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A Prospective Study Evaluating the Effects of Manual Therapy on the Treatment of Patellofemoral Pain

by

Conrad Tang

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

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FACULTY OF GRADUATE STUDIES

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "A Prospective Study Evaluating the Effects of Manual Therapy on the Treatment of Patellofemoral Pain" submitted by Conrad Tang in partial fulfilment of the requirements for the degree of Masters of Science.

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Abstract

Objective: To determine whether manual therapy, specifically myofascial release technique (MRT) and Trigger Point Therapy (TPT) of the quadriceps, directly affects knee-extensor voluntary activation (VA) in patients with patellofemoral pain (PFP).

Methods: This study was a randomized controlled trial (RCT). Thirty patients with PFP were recruited and randomized into group 1, who received MRT; group 2, who received TPT; and group 3, who received a sham ultrasound treatment (control group).

Main outcomes: Voluntary activation, subjective knee pain scores (Visual Analogue Scale), subjective functional knee scores (Anterior Knee Pain Score - AKPS), knee-extensor torque (Biodex machine), and quadriceps electromyography (EMG) were measured over a six week experimental period.

Results: Pain decreased in all three groups over the six week experimental period and there was a significant increase in % VA for the TPT group at 2 weeks post-baseline (9% increase) and 6 weeks post-baseline (10% increase).

Conclusion: The TPT increased % VA, whereas MRT intervention and the control condition did not. Pain decreased in all groups to a similar degree, suggesting that a placebo effect may have produced a decrease in symptoms in this chronic PFP patient group.

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"Character cannot be developed in ease and quiet. Only through experience of trial and suffering can the soul be strengthened ambition inspired and success achieved."

Helen Keller

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List of Symbols, Abbreviations and Nomenclature

Symbol	Definition
AKPS	Anterior Knee Pain Scale
CHREB	Conjoint Health Research Ethics Board
EMG	Electromyography
ITT	Interpolated Twitch Technique
MRT	Myofascial Release Technique
MVC	Maximum Voluntary Contraction
OMB	Office or Medical Bioethics
PFP	Patellofemoral Pain
RCT	Randomized Control Trial
RF	Rectus Femoris
TPT	Trigger Point Therapy
VM	Vastus Medialis
VL	Vastus Lateralis
VA	Voluntary Activation

Chapter One: **Introduction**

1.1 Problem statement

Patellofemoral pain (PFP) is a clinical condition that is characterized by retropatellar and/or peripatellar pain associated with activities involving loading of the lower limb, such as walking, running, jumping, stair climbing, and prolonged sitting and kneeling (1, 2). Patellofemoral pain is the most common overuse injury of the lower extremity, and is particularly prevalent in those who are physically active (1, 2). In a given year, for example, approximately 2.5 million runners will be diagnosed with PFP (3). Patellofemoral pain is a significant problem in the military as well, as it has been reported that 37% of recruits develop PFP while in basic combat training (1, 2). Females are twice as likely to experience PFP than males (1, 2). It is alarming that 70% to 90% of individuals with PFP have recurrent or chronic pain (1, 2). Moreover, PFP has been associated with an increased risk for developing patellofemoral osteoarthritis (1).

It is well established that with knee joint dysfunction, such as PFP, the quadriceps femoris muscles become weak even though there is no direct injury to the muscles (4). This scenario may prevent full voluntary activation of the quadriceps muscles, which results in decreased motor drive to the muscles acting across the knee (5). A decrease in voluntary activation (VA) means that there is an inability to recruit all motor units of a functional muscle group to their full extent during a maximal-effort voluntary contraction (5). This VA deficit has been linked to articular swelling, inflammation, pain, joint laxity, and structural damage (4, 6-8). The relative importance of these factors is not clearly understood. However, it is generally accepted that a VA deficit is caused by a change in the discharge of sensory receptors from the damaged knee (4, 6, 9-12). This is of concern since it may contribute to muscle atrophy, leading to weakness, and

delay effective strengthening, thereby hindering rehabilitation of PFP (13, 14). A decrease in the VA may also limit the functional recovery of muscles and joints, and it has been suggested that one of the early goals in the rehabilitation process should be to reduce or eliminate the VA deficit in order to achieve full recovery of the affected knee joint structures (15). It has been suggested that some rehabilitation protocols fail to achieve 100% recovery of VA, and this remnant reduction in VA is often blamed for the lack of success of treatment modalities (13-15). Research aimed at studying voluntary activation, and how it affects patients with PFP, has improved our understanding of PFP and its rehabilitation, but evidence-based interventions are limited.

1.2 Treatment

Treatments for anterior knee pain commonly include exercise, protective taping, correction of abnormal biomechanics and manual therapy. Physical therapy is the mainstay of non-operative management for PFP (16). For example, Crossley et al. (17) demonstrated that a 6-week program that included a standardized treatment protocol developed in a routine physical therapy practice in Australia (18) was effective in alleviating PFP, measured by a reduction in scores for average pain, worst pain and disability, compared to a placebo group (16). The treatment included patellar taping, retraining of the vastus medialis muscle using a dual-channel surface electromyography (EMG) biofeedback approach, gluteal strengthening exercises, and stretching of soft-tissue structures, such as mobilization of the patella combined with deep friction massage to the lateral soft tissues (16). The data in this study suggest that a combination of treatments is clinically efficient. It is also intuitive that treatments grouped together may provide better results than a single treatment. However, this finding prompts the question of how much manual

therapy alone can affect VA and promote the improvement of PFP. If manual therapy is usually combined with other standard treatments in the clinic, one may not know how effective it is unless it is examined in isolation. Manual therapy is a non-surgical form of management for spinal and extremity dysfunction using the human hand (19). Examples of manual therapy include massage techniques on the patient's soft tissues or the manipulation of the patient's joint, which means the use of the hands to passively move a joint for therapeutic purposes (19). Previous research suggests that manual therapy may improve knee extensor voluntary muscle activation in patients with PFP (5, 15). For example, Suter et al. (15) found anecdotal evidence that in patients with low back and knee joint dysfunction, PFP could be effectively managed through manipulative adjustment of the sacroiliac (SI) joint. Their results showed improved subjective symptoms, a significant increase in knee extensor strength, and a significant improvement in voluntary activation following manipulation treatments (15). These findings motivated a second study by the same group aimed at answering the questions: (i) does SI joint or low back dysfunction contribute to the functional deficiencies observed in the knee extensor muscles of patients with knee joint pathologies, and (ii) does correction of the SI joint dysfunction affect knee extensor voluntary muscle activation (5). The results of the follow-up study suggested that SI joint manipulation improved VA and was an effective treatment (5). However, there are few studies that look at VA in the context of manual therapy treatments, and data in this area of study are rare.

Manual therapy includes joint mobilization, manipulation and and soft-tissue treatments such as massage therapy (20). Soft-tissue techniques such as massage are quite popular and, Perlman et al. (21) showed that massage therapy was significantly better than no intervention for pain and function in individuals with mild to moderate knee osteoarthritis. There are many types of soft-

tissue treatments, and two that are commonly taught to clinicians especially in clinical professional schools are Myofascial release technique and Trigger Point Therapy. These two techniques have been around for decades and were chosen as the two forms of manual therapy used in this study.

1.3 Purpose

The purpose of this study was to determine whether manual therapy, specifically myofascial release technique (MRT) and Trigger Point Therapy (TPT) of the quadriceps, directly affects knee-extensor voluntary activation (VA) in patients with patellofemoral pain (PFP).

1.4 Rationale

Patellofemoral pain causes functional limitations in everyday life and results in loss of work and recreational play (1). Treatment for PFP is often successful in the short term, but long-term results are less promising (1). Patellofemoral pain is typically associated with a reduction in knee extensor voluntary activation (5). One of the goals of rehabilitation is to reduce or eliminate the decreased voluntary activation so that full functional recovery of the knee is obtained (13). Anecdotally, improvements in muscle activation in patients with PFP may be achieved with manual therapy as a common type of treatment option (22). However, there is limited evidence linking manual therapies with improved muscle function, specifically an increase in VA. Patellofemoral pain is the most common knee pathology encountered by orthopedic and sports medicine practitioners (23). While patellofemoral problems are evident in a wide range of individuals, it is particularly prevalent in younger persons who are physically active (2). Taunton et al. (3) suggested that approximately 2.5 million runners are diagnosed every year with PFP.

Patellofemoral pain is also a significant problem in the military, as it has been reported that 37% of recruits develop symptoms while participating in basic training (24). Females are reported to be at higher risk for the development of PFP than their male counterparts (25). Patellofemoral pain is associated with a 70% to 90% recurrence and chronic pain (26). Physicians, especially primary care doctors, are frequently confronted by patients with PFP, but treatment schedules are hard to implement as there is lack of evidence for what is the best and most cost effective treatment (27).

Therefore, it is vital that the cost and efficacy of manual therapy is available to inform clinical practitioners.

1.5 Research Objectives

The overall objective was to determine whether manual therapy, specifically myofascial release technique (MRT) and Trigger Point Therapy (TPT) of the quadriceps, directly affects knee-extensor voluntary activation (VA) in patients with patellofemoral pain (PFP).

Primary objective:

1) To quantify voluntary activation of the quadriceps muscles of PFP patients during a six-week period while patients receive a defined protocol of MRT or TP therapy or no treatment (control group).

Secondary objectives:

2) To measure the subjective pain scores of patients over the study period in the form of a visual analogue scale (VAS), for three situations: during sport activity (VAS-Physical

Activity); knee pain 1 hour after sport activity (VAS-Physical Activity 1hr); and knee pain after sitting with knees bent for 30 min (VAS-Sit).

3) To measure the subjective knee function score of patients over the study period in the form of an "anterior knee pain scale" (AKPS).
4) To measure the patients' knee extensor torque during maximum isometric voluntary contractions.
5) To measure the electromyography of the quadriceps muscles (distal region of the vastus medialis, vastus lateralis and rectus femoris) during maximum isometric voluntary contractions.

We hypothesized that the MRT and TPT manual therapies will increase the quadriceps voluntary activation in a sample of PFP patients. Furthermore, we hypothesize that these manual therapies will improve the subjective knee pain scores (VAS) and knee function scores (AKPS), and increase the knee extensor torque and the quadriceps EMG during maximum voluntary contraction (MVC).

1.6 Summary of the thesis format

In order to address each specific aim, a basic understanding of PFP and voluntary muscle activation is needed. Therefore, this thesis begins with a literature review that discusses the relevant published literature on current theories of PFP and the different types of manual therapies that will be used in this project (Chapter Two). This will be followed by a review of the reliability of the interpolated twitch technique used to quantify voluntary muscle activation (Chapter Three). The methodology for the randomized controlled trial design will be presented in Chapter Four, followed by the results in Chapter Five. A discussion of the results will be

presented in Chapter Six, and the final chapter will offer a summary of the study findings and limitations and recommendations for future research (Chapter Seven).

Chapter Two: **Literature Review**

2.1 Background

2.1.1 Definition of patellofemoral pain

Patellofemoral pain (PFP) is a clinical condition characterized by retropatellar and/or peripatellar pain associated with activities involving loading of the lower limb, such as walking, running, jumping, stair climbing, and prolonged sitting and kneeling (1). For clarity, it is suggested that the definition include all pain-related problems except for knee pain due to intra-articular pathology, peripatellar tendinitis or bursitis, plica syndrome, Sinding Larsen's disease, Osgood Schlatter's disease, neuromas and other rarely occurring pathologies; therefore, all remaining patients with a clinical presentation of anterior knee pain could be diagnosed with patellofemoral pain (PFP) (28).

2.1.2 Epidemiology

Patellofemoral pain is one of the most common lower extremity conditions seen in orthopedic practice (1). It is evident in a wide range of individuals, particularly in younger, physically active people (1). Up to 40% of clinical visits for knee problems are attributed to PFP (29, 30), and approximately 2.5 million runners will be diagnosed with PFP in a given year (1). Fairbanks and colleagues reported PFP symptoms can affect up to 30% of young students (13-19 years) and the symptoms may cause 74% of them to limit their sport activities or cease their participation in sports (31, 32). Patellofemoral pain is also a significant problem in the military as it has been reported that 37% of recruits develop PFP while in basic combat training (1). Historically PFP has been considered a problem of female adolescence (1). However, it has recently been reported

that individuals with patellofemoral osteoarthritis were significantly more likely to have experienced PFP as younger individuals (1). This, along with the reports of recurrence and chronicity of PFP, emphasizes the importance of further improvements in interventions for this population in order to reduce the incidence of disabling patellofemoral osteoarthritis later in life (1).

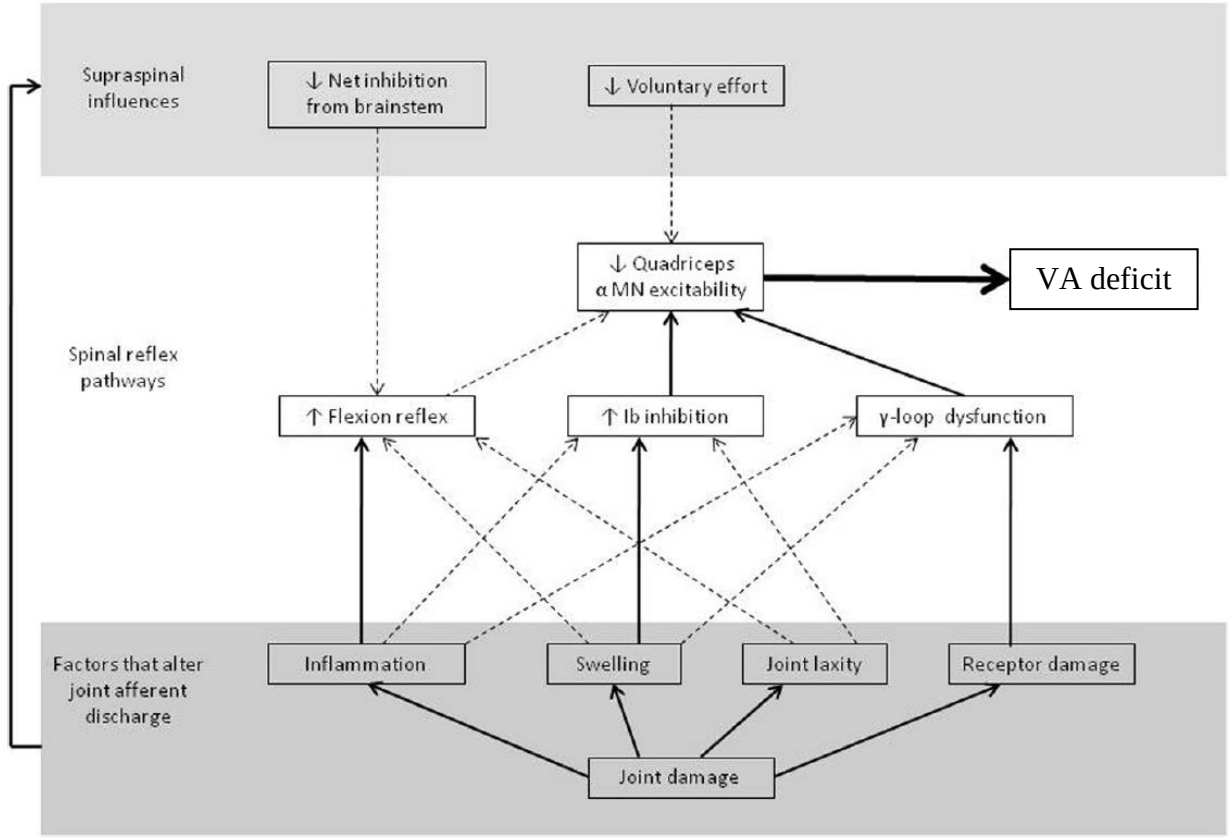
2.1.3 Etiology

The cause of patellofemoral pain (PFP) is believed to be multifactorial (1, 2). However, what seems to be missing in this debate is the source of the pain (2). The structures that may be possible sources are the synovium, lateral retinaculum, subchondral bone, and infrapatellar fat pad (2). Articular cartilage is suggested by Powers et al. (2) to be aneural, and thus provides only an indirect source of pain, perhaps through either synovial irritation or increased subchondral bone stress. There remains no consensus on causative relationships between chronic idiopathic PFP and the different mechanisms that are thought to cause it (2). One current theory is that patellofemoral malalignment and maltracking (pathomechanics) result in PFP (2). Another potential pathway to the pain is patella malalignment/maltracking, which overloads the subchondral bone and results in pain (2). Furthermore, impaired hip strength (extensors, abductors, and external rotators) may underlie the tendency of females with PFP to exhibit altered hip kinematics (2). There is also evidence that acute or chronic knee joint pathology prevents full voluntary activation of the muscles acting across the knee (33, 34). This loss of muscle strength may be caused by muscle atrophy and/or a failure to maximally activate the muscles crossing the joint (33, 34). Failure to activate all motor units of a muscle to their full extent during maximal voluntary effort has been termed a deficit in the voluntary activation (VA) of the muscle (5). The

voluntary activation deficit occurs either because not all motor units of a muscle are being recruited, or because some motor units are recruited at submaximal frequencies during maximal effort contractions (35). A wide range of reductions in VA (5%-80%) have been reported in subjects with past and present knee pathology (14, 36-39). A number of factors such as pain (33, 40), joint effusion (4, 34, 41-44) intra-articular pressure (45), and ligament sprain (46) are thought to contribute to reductions in the VA. Spencer et al. (11) found that the vastus medialis (VM) muscles of non-injured subjects displayed reductions in VA after the injection of 20 to 30mL of sterile saline into the knee joint, and the vastus lateralis (VL) displayed a reduction of VA with 50 to 60 mL of injected saline. This diminished ability for complete recruitment within the motor neuron pool is thought to be caused in part by information originating from joint receptors, primarily mechanoreceptors, which send afferent signals to inhibitory interneurons that synapse on the motor neuron (9). However, the precise mechanisms underlying the VA deficit are unknown, and in particular, the role of pain in the pathogenesis of the VA deficit is not well understood (47). Thomeé et al. (48) demonstrated that patients with PFP may have a moderate reduction of maximal motor unit activation at maximal quadriceps effort as measured during knee extension in the sitting position. Extrapolation from several submaximal levels indicated that an additional 18% (range 0 to 58%) of knee extensor torque could be generated (48). Thomeé et al. (48) stated that it is likely that the estimated additional torque that could be generated at maximal effort among the PFP patients is a result of reflex inhibition caused by afferent signals from the patellofemoral joint, as the patients also reported pain during the strength tests. A number of factors may alter the afferent discharge from the knee joint in patients with knee injury (Figure. 2.1). These factors include swelling, inflammation, joint laxity, and a loss of output from articular sensory receptors due to structural damage (6, 7, 49, 50)(6, 7,

49). In a study by Suter et al., (47) the average quadriceps VA deficit of the affected leg in subjects with unilateral PFP of varying duration was 40% ± 2.5%, measured at a knee angle of 60^{o}. Comparatively normal or healthy subjects had a quadriceps VA deficit of 7% ± 1.1% (51). With respect to knee extensor strength, Callaghan & Oldham (52) reported a lack of correlation between peak torque and the cross sectional area of the quadriceps in PFP patients. This finding indicates that PFP patients have weaker extensor muscle groups that cannot be explained by muscle atrophy alone. Thus, there may be more subtle mechanisms, other than muscle size, limiting quadriceps function. For example, EMG studies on PFP have described a modified neuromuscular control strategy of the quadriceps in patients with PFP; this is not necessarily related to pain inhibition (52).

Figure 2.1. Schematic diagram summarizing the proposed mechanisms contributing to the deficit in quadriceps voluntary activation (VA). Solid lines are mechanisms with stronger evidence to support their existence (13)**. αMN = Alpha motor neuron.**



2.1.4 Symptoms

Symptoms usually consist of diffuse pain originating from the anterior aspect of the knee, most commonly along the medial aspect of the patella; however, retro-patellar and lateral patellar pain are not uncommon (53, 54). Pain is typically reproduced on functions that increase patellofemoral compressive forces such as running, ascending and descending stairs, inclined walking, and squatting, and feels worse after exercise activity or after prolonged inactivity, particularly sitting with the knee in a flexed position ((16, 53, 55, 56). Symptoms of giving way can occur in patients with PFP, with a sudden relaxation due to pain inhibition of the quadriceps

muscles during loading of the patellofemoral joint in standing (28). This should be distinguished from giving way as a result of ligamentous instability of the knee joint or meniscal lesion during a turning movement (28). Giving way and sensations of catching may be misinterpreted in determining whether intra-articular pathology is present in the tibio-femoral joint. Patients with intra-articular pathology in the tibio-femoral joint most often experience giving way while turning (28, 54), while giving way caused by a patellofemoral problem most frequently occurs while ascending stairs or walking down an incline (48, 54). Therefore PFP has a debilitating effect on patients' daily lives by reducing their ability to perform sport- and work-related activities pain-free (57).

2.1.5 Risk factors

A systematic review by Lankhorst et al. (58) provided evidence of two risk factors for PFP: being female, and having lower knee extensor strength (for both men and women). While it is generally accepted that females are at greater risk for PFP, males do experience this injury as well (1). Males have been said to have different knee joint mechanics than females during functional activities such as running, cutting, squatting, and landing (1). Nevertheless, it is not known whether males with PFP move more like females with the disorder, or whether they present with different mechanics (1). If the mechanics are different, then it is possible that interventions for PFP should be gender specific (1). The majority of currently available studies focus on females, and it is agreed in the scientific community that more studies involving males are needed (1).

Witvrouw et al. (59) determined, in an athletic population over a 2-year period, that a shortened quadriceps muscle, an altered vastus medialis muscle reflex response time, decreased explosive

strength, and a hypermobile patella had significant correlations with the incidence of patellofemoral pain. The authors concluded that these four parameters play a dominant role in the genesis of anterior knee pain and therefore deemed them to be risk factors for this disorder (59).

2.1.6 Natural history

Stathopulu and Baildam (26) assessed the long-term outcome of PFP diagnosed in childhood. At a long-term follow-up, the majority of the respondents (91%) in the study were still having knee pain, and the pain affected daily life in 45% of the respondents, with more than one-third of them requiring pain medication. Even if all the non-responders were pain-free at the time of the follow-up, the percentage of subjects still experiencing pain (42%) would be high (26). These results suggest that PFP pain that occurs in childhood may not be a benign condition, as is sometimes thought (26).

Nimon et al. (60) showed that in females who had had pain in adolescence and were followed up at a mean interval of 16 years, 22% had no pain, 71% thought that their symptoms were better than at presentation, 88% used analgesics rarely or not at all, and 90% continued to participate regularly in sports. Nevertheless, about one in four former patients continued to have significant symptoms 20 years after initial presentation (60). No features were identified that predicted those patients in whom symptoms would persist (60).

2.1.7 Anatomy and biomechanics of the patellofemoral joint

The patellofemoral joint consists of the patella, the distal and anterior parts of the femur, the articular surfaces, and surrounding supporting structures (28). The patella is a sesamoid bone of

relatively constant length, width and thickness (28). Seventy-five percent of the posterior surface of the patella is covered by cartilage up to 5mm thick, making it the thickest in the body (28). The patella increases the lever arm for the quadriceps/patellar tendon by up to 50% and also protects the anterior part of the knee joint (54, 61-63). The patella is passively stabilized by its shape, by the femoral trochlea and by the peripatellar retinaculum (28). The dynamic stabilizers of the patella include the pes anserinus and semimembranosus muscles, rotating the tibia inward; the biceps femoris muscle, rotating the tibia outward; the vastus medialis muscle, pulling the patella medially; the vastus lateralis muscle, pulling laterally; and the vastus intermedius and rectus femoris (RF) muscles, pulling proximally/laterally (28). During extension from approximately 30° of knee flexion, the tibia rotates outwards and the patella is guided through the trochlea of the femur by the interacting heads of the quadriceps muscles (28). At full knee extension, the patella rests on the suprapatellar fat pad/bursa (28). During knee flexion from full knee extension, the distal part of the patella comes in contact with the lateral femoral condyle at 10° to 20° of knee flexion, and the patella then follows an S-shaped curve through the trochlea (28). The part of the patellar surface articulating with the femur moves proximally during flexion of the knee and the patellofemoral compression forces increase with increasing knee angles up to 90° of knee flexion; they can reach up to 8 times body weight (54, 61-63).

2.1.8 Interventions

Non-operative treatment is the mainstay of intervention for patellofemoral pain (17). Of the various non-operative management options, physical therapy remains the most commonly used (17). Crossley et al. (64) suggested that the rationale behind the use of physical therapy for alleviation of patellofemoral pain includes restoration of patellar alignment through active or

passive interventions, including protective taping, correction of abnormal biomechanics, and manual therapy. For example Crossley et al. (64) demonstrated that a 6-week program that included a standardized treatment protocol, developed in a routine physical therapy practice in Australia, was efficacious for alleviating PFP as defined by a reduction in scores for average pain, worst pain and disability, compared to a placebo group (16). The treatment included patellar taping, retraining of the VM muscle using dual-channel surface EMG biofeedback, gluteal strengthening exercises, and stretching of soft-tissue structures, such as mobilization of the patella combined with manual therapy to the lateral soft tissues (16). Lieb and Perry (65) suggested that VM muscle retraining is an essential component of treatment because this muscle can provide active medial stabilization of the patella within the femoral trochlea. Reid (66) reported that treatment protocols for PFP commonly include exercise regimens to strengthen the VM, patellar taping, stretching, soft tissue therapy, and correction of abnormal biomechanics. Recall that Powers et al. (2) suggested that impaired hip strength (extensors, abductors, and external rotators) may underlie the tendency of females with PFP to exhibit altered hip kinematics. Therefore, from a treatment standpoint, the focus on hip strength/control is a logical option from a biomechanical perspective (2). This is because control of hip internal rotation can improve patellar tracking, thereby improving contact area, and control of hip adduction can reduce the laterally directed forces on the patellofemoral joint by minimizing the dynamic Q-angle (2). Increases in hip strength are achieved through specific hip muscle exercises.

While some evidence indicates that physiotherapy can reduce the pain associated with PFP (16), there is no conclusive evidence to support the superiority of one physiotherapy intervention over the others (64). Regardless of intervention, it appears that exercises are a generally accepted option for PFP treatment. For example, an eccentric quadriceps strengthening program resulted

in a better treatment response than other forms of strengthening, particularly for functional impairment measures (64). It has been suggested in the literature that even with resistance training, quadriceps strength may remain unchanged or even decline significantly (14, 67-71), an effect attributed to the VA deficit (14, 71). It is possible that another intervention combined with exercise may improve the functional outcomes. Suter et al. (15) suggested that standard physical rehabilitation protocols typically fail to achieve 100% recovery, and decreased VA is often blamed for the lack of success of treatment. Hurley et al. (14) suggested that decreased VA needs to be overcome first, before significant improvement in muscle strength and function can be achieved. Unfortunately, there is no known treatment that reverses decreased VA successfully in a consistent manner (14).

Since manual therapy is widely used in sports medicine clinics, one must question whether manual therapies might be effective in reducing or eliminating VA. Specifically, two manual therapies, the myofasical release technique (MRT) and trigger point therapy (TP), have been suggested as methods to increase voluntary knee-extensor muscle activation (VA) in patients with patellofemoral pain (PFP).

2.1.9 Myofascial Release Technique

The myofascial release technique (MRT) is a form of manual manipulative therapy designed to treat a variety of conditions such as musculoskeletal injuries, somatic pain, fibromyalgia and chronic lymphedema (72-75). Myofascial release has been reported to improve mobility, reduce pain, and reduce inflammation (76-78). In MRT, the practitioner places digital tension on the patient's affected muscles and takes the associated joint (joint crossed by the target muscle) from a fully flexed to a fully extended position (see Appendix G for diagram). Clinical studies

investigating the efficacy of MRT have been inconsistent, with some studies reporting positive clinical outcomes and others showing no difference compared with the conventional standard of care (79-81).

2.2. Trigger Point Therapy

Clinically the trigger point is identified as a localized spot of tenderness in a nodule in a palpable taut band of muscle fibers (82). The spot is tender when pressed and can give rise to characteristic referred pain, motor dysfunction, and autonomic phenomena (82). Histopathologic mechanisms to account for the development of trigger points and subsequent pain patterns have been proposed, however scientific evidence is lacking (83). Researcher has suggested that acute trauma or repetitive microtrauma may lead to the development of a trigger point (83). Occupational or recreational activities that produce repetitive stress on a specific muscle or muscle group commonly cause chronic stress in the muscle fibers, leading to trigger points (83). Examples include an acute sports injury such as a sprain, or trauma caused by moving boxes using improper body mechanics (84). Trigger points can be treated in a variety of ways. Trigger point therapy (TPT) can involve ischemic compression, where the practitioner places a moderate amount of pressure on the affected area of the muscle and holds that pressure for a set period of time, depending on symptom control and practitioner experience (84).

2.2.1 Summary

In summary, PFP is one of the most common lower extremity conditions seen in orthopaedic practice (23). Risk factors include lower knee extension strength (for both men and women), and PFP is observed more often in females than males (58). The natural history of PFP is variable

across studies, and some even suggest that chronic PFP is a clinical syndrome characterized by persistent debilitating symptoms resistant to treatment (60). Beyond two years of chronic PFP, patients often do not improve and continued treatment may not be effective (60). Non-operative treatment is the mainstay of intervention for PFP (17). Of the various non-operative management options, physical therapy remains the most commonly used (17). Crossley et al. (64) suggested the rationale behind the use of physical therapy for alleviation of patellofemoral pain includes restoration of patellar alignment through active or passive interventions, combining different treatments. That being said, manual therapy, such as myofascial release technique and trigger point therapy, is widely used and is purported to show improved subjective symptoms, although the evidence is anecdotal. However, it is difficult to assess how effective MRT and TPT are when they are combined with other treatments in physical therapy regimes. There is an unsubstantiated belief among manual therapists that manual therapy techniques can influence muscle VA. Therefore, evidence-based evaluations of how manual techniques may affect voluntary knee-extensor muscle activation in patients with PFP would make an important contribution.

Chapter Three: **Reliability of the voluntary drive using the interpolated twitch technique**

3.1 Introduction

The assessment of maximal voluntary muscle activation of human muscles has been quantified using several methods. One of these is the so-called interpolated twitch technique (ITT) introduced by Merton (85), which has been used more often in scientific studies than other comparable techniques (13). According to Cheng et al. (86), the ITT is a practical method for determining if impairments in voluntary drive limit muscle force during and following fatiguing efforts. For ITT testing, muscles are stimulated using a maximal electrical twitch (or multiple twitches), preferably through the motor nerve, superimposed upon a maximal voluntary isometric contraction (MVC). The additional force reached during the superimposed twitch, on top of the maximal voluntary force, is thought to represent the extent to which voluntary muscle activation is impaired (86). If a single electrical stimulus is delivered to contracting muscles during a voluntary maximum isometric contraction, a twitch-like increase in force is often observed (87). The size of this 'interpolated twitch' can be used to estimate the level of neural drive to muscles (87). Therefore a large interpolated twitch indicates a low level of voluntary activation, whereas a small or non-existent interpolated twitch indicates near-maximal or truly maximal voluntary activation (85). This method has been used to show that it is possible to produce maximal, or near-maximal, voluntary activation during brief isometric contractions of limb muscles (85, 88-92).

In order to calculate an estimate of voluntary muscle activation, normalizing the superimposed twitch to a resting, control twitch provides an index of voluntary activation (VA):

VA = 1- [ITT/RTF] x 100% (86) (equation 1)

In this equation, ITT is the superimposed twitch force at maximum voluntary effort, and resting twitch force (RTF) is the twitch force measured with the muscle at rest (88). Therefore, a high value for VA means that voluntary muscle activation is nearly maximal, while a small value means that voluntary muscle activation is far from maximal. Voluntary activation for a healthy reference population is about 90% for the knee extensor muscles at a knee flexion angle of 60˚ (5). Knee extensor VA deficits have been shown to depend on the knee angle, with the greatest values at 60˚ of knee flexion, and also to depend on knee joint integrity (12). Patients with knee effusion, pain, osteoarthritis, or ligamentous injuries have been shown to have a much higher VA deficit than healthy subjects, with values averaging about 40% and peak values easily exceeding 60% (47).

The interpolated twitch technique has been validated extensively and is commonly used in neuromuscular research, although its accuracy and repeatability are not perfect and its interpretation is complex and needs to be done carefully (93). Therefore we must question whether the twitch interpolation technique provides a valid measure of voluntary muscle activation. If we accept that voluntary activation of muscles can be measured with the force they produce, then the twitch interpolation provides a valid, albeit not perfectly repeatable and somewhat qualitative, measure of muscle activation (or muscle activation failure; (93)). Any way one looks at it, the ITT gives a measure of the activation drive to muscles, and its underlying principle is sound, although obtaining good ITT results is a skill, and interpretation of ITT results needs to be made with extreme care and with all limitations of ITT in mind (93).

According to Horstman, (94) the interpolated twitch can be a useful tool in patient research as it is a relatively reliable method for specific conditions (95), and is a good way to estimate strength capacity in people who have difficulties with voluntary torque generation. Since the ITT is

known to be variable and patient-dependent (96), it is well recognized that there is inherent variability, and this variability is dependent on how the ITT is implemented. For example, Horstman (94) suggests that using triplet stimulation rather than a single twitch for the superimposed activation gives more consistent results (97), because it limits potential confounding effects, such as the length-dependent changes in calcium sensitivity, post-tetanic potentiation, and it is known to improve the signal-to-noise ratio.

In fact, Suter and Herzog (98) suggest that by adding an increasing number of electrical twitches to the single pulse, the variability in superimposed force disappears completely. However, adding a long superimposed stimulation train onto a voluntary contraction is not advised for the knee extensor muscles because of the pain it causes and the risk of producing injury (98). In accordance with expectations, Suter and Herzog (98) demonstrated that the variability in the interpolated twitch force decreased continuously when going from a single twitch to an increasing number of stimulation pulses.

Horstman (94) agrees with Taylor (93) that the superimposed stimulation technique can be useful in establishing a subject's capacity to voluntarily activate their muscles. However, as mentioned above, the conditions of testing, the equipment used, and the experimenter can affect the variations and reliability of the test. Therefore, in order to understand and assess the reliability of the ITT in our laboratory context, we performed an intra-rater reliability test in the next section.

In the tests performed in this study, the optimal superimposed stimulation was determined by increasing the voltage delivered to the femoral nerve until no further increase in force occurred, indicating that all motor units were successfully activated. Doublet twitches (2 twitch pulses of 0.8ms pulse duration separated by an 8 ms interpulse interval) were used, as that had been shown

in previous work to be the best number of pulses in limiting discomfort to the subjects and reducing variability in superimposed force (98).

3.2 Participants and methods

3.2.1 Study Design

This was a measurement study to establish the reliability of the interpolated twitch technique for a single rater. In this study, we used twitch interpolation to investigate the degree of intra-subject variability in the voluntary drive. We also investigated the repeatability of the measurements of voluntary activation on repeated testing. This information was necessary for application of the interpolated twitch technique since the thesis work was longitudinal in nature or 6 weeks to assess the quadriceps VA in patients with PFP. The voluntary activation results produced by the interpolated twitch technique were analyzed using intra-class correlation (ICC) for a single rater.

3.2.2Purpose

To investigate the degree of intra-subject variability in the voluntary drive.

3.2.3 Participants

All participant recruitment and measurements took place at the University of Calgary, Human Performance Laboratory. Written informed consent was obtained from all subjects and the Conjoint Health Research Ethics Board (CHREB) and the University of Calgary Office of Medical Bioethics (OMB) approved the study. Ten healthy participants, five men and five women whose characteristics are summarized in Table 3.1, participated in this study. All

participants were moderately active and had no knee pathology. They were recruited from among members of the Faculty of Kinesiology at the University of Calgary, Human Performance Laboratory.

Table 3.1: Participant characteristics (N=10)

Gender **Male** **Female**	**n=** **5** **5**
Age	(Mean) (SD)
Male	26.4 (2.7)
Female	24.4 (1.7)
Height	(Mean cm) (SD)
Male	181.8 (8.1)
Female	167.2 (4.6)
Weight	(Mean kg) (SD)
Male	81.3 (8.3)
Female	57.3 (5.9)
Leg tested	
Male	3 right, 2 left
Female	3 right, 2 left

3.2.3 Methods

Voluntary activation during maximal effort, isometric, knee extensor contractions was measured on two separate days. Four repeated measurements were obtained from each subject. Intra-rater reliability was calculated based on these 80 measurements (10 subjects x 4 trials x 2 days). For testing, participants were asked to perform maximum isometric knee extensor contractions with the knee angle fixed at 60^{o} of flexion. The four repeat measurements were separated by a five minute break between contractions. Participants were asked to repeat tests if they felt that the contraction was not maximal. Maximal contraction intensity was attained when two consecutive MVCs differed by less than 5%. In the ITT, a button is pressed to stimulate the femoral nerve

during the peak MVC, and the timing of stimuli was determined by the single rater through visual feedback displayed on a computer monitor to maximize the likelihood that the evoked response would occur close to the peak force achieved. Optimal stimulation was determined by increasing the voltage delivered to the femoral nerve using doublet twitches (0.8 ms pulse duration, 8 ms interpulse interval). Visual feedback of the knee extensor torque produced by the subjects was provided continuously and subjects were loudly exhorted in a standard manner to encourage maximal performance. All measurements were taken by a single rater and the voluntary activation (%) was calculated for each attempted maximal isometric voluntary contraction using equation 1.

3.2 Statistical analysis

Statistical analyses were performed using SPSS (IBM SPSS 20 for Windows). Repeatability of the results of a single rater within subjects across the four trials for the calculated % voluntary activation results on the first testing session (day 1) was quantified with the intraclass correlation coefficient. The same was calculated separately for day 2. Portney (99) has suggested that ICC values above 0.75 indicate good reliability and those below 0.75 indicate moderate reliability.

3.4 Results

3.4.1 Intra-rater reliability on the Interpolated Twitch Technique

The intra-class correlation coefficient for the measurements of the % voluntary activation using the interpolated twitch technique for measurements taken on one day was 0.93, and 48 hours

afterward the ICC was 0.95. The ICC for day one to day two was 0.95. The raw data of the % VA is summarized in Table 3.2 and the ICC is summarized in Table 3.3.

Table 3.2: Results of the % voluntary activation of the quadriceps on two separate days (N=10).

Subject	Voluntary Muscle Activation (%) Day 1 (Trial 1, 2, 3, and 4)	Voluntary Muscle Activation (%) Day 2 (48 hours later) (Trial 1, 2, 3, and 4)
1	88, 87, 89, 90	85, 86, 87, 85
2	90, 85, 87, 88	87, 85, 86, 83
3	92, 93, 88, 87	89, 90, 88, 87
4	89, 86, 88, 90	85, 86, 88, 85
5	83, 82, 81, 80	80, 82, 81, 81
6	88, 87, 90, 89	87, 89, 88, 85
7	92, 88, 87, 90	89, 88, 87, 86
8	86, 85, 86, 89	85, 84, 86, 83
9	95, 96, 92, 93	88, 89, 92, 87
10	83, 84, 85, 82	84, 81, 81, 82

Table 3.3. Results for intra-rater reliability (intraclass correlation - ICC) of the interpolated twitch technique (ITT) (N=10).

Measurement	Day 1	Day 2	Day 1-2
% Voluntary Activation	0.93	0.95	0.95

3.5 Discussion

3.5.1 Intra-rater reliability of the interpolated twitch technique

In this section of the study we looked at the intra-rater reliability of a single rater on the primary outcome measure, which was the % voluntary activation using the interpolated twitch technique. The ICC was > 0.75, (0.93 and 0.95 for days 1 and 2, respectively and 0.95 for day 1 to day 2). Suter et al. (12) suggested that the ITT should be used for repeated testing with multiple observations in single subjects. Additionally, Suter et al. (12) found no systematic difference between the right and left legs. We did not perform the ITT in the contralateral leg because we were interested in the intra-rater reliability of the measurements on the same leg tested, since it would mimic the study protocol for our patients in the study. Suter et al. (12) suggested that if healthy subjects are able to nearly fully activate the quadriceps motor units during maximal contractions, there are no systematic difference between the right and left legs.

One methodological difference between our experimental set-up and that used by Suter et al. (12) was that they used a single electrical stimulus 0.8ms in duration, where as we used a doublet stimulus with the same duration, which would elicit a lower variability with our study measurements, as discussed previously.

3.6 Conclusion

Based on our data, the intra-class correlation for the interpolated twitch technique showed high reliability (ICC>0.75), as suggested by Portney (99). The interpolated twitch techniques examined in this study have both advantages and disadvantages. For clinicians, the goal of using

the ITT is to obtain calculated measures which help determine whether treatment methods have an effect on the muscles of joint pathology, which improve voluntary activation. Test-retest reliability with the same evaluator was reliable enough to be used for the research purposes. Although the interpolated twitch technique has been validated extensively, its accuracy and repeatability are not perfect and its interpretation is complex and needs to be done carefully (93).

Chapter Four: **Methods**

4.1 Research Design

A randomized controlled trial (RCT) design was employed to determine whether manual therapy, specifically myofascial release technique (MRT) and Trigger Point Therapy (TPT) of the quadriceps, directly affects knee-extensor voluntary activation (VA) in patients with patellofemoral pain (PFP).

4.2 Study Participants

4.2.1 Sample size

Literature reviews suggested a study by Hurley and Newham (36), who determined that routine physiotherapy twice per week directly affected knee-extensor voluntary activation (VA) in patients with knee pain. However the limitation of their study was that there was no control group and only 10 patients received the physiotherapy intervention. We estimated the appropriate sample size using the mean difference pre- and post-treatment (19% voluntary activation deficit pre-treatment and 5% voluntary activation deficit post-treatment) and the corresponding standard deviations (±7% pre-treatment and ±3% post-treatment). Based on these results, we estimated that the %VA could improve by a mean difference of 14%, and the sample size was therefore set at the number of patients needed to detect a difference of 14% between the intervention group (MRT or TPT) and the control sham group. An $\alpha = 0.05$ (acceptable type I error) and $\beta = 0.20$ (acceptable type II error) were used. The sample size was calculated to be 2 participants per group or a total of 6. However, we increased the sample size to 10 participants

per group for a total of 30, which was approved by the committee during the research proposal meeting. This would allow for a drop-out rate of up to 4 participants (see Appendix A) (100).

4.2.2 Recruitment

Ethics approval for this study was obtained from the Conjoint Health Research Ethics Board (CHREB) and the University of Calgary Office for Medical Bioethics (OMB) (Ethics ID E15396).

Convenience sampling was used to recruit 30 participants with patellofemoral pain. All recruitment and measurements were performed at the University of Calgary Human Performance Laboratory, Canada. Written informed consent (Appendix C) was obtained from all participants.

4.2.3 Inclusion Criteria

All participants were initially screened via an email or telephone interview in order to see if they fit the inclusion and exclusion criteria, and then booked for an appointment with a sports physician at the University of Calgary sports medicine center for further screening. There were four sources of recruitment: (1) patients referred to primary care and orthopedic sport medicine physicians; (2) University of Calgary campus community and city fitness facilities using bulletin board posters and websites; (3) word of mouth; and (4) media advertising (see Appendix B).

The inclusion criteria were the following:

1. Male and female adults between 18-45 years of age.

2. Participants diagnosed with patellofemoral pain. Diagnosis of PFP was performed by a sports physician at the University of Calgary sports medicine center, based on meeting all of the following historical symptoms, and physical examination:

    2.1 Non-traumatic unilateral and/or bilateral peripatellar or retropatellar knee pain for at least three months but no longer than five years.

    2.2 Patellofemoral pain with and/or after activity, stair ascent or descent, or squatting.

    2.3 Possible patellofemoral pain and/or stiffness after inactivity, especially when sitting with knees in a flexed position.

    2.4 Physically active at least 30 min/day, 3-4 days/week for the past 6 months, and previous to symptoms restricting activities such as pain from running.

    2.5 For bilateral PFP patients, the knee that was considered more severe as reported by the patient was selected as the test and treatment limb.

    2.6 A subjective pain score (Visual Analogue Scale) of ≥4/10 during physical activity (note: if pain limited their activities such as running, walking was considered as physical activity).

3. Participants fluent in English.

4.2.4 Exclusion Criteria

1. Manual therapy including MRT or TPT to the lower limb (4-weeks previously), non-steroidal anti-inflammatories (NSAIDs) (5 days) and corticosteroids (4 months).

2. History of knee surgery or any significant knee injury (including but not limited to patellar

subluxations/dislocations/fractures and ligament including cruciate or other collateral ligament laxity or meniscal injuries, intra-articular pathology, and so forth).

3. Recent injuries which could have affected their knee extensor muscles.

4. Patellar tendon, iliotibial band, or pes anserine tenderness.

5. Positive patellar apprehension sign for patellar instability.

6. Chronic Osgood-Schlatter or Sinding-Larsen-Johanssen syndromes (due to the fact that subjects included are 18 and over, patients with Sinding-Larsen-Johanssen syndrome will be excluded since it only affects the skeletally immature).

7. Hip or lumbar referred pain.

8. History of head injury or vestibular disorder within the last 6 months.

9. Pregnancy.

10. Elite athletes such as nationally ranked individuals and WCB injured patients were also excluded.

The physical examination performed by a licensed sport physician from the University of Calgary Sports Medicine Centre (SMC) included the following tests to confirm the diagnosis of PFP:

1. No articular or soft-tissue periarticular effusion or bursitis (56).

2. No significant joint line tenderness (56).

3. No collateral ligament or cruciate ligamentous instability (56).

4. Peripatellar tenderness +/- mild inferior patellar pole tenderness (56).

4.2.5 Allocation concealment

The PFP patients were randomized into one of three groups following recruitment: the manual therapy treatment interventions (Myofascial Release Techniques or Trigger Point Therapy) and the sham group (unplugged therapeutic ultrasound) (detailed description to follow). Random assignment was done using an equal number of differently labeled cards (MRT, TP and sham) of the same size and weight. A person who was blinded to the study protocol randomly selected one of the labeled cards one at a time from an opaque envelope and attributed that one card to one participant at a time. Once selected, the cards were discarded and not reused. The cards were distributed in strict sequence following the participant's recruitment into the trial. In order to make sure an equal number of males and females were allocated to each group, there were two opaque envelopes, one for each gender (male or female), with the same number of cards labeled MRT, TP or Sham in each. Therefore, if a male participant arrived the person who was blinded to the study protocol chose a card from the gender-specific envelope.

4.2 Study Overview

Subjects who met all inclusion and exclusion criteria based upon the telephone screening and the physical exam by a licensed sport physician from the University of Calgary Sports Medicine Centre (SMC) were accepted into the study. On the first day of testing, participants came to the lab and the informed consent form was reviewed. Only willing participants signed the informed consent form, a copy of which was given to participants for their records. All participants were informed that they could withdraw from the study at any time without prejudice to their healthcare. During this process it was mentioned that participants would be randomly assigned to one of three different intervention groups with no disclosure of the specific types of therapies

used, in order minimize the participants' knowledge of their treatments. Neither the participants nor the evaluator were aware of the type of treatment intervention provided for each participant until after the data were collected, making this a double blinded study.

All questionnaires were answered prior to the testing or treatments. The first session involved answering the baseline questionnaires, which included the following: A 10cm Visual Analogue Pain Scale (VAS) [(a) during physical activity (for example their most recent bout of physical activity such as running or using the elliptical machine: VAS1), (b) 1 hour after physical activity (VAS2), and (c) following 30 minutes of sitting with knees flexed (VAS3)] (Appendix D); and the Anterior Knee Pain Scale (AKPS) (Appendix E). The VAS and AKPS were filled out on all visits, 8 in total within the 6-week study period. Appointment scheduling for subsequent treatment/testing was also included in the first session. Furthermore, participants were instructed to avoid consuming any medication that might influence the study. For example, if patients were to take medications during the study, they were referred back to the sports physician for follow-up and then released from the study.

Treatments alone were given on the next six visits within a two week period: three visits per week for two weeks. For example, treatments would fall on a Monday, Wednesday and Friday and be repeated the following week. There were at least 24 hours, and no more than 72 hours, between each treatment session, in order to mimic a realistic clinical visitation schedule. Quadriceps voluntary activation assessed using the interpolated twitch technique, knee extensor torque at maximum voluntary isometric contraction (Biodex Medical Systems, Shirley, NY, USA) at 60^{o} of knee flexion, and electromyography (vastus medialis, vastus lateralis and rectus femoris) were performed pre- and post-treatment interventions on the first, third and sixth visit. Three repeat measurements were performed (five minute rest between trials to prevent muscular

fatigue). Participants then stepped off the Biodex dynamometer onto a neighboring treatment table and a sports physiotherapist from the University of Calgary Sports Medicine Center or a sports chiropractor provided one of the treatments randomly selected (MRT, TPT or sham treatments). On completion of the treatment, a five minute rest was provided and then the participant went back onto the Biodex dynamometer in order to collect the post- treatment data (same testing as pre-treatment, described above) to end the testing session.

Finally, upon completion of all six treatment visits, two follow-up visits at 4 weeks (visit 7) post-baseline and 6 weeks post-baseline (visit 8 and last) with no treatment were scheduled. The two follow-up visits consisted of three repeated measurements for determining voluntary muscle activation, MVC knee extensor torque, EMG (vastus medialis, vastus lateralis and rectus femoris) of the quadriceps during MVC, VAS and AKPS.

4.4 Data collection

4.4.1 Interpolated Twitch Technique

Voluntary muscle activation was estimated using the interpolated twitch technique (ITT). This technique has been used extensively to evaluate voluntary activation in skeletal muscle (87), and at present is the most direct method to assess the extent of voluntary drive to muscles (86, 93). The ITT quantifies the percentage of voluntary muscle activation using the formula as described previously in Chapter 3, equation 1. In this equation, ITT is the interpolated twitch magnitude during maximal voluntary contraction and RTT is the magnitude of the resting twitch torque obtained after the completion of the maximal voluntary contraction at complete rest, approximately 1 second afterward (101, 102). The ITT involves applying a doublet twitch [two

maximal twitches separated by an 8ms inter-pulse interval] to the femoral nerve during a maximal isometric knee extensor contraction (85, 88). The RTT involves applying this same doublet twitch torque to the subject approximately 1s after complete relaxation from the maximal voluntary contraction. The ITT represents the number of motor units that were not, or not fully, activated during the maximal voluntary contraction. Therefore, a greater ITT is associated with a lesser amount of voluntary activation and if there is no increase in torque by the ITT, then the muscle is assumed to be maximally activated.

Isometric knee extensor contractions were performed with the subject seated and fully strapped into a Biodex dynamometer (Biodex Medical Systems, Shirley, NY, USA) using a standard set-up. Briefly, the lateral epicondyle of the femur was aligned with the axis of rotation of the dynamometer. To stabilize the leg, straps were firmly secured over the distal third of the thigh, the distal part of the shank, and the pelvis and chest to ensure that only the knee extensors contributed to the force measured by the dynamometer. Participants held onto the handle bars of the Biodex chair during each MVC trial. The quadriceps muscles were stimulated using a Grass S88 Muscle Stimulator in combination with an isolation unit approved for human use (Quincy, MA, USA). Carbon-impregnated rubber electrodes (2.5" by 4") thinly coated with a conductive gel were secured to the thigh with adhesive tape. The stimulating electrode was placed over the femoral nerve just distal to the inguinal ligament, and the second electrode was placed over the distal portion of the quadriceps muscle superior to the patella. Double square-wave pulses of magnitude sufficient to activate all motor units of the quadriceps group, and of 0.8 ms duration with an 8ms inter-pulse interval (doublet), were used for stimulation.

4.4.2 Visual Analogue Scale

Subjects filled out a 10 cm visual analog pain scale (52, 103, 104) ranging from *no pain at all* (corresponding to a value of 0 cm) to *the worst pain I can imagine* (10 cm) to indicate how much pain they felt in their knees. The 10 cm VAS is a validated method for assessment of pain (105, 106). The VAS was filled out prior to all eight visits, and was determined for three different situations: (a) during physical activity (for example their most recent bout of exercise such as running or using the elliptical machine; VAS1); (b) 1 hour after physical activity (VAS2); and (c) following 30 minutes of sitting with knees flexed (VAS3). The visual analogue scale can be found in Appendix D.

According to the Canadian Society of Exercise Physiology (107), physical activity is defined as movement that increases heart rate and breathing, or any bodily movement produced by skeletal muscles that requires energy expenditure.

4.4.3 Anterior Knee Pain Scale

Subjects filled out an Anterior Knee Pain Scale to assess knee function (108). The anterior knee pain scale was completed once per visit prior to any testing or treatment. It is a 13-item knee specific self-report questionnaire. It documents responses to six activities thought to be associated specifically with patellofemoral disorders (walking, running, jumping, climbing stairs, squatting, and sitting for prolonged periods with knees bent), as well as symptoms such as limp, inability to weight bear with the affected limb, swelling, abnormal patellar movement, muscle atrophy and limitation of knee flexion (108). The anterior knee pain scale also asks patients about the duration of symptoms and which limb(s) is (are) affected. The maximum score is 100 and lower scores indicate greater pain/disability. Scoring is hierarchical, using various types of

categorization including “no difficulty” to “unable” and “no pain” to “severe pain”. Some sections incorporate a graded scale, for example the distance a patient is able to walk or run without pain. The section on stair climbing distinguishes those with pain only on descending stairs from those who experience pain while ascending and descending stairs (109). The anterior knee pain scale for usual or worst pain is reliable, valid, and responsive for clinical trials or clinical practice in assessing treatment outcomes in patients with patellofemoral pain (110). According to Crossley et al. (110), it is suggested that a change of 10 points (out of 100) on the AKPS reflects a real change in a patient’s symptoms. The anterior knee pain scale can be found in Appendix E.

4.4.4 Knee Extensor Torque

Isometric knee extensor contractions were performed with the leg at an angle of 60° knee flexion (0° = full extension). A knee angle of 60^{o} of flexion was also chosen because this position had been found to be most sensitive to changes in voluntary muscle activation (12). Participants were asked to warm up by extending their tested leg to submaximal intensities within their ability to produce 50, 100 or 150Nm, described in more detail section 4.4.5 (electromyography), as it was a means to standardize a warm-up procedure and ultimately normalize the EMG data at MVC. Once the warm-up was complete the participant was coached on how to perform a maximal voluntary isometric knee extensor contraction at the 60^{o} knee flexion angle and hip angle of 85°. This was followed by three repeated measurements of the MVC with a five minute break between trials. Participants were asked to build up force to reach maximum levels and then hold the maximum contractions for 3-4 s. The ITT was then applied during the peak MVC, and the timing of stimuli was determined by the evaluator through visual feedback displayed on a

computer monitor to maximize the likelihood that the evoked response would occur close to the peak force achieved. A seat-belt strap was positioned across the lap in order to avoid unwanted movement of synergist hip flexors during quadriceps contractions. Maximal contraction intensity was determined when two consecutive MVCs differed by less than 5%. At least 5 minutes rest separated the protocols for measuring isometric strength for appropriate recovery. Isometric knee extensor strength was measured using a multi-joint dynamometer Biodex as described previously. The center of rotation of the knee was aligned with the axis of rotation of the dynamometer's lever arm. The force transducer was positioned with its bottom edge two fingerbreadths proximal to the medial malleolus of the test leg and fixed with a Velcro strap. Additionally, during all contractions participants were instructed to hold the handle bars at the sides.

For both MVC and VA testing, loud verbal encouragement and visual feedback using the real-time digital torque displayed on a computer monitor were used during all contractions to ensure maximal intensity. As well, the maximal torque was calculated by averaging the torques over 500ms just before the ITT. Torque was normalized to body mass to control for sex and body size differences between participants. Subjects were instructed to not smoke, drink alcohol, or exercise at least 6 hours prior to testing and to not eat food or caffeinated beverages for at least 2 hours prior to testing (107).

4.4.5 Electromyography

Self-adhesive silver chloride bipolar EMG surface electrodes were placed on the vastus medialis, vastus lateralis and the rectus femoris. The ground electrodes were applied to the tibial tuberosity. Surgical tape was used to reinforce the electrode position to limit movement during

the study. The electrodes were placed on the muscle belly according to protocols in EMG biofeedback described by Basmajian and Blumenstein (Appendix F) (111). Proper skin preparation with a shaver and alcohol was used and the accepted skin impedance was 10 kΩ or less. EMG signals were amplified 1000x within 10cm of the recording electrode. To keep electrode placements consistent for repeat visits, the electrode placement on the first visit was marked on the participant's skin. EMG data were sampled at 2000 Hz using an analog-to-digital board with a resolution of 12 bits and stored on a Compaq Presario R4000 for signal analysis. A second order, high-pass Butterworth filter (Mathworks Inc, Natiak, Mass) with a cut-off frequency of 10 Hz was used to filter the sampled data. Root mean square (RMS) values of the EMG signal were calculated for a 500ms period just prior to the ITT application. In order to assess possible changes in maximal EMG magnitude (as a measure of muscle activation) across the treatment period, EMGs at defined sub-maximal torques (50, 100, and 150Nm depending on the subject's strength) were measured, and the EMG of the submaximal trial closest to the maximal voluntary contraction was used for estimating the maximal EMG across the different treatment visits. The EMG obtained for the maximal voluntary contraction in the first visit was then assigned a value of 100% and all values from the remaining visits were compared to this initial EMG magnitude. This normalization procedure had to be done because the EMG signal magnitude can change from one visit to the next, even if placement of the recording electrodes is perfectly controlled. In order to achieve accurate submaximal torques of 50, 100, and 150Nm, subjects were provided with visual feedback of their instantaneous torque and the target torque was given by a horizontal line that subjects matched with their instantaneous torque for a 5s period.

4.4.6 Blinding

Participants and the examiner of the data were blinded to the patient's intervention allocation. Participants were blinded in the sense that they did not know the intent of the study, and which was the sham treatment. The examiner was blinded as all data entry was performed in a coded manner, not revealing any patient characteristics.

4.5 Study Intervention

4.5.1 Myofascial Release Technique Treatment

The myofascial release technique (MRT) is a form of manual manipulative therapy designed to treat a variety of conditions such as musculoskeletal injuries, somatic pain, fibromyalgia and chronic lymphedema (72-75). These claims have not been supported by systematic experimental research. Therefore one reason for the current study was to determine whether MRT aimed at the quadriceps muscles affects the voluntary knee-extensor muscle activation in patients with PFP by way of pain reduction. The myofascial release technique involves direct superficial pressure on the soft tissues, or low-load prolonged gentle distraction of the tissues (112-114). In MRT, the involved tissue is passively taken from a shortened position (knee fully extended) to a fully lengthened position (knee fully flexed), while the contact hand holds tension longitudinally along the soft tissue fibers (115). For example, a clinician places her/his thumb on the vastus lateralis muscle and applies tension longitudinally along the muscle fibers while passively bringing the patient's knee from full extension to full flexion (see Appendix G).

The first intervention group (n_1=10) received MRT. Myofascial release technique was applied to the vastus medialis (VM), the vastus lateralis (VL), and the rectus femoris (RF) muscles. Eight

repetitions of MRT, corresponding to eight knee flexion cycles, were applied to each of the target muscles during each treatment visit (6 treatment visits total). A clinician with two years of clinical experience using MRT and trained in MRT treatment and delivery administered all MRT treatments.

4.5.2 Trigger Point Therapy

For clinical comparison, trigger point therapy (TPT) is another soft-tissue technique that has been used frequently by clinicians (116). Trigger point therapy is administered with the clinician putting firm pressure onto the affected area and gradually increasing the pressure until the patient's pain tolerance has been reached; this is called ischemic compression (see Appendix H). For further clarity, only the technique of TPT with ischemic compression was performed, however trigger point is defined as a focus of hyperirritability in a tissue that, when compressed, is locally tender and, if sufficiently hypersensitive, gives rise to a referred pain and tenderness and sometimes to referred autonomic phenomena and distortion of proprioception (82).

The TPT intervention group (n_2=10) was given pressure treatments on the vastus medialis, vastus lateralis, and rectus femoris for the six treatment visits. It consisted of the clinician applying a slowly increasing pressure over areas of elicited pain, tender point(s), for a duration of 15 seconds and five times for each muscle (VM, VL and RF) (see Appendix H).

The rationale for TPT was that myofascial disorders, such as trigger and/or tender points in the quadriceps femoris, overlap with myofascial pain and dysfunction, and that this myofascial pain may be a factor in the etiology, aggravation, and/or treatment of PFP (117). Additionally Hains (118) suggested that PFP may be managed conservatively by myofascial techniques such as TPT to the knee. Furthermore, myofascial trigger point pain has been associated with PFP, resulting

in reductions in voluntary muscle activation, decreased muscle strength, and reduced muscle flexibility (119, 120).

4.5.3 Sham Therapeutic Ultrasound Treatment

In order to compare the two manual therapy treatments (MRT and TPT) and evaluate their clinical efficacy, a control group receiving a sham treatment was added to identify possible placebo effects. Therefore, the third "intervention" group (n_3=10) received a placebo treatment in the form of sham therapeutic ultrasound treatments. The sham ultrasound treatment was applied to the skin on top of the RF, VL, and VM muscles for all six treatment visits. The therapeutic ultrasound was initially turned on so that the participant could hear the sound of the beep and be aware that they would be receiving a form of treatment. When the participants laid supine in the ready position for receiving the treatment, the ultrasound machine was turned off quietly, which did not make any noise. Questioning of the participants after the clinical trials showed that none of the subjects was aware that the ultrasound machine was turned off while they received their expected "treatment" intervention. The clinician then applied the ultrasound head covered with gel over the skin on the entire length of the RF, VL and VM for approximately four minutes per muscle for a total of 12 minutes (see Appendix I). This time frame was used to match studies that have been used for musculoskeletal pain such as knee extensor mechanism disorders, which have been shown to decrease pain in the knee extensor muscles (121, 122).

The sham ultrasound treatment was given because therapeutic ultrasound is frequently used in the treatment of musculoskeletal disorders and has been recommended for the treatment of patients with PFP (123). Ultrasound treatment is assumed to have a thermal and a mechanical effect on the target tissue, resulting in an increased local metabolism, circulation, extensibility of

connective tissues, and help accelerating tissue regeneration (124). Beneficial effects of ultrasound treatments include improvement of pain, swelling, and range of motion (124).
The sham ultrasound treatment was considered a placebo treatment, because the actual treatment effect is associated with the ultrasound penetrating the painful soft tissues, but the ultrasound emission was turned off. In randomized clinical trials, placebo control interventions are designed in such a way as to have the appearance of an experimental intervention, but without the essential or 'specific' content (here, ultrasound emission). We felt that this condition of a proper sham treatment was achieved in this study.

4.6 Outcomes

4.6.1 Primary outcome

The primary outcome variable for this study was the change in the percentage of voluntary muscle activation of the quadriceps muscles of PFP patients, measured from the baseline and up to the six week follow-up. Voluntary activation was measured using the interpolated twitch technique (see Chapter 3). The percentage voluntary muscle activation represents the most direct method to assess the extent of voluntary drive to muscles (125).

4.6.2 Secondary outcomes

There were four secondary outcomes. They included a measure of knee pain, knee function, knee extensor strength and knee extensor activation measured using EMG. These secondary outcome measures are detailed below:

1. Pain: The change in the visual analogue pain scale between baseline and 6 weeks follow-up was assessed. Pain was determined for three situations and was assessed prior to each testing and treatment visit: (a) Pain during physical activity (for example their most recent bout of physical activity such as running or using the elliptical machine) (VAS1); (b) one hour after physical activity (VAS2); and (c) following 30 minutes of sitting with the knees in a flexed position (VAS3).

2. Knee function: The change in knee function between baseline and 6 week follow-up was assessed using a knee function questionnaire (108). The maximum score in this questionnaire is 100, and lower scores indicate greater pain/disability.

3. Strength: The change in knee extensor torque measured during maximal voluntary isometric contractions on the Biodex was determined, between the baseline and the 6 week follow-up.

4. EMG activation: The change in quadriceps (VM, VL and RF) electromyography was analyzed between baseline and the 6 week follow-up.

4.7 Analysis

All data analysis was done using IBM SPSS Statistics 20 statistical software. Differences in demographic characteristics between intervention groups were analyzed with a one-way analysis of variance (ANOVA) with a post-hoc Tukey's test (N=30). Differences in both primary and secondary outcome measures between intervention groups were also analyzed with a one-way ANOVA with a post-hoc Tukey's test (N=30). If we use the %VA as an example, a one way ANOVA was used to analyze differences among the three groups at baseline. Then another one-way ANOVA was used to analyze differences among the three groups at 2-weeks post-baseline. Finally another one-way ANOVA was used to analyze differences among the three groups at 6-

weeks post-baseline. Therefore a total of three one-way ANOVA analyses were made for each outcome measure in order to compare between groups at the three time points (baseline, 2 and 6-weeks post-baseline). Differences in both primary and secondary outcome measures within groups were analyzed with a non-parametric Friedman's test with a pair-wise post-hoc testing (n=10). This was performed at baseline, 2-weeks and 6-weeks post-baseline. Therefore, a total of three Friedman tests were performed for each outcome measure in order to compare within groups at the three time points (baseline, 2 and 6-weeks post-baseline). The level of significance was set at $p<0.05$ for both the ANOVA and Friedman's tests. All figures were described with mean ± standard error of measurements (±SEM) and tables were described with mean ± standard deviations (SD) and 95% confidence intervals.

4.8 Data management

All experiments were performed in the Human Performance Laboratory (HPL) at the University of Calgary. All equipment was available within this facility. Data analysis took place on a laboratory computer within the HPL that had the necessary software installed and ready to go. Technical support for the equipment and data collection was available through the HPL.

The data obtained and the signed consent forms were kept private to conceal all personal information of the participants. The data obtained were stored on a personal computer which was password protected and the informed consent forms were stored in a locked filing cabinet within the HPL. Participants' confidentiality was maintained throughout the study and during the reporting of the results, as the results will be presented without subject identification.

The total time commitment for each subject was approximately three hours per visit for eight visits) for a total of 24 hours (not including any travel and waiting times).

4.9 Ethical Considerations

All participants were required to read, understand, and sign an informed consent form to establish that they were aware of the potential risks of participating in this study. The participants had the right to leave the study at any point without question, and were free from harm at all times. The participants were also able to contact the researcher with any questions at all times during the study either in person or through email.

Participants in the sham treatment group who felt that they had not improved after the study period were given a standard rehabilitative program for PFP patients, which included corrective rehabilitative exercises, manual therapy and patient education by a licensed clinician.

Chapter Five: **Results**

5.1 Participants

Participant characteristics are presented in Table 5.1. Participants in each group were similar in age, height and weight. No significant differences were found in the duration of PFP, normalized knee-extensor strength and % VA.

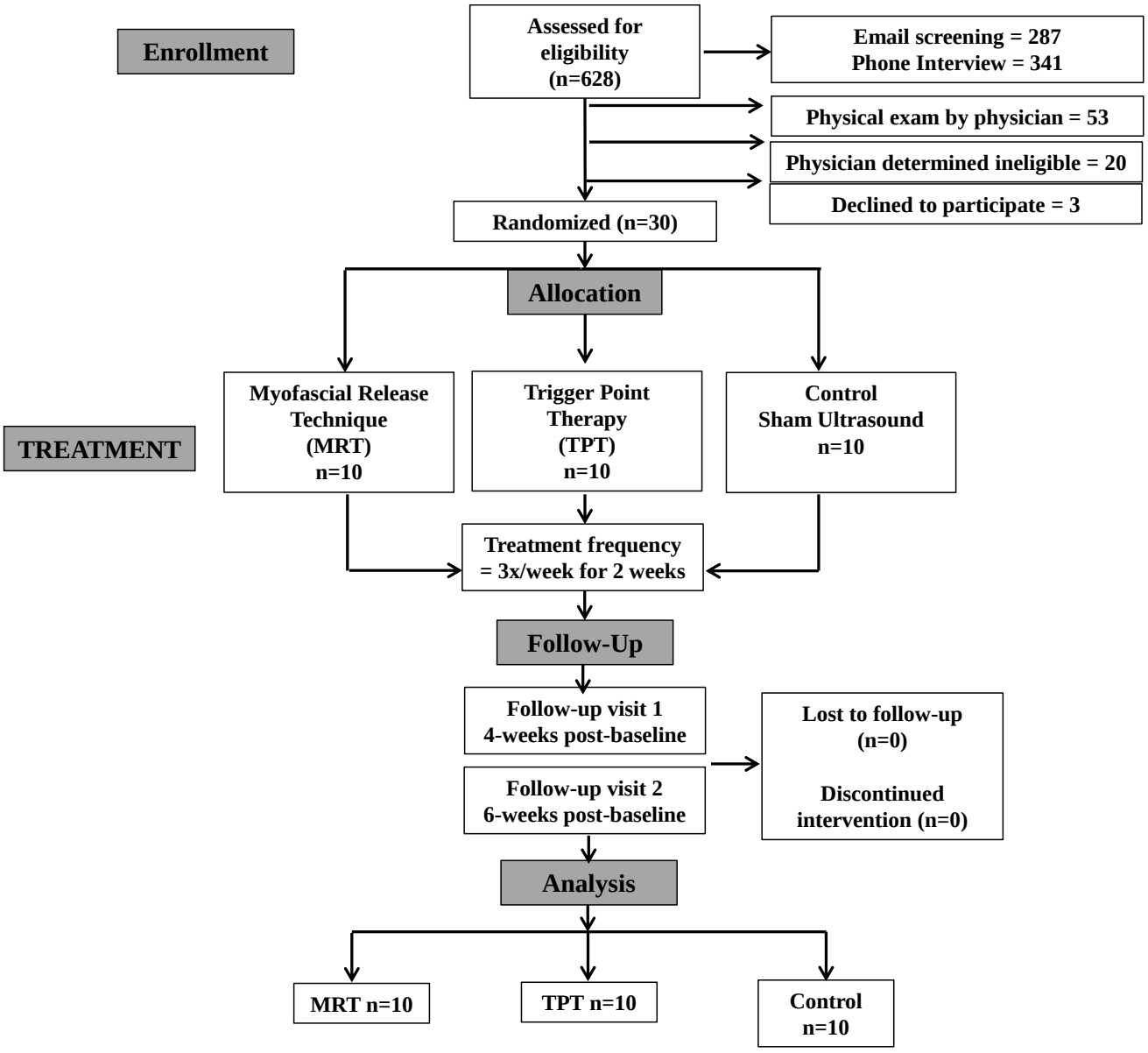
Table 5.1. Participant characteristics (N=30).

	MRT	**TP**	**Control**	**p-value**
Sex (Male/Female)	4/6	5/5	4/6	
Age (years)	31.3 ± 6.6	26.2 ± 9.6	27.7 ± 8.3	0.38
Height (cm)	171.6 ± 6.2	178.9 ± 7.7	175.6 ± 12.3	0.22
Weight (kg)	69.7 ± 14.5	73.5 ± 14.0	72.1 ± 11.9	0.82
Duration of PFP (years)	2.4 ± 1.5	1.4 ± 1.1	1.6 ± 1.4	0.23
% Voluntary Activation (Baseline)	83 (75-90)	82 (78-86)	87 (81-93)	0.44
Visual Analogue Pain Scale (10cm) During Physical Activity (Baseline)	7.5 ± 0.84 (6.9-8.1)	7.2 ± 1.3 (6.3-8.1)	7.1 ± 0.7 (6.6-7.5)	0.64
Visual Analogue Pain Scale (10cm) 1-hr Post-Physical Activity (Baseline) (100mm)	6.6 ± 1.5£ (5.5-7.7)	5.5 ± 0.84 (4.9-6.1)	5.1 ± 0.84* (4.5-5.7)	<0.05
Visual Analogue Pain Scale (10cm) after sitting knees bent 30min (Baseline)	5.1±1.0 (4.4-5.8)	4.9 ± 0.78 (4.3-5.4)	4.9 ± 0.77 (4.3-5.4)	0.80
Anterior Knee Pain Scale Questionnaire (Baseline) (out of 100)	73.9 ± 11.7 (65.5-82.3)	81.0 ± 10.3 (73.6-88.4)	81.9 ± 10.9 (74.1-89.7)	0.22
Strength (Nm/kg) (Baseline)	2.0 ± 0.5 (1.7-2.4)	2.6 ± 0.4 (2.3-2.9)	2.5 ± 0.7 (2.0-3.0)	0.077
EMG Vastus Medialis (Distal region) (mV)	0.60 ± 0.14	0.67 ± 0.20	0.64 ± 0.43	0.64
EMG Vastus Lateralis (mv)	0.29 ± 0.07	0.30 ± 0.10	0.30 ± 0.19	0.84
EMG Rectus Femoris (Baseline) (mV)	0.24 ± 0.07	0.34 ± 0.14	0.32 ± 0.16	0.21

Data are presented as means ±SD, and 95% confidence intervals. Electromyography = EMG and its 95% confidence intervals are presented in Table 5.2. MRT = Myofascial Release Technique; TP = Trigger Point Therapy, Control = Sham Therapeutic Ultrasound. Statistical significance is at the 5% level.
*Significantly different from the MRT group after post hoc testing ($p<0.05$).
†Significantly different from the TP group after post hoc testing ($p<0.05$).
£Significantly different from the control group after post hoc testing ($p<0.05$).

Over 600 participants applied to the study, and 35 were eligible. 590 participants did not fit the criteria and 5 who were eligible declined to take part in the study. See CONSORT flow diagram (Figure 5.1).

Figure 5.1. CONSORT Flow Diagram



5.2 Data presentation

All results are shown for three time points during the experimental period: (i) at baseline, (ii) 2 weeks past the baseline (i.e., at the end of the treatment period), and (iii) at 6-weeks past the baseline (i.e., during the last follow up measurements). Complete results for each data collection session are shown in Appendix J.

5.3 Primary outcome measure

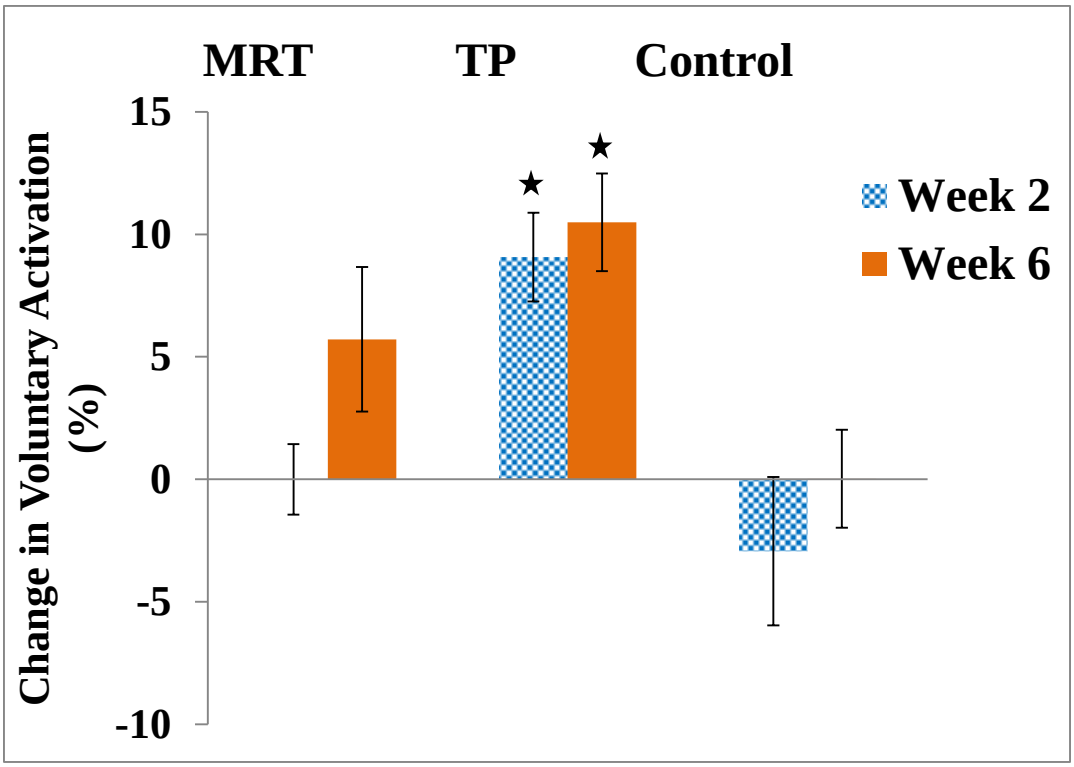
5.3.1 The % Voluntary Activation (VA) measured using the interpolated twitch technique

There was a significant increase in % VA from the baseline to the 2 weeks (9% increase) and the 6 weeks (10% increase) post-baseline evaluation times for the TP treatment group patients (Figure 5.2). MRT group and control group patients did not show significant changes in maximal voluntary knee extensor activation. Recall that normalizing the superimposed twitch to a resting control twitch provides an index of %VA:

%VA = [1-(Superimposed Twitch/Resting Twitch)] x 100% (88)

Figure 5.2. Mean (±1SEM) changes in voluntary activation (%) from baseline to 2 weeks post-baseline and 6 weeks post-baseline.

Asterisks indicate a significant differences between the baseline voluntary activation (%) and 2 weeks post-baseline and/or 6 weeks post-baseline for each treatment ($\alpha<0.05$). Positive values indicate an increase in % voluntary activation. MRT = Myofascial Release Technique; TP = Trigger Point Therapy, Control = Sham Therapeutic Ultrasound.



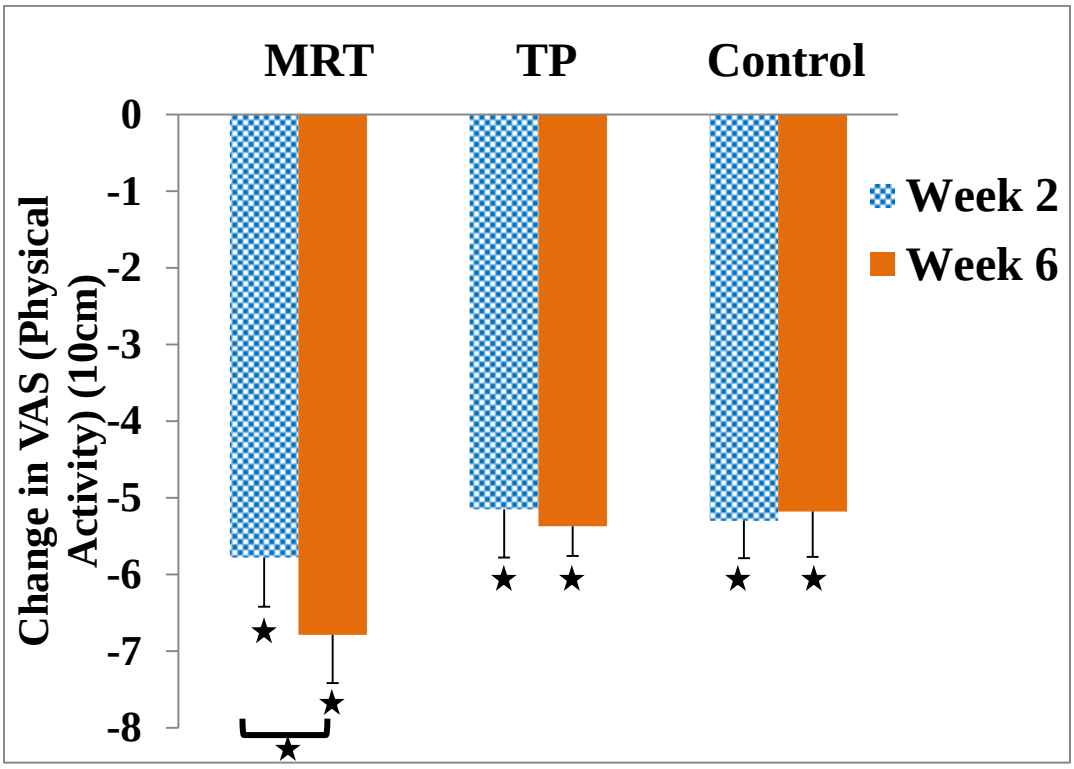
5.4 Secondary Outcome Measures

5.4.1 Visual Analogue Pain Scale (VAS) values during physical activity

There was a significant decrease in pain for all groups from baseline to 2 weeks post-baseline, and a further decrease from 2 weeks to 6 weeks post-baseline for the MRT group patients (Figure 5.3).

Figure 5.3. Mean (±1SEM) changes in Visual Analogue Scale (10cm scale) during physical activity from baseline to 2 weeks post-baseline and 6 weeks post-baseline.

Asterisks indicate a significant difference between baseline VAS values and those recorded either 2 weeks or 6 weeks post-baseline for each treatment group ($\alpha<0.05$). MRT = Myofascial Release Technique; TP = Trigger Point Therapy, Control = Sham Therapeutic Ultrasound.

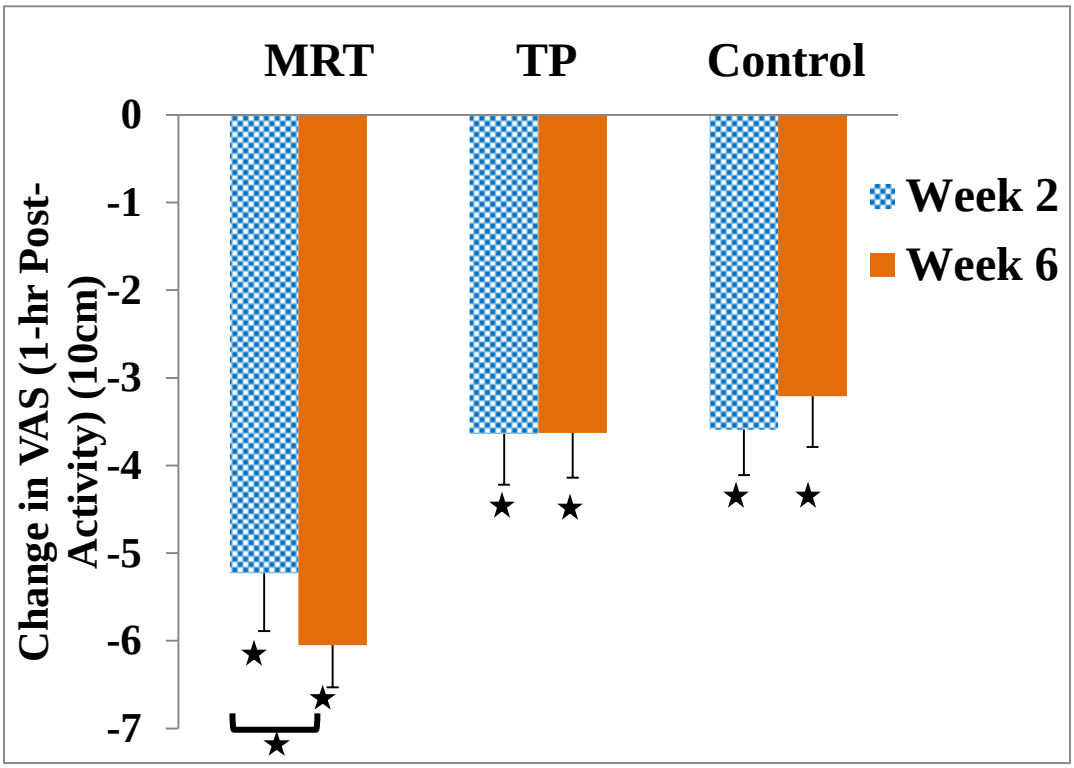


5.4.2 Visual Analogue Pain Scale (VAS) values 1-hr post physical activity

There was a significant decrease in post-activity pain at 2 and 6 weeks post-baseline compared to baseline for all three experimental groups and a further decrease from 2 to 6 weeks post-baseline for the MRT group patients only (Figure 5.4).

Figure 5.4. Mean (±1SEM) changes in Visual Analogue Scale (VAS) (10cm scale) 1-hr post physical activity from baseline to 2 weeks post-baseline and 6 weeks post-baseline.

Asterisks indicate a significant difference between the baseline VAS and 2 weeks post-baseline and/or 6 weeks post-baseline for each treatment ($\alpha<0.05$). Myofascial Release Technique; TP = Trigger Point Therapy, Control = Sham Therapeutic Ultrasound.

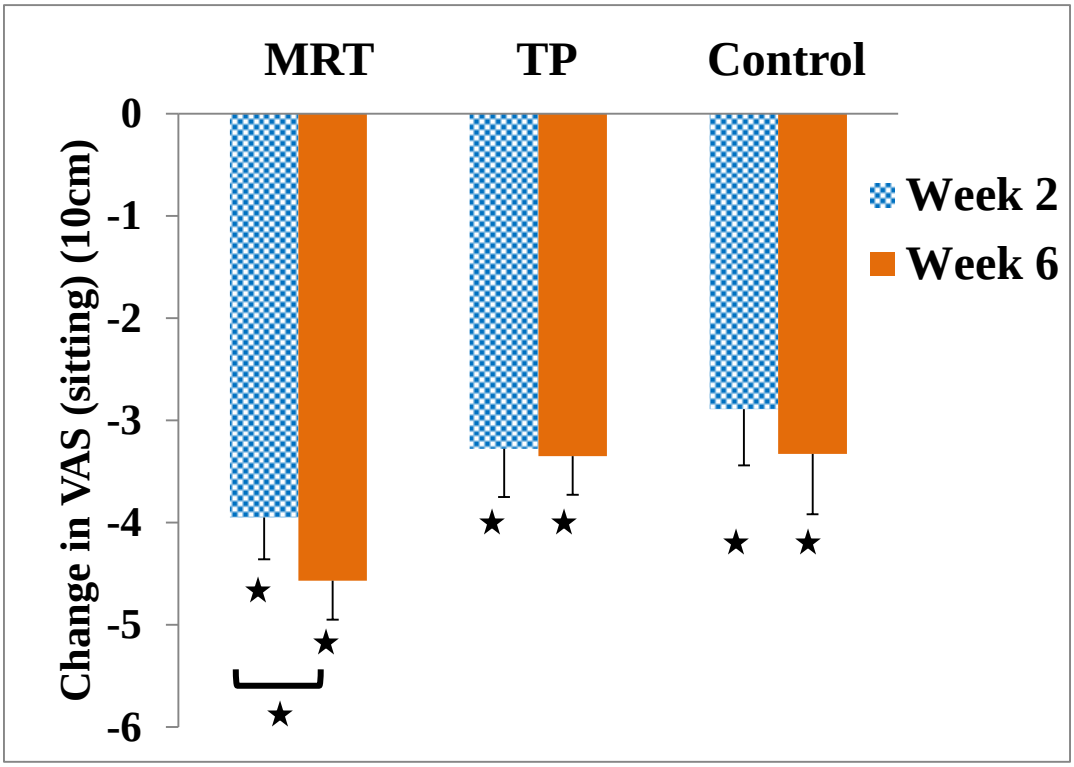


5.4.3 Visual Analogue Scale measured after sitting with knees bent for 30 min

There was a significant decrease in resting VAS values from baseline to 2 and 6 weeks post-baseline values for all three experimental groups and a further decrease from 2 to 6weeks for the MRT group patients (Figure 5.5).

Figure 5.5. Mean (±1SEM) changes in resting Visual Analogue Scale (VAS) (10cm scale) after sitting with knees bent for 30 min from baseline to 2 weeks and 6 weeks post-baseline.

Asterisks indicate a significant difference between the baseline VAS and 2 weeks post-baseline and/or 6 weeks post-baseline for each treatment ($\alpha<0.05$). Myofascial Release Technique; TP = Trigger Point Therapy, Control = Sham Therapeutic Ultrasound.

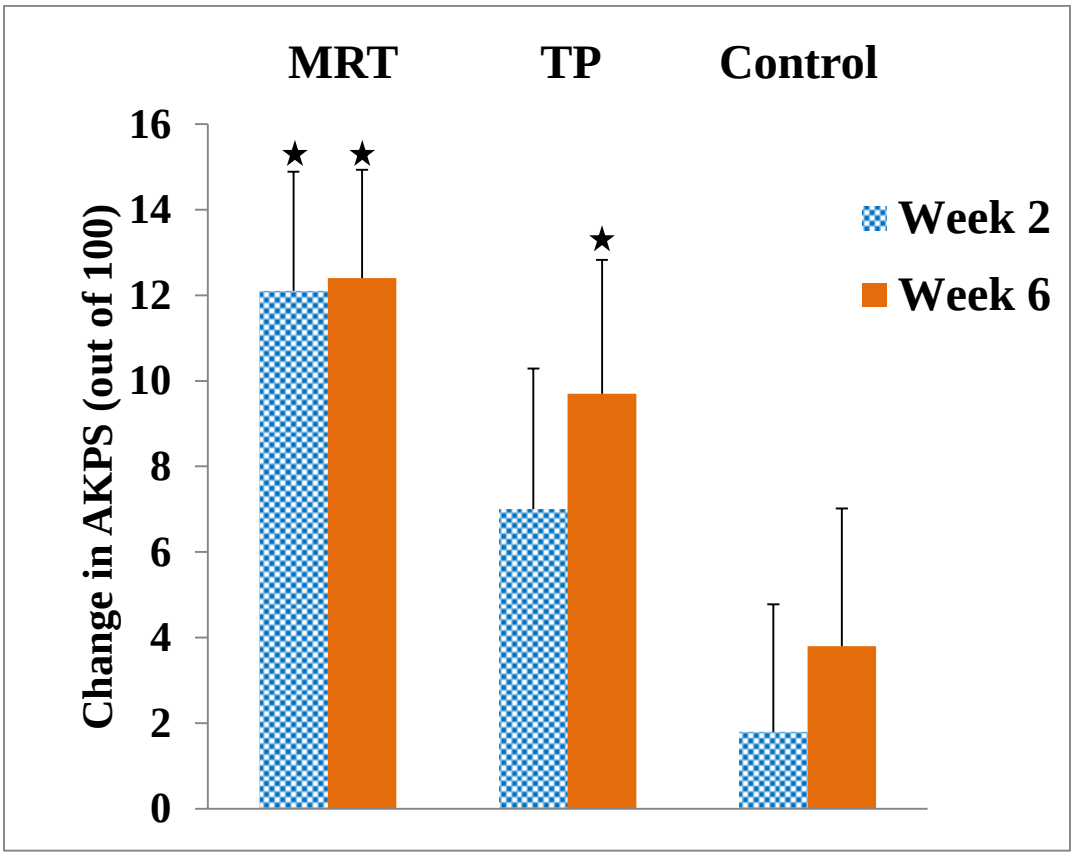


5.4.4 Functional Anterior Knee Pain Score (AKPS).

There was a significant increase in the functional knee joint score for the MRT and TP group patients. For the MRT group patients, this increase in function was found at 2 weeks and 6 weeks post-baseline. For the TP group patients, the increase in functional knee joint score only occurred at the 6-weeks post-baseline evaluation point. Control group patients did not show significant increases in the functional knee joint score at either of the two evaluation times (Figure 5.6).

Figure 5.6. Mean (±1SEM) change in functional knee joint score (AKPS - out of 100) from baseline to 2 weeks and 6 weeks post-baseline.

Asterisks indicate a significant difference between the baseline score and 2 weeks post-baseline and/or 6 weeks post-baseline scores for each treatment group ($\alpha<0.05$). Myofascial Release Technique; TP = Trigger Point Therapy, Control = Sham Therapeutic Ultrasound.

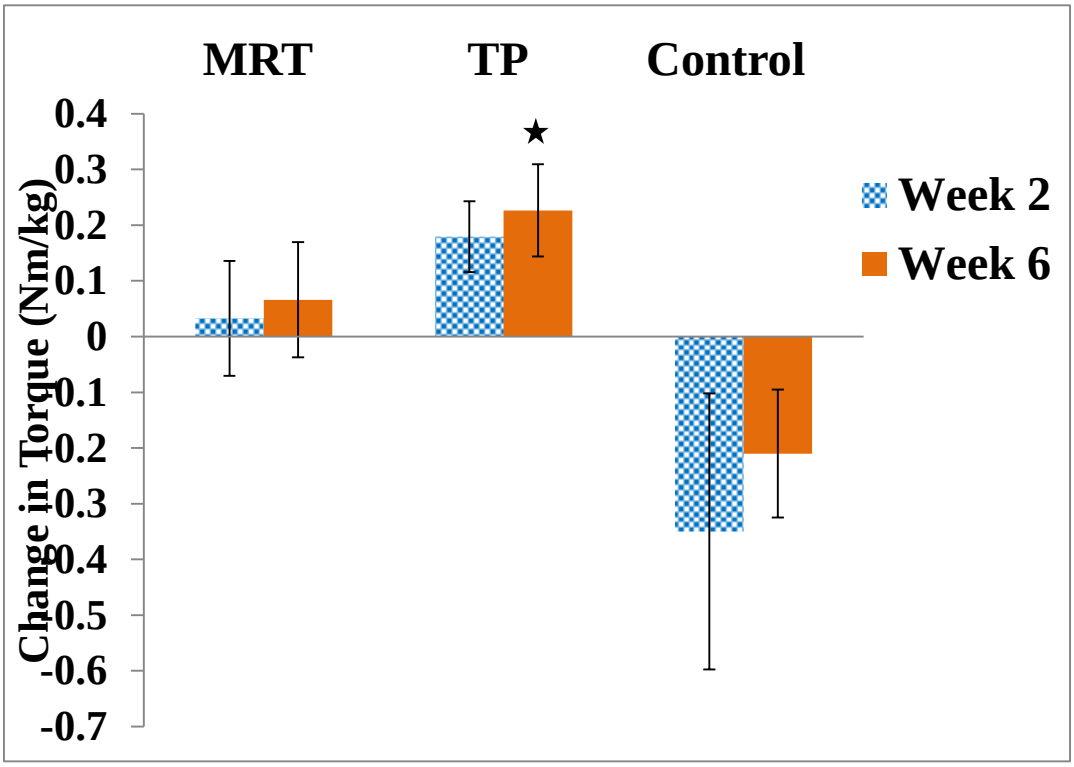


5.4.5 Knee-extensor torque

Isometric knee extensor torques remained unchanged from baseline values for all groups and at both time points of evaluation, except for the values for the TPT group patients which were greater at 2 weeks post-baseline (Figure 5.7).

Figure 5.7. Mean (±1SEM) changes in peak knee-extensor torque normalized to body weight (Nm/kg) from baseline to 2 weeks and 6 weeks post-baseline.

Asterisks indicate a significant difference between the baseline knee extensor torque and 2 weeks post-baseline and/or 6 weeks post-baseline scores for each treatment ($\alpha<0.05$). Myofascial Release Technique; TP = Trigger Point Therapy, Control = Sham Therapeutic Ultrasound.

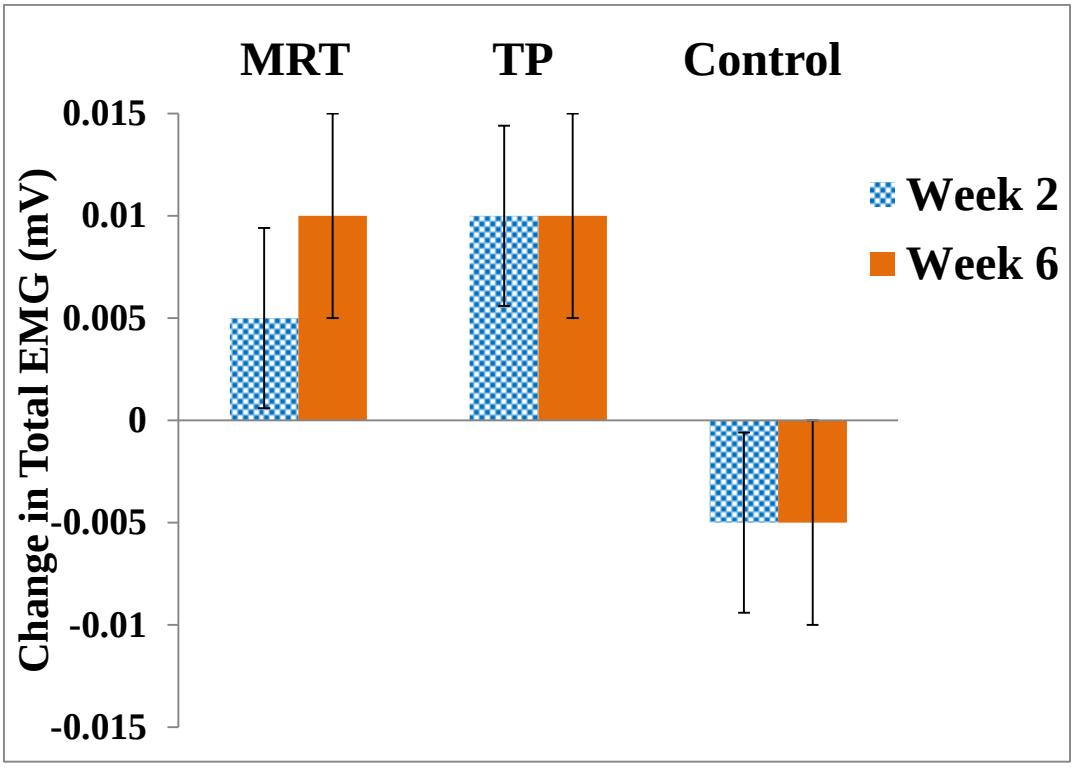


5.4.6 Electromyography (EMG)

RMS values of knee extensor muscle EMGs were the same at 2 weeks and 6 weeks post-baseline as the baseline values for all experimental groups (Figure 5.8).

Figure 5.8. Mean (±1SEM) changes in EMG (mV) from baseline to 2 weeks post-baseline and 6 weeks post-baseline for each treatment.

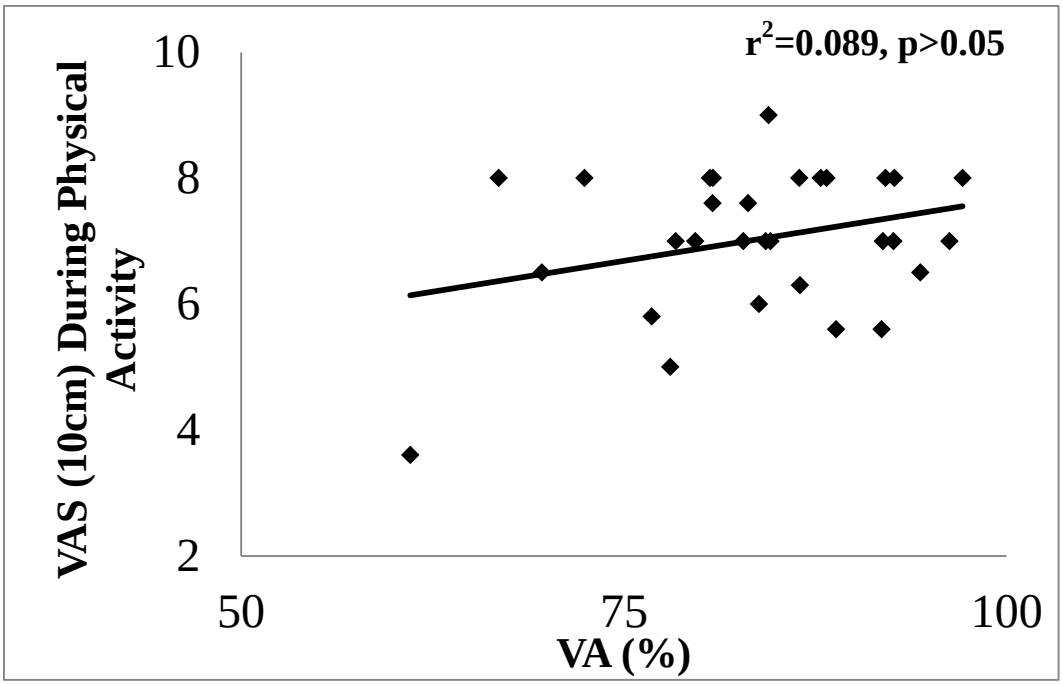
There were no significant changes in EMG from baseline for any of the experimental groups. Myofascial Release Technique; TP = Trigger Point Therapy, Control = Sham Therapeutic Ultrasound.



5.4.7 Correlation between voluntary activation (VA%) and self-reported pain (Visual Analogue Scale - VAS)

Statistical analyses of our results showed a non-significant relationship ($p>0.05$) between VA and pain during physical activity, 1 hour post-physical activity, following 30 min of sitting with knees flexed at baseline, 2 weeks post-baseline, and 6 weeks post-baseline (Figure 5.9).

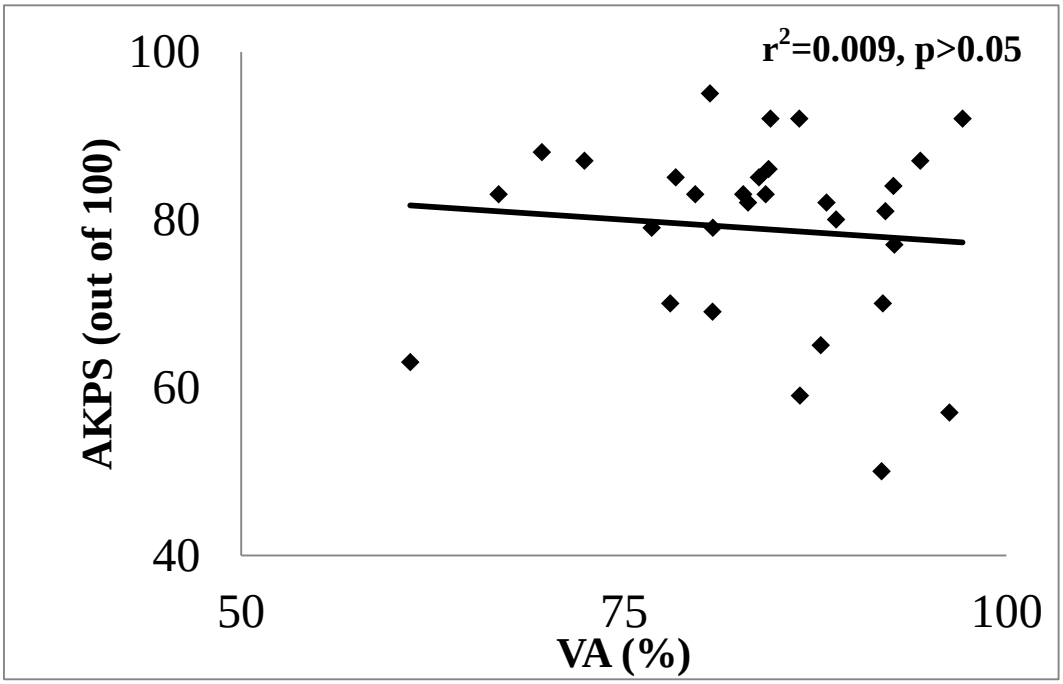
Figure 5.9. Correlation between voluntary activation (VA %) and self-reported pain (Visual Analogue Scale – VAS – 10cm) at baseline during physical activity ($p>0.05$) (N=30).



5.4.8 Correlation between voluntary activation (VA%) and self-reported knee function (Anterior Knee Pain Scale – AKPS)

There was no relationship between VA and anterior knee pain scale scores for knee function at baseline, 2-weeks post-baseline and 6-weeks post-baseline (Figure 5.10).

Figure 5.10. Correlation between voluntary activation (VA %) and self-reported knee function (Anterior Knee Pain Scale – AKPS – out of 100) at baseline (p>0.05) (N=30).



5.4.9 The net change in Anterior Knee Pain Scale (AKPS) compared to baseline for each group

The significant changes in our treatment groups ranged from 9.7 to 12.4 points on the AKPS scale (Table 5.2)

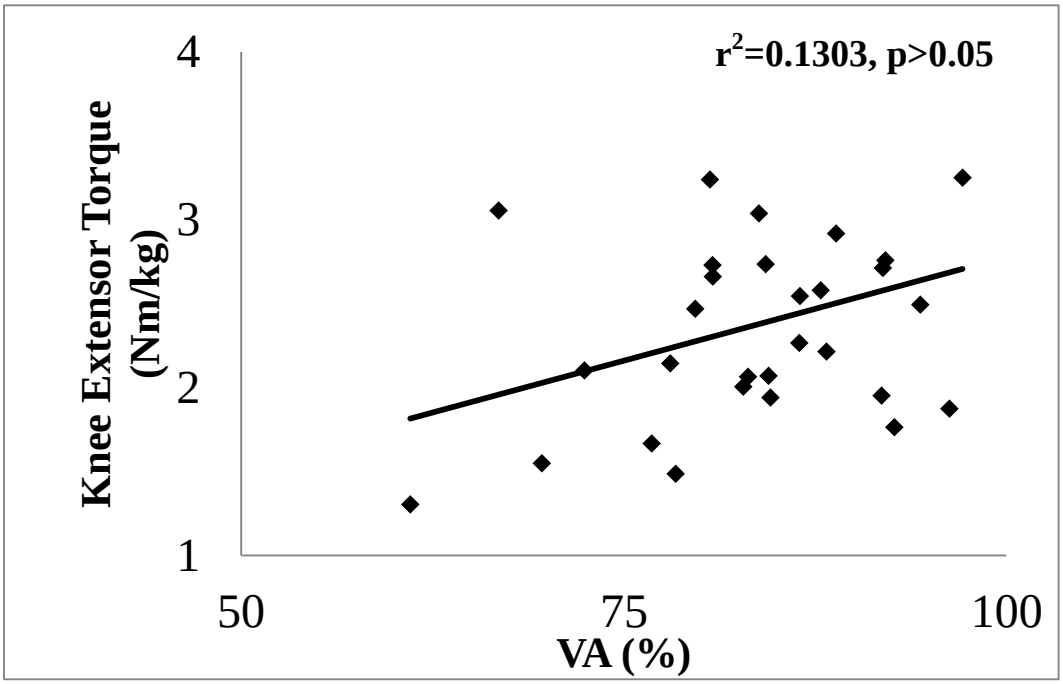
Table 5.2. The net change in the Anterior Knee Pain Scale AKPS (out of 100 points) compared to baseline for each group. Asterisks indicate a significant difference between the baseline AKPS score and 2 weeks post-baseline and/or 6 weeks post-baseline for each treatment group (α<0.05).

Group	2-Weeks Post-Baseline (Net Change Points)	6-Weeks Post-Baseline (Net Change Points)
MRT	12.1 *	12.4 *
TPT	7	9.7 *
Control	1.8	3.8

5.4.10 Correlation between voluntary activation (VA %) and knee extensor torque normalized to body weight (Nm/kg) at baseline

There was a weak but non-significant relationship between VA (%) and knee extensor strength normalized to body weight (Nm/kg) (Figure 5.11).

Figure 5.11. Correlation between voluntary activation (VA %) and knee extensor torque normalized to body weight (Nm/kg) at baseline (p>0.05) (N=30).



5.5 Results Summary

The results are summarized in Table 5.2. In summary, voluntary activation was increased for the TP group patients at 2 and 6 weeks post-baseline, but remained unchanged for the MRT and the Control group patients. Measures of knee pain decreased for patients in all three experimental groups at 2 and 6 weeks post-baseline. Knee function increased in the MRT and TP group patients but not the control group patients. Isometric knee extensor torque remained unchanged

from baseline for patients from all three experimental groups and at both time points of evaluation, except for an increase in torque for the TP group patients at the 6 weeks post-baseline time point.

Table 5.3. Results summary (N=30).

	MRT	TP	Control	p-value
% Voluntary Activation (Baseline)	83 (75-90)	82 (78-86)	87 (81-93)	0.44
% Voluntary Activation (2 weeks post-baseline)	83 (74-91)	91 (88-94)	84 (75-92)	0.15
% Voluntary Activation (6 weeks post-baseline)	89 (83-94)	92 (88-97)	87 (82-92)	0.17
Visual Analogue Pain Scale During Physical Activity (Baseline) (10cm)	7.5 ± 0.84 (6.9-8.1)	7.2 ± 1.3 (6.3-8.1)	7.1 ± 0.7 (6.6-7.5)	0.64
Visual Analogue Pain Scale During Physical Activity (2 weeks post-baseline) (10cm)	1.7 ± 1.5 (0.61-2.8)	2.0 ± 1.7 (0.81-3.3)	1.8 ± 1.5 (0.71-2.8)	0.87
Visual Analogue Pain Scale During Physical Activity (6 weeks post-baseline) (10cm)	0.70 ± 0.54 (0.28-1.1)	1.8 ± 1.3 (0.93-2.7)	1.9 ± 1.8 (0.59-3.2)	0.083
Visual Analogue Pain Scale 1-hr post-physical activity (Baseline) (10cm)	6.6 ± 1.5£ (5.5-7.7)	5.5 ± 0.84 (4.9-6.1)	5.1 ± 0.84* (4.5-5.7)	<0.05
Visual Analogue Pain Scale 1hr post-physical activity (2 weeks post-baseline) (10cm)	1.4 ± 1.0 (0.72-2.0)	1.9 ± 1.5 (0.82-3.0)	1.5 ± 1.2 (0.61-2.4)	0.80
Visual Analogue Pain Scale 1hr post-physical activity (6 weeks post-baseline) (10cm)	0.54 ±0.64 (0.082-1.0)	1.9 ± 1.2 (1.1-2.7)	1.9 ± 1.7 (0.66-3.1)	0.62
Visual Analogue Pain Scale sitting (Baseline) (10cm)	5.1±1.0 (4.4-5.8)	4.9 ± 0.78 (4.3-5.4)	4.9 ± 0.77 (4.3-5.4)	0.80
Visual Analogue Pain Scale sitting (2 weeks post-baseline) (10cm)	1.2 ± 0.53 (0.78-1.5)	1.6 ± 1.2 (0.74-2.5)	2.0 ± 1.4 (0.98-3.0)	0.26
Visual Analogue Pain Scale sitting (6 weeks post-baseline) (10cm)	0.54 ± 0.64 (0.082-1.0)	1.5 ± 1.1 (0.76-2.3)	1.6 ± 1.4 (0.59-2.5)	0.068

	MRT	TP	Control	p-value
Anterior Knee Pain Scale (Baseline) (out of 100)	73.9 ± 11.7 (65.5-82.3)	81.0 ± 10.3 (73.6-88.4)	81.9 ± 10.9 (74.1-89.7)	0.22
Anterior Knee Pain Scale (2 weeks post-baseline) (out of 100)	86 ± 4.4 (76.1-95.9)	88 ± 3.8 (79.4-96.6)	83.7 ± 3.4 (76-91.4)	0.74
Anterior Knee Pain Scale (6 weeks post-baseline) (out of 100)	86.3 ± 3.8 (77.8-94.8)	90.7 ± 1.8 (86.7-94.7)	85.7 ± 2.5 (80.1-91.3)	0.40
Strength (Nm/kg) (Baseline)	2.0 ± 0.5 (1.7-2.4)	2.6 ± 0.4 (2.3-2.9)	2.5 ± 0.7 (2.0-3.0)	0.077
Strength (Nm/kg) (2 weeks post-baseline)	2.1 ± 0.5† (1.7-2.5)	2.7 ± 0.4* (2.5-3.0)	2.2 ± 0.7 (1.6-2.7)	<0.05
Strength (Nm/kg) (6 weeks post-baseline)	2.1 ± 0.6† (1.7-2.5)	2.8 ± 0.4* (2.5-3.1)	2.3 ± 0.7 (1.8-2.8)	<0.05
EMG Vastus Medialis (mV)	0.60 ± 0.14 (0.40-0.70)	0.67 ± 0.20 (0.53-0.81)	0.60 ± 0.43 (0.54-1.2)	0.64
EMG Vastus Lateralis (mV)	0.29 ± 0.07 (0.14-0.44)	0.30 ± 0.10 (0.23-0.37)	0.30 ± 0.19 (0.17-0.44)	0.84
EMG Rectus Femoris **(Baseline) (mV)**	0.24 ± 0.07 (0.19-0.29)	0.34 ± 0.14 (0.24-0.44)	0.32 ± 0.16 (0.20-0.43)	0.21
EMG Vastus Medialis (mV)	0.61 ± 0.11 (0.53-0.69)	0.62 ± 0.15 (0.57-0.78)	0.62 ± 0.34 (0.51-1.0)	0.07
EMG Vastus Lateralis (mV)	0.29 ± 0.07 (0.25-0.35)	0.35 ± 0.08 (0.26-0.38)	0.28 ± 0.16 (0.16-0.39)	0.058
EMG Rectus Femoris **(2 weeks post-baseline) (mV)**	0.24 ± 0.14 (0.24-0.44)	0.35 ± 0.10 (0.28-0.42)	0.31 ± 0.11 (0.23-0.39)	0.10
EMG Vastus Medialis (mV)	0.59 ± 0.25 (0.44-0.70)	0.65 ± 0.16 (0.60-0.83)	0.64 ± 0.34 (0.52-1.0)	0.11
EMG Vastus Lateralis (mV)	0.29 ± 0.06 (0.25-0.34)	0.32 ± 0.07 (0.27-0.37)	0.29 ± 0.18 (0.16-0.42)	0.064
EMG Rectus Femoris **(6 weeks post-baseline) (mV)**	0.26 ± 0.10 (0.27-0.41)	0.35 ± 0.15 (0.25-0.46)	0.28 ± 0.11 (0.23-0.38)	0.11

Data are presented as means ±SD, and 95% confidence intervals. MRT = Myofascial Release Technique; TP = Trigger Point Therapy, Control = Sham Therapeutic Ultrasound.
*Significantly different from the MRT group after post hoc testing ($p<0.05$).
†Significantly different from the TP group after post hoc testing ($p<0.05$).
£Significantly different from the control group after post hoc testing ($p<0.05$).

Chapter Six: **Discussion**

6.1 Study

This randomized controlled trial (RCT) was completed to determine whether manual therapy, specifically myofascial release technique (MRT) and Trigger Point Therapy (TPT) of the quadriceps, directly affects knee-extensor voluntary activation (VA) in patients with patellofemoral pain (PFP). This research contributed to the field by exploring the treatment effects of MRT and TPT specifically on knee-extensor voluntary activation, pain, knee function, knee strength, and quadriceps electromyography.

6.2 Primary outcome: Voluntary activation

The voluntary activation (VA) deficit is thought to prevent full functional recovery of the muscles and the joint after knee injury (14). Hurley et al. (14) showed that patients with a large VA deficit at the onset of a physical rehabilitation program did not show the anticipated improvements during the rehabilitation process. In particular, muscle strength deficits persisted, and there were no significant improvements in VA (14). Therefore reducing or eliminating the VA deficit should be a primary focus of early rehabilitation (14). Suter et al. (47, 126) demonstrated that medical and arthroscopic interventions were successful in reducing pain and subjective symptoms, but complete elimination of the VA deficit was not achieved through these procedures. Therefore, we asked the question whether there are other clinical interventions that would help eliminate the VA deficit in patients with patellofemoral pain syndrome. Drover et al. (22) suggested that clinicians often report strength gains on manual muscle testing immediately following manual therapy treatment. However, these qualitative improvements have not been carefully and systematically investigated.

6.2.1 Myofascial Release Technique (MRT)

The data for the quadriceps voluntary activation in the MRT intervention group was in agreement with the literature, as we did not observe a significant difference in VA 6 weeks post-baseline (Figure 5.2). Non-significant results in VA improvement have been reported by Drover et al. (22). The authors' objective was to determine whether the Active Release Technique (ART®) could be used as an effective way to influence quadriceps voluntary activation and knee extensor strength in athletes with PFP. ART ® is a type of manual therapy that has been patented by Dr. Michael Leahy. The method of MRT, as described in Chapter 4 (section 4.5.1), has some similarities to ART® in terms of how the manual therapy is applied to the tissues. If we use the quadriceps as an example, it is passively taken from a shortened position (knee fully extended) to a fully lengthened position (knee fully flexed), while the contact hand holds tension longitudinally along the muscles fibers (115).

Drover et al. (22) found that a single treatment session using ART® during a one day pre/post trial did not produce statistically significant reductions in the VA deficit or increases in strength in their patients when tested 2 minutes and 20 minutes post-treatment. Drover et al. (22) explained that if the VA deficit was a result of a neuro-inhibitory feedback from the joint receptors within the knee joint capsule, then it was possible that post-treatment, afferent information from the quadriceps muscles is insufficient to overcome this VA deficit. Drover et al. (22) also suggested that direct manipulation of the knee joint complex through knee mobilization may be required to successfully decrease the magnitude of the VA deficit, since they attempted to change the VA by manipulating the soft tissue structures that cross or surround the knee joint, specifically the quadriceps muscles, and the patellar tendon. In the present study,

we also did not mobilize the knee joint and only applied the soft-tissue MRT intervention on the quadriceps muscles.

6.2.2 Trigger point therapy (TPT)

The results of this study suggest that TPT was associated with significant improvements in quadriceps voluntary muscle activation. Improvements of VA of 9% at 2 weeks post-baseline and 10% at 6 weeks post-baseline were observed (Figure 5.2). This improved knee-extensor activation after TPT may be a direct effect of the treatment, as possible confounding factors were controlled as best as possible. For example, a placebo effect was excluded because the sham treated control group did not show an improvement in voluntary activation. Follow-up examinations were performed up to 6 weeks post-baseline, therefore, we do not know how long this possible treatment effect might have persisted beyond our follow-up period. The improved knee-extensor VA after TPT is intriguing and raises questions regarding the connection between PFP and manual therapy, in terms of the mechanisms involved in relieving VA deficits. The mechanisms behind the clinical effectiveness of manual therapy are not established and even though prior studies suggest that manual therapy is an effective treatment for musculoskeletal pain, the mechanisms through which manual therapy exerts its effects have not been systematically established (127). Therefore there is a lack of an identifiable mechanism of action for manual therapy (127). However, one explanation for why TPT may have shown significant differences in the VA is pain reduction, as the VA has been linked to pain (13). More specifically, it is possible that the TPT was able to manage any suspected trigger points on the quadriceps associated with the knee pain. Recall that in Chapter 2 a trigger point is identified as a localized spot of tenderness in a nodule in a palpable taut band of muscle fibers (82). The spot is

tender when pressed and can give rise to characteristic referred pain, motor dysfunction, and autonomic phenomena (82). Therefore any associated trigger and/or tender points in the quadriceps femoris have been purported to be a factor in the etiology, and aggravation, of PFP (117). Additionally, Hains and Hains (118) suggested that PFP may be managed conservatively by myofascial techniques such TPT to the knee. Furthermore, it has been suggested that myofascial trigger point pain is associated with PFP, resulting in reductions in voluntary muscle activation, decreased muscle strength, and reduced muscle flexibility (119, 120). However, the proposed etiology of a trigger point is still in question and requires further systematic investigation.

It should be noted that we tried to control for methodological limitations of the superimposed twitch technique in determining muscle activation. For example, we use a doublet twitch for improved sensitivity and repeatability of the method, verified that all electrical stimulation was supra-maximal (i.e., all motor units were stimulated), and normalized the superimposed twitch to a potentiated control twitch (128), thereby accounting for the fact that the superimposed twitch itself occurs when the muscle is potentiated.

6.3 Secondary outcomes

6.3.1 Visual analogue scale (VAS) subjective knee pain scores

Significant decreases from baseline were observed for Visual Analogue Scale (VAS) subjective knee pain scores for all three groups (MRT, TPT and control) 2 weeks and 6 weeks post-baseline. These results were found for all three pain situations: (a) during physical activity, (b) 1 hour after physical activity, and (c) following 30 minutes of sitting with knees flexed (Figures 5.3, 5.4, and 5.5). Since the control group patients also had a significant reduction in pain, this result may be a placebo effect. Placebo effects have the potential to augment the efficacy of active medical treatments and procedures (129). The placebo effect may account for some of the treatment effects observed in this study, as we did not expect an improvement in pain for the sham control group patients who had chronic patella-femoral pain for an average of 1.6 years (±1.4SD) prior to this study. The placebo effect may be caused by a psychologically and physiologically active process associated with a robust hypoalgesic response (130). The interaction between patient, clinician, and clinical environment may be a factor in the placebo effect. However, since all patients were seen for the same amount of time, on average, and in the same clinical setting, any placebo effects would be expected to be similar across the three patient groups (131).

Nociceptive activity resulting from joint pain has been suggested to increase the VA deficit (132). However, evidence for an association between pain and VA deficit is ambiguous (47). Hurley et al. (14) and Rutherford et al. (38) support the idea that pain may cause a VA deficit. This association was further supported in a study in which the VA deficit was significantly reduced when a local anesthetic was injected into the knee shortly after meniscectomy (133).

However, the same injection administered 2 weeks after the surgery did not appear to reduce the VA deficit despite a decrease in pain. Newham et al. (37) also observed a VA deficit in pathological knees that were free of effusion and pain. Therefore these findings suggest that VA deficits occur, at least partly, from stimuli other than pain.

Statistical analyses of our results showed a non-significant relationship ($p>0.05$) between VA and pain during physical activity, 1 hour post-physical activity, following 30 min of sitting with knees flexed at baseline, 2 weeks post-baseline, and 6 weeks post-baseline (Figure 5.9). This observation supports the conclusion that pain might not be directly related to the amount of the VA deficit, but that other factors are likely to contribute. Suter et al. (47) anticipated that the administration of anti-inflammatory drugs, designed to reduce pain and swelling in the knee, would have a beneficial effect on VA deficits and knee extensor moments. However, this expectation was not met since non-steroidal anti-inflammatory drug (NSAID) intake in a group of patients with unilateral anterior knee pain did not result in an increase in VA or strength of the quadriceps muscles, despite a significant reduction in pain (47). Suter et al. (47) suggested that pain reduction may eventually lead to an improvement in VA, but that the two events are offset in time. The findings also indicate that pain is not the only factor responsible for the origin of the VA deficit, but that other stimuli contribute to the observed VA deficit.

6.3.2 Anterior knee pain scale (AKPS)

There was no relationship between VA and anterior knee pain scale scores for knee function at baseline, 2-weeks post-baseline and 6-weeks post-baseline (Figure 5.10). This was an interesting finding, since there was a significant improvement in knee function for the MRT (2 weeks and 6 weeks post-baseline) and TPT group patients (6 weeks post-baseline) (Figure 5.6). Control group

patients did not show an improvement in AKPS scores. It might be expected that the decrease in visual analog pain scale score observed in control group patients would be associated with a corresponding decrease in AKPS. However, it has been suggested that the AKPS scores do not correlate highly with the visual analogue scale for self-reported pain measures (110). The sensitivity of the AKPS questionnaire has been investigated in a number of studies (110, 134, 135). It has been argued that a 7-point change in the APKS score can be detected reliably Crossley et al. (110), while Bennell et al. (134) and Watson et al. (135) argue for detection of a 10- point change. The significant changes in our treatment groups ranged from 9.7 to 12.4 points on the AKPS scale (Table 5.2), thereby supporting evidence of detectable differences published by others.

The Anterior Knee Pain Scale is a 13-item knee-specific self-report questionnaire, which documents the response to six activities thought to be associated specifically with patellofemoral pain (walking, running, jumping, climbing stairs, squatting, and sitting for prolonged periods with knees bent), as well as symptoms such as limping, inability to weight bear through the affected limb, swelling, abnormal patellar movement, muscle atrophy and limitation of knee flexion (108). Therefore this scale may be better than other measures in detecting PFP-specific improvements, as it relates to detecting change in activities that generally afflict PFP patients, and may be a more realistic measure for tracking progress in PFP than just pain alone.

6.3.3 Knee extensor torque (Knee Strength)

There was a significant increase in knee extensor strength for TPT group patients at 6 weeks post-baseline. This increase in strength was associated with a 10% improvement in quadriceps VA at 6 weeks post-baseline. Since control group patients did not improve in either VA or knee

extensor strength, one can assume that this result was an actual treatment effect. MRT group patients did not increase knee extensor strength with treatment despite a 6% increase in knee extensor VA. Perhaps a minimal amount of improvement in VA is required in order to see significant increases in strength. Our results are in agreement with earlier work on MRT and knee extensor strength by Drover et al. (22), who also did not find an increase in knee extensor strength or voluntary activation following a single MRT treatment, with immediate testing. There was a weak but non-significant relationship between VA (%) and knee extensor strength normalized to body weight (Nm/kg) (Figure 5.11). A greater range of voluntary activation deficits, as found in patients following knee injury or surgical intervention, would likely produce a strong relationship between VA and muscle strength.

6.3.4 Electromyography (EMG)

There were no significant differences in EMG RMS amplitudes of peak isometric force among patients of the three intervention groups. Similarly, Suter et al. (5) did not find significant differences in EMG amplitudes of knee pain patients who received either manual therapy interventions or a sham intervention, despite significantly higher VA of the quadriceps muscles in their manual treatment intervention group. This result demonstrates that activation differences might be detected using the superimposed twitch technique, even when they are not apparent in plain EMG measurements, suggesting that EMG measurements are not a very sensitive measure of muscle activation. Control group patients had a significant decrease in isometric knee extensor torques at 2 and 6 weeks post-baseline, which was accompanied by significant decreases in the corresponding EMG RMS values. We are not certain why the sham treatment in this patient group should have decreased their knee extensor strength and EMG amplitudes.

Surface electromyography has been routinely used to study muscle activation of the quadriceps (e.g., (136)), and muscle imbalances in PFP patients (e.g., (137)). Such studies have produced contradictory results leading to equivocal conclusions due to differences in methodology, materials and large inter-subject variation (138).

It is also accepted that the amplitude of the EMG signal is related, to a certain extent, to the force a muscle generates but is not related to this force in an obvious and consistent manner (139). It is intuitive to think that an increase in force results in an increase in EMG amplitude. However, one problem with maximal voluntary contractions is that they depend not only on the subject's motivation but also on the ability to maximally recruit the muscles (94). As a result, MVCs are usually not an accurate reflection of the maximal torque-generating capacity of the muscle as there are also day to day differences (94). However, one might expect that such differences in maximal activation and force production are reflected in force and EMG similarly, except that EMG signals tend to become saturated at high muscle activation levels and thus are not ideal to distinguish forces at maximal voluntary effort contractions (140).

There are also day to day variations in EMG recordings, which may be associated with differences in electrode application such as minor changes in the position of the recording electrodes over the muscle and differences in skin preparation (141). Komi and Buskirk (142) showed inter-day reliability coefficients to average between 0.60 and 0.81, when studying EMG amplitudes for force levels ranging from 20 to 100% of maximum. As well, intervening factors may influence the MVC and result in a change in the EMG, such as length and timing of sleep, self-reported stress levels and nutritional status (143, 144). According to Youngstedt and O'Connor (144), differences in timing of meal intake prior to tests may be one of the confounding factors affecting diurnal variation in neuromuscular performance. Therefore,

differences in sleep, stress and nutritional status cannot be fully excluded as potential confounding factors in the present study.

In order to control for the variability in MVC, participants were encouraged verbally and through visual biofeedback. Also, subjects were given five minutes of rest between MVC trials. Furthermore, participants were asked to refrain from physical activity on the day of testing.

In order to control variability in EMG data, a transparency outlining the exact location of the electrode placement was drawn and used each time a participant was tested. This procedure ensured a near identical electrode placement for repeat testing sessions. Prior to electrode placement, the skin was carefully prepared by shaving the target area and by using alcohol to clean the skin of dry surface layers and dead cells, thereby reducing the skin impedance, thus enhancing the signal to noise ratio. Skin impedance for all measurements used here was less than 10 kΩ. Variations in day to day recording sensitivity were accounted for using a series of precisely defined sub-maximal contractions and measurement of the corresponding EMG signals. For example, if a 100Nm knee extensor contraction resulted in an RMS of the EMG signal that was 20% greater than the baseline measurement, this was accounted for when comparing RMS values for the maximal voluntary contractions. It was challenging to control for other factors which might affect EMG magnitude from day to day, for example the participants' time of testing, sleep patterns, emotional stress, and nutritional status. All these factors combine to produce variability in day to day EMG measurements, and this inherent variability may have been the reason for a lack of significant differences in EMG data across the testing period.

Contrary to our hypothesis, the MRT intervention showed no significant difference in the voluntary activation, and the control group also experienced a decrease in knee pain scores (VAS). Therefore, this study shows that MRT does not significantly increase the VA. However

since all three interventions yielded favorable results for pain, this may support the power of the placebo effect.

Chapter Seven: **Limitations, future directions and conclusion**

7.1 Limitations

This study was a randomized controlled trial (RCT), therefore randomization and strict inclusion and exclusion criteria were used to control, as well as possible, for potential confounders. Randomization appeared to be successful in establishing equivalent groups based on multiple characteristics, however the participants comprised a convenience sample. Volunteer participation may have brought particularly motivated participants to the study. Participants knew that they were in a scientific experiment, thus they may have had an expectation of getting better, which might have produced a Hawthorne effect. This introduces the possibility of a study bias, and therefore the results of this study may not be generalizable to a similar group of patients undergoing regular treatment but not being part of a scientific study.

For randomization, we did not account for the cause or mechanism of PFP. As the source of PFP is believed to be multifactorial (2), this randomization approach neglects the possibility of a more specific treatment for the underlying cause of the PFP. For example, if foot pronation was causing the PFP (2), then manual therapy to the quadriceps may not show favorable outcomes since it would not address the problem causing the PFP. However, there is no consensus on causative relationships between chronic idiopathic PFP and the different mechanisms that are thought to cause PFP (2). Additionally, the objective of this study was not to look at the mechanisms or cure for PFP, but to determine whether manual therapy, specifically myofascial release technique (MRT) and Trigger Point Therapy (TPT) of the quadriceps, directly affects knee-extensor voluntary activation (VA) in patients with patellofemoral pain (PFP). If the participants were stratified based on the mechanism of PFP, then perhaps these subgroups would be more

clinically homogenous in nature, and some patients, such as those with an underlying quadriceps muscle imbalance or neuromuscular deficiencies, might react better to the MRT and TPT treatment approaches than other patients with different causes for their PFP.

In the ideal case, physical activity levels of the participants would be controlled and kept constant, to exclude physical activity level as a confounding factor. Participants were included if they had been physically active for at least 30 min/day, 3-4 days/week for the past 6 months. The majority of the participants were recreational runners who ran for at least 30 minutes, 3-4 times per week. Being sedentary may result in muscle disuse and atrophy, and cause loss of maximal voluntary strength (145). In the extreme case, a 4-week period of lower limb suspension was shown to reduce isometric forces in knee extensors and plantar flexors by 12% and 22%, respectively (146). On the other hand, participants who thought they received a medical intervention might have increased their volume of training, thereby potentially aggravating symptoms. Therefore, fluctuations in participant physical activity levels were a challenge and a possible confounding factor. We made sure participants communicated their physical and emotional states with us, and if subjects were physically tired or emotionally not ready for testing, they were rescheduled. There were only two cases where participants were unable to perform the testing when asked how they were feeling when they arrived, and then were rescheduled. One was due to fatigue from studying for exams and the other was due to personal matters.

While participants were relatively similar in age and body size (Table 5.1), the MRT and control group had a slightly higher proportion of women than the TPT group. We attempted to control for this discrepancy by normalizing strength to body weight. However it is unknown if the magnitude of the VA deficits reported in each intervention group would be the same if either

men or women were assessed in isolation. We did not possess the necessary study power to analyze subgroups based on sex.

Since there were multiple clinicians (2 physiotherapists and 1 Chiropractor, minimum experience 2 years) providing the treatments, this resulted in inconsistencies in the treatment applications. In order to control for these inconsistencies, all clinicians gathered, decided on an exact procedure to perform each treatment intervention, such as the duration of each therapy applied to the muscle. Additionally, clinicians practiced the treatment interventions on each other until a consensus was made to their satisfaction that each clinician performed the treatments similar to the others.

Cheng et al. (86) suggested that the interpolated twitch technique is a practical method to determine impairments in voluntary drive during and following fatiguing efforts. The interpolated twitch technique has been validated and is extensively used in neuromuscular research. Its accuracy as a measure of neural activation has been questioned. Horstman (94) suggested that a problem with MVCs is that they depend not only on a subject's motivation but also on the ability to maximally recruit their muscles. As a result, MVCs are usually not an accurate reflection of the maximal torque-generating capacity of a muscle or a muscle group (94). According to Herzog (96), defining the maximal voluntary force of a muscle is impossible, as there is no defined standard of when an MVC has been reached. This is further complicated by the fact that maximal voluntary effort contractions give forces that are always much lower than a muscle is inherently capable of, when stimulated artificially through nerve activation. For some reasons, protective mechanisms presumably, the maximal frequency of voluntary motor unit activation is in the range of 60Hz, whereas vast increases in force can be achieved in slow muscles with frequencies up to 120Hz and in fast muscles with frequencies up to 200Hz.

However, these maximal frequencies that would give maximal muscle force cannot be achieved voluntarily (147).

Another problem is that twitch interpolation values are highly variable even under the same conditions and identical force production (96). For example, Suter et al. (12) conducted a study involving 20 healthy subjects performing 20 knee extensor contractions varying from 5 to 100% of the maximal voluntary force. Twitch interpolation values ranged from 0 to 100% for contraction levels of 60% of maximal voluntary force. So despite a constant voluntary force, identical knee joint angles, the same equipment and the same experimenter, the interpolated twitch values varied greatly (12). Suter et al. (12) used a single superimposed twitch, but showed in a later study that using a doublet, triplet and quadruplet superimposed twitch reduced this variability significantly, with a doublet twitch probably being the optimal solution for minimizing pain and reducing variability (98). Therefore, a doublet twitch interpolation protocol was used in this study.

Herzog (96) also suggested that the twitch interpolation is neither an accurate nor a precise measure of the amount of activation, but it does reflect the amount of muscle activation in a qualitative way. Conversely, Horstman (94) suggested that the interpolated twitch can be a useful tool in patient research, as it is a relatively reliable method for specific conditions (95), and is a good way to estimate strength deficits in people who have difficulties with voluntary torque generation. Despite the controversy surrounding the superimposed twitch technique, there is not an obvious alternative method that offers itself for accurate and reliable measurement of *in vivo* muscle activation.

7.2 Recommendations for Future Research

An interesting topic for future study would be to determine whether a subgroup classification based on the causes of PFP would result in better treatment success, if treatment options addressed the specific cause of the PFP. Flynn et al. (148) and Cleland et al. (149) have also suggested that we would gain a better understanding if we were to predict the manual therapy outcomes after looking at clinical prediction rules based on signs and symptoms, in order to identify responders to the manual therapy. For example, Witvrouw et al. (59) suggested that an intrinsic risk factor for PFP is decreased quadriceps flexibility, confirmed by measurement in a prone position with a goniometer. It would be interesting to see how this group would react if treated exclusively with TPT, compared to the combination of TPT and dynamic exercises, or to exercise interventions alone.

7.3 Conclusion

The purpose of this study was to determine whether manual therapy, specifically myofascial release technique (MRT) and Trigger Point Therapy (TPT) of the quadriceps, directly affects knee-extensor voluntary activation (VA) in patients with patellofemoral pain (PFP). Thirty patients with PFP were recruited and randomized into group 1 who received myofascial release technique treatments, group 2 who received trigger point therapy, and group 3 who received a sham ultrasound treatment (control group). We found that the % voluntary activation in the quadriceps improved for the trigger point therapy intervention at 2 weeks and 6 weeks post-baseline by 9% and 10%, respectively. We did not find any significant changes in quadriceps voluntary activation with the myofascial release technique. Interestingly, the self-reported visual analogue knee pain scores improved for patients in all three groups (MRT, TPT and control).

This latter finding may partly be a placebo effect. The reduction in pain corresponded to an improvement in knee function as measured with the Anterior Knee Pain Scale, for the MRT group at 2 and 6 weeks post-baseline and for the TPT group at 6 weeks post-baseline. The increase in voluntary activation for TPT group patients corresponded to a significant improvement in knee extensor strength at 6 weeks post-baseline. Surface EMG signals remained unchanged for all muscles and all three groups for all assessment times. Although voluntary activation improved for the TPT group patients, there is a widespread, but unsubstantiated belief among clinicians that MRT increases strength immediately following treatment. Our study does not support that belief.

References

1. Davis IS, Powers CM. Patellofemoral pain syndrome: Proximal, distal, and local factors, an international retreat, april 30-may 2, 2009, fells point, baltimore, MD. J Orthop Sports Phys Ther. 2010 Mar;40(3):A1-16.
2. Powers CM, Bolgla LA, Callaghan MJ, Collins N, Sheehan FT. Patellofemoral pain: Proximal, distal, and local factors, 2nd international research retreat. J Orthop Sports Phys Ther. 2012 Jun;42(6):A1-54.
3. Taunton JE, Ryan MB, Clement DB, McKenzie DC, Lloyd-Smith DR, Zumbo BD. A retrospective case-control analysis of 2002 running injuries. Br J Sports Med. 2002 Apr;36(2):95-101.
4. DEANDRADE JR, GRANT C, DIXON AS. Joint distension and reflex muscle inhibition in the knee. J Bone Joint Surg Am. 1965 Mar;47:313-22.
5. Suter E, McMorland G, Herzog W, Bray R. Conservative lower back treatment reduces inhibition in knee-extensor muscles: A randomized controlled trial. J Manipulative Physiol Ther. 2000 Feb;23(2):76-80.
6. Konishi Y, Fukubayashi T, Takeshita D. Possible mechanism of quadriceps femoris weakness in patients with ruptured anterior cruciate ligament. Med Sci Sports Exerc. 2002 Sep;34(9):1414-8.
7. Young A. Current issues in arthrogenous inhibition. Ann Rheum Dis. 1993 Nov;52(11):829-34.
8. Beryl Harding A. AN INVESTIGATION INTO THE CAUSE OF ARTHRITIC MUSCULAR ATROPHY. The Lancet. 1929;213(5505):433-4.
9. Hopkins J, Ingersoll C. Arthrogenic muscle inhibition: A limiting factor in joint rehabilitation. J Sport Rehab. 2000;9(2):135-59.
10. Iles JF, Stokes M, Young A. Reflex actions of knee joint afferents during contraction of the human quadriceps. Clin Physiol. 1990 Sep;10(5):489-500.
11. Spencer JD, Hayes KC, Alexander IJ. Knee joint effusion and quadriceps reflex inhibition in man. Arch Phys Med Rehabil. 1984 Apr;65(4):171-7.
12. Suter E, Herzog W. Extent of muscle inhibition as a function of knee angle. J Electromyogr Kinesiol. 1997 Jun;7(2):123-30.
13. Rice DA, McNair PJ. Quadriceps arthrogenic muscle inhibition: Neural mechanisms and treatment perspectives. Semin Arthritis Rheum. 2010 Dec;40(3):250-66.
14. Hurley MV, Jones DW, Newham DJ. Arthrogenic quadriceps inhibition and rehabilitation of patients with extensive traumatic knee injuries. Clin Sci (Lond). 1994 Mar;86(3):305-10.
15. Suter E, McMorland G, Herzog W, Bray R. Decrease in quadriceps inhibition after sacroiliac joint manipulation in patients with anterior knee pain. J Manipulative Physiol Ther. 1999 Mar-Apr;22(3):149-53.
16. McConnell J. Management of patellofemoral problems. Man Ther. 1996;1(2):60-6.
17. Crossley K, Bennell K, Green S, Cowan S, McConnell J. Physical therapy for patellofemoral pain: A randomized, double-blinded, placebo-controlled trial. Am J Sports Med. 2002 Nov-Dec;30(6):857-65.
18. Farrell JP, Jensen GM. Manual therapy: A critical assessment of role in the profession of physical therapy. Phys Ther. 1992 Dec;72(12):843-52.

19. Cyriax J. Textbook of orthopaedic medicine, volume I: Diagnosis of soft-tissue lesions. 8th ed. London, England: A Bailliere Tindall; 1982.
20. French HP, Brennan A, White B, Cusack T. Manual therapy for osteoarthritis of the hip or knee - a systematic review. Man Ther. 2011 Apr;16(2):109-17.
21. Perlman AI, Sabina A, Williams AL, Njike VY, Katz DL. Massage therapy for osteoarthritis of the knee: A randomized controlled trial. Arch Intern Med. 2006 Dec 11-25;166(22):2533-8.
22. Drover JM, Forand DR, Herzog W. Influence of active release technique on quadriceps inhibition and strength: A pilot study. J Manipulative Physiol Ther. 2004 Jul-Aug;27(6):408-13.
23. Wood L, Muller S, Peat G. The epidemiology of patellofemoral disorders in adulthood: A review of routine general practice morbidity recording. Prim Health Care Res Dev. 2011 Apr;12(2):157-64.
24. Boling MC, Padua DA, Marshall SW, Guskiewicz K, Pyne S, Beutler A. A prospective investigation of biomechanical risk factors for patellofemoral pain syndrome: The joint undertaking to monitor and prevent ACL injury (JUMP-ACL) cohort. Am J Sports Med. 2009 Nov;37(11):2108-16.
25. Boling M, Padua D, Marshall S, Guskiewicz K, Pyne S, Beutler A. Gender differences in the incidence and prevalence of patellofemoral pain syndrome. Scand J Med Sci Sports. 2010 Oct;20(5):725-30.
26. Stathopulu E, Baildam E. Anterior knee pain: A long-term follow-up. Rheumatology (Oxford). 2003 Feb;42(2):380-2.
27. Tan SS, van Linschoten RL, van Middelkoop M, Koes BW, Bierma-Zeinstra SM, Koopmanschap MA. Cost-utility of exercise therapy in adolescents and young adults suffering from the patellofemoral pain syndrome. Scand J Med Sci Sports. 2010 Aug;20(4):568-79.
28. Thomee R, Augustsson J, Karlsson J. Patellofemoral pain syndrome: A review of current issues. Sports Med. 1999 Oct;28(4):245-62.
29. Natri A, Kannus P, Jarvinen M. Which factors predict the long-term outcome in chronic patellofemoral pain syndrome? A 7-yr prospective follow-up study. Med Sci Sports Exerc. 1998 Nov;30(11):1572-7.
30. Kannus P, Aho H, Jarvinen M, Niittymaki S. Computerized recording of visits to an outpatient sports clinic. Am J Sports Med. 1987 Jan-Feb;15(1):79-85.
31. Blond L, Hansen L. Patellofemoral pain syndrome in athletes: A 5.7-year retrospective follow-up study of 250 athletes. Acta Orthop Belg. 1998 Dec;64(4):393-400.
32. Fairbank JC, Pynsent PB, van Poortvliet JA, Phillips H. Mechanical factors in the incidence of knee pain in adolescents and young adults. J Bone Joint Surg Br. 1984 Nov;66(5):685-93.
33. Morrissey MC. Reflex inhibition of thigh muscles in knee injury. causes and treatment. Sports Med. 1989 Apr;7(4):263-76.
34. Stokes M, Young A. The contribution of reflex inhibition to arthrogenous muscle weakness. Clin Sci (Lond). 1984 Jul;67(1):7-14.
35. Herzog W, Suter E. Muscle inhibition following knee injury and disease. Sportverletz Sportschaden. 1997 Sep;11(3):74-8.
36. Hurley MV, Newham DJ. The influence of arthrogenous muscle inhibition on quadriceps rehabilitation of patients with early, unilateral osteoarthritic knees. Br J Rheumatol. 1993 Feb;32(2):127-31.
37. Newham D, Hurley M, Jones D. Ligamentous knee injuries and muscle inhibition. Journal of Orthopedic Rheumatism. 1989;2:163-73.

38. Rutherford OM, Jones DA, Newham DJ. Clinical and experimental application of the percutaneous twitch superimposition technique for the study of human muscle activation. J Neurol Neurosurg Psychiatry. 1986 Nov;49(11):1288-91.
39. Snyder-Mackler L, De Luca PF, Williams PR, Eastlack ME, Bartolozzi AR,3rd. Reflex inhibition of the quadriceps femoris muscle after injury or reconstruction of the anterior cruciate ligament. J Bone Joint Surg Am. 1994 Apr;76(4):555-60.
40. Hurley M, Jones D, Wilson D, Newham D. Rehabilitation of quadriceps inhibited due to isolated rupture of the anterior cruciate ligament. Orthopaedic Rheumatolog. 1992;5:145-54.
41. Fahrer H, Rentsch HU, Gerber NJ, Beyeler C, Hess CW, Grunig B. Knee effusion and reflex inhibition of the quadriceps. A bar to effective retraining. J Bone Joint Surg Br. 1988 Aug;70(4):635-8.
42. Krebs DE, Staples WH, Cuttita D, Zickel RE. Knee joint angle: Its relationship to quadriceps femoris activity in normal and postarthrotomy limbs. Arch Phys Med Rehabil. 1983 Oct;64(10):441-7.
43. McNair PJ, Marshall RN, Maguire K. Knee effusion and quadriceps muscle strength. Clin Biomech (Bristol, Avon). 1994 Nov;9(6):331-4.
44. Stratford P. Electromyography of the quadriceps femoris muscles in subjects with normal knees and acutely effused knees. Phys Ther. 1982 Mar;62(3):279-83.
45. EKHOLM J, EKLUND G, SKOGLUND S. On the reflex effects from the knee joint of the cat. Acta Physiol Scand. 1960 Oct 31;50:167-74.
46. Hirokawa S, Solomonow M, Lu Y, Lou ZP, D'Ambrosia R. Anterior-posterior and rotational displacement of the tibia elicited by quadriceps contraction. Am J Sports Med. 1992 May-Jun;20(3):299-306.
47. Surer E, Herzog W, De Souza K, Bray R. Inhibition of the quadriceps muscles in patients with anterior knee pain. Journal of Applied Biomechanics. 1998;14:360-73.
48. Thomee R, Grimby G, Svantesson U, Osterberg U. Quadriceps muscle performance in sitting and standing in young women with patellofemoral pain syndrome and young healthy women. Scand J Med Sci Sports. 1996 Aug;6(4):233-41.
49. Hurley MV. The effects of joint damage on muscle function, proprioception and rehabilitation. Man Ther. 1997 Feb;2(1):11-7.
50. Grigg P, Schaible HG, Schmidt RF. Mechanical sensitivity of group III and IV afferents from posterior articular nerve in normal and inflamed cat knee. J Neurophysiol. 1986 Apr;55(4):635-43.
51. Krishnan C, Williams GN. Quantification method affects estimates of voluntary quadriceps activation. Muscle Nerve. 2010 Jun;41(6):868-74.
52. Callaghan MJ, Oldham JA. Quadriceps atrophy: To what extent does it exist in patellofemoral pain syndrome? Br J Sports Med. 2004 Jun;38(3):295-9.
53. Powers CM. Rehabilitation of patellofemoral joint disorders: A critical review. J Orthop Sports Phys Ther. 1998 Nov;28(5):345-54.
54. Grayson T, editor. Disorders of the patellofemoral joint. 2nd ed. Baltimore (MD): Williams and Wilkins; 1990.
55. Levine J. Chondromalacia patellae. Phys Sportsmed. 1979;7:41-9.
56. Lun VM, Wiley JP, Meeuwisse WH, Yanagawa TL. Effectiveness of patellar bracing for treatment of patellofemoral pain syndrome. Clin J Sport Med. 2005 Jul;15(4):235-40.

57. Fagan V, Delahunt E. Patellofemoral pain syndrome: A review on the associated neuromuscular deficits and current treatment options. Br J Sports Med. 2008 Oct;42(10):789-95.
58. Lankhorst NE, Bierma-Zeinstra SM, van Middelkoop M. Risk factors for patellofemoral pain syndrome: A systematic review. J Orthop Sports Phys Ther. 2012 Feb;42(2):81-94.
59. Witvrouw E, Lysens R, Bellemans J, Cambier D, Vanderstraeten G. Intrinsic risk factors for the development of anterior knee pain in an athletic population. A two-year prospective study. Am J Sports Med. 2000 Jul-Aug;28(4):480-9.
60. Nimon G, Murray D, Sandow M, Goodfellow J. Natural history of anterior knee pain: A 14- to 20-year follow-up of nonoperative management. J Pediatr Orthop. 1998 Jan-Feb;18(1):118-22.
61. Ficat R, Hungerford D. Disorders of the patello-femoral joint. Baltimore (MD): Williams & Wilkins; 1977.
62. Hughston J, Walsh W, Puddu G. Patellar subluxation and dislocation. Philadelphia (PA): WB Saunders; 1984.
63. Hungerford DS, Barry M. Biomechanics of the patellofemoral joint. Clin Orthop Relat Res. 1979 Oct;(144)(144):9-15.
64. Crossley K, Bennell K, Green S, McConnell J. A systematic review of physical interventions for patellofemoral pain syndrome. Clin J Sport Med. 2001 Apr;11(2):103-10.
65. Lieb FJ, Perry J. Quadriceps function. an anatomical and mechanical study using amputated limbs. J Bone Joint Surg Am. 1968 Dec;50(8):1535-48.
66. Reid D. Sports injury assessment and rehabilitation. New York: Churchill Livingstone; 1992.
67. Keays SL, Bullock-Saxton J, Keays AC. Strength and function before and after anterior cruciate ligament reconstruction. Clin Orthop Relat Res. 2000 Apr;(373)(373):174-83.
68. Keays SL, Bullock-Saxton JE, Newcombe P, Keays AC. The relationship between knee strength and functional stability before and after anterior cruciate ligament reconstruction. J Orthop Res. 2003 Mar;21(2):231-7.
69. Rossi MD, Brown LE, Whitehurst M. Early strength response of the knee extensors during eight weeks of resistive training after unilateral total knee arthroplasty. J Strength Cond Res. 2005 Nov;19(4):944-9.
70. Rossi MD, Brown LE, Whitehurst M, Charni C, Hankins J, Taylor CL. Comparison of knee extensor strength between limbs in individuals with bilateral total knee replacement. Arch Phys Med Rehabil. 2002 Apr;83(4):523-6.
71. Stevens JE, Mizner RL, Snyder-Mackler L. Quadriceps strength and volitional activation before and after total knee arthroplasty for osteoarthritis. J Orthop Res. 2003 Sep;21(5):775-9.
72. Bron C, de Gast A, Dommerholt J, Stegenga B, Wensing M, Oostendorp RA. Treatment of myofascial trigger points in patients with chronic shoulder pain: A randomized, controlled trial. BMC Med. 2011 Jan 24;9:8,7015-9-8.
73. Deyle GD, Allison SC, Matekel RL, Ryder MG, Stang JM, Gohdes DD, et al. Physical therapy treatment effectiveness for osteoarthritis of the knee: A randomized comparison of supervised clinical exercise and manual therapy procedures versus a home exercise program. Phys Ther. 2005 Dec;85(12):1301-17.
74. Deyle GD, Henderson NE, Matekel RL, Ryder MG, Garber MB, Allison SC. Effectiveness of manual physical therapy and exercise in osteoarthritis of the knee. A randomized, controlled trial. Ann Intern Med. 2000 Feb 1;132(3):173-81.

75. Walker MJ, Boyles RE, Young BA, Strunce JB, Garber MB, Whitman JM, et al. The effectiveness of manual physical therapy and exercise for mechanical neck pain: A randomized clinical trial. Spine (Phila Pa 1976). 2008 Oct 15;33(22):2371-8.
76. Eisenhart AW, Gaeta TJ, Yens DP. Osteopathic manipulative treatment in the emergency department for patients with acute ankle injuries. J Am Osteopath Assoc. 2003 Sep;103(9):417-21.
77. Cubick EE, Quezada VY, Schumer AD, Davis CM. Sustained release myofascial release as treatment for a patient with complications of rheumatoid arthritis and collagenous colitis: A case report. Int J Ther Massage Bodywork. 2011;4(3):1-9.
78. LeBauer A, Brtalik R, Stowe K. The effect of myofascial release (MFR) on an adult with idiopathic scoliosis. J Bodyw Mov Ther. 2008 Oct;12(4):356-63.
79. Desmeules F, Cote CH, Fremont P. Therapeutic exercise and orthopedic manual therapy for impingement syndrome: A systematic review. Clin J Sport Med. 2003 May;13(3):176-82.
80. Licciardone JC, Stoll ST, Fulda KG, Russo DP, Siu J, Winn W, et al. Osteopathic manipulative treatment for chronic low back pain: A randomized controlled trial. Spine (Phila Pa 1976). 2003 Jul 1;28(13):1355-62.
81. Posadzki P, Ernst E. Osteopathy for musculoskeletal pain patients: A systematic review of randomized controlled trials. Clin Rheumatol. 2011 Feb;30(2):285-91.
82. Simons D, Travell J, Simons L. Myofascial pain and dysfunction: The trigger point manual. 2nd ed. Baltimore: Williams and Wilkins; 1999.
83. Alvarez DJ, Rockwell PG. Trigger points: Diagnosis and management. Am Fam Physician. 2002 Feb 15;65(4):653-60.
84. Rachlin E. Trigger points. myofascial pain and fibromyalgia: Trigger point management. St. Louis: Mosby; 1994.
85. MERTON PA. Voluntary strength and fatigue. J Physiol. 1954 Mar 29;123(3):553-64.
86. Cheng AJ, Dalton BH, Harwood B, Power GA. 'SIT' down and relax: The interpolated twitch technique is still a valid measure of central fatigue during sustained contraction tasks. J Physiol. 2013 Aug 1;591(Pt 15):3677-8.
87. Gandevia SC, Herbert RD, Leeper JB. Voluntary activation of human elbow flexor muscles during maximal concentric contractions. J Physiol. 1998 Oct 15;512 (Pt 2)(Pt 2):595-602.
88. Belanger AY, McComas AJ. Extent of motor unit activation during effort. J Appl Physiol Respir Environ Exerc Physiol. 1981 Nov;51(5):1131-5.
89. Bigland-Ritchie B, Furbush F, Woods JJ. Fatigue of intermittent submaximal voluntary contractions: Central and peripheral factors. J Appl Physiol (1985). 1986 Aug;61(2):421-9.
90. Gandevia SC, McKenzie DK. Activation of human muscles at short muscle lengths during maximal static efforts. J Physiol. 1988 Dec;407:599-613.
91. Herbert RD, Gandevia SC. Muscle activation in unilateral and bilateral efforts assessed by motor nerve and cortical stimulation. J Appl Physiol (1985). 1996 Apr;80(4):1351-6.
92. Allen GM, Gandevia SC, McKenzie DK. Reliability of measurements of muscle strength and voluntary activation using twitch interpolation. Muscle Nerve. 1995 Jun;18(6):593-600.
93. Taylor JL. Point: The interpolated twitch does/does not provide a valid measure of the voluntary activation of muscle. J Appl Physiol (1985). 2009 Jul;107(1):354-5.
94. Horstman AM. The interpolated twitch can be a useful tool in patient research. J Appl Physiol (1985). 2009 Jul;107(1):359; discussion 367-8.

95. de Haan A, Gerrits KH, de Ruiter CJ. Counterpoint: The interpolated twitch does not provide a valid measure of the voluntary activation of muscle. J Appl Physiol (1985). 2009 Jul;107(1):355,7; discussion 357-8.
96. Herzog W. Twitch interpolation represents muscle activation in a qualitative manner only. J Appl Physiol (1985). 2009 Jul;107(1):360,1; discussion 367-8.
97. de Haan A. The influence of stimulation frequency on force-velocity characteristics of in situ rat medial gastrocnemius muscle. Exp Physiol. 1998 Jan;83(1):77-84.
98. Suter E, Herzog W. Effect of number of stimuli and timing of twitch application on variability in interpolated twitch torque. J Appl Physiol (1985). 2001 Mar;90(3):1036-40.
99. Portney L, Watkins M. Foundations of clinical research, applications to practice. 3rd ed. Cohen M, editor. Upper Saddle River, New Jersey: Julie Levin Alexander; 2009.
100. Lachin JM. Introduction to sample size determination and power analysis for clinical trials. Control Clin Trials. 1981 Jun;2(2):93-113.
101. Allen GM, McKenzie DK, Gandevia SC. Twitch interpolation of the elbow flexor muscles at high forces. Muscle Nerve. 1998 Mar;21(3):318-28.
102. Bellemare F, Bigland-Ritchie B. Assessment of human diaphragm strength and activation using phrenic nerve stimulation. Respir Physiol. 1984 Dec;58(3):263-77.
103. Ohnhaus EE, Adler R. Methodological problems in the measurement of pain: A comparison between the verbal rating scale and the visual analogue scale. Pain. 1975 Dec;1(4):379-84.
104. Scott J, Huskisson EC. Graphic representation of pain. Pain. 1976 Jun;2(2):175-84.
105. Flandry F, Hunt JP, Terry GC, Hughston JC. Analysis of subjective knee complaints using visual analog scales. Am J Sports Med. 1991 Mar-Apr;19(2):112-8.
106. Huskisson EC. Measurement of pain. Lancet. 1974 Nov 9;2(7889):1127-31.
107. Canadian society of exercise physiology (CSEP) - canadian physical activity guidelines (all ages)

[homepage on the Internet]. Canada: . 2014. Available from: http://www.csep.ca/english/view.asp?x=890.
108. Kujala UM, Jaakkola LH, Koskinen SK, Taimela S, Hurme M, Nelimarkka O. Scoring of patellofemoral disorders. Arthroscopy. 1993;9(2):159-63.
109. Singer B, Singer K. Anterior knee pain scale. Aust J Physiother. 2009;55(2):140.
110. Crossley KM, Bennell KL, Cowan SM, Green S. Analysis of outcome measures for persons with patellofemoral pain: Which are reliable and valid? Arch Phys Med Rehabil. 2004 May;85(5):815-22.
111. Basmajian J, Blumenstein R. Electrode placement in EMG biofeedback. Baltimore: Williams and Wilkins; 1980.
112. Barnes I. Therapeutic insight. Phys Ther Forum. 1987:423-9.
113. Barnes I. Myofascial release seminar. PA, Pain and Stress Control Center: ; 1988.
114. Manheim C, Lavett D. The Myofascia1 release manual. Thorofare, NJ: Slack Incorporated; 1989.
115. Hanten WP, Chandler SD. Effects of myofascial release leg pull and sagittal plane isometric contract-relax techniques on passive straight-leg raise angle. J Orthop Sports Phys Ther. 1994 Sep;20(3):138-44.

116. Schneider MJ. Tender points/fibromyalgia vs. trigger points/myofascial pain syndrome: A need for clarity in terminology and differential diagnosis. J Manipulative Physiol Ther. 1995 Jul-Aug;18(6):398-406.
117. Dippenaar D, Korporaal C, Jones A, Globe G, Snyder W. Myofascial trigger points in the quadriceps femoris muscle of patellofemoral pain syndrome subjects assessed and correlated with NRS-101, algometry, and piloted patellofemoral pain severity and myofascial diagnostic scales. Journal of the American Chiropractic Association. 2008:16-28.
118. Hains G, Hains F. Patellofemoral pain syndrome managed by ischemic compression to the trigger points located in the peri-patellar and retro-patellar areas: A randomized clinical trial. Clinical Chiropractic. 2010;13:201-9.
119. Stakes N, Myburgh C, Brantingham J, Moyer R, Jensen M, Globe G. A prospective randomized clinical trial to determine efficacy of combined spinal manipulation and patella mobilization compared to patella mobilization alone in the conservative management of patellofemoral pain syndrome. J Amer Chiropr Assoc. 2006;43(7):11-8.
120. Travell J, Simmons D. Myofascial pain and dysfunction: Trigger point manual. the lower extremities. vol 2. Media, PA: Williams and Wilkins; 1983.
121. Antich TJ, Randall CC, Westbrook RA, Morrissey MC, Brewster CE. Evaluation of knee extensor mechanism disorders: Clinical presentation of 1 12 patients*. J Orthop Sports Phys Ther. 1986;8(5):248-54.
122. Ulus Y, Tander B, Akyol Y, Durmus D, Buyukakincak O, Gul U, et al. Therapeutic ultrasound versus sham ultrasound for the management of patients with knee osteoarthritis: A randomized double-blind controlled clinical study. Int J Rheum Dis. 2012 Apr;15(2):197-206.
123. Roebroeck ME, Dekker J, Oostendorp RA. The use of therapeutic ultrasound by physical therapists in dutch primary health care. Phys Ther. 1998 May;78(5):470-8.
124. van der Windt DA, van der Heijden GJ, van den Berg SG, ter Riet G, de Winter AF, Bouter LM. Ultrasound therapy for musculoskeletal disorders: A systematic review. Pain. 1999 Jun;81(3):257-71.
125. Herbert RD, Gandevia SC. Twitch interpolation in human muscles: Mechanisms and implications for measurement of voluntary activation. J Neurophysiol. 1999 Nov;82(5):2271-83.
126. Suter E, Herzog W, Bray RC. Quadriceps inhibition following arthroscopy in patients with anterior knee pain. Clin Biomech (Bristol, Avon). 1998 Jun;13(4-5):314-9.
127. Bialosky JE, Bishop MD, Price DD, Robinson ME, George SZ. The mechanisms of manual therapy in the treatment of musculoskeletal pain: A comprehensive model. Man Ther. 2009 Oct;14(5):531-8.
128. Shield A, Zhou S. Assessing voluntary muscle activation with the twitch interpolation technique. Sports Med. 2004;34(4):253-67.
129. Kisaalita NR, Roditi D, Robinson ME. Factors affecting placebo acceptability: Deception, outcome, and disease severity. J Pain. 2011 Aug;12(8):920-8.
130. Vase L, Petersen GL, Riley JL,3rd, Price DD. Factors contributing to large analgesic effects in placebo mechanism studies conducted between 2002 and 2007. Pain. 2009 Sep;145(1-2):36-44.
131. Bialosky JE, Bishop MD, George SZ, Robinson ME. Placebo response to manual therapy: Something out of nothing? J Man Manip Ther. 2011 Feb;19(1):11-9.

132. STENER B, PETERSEN I. Electromyographic investigation of reflex effects upon effects upon stretching the partially ruptured medial collateral ligament of the knee joint. Acta Chir Scand. 1962 Nov;124:396-411.
133. Shakespeare DT, Stokes M, Sherman KP, Young A. Reflex inhibition of the quadriceps after meniscectomy: Lack of association with pain. Clin Physiol. 1985 Apr;5(2):137-44.
134. Bennell K, Bartam S, Crossley K, Green S. Outcome measures in patellofemoral pain syndrome: Test retest reliability and inter-relationships. Physical Therapy in Sport. 2000;1(2):32-41.
135. Watson CJ, Propps M, Ratner J, Zeigler DL, Horton P, Smith SS. Reliability and responsiveness of the lower extremity functional scale and the anterior knee pain scale in patients with anterior knee pain. J Orthop Sports Phys Ther. 2005 Mar;35(3):136-46.
136. Lieb FJ, Perry J. Quadriceps function. an electromyographic study under isometric conditions. J Bone Joint Surg Am. 1971 Jun;53(4):749-58.
137. Souza DR, Gross MT. Comparison of vastus medialis obliquus: Vastus lateralis muscle integrated electromyographic ratios between healthy subjects and patients with patellofemoral pain. Phys Ther. 1991 Apr;71(4):310,6; discussion 317-20.
138. Herrington L. The role of vastus medialis oblique in patellofemoral pain syndrome. Critical Reviews™ in Physical and Rehabilitation Medicine. 1998;10(3).
139. Kollmitzer J, Ebenbichler GR, Kopf A. Reliability of surface electromyographic measurements. Clin Neurophysiol. 1999 Apr;110(4):725-34.
140. Doorenbosch CA, Harlaar J. A clinically applicable EMG-force model to quantify active stabilization of the knee after a lesion of the anterior cruciate ligament. Clin Biomech (Bristol, Avon). 2003 Feb;18(2):142-9.
141. Kankaanpaa M, Taimela S, Laaksonen D, Hanninen O, Airaksinen O. Back and hip extensor fatigability in chronic low back pain patients and controls. Arch Phys Med Rehabil. 1998 Apr;79(4):412-7.
142. Komi PV, Buskirk ER. Reproducibility of electromyographic measurements with inserted wire electrodes and surface electrodes. Electromyography. 1970 Nov-Dec;10(4):357-67.
143. Sedliak M, Finni T, Cheng S, Haikarainen T, Hakkinen K. Diurnal variation in maximal and submaximal strength, power and neural activation of leg extensors in men: Multiple sampling across two consecutive days. Int J Sports Med. 2008 Mar;29(3):217-24.
144. Youngstedt SD, O'Connor PJ. The influence of air travel on athletic performance. Sports Med. 1999 Sep;28(3):197-207.
145. Clark BC. In vivo alterations in skeletal muscle form and function after disuse atrophy. Med Sci Sports Exerc. 2009 Oct;41(10):1869-75.
146. Clark BC, Pierce JR, Manini TM, Ploutz-Snyder LL. Effect of prolonged unweighting of human skeletal muscle on neuromotor force control. Eur J Appl Physiol. 2007 May;100(1):53-62.
147. Basmajian J, de Luca C. Muscles alive: Their functions revealed by electromyography. Baltimore, MD: Williams and Wilkins; 1985.
148. Flynn T, Fritz J, Whitman J, Wainner R, Magel J, Rendeiro D, et al. A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with spinal manipulation. Spine (Phila Pa 1976). 2002 Dec 15;27(24):2835-43.

149. Cleland JA, Childs JD, Fritz JM, Whitman JM, Eberhart SL. Development of a clinical prediction rule for guiding treatment of a subgroup of patients with neck pain: Use of thoracic spine manipulation, exercise, and patient education. Phys Ther. 2007 Jan;87(1):9-23.

APPENDIX A: SAMPLE SIZE CALCULATION

Literature reviews suggested a study by Hurley and Newham (36) who determined that routine physiotherapy twice per week, increased the knee-extensor voluntary activation (VA) in patients with knee pain. However the limitation of this study was that there was no control group and only 10 patients received the physiotherapy intervention. For the determination of the sample size (100), we have estimated this by using the mean difference pre and post treatment (19% voluntary activation deficit pre-treatment and 5% voluntary activation deficit post-treatment) and the corresponding standard deviations (±7% pre-treatment and ±3% post-treatment). Based on these results, we estimated that the %VA could improve by a mean difference of 14% and the sample size calculation was therefore estimated to detect a difference of 14% between the intervention group (MRT or TPT) and the control sham group. An $\alpha = 0.05$ (acceptable type I error) and $\beta = 0.20$ (acceptable type II error) were used. The sample size calculation was calculated to be 2 participants per group or a total of 6, however we increased the sample size to 10 participants per group to a total of 30, which was approved by the committee during the research proposal meeting and sufficient for a Master's thesis. This would account for a drop-out rate of 20%.

$$N = \left[\frac{Z_{\alpha}\,\sigma_0 + Z_{\beta}\,\sigma_1}{\mu_1 - \mu_0} \right]^2$$

$$N = \left[\frac{(1.645)(7) + (0.85)(3)}{(19-5)} \right]^2$$

$$N = \left[\frac{(11.515) + (2.55)}{14} \right]^2$$

N = 1.009 (Will round to 2 to increase sample)

N = 2 participants/group

N = 2 x 3 groups

N = 6 Total, however will increase to **10/group**

N = Sample Size
σ_0 = Standard deviation (Pre)
σ_1 = Standard deviation (Post)
μ_0 = Mean (Pre)
μ_1 = Mean (Post)
Z_{α} = with $\alpha = 0.05 = 1.645$
Z_{β} = with $\beta = 0.20$ (power = 0.80) = 0.85

APPENDIX B: RECRUITMENT POSTER

DO YOU HAVE KNEE

PAIN?

Interested in participating in a 6-8 week treatment program for your knee pain?

Located at the University of Calgary, Human Performance Laboratory.

If you would like to be involved are active 30min/day & between 18-45 years of age please contact:

Conrad Tang

403-220-7119

ctang@kin.ucalgary.ca

The Roger Jackson Center for Health & Wellness Research

Assessment & Treatment Included

Conrad Tang 403-220-7119 ctang@kin.ucalgary.ca	Conrad Tang 403-220-7119 ctang@kin.ucalgary.ca	Conrad Tang 403-220-7119 ctang@kin.ucalgary.ca	Conrad Tang 403-220-7119 ctang@kin.ucalgary.ca	Conrad Tang 403-220-7119 93 ctang@kin.ucalgary.ca	Conrad Tang 403-220-7119 ctang@kin.ucalgary.ca	Conrad Tang 403-220-7119 ctang@kin.ucalgary.ca	Conrad Tang 403-220-7119 ctang@kin.ucalgary.ca	Conrad Tang 403-220-7119 ctang@kin.ucalgary.ca	Conrad Tang 403-220-7119 ctang@kin.ucalgary.ca

APPENDIX C: INFORMED CONSENT

Subject Informed Consent

Title: Biomechanics of Manual Therapy
Investigators: Walter Herzog PhD, Sarah Wuest DC, and Conrad Tang DC.
This consent form, a copy of which has been given to you, is only part of the process of informed consent. It should give you the basic idea of what the research is about and what your participation will involve. If you would like more details about something mentioned here, or information not included here, you should feel free to ask. Please take the time to read this carefully.

Purpose
The purpose of this experiment is to gather information concerning the amount of change in knee pain, functional measurements, quadriceps muscle inhibition, strength, and muscle activity to the patient with anterior knee pain during manual soft-tissue treatments.

Explanation of Patient's Involvement
You will be randomly placed into three groups all of which will receive three different types of commonly used manual therapy treatments for knee pain. The specific type of manual therapy treatment will not be disclosed to you until the trial period is complete in order to prevent any perceptions that may alter the outcome of the study. Additionally we request that you not reveal any information regarding your treatment to the outcome assessor, Conrad Tang during this period. At the completion of the trial, before un-blinding, participants will also be asked to indicate to which group they thought they had been allocated.
You will be asked to attend a testing session of approximately 1 hour. During this session, we may measure surface electromyographical (EMG) activity of selected trunk or limb muscles. If this is done, disposable electrodes will be attached in order to measure muscle activity in selected muscle groups. No penetration of the skin by needles is required. Then you will perform a series of maximal muscle contractions.
During the contractions, we will use the "interpolated twitch technique" which measures your ability to fully activate specific muscle groups. This technique requires that a series of electrical stimulations be applied to your muscle. To do this, two padded electrodes will be placed on the upper and lower part of the muscle and fixed to the skin with tape. A conducting gel will be placed on the electrodes to help transmit the electrical stimuli to your muscles. This will be washed off with water following the procedure.
Following these measurements a clinician will conduct an assessment of your knee and then, will give you a commonly used manual therapy treatment. Following the assessment and treatment the measurements with surface electromyographical (EMG) activity of selected trunk or limb muscles and the interpolated twitch technique will be taken again.

Risks And Discomforts

The risks involved are minimal. The manual therapy treatments performed by the clinician are all standard procedures. Side effects could relate to a brief discomfort or minor muscle soreness after the manual therapy treatment. There might be some discomfort of post-test joint or muscle pain of short duration. There is some potential for minor muscle strain or slight worsening of your condition. The electrical stimuli applied are very brief and may feel unusual and slightly uncomfortable. Voltage control during stimulation is ensured by an isolation unit, which will shut off if the applied voltage exceeds 240V. The conducting gel used for the stimulation electrodes may cause some minor skin irritation. In the event that you suffer injury as a result of participating in this research, no treatment or compensation will be provided for you by the University or the Researchers. You still have all of your legal rights. Nothing said here about treatment or compensation in any way alters your right to recover damages.

Doctors of Chiropractic, Medical doctors, and Physiotherapists who use manual therapy techniques are required to advise patients that there are some risks associated with such treatment. In particular, you should note:

a) While rare, some patients have experienced temporary pain or mild discomfort, and swelling after soft-tissue treatment;
b) There have been reported cases of bruising, fatigue and headaches after soft-tissue treatment.
c) Subsequent heating of the therapeutic ultrasound applicator could have a risk of causing a burn.
d) Prolonged standing waves of the therapeutic Ultrasound might result in blood flow arrest and cause possible damage to the endothelial cells in the blood vessel walls. This could also lead to the formation of blood clots.
e) If any pain or uncomfortable "prickly" sensation is felt, this may be an indication that the bones or nerve endings in the vicinity of the therapeutic ultrasonic beam are becoming, or are already, overheated.

Benefits To Be Expected

This study will allow us to quantify muscle inhibition and reflex responses in patient populations suffering from musculoskeletal pathologies. Furthermore, we will be able to understand the mechanisms responsible for muscle inhibition and reflex responses and the information we get from this study may help us to improve treatment programs involving manual therapy treatments for knee pain in the future. We expect that the results of this study may be directly used to improve manual therapy programs designed to restore normal muscle function following joint injuries.

Use Of Personal Information

Information obtained during this research project is confidential. It will not be released without your written consent. The information however, may be used for statistical analysis or scientific purposes with your right to privacy retained. If you are presently or have been a patient at the University of Calgary Sports Medicine Centre, Conrad Tang will be allowed to review your health records, including any images, for research purposes. This would only be permitted with your written consent in advance. This process would allow us to clarify and to review any medical conditions that may provide us with more information which may be relevant to the research project.

Freedom Of Consent

Your signature on this form indicates that you have understood to your satisfaction the information regarding participation in the research project and agree to participate as a subject. In no way does this waive your legal right nor release the investigator, sponsors, or involved institutions from their legal and

professional responsibilities. You are free to withdraw from the study at any time without jeopardizing your health care. Your continued participation should be as informed as your initial consent, so you should feel free to ask for clarification or new information throughout your participation. If you have further questions concerning matters related to this research, please contact:

Dr. Walter Herzog (403) 220-8525

If you have any questions concerning your rights as a possible participant in this research, please contact the Director, the Office of Medical Bioethics, University of Calgary, at 403-220-7990.

_____	_____
Participant' Name	Signature and Date
_____	_____
Investigator/Delegate's Name	Signature and Date
_____	_____
Witness' Name	Signature and Date

The University of Calgary Conjoint Health Research Ethics Board has approved this research study.

A signed copy of this consent form has been given to you to keep for your records and reference.

APPENDIX D: VISUAL ANALOGUE SCALE

Patient Name: _____ Date: _____

Visual Analogue Scale (VAS) – During physical activity

_____

NO PAIN — WORST PAIN IMAGINABLE

Visual Analogue Scale (VAS) – 1 hour after physical activity

_____

NO PAIN — WORST PAIN IMAGINABLE

Visual Analogue Scale (VAS) – Following 30 minutes of sitting with knees flexed

_____

NO PAIN — WORST PAIN IMAGINABLE

APPENDIX E: ANTERIOR KNEE PAIN SCALE

Name: _____
Date: _____
Age:
Knee: L/R
Duration of symptoms: __ years __ months

For each question, circle the letter choice which corresponds to your knee symptoms.

1. Limp
(a) None (5)
(b) Slight or periodical (3)
(c) Constant (0)

2. Support
(a) Full support without pain (5)
(b) Painful (3)
(c) Weight bearing impossible (0)

3. Walking
(a) Unlimited (5)
(b) More than 2 km (3)
(c) 1-2 km (2)
(d) Unable (0)

4. Stairs
(a) No difficulty (10)
(b) Slight pain when descending (8)
(c) Pain both when descending and ascending (5)
(d) Unable (0)

5. Squatting
(a) No difficulty (5)
(b) Repeated squatting painful (4)
(c) Painful each time (3)
(d) Possible with partial weight beating (2)
(e) Unable (0)

6. Running
(a) No difficulty (10)
(b) Pain after more than 2 km (8)
(c) Slight pain from start (6)
(d) Severe pain (3)

(e) Unable (0)

7. Jumping
(a) No difficulty (10)
(b) Slight difficulty (7)
(c) Constant pain (2)
(d) Unable (0)

8. Prolonged sitting with the knees flexed
(a) No difficulty (10)
(b) Pain after exercise (8)
(c) Constant pain (6)
(d) Pain forces to extend knees temporarily (4)
(e) Unable (0)

9. Pain
(a) None (10)
(b) Slight and occasional (8)
(c) Interferes with sleep (6)
(d) Occasionally severe (3)
(e) Constant and severe (0)

10. Swelling
(a) None (10)
(b) After severe exertion (8)
(c) After daily activities (6)
(d) Every evening (4)
(e) Constant (0)

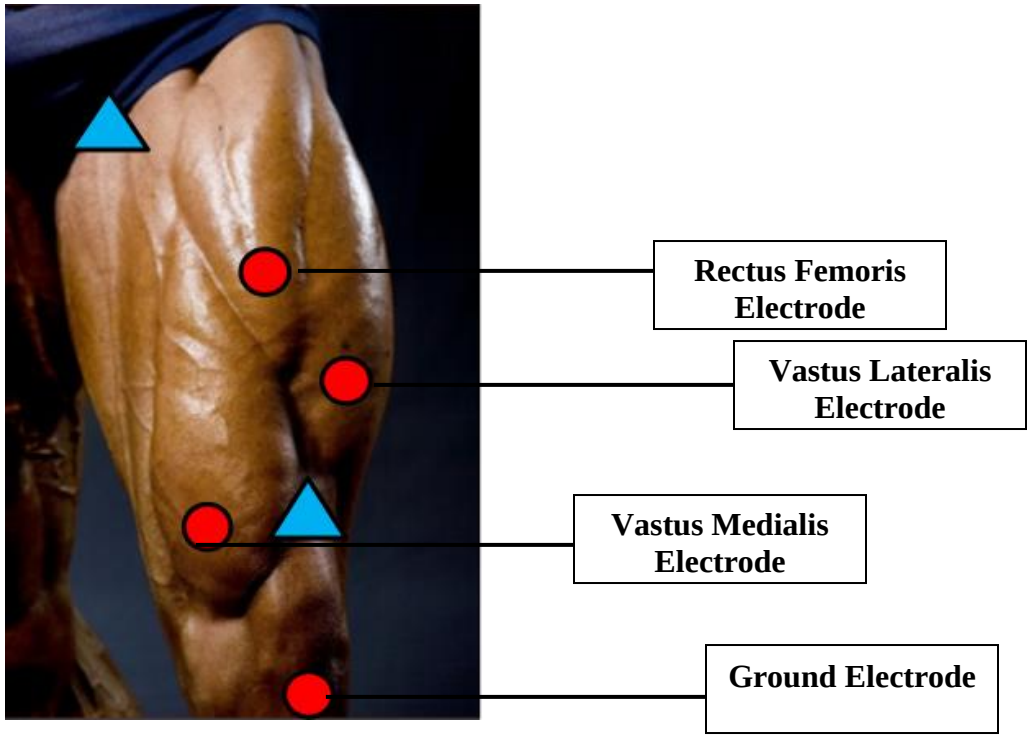
11. Abnormal painful patellar (knee-cap) movements (subluxation = partial dislocation)
(a) None (10)
(b) Occasionally in sports activities (6)
(c) Occasionally in daily activities (4)
(d) At least one documented dislocation (2)
(e) More than two dislocations (0)

12. Atrophy of thigh (Decrease in size of thigh muscle)
(a) None (5)
(b) Slight (3)
(c) Severe (0)

13. Flexion (bending) deficiency
(a) None (5)
(b) Slight (3)
(c) Severe (0)

APPENDIX F: LOCATION OF ELECTRMYOGRAPHY (EMG) PLACEMENT ON THE QUADRICEPS FEMORIS

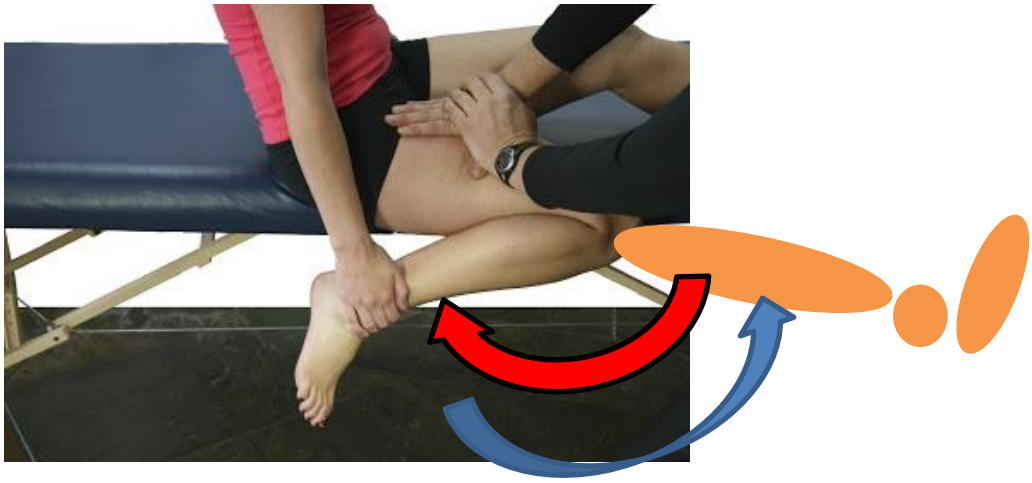
- Circles represent the EMG electrodes
- Triangles represent the Interpolated Twitch Rubber Electrodes (The proximal triangle is located on top of the femoral nerve and the distal electrode is just superior to the patella)



APPENDIX G: DIAGRAM AND DESCRITIPTION OF THE MYOFASCIAL RELEASE TECHNIQUE (MRT)

In the myofascial release technique the involved tissue is taken passively from a shortened position (knee at full extension) to a fully lengthened position (knee fully flexed) while the contact hand holds tension longitudinally along the soft tissue fibers (115). For example, a clinician places her/his thumb on the vastus lateralis muscle and applies tension longitudinally along the muscle fibers while passively bringing the patient's knee from full extension to full flexion.

The first intervention group (n_1=10) received MRT. Myofascial release technique was applied to the vastus medialis (VM), the vastus lateralis (VL), and the rectus femoris (RF) muscles. Eight repetitions of MRT, corresponding to eight knee flexion cycles, were applied to each of the target muscles during each treatment visit (6 treatment visits total).



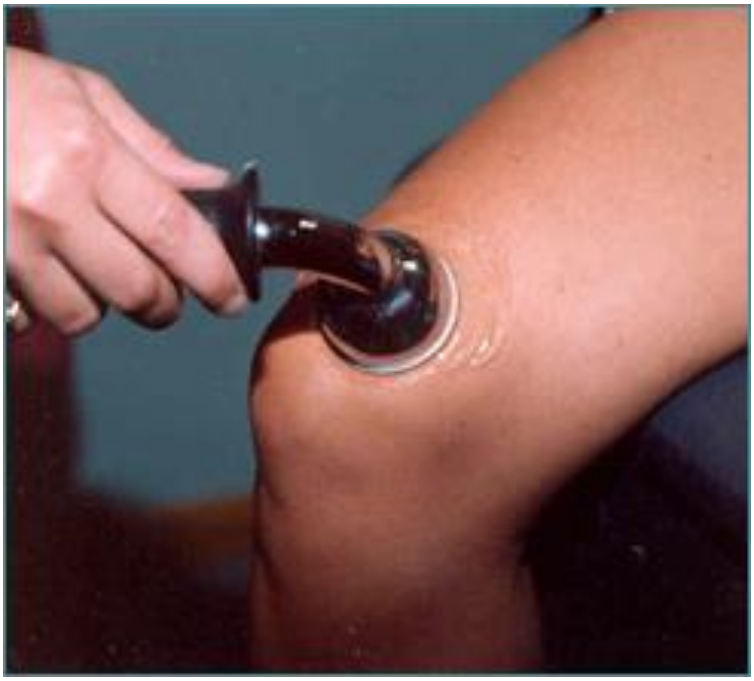
APPENDIX H: DIAGRAM AND DESCRITIPTION OF THE TRIGGER POINT THERAPY (TPT)

The Trigger Point Therapy is administered with the clinician putting firm pressure onto the affected area and gradually increasing the pressure until the patient's pain tolerance has been reached called ischemic compression (82).

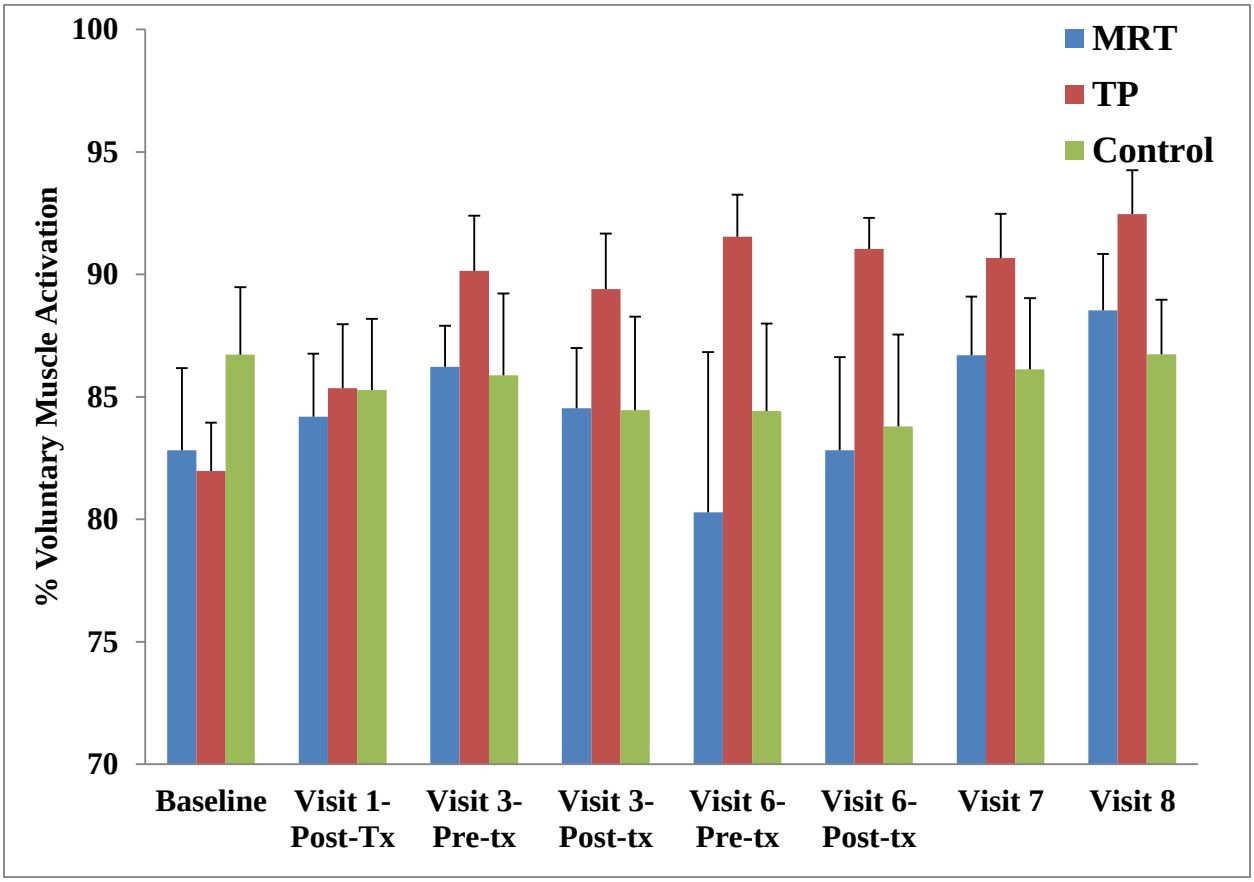
The TPT intervention group (n_2=10) was given pressure treatments on the vastus medialis, vastus lateralis, and rectus femoris for the six treatment visits. It consisted of the clinician applying a slowly increasing pressure over areas of elicited pain, tender point(s), for a duration of 15 seconds and five times for each muscle (VM, VL and RF).

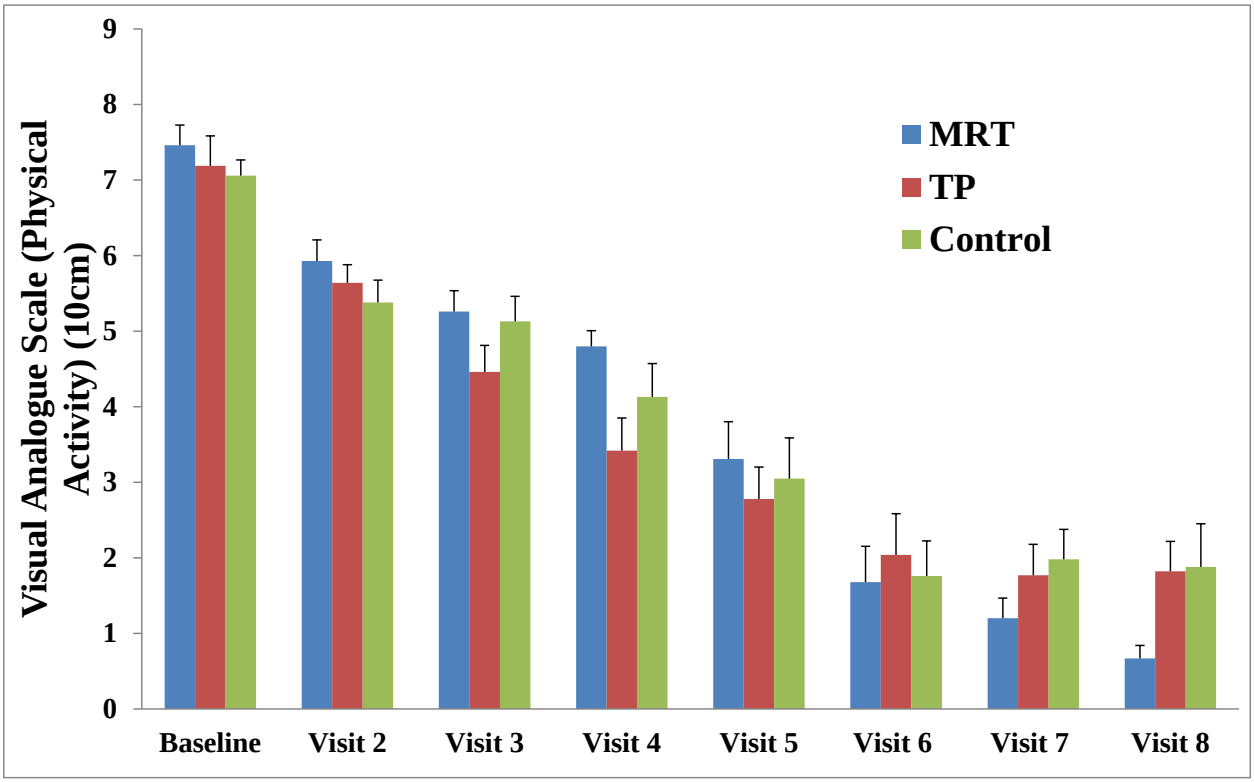
APPENDIX I: DIAGRAM AND DESCRITIPTION OF THE CONTROL SHAM THERAPEUTIC ULTRASOUND

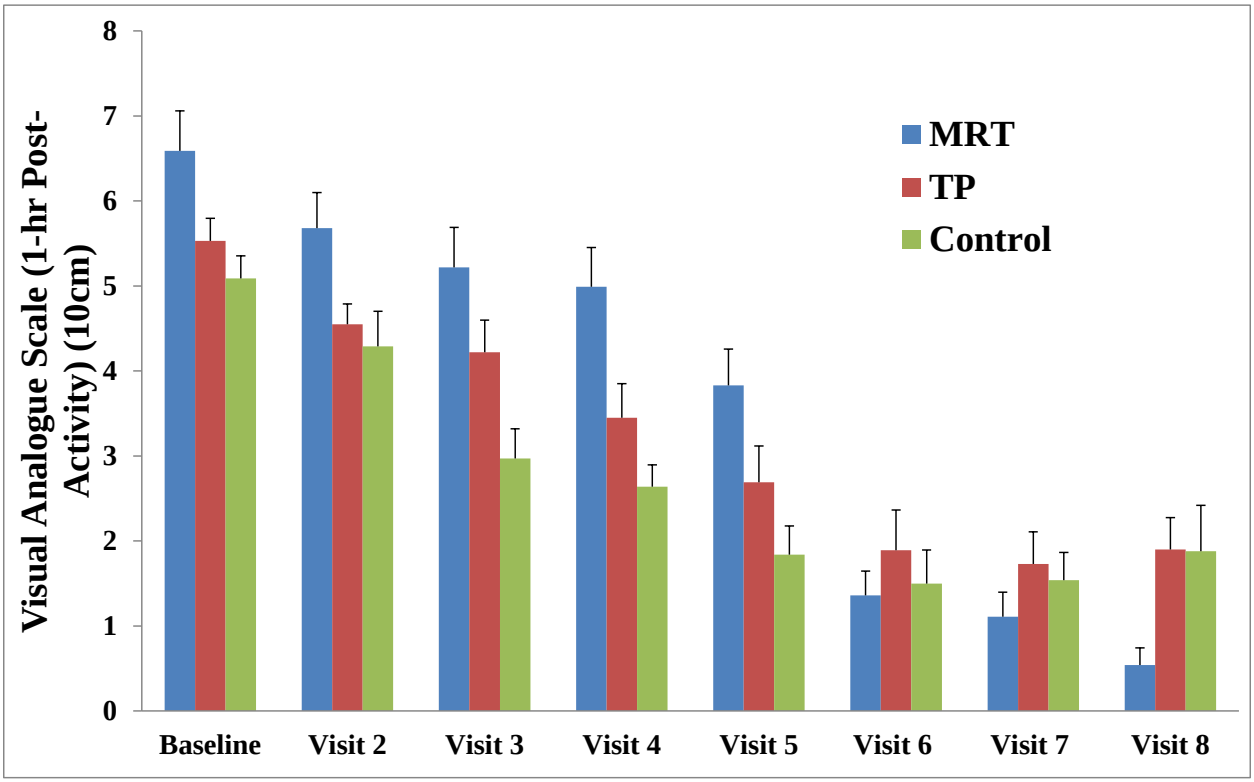
The therapeutic ultrasound was initially turned on so that the participant could hear the sound of the beep and be aware that they would be receiving a form of treatment. When the participants laid supine in the ready position for receiving the treatment, the ultrasound machine was turned off quietly. Questioning of the participants after the clinical trials showed that none of the subjects were aware that the ultrasound machine was turned off while they received their expected "treatment" intervention. The clinician then applied the ultrasound head covered with gel over the skin on the entire length of the RF, VL and VM for approximately four minutes per muscle for a total of 12 minutes. This time frame was used to match studies that have been used for musculoskeletal pain such as knee extensor mechanism disorders, which have been shown to decrease pain in the knee extensor muscles (121, 122).

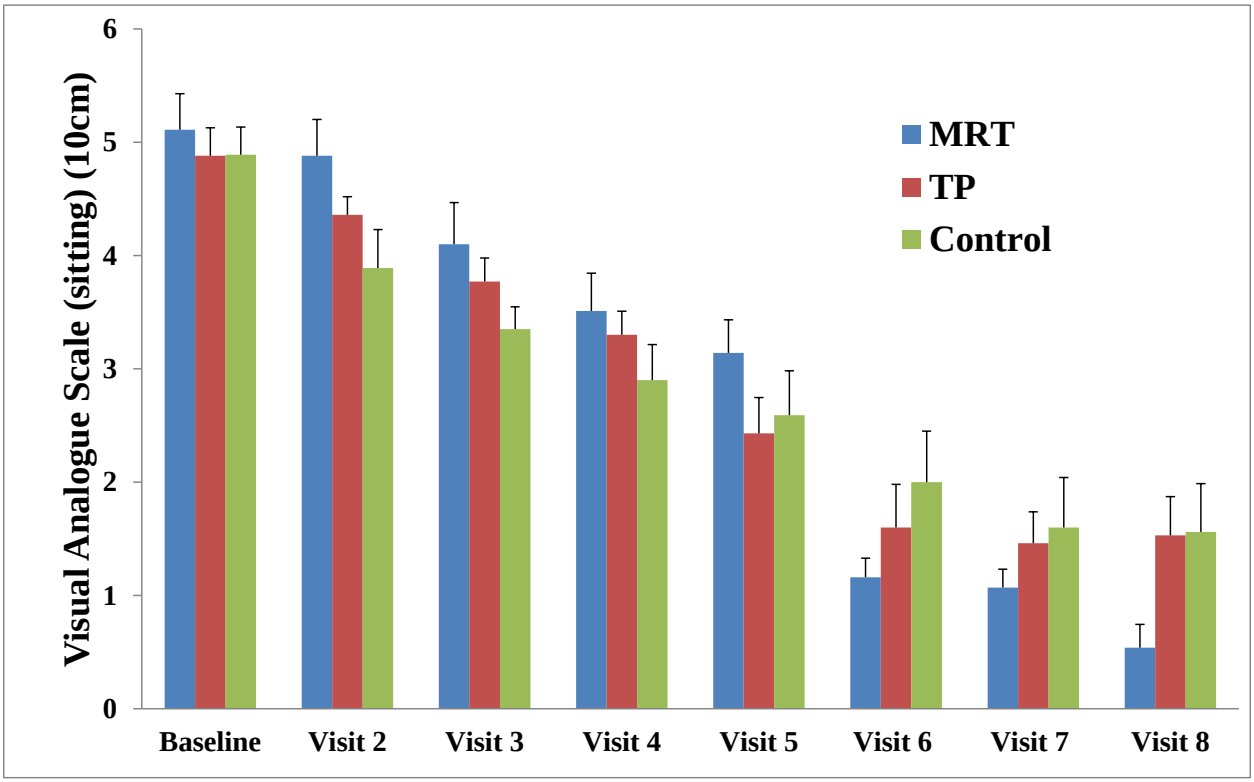


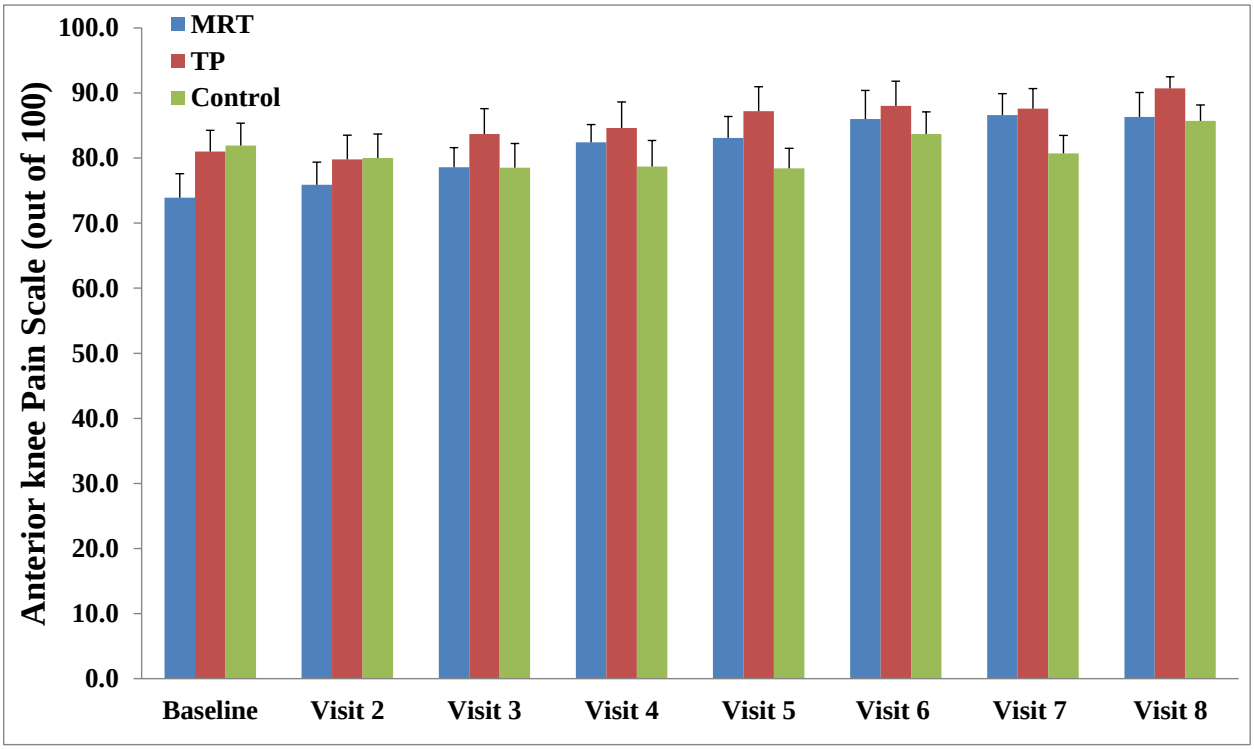
APPENDIX J: RESULTS

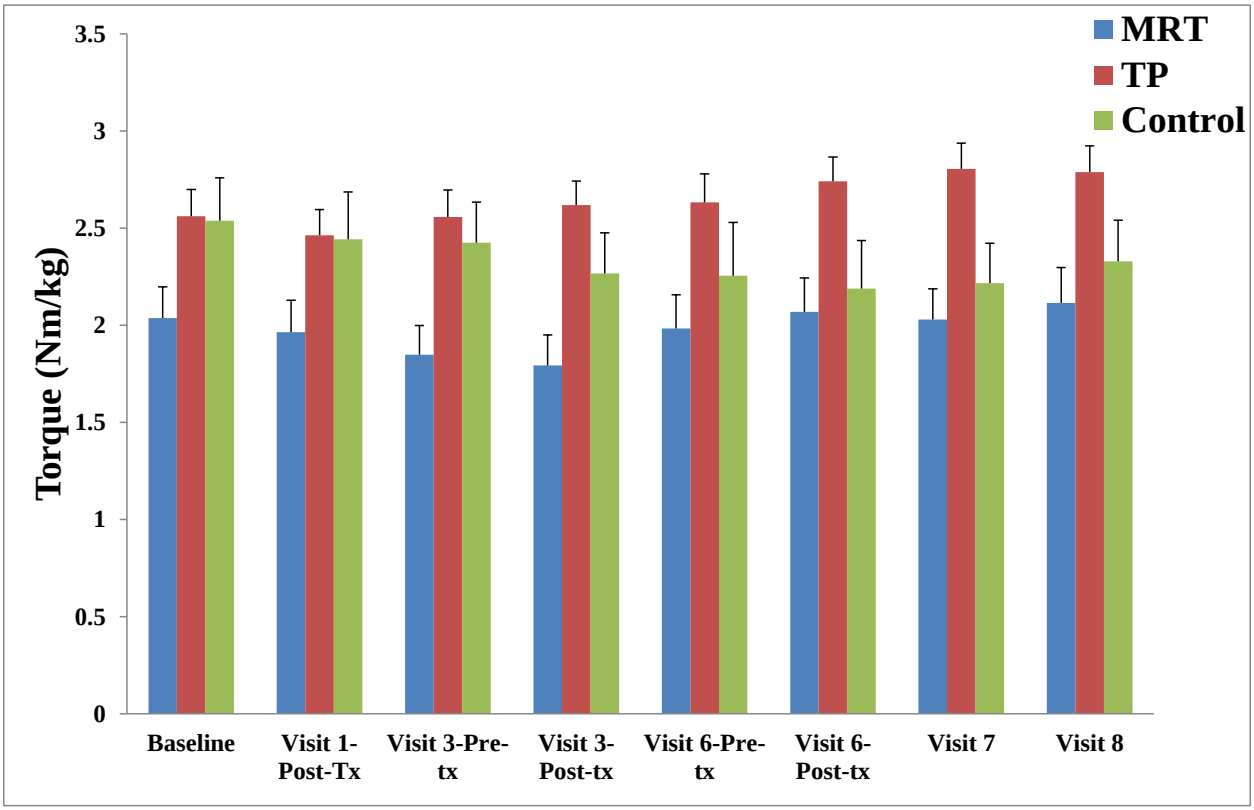


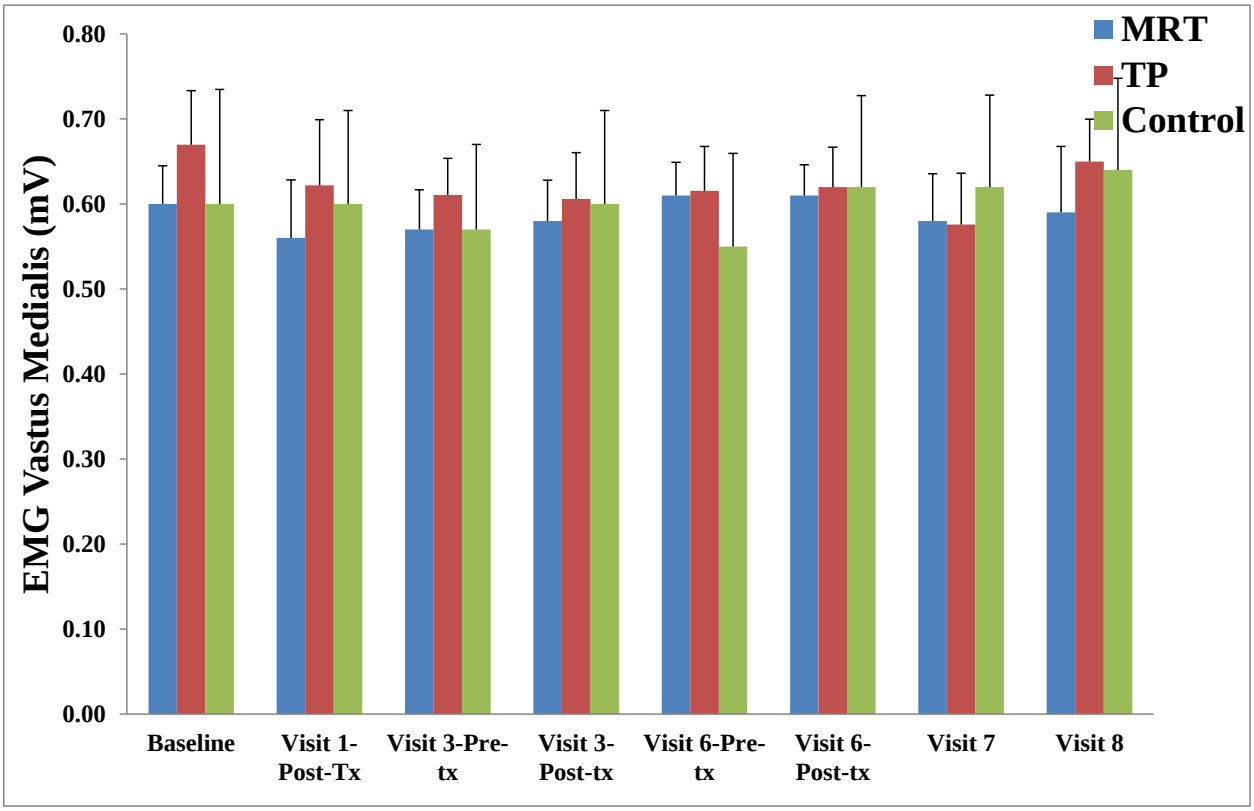

Visual Analogue Scale (Physical Activity) (10cm)
9
8
7
6
5
4
3
2
1
0
MRT
TP
Control
Baseline
Visit 2
Visit 3
Visit 4
Visit 5
Visit 6
Visit 7
Visit 8

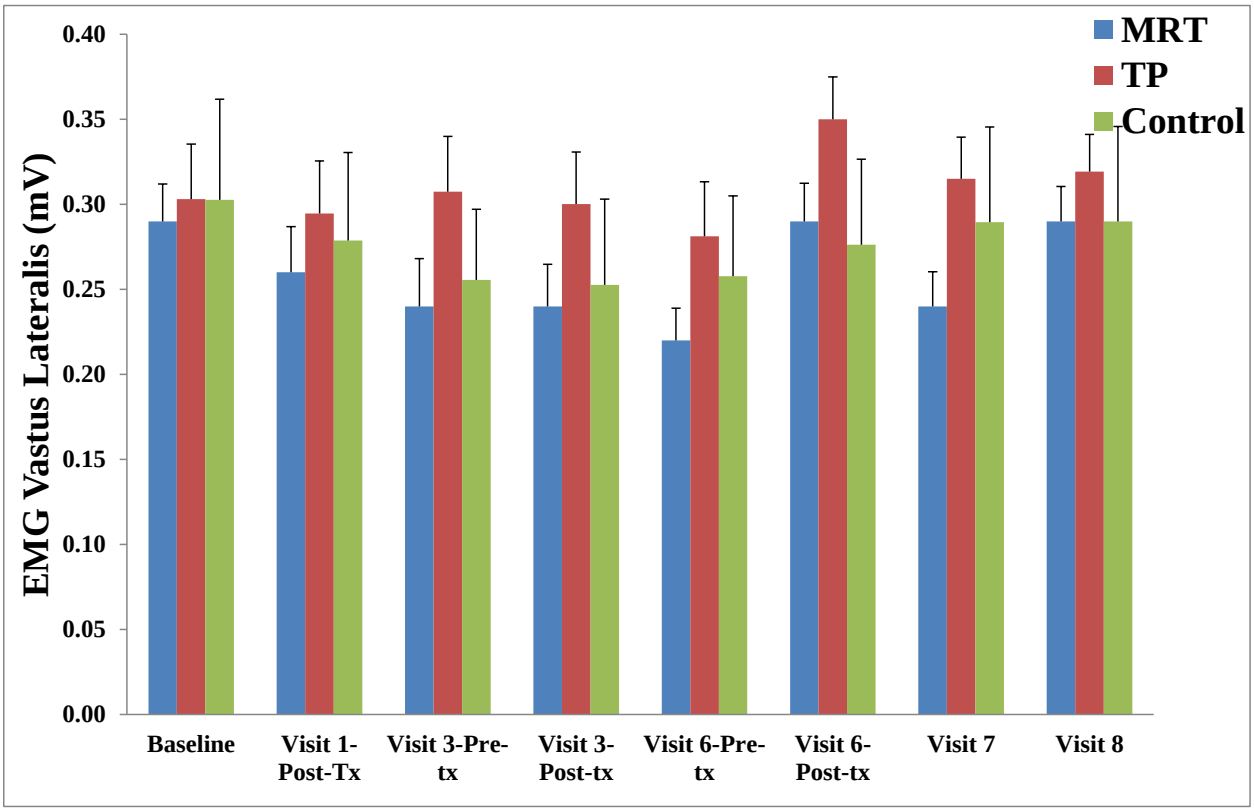

Visual Analogue Scale (1-hr Post-Activity) (10cm)
8
7
6
5
4
3
2
1
0
MRT
TP
Control
Baseline
Visit 2
Visit 3
Visit 4
Visit 5
Visit 6
Visit 7
Visit 8

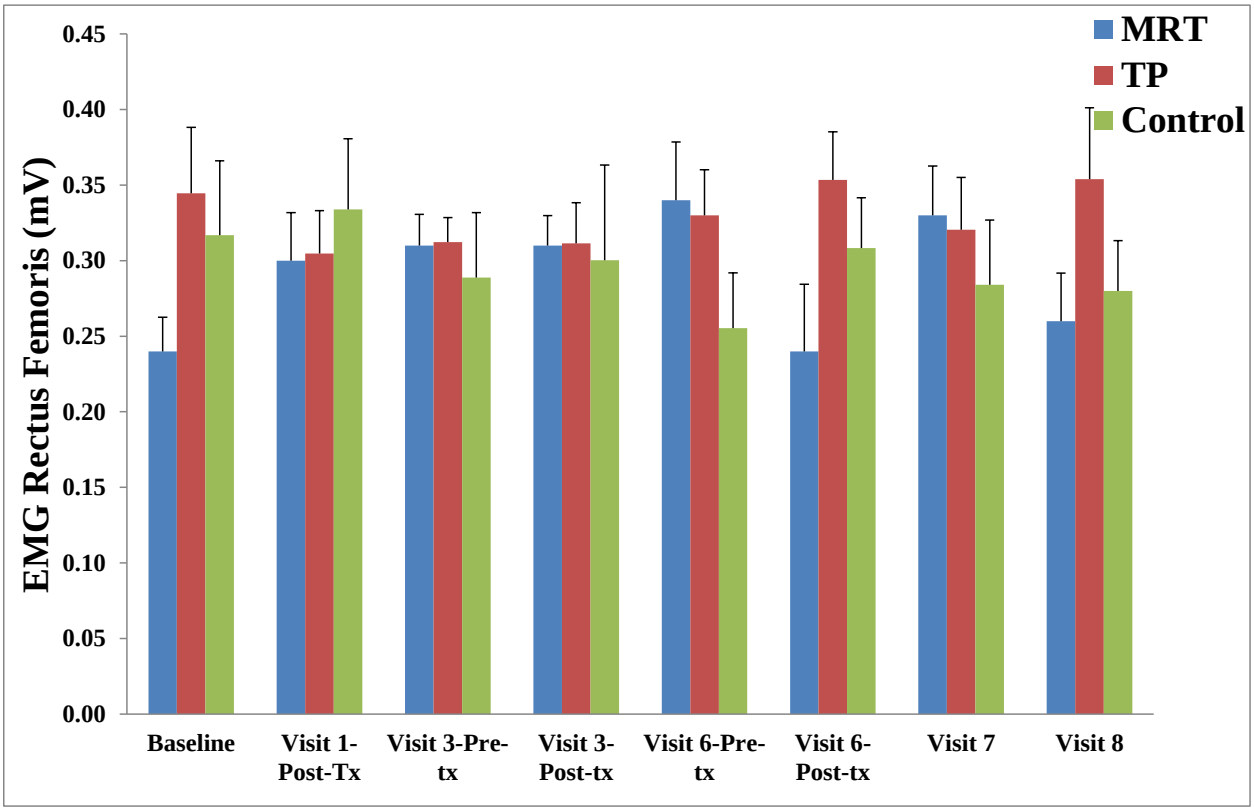

Visual Analogue Scale (sitting) (10cm)
6
5
4
3
2
1
0
MRT
TP
Control
Baseline
Visit 2
Visit 3
Visit 4
Visit 5
Visit 6
Visit 7
Visit 8


MRT
TP
Control
Anterior knee Pain Scale (out of 100)
100.0
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
0.0
Baseline
Visit 2
Visit 3
Visit 4
Visit 5
Visit 6
Visit 7
Visit 8


MRT
TP
Control
Torque (Nm/kg)
3.5
3
2.5
2
1.5
1
0.5
0
Baseline
Visit 1-Post-Tx
Visit 3-Pre-tx
Visit 3-Post-tx
Visit 6-Pre-tx
Visit 6-Post-tx
Visit 7
Visit 8


MRT
TP
Control
EMG Vastus Medialis (mV)
0.80
0.70
0.60
0.50
0.40
0.30
0.20
0.10
0.00
Baseline
Visit 1-Post-Tx
Visit 3-Pre-tx
Visit 3-Post-tx
Visit 6-Pre-tx
Visit 6-Post-tx
Visit 7
Visit 8


EMG Vastus Lateralis (mV)
0.40
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
MRT
TP
Control
Baseline
Visit 1-Post-Tx
Visit 3-Pre-tx
Visit 3-Post-tx
Visit 6-Pre-tx
Visit 6-Post-tx
Visit 7
Visit 8


MRT
TP
Control
EMG Rectus Femoris (mV)
0.45
0.40
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
Baseline
Visit 1-Post-Tx
Visit 3-Pre-tx
Visit 3-Post-tx
Visit 6-Pre-tx
Visit 6-Post-tx
Visit 7
Visit 8