2007

A COMPARISON OF SELECT TRUNK MUSCLE THICKNESS CHANGE BETWEEN SUBJECTS WITH LOW BACK PAIN CLASSIFIED IN THE TREATMENT-BASED CLASSIFICATION SYSTEM AND ASYMPTOMATIC CONTROLS

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ABSTRACT OF DISSERTATION

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ABSTRACT OF DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy
in the College of Health Sciences
at the University of Kentucky

By
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A COMPARISON OF SELECT TRUNK MUSCLE THICKNESS CHANGE BETWEEN SUBJECTS WITH LOW BACK PAIN CLASSIFIED IN THE TREATMENT-BASED CLASSIFICATION SYSTEM AND ASYMPTOMATIC CONTROLS

The purposes of this dissertation were to determine: 1) the relationship between muscle thickness change (MTC) as measured by rehabilitative ultrasound imaging (RUSI) and EMG activity in the lumbar multifidus (LM), 2) if motor control changes produced by experimentally induced pain are measurable with RUSI, 3) if a difference exists in MTC between subjects with low back pain (LBP) classified in the treatment-based classification system (TBC) system and controls, 4) if MTC improves following intervention.

Current literature suggests sub-groups of patients with LBP exist and respond differently to treatment, challenging whether the majority of LBP is "nonspecific". The TBC system categorizes subjects into one of four categories (stabilization, mobilization, direction specific exercise, or traction). Currently, only stabilization subjects receive an intervention emphasizing stability. Because recent research has demonstrated that motor control impairments of lumbar stabilizing muscles are present in most subjects with LBP, it is hypothesized that impairments may be present across the TBC classifications.

Study 1: Established the relationship between MTC as measured by RUSI and EMG in the LM. Study 2: Assessed MTC of the LM during control and painful conditions to determine if induced pain changes in LM and transverse abdominis (TrA) are measurable with RUSI. Study 3: Measured MTC of the LM and TrA in subjects with LBP classified in the TBC system and 20 controls. Subjects completed a stabilization program and were re-tested.

The inter-tester reliability of the RUSI measurements was excellent ($ICC_{3,3}$ =.91, SEM=3.2%). There was a curvilinear relationship (r = .79) between thickness change and EMG activity. There was a significant difference ($p < .01$) between control and painful conditions on 4 of the 5 LM tasks tested and on the TrA task. There was a difference in MTC between subjects and controls on the loaded LM test which varied by level and category. All categories were different from control on the TrA. Following

intervention the TrA MTC improved ($p < .01$). The LM MTC did not (p values from .13-.86).

These findings suggest MTC can be clinically measured and that deficits exist within TBC system. Significant disability and pain reduction were measured.

KEY WORDS: Lumbar Multifidus, Motor Control, Stabilization Training, Transverse Abdominis, Ultrasound Imaging

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Chapter I

Introduction

The current era of evidence-based practice has made health-care practitioners reflect on current practices for management of low back pain (LBP). The lifetime prevalence rate for an adult suffering an acute low back pain episode has been reported to be as high as 80% and the one-year prevalence rate approximately 65%. Point prevalence rate estimates range from 12-33%.[114] Current practice guidelines, such as those published by the Agency for Health Care Policy and Research[37] and the American Academy of Family Physicians[89] for the management of acute low back pain call for practitioners to reassure the patient that the episode will take a favorable course and to maintain their current level of activity.

It is a widely held belief that an acute low back pain episode will spontaneously resolve within 6 weeks regardless of intervention. This myth has been perpetuated in the literature. Recent studies describing the true course of a LBP episode suggest the majority do not spontaneously resolve. Pengel et al [90] included 15 relevant studies in a systematic review and reported a 58% (pooled mean) reduction in pain and disability could be expected over one month, and that pain and disability unresolved at three months persists. Abbott and Mercer[1] included 20 studies in their review on acute LBP in primary care, and concluded that although the belief that 80-90% of acute LBP will resolve in 6 weeks is widely perpetuated, there is considerable evidence that this is not the case. They state that persisting and recurring LBP is often "hidden" as many patients do not return to the health care system, and the natural course of acute LBP and associated disability is persistent and episodic. A review by Hestbaek et al [40] also found no evidence to support the claim that 80%-90% of patients become pain-free within one month. Instead they found, on average, that 62% of patients still experienced symptoms after 12 months and between 44% and 78% of subjects experienced relapses, Pengel[90] reported a 12-month recurrence rate of 73% (pooled mean).

Retrospective reviews have dispelled the myth of spontaneous recovery and new research exists to describe the natural course of LBP. Mortimer et al[79] followed LBP

subjects for a five-year period and found an average pain level of 28.5/100 with only 37% of the subjects reporting no disability after 5 years. Additionally, the researchers measured physical exercise levels and determined that the level of nonspecific physical exercise did not correlate with recovery. Dunn et al[26] followed LBP patients in primary care for one year and identified 4 distinct patterns for patients with LBP including; "persistent mild" (36%) patients had stable, low levels of pain, "recovering" (30%) started with mild pain, progressing quickly to no pain, "severe chronic" (21%) patients had permanently high pain, and "fluctuating" (13%) pain varied between mild and high levels. These distinctive patterns were maintained at one year and statistically significant differences in disability, psychological status, and work absence between groups were reported.

These findings suggest that the true course of LBP is not one of complete recovery and is not related with physical activity levels, but varies between subjects in a potentially predictable manner. Research further suggests that subjects with LBP are, in part, a heterogeneous group; highlighting the need for classification and challenging the validity of studies grouping subjects by duration of symptoms alone.[24]

The importance of identifying sub-groups of patients with LBP to guide clinical intervention and research has been highlighted as a research priority since 1996.[9, 10] Because of the difficulty grouping patients with LBP into relevant pathoanatomical categories,[2] classification schemes derived from clinical examination findings and historical factors have evolved. The Treatment-Based Classification (TBC) system, initially proposed by Delitto et al[25] in 1995, suggests that identifiable sub-categories of LBP patients exist. Research published since 2002 has validated this premise by demonstrating that sub-groups of patients with low back pain exist and respond differently to treatment. These groups include those who respond to manipulation,[15, 30] stabilization training,[41] and direction specific exercises.[11, 68] This line of inquiry has helped challenge the assertions that the majority of low back pain is “nonspecific” and that the watchful waiting treatment approach is superior to classification driven intervention.

The TBC system utilizes relevant historic factors, current disability and pain levels, and key clinical exam findings to classify patients into one of four categories;

direction specific exercise (flexion or extension), mobilization (lumbar or sacroiliac (SI) mobilization/manipulation), stabilization (core stabilization program) or traction (Table 1.1). The reliability of clinicians classifying subjects into each of the categories of TBC system has been established in two studies. Kappa values ranged from .56-.60 which is considered substantial.[33, 34] George and Delitto[35] identified those factors most powerful for discriminating between categories (see Table 1.1).

Treatment studies have demonstrated that clinical outcomes are superior when subjects receive an intervention matched to their specific classification as compared to subjects receiving unmatched intervention.[11, 15, 41] Additionally, a randomized trial has provided preliminary evidence that interventions based on the TBC produce superior outcomes when compared to LBP interventions based on current medical treatment guidelines.[32]

Motor Control

In addition to the importance of providing proper classification, emerging research suggests the need to address commonly identified motor control deficits thought to be present in nearly all types of patients with LBP. A growing body of neurophysiologic and clinical evidence suggests that the deep stabilizing muscles of the spine are impaired in those with LBP[29, 71, 94] which has led to the development of the motor control intervention approach for LBP.[58] The motor control model of spinal stabilization focuses on the function of deep spinal muscles because these structures are thought to have the ability to control motion between vertebral segments. The motor control approach emphasizes that subjects learn isolated volitional activation of deep trunk muscles,[93] primarily the transverse abdominis (TrA) and lumbar multifidus (LM). A recently published systematic review summarizes the clinical evidence to date supporting the motor control model of intervention.[29] It is not known whether motor control deficits are present across the different categories of the TBC system.

The motor control exercise approach is based upon the theory of spinal stabilization proposed by Bergmark.[7] Bergmark hypothesized the presence of two muscle systems responsible to maintain stability of the spine (1) the “global muscle system” consisting of large torque producing muscles that act on the spine without directly attaching to it. These muscles provide general trunk stabilization without the

capacity to control intersegmental motion, and (2) the "local muscle system" consisting of muscles that directly attach to the lumbar vertebra and are responsible for providing segmental stability and control such as the TrA and LM.

Panjabi [86, 87] supports Bergmark's theory with a more clinically relevant model of how spinal instability deficits can become pain producing. He defines spinal stability as a combination of the passive (osseous, articular, and ligamentous), active (force-generating capacity of muscles), and neural control (integration of afferent and efferent information) subsystems. This model describes the three subsystems as interdependent whereby one system is capable of compensating for deficits in another system. In this context, Panjabi redefines spinal instability to include a "neutral zone". The neutral zone is a region of intervertebral motion around the neutral posture (neither in flexion or extension) where little resistance is offered by the passive spinal column.[88] The components of the passive subsystem can only provide stability toward the ends of ranges of motion as the ligaments develop tension that resist spinal motion. Substantial stability to the spine in the vicinity of the neutral zone is thought to be provided by the active subsystem with contribution from the neural subsystem. It is hypothesized that the neural subsystem provides afferent information related to intersegmental joint position while in the neutral zone. In the presence of normally functioning subsystems, the size of the neutral zone is maintained, providing mechanical stability of the spine for normal functional movement. The size of the neutral zone has been shown to increase with ligamentous injury and intervertebral disc degeneration[78] and is thought to increase gradually due to dysfunction of any of the subsystems. The consequence is chronic pain and disability.

Major contributions by Bergmark (local muscle system) and Panjabi (inclusion of the neural subsystem's role in regulating normal spinal stability) has led to a line of research dedicated to the understanding of key muscle activation characteristics and how muscle activation is altered in the presences of LBP. Research has documented impairments in these deep muscles including atrophy,[6, 43, 44, 59, 64, 119] delayed activation,[28, 50, 52] and lack of volitional control,[39] and has exposed links between low back pain and various impairments in the muscles of the local system.[44-46, 52, 54, 67] Delays in muscle activation during limb movements as well as lower than expected levels of activation

during exercise [22, 50, 52, 53, 56, 82, 100] are thought to expose vertebral segments to abnormal translation and shear forces, eventually contributing to pain of spinal origin and disability.

Isolated atrophy of the LM muscle has been identified in subjects with acute LBP.[44] This atrophy has been shown to be selective to the side and level of pain in both acute and chronic[43] LBP subjects and may not reverse upon resolution of symptoms.[46] Using an porcine model, Hodges et al[48] identified specific patterns of atrophy when comparing a simulated disc lesion with a nerve lesion (transaction of the medial branch of the dorsal ramus). Cross-sectional area of the LM was reduced at the level of the lesion for the disc condition. The nerve lesion condition atrophy followed the innervations pattern of the LM, 1-3 levels below the level of the lesion. Lumbar multifidus atrophy has also been associated with leg pain,[59] and histological changes within the muscle have been identified in chronic LBP subjects, where fatty deposits replace multifidus muscle tissue.[6, 64, 77]

Rehabilitative Ultrasound Imaging

Clinical assessment of deep muscle performance to help guide clinical intervention is difficult. Electromyography (EMG), utilizing fine wire electrodes, has traditionally been used to assess the magnitude and timing of the TrA and LM providing useful information related to motor control. Unfortunately, the invasiveness of these procedures limits their routine clinical use[108] and so researchers and clinicians have relied on manual palpation techniques with limited evidence of assessment validity.

There is emerging research evidence supporting the use of ultrasound imaging as a non-invasive tool to assess deep muscle activation.[57] The application of ultrasound imaging for the purposes of biofeedback and muscle performance measurement by rehabilitation professionals is referred to as Rehabilitative Ultrasound Imaging (RUSI).[107]

RUSI can be used to assess muscle and other structures of interest during volitional activation or active movements. Several of these dynamic measures have been described in the literature including measurement of bladder [84, 110] (indirect assessment of pelvic floor muscle function), transverse abdominis[28], and lumbar multifidus movement.[61] Others researchers have described using RUSI during dynamic tasks to measure muscle length and fatigue.[101]

There are several architectural properties of muscle that can be measured during dynamic tasks including fascicle length, pennation angle,[69,74] and thickness.[57] Muscle thickness change (MTC) is the most common parameter measurable with RUSI that relates to muscle activation. Several researchers have utilized MTC as an indicator of muscle activation for the TrA [12, 13, 28, 39, 96, 108] and LM.[46, 112]The reliability of measuring muscle with RUSI has been reported by several authors[13, 39, 65, 76, 92, 103, 106, 108, 112] and is considered to be good to excellent. It should be noted that the majority of studies have assessed intra-tester reliability.

To validate the use of RUSI as a measurement tool for muscle contraction, thickness change has been compared to EMG activity of the gastrocnemius[73], transverse abdominis,[55, 76] external oblique, internal oblique, tibialis anterior, biceps brachii, and brachialis.[55] Although the relationship between MTC and EMG varies slightly between muscles and experimental protocols utilized, in general it is considered to be curvilinear.[57] Thickness change and EMG activity is relatively linear at lower levels of activation, then plateaus as EMG activity continues to increase.[55] The validity of using MTC as a measurement of muscle activation has been demonstrated in the TrA[55, 76] in an asymptomatic population by comparing thickness change to fine-wire EMG. Ferreria et al[28] demonstrated concurrent thickness and EMG attenuation of the TrA during an automatic recruitment task in subjects with LBP when compared to controls.

Limited data exist describing the use of RUSI to measure the paraspinal musculature during dynamic tasks. One study measuring thickness change of the lumbar paraspinals was performed by Wanatbe et al.[116] In this study the thickness of the erector spinae muscle was taken in the sagital plane over the transverse process. Subjects were seated and measures were obtained in neutral, flexed and extended postures. Results suggested that changes in muscle thickness could be reliably measured by ultrasound and that significant differences in thickness were present between positions. No EMG data were collected. Van et al[112] utilized RUSI to measure MTC change of the LM in a motor learning study and demonstrated that visual feedback from RUSI improved subjects' ability to learn how to volitionally activate the LM. Both of these studies add validity of RUSI as a noninvasive measurement tool for clinical assessment of muscle activation.

Purpose

Non-invasive measurement protocols using RUSI have been developed and validated for the TrA. There is a need to develop a similar measurement protocol for the LM. Classification systems for subjects with LBP, such as the TBC system, have been developed and validated. But, emerging evidence suggests motor control deficits are present in a wide variety of subjects with LBP and may be present across LBP categories. Therefore, the purposes of this dissertation are to:

1) Explore the relationship between MTC (as measured by RUSI) and EMG activity in the LM

2) Determine if motor control changes produced by experimentally induced pain can be measured by RUSI

3) Determine if there is a difference in MTC (as measured by RUSI) between subjects with LBP classified using the TBC system and asymptomatic controls.

4) Determine if abnormal MTC is altered after completion of a standardized lumbar stabilization intervention program.

Each study is described in the chapters that follow. The first study assessed the relationship between MTC and EMG activity. In the second study, an experimentally induced pain model was used to determine if RUSI could detect pain induced changes in the LM and TrA. The third study was designed to investigate potential differences in MTC across categories of the TBC and to assess changes pre-post intervention.

TABLE 1.1 Key examination findings and interventions for the Treatment-Based Classification System adopted from Delitto et al[25]

Classification	Key Examination Findings and Discriminating Factors in Bold[35]	Intervention
Stabilization	**Duration of symptoms greater than average of other categories (23 days)** **Pain intensity less than other categories (4.8)**	
	Positive on Clinical Predictive Rule for Stabilization Training Frequent prior episodes of low back pain due to minimal perturbations History of frequent manipulations History of trauma Positive response to prior use of brace or corset Generalized ligamentous laxity "Instability catch" during lumbar flexion or return from flexion Positive Prone Instability test	General stabilization program progression
Mobilization	**Average pain intensity 5.5, average duration 14.5**	
Sacroiliac Mobilization	Asymmetry of pelvic landmarks (ASIS, PSIS, iliac, iliac crest) in standing Positive standing flexion test Asymmetry of the PSIS in sitting Positive long-sit test Positive prone knee bend test	Sacroiliac region manipulation or muscle energy technique ROM exercises
Lumbar Mobilization	Positive on CPR for Manipulation Localized, unilateral low back pain Presence of an "opening" pattern (painful restricted flexion and contralateral side-bending) or "closing" pattern (painful and restricted ipsilateral side-bending) of active range of motion restrictions.	Lumbar regions manipulation and ROM exercises
Direction Specific Exercise	**Average pain intensity 6.2, more likely to have leg pain, average duration 14 days**	

Extension Syndrome	Symptoms centralize with lumbar extension	Extension exercises
Flexion Syndrome	Symptoms centralize with lumbar flexion	Avoidance of extension
Lateral shift	Visible frontal plane deviation of the shoulders relative to the pelvis Asymmetrical side-bending range of motion	Pelvic translocation exercises/then to extension program

Chapter II

Measurement of Lumbar Multifidus Muscle Contraction with Ultrasound Imaging

Background

This study was performed to establish the reliability of using ultrasound imaging to measure the thickness of the lumbar multifidus muscle and to establish the criterion validity of using thickness change as a measurement of muscle activation.

The reliability portion was performed on 8 asymptomatic subjects. The first measurements were taken, then subjects were repositioned and the measurements were repeated. For this study one single measurement was used. The reliability for the LM was good; however, in a study using ultrasound to measure thickness of the lateral abdominal wall muscles it was reported that using an average of 3 measures decreased the SEM substantially. Therefore, when establishing the reliability of the measurement in subjects with low back pain, an average of 3 measures was used.

In pilot work it was observed that the thickness of the multifidus tended to increase with increased loads when subjects lifted either their upper or lower extremity while in the prone position. Because of movement artifact that occurred in some subjects while the lower extremity was lifted, the prone upper extremity arm lifting model (subject prone with upper extremity in 120° of abduction, lifting extremity off of table) was used to compare EMG activity and muscle thickness change in the lumbar multifidus. Indwelling electrodes were placed into the deep fibers of the multifidus at the L4 level and simultaneous ultrasound and EMG data were collected to establish the relationship between thickness change and EMG activity. Data were collected on 12 subjects, but approximately one half of the data was not usable because of an equipment problem while performing the MVIC procedure. Therefore this study includes data on 5 subjects.

Chapter Synopsis

Rehabilitative ultrasound imaging (RUSI) has been validated as a noninvasive method to measure activation of selected muscles. The purpose of this study was to determine the relationship between muscle thickness change, as measured by RUSI, and EMG activity of the lumbar multifidus muscle in normal subjects.

Bipolar fine wire electrodes were inserted into the multifidus muscle at the L4 level of 5 subjects. Simultaneous EMG and RUSI data (muscle thickness) were collected while subjects performed 4 increasingly demanding postural response tasks known to activate the multifidus muscle. The change in muscle thickness between rest and activation was compared to EMG output over the four tasks. Additionally, normalized EMG data were correlated to normalized RUSI data.

Mean EMG data showed increasing levels of activation across tasks (19% to 34% of MVIC). There was a significant difference between tasks for EMG activity. Muscle thickness change as measured by RUSI was highly correlated with LM EMG activity of LM in asymptomatic subjects ($r = .79$ $p < 0.001$).

Results suggest that measurement of muscle thickness change using RUSI is a valid and practical method to measure activation of the lumbar multifidus muscle in a narrow range (19-34% of MVIC) for an asymptomatic population.

Introduction

Lumbar paraspinal musculature plays a key role in providing stability during dynamic tasks.[17] Of particular interest recently has been study of the lumbar multifidus muscle. Altered characteristics of the lumbar multifidus identified in low back pain subjects include histological changes,[118, 119, 122] girth changes,[45, 59] and deficits in motor control, recruitment, and endurance.[8, 22, 51]

Quantification of lumbar multifidus (LM) activation in those with low back pain may be helpful in determining effective intervention. The gold standard measurement tool used to assess muscle activation is electromyography (EMG). EMG measures the electrical activity in the muscle and can be interpreted to represent muscle activation. To ensure a reliable signal is obtained from the multifidus, an indwelling electrode should be used.[104] Ultrasonography offers a noninvasive method to measure muscle activation[55, 76] and has gained popularity in various aspects of low back pain rehabilitation.[13, 20, 42, 45-47] The application of ultrasound imaging for the purposes of biofeedback and muscle performance measurement by rehabilitation professionals is referred to as Rehabilitative Ultrasound Imaging (RUSI). Ultrasonography is an imaging technique utilizing high-frequency sound waves to evaluate tissue properties such as thickness. Ultrasound examination is considered low risk. According to the safety committee of the European

Committee for Medical Ultrasound (ECMUS), "*Based on scientific evidence of ultrasonically induced biological effects to date, there is no reason to withhold scanning for any clinical application*".

It is known that muscle thickness changes when the muscle is activated.[55] The amount of thickness change that occurs with muscle activation can be quantified by RUSI, comparing resting muscle thickness values to those obtained during muscle activation. Measurement of muscle thickness change compared to EMG activity has been performed on the gastrocnemius muscle,[73] on the transverse abdominis[76] and on other trunk and peripheral muscles.[55] To our knowledge no comparison has been performed on the lumbar multifidus. The purpose of this study is to determine the relationship between muscle thickness change, as measured by RUSI, and EMG activity of the LM in normal subjects.

Methods

Subjects

Five healthy subjects, 3 of which were female (mean age = 28.0 years SD 5.6, mean height = 170.7 cm SD 9.4, mean mass = 70.3 kg SD 15.9) volunteered for this study. Subjects were excluded if they had current or recent history (within 6 months) of LBP or hip pain, a history of lumbar/sacral surgery, congenital lumbar/sacral condition such as spondylolithesis, or spina bifida, or bony pathology such as a fracture. All volunteering subjects signed an institutional-review-board-approved consent form following verbal instructions of the procedure.

Procedures

Subjects were positioned prone on a standard plinth. An inclinometer was placed longitudinally over the lumbo/sacral junction and pillows were used to flatten the lumbar curve to less than 10 degrees. Subjects were then oriented to and practiced the maximum voluntary isometric contraction procedure performed with the elbows flexed to approximately 90 degrees and shoulders abducted to approximately 120 degrees. Subjects then lifted their head, trunk, and upper extremities and held with maximum effort against a load applied at the elbow by one of the researchers. The contralateral upper extremity lifting movement, used to activate the LM, was then practiced. This

consisted of the upper extremity lift with four levels of graded resistance as described below.

To study the LM, fine wire (California Fine Wire Company, Grover Beach, CA) electrodes were fabricated from pairs of nylon coated 50µm wires which were inserted into a 27ga hypodermic needle. Approximately 1–2mm of coating was removed from the tip of the wire, the tips were bent back at 2-3mm and 3-4mm respectively, and the needle and wires were sterilized. The L4 spinous process was identified, and the needle was inserted just lateral to the spinous process to the depth of the lamina, then withdrawn, leaving the electrode in the deepest portion of the LM muscle. A surface ground electrode was placed over the subject's lateral malleolus.

The ultrasound images were generated at 25Hz utilizing computerized ultrasonography (Sonosite 180plus, Sonosite Inc, Bothell, WA). The primary investigator operated the ultrasound unit and performed the scanning for this study. A 70mm 5-MHz curvilinear transducer was placed longitudinally along the spine with the mid- point over the L4 spinous process. It was moved laterally and angled slightly medially until the L4/5 zygapophyseal joint could be identified. This scan point is directly over the LM and a measurement from the most posterior portion of this landmark to the plane between the muscle and subcutaneous tissue was used for the linear measurement of the LM[98] at rest and during activation. An on-screen caliper was used to obtain the resting measurement, captured simultaneously with resting EMG data. The reliability of this measurement was established in a pilot study of 8 asymptomatic subjects ($ICC_{3,1}$=.85) and represents the ability to reliably capture and measure a given image. Subsequent images taken during the arm lifting tasks were saved and printed for off screen manual measurement. The reliability of this measurement ($ICC_{3,1}$=.95) represents the ability to consistently measure the same image off-screen (intraimage reliability). The sonograms of the LM captured during the arm lifting tasks were printed and measured off-screen to limit the total time of the tasks and limit fatigue. The muscle thickness measurements obtained during each task were normalized to the resting measurement and percent change from rest was calculated (Activity – Rest/Rest x 100). This percent change in muscle thickness from rest to activation represented muscle activation as measured by RUSI.

The MVIC data were collected as the subject performed the maximum upper extremity and trunk lift described. Two trials of 5 seconds each were performed and the greatest root mean square (RMS) peak .5 second MVIC recording was used to normalize the EMG activity. Normalization provides a standard reference of electrical activity and all data are reported as a percentage of the MVIC.

The contralateral arm lifting tasks were performed in the same plane as the MVICs. The subjects were instructed to lift their extremity straight up off of the table and hold for 8 seconds (see Figure 1). Two trials each of the 4 levels of increasingly demanding upper extremity lifting tasks were performed while EMG data and images were obtained simultaneously. The first level (no load) had resistance of only the limb; the next three levels (low, medium, and high load) had graded resistance based on the subject's body mass (see Table 2.1). The average of the two trials for each task was used for analysis.

EMG Analysis

The EMG data were sampled at 2000Hz using the Biopac II Student Lab Pro (Biopac System, Inc. Santa Barbara, CA) amplified 1000x and filtered at 30-500Hz. The Biopac has a signal to noise ratio of > 90dB and an input impedance of 1.0 MΩ. The data were saved and imported to a PC for analysis with Datapac software (Run Technologies, Mission Viejo, CA). RMS peak amplitudes were calculated for each 0.5 second period. Data from the middle three seconds of each trial were averaged and expressed as a percent of MVIC**.** The average of the two trials for each task was used for analysis.

Statistical Analysis

To determine if the individual tasks adequately increased muscle activation, a repeated measures analysis of variance with post hoc analysis (alpha level .05) was performed on the EMG data.

To determine if a correlation existed between the EMG and RUSI data points, a Pearson's correlation coefficient was calculated and a regression line was fit.

Results

The tasks studied were significantly different from each other ($F_{3,12} = 25.39$ $P < 0.001$). Post hoc analysis utilizing Bonferroni correction revealed significant differences between the no-load and medium and high load tasks, and between low load

and high load tasks. Muscle activation as measured by EMG correlated highly (r = .79 $P< 0.001$) with thickness change as measured by RUSI. There was a 0.01 improvement in r value between the first and second order regression equations (Figure 2.1). Table 2.2 includes values of muscle thickness in centimeters (cm) and EMG as a percent of MVIC for each task.

Discussion

Our main finding was that EMG activity and thickness change in the LM muscle during functional contractions is highly correlated. This result adds to the limited body of knowledge related to the use of RUSI as a measurement tool for muscle activation. A curvilinear relationship between thickness change in the LM muscle and EMG activity during the graded contralateral upper extremity lifting tasks was demonstrated, suggesting that RUSI may provide an alternative technique to measure LM muscle contraction. Previous research assessing the relationship between muscle thickness change and EMG activity in the transverses abdominis muscle used volitional activation matched to percent of MVIC[55, 76] through a large range of activation levels. For this study, the selected tasks produced, on average, a narrow range of activation from 19% to 34% of MVIC. EMG activation changed as expected based on the level of the task. There was a difference between no-load and medium and high load tasks and between low load and high load indicating these are true differences. Although not statistically significant, the difference between the no load and low task was 5%, and consistent with increases between levels of activation in previously cited studies of the transverse abdominis. Isolated volitional activation of the LM is discussed in the literature,[46] studying subjects trained to perform this activity may be a method for future research to study a broader range of activation levels.

Direct comparison of our EMG results is not possible as earlier studies that isolated EMG activity of the multifidus during contralateral limb movement in the prone position could not be identified. Arokoski et al in two separate papers[4, 5] studied a variety of stabilization exercises and reported an average of 41% MVIC for the lumbar multifidus during a standing, alternating shoulder flexion movement with an average load of 1.5 kilograms. Our average load across each task was 0.8 kilograms, which produces an average output of 28% of MVIC. Despite these methodological differences, research

to measure multifidus activity during various lumbar stabilization exercises, involving loaded limb movements, has shown somewhat similar activation levels to the present study.

Previous studies measuring thickness change and EMG activity of other muscles have reported conflicting results. Hodges et al[55] compared EMG activity to architectural change measured by RUSI in several muscles across a broad range of activation levels. They measured thickness change and EMG activity of the tibialis anterior, biceps brachii, brachialis, internal oblique and transverse abdominis and reported a curvilinear relationship where RUSI was useful to detect changes at low levels of contraction (up to approximately 20% of MVIC) and higher levels of contraction produce little further thickness change. McMeeken et al[76] measured the transverse abdominis during abdominal hollowing from 5% to 80% of MVIC and demonstrated a linear relationship between thickness change and EMG activity across all levels of activation measured.

Our methods reported here differed somewhat from similar research in that they did not include matching a volitional contraction to a set level of activation; rather, tasks thought to activate the LM at progressively greater levels were included. This resulted in measurement in a narrow range of muscle activation and is a limitation of the study. We cannot assume this relationship exists across the entire range of muscle activation since we tested a narrow range. With the limits of our study RUSI can detect changes in LM EMG activity from an average of 19% of MVIC (no load) to of 34% of MVIC (high load).

Further research is needed to determine if RUSI is a valid measure of LM activation across a greater range of activation levels, and in individuals with low back pain. If RUSI can be validated as a noninvasive measurement of LM muscular activity in the low back pain population, it may be useful for clinicians who use therapeutic exercise as an intervention in this population. RUSI could be used to measure potential LM activation impairment and how various interventions effect the impairment.

Conclusion

These results provide preliminary data on the potential use of RUSI to measure LM muscle activation. The measurement of muscle thickness change utilizing RUSI appears to be a noninvasive method to measure activation of the LM muscle as it is

highly correlated with EMG in a limited range (19-34% of MVIC) in an asymptomatic population.

Table 2.1

Graded resistance levels for upper extremity lifting tasks in kilograms.

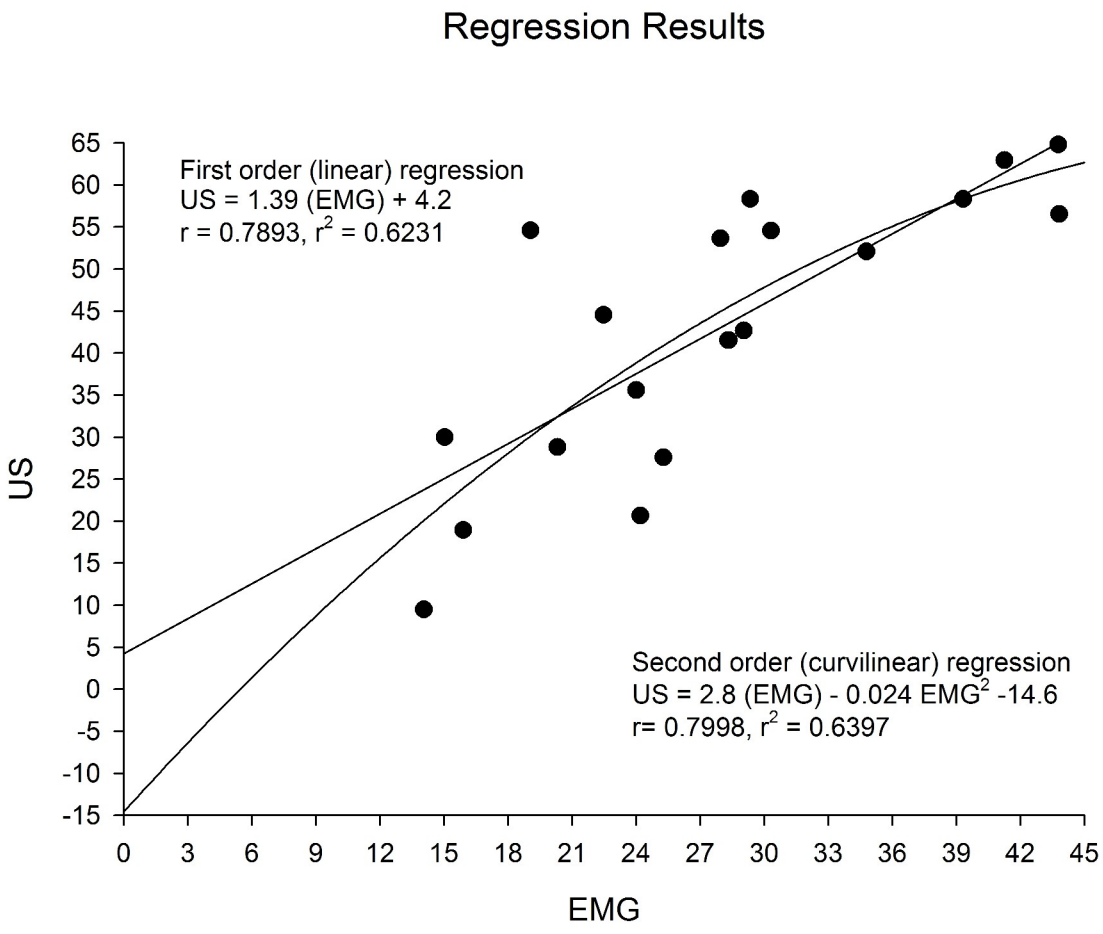
Subject Mass (Kg)	Low	Medium	High
< 68.2	.45	.68	.90
68.2-79.5	.45	.68	1.14
79.5-90.9	.45	.90	1.14
>90.9	.45	.90	1.36

Table 2.2

Mean and standard deviation values for the lumbar multifidus muscle during rest and each of the lifting task conditions. Ultrasound values are thickness measured in centimeters and EMG values are expressed as a percent of MVIC.
* indicates values are significantly different from No Load condition.
^indicates values are significantly different from Low Load condition.

Instrumentation	Lifting Task Conditions (X, SD)				
	Rest	No Load	Low	Medium	High
Ultrasound	2.48 (.19)	3.28 (.35)	3.50 (.29)	3.60 (.33)	3.68 (.29)
EMG	na	19.50 (5.94)	25.31* (7.15)	32.21* ^(7.58)	34.31* (8.85)

Figure 2.1 Regression between thickness change and EMG in the lumbar multifidus



Chapter III

Rehabilitative Ultrasound Measurement of Select Trunk Muscle Activation During Induced Pain

Background

The first study established the relationship between thickness change as measured by ultrasound imaging and EMG at relative low levels of activation (mean values were 19-34% of MVIC). It is thought that these lower levels of activation are all that is required of deep stabilizing muscles such as the multifidus to create adequate segmental stabilization.

Prior to utilizing this measurement in a clinical study, it was important to determine if the measurement was sensitive enough to detect changes in muscle performance thought to be present in subjects with LBP. It is difficult to recruit homogenous subjects with LBP, therefore, it has been recommended in the literature that an experimentally induced pain model be used when studying pain-related motor control issues. This allows for the control of pain levels as well as controlling for muscle performance changes that are thought to occur over time in subjects with LBP. Therefore, this study was performed to determine if ultrasound imaging was sensitive enough to measure pain related changes in the multifidus and transverse abdominis muscles.

There is a fairly large body of literature describing the use of ultrasound to measure muscle thickness of the transverse abdominis including two studies describing the relationship between thickness change and EMG activity. We added assessment of the transverse abdominis because of our desire to include this in a clinical study.

Chapter Synopsis

Rehabilitative ultrasound imaging (RUSI) is considered a valid method to measure muscle activation in key spinal muscles in asymptomatic subjects. Research measuring muscle activation with RUSI in painful subjects is limited. The aim of this study was to determine if changes in muscle activation from experimentally induced pain can be measured by RUSI.

Six male subjects performed tasks known to activate the transverse abdominis (TrA) and lumbar multifidus (LM) while RUSI measurements of muscle thickness were obtained during control and hypertonic saline conditions. The abdominal draw-in maneuver was used to volitionally activate the TrA and a series of upper extremity lifting tasks were used to automatically activate the LM. Pain was induced by injecting 5% hypertonic saline into the longissimus muscle adjacent to the LM at the L4 level. The percent change in muscle thickness from rest to contraction represented muscle activation.

Activation was significantly less ($p < 0.01$) during the painful condition on 4 of the 5 tasks performed for the LM and on the task performed for the TrA. These results indicate that RUSI can be used to measure pain-related changes in deep trunk muscle activation. Future research should include a larger sample size and women.

Introduction

Contemporary rehabilitation for low back pain (LBP) subjects includes specific exercise aimed at restoring motor control of key stabilizing muscles including the transverse abdominis (TrA) and the lumbar multifidus (LM).[45, 80, 83, 85, 95] Surface electromyography does not accurately measure activation characteristics of these deep spinal muscles,[75, 104] requiring invasive measurement techniques not routinely used in the clinical setting.[109] Rehabilitative ultrasound imaging (RUSI) can be used to assess muscle activation by measuring change in muscle geometry during contraction. The most common measurement utilized to assess muscle activation is change in muscle thickness.[49] Muscle thickness change has been shown to represent muscle activation by simultaneous EMG recording in the TrA muscle[55, 76] and the LM muscle[61] in normal subjects.

Few studies have been conducted to measure muscle thickness change with RUSI in subjects with LBP. Ferreira[28] et al demonstrated thickness change of the TrA is less in asymptomatic subjects with a history of LBP. This study utilized a loaded lower extremity task, similar to recumbent biking, to measure automatic recruitment of TrA over the course of the task. Critchley and Coutts[19] used RUSI to measure thickness change in the TrA in chronic LBP subjects performing the abdominal draw-in maneuver.

The magnitude of thickness change in the LBP subjects was significantly less than asymptomatic age matched control subjects. Thickness change of the LM has not been measured with RUSI in the LBP population. RUSI has been used as biofeedback during intervention in an acute LBP population where thickness change was thought to represent activation.[46]

Many researchers have reported changes in muscle activation in LBP subjects as compared to asymptomatic control subjects.[111] The majority of studies have utilized surface EMG to assess the response to pain in superficial muscles. Results vary widely and are in part dependent on the task studied, with some demonstrating hyperactivity and other demonstrating hypoactivity in the presence of pain. These results have been used to support and refute the two primary theories of how pain affects motor control 1) the pain-spasm-pain model (predicts pain increases activity as a protective response) and 2) the pain-adaptive model (predicts pain will cause an increase muscle activity when the muscles act as antagonist and decreases activity when the muscle is active as an agonist). van Dieen et al111 concluded that "lumbar erector spinae EMG activity in LBP subjects is highly variable and thought to depend upon the task studied."

Researchers demonstrating the effects of induced pain on trunk muscle activation also offer no consistent findings, with results appearing to vary depending on the task. Arendt-Nielsen et al[3] induced pain with hypertonic saline and demonstrated an increase in erector spinae activity during walking. Zedka et al[121] measured erector spinae activity during trunk flexion and extension before and after hypertonic saline induced pain and found an increase in activity when EMG activity was normally silent and a decrease or no change when EMG activity was normally high.

More recent work has focused on deep muscle activation, in particular on the timing of activation in the presence of pain. Delays in activation of the TrA, in response to rapid limb movement, have consistently been demonstrated in subjects with LBP,[54] subjects with a history of LBP in remission at the time of testing [50, 52] and in asymptomatic subjects when pain is experimentally induced.[53] There are several studies demonstrating various impairments of the LM in subjects with LBP including selective morphologic changes such as decreased girth and fatty infiltrate development.[21, 44, 46, 59, 119] Despite these consistent findings, muscle activation deficits of the LM have not been

consistently identified. Hodges et al[53] failed to show recruitment differences in the deep portions of the LM in response to rapid limb movements during induced pain. Other studies have shown diminished EMG activity in the LM during forward and backward bending[102] and a reduction in fatigue resistance.[99] Measurement of changes in muscle activation associated with LBP may be beneficial to the clinician in development of select intervention to reverse the identified impairment.

Experimental pain can be induced by many methods, but hypertonic saline-induced pain has been used extensively to test the effects of pain on various aspects of motor control [38] and utilized specifically to study the effects of pain on motor control of spinal muscles.[3, 53, 121] Intramuscular injection of hypertonic saline is thought to produce pain by primarily exciting nociceptive fibers and possibly by increasing the intramuscular sodium and potassium concentrations. Other possible contributors to the pain response are increases in intramuscular pressure and a nonspecific excitation of non-nociceptive afferents.[38] Interesting, it has been shown that injection of isotonic saline does not produce pain beyond that associated with the injection itself.[53] Using intramuscular injection of hypertonic saline to produce pain is considered safe, reliable and comparable to clinical pain.[38] The advantage of using experimental pain applied to healthy subjects over patients in clinical studies is the control obtained for pain intensities and duration. Such control may be important when measuring the LM because of its tendency to become inhibited quickly in those with acute LBP[46] and because of known morphological changes in chronic LBP subjects [59, 119, 120] which may affect measurement accuracy. To our knowledge, no study has demonstrated if RUSI can detect change in muscle activation in those with acute pain at the time of testing. Therefore, the aim of this study was to determine if changes in muscle activation from experimentally induced pain can be measured with RUSI.

Methods

Subjects

A convenience sample of 7 healthy male subjects (mean age = 26.0 years SD 7.3, mean height = 176.9 cm SD 10.7, mean weight = 83.0 kg SD 11.7) volunteered for this study. Females were not included because of known differences in LM activation levels.4 Potential subjects were also excluded if they had a history of LBP or hip pain,

spondylolithesis, or a congenital lumbar/sacral condition such as spina bifida. All volunteering subjects signed an institutional-review-board-approved consent form following verbal instructions of the procedure.

Procedures

Ultrasound measurements

Rest and activation measures (control and hypertonic saline conditions) of thickness of the TrA and LM were obtained using the Sonosite 180 Plus sonography unit, (Sonosite Inc, Bothell, WA) with a 70mm 2-5 MHz curvilinear transducer. The TrA measurements as described by Richardson et al were taken with the subjects in the supine hooklying position with the transducer placed just superior to the iliac crest along the axillary line.[97] To ensure measurements were taken at similar points along the TrA, the transducer was adjusted until the medial most portion of the TrA was visualized in the far left portion of the screen [39] (Figure 3.1). Subjects were then taught to preferentially activate their TrA by performing the abdominal draw-in maneuver with visual feedback from the ultrasound. Once the skill had been adequately learned (isolated TrA activation as determined by the tester viewing the RUSI) the resting measure was captured at the end of quiet expiration followed by the activation measure.

The LM measurements were taken with the subjects positioned prone on a standard plinth. An inclinometer was placed longitudinally over the lumbo/sacral junction and pillows were used to flatten the lumbar curve to less than 10 degrees. The L4 spinous process was identified by palpation and marked for reference. Then the transducer was placed longitudinally along the spine, moved laterally, and then angled slightly medial until the L4/5 facet joint could be identified. This scan point was directly over the lumbar multifidus. A measurement from this landmark to the plane between the muscle and subcutaneous tissue was used for the thickness measurement of LM at rest and during activation (Figure 3.2).[106]

To activate the LM, 2 trials each of 5 increasingly demanding contralateral upper extremity lifting tasks were performed while ultrasound images were obtained. The first task had resistance of only the limb with the shoulder adducted and the elbow fully flexed; next the shoulder was abducted to 120 degrees and lifted with just resistance from the limb, then graded resistance was added for the next 3 lifts based on the subject's body

weight (see Table 3.1). The average of the two trials for each task was used for analysis. A percent change from rest was calculated [(Activity – Rest)/Rest *100] for muscle thickness measures obtained during each task. This percent change in muscle thickness represented muscle activation as measured by RUSI. Resting and all TrA measurements were performed with the on-screen calipers. The intratester reliability of these measures was established in a pilot study (TrA ICC3,1 = 0.95, LM ICC3,1 = 0.85) performed on 8 asymptomatic subjects. LM images captured during the UE lifting tasks were saved and printed for off screen manual measurement. The intra-image reliability of this measurement was (ICC3,1=.95). The off screen LM activation measurements were taken by a researcher who was blind to both task and condition.

Induced pain

After completion of the measurements during the control condition, subjects remained positioned on the plinth. To induce acute pain, a 1.5ml bolus of hypertonic saline (5%) was injected into the longissimus muscle 6cm lateral to the L4 spinous process at a depth of approximately 3.5cm as described by Hodges et al[53.] Pain was measured on a 0-10 point visual analog scale at 60 seconds post injection and every 60 seconds thereafter. Reported pain scores had to reach = 4/10 and maintain that level throughout the hypertonic condition data collection. If reported pain dropped below the pre-determined threshold of 4/10, an additional 0.5ml bolus was administered. Subjects were offered a 0.5ml subcutaneous injection of 1% lidocaine to diminish the superficial pain associated with the subsequent saline injection.

Statistical Analysis

Paired t-tests were used to determine if muscle activation was different between the two conditions on each of the 5 activation tasks for the LM and on the volitional TrA contraction. The alpha level was set at = 0.05 and a Bonferroni correction was performed on the LM data to diminish the risk of committing a Type I error due to multiple comparisons. The correction was done by dividing the alpha level of 0.05 by the number of comparisons which was five. Therefore, the alpha level for acceptance for the LM was = 0.01 and remained at = 0.05 for the TrA.

Results

Of the seven subjects enrolled in the study, one did not reach the required pain therefore six subjects completed all aspects of the study. The results of the paired t-test indicated a significant difference ($p = < 0.01$) in LM muscle activation between the control and hypertonic saline conditions for all but the second activation task (see Figure 3.3). There was also a significant difference for the TrA between conditions ($p = < 0.01$) see Figure 3.4. Table 3.2 includes mean and SD values in centimeters for all measurements.

Discussion

The results of the present study indicate that induced pain attenuates the thickness change of the LM muscle during an automatic recruitment task and the TrA muscle during a volitional recruitment task. Research to establish the relationship between muscle thickness change and muscle performance measures such as EMG has been conducted on a variety of muscles including the TrA[55, 76] and LM. Hodges et al[55] reported a curvilinear relationship where maximum muscle thickness is reached at approximately 20% of MVIC. McMeeken et al[76] demonstrated a more linear relationship where thickness change can be observed up to 80% of MVIC. Thickness change in the TrA is considered a valid measure of muscle activation although the linearity of the relationship is controversial.[109]

Little research has been conducted on thickness change of the LM. In previous work, we demonstrated a curvilinear linear relationship ($r = .79$ $p < 0.001$) between LM thickness change and EMG activity across a narrow span of activation levels (19-34% of MVIC, see chapter 2). One study[116] utilized RUSI to evaluate thickness change in the lumbar erector spinae. The scan point for this study was lateral to the point used in the current study, over the transverse process, measuring thickness of the erector spinae group as a whole. This study did not include EMG, but did report intra and interrater reliability of the muscle thickness measurement ($R = 0.90$) and significant differences in muscle thickness between sitting flexion, neutral, and extended spinal positions across all lumbar levels.

The importance of LM function in LBP has been established,[23, 44, 45, 59, 119] and several authors discuss the use of RUSI to measure activation and provide feedback for

select training of the deep portion of the LM.[45, 60, 66] Although researchers have demonstrated a structural[72] and functional[81] differentiation between the deep and superficial fibers of the LM, the anatomical differentiation between the fibers is difficult to identify with RUSI and we did not attempt this. The measurement we utilized, directly over the facet joint, is thought to encompass the entire LM and the contralateral UE lifting task is likely to activate the paraspinal muscles en mass. Therefore, our measurement included both the deep and superficial portions of the LM. Refinement of select deep LM measurement and training with RUSI requires further research.

Our findings are consistent with previous studies which have demonstrated reduced thickness change in the TrA as measured by ultrasound imaging in those with chronic LBP. Critchley and Coutts[19] reported a mean thickness change of 15% in chronic LBP subjects (duration of symptoms 54.1 months) compared to a 50% change in pain-free controls during volitional muscle activation.

Ferreira et al[28] also demonstrated a significant difference in thickness change of the TrA between controls and subjects with a history of LBP during an automatic recruitment task of a loaded recumbent biking-type activity. In contrast, Teyhen et a[108] found LBP subjects (duration of symptoms 3.3 months) were able to volitionally activate the TrA as measured by RUSI demonstrating a mean 109% thickness change from rest to activation. Substantial differences between studies may be due to differences in resting measures. Critchley and Coutts reported a mean resting thickness of .51cm while Teyhen et al reported a mean resting thickness of .21cm. Mean thickness values during volitional activation were reported at .67cm and .44 cm respectively. Our data are similar to Critchley and Coutts as we both report approximately a 50% change in TrA thickness in pain-free subjects. Neither study reported pain levels at the time of testing and there was a substantial difference in duration of symptoms.

We are aware of no study that has measured thickness change of the LM in subjects with LBP. Hides et al[45] reported significant differences in cross-sectional area of the LM, at the spinal level of pain, in those with first time acute LBP. These subjects were then randomized to either the control group which received standard medical care of medication and education or the intervention group which added motor control exercise. This exercise protocol utilized RUSI for feedback to the subjects as they learned to

volitionally activation both the TrA and LM. Thickness change of the LM was used as feedback for activation, but no thickness measurements were reported.

A single case-study reported a 64% contralateral difference in LM thickness change, as measured by RUSI, with the painful side changing less than the non-painful side. Following exercise intervention, the activation difference was reported to be resolved and the patient remained symptom-free 12 months following intervention.[63]

Previous research demonstrates experimentally induced pain alters muscle activity, including delays in the timing of TrA activation53 and either an increase or decrease in erector spinae activity dependent upon the phase of the movement task tested. The pain-adaptation model[70] predicts pain will alter muscle activity depending on a given muscle's role as an agonist or antagonist to control movement for protection. This model is described by Graven-Nielsen et al[38] in a review article as the current best explanation of how pain likely alters motor control. It is difficult to categorize the role of LM in the prone UE lifting task used in this study as either agonistic or antagonistic. As an example, the pain-adaptation model predicts increased activity when a muscle would normally be silent and decreased activity when a muscle would normally be active, therefore a decrease in LM activity could be expected. Hodges et al[53] reported an initial increase in deep lumbar multifidus EMG amplitude following saline injection during rapid arm lifting. Differences may be related to the position of subjects. The authors postulate that because subjects were in the standing position, an initial increase in activity of the LM may have been part of a protective trunk splinting response.

Limitations of this study include the small sample size as well as the lack of EMG data. Measuring if EMG also changes during the painful condition would add validity to the study as well as to the use of thickness change as a measure of muscle activation. Additionally, the strength of contraction was not measurable and maximal contraction could not be confirmed in either muscle tested. This may not be relevant from a clinical perspective as high force contractions are not functional in that the stabilizing role of deep muscles is thought to occur at relatively low forces.

Conclusion

The results of this study provide preliminary data indicating RUSI can be used to measure pain-related change in select trunk muscle activation. This adds to the validity

of using RUSI in the clinical setting and may help to expand its use beyond that of feedback and measurement for the TrA. Additionally, the decreased activation as measured by RUSI supports the pain model describe by previous authors Lund and Graves-Nielsen.

Table 3.1. Graded resistance levels for upper extremity lifting tasks in kilograms.

Subject Mass (Kg)	Level 1 UE in add. with elbow flexed	Level 2 UE only at 120° of abd.	Level 3	Level 4	Level 5
< 68.2	_	_	.45	.68	.90
68.2-79.5	_	_	.45	.68	1.14
79.5-90.9	_	_	.45	.90	1.14
>90.9	_	_	.45	.90	1.36

Table 3.2. Mean and SD of muscle thickness (cm) during control and hypertonic conditions. The bold dash indicates no data was collected by study design.

	Control		Hypertonic Saline		
	TrA	LM	TrA	LM	**Δ**
Rest	0.46±0.07	3.27±0.04	—	—	—
Draw-in	0.68±0.08	—	0.59±0.07	—	0.09
UE Lift 1	—	3.70±0.57	—	3.44±0.49	0.26
UE Lift 2	—	4.02±0.60	—	3.72±0.63	0.30
UE Lift 3	—	4.17±0.57	—	3.87±0.54	0.30
UE Lift 4	—	4.25±0.63	—	3.93±0.47	0.32
UE Lift 5	—	4.33±0.66	—	4.04±0.54	0.29

Figure 3.1 Sonogram of the anterior abdominal wall demonstrating measurement of the TrA at rest (left panel) and during volitional abdominal draw-in (right panel).

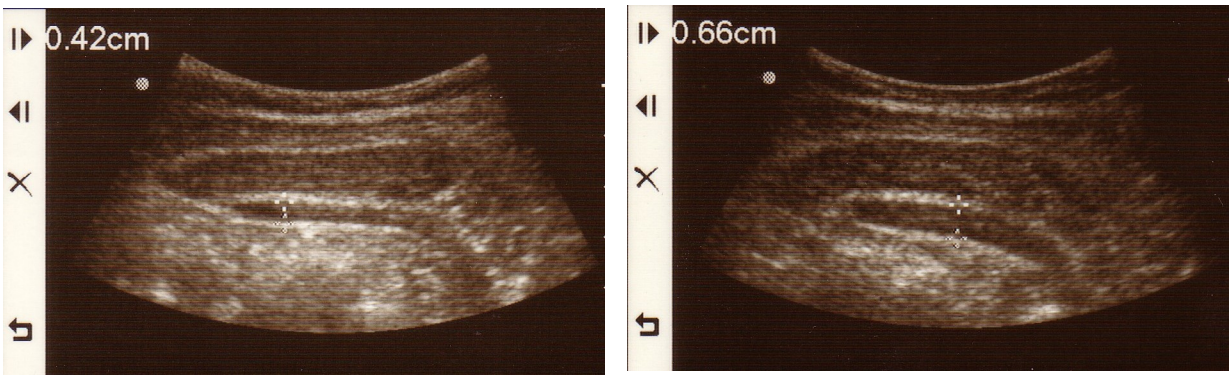


Figure 3.2 Sonogram of a parasagital view of lumbar spine with the L4/5 facet joint in the center. Measurement of the LM at rest (left panel) and during automatic recruitment (right panel) via contralateral arm lifting while in prone position.

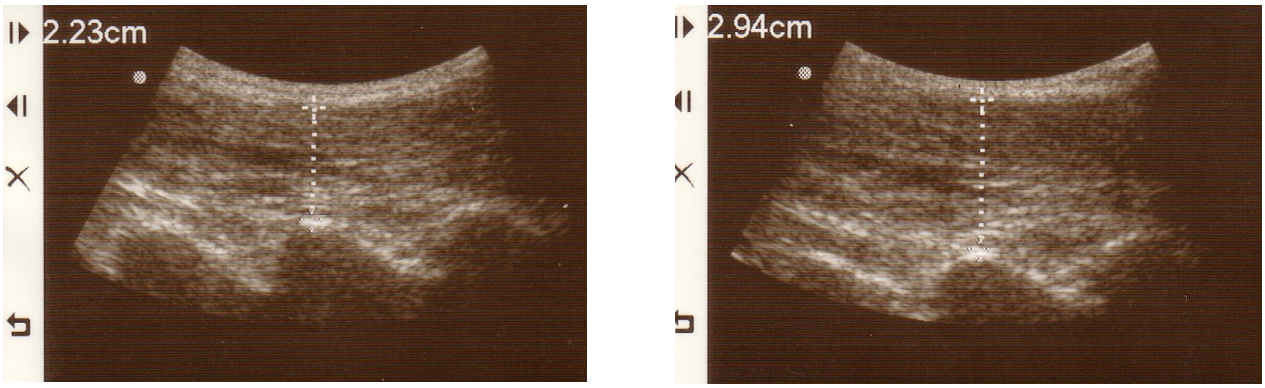


Figure 3.3 Lumbar multifidus thickness change expressed as a % change from rest. The X axis represents each of 5 prone UE lifting tasks with increasing levels of load. * indicates thickness change during the hypertonic saline condition when significantly different from the control condition ($p < 0.01$).

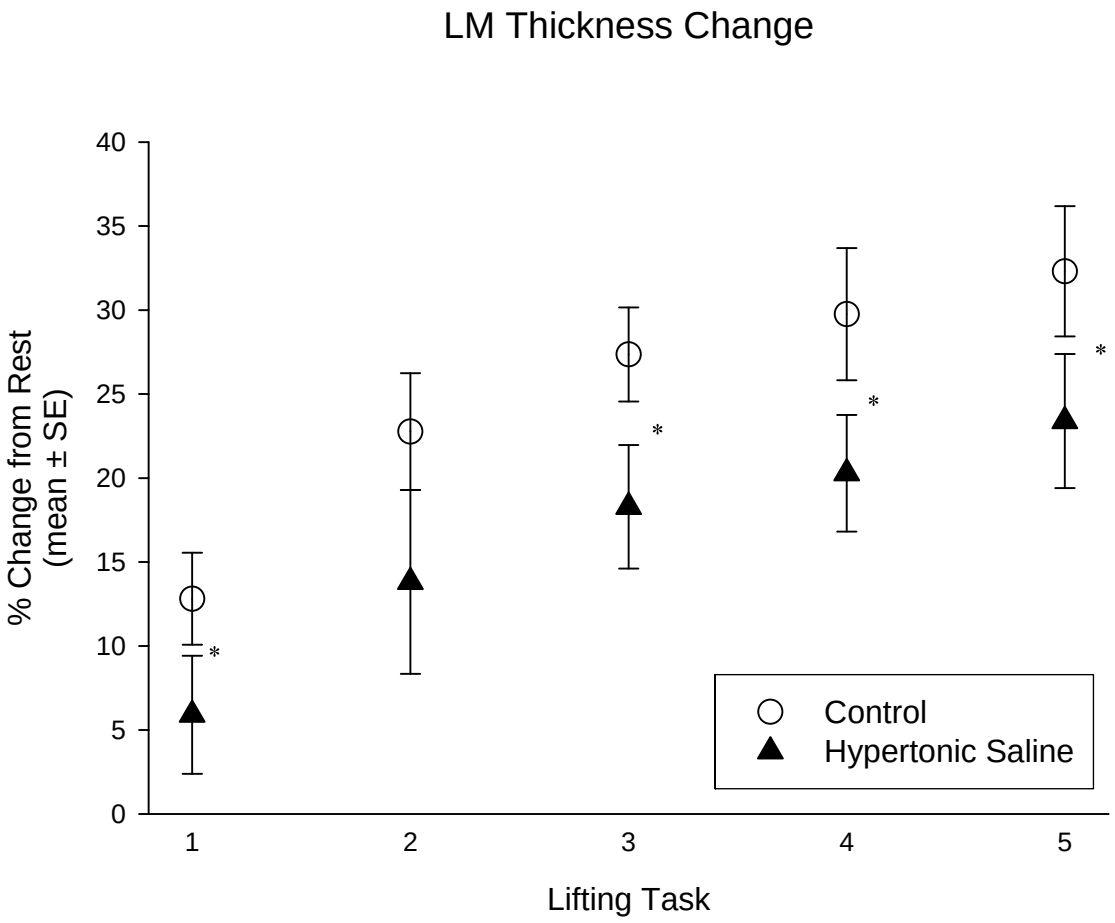
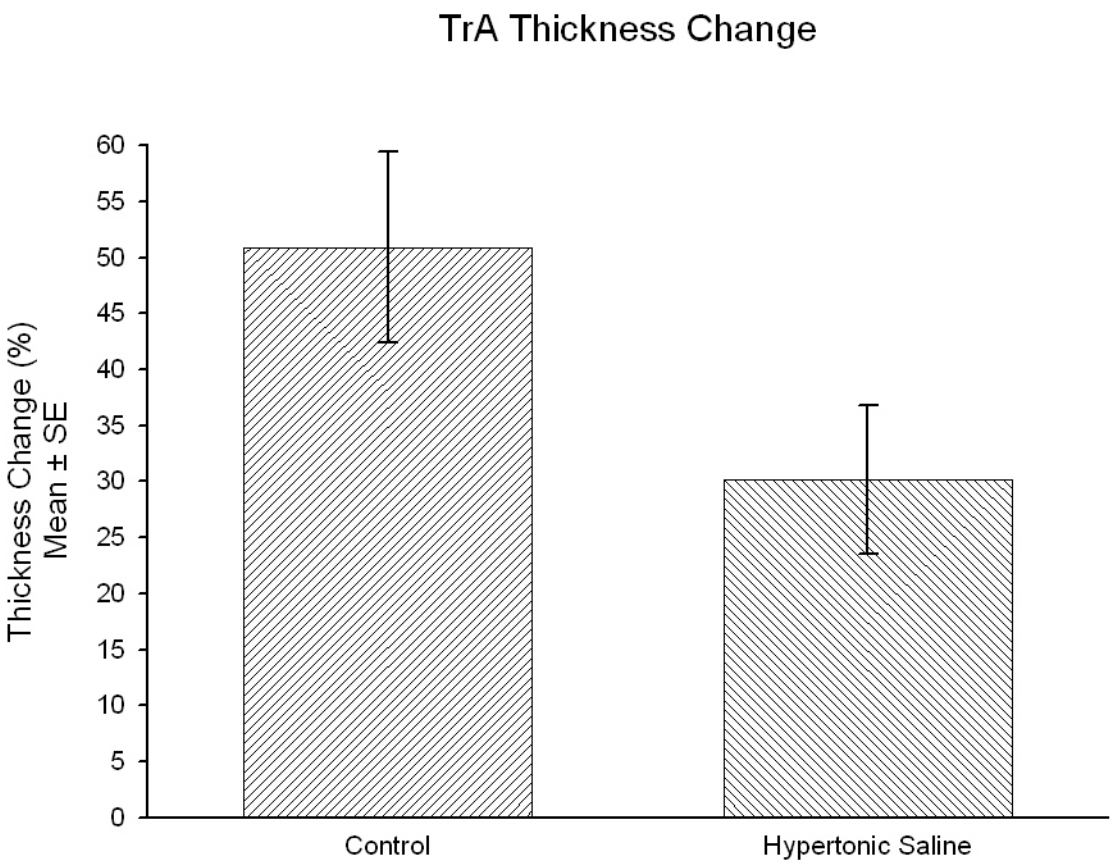


Figure 3.4. Transverse abdominis thickness change expressed as a % change from rest during the volitional abdominal draw-in activity. Thickness change during the hypertonic saline condition was significantly different from the control condition ($p < 0.01$).



Chapter IV

Clinical Study

Chapter Synopsis

The aim of this study was to determine if a difference in thickness change (TC) of the transverse abdominis (TrA) and the lumbar multifidus (LM) as measured by rehabilitative ultrasound imaging (RUSI) exists between subjects with low back pain (LBP) and controls. Researchers have demonstrated that sub-groups of patients with LBP exist and respond differently to treatment, challenging the assertion that LBP is "nonspecific".

The Treatment-Based Classification (TBC) system uses four categories (stabilization, mobilization, direction specific exercise, or traction) to sub-group patients. Only subjects in the stabilization category receive intervention emphasizing stabilization exercises. Recent research has demonstrated impairments of the TrA and LM in those with LBP, therefore, we hypothesize impairments may be present across categories.

RUSI was utilized to measure TC of the TrA and LM in 56 subjects with LBP classified in the TBC system and 20 asymptomatic controls. A standardized intervention was applied to those with deficits.

Control subjects demonstrated a significantly greater TC for the LM (L4, $P = .03$, L5, $P = .04$) during the prone upper extremity lifting task when loaded (hand weight) and the TrA ($P < .01$) during volitional activation. Post-hoc testing revealed the differences were between controls and subjects in the direction specific and stabilization categories for L4, between control and direction specific for L5 and between controls and all subjects for the TrA. There was a significant change in TrA thickness change after the intervention ($P = .02$) and no change in the LM.

These findings suggest a TC deficit exists across categories of the TBC system. Intervention studies should be performed to determine if intervention can correct these deficits and its relationship with outcomes.

Introduction

The importance of identifying sub-groups of patients with low back pain (LBP) to guide clinical intervention and research has been highlighted as a research priority since 1996.[9, 10] Because of the difficulties of grouping patients with LBP into relevant

pathoanatomical categories, classification schemes derived from clinical examination findings and historical factors have evolved. The Treatment-Based Classification (TBC) system, initially proposed by Delitto et al[25] in 1995, suggests that identifiable sub-categories of LBP patients exist. Research published since 2002 has served to validate this premise by demonstrating that sub-groups of patients with low back pain exist and respond differently to treatment.[11, 15, 30, 36, 41, 68] This line of inquiry has helped challenge the assertion that the majority of low back pain is "nonspecific" and that the watchful waiting treatment approach is superior to classification driven intervention.

The TBC system utilizes relevant historic factors, current disability and pain levels, and key clinical exam findings to classify patients into one of the four categories; direction specific exercise (flexion or extension), mobilization (lumbar or SI mobilization/manipulation), stabilization (core stabilization program) or traction. Reliability of clinicians classifying subjects into each of the categories of TBC system[33, 34] as well as which factors are the most useful to discriminate between categories has been established.[35] Treatment studies have demonstrated that clinical outcomes are superior when subjects receive an intervention which is matched to their category as compared to those subjects receiving unmatched intervention.[11, 15, 41] Additionally, a randomized trial has provided preliminary evidence demonstrating that interventions based on the TBC produce superior outcomes when compared to LBP interventions based on current medical treatment guidelines.[32]

Motor Control

In addition to the importance of providing proper classification, is the ability to identify and correct impaired motor control. A growing body of neurophysiologic and clinical evidence suggests that the deep stabilizing musculature of the spine is impaired in those with LBP.[29, 71, 94] The motor control model of spinal stabilization focuses on the function of deep spinal muscles because these structures are thought to have the ability to control motion between vertebral segments. Research demonstrating impairments in these deep muscles including atrophy,[6, 43, 44, 59, 64, 119] delayed activation,[28, 50, 52] and lack of volitional control,[39] has led to the development of the motor control model of stabilization training. The motor control approach emphasizes that subjects learn isolated volitional activation of deep trunk muscles,[93] primarily the transverse abdominis (TrA)

and lumbar multifidus (LM). For the interested reader, a recent systematic review summarizes the clinical evidence to date supporting the motor control model of intervention.[29]

Clinical assessment of deep muscle performance to help guide clinical intervention is difficult. Electromyography (EMG), utilizing fine wire electrodes, has traditionally been used to assess the magnitude and timing of the TrA and LM providing useful information related to motor control. Unfortunately, the invasiveness of these procedures limits their routine clinical use[108] and researchers and clinicians have relied on manual palpation techniques with limited evidence regarding their validity.

There is emerging research evidence supporting the use of ultrasound imaging as a non-invasive tool to assess deep muscle activation.[57] The application of ultrasound imaging for the purposes of biofeedback and muscle performance measurement by rehabilitation professionals has been named Rehabilitative Ultrasound Imaging (RUSI).[107] The most common parameter measurable with RUSI that relates to muscle activation is muscle thickness change. Several researchers have utilized thickness change as an indicator of muscle activation for the TrA [12, 13, 28, 39, 96, 108] and LM.[46, 112]

The validity of utilizing muscle thickness change as a measurement of muscle activation has been demonstrated in the TrA[55, 76] and LM[61] in an asymptomatic population by comparing thickness change to fine-wire EMG. Ferreria et al[28] demonstrated concurrent thickness and EMG attenuation of the TrA in subjects with LBP as compared to controls and we demonstrated experimentally induced pain reduces the thickness change of the LM during an automatic recruitment task (chapter 3).[62] These studies add validity to both the use of RUSI as a measurement tool for muscle activation as well as the motor control model of spinal stabilization.

Commonly used techniques to measure muscle thickness change during volitional activation tasks such as the abdominal draw-in, or automatic recruitment tasks such as the prone arm lifting model used for the LM, do not capture the timing of muscle activation which is considered a key motor control variable of interest.[56] The relationship between the timing of activation and muscle thickness change is unknown. There are ultrasound measurement techniques under development that have been shown to accurately measure the timing of muscle activation. Using high frequency M-mode (motion mode)

ultrasound, Vesseljen et al [113] demonstrated the onset of thickness change in the LM was correlated to the onsets measured by EMG. The measurement of thickness change between rest and activation used in the current study does not assess the timing of muscle activation, but is a measurement that can be routinely performed clinically and may have the potential for use as an impairment measure in future clinical research.

Motor control deficits may, in part, be caused by pain, irrespective of the source,[56] supporting the concept that motor control deficits may be present across all LBP classifications. If differences exist in the performance of deep stabilizing muscles between subjects with LBP in any TBC category and asymptomatic control subjects, this would suggest that motor control training may be appropriate across the different categories of the TBC system. The purposes of this study were to 1) report the reliability of RUSI measurements in subjects with LBP 2) determine if there is a difference in muscle performance of deep lumbar stabilizing muscles (thickness change as measured by RUSI) between subjects with LBP classified in the TBC system and asymptomatic controls 3) determine if muscle thickness change improved following a standardized intervention 4) determine if disability and pain change following the intervention period

Methods

Subjects

Subjects age18-60 years of age with a modified Oswestry (ODQ) score of = 25% referred to one of 5 physical therapy clinics for treatment of LBP were recruited for this study. Power calculations indicated that with a sample size of 14 in each group, a 1-way ANOVA would have 80% power to detect a difference in muscle thickness between subjects and controls at the 0.05 level. Exclusion criteria included being classified into the traction category of the TBC, prior lumbar surgical intervention, overt neurological compromise including lower limb reflex loss or gross myotomal strength loss, known fracture, infection, tumor, pregnancy or recent ingestion of a contrast medium (which is a contraindication to RUSI application). A total of 56 subjects were included in the analysis. The mean (± SD) age was 43.1 (10.9) years, height 149.9 (9.4) cm, mass 83.8 (20.8) kg, and 63% of the subjects were female. The control group was similar for demographic data and activity level. See Table 4.1 for descriptive statistics.

Procedures

Eight physical therapists, all familiar with the TBC system, participated in this study. All therapists completed a training session to review the TBC criteria and study protocol. Once enrolled, the treating therapist classified the subject into the appropriate category based on the algorithm described by Fritz et al[33]. The subjects received initial treatment based on their category and were scheduled for their RUSI exam. The exam was scheduled as soon as possible after the subject was enrolled and the majority took place less than one week after initial assessment. To determine the reliability of classifying subjects into the categories of the TBC between the participating clinicians and the Principal Investigator (PI), all subjects who did not change more than the minimal detectable change on the ODQ (6 points)[31] were also classified by the PI after completion of the RUSI exam. A total of 30 subjects met this criteria and were included in the TBC reliability analysis.

All subjects received intervention based on their respective category. For the direction specific exercise and mobility categories, a pragmatic approach was taken and clinicians were free to utilize manual therapy and exercise techniques at their discretion. If subjects were determined to have a thickness change deficit as measured by RUSI, they received a standardized stabilization exercise progression as outlined by Hicks et al[41] (see Table 4.2) in addition to their respective category specific program. Subjects classified in the stabilization category received only the standardized stabilization program. The operational definition of muscle thickness change deficit for the TrA includes either side demonstrating = 75% change in thickness from rest to activation. The LM deficit was either side or level demonstrating = 15% thickness change from rest to contraction on the low load test and/or either side or level tested demonstrating = 20% change from rest to contraction on the high load test. These values were derived from data collected in the first two thickness change studies for the LM and from the current literature suggesting approximately a 100% change in TrA is normal.[108]

Ultrasound Exam

RUSI measurements were obtained using the Sonosite 180 Plus (Sonosite, Inc, Bothell, WA) computerized sonography unit with a 2 to 5-MHz curvilinear probe. The

TrA measurement was performed with the subject in the supine hooklying position with the transducer placed along the lateral abdominal wall just superior to the iliac crest along the mid-axillary line[98] and adjusted so the medial portion of the muscle was on the left side of the screen as described by Henry et al.[39] Once an adequate image was obtained (Figure 4.1), 3 rest measures were recorded at the end of inspiration. Next, the TrA activation measurements were recorded while the subject performed the abdominal draw-in maneuver. Subjects were instructed to "*exhale and gently draw your lower stomach in toward your spine*". This was taught to the subjects by the PI and common errors were corrected. Once the PI was confident that the subject understood the correct procedure, 5 practice repetitions were performed before the start of data collection. Data were collected on the left and right sides on all subjects with the tester blinded to TBC category and painful side. The mean of the 3 measures, which has been shown to reduce the standard error of the measure by approximately 50 percent,[103] was used for all RUSI measurements. The test was repeated at the end of intervention period.

To activate the LM, a prone upper extremity lifting model, modified from a our previous study was utilized (see chapter 2). The measurement is performed with the subject in the prone position with pillow(s) placed under the abdomen to flatten the lumbar spine such that the lumbosacral junction is = 10°. The transducer was placed longitudinally along the mid-line of the spine first over the L4 level then moved laterally and tilted slightly until the L4/5 facet joint could be visualized. A measurement from the hyperechogenic facet joint to the plane between the subcutaneous tissue and the multifidus muscle is considered LM thickness (Figure 4.2). This "parasagital view" of the LM has been described by both Richardson et al[98] and Stokes et al[106] and thickness change from rest to activation (during contralateral upper extremity lifting) has been shown to be correlated highly ($r=.79$ $P< 0.001$) with EMG activity in asymptomatic subjects (chapter 2). Measurements were taken at the L4/5 and L5/S1 levels bilaterally with no load (upper extremity abducted to 120° with the elbow flexed to 90°) and with a load (same position using .68, .90, or 1.36 kilograms of resistance based on body mass). Previous work has demonstrated that EMG activity during these activation tasks (arm lift and load) are significantly different from each other in asymptomatic subjects.[61]

All measurements were obtained via the on-screen calipers and recorded on a data sheet, then entered into a spread sheet. TrA and LM thickness change was calculated as activation – rest / rest x 100. Same day intratester reliability of the RUSI measurements were calculated using data obtained from 15 subjects with LBP. The RUSI exam was performed as described above. The subjects were then repositioned and the exam was repeated. This study was approved by the institutional review boards at the University of Kentucky and the University of Evansville. All subjects provided informed consent and their rights were protected at all times.

Statistical Analysis

To determine the reliability of the RUSI measures the interclass correlation coefficient was calculated using model 3 and the average of the 3 measures. For the reliability of classifying subjects into the TBC system, a kappa statistic and corresponding percent agreement were calculated. To determine if a difference existed in thickness change of the LM and TrA between subjects with LBP classified in each of the 3 TBC categories and controls, separate one-way analyses of variance (ANOVA) were conducted using group assignment as the between subjects factor. This analysis allows for interpretation related to how the arm lift or arm lift with load performs irrespective of each other and was chosen to maximize the clinical meaning of the measures. There were no differences (*P* values ranging from .47 to .91) in muscle thickness change between the painful and non-painful sides for any of the 5 measurements obtained, therefore an average of the two measurements was utilized in the analysis. The Games-Howell post-hoc test was used because the assumption of equal variance was not met for 3 of the 5 variables and the sample sizes were different. The level of significance was set at .05.

To determine if a difference existed between muscle thickness change after the intervention period, separate paired t-tests were run. Because of multiple comparisons of the LM a Bonferroni correction was applied, lowering the significance level to 0.01. To analyze change in pain and disability, repeated-measures ANOVAs were run using initial, 6-week and 6-month time points. The results are expressed using an intention-to-

treat analysis as well as analyzing just those who completed the study. SPSS version 14.0 (SPSS, Inc, Chicago, IL) statistical software was used for all analyses.

Results

The intratester reliability and SEM for each of the measurements assessed were calculated and are reported in Tables 4.3 and 4.4. The results for percent change in muscle thickness from rest to activation for the TrA was $ICC_{3,3}$ = 0.96, SEM 6.26%. For the LM at L4 the reliability was $ICC_{3,3}$ = 0.98, SEM=2.96% and for the LM at the L5 the results were $ICC_{3,3}$ = 0.93, SEM=2.49%. The reliability of the classification of subject into the categories of the TBC between the clinicians and the PI was Kappa = 0.65 (agreement = 77%) with a 95% confidence interval of 0.42 to 0.87.

There was a difference in muscle thickness change between subjects with LBP and controls for the loaded LM test at L4 (F=3.24, *P* = .03) and L5 (F = 3.01, *P* = .04) and for the TrA test (F=14.53, *P* <.01). Post-hoc testing revealed the differences were between controls and LBP subjects in the direction specific (*P* = .04) and stabilization (*P* = .01) categories for L4, between control and direction specific (*P* = .05) for L5 and between control and all subjects with LBP for the TrA (*P* < .01) see Figures 3-5. No differences were identified between TBC categories (see Table 4.5).

All but 3 subjects (one from each TBC category) met the definition of having a muscle thickness change deficit. There was a 43% dropout rate as 32 subjects completed the intervention and underwent the post-intervention RUSI exam. Of these 32 subjects 19 returned the 6-month questionnaires.

There was no difference between muscle thickness change after the intervention period for any of the LM measures (*P* values ranging from .13-.87) see Figures 4.6 and 4.7. The subjects did demonstrate a greater thickness change of the TrA after intervention (*P* =.02, see Figure 4.8). The intention-to-treat analysis of disability and pain revealed there was a significant decrease in both variables following intervention (P < .01) see figures 4.9 and 4.10. There were no differences between the subjects in each TBC categories.

Discussion

The same-day reliability of the RUSI was good to excellent and considered clinically meaningful according to standards suggested by Portney and Watkins.[117] When considering the clinical accuracy of a measurement tool, it is important to consider the SEM (standard error of the measure) as well. For the TrA our results for rest and activation were 0.01 and 0.02 cm which are consistent to errors reported by Teyhen et al[103] for similar RUSI measurement when the mean of 3 measures is utilized. For the overall measure of percent thickness change of the TrA the SEM = 6.26%. The mean thickness change for subjects with LBP was 61.1% (27.9) indicating RUSI has the ability to assess TrA thickness change beyond measurement error. Findings were similar for LM where the mean thickness change for subjects with LBP during the loaded tests was 16.8% (9.7) at the L4-5 level and 11.8% (6.3) for the L5-S1 level. The SEM was 2.49% and 2.02% respectively, indicating that RUSI can detect thickness change of the LM beyond measurement error. Our findings for reliability were consistent with Van et al[112] who reported an ICC of 0.98 and a SEM 0.31cm utilizing the same measurement technique in asymptomatic subjects at the L4-5 level. Our SEM values were lower (0.07 cm) probably because we used the mean of 3 measures. These results are for the same rater on the same day only. Further research is required to establish reliability between raters and on subjects between days.

The reliability of classifying subjects into the different categories of the TBC system was kappa = 0.65 (77% agreement). According to Portney and Watkins[117] this value is on the lower range of what is considered to represent substantial agreement (.61-.80). Our findings are consistent with other reliability studies of the TBC system. Fritz and George reported a kappa value of 0.56 in an interrater reliability study of 120 subjects with LBP[34] and Fritz et al[33] who reported an overall kappa of 0.60 when utilizing a newly developed algorithm for classification which was used in the current study. The greatest source of error in classifying subjects was a discrepancy when categorizing subjects into either the mobilization or stabilization category. In four cases a clinician placed subjects into the stabilization category when the PI had placed them into the mobilization category. The presence of aberrant movement patterns is an individual exam item which has been previously shown to have only fair reliability and may be

variable day to day even in an otherwise stable subject.[33] This important variable to distinguish between the stabilization or mobilization category may have contributed to our lack of agreement.

Intervention

The thickness change of the LM did not significantly improve following intervention and none of the 4 measures approached statistical significance. The SEM is 2.5% and the minimal detectable change (MDC) is = 3.5%. It would take approximately a 6.9% (3.5 x 1.96) change to be 95% confident the error in the measurement was exceeded. There were 19 of subjects who did exceed the 7% change threshold on at least one of the LM measures, but this was not associated with any outcome variable.

The results of this study support the hypothesis that muscle thickness change, as measured by RUSI, is different between subjects with LBP and asymptomatic controls, but not different between categories. However, there was substantial variation in muscle thickness change of the LM between subjects, levels, and between sides. We had subjects self-report their more painful side and tested the hypothesis that the more painful side would demonstrate a greater thickness change impairment. Previous studies have identified a consistent pattern of LM atrophy on the symptomatic side in acute[46] and chronic[43] LBP subjects. These data do not support this hypothesis as we did not identify a meaningful pattern when we considered category, duration of symptoms, pain or disability level, and magnitude of side to side asymmetry. A key finding of this study was that thickness change of the LM between levels and sides is highly variable in subjects with LBP. Additional research could explore the relationship between LM cross-sectional area and thickness change to better elucidate this finding.

Recent clinical trials have demonstrated no improvement in clinical outcomes when motor control training is compared to conventional exercise [14] for subjects with recurrent LBP or when compared to conventional exercise and manual therapy in patients with chronic nonspecific LBP.[27] Because motor control deficits are highly variable, it is not surprising that non-significant findings are reported from studies which randomize subjects who likely have somewhat heterogeneous clinical presentation (“recurrent” or “chronic non-specific”) into different treatment groups. Some subjects in the general exercise or manual therapy groups may need a program emphasizing motor control

training while some subjects in the motor control group may need an emphasis on general exercise or manual therapy.

There was a significant change in the subject's ability to volitionally activate the TrA but no difference in LM thickness change and no association with pain or disability. This suggests that pain is not the main factor responsible for the LM thickness change deficit as pain improved but the thickness change did not. This is consistent with other LBP research which fails to correlate impairments measures with disability. Also the intervention program utilized did include volitional isolation of the TrA throughout the program but did not have motor control activities targeted directly at the LM. Future research should be conducted to determine what interventions best reverse motor control deficits and if individual deficits have a relationship with outcomes not tested in this study, such as recurrence.

Limitations to this study include the high dropout rate which limits the finding of the intervention portion. The dropouts were evenly distributed across classification (30% direction specific, 40% mobility, and 30% stability). Maturation may also have been a factor as the time between initial classification and the initial RUSI exam varied between subjects. This may have affected the muscle activation tests as most subjects received treatment on the initial visit and had been performing their initial home exercises for at least a short period prior to the RUSI exam.

Gender difference may have been a confounding factor due to what some have reported as difference in LM activation levels (see chapter 3). We did not identify a difference in any of the primary variables between genders ($p = 0.42$-0.89). Additionally, we did not control for days of onset of current LBP episode, therefore 44% of our subjects would be considered to have acute LBP (4 weeks), 21% subacute (4-12 weeks), and 35% chronic LBP (> 12 weeks) by the classic duration of symptoms definition. There was a statistical difference again for the loaded LM test at the L4 level ($P = .03$), L5 level ($P = .03$) and TrA ($P < .01$) when we compared muscle thickness change by chronicity as defined. Post-hoc testing revealed differences were between the control group and the chronic group at the L4 level and between the control and acute group for L5. For the TrA, all groups were different than controls and the chronic group demonstrated significantly less thickness change than the subacute group.

Conclusion

The findings of this study suggest muscle thickness change can be measured clinically utilizing RUSI and that deficits exist in subjects with LBP. The patterns of thickness changed varied widely between subjects and to a lesser extent than controls. Future research should be performed to determine if directed intervention can normalize muscle thickness change deficits in LBP subjects and if this has a meaningful relationship with clinical outcomes.

Table 4.1 Descriptive statistics. Values represent mean (SD).

	Control (n = 20)	Direction Specific Exercise (n = 16)	Mobilization (n = 22)	Stabilization (n = 18)
Age (y)	41.2 (8.6)	41.6 (11.7)	44.1 (9.8)	42.9 (12.0)
Height (cm)	170.6 (11.2)	169.7 (8.0)	170.0 (9.6)	166.4 (9.1)
Mass (kg)	79.1 (15.0)	86.8 (22.3)	80.8 (18.6)	77.7 (25.2)
Baecke activity score	38.1 (3.8)	40.0 (6.8)	42.5 (6.54)	36.2 (8.9)
Oswestry score		42.6 (11.6)	37.7 (15.5)	34.8 (11.2)
Pain rating		6.1 (1.8)	5.1 (1.7)	5.3 (1.9)
Duration of symptoms (m)		2.7 (3.9)	2.9 (4.1)	3.2 (3.6)
Fear-avoidance beliefs questionnaire (work)		14.7 (12.2)	16.4 (11.7)	15.2 (9.8)
Fear-avoidance beliefs questionnaire (activity)		40.4 (22.2)	45.2 (19.3)	36.9 (15.4)

Table 4.2. Stabilization Exercises With Criteria for Progression of Each Exercise

Primary Muscle Group*	Exercises	Criteria for Progression
Transversus abdominus	Abdominal bracing	30 repetitions with 8-s hold
	Bracing with heel slides	20 repetitions per leg with 4-s hold
	Bracing with leg lifts	20 repetitions per leg with 4-s hold
	Bracing in standing	30 repetitions with 8-s hold, then progress to 1 leg
	Bracing with standing row exercise	30 repetitions with 8-s hold
	Bracing with walking	20 repetitions per side with 6-s hold
Erector spinae/multifidus	Quadruped arm lifts with bracing	30 repetitions with 8-s hold one each side
	Quadruped leg lifts with bracing	30 repetitions with 8-s hold one each side
	Quadruped alternate arm and leg lifts with bracing	30 repetitions with 8-s hold one each side
Quadratus lumborum	Side support with knees flexed	30 repetitions with 8-s hold one each side
	Side support with knees extended	30 repetitions with 8-s hold one each side
Oblique abdominals	Side support with knees flexed	30 repetitions with 8-s hold one each side
	Side support with knees extended	30 repetitions with 8-s hold one each side

* Although certain muscle groups are preferentially activated with each exercise sequence, each exercise progression will promote stability by producing motor patterns of cocontraction among all spinal stabilizing muscles.

Table 4.3 Same-day intratester reliability (n = 15) for measuring the lumbar multifidus with RUSI.

Condition	L4-L5		L5-S1	
	ICC CI_{95}	SEM	ICC CI_{95}	SEM
Rest	0.99 (0.97-0.99)	0.07	0.99 (0.97-0.99)	0.07
Arm Lift	0.98 (0.92-0.99)	0.09	0.99 (0.96-0.99)	0.07
Load Lift	0.86 (0.60-0.95)	0.09	0.97 (0.92-0.99)	0.07
% Δ Lift	0.98 (0.96-0.99)	2.96	0.99 (0.96-0.99)	1.20
% Δ Load	0.93 (0.80-0.97)	2.49	0.93 (0.91-0.97)	2.02

Abbreviations: ICC, interclass correlation coefficient; CI_{95}, 95% confidence interval; SEM, standard error of the measure; % Δ Lift, percent thickness change from rest to activation during arm lifting; % Δ Load, percent thickness change from rest to activation during loaded arm lifting.

Table 4.4. Same day intratester reliability (n = 15) for measuring the transverse abdominis with RUSI.

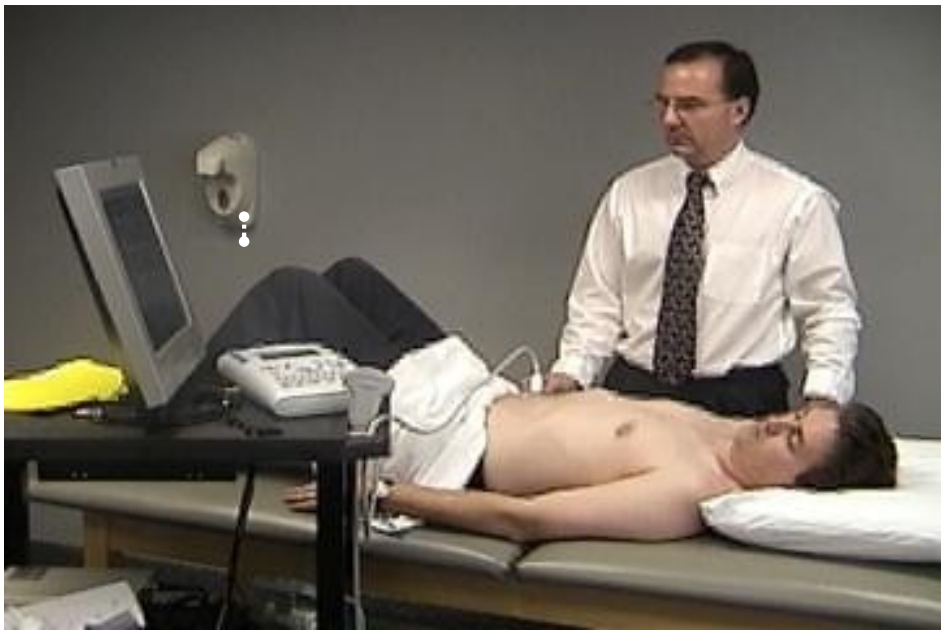
Condition	ICC CI_{95}	SEM
Rest	0.98 (0.91-0.99)	0.01
Abdominal Draw-in	0.97 (0.91-0.98)	0.02
% Δ	0.96 (0.91-0.99)	6.26

Abbreviations: ICC, interclass correlation coefficient; CI_{95}, 95% confidence interval; SEM, standard error of the measure; % Δ, percent thickness change from rest to activation during the abdominal draw-in maneuver.

Table 4.5. Mean (SD) and 95% confidence interval data for thickness change between groups. * Indicates value is significantly different from control ($P < .05$).

Muscle	Segment/Mode	Group	Mean	95% CI
TrA		Control	99.4 (15.4)	91.2 – 107.6
		Direction Specific*	66.8 (31.8)	45.4 – 88.2
		Mobilization*	65.8 (26.7)	52.1 – 79.5
		Stabilization*	48.9 (19.6)	37.0 – 60.7
LM	L4 Lift	Control	19.0 (5.9)	15.9 – 22.2
		Direction Specific	14.5 (7.9)	9.1 – 19.8
		Mobilization	14.5 (10.5)	9.1 – 19.9
		Stabilization	14.7 (6.5)	10.7 – 18.6
	L4 Load	Control	25.0 (7.5)	21.0 – 29.0
		Direction Specific*	17.8 (9.6)	11.4 – 24.2
		Mobilization	17.9 (12.1)	11.7 – 24.2
		Stabilization*	16.6 (8.2)	11.6 – 21.5
LM	L5 Lift	Control	12.5 (4.3)	10.3 – 14.8
		Direction Specific	8.4 (5.2)	4.9 – 11.9
		Mobilization	9.1 (7.0)	5.5 – 12.7
		Stabilization	13.7 (8.2)	8.7 – 18.6
	L5 Load	Control	17.4 (6.3)	14.1 – 20.7
		Direction Specific*	11.1 (5.5)	7.4 – 14.8
		Mobilization	11.7 (7.6)	7.7 – 15.6
		Stabilization	12.9 (6.0)	9.2 – 16.5

FIGURE 4.1 Researcher collecting TrA ultrasound data during abdominal draw-in. Below are sonograms of the anterior abdominal wall demonstrating an 87% thickness change of the TrA between rest (left panel) and volitional abdominal draw-in (right panel).



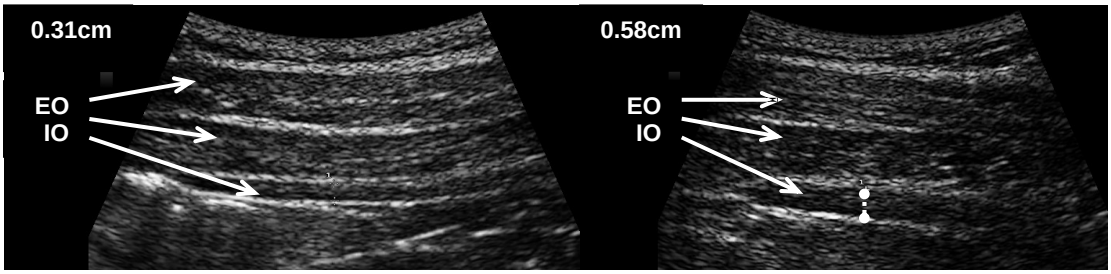


Figure 4.2 Researcher collecting LM ultrasound data during arm lift with no load. Lower panels are subsequent sonogram of a parasagital view of lumbar spine with the L5-S1 facet joint in the center. Measurement demonstrating a 28% thickness change of the LM between rest (left panel) and automatic recruitment (right panel) via contralateral arm lifting with load.

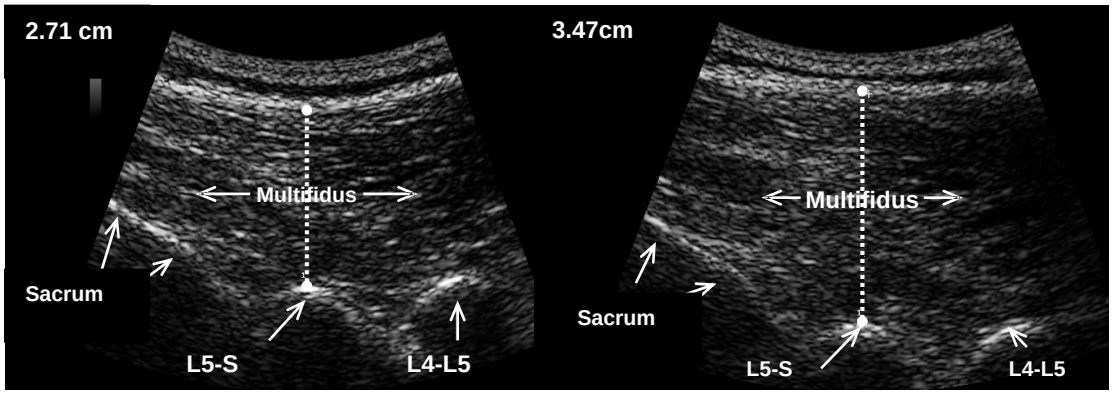
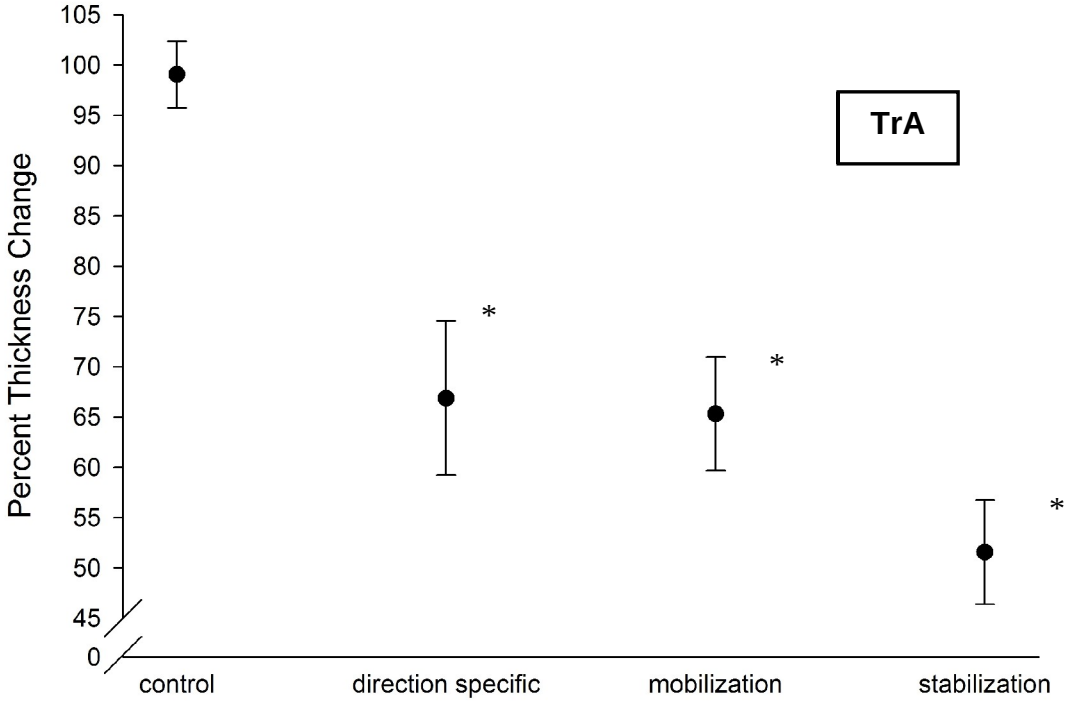
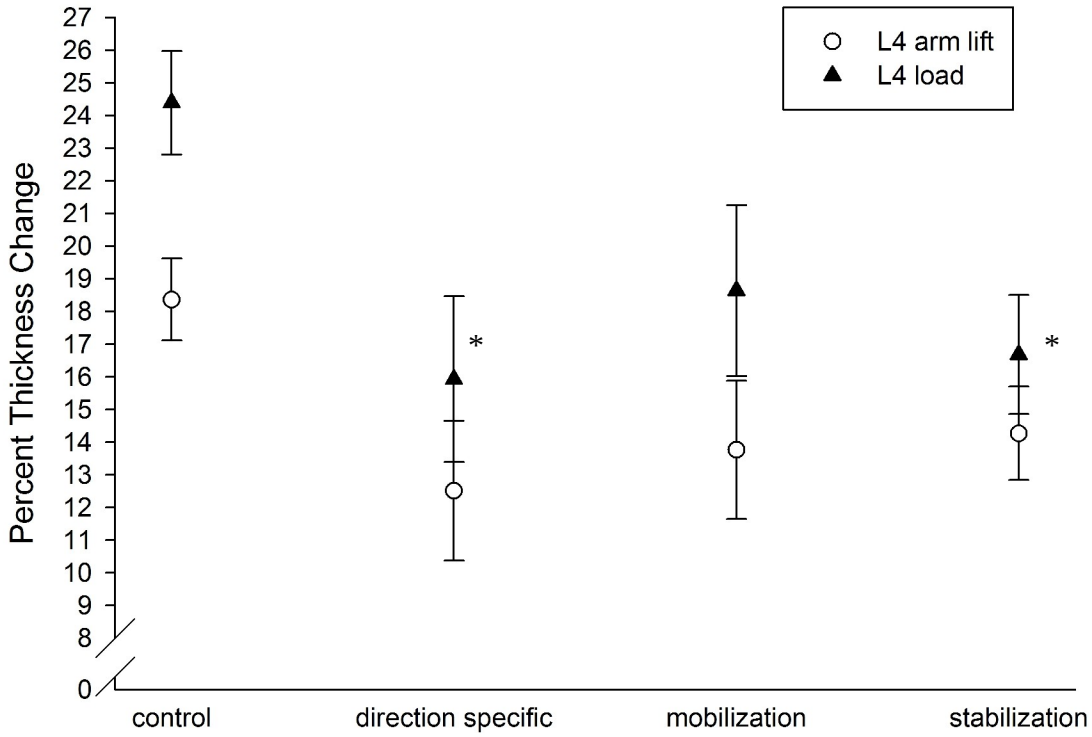


Figure 4.3 Graph of percent thickness change of the TrA from rest to activation during the abdominal draw-in maneuver between the control group and subjects classified into the different categories of the Treatment-Based Classification System.



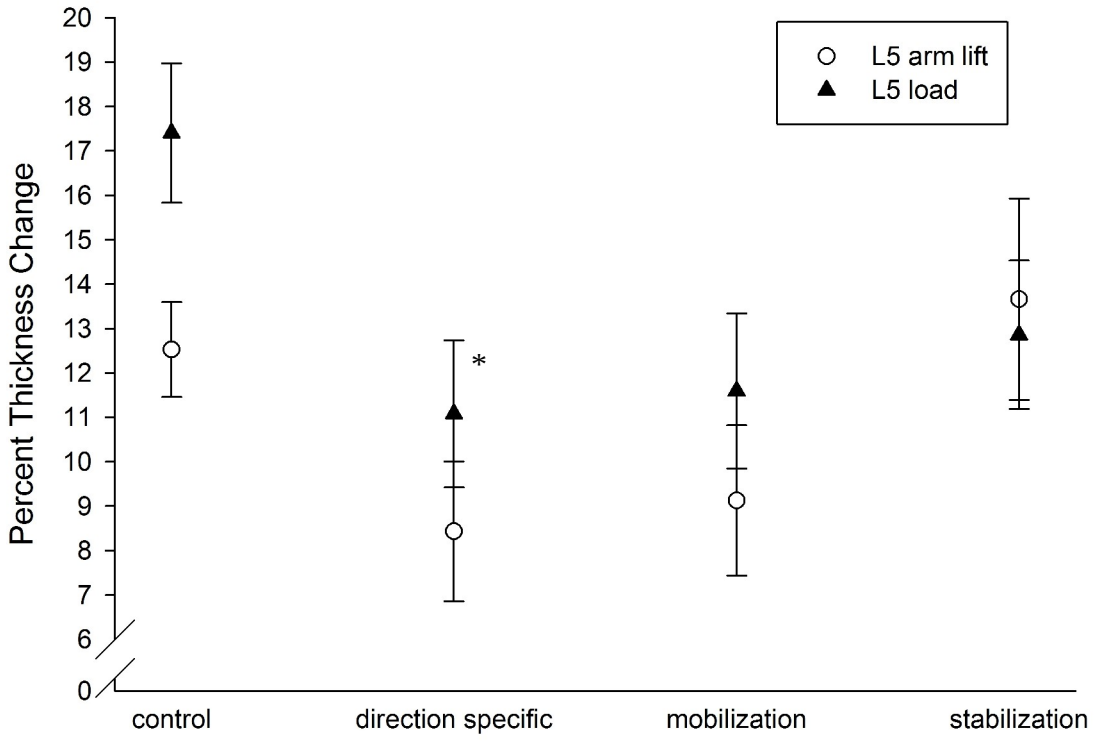
* Indicates value is significantly different from control ($P < .05$).

Figure 4.4 Graph of percent thickness change of the LM at the L4 level from rest to activation during the prone arm lift and lift with load between the control group and subjects classified into the different categories of the Treatment-Based Classification System.



* Indicates value is significantly different from control ($P < .05$).

Figure 4.5 Graph of percent thickness change of the LM at the L5 level from rest to activation during the prone arm lift and lift with load between the control group and subjects classified into the different categories of the Treatment-Based Classification System.



* Indicates value is significantly different from control ($P < .05$).

Figure 4.6 Graph of muscle thickness change of all subjects completing the intervention of the L4 LM before and after intervention.

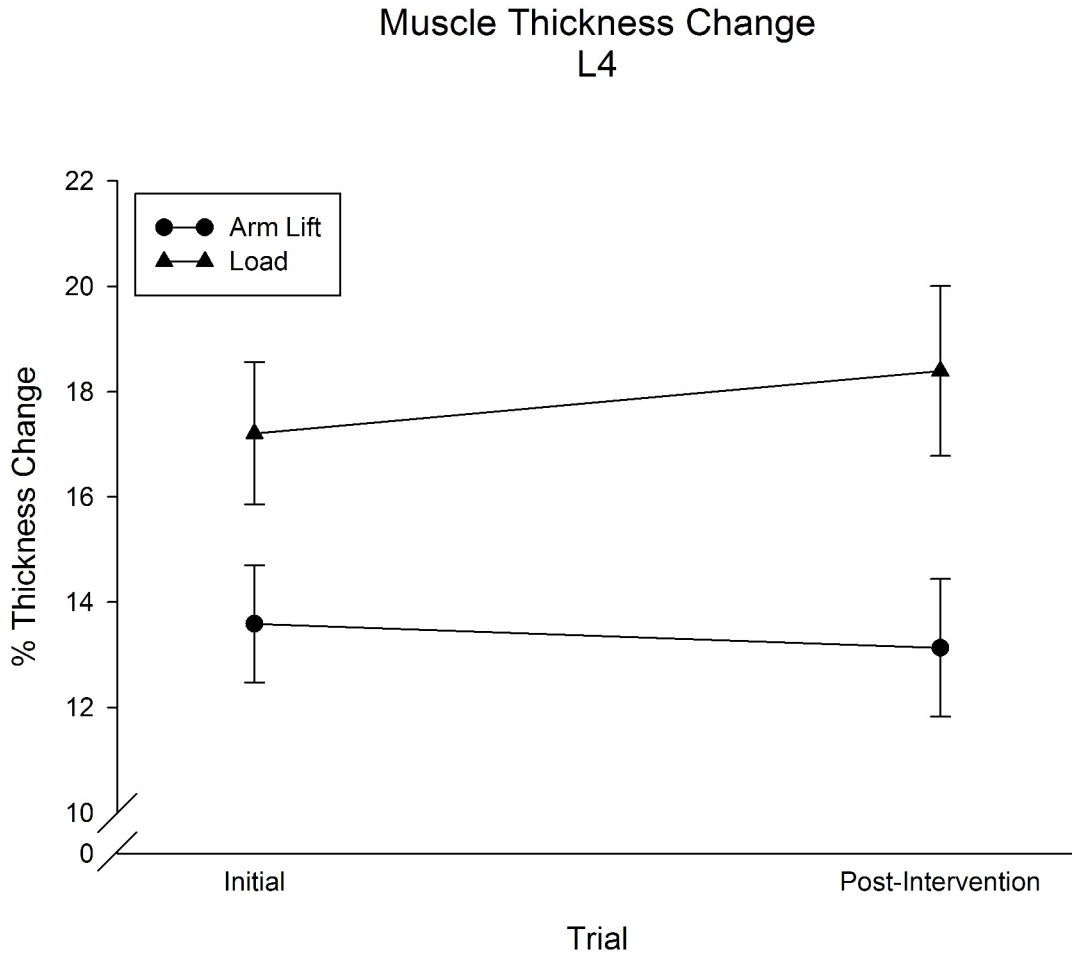


Figure 4.7. Graph of muscle thickness change of all subjects completing the intervention of the L5 LM before and after intervention.

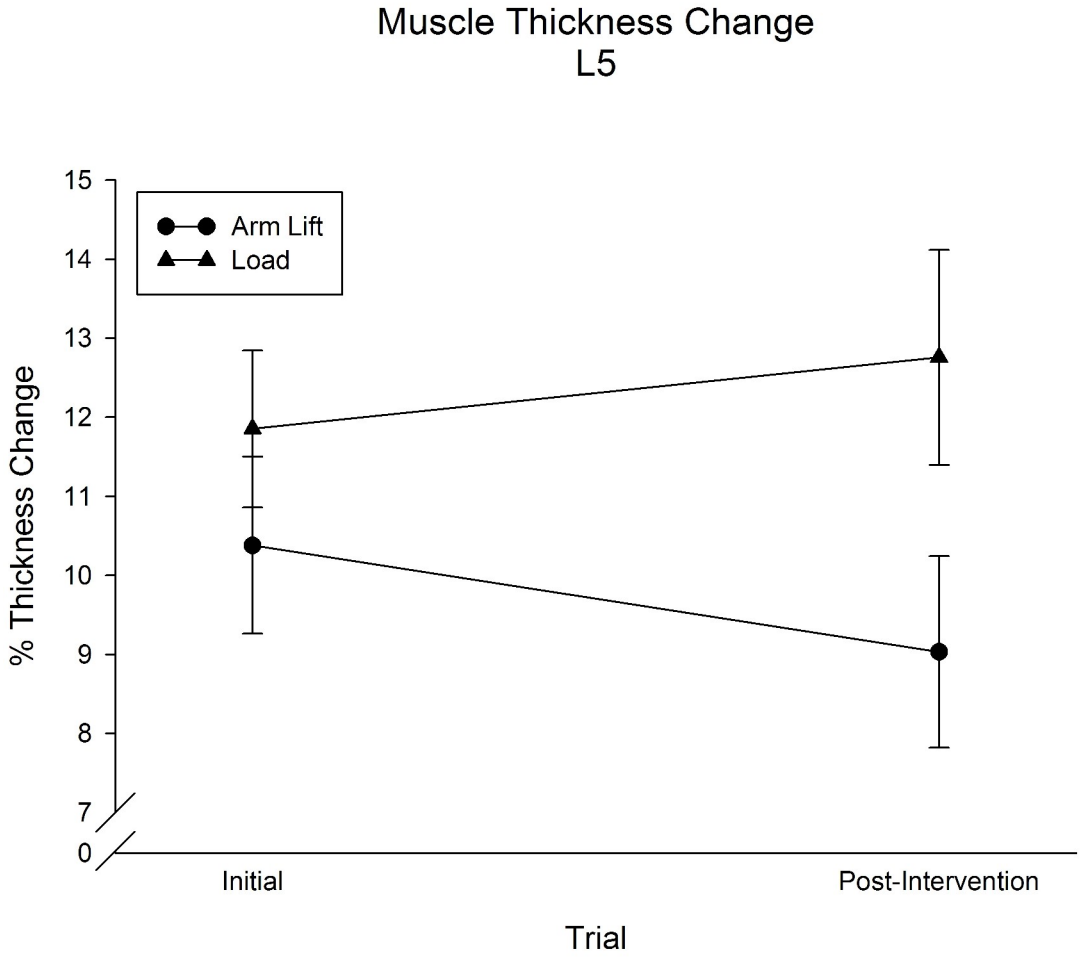
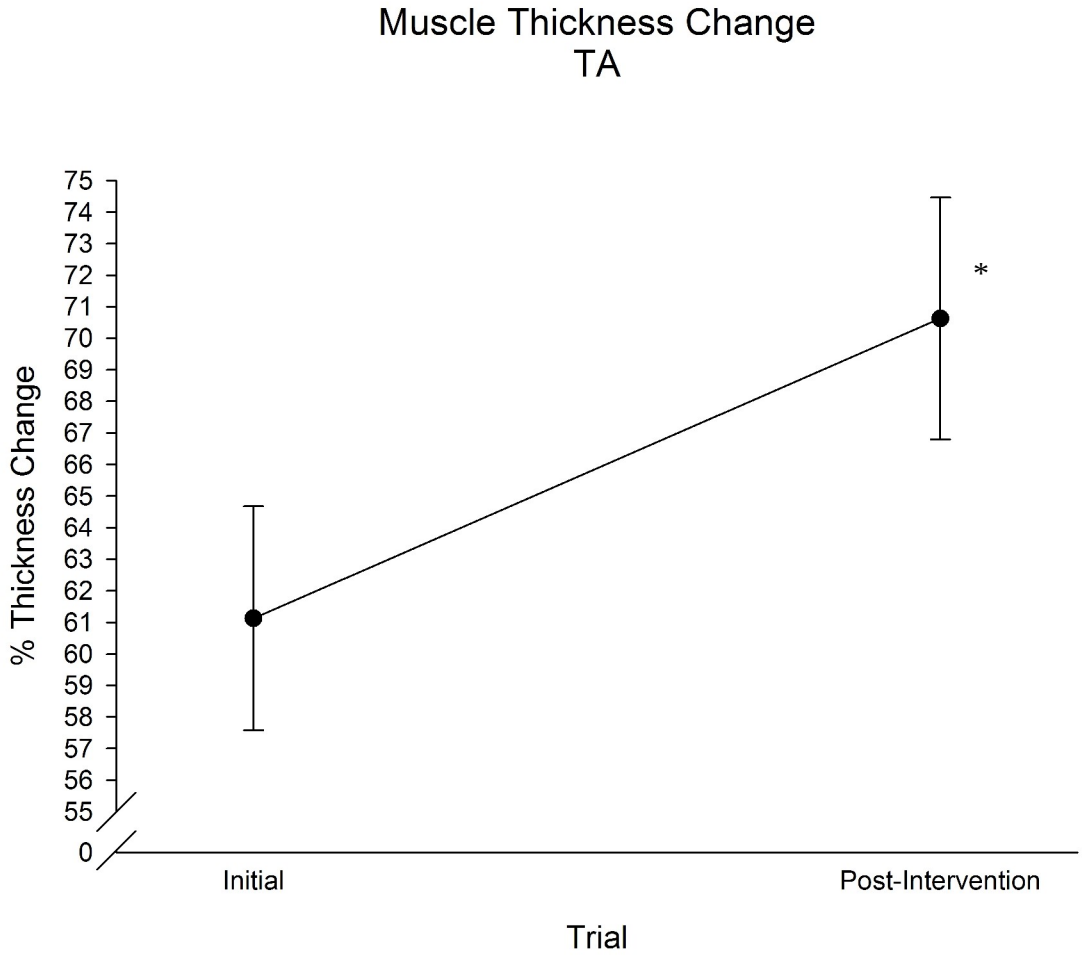
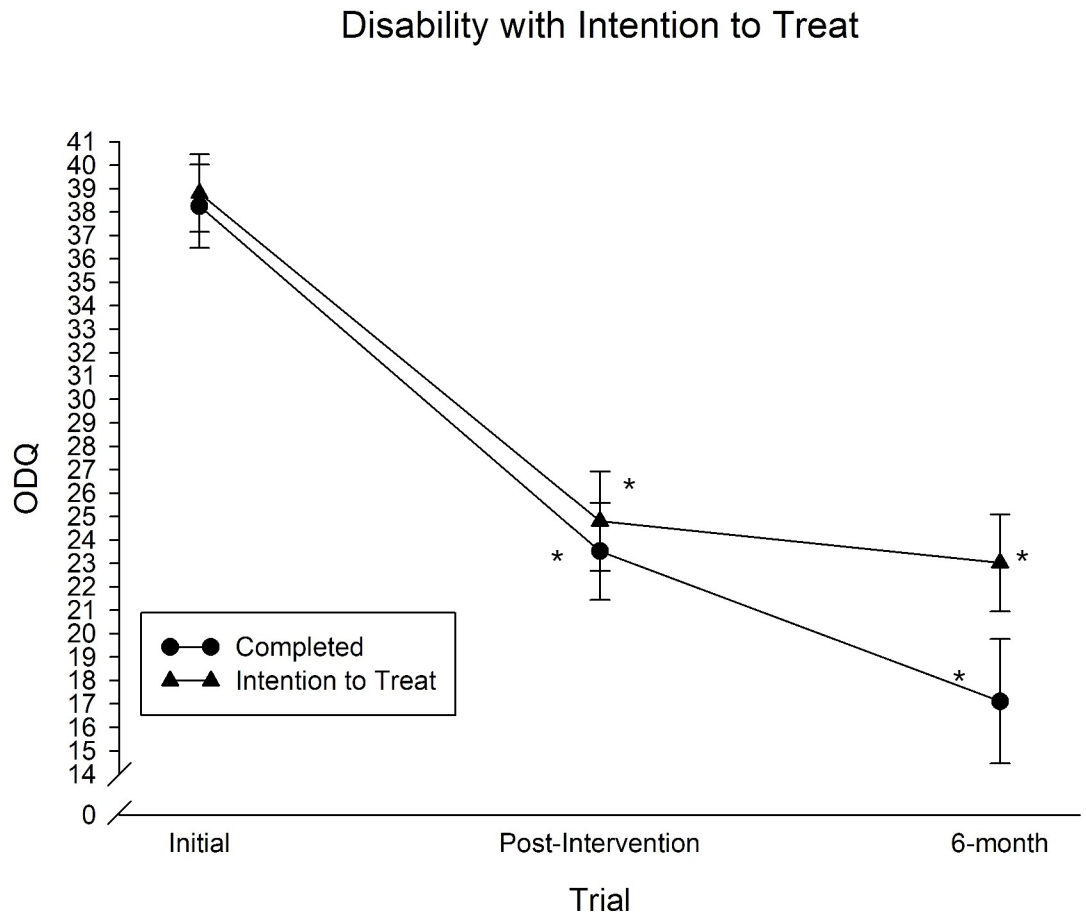


Figure 4.8. Graph of muscle thickness change of all subjects completing the intervention of the TrA before and after intervention.



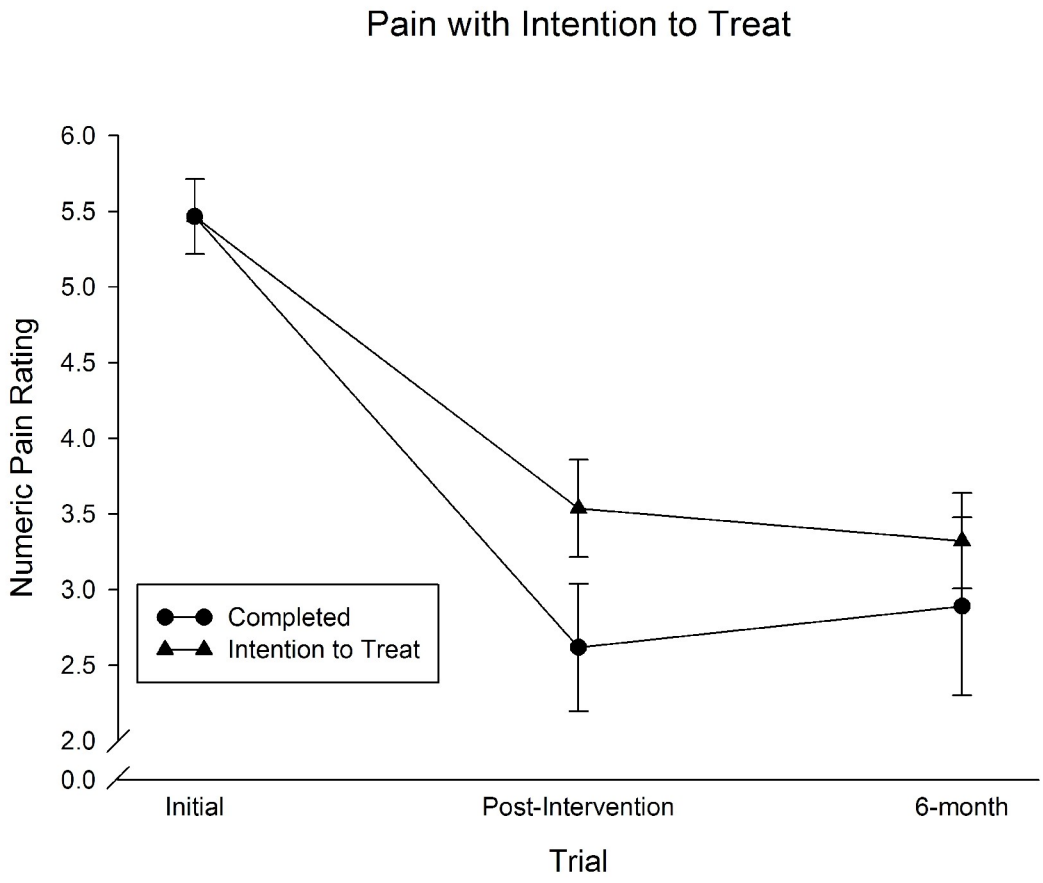
* Indicates value is significantly different ($P < .01$).

Figure 4.9. Graph of all subjects disability change (Intention to treat) and just those completing the all aspects of the study (completed).



*Indicates value is significantly different from initial ($P < .01$).

Figure 4.10. Graph of all subjects numeric pain rating change (intention to treat) and just those completing the all aspects of the study (completed).



Chapter 5
Clinical Applications and Conclusion

To date, RUSI of the LM has been limited to girth measurement and for biofeedback purposes as subjects learn volitional activation. The main purpose of this dissertation was to determine if RUSI could be used to assess the magnitude of activation of the LM. A prone arm lifting model was developed, utilizing percent thickness change from rest to activation, which was shown to generate measurable activity in the LM while controlling for movement artifact during image acquisition.

Thickness Change

The results of the first study demonstrated the relationship between muscle thickness change as measured by RUSI during contralateral arm lifting and EMG activity. The key findings from this study were the positively correlated, curvilinear relationship between thickness change and EMG ($r = 0.79$) and that when the limb is loaded with small relative loads (1-3 lbs.) the LM EMG and thickness change increase.

Previous studies measuring thickness change and EMG activity of other muscles have reported conflicting results. Hodges et al[55] compared EMG activity to architectural change measured by ultrasonography in several muscles across a broad range of activation levels. This study measured thickness change and EMG activity of the tibialis anterior, biceps brachii, brachialis, internal oblique and transverse abdominis and reported a curvilinear relationship where ultrasonography could detect changes at low levels of contraction (up to 20-30% of MVIC) and higher levels of contraction produce little further thickness change. McMeeken et al[76] measured the transverse abdominis during abdominal hollowing from 5% to 80% of MVIC and demonstrated a linear relationship between thickness change and EMG activity across all levels of activation measured ($P < 0.001$, $R^2 = 0.87$). The prone arm lifting model utilized in our study produced contractions from 19% to 43% of maximum, with a high correlation ($r=0.79$, $p<0.001$) between thickness change and EMG activity. There was no significant difference in thickness change between the last two levels of activation, indicating that EMG continued to increase with load but thickness change was nearing its maximum. It is likely that during this isometric contraction, the point is reached at which tendon

stiffness precludes further tendon stretch and the muscle continues to form cross bridges and increase electrical activity but with minimal further change in length, and therefore thickness. Muscles are considered to reach their maximum thickness at relatively low EMG values (~20-30% of MVIC)[55] and this likely occurred in our study at 34% of MVIC on average. It is thought that adequate joint compression can be achieved with low level muscle activation (~20% of MVIC) which is in the most linear part of the relationship where EMG activity reflects thickness change (see chapter 2). Because adding a load significantly increased EMG activity that was still measurable with the ultrasound, unloaded and loaded tests were included in the clinical study to determine which test had more clinical meaning.

Pain Related Changes

The next step was to determine if pain-related changes in muscle activation could be measured with the ultrasound. Current literature suggests utilizing an experimentally induced pain model to measure how pain affects different aspects of motor control. Experimental pain can be induced by many methods, but hypertonic saline-induced pain has been used extensively to test the effects of pain on various aspects of motor control [38] and specifically to study the effects of pain on motor control of spinal muscles.[3, 53, 121] In the presence of pain, the pain-adaptation model[70] predicts increased activity when a muscle would normally be silent and decreased activity when a muscle would normally be active. Therefore a decrease in LM thickness change was expected.

The same prone arm lifting model was used and as in the first study demonstrated similar thickness changes in the LM, increasing with load. During the induced-pain condition, thickness change was significantly reduced across all but the second load level ($P = .01$). This level had more variability than the other 4 and with a sample size of only 6 subjects, resulted in a non-significant outcome.

The standard error of the measure (SEM) must be considered when assessing the clinical application of using this model to measure LM activation. The average thickness change difference across the arm lifting tasks between the control and painful condition was 7.7% (7.5-8.1%). The SEM calculated from patients in study 3 was, on average 2.16%. The SEM is much smaller than the amount of change measured in the LM,

suggesting the thickness change measure has sufficient precision to detect if a thickness change deficit is present in a given patient.

There are few studies which directly measure LM activity during induced pain. Hodges et al[53] measured activity in the LM during induced pain and reported an increase in activity during rapid arm lifting. A decrease in activity was expected and the authors postulated that because subjects were in the standing position, the increased LM activity may have been part of a protective trunk splinting response.

The majority of studies reported have used surface EMG to assess response to pain in superficial muscles. Arendt-Nielsen et al[3] induced pain with hypertonic saline and demonstrated an increase in erector spinae activity during walking. They did not report EMG activity as a function of the phases of the gait cycle so it is difficult to interpret their findings. Zedka et al[123] measured erector spinae activity during trunk flexion and extension found an increase in activity when EMG activity was normally silent and a decrease or no change when EMG activity was normally high. The studies suggest that position and task may affect whether muscle activity increases or decreases in the presence of pain.

Clinical study

Using the same prone arm lifting model, the final study was designed to measure LM activation and to accommodate the need to classify subjects with LBP into pertinent subgroups. The traditional approach of considering mainly duration of symptoms as the primary between group factor in LBP intervention studies has been highly criticized of late and has caused somewhat misleading research conclusions. The Treatment-Based Classification was utilized because it has a growing body of reliability and validity literature and because of clinical observation that subjects in each of the 3 main treatment categories demonstrate deep muscle activation deficits.

The reliability and stability of the RUSI measure was established in a patient group and found acceptable for clinical use. On average subjects with LBP did demonstrate a thickness change difference when compared to controls. Differences were identified on the loaded tests only. Post hoc testing revealed the differences were in the direction specific and stabilization groups at the L4 level and in the direction specific group at the L5 level.

A key finding was that only the loaded tests showed a difference in LM thickness change. Many subjects with LBP do not respond to a small load in the same manner as control subjects. The finding that thickness change on the loaded test for the mobilization category was not significantly different from the control, was likely due to a high degree of variability. The mean (SD) thickness change for the mobilization group for the L4 loaded test for example, was 17.9 (12.1) compared to the control of 25.0 (7.5). The direction specific category was 17.8 (9.6) (see table 4.5). Because of variability in the data the mobilization group was not significantly different, but many individual subjects in the group demonstrated thickness change well below the mean of the control (Figures 5.1-5.3). The mobilization group demonstrated the greatest variability, but there were substantial individual differences in the other categories as well. Based on these differences, it may be best to interpret these data from the perspective that any given patient may have a meaningful thickness change deficit. For future intervention studies it will be important to include only those subjects who have a deficit. With a larger sample size of homogenous subjects with LM deficit, it will be more likely that a relationship between the loaded LM thickness change impairment and a meaningful outcome measure could be established. Subsequent studies could be conducted to determine if/what intervention could reverse this dysfunctional response. Such an approach speaks to the importance of meaningful classification and matching patients to the specific intervention they need.

Measurements of the TrA were also included in the induced pain study and the clinical study. The clinically popular abdominal draw-in technique was used. All subjects with LBP had thickness change deficits when compared to controls. The post-intervention testing revealed a significant improvement (75% thickness change), but the mean value was still significantly below that of the control group (99%, $P = < 0.01$).

There was a significant change in subjects' ability to volitionally activate the TrA but no difference in LM thickness change and no association with pain or disability. This suggests that pain is not the main factor responsible for a LM thickness change deficit since pain improved but thickness change did not.

The intervention program here did include volitional isolation of the TrA throughout; however, it did not include isolated motor control activities targeted directly

at the LM. No reported study has directly compared volitional isolation exercises with isolated activities to determine which is best for normalizing a dysfunctional LM. It has been shown that when RUSI is used for real-time feedback, asymptomatic subjects learn how to volitionally activate the LM sooner than those who do not receive feedback.[112] This line of research needs to be extended to a patient population to establish the most effective intervention for LM dysfunction.

Recurrence of LBP occurs in up to 73% of subjects within one year following an acute episode and has been shown to contribute disproportionately to the overall cost of treating this disorder in the United States.[115] Recurrence has been shown to be associated with LM girth in one study[45] and recurrence is being measured at 6 months. To date 19 of the 6-month questionnaires have been returned and 11 (58%) subjects have reported a recurrence. Several variables including LM thickness change, TrA thickness change, chronicity, and disability levels were explored among subjects reporting recurrence and those with no report of recurrence. Only the 6-month disability score was found to be significantly different ($P = .05$) between groups. The mean Oswestry of subjects who reported recurrence was 21 (SD 11) compared to those who did not (M = 10, SD 9). A trend was noted in the loaded L5 test; those who recurred had a mean of 9% change compared to a 12% change in subjects who did not recur ($P = .15$). When the data set is complete, this variable may warrant future research.

Following intervention, a significant reduction in pain and disability was achieved, even when dropouts were considered using an intention-to-treat analysis. The design here did not include a control group and because of the high dropout rate, these data must be considered pilot data for future research. The disability reduction measured in this study is consistent with other research. Fritz et al[32] compared the TBC system against standard medical care in a group of workers with acute LBP. The group receiving care based on the TBC system experienced a significant reduction in disability with their Oswestry scores improving from 43 to 21 in 4 weeks. Oswestry scores in the current study improved from 39 to 25 in approximately 6 weeks. Long et al[68] studied the effect of direction specific exercise on subjects with LBP and reported a 35% reduction in disability (Rolland-Morris improved from 17 to 11) which is consistent with the 36% change seen in the current study.

Clinical Application

In study 2 we measured how induced pain produces short-term changes in the activation of the TrA and LM. But longer-term changes in motor control in subjects with LBP have been identified and are encountered more often clinically. There is a growing number of studies that have identified an increase in EMG activity in subjects with LBP or with a history of LBP in superficial muscles during either functional tasks[91] or when small loads are applied to the trunk. Measuring EMG of the trunk muscles during trunk loading, Cholewicki demonstrated athletes with a recent history of LBP shut off fewer muscles and did so with delayed latencies when compared to matched controls.[18] In a prospective study with a 3-year follow-up period, Cholewicki et al[16] again measured muscle reflex latencies in response to trunk loading into flexion, extension, and lateral bending in college athletes to determine if muscle reflex latency had a relationship with developing LBP. There were 292 athletes used for the final analysis and 11% developed LBP. A regression model, consisting of history of LBP, body weight, and the latency of muscles shutting off during flexion and lateral bending predicted 74% of LBP episodes. The odds of developing LBP increased 3x when a history of LBP was present, but also increased by 3% with each millisecond of abdominal muscle shut-off latency. It may be that delayed latencies reflect a preexisting risk factor and are not the effect of a LBP episode.

Using a similar trunk loading model, Stokes et al[105] showed subjects with a history of LBP generated higher EMG activity, compared to controls, just prior to the trunk load being applied. Additionally, subjects demonstrated greater relative paraspinal muscle activity during a ramped maximal effort task.

Pirouz et al[91] measured the magnitude of activity in superficial muscle during sub-maximal rotational activities in subjects with chronic LBP and compared them to controls. Subjects with LBP demonstrated a consistent increase activity in the gluteals, hamstrings and erector spinea during the rotational tasks tested. These findings support the hypothesis that increased activity in superficial muscles may be a motor control impairment related to LBP. Increased superficial muscle activity seems to be retained after a LBP episode and may be a potential cause of LBP. These data, coupled with the research demonstrating dysfunction of the LM and TrA (see Introduction), suggest motor

control changes of trunk muscles related to LBP are somewhat complex and therefore may not respond to a basic progressive resistance therapeutic exercise approach.

The clinical study (study 3) demonstrated that most subjects with LBP did not respond to small loads the same way that controls did. It took the load to expose the difference in thickness change between subjects and controls. Subjects with LBP demonstrated a higher level of activation of superficial muscles and had longer latencies during perturbation testing. This suggests that the CNS adapts a rigid protective strategy resulting in an increased neural drive to superficial muscles. Increased neural drive to superficial muscles may lead to a concurrent decrease in drive to deeper muscles and, in some subjects with LBP, this strategy may be retained after the related pain and disability resolves. Perhaps LM thickness change reduction during load is a motor control impairment that can be clinically measured to identify subjects who need select LM motor control intervention.

Conclusion

Thickness change and EMG is considered to be curvilinear across the entire span of activation levels for most muscles. Study one demonstrated that at lower levels of EMG activity the relationship is linear, but can be considered curvilinear across the entire range of activation. Because LM and TrA are thought to produce adequate joint stabilization at lower levels of activation, measurement with RUSI at these lower levels may provide meaningful information on the function of these muscles. Using RUSI to measure muscle thickness change has been shown to be reliable and precise enough to detect differences between subjects with LBP and control subjects.

The results from the clinical study demonstrated that on average, subjects with LBP in two of the three categories had LM deficits on the loaded test. There was significant variation between subjects with a majority demonstrating at least one level or side that was different from controls. The standardized intervention did not stimulate a change in the LM as measured. The program had more motor control activities for the TrA which did significantly improve, but did not include motor control activities for the LM. Emerging evidence suggests greater superficial muscle activity is present in subjects with LBP. Perhaps training the superficial muscles in such cases only serves to further the dysfunction by making the superficial muscles more hyperactive and

concurrently decreasing the stimulus to the LM. Future research should randomize subjects with known LM deficits into different intervention groups and test to see which program is more effective in normalizing LM function. It would also be meaningful to test subjects with LM deficits and see if they have a greater frequency of superficial muscle hyperactivity.

These research studies suggest that:

1) lumbar multifidus thickness change is highly correlated with EMG activity, is sensitive to pain-related changes and can be measured clinically with RUSI.

2) TrA activation deficits exist in subjects with LBP across the TBC categories and resolved following intervention

3) although quite variable, LM activation deficits exist in two of the three TBC categories for the loaded test only

4) LM thickness change did not improve (normalize) following the applied intervention

5) Future research should identify if selected intervention can normalize LM muscle thickness change deficits and if normalized LM thickness has a meaningful relationship on clinical outcomes

Figure 5.1. Lumbar multifidus thickness change for the L4 level during the loaded test. Individual data points are shown with mean and standard error.

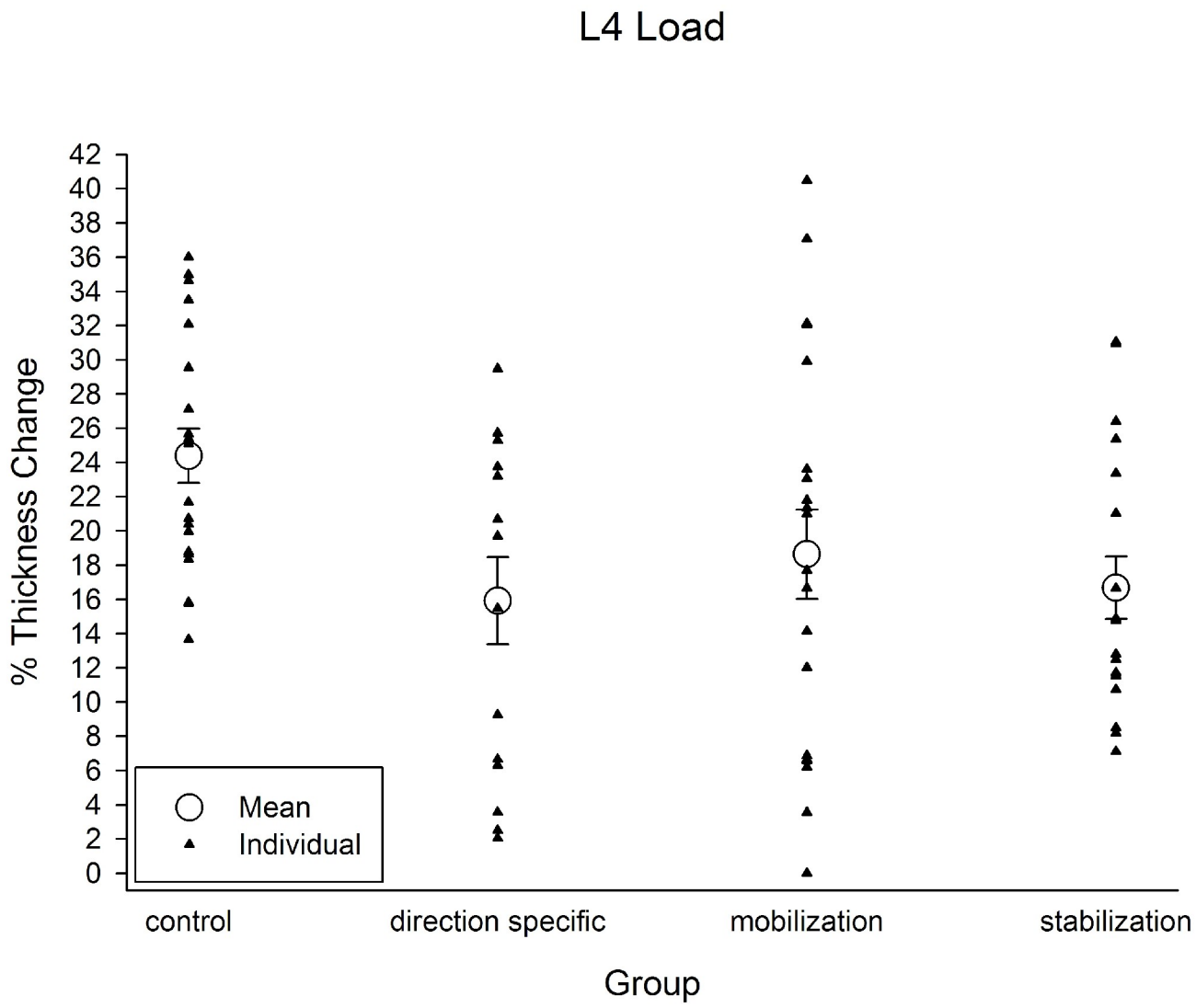


Figure 5.2. Lumbar multifidus thickness change for the L5 level during the loaded test. Individual data points are shown with mean and standard error.

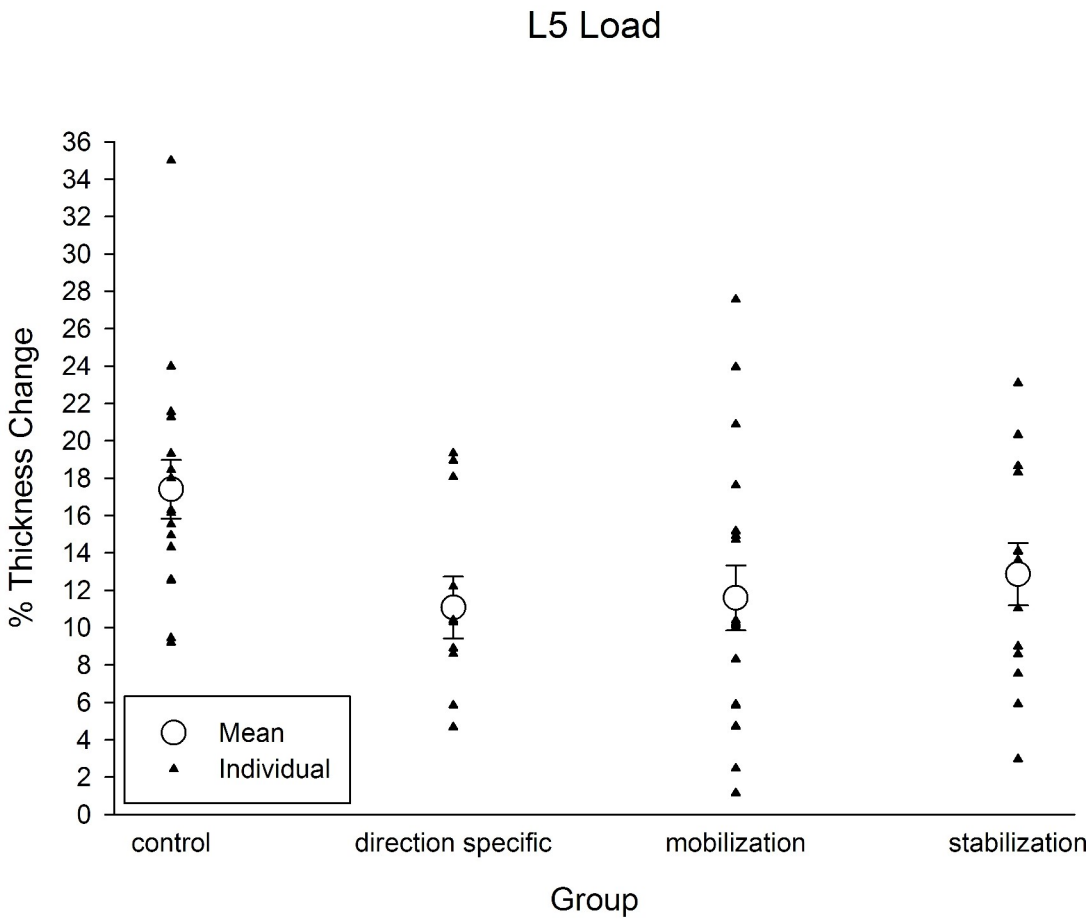
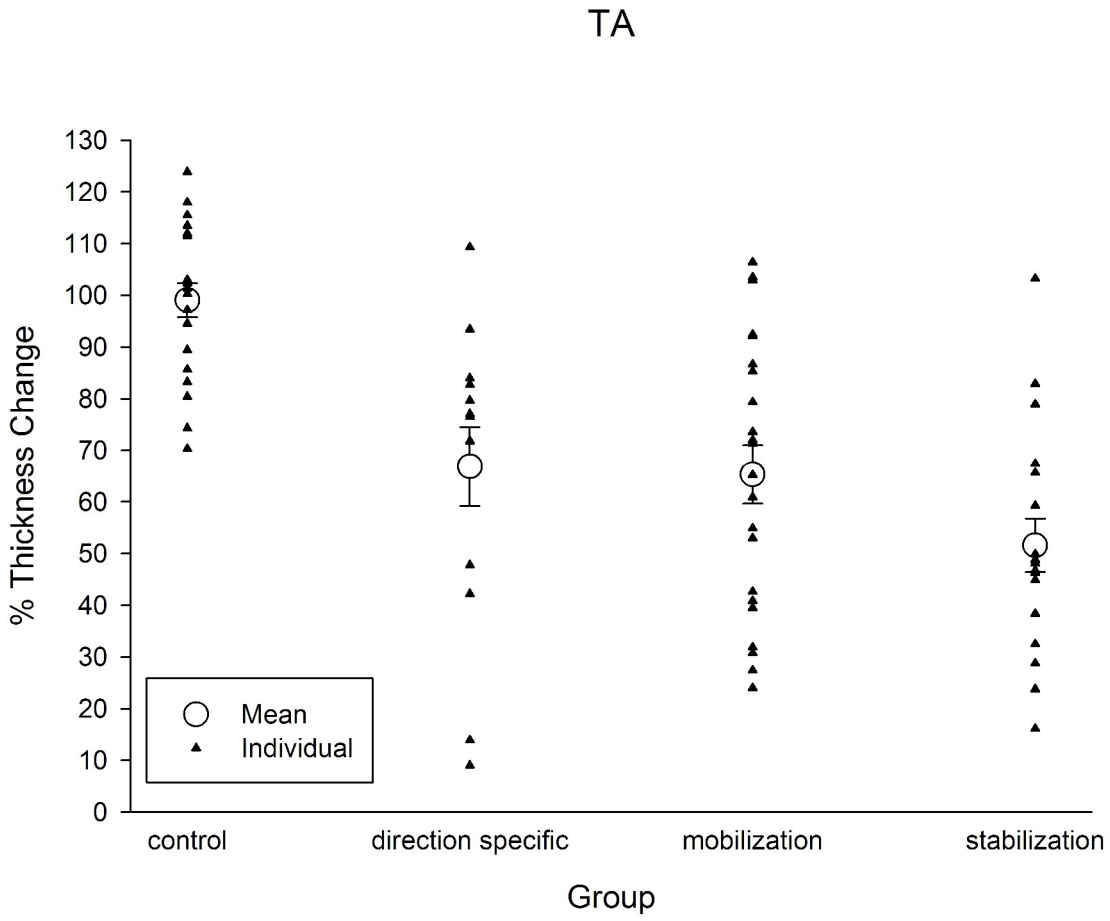


Figure 5.3. Transverse abdominis thickness change during the abdominal drawing-in maneuver. Individual data points are shown with mean and standard error.



Appendix A

Consent to Participate in a Research Study

Title: Measurement of Select Trunk Muscle Thickness Change in Subjects with Acute Low Back Pain Classified in the Treatment-Based Classification System

WHY ARE YOU BEING INVITED TO TAKE PART IN THIS RESEARCH?

You are being invited to take part in a research study about muscle contraction in low back pain (LBP) patients. You are being invited to take part in this research study because you have been diagnosed with LBP or you have not had back pain and your measurements will be considered normal and used to compare to those with LBP. If you volunteer to take part in this study, you will be one of about 80 people to do so.

WHO IS DOING THE STUDY?

The person in charge of this study is Kyle Kiesel MPT from ProRehab PC and University of Evansville. He is being guided in this research by his doctoral committee chairperson Terry Malone EdD, PT from the University of Kentucky. There will be other people on the research team assisting at different times during the study.

WHAT IS THE PURPOSE OF THIS STUDY?

The purpose of the this study is to learn how muscle contractions of certain deep muscles are different in people with LBP compared to those people without LBP and to learn how people with a problem with deep muscle contractions respond to an exercise program. By doing this study, we hope to learn how muscle contractions differ in people with LBP and if the new exercise program works better to treat LBP and prevent future LBP episodes.

WHERE IS THE STUDY GOING TO TAKE PLACE AND HOW LONG WILL IT LAST?

The research procedures will be conducted at ProRehab, PC an outpatient physical therapy clinic where you have been referred for physical therapy treatment for your LBP. You will need to come to the Indian St. clinic which is at 7300 East Indian St. Suite 102 or we will arrange for testing at the ProRehab location where you are receiving your physical therapy. If you participate in the study, you will need to attend two testing sessions, lasting 30-45 minutes each. One will be at the start of your physical therapy and one at the end. If the results of the deep muscle test reveal that you have weakness in or difficulty contracting

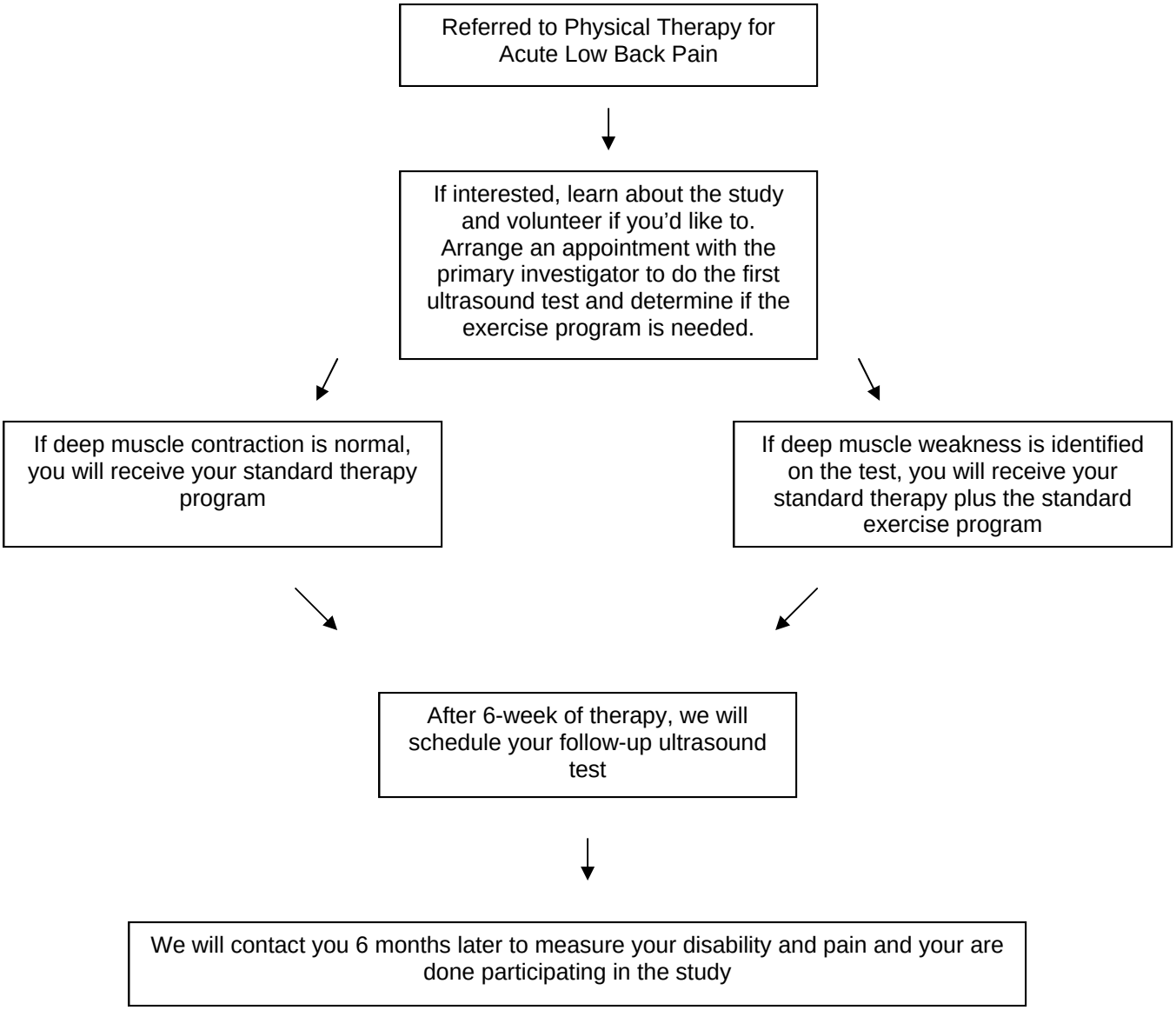
the deep muscles, you will be placed on a standard exercise program that has been used in other studies and proven to be effective. The exercises will be part of your regular physical therapy which you will attend two times a week for approximately 6 weeks. The total amount of time you will be asked to volunteer for this study includes the 6 weeks of physical therapy and we will contact you by mail or phone 6 months after you complete the therapy to keep track of how you are doing.

WHAT WILL YOU BE ASKED TO DO?

If you agree to participate in this study, we will first test your ability to contract two deep spinal muscles using ultrasound imaging. Ultrasound imaging is the same thing doctors use to look at a baby in a mothers stomach, we just use it to look at muscles and how they thicken during contractions. (This is part of the experiment; we think these contractions may be different in people with LBP, so we will compare the measurements of people with LBP to those without LBP to find out). You do not feel anything from the ultrasound waves, just light pressure from the sound head and cold from the gel. To test the deep stomach muscle, you will lie on your back on the table and we will put a little bit of gel over the outside part of your stomach. We will then teach you how to contract the deep stomach muscle we want to measure. This is done by doing the “abdominal draw-in maneuver”. You simply draw your lower stomach gently in toward your spine. Once you have learned how to do this, we will measure the muscle at rest, then during a contraction. This will be repeated on the other side. For the other test we will have you lie on your stomach on the table over one or two pillows used to flatten your low back. We will then place gel on your back and use the ultrasound to see a deep back muscle. You will lift your arm up, a few inches from the table and that makes the deep back muscle contract so we can measure it. We will do it two times on each side, first with no weight in your hand, then with a small (1-2.5 lbs) weight in your hand. All the gel will be wiped off and the ultrasound measurements will then be completed. You will then go through another physical therapy examination which will be about the same as the one you did your first day of physical therapy. This will allow us to determine what type of LBP you have and which type of manual therapy and range of motion exercise techniques will be best for your type of LBP.

The results of the ultrasound test will allow us to determine if your deep muscles are normal or need some exercise training to improve the contractions. If your muscles contractions need exercise training, you will be progressed through a standard exercise program by your physical therapist. You will be asked to fill out the same pain and disability questionnaires you did the first day after two weeks of treatment and at the end. After you complete your 6-weeks of therapy, we will repeat the ultrasound test, just like we did the first time. You are then done with the active part of the study. We will contact you by mail and/or phone 6 months later to have you fill out the questionnaires again so we can measure your progress.

Time-line Chart:



ARE THERE REASONS WHY YOU SHOULD NOT TAKE PART IN THIS STUDY?

There are a few reasons why you should not participate in this study. They are primarily based on the type of LBP you have, but if you are less than 18 or older than 60 years of age, you do not qualify for the study. If your LBP is causing pressure on a nerve, like a pinched nerve, and that is giving you weakness in your leg or changing your knee or ankle jerk reflex, we will keep you out of the study. If your LBP is caused by an infection, cancer, fracture or some type of congenital problem you do not qualify for this study. If you are pregnant, please tell us and you would not qualify for this study. You should not participate in this study if you have recently ingested any type of contrast material for another medical test, such as barium. This is because we should not do the ultrasound test on you if you have had this in the past two days.

WHAT ARE THE POSSIBLE RISKS AND DISCOMFORTS?

There are no risks beyond that of standard physical therapy treatment for the exercise and manual therapy procedures we are using in this study. For the ultrasound, there is a risk if you have taken a contrast material as described above or if the ultrasound is applied continuously too long. We will not exceed the recommended time for ultrasound application. There is always a chance that any medical treatment can harm you, and the treatment in this study is no different. We will do everything we can to keep you from being harmed. In addition to the risks listed above, you may experience a previously unknown risk or side effect.

WILL YOU BENEFIT FROM TAKING PART IN THIS STUDY?

There is no guarantee that you will get any benefit from taking part in this study. However, most people have improvement with their LBP when receiving this type of physical therapy treatment. We cannot and do not guarantee that you will receive any personal benefits from taking part in this study. Your willingness to take part, however, may, in the future, help physical therapists better understand and/or treat others with LBP.

DO YOU HAVE TO TAKE PART IN THE STUDY?

If you decide to take part in the study, it should be because you really want to volunteer. You will not lose any benefits or rights you would normally have if you choose not to volunteer. You can stop at any time during the study and still keep the benefits and rights you had before volunteering. If you decide not to take part in this study, your decision will have no effect on the quality of physical therapy care you receive.

IF YOU DON'T WANT TO TAKE PART IN THE STUDY, ARE THERE OTHER CHOICES?

If you do not want to be in the study, there are no other choices except not to take part in the study.

WHAT WILL IT COST YOU TO PARTICIPATE?
You and/or your insurance company, Medicare or Medicaid will be responsible for the costs of all care and treatment you receive during this study that you would normally receive for your condition. These are costs that are considered medically reasonable and necessary and will be part of the care you receive if you do not take part in this study.
ProRehab PC may not be allowed to bill your insurance company, Medicare, or Medicaid for the medical costs of procedures done strictly for research. We will not bill you for the time it takes to do the ultrasound testing or the repeat physical therapy examination. The rest of the study is part of your physical therapy treatment and normal charges will be billed.
If you have questions about whether your insurance company, Medicare, or Medicaid will pay these costs, you should ask them if they will agree to pay these costs.

WHO WILL SEE THE INFORMATION THAT YOU GIVE?
We will keep private all research records that identify you to the extent allowed by law.
Your information will be combined with information from other people taking part in the study. When we write about the study to share it with other researchers, we will write about the combined information we have gathered. You will not be identified in these written materials. We may publish the results of this study; however, we will keep your name and other identifying information private.
We will make every effort to prevent anyone who is not on the research team from knowing that you gave us information, or what that information is. For example, your name will be kept separate from the information you give, and these two things will be stored in different places under lock and key. You should know, however, that there are some circumstances in which we may have to show your information to other people. For example, the law may require us to show your information to a court.

CAN YOUR TAKING PART IN THE STUDY END EARLY?
If you decide to take part in the study you still have the right to decide at any time that you no longer want to continue. You will not be treated differently if you decide to stop taking part in the study.
The individuals conducting the study may need to withdraw you from the study. This may occur if you are not able to follow the directions they give you or if they find that your being in the study is more risk than benefit to you.

WHAT HAPPENS IF YOU GET HURT OR SICK DURING THE STUDY?
If you believe you are hurt or if you get sick because of something that is done during the study, you should call Kyle Kiesel at (812) 589-5826 immediately. It is important for you to understand that the University of Kentucky or the University of Evansville will not pay for the cost of any care or treatment that might be necessary because you get hurt or sick while taking part in this study. That cost will be your

responsibility. Also, neither university will pay for any wages you may lose if you are harmed by this study.
Medical costs that result from research-related harm can not be included as regular medical costs. Neither university will be allowed to bill your insurance company for such costs. You should ask your insurer if you have any questions about your insurer's willingness to pay under these circumstances. Therefore, the costs related to your care and treatment because of something that is done during the study, such as the need for additional treatment for your LBP will be your responsibility.

WILL YOU RECEIVE ANY REWARDS FOR TAKING PART IN THIS STUDY?
You will receive a payment of $15 when you complete the first ultrasound test and an additional $25 when you complete the second ultrasound test.

WHAT IF YOU HAVE QUESTIONS?
Before you decide whether to accept this invitation to take part in the study, please ask any questions that might come to mind now. Later, if you have questions about the study, you can contact the primary investigator, Kyle Kiesel at (812) 589-5826. If you have any questions about your rights as a volunteer in this research, contact the staff in the Office of Research Integrity at the University of Kentucky at (859) 257-9428 or toll free at 1-866-400-9428. You may also call the General Clinical Research Center Subject Advocate if you have questions about your rights and welfare at (859) 323-5049, ext. 230. We will give you a signed copy of this consent form to take with you.

WHAT ELSE DO YOU NEED TO KNOW?
You will be told if any new information is learned which may affect your condition or influence your willingness to continue taking part in this study.

_____ _____
Signature of person agreeing to take part in the study Date

_____
Printed name of person agreeing to take part in the study

_____ _____
Name of [authorized] person obtaining informed consent Date

_____
Signature of Investigator

Appendix B

MODIFIED OSWESTRY LOW BACK PAIN DISABILTY QUESTIONNAIRE

This questionnaire has been designed to give your therapist information as to how your back pain has affected your ability to manage in everyday life. Please answer every question by placing a mark in the one box that best describes your condition today. We realize you may feel that 2 of the statements may describe your condition, but please mark only the box that most closely describes your current condition.

Pain Intensity

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

Personal Care (e.g., Washing, Dressing)

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

Lifting

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

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 ½ mile.
□Pain prevents me from walking more than ¼ mile.
□I can only walk with crutches or a cane.
□I am in bed most of the time and have to crawl to the toilet.

Sitting □I can sit in any chair as long as I like. □I can only sit in my favorite chair as long as I like. □Pain prevents me from sitting for more than 1 hour. □Pain prevent me from sitting for more than ½ hour. □Pain prevents me from sitting for more than 10 minutes. □Pain prevent me from sitting at all.	**Social Life** □My social life is normal and does not increase my pain. □My social life is normal, but it increases my level of pain. □Pain prevents me from participating in more energetic activities (e.g., sports, dancing) □Pain prevents me from going out very often. □Pain has restricted my social life to my home. □I have hardly any social life because of my pain.
Standing □I can stand as long as I want without increased pain. □I can stand as long as I want, but it increases my pain. □Pain prevents me from standing more than 1 hour. □Pain prevents me from standing more than ½ hours. □Pain prevents me from standing more than 10 minutes. □Pain prevents me from standing at all.	**Traveling** □I can travel anywhere without increased pain. □I can travel anywhere, but it increases my pain. □My pain restricts my travel over 2 hours. □My pain restricts my travel over 1 hour. □My pain restricts my travel to short necessary journeys under ½ hour. □My pain prevents all travel except for visits to the physician/therapist or hospital.
Sleeping □Pain does not prevent me from sleeping well. □I can sleep well only by using pain medication. □Even when I take pain medication, I sleep less than 6 hours. □Even when I take pain medication, I sleep less than 4 hours. □Even when I take pain medication, I sleep less than 2 hours. □Pain prevents me from sleeping at all.	**Employment/Homemaking** □My normal homemaking/job activities do not cause pain. □My normal homemaking/job activities increase my pain, but I can still perform all that is required of me. □I can perform most of my homemaking/job duties, but pain prevents me from performing more physically stressful activities (e.g.,lifting, vacuuming). □Pain prevents me from doing anything but light duties. □Pain prevents me from doing even light duties. □Pain prevents me from performing any job or homemaking chores.

Utilization: Minimum clinically important difference is 6 points
(Sensitivity=91%; specificity=83%)
Test-retest reliability high {ICC(2,1)=0.90}
Scoring: Each section is scored 0 to 5. The first statement has the score of 0 assigned, the second statement a 1, and so forth so that the last statement in each section is assigned a score of 5. Patients are instructed to mark only one statement in each section. All section scores are totaled and then doubled to obtain the final percentage score. If two or more statements in a section is marked, the highest scoring state is used.

Interpretation of score:	0-20%	Minimal disability
	20%-40%	Moderate disability
	40%-60%	Severe disability
	60%-80%	Crippled
	80%-100%	Either bed-bound or exaggerating symptoms

Appendix C

FEAR-AVOIDANCE BELIEFS QUESTIONNAIRE

1. My pain was caused by physical activity……..
2. Physical activity makes my pain worse……..
3. Physical activity might harm my back……
4. I should not do physical activities which (might) make my pain worse……..
5. I cannot do physical activities which (might) make my pain worse……..

The following statements are about how your normal work affects or would affect your back pain.

6. My pain was caused by my work or by an accident at work……
7. My work aggravated my pain……
8. I have a claim for compensation for my pain……
9. My work is too heavy for me……
10. My work makes or would make my pain worse……..
11. My work might harm my back……..
12. I should not do my normal work with my present pain……
13. I cannot do my normal work with my present pain……
14. I cannot do my normal work till my pain is treated……
15. I do not think that I will be back to my normal work within 3 months……..
16. I do not think that I will ever be able to go back to that work…….

Work subscale is #'s 6, 7, 9, 10, 11, 12, and 15

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Publications

Kiesel, K.; Uhl, T.; Underwood, F.; Rood, D.; and Nitz, A.: Measurement of Lumbar Multifidus Muscle Contraction with Ultrasound Imaging. *Manual Therapy,* Sept. 2006.

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