The responses of trunk muscles to perturbations before and after Active Release Technique[®] of the hip flexor

by

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Descriptive Note (page 2)

MASTER OF Science (2010)McMaster University(Kinesiology)Hamilton, OntarioTITLE: The responses of trunk muscles to perturbations before and after Active ReleaseTechnique® of the hip flexorAUTHOR: Daniel Avrahami (McMaster University)SUPERVISOR: James Robert Potvin

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The purpose of this study was to investigate the influence of a passive myofascial therapy treatment (Active Release Technique[®] (A.R.T[®])) on various outcome measures with an asymptomatic tight hip flexor group (A-THF) (n=8) and a low back pain tight hip flexor group (LBP-THF) (n=10). These two groups were also compared with a control group (CON) (n=8) The outcome measures for this study were: 1) Trunk muscle EMG measurements monitoring unloading perturbations (Unknown Timing (UT) and Known Timing (KT)) and Unstable Standing (US) perturbations. 2) Maximum voluntary trunk flexor and extensor moments (Flex_{Max} and Ext_{Max}), 3) disability and pain measurements (RMDQ and VAS) with self-efficacy (PSEQ) evaluated as a covariate. 4) Hip extension mobility.

The results from this study demonstrated both significant short term and sustained improvements in trunk Flex_{Max} and Ext_{Max} . For the THF groups, A.R.T[®] resulted in significant acute (within session) increases in Ext_{Max} of 20.6%, 11.9% and 12.3% on days 1, 3 and 4, respectively. After the 2 week treatment program was completed, the THF Groups demonstrated an average increase in Ext_{Max} of 25% compared with their baseline values. After the treatment, the LBP-THF group had trunk Flex_{Max} and Ext_{Max} values that were 34% and 32.3%, respectively, compared with the CON group.

The full treatment program of the LBP-THF group was associated with a reduction in disability by 2.8 points and pain by 2.9 cm. Interestingly, self-efficacy was found to be a significant covariate for the disability outcome measure. For the passive hip extension measurements, the LBP-THF group and the A-THF groups increased their values by 13.1° and 8.0°, respectively. In addition, the other two hip extension tests (knee extended and knee bent tests) demonstrated significant increases in measured passive hip extension values over the

course of the A.R.T[®] treatment and, by the end of the study, the LBP-THF groups' hip extension values were not significantly different from that of the CON group.

The US perturbation trials showed a significant decrease in average muscle activity for the TES, LES, MULT and GLUT muscles over the course of the treatment program, but there were no significant and clinically relevant changes observed in anticipatory adjustment and baseline muscle activity for the KT and UT perturbation trials. However, after the treatment program, there was a trend for the THF Groups' baseline and anticipatory adjustment EMG ampltidues to shift closer to those of the CON group for most muscles.

The results from this study suggest that the utilization of A.R.T[®] can result in clinically important benefits, for patients with tight hip flexors and low back pain ,by decreasing pain and disability while improving trunk strength and hip extension flexibility.

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ABBREVIATIONS

CNS - Central Nervous System

- ERS Erector Spinae
- EO External Oblique
- GM Gluteus Maximus
- GMED Gluteus Medius
- IL Iliocostalis Lumborum
- IO Internal Oblique
- LAT Latissimus Dorsi
- LT Longissimus Thoracis
- LERS Lumbar Erector Spinae
- LM Lumbar Multifidus
- LS Lumbar Spine
- MVE Maximum Voluntary Exertion
- PM Psoas Muscle
- QL Quadratus Lumborum
- RA Rectus Abdominis
- TERS Thoracic Erector Spinae
- TrA Transverse Abdominis
- L1 to $L5 1^{st}$ to 5^{th} Lumbar Vertebrae
- S1 to $S5 1^{st}$ to 5^{th} Sacral Vertebrae

1.0INTRODUCTION

There is a high prevalence of people with low back pain in Canada and, therefore, it has a large socioeconomic impact on society(McIntosh *et al.*, 2000). Some of the risk factors include: vibration (Okunribido *et al.*, 2008), repetitive cyclical loading (Navar *et al.*, 2006), muscular fatigue (Moffroid, 1997) and sudden unexpected movements (Manning *et al.*, 1984), such as slips or falls, that can cause the neuromuscular system to overreact, causing injury to soft tissues containing nociceptors and proprioceptors (Lavender *et al.*, 1993b). In addition, epidemiological studies have attributed low back pain to heavy lifting, regular bending and repetitive movements (Roelen *et al.*, 2008). Therefore, it would seem logical that a combination of these risk factors may impose a heightened health risk on individuals which could contribute to low back pain and even disability.

Physical impairments are not the only contributing factor to developing chronic low back pain and disability. Brox *et al.*, (2005) have shown that self-efficacy for pain management, and fear-avoidance beliefs for work, were shown to significantly influence outcomes in patients with chronic low back pain. Self-efficacy refers to confidence in the ability to perform a particular task to bring about a particular outcome (Bandura, 1977). Brox's study also demonstrated that there was a stepwise increase in disability and impairment from healthy controls to patients with chronic low back pain. The World Health Organization defines disability as "the outcome or result of a complex relationship between an individual's health condition and personal factors, and of the external factors that represent the circumstances in which the individual lives". Impairments are defined as "problems in body function or structure such as significant deviation or loss"(Imrie, 2004). Woby *et al.*, (2007) suggest that, when self-efficacy for managing pain is high, elevated pain related fear might not lead to greater pain and disability. Therefore, self-

efficacy of the patients ability to perform activities of daily living should be taken into account when treating low back pain patients (Nicholas, 2007b).

One way to decrease low back injuries is to increase spinal stability. The definition of spinal instability is not always clear. Panjabi defines it as the degree of motion that prevents pain, neurological deficit, and abnormal angulation (Panjabi *et al.*, 1989). Mechanically speaking, the spinal system has generally been classified as being stable or unstable (Bergmark, 1989). However, clinical stability is viewed on a continuum (Panjabi and White, III, 1980). The premise behind the clinical theory of spinal stability is that the spine can vary in its stability and, therefore, it is important to continually strive to promote the patient to a more stable scenario. Whether we are discussing mechanical or clinical stability of the spine, stability must be assured to avoid risk of injury during daily activities (Panjabi, 1992a).

There are at least three different aspects of muscle activity that can contribute to spinal stability: 1) muscle moment production, 2) muscle timing and 3) muscle coordination. Each interacts with the others to vary the stiffness of spine, develop spinal stability and protect the body from any unforeseen situations that may occur (Hodges and Richardson, 1998; van Dieen and de Looze, 1999). A perturbation to the spinal system, without adequate muscular contributions to spine joint rotational stiffness, can lead to decreased stability of the spine and may can contribute to low back injury (van der Burg *et al.*, 2000). Timing of muscle activity has been shown to be altered in low back pain patients compared with healthy individuals (Hodges and Richardson, 1998). Research has shown that patients with low back pain, in contrast to healthy control subjects, demonstrated later onset of muscle response pattern in response to perturbations (Hodges and Richardson, 1998; Radebold *et al.*, 2001). This alteration in muscle activity in the low back pain patients can hamper spine stability and lead to injury through spine

buckling or tissue damage and failure (Cholewicki and McGill, 1996). Finally, increased trunk stiffness, due to cocontraction, has been shown to increase spine stability which protects it against perturbations (van Dieen *et al.*, 2003a).

A perturbation to an individual is one way to test the stability of the spine. Perturbations, especially those that are rapid and unexpected, will create a force to the spinal system and test the spine's ability to control its motion. Perturbations to the spine will trigger the central nervous system (CNS) to respond and restore the body to postural equilibrium. The system's response can be characterized by the onset rate of muscle activity and an increase in the number of muscles activated and the intensity of muscle activity (Lavender *et al.*, 1993b).

Many researchers have disturbed an individual's neuromuscular equilibrium and study the body's reaction to these perturbations. Researchers have manipulated numerous variables including self-imposed or external subject stimuli, addition of safety equipment, along with perturbations that are expected or unexpected, as well as symmetrical or asymmetrical (Zattara and Bouisset, 1988; Lavender *et al.*, 1993b; Cresswell *et al.*, 1994; Thomas *et al.*, 1998; Cholewicki and VanVliet, 2002; Stokes *et al.*, 2006).

Sudden perturbation to pain-free, healthy individuals have been shown to increase trunk muscle activity and create greater compressive loads on the spine, which can serve to increase spinal stability (Cholewicki and McGill, 1995a). However, excessive loads on the spine can compromise the spinal stability during everyday tasks and injure the tissues of the spine such as ligaments, bone and intervertebral discs (Lavender *et al.*, 1989; Cholewicki and McGill, 1996). Cholewicki & McGill (1996) and Janevic *et al.* (1991) suggest that compression forces from the psoas will create segmental stiffness and increased spinal stability. However, too much compression force from the psoas can have a detrimental effect on the spine's health (Juker *et*

al., 1998). Therefore, a compressive force from the psoas, which is neither too great nor too little, is important to developing spine stability. Juker *et al.* (1998) confirmed that a relatively low psoas muscle activity might create sufficient spinal stiffness.

In addition to muscle activity, the architecture of the spinal muscle can contribute considerably to spinal stability. Muscles that span multiple vertebral segments tend to be larger in cross-sectional area, volume and length (Bergmark, 1989). When recruited, these muscles balance the external loads to develop posture and movement. One multi-segmental muscle that has a significant effect on stability, posture and movement is the psoas which spans from the thoracolumbar region, across the lumbar spine and pelvis, to the femur attachment (Andersson *et al.*, 1995).

Segmental muscles, such as the multifidus, enhance spine stability by increasing intervertebral stiffness (Bergmark, 1989) and provide proprioceptive feedback to the spine via their mechanoreceptor properties (Brumagne *et al.*, 2000). The psoas is not considered a segmental muscle. However, the psoas attaches to the anterior portion of each successive lumbar transverse process along with the anterior and medial portion of each successive lumbar intervertebral discs and the lumbar vertebral bodies (Bogduk, 1992).

Since both structure and function of the neural, muscular and skeletal systems is crucial to controlling spinal stability, the combination of sufficient muscle force, correct muscle recruitment and timing patterns are important towards preventing episodes of low back pain. The psoas muscle is one of the muscles that are structurally and functionally important in providing spine stability by providing adequate spine compression (Juker *et al.*, 1998). Many studies have looked at this muscle in low back pain patients and several imaging studies have observed psoas atrophy in these patients (Cooper *et al.*, 1992; Parkkola *et al.*, 1993; Flicker *et al.*, 1993;

Dangaria and Naesh, 1998; Barker *et al.*, 2004; Kamaz *et al.*, 2007; Hides *et al.*, 2007). Clinically, dysfunction of the lumbar spine and pelvic muscles has been documented and these impairments have been characterized by decreased extensibility of hip flexors and back extensors, and weakness of the abdominals and hip extensor muscles (Jull, 1987). This cluster of impairments has been classified as "Pelvic Crossed Syndrome" (Jull, 1987). One of the key components of this syndrome is shortening and weakness of the psoas. Janda (1986) has shown that low back pain patients with tight hip flexors tend to have a hypoactive, hypotonic and weak gluteal muscles. Some of the contributing factors to this dysfunctional muscle include overexertion through repetitive hip flexion, continuous sitting or sleeping in the fetal position. Janda (1986) postulated that it is important to stretch the tight muscles, such as the psoas, prior to strengthening the weak muscles, such as the gluteal muscles.

Therefore, reasonable approach is for clinicians to stretch muscles that are tight and shortened, like the iliopsoas complex, and then strengthen the muscles that are inhibited and weak, such as the gluteal muscles. One common method of lengthening the iliopsoas complex is through Active Release Technique[®] (A.R.T. [®]). A.R.T. [®] is a widely used treatment technique utilized by over 10,000 health care professionals, of which approximately 50% are chiropractors and approximately 50% are physiotherapists, registered massage therapists and other health care professionals (Leahy, 2009). A.R.T. [®] is a therapy that targets the muscle and fascial systems, and promotes flexibility and mobility of the body's connective tissues (Leahy, 1995). It has been proposed that this type of therapy removes adhesions between the musculo-fascial interface and promotes restoration of normal tissue extensibility (Leahy, 1995). During this treatment, the therapist places the targeted muscle in a shortened position and then places their finger contact on the targeted treatment area. The therapist places light tension in the opposite direction of the

body movement and the muscle is subsequently lengthened. A.R.T[®] has been successful in treating trigger thumb, lateral epicondylosis and hamstring flexibility (George *et al.*, 2006; Howitt, 2006; Howitt *et al.*, 2006). However, there has been no known research to support the use of A.R.T[®] for low back pain patients.

There is only a small amount of research directed towards the contribution of the psoas, iliacus and iliopsoas complex to spine stability (Janevic *et al.*, 1991; Santaguida and McGill, 1995; Cholewicki and McGill, 1996; Juker *et al.*, 1998). In addition, there is no research examining perturbations of subjects with and without low back pain, and their muscle reflexes before and after treatment to the hip flexor complex.

The purpose of this study was to investigate the influence of a passive myofascial therapy treatment (Active Release Technique[®] (A.R.T[®])) on various outcome measures with an asymptomatic tight hip flexor group (A-THF) and a low back pain tight hip flexor group (LBP-THF) (n=10) and compare these two groups with a control group (CON).

2.0 LITERATURE REVIEW

The literature review will begin with a brief anatomical overview of all the structures that make up the trunk, including an important overview of the iliopsoas complex. This review will then describe the concept of spine stability and the factors that contribute to spine stability. One method that researchers have extensively used to study spine stability is to apply various types of perturbations to the spine and its surrounding structures. This literature review will examine some of the key studies that contributed to discovering important information that has furthered the body of knowledge on spine stability. Following this section, the importance of muscle dysfunction and its relationship with low back pain will be examined. One of the key muscle complexes that have been implicated in low back pain is the iliopsoas complex. The majority of the literature has simply examined the psoas and its' relationship with spine stability and low back pain. This review will examine all available literature regarding the psoas, iliacus and iliopsoas complex with respect to its role in spine stability, low back pain, pelvic cross syndrome and treatments that have been used to decrease low back pain in individuals with a tight iliopsoas complex. Finally, evidence indicating the importance of self-efficacy in decreasing low back pain will be presented.

2.1 Anatomy of the Iliopsoas Complex

The iliopsoas is a complex structure composed of 2 major portions, iliacus and psoas, originating from the iliac bone and lumbar spine respectively. This complex has one common insertion onto the lesser trochanter of the femur (Figure 1).

Dissection has shown that the anterior and posterior fascicles have a separate nerve supply. The posterior fascicles are supplied by the ventral rami of spinal nerves T12 through L4. The anterior fascicles are supplied by branches of the femoral nerve from L2, 3 and 4. It has also

been stated that psoas is innervated by the anterior rami of the lumbar nerves (L1-L3) and the iliacus is innervated by the femoral nerve (L2-3) (Moore, 1992).

The psoas attaches to the anterior portion of the lumbar transverse processes along with the anterior and medial portion of the lumbar intervertebral discs and the lumbar vertebral bodies (Bogduk, 1992). The fibres of the muscle course inferior and laterally to a tendon that follows the pelvic brim and attaches with the iliacus onto the lesser trochanter (Bogduk, 1992). The angle of pennation in the psoas major was shown to have the upper fibres, that originate from the lumbar spine, vertically oriented compared with the lower fibres (Santaguida and McGill, 1995). Santaguida & McGill also showed that these lower fibres did not cross the LS/SI level and tend to insert onto the posterior and medial aspect of the tendon and extended up two lumbar levels.

The lateral component of the psoas moment arm increases from upper to lower lumbar levels bilaterally. The moment arm running in the anterior/posterior direction is largest at the upper and lower end of the muscle and smallest where it reverses direction from posterior to anterior of the fulcrum at the L4/L5 level bilaterally (Santaguida and McGill, 1995).

The psoas has an intricate interaction with the fascial system. The psoas has connections with fascia that connects with the medial arcuate ligament and continues superiorly to the diaphragm. The left and right crus attach from the diaphragm to antero-lateral component of the upper three vertebrae and bodies. The crus and the fascia overlap the psoas muscle. They appear continuous with psoas and blend with the anterior longitudinal ligament. The fascia becomes thicker as it descends and it is continuous with the pelvic floor fascia. The fascia also forms a link with the conjoint tendon, transverse abdominis (TrA), and the internal oblique (IO). As the psoas passes over the pelvic brim, the fascia of the posterior fascicles attaches firmly to the pelvic brim (Moore, 1992).

According to Bogduk (1992) the iliopsoas is a muscle of the thigh and therefore its primary action is flexion of the hip. He also mentioned that in a fixed thigh position, such as in the sit-up position, the iliopsoas flexes the lumbar spine. However, biomechanical studies have shown that psoas has minimal influence on lumbar spine movement (Andersson et al., 1995; Santaguida and McGill, 1995) . Andersson et al. (1995) reported that both psoas and iliacus activity is linked with the generation of hip flexion moment and act as a postural muscle during upright sitting, as it is activated in a lordotic upright sitting posture and not activated in a slumped sitting posture. The global resultant of the psoas activation is inclined to increase lordosis and, hence, actively engage anterior longitudinal ligament support (Santaguida and McGill, 1995). Santaguida & McGill (1995) suggested that the architecture of the psoas is mechanically oriented to flex the hip. They also showed that the psoas muscle creates considerable axial compression throughout the lumbar spine and anterior shear at L4/L5 and L5/S1. Juker et al. (1998) showed that the highest muscle activity they found for this muscle was during maximal isometric hip flexion (hip at 90 °) in standing posture and pushing down the flexed thigh. It also flexes the femur and rotates it outwards. Psoas activation will create spinal stiffness (Janevic et al., 1991) increase intradiscal pressure when it is contracted (Nachemson, 1966) and resist shear forces (Santaguida and McGill, 1995). Some researchers suggest that the primary role of this muscle is to create lumbar spine stability and hip stability by pulling the femoral head into the acetabulum (Yoshio et al., 2002).



Figure 1. The lumbar spine, psoas and iliacus muscles (Netter F.H., 2003).

2.2 Stability

2.2.1 Stability of the Spine

Spinal stability is developed to limit inter-vertebral motions that can damage to structures of the spine (ie. neural elements, ligaments, joint capsules, annular fibres and endplates). Since the spine has high levels of nociceptors (Wyke, 1970), this damage can cause pain (Panjabi, 1992a).

There have been numerous definitions of spine stability. One definition of spinal stability commonly used by surgeons and engineers relates to mechanical stability, which relates to the ability of a loaded structure to maintain static equilibrium even with fluctuations around the equilibrium position. Once mechanical stability has been compromised, a small change of the position can cause buckling and the structure will move away from equilibrium (Bergmark, 1989). In other words, mechanically, a system can be considered to be either stable or unstable.

In contrast, the concept of clinical stability relates to the ability of a loaded spine to limit displacement so there is no damage or irritation to the spinal cord and nerve roots or the passive spinal tissues (ligaments, discs, facets) such that pain can be avoided. Damage to the spine compromises its' mechanical function and stability potential (White and Panjabi, 1978). However, by definition, the degree of clinical stability is often placed on a continuous scale. This signifies that each individual's spine is unique and their stability is located at a unique location on the stability scale. Therefore, it is the goal of the clinician to constantly promote their patient further up the stability spectrum, continuously moving towards a more stable condition.

Panjabi (1992a) divided the spinal stabilizing system into three subsystems; 1) passive, 2) active and 3) neural. The passive musculoskeletal subsystem consists of the vertebrae, facet articulations, intervertebral discs, spinal ligaments, joint capsules and the passive mechanical properties of the muscles. It is called the passive subsystem because it does not supply energy to the system. This aspect of the three-tier system helps maintain stability near the end ranges of motion. The active musculoskeletal subsystem are muscles. This system produces forces that help keep the system stable and avoids the end ranges of motion. The neural feedback system has proprioceptive receptors in ligaments, tendons and muscles along with the nervous control system. This control system takes information from the other two subsystems, processes the information and responds by sending signals to maintain spinal stability.

These interdependent systems work together to provide stability of the spine (Panjabi, 1992a). It has been proposed that dysfunction to any part of the spinal stabilizing system can

compromise the stability of the spine. As a result, injury to elements within the system can occur and possibly create pain, muscles spasm, atrophy, tears, degeneration and fatigue (Panjabi, 1992a).

To theoretically decrease the body's susceptibility to injury, the three subsystems work together to create a compensatory co-contraction mechanism. Perturbations to the system create an anticipatory co-activation of trunk muscles that is used to increase trunk stiffness (Gardner-Morse and Stokes, 1998). As part of this co-contraction, trunk antagonist muscle activation is commonly observed (Andersson *et al.*, 1977). A small amount of antagonist activation is necessary to maintain spine stability (Cholewicki *et al.*, 1997).

2.2.2 Trunk Muscles and Spine Stability

The lumbar spine is inherently unstable, particularly around the neutral zone (Panjabi, 1992b). The neutral zone is a region of intervertebral motion around the neutral posture where little resistance is offered by the passive spinal column (Panjabi, 1992b). The control of this feature, and the contribution of the surround muscles, are of paramount importance. Muscles can provide stability to a joint through their stiffness and the provide stiffness through their passive and active biomechanical properties (Cholewicki and McGill, 1995b; Bergmark, 1989). Co-activation of muscles surrounding the trunk can provide stability even under very high loading conditions (Cholewicki and McGill, 1996). Kumar *et al.* (1996) quantified the patterns of muscle activity during reciprocal axial rotations by healthy subjects against no resistance. They found that the agonists contributed 65% and the antagonists contributed 35% the total muscle activity and stability of the spine.

Historically, Bergmark's (1989) biomechanical model was one of the first complex representations of spinal stability. He compartmentalized muscles to act as either `local' or

global' stabilizers. In Bergmark's biomechanical model the `local' system is where those with attachments to the lumbar vertebrae. These muscles influence inter-segmental control of the spine. The psoas muscle was not included in the local system, even with its segmental attachments. In contrast, the `global' systems are muscles which transfer the load directly between the thoracic cage and the pelvis and are suitable for control of external forces acting on the spine (Figure 2). These muscles are thought to control spinal orientation. With its mechanical role, the psoas could also be considered a global muscle as it runs from the lumbar spine to the femur.



Figure 2. The global system is formed by the muscles and the intra-abdominal pressure. ESg – global erector spinae muscles, IO – internal oblique muscle, EO – external oblique muscle, RA – rectus abdominis muscles. Not shown is the intra-abdominal pressure, quadratus lumborum and the lumbar spine (Bergmark, 1989).

The lumbar multifidus provides up to two thirds of the control of inter-segmental motion in certain directions (Wilke *et al.*, 1995). This muscle would be considered a 'local' spinal stabilizer. However, this muscle has its limitations as it does not contribute much to the control of lumbar rotation (Wilke *et al.*, 1995). The TrA may contribute to inter-segmental stability, in a

general and non-direction specific manner, through either fascial tensioning or generation of intra-abdominal pressure (Tesh *et al.*, 1987), or a combination of both (Hodges and Richardson, 1998). Although TrA was not considered in Bergmark's model, the evidence is consistent with the classification of TrA in the `local' group. The interspinal and the intertransverse muscles are two other muscles that are considered part of the local system.

The muscles that are categorized in the global system are the erector spinae muscles, the internal and external obliques, the rectus abdominal muscles and the lateral parts of the quadratus lumborum muscles. The psoas and latissimus dorsi muscles were not included in Bermark's model.

In contrast to Bergmark's model, Cholewicki & VanVliet IV (2002) found that all trunk muscles contribute to spine stability and that it depends on the state of the system at that point in time. They suggest that the "local" and "global" system of stability proposed by Bergmark is incorrect and that a muscle's contribution to spinal stability depends on many variables (joint properties, load, loading conditions, posture etc.) applied to the state of the entire system. Cholewicki & Van Vliet IV (2002) did not find a great difference in spine stabilization of the inter-segmental muscles compared to the multi-segmental muscles. The co-activation of agonistic and antagonistic muscle groups has been shown to stiffen and stabilize the lumbar spine (Quint *et al.*, 1998; Cholewicki and McGill, 1996; Bergmark, 1989; Radebold *et al.*, 2000). The continued contraction of agonistic muscles in the low back pain group may promote an increase in joint stability. This may serve as a compensatory mechanism to protect the individual from pain and stall the progression of damage to the spine.

Penning (2000) has hypothesized that the psoas may also serve to stabilize the lumbar spine (LS) in an upright stance. Nachemson (1968) suggested that the osseo-ligamentous of the

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LS, in the upright position, must be stabilized by extrinsic factors such as the psoas. The psoas is positioned to contribute to the prevention of spine buckling and to the control of lordosis and pelvic tilt through its femur attachment (Hadjipavlou *et al.*, 1996).



Figure 3. Comparison of experimental specific pull and psoas orientation. The general direction of the psoas major corresponds well with the experimental direction of specific pull (Penning, 2000).

2.2.3 Fascia and Spine Stability

The fascial system has also been shown to contribute towards spinal stability. It has been shown in one biomechanical study of 17 lumbar segments, from 9 unembalmed cadavers, that tension on the lumbar fasciae simulating moderate contraction of TrA affects increases spinal stiffness and may decrease inter-segmental motion (Barker *et al.*, 2004).

2.2.4 Neutral Zone and Stability

The neutral zone is a theory proposed by Panjabi (1992b). It involves the range of physiological intervertebral motion, measured from the neutral position, within which the spinal motion is produced with a minimal internal resistance (Panjabi, 1992b). Panjabi has also described the

neutral zone as the zone of high flexibility or laxity. When the spine is in the neutral zone, there is minimal amount of internal stresses in the spinal column and minimal muscular effort exerted in this range (Panjabi, 1992b). The elastic zone extends from the end of the neutral zone to the physiological limits. This region has a higher stiffness and the two zones combined are considered the physiological ROM of a joint (Panjabi, 1992b).

The neutral zone is hypothesized to be affected by resting muscle tone. Theoretically, muscles have the potential to decrease the neutral zone and minimize degeneration or trauma to the spine (Panjabi, 1992b). It has been shown by Wilke *et al.* (1995) that simulated muscle forces around the trunk strongly influence load-deformation by decreasing the range of motion and the neutral zone of the motion segments.

2.2.5 Clinical Instability

Clinical instability is defined by Panjabi (1992b) as: "...a significant decrease in the capacity of the stabilizing system of the spine to maintain the intervertebral neutral zones within the physiological limits so that there is no neurological dysfunction, no major deformity, and no incapacitating pain." A diagrammatic representation of the neutral zone has been illustrated by Panjabi (Panjabi, 1992b). It has an upper limit which is represented by micro deformations of soft tissues causing pain or the stretching and compression of the neural elements due to deformation of the spinal column (Panjabi, 1992b). It also has a lower limit which is described as excessive muscular effort causing muscle fatigue (Panjabi, 1992b). An injury may prevent an individual's ability to maintain their intervertebral neutral zone and, in turn, may lead to low back pain and further injury.



Figure 4. Neutral zone size is a function of passive (spinal column) and active (spinal muscles) components of the spinal stabilizing system. Point P represents the normal value of the neutral zone for an individual for a particular spinal motion. An injury or an increase in the neutral zone causes point P to move up and down the on the surface (line a). An increase or decrease in muscle function causes point P to move along the b line (Panjabi, 1992b).

Low back pain during benign activities, requiring minimal or submaximal efforts can cause spinal buckling (segmental hypermobility) (Preuss and Fung, 2005). Spine buckling has been interchangeably referred to as clinical instability. Researchers have not been able to elucidate a cause-effect relationship for dynamic clinical instability. However, there has been numerous clinical reports of spine buckling in individuals with low back injuries related to simple tasks such as picking up a pencil from the floor (McGill, 2002; Cholewicki and McGill, 1996). It has been proposed that it is more like a chain of events where each link must be in place for spinal buckling to occur. This theory is thought to occur due to loss of stability at a segment and transient loss of coordination or control of one or more inter-segmental muscles (Preuss and Fung, 2005). It has been theorized that error of the central nervous system and spine buckling can occur at large and light loads. Modelling studies have found that inactivity of the local, inter-segmental musculature will lead to instability and buckling at the affected segment (Cholewicki and McGill, 1996). This is similar to what was observed using video fluoroscopy under strenuous conditions (Cholewicki and McGill, 1996). Inappropriate levels of muscle force and stiffness at a given spine segment can compromises the segmental spine stability (McGill, 2002). Compromised stability may lead to transient intersegmental buckling and excess ROM and loading of the surrounding soft tissues (i.e. ligaments, discs) (Preuss and Fung, 2005).

The diagnosis of clinical instability has been reported in the literature (O'Sullivan *et al.*, 1997). Questionnaire data gathered by patients diagnosed with lumbar clinical instability showed that half of the subjects reported back pain after a single event. The other half presented with back pain gradually after many minor traumatic incidents (O'Sullivan *et al.*, 1997). The descriptors of the back pain by these patients included: recurrent (70%), constant (55%), `catching' (45%), `locking' (20%), `giving way' (20%) or accompanied by a feeling of `instability' (35%) (O'Sullivan *et al.*, 1997).

2.3 Perturbations to the Spine

Perturbations to the spine, expected or unexpected, is used to study spinal stability. Perturbations are forces or loads applied to the spine and trigger the central nervous system (CNS) to respond and restore the body to postural equilibrium. Spinal equilibrium or spinal stability can be preserved in two ways: pre-activated muscles in the spine that are preset through preparatory, feed-forward muscle contraction (Hodges and Richardson, 1997b) or through

afferent feedback that causes the trunk muscles to respond to perturbations and create muscular force and stiffness (Cresswell *et al.*, 1994).

2.3.1 Expected and Unexpected Loading and Unloading Tasks of the Spine

Investigating the recruitment of trunk muscles in a predictable task is one method to examine spinal stability. This can be achieved by the looking at spinal control associated with limb movement. The body is altered with limb movement and, in turn, reactive forces are imposed on the body that are equal in magnitude, but opposite in direction, to those producing the movement (Zattara and Bouisset, 1988).

There have also been numerous studies investigating unexpected perturbations on the spine. Marras *et al.* (1987) was one of the first research groups to look at sudden unexpected loading and the body's muscular forces upon the trunk. Twelve male subjects were asked to hold a box in a static lift position while masses ranging from 2.27 to 9.07 kg were dropped into the box from a constant height. Under some conditions, the subject was allowed to observe the weight drop, while in other conditions (unexpected) the subjects were deprived of visual and auditory cues during the drop. Peak and mean muscle forces were considerably higher in the unexpected condition compared to the expected condition. Peak muscle forces in the unexpected condition were on average 70% greater.

Cresswell *et al.* (1994) examined both unexpected and expected perturbations on trunk muscle activity and intra-abdominal pressure by adding a weight to a harness over the shoulders. They showed that muscle pre-activation and increased intra-abdominal pressure (IAP) were utilized as stabilizing strategies to brace the spine prior to any perturbation. Interestingly, they found that, during the unexpected trunk flexion, TrA was active prior to the erector spinae (ERS) by an average of 24 milliseconds. The authors of this study suggested that increasing IAP and

recruiting the TrA is a strategy used by individuals to increase spine stability by making the entire segment more rigid.

The activation of the TrA is affected by variations in force magnitude and speed. Rapid and intermediate limb movements showed a feed-forward activation of the TrA. However, slow movements did not produce a feed forward activation of the TrA (Hodges and Richardson, 1997c). In addition, they found that the onset of TrA activity precedes that of deltoid by approximately 30 milliseconds (Hodges and Richardson, 1997b). During leg motions, greater reactive magnitude forces, activation of TrA precedes that of deltoid by more than 100 milliseconds (Hodges and Richardson, 1997a).

Lavender *et al.* (1993a) looked at task experience and expectancy in the development of preparatory strategies to stabilize the spine, minimize spinal loading and minimize postural disturbances. He found that subjects developed preparatory strategies to handle the periodic sudden loading, decrease the destabilization of the torso and reduce the muscle forces compressing the spine during loading. This preparatory response strategy allowed the subject to deal with the sudden loading and protect the body from the forces exerted on the spine.

van Dieen & de Looze (1999) studied anticipatory trunk muscle activity patterns of familiar and unfamiliar mass locations. The subjects were given a warning signal prior to lifting a weighted box in an expected and unexpected trial. The reaction time of the subjects, in unexpected trial (mean = 274 ms), tended to be longer compared to the expected trial (mean = 231 ms). They found anticipatory muscle onset for the extensor group. There was no anticipatory muscle activity for the flexor muscle group. In comparing loading situations with regards to mass location, during the trials where the mass was in an unfamiliar location, the subjects activated both sides of their trunk extensors equally. However, when the mass location was known, the

subject selectively recruited their trunk extensor muscle activity prior to loading. This study demonstrated that anticipatory control of trunk muscles appears to be specifically tuned to counteract expected perturbations. In unknown loading situations, stiffness of the spine is increased by bilateral activity.

One series of studies performed by Hodges & Richardson (1997c; 1997a; 1997b) looked at the activation of the abdominal and ERS muscles with unilateral arm (Figure 5) and leg movements. The results from these studies showed that the TrA was consistently the first muscle activated. The authors speculated that the TrA must be pre-programmed by the CNS and is contributory to developing spinal stability in response to perturbations. These results provide evidence that the CNS will react to perturbations to spinal stability by initiating preparatory motion of the spine to `dampen' the forces rather than simply making the trunk rigid.



Figure 5. Recruitment of the abdominal muscles (TrA – transverse abdominis, OI – oblique internus abdominis, OE – oblique externus abdominis, RA – rectus abdominis and ES – erector spinae). (A) Electromyography (EMG) set up. (B) Muscle activity of TrA prior to that of deltoid.

(C) Mean time of EMG onset of each trunk muscle relative to that of deltoid for upper limb movement in various directions (Hodges and Richardson, 1997c).

Thomas *et al.* (1998) investigated both expected/unexpected and asymmetric/symmetric loads applied to the torso using EMG to study the activation of the trunk muscles prior to loading, trunk stiffness and peak muscle activity. They found that, when the applied load was expected, the peak latencies of the posterior trunk muscles always preceded the peak latencies of the anterior trunk muscles. The difference in the latency periods in the unexpected loading conditions, with the exception of the left longissimus thoracis, was so small that the peak response of the trunk extensors and flexors appeared to occur simultaneously. Thomas *et al.* (1998) suggested that the muscle group created a preparatory stabilizing strategies that could potentially contribute to trunk stabilization following the loading event.

van der Burg (2000) investigated unexpected and expected lateral mass placement in lifting. Their study looked at spine loading while subjects lifted a crate with known and unknown weight placements. In the unexpected condition, the subject start to exert a net lateral bending moment later in time than in the expected condition. The unexpected condition also created a lower stiffness and a lower moment compared to the expected condition. Since the total muscle force is lower and there is an increased angular excursion, the authors concluded that there was an increased risk of developing spinal pathology due to decreased lumbar stability.

In testing expected and unexpected perturbations many authors examine trunk muscle activity during loading. Interestingly, Brown *et al.* (2003) examined trunk muscle activity of the unloading phase of a 6.8 kg box in three testing situations: (1) subject initiated perturbation, (2) known experimenter initiated perturbation, and (3) unknown experimenter initiated perturbation. They examined the baseline, the anticipatory responses and the post unloading responses of
selected trunk and leg muscles. It was found that, as timing uncertainty increased, there was a decreased ability to make anticipatory adjustments to the upcoming unloading perturbation. As a result, the subjects in the unknown perturbation situations increased their trunk muscle responses. It was hypothesized that the subjects in these unknown situations do not have enough information to exert precise muscular activation to stabilize the spine. Furthermore, anticipatory muscle activity increased as the knowledge of the unloading timing decreased. Therefore, preparatory adjustments were used to reduce the overall postural movement to the body and increase spinal stability.

Grondin & Potvin (2008) investigated fatigued trunk muscle responses to sudden loading of fifteen female subjects. The subjects received sudden loads to the hands, at both known and unknown times. Pre-activation was not shown to be significant in any of the testing situations. However, it was found that subjects in the unexpected condition exerted greater forces compared with the other groups. It was hypothesized that unexpected conditions do not give the subject accurate enough information to give precise muscle activation to stabilize the trunk. The authors from this study hypothesized that preparations in this condition must have taken place prior to the anticipated perturbations. The other important finding in this study was that there was heightened baseline activity with fatigue which implies increased spinal stiffness. Therefore, the spine, in fatigued situations, attempts to increase co-contraction to maintain stability.

2.3.2 Expected and Unexpected Loading of the Spine in Subjects with Back Pain

Impaired motor control of the trunk has been suggested as one of the possible mechanisms related to individuals with low back pain (Panjabi, 1992a; Panjabi, 1992b; Cholewicki and McGill, 1996). Buckling instability cannot be provoked, experimentally, by a perturbation (Stokes *et al.*, 2006). Since it is thought that people with LBP might respond differently to the

anticipation of the perturbation, the perturbation itself or the unloading response of a perturbation, the onset of muscle activity has been tested with sudden trunk loading in subjects with low back pain.

Magnusson *et al.* (Magnusson *et al.*, 1996) and Wilder *et al.* (Wilder *et al.*, 1996) demonstrated that these LBP subjects had delayed reaction time of the erector spinae group compared with healthy control subjects. Hodges *et al.* (1997c; 1997a; 1997b) have extensively researched the contribution towards spine stability and have suggested that the delayed onset of the TrA in subjects with LBP is due to a deficient motor control system.

Radebold *et al.* (2000) performed sudden trunk-loading experiments to measure the response latencies of 12 major trunk muscles in patients with chronic LBP (Figure 6). They unloaded the spine by unexpectedly releasing a cable that resisted the subjects' isometric exertions in 3 planes (flexion, extension and lateral bending). Overall, the subjects responded to the load release with longer reaction times than did healthy control subjects. Healthy subjects shut-off their agonistic muscles (with a latency of 53 msec) before the switch-on of antagonistic muscles (latency = 70 msec). Patients had longer muscle reaction times for muscles shutting off (70 msec) and switching on (83 msec). Furthermore, the recruitment pattern of individual muscles was relatively homogeneous in the healthy control group, whereas the LBP pain subjects showed large variability. Healthy subjects shut off most of their agonistic muscles and subsequently activated almost all of their antagonists. The chronic low back pain subjects. The subjects with LBP were more likely to co-contract their agonistic and antagonists in response to the load release.



Figure 6. Quick-release perturbation apparatus (Radebold et al., 2000).

Cholewicki *et al.* (2002) examined impaired neuromuscular function in athletes with and without a recent history of acute low back injury. Their study included 17 subjects who recently experienced a low back injury along with 17 control subjects. All the subjects were pain free at the time of the study and had returned to athletic competition. A sudden perturbation was imposed on the lumbar spine after each subject exerted isometric trunk holds in flexion, extension, and left and right lateral bending. The authors in this study found that the recent LBP subjects shut off fewer muscles, with an increased latency compared to the control subjects. Did the muscle dysfunction occur prior to or after the onset of the low back pain? This study demonstrated that neuromuscular deficiencies remain in subjects that have recently experienced LBP.

A prospective study by Cholewicki *et al.* (2005) was performed, with a 2- to 3-year follow-up, to determine whether delayed muscle reflex responses to sudden trunk loading are a

result of, or a risk factor for, sustaining a low back injury. A force was applied to the trunk of college athletes in flexion, extension, and lateral bending. Their muscle reflex latencies were measured. Using regression analysis, the authors were able to correctly predict 74% of low back injuries during the follow-up using a combination of three variables: (1) history of low back injury, (2) body weight, and (3) latency of muscles shutting off during flexion and lateral bending load releases. They discovered that the odds of sustaining LBP increased 2.8-fold when a history of LBP was present. They also learned that delayed muscle reflex response latencies have a significant influence on predicting future low back injuries.

Stokes *et al.* (2006) also examined the role of muscle activation and responses to force perturbations in persons with and without a history of low back pain. Subjects were tested while seated in an apparatus with the pelvis immobilized. Resistance was provided by a horizontal cable secured to the thorax to one of five points on a wall. Comparing a LBP group to an asymptomatic group, these investigators studied EMG in a ramped effort task. They found that the LBP subjects provided substantial muscle activation prior to perturbations in an attempt to stiffen and stabilize the trunk. The largest limitation associated with this study is that the maximum efforts generated in the maximum effort trials, averaged over subjects and angles, was 575 N for the healthy group and 403 N the LBP subjects. The authors are unsure if this is due to pain inhibition and a lack of motivation or if it is due to anatomical differences.

2.4 Muscle Dysfunction

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2.4.1 Muscle Dysfunction and Low Back Pain

The importance of individual flexibility and muscle weakness has been suggested to be an important determining factor in those that develop low back pain (Janda, 1964; Janda, 1978; Sahrmann, 1992; Hultman *et al.*, 1993). One symptom that many low back pain patients

experience is increased muscle tone in the area of pain (Travell J., 1952). According to Travell & Rinzler (1952), myofascial trigger points are small hypersensitive regions within a muscle from which impulses bombard the central nervous system and give rise to referred pain. A trigger point in a skeletal muscle is identified by a localized deep tenderness in a palpably firm band of muscle. Deep palpation provokes a positive 'jump sign' as an indicator of increased irritability of the shortened muscle band. A second type of increased muscle tone refers to muscle hypertonicity (spasm) due to an uncoordinated muscle contraction as a result of the impaired function of the interneuron on the spinal segmental level (Janda, 1991). It has been defined by Emre (1988) as "involuntary and inappropriate, reversible, prolonged bracing of a muscle or group of muscles, attributable to over-activity of motor units or changes of excitability of muscle fibres." A third type of increased muscle tone refers to the overuse muscle tightness syndrome. This has been described with changed elasticity of the muscle and usually described as muscle tightness (Janda, 1991). A fourth type of increased muscle tone has been described as a response to pain irritation (Janda, 1991). Finally, dysfunction of the limbic system can also create increased muscle tone (Schneider, 1995).

Clinically speaking, the tendency of some muscles to develop tightness or weakness is well published by Vladimir Janda (Janda, 1964; Janda, 1986; Janda, 1991). Sherrington's law (1947) states that tight muscles have an inhibitory influence on their antagonists (Janda, 1978). Therefore, tight muscles tend to be stuck in a vicious cycle: during training, or postural shortening of a muscle and/or prolonged overuse, the muscle will get shorter, stronger and readily activated (Sahrmann, 1987). Eventually, the continued muscle tightness will lead to muscle strength decreases. Therefore, these tight muscles are weak and require lengthening to restore proper muscle strength (Sahrmann, 1987).

2.4.2 Therapy for Muscle Dysfunction

One widely used therapy for lengthening a tensioned muscle is called Active Release Technique[®] (A.R.T[®]). The originator of A.R.T[®], Michael Leahy, claims that it is a therapy that targets the muscle and fascial systems and promotes flexibility and mobility of the body's connective tissues (George *et al.*, 2006). Leahy suggests that it can be used to mobilize fibrous adhesions, and to reduce the severity and sensitivity of scarring caused by injury or surgery (George *et al.*, 2006). Leahy (1995) proposes that this type of therapy releases the adhesions between the musculo-fascial interface and provides functional improvement sufficient to enhance healing and performance.

This therapy involves placing the muscle of interest in a shortened position, applying a light tension to a specific area of the muscle and lengthening of the muscle while maintaining tension in opposing direction of the muscle lengthening direction. The muscle is subsequently lengthened while the therapist maintains tension in the opposing direction. Unfortunately, there has been little research to support the mechanisms or effectiveness of A.R.T[®]. However, there is some evidence that A.R.T[®] has been successful in treating trigger thumb, lateral epicondylosis and hamstring flexibility (George *et al.*, 2006; Howitt, 2006; Howitt *et al.*, 2006). However, the author is aware of no published research to support the use of A.R.T[®] for low back pain patients.

2.5 The Iliopsoas Complex

2.5.1 Electromyography of the Iliopsoas Complex

Psoas is relatively inaccessible to electromyography surface recording, but has been recorded intramuscularly with wire electrodes (Andersson *et al.*, 1995; Andersson *et al.*, 1997). Andersson *et al.* (1995) used thin intramuscular wire electrodes inserted under guidance of high-resolution ultrasound to record myoelectric activity simultaneously from the psoas and iliacus for subjects

in the lying, seated and standing positions. McGill *et al.* (1996) used strategically placed surface electrodes to act as surrogates for the psoas and quadratus lumborum. Obviously, indwelling electrodes would have been more acute but McGill *et al.* (1996) suggests this method is an alternative to indwelling electrode placement.



Figure 7. Examples of various tasks that subjects performed while recording EMG activity of the psoas and iliacus muscles.

2.5.2 The Iliopsoas Complex and Spine Stability

The iliopsoas muscle is one muscle that simultaneously contributes to stability and movement of the trunk, pelvis and leg. The iliopsoas is a complex of particular interest for low back pain and rehabilitation due to its comprehensive nature spanning from the thoracolumbar region, across the lumbar spine and pelvis, to the femur attachment. However, much of the function of this muscle complex is largely unknown, mainly due to the relative inaccessibility of the muscle complex for EMG recordings (Andersson *et al.*, 1995).

It is important to understand the functions and limitations of the lumbar spine during loading tasks and its influence on spine stability. Spine stability can be compromised when there is excess loading of tissue tolerance during everyday tasks (McGill and Norman, 1986; Cholewicki et al., 1991). The spine's integrity can also be compromised during extremely light lifting tasks, such as lifting a pencil (Cholewicki and McGill, 1996). If the neuromuscular system is compromised in any way, even the smallest external loads can create problems for individuals with and without low back pain. Therefore, mechanical stability of the spinal system must be of concern at all levels of external loading to avoid inter-segmental buckling. Nachemson (1968) is one of the first researchers to propose that spinal stability required psoas activity even though it can only produce small moments in the sagittal plane (Santaguida and McGill, 1995). In most cases, the stabilizing potential of the psoas has bee attributed to the spinal compression it produces. Cholewicki & McGill (1996) suggest that compression from the psoas will create segmental stiffness. They used a biomechanical model of the lumbar spine to estimate the lumbar spine stability. Individual muscle forces, their associated stiffness estimated from the EMGassisted optimization algorithm and external forces were used for calculating the relative stability index of the lumbar spine for three subjects. They found that there is a stability safety margin during tasks that demand a high muscular effort and the psoas is one of the important muscles that assists in developing spinal stiffness. In addition, bilateral contraction of the psoas majors provides equal and opposite moments about the lateral bend and axial rotation. These equal and opposite muscle actions have been described to act like guy wires (i.e. psoas major muscles) to stabilize the mast (i.e. the lumbar spine) during various movements such as lifting (Santaguida and McGill, 1995). The psoas major has been described extensively by Bogduk et al. (1992). The fascicular anatomy of the psoas major was determined by dissection in three cadavers. They

examined its actions on the lumbar spine in the sagittal plane. They modelled the lumbar spine in a neutral spine position, flexed position, and extended position with ten adult males. According to Bogduk, the psoas serves as a major compressor of the spine due to the action of the short moment arms that the fascicles exert as they pass near the corresponding flexion-extension centres. The large compressive forces orient the spine into lordosis and shear the L5/S1 level.

Andersson *et al.* (1995) also looked at the relationship of the iliopsoas muscle towards compressive spine stability. His research team took the psoas and iliacus muscle activation patterns of 7 subjects doing a variety of tasks in standing, sitting and lying positions. The psoas and iliacus muscles, under most conditions, were shown to have a common activation pattern. Interestingly, the authors in this study found that the iliopsoas complex had stabilizing effects on the lumbar spine. They found that muscle contraction of the iliacus stabilized the pelvis in contralateral hip extension during standing and psoas contraction stabilized the spine during contralateral loading situations in the frontal plane.

The psoas muscle has been shown to be active during leg lifts in a supine position and during sitting with a lordotic curve in lumbar spine (Andersson *et al.*, 1995). On the other hand, the iliopsoas complex was silent, and a lower disc pressure has been found, during sitting with the back in a relaxed kyphotic or forward flexed position. These results are consistent with another study by the same research group (Andersson *et al.*, 1974). The compressive force on the lumbar spine could possibly contribute towards spine stability. While the psoas contributes towards spinal stability, too much compression can have a detrimental effect on the spine's health. Juker *et al.* (1998) confirmed these observations and suggested that relatively low psoas muscle activity might be enough to create sufficient stiffness of the lumbar spine for demanding activities.

Bogduk *et al.* (1992) described the psoas as having two different actions. The lowest fascicles of the psoas major flex the lumbar spine and the upper fascicles extend the lumbar spine. Penning (2000) also found that the upper portion of the psoas actively pulls the upright lumbar spine into more lordosis and the lower portion flexes the spine. Penning used a cadaver model with vertically placed elastic metal strips modelled into a lordotic configuration to imitate the lumber spine. Penning also suggested that, since the psoas has attachments at each lumbar vertebra, it has a second function to passively stabilizing the lumbar spine segmentally. Penning suggests that each individual fascicle tightens to stabilize the spine in a lordotic position.

The psoas has been show to create stability at low levels of exertion. Kimura *et al.* (1991) demonstrated that the psoas muscle has been shown to contain muscle fibres that act in tonic contraction. The minimal activity of the muscle during upright standing contributes to the stabilizing effect on the spine in any given degree of lumbar spine lordosis (Hadjipavlou *et al.*, 1996; Nachemson, 1966). In upright standing, the segments of the lumbar spine are aligned with the line of gravity. This position creates minimal moments around the trunk and therefore the spine is stabilized with minimal muscular energy expended (Penning, 2000). Penning emphasized that the stabilizing effect of the psoas is based on the assumption that both sides are efficiently functioning to stabilize the lumbar spine in the frontal plane with efficient energy expenditure.

During walking and running, the psoas has a stabilizing effect on the spine (Andersson *et al.*, 1997). Intramuscular EMG the psoas during these activities demonstrates two spurts of activity: the first is during the onset of ipsilateral hip flexion and the second is related to control and stabilization of the movements of the trunk in the frontal plane (Andersson *et al.*, 1997).

The psoas has been shown to contribute to intersegmental stiffness that is required for spinal stability (Panjabi et al., 1989; Jemmett et al., 2004; Wilke et al., 1995; Quint et al., 1998; Penning, 2000). Panjabi et al. (1989) performed an in vitro experiment to investigate the effect of simulated intersegmental muscle forces on spinal instability. Intact and sequentially injured lumbar functional spinal units were subjected to three-dimensional biomechanical tests with increasing muscle forces. The muscle forces applied to the spinous process in the form of two equal and symmetrical vectors directed laterally, anteriorly, and inferiorly. They found that the action of the intersegmental muscle forces is to maintain or decrease intervertebral motions, with the exception of the flexion range of motion. They also were able to determine that the ability of an individual to stay within the neutral zone is a better indicator of spinal stability compared to range of motion. They concluded that the action of the intersegmental muscle forces is to maintain or decrease intervertebral motions after injury. The exception of this conclusion was in the flexion ROM, which increased with the application of muscle forces. In addition to the work performed by Panjabi et al., (1989) Wilke et al., (1995) demonstrated that the spinal muscles, including the psoas, play a crucial role in decreasing the range of motion and neutral zone by lowering the segmental motion. Quint et al. (1998) modeled the effects of co-activation of psoas and multifidus muscles on L4-L5 mobility and found that it decreased the range of motion and, conversely, increased spine stability by 20% during lateral bending. Experimentally, Penning (2000) was able to show that the psoas action necessitates individual tuning of separate fascicles to the generate spine stability. The findings in this study suggest that each of the psoas fascicles is able to function relatively independently of the other fascicles. Jemmett et al. (2004) performed a dissection of the lumbar spine to document the attachments of the deep vertebral muscles. Their primary goal was to demonstrate the characteristics of several muscles, including

the psoas, in the context of multi-planar segmental motion. They found that the architecture of the psoas muscle is suited to generate intersegmental stiffness across multiple planes of segmental motion. It total, these studies show that the psoas has a contributory effect on intersegmental lumbar spine stability.

In addition to joint stiffness, the psoas has been shown to provide spine stability through the motor control of the system under various spine-loading conditions (Cholewicki and VanVliet, 2002). Cholewicki & Van Vliet IV (2002) showed that the iliopsoas contributed to spine stability under a combination of loading magnitude and direction, particularly during flexion type trunk motions.

2.5.3 The Iliopsoas Complex and Low Back Pain

Lewis *et al.* (2007) developed a musculoskeletal model that showed a decrease in force contribution from the gluteal muscles, during active hip extension, and from the iliopsoas complex, during active hip flexion, would produce greater anterior hip joint force. As a result of these muscle contributions, the model predicted an increase in semimembranosis, tensor fascia lata and sartorius muscles activation. Repeatedly, over time, these changes in muscle force contributions result in an increase in the anterior hip joint force and may lead to hip pain, instability, and a tear of the acetabular labrum. It has been shown that hip pain can radiate to the low back region and may present itself as low back pain (Magora, 1975).

The psoas has been researched in several studies of patients receiving hip replacement surgery. Di Lorenzo *et al.* (2007) reported that 37 out of 100 extracapsular hip fracture surgical patients showed significant altered density in the ipsilateral psoas muscle. They showed that every hip fracture had clear tomographic evidence of fibroadipose degeneration. The authors

concluded that these psoas muscle deficiencies may be related to the persistent postextracapsular hip fracture.

Chronic psoas shortening and weakness may occur due to sleeping in the fetal position, exercise programs emphasizing repetitive hip flexion, and sedentary life style (Bachrach, 1988). Most activities of daily living and many sports activities emphasize a forward orientation, repetitive sitting and repetitive hip flexion. These repetitive activities can lead to an iliopsoas shortening if the repetitive hip flexion movements are not offset by stretching (Bachrach, 1988). Bachrach states that a patient with iliopsoas tightness may present with pain at the thoracolumbar, lower lumbar or sacroiliac area, sometimes referring pain to the knee. They state that the pain is usually located unilaterally. He also mentioned that the pain is often relieved by sitting. This is consistent with Andersson *et al.* (1995) who demonstrated that the iliopsoas is inactive when the person is in a relaxed kyphotic sitting position.

2.5.4 Imaging Studies, Iliopsoas Complex and Back Pain

Most studies have shown a decrease in cross sectional area (CSA) of the iliopsoas in individuals with LBP (Cooper *et al.*, 1992; Lee *et al.*, 1992; Parkkola *et al.*, 1993; Flicker *et al.*, 1993; Dangaria and Naesh, 1998; Barker *et al.*, 2004; Kamaz *et al.*, 2007). Interestingly, there is one study by Danneels *et al.* (2000) that showed no significant change in the CSA of the psoas muscle when comparing non-surgical chronic LBP patients and matched control subjects.

Cooper *et al.* (1992) demonstrated a significant reduction in the dimension of the ERS muscles and the psoas muscle in patients with chronic low back pain. They showed that patients that have recently developed low back pain did not demonstrate these significant reductions in size. The size of these muscles was hypothesized to contribute towards spinal instability and dysfunction.

Parkkola *et al.* (1993) looked at isometric strength and size of trunk muscles in healthy and LBP subjects. Using MRI, they found that the psoas and trunk extensor muscles were smaller in the LBP population compared with the healthy volunteers. Similarly, Flicker *et al.* (1993) also conducted an MRI study in patients with chronic low back pain. They looked at both isometric and concentric lumbar paraspinal muscle activity (psoas, multifidus, and longissimusiliocostalis) during a back extension exercise in five normal volunteers, five chronic LBP patients without surgery, and five chronic LBP patients with surgery. MRI results showed differences in lumbar paraspinal musculature in chronic LBP subjects compared to normal subjects. The study also demonstrated a decrease in the CSA of LBP patients with surgery compared with the other two groups.

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Dangaria & Naesh (1998) looked at 15 healthy volunteers and 25 patients with unilateral sciatic pain from single-level disc herniation with magnetic resonance imaging (MRI) of the lumbar spine. The cross-section area (CSA) of the psoas was recorded on both sides. There was significant reduction in the CSA of psoas muscle in patients with disc herniation on the ipsilateral side, most prominently at the level of the affected disc. However, no direct correlation between the amount of disc herniation and reduction in CSA could be demonstrated at any level.

More recently, Barker *et al.* (2004) took MRIs of fifty patients with unilateral back pain. They examined the relationship between changes in the cross-sectional area of the psoas and multifidus muscles. They found segmental atrophy in psoas major and lumbar multifidus in subjects with unilateral low back pain. Kamaz *et al*, (2007) also compared healthy and chronic LBP patients' cross-sectional area changes of the paraspinal, isolated multifidus, quadratus lumborum, psoas, and the gluteus maximus muscles. In contrast, they used CT imaging to

determine the changes in CSA. They found that the patient group had smaller CSA in the multifidus, psoas, and quadratus lumborum compared with the control group.

Interestingly, Hides *et al.* (2007) found that the CSA of the psoas muscle was unexpectantly increased in bed-rested healthy subjects. It was suggested that these MRI findings could reflect increases in muscle tone or could relate to the flexed trunk position, thus reflecting muscle shortening.

2.5.5 Pelvic Cross Syndrome

In the clinical setting, Janda (1986) and Jull (1987) have studied common muscular impairments of the lumbar spine and pelvic region and have found a common pattern of muscle impairment, characterized by decreased extensibility of hip flexor and back extensor, and weakness of abdominal and hip extensor muscles. They have termed this condition as "Pelvic Cross Syndrome" (PCS). A similar pattern of muscle dysfunction has also been described by Chaitow (2002), which he termed "Lower Crossed Syndrome." This syndrome categorizes lumbo-pelvic muscles into two categories based on their functions; phasic and postural muscles. The phasic groups. The postural muscles tend to be tight and shortened. These muscles include the back extensors, iliopsoas and hamstring muscles.

Nourbakhsh (2006) looked at the relationship between PCS, lumbar lordosis angle and LBP. They found no significant difference in the degree of lumbar lordosis in subjects with and without patterns of PCS, or in subjects with and without LBP. Their findings did not support the theory that muscle impairments in PCS lead to excessive lumbar lordosis and LBP. In spite of these findings, they did show a significant difference in the strength of abdominal and gluteal muscles between subjects with and without LBP. In addition, they also found significant

differences between subjects with and without LBP for the length of hip flexor and hamstring muscles.

2.5.6 Treatment for the Tight Iliopsoas Complex

It has been shown that individuals with LBP displayed less passive hip extension than people without LBP and this has been related to PCS (Van Dillen *et al.*, 2000). Van Dillen *et al.* (2000) provided evidence to show that, during the hip flexor length test, changing the knee joint angle in the sagittal plane, and the hip joint angle in the frontal plane, can affect the amount of passive hip extension ROM. The contribution of specific hip flexor muscles to a hip extension limitation may differ depending of the individual. Therefore, the specific hip flexor lengths are important in determining the amount of hip extension.

Based on these clinical findings, many clinicians combat low back pain by stretching the tight and shortened muscles, and then subsequently strengthen the weak muscles. Janda postulated that low back pain patients with tight hip flexors tend to have a hypoactive, hypotonic and weak gluteal muscles (Janda, 1986). Stretching treatment of the iliopsoas complex has been shown to increase hip extension, reduce pain and aid the return to normal activity for patients with low back pain (Ingber, 1989; Winters *et al.*, 2004). A.R.T[®] is a widely used therapy for lengthening a tensioned muscle such as the iliopsoas complex. Unfortunately, there has been no research to support the use of A.R.T[®] for treating low back pain patients with a tight iliopsoas complex.

2.6 Self-Efficacy

The treatment for a patient that experiences low back pain does not simply include passive therapy to control and repair structural damage. A large component that affects the progress of a patient with low back pain, involves the patients' psychosocial construct. Some of the most

relevant components include one's own beliefs, attitudes, values and behaviour modifications (Bandura, 2004). Dysfunctional beliefs about pain and management can affect the progression of the patient and their outcome. Amongst the various beliefs in the management of low back pain, self-efficacy is very important and must be carefully considered with this population. Bandura (1977) developed the concept of self-efficacy and stressed its' importance because it affects health behaviour directly and influences other determinants. Self-efficacy is the belief that one has the ability to successfully perform certain tasks or behaviours in order to produce a desired outcome (Bandura, 1977). Bandura (1977) points to four sources affecting self-efficacy; (1) Experience is an important factor in deciding a person's self-efficacy. Simply put, success raises self-efficacy and failure lowers it. Furthermore, this experience is task specific. (2) Vicarious Experience is a process of comparison between a person and someone else. If one experiences others having success with something in particular, their self-efficacy will increase; and where they see people failing, their self-efficacy will decrease. (3) Social Persuasions relate to encouragements/ discouragements. Positive persuasions increase self-efficacy and negative persuasions decrease it. (4) Physiological factors, such as aches and pains, fatigue, fear, nausea during stressful situations can markedly alter a person's self-efficacy.

The relationship of self-efficacy for various behaviours and pain, in this case more specifically back pain, has been fairly well researched in recent (Woby *et al.*, 2007; Denison *et al.*, 2004; Nicholas, 2007b; Salvetti and Pimenta, 2007; Saunders, 2004; Rapley and Fruin, 1999). Denison *et al.* (2004) studied the relationship between disability, self-efficacy, fear avoidance and pain intensity. A multiple hierarchical regression analyses showed that selfefficacy explained the largest proportion of the variance in disability scores. The results from this study suggest that one of the most significant predictors of disability is the patients' beliefs, such

as their own self-efficacy. Therefore, examination of individuals with low back pain should not solely focus on physical impairments, but should also focus on psychological factors.

Woby *et al.* (2007) performed a cross-sectional study with chronic low back pain patients. Without intervention, 102 chronic low back pain patients completed measures for pain, disability, self-efficacy and pain-related fear (fear of movement and catastrophizing). A multistep regression analyses was performed on measures including pain, disability, self-efficacy and pain-related fear. They found that greater pain-related fear and intensity will lead to lower pain self-efficacy and, in turn, greater pain and disability.

Self-efficacy is an important facet in patients dealing with pain because it affects the way a patient faces obstacles and deals with aversive experiences. Self-efficacy beliefs are important for individuals who deal with pain on a daily basis. It is important that these individuals have the expectation that they can perform a particular task and have the confidence in being able to do it. These aspects of the self-efficacy construct have been included in a pain questionnaire, called the Pain Self-Efficacy Questionnaire, and has been validated in the literature for people with back pain (Nicholas, 2007b; Jensen, 2003).

The ten items on the questionnaire reflect a wide variety of classes of activities and tasks, with indicative examples, commonly reported as problematic by patients with chronic pain. All items include mention of performing the activities despite their pain (e.g., "I can do most of the household chores (e.g., tidying-up, washing dishes), despite the pain". The questionnaire asks the respondents to rate how confident they are that they can do each of the 10 activities or tasks at present despite the pain they are experiencing. They select a number on a 7-point scale, where 0 equals "not at all confident" and 6 equals "completely confident". A total score is calculated

by summing the scores for each of the 10 items, yielding a maximum possible score of 60. Higher scores reflect stronger self-efficacy beliefs.

3.0 PURPOSE AND HYPOTHESES

3.1 Statement of Purpose

The purpose of this study was to investigate the influence of a passive myofascial therapy treatment (Active Release Technique[®] (A.R.T[®])) on various outcome measures with an asymptomatic tight hip flexor group (A-THF) (n=8) and a low back pain tight hip flexor group (LBP-THF) (n=10). These two groups were also compared with a control group (CON) (n=8) The outcome measures for this study were: 1) Trunk muscle EMG measurements monitoring unloading perturbations (Unknown Timing (UT) and Known Timing (KT)) and Unstable Standing (US) perturbations. 2) Maximum voluntary trunk flexor and extensor moments (Flex_{Max} and Ext_{Max}), 3) disability and pain measurements (RMDQ and VAS) with self-efficacy (PSEQ) evaluated as a covariate. 4) Hip extension mobility.

Due to the difficulty in isolating the psoas and iliacus muscle, this study examined a passive treatment on the two muscles: the iliopsoas complex. Through EMG, the muscles examined included the internal oblique (IO), external oblique (EO), thoracic erector spinae (TES), lumbar erector spinae (LES), multifidus (MULT) and gluteus maximus (GLUT) muscles.

3.2 Statement of Hypotheses

1) For the trunk MVE trials, prior to treatment, it is hypothesized that both the CON group and the A-THF group will have the highest Ext_{Max} and Flx_{Max} values, compared to the LBP-THF group. It is further hypothesized that the differences found between the LBP-THF treatment group and both the CON and A-THF group will become less pronounced over the course of the treatment program. Thus, it is hypothesized that MVE testing will show a statistically significant interaction (p<0.05) between the groups of subjects and the treatment time. Post hoc analysis will reveal that there will be no significantly different moments produced between the A-THF group and the CON group. Post hoc analysis will also demonstrate significantly different moments produced between the LBP-THF group and the two non-painful groups. By the end of the treatment program, LBP-THF group Ext_{Max} and Flx_{Max} values will not be significantly different from the CON and A-THF groups.

It has been shown that subjects with low back pain exhibit altered muscle activation and recruitment strategies compared with control subjects (Hodges and Richardson, 1998; Radebold *et al.*, 2000; Radebold *et al.*, 2001). More importantly, it has been shown that moments produced by low back pain patients are 40% lower than the pain-free healthy group and electromyography amplitude was 60% lower than the pain-free healthy group (Kramer *et al.*, 2005). Treatment of LBP patients has been shown to decrease pain and increase trunk muscle flexion and extension moment production (Hanrahan *et al.*, 2005). Therefore, it is hypothesized that decreasing the patients' pain through treatment will allow them to increase their flexion and extension moments produced.

2) The low back pain group will have significantly higher levels of disability and pain measures

before beginning the treatment program. It is hypothesized that, over the course of the treatment period, the RMDQ and 10-VAS scores will decrease significantly. Further, it is hypothesized that there will be a significant main effect for Time (p<0.05) that will demonstrate a decrease in disability and pain over the course of the treatment period,. Finally, it is hypothesized that the PSEQ self-efficacy score will be a significant covariate for disability and pain changes over the course of the treatment program.

It has been shown that individuals with LBP displayed less passive hip extension than people without LBP. Stretching treatment of the iliopsoas complex has been shown to increase hip extension, reduce pain and aid the return to normal activity for patients with low back pain (Ingber, 1989; Winters *et al.*, 2004). Therefore, it is expected that, by the end of the treatment program the differences found in disability and pain between the CON group and the LBP-THF treatment group will decrease significantly.

Self-efficacy plays an important role in coping with adversity (Benight and Bandura, 2004). People with greater self-efficacy can perform a particular behaviour or task and have greater confidence in being able to do that task or behaviour despite any pain they may be experiencing (Nicholas, 2007a). Therefore, it is expected that by the end of the treatment program the greatest improvements in the LBP-THF treatment group will be from the subjects with the greatest self-efficacy.

3) Measuring passive hip extension, the CON group will have significantly greater hip extension than the THF groups. At the end of the treatment period, the differences between the CON and THF groups will be less noticeable. Hip extension measurements will show a statistically significant interaction (p<0.05) between the groups of subjects and the treatment time. Post hoc analysis will reveal that there will be a significantly different hip extension measurements. Post hoc analysis will also demonstrate, after the completion of the treatment program, no significantly different measurements between the THF groups and the CON group at baseline measurement.</p>

Decreased hip flexor flexibility has been shown to have a relationship with pain and musculoskeletal impairments(Winters *et al.*, 2004). Passive and active stretching has been shown to help improve hip range of motion (Christiansen, 2008; Zakas *et al.*, 2006; Aalto *et al.*, 2005; Winters *et al.*, 2004). Therefore, one can hypothesize that after, a two week treatment period of manual stretching therapy, the subjects in the THF groups will have increased hip range of motion.

4) In the perturbation trials (UT, KT and US), prior to treatment, it is hypothesized that the control group (CON) will have the lowest baseline muscle activity and anticipatory adjustment muscle activity during known and unknown unloading and the lowest average trunk muscle activity during unstable standing, which will be exceeded by the low back pain tight hip-flexor group (LBP-THF). It is hypothesized that the differences found between the

LBP-THF treatment group and the CON group will become less pronounced over the course of the treatment program. It is hypothesized that there will be no differences between the CON group and the A-THF group.

Baseline, anticipatory adjustment during sudden unloading (UT and KT) and average trunk muscle activity during unstable standing (US)_will show a statistically significant interaction (p<0.05) between the groups of subjects and the treatment time. Post hoc analysis will reveal that, at baseline, there will be a significant difference between the CON group and the LBP-THF group, but at the completion of the four treatment sessions, anticipatory adjustment and average trunk muscle activity in the LBP-THF treatment group will show no significant difference compared with the control group.

Research has studied the effects of pain induced by the injection of hypertonic saline in the LES reported an increase of the EMG amplitude of this muscles with increasing pain intensity and a subsequent reduction in EMG amplitude with diminishing pain (Cobb et al., 1975). It has been suggested that people with back pain increase their trunk muscle amplitude to protect the spine from further damage (van Dieen et al., 2003b). Poor spine kinaesthesia in participants with low back pain have been also demonstrated in the literature (Radebold et al., 2001). It has also been shown that LBP patients have delayed onset of the gluteus maximus and transverse abdominis muscles and hypoactivity in the gluteus maximus and medius muscle (Janda, 1986; Bullock-Saxton et al., 1993; Hodges and Richardson, 1998). In addition, patients with low back pain, in contrast to healthy control subjects, have shown patterns of co-contraction and longer reaction times in trunk muscle activity responses to sudden load release (Radebold et al., 2000). In order to stabilize and protect the spine, individuals increase trunk muscle activity prior to perturbations (Andersson et al., 1995). Pre-activation is one way to protect the spine during perturbations to the spine. Janda (1986) has shown that stretching the tight agonist trunk muscles can dis-inhibit the inhibited trunk muscles.

It is hypothesized that stretching the iliopsoas will decrease the pain that the participant is experiencing; decrease the inhibited trunk muscles, decrease the preactivation amplitude of the trunk muscles (%MVE for trunk muscles) during sudden unloading, and decrease the average trunk muscle activation during unstable standing.

4.0 METHODS AND PROCEDURES

The scope of this research project involved: 1) the recruitment and screening of three different groups of subjects: (i) low back pain subjects with tight hip flexors (LBP-THF) (n = 10), (ii) asymptomatic subjects with tight hip flexors (A-THF) (n = 8), and (iii) control subjects (CON) (n = 8); 2) treatment for the tight hip flexor groups, 3) measuring outcomes (including disability, pain, MVE, passive hip extension and trunk muscle response to the three perturbations) over a 2 week passive treatment program.

4.1 Study Design

i.

The study performed was an unblinded, 3 group intervention study that involved participation in 5 sessions over a 2 week period. Both THF groups were treated identically over the course of the study. Prior to treatment, various outcome measures were recorded on Day 1, including: passive hip extension angle, palpatory pain, jump sign pain, self-reported pain scale, self-reported disability questionnaire and self-reported self-efficacy questionnaire. The subject performed 3 randomized perturbation trials and two MVE trials. After completion of one full set of outcome measurements, the subject received treatment. Subsequently, the subjects repeated another full set of outcome measures in reverse order without the palpatory pain, jump sign pain, disability and self-efficacy measures. The second, third and fourth testing days were performed with at least 24 hours of rest between sessions. During these sessions, the subjects completed the self-reported pain scale (10-cm Visual Analogue Scale), performed 2 MVE trials. Day 5 was completed within two weeks of the initial testing day. This session consisted of replicating one complete set of outcome measures from Day 1 with no treatment. The CON group completed

one testing session with one complete set of outcome measures with no treatment. Please refer to Figure 10 and 11 for a schematic diagram outlining the study design.

Specifically, the outcome measures for the study included: (1) perturbation testing (2) disability and pain outcomes, (3) MVE testing, (4) hip extension angle. Self-efficacy was measured as covariate in all participants. The trunk muscle activity for the IO, EO, TES, LES, MULT and GLUT muscles was measured through three perturbation scenarios: 1) <u>Unloading with Known Timing (KT)</u>. Subjects were exposed to unloading with KT, where they dropped a handheld load at a time of their own choosing. 2) <u>Unloading with Unknown Timing (UT)</u>. The weight that the subjects held was attached to a pulley system that was used to suddenly unload the weight from the handheld box (Figure 12). Baseline activity and anticipatory trunk muscle adjustments was monitored. 3) <u>Standing on an unstable surface</u>: Subjects stood on an unstable surface (US). A rocker board was used for the US (Figure 15). The average trunk muscle activity was examined for this condition.

Disability and pain were measured using 1) Roland-Morris Disability Questionnaire (RMDQ) (Roland and Morris, 1983) and 2) 10-cm Visual Analogue Scale (VAS)(Scott and Huskisson, 1976). Self-efficacy was measured with the Pain Self-Efficacy Questionnaire (PSEQ)(Nicholas, 2007b) on the first and fifth testing day. Self-efficacy was used as covariate in the study. In addition, maximal trunk flexor and extensor moment production was measured before and after each treatment along with the final testing session on Day 5.

4.2 Subject Information

Twenty-four male athletes, between the ages of 17-29 years old, were recruited for this study. They represented a homogeneous group with respect to age, health and fitness level. The participants were recruited by advertisements that were placed on campus and the University website. All participants were placed into three separate groups (LBP-THF, A-THF and CON). All subjects were evaluated by a doctor of chiropractic and placed in the following groups: <u>CON</u>: Healthy control subjects were defined as persons who had never experienced back pain lasting longer than 3 consecutive days. <u>LBP-THF</u>: Mechanical Low back pain subjects with episodic or constant LBP for more than 3 months and tight hip flexors. Subjects with mechanical LBP, or nonspecific LBP, had no serious underlying pathology or nerve root compromise. Furthermore, they had no structural deformities, genetic spinal disorders, or previous spinal surgery. Subjects understood that they were able to discontinue the study at anytime and were allowed to take rest periods whenever they felt it was necessary. <u>A-THF</u>: Asymptomatic subjects had tight hip flexors but did not have any LBP.

Subjects in the two tight hip flexor (THF) groups were required to test positive on the modified Thomas test to be included in the study. The following tests were performed on each subject:

The Modified Thomas Test (MTT)

The modified Thomas test, which has been shown to have good intra-rater reliability in the literature, was used test for a short iliopsoas complex. This is the only objective and reliable orthopaedic test in the literature for this purpose. The subject sat on the end of a table, rolled back on to the table while holding both knees to their chest. This ensured that the lumbar spine was flat on the table and the pelvis was in posterior rotation. The subject held the contralateral hip in maximal flexion with their arms, while the tested limb was lowered towards the floor (Figure 8). The test was considered "positive" if a short iliopsoas complex existed when the hip flexion angle was greater than 0° and knee was bent. The examiner made sure that the leg was neither abducted nor adducted. A positive Modified Thomas Test was required to be included in the THF group for this study. Three measurements were taken on both sides and the average of each side was recorded. The amount of hip flexion was measured using a goniometer placed over the greater trochanter. The goniometer has been shown to have intra-rater reliability values that fall within 4-5 degrees of each other 95% of the time (Ellis and Bruton, 2002). In addition, the goniometer has been found to be a reliable instrument for measuring hip extension flexibility (Clapis *et al.*, 2008).



Figure 8. Modified Thomas Test

Additional Participant Tests:

Passive hip extension on both sides was measured with the participant in the prone position. The leg was passively extended with the knee straight and in a 90° bent position. A goniometer placed at the greater trochanter was used to measure passive hip extension in both positions. Three measurements were taken for the straight (ProneExt.) and bent (ProneBent) knee position and the average hip extension angle was recorded.

At the commencement of the study, the subjects completed a questionnaire regarding personal data (sex, height, weight, age, medical history including low back pain characteristics, and work status), training hours per week and previous treatment procedures.

4.3 Subject Orientation

Participants that met the inclusion criteria for the experiment were given a brief orientation to the experimental protocol. Specifically, subjects were given an opportunity to practice balancing on the rocker board to ensure they were comfortable performing the task. Subjects were shown the unloading apparatus and the protocol was explained to them in detail. In addition, subjects were given the opportunity to try the unloading apparatus prior to commencement of the trials. Finally, the subjects were given a chance to try the maximum trunk flexion (Flex_{Max}) and extension moment (Ext_{Max}) protocol.

At this point, the subjects were allowed to ask questions or voice concerns that they might have had regarding the study and their participation. Only subjects that agreed to participate in the experimental protocol were included in the study. Subjects signed an approved in the study of the McMaster Research Ethics Board (Appendix A).

4.4 Experimental Protocol

4.4.1 Instrumentation and Data Acquisition

There were several devices used to collect the data for this study. The electromyography (EMG) signals was collected with customized LabView software (National Instruments, Austin, TX.) using a PC compatible computer. Analog signals obtained from each instrument were converted to digital signals using a 12-bit A/D card (National Instruments, Austin, TX.). For the unloading trials, a force gauge (MLP-500-CO, A-Tech Instruments, Scarborough, Canada) was placed in one of the handles to determine when the unloading occurred.

4.4.2 Electromyography

Six channels of surface EMG were used to measure trunk muscle activity, bilaterally. After shaving and scrubbing the EMG recording sites with isopropyl alcohol, disposable Ag-AgCl surface electrodes (Medi-trace disposable electrodes, Graphic Controls, Gananoque, ON,) with an inter-electrode distance of 3 cm, they were affixed to the skin parallel to the muscle fibre orientation over the muscle bellies of the following muscles on each side of the body. Electrode placement were taken from Goodgold (1974) and Radebold et al. (Radebold et al., 2000). The muscles examined included: external oblique (EO - approximately 15 cm lateral to the umbilicus), internal oblique (IO - approximately midway between the anterior superior iliac spine and symphysis pubis, above the inguinal ligament), thoracic erector spinae (TES - 5 cm lateral to the T9 spinous process), and lumbar erector spinae (LES - 3 cm lateral to the L3 spinous process), the gluteus maximus (GLUT - one-third from the lateral edge, on a line from the upper border of the greater trochanter of the femur to the ischial tuberosity), and lumbar multifidus (MULT - located adjacent to the spinous process of the 4th lumbar vertebrae). Surface EMG signals were processed through a differential amplifier (gain = 1000-5000, input mpedance = 10 G Ω s, 10-1000 Hz, CMRR = 115 dB at 60 Hz, Bortec, Octopus AMT-8, Calgary, AB, Canada).

4.4.3 EMG Data Normalization

Once the electrodes were placed on each participant, the appropriate electrode placement was verified by having the subjects perform manual muscle testing of each muscle and using an oscilloscope to give the researcher accurate feedback. Subsequently, the participant performed a quiet noise trial by lying on the treatment table for a 20 second period. Following the noise trial, subjects performed three $Flex_{Max}$ and Ext_{Max} efforts against resistance. For the $Flex_{Max}$ and

 Ext_{Max} , subjects performed these efforts against a padded wood plank that was attached with a metal chain to a metal beam supported by the wall. The participants exerted three maximum back extension exertions within a 20 second period and then repeated this procedure with three flexion trials. In addition, subjects sat in a sit-up position and performed 6 maximal voluntary flexion exertions against resistance within a 20 second period (2 - direct flexion, 2 - right rotation with flexion, 2 - left rotation with flexion). To obtain the GLUT MVC, the subjects lay in a prone position and extended the hip isometrically against the examiners resistance 3 times for each side. Maximum force was selected as the average of the highest peaks achieved for the three exertions. Approximately one minute was allowed for rest between each exertion. The Flex_{Max} and Ext_{Max} were repeated before and after treatment on the first testing session and during the final testing day.

4.4.4 Testing Protocols

Once subjects were familiar with the protocol, signed the consent form, completed the screening protocol (history, physical exam and group placement), completed the forms (PSEQ, VAS, RMDQ, anthropometric data), EMG electrodes were placed on the participant and normalization trials were completed. Following EMG normalization, Flex_{Max} and Ext_{Max} were tested (Figure 9). After this was completed, the three perturbation scenarios were presented to the subject (in a randomized order) both before and after the treatment in the first session and last session (Figure 10 and 11).



Figure 9. MVE apparatus: Subject performing $Flex_{Max}$ and Ext_{Max} efforts.



Figure 10. Schematic showing the timeline of testing the THF Groups on Day 1.



Figure 11. B) Schematic showing the timeline of testing the THF Groups on Day 2, 3 and 4. C) Day 5 sequence of testing for THF Groups.

<u>Unloading Perturbations</u>: Each subject held a wood platform with handles on either side using a palmar grip in a comfortable, shoulder width stance. The mass of the platform with handles was 0.6 kg. The load on top of the wood platform was a 6.3 kg (figure 11).



Figure 11. Handheld apparatus for Known Timing and Unknown Timing perturbations.

For the Known Timing (KT) condition, the subject voluntarily dropped the load. This KT procedure was completely dependent on the subject themselves and they were in full control of the unloading timing (Figure 12).



Figure 12. Known Timing unloading perturbation. Subject is shown just before dropping the load.

For the Unknown Timing (UT) condition, an unloading apparatus was used which contained one pulley and a black curtain in front of the participant. The participant stood facing the pulley system and the black curtain. For this condition, the subjects were aware that the researcher on the other side of the curtain would unload the load after a random duration between 1 and 15 seconds (Figure 14). Ten consecutive trials were collected for each timing condition.



Figure 13. Unknown Timing unloading perturbation. The subject is standing behind a black curtain while holding the load. The weighted apparatus is attached to a pulley system hanging from the ceiling. The researcher is waiting to unload the handheld apparatus.

Unstable Perturbations: The third perturbation condition had participants stand on an unstable rocker board (Forza Equipment, Spokane Valley, WA.) (Figure 15). A safety railing surrounded the subject, providing support in the event that balance was lost. Subjects were instructed to maintain balance while standing upright. Subjects performed five 20-second trials with eyes opened. A 30-second rest was given between trials. Subjects were asked to hold on to the safety railing at all times between the trials to prevent additional learning or fatigue. (figure 16). The order of presentation of the perturbation conditions was randomized for each subject.



Figure 14. Rocker board for Unstable Standing perturbations.



Figure 15. Unstable Standing perturbation apparatus.

4.5 Treatment

All treatment therapy was provided by a Doctor of Chiropractic. The treatment used was A.R.T with the goal of stretching the myofascial iliopsoas complex. A.R.T was used with the subject in the side-lying position. The targeted muscle, the iliopsoas complex, was placed in a shortened position (hip flexion). The fingers of the chiropractor were placed on the targeted treatment area. The finger contact was light compression and tension in the opposite direction of the eventual lengthening of the muscle, in this case cephaladly (toward the head). The muscle was
subsequently lengthened, moving the hip into extension until the end of the subject's range of motion, while the chiropractor maintained tension in the opposing direction (figure 17). This was considered to be one pass. Three locations were used for the myofascial release protocol including: next to navel, on the inside of the iliac crest and half way between the anterior superior iliac spine and the pubic bone (just below the inguinal ligament and medial to the Sartorius) (Cyriax J, 1982). Three passes were performed at each location. After the nine passes, the subject was asked which location provided the most benefit. The chosen location received an addition three passes. Subject received treatment on both the right and left iliopsoas complex. Subjects were required to perform 4 treatment sessions within a two week period, with at least 24 hours in-between each session.



Figure 16. Active Release Technique® (A.R.T®) of the iliopsoas complex.

4.6 Subject Data

Data from the outcome measures (palpation, hip extension angles, disability, pain and selfefficacy) were entered into a spreadsheet. Following the completion of the study, these data were grouped accordingly and stored on a PC for further analysis.

4.7 Data Analysis

4.7.1 Electromyography Data Analysis

Signal bias was quantified and removed from experimental noise trials. For each trial, all surface EMG data will be amplified 500 to 1000 times (CMRR > 80db) prior to sampling at 2100 Hz. EMG data digitally band-pass filtered (140-500 Hz, 6^{th} order), full-wave rectified and low-pass filtered using a 2nd order Butterworth filter with a frequency cut-off of 2 Hz. These data were analyzed in conjunction with the outputs from the normalization data enabling muscle activation levels to be compared to force output.

4.7.2 Unloading Task

The dependent variables for the unloading perturbation were the average EMG (%MVE) for baseline and the anticipatory adjustment for with and without the CON group. The anticipatory adjustment was defined as the pre-perturbation levels averaged over the last 15 ms prior to the unloading. Figure 18 represents a schematic diagram of the baseline and anticipatory trunk muscle activity (%MVE).



Figure 17. Schematic diagram of the determination of dependent variable: baseline and anticipatory adjustment of muscle activity.

4.7.3 Unstable Standing Task

The dependent variable for Unstable Standing was the average rectified, EMG data, normalized for MVE. This was found for each muscle using a 7 second sliding window for each 20 second trial. The coefficient of variation (CV) was calculated for each 7 s window and the window with the lowest average CV found, across all muscles, was used to calculate the representative average activation for each muscle. Since there were 5 trials, the average of all 5 trials was used in the data analysis.

4.7.4 Disability, Pain and Self-Efficacy Measures

The dependent variable for the disability and pain measures was the score of the RMDQ (Roland and Morris, 1983) and VAS(Scott and Huskisson, 1976). Self-efficacy was obtained with the

PSEQ questionnaire (Nicholas, 2007b). The disability and pain scores were tabulated individually and were compared with each other using an analysis of variance (ANOVA) and an analysis of co-variance (ANCOVA). The self-efficacy scores were used as the covariate for the ANCOVA analysis.

4.7.5 Trunk Flexor and Extensor Exertions

The dependent variable for these trials was the maximum moment produced during maximum voluntary flexion and extension exertions ($Flex_{Max}$ and Ext_{Max}). To obtain the maximum moment the subjects isometrically flexed and extended, in two separate trials, against an immovable object. The maximum Newtons obtained was recorded for each direction. The highest force attained for the $Flex_{Max}$ and Ext_{Max} was used in the data analysis.

4.7.6 Change in Hip Extension Angle

The dependent variables for this outcome measure was the change in hip extension angle using 3 different methods: (1) Modified Thomas Test, (2) passive hip extension in the prone position with the knee extended and (3) passive hip extension in the prone position with the knee bent to 90°.

4.8 Statistical Analysis

For all statistical analyses, any significant main effects or interaction effects, a Tukey's *post hoc* pairwise comparison test was performed to determine the significance of individual mean differences between the conditions. Significance was set at p < 0.05. Each of the four types of ANOVAs used are presented in Table 1.

For both the $Flex_{Max}$ and Ext_{Max} data, both a 'THF Groups' and a 'All Groups' analysis were performed. For the 'THF Groups' analysis, a 2-way (2 X 9) ANOVA was performed and the independent variables were: 1) *Subject Groups* as a between variable (LBP-THF, A-THF) and 2) *Treatment Time* as a within variable (Pre-treatment Day 1 (or Pre-D1), Post-treatment Day 1 (or Post-D1), Pre-D2, Post-D2, Pre-D3, Post-D3, Pre-D4, Post-D4, Day 5 (or D5)). For the 'All Groups' analysis a 1-way (5) ANOVA was performed and the independent variable was: 1) *Subject Groups* as a between variable (LBP-THF Pre-D1, LBP-THF D5, A-THF Pre-D1, A-THF Pre-D5 and CON). The dependent variables for both the 'THF Groups' and 'All Groups' ANOVAs were the Flex_{Max} and Ext_{Max} moment production. For each subject, the maximum trial for each session was used in the ANOVAs.

For Known Timing (KT) and Unknown Timing (UT) perturbation trials, Unstable Standing trials and the three hip extension measurement measurements (MTT, Knee Bent and Knee Extended), both a 'THF Groups' and a 'All Groups' analysis were performed. For the 'THF Groups' analysis a 3-way (2 X 3X 2) mixed ANOVA was performed and the independent variables were: 1) Subject Groups as a between variable (LBP-THF, A-THF), 2) Treatment Time as a within variable (Pre-D1, Post-D1 and D5) and 3) Side as a between variable (Rt. and Lt.). For the 'All Groups' analysis, a 2-way (5 X 2) ANOVA was performed and the independent variables were: 1) Subject Groups as a between variable (LBP-THF Pre-D1, LBP-THF D5, A-THF Pre-D1, A-THF Pre-D5 and CON) and 2) Side of Pain as a between variable (Rt. and Lt.). For both the KT and UT perturbations, the dependent variables were Baseline (BL) muscle activity and Anticipatory Adjustment (AA) muscle activity for each of the six muscles monitored. For the Unstable Standing trials, the dependent variable was average muscle activity for each of the six muscles monitored, determined over the selected time window. For the three hip extension measurements, the dependent variable was passive hip extension. In all cases, multiple trials were averaged within subjects and these averages represented the subject scores for each ANOVA.

The score for the RMDQ was out of 24. A score of 0 would indicate no disability and a score 24 would indicate complete disability. The PSEQ questionnaire score was out of 60. A score of 0 would indicate no self-efficacy and a score of 60 would indicate the highest self-efficacy that can be reported by this questionnaire. The 10-cm VAS score is a score from 0 cm to 10 cm. 0 cm would indicate no pain.

Using self-efficacy as a covariate for the RMDQ and VAS outcome measures, a 2-way (2x1) ANCOVA was performed with the independent variables being recorded for Time (n=2) and subject Group (n=1). The dependent variables were disability and pain (RMDQ and VAS). The analysis was re-run for the RMDQ and VAS outcome measure, calculating a 2-way (9x1) ANOVA with the independent variables being the recorded Time (n=9) and subject Group (n=1). The dependent variables being the recorded Time (n=9) and subject Group (n=1).

Table 1: Summary of the four types of ANOVA models used for the various dependent variables in this study.

THF Groups ANOVA

(Flexion MVE, Extension MVE)

	Day 1		Day 2		Da	y 3	Da	Doy 5	
	Before	After	Before	After	Before	After	Before	After	Days
A-THF	X	Х	X	X	Х	X	• X	X	X
LBP-THF	X	Х	X	X	Х	X	Х	X	X
CON									

THF Groups ANOVA

(KT perturbation, UT perturbation, Unstable Standing and Hip Extension with MTT, bent knee and extended knee)

			y 1	Day 2		Day 3		Da	Dou 5	
		Before	After	Before	After	Before	After	Before	After	Day 5
	A-THF	X	X							X
Right	LBP-THF	X	Х							X
	CON									
	A-THF	X	X							Х
Left	LBP-THF	X	X							X
	CON									

All Groups ANOVA

(Flexion MVE, Extension MVE)

	Day 1		Day 2		Da	у З	Da	Days	
	Before	After	Before	After	Before	After	Before	After	Day 5
A-THF	X								X
LBP-THF	X				Salata.				Х
CON	X		e ned s						

All Groups ANOVA

(KT perturbation, UT perturbation, Unstable Standing, Flexion MVE, Extension MVE

and Hip Extension with MTT, bent knee and extended knee)

-		Da	y 1	Day 2		Day 3		Da	David	
		Before	After	Before	After	Before	After	Before	After	Day 5
	A-THF	X					19. A.			х
Right	LBP-THF	X							4 A A	X
	CON	X								
	A-THF	X								Х
Left	LBP-THF	X								Х
	CON	X								

5.0 RESULTS

The results of this study are divided into four sections. The first describes the flexion and extension maximum voluntary exertions and the second will look at the questionnaire results (RMDQ and VAS). The third section will present the EMG data and the fourth will present the changes in passive hip extension.

5.1 Trunk Flexion and Extension Exertions (MVE)

The dependant variables for this analysis were the peak moments produced during maximum voluntary trunk flexion and extension exertions. The maximum trunk flexion ($Flex_{Max}$) and extension moment (Ext_{Max}) values were analyzed, over the course of the treatment period, using an All Groups ANOVA and a THF Groups ANOVA. The significant results for these two analyses are displayed in Table 2 and 3.

Table 2. THF Groups' analysis: There was a statistically significant effect of time for maximum trunk extension (Ext_{Max}) and flexion moment ($Flex_{Max}$) (p<0.05). *Post hoc* results that are statistically significant are presented with "E" for extension and "F" for flexion trials. Differences large enough to be deemed "clinically relevant" are indicated by bolded letters with an asterisk.

Trunk Extension and Flexion Trials:			28E) Z		P¢	st-Treatment				1. S.
Post Hoc Results for	THF Groups	Day 1 - Before	Day 1 - After	Day 2 - Before	Day 2 - After	Day 3 - Before	Day 3 - After	Day 4 - Before	Day 4 - After	Day S
	Day 1 - Before	:: .,	. E*	E*	E	E‡	E	E*	🖉 🔮 🗄 😒	E †
	Day 1 - After	F*			1					
	Day 2 - Before									
	Day 2 - After	F*				÷Ë		E		
Pre-Treatment	Day 3 - Before	S F	tin an				E4		. e	
	Day 3 - After									
	Day 4-8efore	F			F				. E #	₩
	Day 4 - After	_								
	Dav 5	A. 284.	- <i>1</i>		1. Same 7.	1.4.3.4.3.3.3	2	The gelote States	the souther "	

Table 3. 'All Groups' analysis: There was a statistically significant effect of time for maximum trunk extension (Ext_{Max}) and flexion moment ($Flex_{Max}$) (p<0.05). *Post hoc* results that are statistically significant are presented with "E" for extension and "F" for flexion trials. Differences large enough to be deemed "clinically relevant" are indicated by bolded letters with an asterisk.

Trunk Extension a	nd Flexion Trials:			Groups		
Post Hoc Result	s for All Groups	A-THF D1	A-THF D5	CON	LBP-THF D1	LBP-THF.D5
	A-THFD1			2	2 2	E
	A-THF DS					
Groups	CON					E‡
	LBP-THF D1					E‡
	LBP-THEDS	S. ()		F¥		

5.1.1 Trunk Extension and Flexion Moments: THF Groups

For Ext_{Max}, the THF Groups' analysis showed a significant main effect of Time (P < 0.0001). The *post* hoc analysis revealed long and short term improvements in Ext_{Max}. When comparing baseline values with day 2, 3, 4 and 5, there was sustained increase of 18.3%, 14.2%, 12.9% and 25.0% respectively. Furthermore, when assessing the effect immediately after each treatment, there was a significant increase in Ext_{Max} on day 1, 3 and 4 by 20.6%, 11.9% and 12.3% respectively (Figure 18).



Figure 18. THF Groups' analysis: The short term and sustained increases in maximum trunk extension moment for the THF groups over the course of the treatment program (n = 18). Standard error bars are presented. Short term increases in trunk extension moment were observed on day 1, 3 and 4. Sustained increases in trunk extension moment were found from the baseline values on day 1 compared to days 2, 3, 4 and 5. Significant results are displayed with an arrow.

For Flex_{Max}, the THF Groups' analysis showed a significant main effect with Time (P < 0.001). The *post* hoc analysis revealed long and short term improvements in Flex_{Max}. When comparing baseline values with day 1 and day 2 post-treatment results, there was an increase of 12.8% and 10.9% respectively (Figure 19).



Figure 19. THF Groups' analysis: This figure demonstrates the increases in maximum trunk flexion moment for the THF groups over the course of the treatment program (n = 18). Standard error bars are also presented. Significant results are displayed with an arrow.

5.1.2 Trunk Extension and Flexion Moments: All Groups

For Ext_{Max}, the All Groups' analysis demonstrated a significant main effect of Group (p<0.05). The *post hoc* analysis revealed no differences comparing both the CON group and the A-THF group on day 1 with the LBP-THF group on day 1. However, with the addition of the treatment program to the LBP-THF group, this group was able to exert 32.4% and 27.5% greater Ext_{Max} on day 5 of the treatment period compared with the baseline CON and A-THF groups respectively (Figure 20).



Figure 20. All Groups' analysis: This figure demonstrates the maximum trunk extension moment for CON and the change in the LBP-THF groups over the course of the treatment program (n = 26). Standard error bars are also presented. The arrow shows significant differences between groups.

For Flex_{Max}, the All Groups' analysis demonstrated a significant main effect for Group (p<0.05). The *post hoc* analysis revealed no differences comparing the CON group with the LBP-THF on day 1. However, on day 5, the LBP-THF group was able to exert 32.3% greater Flex_{Max} compared with the baseline CON group (Figure 21).



Figure 21. All Groups' analysis: The change in maximum trunk flexion moment for the LBP-THF group over the course of the treatment program compared with the CON groups' baseline values (n = 26). Standard error bars are also presented. The arrow demonstrates a significant difference between CON group D1 and LBP-THF group D5.

5.2 Disability and Pain Questionnaires

The dependent variable for the disability outcome measure was the RMDQ score and the covariate for this analysis was self-efficacy (PSEQ score). The dependent variable for the pain outcome measure was the 10-cm VAS score and the covariate for this analysis was self-efficacy (PSEQ score). For each analysis, the significant results are described below.

5.2.1 Roland-Morris Disability Questionnaire

For the RMDQ analysis there was a significant main effect of Time (p<0.05) and a significant covariate of self-efficacy (p<0.05). Prior to treatment, the average RMDQ score for the LBP-THF group was 5.1/24 (±2.8). After 4 treatments the average RMDQ score was 2.3/24 (±1.1). This represents a 55% reduction in this disability score (Figure 22).



Figure 22. This figure reveals *post hoc* results for the RMDQ/PSEQ analysis (n = 10). Standard deviation bars are presented. There was a statistically significant decrease in disability demonstrated over the course of the treatment period for the LBP-THF group.

5.2.2 10-cm Visual Analogue Scale

For the 10-cm VAS scale there was a significant main effect for Time (p<0.001) and self-

efficacy was not found to be a significant covariate (p>0.05). Prior to treatment, the average 10-

cm VAS score for the LBP-THF group was $4.2/10 (\pm 1.8)$. After 4 treatments the average 10-cm VAS score was $1.3/10 (\pm 1.0)$, representing a 65% reduction in pain (Figure 23).



Figure 23. This figure reveals *post hoc* results for the 10-cm VAS/PSEQ analysis (n = 10). Standard deviation bars are presented. There was a statistically significant decrease in this pain score over the course of the treatment period for the LBP-THF group.

5.3 Hip Extension

The dependant variable for this analysis was the passive hip extension angle from 3 different tests: 1) Modified Thomas Test (MTT), 2) Knee Extended and 3) Knee Bent. For each test, a one-way All Groups' ANOVA was run along with a two-way THF Groups' ANOVA. For each analysis, the significant results are described below.

5.3.1 Passive Hip Extension Tests: All Groups

For the passive hip extension tests, the All Groups' analysis revealed a significant main effect of Group (P < 0.001) for the Modified Thomas Test. The *post hoc* analysis revealed a significant difference between the CON group and the baseline values of the THF groups. There was a significant increase in passive hip extension over the course of the treatment program for the LBP-THF and A-THF groups by 13.1° ($\pm 1.1^{\circ}$) and 8.0° ($\pm 1.0^{\circ}$) respectively. After the completion of the treatment plan, the LBP-THF groups' hip extension value ($-8.3^{\circ} \pm 1.3^{\circ}$) was not significantly different than the CON group ($-9.1^{\circ} \pm 1.0^{\circ}$) (Figure 24). There were no significant findings for the All Groups' analysis with the Knee Extended and Knee Bent tests.



Figure 24. All Groups' analysis: This figure demonstrates the changes in passive hip extension values, using the MTT, for the THF groups over the course of the treatment program compared with the CON groups' baseline values (n = 26). Standard error bars are presented. The arrows demonstrate significant differences in passive hip extension angle.

5.3.2 Passive Hip Extension Tests: THF Groups

For the MTT, Knee Extended and Knee Bent tests, the THF Groups' analysis demonstrated a significant main effect of Time (p<0.001, p<0.001 and p<0.01 respectively). The treatment program revealed sustained increases in passive hip extension by 9.7° , 7.0° and 3.6° respectively. (Figure 25).



Figure 25. THF Groups' analysis: Using the MTT, Knee Extended and Knee Bent tests as outcome measures, this figure demonstrates the changes in passive hip extension for day 1 and day 5 of the treatment program (n = 18). Standard error bars are also presented. Significant results are displayed with arrows.

5.4 EMG Data

The EMG dependant variables for the known and unknown timing perturbation trials were baseline (BL) and anticipatory adjustment (AA). The dependent variable for the unstable standing task was the average rectified, EMG data. All significant effects and interesting trends are shown in Table 4. For this study, all statistically significant changes less than 1% of MVE were considered to be clinically irrelevant and were disregarded in the discussion. Furthermore, some changes that were not statistically significant, but did show an interesting trend, were considered in the Results and Discussion sections.

Side was one of the variables analyzed in the ANOVA. Generally speaking, differences between the right and left side were less than 1% (%MVE). Therefore, all significant Side results were not deemed to be clinically relevant and were not reported in the results or the discussion. Thus, EMG data were pooled across the right and left side.

Table 4. ANOVA results for the perturbation protocols with the 'THF Groups' and the 'All Groups' analysis. All statistically significant results are presented in the table below. The clinically relevant results are bolded with a star. The bolded results that are not statistically significant (P>0.05) demonstrated interesting trends and will be discussed in further detail within the discussion section.

		Tight Hip Flexor Groups								All Groups			
		Muscles	Group	Side	Time	Side* Group	Time* Group	Side* Time	Side* Time* Group	Group/ Time	Side	Group/ Time* Side	
		EO											
		- IO -								See See			
	B arratia a	TES											
	Basellite	LES											
16 m manage		MULT											
Known		GLUT			<0.001			<0.05					
riming Restucted		EO							<0.05				
Perturbation		io											
1	Anticipatory	TES				1						T	
	Adjustment	LES		1.		s Santa inci			- 19-2				
		MULT							<u> </u>				
		GLUT			1000				1				
	Baseline	EO								- <u>,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,</u>			
		0		535			<0.05		222	1.56			
		TES							T			>0,05	
		LES		1.0			1.200				<0.05	>0.05	
1.6.1		MULT						-				<0.01*	
Unknown		GLUT							1.20	1228			
liming		EO	[1		<u> </u>		
Perturbation		10		0.2.5		1.200	<0.01						
	Anticipatory	TES							<0.05	1			
	Adjustment	LES	14-15-	0.00									
		MULT	1	\square		<0.05							
		GLUT			<0.05		1990			\$ \$2.22			
		EO		1	1		and the second second		1			1	
		10	385	34.3			5.20			>0.05			
Unstable	Average	TES		1	<0.01*	<0.05	<u>, p. 17 343</u>		1	>0.05	1	1	
Standing	Muscle	LES		1	<0.05*	0.02			5	>0.05			
Perturbation	Activity	MULT		T	<0.01*					>0.05	1		
		GLUT			<0.001	1995	12.00	≪0.05		524	<0.09		

5.4.1 Known Timing

For the THF Groups' analysis of BL values, only one muscle, Gluteus, had significant effects. The gluteus had a main effect of Time (p>0.001). The largest difference between Times was 1.7% MVE and is presented in Figure 26.



Figure 26. Known Timing Perturbation: This figure reveals *post hoc* results for the baseline THF Groups' analysis (n = 18). Standard error bars are also presented. A statistically significant decrease in baseline muscle activity (%MVE) over the course of the treatment program (Day 1 and Day 5) is noted. The significant result is displayed with an arrow.

There were no significant results or interesting trends to report for the KT perturbation

with the All Groups' analysis.

5.4.2 Unknown Timing

For the UT perturbations, the All Groups' analysis for the BL variable showed no significant effects.

For the UT perturbations, the All Groups' analysis for the AA outcome measure revealed no significant results or interesting trends to report.

For the UT perturbations, the THF Groups' analysis for the BL outcome measure revealed only one muscle, IO, with significant effects. The IO had an interaction effect of Time*Group (p<0.05). There was less than a 1% (%MVE) decrease in muscle activity over the course of the treatment program for the LBP-THF group. Thus, these results were not deemed to be clinically relevant.

For the UT perturbations, the THF Groups' analysis for the AA variable revealed several significant results. The GLUT muscles had a main effect of Time (p<0.05). However, this result was not deemed clinically relevant as there was less than a 1% (%MVE) decrease in muscle activity over the course of the treatment program. The IO muscles had an interaction effect with Time*Group (p<0.01). However, the IO muscle activity was less than 1% (%MVE) and was considered not clinically relevant.

5.4.3 Unstable Standing

For the US perturbation, the THF Groups' analysis demonstrated a significant main effect was found for the TES, LES, MULT and GLUT muscles for Time (p<0.01, <0.05, <0.01 and <0.001 respectively – see Figure 27). Over the course of the treatment program, the muscle activity (%MVE) for the aforementioned muscles decreased by 1.8%, 1.6%, 2.3% and 1.5% respectively



Figure 27. Unstable Standing Perturbation: This figure reveals *post hoc* results from the THF Groups' analysis (n = 18). Standard error bars are also presented. There was a statistically significant decrease in the TES, LES, MULT and GLUT muscle activity (%MVE) for the A-THF and LBP-THF groups over the course of the treatment program (Day 1 and Day 5). The significant results are displayed with an arrow.

For the US perturbation, the All Groups' analysis demonstrated a generalized decreasing trend noted with the EO, IO, TES, LES, MULT and GLUT muscles for Group/Time (p>0.05). (Figure 28). The A-THF group experienced a decline in muscle activity (%MVE) over time for the EO muscle by 1.0%, for the IO muscle by 3.2%, for the TES muscle by 1.6%, for the LES muscle by 1.9%, for the MULT muscle by 2.4% and for the GLUT muscle by 1.6%. The LBP-

1.0%, for the IO muscle by 0.2%, for the TES muscle by 1.9%, for the LES muscle by 1.4%, for the MULT muscle by 2.2% and for the GLUT muscle by 1.4%.



Figure 28. Unstable Standing Perturbation: This figure reveals the results from the All Groups' analysis (n = 26). The Day 1 and Day 5 A-THF and LBP-THF muscle activity (%MVE) was subtracted from the CON group muscle activity. This figure demonstrates the general shift of the muscle activity towards the CON group after the treatment (towards the 0% mark). Interestingly, the only muscle that was below the CON group did not show a trend to shift downwards after the treatment period. These results were not statistically significant.

6.0 DISCUSSION

The current study was designed to examine the the influence of a passive treatment program (Active Release Technique) of the iliopsoas complex on two groups of subjects with tight hip flexors (LBP-THF and A-THF). Various outcome measures (trunk muscle responses to the three perturbations (%MVE), disability, pain, trunk flexion and extension strength and changes in passive hip extension) were compared between the two tight hip flexor groups over a 2 week passive treatment program. The outcome measures of the two THF groups were compared with the baseline outcome measures of a control group.

The results from this study demonstrated both significant short term and sustained improvements in trunk extension and flexion strength over time. The THF Groups significantly improved their pre-post treatment trunk maximum trunk extension moment on day 1, 3 and 4 by 20.6%, 11.9% and 12.3% respectively. After the 2 week treatment program was completed, the THF Groups were able to increase their trunk maximum trunk extension moment by 25% compared with their baseline values. When comparing the LBP-THF group with the CON group, there was a 34% and 32.3% increase in Ext_{Max} and Flex_{Max}, respectively, over the course of the treatment program.

Over the course of the treatment program, the LBP-THF group was able to reduce their disability by 2.8 points and pain by 2.9 cm. Interestingly, self-efficacy was found to be a significant covariate. For the passive hip extension measurements, the LBP-THF group and the A-THF groups increased their values by 13.1° and 8.0°, respectively. After the completion of the treatment plan, the LBP-THF groups' hip extension values were not significantly different to the CON group.

Over the course of the treatment program, the unstable standing trials showed a

significant decrease in average muscle activity (%MVE) for the TES, LES, MULT and GLUT

muscles over the course of the treatment program. There were no significant difference found in

anticipatory adjustment and baseline muscle activity (%MVE) for both the known and unkwon

timing sudden unloading trials. However, after the treatment program there was a trend for the

THF Groups' AA and BL values to shift closer to those of the CON group.

Hypotheses Revisited

In this section, each of the hypotheses will be addressed with regard to the current results.

Previous literature will be compared to the findings of the current study.

6.1 Trunk Flexion and Extension Exertions (MVE)

For the trunk MVE trials, prior to treatment, it is hypothesized that both the CON group and the A-THF group will have the highest Ext_{Max} and Flx_{Max} values, compared to the LBP-THF group. It is further hypothesized that the differences found between the LBP-THF treatment group and both the CON and A-THF group will become less pronounced over the course of the treatment program. Thus, it is hypothesized that MVE testing will show a statistically significant interaction (p<0.05) between the groups of subjects and the treatment time. Post hoc analysis will reveal that there will be no significantly different moments produced between the A-THF group and the CON group. Post hoc analysis will also demonstrate significantly different moments produced between the LBP-THF group and the two non-painful groups. By the end of the treatment program, LBP-THF group Ext_{Max} and Flx_{Max} values will not be significantly different from the CON and A-THF groups

6.1.1 Trunk Extension and Flexion Moments: THF Groups

Passive lengthening of the iliopsoas complex in the THF groups, using A.R.T[®], showed marked short term and sustained improvements for trunk extension and trunk flexion strength. One theory that explains the mechanism behind this type of treatment is that it allows the fasciomuscular interface to slide freely between each other promoting restoration of normal tissue extensibility (Leahy, 1995). Increasing the extensibility of the THF groups will restore the iliopsoas complex to a more normalized muscle length. This improved fascio-muscular extensibility has the potential to decrease its inhibitory effect on the antagonist and surrounding trunk musculature. This will allow the antagonist and surrounding trunk muscles to function closer to their full potential.

During trunk extension trials, healthy subjects have been shown to have significant activity of the lumbar multifidus and lumbar erector spinae muscles (Dickx *et al.*, 2008). However, certain muscles have a predictable tendency to become tense. It is common for the iliopsoas complex to develop tightness and decreased extensibility. This dysfunction has been grouped together within Pelvic Cross Syndrome, a common pattern of muscle impairment characterized by decreased extensibility of hip flexor and back extensor; and weakness of abdominal and hip extensor muscles (Janda, 1964; Jull, 1987). Sherrington's law (1947) states that tight muscles have an inhibitory influence on their antagonists (Janda, 1978). During prolonged training or continued postural shortening of a muscle, overuse will cause the muscle to become shorter, stronger and readily activated (Sahrmann, 1987). Unfortunately, if the overuse activities continue, the muscle tightness will lead to muscle strength decreases. A common practice for restoring muscle strength is to induce a lengthening treatment (Sahrmann, 1987).

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It would seem sensible to speculate that A.R.T[®], a type of fascio-muscular lengthening therapy, might have restored the iliopsoas complex to a normalized length and extensibility. The iliopsoas complex is an integral component towards maintaining spine stability (Santaguida and McGill, 1995). To change the function of a key trunk stabilizing muscle, such as the iliopsoas complex, would change the relative contribution of the antagonist and supporting trunk muscles contribution to spine stability. The central nervous system will choose the best muscle activation patterns in order to optimize the relationship between spine loading and spine stability (Brown

and Potvin, 2005). Normalizing the iliopsoas complex might decrease the inhibition of other trunk stabilizing muscles and give individuals the ability to produce greater moment production during MVE trials. This may help explain how the THF groups were able to increase their moment production after this type of therapy.

6.1.2 Trunk Extension and Flexion Moments: Comparing Across All Groups

After the course of treatments, over the 2 week period, there was a significant increase in maximum trunk extension moment for LBP-THF group compared with their baseline values. While there were no significant differences between all the groups' baseline extension moment values, after the treatment the LBP-THF group was able to significantly generate greater extension moment than the CON groups' baseline values.

As seen in the trunk extension trials, the baseline trunk flexion trials showed no difference between the CON and LBP-THF groups. Likewise, after the treatment program was completed, the LBP-THF group was able to significantly produce more flexion moment compared with the CON groups' baseline values.

The subjects were asked to maintain their normal daily activities over the course of the 2 week treatment period. However, the subjects were able to produce greater moment values immediately after treatment and over the course of the entire treatment schedule. There are possibly 2 different explanations that may explain the changes that occurred: (1) pain-adaptation theory and (2) that pain produces muscle inhibition, which is a subconscious protection mechanism.

The RMDQ and 10-cm VAS demonstrated that, over the course of the treatment program, there was a significant decrease in disability and pain for the LBP-THF group. Painrelated fear can lead to avoidance behaviours and disuse. These individuals avoid activation of

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their trunk muscles during maximal isometric trunk exertions (Thomas *et al.*, 2008). The painadaptation theory follows this premise closely. It hypothesizes that, when pain is present, there is a decreased activation of muscles during movements in which they act as agonists and increased activation during movements in which they are antagonists (Lund *et al.*, 1991). This theory has been supported by several studies (Arendt-Nielsen *et al.*, 1996; Graven-Nielsen *et al.*, 1997).

It is possible that individuals with low back pain will not use their trunk muscles to their functional capacity due to fear-avoidance behaviours. This apprehension will decrease the possibility of developing further injury. As a result, these individuals would not be able to perform optimally tasks such as the maximum trunk extension and flexion trials recorded before the treatement. The therapy for the LBP-THF group may have helped decrease their fear-avoidance behaviour by lowering their pain and disability, lessening trunk muscle inhibition. This could explain the greater moment production following treatment.

Another possible mechanism for the decreased muscle activity in low back pain patients might be due to the body's subconscious protective mechanism against further harm. It is well recognized that low back pain patients tend to exhibit inhibition of their trunk muscles (Hides *et al.*, 1996). The erector spinae muscle has been shown to have varying EMG signals ranging from increased activity to decreased activity and no change in activity in LBP (Hodges and Moseley, 2003; van Dieen *et al.*, 2003b). However, the pain that one experiences during a bout of low back pain may create a subconscious compensatory mechanism that inhibits trunk muscle activity so that no further damage is caused. One important study that illustrates this point was performed by Dickx and colleagues (2008). Muscle activity measured in healthy subjects, with muscle functional MRI, showed significant activity of the lumbar multifidus, lumbar erector spinae muscles and the psoas muscles during trunk extension trials. In the same study,

experimentally induced unilateral muscle pain was induced in these healthy subjects. It was found that the induced pain influenced muscle activity of the aforementioned muscles. They demonstrated hypoactivity of diverse stabilizing muscles, not limited to the side and level of the pain, during trunk extension trials (Dickx *et al.*, 2008). Therefore, it would be reasonable to suggest that individuals will reduce trunk muscle activity while experiencing pain as protective mechanism against further harm. It is suggested that the addition of the treatment program for the LBP-THF group helped decrease the pain and disability they were experiencing which, in turn, allowed these subjects to produce greater moment production during their post-treatment MVE trials.

6.2 Disability and Pain Questionnaires

The low back pain group will have significantly higher levels of disability and pain measures before beginning the treatment program. It is hypothesized that, over the course of the treatment period, the RMDQ and 10-VAS scores will decrease significantly. Further, it is hypothesized that there will be a significant main effect for Time (p<0.05) that will demonstrate a decrease in disability and pain over the course of the treatment period,. Finally, it is hypothesized that the PSEQ self-efficacy score will be a significant covariate for disability and pain changes over the course of the treatment program.

6.2.1 Roland-Morris Disability Questionnaire

Over the course of the treatment program the average disability rating for the LBP-THF group was reduced by more than half of the baseline score. The minimal important difference for change in the RMDQ score has varied in the literature. One study suggested a change score of 5 on the RMDQ has been recommended as the smallest change that is important to patients over a 3-6 week treatment program(Stratford *et al.*, 1998). However, this study recommended further research into baseline score-specific minimum importance difference from anchor-based methods. Beurskens *et al.* (Beurskens *et al.*, 1996) recommended a change of 2.5–5 points for

signalling improvement after 5 weeks of treatment, All these studies were looking at treatment plans that were longer then the treatment program instituted in this study. A more recent study examined recommended 3 different categories: (1) definitely improved patients - rating their back pain as at least better at 6 months and with a reduction of at least 30% on their RMDQ score from baseline, (2) possibly improved patients – those who have a RMDQ score at least 30% reduced at 6 months but who have not rated their back pain as better, and (3) not improved patients - those with less than a 30% reduction in RMDQ score at 6 months (Jordan *et al.*, 2006). The most recent study analyzed the literature which demonstrated, when the baseline score is taken into account, a 30% improvement (2 to 8.6 points) was considered a useful threshold for identifying clinically meaningful improvement on each of these measures (Ostelo *et al.*, 2008). However, the authors from this paper suggested 3.0 to 6.0 point change and 20% to 30% improvement to be considered a minimal important difference. Applying the most recent literature to the changes found in this study, the results from this study are encouraging results for this type of treatment therapy for LBP patients with THFs.

The decreased disability scores in this population can be hypothesized to stem from releasing excess compression on the damaged structures of the spine. The LBP population will generate co-contraction to stiffen and stabilize the lumbar spine (Quint *et al.*, 1998; Cholewicki and McGill, 1996; Bergmark, 1989; Radebold *et al.*, 2000). Muscle force that exceeds the necessary requirements to create spine stiffness can compromise spine stability (McGill, 2002). This excess joint stiffness can damage the spines' surrounding tissues (Panjabi, 1992a). Therefore, relieving pressure on the spine by lengthening the iliopsoas complex may decrease the sensitivity to the irritated structures. This will enable the LBP population to perform their daily activities with less pain, increased range of motion and decreased disability.

An interesting finding for the disability analysis was that self-efficacy was found to be a significant covariate. Primarily, there are two factors that affect an individual's ability to execute a desired task: 1) the ability to physically complete the task and 2) the belief system of the individual to complete that task. Self-efficacy is the belief that one has the ability to successfully perform certain tasks or behaviours in order to produce a desired outcome (Bandura, 1977). The relationship between disability, self-efficacy, fear avoidance and pain intensity has been previously researched (Denison *et al.*, 2004). Denison's study demonstrated that self-efficacy explained the largest proportion of the variance in disability scores.

The results from Denison's (2004) study are congruent with the results from the current research. Self-efficacy is a crucial factor that can help a LBP patient deal with their pain. It affects the way LBP patients face aversive painful experiences. LBP patients need to have confidence in their abilities and expect that they can execute a particular task. These findings stress the importance of evaluating a LBP patient's self-efficacy prior to the commencement of a treatment program, as it will affect their progress.

6.2.2 10-cm Visual Analogue Scale

The 10-cm VAS is a tool for monitoring a patient's perceived pain and has been proven to be valid, reliable and appropriate for use in clinical practice (Williamson and Hoggart, 2005). The minimally important difference has been suggested to be 15/100 (or 1.5/10) and 30% improvement from baseline scores (Ostelo *et al.*, 2008). The 10-cm VAS was used as a pain outcome measure in this study. At the conclusion of the 2 week treatment period, there was an average change of 2.9 out of 10 cm on this scale. Self-efficacy was not found to be a covariate for changes in pain. Unfortunately, the results from this study cannot be compared with other studies as there is no known research examining A.R.T[®] for the treatment LBP patients with

THFs. However, the results from this study are promising for the benefits of A.R.T for the LBP-THFs population.

Many LBP patients have injuries to their spinal structures. Low back pain patients have been shown to exhibit a pattern of spine co-contraction as a protective mechanism(Radebold *et al.*, 2000). Co-contraction of the trunk muscles has been shown to create spine stability (Cholewicki and McGill, 1996). The iliopsoas complex is one of the key spine stabilizing muscles by way of compression (Santaguida and McGill, 1995; Juker *et al.*, 1998). Unfortunately, excess pressure on the spine can cause more damage to the painful structures creating the pain-spasm-pain model (Panjabi, 1992a; van Dieen *et al.*, 2003b). One possible explanation for the mechanism of this treatment is breaking the painful loop by relieving compression on the painful spinal structures. It has been speculated that this treatment removes adhesions between the musculo-fascial interface and promotes restoration of normal tissue extensibility (Leahy, 1995). Lengthening the iliopsoas complex might decrease spine compression, give the spinal structures a chance to heal and contribute to breaking the painful cycle.

6.3 Hip Extension

Measuring passive hip extension, the CON group will have significantly greater hip extension than the THF groups. At the end of the treatment period, the differences between the CON and THF groups will be less noticeable. Hip extension measurements will show a statistically significant interaction (p<0.05) between the groups of subjects and the treatment time. Post hoc analysis will reveal that there will be a significantly different hip extension measurement between the THF groups and the CON group at baseline measurements. Post hoc analysis will also demonstrate, after the completion of the treatment program, no significantly different measurements between the THF groups and the CON group.

6.3.1 Modified Thomas Test: All Groups

The MTT is the only known test to show good intra-rater reliability for measuring the length of the iliopsoas complex and, therefore, was used as the outcome measure for measuring changes in hip extension over the course of the treatment program. It was found that there was a significant increase in passive hip extension in the THF groups over time. After the completion of the treatment, the LBP-THF groups' hip extension values were not significantly different from the CON group.

Hip extension and low back pain have been briefly touched upon in the research literature. One study showed that individuals with LBP displayed less passive hip extension than people without LBP (Van Dillen *et al.*, 2000). Clinicians have postulated that low back pain patients can have tight hip flexors (Janda, 1986). Stretching treatment of the iliopsoas complex has been shown to increase hip extension, reduce pain and aid the return to normal activity for patients with low back pain (Ingber, 1989; Winters *et al.*, 2004).

Improving the extensibility of the hip flexor complex in low back pain patients can help alleviate stress on the spine and decrease the pain experienced by these individuals. Athletes with THFs and low back pain can benefit greatly from this type of treatment prior to practice and competition. These athletes will be able to concentrate on the sport instead of the pain. In addition, this type of treatment may provide the athletes with increased hip range of motion and might facilitate improved performance.

6.3.2 Modified Thomas Test, Knee Extended and Knee Bent Tests: THF Groups

There has been no known research regarding sensitivity and reliability of the knee extended and knee bent test for measuring passive hip extension. Both tests have the subject lying in the prone position while the examiner passively extends the hip with the knee extended and bent. The lumbar spine must be in a neutral position during hip extension measurements. All three tests

(MTT, knee extended and knee bent tests) for the THF groups demonstrated significant increases

in hip extension measurements when comparing the baseline values with the post-treatment

values.

6.4 Sudden Unloading and Unstable Standing Perturbation Trials

In the perturbation trials (UT, KT and US), prior to treatment, it is hypothesized that the control group (CON) will have the lowest baseline muscle activity and anticipatory adjustment muscle activity during known and unknown unloading and the lowest average trunk muscle activity during unstable standing, which will be exceeded by the low back pain tight hip-flexor group (LBP-THF). It is hypothesized that the differences found between the LBP-THF treatment group and the CON group will become less pronounced over the course of the treatment program. It is hypothesized that there will be no differences between the CON group and the A-THF group.

Baseline, anticipatory adjustment during sudden unloading (UT and KT) and average trunk muscle activity during unstable standing (US)_will show a statistically significant interaction (p<0.05) between the groups of subjects and the treatment time. Post hoc analysis will reveal that, at baseline, there will be a significant difference between the CON group and the LBP-THF group, but at the completion of the four treatment sessions, anticipatory adjustment and average trunk muscle activity in the LBP-THF treatment group will show no significant difference compared with the control group.

6.4.1 Unloading with Known Timing

There were no significant results for the KT condition that were considered to be clinically relevant. However, there was an increasing trend noted for the thoracic and lumbar extensor muscle groups of the THF subjects over the course of the treatment program. The day 1 muscle activity of the TES and LES muscles for the control group had a greater anticipatory adjustment compared with the day 1 THF groups. There was a greater tendency for the control subjects to relax their back extensor muscles prior to dropping the load compared with the THF groups. Even though these results were not show to be significant, over the course of the treatment program, there was a trend for THF groups' anticipatory adjustment to become more similar to that of the CON group.

There have been few studies in the literature examining unloading perturbations with known timing. Unloading tasks create moments that are completely opposite to those in response to loading tasks. Therefore, it has been suggested that muscle activation patterns will be different than those of loading tasks (Brown *et al.*, 2003).

In one relevant study, subjects maintained a 1-kg weight with their postural forearm, attached via an electromagnet. The researchers in this study repeatedly unloaded the forearm. The researchers desmonstrated an increase in the anticipatory inhibition in the EMG of the biceps muscle with the unloaded arm (Kazennikov *et al.*, 2007). It has also been shown that inhibition of the erector spinae activity was found during the 1500 first milliseconds (anticipatory adjustment) of the sitting movement in healthy individuals (Cheynel *et al.*, 2002). Finally, another study has shown that the LES and TES of healthy subjects significantly decreased anticipatory activity as knowledge of the unload timing increased (Brown *et al.*, 2003). Unfortunately, there are no known studies in the literature that have examined expected unloading tasks for subjects with low back pain or tight hip flexors and the effects of a treatment program on these populations.

Similar to the aforementioned known timing unloading studies, the control group also exhibited greater inhibition of the trunk extensors compared with the THF groups (including the low back pain population) during the anticipatory adjustment phase of the known unloading task. This muscle activity pattern prior to spine perturbations has an important effect on developing spine stability. Stability of the spine is reliant on the active, passive and neural subsystems to develop trunk stiffness(Panjabi, 1992a). Elevated trunk muscle activity increases compressive forces acting on the spine. The function of the anticipatory adjustment is to counteract the consequence of the unloading perturbation. Therefore, maintaining muscle activity prior to
unloading an object will decrease the postural disturbance and increase spine stability. These strategies have been shown to be most effective in situations where the perturbation timing is precisely known (Brown *et al.*, 2003).

6.4.2 Unloading with Unknown Timing

The comparison of the All Groups' analysis for the unknown timing perturbation demonstrated a statistically significant decrease in MULT baseline muscle activity (%MVE) for the A-THF and LBP-THF groups over the course of the treatment program (Day 1 and Day 5). It was also noted that there was a decreasing trend in the TES and LES baseline muscle activity (%MVE) for the A-THF and LBP-THF groups over the course of the treatment period..

There have been few known studies in the literature examining unknown unloading perturbations. Brown et al. (2003) demonstrated that, during unknown unloading tasks, the subjects tended to increase their baseline muscle activity prior to unloading a mass. There has also been research examining muscle activity during perturbation tasks for low back patients. Low back pain patients exhibit delayed muscle responses (Radebold *et al.*, 2001), altered muscle recruitment patterns (Radebold *et al.*, 2000), increased muscle shut-off latency and significantly more muscles active during perturbation tasks (Cholewicki *et al.*, 2002). However, there are no known studies in the literature that look at the influence of a passive treatment program, such as A.R.T[®], on the trunk muscle recruitment during unknown unloading tasks.

Muscle spasm or increased resting muscle activity is often considered to be a prominent feature of the low back pain population (Kravitz *et al.*, 1981). Kavitz (1981) has demonstrated that LBP patients exhibit higher levels of back muscle activity as compared to non-painful populations. This is a protective mechanism to increase spine stability and prevent further damage. Therefore, it is not surprising that compared with the healthy population; the LBP-THF

group had higher baseline muscle activity. Furthermore, at the conclusion of the treatment program, there was a decrease in the resting muscle activity. Even though the multifidus was the only muscle to exhibit a significant decrease in the resting muscle activity, this study demonstrated a clear decreasing trend for all 3 trunk extensor muscles towards the values of the CON group.

Another possible explanation for the effects of this treatment may lie in the extensibility of the iliopsoas complex. Cholewicki & McGill (1996) has found that compression from the psoas will create segmental stiffness. Juker *et al.* (1998) has suggested that relatively low psoas muscle activity might be enough to create sufficient stiffness of the lumbar spine for demanding activities and too much compression can have a detrimental effect on the spine's health. Lengthening the tight fascio-muscular structures may decrease the stress on the spine and normalize the function of the antagonist muscles, such as the TES, LES and MULT.

6.4.3 Unstable Standing

For the unstable standing trials, the THF groups had decreases in the tonic muscle activity of the TES, LES, MULT and GLUT muscles.

The low back pain population has been observed to have altered proprioceptive postural control (Radebold *et al.*, 2001; Brumagne *et al.*, 2000; Brumagne *et al.*, 2008). These patients seem to adopt a body and trunk stiffening strategy and rely more on ankle proprioception to control their posture during quiet upright standing and unstable standing conditions (Brumagne *et al.*, 2008). For these reasons, clinically speaking, postural stability and proprioception tests are now being used to examine low back pain patients (Tidstrand and Horneij, 2009). Therefore, the LBP population will generate co-activation of agonistic and antagonistic muscle groups to stiffen and stabilize the lumbar spine (Quint *et al.*, 1998; Cholewicki and McGill, 1996; Bergmark,

1989; Radebold *et al.*, 2000). This may serve as a compensatory mechanism to protect the individual from pain and delay the progression of damage to the spine. However, excess levels of muscle force and stiffness at a given spine segment can compromises the segmental spine stability (McGill, 2002). Excess joint loading from the surrounding soft tissues can cause damage to the surrounding joint structures (i.e. ligaments, discs) (Preuss and Fung, 2005). Therefore, it is suggested that lengthening the fascio-muscular complex might reduce the stresses imposed on the spine. With less compression on the spine, there would be less need to adopt a trunk stiffening strategy.

The CON group exhibited lower IO, TES, LES and MULT tonic muscle activity compared with the THF groups during unstable standing. However, after the treatment program the THF groups' muscle activity showed a trend to shift towards that of the CON group. Stability of the spine is developed from varying coordinated muscle activation patterns involving many trunk muscles. These recruitment patterns change depending on the task at hand and the condition of the surround muscle activity (McGill *et al.*, 2003a). One could estimate that lengthening one of the spine stabilizing trunk muscles, such as the iliopsoas complex, might change the muscle activity of the surrounding trunk muscles. In this instance, the opposing trunk muscles would have to change their activity to offset the changes in the hip flexor complex.

7.0 LIMITATIONS AND RECOMMENDATIONS FOR FUTURE RESEARCH

7.1 Limitations

There are several limitations associated with this research study. The first is related to the subject population tested. There was a wide range of subjects examined with varying athletic abilities, varying sizes and weights and varying duration and severity of LBP. The different types of subjects may have influenced the results and future studies should aim to standardize their subject population more closely.

The treatment program can also be scrutinized as an area of limitation. Even though there were several promising results attained from this research, the treatment program only consisted of 4 treatments over a 2 week period. Considering many of the subjects have had LBP and/or THFs for a long period of time, this treatment program may not have been enough to elicit the true benefits from this type of therapy. For example, the results for the KT and UT were not deemed to be significant, but might have been more pronounced if the concentration and length of the treatment program were increased. Also, when designing the perturbation tasks, extreme caution was employed such that the perturbation loads were small to protect subjects with LBP. Unfortunately, only minimal trunk muscle activity was needed during these unloading perturbation tasks. An unstable standing surface that imposed a greater challenge to the subjects might elicit more trunk muscle activity and greater changes in pre-post treatment results. Regarding the KT and UT perturbations, the mass might not have been heavy enough and was not relatively proportioned for each individual. This might have influenced the results of this perturbation scenario as the subjects may have not been stressed enough to tease out the true effects of the therapy.

There may have been a limitation in the CON group tested. During the MVE trials, it was found that the baseline trunk extension and flexion values of the CON group were similar to both THF groups. After the introduction of the treatment program, the THF groups (including the LBP-THF group) were able to produce greater moment production. The CON group might have been a weaker group of subjects and, therefore, the group tested might not have been a good representation of the general athletic population. Interestingly, several researchers have found LBP subjects to have had higher strength measures (Stevenson *et al.*, 2001; McGill *et al.*, 2003b). It has been suggested that these individuals chose to move with more spine motion and activate muscle in a way that causes higher back loads. Closer attention to subject strength abilities should be noted for future research in this area.

One final limitation of note was the specificity of the targeted structure of treatment. There are various tissues between hand contact of the therapist and the targeted iliopsoas complex and it possible that the effects of the treatments might not be solely related to the targeted myofascial complex of the hip flexor. In addition, the patient is put through a fairly large range of motion throughout the treatment session. Therefore, the benefits may arisen from the passive mobilization of the lumbar spine and/or passive stretching of other hip related structures.

7.2 Recommendations for Future Research

Since there are several theoretical hypotheses presented in this thesis, there are many options for future research that could help to elucidate some of the questions posed here. One of the first follow-up research issues, that must be addressed, is to find out the prevalence and association between LBP suffers and THFs. Many clinicians treat the THF complex for individuals with LBP. However, it is not understood which types of populations (i.e. athletes, desk employees, age, gender etc.) have the tendency to develop this problem or why they are developing this

dysfunction in the first place. Currently, it is not understood if the tight hip flexor complex is causing low back pain or if the tight hip flexor complex is a compensatory mechanism for other spinal pathology such as multifidus dysfunction or injury to other spinal structures (i.e. ligaments, capsules, discs etc.).

During the known and unknown timing unloading perturbations, examination of other important spine stabilizing muscles, such as the iliopsoas and quadratus lumborum, by way of indwelling EMG may help clarify the trunk muscles relative contribution toward spine stability. Other considerations regarding the known and unknown perturbation scenario would be to incorporate a greater mass, or a mass that is proportional to each individual subject. Other methods of perturbation can also be imposed on individuals. For example, analysing a dynamic loading or unloading task that is typically experienced in the workplace or athletic environment might give us new information on more practical everyday tasks. A particular interest to the author is the relative contribution of the gluteus maximus to spine stability and LBP patients. A better perturbation to test the effects of this treatment on the gluteus maximus might give us knew information that is important when examining these populations.

An improved treatment protocol for LBP subjects may elicit improved clinically relevant results. For example, increasing the number of treatments over a longer time period may bring about improved results. In addition, a long term follow-up study would be important to examine if the changes found are short-term or long lasting.

Active exercise therapy is a very important component in patient recovery. Therefore, future studies should focus on complementing passive and active therapy within a treatment program for low back pain patients.

From a sports performance perspective, future research studies may endeavour to examine the effects of this type of treatment on sporting performance measurements. Some relevant questions include: Does this treatment help improve sporting performance? If so, is this type of treatment solely beneficial for LBP athletes or does this treatment help improve the performance of all THF groups?

8.0 SUMMARY

There are numerous medications and non-pharmacologic therapies for LBP patients and many of these treatments have been validated in the research literature (Chou and Huffman, 2007a; Chou and Huffman, 2007b). However, there have been few known studies examining the iliopsoas complex, low back pain and passive treatments, such as A.R.T[®], for treating this type of muscular dysfunction. The studies, found in this area suggest, that a stretching treatment of the iliopsoas complex has been shown to increase hip extension, reduce pain and aid the return to normal activity for patients with low back pain (Ingber, 1989; Winters *et al.*, 2004). There is evidence to support A.R.T[®] for treating trigger thumb, lateral epicondylosis and hamstring flexibility (George *et al.*, 2006; Howitt, 2006; Howitt *et al.*, 2006). However, there has been no known research to support this commonly used therapy for treating low back pain patients with THFs.

Three groups of subjects participated in this study: 1) low back pain subjects with tight hip flexors (LBP-THF), 2) asymptomatic subjects with tight hip flexors (A-THF), and 3) control subjects (CON). Subjects from the THF groups received A.R.T[®], a therapy for improving the extensibility of their tight hip flexors. Changes in various outcome measures were examined

including EMG measures during perturbation tasks, disability, pain, trunk Ext_{Max} and $Flex_{Max}$ trials and passive hip extension measurements over a 2 week period.

The results from this study demonstrated significant short term and sustained improvements in trunk Flex_{Max} and Ext_{Max} . The THF Groups significantly improve their pre-post treatment trunk maximum trunk extension moment on day 1, 3 and 4 by 20.6%, 11.9% and 12.3% respectively. After the 2 week treatment program was completed, the THF Groups were able to increase their trunk maximum trunk extension moment by 25% compared with their baseline values. When comparing the LBP-THF group with the CON group, there was a 34% and 32.3% increase in trunk extension and flexion moment production over the course of the treatment program.

Over the course of the treatment program, the LBP-THF group was able to reduce their disability by 2.8 points and pain by 2.9 cm. Interestingly, self-efficacy was found to be a significant covariate for the disability outcome measure. For the passive hip extension measurements, the LBP-THF group and the A-THF groups increased their values by 13.1° and 8.0°, respectively. In addition, the other two hip extension tests (knee extended and knee bent tests) demonstrated significant increases in measured passive hip extension values over time. After the completion of the treatment plan, the LBP-THF groups' hip extension values were not significantly different from the CON group.

Interesting trends and significant results were found during the various perturbation outcome measures (KT, UT and US) for the A-THF and LBP-THF group over the course of the treatment program. The US perturbation trials showed a significant decrease in average muscle activity for the TES, LES, MULT and GLUT muscles over the course of the treatment program.

On day 1, prior to the commencement of the treatment program, the CON group tended to have a greater anticipatory adjustment and lower baseline values for the KT and UT trials compared with the THF groups. Over the course of the treatment program, there were no significant difference found in anticipatory adjustment and baseline muscle activity for the KT and UT perturbation trials. However, after the treatment program there was a trend for the THF Groups' AA and BL values to shift closer to those of the CON group.

REFERENCES

Aalto, T.J., Airaksinen, O., Harkonen, T.M., and Arokoski, J.P. (2005) Effect of passive stretch on reproducibility of hip range of motion measurements. *Arch.Phys.Med Rehabil.* **86**, 549-557.

Andersson, B.J.G., Ortengren, R., Nachemson, A., and Elfstrom, G. (1974) Lumbar disc pressure and myoelectric back muscle activity during sitting. *Scandinavian Journal of Rehabilitation Medicine* **6**, 128-133.

Andersson, E., Oddsson, L., Grundstrom, H., and Thorstensson, A. (1995) The role of the psoas and iliacus muscles for stability and movement of the lumbar spine, pelvis and hip. *Scand.J Med Sci.Sports* 5, 10-16.

Andersson, E.A., Nilsson, J., and Thorstensson, A. (1997) Intramuscular EMG from the hip flexor muscles during human locomotion. *Acta Physiol Scand.* **161**, 361-370.

Andersson, G.B.J., Ortengren, R., and Herberts, P. (1977) Quantitative electromyographic studies of back muscle activity related to posture and loading. *Orthop.Clin.North Am.* **8**, 85-96.

Arendt-Nielsen,L., Graven-Nielsen,T., Svarrer,H., and Svensson,P. (1996) The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. *Pain* **64**, 231-240.

Bachrach, R.M. (1988) Team physician #3. The relationship of low back/pelvic somatic dysfunctions to dance injuries. *Orthop.Rev.* 17, 1037-1043.

Bandura, A. (1977) Self-efficacy: toward a unifying theory of behavioral change. *Psychol.Rev.* **84,** 191-215.

Bandura, A. (2004) Health promotion by social cognitive means. *Health Educ.Behav.* **31**, 143-164.

Barker,K.L., Shamley,D.R., and Jackson,D. (2004) Changes in the cross-sectional area of multifidus and psoas in patients with unilateral back pain: the relationship to pain and disability. *Spine* **29**, E515-E519.

Benight, C.C. and Bandura, A. (2004) Social cognitive theory of posttraumatic recovery: the role of perceived self-efficacy. *Behav.Res.Ther.* **42**, 1129-1148.

Bergmark, A. (1989) Stability of the lumbar spine. A study in mechanical engineering. *Acta Orthop.Scand.Suppl* **230**, 1-54.

Beurskens, A.J., de Vet, H.C., and Koke, A.J. (1996) Responsiveness of functional status in low back pain: a comparison of different instruments. *Pain* **65**, 71-76.

Bogduk, N.P.M.H.G. (1992) Anatomy and biomechanics of psoas major. *Clin.Biomech.* 7, 109-119.

Brown,S.H.M., Haumann,M.L., and Potvin,J.R. (2003) The response of leg and trunk muscles to sudden unloading of the hands: implications for balance and spine stability. *Clin.Biomech.* **18**, 812-820.

Brown,S.H.M. and Potvin,J.R. (2005) Constraining spine stability levels in an optimization model leads to the prediction of trunk muscle co-activity and improved predictions of spine compression. *J.Biomech.* **38**, 745-754.

Brox, J.I., Storheim, K., Holm, I., Friis, A., and Reikeras, O. (2005) Disability, pain, psychological factors and physical performance in healthy controls, patients with sub-acute and chronic low back pain: a case-control study. *J.Rehabil.Med.* **37**, 95-99.

Brumagne,S., Cordo,P., Lysens,R., Verschueren,S., and Swinnen,S. (2000) The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. *Spine* **25**, 989-994.

Brumagne, S., Janssens, L., Knapen, S., Claeys, K., and Suuden-Johanson, E. (2008) Persons with recurrent low back pain exhibit a rigid postural control strategy. *Eur.Spine J* **17**, 1177-1184.

Bullock-Saxton, J.E., Janda, V., and Bullock, M.I. (1993) Reflex activation of gluteal muscles in walking. An approach to restoration of muscle function for patients with low-back pain. *Spine* **18**, 704-708.

Chaitow, L.W.D.J. Clinical Application of Neuromuscular Techniques. 31-72. 2002. Churchill Livingstone, China. Ref Type: Generic

Cheynel,N., Mourey,F., Peschaud,F., Durand-Fontanier,S., Didier,J.P., and Trouilloud,P. (2002) [Standing-up/sitting-down movement. Electromyographic analysis of 4 muscles of lower limb and the erector spinae muscle: study of anticipatory postural adjustments]. *Morphologie.* **86**, 23-26.

Cholewicki, J., Panjabi, M.M., and Khachatryan, A. (1997) Stabilizing function of trunk flexorextensor muscles around a neutral posture. *Spine* **22**, 2207-2212.

Cholewicki, J., Greene, H.S., Polzhofer, G.K., Galloway, M.T., Shah, R.A., and Radebold, A. (2002) Neuromuscular function in athletes following recovery from a recent acute low back injury. *J.Orthop.Sports Phys.Ther.* **32**, 568-575.

Cholewicki, J. and McGill, S.M. (1995a) Relationship between muscle force and stiffness in the whole mammalian muscle: a simulation study. *J Biomech.Eng* **117**, 339-342.

Cholewicki, J. and McGill, S.M. (1995b) Relationships between muscle force and stiffness in the whole mammalian muscle: A simulation study. *J.Biomech.Eng.* **117**, 339-342.

Cholewicki, J. and McGill, S.M. (1996) Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. *Clin.Biomech.* **11**, 1-15.

Cholewicki, J., McGill, S.M., and Norman, R.W. (1991) Lumbar spine loads during the lifting of extremely heavy weights. *Med.Sci.Sports Exer.* 23, 1179-1186.

Cholewicki, J., Silfies, S.P., Shah, R.A., Greene, H.S., Reeves, N.P., Alvi, K., and Goldberg, B. (2005) Delayed trunk muscle reflex responses increase the risk of low back injuries. *Spine (Phila Pa 1976.)* **30**, 2614-2620.

Cholewicki, J. and VanVliet, J.J. (2002) Relative contribution of trunk muscles to the stability of the lumbar spine during isometric exertions. *Clin.Biomech.(Bristol., Avon.)* **17**, 99-105.

Chou, R. and Huffman, L.H. (2007a) Medications for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. *Ann.Intern.Med* **147**, 505-514.

Chou, R. and Huffman, L.H. (2007b) Nonpharmacologic therapies for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. *Ann.Intern.Med* **147**, 492-504.

Christiansen, C.L. (2008) The effects of hip and ankle stretching on gait function of older people. *Arch.Phys.Med Rehabil.* **89**, 1421-1428.

Clapis, P.A., Davis, S.M., and Davis, R.O. (2008) Reliability of inclinometer and goniometric measurements of hip extension flexibility using the modified Thomas test. *Physiother.Theory.Pract.* **24**, 135-141.

Cobb,C.R., DeVries,H.A., Urban,R.T., Luekens,C.A., and Bagg,R.J. (1975) Electrical activity in muscle pain. *Am.J Phys.Med* 54, 80-87.

Cooper, R.G., St Clair, F.W., and Jayson, M.I. (1992) Radiographic demonstration of paraspinal muscle wasting in patients with chronic low back pain. *Br.J Rheumatol.* **31**, 389-394.

Cresswell,A.G., Oddsson,L., and Thorstensson,A. (1994) The influence of sudden perturbations on trunk muscle activity and intra-abdominal pressure while standing. *Exp.Brain Res.* **98**, 336-341.

Cyriax J (1982) *Textbook of Orthopaedic Medicine: Diagnosis of Soft Tissue Lesions*. London: Bailliere Tindall.

Dangaria, T.R. and Naesh, O. (1998) Changes in cross-sectional area of psoas major muscle in unilateral sciatica caused by disc herniation. *Spine* **23**, 928-931.

Danneels,L.A., Vanderstraeten,G.G., Cambier,D.C., Witvrouw,E.E., and De Cuyper,H.J. (2000) CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. *Eur.Spine J* 9, 266-272.

Denison, E., Asenlof, P., and Lindberg, P. (2004) Self-efficacy, fear avoidance, and pain intensity as predictors of disability in subacute and chronic musculoskeletal pain patients in primary health care. *Pain* **111**, 245-252.

Di Lorenzo, L., Forte, A., Landolfi, A., Di Mario, M., Formisano, R., and Gatto, S. (2007) [Chronic lumbago after unstable intertrochanteric femoral fracture: a new syndrome or sporadic feature of hip biomechanics after surgery? A case report]. *G.Ital.Med Lav.Ergon.* **29**, 210-213.

Dickx,N., Cagnie,B., Achten,E., Vandemaele,P., Parlevliet,T., and Danneels,L. (2008) Changes in lumbar muscle activity because of induced muscle pain evaluated by muscle functional magnetic resonance imaging. *Spine (Phila Pa 1976.)* **33**, E983-E989.

Ellis, B. and Bruton, A. (2002) A study to compare the reliability of composite finger flexion with goniometry for measurement of range of motion in the hand. *Clin.Rehabil.* **16**, 562-570.

Emre, M. (1988) Symptomatology of muscle spasm. In: Emre, M. Mathies, H. (eds) Muscle spasm and pain.

Flicker, P.L., Fleckenstein, J.L., Ferry, K., Payne, J., Ward, C., Mayer, T., Parkey, R.W., and Peshock, R.M. (1993) Lumbar muscle usage in chronic low back pain. Magnetic resonance image evaluation. *Spine* **18**, 582-586.

Gardner-Morse, M.G. and Stokes, I.A.F. (1998) The effects of abdominal muscle coactivation on lumbar spine stability. *Spine* 23, 86-92.

George, J.W., Tunstall, A.C., Tepe, R.E., and Skaggs, C.D. (2006) The effects of active release technique on hamstring flexibility: a pilot study. *J Manipulative Physiol Ther.* **29**, 224-227.

Goodgold, J. (1974) Anatomical correlates of clinical electromyography. The Williams & Wilkins Co., Baltimore, MD.

Graven-Nielsen, T., Svensson, P., and Arendt-Nielsen, L. (1997) Effects of experimental muscle pain on muscle activity and co-ordination during static and dynamic motor function. *Electroencephalogr. Clin. Neurophysiol.* **105**, 156-164.

Grondin, D.E. and Potvin, J.R. (2008) Effects of trunk muscle fatigue and load timing on spinal responses during sudden hand loading. *J Electromyogr. Kinesiol*.

Hadjipavlou, A., Enker, P., Dupuis, P., Katzman, S., and Silver, J. (1996) The causes of failure of lumbar transpedicular spinal instrumentation and fusion: a prospective study. *Int.Orthop.* **20**, 35-42.

Hanrahan, S., Van Lunen, B.L., Tamburello, M., and Walker, M.L. (2005) The Short-Term Effects of Joint Mobilizations on Acute Mechanical Low Back Dysfunction in Collegiate Athletes. *J Athl. Train.* **40**, 88-93.

Hides, J.A., Belavy, D.L., Stanton, W., Wilson, S.J., Rittweger, J., Felsenberg, D., and Richardson, C.A. (2007) Magnetic resonance imaging assessment of trunk muscles during prolonged bed rest. *Spine* **32**, 1687-1692.

Hides, J.A., Richardson, C.A., and Jull, G.A. (1996) Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine (Phila Pa 1976.)* **21**, 2763-2769.

Hodges, P. and Moseley, G.L. (2003) Pain and motor control of the lumbopelvic region: Effect and possible mechanisms. *Journal of Electromyography and Kinesiology* **13**, 361-370.

Hodges, P.W. and Richardson, C.A. (1997a) Contraction of the abdominal muscles associated with movement of the lower limb. *Phys.Ther.* **77**, 132-142.

Hodges, P.W. and Richardson, C.A. (1997b) Feedforward contraction of transversus abdominis is not influenced by the direction of arm movement. *Exp.Brain Res.* **114**, 362-370.

Hodges, P.W. and Richardson, C.A. (1997c) Relationship between limb movement speed and associated contraction of the trunk muscles. *Ergonomics* **40**, 1220-1230.

Hodges, P.W. and Richardson, C.A. (1998) Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. *J.Spinal Disord.* **11**, 46-56.

Howitt,S., Wong,J., and Zabukovec,S. (2006) The conservative treatment of Trigger Thumb using Graston Techniques and Active Release Techniques(R). *JCCA.J Can.Chiropr.Assoc* **50**, 249-254.

Howitt,S.D. (2006) Lateral epicondylosis: a case study of conservative care utilizing ART and rehabilitation. *JCCA.J Can. Chiropr.Assoc* **50**, 182-189.

Hultman,G., Nordin,M., Saraste,H., and Ohlsen,H. (1993) Body composition, endurance, strength, cross-sectional area, and density of MM erector spinae in men with and without low back pain. *J Spinal Disord.* **6**, 114-123.

Imrie, R. (2004) Demystifying disability: a review of the International Classification of Functioning, Disability and Health. *Sociol.Health Illn.* **26**, 287-305.

Ingber, R.S. (1989) Iliopsoas myofascial dysfunction: a treatable cause of "failed" low back syndrome. *Arch.Phys.Med Rehabil.* **70**, 382-386.

Janda, V. (1964) Movement patterns in pelvic and hip region in pathogenesis of vertebrogenic disease. Charles University Thesis.

Janda, V. (1978) Muscles, central nervous motor regulation and back problems. In: Korr I M (ed) The neurobiologic mechanisms in manipulative therapy. Pp. 27-41. Plenum Press, New York.

Janda, V. (1986) Muscle weakness and inhibition (pseudoparesis) in back pain syndromes. Modern Manual Therapy of the Vertebral Column. Churchill-Livingston., New York.

Janda, V. (1991) Muscle spasm – a contribution to differential diagnosis. *Manual Medicine* **6**, 136-139.

Janevic, J., Ashton-Miller, J.A., and Schultz, A.B. (1991) Large compressive preloads decrease lumbar motion segment flexibility. *J.Orthop.Res.* **9**, 228-236.

Jemmett,R.S., Macdonald,D.A., and Agur,A.M. (2004) Anatomical relationships between selected segmental muscles of the lumbar spine in the context of multi-planar segmental motion: a preliminary investigation. *Man.Ther.* **9**, 203-210.

Jensen, M.P. (2003) Questionnaire validation: a brief guide for readers of the research literature. *Clin.J Pain* **19**, 345-352.

Jordan, K., Dunn, K.M., Lewis, M., and Croft, P. (2006) A minimal clinically important difference was derived for the Roland-Morris Disability Questionnaire for low back pain. *J Clin.Epidemiol.* **59**, 45-52.

Juker, D., McGill, S., Kropf, P., and Steffen, T. (1998) Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks. *Med.Sci.Sports Exerc.* **30**, 301-310.

Jull,G.J.V. (1987) Muscle and motor control in low back pain: Assessment and management, in: *Physical Therapy for the Low Back. Clinics in Physical Therapy*. L.T. Twomey and J.R. Taylor, eds, Churchill Livingston, New York.

Kamaz, M., Kiresi, D., Oguz, H., Emlik, D., and Levendoglu, F. (2007) CT measurement of trunk muscle areas in patients with chronic low back pain. *Diagn.Interv.Radiol.* **13**, 144-148.

Kazennikov,O.V., Solopova,I.A., Talis,V.L., and Ioffe,M.E. (2007) Anticipatory postural adjustment before bimanual unloading reactions: the role of the motor cortex in motor learning. *Neurosci.Behav.Physiol* **37**, 651-657.

Kimura T,K.M.I.M. (1991) Myofibrous organization in human psoas major muscle. *J Showa Med Assoc* **51**, 509-513.

Kramer, M., Ebert, V., Kinzl, L., Dehner, C., Elbel, M., and Hartwig, E. (2005) Surface electromyography of the paravertebral muscles in patients with chronic low back pain. *Arch.Phys.Med Rehabil.* **86**, 31-36.

Kravitz, E., Moore, M.E., and Glaros, A. (1981) Paralumbar muscle activity in chronic low back pain. *Arch.Phys.Med Rehabil.* **62**, 172-176.

Kumar, S., Narayan, Y., and Zedka, M. (1996) An electromyographic study of unresisted trunk rotation with normal velocity among healthy subjects. *Spine* **21**, 1500-1512.

Lavender, S.A., Marras, W.S., and Miller, R.A. (1993a) The development of response strategies in preparation for sudden loading the torso. *Spine* **18**, 2097-2105.

Lavender, S.A., Marras, W.S., and Miller, R.A. (1993b) The development of response strategies in preparation for sudden loading to the torso. *Spine* **18**, 2097-2105.

Lavender, S.A., Mirka, G.A., Schoenmarklin, R.W., Sommerich, C.M., Sudhakar, L.R., and Marras, W.S. (1989) The effects of preview and task symmetry on trunk muscle response to sudden loading. *Human Factors* **31**, 101-115.

Leahy, M. Active Release Technique. 2009. Ref Type: Personal Communication

Leahy, P.M. (1995) Improved treatments for carpal tunnel and related syndromes. *Chiropractic Sports Medicine* 9, 6-9.

Lee, D.J., Stokes, M.J., Taylor, R.J., and Cooper, R.G. (1992) Electro and acoustic myography for noninvasive assessment of lumbar paraspinal muscle function. *Eur.J Appl.Physiol Occup.Physiol* **64**, 199-203.

Lewis, C.L., Sahrmann, S.A., and Moran, D.W. (2007) Anterior hip joint force increases with hip extension, decreased gluteal force, or decreased iliopsoas force. *J Biomech.* **40**, 3725-3731.

Lund, J.P., Donga, R., Widmer, C.G., and Stohler, C.S. (1991) The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. *Can.J Physiol Pharmacol.* **69**, 683-694.

Magnusson,M.L., Aleksiev,A., Wilder,D.G., Pope,M.H., Spratt,K., Lee,S.H., Goel,v.K., and Weinstein,J.N. Unexpected load and asymmetric posture as etiologic factors in low back pain. Eur Spine J 5[1], 25-35. 1996. Ref Type: Abstract

Magora, A. (1975) Investigation of the relation between low back pain and occupation. VII. Neurologic and orthopedic condition. *Scand.J Rehabil.Med* **7**, 146-151.

Manning, D.P., Mitchell, R.G., and Blanchfield, L.P. (1984) Body movements and events contributing to accidental and nonaccidental back injuries. *Spine* **9**, 734-739.

Marras, W.S., Rangarajulu, S.L., and Lavender, S.A. (1987) Trunk loading and expectation. *Ergonomics* **30**, 551-562.

McGill,S. (2002) *Low Back Disorders. Evidence-Based Prevention and Rehabilitation*. Human Kinetics, Champaign, IL.

McGill,S., Grenier,S., Kavcic,N., and Cholewicki,J. (2003a) Coordination of muscle activity to assure stability of the lumbar spine. *Journal of Electromyography and Kinesiology* **13**, 353-359.

McGill,S., Grenier,S., Bluhm,M., Preuss,R., Brown,S., and Russell,C. (2003b) Previous history of LBP with work loss is related to lingering deficits in biomechanical, physiological, personal, psychosocial and motor control characteristics. *Ergonomics* **46**, 731-746.

McGill,S., Juker,D., and Kropf,P. (1996) Quantitative intramuscular myoelectric activity of quadratus lumborum during a wide variety of tasks. *Clin.Biomech.* **11**, 170-172.

McGill,S.M. and Norman,R.W. (1986) Partitioning of the L4-L5 dynamic moment into disc, ligamentous, and muscular components during lifting. *Spine* **11(3)**, 666-678.

McIntosh,G., Frank,J., Hogg-Johnson,S., Bombardier,C., and Hall,H. (2000) Prognostic factors for time receiving workers' compensation benefits in a cohort of patients with low back pain. *Spine* **25**, 147-157.

Moffroid, M.T. (1997) Endurance of trunk muscles in persons with chronic low back pain: assessment, performance, training. *J.Rehabil.Res.Dev.* **34**, 440-447.

Moore, K.L. (1992) Clinically oriented anatomy. Williams & Wilkins, Baltimore, MD.

Nachemson, A. (1966) [The significance of the psoas muscle for lumbosacral stability. Electromyographic study]. *Nord.Med* **76**, 895.

Nachemson, A.L. and Evans, H.J. (1968) Some mechanical properties of the third lumbar interlaminar ligament. *J.Biomech.* **1**, 211-220.

Navar, D., Zhou, B.H., Lu, Y., and Solomonow, M. (2006) High-repetition cyclic loading is a risk factor for a lumbar disorder. *Muscle Nerve* **34**, 614-622.

Netter F.H. (2003) Atlas of Human Anatomy. Icon Learning Systems, Teterboro.

Nicholas, M.K. (2007a) The pain self-efficacy questionnaire: Taking pain into account. *Eur.J.Pain* **11**, 153-163.

Nicholas, M.K. (2007b) The pain self-efficacy questionnaire: Taking pain into account. *Eur.J Pain* **11**, 153-163.

Nourbakhsh,M.R.A.A.M.S.M. The relationship between pelvic cross syndrome and chronic low back pain. Journal of Back and Musculoskeletal Rehabilitation 19, 119-128. 2006. Ref Type: Generic

O'Sullivan, P.B., Phyty, G.D., Twomey, L.T., and Allison, G.T. (1997) Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine* **22**, 2959-2967.

Okunribido,O.O., Magnusson,M., and Pope,M.H. (2008) The role of whole body vibration, posture and manual materials handling as risk factors for low back pain in occupational drivers. *Ergonomics* **51**, 308-329.

Ostelo,R.W., Deyo,R.A., Stratford,P., Waddell,G., Croft,P., Von Korff,M., Bouter,L.M., and de Vet,H.C. (2008) Interpreting change scores for pain and functional status in low back pain:

towards international consensus regarding minimal important change. *Spine (Phila Pa 1976.)* **33,** 90-94.

Panjabi, M.M. (1992a) The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *J.Spinal Disord.* **5**, 383-389.

Panjabi, M.M. (1992b) The stabilizing system of the spine. Part II. Neurtral zone and instability hypothesis. *J.Spinal Disorders* **5**, 390-397.

Panjabi,M.M., Abumi,K., Duranceau,J., and Oxland,T. (1989) Spinal stability and intersegmental muscle forces: a biomechanical model. *Spine* **14(2)**, 194-199.

Panjabi, M.M. and others (1989) Spinal stability and intersegmental muscle forces. *Spine* 14, 194-200.

Panjabi, M.M. and White, A.A., III (1980) Basic biomechanics of the spine. *Neurosurgery* 7, 76-93.

Parkkola, R., Rytokoski, U., and Kormano, M. (1993) Magnetic resonance imaging of the discs and trunk muscles in patients with chronic low back pain and healthy control subjects. *Spine* **18**, 830-836.

Penning,L. (2000) Psoas muscle and lumbar spine stability: a concept uniting existing controversies. Critical review and hypothesis. *Eur.Spine J* 9, 577-585.

Preuss, R. and Fung, J. (2005) Can acute low back pain result from segmental spinal buckling during sub-maximal activities? A review of the current literature. *Man. Ther.* **10**, 14-20.

Quint,U., Wilke,H.J., Shirazi-Adl,A., Parnianpour,M., Loer,F., and Claes,L.E. (1998) Importance of the intersegmental trunk muscles for the stability of the lumbar spine. *Spine* 23, 1937-1945.

Radebold,A., Cholewicki,J., Panjabi,M.M., and Patel,T.C. (2000) Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine* **25**, 947-954.

Radebold, A., Cholewicki, J., Polzhofer, G., and Greene, H. (2001) Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. *Spine* **26**, 724-730.

Rapley, P. and Fruin, D.J. (1999) Self-efficacy in chronic illness: the juxtaposition of general and regimen-specific efficacy. *Int.J Nurs.Pract.* **5**, 209-215.

Roelen, C.A., Schreuder, K.J., Koopmans, P.C., and Groothoff, J.W. (2008) Perceived job demands relate to self-reported health complaints. *Occup.Med.*(*Lond*) **58**, 58-63.

Roland, M. and Morris, R. (1983) A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low-back pain. *Spine* **8**, 141-144.

Sahrmann, S.A. (1987) Posture and muscle imbalance. Faulty lumbar pelvic alignments. *physical therapy* **67**, 1840-1844.

Sahrmann, S.A. (1992) Posture and muscle imbalance: Faulty lumbar-Pelvic alignment and associated musculo-skeletal pain syndromes. *Orthopaedic Division Review -Physical Therapy* 13-20.

Salvetti, M.G. and Pimenta, C.A. (2007) [Chronic pain and the belief in self-efficacy]. *Rev.Esc Enferm.USP.* **41**, 135-140.

Santaguida, P.L. and McGill, S.M. (1995) The psoas major muscle: A three-dimensional geometric study. *J.Biomech.* **28**, 339-345.

Saunders, D. (2004) Coping with chronic pain: what can we learn from pain self-efficacy beliefs? *J Rheumatol.* **31**, 1032-1034.

Schneider, M.J. (1995) Tender points/fibromyalgia vs. trigger points/myofascial pain syndrome: a need for clarity in terminology and differential diagnosis. *J Manipulative Physiol Ther.* **18**, 398-406.

Scott, J. and Huskisson, E.C. (1976) Graphic representation of pain. Pain 2, 175-184.

Sherrington, C.S. (1947) The Integrative Action of the Nervous System. New Haven.

Stevenson, J.M., Weber, C.L., Smith, J.T., Dumas, G.A., and Albert, W.J. (2001) A longitudinal study of the development of low back pain in an industrial population. *Spine (Phila Pa 1976.)* **26**, 1370-1377.

Stokes, I.A., Fox, J.R., and Henry, S.M. (2006) Trunk muscular activation patterns and responses to transient force perturbation in persons with self-reported low back pain. *Eur.Spine J.* **15**, 658-667.

Stratford, P.W., Binkley, J.M., Riddle, D.L., and Guyatt, G.H. (1998) Sensitivity to change of the Roland-Morris Back Pain Questionnaire: part 1. *Phys. Ther.* **78**, 1186-1196.

Tesh,K.M., Dunn,J.S., and Evans,J.H. (1987) The abdominal muscles and vertebral stability. *Spine* **12**, 501-508.

Thomas, J.S., France, C.R., Sha, D., and Wiele, N.V. (2008) The influence of pain-related fear on peak muscle activity and force generation during maximal isometric trunk exertions. *Spine* (*Phila Pa 1976.*) **33**, E342-E348.

Thomas, J.S., Lavender, S.A., Corcos, D.M., and Andersson, G.B. (1998) Trunk kinematics and trunk muscle activity during a rapidly applied load. *J.Electromyogr.Kinesiol.* **8**, 215-225.

Tidstrand, J. and Horneij, E. (2009) Inter-rater reliability of three standardized functional tests in patients with low back pain. *BMC.Musculoskelet.Disord.* **10**, **5**8.

Travell J., R.S.H. (1952) The myofascial genesis of pain. Postgraduate Medicine 11, 425-434.

van der Burg, J.C.E., van Dieen, J.H., and Toussaint, H.M. (2000) Lifting an unexpectedly heavy object: the effects on low-back loading and balance loss. *Clin.Biomech.* **15**, 469-477.

van Dieen, J.H. and de Looze, M.P. (1999) Directionality of anticipatory activation of trunk muscles in a lifting task depends on load knowledge. *Exp.Brain Res.* **128**, 397-404.

van Dieen, J.H., Kingma, I., and van der, B.P. (2003a) Evidence for a role of antagonistic cocontraction in controlling trunk stiffness during lifting. *J Biomech.* **36**, 1829-1836.

van Dieen, J.H., Selen, L.P., and Cholewicki, J. (2003b) Trunk muscle activation in low-back pain patients, an analysis of the literature. *J Electromyogr. Kinesiol.* **13**, 333-351.

Van Dillen, L.R., McDonnell, M.K., Fleming, D.A., and Sahrmann, S.A. (2000) Effect of knee and hip position on hip extension range of motion in individuals with and without low back pain. *J Orthop.Sports Phys.Ther.* **30**, 307-316.

White, A.A. and Panjabi, M.M. (1978) The basic kinematics of the human spine. A review of past and current knowledge. *Spine* **3**, 12-20.

Wilder, D.G., Aleksiev, A.R., Magnusson, M.L., Pope, M.H., Spratt, K.F., and Goel, v.K. Muscular response to sudden load. A tool to evaluate fatigue and rehabilitation. Spine 21[22], 2628-2639. 1996.

Ref Type: Abstract

Wilke,H.J., Wolf,S., Claes,L.E., Arand,M., and Wiesend,A. (1995) Stabillity increase of the lumbar spine with different muscle groups; A biomechanical in vitro study. *Spine* **20**, 192-198.

Williamson, A. and Hoggart, B. (2005) Pain: a review of three commonly used pain rating scales. *J Clin.Nurs.* 14, 798-804.

Winters, M.V., Blake, C.G., Trost, J.S., Marcello-Brinker, T.B., Lowe, L.M., Garber, M.B., and Wainner, R.S. (2004) Passive versus active stretching of hip flexor muscles in subjects with limited hip extension: a randomized clinical trial. *Phys. Ther.* **84**, 800-807.

Woby, S.R., Urmston, M., and Watson, P.J. (2007) Self-efficacy mediates the relation between pain-related fear and outcome in chronic low back pain patients. *Eur.J Pain* **11**, 711-718.

Wyke,B. (1970) The neurological basis of thoracic spinal pain. *Rheumatol.Phys.Med* **10**, 356-367.

Yoshio, M., Murakami, G., Sato, T., Sato, S., and Noriyasu, S. (2002) The function of the psoas major muscle: passive kinetics and morphological studies using donated cadavers. *J Orthop.Sci.* **7**, 199-207.

Zakas, A., Grammatikopoulou, M.G., Zakas, N., Zahariadis, P., and Vamvakoudis, E. (2006) The effect of active warm-up and stretching on the flexibility of adolescent soccer players. *J Sports Med Phys. Fitness* **46**, 57-61.

Zattara, M. and Bouisset, S. (1988) Posturo-kinetic organisation during the early phase of voluntary upper limb movement. 1. Normal subjects. *J.Neurol.Neurosurg.Psychiatry* **51**, 956-965.

Appendix A



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Letter of Information and Consent

The responses of trunk muscles to unloading perturbations and unstable standing before and after myofascial release of the hip flexor

Investigators: Dr. Daniel Avrahami & Dr. James Potvin

Principal Investigator:

Daniel Avrahami, BPHE, DC Department of Kinesiology McMaster University Hamilton, Ontario, Canada (905) 525-9140 ext. 20175

Research Co-Ordinator Dr. Daniel Avrahami

Research Sponsor: None

Purpose of the Study

The human body controls the muscles surrounding the lumbar joints to provide spine stability. Some of these muscles, such as the hip flexor complex, are extremely tight in low back pain patients. One way to treat these patients is by stretching out the hip flexor complex. The purpose of this study is to examine trunk muscle activity before and after treatment sessions of stretching the tight hip flexor complex.

Procedures involved in the Research

You will be asked to attend four treatment sessions with testing before and after the first and fourth treatment. The testing sessions will require that you to hold a box with a weight you and perform 3 different unloading situations. You will also be asked to stand on an unstable surface surrounded by hand supports. While you perform the task, we will record the activity of your upper body muscles using a technique called Electromyography, which involves recording electrical activity via stick electrodes on your skin. The first and fourth testing/treatment sessions will be approximately 2 hours and the second and third treatment session will be approximately 30 minutes.

Potential Harms, Risks or Discomforts

There is a risk associated with participation in this study. Subjects may experience slight muscle soreness proceeding the testing sessions as a result of perturbations to the spine. There is a risk in aggravating the low back pain subjects symptoms. Although very rare, subjects may experience a temporary reaction to the adhesive from the surface electrodes. Due to the nature of the protocol you will not be allowed to participate if you have been diagnosed with high blood pressure or neurologic deficits and have no structural deformities, genetic spinal disorders, previous spinal surgery or L1, L2 or L3 nerve root involvement.

Potential Benefits

There are benefits to the participants. The participants will recieve treatment that will aim to decrease and hopefully resolve their low back pain. There are potential benefits to the scientific community as this study will quantify the effect of this treatment on low back pain patients and bring attention to the importance of the hip flexor in low back pain patients.

Payment or Reimbursement: No payment will be offered for participation in this study. However, subjects will receive complimentary treatment.

Confidentiality:

Subject identity will be kept confidential and the data collected will be used for teaching and research purposes only. The information directly pertaining to you will be locked in a cabinet for a maximum of 10 years. Information obtained will be kept confidential to the full extent of the law and I will treat all information provided to me as subject to researcher-participant privilege.

Participation:

Your participation in this study is voluntary. If you decide to participate, you can decide to stop at any time, even after signing the consent form or part-way through the study. If you decide to stop participating, your data will be deleted and there will be no consequences to you.

Information About the Study Results:

You may obtain information about the results of the study by contacting Dr. Daniel Avrahami, Dr. Potvin or research lab members.

Information about Participating as a Study Subject:

If you have questions or require more information about the study itself, please contact Dr. Daniel Avrahami or Dr. Potvin.

This study has been reviewed and approved by the McMaster Research Ethics Board. If you have concerns or questions about your rights as a participant or about the way the study is conducted, you may contact:

McMaster Research Ethics Board Secretariat Telephone: (905) 525-9140 ext. 23142 c/o Office of Research Services E-mail: ethicsoffice@mcmaster.ca

See next page for consent form

CONSENT

I have read the information presented in the information letter about a study being conducted by Daniel Avrahami and Dr. James Potvin, of McMaster University. I have had the opportunity to ask questions about my involvement in this study, and to receive any additional details I wanted to know about the study. I understand that I may withdraw from the study at any time, if I choose to do so, and I agree to participate in this study. I have been given a copy of this form.

Name of Participant

In my opinion, the person who has signed above is agreeing to participate in this study voluntarily, and understands the nature of the study and the consequences of participation in it.

Signature of Researcher or Witness

Appendix B

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Group	Sex	Age (yrs)	Height (cm)	Weight (Kg)	Training/Week (Hrs.)	Moment Arm (cm)	Pain Scores (Pre-D1, D5)	Disability Scores (Pre-D1, D5)	Self-Efficacy Scores (Pre-D1, D5)	Sport
A-THF	Male	20	132	88	15	42	0,0	0,0		Wrestling, Jiu-Jitsu
A-THF	Male	22	175.3	81.6	18.5	46	0, 0	0, 0		Football, baseball
A-THF	Male	20	182.9	65.8	15	47	0, 0	0, 0		Cross Country/Track (Running)
A-THF	Male	23	180.3	83	7	41	0, 0	0,0		Runner, Cyclist
A-THF	Male	22	185.4	102.1	12	39	0,0	0,0		Rugby
A-THF	Male	26	175.3	74.8	8	42	0,0	0,0		Biking, Kickboxing
A-THF	Male	22	180	93	7.5	36	0,0	0,0		Lacrosse, Rugby
A-THF	Male	25	170.2	83.9	15	37	0,0	0,0		Wrestling
Control	Male	24	185.4	83.9	10	38	0, 0	0,0		Soccer
Control	Male	21	170	70.5	8	35	0,0	0,0		Kick Boxing, Soccer
Control	Male	30	172.7	64.4	10	35	0, 0	0, 0		Rock Climbing, Mountain Biking
Control	Male	26	175,3	74.8	10	37	0,0	0, 0		Swimming
Control	Male	23	180.3	88.5	3	43	0, 0	0, 0		Ultimate Frisbee
Control	Male	22	185,4	93	8	42	0,0	0, 0		Badminton, Muay Thai
Control	Male	20	190.5	106	12.5	23	0, 0	0, 0		Rugby
Control	Male	21	190	95	б	38	0,0	0,0		Cheerleading
LBP-THF	Male	18	182.9	78.5	20	50	1.5, 0.4	5,3	60, 59	Basketball
LEP-THF	Male	21	175.3	79.4	10	48	2.8, 0.6	1, 1	53, 60	Wrestling
LBP-THF	Male	22	188	131.5	20	50	3.6, 1.2	5, 3	43, 49	Football
LBP-THF	Male	19	133	86,2	8	50	6,6, 3,7	3, 2	60, 60	Football
LBP-THF	Male	21	185.4	92	8	37	4.0, 1.2	5, 2,	59, 59	Rugby
LBP-THF	Male	27	175.3	81.6	б	43	3.6, 1.0	7, 2	47, 50	Hockey, Weight Lifting
LBP-THF	Male	29	155	60	8	42	7.6, 5.0	4,0	36, 50	Wrestling
LBP-THF	Male	17	198.1	93	20	48	2.8, 1.5	4, 3	57, 60	Basketball
LBP-THF	Male	20	182.9	88.5	10	41	5.0, 1.0	5, 3	48, 44	Wrestling
LBP-THF	Malé	28	182	68	8	40	5:3, 2.5	12, 4	37, 43	Triathlon
	AVG	22.7	178.6	84.9	10.9	41.2				
	STDEV	3.3	12.7	14.8	4.7	6.0				

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Appendix C

Group		Age (yrs)	Height (cm)	Weight (Kg)	Training/Week (Hrs.)	Moment Arm (cm)	Pain Scores (Pre-D1, D5)	Disability Scores (Pre-D1, D5)	Self-Efficacy Scores (Pre-D1, D5)
A-THF	AVG	22.5	172.7	84.0	12.3	41.3	0,0	0,0	
	STDEV	2.1	17.1	11.0	4.3	3.9	0, 0	0, 0	
Control	AVĠ	23.4	181.2	84.5	8.4	36.4	0, 0	0,0	-
	STDEV	3.3	7.9	13.9	2.9	6.1	0,0	0,0	
LBP-THF	AVG	22.2	181.3	85.9	11.8	44.9	4.3, 1.3	5.1, 2.3	50,5, 53,4
	STDEV	4.3	11.3	19.1	5.8	4.8	1.8, 1.0	2.8, 1.1	9.5, 6.9

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