

Can Altering Hip Joint Fluid Volume and Intra-Capsular Pressure
Influence Muscle Activation Patterns?
Neuromuscular Implications on Clinical Practice

by
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Author's Declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

I understand that my thesis may be made electronically available to the public.

Abstract

Although the integrated relationship that exists between the lumbar spine and hip joints is frequently acknowledged in scientific journals and by medical professionals, specific functional and injury relationships, are speculative and have not been substantiated. Lumbar spine and hip dysfunctions are suspected to be associated with inhibition of the surrounding extensor musculature, particularly the gluteal muscles, and facilitation of the flexor musculature. This phenomenon has been observed in other joints following effusion and is often termed 'arthrogenic inhibition'. Its apparent occurrence about the hip has never been validated. The primary objective of this thesis was to investigate whether arthrogenic inhibition occurred about the hip. If inhibition was found to exist, its relationship with volume vs pressure was investigated to determine if either of these factors were a more appropriate predictor of inhibition. Finally, compensatory motor patterns in response to apparent inhibition were of interest.

Participants were allocated to the following groups: 1) Control 2) Intervention I (magnetic resonance arthrogram) or 3) Intervention II (therapeutic arthrogram). Electromyography was collected on the rectus abdominis, erector spinae, gluteus maximus and semimembranosus bilaterally during hip rehabilitation exercises prior to and following the intervention. Intra-capsular pressure was measured during the intervention.

The findings provided support for the presence of extensor-inhibition in the hip following infusion of intra-articular fluid with intra-capsular pressure being the most appropriate predictor of the magnitude of inhibition. Hip extensor inhibition appeared to be compensated for by lumbar spine extensors during the selected tasks. Arthrogenic inhibition should be considered in the clinical evaluation and management of patients with hip joint effusions and/or elevated intra-capsular pressure.

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With faith all things are possible. I thank God for placing the following people in my life and orchestrating the events that made this thesis possible.

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Anthony Mascia and his medical team for their assistance with participant scheduling, patient interventions and their unrelenting patience throughout the research collection process. I would like to take this opportunity to offer a special thanks to Anthony for your dedicated attention to this project and to our irreplaceable friendship. The integrity with which you approach your life makes me thankful that you are such an important part of mine.

Finally, thank you to my parents, mom who was continuously pushing so that I would amount to something and dad who would have continuously loved me even if I amounted to nothing. I only hope that my achievements and my character are a reflection of your continued love and support.

"I can do things you cannot, you can do things I cannot; together we can do great things." ~ Mother Teresa

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List of Acronyms

ABD – hip abduction

ASLR – active straight leg raise

CNS – central nervous system

EMG – electromyography

ES – erector spinae (refers to the lumbar erector spinae in the context of this proposal)

EXT – hip extension

FAI – femoroacetabular impingement

GM – gluteus maximus

LBP – low back pain

MRI – magnetic resonance imaging

MRA – magnetic resonance arthrogram

ODI – Oswestry Disability Index

OHS – Oxford Hip Score

PB – pelvic bridge

RA – rectus abdominis

SM – semimembranosus

TA – therapeutic arthrogram

VAS – visual analog scale

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Chapter 1: Introduction

The Scope of the Problem

The overwhelming physical and financial impact of LBP (low back pain) on society is well established. This motivates research and clinical investigations, which attempt to contribute insight into specific injury mechanisms. It is accepted amongst these investigations that the etiology of lumbar spine injuries and pain is multifactorial, consisting of structural biomechanical, neurological, physiological and psychosocial factors (Davis & Jorgensen 2005, Sakamoto et al. 2009). Although there is also strong agreement between scientists and practitioners that lower back pain may precipitate, or be precipitated by, pathology at another joint, this concept has only recently been described as ‘regional interdependence’ (Reiman et al. 2009). In particular, the intimate relationship that exists between the lumbar spine and hip joints is frequently acknowledged in scientific journals and by medical professionals (Reiman et al. 2009). Specific functional and injury relationships, however, are speculative and have not been substantiated. Only within the last couple of years have publications begun to investigate specific questions surrounding this relationship (Reiman et al 2009, Harris-Hayes et al 2009, Sakamoto et al 2009). This thesis represents an attempt to better understand the connections between hip and back pain.

Furthermore, lumbar spine and hip pain are suspected to be associated with inhibition of the surrounding extensor musculature and facilitation of the flexor musculature. This may be considered a broad neurological truism in that this pattern of inhibition-facilitation, often termed arthrogenic or neurological inhibition, has been documented extensively following injuries, surgical intervention and/or intra-articular fluid

administration to the knee, and to a lesser degree in other peripheral joints (Palmieri et al. 2003, 2004, 2005, 2007). Arthrogenic inhibition has been generalized to occur at all joints, but its apparent occurrence at the lumbar spine and/or hip joints has never been validated. This thesis serves as a preliminary investigation providing insight into the underlying mechanisms; specifically, neuromuscular/mechanical compensations that occur in response to changes in hip joint intra-articular volume and pressure (via either magnetic resonance arthrogram or therapeutic arthrogram of the hip) will be investigated.

Purpose & Proposal Hypotheses

This investigation sought to provide preliminary contributions to the integrated relationships between lumbar spine and hip joint functions and related injury mechanisms. Specifically, investigating the transient effects of altering hip joint capsular volume and pressure on activation patterns of the lumbar spine and hip musculature was the primary objective. It is hypothesized that extensor-inhibition may exist about the hip joint with administration of intra-articular joint fluid volume. The secondary purpose of this investigation was to evaluate whether level of inhibition, if present, was associated with fluid volume and/or intra-capsular pressure. It was hypothesized that pressure might be a better predictor of inhibition than volume. The third objective of this thesis was to assess changes in muscular activation in response to noted inhibition of certain musculature. Surrounding extensor musculature was hypothesized to increase in a compensatory manner with inhibition. Finally, comparison of muscle activation patterns about the lumbar spine and hips during frequently utilized rehabilitation exercises between healthy and painful populations was possible.

These objectives were accomplished by assessing hip pathology patients prior to, and following, a scheduled medical arthrogram procedure (magnetic resonance arthrogram or therapeutic arthrogram) and examining whether muscle activation patterns about the lumbar spine and hips were affected. Because these procedures modulate hip joint fluid volume and intra-capsular pressure, it was hypothesized that, if present, these effects would provide insight into the relationships between hip joint fluid, pressure and arthrogenic inhibition.

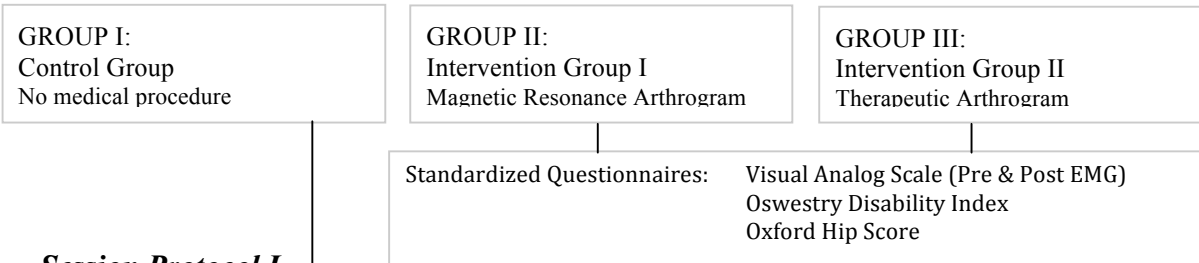
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Thesis Questions:

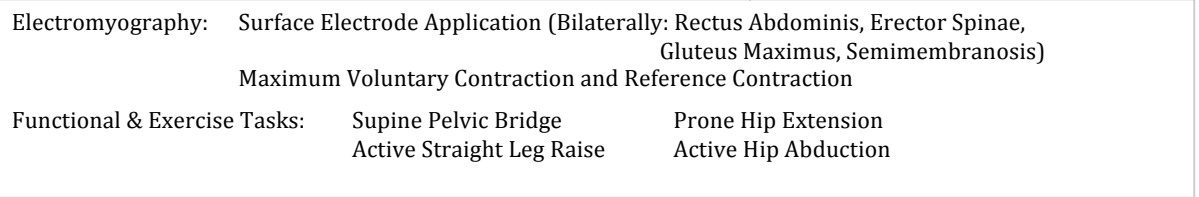
- Does flexion-facilitation and extensor-inhibition occur about the hip joint following induced joint effusion?
- Does the intensity of extensor-inhibition vary proportionally with fluid volume and/or capsular pressure?
- Does extensor-inhibition at one joint result in increased muscle activation at adjacent joints?

Overview of research design

Participant Recruitment

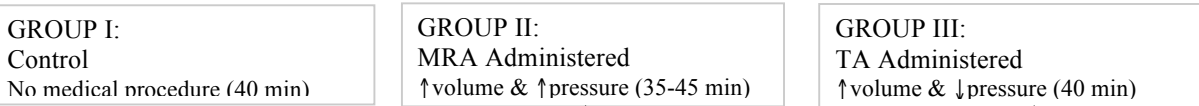


Session Protocol I

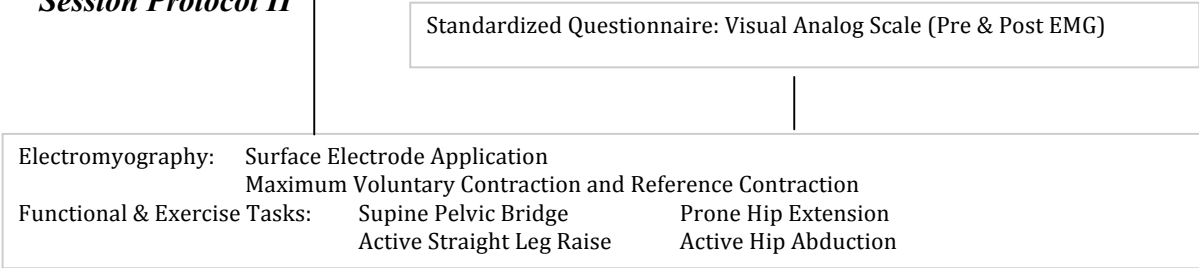


Passive Hip ROM: Flexion, Extension, External Rotation and Internal Rotation

Medical Procedure



Session Protocol II



Passive Hip ROM: Flexion, Extension, External Rotation and Internal Rotation

Figure 1: Schematic overview outlining central research questions & summarizing study designs

GROUP I: Control (No Medical Procedure)

Using healthy normal subjects, it was hypothesized that muscle activation would not differ significantly between the 2 session protocols within muscles. As these were healthy adults performing symmetrical tasks or repeating asymmetrical tasks bilaterally, no differences in muscle activation from right to left sides were anticipated. The results from this control group served to provide baseline values for studies involving pathological participants.

GROUP II: Intervention Group I (Magnetic Resonance Arthrogram Procedure)

A magnetic resonance arthrogram (MRA) procedure of the hip was performed unilaterally. Because it increased hip joint fluid volume and intra-articular pressure, facilitation of the flexors and inhibition of the extensors (ipsilaterally) was expected to occur in Session Protocol II relative to Session Protocol I. Specifically, gluteus maximus activation was anticipated to diminish, while compensatory increases in erector spinae and semimembranosus activation were anticipated.

GROUP III: Intervention Group II (Therapeutic Arthrogram Procedure)

A therapeutic arthrogram (TA) procedure of the hip was performed unilaterally and increased fluid volume in order to release the capsular contracture. Thus, lower intra-capsular pressure was expected. It was hypothesized that the intensity of extensor-inhibition would increase from Session Protocol I to II if arthrogenic inhibition is primarily a result of increased joint effusion. If intra-capsular pressure was a primary contributor to extensor-inhibition, it was also hypothesized that the magnitude of inhibition would decrease from Session Protocol I to II.

Interventions of Opportunity:

It should be noted that this study was not intended to evaluate the clinical efficacy of the described interventions. These procedures have been selected for this use in this thesis because of their effects on intra-articular volume and pressure. In fact, the MRA is considered medically diagnostic while TA is utilized therapeutically. Therefore, these interventions may be considered ‘interventions of opportunity’ for the purposes of this thesis, as their clinical diagnostic and therapeutic effects are not being evaluated directly.

Chapter 2: Review of the Literature

Since this thesis attempted to elucidate potential associations between hip joint fluid, intra-capsular pressure, neuromuscular inhibition and compensatory muscular recruitment patterns, these relevant topics will be introduced and/or critically reviewed from anatomical, biomechanical and neurological perspectives.

General Anatomy: Lumbar Spine & Hip Joints

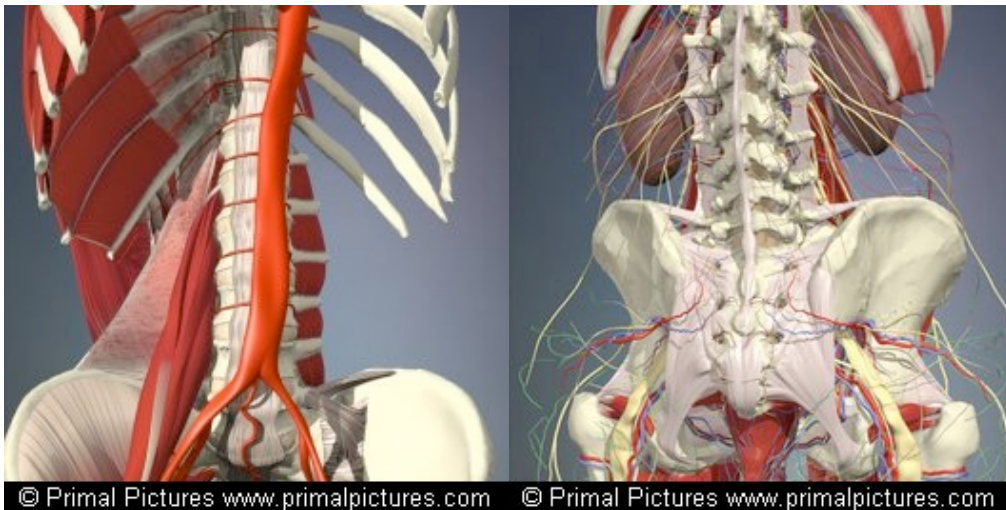


Figure 2: Lumbar Spine Anatomy

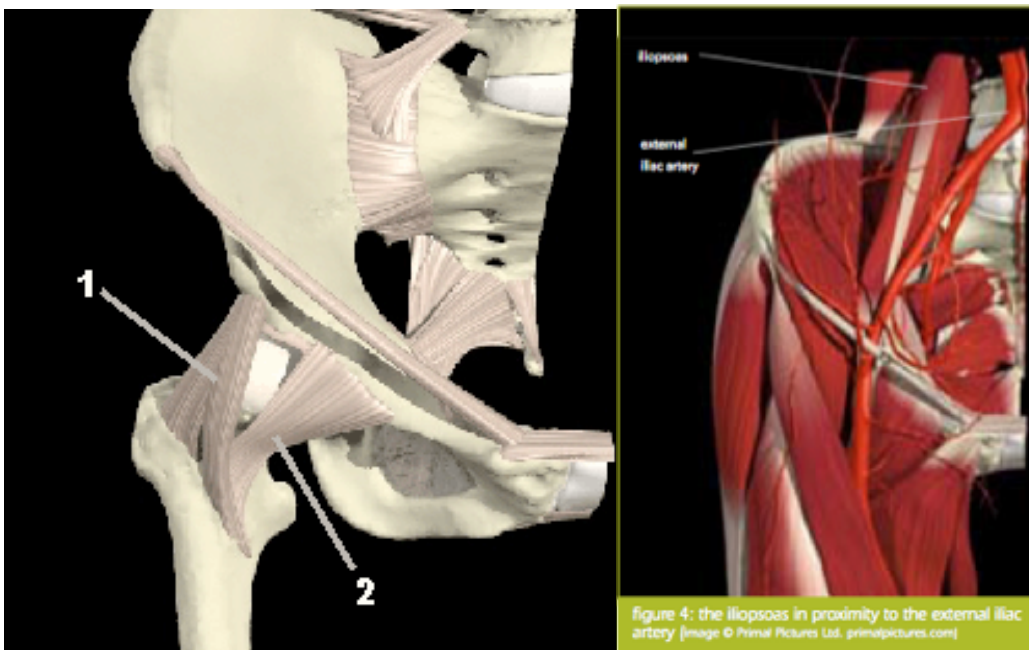


Figure 3: Hip Anatomy

Bony Structures

Lumbar Spine

The lumbar spine consists of 5 vertebrae. The vertebral body is kidney-shaped in the transverse plane and is comprised of cortical bone superficially, which encases the cancellous bone interior. The posterior elements of the vertebrae include the pedicles, laminae, spinous processes and facet joints. The oval to triangular-shaped foramen is formed by the neural arch, comprised of pedicles and laminae. Each vertebrae has 2 transverse processes, which project laterally and posteriosuperiorly and a spinous process, which projects posteriorly. (McGill 2002 and Moore & Dalley 1999).

Hip Joint

The hip joint (ox coxae) is a synovial multiaxial ball and socket articulation (enarthrosis) formed between 2 bony structures: the head of the femur and acetabulum (Torry 2006 & Phillipon 2001). While the femoral head forms approximately 2/3 of a sphere, it contains a flattened portion where it inherits the greatest loads from the acetabulum. The convergence of 3 pelvic bones, the ilium, ischium and pubis, occurs within the acetabulum which is oriented approximately 45° caudally and 15° to 20° anteriorly (Phillipon 2001 & Torry 2006). Decreased acetabular anteversion is considered to be 10° to 14° while increased anteversion is 21° to 25°(Torry 2006).

Cartilaginous Structures: Intervertebral Discs and Acetabular Labrum

Intervertebral Discs

The intervertebral disc is considered to have 3 major components: the nucleus pulposus, annulus fibrosus and end plates. The nucleus pulposus is contained within lamellae of the annulus fibrosus. While the inner fibers of the lamellae attach the superior and inferior end plates, the outer fibers attach the vertebral body. (McGill 2002)

Acetabular Labrum

The cartilaginous labrum of the hip contains circumferential fibers that surround the peripheral portion of the acetabulum, except inferiorly where it blends with the transverse acetabular ligament (also Bowman et al 2010). The labrum consists of triangular fibrocartilage; its base attached to the acetabular rim and femoral surface is concave. Free nerve endings and sensory end organs have been observed to innervate the superficial layers (approximately outer 1/3) of both annular fibers of the intervertebral discs and acetabular labrum (Bogduk 1983 & Phillion 2001).

Ligamentous & Capsular Structures

Ligaments of the Lumbar Spine

The spine has an anterior and posterior longitudinal ligament, which course along the vertebral bodies and their intervertebral discs. The ligamentum flavum also has longitudinal fibers, but lies posterior to the spinal cord. The supraspinous ligament connects the posterior tip of spinous processes of adjacent vertebrae, while the interspinous ligament is oriented obliquely between spinous processes. (McGill 2002)

Hip Joint Capsule & Ligaments

The hip joint fibrous capsule encases the femoral head, acetabulum and labrum. It has 3 primary ligaments: the iliofemoral ligament, pubofemoral ligament and ischiofemoral ligament. The iliofemoral ligament (ligament of Bigelow) lies anteriorly in the shape of an inverted “Y” and is approximately 12-14 mm in thickness. It attaches proximally to the lower portion of the anterior-inferior iliac spine, from which its fibers diverge distally to attach the intertrochanteric line. It may be described as having medial and lateral portions (Phillipon 2001 & Torry 2006 & Martin 2008). The pubofemoral ligament lies in the anterior and inferior aspect of the hip. Proximally, it is attached to the pubic portion of the acetabular rim, then passes below the neck of the femur and blends with the inferior fibers of the iliofemoral ligament. The ischiofemoral ligament originates at the ischial portion of the acetabular rim, has a spiraling pattern before inserting medial to the greater trochanter and along the posterior intertrochanteric line. In contrast to these ligaments that have a longitudinal orientation, a deep layer of fibers, termed the zona orbicularis, may exist in approximately 30 percent of the population within the deep layer of the capsule that encircles the neck of the femur (Kalhor 2009).

Relevant Neuroanatomy of the Hip Capsule

Mechanoreceptors are present in several soft tissues throughout the body and provide afferent information to the central nervous system (CNS), which integrates this afferent input and modulates subsequent efferent responses of joints and muscles appropriately (Dee 1969 and Wyke 1972). The presence and concentration of specific mechanoreceptor types depends on the structure and function of various tissues. Although types I (Ruffini corpuscles), II (Pacinian corpuscles) and IV (free nerve endings) are prevalent in joint capsules throughout the body, the hip capsule contains primarily type I receptors. These

are considered low-threshold, slowly-adapting receptors whose CNS afferents influence muscular tone and perceptual awareness. Surrounding ligamentous structures are prominent in type III, high-threshold, slowly-adapting, receptors whose activity reflexively inhibits prime mover muscles at end ranges of joint motion (Dee 1969, He et al 1998 and Wyke 1972).

Muscular Structures

Although the lumbar spine, pelvic and hip regions contains numerous muscles, this is not intended as a comprehensive review. Shared muscles of the lumbar spine and hip joints, which are relevant to their relationship include psoas major and minor and quadratus lumborum. However, muscles most relevant to this study have been described in further detail and include the primary flexors and extensors of the lumbar spine and hips.

Rectus Abdominis

The rectus abdominis is contained in the anterior abdomen and has vertically oriented fibers that originate on the pubic symphysis and pubic crest and insert on the xiphoid process and 5th – 7th costal cartilages. It is partitioned longitudinally by the linea alba and contains 3 or more transverse tendinous partitions (Moore & Dalley 1999).

Erector Spinae

The erector spinae muscles are considered the intermediate layer of back muscles and lie along the vertebral column. The erector spinae consists of 3 columns (from medial to lateral): spinalis, longissimus and iliocostalis; only the lumbar portions were considered in this thesis. The common origin of the erector spinae muscles is a broad tendon that

attaches inferiorly to the posterior iliac crest, posterior sacrum, sacrospinous ligaments and sacral and inferior lumbar spinous processes (Moore & Dalley 1999).

Iliopsoas

The iliopsoas muscle is comprised of 3 portions that may be considered as separate muscles: psoas major, psoas minor and iliacus.

Psoas Major & Minor

Psoas major originates on the lateral aspect of vertebral bodies T12-L5, their related intervertebral discs and transverse processes and inserts on the lesser trochanter of the femur. Psoas minor originates on the lateral aspect of vertebral bodies T12-L1 and their intervening discs and has insertions on the pectineal line and iliopectinal eminence.

Iliacus

Iliacus originates on the iliac crest, iliac fossa, ala of the sacrum and anterior sacroiliac ligaments, travels distally to merge with the psoas major tendon and inserts on the lesser trochanter of the femur (Moore & Dalley 1999).

Gluteus Maximus

The gluteus maximus originates on the posterior ilium, dorsal sacrum and coccyx and sacrotuberous ligament. Its fibers travel inferolaterally where most insert onto the iliotibial tract, which inserts distally on the condyle of the femur. The remaining gluteus maximus fibers insert onto the gluteal tuberosity of the femur.

Gluteus Medius & Minimus

Gluteus medius and minimus attach the external surface of the ilium between the anterior and posterior gluteal lines and anterior and inferior gluteal lines, respectively. The gluteus

medius inserts on the lateral surface and the gluteus medius on the anterior surface of the greater trochanter.

Semimembranosis

The semimembranosis originates on the ischial tuberosity and inserts distally on the posterior medial condyle on the tibia.

Functional-Anatomical Relationships between the Lumbar Spine & Hips

Dynamic movement that occurs at the lumbar spine and hip joints can be characterized and constrained by the anatomy (Torry et al 2006). Abnormal anatomical structure and/or function of either the lumbar spine or hip joints may lead to abnormal stresses and functioning and facilitate the onset of injury at adjacent joints.

Bony Structures:

Although the lumbar spine affords segmental motions of flexion, extension, lateral flexion and rotation at each intervertebral joint, excessive loading (magnitude or frequency) especially at end ranges of motion, has been shown to be a mechanism for injury. On the contrary, the hip joint possesses inherent stability due to osseous congruency. Nearly all motion between the femoral head and acetabulum is rotation due to its congruency (Bowman et al 2010). Stability depends on 3 biomechanical and geometrical factors relating to the acetabulum: acetabular anteversion, appropriate femoral head-neck-offset and acetabular coverage of the femoral head (Torry et al 2006). Acetabular anteversion is associated with proportional hip flexion and internal rotational capabilities. It is unknown whether reduced anteversion, often related to femoroacetabular impingement (FAI), relates to and increased need for lumbar spine

flexion and/or rotations. The McKibbin instability index was developed in an attempt to predict instability and was based on the sum of femoral and acetabular anteversion angles. Of the 290 hips to which this prediction index was applied, 38% and 6% had low and high indices of instability, respectively (Torry et al 2006). No correlation of these indices with lumbar spine instability exists, although it is assumed that they are inversely proportional. Alterations in femoral head-neck offset and acetabular coverage of the femoral head are also predispositions for FAI. Finally, hip joint stability resulting from osseous congruency also depends on joint position, and is maximalized in a position of flexion and lateral rotation. This is, however, considered the loose-packed position with respect to capsular ligaments (Martin et al 2008 and Philippon 2001). It would be of interest to understand how bony congruency and hip position influence the lumbar spine.

Cartilaginous Structures: Intervertebral Discs and Acetabular Labrum:

Although intervertebral disc injury mechanisms to the lumbar spine are prevalent in the literature, injuries to the acetabular labrum have only recently received increasing attention.

The labrum contributes stability to the hip joint by augmenting the femoral head coverage and creating a seal, which provides negative intra-articular pressure (Bowman 2010).

Damage to the acetabular labrum typically occurs anteriorly and superiorly and is associated with pain in extremes of flexion and extension. However, there are no evaluations in the literature investigating compensatory (likely increased) motions or loads at the lumbar spine during tasks requiring various levels of trunk flexion. If increased spine flexion and extension (magnitude and repetition of motion) are required,

the potential for intervertebral disc damage is increased. The acetabular labrum does not increase contact area, distribute load or reduce contact stresses in the hip during double or single limb stance (Philippon 2001). Similarly to the lumbar intervertebral discs, nerve endings contained in the acetabular labrum appear to serve nociceptive and proprioceptive mechanisms.

Ligamentous & Capsular Structures:

As the specific functions of the lumbar spine ligaments are not directly relevant to this thesis, this section will focus on the hip joint capsule, which will be directly affected. Despite the inherent stability of the hip joint due to bony congruency, the hip joint requires additional passive and active restraints to motion. In the absence of ligamentous restraint, the hip joint subluxes anteriorly in 48° of external rotation. Although the motions prevented by individual ligaments will be described, they are considered to act together to provide stability in all directions of hip motion (Torry et al 2006).

The iliofemoral ligament provides support to the capsule anteriorly and resistance to hip extension beyond neutral. It contributes over 50% of the resistance to external femoral rotation in all angles of hip flexion and extension and to internal femoral rotation in extension (Martin et al 2008, Philippon 2001 and Torry et al 2006).

The pubofemoral ligament supports the capsule inferiorly and anteroinferiorly and resists hip extension and abduction (Torry et al 2006). It also provides resistance to external rotation, particularly in non-neutral hip positions of approximately 30° of flexion and extension (Martin et al 2008).

The ischiofemoral ligament supports and reinforces the capsule posteriorly (Torry et al 2006). It contributes approximately 60% of the resistance to internal femoral rotation in positions of flexion and extension (Martin et al 2008).

Finally, the zona orbicularis may have a constrictive effect within the capsule assisting in maintaining the femoral head within the acetabulum.

Globally, the greatest gains in hip motion when all 4 ligaments are removed are in rotation, implying that they contribute to limiting rotation (Martin et al 2008).

Hip joint position also influences the ligamentous contributions to stability. Since the twisted orientation of the capsular ligaments surrounding the hip joint have a 'screw home' effect in full extension, this is considered the position of maximum ligamentous stability; notice this is in contrast to the position of maximum articular contact. Although rare, the greatest risk for traumatic dislocation of the hip is in a flexed and adducted position (ie. when the joint surfaces are not maximally congruent and the ligaments are close-packed).

Muscular Structures

Iliopsoas

The function of this muscle in the literature continues to be controversial. It is generally agreed upon that it is a hip flexor and may have some influence on the lumbar vertebra and pelvis in maintaining appropriate postures (Torry et al 2006). In the standing position, psoas and iliacus muscles were active in ipsilateral leg extension but contralateral leg extension resulted in selective recruitment of iliacus. Both psoas and iliacus were active during maximal thigh abduction, but no postural activity occurred in either muscle during quiet standing with the trunk flexed 30° at the hip.

In the supine position, iliacus shows notable activity during a 'sit-up in supine' but minimal or no activity in the during the first 30° of hip flexion.

In standing, psoas and iliacus muscles have shown to be active in ipsilateral leg extension, whereas contralateral leg extension has resulted in selective recruitment of iliacus. Both psoas and iliacus were shown to be active during maximal thigh abduction, though no postural activity occurred in either muscle during quiet standing when the trunk was flexed to about 30° at the hip. In the supine position, iliacus shows notable activity during a 'sit-up' but minimal to no activity during the first 30° of hip flexion.

Gluteus Maximus

The gluteus maximus muscle has been shown to be active during extension of the hip and external rotation and abduction against heavy resistance when the hip was flexed to 90°. Significant activation has also been observed while bending forward and standing up from the toe-touching position. Finally, single leg stance activates gluteus maximus ipsilaterally (Joseph & Williams 1957).

Gluteus Medius & Minimus

These muscles are primarily considered hip abductors. In healthy individuals, these muscles have been shown to be 'quiet' (i.e. low activity) during relaxed standing. During single leg stance and gait, it has been hypothesized that they prevent the Trendeleburg sign and contribute to medial rotation and control hip adduction (Joseph & Williams 1957).

Biomechanical Relationships between the Lumbar Spine and Hip Joints

Evidence of Motion Compensations

Reduced joint motion, whether a result of a structural and/or functional limitation, influences motion and motor patterns at surrounding joints. Numerous studies involving surgical joint fusions reveal that radiographic evidence of early degenerative changes in adjacent joints or motion segments is common (Hilibrand and Robbins 2004).

Specifically, this is observed following fusion of 2 vertebral bodies and their intervening intervertebral disc. For example, fusion of the L3-L4 motion segment is expected to result in early degenerative changes in the facet joints and discs at L2-L3 and L4-L5, evident only a few years after fusion (Ghiselli et al 2004 and Yochum & Rowe 2005).

Although outcomes support the success of fusions in providing transient relief of symptoms, resultant degenerative changes are thought to be associated with increased compensatory motion and may later become symptomatic.

Arthrodesis or hip joint fusion procedures were first attempted in 1866 and for 50 years remained the preferred procedure for painful hips. Short-term progress reports were positive as the procedure rendered hip joints stable and non-painful. However, retrospective analyses of patients who underwent arthrodeses 17-50 years previously revealed radiographic signs of early degeneration, often with associated symptoms, in surrounding joints. Over 60% of patients demonstrated ipsilateral knee and lower back pain (most frequently associated with a disc herniation) with average times of onset 23-25 years post-surgery, respectively. These complaints were severe enough to cause patients to seek medical attention. It is of interest that knee pain was more prevalent in labourers, while back pain occurred in workers with sedentary work involving prolonged

sitting. Physical examination demonstrated ipsilateral knee laxity in the anterior-posterior (75%) and medial-lateral (80%) directions (Callaghan et al 1985).

These studies documenting long-term implications of surgical fusions on adjacent motion segments are of use in evaluating the efficacy of specific joint fusions and provide insights into the relationship between lumbar spine and hip joint motion. However, these are based on radiographic findings, clinical assessments and reports. The collection of muscle activation and kinematic data would provide useful contributions to the mechanisms leading to these observed compensatory findings.

Evidence of Muscular Compensations

The influence of pain on muscle recruitment and activation patterns is also relevant to injury mechanisms, particularly of the lumbar spine and hips. A musculoskeletal model was utilized to estimate hip joint forces during prone hip extension and supine hip flexion (Lewis et al 2007). The influence of muscle contributions to force production and hip joint position were evaluated. In particular, when decreased gluteus maximus force production during extension and iliopsoas force production during flexion were modeled, the result was a significant increase in anterior gliding of the femoral head contributing to increased anterior hip joint forces (Lewis et al 2007). Anterior hip joint forces were greater when the hip was positioned in extension irrespective of muscle contributions to force. This has clinical correlations as hip extension and external femoral rotation typically causes pain in patients with anterior hip pain and instability. The information provided by this musculoskeletal model correlates with frequent clinical observations associating lower back and/or hip pain with decreased gluteal contracture, during prone hip extension and supine pelvic bridge tasks for example (Janda 1988). The model

evaluated muscular contributions to force, which would be well complemented by muscular activation investigations during similar tasks.

Neurological Relationships between the Lumbar Spine and Hip Joints

The neurological system is integral in maintaining the appropriate position between the acetabulum and femoral head. This is done through feedback loops that balance neuromuscular regulation voluntarily and involuntarily (Bowman 2010). Because articular and muscular responses are modulated by the CNS, it would be expected that alterations in factors that affect afferent input, such as joint fluid or pressure, might influence these responses.

Arthrogenic (Neurological) Inhibition

Lumbar spine and hip pathologies are suspected to be associated with inhibition of the surrounding extensor musculature and facilitation of the flexor musculature, which further exacerbate the injury process. It is not uncommon for clinicians to report observation of patients holding their affected joint in a flexed posture and/or avoiding joint extension. This pattern of inhibition-facilitation, often termed arthrogenic inhibition, is considered to be a continuing pre-synaptic reaction to the musculature surrounding a joint following distension or damage to the structures of the joint itself (Hopkins et al 2002 & Palmieri et al 2004).

Arthrogenic inhibition has been documented extensively following injuries and/or intra-articular fluid administration to the knee, and to a lesser degree in other peripheral joints. Initially, traditional methods measuring inhibition following knee injuries relied on changes in thigh circumference. When facilitation of flexor musculature was later

realized, needle biopsies and advanced imaging techniques (computed axial tomography and magnetic resonance imaging) confirmed the extent of previously underestimated extensor-inhibition of the quadriceps muscles. Currently, arthrogenic inhibition is characterized by reduced tone and evaluated based on neurophysiological measures, such as H-reflex and M-response, and the amplitude of maximum voluntary contraction (Young et al 1987).

Numerous studies have documented quadriceps inhibition in acute and chronic phases following knee injuries and pathologies, such as anterior cruciate ligament tears, meniscal damage and osteoarthritis (Palmieri-Smith et al 2007, Tarasevicius et al 2007). Despite previous reports that inhibition may be considerable without pain and clinically undetectable effusion, in the aforementioned injuries the presence of pain cannot be eliminated as a potential source of observed inhibition and compensatory mechanisms (Young et al 1987).

Several authors have experimentally induced knee joint effusions in an attempt to remove pain as a contributing factor producing arthrogenic muscle inhibition. Research contributions by Palmieri et al (2003 and 2004) have provided insights in this area.

Extensor-inhibition of the quadriceps muscles was exhibited unilaterally by neurophysiological measures (H_{max}/M_{max} ratio) following subcutaneous injection of 3 mL of lidocaine and intra-articular injection of 60 mL sterile saline. This inhibition was evident at 10, 20 and 30 minutes post-injection relative to the pre-injection baseline values and was isolated to the ipsilateral knee (Palmieri et al 2003). Facilitation responses in the gastrocnemius and soleus musculature, which may contribute to knee flexion have been documented. Following subcutaneous injection of 3 mL of 1% xylocaine and intra-

articular injection of sterile saline, ipsilateral soleus muscle facilitation was shown to occur and persist at 25 min and 45 min post-injection as compared to baseline (Palmieri 2004).

Although studies involving experimentally-induced knee joint effusions eliminate pain as a potential cause of inhibition, the effects of anaesthetic administered (although subcutaneous) may warrant further investigations. Additionally, the relationship between fluid volume and extent of arthrogenic inhibition was not examined. This may be statistically and clinically relevant, as the extent of inhibition has been shown to be proportional to the fluid volume. Specifically, it has been suggested that approximately 30 mL of knee joint effusion is required to selectively inhibit the vastus medialis, while 60 mL also inhibits the vastus lateralis and rectus femoris (Palmieri-Smith et al 2007). Later research studies improved upon previous work and discovered that not only is experimentally induced knee joint effusion associated with extensor-inhibition during passive collection neurophysiological measurements with the participant relatively static, but it may also have functional implications. With the addition of an intervention group (ie. receiving subcutaneous anaesthetic only and categorizing intra-capsular injection participants into 'low' (30 mL) and 'high' (60 mL) effusion groups) and a functional task (ie. single-leg drop landing), further knowledge was obtained. The effusion interventions induced both vastus medialis and lateralis inhibition, but only the 'high' joint effusion also revealed increases in peak ground reaction forces and decreases in peak knee flexion angle and net knee extension moments upon landing (Palmieri-Smith et al 2007). These studies have contributed significantly to establishing the existence of arthrogenic inhibition in the knee and its association with fluid in the absence of

pain. However, fluid has been assumed as the causative factor for the selective inhibition displayed. Intra-capsular pressure was not considered nor measured in these studies even though it typically varies with fluid administration to a joint. Elevated intra-capsular pressure may also contribute to capsular distension, which is deemed to be a factor in the development of arthrogenic inhibition. Furthermore, the presence of arthrogenic inhibition has been generalized to occur at all joints, but its apparent occurrence at the lumbar spine and/or hip joints has never been validated.

Relationship between Intra-Capsular Pressure and Joint Fluid Volume

Hip joint effusion and subsequent intra-capsular pressure may cause pain, limit range of motion, alter muscular recruitment about the hip and elicit compensatory mechanisms across other joints.

Cadaveric investigations have evaluated the relationship between volume of fluid administered to the hip joint and the resulting intra-capsular pressure. The effects of hip joint position on this relationship are also of importance.

Schwarz et al 1988 recorded hip intra-articular pressure following instillations of 2.5 mL, 5 mL, 7.5 mL and 10 mL of Ringer's solution and in 6 hip positions: flexion (45° and 90°), extension, internal rotation to (40°), external rotation to (40°) and abduction to (45°). Hip joint position was the most significant factor influencing intra-capsular pressure. In particular, positions of rotation resulted in the highest pressure, while a flexed posture (45°) produced the lowest pressure values across fluid volumes. A 45° flexed hip posture was considered relieving as it corresponded to the position with the largest capsular volume or lowest intra-articular pressure (Schwarz et al 1988).

In this preliminary work, a neutral (control) hip position was elected for use and therefore depiction of the pressure-volume relationship was not clear and cannot be utilized clinically to predict intra-capsular pressure from fluid volume. It has been reported clinically that at least 10 mL of fluid is required for an injection intended to distend the hip joint capsule in-vivo.

Yen et al 2009 replicated the concept of investigating the relationship between hip joint fluid volume and intra-capsular pressure across hip joint positions cadaverically and produced more clinically useful documentation. Differences in methodology included the use of normal saline, incremental instillations of 2 mL to a maximum volume of 14 mL and 6 slightly different hip positions: neutral (control), flexion (45° and 90°), full internal and external rotation (in 0° hip extension) and full abduction (in 0° hip extension). When the hip was maintained in a neutral (control) position, the intra-capsular pressure was unchanged under 10 mL and then increased exponentially. When compared to neutral hip position and at a volume of 12 mL, positions of full abduction and full rotation (internal and external) increased intra-capsular pressure by 2 and 4 fold, respectively. Specifically, full abduction tightened the ischiofemoral ligament, internal rotation tightened the ischiofemoral ligament and lateral iliofemoral ligament and external rotation tightened the iliofemoral ligament. In contrast, positions of flexion decreased capsular pressure. Hip flexion to 90° resulted in a 19% decrease, while hip flexion to 45° resulted in a 81% decrease in capsular pressure. At 14 mL fluid volume, intra-capsular pressure increased significantly, even in positions of hip flexion; this volume was considered dangerously high (Yen 2009).

These studies confirm that hip position is significant to the relationship between intra-articular hip fluid volume and capsular pressure and that adopting a 45° flexed posture may relieve intra-articular capsular pressure. These investigations are limited by use of a cadaveric population and these findings therefore it may be difficult to generalize to a living population.

Tarasevicius et al (2007) investigated intra-capsular pressure and elasticity of the hip joint capsule in osteoarthritis patients during hip arthroplasty. The intra-capsular pressure was measured peri-operatively following 1 mL incremental instillations of 0.9% saline solution and averaged across 4 hip positions: flexion (45°), extension, internal and external rotation. Similarly to the cadaveric studies conducted, the position of 45° flexion produced intra-capsular pressure values substantially lower than any other hip position.

Although this study was conducted in-vivo, several limitations exist with this investigation. The patient population of interest was those diagnosed with osteoarthritis (OA) and were undergoing arthroscopic procedures. These results may not be generalizable to healthy individuals or those with alternative causes of hip pain.

Collection of muscle activation data associated with intra-capsular pressure and joint fluid volume was not possible in cadaveric studies and has not been considered in studies conducted in-vivo. Since pain, joint effusion and altered intra-articular pressure appear to have contributions to arthrokinetic inhibition and influence alterations in muscular activation, studies investigating muscular activation responses would contribute further insights into the functional implications of these relationships.

Electromyography (EMG)

Electromyography is a technique used to measure the electrical excitation of a specific muscle. It is important to note that EMG cannot provide direct information regarding the amount of force production or moment contribution from a specific muscle. Calculation of muscular force from EMG includes considerations surrounding the maximum voluntary contraction (MVC), maximum producible force by a muscle, physiological cross-sectional area, the length-tension and velocity-tension relationships and the passive elastic component. This underscores the limitation of EMG data in the absence of other collection measures (Torry et al 2006).

Medical Imaging Review

Magnetic Resonance Imaging & Magnetic Resonance Arthrogram

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) and radiography are imaging essentials in evaluating intra-articular hip pathology and extra-articular sources of hip pain (Armfield et al 2006).

MRI is the imaging modality of choice for investigating soft tissue injuries as it has been shown to be reliable in detecting and differentiating between diagnoses with similar clinical presentations. MRI is particularly useful if the following pathologies are suspected: soft tissue inflammation, synovitis, lesions to the articular cartilage, loose bodies, neoplasm, infection and stress fractures.

The sensitivity, specificity and accuracy of MRI have been shown to be 100% for femoral neck fractures. This sensitivity was superior to that of bone scan, which is often used due to accessibility and high sensitivity, but low specificity. In addition, MRI has been shown higher specificity in the detection of avascular necrosis, bursitis or tendinitis (Scopp et al 2001).

Summary Description of Medical Procedures

Magnetic Resonance Arthrogram

Generalized MRI of the pelvis may be useful for evaluating surrounding muscle, tendon and bone marrow abnormalities, but is sufficient for evaluating internal derangement of the hips, particularly the labral structure. A magnetic resonance arthrogram (MRA) is an advanced diagnostic procedure that may be performed by an interventional musculoskeletal radiologist if warranted. High-resolution unilateral direct MRA is preferred for intra-articular hip pain. This involves fluoroscopically-guided injection of the hip prior to MRI. This injection is performed in a hospital setting under sterile conditions with a 22-gauge spinal needle inserted via an anterolateral approach toward the proximal femoral neck. Confirmation of intra-articular needle position should be confirmed prior to instillation of fluid as the intent is to distend the joint capsule, which improves visualization of the underlying structures; the flow of (gadolinium) substance into underlying pathology, such as a labral tear, also highlights pathology on the MR images. Research studies reveal improved detection of intra-articular pathology and a high positive predictive value with MRA over MRI. The sensitivity and specificity of MRA, 90% and 91% respectively, is superior to that of MRI, 30% and 36% respectively (Armfield et al 2006).

Therapeutic Arthrogram

A therapeutic arthrogram (TA) is a moderately invasive procedure selected for the management of contraction or adhesion of the synovial joint capsule. It typically follows a course of unsuccessful conservative care and may prevent surgical intervention. This procedure is also performed in a hospital setting under sterile conditions, and also involves a fluoroscopically-guided injection administered into the hip joint with a 22-gauge spinal needle. In contrast to an MRA, this procedure is used therapeutically for the purpose of releasing capsular contracture and MRI is not performed following the intra-articular injection.

Medical Indications for the Performance of Arthrogram Procedures

Medical indications for MRA include clinical suspicion of labral pathology, synovial disease, intra-articular loose bodies and post surgical consultation. Medical indications for TA include clinically diagnosed adhesive capsulitis. Please see Table 3 for injury descriptions.

A Note on Arthrogram Injections and Time to Fluid Dissipation

Radiological investigations performing repeated MR images over time following an arthrogram injection revealed adequate containment of fluid volume for diagnosis at 1, 2.5 and 4 hours following injection. Full dissipation of fluid volume occurs in approximately 24 hours (Wagner, Wiener et al 2009 and Andreisek et al 2007).

Rehabilitation of Lumbar Spine & Hips: Selection of Exercise Tasks

The importance of rehabilitation of the lumbar spine and hip joints prior to and following injury or surgical intervention is underscored in the literature and clinical settings.

Although surgical and conservative care may be indicated to address some mechanical spine and hip disorders, functional deficits require correction through rehabilitation (Griffin 2001). Furthermore, rehabilitation programs for the lumbar spine and hip joints are typically described and prescribed in isolation despite acknowledgement of their integrated relationship. Knowledge of their inter-related functions would assist in the development of specific rehabilitation and prevention protocols and progressions.

Minimal scientific support exists to support the profound prevalence of their use as both diagnostic and therapeutic tools.

Non-Weight-Bearing Exercises

Numerous studies describe lying leg raises (ie. active straight leg raise in the supine position, active hip abduction performed side-lying and active hip extension in a prone posture) and supine pelvic bridges as introductory rehabilitation exercises (Stalzer et al 2006, Lewis et al 2007, Nelson-Wong et al 2009). As leg lift exercises are recommended for THR or arthroscopy patients 2-3 weeks post surgery and progressed to include double and single leg squat exercises, it is expected they will not be too demanding for the population recruited for this study.

Active Straight Leg Raise (ASLR)

An ASLR is commonly utilized in the clinical diagnoses of lumbar spine pathology, but may provide insights into hip musculature functions. Lewis et al (2007) modeled the effect of decreased iliopsoas contribution to hip flexion force production during an ASLR

to contribute to anterior hip joint forces. The model predicted compensatory increases to flexion force production from ipsilateral tensor fascia latae and sartorius muscles. The observable clinical manifestation thought to be associated with this compensatory recruitment pattern is increased internal femoral rotation during an ASLR.

Prone Hip Extension

The prone hip extension test was described clinically by Janda to evaluate gluteus maximus function as a hip extensor. Visual observation of decreased gluteal contracture was associated with excessive ipsilateral hamstring and contralateral lumbar and thoracic extensor muscular contraction; this pattern was termed ‘Pelvic Crossed Syndrome’.

Recently, decreased gluteus maximus contributions to hip extension force during this exercise were predicted to increase anterior hip joint forces when modeled by Lewis et al (2007). Compensatory increases in hamstring contributions to extension force, particularly semimembranosis, were also predicted. In order to evaluate the hamstrings contribution to extensor force clinically, Janda flexed the ipsilateral knee to 90° in an attempt to exploit the muscle length-tension relationship and reduce the hamstrings as a contributor to force. There has been no scientific quantification to support or deny these theories. EMG investigations into muscle activation during this task would complement the work of Lewis et al (2007).

Active Hip Abduction

Active hip abduction was also described clinically by Janda to functionally evaluate gluteus medius and minimus. Dysfunction or decreased contributions to force in the gluteus medius and minimus were thought to be compensated for by increases in tensor

fascia latae, quadratus lumborum and psoas muscles, manifesting as any of the following: posterior pelvic rotation, increased external femoral rotation or increased hip flexion.

Recently, Nelson-Wong et al (2009) revealed that the active hip abduction test was the only clinical assessment tool that predicted the development of lower back pain during prolonged standing. This test appears to evaluate trunk control during gluteal musculature contracture. Poor active hip abduction performance was correlated with the subjective reports of pain during prolonged standing. Preceding the onset of pain, diminished trunk flexor/extensor co-activation and increased compensatory hip musculature activation was documented (Nelson-Wong et al 2009).

Supine Pelvic Bridge

The supine pelvic bridge, as described by Janda, may be utilized to evaluate gluteus maximus contributions to hip extensor force. A similar description of increased compensatory hamstring and lumbar spine extensor muscle contributions with gluteus maximus dysfunction during a prone hip extension was defined as ‘Pelvic Crossed Syndrome’.

Summarizing Review of the Literature

The existence of a relationship between the lumbar spine and hip joints, which comprises anatomical, biomechanical and neurological components, is well accepted. There is also strong agreement between scientists and practitioners that lower back pain may precipitate, or be precipitated by, pathology at another joint. Although this relationship is frequently acknowledged by researchers in scientific journals and by medical professionals in clinical settings, scientific support for the specific functional and injury mechanisms is scarce.

Lumbar spine and hip pain are suspected to contribute to inhibition of the surrounding extensor musculature and facilitation of the flexor musculature. While this is a neurological “truism” among many clinicians, direct experimental evidence is very circumstantial. There is extensive documentation surrounding ‘arthrogenic inhibition’ in the knee and this has led to generalization of its occurrence at all joints, including the lumbar spine and hips. But his leap remains speculative. Despite progress to demonstrate this pattern of flexor-facilitation and extensor-inhibition in the knee across various conditions, limitations exist in determining the specific mechanism for its existence, as the contribution of certain factors may have been overlooked. When arthrogenic inhibition was first observed clinically following knee injuries and/or corrective surgeries, both pain and joint effusion were considered the causative factors. Subsequent investigations induced experimental joint effusions in the absence of reported pain and documented the persisting presence of arthrogenic inhibition via neurophysiological measurements. These methods were advanced to include observation of arthrogenic inhibition during functional tasks and its effects on an individual’s kinematics and kinetics.

Following the removal of pain as a contributing factor to arthrogenic inhibition, it appears that fluid volume was assumed to be the only remaining contributing factor. However, administration of fluid into a joint may create capsular distension and contribute to alterations in intra-capsular pressure. These factors have never been considered in contributing to arthrogenic inhibition in any joint.

Furthermore, cadaveric investigations of the hip reveal the importance of hip joint position on intra-capsular pressure. Flexion of the hip to 45 ° produces intra-capsular pressure values substantially lower than other postures, including neutral.

Provided that those who present with hip pain and clinical suspicion of gluteal inhibition commonly adopt a flexed hip posture, several considerations are warranted. If the extent of arthrogenic inhibition varies proportionally with intra-capsular pressure, arthrogenic inhibition might be considered a functional adaptation to increased pressure, which predisposes the affected joint to a position that counteracts elevated intra-capsular pressure.

Even if this inhibition is considered functional with respect to intra-capsular hip joint pressure, the implications of diminished gluteus maximum activation and/or force contribution during hip extension warrants investigation. Application of anatomical and biomechanical principles suggests that decreased motion at one joint or motion segment results in increased compensatory motion at adjacent motion segments. This is evident in joint fusion studies that reveal clinical presentations of laxity and radiographic signs of advanced degeneration at motion segments adjacent to fusion. However, how these compensatory increases in motion are produced remains unclear. In summary, this literature review has highlighted what is reasonably well known and what remains clinical speculation. The clinically popular notion of extensor muscle inhibition needs empirical investigation regarding hip and back pain. Clearly hip mechanics and low back mechanics are related. Thus the focus of this thesis was directed towards better understanding whether decreased gluteus maximus activation during activities requiring

trunk extension lead to compensatory increases in hamstring or lumbar spine extensor muscle activation, which may precipitate abnormal stresses and eventual injury.

Chapter 3: Proposed Studies and Methodology

Brief introduction of the methodology

The objective of this thesis was to assess the effects of hip capsule fluid volume and resultant pressure on lumbar spine and hip muscle activity. This necessitated a control group and two experimental groups of convenience formed by patients who were appropriate candidates for undergoing an MRA or TA procedure. The influence of hip capsule volume and pressure on muscle activation profiles were assessed.

A flow chart linking the phases of the study, and the experimental groups, together with an overview of the experimental design is provided in Figure 1.

EXPERIMENTAL PROTOCOL

Participant Recruitment and Group Assignment

Control Group

Nine healthy participants (4 males and 5 females) with an average age, height and body mass of 31.0 ± 5.0 years, 1.76 ± 0.10 m and 72.5 ± 23.6 kg, respectively, were recruited for participation in this study. These participants had no reported history of low back or hip pain requiring medical intervention or time off occupational duties for longer than 3 days. All participants reportedly engaged in physical activity at least 3 days per week and 6 were currently or had previously been involved in competitive sport. Participant recruitment and data collection procedures were performed in accordance with the University's Office of Research and Ethics guidelines.

Intervention Groups

A total of 19 patients scheduled for medical arthrogram procedures performed by Dr. Anthony Mascia, interventional musculoskeletal radiologist at Humber River Regional

Hospital, were recruited to participate in this study. They were assigned to an intervention group, based on the procedure for which they were recommended by a referring physician. Twelve participants (4 males and 8 females) were scheduled for a magnetic resonance arthrogram (MRA) procedure and comprised Intervention Group I. Their average age, height and body mass were 33.6 ± 7.6 years, 174.4 ± 9.5 cm and 71.2 ± 16.5 kg, respectively.

Seven (3 males and 4 females) were scheduled for a therapeutic arthrogram (TA) procedure and were assigned to Intervention Group II. The average age, height and body mass of this group were 38.9 ± 11.5 years, 177.4 ± 7.6 cm and 86.0 ± 18.4 kg, respectively.

Intervention group exclusion criteria included any prior history of lumbar spine or hip surgery.

Standardized Questionnaires & VAS

Samples of the standardized questionnaires are included in Appendix A. The intervention groups were provided with 3 standardized self-questionnaires. The Oswestry Disability Index (ODI) and Oxford Hip Score (OHS) were administered to any participant who reported a history or current episodes of lower back and/or hip pain, respectively. Visual analog scale (VAS) scores were obtained 4 times throughout the study: prior to, and following, each exercise session.

Electromyography

Signal Acquisition

Participants had their skin prepared for electrode placement using standard laboratory protocols of shaving and light abrasion with rubbing alcohol. Pre-cut double-sided

medical grade tape was applied to the Ag-AgCl electrodes (Biometrics DataLOG, Nexgen, Calgary) and then adhered to the skin over 4 muscle groups bilaterally: rectus abdominis (RA), lumbar erector spinae (ES), gluteus maximus (GM) and semimembranosus (SM). The electrodes provided a fixed 2 cm centre-to-centre inter-electrode distance, which remained unchanged across collection sessions. All electrodes were placed over the muscle belly in line with the direction of muscle fibers and were confirmed through combined palpation with applied manual resistance. Table 1 provides a list of the muscles that were investigated and their associated electrode placements and a more detailed diagram of these placements is contained in Appendix B. An adjustable ground (Earthing) strap (R206) was applied to the right wrist throughout EMG collections.

Table 1: Description of Surface EMG Electrode Placements

EMG Lead #	Muscle	Electrode Placement
1 (R), 2 (L)	RA	2 cm lateral to midline and 1 cm above the umbilicus
3 (R), 4 (L)	ES	2 cm lateral to midline at the level of L3
5 (R), 6 (L)	GM	mid-point between sacrum and greater trochanter
7 (R), 8 (L)	SM	mid-point between the ischial tuberosity and crease of knee joint; 1-2 cm medial to midline

Prior to data collection, maximum voluntary contractions (MVCs) were collected according to participant tolerance. Reference contractions were also collected for participants in the intervention groups for appropriate subsequent comparisons. However, the same positions were used for reference contractions and MVCs and all participants were capable of performing an MVC. Because the MVCs produced higher peak levels of EMG as compared to the reference contractions, these were considered to better reflect recruitment capabilities and therefore, used for normalization. Manual resistance was applied to obtain MVCs for EMG normalization with a 10 second ramped contraction in

each of the described positions (Please refer to Table 2). Resting trials of 10 seconds duration were also collected while the participants lay quietly in a supine position; participants were instructed to relax completely for determination of the resting activation level of the monitored muscles.

Table 2: Description of MVC Positions

Muscle(s)	Position
RA	The participant was seated with knees bent and feet flat on the floor. The trunk was positioned 45° to the horizontal.
ES, GM, SM	The participant was positioned prone with the pelvis and thighs on a table. The torso and upper body was positioned off the end of the table and maintained in a position horizontal to the floor.

For MVC contractions, manual resistance was applied (ie. resisted trunk flexion for RA and resisted trunk extension for ES, GM and SM) and the participant generated a maximum voluntary isometric contraction. For reference contractions, the participant was asked to statically hold the above mentioned positions against gravity.

Description of obtaining Maximum Voluntary Contractions and Reference Contractions

The rectus abdominis MVCs were obtained through a modified sit-up position (45° to the horizontal) in which the participants isometrically contracted against manual resistance. Trunk and hip extensor (ES, GM, SM) MVCs were obtained with the prone participants' torso balanced off the end of a bench to which their legs were tightly secured. In this position the participants attempted extend their trunk extension against manual resistance. Following collection of maximum voluntary contractions, the participants were given 3 minutes rest and at which time they were shown the tasks that would be performed.

Signal Processing

The raw EMG signals were collected and pre-amplified with a DataLOG (Biometrics DataLOG P3X8, Nexgen, Montreal, Canada; bandwidth 15-450 Hz, CMRR 92 dB at 60

Hz, input impedance $>10\text{ M}\Omega$) and sampled at a frequency of 2000 Hz using a 13-bit A/D card with a $\pm 3\text{ V}$ range.

The collected data was automatically stored on a 2 GB micro SD memory card and then imported onto a computer using DataLINK software (Version 3.0, 2002). Signals were viewed and exported into excel for conversion from A/D units into Volts.

Customized Labview (Version 8.5, National Instruments) software was to process the data. EMG signals were high pass filtered with a second-order Butterworth filter with a cut-off frequency of 30 Hz (Drake et al 2006). The zero-bias was subtracted from the raw signals before full-wave rectification. Signals were linear enveloped using a second-order low-pass Butterworth filter with a cut-off frequency of 2.5 Hz then normalized to the peak amplitude of a maximum voluntary contraction.

All trials collected were normalized to the initial MVC trials collected as it was noted that patients were not providing comparable maximal efforts following the administered intervention. This was evidenced by differing levels of raw muscle activation during collection of MVCs prior to the post-intervention exercise session; relatively lower and higher levels were displayed following the intervention in Intervention groups I and II, respectively. Participants appeared hesitant following administration of the intervention likely due to fear of pain production. However, the consideration that the changes observed in raw EMG during the second exercise session could be a direct result of the intervention is warranted. If an intervention alters the magnitude of raw EMG during an MVC, which is intended to represent the maximum capabilities of an individual, this may provide insights into impact of the intervention on an individual's activation capabilities, Given this, the raw EMG collected during MVC trials reveals that the activation

capabilities of participants diminished following Intervention I, but elevated following Intervention II. The fact that maximum capabilities were influenced by each intervention is important and therefore, normalizing all exercise trials to a consistent baseline was considered more appropriate for comparing muscle activation patterns across sessions. The peak levels of activation displayed in the pre-intervention MVC collection session were considered representative of the participants' maximum activation capabilities upon arriving to participate in the study and were likely a more reasonable representation of their capabilities leading up to the intervention procedure. Hence, these trials were used for normalization of exercise tasks from both sessions.

Surface electrode removal was required for the intervention participants in accordance to sterile procedures guidelines, to perform the arthrogram procedures. Electrode placement was traced onto the skin in the control and intervention groups prior to electrode removal at the conclusion of the first exercise session protocol. Tracings were used for re-application of surface electrodes for during the second collection session.

The 3 trials collected for each task were averaged for each participant. In order to do this, customized Labview programs were used initially to define the start and end points of individual repetitions. The 'start' was considered to occur when the first of the extensor muscles (ie. ES, GM or SM) was activated above 5 % of MVC and remained above this level for 1000 frames, while the 'end' of the repetition was defined as the point at which all extensor musculature decreased below 5 % of MVC and remained below this level for 1000 frames. Individual and group mean and maximum values were then calculated.

Maximum values were examined because they appeared most appropriate to represent 'neurogenic inhibition', which has been characterized by diminished maximum

(electrically stimulated and/or actively generated) muscle activity in previous research. As mean EMG was considered relevant to clinical interpretation and was less variable than the peak values, it was also analyzed. As the maximum and mean values provided similar results for interpretation, the peak values and associated statistical analyses were presented.

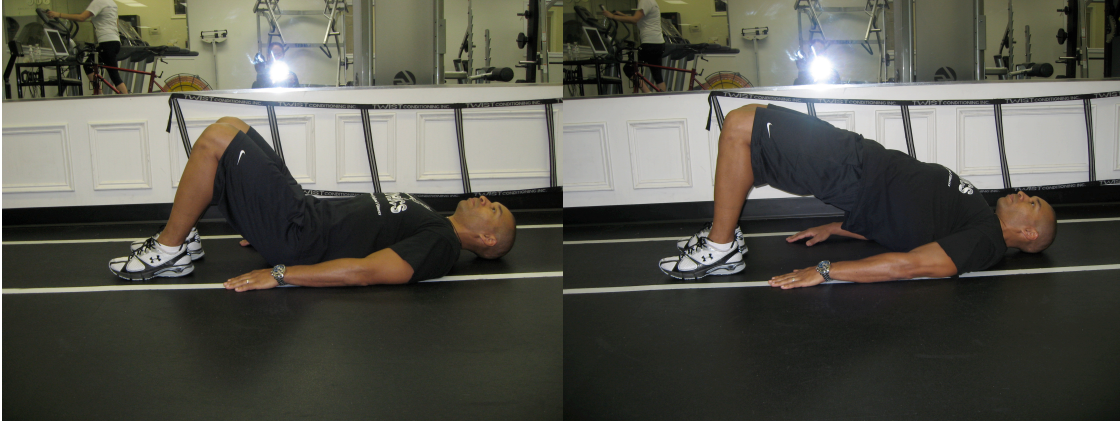
Rehabilitation and Functional Exercise Session Protocols

Selection of tasks for this thesis was based on introductory motions or exercises frequently used in clinical settings in the evaluation and rehabilitation of low back and/or hip injuries.

Participants were asked to perform 3 repetitions each of the following functional and exercise tasks prior to and following intervention administration:

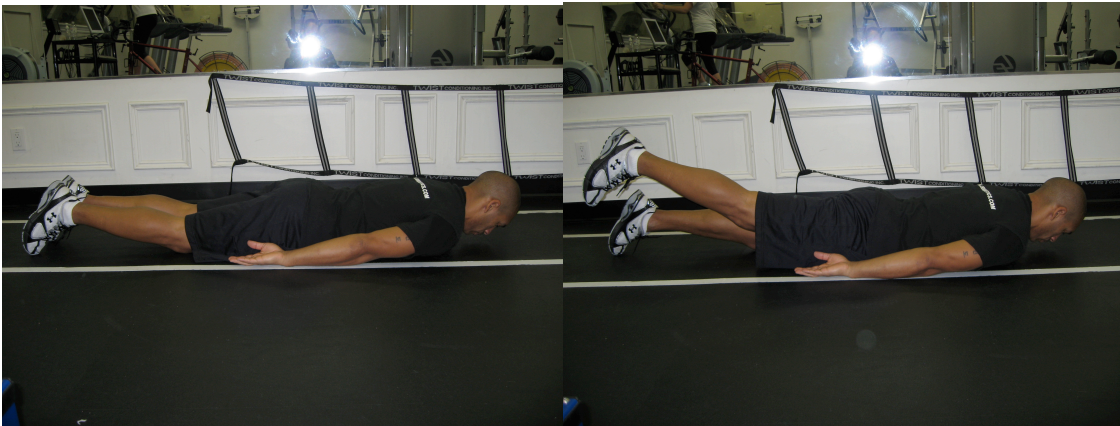
- 1) Supine pelvic bridge (PB)
- 2) Prone hip extension (EXT)
- 3) Active straight leg raise (ASLR)
- 4) Active hip abduction (ABD)

Rehabilitation and Functional Exercises



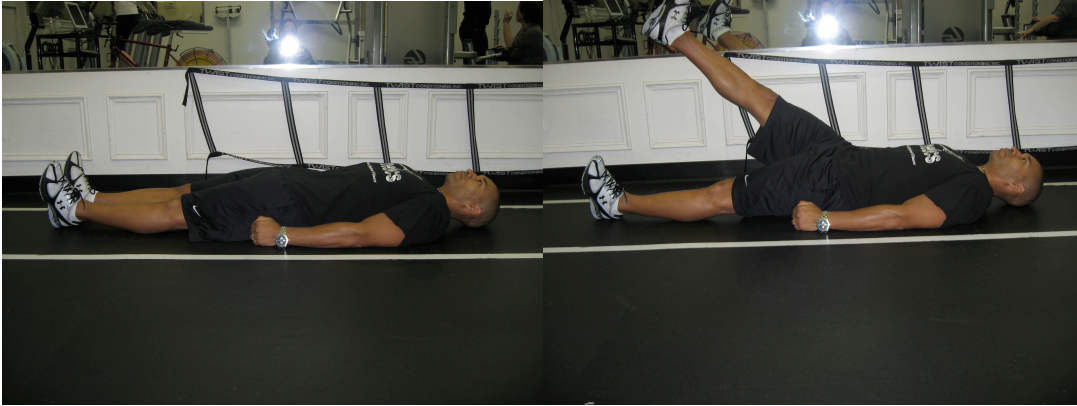
Pelvic Bridge

The participant lay in the supine position with their hips in 45° of flexion and their feet on the floor. The pelvis was lifted off the floor to neutral hip position or within participant tolerance.



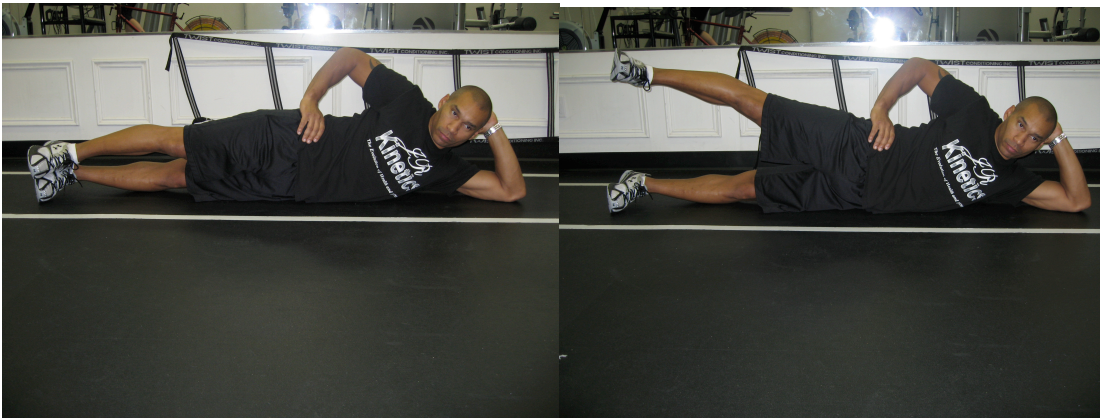
Prone Hip Extension

The patient was positioned prone and slightly flexed at the hips on a bench (not shown above). One leg is raised (i.e. hip extended) while maintaining knee extension.



Active Straight Leg Raise

The participant lay in the supine position. One leg was raised to 90 degrees of hip flexion or within patient tolerance while maintaining knee extension. The hip flexion angle at which the lumbar spine began to flex was recorded (based on clinical observation). This was performed bilaterally.



Active Hip Abduction

The participant was positioned in side-lying and the top leg is raised to approximately 45° while maintaining knee extension.

Intervention: Control Wait Time or Medical Procedure

Control

The control participants were asked to wait in the supine position for 40 minutes. This position was similar to that adopted by those participants in either intervention group.

Medical Arthrogram Procedures

The MRA and TA procedures were conducted in accordance with standard hospital procedures and remained completely unaffected by conduction of this research. Below is an illustration of the procedure room:

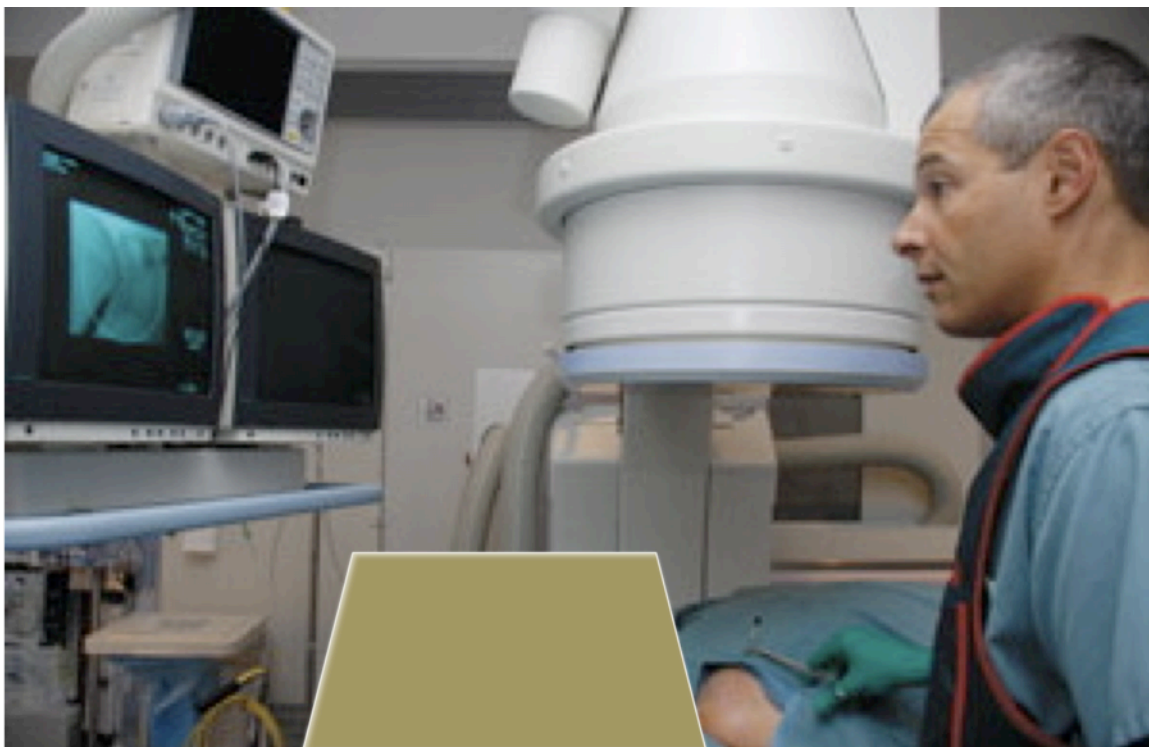


Figure 4: Illustration of Arthrogram Procedure Room, including Fluoroscopic Imaging Equipment with Imaging Display and Mean Arterial Blood Pressure Device for Obtaining Intra-capsular Pressure.

Magnetic Resonance Arthrogram

Participants were escorted to the sterile procedures room by the appropriate medical staff where they were prepared (ie. gowned, local skin sterilization, medical procedure informed consent obtained). Under fluoroscopic guidance, a sterilized 22-gauge spinal needle was inserted into the hip joint, by our interventional musculoskeletal radiology specialist, using an anterolateral approach. This was attached to an arterial blood pressure monitor via 3-way stop-cock in order to capture instantaneous intra-capsular pressure values throughout the procedure. A 0.5% solution of xylocaine was injected into epidural and subcutaneous layer. Avoidance of deeper injection to muscle and joint was given careful attention. Next, confirmation of needle position into the hip joint was confirmed via injection of 1-2 ccs of fluoroscopic radiopaque contrast, Omnipaque (Optiray 320). Upon confirmation of intra-articular needle position, a pressure reading was obtained and recorded in mmHg; this was considered the “Opening Pressure”. A 250:1 ratio of sterile saline and MR contrast, Gadolinium (Magnivist 460), solution was instilled directly into the joint. Instantaneous intra-articular pressure values and their corresponding fluid volumes were acquired and documented at intervals of every 1-5 ccs of fluid administered. As near full capsular distension was observed visually and determined by clinical expertise, 1-2 cc’s of the radiopaque contrast was injected to confirm that the position within the joint was maintained. At this time the “Final Pressure” value was taken. Finally, the patient was assisted by medical staff and transported via wheelchair to the magnetic resonance imaging tube. Magnetic resonance images of the hip were taken as medically indicated. This medical procedure, including preparation, injection and imaging, typically took approximately 40 minutes.

Therapeutic Arthrogram

Participants were escorted to the sterile procedures room by the appropriate medical staff where they were prepared (ie. gowned, local skin sterilization, procedure informed consent obtained). Under fluoroscopic guidance, a sterilized 22-gauge spinal needle was inserted into the hip joint, by our interventional musculoskeletal radiology specialist, using an anterolateral approach. This was attached to an arterial blood pressure monitor via 3-way stopcock in order to capture instantaneous intra-capsular pressure values throughout the procedure. A 0.5% solution of xylocaine was injected into epidural and subcutaneous layer. Avoidance of deeper injection to muscle and joint was given careful attention. Next, confirmation of needle position into the hip joint was confirmed via injection of 1-2 ccs of fluoroscopic radiopaque contrast, Omnipaque (Optiray 320). Upon confirmation of intra-articular needle position, a pressure reading was obtained and recorded in mmHg; this was considered the “Opening Pressure”. Similar to an MRA, a 250:1 ratio of sterile saline and MR contrast, Gadolinium (Magnivist 460), solution was instilled directly into the joint. Dissimilar to the MRA procedure, the TA intends to release capsular contractures or adhesions. Thus, it was necessary for the needle position to be adjusted throughout the procedure toward constricted areas and for air to replace Gadolinium fluid volume at later stages of the procedure; this was all done at the discretion of the radiologist. Instantaneous intra-articular pressure values and their corresponding fluid/air volumes were acquired and documented at intervals of every 1-10 cc’s of fluid/air administered. In order for release or rupture to be achieved, fluid/air volume was injected beyond capsular distension. When the procedure was deemed sufficient by the radiologist, 1-2 cc’s of the radiopaque contrast was injected to confirm

that the position within the joint was maintained. At this time the “Final Pressure” value was taken.

Clinical notes recorded with the arthrogram procedures included: affected side, fluid/air volume injected, instantaneous intra-capsular pressures (at least “Opening” and “Final” pressures), psoas adherence (or lack of adherence) to anterior hip capsule. Final radiographical diagnoses and time taken for medical procedures were also recorded.

Upon completion of the medical procedure, participants were returned to the ‘Nurses Holding Area’ where the remainder of the study took place.

Statistical Analyses

SAS version 9.1 (SAS Inc., Toronto, Ontario, Canada) was used for all statistical analyses. The EMG data were analyzed considering the within factors of session and side and the between factor of group, where indicated. Regression analyses were performed to evaluate the relationship between changes in EMG and changes in pressure and volume. The level for significance was set at $p < 0.05$ for all statistical tests unless otherwise stated. Consultation and assistance with the statistical analyses was provided by Erin Harvey, University of Waterloo, Department of Statistics and Actuarial Science.

Step I: Muscle Activation and the Control Group

The intent was to reveal inherent differences in a healthy control population. If no significant differences were observed, then it appeared a more reasonable assumption that any changes observed in the intervention groups are related to the intervention, rather than occurring by chance.

A 2-way repeated measures ANOVA was used to evaluate the inherent variability in the control group to establish normal values for a healthy population. This analysis included independent variables, session (2 levels: pre and post) and side (2 levels: right and left). The dependent variable, peak level of muscle activation (expressed as a % MVC), was measured for each muscle across all tasks.

Step II: Comparison of Muscle Activation in the Control and Intervention Groups

The intent was to examine whether differences were present between the control and intervention groups. In order to compare the control group to the intervention groups, the average of 'right' and 'left' sides were calculated for the control group (as no differences were observed in Step I and was considered to represent the 'affected' (ipsilateral) and

‘unaffected’ (contralateral) side. Whether the ‘unaffected’ side could be considered a within-subject control was also of interest. Changes occurring on the unaffected side could represent a ‘carry-over’ effect of inhibition or compensatory activation, for example.

Control and treatment groups were analyzed using a 2-way repeated measures ANOVA. This included the between-factor, intervention (3 levels: Control, Intervention I and Intervention II) and session (2 levels: pre and post). Each side (2 levels: affected and unaffected) was considered separately and the p-values were Bonferoni corrected ($0.05/2$) to 0.025. Tukey post-hoc tests were performed on main effects involved in the significant interactions. Post-hoc testing enabled comparisons of peak values within a group over time and comparison of all groups at the same time. Peak EMG was the dependent variable in the analysis.

Step III: Inhibition-Volume vs Inhibition-Pressure Relationship

Simple linear regression analyses were used to evaluate the relationship between 1) inhibition and change in volume and 2) inhibition and change in pressure. The change in peak gluteus maximus EMG within a task across sessions was considered to represent levels of inhibition or release of inhibition. The association between this value and change in volume and change in pressure recordings was investigated.

Step IV: Intervention Groups VAS Scores and Passive ROM

VAS scores were analyzed using a way 1-way ANOVA, within factor session (pre and post intervention) on each side (2 levels: affected and unaffected).

Passive ROM was analyzed using a 2-way ANOVA, with between factor of group (2 levels: Intervention I and Intervention II) and within factor, session (2 levels: pre and post

intervention). Tukey post-hoc tests were performed on main effects in significant interactions.

Chapter 4: Experimental Results

The results are presented in sections, starting with the questionnaires (VAS, OHS and ODI) and passive hip ROM values. Next, EMG data is displayed, followed by the Pressure-Volume-Inhibition results.

Standardized Questionnaires

Visual Analog Scale

In the majority of cases, the individual VAS scores were unchanged throughout the study (Intervention I, $p=0.07$; Intervention II, $p=0.9995$). Each time a score was obtained (ie. prior to and following 2 distinct EMG collection sessions), the participant was at rest in a seated position. The average VAS score and SD (standard deviation) per group during each session were as follows:

Table 2: Summary of Average VAS scores

Intervention Group	Time	VAS Score: Hip	SD
I	1	0.82	1.78
I	2	0.82	1.78
I	3	0.82	1.78
I	4	0.73	1.79
II	1	2	3.42
II	2	2.13	3.64
II	3	2	3.42
II	4	2.13	3.68

Oswestry Disability Index and Oxford Hip Score

All the participants within the Intervention Groups reported hip pain or disability and filled out the OHS questionnaire. Many participants relayed that they had minimal pain at rest and/or only with intensive work or athletic activities. Interestingly, the average OHS for both intervention groups indicated minimal functional disability and. Intervention Group I contained 4 participants who reported a history of low back troubles, while there

were 5 participants in Intervention Group II. Participants who reported no history of back pain were assigned a score of 0 for percentage of disability.

The average ODI and OHS and associated SD are shown below for each group:

Table 3: Summary of ODI and OHS scores

Intervention Group	Average ODI Score	SD
I	12.73	20.73
II	22.5	22.52

Intervention Group	Average OHS	SD
I	42.64	4.27
II	40.78	4.50

Passive Range of Hip Motion

On the affected hip, main effects of group nor session were observed for the intervention groups, but interactions indicated the ROM in each group were different over time in flexion ($p < 0.0001$), extension ($p < 0.0001$), ER ($p = 0.0005$) and IR ($p = 0.0030$).

Intervention group I showed differences in ROM from session 1 to 2 in flexion ($p = 0.0247$) and ER ($p = 0.0210$) but not in extension (0.0766) and IR ($p = 0.1677$).

Intervention II revealed differences in flexion ($p < 0.0001$), extension ($p < 0.0001$), ER ($p < 0.0001$) and IR ($p < 0.0001$).

No differences in ROM were seen between the groups over time in the unaffected hip of Intervention Group participants (flexion $p = 0.379$, extension $p = 0.7274$, ER $p = 0.2123$ and IR $p = 0.7163$). The direction of change was opposite for the interventions, with ROM decreasing after Intervention I and increasing after Intervention II.

The average hip ROM are displayed in Figure 4 and values provided in Appendix C.

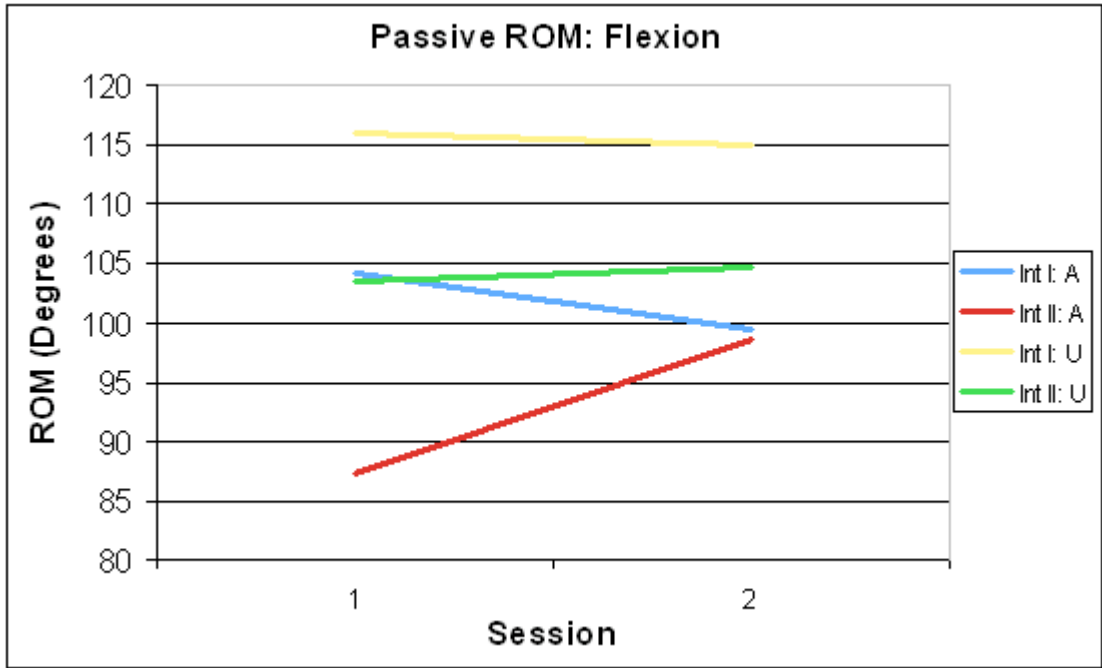


Figure 5a: Group Average Passive Hip ROM Prior to and Following the Intervention. Flexion Values for the Affected (A) and Unaffected (U) Hip are Displayed.

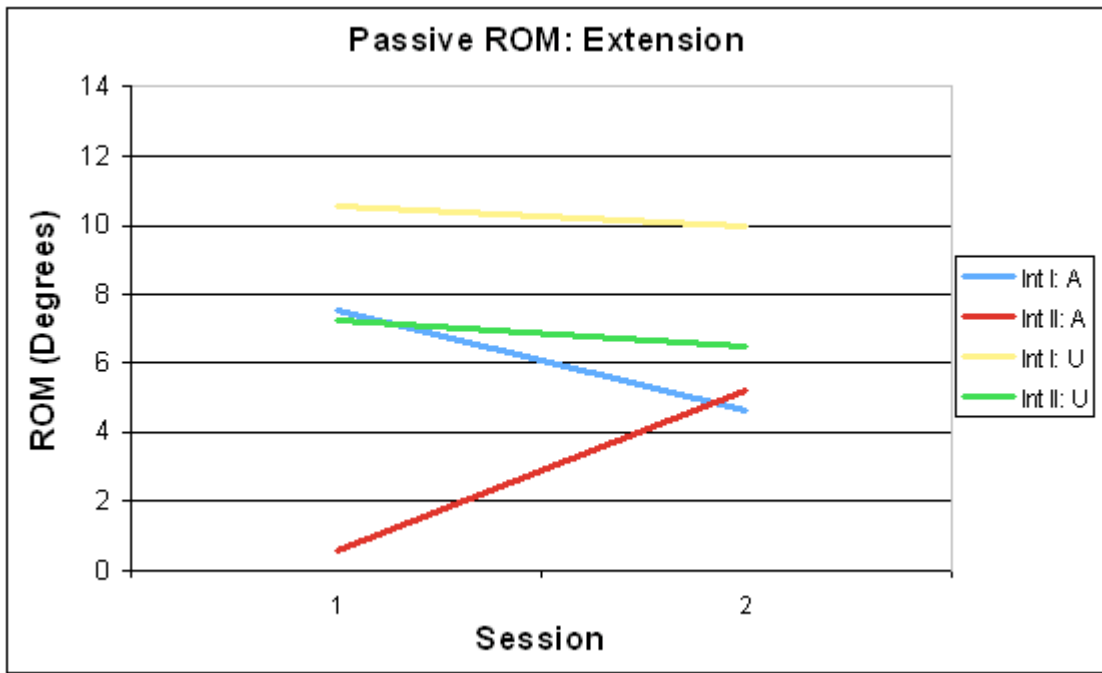


Figure 5b: Group Average Passive Hip ROM Prior to and Following the Intervention. Extension Values for the Affected (A) and Unaffected (U) Hip are Displayed.

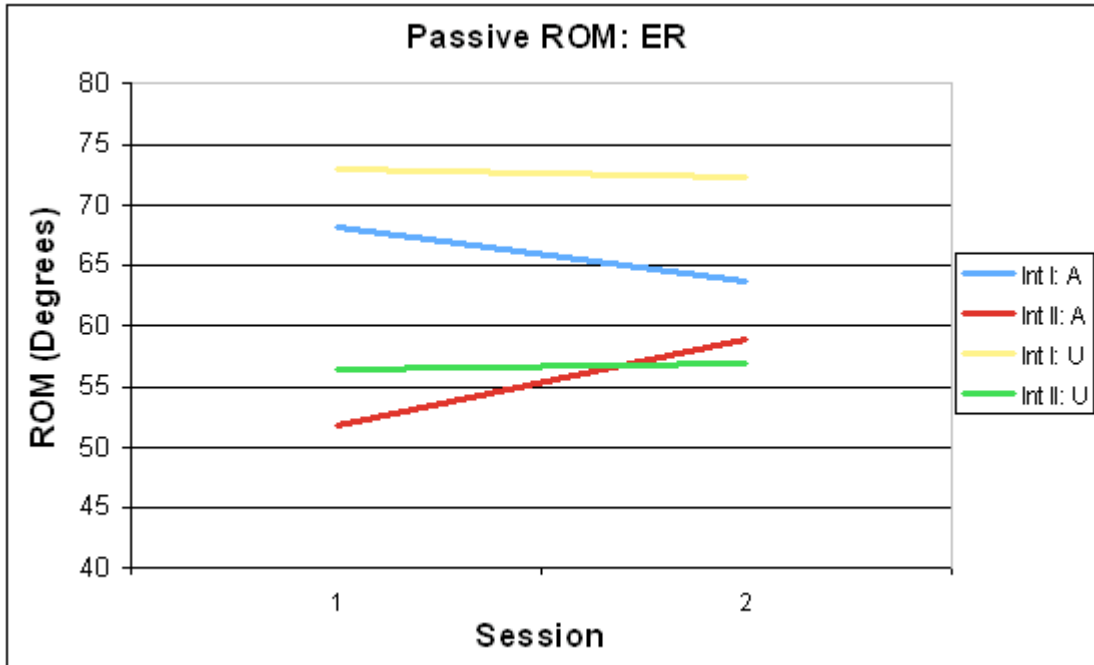


Figure 5c: Group Average Passive Hip ROM Prior to and Following the Intervention. External Rotation Values for the Affected (A) and Unaffected (U) Hip are Displayed.

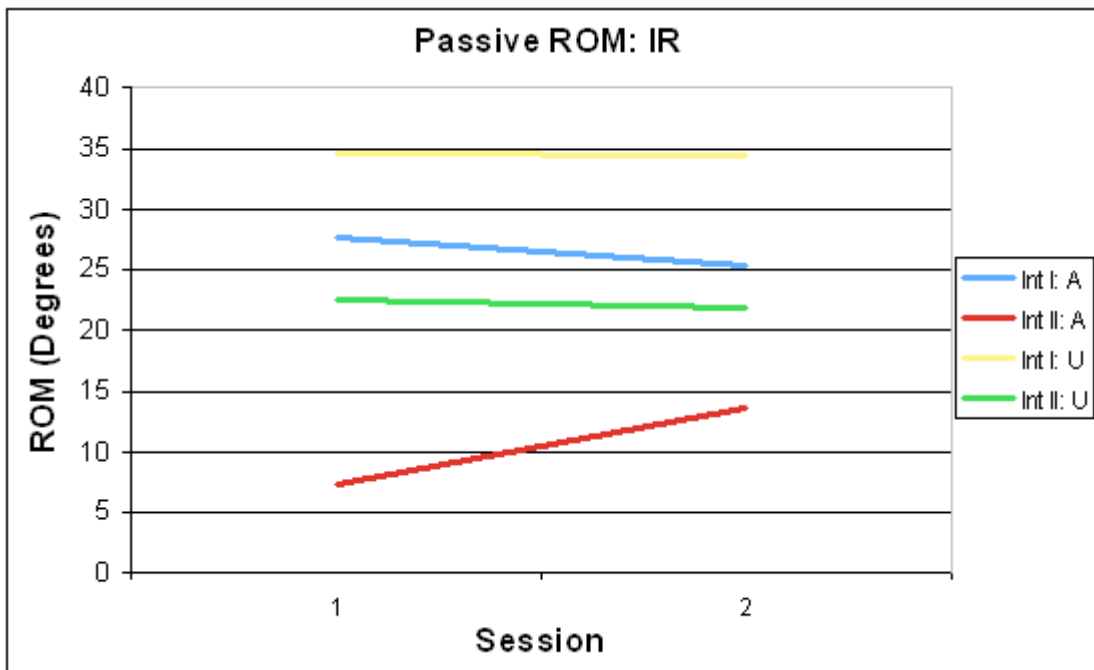


Figure 5d: Group Average Passive Hip ROM Prior to and Following the Intervention. Internal Rotation Values for the Affected (A) and Unaffected (U) Hip are Displayed.

Electromyography

Control Population

Analyses revealed no main effects of side or and no side by session interaction for GM (side $p=0.3830$, session $p=0.9893$, session by side $p=0.7148$), ES (side $p=0.1637$, session $p=0.6179$, session by side $p=0.8356$) or SM (side $p=0.1726$, session $p=0.7872$, session by side $p=0.5110$) during PB; the same was observed during EXT, ASLR and ABD exercises. Side and session were independent variables considered, while peak muscle activation was the dependent measure. This was expected and was important to enable investigation of the rest of the data set. For asymmetrical exercises (EXT, ASLR and ABD), the activation of the elevated leg musculature (GM, ES, SM) differed from that of the stationary leg, although side comparisons were performed during equitable activities (ie. the right GM during right hip extension was compared to the left GM during left hip extension, for example).

Comparison of Control and Intervention Groups

As no side differences were observed in the analysis of the control group, the left and right sides were averaged and used for comparison to the intervention groups. This average was used to represent both the 'affected' and 'unaffected' sides, since these classifications did not exist in the control group participants. Combined analyses of the control and both intervention groups did not reveal main effects of session or group. Any exceptions have been noted in the appropriate sections below.

Pronounced differences exhibited in muscle activation between the control and intervention groups over time have been reported. These differences occurred primarily on the side of the body ipsilateral to the administered intervention. Group and session were

independent variables considered, while peak muscle activation was the dependent measure

Gluteus Maximus

There were no main effects of group or session observed during the PB (affected/unaffected: group $p=0.7428/0.3164$, session $p=0.5269/0.6974$) or EXT (group $p=0.2554/0.1228$, session $p=0.5737/0.0956$). An interaction of group by session was significant only on the affected side during both the PB (affected/unaffected $p=0.0192/0.9654$) and EXT ($p<0.0001/0.0826$) on the affected side. Post hoc testing on the significant interaction revealed that the affected gluteus maximus diminished over time in Intervention I (PB/EXT $p=0.0238/<0.0001$) and increased in Intervention II (PB $p=0.0076/<0.0001$). This supports the primary hypothesis inherent in this thesis investigating the presence of extensor-inhibition about the hip joint. A sample summarizing all participant EMG values can be seen in Appendix D, while the group average of peak activation levels are displayed in Figures 6 (PB) and 7 (EXT).



Figure 6a: Group average of GM peak muscle activation on the affected hip of participants while performing the PB

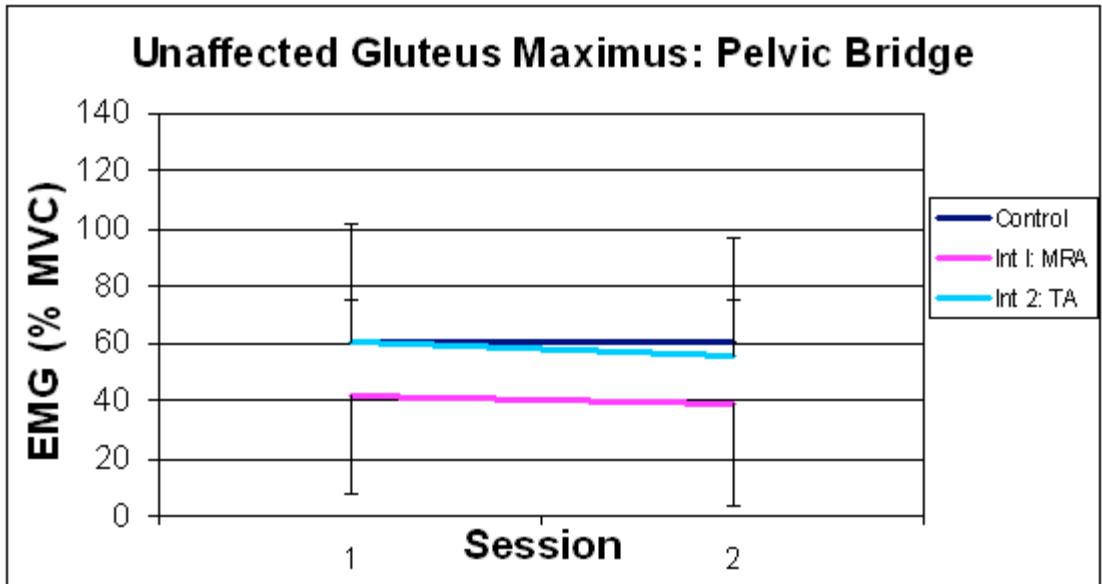


Figure 6b: Group average of GM peak muscle activation on the unaffected hip of participants performing the PB

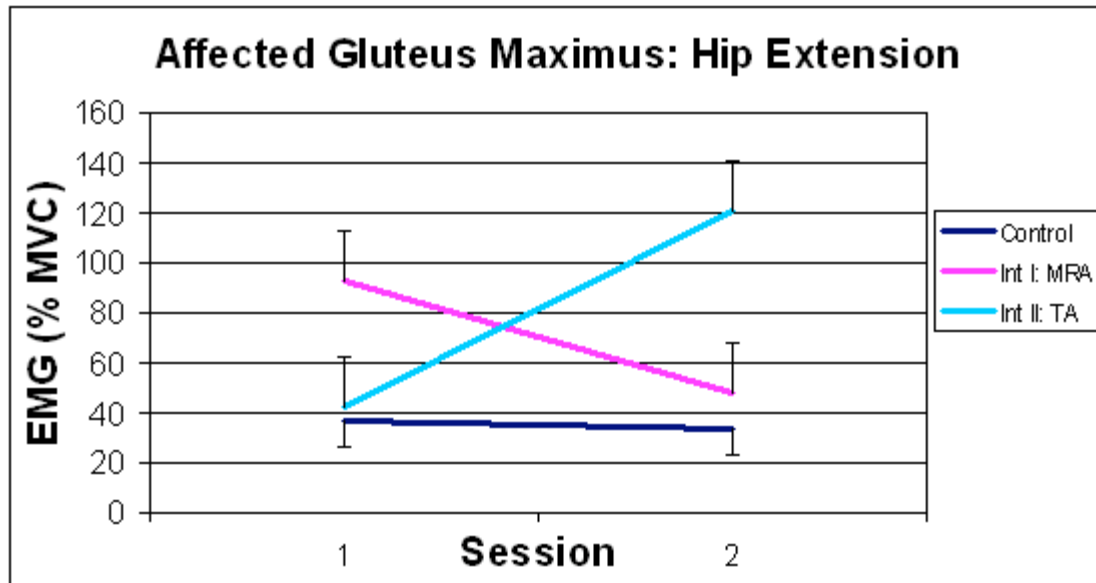


Figure 7a: Group average GM peak muscle activation on the affected hip of participants while performing EXT.

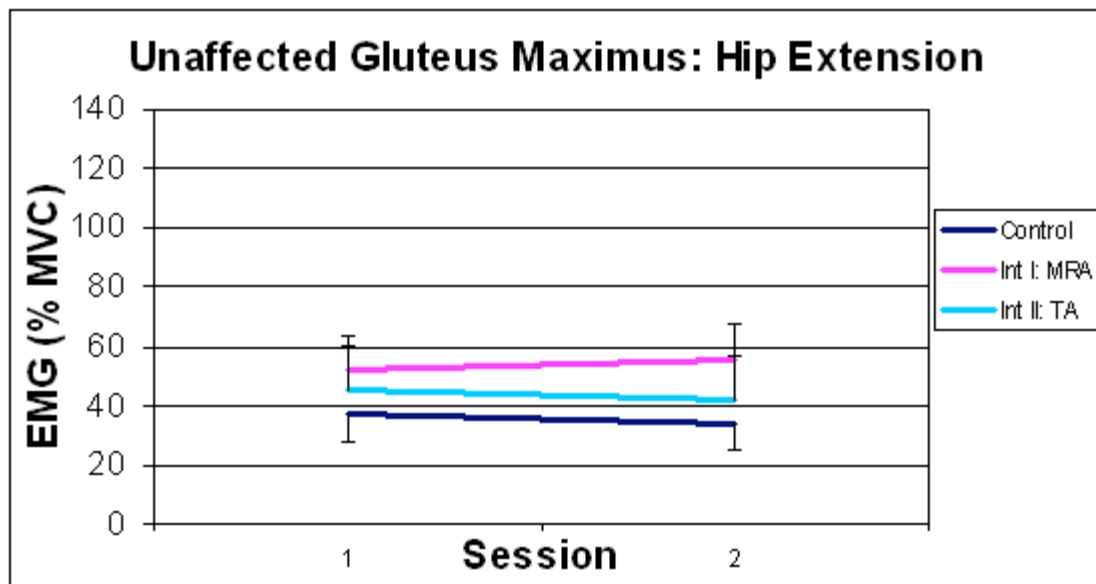


Figure 7b: Group average GM peak activation on the unaffected hip of participants while performing EXT.

Initially (ie. session 1), during the PB the control group did not differ from either intervention group (I $p=0.4677$, II $p=0.3491$) and the intervention groups were similar ($p=0.7944$). However, following the intervention the control and intervention groups differed (control to I/II $p=0.0064/0.0005$ and I/II $p=0.0005$). During EXT, the control and Intervention I groups were different ($p=0.0004$) as were Intervention groups I and II ($p=0.0007$) initially, while the differences existing following the intervention were between the control and Intervention II groups ($p<0.0001$) and Intervention I and II ($p<0.0001$).

No significant changes occurred in the GM during the ASLR and although the direction of peak EMG in the GM during the ABD exercise reflected that during the PB and EXT, these changes were not found to be significant. In asymmetrical exercises that involved one leg to remain on the supporting surface (ie. EXT, ASLR and ABD), no differences were observed in the affected or unaffected side while in this static position.

Erector Spinae

Although peak ES activation appeared different bilaterally for the groups over sessions in Figures 7 and 8 during the PB (affected/unaffected $p=0.0083/0.1452$), EXT (affected/unaffected $p=0.0035/0.0584$), these interactions were only significant on the side contralateral to the affected hip. In contrast to the GM muscle activation, the ES activation increased following Intervention I (PB/EXT $p=0.0234/0.0204$), but diminished on following Intervention II (PB/EXT $p=0.0042/0.0038$). No differences existed between groups initially during the PB, but the Intervention I and II were different following the intervention ($p=0.0202$); these differences were not observed during EXT. The group averages of peak muscle activation are displayed in Figures 8 (PB) and 9 (EXT).

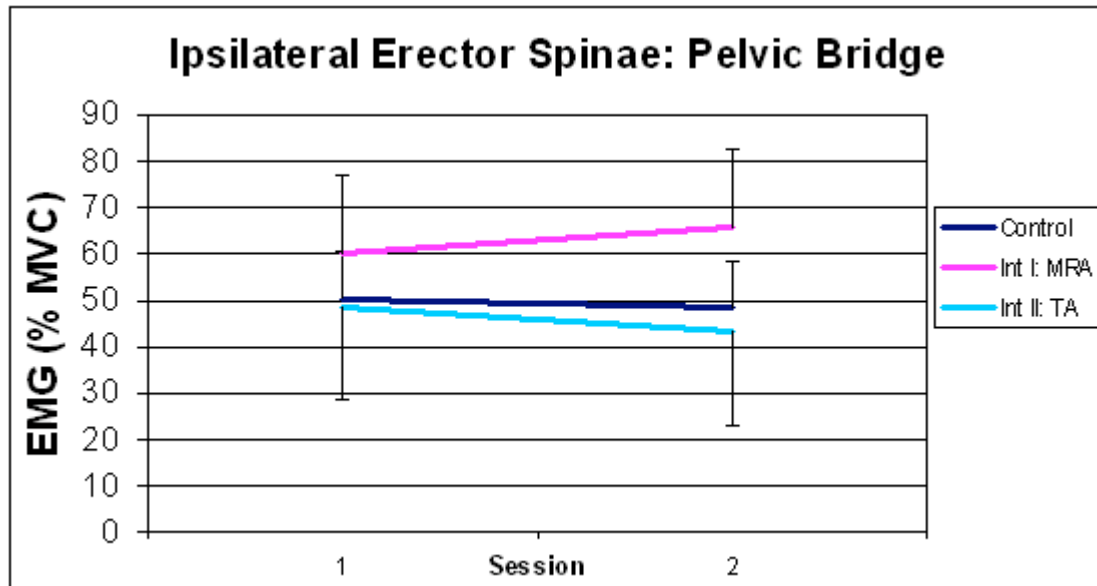


Figure 8a: Group average of ES peak muscle activation ipsilateral to the affected hip of participants while performing PB

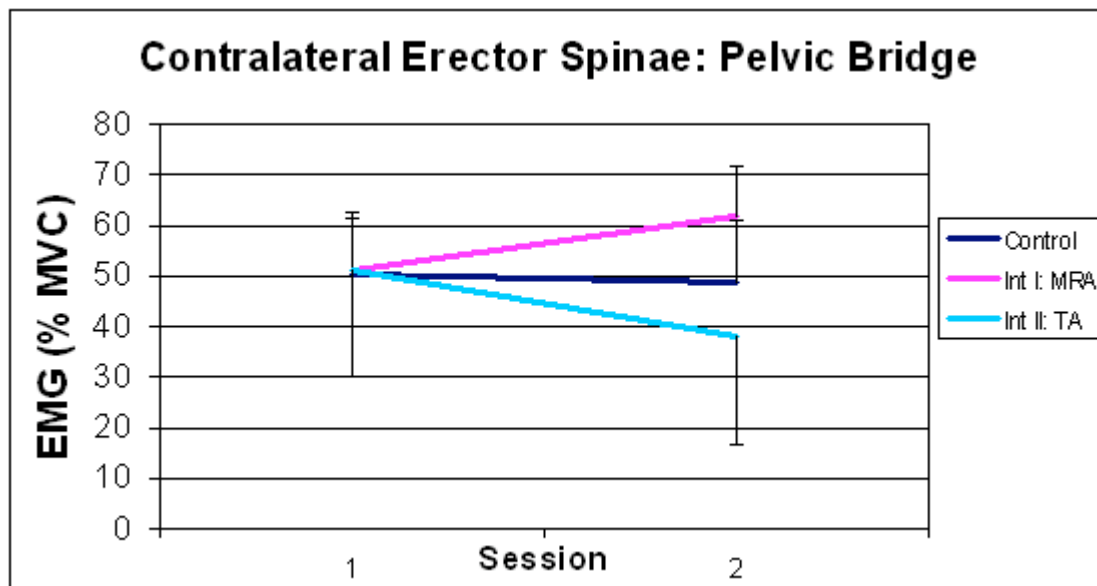


Figure 8b: Group average of ES peak muscle activation contralateral to the affected hip of participants while performing PB.

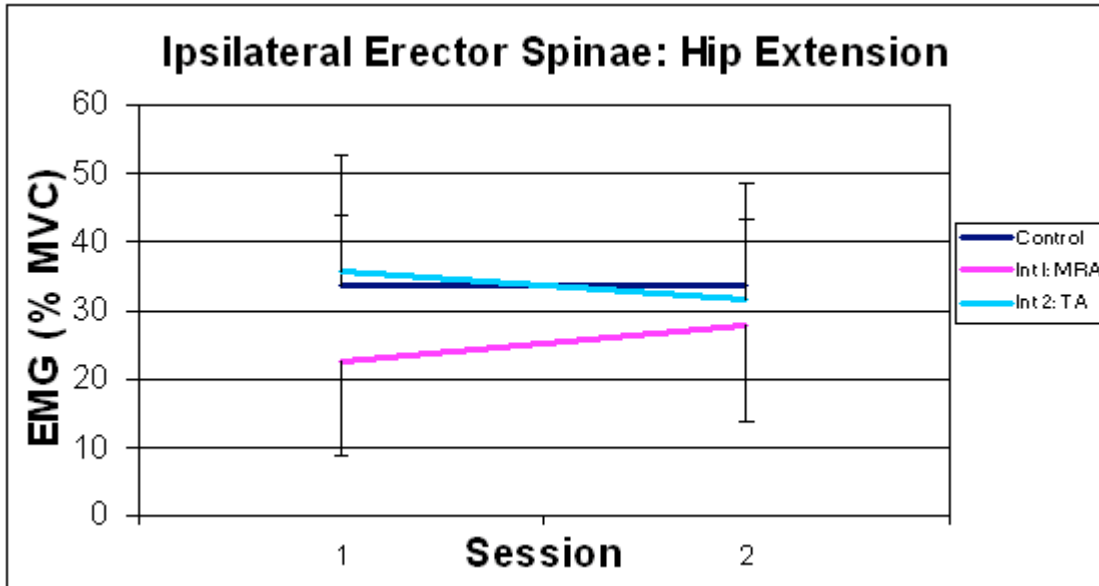


Figure 9a: Group average of ES peak muscle activation ipsilateral to the affected hip of participants while performing EXT.

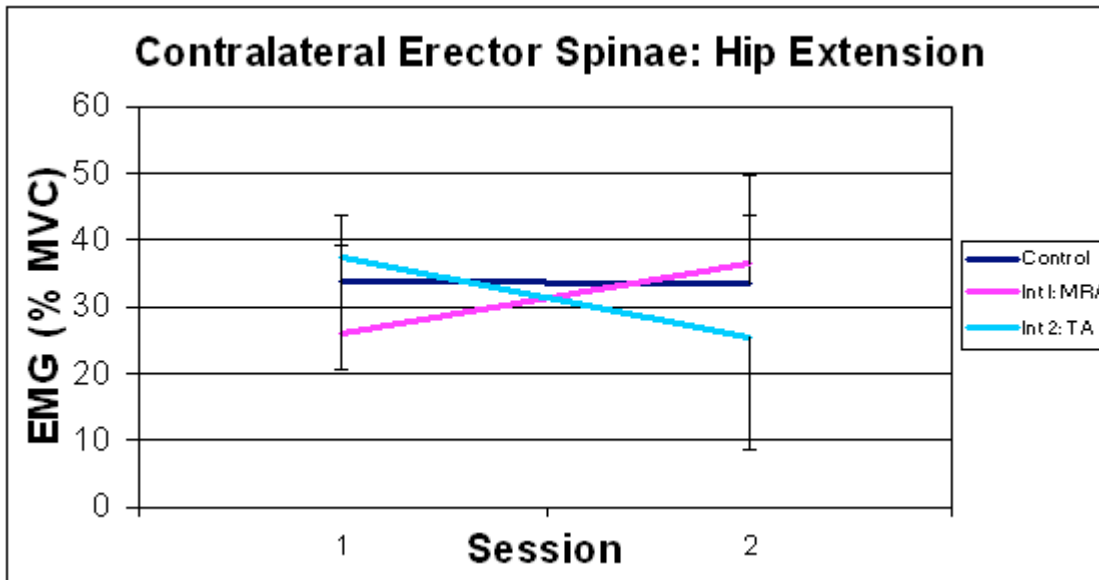


Figure 9b: Group average of ES peak muscle activation contralateral to the affected hip of participants while performing EXT.

Semimembranosis

Main effects of group and time were not observed in the SM peak muscle activation in either hip during PB or EXT. An interaction effect of group by time was observed only on the side ipsilateral to the administered intervention (affected/unaffected $p=0.0060/0.5052$). Further analyses indicated that activation decreased following Intervention I (PB/EXT $p<0.0001/0.0286$) and was elevated following Intervention II (PB/EXT $p=0.0057/0.0189$) and the direction of these changes was similar to that of the GM these exercises. Please see Figures 10 (PB) and 11 (EXT) for a summary of these results.

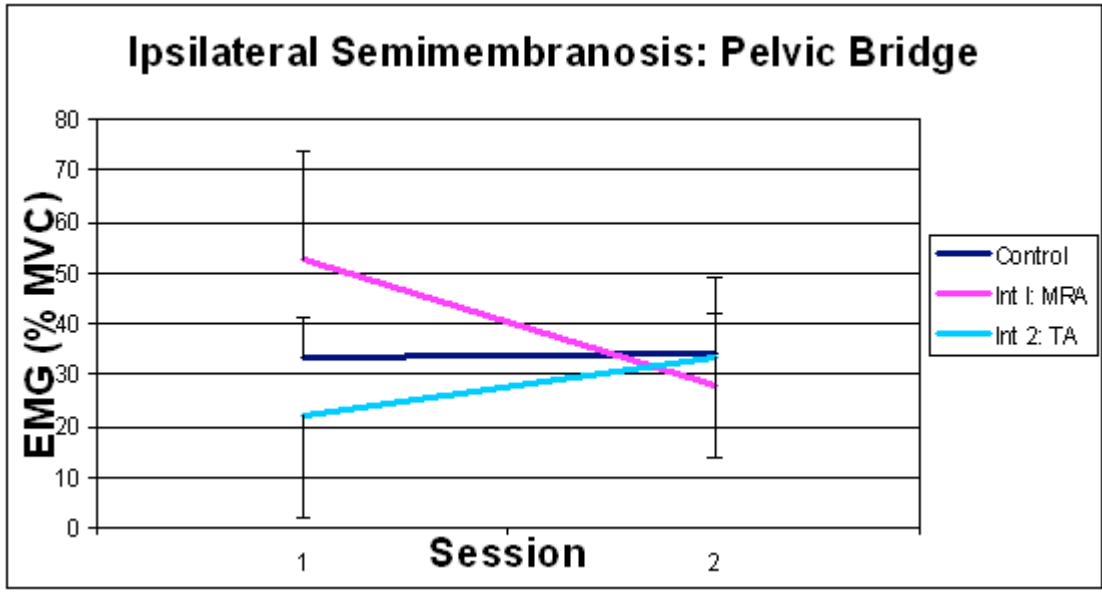


Figure 10a: Group average SM peak muscle activation ipsilateral to the affected hip of participants while performing PB.

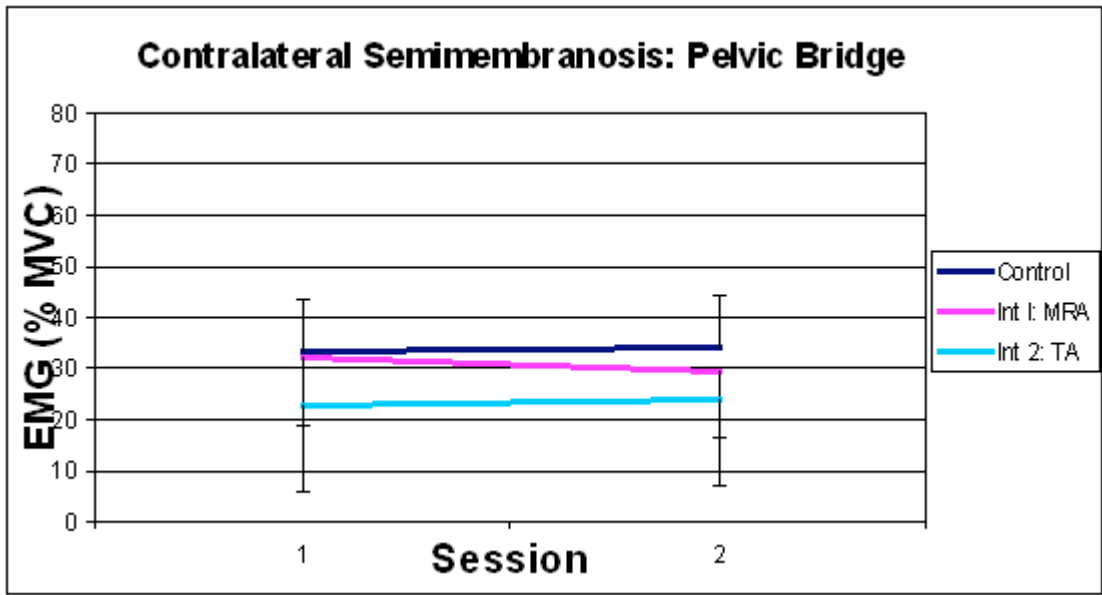


Figure 10b: Group average SM peak muscle activation contralateral to the affected hip of participants in Intervention Group I while performing PB.

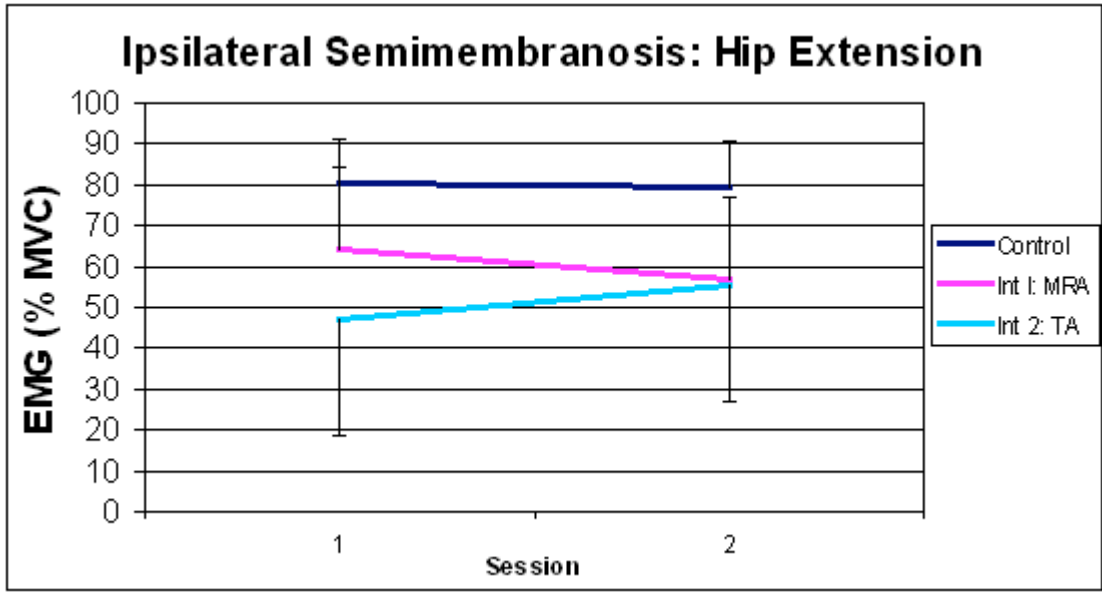


Figure 11a: Group average SM peak muscle activation ipsilateral to the affected hip of participants while performing EXT.

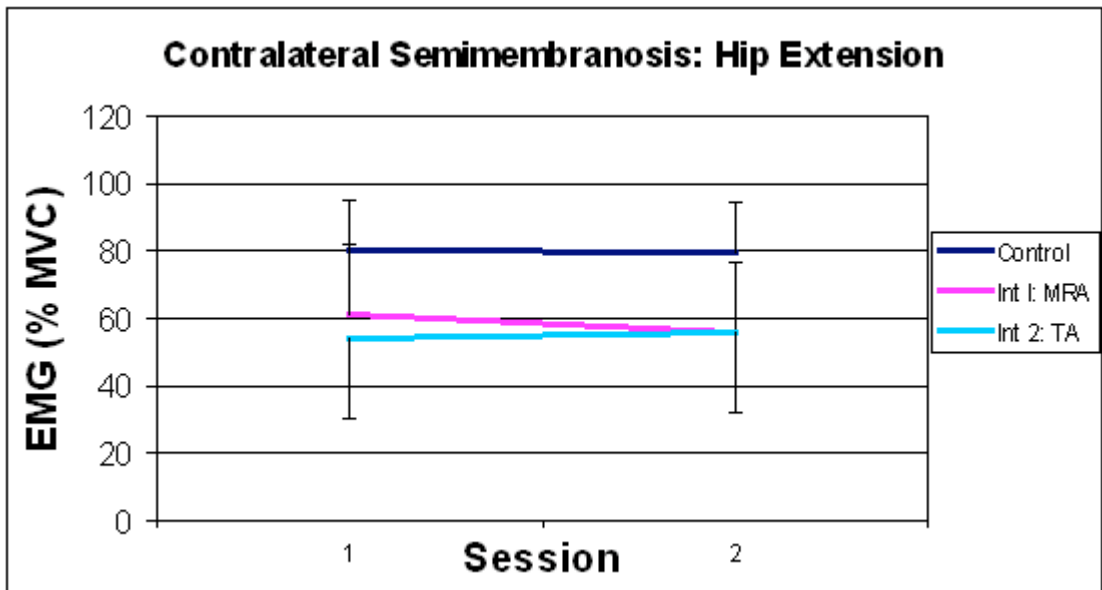


Figure 11b: Group average SM peak muscle activation contralateral to the affected hip of participants while performing EXT.

This activation pattern was documented during the ASLR on the affected side, without attaining statistical significance. The unaffected SM displayed no differences throughout the tasks performed, except for a treatment effect during the active straight leg raise exercise.

Rectus Abdominis

Rectus abdominis activation levels were substantially lower during these tasks. This was expected as only minimal active trunk flexion was required for the selected tasks. Low activation levels (< 5% MVC) of rectus abdominis were observed throughout the tasks. As activation levels did not vary during the exercise tasks from resting levels, there are no differences to report for this muscle in the control or intervention groups.

Volume, Pressure and Inhibition Relationships

Pressure-Volume Relationship

Instillation of joint fluid volume was associated with intra-articular pressure values increasing until maximal intra-capsular volume and capsular distension were achieved. This relationship was exhibited during the MRA procedure as the intention was capsular distension for diagnostic purposes. An average of total volume of 9.4 ± 1.78 cc was instilled and was associated with an average total pressure increase of 18.2 ± 11.22 mmHg.

Since the objective of the TA procedure was to release the capsular structure, fluid/air volume was instilled beyond maximum capsular volume and distension. These higher levels of fluid/air volume were associated with subsequent decrements in intra-articular pressure recordings. The average total volume instilled was 28.9 ± 3.64 cc with an associated decrease in total pressure of 34.0 ± 48.0 mmHg.

Absolute values for maximum intra-capsular volume capacity and intra-articular pressure (“Opening” and “Final” pressures for example) values appear unique to the individual, but the pressure-volume relationship exhibits similar behavior across individuals, irrespective of individual differences.

Intervention Group I

Pressure-Volume Relationship

All subjects within this intervention group displayed a higher “Final” intra-articular pressure when compared to “Opening” pressure values. Typically, this relationship followed a linear pattern until an injected volume of 8-10 mL was achieved, after which the related pressure appeared to increase exponentially.

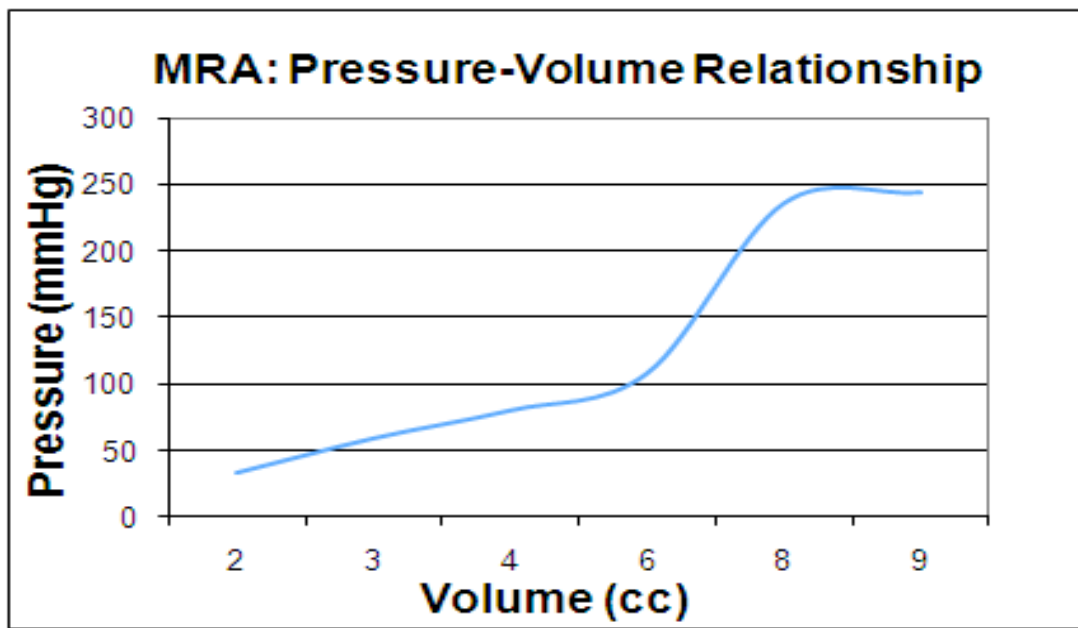


Figure 12: A Sample Pressure-Volume Relationship during Magnetic Resonance Arthrogram for an Individual Participant

Inhibition: Relationship to Change in Pressure vs Change in Volume

Inhibition was considered as the mean change in GM EMG within an exercise task across sessions. Generally, GM activation decreased following Intervention I, displaying an inverse relationship with both pressure and volume.

Regression analyses revealed a significant relationship between inhibition and pressure (PB/EXT $r=0.9925/0.9831$ and $p<0.0001/<0.0001$) but not with volume (PB/EXT $r=-0.4783/0.1659$ and $p=0.1620/0.6470$).

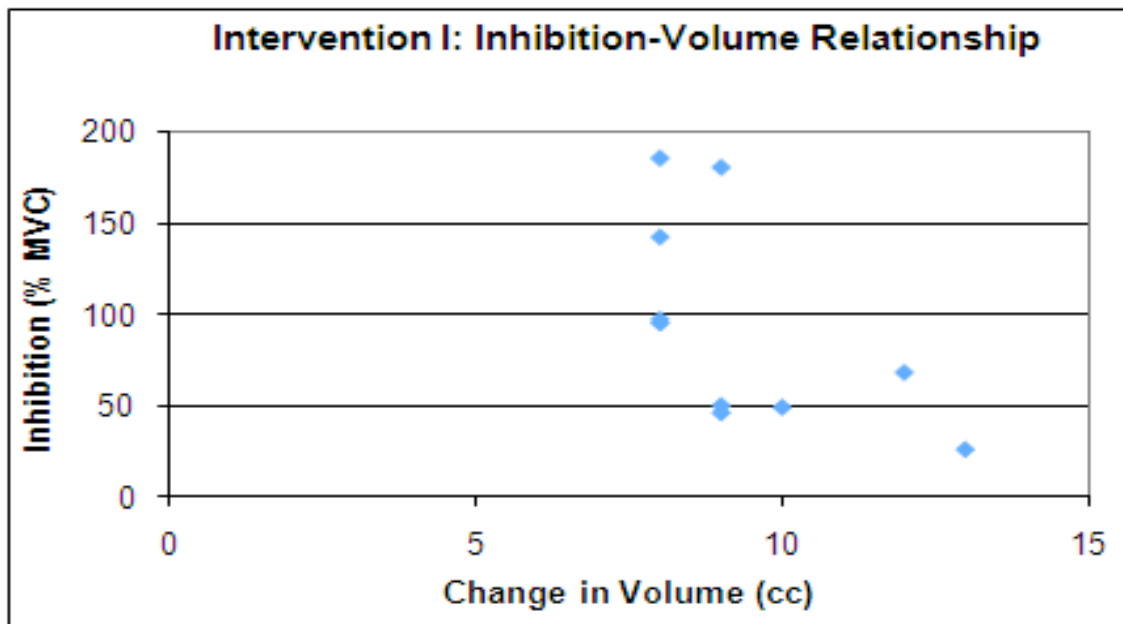


Figure 13: Inhibition-Volume Relationship during the Pelvic Bridge with Each Individual Participant in Intervention I Represented by a Single Point

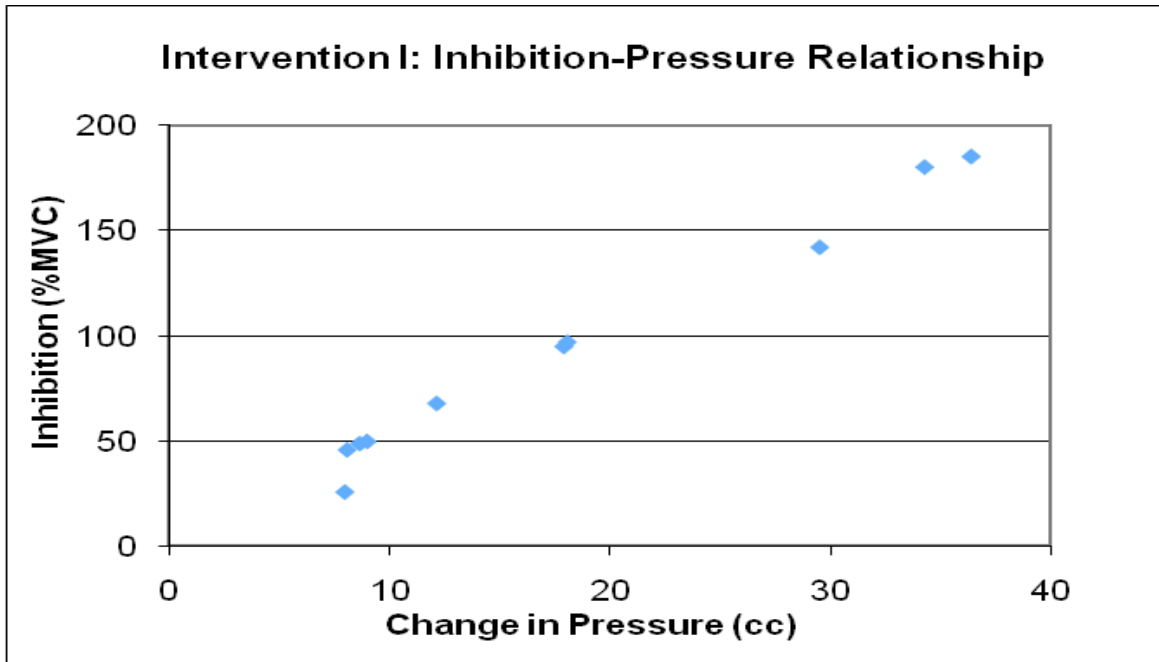


Figure 14: Inhibition-Volume Relationship during the Pelvic Bridge with Each Individual Participant in Intervention I Represented by a Single Point

Intervention Group II

Pressure-Volume Relationship

All subjects within this intervention group displayed a lower “Final” intra-articular pressure when compared to “Opening” pressure values

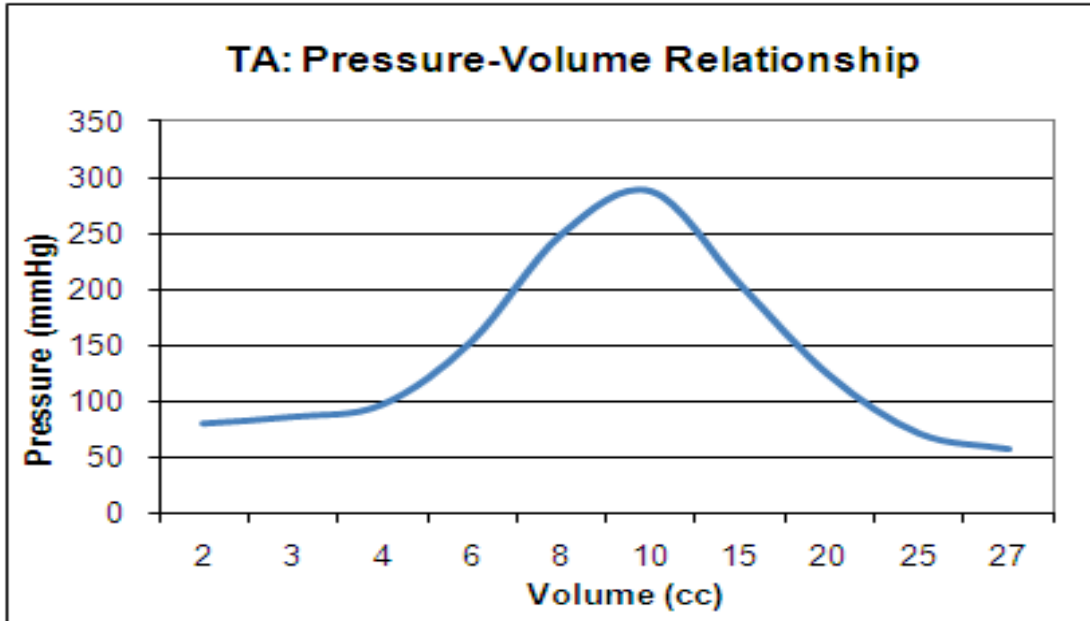


Figure 15: A Sample Pressure-Volume Relationship during Therapeutic Arthrogram for an Individual Participant

Inhibition: Relationship to Change in Pressure vs Change in Volume

Generally, GM activation appeared to increase following Intervention II. Regression analyses revealed that inhibition exhibited a significant relationship with both pressure ($r=0.7339$ and $p=0.0382$) and volume ($r=-0.7452$ and $p=0.0339$) during the PB task. During the EXT task significance was observed for inhibition with pressure only ($r=0.8970$ and $p=0.0025$) as the relationship between inhibition and volume was not found to be significant ($r=0.3276$ and $p=0.3276$).

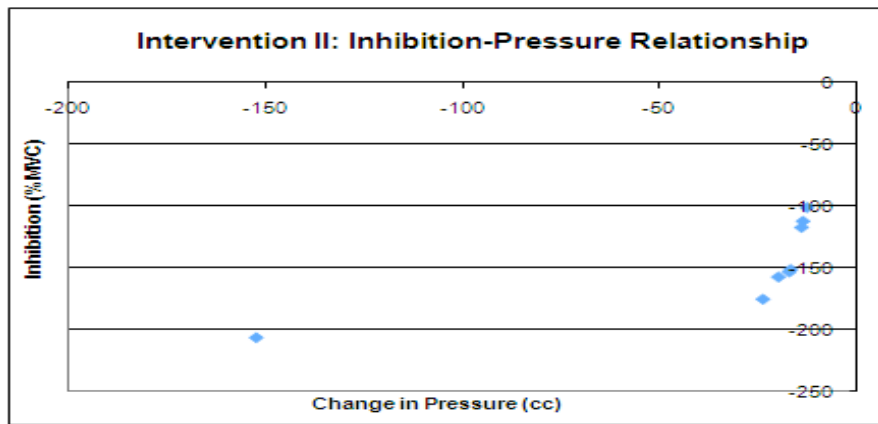


Figure 16 a: Inhibition-Pressure Relationship during Therapeutic Arthrogram with Individual Participants from Intervention II Displayed as a Single Point. Please also see Figure 15 b as the scale has been adjusted for improved viewing of this graph.

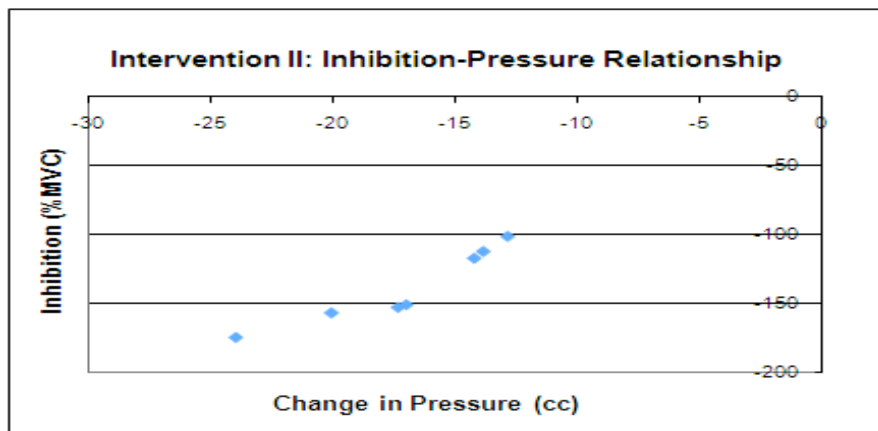


Figure 16 b: Inhibition-Pressure Relationship during Therapeutic Arthrogram with Individual Participants from Intervention II Displayed. The same data can be viewed in Figure 15 a, which includes one additional participant. Note: the scale has been adjusted as mentioned in Figure 16 a.

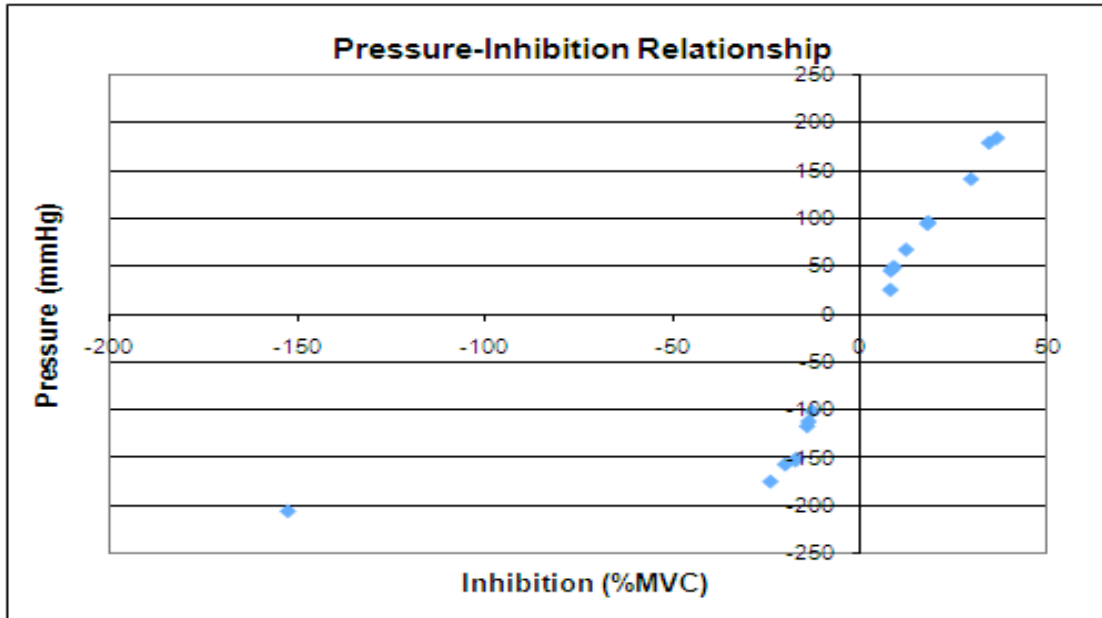


Figure 16 c: Alternative View Summarizing the Inhibition-Pressure Relationship during Magnetic Resonance and Therapeutic Arthrograms with Each Individual Participant from Interventions I and II Displayed as a Single Point.

Notes for Analysis

Relevant Clinical Observations from the Arthrogram Procedures

Five MRA participants and all of the TA participants exhibited abnormal psoas adherence to the anterior capsule of the hip. This was palpable by the radiologist on needle insertion and lack of psoas mobility on fluoroscopy.

Justification for Removal of Gender as a Factor:

Initially, the statistical analyses were performed including gender as a between factor. Gender was determined statistically insignificant for all conditions, with the exception of GM during the PB in the control group. It was concluded that this was not sufficient justification to include gender in the analyses as it was not shown to be statistically significant in the intervention groups and its inclusion would have decreased the number of participants per group, thereby diminishing statistical power.

Justification on Removal of an Outlier: An Interesting Case

Preliminary statistical analyses revealed an outlier amongst participants in Intervention I group. This participant appeared to contaminate the statistical analyses as the direction of change in GM EMG was opposite to the remainder of the group.

This participant was age-matched with no pathology observed on the MR images. Closer examination of this participant revealed over-distension of the capsular structure on fluoroscopic images acquired during the arthrogram procedure. As this study investigated the effects of intra-articular hip fluid volume and pressure on local muscle activation patterns, inclusion of this participant would have disrupted the homogeneity of the group.

Chapter 5: Discussion

The purpose of this thesis was to provide foundational insights with respect to arthrogenic inhibition about the hip joint and address the primary hypothesis that extensor-inhibition (ie. GM) about the hip may exist following intra-articular administration of fluid. If inhibition was detected, its magnitude was hypothesized to vary with intra-articular pressure. Compensatory muscular activation strategies and implications of these hip findings on the lumbar spine were of secondary interest. Compensatory increases in muscle activation (ie. ES and SM) at adjacent joints was hypothesized to occur in association with extensor-inhibition. These hypotheses will be addressed and clinical implications discussed.

Primary Hypothesis

Arthrogenic Inhibition: Existence in the Hip Joint?

Based on the results of this study, extensor-inhibition was evident in the GM muscle ipsilaterally during tasks involving primarily hip extension, such as PB and EXT. Support for the presence of inhibition was observed in Intervention Group I, following fluid instillation causing capsular distension and subsequent elevation in intra-capsular pressure. Not only was the existence of GM inhibition demonstrated as a transient effect secondary to induced effusion but was also apparent in Intervention Group II, which contained a population with prolonged limited hip function. Interestingly, this population exhibited substantial restoration of GM activation following over-distension of the capsule via instillation of joint fluid resulting in an overall decrease in intra-articular pressure.

Secondary Hypothesis

Arthrogenic Inhibition: Relationship with Intra-Articular Volume and/or Pressure

Given that inhibition correlated more closely with changes in intra-articular pressure than with volume and VAS scores were unchanged, intra-articular pressure appears to be the most powerful predictor of GM inhibition amongst these variables, This has tremendous implications on clinical practice since intra-articular pressure has not even been considered previously as a factor influencing extensor-inhibition in any joint.

Investigations by Aloisi et al (1988) who found that the type I hip capsular receptors in the cat were specifically sensitive to pressure (and not vibratory stimulus) applied perpendicular to the capsular fibers, may provide some explanation for the findings in this thesis. It appears reasonable that the capsular receptors and hip joint afferents sensitive to pressure may be involved in the neuromuscular modulation of GM response.

Cyclical Nature of Hip Dysfunction

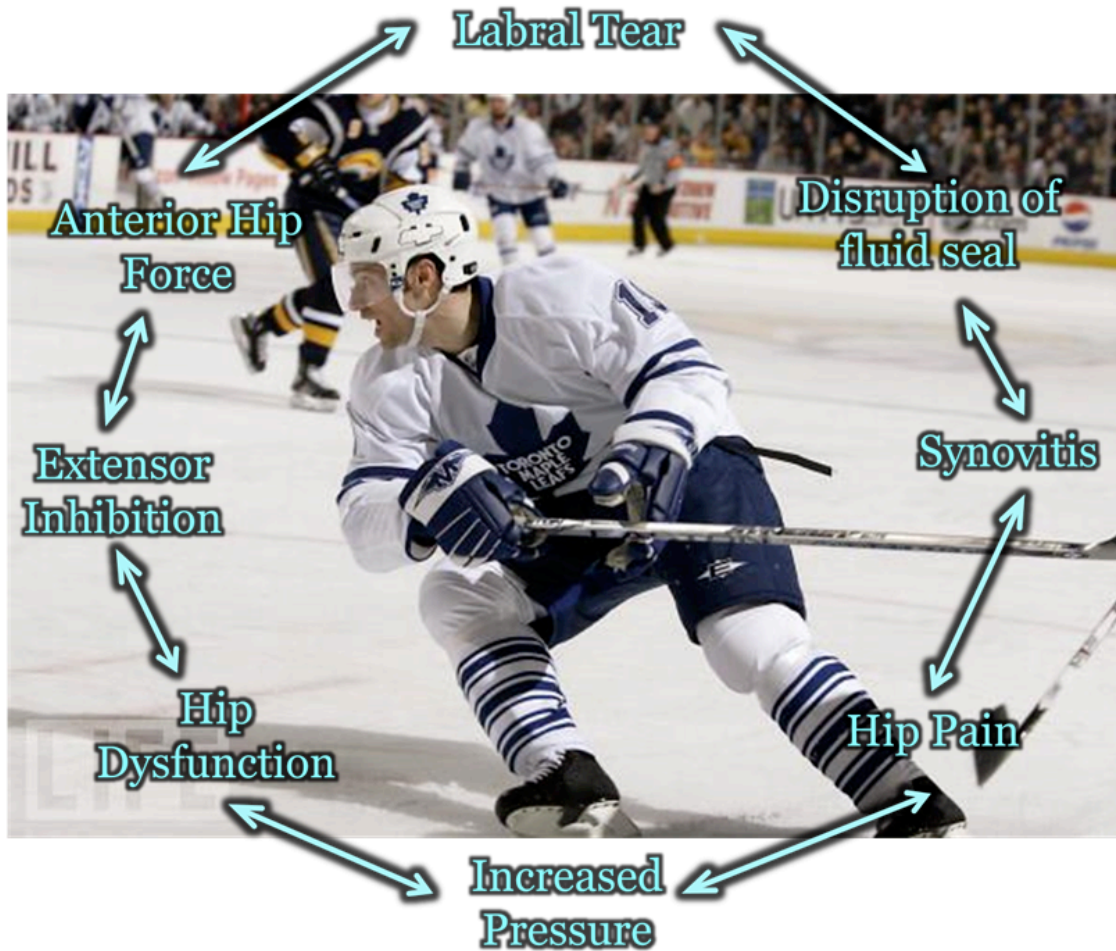


Figure 18: Illustration of Possible Cyclical Nature of Hip Dysfunction

The existence of extensor-inhibition (ie. GM) about the hip joint may have a pronounced influence on precipitation and/or progression of injury. Diminished GM activation may precipitate a hip injury as it leads to increased anterior femoral translation, where most hip pathology has been found to occur (Lewis 2007). This is particularly important in the development of injuries involving the anterior-superior acetabular labrum, prevalent in physically active populations such as hockey players and runners. In the acute phase, the pain associated with labral tears may be attributed to the associated synovitis, a prevalent radiographic finding associated with labral tears. An important function of the acetabular

labrum is to create a fluid seal so that negative pressure may be maintained within the hip capsule. It is probable that both synovitis and disruption of the acetabular labrum (and fluid seal) leads to a relative increase in intra-capsular pressure. Prolonged elevation of intra-articular pressure may exacerbate GM inhibition and thus, increased anterior hip joint forces. If further anterior femoral translation activates type I afferents in the anterior capsule, extensor-inhibition needs to be considered a significant factor in the cyclical decline in hip function.

Tertiary Hypothesis

Arthrogenic Inhibition: Compensatory Motor Patterns

Although levels of ES activation increased in response to GM inhibition as hypothesized, SM diminished with GM inhibition, despite the common belief that GM inhibition results in hamstring facilitation. With the release of GM inhibition, the ES decreased and SM increased.

Interestingly, a unilateral decrement in GM activation resulted in bilateral (although only significant on the side contralateral to the intervention) elevation of ES activation during hip extension exercises. This finding warrants consideration of the influence of a unilateral hip dysfunction on the lumbar spine bilaterally. Unilateral GM inhibition might result in compensatory increased lumbar spine compression loads due to elevated ES activation. Furthermore, if this increased ES activation was associated with increased spine motion in the sagittal plane, injuries to the intervertebral discs and facet joints bilaterally, might be of concern. Given that SM did not appear to increase in response to GM inhibition, it is possible that participants accomplished the extension tasks via

lumbar spine, rather than hip motion, which could contribute to these injuries to the spine. Furthermore, if diminished GM activation during extension activities is not compensated for by elevated SM activation, even greater loads than originally thought might be imposed on the lumbar spine, potentially accelerating the rate of future injury. Contrary to popular thought, SM activation did not increase in a compensatory manner to diminished GM activation. One possible explanation for this pattern of activation is that even though GM is the primary extensor of the hip and SM is often considered primarily as a knee flexor, it also contributes to the generation of hip extension. Lewis et al (2007) report that it is plausible for the semitendinosus muscle to contribute 50% of hip extensor torque during hip extension activities. Provided extensor-inhibition selectively inhibits extensor musculature and SM functions as a hip extensor, perhaps changes in its activation profile reflecting that of the ipsilateral GM should not be surprising. Facilitation of the gastrocnemius and soleus muscles was documented in investigations by Palmieri et al (2004) surrounding arthrogenic inhibition of the knee. This was presumed to occur as the gastrocnemius may contribute to knee flexion, which was thought to be facilitated. This could support the possibility for synergistic inhibition of the SM in arthrogenic inhibition of the hip. It cannot be concluded, however, that the remaining hamstring musculature (semitendinosus, biceps femoris and the hamstring portion of adductor magnus) becomes inhibited or facilitated with increasing intra-articular hip pressure or that this pattern occurs across other activities.

Limitations

Although it could be argued that administration of a subcutaneous anesthetic, which may modulate pain, could contribute to this re-establishment of GM activation, it is unlikely as VAS scores were unchanged throughout.

This study did not collect kinematic data to confirm this notion as the primary objective was to quantify muscle activity rather than motion. However, acquisition of kinematic data would be a useful addition for future research.

Future Directions

This thesis underscores the importance of clinical evaluation and rehabilitation following hip dysfunction. Intra-articular hip injuries with associated effusion or synovitis may elevate intra-articular pressure and resultant GM inhibition. Even in the absence or dissipation of pain, appropriate motor re-training appears necessary to restore symmetrical muscle activation. Knowing that hip position influenced intra-articular pressure as discovered by Yen et al (2009), it might be wise for clinicians to begin gluteal re-training and/or hip rehabilitation exercise in positions of hip flexion (as this was associated with the lowest pressure values across similar fluid volumes) and progress exercises by positioning the hip in relatively decreasing degrees of flexion. Specifically, PB to EXT appears a reasonable progression.

Additionally, in the selection of clinical evaluation tests and subsequently assigned rehabilitation protocols, consideration of the physical demands specific to the individual are essential for improved outcomes. This is supported by previous research that found classification of LBP patients into ‘hypermobile’ vs ‘hypomobile’ positively predicted which patients would benefit from stabilization vs mobilization therapy (Hicks et al 2004). Of interest, hip ROM (especially rotation) was a useful factor in patient subclassification. Recently, Harris-Hayes et al (2009) investigated athletic populations with and without reported LBP. Of those athletes with LBP, those involved in rotational sports (specifically golf and tennis) presented with restrictions in passive hip rotational ROM, whereas these restrictions were not found in athletes with LBP and minimal rotational demands and those without LBP. This supports that in order to be able to better predict whether deficits identified, in GM activation for example, will lead to reported

compensatory dysfunction, such as LBP, the deficit needs to be matched to biomechanical demands. In the case of this thesis, it appears reasonable that those with extensor-inhibition of the hip who participate in hip flexion-extension activities would be predisposed to lower back injuries more so than those whose lifestyles do not include these demands. This could also explain why GM inhibition was evident in PB and EXT, which require extensor torque generation, and not ASLR and ABD. Finally, those patients experiencing capsular contracture or adhesion, procedures such as the TA which intend to normalize joint pressures, may be needed in addition to conservative care in order for full restoration of motor patterns to be accomplished.

Conclusions

This thesis was designed to provide foundational insights into the inter-related neuromuscular relationships between the lumbar spine and hip joints. The findings provide substantial support for the concept of arthroscopic inhibition following infusion of intra-articular fluid may be generalized to the hip joint. The clinical importance of decreased hip extensor (GM and SM) activation associated with elevated intra-articular pressure in the hip joint has tremendous implications to hip evaluation and rehabilitation programs.

Furthermore, compensatory increases in lumbar spine extensor muscular activation were observed bilaterally during extension exercises that are frequently included in lumbar spine and hip rehabilitation programs. Diminished activation of the hip extensor musculature unilaterally may not only facilitate further hip joint injury but may also impose unnecessarily high compressive loads to the spine which is known to precipitate eventual injury.

In clinical cases where appropriate GM activation (and possibly activation capability) is inadequate, interventions such as the TA procedure may be necessary prior to, or in conjunction with, manual therapies and rehabilitative exercise to enable complete restoration of muscle activation patterns and promote functional improvements.

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Appendix A: Standardized Questionnaire Samples

Visual Analog Scale

0 1 2 3 4 5 6 7 8 9 10

On the above scale please rate your pain (0 indicates no pain; 10 indicates worst imaginable pain)

Oswestry Disability Questionnaire

This questionnaire has been designed to give us information as to how your back or leg pain is affecting your ability to manage in everyday life. Please answer by checking **one box in each section** for the statement which best applies to you. We realise you may consider that two or more statements in any one section apply but please just shade out the spot that indicates the statement **which most clearly describes your problem.**

Section 1: Pain Intensity

- I have no pain at the moment
- The pain is very mild at the moment
- The pain is moderate at the moment
- The pain is fairly severe at the moment
- The pain is very severe at the moment
- The pain is the worst imaginable at the moment

Section 2: Personal Care (eg. washing, dressing)

- I can look after myself normally without causing extra pain
- I can look after myself normally but it causes extra pain
- It is painful to look after myself and I am slow and careful
- I need some help but can manage most of my personal care
- I need help every day in most aspects of self-care
- I do not get dressed, wash with difficulty and stay in bed

Section 3: Lifting

- I can lift heavy weights without extra pain
- I can lift heavy weights but it gives me extra pain
- Pain prevents me lifting heavy weights off the floor but I can manage if they are conveniently placed
eg. on a table
- Pain prevents me lifting heavy weights but I can manage light to medium weights if they are conveniently positioned
- I can only lift very light weights
- I cannot lift or carry anything

Section 4: Walking*

- Pain does not prevent me walking any distance
- Pain prevents me from walking more than 2 kilometres
- Pain prevents me from walking more than 1 kilometre
- Pain prevents me from walking more than 500 metres
- I can only walk using a stick or crutches
- I am in bed most of the time

Section 5: Sitting

- I can sit in any chair as long as I like
- I can only sit in my favourite chair as long as I like
- Pain prevents me sitting more than one hour
- Pain prevents me from sitting more than 30 minutes

- £ Pain prevents me from sitting more than 10 minutes
- £ Pain prevents me from sitting at all

Section 6: Standing

- £ I can stand as long as I want without extra pain
- £ I can stand as long as I want but it gives me extra pain
- £ Pain prevents me from standing for more than 1 hour
- £ Pain prevents me from standing for more than 30 minutes
- £ Pain prevents me from standing for more than 10 minutes
- £ Pain prevents me from standing at all

Section 7: Sleeping

- £ My sleep is never disturbed by pain
- £ My sleep is occasionally disturbed by pain
- £ Because of pain I have less than 6 hours sleep
- £ Because of pain I have less than 4 hours sleep
- £ Because of pain I have less than 2 hours sleep
- £ Pain prevents me from sleeping at all

Section 8: Sex Life (if applicable)

- £ My sex life is normal and causes no extra pain
- £ My sex life is normal but causes some extra pain
- £ My sex life is nearly normal but is very painful
- £ My sex life is severely restricted by pain
- £ My sex life is nearly absent because of pain
- £ Pain prevents any sex life at all

Section 9: Social Life

- £ My social life is normal and gives me no extra pain
- £ My social life is normal but increases the degree of pain
- £ Pain has no significant effect on my social life apart from limiting my more energetic interests e.g. sport
- £ Pain has restricted my social life and I do not go out as often
- £ Pain has restricted my social life to my home
- £ I have no social life because of pain

Section 10: Traveling

- £ I can travel anywhere without pain
- £ I can travel anywhere but it gives me extra pain
- £ Pain is bad but I manage journeys over two hours
- £ Pain restricts me to journeys of less than one hour
- £ Pain restricts me to short necessary journeys under 30 minutes
- £ Pain prevents me from traveling except to receive treatment

Score: / x 100 = %

Scoring: For each section the total possible score is 5: if the first statement is marked the section score = 0, if the last statement is marked it = 5. If all ten sections are completed the score is calculated as follows:

Example: 16 (total scored)

$$50 \text{ (total possible score)} \times 100 = 32\%$$

If one section is missed or not applicable the score is calculated: 16 (total scored)

$$45 \text{ (total possible score)} \times 100 = 35.5\%$$

Minimum Detectable Change (90% confidence): 10%points (Change of less than this may be attributable to error in the measurement)

Source: Fairbank JCT & Pynsent, PB (2000) The Oswestry Disability Index. *Spine*, 25(22):2940-2953.

Davidson M & Keating J (2001) A comparison of five low back disability questionnaires: reliability and responsiveness. *Physical Therapy* 2002;82:8-24.

Oxford Hip Score

1. **During the past 4 weeks**, how would you describe the pain you **usually** had from your hip?

None ¹	Very mild ²	Mild ³	Moderate ⁴	Severe ⁵

2. **During the past 4 weeks**, have you had any trouble with washing and drying yourself (all over) **because of your hip**?

No trouble at all ¹	Very little trouble ²	Moderate trouble ³	Extreme difficulty ⁴	Impossible to do ⁵

3. **During the past 4 weeks**, have you had any trouble getting in and out of a car or using public transport **because of your hip**?

No trouble at all ¹	Very little trouble ²	Moderate trouble ³	Extreme difficulty ⁴	Impossible to do ⁵

4. **During the past 4 weeks**, have you been able to put on a pair of socks, stocking or tights?

Yes, easily ¹	With little difficulty ²	With moderate difficulty ³	With extreme difficulty ⁴	No, impossible ⁵

5. **During the past 4 weeks**, could you do the household shopping **on your own**?

Yes, easily ¹	With little difficulty ²	With moderate difficulty ³	With extreme difficulty ⁴	No, impossible ⁵

6. **During the past 4 weeks**, for how long have you been able to walk before **pain from your hip** becomes severe (with or without a stick)?

No pain/more than 30 minutes ¹	16 – 30 minutes ²	5 – 15 minutes ³	Around the house only ⁴	Not at all – pain severe on walking ⁵

7. **During the past 4 weeks**, have you been able to climb a flight of stairs?

Yes, easily ¹	With little difficulty ²	With moderate difficulty ³	With extreme difficulty ⁴	No, impossible ⁵

8. **During the past 4 weeks**, after a meal (sat at a table), how painful has it been for you to stand up from a chair **because of your hip**?

Not at all painful ¹	Slightly painful ²	Moderately painful ³	Very painful ⁴	Unbearable ⁵

9. **During the past 4 weeks**, have you been limping when walking **because of your hip**?

Rarely/never ¹	Sometimes, or just at first ²	Often, not just at first ³	Most of the time ⁴	All of the time ⁵

10. **During the past 4 weeks**, have you had any sudden or severe pain – ‘shooting’, ‘stabbing’, or ‘spasms’ – **from the affected hip**?

No days ¹	Only 1 or 2 days ²	Some days ³	Most days ⁴	Every day ⁵

11. **During the past 4 weeks**, how much has **pain from your hip** interfered with your usual work (including housework)?

Not at all ¹	A little bit ²	Moderately ³	Greatly ⁴	Totally ⁵

12. **During the past 4 weeks**, have you been troubled by **pain from your hip** in bed at night?

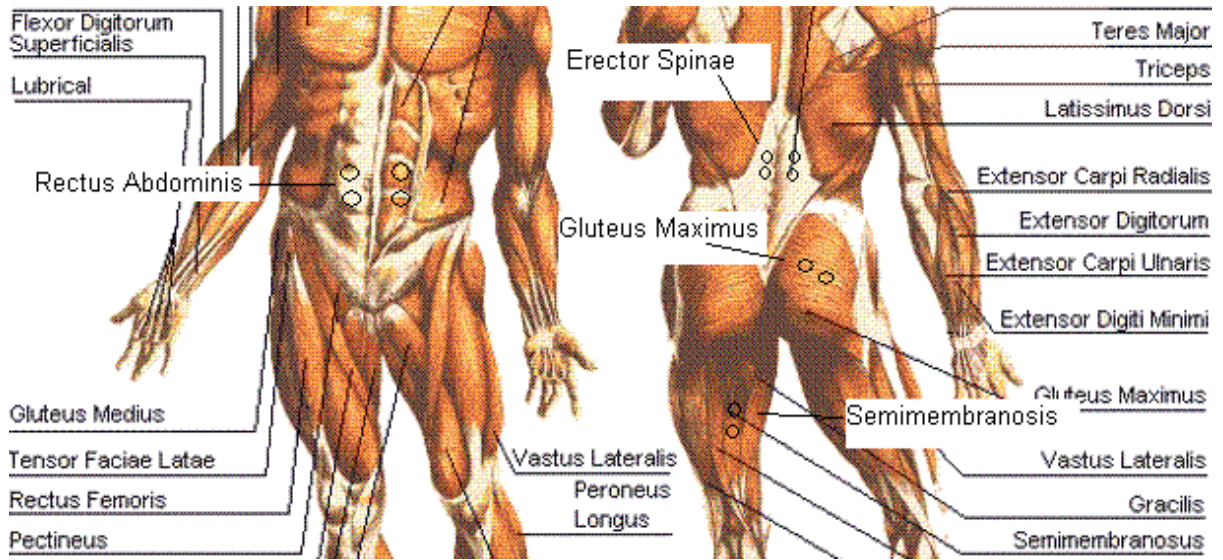
No nights ¹	Only 1 or 2 nights ²	Some nights ³	Most nights ⁴	Every night ⁵

Grading for the Oxford Hip Score

Score 0 to 19	May indicate severe hip arthritis. It is highly likely that you may well require some form of surgical intervention, contact your family physician for a consult with an Orthopaedic Surgeon.
Score 20 to 29	May indicate moderate to severe hip arthritis. See your family physician for an assessment and x-ray. Consider a consult with an Orthopaedic Surgeon.
Score 30 to 39	May indicate mild to moderate hip arthritis. Consider seeing your family physician for an assessment and possible x-ray. You may benefit from non-surgical treatment, such as exercise, weight loss, and/or anti-inflammatory medication.
Score 40 to 48	May indicate satisfactory joint function. May not require any formal treatment.

Reference for Score: Dawson J, Fitzpatrick R, Carr A, Murray D. Questionnaire on the perceptions of patients about total hip replacement. *J Bone Joint Surg Br.* 1996 Mar;78(2):185-90. [Link](#)

Appendix B



Appendix C

Average ROM (in flexion, extension, external rotation and internal rotation) for Intervention Groups prior to and following the administered intervention procedure.

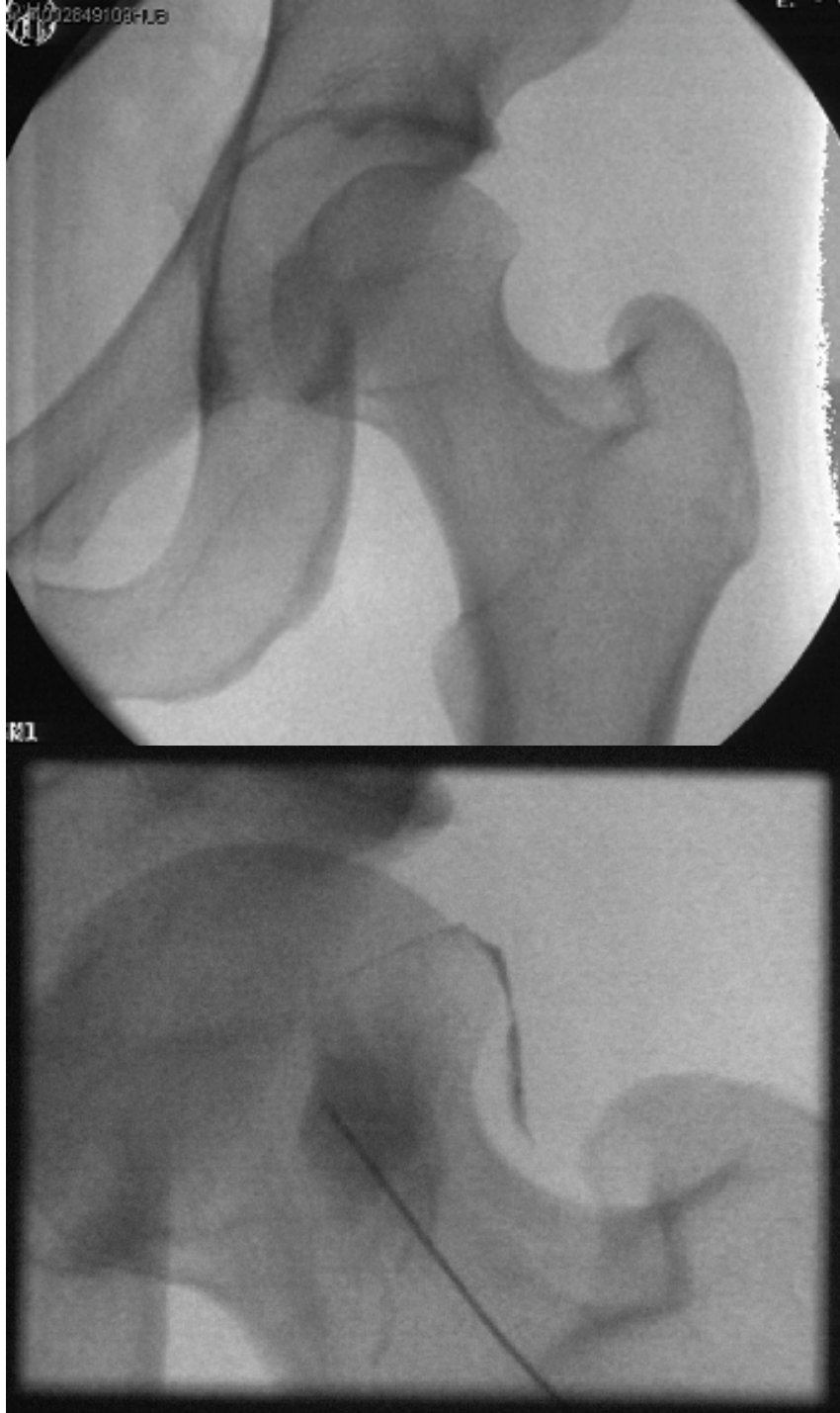
Intervention Group I: Affected Hip		
Range of Motion: Degrees (SD)	Pre-Intervention	Post-Intervention
Flexion	104.1 (20.09)	99.5 (19.24)
Extension	7.5 (7.10)	4.6 (5.33)
External Rotation	68.1 (18.81)	63.6 (17.82)
Internal Rotation	27.6 (29.22)	25.4 (27.07)
Intervention Group I: Unaffected Hip		
Flexion	115.9 (18.56)	115.0 (19.56)
Extension	10.5 (5.50)	10 (4.96)
External Rotation	72.9 (19.45)	72.3 (20.15)
Internal Rotation	34.5 (28.50)	34.4 (28.34)

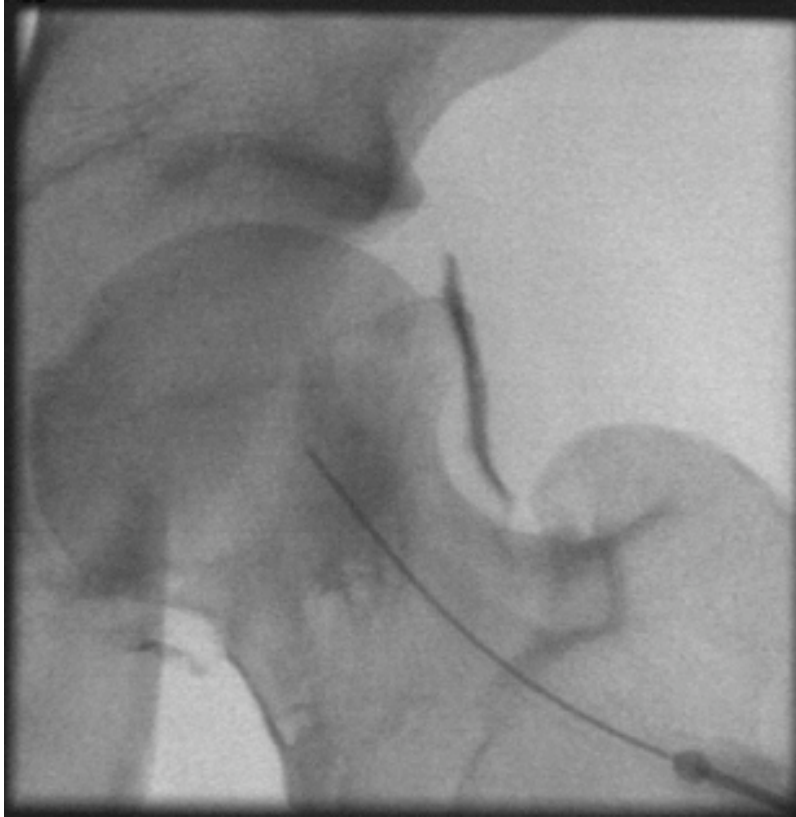
Intervention Group II: Affected Hip		
Range of Motion (degrees)	Pre-Intervention	Post-Intervention
Flexion	87.3 (18.75)	98.8 (17.09)
Extension	0.63 (9.56)	5.3 (7.27)
External Rotation	51.9 (19.02)	59.0 (17.58)
Internal Rotation	7.4 (14.35)	13.5 (14.59)
Intervention Group II: Unaffected Hip		
Flexion	103.5 (18.23)	104.8 (17.40)
Extension	7.3 (7.13)	6.5 (7.25)
External Rotation	56.4 (25.96)	56.9 (24.77)
Internal Rotation	22.4 (21.67)	22.0 (21.19)

Appendix D: Flourosopic Arthrogram Images

Magnetic Resonance Arthrogram

Sequential Flourosopic Images Showing Capsular Distension:





Subsequent MRI showing fluid volume maintained within the hip joint

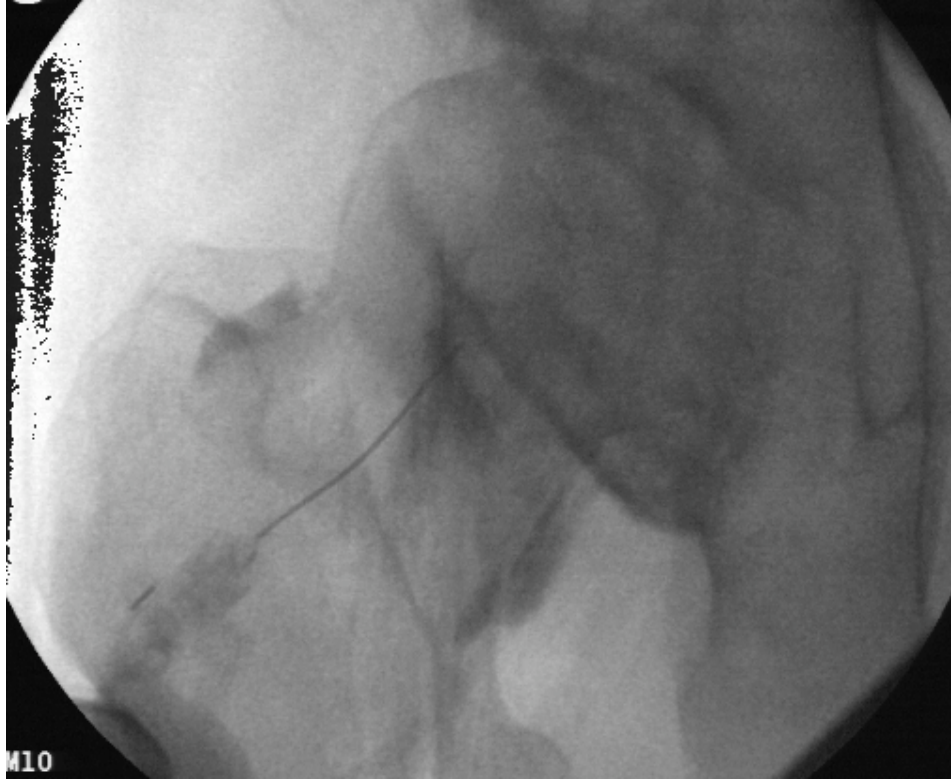


Therapeutic Arthrogram

Sequential Fluoroscopic Images showing Capsular Distension and Subsequent Capsular Rupture:

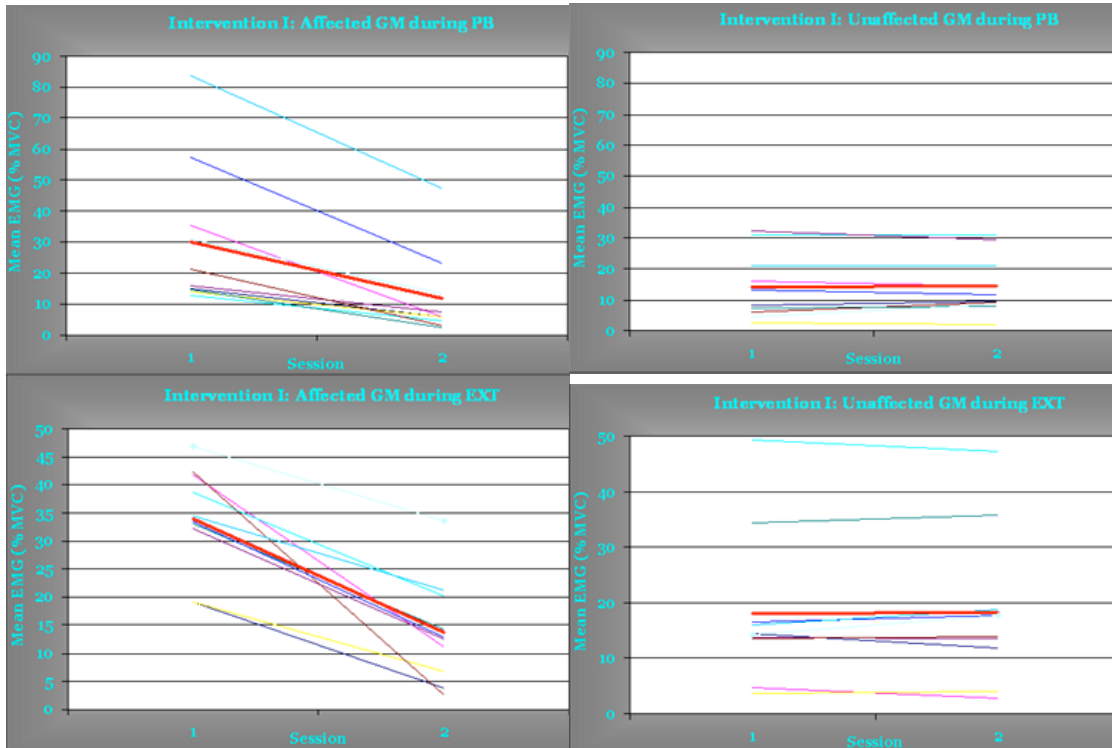




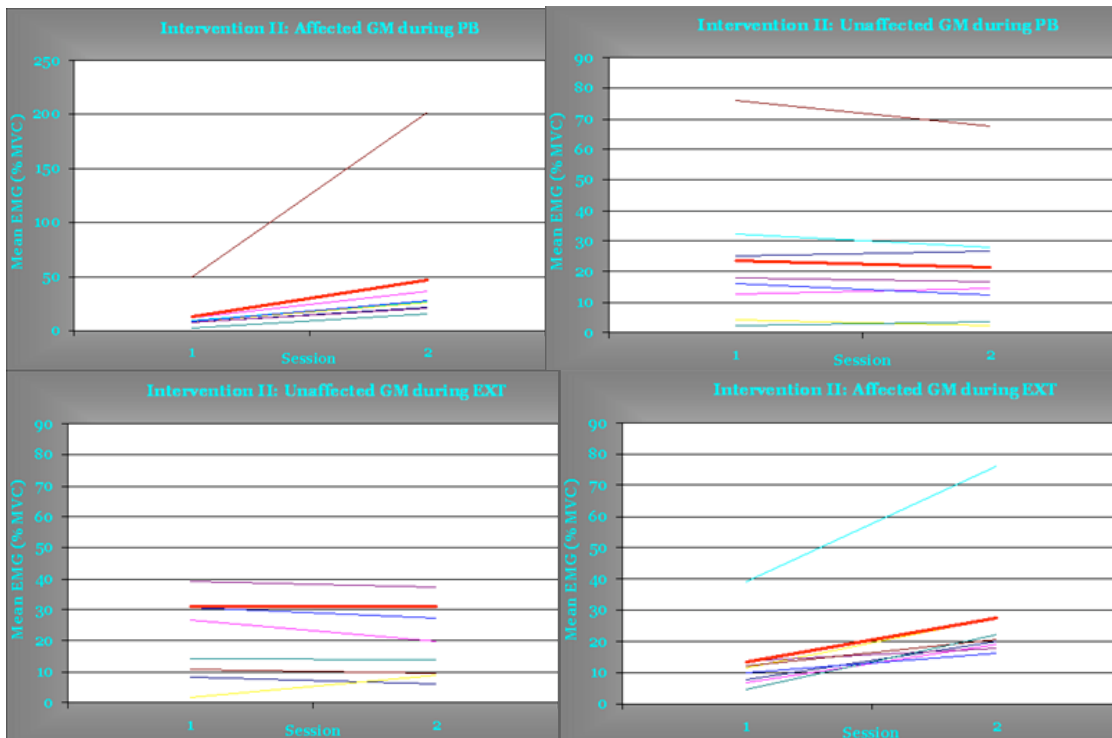


Appendix E

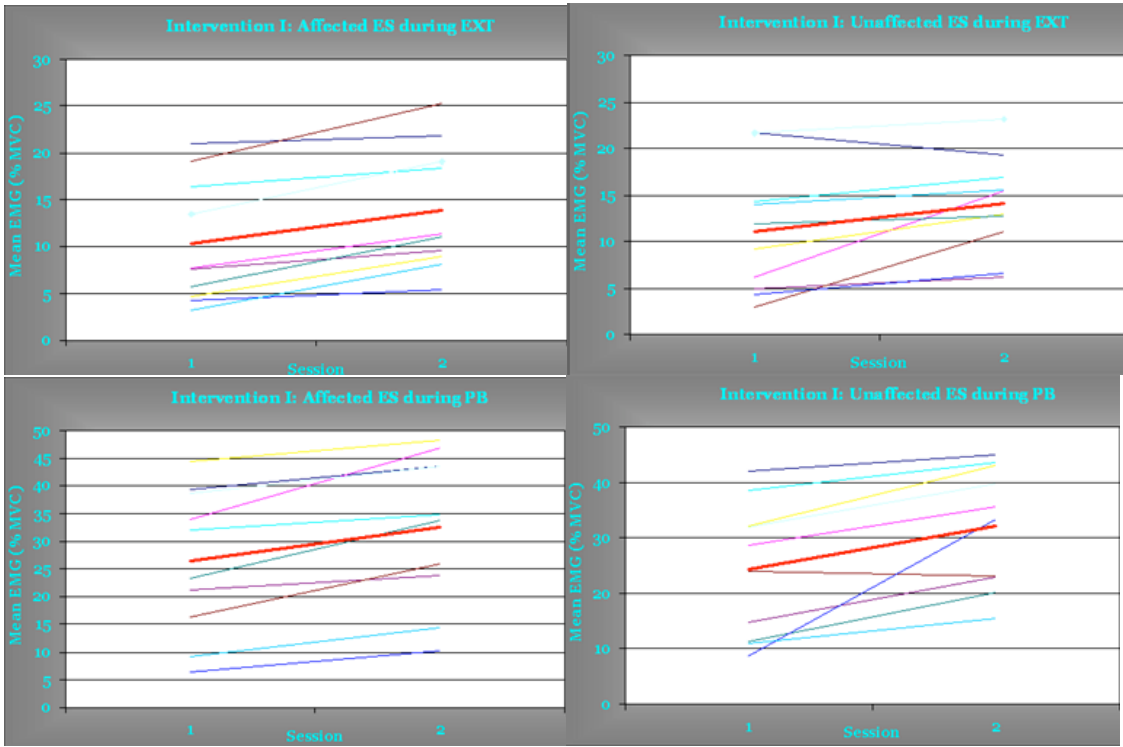
GM: Intervention I



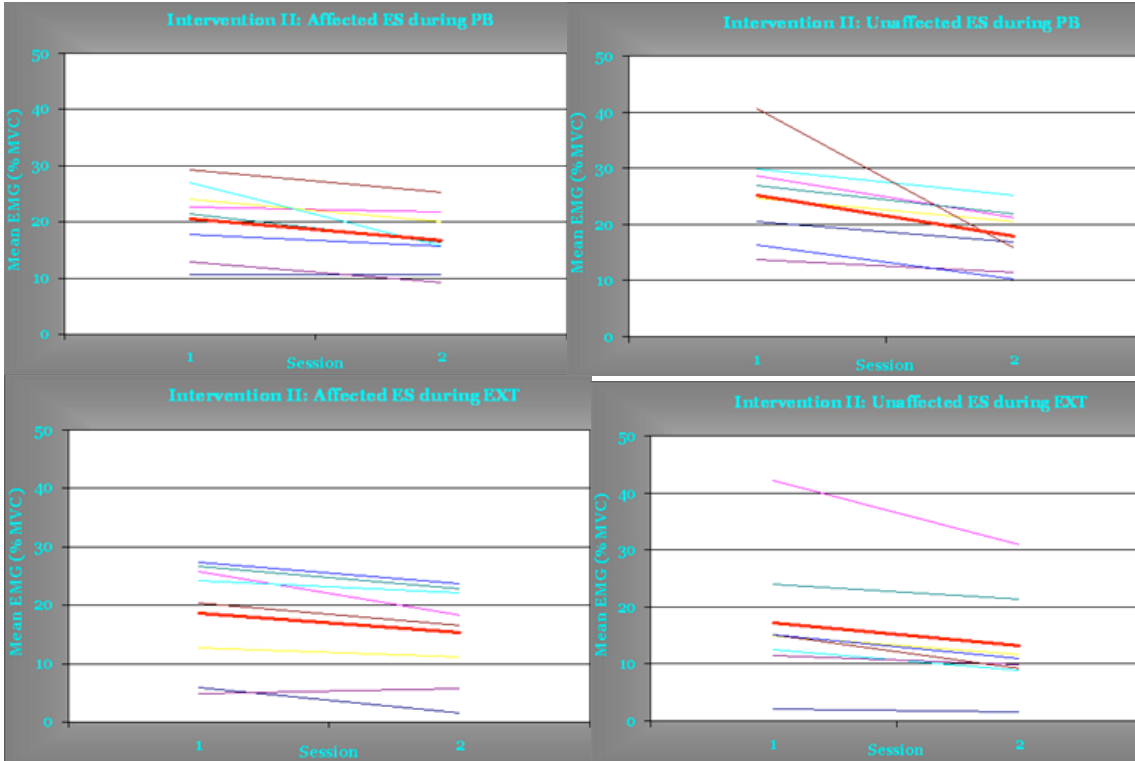
GM: Intervention II



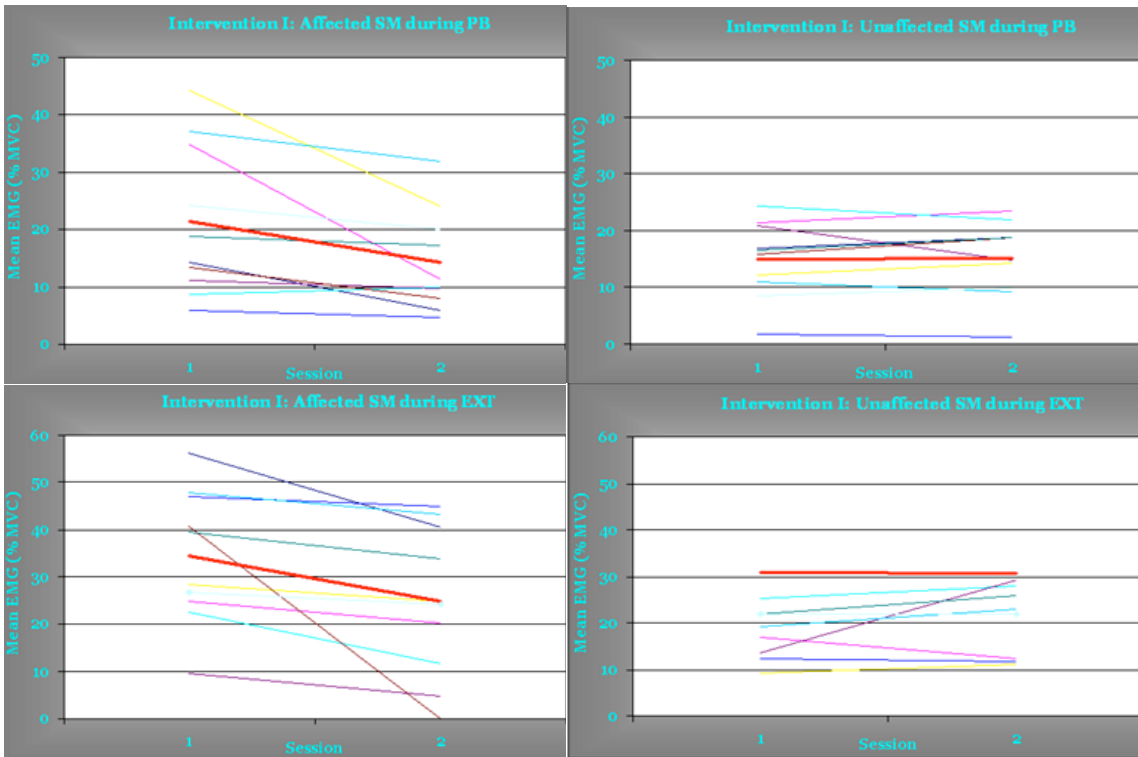
ES: Intervention I



ES: Intervention II



SM: Intervention I



SM: Intervention II

