.6) LMES t(8)=1.46, p=.15, d=.19. There was also no significant difference in onset of pelvic segment domination of the movement (T) pre (mean=36% ± 21%) and post (mean=32% ± 24%) following LMES, t(8)=1.33, p=.19, d=.18.

Discussion

The hypothesis that LMES mediated fatigue would result in aberrant forward bending across all individuals was not supported. Participants 1, 2, and 8 developed an aberrant pattern following LMES, while participants 5,6, and 7 extinguished aberrant movement following LMES.

There was no significant difference in the values of kinematic variables (Lmin, T) pre and post LMES with small effect sizes. This would also suggest that participants with

aberrant movement pre LMES, did not have any *worsening* of their bending post LMES The presence of kinematic rated aberrant movement prior to fatiguing the LM confounds results of this study, as the intent was to determine if fatigue would result in production of aberrant movement. Participants were selected based on visual screening for absence of aberrant movement prior to them going through the study procedures. However, despite not observing aberrant motion in these individuals, a majority of participants were rated to have aberrant forward bending based on their kinematic pattern prior to the LMES. Kinematic analysis classified 2 individuals with aLPR and five with judder. The level of agreement between the investigator and kinematic derived classifications for aLPR was comparable to the percent agreement of 75% (K=.47) between visual rating and kinematic rating identified by Wattananon (2014). However, the level of agreement between the investigator and the kinematic derived classification was lower for judder than the percent agreement of 86% (K=.50) established by Wattananon (2014).

The disagreement between observer and kinematic rating for judder may be attributable to various sources. Visual screening for inclusion into the study was performed prior to application of kinematic sensors that may have altered movement patterns that the criteria for judder may have been more sensitive to. The kinematic rating for judder has no set threshold of change that constitutes a significant shift in velocity. If the velocity profile of an individual has small amplitude changes in lumbar angular velocity it might not be appreciated through visual observation, but would be counted according to the kinematic criteria. Lastly, the investigator performing the visual rating (WS) may have been more proficient at detecting aLPR than judder. Interestingly, if a participant was classified with aberrant movement they only demonstrated 1 type of

aberrant movement. Diagnostic accuracy in patients with LBP improves with the presence of two aberrant movements in forward bending and therefore despite the findings of aberrant patterns our healthy participants did not reach the diagnostic threshold [6,12].

The results of the chi-square analysis was significant with a large effect size (phi=.707). There were three individuals who demonstrated an elimination of judder following LMES. These individuals (participants 5, 6, and 7) had mean pre-stimulation peak lumbar angular velocities that were 1/3 the velocity of the study sample average. Following LMES, their mean peak lumbar angular velocities nearly doubled. While the reason for this is difficult to determine. LM fatigue may have reduced their ability to control forward bending, resulting in increased velocity.

The lack of significant findings may have occurred because we could not induce fatigue of the LM with the ES protocol. It was not possible to test for fatigue using parameters such as EMG median frequency shifts. The time needed to perform that verification may have led to recovery of muscle function negating any effects of the LMES. However, prior work in our lab did demonstrate the ability to produce preferential fatigue of the LM compared to the LES [24] (Appendix K). There was median frequency reduction as well as force reduction as mentioned in the methods, suggesting that LMES is capable of producing LM fatigue. It may be possible that the individuals in this study recovered from fatigue prior to the post LMES forward bend trials.

Individuals without LBP may also have a robust neuromuscular system that can better compensate for LM fatigue without resultant aberrant movements. This may include substitution with other muscles such as the erector spinae or subtle changes

involving movement of other segments. The current algorithms, which were intended to detect the presence of aberrant movements in individuals with LBP, identify segment alterations between the lumbar and pelvis. It may not have the ability to identify changes in movement patterns of individuals without LBP who may have more robust compensatory mechanisms to fatigue that may possibly involve the thoracic spine segment (thoracic to lumbar sensors) or hip (pelvis to femur sensors) as well.

As an example, participant 2 was classified as not having aLPR pre LMES. This participant begins with a majority of forward bend trials starting well below the 58 degree coupling angle cut point prior to 38% of movement representative of a lumbar dominant pattern (Figure 2). As forward bending progresses, there is an increase in coupling angle as the pelvis begins to contribute more to the movement. Following LMES, this individual was classified as demonstrating aLPR. The movement is pelvic dominant and starts above the 58 degree point for a majority of the movement. In comparison to participant 2, participant 6 demonstrates a similar shift in the patterns with more pelvic contribution to the movement following LMES. While a few of the forward bend trials meet the kinematic cut points, it is clear that there was a shift in the pattern of forward bending that is present throughout the post LMES trials. The shift in pattern may have involved changes in movement within other segments of the trunk or the hips.

These shifts in pattern are also seen in the phase-plane plots used for kinematic ratings for judder (Figure 3). Participant 4 demonstrated a flattening of the velocity curve post LMES. This indicates that the lumbar segment stayed at a constant speed, and another segment became responsible for the steady acceleration and deceleration of forward bending seen in the phase-plane plots of other participants. However, there was

no difference in the average Lmin count pre and post LMES that could quantify a change in this pattern. There were lumbar velocity pattern changes in some individuals following LMES, but the current kinematic criteria focuses solely on shifts in velocity using Lmin and was unable to detect other changes in pattern. . Refinement of the current approach using kinematic algorithm with definitive cut points that account for these pattern changes is likely necessary to study more subtle changes in movement pattern coordination and control. Other supplemental information such as analyses of EMG activity during forward bending may also help in improving detection of changes in forward bend movement patterns.

Conclusions

The current study was not able to support the hypothesis that fatigue induced LMES would result in aberrant movement using the current kinematic algorithm. This may be attributable to several factors. We may not have obtained adequate fatigue of the LM or the participants in this study may have recovered from fatigue prior to post testing. Individuals without LBP may also have redundant systems with greater degrees of freedom to prevent the formation of aberrant movements. However, there does appear to be some changes that occurred in the forward bending pattern. Individuals without LBP may have a more robust neuromuscular system that allows for compensations that are more evenly distributed through the movement system, than the segments that are monitored with the current kinematic algorithm. Identifying how individuals without LBP compensate to a temporary impairment in muscle function may assist in the intervention of patients with LBP that demonstrate changes in movement that appear to occur predominantly at the lumbopelvic segment[4,27].

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Tables and figures

Table 4.1 Mean Peak segment excursion angle (standard deviation) and mean peak segment velocity (standard deviation) during forward bending pre and post electrical stimulation fatigue.

	Segment Excursion (degrees)		Segment Velocity (deg/sec)	
	Pre	Post	Pre	Post
Thoracic	6.1 (7.4)	3.2 (2.7)	6.4 (2.5)	6.6 (4.2)
Lumbar	56 (6.8)	54.4 (8.1)	31.6 (11.8)	33.2 (15.2)
Pelvis	54.9 (14.8)	52.5 (16.3)	54.9 (14.9)	52.5 (15.3)

Table 4.2. Classification of movement patterns [altered lumbopelvic rhythm (aLPR) or lack of smooth control (judder)] during forward bending, pre and post electrical stimulation fatigue. Changes in classification following electrical stimulation are shaded in grey. Participants 1,2, and 8 developed aberrant movement following LMES. Participants 5,6, and 7 extinguished aberrant movement following LMES.

Participant	**aLPR**		**Judder**	
	Pre	**Post**	**Pre**	**Post**
1	Yes	Yes	No	Yes
2	No	Yes	Yes	Yes
3	Yes	Yes	No	No
4	No	No	Yes	Yes
5	No	No	Yes	No
6	No	No	Yes	No
7	No	No	Yes	No
8	No	No	No	Yes
9	No	No	No	No

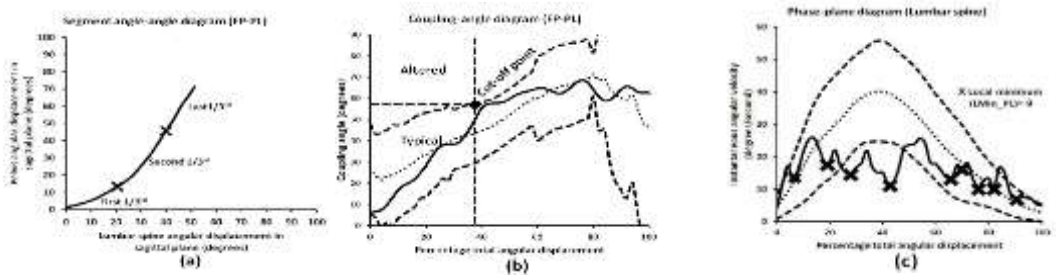

Figure 4.1 4.1a) Pelvis angle-lumbar angle plot used to derive: 4.1b) coupling angle plot to determine altered lumbopelvic rhythm 4.1c) phase plane plot of lumbar flexion angular velocity vs lumbar flexion angle used to determine judder. Adapted with permission from Wattananon 2014.

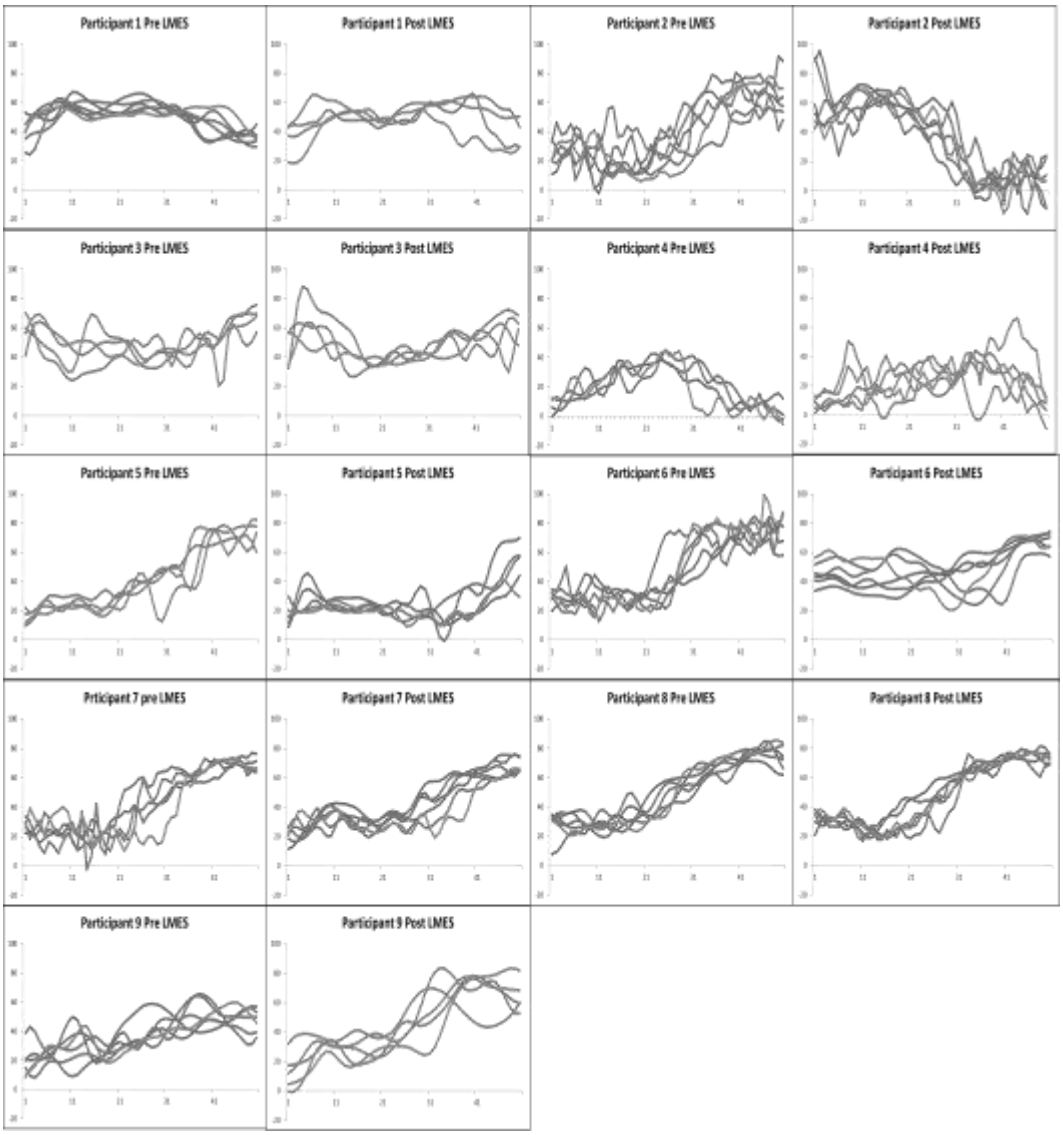

Figure 4.2 Graphs of coupling angles. Y axis is the coupling angle and X-axis is the percent of forward bend motion. Coupling angles that exceeded 58 degrees prior to 38% of forward bend motion were rated as altered lumbopelvic rhythm (aLPR). Participant 2 was classified as having a typical forward bending pattern with respect to lumbopelvic rhythm pre LMES, and classified as having aLPR following LMES fatigue.

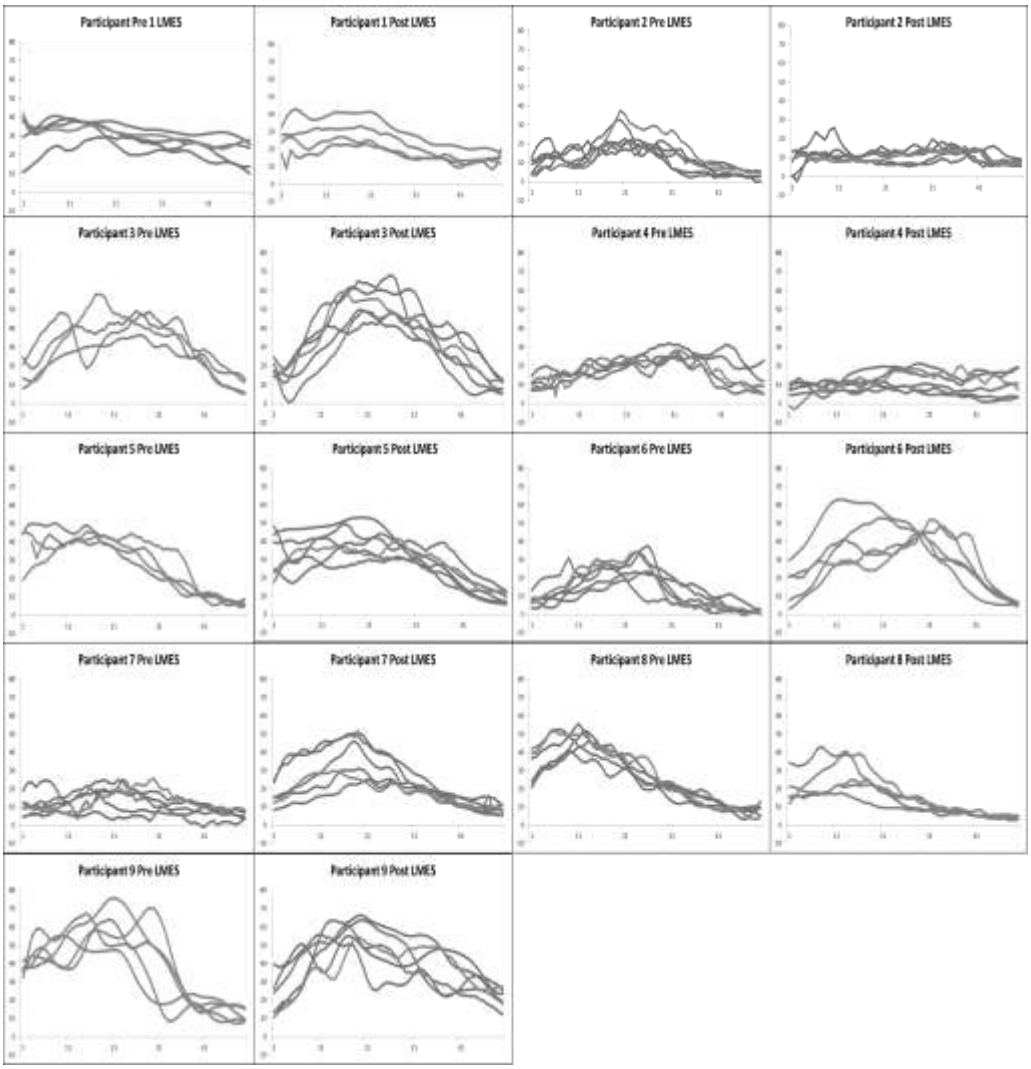

Figure 4.3 Lumbar flexion angular velocity graph for rating of judder. Velocity is on the y-axis and time normalized motion of the forward bend phase on x-axis. A forward bend trial was rated as judder if it had 7 or more deceleration events, or local velocity minima. Participants 1 and 8 were classified as having judder post LMES. Participants 5,6, and 7 were classified as having extinguished judder following LMES.

Chapter 5: Summary

This chapter provides a brief summary of the rationale and specific aims, discussion, and conclusions of the aims originally proposed in Chapter 1. Conclusion of each specific aim will be addressed in addition to discussions of limitations, implications for clinical practice, and recommendations for future research. Changes from the original proposal and rationale will also be discussed.

The purpose of this dissertation was to identify mechanisms underlying clinical tests that are used to predict a patient's success with trunk stabilization exercises: aberrant movements during forward bending and the prone instability test. The rationale was that identifying impairments driving positive findings on these tests could help improve test interpretation and intervention prescription.

The following two specific aims were addressed in this dissertation (**Chapter 1)**:

1) Characterize lumbar extensor muscle neuromuscular control during active forward bending and the prone instability test (PIT);

2) Validate clinical assumptions of the role that lumbar multifidus muscle activity has in aberrant movements patterns during a forward bend task and a positive prone instability test.

Chapter 2 addressed specific aim 1 and focused on identifying motor control strategy utilized by patients with aberrant forward bending and comparing it to healthy subjects with typical forward bend. **Chapter 3** investigated the PIT relative to stiffness change and neuromuscular control in patients with low back pain. Data from healthy control subjects were used to compare and contrast stiffness and neuromuscular control findings (specific aim 1). To determine the lumbar multifidus (LM) muscles' ability to reproduce

positive PIT results and increase spinal stiffness, neuromuscular electrical stimulation was also used to elicit LM contraction in a prone position (specific aim 2). **Chapter 4** addresses the role of the LM in controlling forward bending. In this experiment neuromuscular electrical stimulation was used to fatigue LM and determine if limiting the muscles' contribution in forward bending resulted in aberrant movement (specific aim 2).

Conclusions

The long term goal of this dissertation was to identify mechanisms associated with impaired neuromuscular control in LBP patients in order to improve diagnostic criteria and intervention efficacy. Because interventions are guided by the quality of forward bending and results of the PIT, it was believed that understanding the mechanisms responsible for these tests would help to improve treatment efficacy. Aim 1 identified muscle activation differences in individuals with low back pain that suggest addition of interventions to address motor control and muscle capacity may prove useful. Aim 2 results would indicate that electrical stimulation can produce spinal stiffness changes that exceed that achieved through volitional activation in individuals with LBP and may be an adjunctive treatment.

Aim 1

Analysis of forward bending identified 2 subgroups of aberrant movement in individuals with low back pain (LBP): alteration of lumbopelvic rhythm (aLPR) and a combination of aLPR and rapid lumbar segment acceleration and deceleration during movement (aLPRJUD). Individuals with aLPR and aLPRJUD had higher LM and lumbar erector spinae (LES) activation compared to individuals without LBP that had typical

movement. Individuals with aLPRJUD had higher thoracic erector spinae (TES) activation compared to individuals with just aLPR and individuals without LBP. There were also differences in the point during forward bending that these muscles reached peak activation compared to individuals without LBP.

Analysis of prone instability mechanism yielded increase in spinal stiffness to a posterior to anterior (PA) force from prone to the prone instability leg raising task (PITLR). Stiffness changes from prone to PITLR were not different between groups. However, EMG analysis did demonstrate a reduction in LM and LES activity in individuals with LBP. There was also a difference in the muscle synergy patterns used to achieve pain reduction during the test. Individuals without LBP had a higher percentage of variance accounted for by the LM, LES, and LD compared to those with LBP. While individuals with LBP were able to reduce pain from the PA force, they achieved this using a different neuromuscular strategy.

Comparing LM activation during the prone instability test and forward bending offer interesting contrasts. LM activation was higher in individuals with LBP that had aberrant movements during forward bending compared to a control group. However, it was lower during the prone instability test compared to a control group, along with a different synergy to accomplish a positive test. While participants in these two studies were from a different sample, they were of similar age and anthropometric characteristics. They had similar pain and functional outcome scores. Lastly, both samples were drawn from a population of individuals with LBP in an acute to subacute phase of symptoms. Their baseline pain tended to be low with pain exacerbation during functional activities. Lastly, both samples were recruited using the same inclusion and

exclusion criteria. Therefore, they are assumed to be similar in clinical presentation with similar impairments in neuromuscular control. In the forward bend, there are very few ways that a person can modify the task. They can change how the thoracic, lumbar, and pelvic segments move relative to each other compared to a typical forward bending pattern and/or increase muscle output to complete the task. In the prone instability test, the table supports the trunk and the limbs may be able to contribute a larger role in stiffening or stabilizing the spine. This may allow the individual to change how muscles are activated and to what degree since more muscles may participate during the test. The individual may be demonstrating the capacity to successfully compensate, perhaps with suboptimal neuromuscular control, in order to stiffen the spine and reduce pain to external perturbations. In both cases, the individual with LBP is able to alter or reorganize some aspect of the neuromuscular system to accomplish or reduce symptoms during the task. Perhaps this is why these tests are able to identify an individual with LBP that responds favorably to trunk stabilization exercises. It may be the case, that individuals who are predicted to fail with trunk stabilization exercises lack these adaptation strategies.

Aim 2

The use of an electrically stimulated contraction of the LM, in individuals with LBP, was able to reproduce the pain reduction and spinal stiffening obtained in the prone instability test. The stiffening of the spine that was achieved with electrically elicited contraction was similar across individuals with and without LBP. While results of aim 1 demonstrated a combination of muscles contributing to a positive finding on the prone

instability test, the results of aim 2 would suggest that LM can play a major role in spinal stiffening and subsequent pain reduction. We were not able to support the hypothesis that isolated fatigue of the LM would result in aberrant forward bending movement patterns in individuals with typical movement prior to fatigue. However, these findings may have been due to suboptimal kinematic criteria used to rate forward bending quality. It may also have been due to the ability of individuals without LBP to use their intact and robust neuromuscular system to compensate for fatigue of an isolated muscle. Therefore, we cannot rule out that impairments in LM function play a significant role in aberrant movement.

Summary of Modifications

Prone instability Test

Delivery of Lumbar Multifidus Electrical Stimulation

Initially, the electrical stimulation to the lumbar multifidus was to be administered in the resting position of the prone instability test. In this position, the participant lies partially prone with the upper body supported on the table, with the feet off the floor. This was initially proposed because it would best replicate muscle activation during the prone instability test. The methods were modified to administer electrical stimulation in prone. The standard error of measure was smaller in the prone position, allowing for better detection of spinal stiffness changes resulting from stimulation.

EMG Collection and Data Reduction

The original proposal called for collection of EMG data from the right gluteus medius. However, equipment failure mid study resulted in the loss of 1 EMG lead. Examination of the EMG data to that point revealed that the gluteus medius and external

oblique had the lowest amplitudes compared to normalization tasks. We wanted to maintain representation from the trunk flexors muscles during testing. Therefore, the decision was made to omit gluteus medius from the remainder of the protocol for all participants.

The proposal stated that submaximal isometric contractions would be used to normalize EMG signals collected during the prone instability test protocol. However, during initial practice trials as well as pilot work with 1 participant with LBP and 1 participant without LBP, participants were not able to maintain the testing positions against the resisting forces applied by the examiner, resulting in a maximal test. Therefore the decision was made to use maximal force testing of all participants (break testing) to obtain maximal volitional contraction (MVC) testing. This would allow for consistent comparison of EMG across participants and conditions.

Limitations and Future Studies

Forward bending muscle activation patterns reported in chapter 2 were based on the lowering phase of the forward bending movement where eccentric contraction of the extensor muscles is presumed to control motion. The return phase where concentric activation of extensors would dominate was not investigated. This lowering phase was chosen because the kinematic criteria that detected aberrant movement were developed in this phase of forward bending. The LM is functioning in eccentrically during the lower phase. The muscle fibers of LM have a high concentration of titin, which is thought to be responsible for the higher force generation during eccentric contractions such as forward bending[1-3]. This attribute may allow for LM to have large contributions in controlling

forward bend movement. Eccentric movements are also thought to require greater motor planning compared to concentric movement because of greater coordination between the movement target and movement trajectory [4]. These movements require a large amount of sensory feedback to coordinate agonist-antagonist force generation in order to move towards a target at controlled speeds [5] which may be provided by the LM [6-8]. Therefore, it was thought that the lowering phase of forward bending would be more indicative of movement coordination impairments.

The concentric return phase may yield further information on neuromuscular control patterns. Nelson-Wong, et al. (2012) identified a different order of muscle activation in individuals with LBP on the return phase compared to individuals without LBP. This study used cross correlation analysis to identify activation timing and did not look at muscle activation amplitude patterns. Further work should consider using methods similar to chapter 2 on the return phase of forward bending to obtain further information on neuromuscular control in these patients. Individuals with LBP that demonstrated aberrant forward bending have demonstrated improvement in forward bending quality following trunk stabilization exercises[10]. Therefore, it does appear that this movement pattern can improve, even if practice of the task is not performed in the clinic. Comparison of neuromuscular control during forward bending in individuals who had pain reduction and improvement in function following rehabilitation versus those that were not successful may therefore yield valuable mechanistic information. This type of analysis may help identify mechanism responsible for successful rehabilitation to help refine intervention planning.

In the studies on the prone instability test all of the LBP participants had successful pain reduction during the test. No LBP participant had a negative test. While these results help with identification of neuromuscular control in these patients, it still leaves a gap. We do not know what factors are responsible for those patients who do not have a reduction in pain during the test. This is important as these patients are associated with failure with trunk stabilization exercises. This may be a result of a patient's inability to make necessary neuromuscular adaptations to reduce pain. Perhaps these are the patients that are at the highest risk for recurrence and need the most attention. Identification of factors that contribute to a negative response (no pain reduction during the test) may help adapt intervention planning to allow for success with trunk stabilization exercises. A study designed to compare neuromuscular control of patients with a positive and negative prone instability test response would help to achieve this task.

The prone instability test study also identified the ability to obtain spinal stiffening and pain reduction against posterior to anterior forces on the spine with application of lumbar multifidus electrical stimulation (LMES). Therefore it is plausible that this modality has potential to be an effective adjuvant in clinical care. This would be supported by the work of Hicks, et al. (2016) in geriatric patients with chronic low back pain. However, unlike studies in the knee, where stimulation dosage can be adjusted to produce a measured force[12], this was not pragmatically possible in this testing condition. ES was applied initially at 40mA and was increased for subjects as necessary to promote a contraction. No subject exceeded 50mA, and only 2 subjects required ES at greater than 40mA to obtain a tetanic contraction. However, there is likelihood that there was

variability in the force generated among the subjects with the LMES. Refining the method to identify dosage of LMES to obtain specific dosage would be beneficial for future application of LMES.

We were unable to detect production or worsening of aberrant movement during forward bending following fatigue of the LM in individuals without LBP. There did appear to be movement pattern changes, but these changes were not relative to alteration in movement pattern that were captured using our current kinematic algorithm. Refining the kinematic rating methods or using alternative methods such as dynamic systems analysis or center of rotation changes during movement may allow for better detection of movement pattern change or identification of clinically observed aberrant movement. These approaches could be used this and future studies on forward bending. While kinematic analysis was not able to detect changes in forward bending quality, EMG analysis during forward bending post fatigue may also be helpful in detecting any change in muscle activation of synergies associated with control of this movement.

Rehabilitation implications

During forward bending, LM activity increase separated the aberrant movement groups from study participants with typical forward bending. Synchronization of motor units resulting in higher EMG amplitudes may could have resulted from several factors. There may have been an increase in vertebral segment movement requiring increased muscle activity. Reduction in muscle capacity may have been present requiring activation of additional motor units to accomplish the task. There may also have been sensory feedback impairments that resulted in improper modulation of muscle activation. Results of EMG analysis during the prone instability test suggest there was reorganization of

muscle synergies to achieve the task. While findings resulted in pain reduction, the solution may be a suboptimal plan where global trunk stabilizing muscles are used to compensate for limitations within intrinsic muscles.

These results make the case that interventions should address the specific impairments. However, tests to identify specific impairments in muscle coordination, timing, and capacity may be difficult, in an interrelated movement system that requires all of the above to successfully execute movement. Without clear tests to identify specific impairments in neuromuscular control, it may be best to implement an intervention plan that incorporates all facets of control. In this case, interventions with proper exercise prescription and dosage directed toward enhancing muscle capacity (strength and endurance), with perhaps a specific focus on LM, may need to be considered in treating these patients. It would also be reasonable to attempt restoring the movement coordination in patients to obtain similar movement and muscle activation patterns to those demonstrated by healthy participants. This may be obtained through exercises that focus on improved movement coordination between the thoracic, lumbar, and pelvic segments as proposed by Sahrmann (2001) and O'Sullivan (2000). Intervention planning may have to go beyond maintaining a neutral spine position during stabilization exercises and involve more coordinated movement through the available range to promote strength, endurance, and movement coordination as described by these investigators. In patients with LBP that have impaired LM activity, use of LMES may be a helpful adjunct to intervention. It may help to address muscle activation impairments to allow the muscle to properly function during exercises.

Summary

Aberrant movement during forward bending and a positive prone instability test are able to predict individuals with LBP that would benefit from trunk stabilization exercises. The results may suggest that these individuals are able to succeed with this intervention because they have the ability to modify or reorganize their neuromuscular system using exercises associated with this approach. The results support the role of the LM in forward bending and the prone instability test. The findings also demonstrate that LES works in synergy with LM to control movement and stiffens the lumbar spine. Adaptations in neuromuscular control during forward bending and the prone instability test in individuals with LBP suggest that exercises that include movement control and coordination may be necessary within the intervention. Electrical stimulation may also be an important adjuvant to rehabilitation in some patients, however this requires further study.

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Chapter 6: Appendices

6.1 Appendix A:Timeline

April 2015

Proposal Defense

May 2015-October 2015

Data Reduction and Analysis for Aim 1a data: May 2015-August 2015
Data Collection on Aim 1b, 2A, 2b: June 2015 – December 2015
Data Reduction on Aim 1b, 2A, 2b: December 2015
Data Analysis on Aim 1b, 2A, 2b: January 2016-March 2016

October 2015-April 2016

Manuscript Preparation:
Aim 1a: September 2015-November 2015
Aim 1b, 2A, 2b: December 2015-March 2016
Dissertation Defense: April 2016

6.2 Appendix B: Resources

Personnel
Sheri P. Silfies, PT, PhD
David Ebaugh, PT, PhD
Susan S. Smith, PT, PhD
Gregory Hicks, PT, PhD
Peemongkon Wattananon, PT, PhD
Scott Stackhouse, PT, PhD
Marco Canella, PhD

Primary Data Collection was funded by:
Aim 1a: National Institute of Child Health & Human Development, National Institutes of Health (K01HD053632).

6.3 Appendix C: Testing protocol and results for within day reliability of posterior to anterior force application for the prone instability test

<table>
<tr><td></td><td>Testing Position</td><td>Load</td><td></td></tr>
<tr><td rowspan="6">Fully prone: Subject is in prone position on an examination table. 2 minutes rest between trials, 5 minutes between load change</td><td>Prone Trial 1</td><td>22 N</td><td rowspan="6">20 seconds data acquisition; Audible test initiation marker at 5 seconds, where examiner applies load, ramping up force to visual markers on screen within 5-10 seconds</td></tr>
<tr><td>Prone Trial 2</td><td>22 N</td></tr>
<tr><td colspan="2">Subject rest 5 min</td></tr>
<tr><td>Prone Trial 1</td><td>44N</td></tr>
<tr><td>Prone Trial 2</td><td>44N</td></tr>
<tr><td colspan="2">Subject rest 5 min</td></tr>
<tr><td rowspan="7">Prone Instability Resting Position (RPIT): Subject is in testing position with leg in resting position through whole session. 2 minutes rest between trials, 5 minutes between load change</td><td>Testing Position</td><td>Load</td><td rowspan="7">20 seconds data acquisition; Audible test initiation marker at 5 seconds, where examiner applies load, ramping up force to visual markers on screen within 5-10 seconds</td></tr>
<tr><td>PIT Resting Trial 1</td><td>22 N</td></tr>
<tr><td>PIT Resting Trial 2</td><td>22 N</td></tr>
<tr><td colspan="2">Subject rest 5 min</td></tr>
<tr><td>PIT Resting Trial 1</td><td>44N</td></tr>
<tr><td>PIT Resting Trial 2</td><td>44N</td></tr>
<tr><td colspan="2">Subject rest 5 min</td></tr>
<tr><td rowspan="7">Prone Instability Leg Raise Session (PITLR): Subject is in testing position with leg in resting position until an auditory marker is heard. 2 minutes rest between trials, 5 minutes between load change</td><td>Testing Position</td><td>Load</td><td rowspan="7">20 seconds data acquisition; Audible test initiation marker at 5 seconds, where subject raising legs to 18 inch high gate at the marker. Examiner applies load, ramping up force to visual markers on screen within 5-10 seconds</td></tr>
<tr><td>PIT Trial 1</td><td>22 N</td></tr>
<tr><td>PIT Trial 2</td><td>22 N</td></tr>
<tr><td colspan="2">Subject rest 5 min</td></tr>
<tr><td>PIT Trial 1</td><td>44N</td></tr>
<tr><td>PIT Trial 2</td><td>44N</td></tr>
</table>

Subjects: 5 (3 male, 2 female)
Mean age: 30 +/-
Mean weight:
Mean height:
1 subject with no low back pain
4 subjects with low back pain

	Mean (test and retest)	SD test	SD retest	$ICC_{(2,)2}$	MDC (95%CI)	MDC (90%CI)
Prone	6.68	6.9	6.5	0.902	5.81	4.89
PIT resting position	14.58	14.3	6.2	0.79	13.02	10.96
PIT leg raise position	18.8	4.9	3.73	0.945	2.81	2.36

6.4 Appendix D: Fidelity testing of posterior to anterior force onset suring the prone instability test

Five random subjects were drawn from the pool of participants in the prone instability test study. Three data samples were taken of their prone, RPIT and PITLR trials. Each data stream was 2 seconds long and was captured on the load application at random points. The posterior force and time were taken these points and force application velocity were calculated. This is expressed in newtons per second (n/s)

Coefficient of variation was calculated for the posterior to anterior force application across the subjects

Table 6.1 Rate of posterior to anterior force application samples (newtons/second) for 5 random subjects

Subject	Sample	Prone	RPIT	PITLR
1	Sample 1	3.11	3.40	3.24
1	Sample 2	3.24	3.17	2.99
1	Sample 3	3.33	2.87	2.81
2	Sample 1	3.19	3.23	3.13
2	Sample 2	2.99	3.24	3.11
2	Sample 3	2.99	3.12	3.21
3	Sample 1	3.19	3.45	3.16
3	Sample 2	3.25	3.21	3.19
3	Sample 3	2.91	3.24	3.10
4	Sample 1	3.28	3.11	3.12
4	Sample 2	3.45	3.21	3.23
4	Sample 3	3.19	2.98	3.34
5	Sample 1	3.11	3.28	3.19
5	Sample 2	3.23	3.12	3.25
5	Sample 3	3.19	3.33	3.18

Table 6.2 ,Mean, standard deviation, and coefficient of variation for rate of force application.

	Prone	RPIT	PITLR
Mean	3.2	3.2	3.2
S.D.	0.14	0.15	0.12
CV	4%	5%	4%

Table 6.3 Mean, standard deviation, and coefficient of variation for rate of force application for individual subjects across conditions (newtons/second).

	Prone			RPIT			PITLR		
Subject	Mean	S.D.	CV	Mean	S.D.	CV	Mean	S.D.	CV
1	3.2	0.11	3%	3.1	0.27	8%	3.0	0.22	7%
2	3.1	0.12	4%	3.2	0.07	2%	3.2	0.05	2%
3	3.1	0.18	6%	3.3	0.13	4%	3.2	0.05	1%
4	3.3	0.13	4%	3.1	0.12	4%	3.2	0.11	3%
5	3.2	0.06	2%	3.2	0.11	3%	3.2	0.04	1%

6.5 Appendix E. IRB approval documents

Use of FNIR to determine the ability of NMES to selectively activate lumbar multifidus.

Drexel UNIVERSITY
Human Research Protection

APPROVAL OF PROTOCOL

April 16, 2014

Sheri P. Silfies, PhD
Mail Stop: Three Parkway; Mail Stop 7-502

Dear Dr. Silfies:

On 04/16/2014 the IRB reviewed the following protocol:

Type of Review:	Initial Expedited
Title:	Use of EMG, fNIR and NMES to Study Muscle Activity of the Lumbar Multifidus During 3 Commonly Used Lumbar Spine Clinical Tests: A Pilot Study
Investigator:	Sheri P. Silfies, PhD
IRB ID:	1404002752
Funding:	None
Grant Title:	None
Grant ID:	None
IND, IDE or HDE:	None
Documents Reviewed:	HRP 211 Application for Initial Review, HRP 201 Contact Information Forms, Form 1 Financial Interest Disclosure Forms, HRP 503 Protocol Version Revised April 3, 2014; Research Proposal; Recruitment Flyer Version 1; HRP 502 Informed Consent Version 1 Revision Date 04-04/2014; Training Certifications

The IRB approved the protocol from 04/16/2014 to 04/15/2015 inclusive.

On 04/16/2014 – According to 45 CFR 46.110 this study has been Approved Expedited Category 4.
Approval Includes: Recruitment of 10 Subjects into the study; HPR 503 Protocol Version Revised April 3, 2014; Recruitment Flyer Version 1; Informed Consent Version 1 Revision Date: 04-04-2014.
Risk Level of Devices: Non-Significant Risk, According to 21 CFR 812.2(b)

Before 03/01/2015, which is 45 days prior to study closure, you are to submit a completed "FORM: Continuing Review Progress Report (HRP-212)" and required attachments to request continuing approval or closure.

If continuing review approval is not granted before the expiration date of 04/15/2015 approval of this protocol expires on that date.

Attached are stamped approved consent documents. Use copies of these documents to document consent.

1601 Cherry Street, 3 Parkway Building, Suite 10644, Philadelphia, PA 19103 | Tel: 215.255.7807 | Fax: 215.255.7074
drexel.edu/research/hrp | hrpp@drexel.edu

In conducting this protocol you are required to follow the requirements listed in the INVESTIGATOR MANUAL (HRP-103).

Sincerely,

Andrew S. Wechsler, M.D.
IRB Chair

Drexel University
Consent to Take Part
In a Research Study

1. Title of research study: Use of EMG, fNIR and NMES to study muscle activity of the lumbar multifidus during 3 commonly used lumbar spine clinical tests: a pilot study.

2. Researcher: Sheri P. Silfies PT, PhD

3. Why you are being invited to take part in a research study

We invite you to take part in a research study because you are a healthy individual between the ages of 18-50 years of age, with no history of low back pain for which you got healthcare services or caused you pain or loss of normal function for greater than 3 days.

4. What you should know about a research study

- Someone will explain this research study to you.
- Whether or not you take part is up to you.
- You can choose not to take part.
- You can agree to take part now and change your mind later.
- If you decide to not be a part of this research no one will hold it against you.
- Feel free to ask all the questions you want before you decide.

5. Who can you talk to about this research study?

If you have questions, concerns, or complaints, or think the research has hurt you, talk to the research team by contacting Dr. Sheri Silfies at 215-553-7018.

This research has been reviewed and approved by an Institutional Review Board (IRB). An IRB reviews research projects so that steps are taken to protect the rights and welfare of human subjects taking part in research. You may talk to them at (215) 255-7857 or email HRPP@drexel.edu for any of the following:

- Your questions, concerns, or complaints are not being answered by the research team.
- You cannot reach the research team.
- You want to talk to someone besides the research team.
- You have questions about your rights as a research subject.
- You want to get information or provide input about this research.

6. Why are we doing this research?

Exercise is one of the most effective ways to address low back pain. Very specific muscles of the low back have been identified as playing an important role in the recovery of people who suffer from low back pain. The purpose of our study is to investigate how well different muscle recording instruments are able to detect muscle activity during the use of common clinical tests that identify the kinds of exercises a person should do in therapy. We will also be studying the ability of a these instruments to detect the activity of specific low back muscles that will be activated by electronically stimulating the

muscles. This will be done using a clinical muscle stimulation unit and a set up commonly used in treating patients with low back pain.

7. How long will the research last?

We expect that you will be in this research study for 2 hours.

8. How many people will be studied?

We expect about 10 people will be in this research study. This study will only occur in the Philadelphia area.

9. What happens if I say yes, I want to be in this research?

The **first step**: We will ask you some simple questions about your low back and general health to make sure you meet all the criteria for the study.

If you have a condition that meets the exclusion criteria, then you will not be able to participate in the study.

If you meet the criteria, the **second step** will be to collect some information about you:

> We will collect general information about you including your age, gender, measurement of your height, length of the back and weight.

The **third step** will be laboratory testing. Testing takes place at the Rehabilitation Sciences Labaratory at Drexel University on the 2nd floor of 1601 Cherry Street, Philadelphia. This testing will take place during one session lasting approximately 2 hours.

> You will be asked to wear shorts and sports bra/halter top during the procedure. Clean clothing will be provided for you by the researcher if necessary.

Setup Procedure:

Sensors will be taped to your back, legs and stomach that detect muscle activity.

Sensors that measure blood flow to your muscles using infrared light will be taped to your back. Infrared light is similar to types of light found in sun light.

You will be asked to lie on your stomach on a examination table. The head of the table will be lowered to place your back in a slightly bent position, but you will still be supported by the table. Supporting straps will be placed across your legs to make sure you are steady in this position.

The setup for this will take 20 minutes. The sensors and electrodes will stay on you for the remainder of the experiments.

Back Muscle Tests

> The back muscle testing will consist of 3 different tests.
>
> 1) Back Raising Test:
>
> You will be asked to cross your arms across your chest and asked to lift your chest off the table so that your back is parallel to the floor. You will hold this position for 10 seconds, and then

return to a resting position. You will perform 10 repetitions of this activity with 30 second rest between each test.

You will then be asked to lie on your side, with the head of the table still in the lowered position with the steadying straps around your legs, and raise your trunk so that it is parallel to the floor. You will hold this position for 10 seconds on the right and 10 seconds on your left. This will be done for 1 repetition.

This will take 10 minutes.

2) Arm Raising Test:

The head of the table will be straightened and you will continue to lie on your stomach. You will be asked to place your dominant arm in an overhead position while holding a 1lb. weight. You will be asked to pick your arm up off the table and hold that position for 10 seconds and then return to a resting position. You will perform 10 repetitions of this activity with a 30 second rest between each arm raise.

This test will be repeated two more times, 10 arm raises each using the following weights:

10 repetitions with 30 second rest holding between 1.5-2 lbs

10 repeitions with 30 second rest holding between 2-3 lbs.

Determination of the weight for the second and third trials of this test, will be based on your body weight.

This will take 30 minutes

3) Leg Raising Test

You will be asked to lie on the table with your upper body supported on the table. Your legs will be off the table. The height of the table is adjustable and will be raised or lowered so that your feet are touching the ground and you are comfortable. You will be asked to hold on to the table with your hands and raise your legs up so your feet are off the ground. You will hold your legs up for 10 seconds followed by a 30 second rest. You will perform 10 repetitions of this activity with a 30 second rest between each leg raise.

This will last 10 minutes.

Muscle Stimulation Test

An battery powered muscle stimulator which is commonly used in physical and occupational therapy treatments will be used to cause contractions of your back muscles. This unit uses "AA" batteries to cause an electrical signal that causes muscle to contract.

You will be asked to lie on the examination table your stomach. Self adhesive muscle electrodes will be placed along the muscles of your low back. The electrodes used to measure muscle activity will be removed from your back. The infrared sensors used to measure blood flow levels in your muscles will stay on you. Pillows will be placed under your hips to make sure your back is in the right position for testing as well as for your comfort. A belt will be placed across your pelvis to keep it steady during the stimulation. The muscle stimulator will be turned on until a visible muscle contraction is seen and felt by the examiner. The muscle stimulator will be on for 10 seconds. You may feel a sensation similar to

a muscle cramp while the stimulator is on. After the contraction, there will be a 50 second rest period. There will be 10 repetions of this activity.

This will last 15 minutes.

10. What are my responsibilities if I take part in this research?

If you take part in this research, it is very important that you:

- Follow your researcher's instructions.
- Tell the researcher right away if you have a complication or injury.

11. What happens if I do not want to be in this research?

You may decide not to take part in the research and it will not be held against you.

12. What happens if I say yes, but I change my mind later?

You agree to take part in the research now and stop at any time it will not be held against you.

13. Is there any way being in this study could be bad for me?

Risks:

Muscle Soreness (common): There is some risk of muscle soreness after the testing. This is similar to the soreness you will experience after exercising and should fade within 12-36 hours.

Skin Irritation (rare): There is risk of skin irritation during the electrical stimulation and from the adhesive on the muscle sensors. We will minimize these risks by making sure your skin is prepared properly prior to the testing by cleansing the area with soap and water. We will also use a hypoallergenic electrode pads.

Sensitivity to light (rare): Sensitivity to light (same as being exposed to sunlight without a hat or sunglass): the brightness of the near infrared light that will be used is very low, and is equivalent to spending the same amount of time under sunlight without a hat. The total amount of time you will be exposed to this type of light will be under 15 minutes. Although there may be some as yet unknown effects from exposure to near infrared light, these risks are thought to be the same as those associated with exposure to sunlight. Also, the power of the laser light used in this research is low and is less than the FDA's (Food and Drug Administration) safety limit so, thermal damage to skin is minimum. Due to the high sensitivity of eyes to the laser beam, special caution will be taken to never emit laser light directly to your eyes.

Shock from Infrared light sensor pad (rare): one participant (out of many hundred hours of research) reported a shock from a near infrared sensor pad that was not peoperly manufactured. The researchers have implemented a number of inspection and training measures to prevent any such occurrence in the future

14. Do I have to pay for anything while I am on this study?

There is no cost to you for participating in this study.

15. Will being in this study help me any way?

We cannot promise any benefits to you or others from your taking part in this research. However, possible benefits to others include the ability for scientists to better understand back pain and help patients with back pain.

16. What happens to the information we collect?

Efforts will be made to limit your personal information, including research study and medical records, to people who have a need to review this information. We cannot promise complete secrecy. Organizations that may inspect and copy your information include the IRB and other representatives of this organization.

The auditors, and the IRB will be granted direct access to your records for verification of the research procedures and date. By signing this document you are authorizing this access.

We may publish the results of this research. However, we will keep your name and other identifying information confidential.

17. Can I be removed from the research without my OK?

The person in charge of the research study or the sponsor can remove you from the research study without your approval. Possible reasons for removal include:

a) you do not meet the study criteria

b) you fail to adhere to safety requirements set by the researcher during the study

c) a change occurs in your medical status during testing that would make tesing unsafe for you

d) if all or part of the study is discontinued for any reason by the investigator or university.

18. What else do I need to know?

This research study is being done by Drexel University.

Consent to Take Part in a Human Research Study Page 6 of 6

Signature Block for Capable Adult

Your signature documents your permission to take part in this research.

DO NOT SIGN THIS FORM AFTER THIS DATE → April 15, 2015 OHR

Signature of subject	Date
Printed name of subject	
Signature of person obtaining consent	Date

APPROVED
Human Research Protection
Protocol #[redacted]
Approval Date: [redacted]
Expiration Date: [redacted]

Drexel University
Recruiting Healthy Volunteers for a Research Study

Research Title: Use of EMG, fNIR and NMES to study muscle activity of the lumbar multifidus during 3 commonly used lumbar spine clinical tests: a pilot study.

Research Objectives: The purpose of our study is to investigate how well different muscle recording instruments are able to detect muscle activity during common clinical tests that are used to identify the kinds of exercises a person should do in therapy. The study involves:

- 1 testing session lasting 2 hours.
- Non-invasive recording of your trunk muscle activity during strength testing and three clinical tests of back muscle activity.
- Electrical stimulation to the back muscles while recording surrounding back muscle activity.

Information for Research Subject Eligibility:
You may qualify for this research study if you are between 18-50 years old, have NO history of low back pain for which you got medical treatment or that limited your function for more than 3 days and NO history of spine, hip or shoulder surgery.

Location of Research and Person to Contact for Further Information:
This research is approved by the Institutional review board.
If you are interested in participating in this study, please contact

Dr. Won Sung, PT, DPT:
215-762-3589 *(wss26@drexel.edu)*
2nd Floor, Three Parkway, 1601 Cherry Street
This research is being conducted by a researcher who is a member of Drexel University.

(Version 1)

215-762-3589 wss26@drexel.edu Back Muscle Study	215-762-3589 wss26@drexel.edu Back Muscle Study	215-762-3589 wss26@drexel.edu Back Muscle Study	215-762-3589 wss26@drexel.edu Back Muscle Study

Drexel UNIVERSITY
Office of Research

APPROVAL OF PROTOCOL

June 29, 2015

Sheri Silfies, PhD
Grad - Physical Therapy 1505
Race St Rm 925
Philadelphia, PA 19102
Phone: (215) 762-5136
sheri.p.silfies@drexel.edu

Dear Dr. Silfies:

On <06/29/2015> the IRB reviewed the following protocol:

Type of Review:	*Initial Protocol Application*
Title:	*Validating the Role of the Lumbar Multifidus Muscle in the Prone Instability Clinical Test*
Investigator:	*Silfies, Sheri P*
IRB ID:	*1506003709*
Funding:	*CNHP - Physical Therapy Internal*
Grant Title:	*None*
Grant ID:	*None*
IND, IDE or HDE:	*None*
Documents Reviewed:	*HRP 211 Initial Submission form, HRP 201 and FCOI forms for all personnel, HRP 503 Protocol 05/31/2015, HRP 502 Consent 05/31/2015 and Flyer*

On 06/29/2015 Approved Initial
Approval includes: Recruitment of 30 subjects: 15 healthy control subjects and 15 subjects with current/previous low back pain. HRP 503 Protocol 05/31/2015, HRP 502 Consent 05/31/2015 and Flyer.
According to 45 CFR 46.110, this study is Approved Expedited Category 4 and 7.

The IRB approved the protocol from <06/29/2015> to 06/28/2016 inclusive.

Before <05/14/2016> which is 45 days prior to study closure, you are to submit a completed "FORM: Continuing Review Progress Report (HRP-212)" and required attachments to request continuing approval or closure. If continuing review approval is not granted before the expiration date of 06/28/2016 approval of this protocol expires on that date. Attached are stamped approved consent documents. Use copies of these documents to document consent. In conducting this protocol you are required to follow the requirements listed in the INVESTIGATOR MANUAL (HRP-103).

Sincerely,

Melissa A. Casey BS, MB, CIP
Institutional Review Board (IRB #1)

Drexel University
Consent to Take Part
In a Research Study

1. Title of research study: Validating the role of the lumbar multifidus muscle in the prone instability clinical test.

2. Researcher: Sheri P. Silfies PT, PhD

3. Why you are being invited to take part in a research study

We invite you to take part in a research study because you are either:

a healthy individual between the ages of 18-45 years of age, with no history of low back pain for which you got healthcare services or caused you pain or loss of normal function for greater than 3 days;

OR

between the age of 18-45 with current low back pain or a history of low back pain that has limited your physical activitywithin the last 12 months.

4. What you should know about a research study

- Someone will explain this research study to you.
- Whether or not you take part is up to you.
- You can choose not to take part.
- You can agree to take part now and change your mind later.
- If you decide to not be a part of this research no one will hold it against you.
- Feel free to ask all the questions you want before you decide.

5. Who can you talk to about this research study?

If you have questions, concerns, or complaints, or think the research has hurt you, talk to the research team by contacting Dr. Sheri Silfies at 215-553-7010.

This research has been reviewed and approved by an Institutional Review Board (IRB). An IRB reviews research projects so that steps are taken to protect the rights and welfare of human subjects taking part in research. You may talk to them at (215) 255-7857 or email HRPP@drexel.edu for any of the following:

- Your questions, concerns, or complaints are not being answered by the research team.
- You cannot reach the research team.
- You want to talk to someone besides the research team.
- You have questions about your rights as a research subject.
- You want to get information or provide input about this research.

6. Why are we doing this research?

Exercise is one of the most effective ways to treat low back pain. Special clinical tests have been identified that are able to predict a patient's success with reducing low back pain through exercise. The

Subject Initials: _____

purpose of this study is to better understand what it is about these tests that helps physical therapists predict a person's success with exercise. We are hopeful that this information will actually help to improve exercise selection for patients with low back pain.

7. How long will the research last?

We expect that you will be in this research study for 2 hours.

8. How many people will be studied?

We expect about 30 people will be in this research study. This study will only occur in the Philadelphia area.

9. What happens if I say yes, I want to be in this research?

The **first step**: We will ask you some simple questions about your low back and general health to make sure you meet all the criteria for the study.

If you have a condition that meets the exclusion criteria, then you will not be able to participate in the study.

If you meet the criteria, the **second step** will be to collect some information about you:

> We will collect general information about you including your age, gender, measurement of your height and weight, as well as the length of your back. You will also be asked to complete a questionnaire on your beliefs about pain.
>
> If you have low back pain or a history of low back pain, you will also complete questionnaires about your back pain and how back pain effects your daily activity.

The **third step** will be laboratory testing. Testing takes place at the Rehabilitation Sciences Labaratory at Drexel University on the 2nd floor of 1601 Cherry Street, Philadelphia. This testing will take place during one session lasting approximately 2 hours.

> You will be asked to wear shorts and sportsbra/halter top during the procedure. Clean clothing will be provided for you by the researcher if necessary.

Setup Procedure:

Sensors will be taped to your back, legs and stomach muscle that detect muscle activity during the testing.

Sensors that detect movement will be taped to several locations on your body: outside of your knee, over your pelvis, at your midback, and just below your neck.

The setup for this will take 60 minutes. The sensors and electrodes will stay on you for the remainder of the experiments, with the exception of 2 low back electrodes that will be removed during the experiment for the muscle stimulation experiment.

Back Muscle Tests

> The back muscle testing will consist of 5 different tests. These tests will take 10 minutes.
>
> 1) You will lie on your stomach with cross your arms across your chest and asked to lift your chest off the table so that your back is parallel to the floor. You will hold this position for 10

Subject Initials: _____

seconds, and then return to a resting position an the examiner will apply pressure to back to make the muscles work harder to hold you in position. You will perform 2 repetitions of this activity with 30 second rest between each test.

2) You will lie on your back with your hips and knees bent to a comfortable position. You will be asked to lift your buttocks off the table and then straighten one knee. You will hold this position for 5 seconds and then the examiner will push down on your leg to make the muscles work harder to hold you in position. Two repetitions will be performed for each side and there will be 30 seconds rest between each repetition.

3) You will lie on your back with your back supported. You will be asked to perform a curl up activity for 5 seconds and the examiner will try to push you back toward the support. Two repetitions will be performed with 30 seconds rest between each repetition.

4) You will be standing while holding a plastic pipe, with your arms in front of you and level with the floor. The examiner will apply a resisting force upward through the pipe, while you try to keep you arms in the same position. Two repetitions will be performed for each side and there will be 30 seconds rest between each repetition.

5) You will be standing while holding a plastic pipe, with your arms over your head. The examiner will apply a force downard through the pipe, while you try to keep your arms in the same position over your head. Two repetitions will be performed for each side and there will be 30 seconds rest between each repetition.

Prone Instability Test

This is a commonly performed clinical tests used in physical therapy for patients with low back pain. During this test, you will be asked to lie on your stomach for the examiner to identify painful areas on your back. A force gauge will record the amount of force the examiner uses to cause discomfort over different levels of your low back. Once any painful area is identified, you will lie on your stomach with your feet supported on the ground. Pressure will be applied to the same area that was identified previously. This will be repeated for up to three trials with 2 minutes in between trials.

You will then be asked to raise your legs off the floor to contract the muscles across the back, and the same force will be applied to the back. This will be performed for up to 4 trials with 2 minutes rest between trials.

During these tests, we will be collecting information from the sensors to determine changes in spine position during the test, the muscles that are working as you perform this test, and your pain levels during this test. This test will take approximately10 minutes.

Muscle Stimulation Test

You will be asked to lie on your stomach. Using the same force as applied in the first test, up to 4 compressions at the same level will be applied with 2 minutes rest in between.

An electric muscle stimulator which is commonly used in physical and occupational therapy treatments will be used to cause contractions of your back muscles. This unit uses three "AA" batteries to provide an electrical signal that causes muscle to contract.

You will be asked to lie on the examination table on your stomach. The electrodes used to measure muscle activity will be removed from your back. Self adhesive muscle stimulation electrodes will be

Subject Initials: _____

placed along the muscles of your low back. Pillows will be placed under your hips to make sure your back is in the right position for testing as well as for your comfort. The muscle stimulator will be turned on until a visible muscle contraction is seen and felt by the examiner. The muscle stimulator will be on for up to 20 seconds. You may feel a sensation similar to a muscle cramp while the stimular is on. While the muscle stimulator is turned on, the same force used during prior tests will be applied to your back. After the contraction, there will be a 2 minute resting period. There will be up to 4 repetions of this activity. This test will take approximately 20 minutes.

10. What are my responsibilities if I take part in this research?

If you take part in this research, it is very important that you:

- Follow your researcher's instructions.
- Tell the researcher right away if you have a complication or injury.

11. What happens if I do not want to be in this research?

You may decide not to take part in the research and it will not be held against you.

12. What happens if I say yes, but I change my mind later?

You agree to take part in the research now and stop at any time it will not be held against you.

13. Is there any way being in this study could be bad for me?

Risks:

Muscle Soreness and/or joint (common): There is some risk of muscle soreness or joint related soreness after the testing. This is similar to the soreness you will experience after exercising and should fade within 12-36 hours.

Electrical stimulation (rare): There is risk of skin irritation during the electrical stimulation. We will minimize these risks by making sure your skin is prepared properly prior to the testing by cleansing the area with soap and water. We will also use a hypoallergenic electrode pad.

14. Do I have to pay for anything while I am on this study?

There is no cost to you for participating in this study.

15. Will being in this study help me any way?

We cannot promise any benefits to you or others from your taking part in this research. However, possible benefits to others include the ability for scientists to better understand back pain and help patients with back pain.

16. What happens to the information we collect?

Efforts will be made to limit your personal information, including research study data, to people who have a need to review this information. We cannot promise complete secrecy. Organizations that may inspect and copy your information include the IRB and other representatives of this organization.

The auditors and the IRB will be granted direct access to your records for verification of the research procedures and date. By signing this document you are authorizing this access.

Subject Initials: _____

We may publish the results of this research. However, we will keep your name and other identifying information confidential.

17. Can I be removed from the research without my OK?

The person in charge of the research study or the sponsor can remove you from the research study without your approval. Possible reasons for removal include:

a) you do not meet the study criteria
b) you fail to adhere to safety requirements set by the researcher during the study
c) a change occurs in your medical status during testing that would make tesing unsafe for you
d) If all or part of the study is discontinued for any reason by the investigator or university

18. What else do I need to know?

This research study is being done by Drexel University.

Authorization to Use and Disclose Protected Health Information

Federal law provides additional protections of your personal information that are described here.

A. Individually Identifiable Health Information That Will Be Collected

The following personal health information about you will be collected and used during the research study and may be given out to others:

- Your name, telephone number, date of birth;
- LBP medical history;
- Information from clinical examination and other tests or procedures described in this consent form.
- Information learned during telephone calls, surveys, questionnaires done as part of this research study;

B. Who Will See and Use Your Health Information within Drexel University

The researcher and other authorized individuals involved in the research study at Drexel University will see your health information and may give out your health information during the research study. These include the researcher and the research staff, the institutional review board and their staff, legal counsel, research office and compliance staff, officers of the organization and other people who need to see the information in order to conduct the research study or make sure it is being done properly. Your health information may be disclosed or transmitted electronically.

C. Who Else May See and Use your Health Information

Other persons and organizations outside of Drexel University may see and use your health information during this research study. These include:

- Governmental entities that have the right to see or review your health information, such as The Office for Human Research Protections, and the Food and Drug Administration
- The sponsors of this research study and persons that the sponsor may hire to work on the research study.

Subject Initials: _____

If your health information is given to someone not required by law to keep it confidential, then that information may no longer be protected, and may be used or given out without your permission.

D. Why your health information will be used and given out

Your health information will be used and given out to carry out the research study and to evaluate the results of the study.

E. If you do not want to give authorization to use your health information

You do not have to give your authorization to use or give out your health information. However, if you do not give authorization, you cannot participate in this research study.

F. How to cancel your authorization

At any time you may cancel your authorization to allow your health information to be used or given out by sending a written notice to Human Research Protection at 1601 Cherry Street, 3 Parkway Bldg., Mail Stop 10-444, Philadelphia, Pennsylvania, 19102. If you leave this research study, no new health information about you will be gathered after you leave. However, information gathered before that date may be used or given out if it is needed for the research study or any follow-up.

G. When your authorization ends

Your authorization to use and give out health information will continue until you withdraw or cancel your authorization.

After the research study is finished, your health information will be maintained in a research database. Drexel University shall not re-use or re-disclose the health information in this database for other purposes unless you give written authorization to do so. However, the Drexel University Institutional Review Board may permit other researchers to see and use your health information under adequate privacy safeguards.

H. Your right to inspect your medical and research records

You will not be able to look at your *research* records while you are taking part in this research study. You have the right to look at your *medical* records at any time during this research study. Your personal information will be made available in an emergency if doctors need this information to treat you. You can have access to your *research study information* when the *study is over*. However, the researcher does not have to release research information to you if it is not part of your medical record.

Subject Initials: _____

Permission to Take Part in a Human Research Study

Signature Block for Capable Adult

Your signature documents your permission to take part in this research.

DO NOT SIGN THIS FORM AFTER THIS DATE → **06/28/2016** HRP

Signature of subject	Date
Printed name of subject	
Signature of person obtaining consent	Date

Drexel UNIVERSITY
Office of Research

APPROVAL OF PROTOCOL

June 29, 2015

Sheri Silfies, PhD
Grad - Physical Therapy 1505
Race St Rm 925
Philadelphia, PA 19102
Phone: (215) 762-5136
sheri.p.silfies@drexel.edu

Dear Dr. Silfies:

On <06/29/2015> the IRB reviewed the following protocol:

Type of Review:	*Initial Protocol Application*
Title:	*Validating the Role of the Lumbar Multifidus Muscle in Controlling Forward Bend of the Lumbar Spine*
Investigator:	*Silfies, Sheri P*
IRB ID:	*1506003710*
Funding:	*CNHP - Physical Therapy Internal*
Grant Title:	*None*
Grant ID:	*None*
IND, IDE or HDE:	*None*
Documents Reviewed:	*HRP 211 Initial Submission form, HRP 201 and FCOI forms for all personnel, HRP 503 Protocol 05/31/2015, HRP 502 Consent 05/31/2015 and Flyer*

On 06/29/2015 Approved Initial
Approval includes: Recruitment of 15 healthy control subjects. HRP 503 Protocol 05/31/2015, HRP 502 Consent 05/31/2015 and Flyer. According to 45 CFR 46.110, this study is Approved Expedited Category 4 and 7.

The IRB approved the protocol from <06/29/2015> to 06/28/2016 inclusive.

Before <05/14/2016> which is 45 days prior to study closure, you are to submit a completed "FORM: Continuing Review Progress Report (HRP-212)" and required attachments to request continuing approval or closure. If continuing review approval is not granted before the expiration date of 06/28/2016 approval of this protocol expires on that date. Attached are stamped approved consent documents. Use copies of these documents to document consent. In conducting this protocol you are required to follow the requirements listed in the INVESTIGATOR MANUAL (HRP-103).

Sincerely,

Melissa A. Casey BS, MB, CIP
Institutional Review Board (IRB #1)

Drexel University
Consent to Take Part
In a Research Study

1. Title of research study:

Validating the role of the lumbar multifidus muscle in controlling the forward bend of the lumbar spine.

2. Researcher: Sheri P. Silfies PT, PhD

3. Why you are being invited to take part in a research study

We invite you to take part in a research study because you are a healthy individual between the ages of 18-45 years of age, with no history of low back pain for which you got healthcare services or caused you pain or loss of normal function for greater than 3 days.

4. What you should know about a research study

- Someone will explain this research study to you.
- Whether or not you take part is up to you.
- You can choose not to take part.
- You can agree to take part now and change your mind later.
- If you decide to not be a part of this research no one will hold it against you.
- Feel free to ask all the questions you want before you decide.

5. Who can you talk to about this research study?

If you have questions, concerns, or complaints, or think the research has hurt you, talk to the research team by contacting Dr. Sheri Silfies at 215-553-7010.

This research has been reviewed and approved by an Institutional Review Board (IRB). An IRB reviews research projects so that steps are taken to protect the rights and welfare of human subjects taking part in research. You may talk to them at (215) 255-7857 or email HRPP@drexel.edu for any of the following:

- Your questions, concerns, or complaints are not being answered by the research team.
- You cannot reach the research team.
- You want to talk to someone besides the research team.
- You have questions about your rights as a research subject.
- You want to get information or provide input about this research.

6. Why are we doing this research?

Exercise is one of the most effective ways to address low back pain. Special clinical tests have been identified that are able to predict a patient's success with reducing low back pain through exercise. The purpose of this study is to better understand what it is about these tests that helps physical therapists

Subject Initials: _____

predict a person's success with exercise. We are hopeful that this information will actually help to improve exercise selection for patients with low back pain.

7. How long will the research last?

We expect that you will be in this research study for 2 hours.

8. How many people will be studied?

We expect about 15 people will be in this research study. This study will only occur in the Philadelphia area.

9. What happens if I say yes, I want to be in this research?

The **first step**: We will ask you some simple questions about your low back and general health to make sure you meet all the criteria for the study.

If you have a condition that meets the exclusion criteria, then you will not be able to participate in the study.

If you meet the criteria, the **second step** will be to collect some information about you:

> We will collect general information about you including your age, gender, measurement of your height and weight, as well as the length of your back.

The **third step** will be laboratory testing. Testing takes place at the Rehabilitation Sciences Labaratory at Drexel University on the 2nd floor of 1601 Cherry Street, Philadelphia. This testing will take place during one session lasting approximately 2 hours.

> You will be asked to wear shorts and sportsbra/halter top during the procedure. Clean clothing will be provided for you by the researcher if necessary.

Setup Procedure:

Sensors will be taped over muscles on your back, legs and stomach. These sensor detect muscle activity during the testing.

Sensors that detect movement will be taped to several locations on your body: outside of your knee, over your pelvis, at your midback, and just below your neck.

The setup for this will take 60 minutes. The sensors will stay on you for the remainder of the experiments, with the exception of 2 low back sensors that will be removed midway through the experiment for muscle stimulation. Once the muscle stimulation study is completed, the 2 low back electrodes will be re-applied for the duration of the study.

Back Muscle Tests

> The back muscle testing will consist of 5 different tests. These tests will take 10 minutes.
>
> 1) You will lie on your stomach with cross your arms across your chest and asked to lift your chest off the table so that your back is parallel to the floor. You will hold this position for 10 seconds, and then return to a resting position an the examiner will apply pressure to back to make the muscles work harder to hold you in position. You will perform 2 repetitions of this activity with 30 second rest between each test.

Subject Initials: _____

2) You will lie on your back with your hips and knees bent to a comfortable position. You will be asked to lift your buttocks off the table and then extend one knee. You will hold this position for 5 seconds and then the examiner will push down on your leg to make the muscles work harder to hold you in position. Two repetitions will be performed for each side and there will be 30 seconds rest between each repetition.

3) You will lie on your back with your back supported. You will be asked to perform a curl up activity for 5 seconds and the examiner will try to push you back toward the support. Two repetitions will be performed with 30 seconds rest between each repetition.

4) You will be standing while holding a plastic pipe, with your arms in front of you and level with the floor. The examiner will apply a resisting force upward through the pipe, while you try to keep you arms in the same position. Two repetitions will be performed for each side and there will be 30 seconds rest between each repetition.

5) You will be standing while holding a plastic pipe, with your arms over your head. The examiner will apply a force downard through the pipe, while you try to keep your arms in the same position over your head. Two repetitions will be performed for each side and there will be 30 seconds rest between each repetition.

Once the back muscle tests have been completed, you will be asked to stand and perform 6 forward bends and return to standing. Following the forward bends, you will be asked to lie on your stomach. Two sets of muscle recording electrodes will be removed, and replaced with 2 sets of muscle stimulating electrodes. You will receive muscle stimulations until the muscles are fatigued and no longer able to make a visible contraction using the muscle stimulator. The muscle stimulating electrodes will then be replaced with 2 sets of muscle recording electrodes. You will be asked to perform 6 forward bends.This portion of the testing will take approximately 20 minutes.

10. What are my responsibilities if I take part in this research?

If you take part in this research, it is very important that you:

- Follow your researcher's instructions.
- Tell the researcher right away if you have a complication or injury.

11. What happens if I do not want to be in this research?

You may decide not to take part in the research and it will not be held against you.

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Risks:

Muscle Soreness and/or joint (common): There is some risk of muscle soreness or joint related soreness after the testing. This is similar to the soreness you will experience after exercising and should fade within 12-36 hours.

Subject Initials: _____

Electrical stimulation (rure): There is risk of skin irritation during the electrical stimulation. We will minimize these risks by making sure your skin is prepared properly prior to the testing by cleansing the area with soap and water. We will also use a hypoallergenic electrode pad.

14. Do I have to pay for anything while I am on this study?

There is no cost to you for participating in this study.

15. Will being in this study help me any way?

We cannot promise any benefits to you or others from your taking part in this research. However, possible benefits to others include the ability for scientists to better understand back pain and help patients with back pain.

16. What happens to the information we collect?

Efforts will be made to limit your personal information, including research study and medical records, to people who have a need to review this information. We cannot promise complete secrecy. Organizations that may inspect and copy your information include the IRB and other representatives of this organization.

The auditors and the IRB will be granted direct access to your records for verification of the research procedures and date. By signing this document you are authorizing this access.

We may publish the results of this research. However, we will keep your name and other identifying information confidential.

17. Can I be removed from the research without my OK?

The person in charge of the research study or the sponsor can remove you from the research study without your approval. Possible reasons for removal include:

a) you do not meet the study criteria

b) you fail to adhere to safety requirements set by the researcher during the study

c) a change occurs in your medical status during testing that would make tesing unsafe for you

d) If all or part of the study is discontinued for any reason by the investigator or university

18. What else do I need to know?

This research study is being done by Drexel University.

Subject Initials: _____

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Federal law provides additional protections of your personal information that are described here.

A. Individually Identifiable Health Information That Will Be Collected

The following personal health information about you will be collected and used during the research study and may be given out to others:

- Your name, telephone number, date of birth;
- LBP medical history;
- Information from clinical examination and other tests or procedures described in this consent form.
- Information learned during telephone calls, surveys, questionnaires done as part of this research study;

B. Who Will See and Use Your Health Information within Drexel University

The researcher and other authorized individuals involved in the research study at Drexel University will see your health information and may give out your health information during the research study. These include the researcher and the research staff, the institutional review board and their staff, legal counsel, research office and compliance staff, officers of the organization and other people who need to see the information in order to conduct the research study or make sure it is being done properly. Your health information may be disclosed or transmitted electronically.

C. Who Else May See and Use your Health Information

Other persons and organizations outside of Drexel University may see and use your health information during this research study. These include:

- Governmental entities that have the right to see or review your health information, such as The Office for Human Research Protections, and the Food and Drug Administration
- The sponsors of this research study and persons that the sponsor may hire to work on the research study.

If your health information is given to someone not required by law to keep it confidential, then that information may no longer be protected, and may be used or given out without your permission.

D. Why your health information will be used and given out

Your health information will be used and given out to carry out the research study and to evaluate the results of the study.

E. If you do not want to give authorization to use your health information

You do not have to give your authorization to use or give out your health information. However, if you do not give authorization, you cannot participate in this research study.

Subject Initials: _____

F. How to cancel your authorization

At any time you may cancel your authorization to allow your health information to be used or given out by sending a written notice to Human Research Protection at 1601 Cherry Street, 3 Parkway Bldg., Mail Stop 10-444, Philadelphia, Pennsylvania, 19102. If you leave this research study, no new health information about you will be gathered after you leave. However, information gathered before that date may be used or given out if it is needed for the research study or any follow-up.

G. When your authorization ends

Your authorization to use and give out health information will continue until you withdraw or cancel your authorization.

After the research study is finished, your health information will be maintained in a research database. Drexel University shall not re-use or re-disclose the health information in this database for other purposes unless you give written authorization to do so. However, the Drexel University Institutional Review Board may permit other researchers to see and use your health information under adequate privacy safeguards.

H. Your right to inspect your medical and research records

You will not be able to look at your *research* records while you are taking part in this research study. You have the right to look at your *medical* records at any time during this research study. Your personal information will be made available in an emergency if doctors need this information to treat you. You can have access to your *research study information* when the *study is over*. However, the researcher does not have to release research information to you if it is not part of your medical record.

Subject Initials: _____

Permission to Take Part in a Human Research Study

Signature Block for Capable Adult

Your signature documents your permission to take part in this research.

DO NOT SIGN THIS FORM AFTER THIS DATE → **06/28/2016** HRP

Signature of subject	Date
Printed name of subject	
Signature of person obtaining consent	Date

Drexel University

Recruiting Healthy Volunteers for a Research Study

Research Title: Validating the role of the lumbar multifidus muscle in controlling forward bend of the lumbar spine.

Research Objectives: The purpose of our study is to investigate the effects of muscle fatigue during forward bending. The study involves:

- 1 testing session lasting 2 hours.
- Non-invasive recording of your trunk muscle activity during strength testing and forward bending.
- Electrical stimulation to the back muscles to fatigue the back muscles.

Information for Research Subject Eligibility:

You may qualify for this research study if you are:

1) Between 18-45 years old
2) Have NO history of low back pain for which you received medical treatment or that limited your function for more than 3 days
3) NO history of spine or hip surgery

Location of Research and Person to Contact for Further Information:

This research is approved by the Institutional review board.
If you are interested in participating in this study, please contact

Dr. Won Sung, PT, DPT:
215-762-3589 *(wss26@drexel.edu)*
2nd Floor, Three Parkway, 1601 Cherry Street

This research is being conducted by a researcher who is a member of Drexel University.

215-762-3589 wss26@drexel.edu Back Muscle Study	215-762-3589 wss26@drexel.edu Back Muscle Study	215-762-3589 wss26@drexel.edu Back Muscle Study	215-762-3589 wss26@drexel.edu Back Muscle Study

6.6 Appendix F: RUSI Analysis Method

Images were saved from RUSI unit, to AVI files. These clips were converted in imageJ offline to a continuous grey scale image. Analysis of contraction time and thickness were performed using the scale setting feature, which takes a pixel count of a marked area with a known distance. Marker scales on the RUSI video were used to provide imageJ a known length and duration to calculate pixel count as seen below. A line is drawn in relation to the time scale below. The known distance of that line in seconds is entered to set scale. This is also done to scale for length using depth markers on the image.

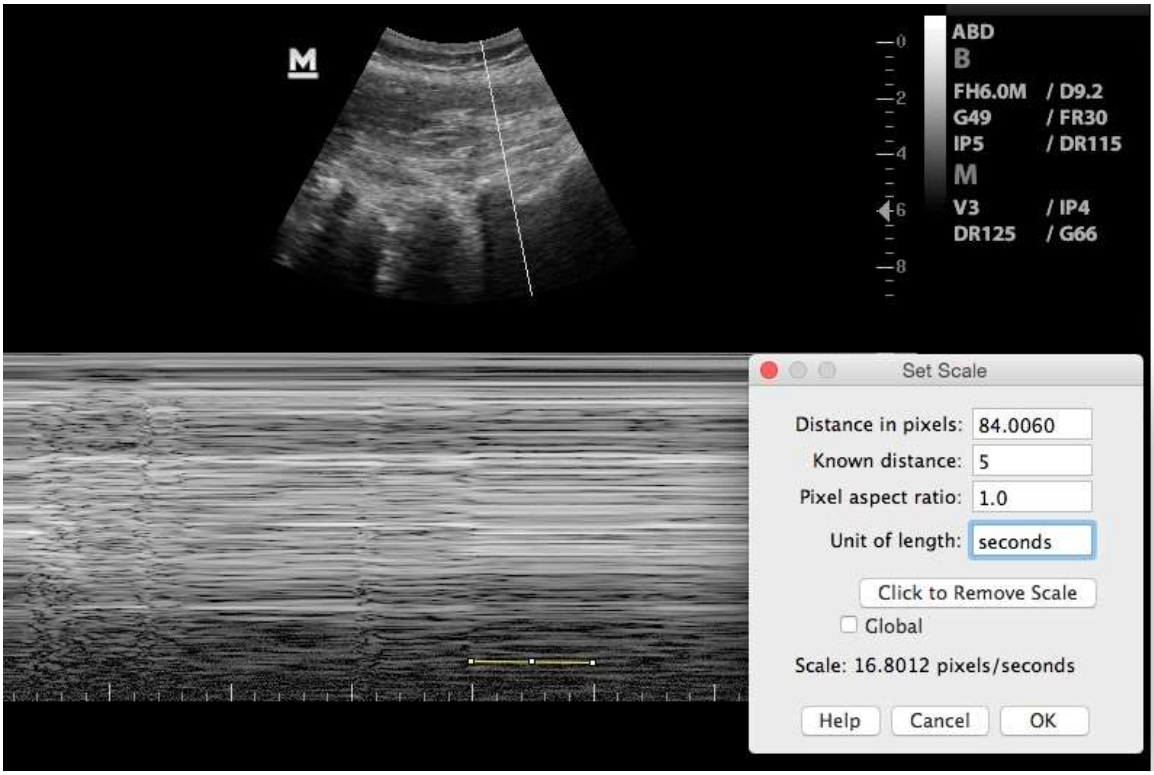

Figure 6.1 Image of Lumbar erector spinae. Erector spinae can be distinguished through the presence of the transverse process which is different in appearance to the

Identifying contraction times:

Within imageJ, a scanning window of 1 second duration and equal thickness of the LM/LES at rest was created to measure mean grey scale histogram. The b-mode image above the time series, gave the rater an indication of when the muscle underwent contraction, through visualized thickness change of the muscle. This thickness change would cause a disturbance in the m-mode scan that was measured for contraction duration. This was performed by sliding the image through the histogram scanning window as seen below.

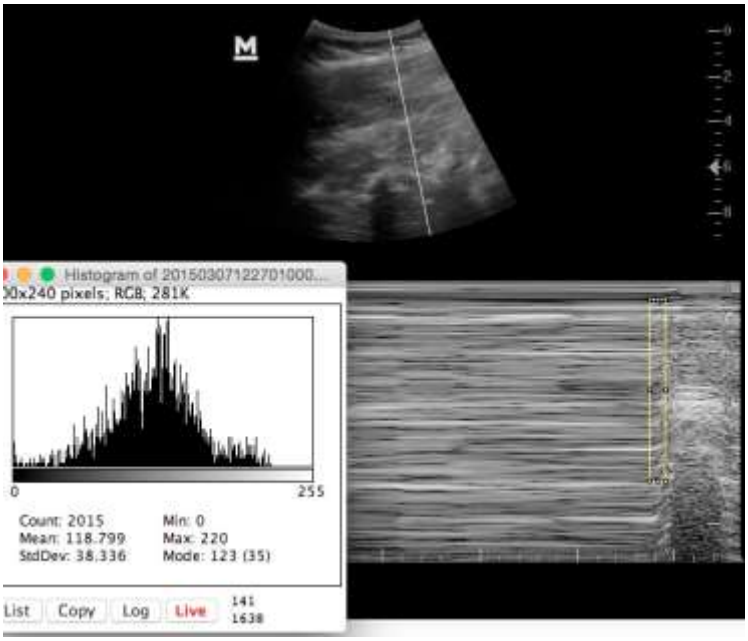

Figure 6.2 Muscle at rest prior to the contraction was analyzed for mean grey scale, using the b-mode video above to confirm there was a contraction.

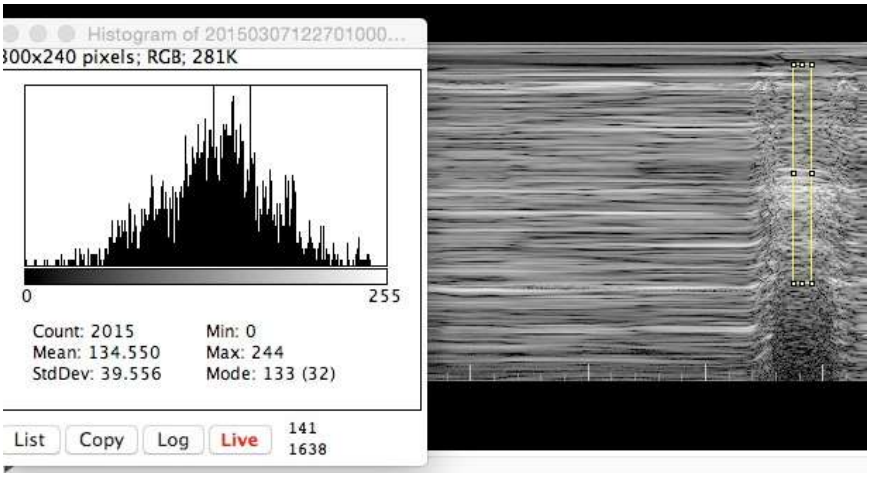

Figure 6.3 The video was passed through a sliding window with a live view of the histogram with changing grey scale.

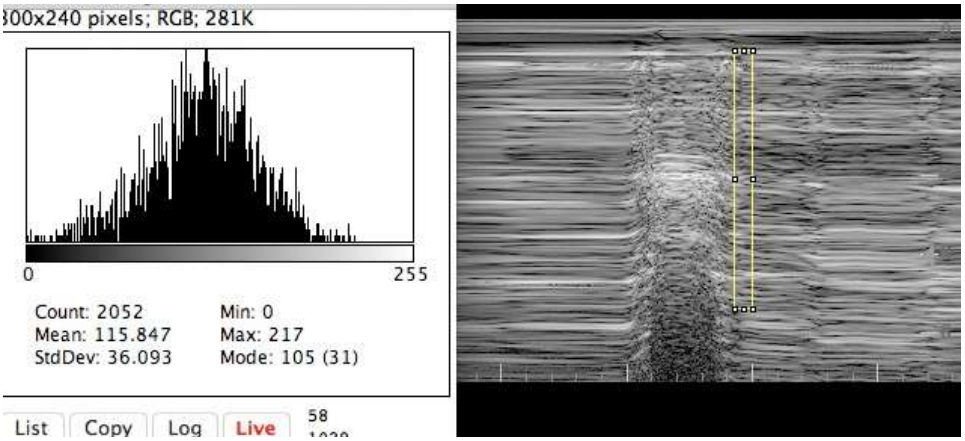

Figure 6.4 The offset of the contraction was confirmed by b-mode image showing relaxation of the muscle. This was associated with a return to mean grey scale values within 5% of the resting values as seen above.

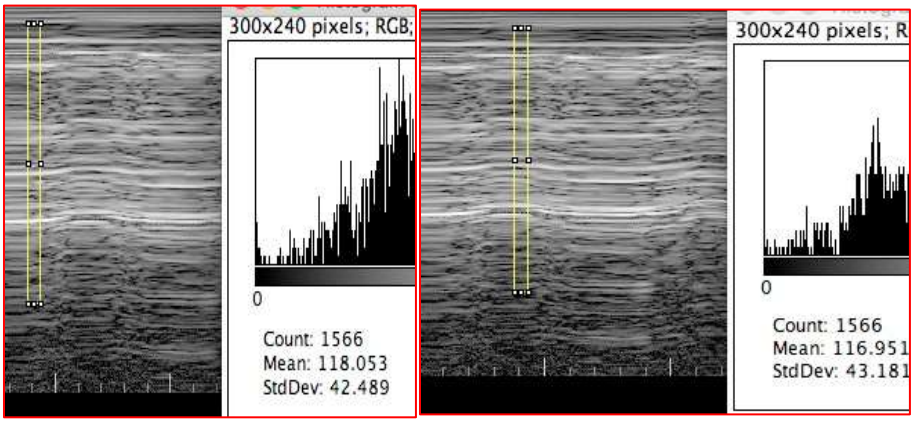

Figure 6.5 RUSI video image of movement artifact.

This process was repeated with a video clip of RUSI sound head compression along the surface of the skin. As seen above, the change in mean grey scale before the m-mode disturbance and during the m-mode disturbance does not exceed 5% of the baseline mean grey scale. This is beneficial when detecting the presence of contraction or disturbance in the m-mode time series from motion artifact such as breathing or skin movement along the probe from contraction distal to the area of inspection.

This method proved to be essential as during LM stimulation, there was m-mode disturbance at the LES probe. This could be from skin movement, movement of the pelvic during LM contraction, or pull of the LM fasciae onto the LES fasciae. However, the two step confirmation of grey scale analysis and confirmation with b-mode view above the m-mode time series was able to confirm if this was contraction or artifact.

An m-mode disturbance was classified as a contraction when there was a mean grey-scale change that exceeded 5% of baseline and there was confirmation with the b-mode image. Markers were used to signal the start of the onset and the offset of the contraction, and measured to determine the duration of contraction time, and were measured in seconds.

6.7 Appendix G: Subject preparation for kinematic data collection (adapted from Wattananon 2014)

This protocol was used for kinematic data used for preliminary study 5.1, and will also be used for prospective kinematic data collection during the forward bend.

1. Each subject exposed his/her back and his/her left knee.
2. Male subjects simply took off their shirt. Female subjects wore tank top or sport bra
3. All subjects wore shorts or loose fitting sweat pants to expose left knee.
4. All subjects wore their shoes throughout the test protocol.
5. The following body landmarks were identified and marked with a skin pen:
    - Femur sensor location: 15 cm. superior and 5 cm medial to left lateral epicondyle.
    - Pelvis/sacrum sensor location: over S2 spinous process located by palpating the PSISs and move medially to the spinous process between the PSISs.
    - Lumbar spine sensor location: over L1 spinous process located by palpating the level of the iliac crests and moving medially to the adjacent spinous process interspace which was L4-5 interspace. Palpating the L4 spinous process above the interspace and moving superiorly until the L1 spinous process was reached.
    - Thoracic spine sensor location: over T3 spinous process located by palpating the C7 spinous process or vertebra prominent. The C6

spinous process was identified by its anterior translation with cervical extension. The C7 was inferior to the C6. The spinous processes were counted inferiorly until the T3 spinous process was reached.

- Sternal notch, xyphoid process, C7, T8, T10, L3, L4, L5, ASISs, PSISs, and left medial and lateral epicondyles were also marked for digitization.

6. Any hair in the area around the mark was shaved.
7. The four sensors were mounted on a small piece of orthoplast.
8. Double-sided adhesive tape was cut to the same size of the orthoplast.
9. The backing on the tape was removed and the tape was affixed to the sensor.
10. The femur sensor was attached with the subject standing normally.
11. The pelvic and lumbar sensors were attached with the subject in a slightly forward bend position to prevent excessive sensor movement due to skin movement.
12. The thoracic sensor was attached with the subject's head and upper back in a slightly forward bend position and shoulders rounded.
13. The sensor itself was ensured to be right on top of the skin mark.

6.8 Appendix H: Kinematic Data Conversion and Calculation

- Rotation Convention

  α: Rotations about x-axis- flexion/extension

  β: Rotations about y- axis- lateral bending

  γ: Rotations about z- axis- axial rotation

  s: Sine

  c: Cosine

  Femur, Pelvis, Lumbar Spine, Thoracic Spine (x – y' – z")

Rotation Matrix

$$\begin{bmatrix} \mathbf{1} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \boldsymbol{c\alpha} & \boldsymbol{-s\alpha} \\ \mathbf{0} & \boldsymbol{s\alpha} & \boldsymbol{c\alpha} \end{bmatrix} \cdot \begin{bmatrix} \boldsymbol{c\beta} & \mathbf{0} & \boldsymbol{s\beta} \\ \mathbf{0} & \mathbf{1} & \mathbf{0} \\ \boldsymbol{-s\beta} & \mathbf{0} & \boldsymbol{c\beta} \end{bmatrix} \cdot \begin{bmatrix} \boldsymbol{c\gamma} & \boldsymbol{-s\gamma} & \mathbf{0} \\ \boldsymbol{s\gamma} & \boldsymbol{c\gamma} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{1} \end{bmatrix} =$$

$$\begin{bmatrix} \boldsymbol{c\gamma.c\beta} & \boldsymbol{-s\gamma.c\beta} & \boldsymbol{s\beta} \\ \boldsymbol{c\gamma.s\beta.s\alpha + s\gamma.c\alpha} & \boldsymbol{-s\gamma.s\beta.s\alpha + c\gamma.c\alpha} & \boldsymbol{-c\beta.s\alpha} \\ \boldsymbol{-c\gamma.s\beta.c\alpha + s\gamma.s\alpha} & \boldsymbol{s\gamma.s\beta.c\alpha + c\gamma.s\alpha} & \boldsymbol{c\beta.c\alpha} \end{bmatrix}$$

Angle Calculations

$$c\beta = \sqrt{(R_{12})^2 + (R_{22})^2} \qquad s\beta = R_{02}$$

$$c\alpha = \frac{R_{22}}{c\beta} \qquad s\alpha = -\frac{R_{12}}{c\beta}$$

$$c\gamma = \frac{R_{00}}{c\beta} \qquad s\gamma = -\frac{R_{00}}{c\beta}$$

Kinematics adapted from Polhemus Manual: Shoulder Kinematics Program written by Andrew Karduna, PhD, MCP Hahnemann University (1997)

- Vector coding

  Vector coding (VC) is calculated from the angle-angle plot which contains one state of variable (spatial). In angle-angle plot, the x coordinate represents angular displacement in degree from one segment (θ_1) and the y coordinate represents

angular displacement in degree from another segment (θ_2). The vector coding is calculated as follows:

$$\Theta_{VC}(i) = tan^{-1}\left[\frac{\theta_2(i+1) - \theta_2(i)}{\theta_1(i+1) - \theta_1(i)}\right], i = 1, 2, \dots, n-1$$

- Continuous relative phase (CRP) is performed on relative phase angle between distal and proximal segments which are derived from phase plane plots. The state of each signal is described by two state variables (spatial and temporal). In phase plane plot, the x coordinate represents either segmental angular displacement in degree ($\tilde{\theta}$) and the y coordinate represents segmental angular velocity ($\tilde{\omega}$). The phase angle is calculated as follows:

$$\Phi(i) = tan^{-1}\left[\frac{\tilde{\omega}(i)}{\tilde{\theta}(i)}\right], i = 1, 2, \dots, n$$

The relative phase angle (the difference between the phase angles of two signals) is performed as follows:

$$\theta_{CRP}(i) = \left|\Phi_{distal\ segment}(i) - \Phi_{proximal\ segment}(i)\right|$$

However, the derivation of θ_{CRP} requires that the state variables ($\tilde{\theta}$ and $\tilde{\omega}$) have the same amplitude and frequency. Therefore, the phase plane plot (θ vs. ω) should be scaled to the phase plane plot ($\tilde{\theta}$ vs. $\tilde{\omega}$) in order to account for amplitude and frequency differences in the state variables.

$$\tilde{\theta} = 2\left[\frac{\theta - \min(\theta)}{\max(\theta) - \min(\theta)}\right] - 1$$

$$\tilde{\omega} = \frac{\omega}{\max(|\omega|)}$$

- Angular deviation (AD) is used to determine variability in the VC and CRP measurement because both $\theta_{VC}$and θ_{CRP} are directional in nature. Angular deviation is calculated as follows:

  1. Decompose the angle of the vector coding (θ_{VC}) and the angle of the relative phase (θ_{CRP}) into a unit vector for each time point.

$$\theta_i = \begin{bmatrix} \cos\theta_i \\ \sin\theta_i \end{bmatrix}$$

  2. Find mean resultant vector for that time point across the multiple repetition.

$$\bar{\theta}_{unit\ vector} = \frac{1}{N}\sum_{i=N}^{N}\theta_i$$

  3. Find mean resultant direction by transforming the mean resultant vector using the four quadrant inverse tangent function.

$$\hat{\theta}_{angle} = 2tan^{-1}\frac{y}{\sqrt{x^2+y^2}+x}$$

  4. Find the length of the mean resultant vector. This critical quantity is used for the measurement of circular variability and/or hypothesis testing in directional statistics.

$$R_\theta = \left\|\bar{\theta}_{unit\ vector}\right\|$$

$$R_\theta = \sqrt{x^2+y^2}$$

  5. Find angular deviation or circular standard deviation

$$AD = \sqrt{2(1-R_\theta)}$$

6.9 Appendix I: Detailed methods for kinematic identification of aberrant movements.

Once kinematic data were reduced, coupling-angle diagrams and phase-plane diagrams were utilized to characterize movement. Wattananon 2014 used kinematic data that was collected simultaneously with examiner observation of the forward bend to detect aberrant movement. Cutoffs for kinematic detection of aberrant movement were based on agreements between the kinematic variables and the observed aberrant movement.

Graph Type	Definition
Coupling-angle diagram	The percentage of total angular displacement in the sagittal plane on x axis is plotted against coupling angle derived from segment angle –angle plot of the lumbar and pelvic segments on the y axis. Coupling-angle diagrams can be used to quantify relative or intersegment movement coordination and represent the point in time (% of movement) when hip/pelvic segment dominance occurred. This plot was used to determine the presence of altered lumbo-pelvic rhythm. Motion ratio between 2 segments greater than 45 degrees indicated proximal segment dominance, and less than 45 degrees indicated distal segment dominance (Figure 1a). Figure 1b depicts cut off points for altered lumbo-pelvic rhythm.
Phase Plane Diagram	The percentage of total angular displacement in sagittal plane (x axis) is plotted against instantaneous angular velocity (y axis). Phase-plane diagram can be used to capture movement control (smoothness of movement) during standing forward bend. This plot was used to determine when judder occurred. Figure 1c

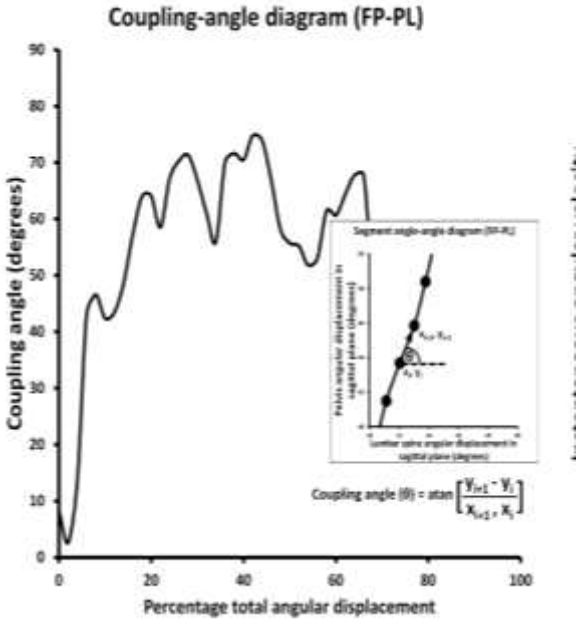

Figure 6.6: Coupling angle diagram of lumbar vs pelvic excursion during forward bending

Angle-Angle plot (insert) depicts a plot of angular displacement of the lumbar versus pelvic segments. A straight diagonal line on an angle-angle plot demonstrates 1:1 angular velocity of the 2 segments. A coupling angle diagram allows for standardization of the angle-angle plot by calculating a vector between two adjacent points. The diagram represents the coordination between segments quantifying the shape or trajectory of movement coordination between two segments relative to the percent of movement. The coupling-angle diagram can therefore, indicate when during the forward bend, when the alteration of lumbo-pelvic rhythm occurred. Figure and text adapted from Wattananon 2014.

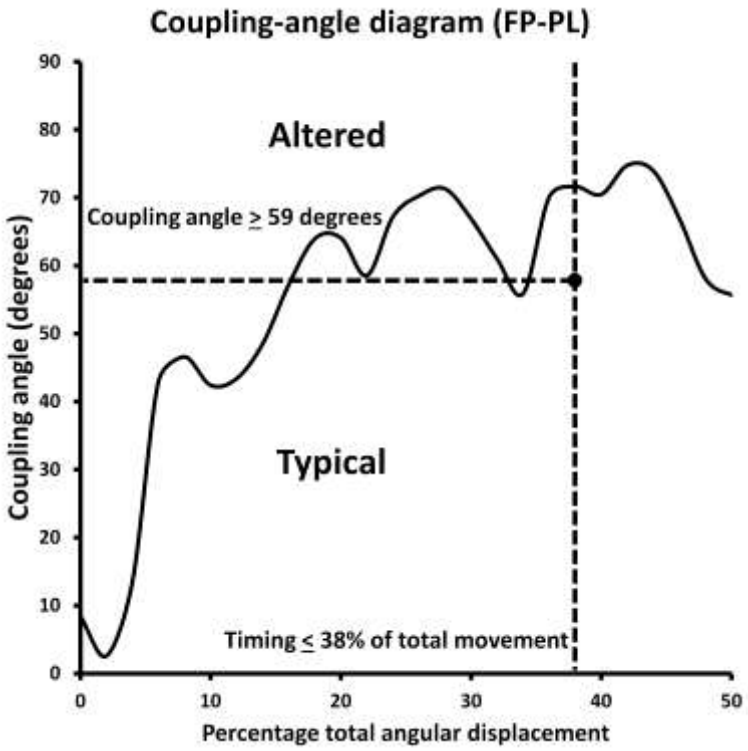

Figure 6.7 Example of a forward bend defined as demonstrating altered lumbo-pelvic rhythm.

A coupling angle of 59 degrees or greater that occurs within the first 38% of the forward bending movement was identified as having an altered lumbopelvic rhythm. MDC_{90} was determined to be 11.9 seconds. Figure and text adapted from Wattananon 2014.

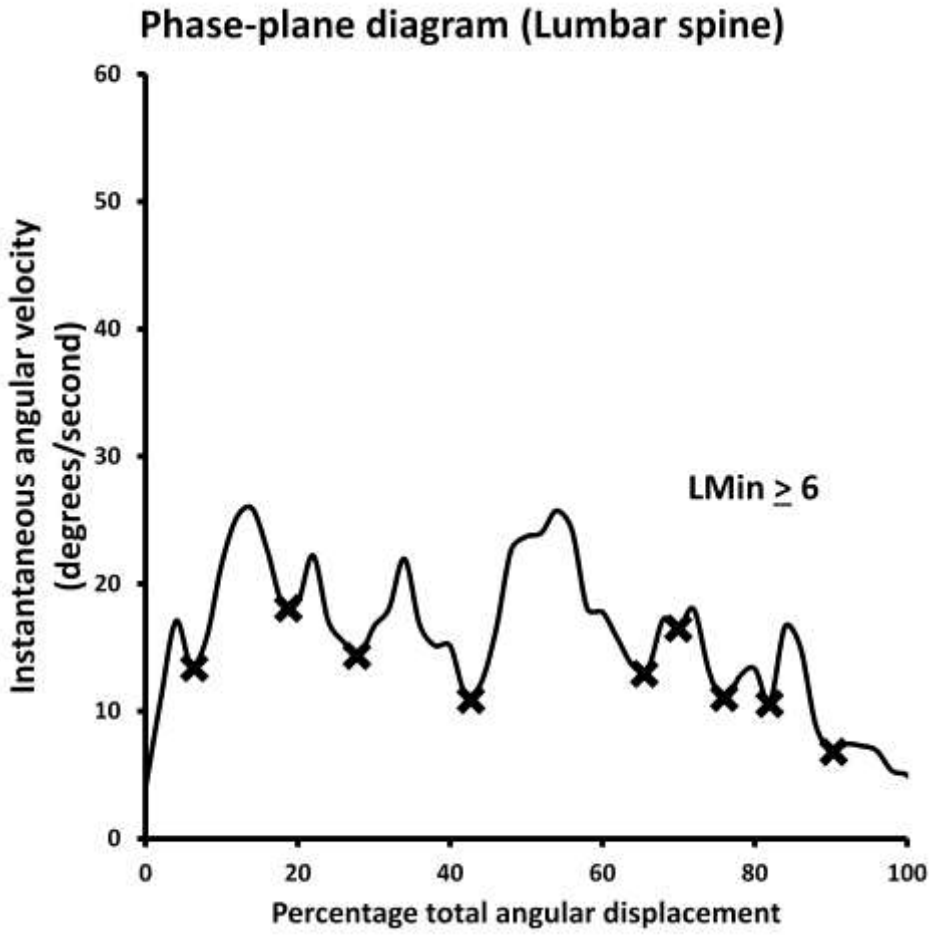

Figure 6.8 Phase plane diagram.

Plots angular velocity of the lumbar segment over the percentage of total angular displacement during the forward bend. The total local minima is used to determine the presence of judder: the presence of 6 or greater local minima classify a forward bend as having judder. The MDC_{90} for Lmin was found to be 1.58. Figure and text adapted from Wattananon 2014.

6.10 Appendix J: Methods for obtaining submaximal isometric volitional contractions of the trunk.

Submaximal Isometric Volitional Contraction (sMVIC) motions utilized for normalizing EMG data during preliminary study 5.4 and 5.5 .

1) Trunk flexion: subject lied on their back with knees and hips bent to a comfortable position. They were then asked to raise their trunk up so their shoulder blades rose above the surface. Subjects held that position for 10 second.

2) Unilateral bridge: subject lied on their back with knees and hips bent to a comfortable position. They were then asked to raise their right leg off the table and perform a bridge with the left leg by lifting the buttocks off the table until the pelvis and trunk were in a neutral position. This was then repeated with the opposite side.

3) Modified Biering-Sorensen (Coorevits et al., 2008): The subject was positioned in a prone so that the lower trunk was supported on an exam table. Support straps were placed around the thighs to stabilize subjects to the table. The subject's upper trunk was supported through their arms on a chair in front of the table. The subject was then asked to bring their arms across their chest and maintain the prone position using the trunk extensors for 10 seconds.

4) Seated row: The subject placed in a long sitting position with the trunk supported at a 45 degree angle. The subject held onto a 12 foot strap that was secured around the feet. The subject was then asked to use their arms to bring themselves to an upright seated position, and hold for 5 seconds.

Strength tests to performed to normalize EMG data for aim 1b:

Maximal resistance was applied using the "break test" method.

1) Trunk flexion, unilateral bridge, and modified Sorensen tests were performed as described above. However, for all tests, the tester applied maximal resistance until the subject could not hold the testing position.
2) Bilateral Shoulder Extension: While standing, subjects raised both arms to 90 degrees of shoulder flexion while holding onto a PVC pipe in their hands. The examiner resisted bilateral shoulder extension from this position (to avoid potential changes in SEMG data from examiner contact) until the subject could not hold the testing position
3) Bilateral Shoulder flexion: While standing, subjects brought their arms overhead while holding onto a PVC pipe in both hands. The examiner applied a downward force through the pipe, until the subject could not hold the testing position

6.11 Appendix K: NMES Pain Pressure threshold, force fatigue and EMG median frequency response to NMES

Pain gaiting mechanism of NMES: Pain Pressure and Pain threshold tests

Instrument: Wagner algometer with rubber tip with a load range from 4-44 lbs range was utilized. Gauge was marked in 0.5 lb increments.

Methods:

Pain pressure threshold was measured first. Pressure was applied to the L3 spinous process and subjects were asked to indicate when they first perceived the onset of pain. Pain tolerance was then assessed. Pressure was applied to the L3 spinous process to the maximum level the subject could tolerate. Numerical Pain rating scale (NPRS, 0-10) was collected with each application of pressure.

Both Pain pressure threshold and pain tolerance were done prior to the delivery of the NMES (pre-NMES) and during the NMES (NMES). 1 trial was performed for each condition. Pressure and pain ratings were compared using paired t-tests (alpha=.025) for pain pressure threshold and maximum pain tolerance.

Table 6.4 Pain pressure threshold. There were no statistically significant differences in *pressure thresholds* or *numerical pain rating scale* between pre-NMES to NMES conditions. *Only 1 subject (#7) had pain reduction with NMES during the pain pressure threshold measurement that exceeded the 2 point MDC for the NPRS.

Pain Pressure Threshold

	Pressure Applied (lbs)		**Pain Rating (NPRS)**	
Subject	Pre-NMES	NMES	Pre-NMES	NMES
1	8	8	2	2
2	6.5	7	2	2
3	9	9	1	2
4	10	10.5	2	2
5	6	7	3	2
6	4	4.5	2	2
7*	6	6	3	1
Mean (SD)	7.1 (2)	7.4 (1.9)	2.1 (0.7)	1.9 (0.4)

Maximal Pain Tolerance

	Pressure Applied (lbs)		**Pain Rating (NPRS)**	
Subject	Pre NMES	NMES	Pre NMES	NMES
1	12	13	7	7
2	16	14	8	7
3	11	11	6	6
4	18.5	19	7	7
5	17	21.5	7	8
6	9.5	11	6	8
7	12.5	11	7	7
Mean (SD)	13.8 (3.4)	14.4 (4.2)	6.9 (0.7)	7.1 (0.7)

There were no statistically significant differences in *maximal pain tolerance pressure* or *NPRS* between pre-NMES to NMES conditions. One subject (#6) exceeded the 2 point MDC for pain with NMES. This subject's pain increased during the NMES but also had a 1.5lbs increase in the maximal pressure tolerated.

Confirmation of fatigue with NMES

6 subjects underwent 2 testing sessions, at least 5 days apart to determine 1) force reduction with NMES and 2) EMG median frequency analysis of the lumbar multifidus and erector spinae after NMES.

Day 1: Subjects were tested for lumbar extensor strength using a modified Sorensen position, using a hand held dynamometer as recommended by committee members. Strength was measured using a 5 second hold prior to the delivery of NMES. NMES parameters were as described in the proposal. Strength testing was repeated every 10 stimulations.

Results

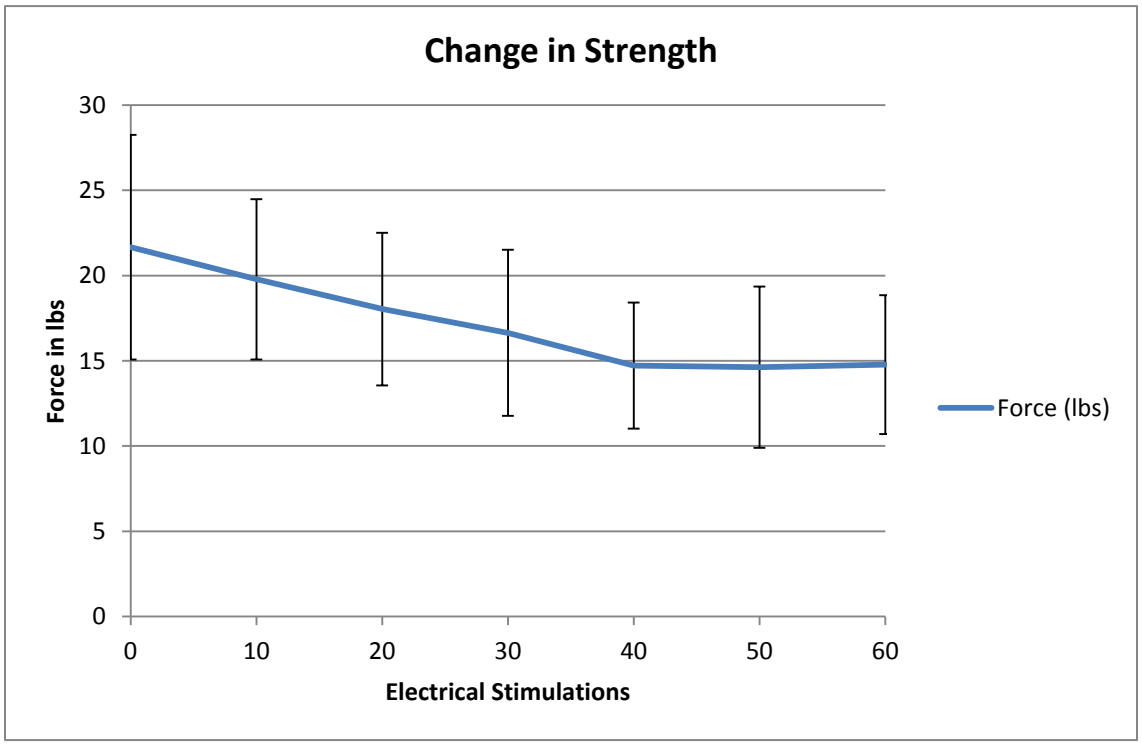

Figure 6.9 Strength reduction through iterative stimulations. Strength was measured every 10 electrical stimulations.

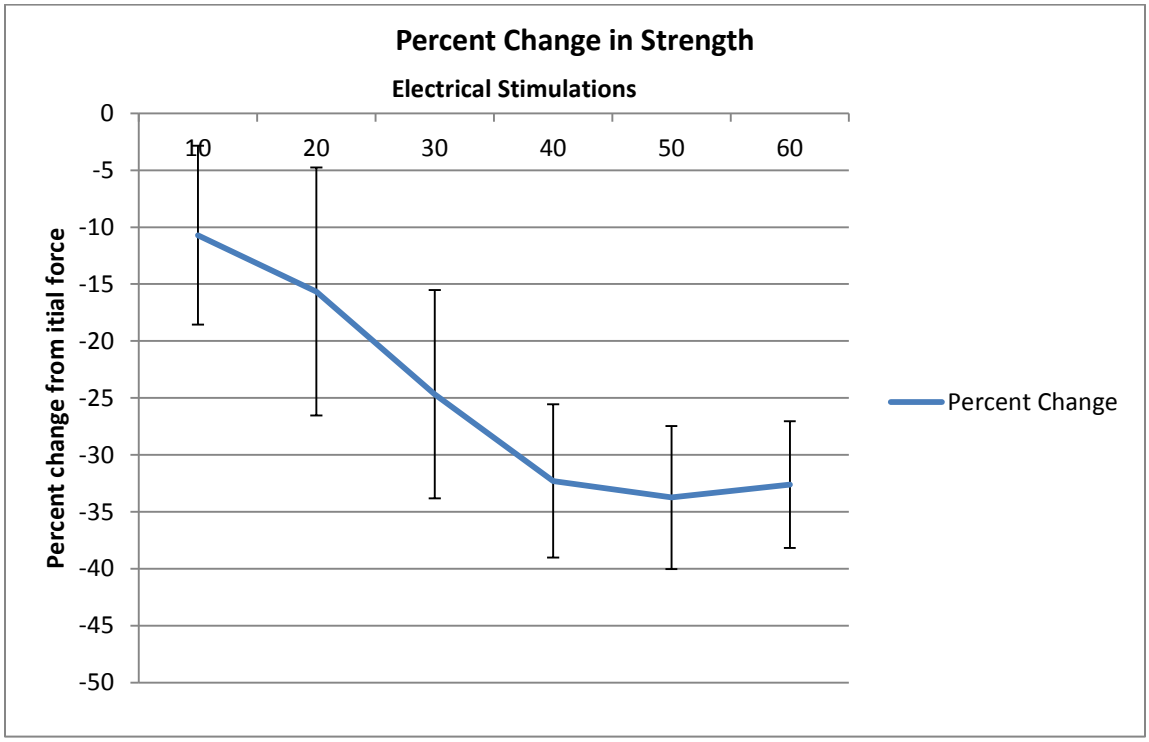

Figure 6.10 Percent change in strength through iterative stimulations. Subjects had on average, 32% reduction in strength by the 40th stimulation. Percent change is negative to reflect reduction in strength through iterative stimulations, compared to the first strength measure prior to NMES.

Conclusions:

Force reduction of trunk extensors reached a plateau after 40 stimulations. To ensure fatigue, it was decided to maintain 60 electrical stimulations.

Day 2.

After a rest period of at least 5 days, to allow for muscular recovery, subjects returned for testing. EMG electrodes were placed on the lumbar multifidus (LM) and lumbar erector spinae (LES) bilaterally, as described in previous methods in the proposal. EMG was collected during resisted trunk extension and a non-resisted 20 second extensor endurance test. They then received

60 electrical stimulations to the lumbar multifidus. Subjects were re-tested for extensor strength and endurance.

Of the 6 subjects, 4 subjects were able to maintain an isometric contraction during strength testing. Only 1 subject was able to maintain an extensor endurance position post NMES. Therefore, the isometric extensor strength testing of the subjects was used for to investigate median frequency changes of the extensors.

Results:

EMG median frequency of the LM and LES were averaged by side. Median frequency of the LM was reduced by an average of 32%±12% while the LES was reduced by an average of 8%± 4% among the 4 subjects able to assume the testing position and maintain an isometric contraction. Of the 2 subjects that were not able to maintain testing position, 1 subject had an increase in median frequency across both LES and LM, while the remaining subject demonstrated no change. In these 2 subjects, the median frequency response is likely due to a change from an isometric contraction to an eccentric contraction as load was applied.

Conclusion:

The 32% median frequency reduction across the LM is consistent with relevant literature on muscle fatigue. It suggests that we are able to preferentially recruit LM with the NMES. The median frequency reduction along with the force reduction supports our ability to fatigue the LM with NMES.

6.12 Appendix L: Forms and outcomes.

Numerical Pain Rating Scale

Subject Number _____ Testing Date_____

0-10 Numeric Pain Intensity Scale

0	1	2	3	4	5	6	7	8	9	10
No Pain					Moderate Pain					Worst Possible Pain

Spine Research Lab at Drexel University

OSWESTRY PAIN QUESTIONNAIRE

Subject Number:_____ Location:_____ Examiner: _____ Date:_____

Please read:
This questionnaire has been designed to give the therapist information as to how your back pain has affected your ability to manage in everyday life. Please answer every section, and mark in each section only the one box which applies to you. We realize you may consider that two of the statements in any one section relate to you, but please just *mark the box which most closely describes your problem.*

Section 1— Pain Intensity
- ☐ I have no pain at the moment
- ☐ The pain is very mild at the moment
- ☐ The pain is moderate at the moment
- ☐ The pain is fairly severe at the moment
- ☐ The pain is very severe at the moment
- ☐ The pain is the worst imaginable at the moment

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

Section 3— Lifting
- ☐ I can lift heavy weights without extra pain
- ☐ I can lift heavy weights but it causes extra pain
- ☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, i.e. 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
- ☐ Pain prevents me from walking more than 1/2 mile
- ☐ Pain prevents me from walking more than 1/4 mile
- ☐ I can only walk using a cane or crutches
- ☐ 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 more than 1 hour
- ☐ Pain prevents me from sitting more than 1/2 hour
- ☐ Pain prevents me from sitting more than 10 minutes
- ☐ Pain prevents me from sitting at all

Section 6— Standing
- ☐ I can stand as long as I want without extra pain
- ☐ I can stand as long as I want but it causes extra pain
- ☐ Pain prevents me from standing for more than 1 hour
- ☐ Pain prevents me from standing for more than 30 minutes
- ☐ Pain prevents me from standing for more than 10 minutes
- ☐ Pain prevents me from standing at all

Section 7— Sleeping
- ☐ I have no trouble sleeping
- ☐ My sleep is slightly disturbed (less than 1 hour sleep loss)
- ☐ My sleep is mildly disturbed (1-2 hours sleep loss)
- ☐ My sleep is moderately disturbed (2-3 hours sleep loss)
- ☐ My sleep is greatly disturbed (3-5 hours sleep loss)
- ☐ My sleep is completely disturbed (5-7 hours sleep loss)

Section 8— Social Life
- ☐ My social life is normal and gives me no extra pain
- ☐ My social life is normal but gives me extra pain
- ☐ Pain has no significant effect on my social life apart from limiting my more energetic interests, e.g. dancing, etc.
- ☐ Pain has restricted my social life and I do not go out as often
- ☐ Pain has restricted my social life to my home
- ☐ I have no social life because of pain

Section 9— Traveling
- ☐ I can travel anywhere without extra pain
- ☐ I can travel anywhere but I experience extra pain
- ☐ Paint is bad but I manage journeys of greater than one hour
- ☐ Pain restricts me to journeys of less than one hour
- ☐ Pain restricts me to short, necessary journeys under 30 minutes
- ☐ Pain prevents me from traveling except to the doctor or hospital

Section 10— Employment/Homemaking Duties
- ☐ My normal job/homemaking duties do not cause pain
- ☐ My normal job/homemaking duties cause pain but I can still do all that is required of me
- ☐ I can perform most of my job/homemaking duties but pain prevents me from performing more demanding tasks such as lifting, vacuuming, etc.
- ☐ Pain prevents me from doing anything except light activity
- ☐ Because of pain I cannot even perform light activity
- ☐ Pain prevents me from doing any job/homemaking activity

K01 12/08

Spine Research Lab at Drexel University

Fear Avoidance Beliefs Questionnaire

Subject Number:_____ Location:_____ Examiner: _____ 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 activities such as bending, lifting, walking or driving affect or would affect your back pain.

	Completely Disagree			Unsure			Completely Agree
1) My pain was caused by physical activity	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
2) Physical activity makes my pain worse	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
3) Physical activity might harm my back	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
4) I should not do physical activities which (might) make my pain worse	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
5) I cannot do physical activities which (might) make my pain worse	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6

The following statements are about how your normal work affects or would affect your back.

	Completely Disagree			Unsure			Completely Agree
6) My pain was caused by my work or by an accident at work	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
7) My work aggravated my pain	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
8) I have a claim for compensation for my pain	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
9) My work is too heavy for me	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
10) My work makes or would make my pain worse	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
11) My work might harm my back	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
12) I should not do my regular work with my present pain	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
13) I cannot do my normal work with my present pain	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
14) I cannot do my normal work until my pain is treated	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
15) I do not think that I will be back to my normal work within 3 months	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6
16) I do not think that I will ever be able to go back to work	□ 0	□ 1	□ 2	□ 3	□ 4	□ 5	□ 6

Borg Scale of Perceived Exertion

6	No exertion at all
7	Extremely light
8	
9	Very light
10	
11	Light
12	
12	Somewhat hard
14	
15	Hard (heavy)
16	
19	Extremely hard
20	Maximal Exertion

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Peer Reviewed Proceedings
Sung W, Silfies, S. (2016). Trunk Muscle Synergy Patterns During the Multifidus Lift Test and Prone Instability Test. APTA Combined Sections Meeting, Anaheim, CA.

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