IMPLEMENTATION OF EVIDENCE-BASED PRACTICE WITHIN AN ATHLETIC TRAINING ATHLETIC MODEL: A DISSERTATION OF CLINICAL PRACTICE IMPROVEMENT

A Dissertation
Presented in Partial Fulfillment of the Requirements for the
Degree of Doctor of Athletic Training
with a
Major in Athletic Training
in the
College of Graduate Studies
University of Idaho
by
Valerie Stevenson

Major Professor: Russell Baker, DAT
Committee Members: James May, DAT; Alan Nasypany, EdD; Don Reordan, PT
Department Administrator: Phillip W. Scruggs, PhD

July 2016

AUTHORIZATION TO SUBMIT DISSERTATION

This dissertation of Valerie Stevenson, submitted for the degree of Doctor of Athletic Training with a Major in Athletic Training and titled "Implementation of Evidence-Based Practice within an Athletic Training Athletic Model: A Dissertation of Clinical Practice Improvement," has been reviewed in its final form. Permission, as indicated by the signatures and dates given below, is now granted to submit final copies to the College of Graduate Studies for approval.

Major Professor: ______________________________ Date: ____________________

Russell Baker, D.A.T

Committee Members: ______________________________ Date: ____________________

James May, D.A.T

______________________________ Date: ____________________

Alan Nasypany, Ed.D

______________________________ Date: ____________________

Don Reordan, P.T

Department Administrator: ________________________ Date: ____________________

Phillip W. Scruggs, Ph.D

ABSTRACT

The Dissertation of Clinical Practice Improvement (DoCPI) is a culmination of clinical practice growth and scholarly development. Doctorate of Athletic Training (DAT) students are encouraged to examine not only their patient care, but also reflect upon themselves in a meaningful and practical manner. Reflection upon patient outcomes and clinical strengths and weaknesses combined to contribute to the chapters that are contained within this DoCPI. The final research multi-site study reflects the philosophy of the DAT in its mission to emphasize collection and use of patient outcomes to drive clinical decision-making. The examination of the effects of the Mulligan “Squeeze” Technique on the symptoms of meniscus tears has preliminarily provided a means to treating a very common and cost intensive injury with manual therapy. Thoughtfully identifying common injuries and seeking out viable and effective treatments is the foundation of action research in clinical practice and research. The following DoCPI provides evidence of how action research can be implemented and utilized in a systematic and clinically meaningful way as well as detail my student journey to from novice athletic trainer to advanced practitioner.

ACKNOWLEDGEMENTS

Obtaining a doctorate is not simply a one-person task. Many brilliant and knowledgeable people were instrumental in assisting me with this life-long goal. I would like to thank my Major Professor, Dr. Russell Baker, for his honest guidance and hard work in providing a road map for successful completion of the Doctorate of Athletic Training Program (DAT). Your dedication and patience throughout this process was the stability I needed to get to this point.

Dr. James May, the diligent and positive voice, thank you for your meaningful feedback and for offering a different perspective on many topics of clinical practice. You challenged me to examine concepts from all angles and I have grown because of that.

Dr. Alan Nasypany, your focus on improving clinical practice through self-examination has been transformative. I appreciate your willingness to share so many advanced and alternative clinical pearls through my student journey. Your insight was a driving force to developing my new clinical philosophy.

Don Reordan, thank you for your expert advice and always being available for consultation throughout this process. Your knowledge of the Mulligan Concept is extensive and I am honored to have been taught by one of the best.

Texas Woman's University thank you for providing an environment in which to grow and explore new scholarly approaches to my patient care.

DEDICATION

I would like to dedicate this dissertation to many people who have inspired and supported me throughout this process.

To my family, my eternal support system. I would not be where I am today without the example that has been set by all those who came before me. Your words of encouragement pushed me to begin and your arms of support held me through the finish.

Katie, you will never know how much your unwavering support has meant to me from start to finish. I am certain that I would not have been sane through this journey without you. You have been my yellow umbrella through all the storms. I love you to the moon and back.

Team Meniscus, you all allowed me to be me and I could not have asked for a better group of nerds to share this DAT journey with.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER 1

Narrative Summary

Athletic Training (AT) is my true, professional calling. I began my journey into the profession over 15 years ago, as a student at the University of Memphis. After obtaining a Bachelor of Science degree in Exercise and Sports Science, I continued my education with an entry-level Master of Athletic Training degree at the University of Arkansas. Despite numerous years of didactic coursework, clinical applications of orthopedic evaluations, therapeutic rehabilitation, and sideline discoveries of interventions for expedited return-to-play situations, I still had the desire to expand and advance my knowledge. After critical self-reflection, I decided that my clinical practice needed further refinement, because I was not satisfied with the patient outcomes I was producing and I knew I was capable of meeting a higher standard.

Education has always been a priority in my life. I come from a family of educators who teach at every level, from preschool to higher education, and I am constantly reminded how proper learning can be life-altering if you give it your undivided attention. When I discovered the Doctor of Athletic Training (DAT) program at the University of Idaho (UI), I knew it was what I needed to reach my goal of advancing my clinical practice and improving my patient care. The program's advance-practice philosophy supports a patient-centered clinical practice in which clinical decisions are based on the evidence provided by patient outcomes. After speaking with the DAT program's first graduate at a professional conference, I was convinced that I would advance my theoretical knowledge as well as my clinical skillset by pursuing a post-professional, terminal degree.

The DAT program at the University of Idaho is an advanced professional practice degree (PPD) wherein students, who are taught by actively practicing athletic training faculty, apply their knowledge and hone their skills through clinical practice. Each semester of the DAT program is designed to systematically and logically progress students toward advanced practice, and each element of advanced AT that is taught within a single semester intertwines with the next element to provide a strong foundation upon which students can build (Willis, Inman, & Valenti, 2010). The culminating project of the DAT program is the Dissertation of Clinical Practice Improvement (DoCPI). The DoCPI, which is reflective in nature, is intended to provide evidence of a student's clinical practice improvement and development as a scholar over the course of the DAT program. The process of attaining a PPD and revealing my growth through a DoCPI has improved my personal clinical practice and has shifted my professional outlook on patient care. I am no longer a novice, but a professional AT whose practice will continue to evolve as I continue to learn.

The innovative structure and philosophy of the UI DAT program represents the progressive potential of advanced clinical practice education in AT. The program incorporates an evidenced-based practice (EBP) philosophy, which emphasizes action research (AR). Action research involves addressing a local problem/topic and finding a solution to that problem/topic by producing answers within the parameters and confines of the location of the problem (Koshy, Koshy, & Waterman, 2011). The principle of recognizing a local, practical issue and finding a solution through practitioner-led interventions is the foundation of AR, as well as one of its strengths (Meyer, 2000).

Student residents utilize the principals and ideals of AR in their clinical sites during the clinical residency portion of the DAT. Within his or her clinical residency, a student develops methods for the systematic collection of patient outcomes and reflects on the most effective and appropriate manner to analyze and share the resulting clinical evidence. Before I enrolled in the DAT program, my patient care philosophy was emotion-based and relied heavily on anecdotal evidence. In other words, I determined my methods for treating a patient based on how I had treated similar injuries in the past. I paid little heed to whether or not a treatment was effective. I have since developed my own evidence-based patient care philosophy in which I implement the collection of patient outcomes. I use a combination of both electronic and paper forms to track patient data, which has proven to be the most streamlined approach in my practice for analyzing data completely and having direct interaction with my patients. Moreover, collecting outcomes in this manner has been pivotal to the composition of the case studies and the randomized controlled study (RCT) presented in this dissertation.

The research studies presented in chapters 2, 3, and 5 of this DoCPI serve as evidence of my development as a clinician. Although the injuries were common to the practice in which I worked before I entered the DAT program, I had always treated injuries of that particular type without much thought as to the underlying mechanisms involved. Once I began my studies in the DAT program, I developed a deeper level of understanding of the theories behind various treatment techniques, and I acquired hands-on training in those paradigms. A combination of grasping theory and moving beyond my novice experience allowed me to identify the most appropriate interventions for the patients referenced in chapters 2, 3, and 5, based on their clinical exams. Because the interventions that I used to treat these patients were performed in busy AT clinics during normal operating hours, and the injuries were common, local problems, the outcomes that were observed and reported on are relatable and applicable to other athletic trainers and healthcare workers and could assist these professionals in improving their own patient outcomes. The focus on conducting AR and using its guiding principles within my personal clinical practice has resulted in a personal mandate to collect patient outcomes for the duration of my professional career. Collecting patient outcomes will help me to continue to identify, analyze, and improve the areas of my practice that are weak. Honest contemplation of all components of my clinical practice, as detailed in my Plan of Advanced Practice (PoAP) (Appendix A), has resulted in a clinical practice philosophy that is evolving even as my skills as an AT are growing.

My PoAP serves as a reminder of my background as a student and practicing professional as well as a permanent guide to help formulate future professional goals. Consistently consulting and adjusting those goals helps to guide me toward continual growth and provides me with evidence of progress I have made along the way. Because my PoAP is an ever-evolving document, I refer back to its contents for direction and for assurance that I am actively seeking, reaching, and setting new professional goals. The plan contains the following: 1) details on my professional and clinical strengths and weaknesses; 2) an explanation of my advanced practice areas, the goals I have set to continue to improve in those areas, and which focus areas I plan to develop in the future; and 3) a description of my rehabilitation and low back pain treatment philosophies. Composing this PoAP revealed that I had not previously identified my personal clinical practice philosophy; therefore, I created one that is tangible and can be adjusted as I am exposed to new ideas and theories.

I was introduced to many new theories of efficacy for paradigms that were previously unknown to me during my doctorate education such as the Mulligan Concept (MC), Positional Release Technique (PRT), Primal Reflex Release Technique™ (PRRT™), MyoKinesthetic™ (MYK™) System, and Total Motion Release (TMR). Because I studied those theories and discussed application indications with fellow DAT student, I was able to begin treating common patient complaints and injuries more effectively and efficiently than I had during my previous clinical care. I began to identify common presentations in my patients, and I made note of how they responded to techniques that addressed specific mechanical or physiological injuries.

I used the aforementioned paradigms on their own, but I also used them in conjunction with each other, because most patient complaints involve more than one musculoskeletal entity. For example, a common injury, such as a lateral ankle sprain, may require a correction of a possible positional fault of the fibula on the talus. The positional fault could be corrected with the MC. At the same time, a minor tissue injury associated with that same sprain may require the reduction of trigger or tender points (TrP). Reduction of TrP could be done using PRT. Through collecting patient-specific and region-specific outcomes, and making adjustments based on the results of those outcomes, I have improved my patient care and have also developed a patient-centered foundation for my current treatment philosophy.

Patient-centered care, as well as a holistic approach to both the patient's and the clinician's well-being, is emphasized in the curriculum and philosophy of the DAT program. One of the often-overlooked components of advanced patient care is the clinician's mental presence or lack of mental presence during examination and treatment. Incorporating an "all-in" attitude into my clinical practice has improved my patient outcomes as well as my patients' overall care. Being "all-in" has required me to pay purposeful attention to all components of my patients' care. In class discussions, the DAT program faculty and students explored the negative effects that a distracted and disconnected clinician can have on a patient's well-being. The faculty also introduced psychosocial interventions that correct the disconnect between the clinician and the patient, which can be beneficial to both parties.

Through mind-body wellness-promoting psychosocial interventions, such as Tension and Trauma Releasing Exercises (TREs), Reflexercise, and Emotional Freedom Technique (EFT), I was able to start addressing my patients' physiological ailments while remaining connected with the patients during the duration of their treatments. For my patients to reach a level of healing not experienced through manual therapy interventions, the aforementioned techniques required that I be physically, mentally, and, in some cases, emotionally available to my patients. Improving my selection of manual therapy techniques, my accuracy in using these techniques, and my ability to recognize indications for the use of psychosocial interventions are all components of the global- and regional-interdependent (RI) components of my new clinical approach to patient care.

Regional interdependent assessments and treatment applications are a cornerstone of the DAT program. They were introduced and expounded on through numerous class discussions, Wordpress® blog posts, and instructor-led laboratory experiences. Using advanced paradigms in a global and RI manner has transformed my clinical practice entirely. A global and RI approach to patient care involves relating both injury assessments and treatments to possible dysfunctions in other areas of the body (Sueki, Cleland, & Wainner, 2013). Other dysfunctional components—either physical or psychological—may cause perceived pain in areas not directly related to the areas of dysfunction.

For instance, shoulder pain may be a result of an anteriorly rotated innominate, and central sensitization of the nervous system could be the cause of chronic pain when normal tissue healing time has elapsed. Currently, when I use a global and RI approach on hip flexor- and adductor-pain patients, I assess breathing function and primal reflexes in my initial evaluation. My decision is based on my patients' outcomes, which I collected after applying PRRT in previous cases. When I have difficulty maintaining the long-lasting effects of the MC, I also assess and clear any unbalanced breathing patterns to determine if a dysfunctional diaphragm caused the return of the pain. To assess for possible psychosocial implications and to determine if there is a need for psychosocial interventions, I administer assessments such as the Fear Avoidance Belief Questionnaire (FABQ). Evidence of my use of global and RI assessments and interventions, as well as of my use of AR principles to guide my clinical practice, are provided in Chapter 2 and Chapter 3 of this dissertation.

Chapter 2 is a case study entitled, "Using the MyoKinesthetic™ (MYK) System to Treat Bilateral Chronic Knee Pain." The purpose of this case study was to report on the effects of the MYK™ system on a patient who presented with bilateral chronic knee pain associated with knee osteoarthritis (OA). The MYK™ system's lower body postural assessment involves assessing the positions of the hip, knees, ankles, feet, and toes to determine the correct treatment intervention for a patient's primary complaint. The postural exam portion of the paradigm is an RI assessment technique, wherein the clinician identifies dysfunction in other areas of the body that could be contributing to the location of pain as described by a patient. By treating the musculature of the patient's entire lower body, knee pain and dysfunction were reduced in my patient, and function was improved.

Chapter 3 is case series entitled, "A Novel Approach to Treating Groin and Hip Flexor Pain Using Primal Reflex Release Technique™: A Case Series." I collected data over a span of three months from six patients who complained of general groin area pain associated with hip flexor pain. I designed this as an *a priori* study, because I wanted to begin exploring a technique to treat hip flexor pain. I identified hip flexor pain as a very common local problem in my patient population, especially during athletes' preseasons.

I chose three separate Primal Reflex Release Techniques (first rib reset, sixth and seventh rib reset, and eleventh and twelfth rib reset) to treat the general groin area pain and hip flexor pain, because those techniques reflexively reset structures and muscles that are involved in respiration. I identified dysfunctional respiration as a possible reason for anterior hip flexor pain due to a common characteristic in the patients' sports: quick, short bursts of sprinting followed by an abrupt stop. I theorized that breathing may have become dysfunctional in those patients, because labored breathing patterns during sprinting can lead to a mild hyperventilation state, which leads to muscle pain and fatigue (Bradley & Clifton-Smith, 2005). I decided to use PRRT™ to treat these patients, because dysfunctional breathing can have long-lasting, painful physical and psychological effects throughout the body (Chitow, Bradley, & Gilber, 2014). By treating dysfunctional breathing structures, hip flexor and general groin pain were reduced in less time than had been reported in current literature that described the use of traditional, local treatments. Primal Reflex Release Technique™ uses both local and global physiological assessments and treatments to achieve effective and efficient patient outcomes.

While the case studies in chapters 2 and 3 are evidence of my implementation of AR and RI approaches to patient care, chapters 4 and 5 contain a unique component of the UI DAT program, which is the use of multi-site studies for the DoCPI's final research study requirements. Implementing multi-site studies is valuable for many reasons. For example, collaborating with other athletic trainers from a variety of clinical backgrounds, clinical experiences, and diverse workplace settings allows for a larger sample size for the final research study. It also provides a varied demographic pool of participants. Because the members of my own multi-site research group collected data from different genders, activity levels, body mass indexes (BMI), and ages, the results of our study are more applicable to a larger population of patients than they would be had I worked on my own.

Working collectively as a research team is very representative of the culture of athletic training. I, like most athletic trainers, rarely work in a clinic on my own; generally, I treat patients in busy athletic training clinics, with multiple staff members and large numbers of patients present. Athletic trainers typically share information regarding treatment interventions and the effects of paradigms on both common injuries and special cases. The UI DAT faculty have created another dimension of advanced clinical practice education by providing a way for students to network and sharpen intrapersonal and interpersonal professional relationships through a multi-site research project.

Scholarly development through the multi-site project was attained not only by focusing on the effects of the Mulligan Concept on meniscus tears, but also by examining the reasoning behind the possible theories of efficacy of the “Squeeze” technique. According to the National Athletic Training Association (NATA) Post-Professional Education Council, theoretical understanding is one of the components of graduate education that leads to “scholarly competence, inquiry, and discovery” (Neibert, 2009). While clinical applicability is a goal of evidence-based research, a theoretical basis of the application is needed, initially, to ensure appropriateness and the likelihood of positive outcomes of the intervention. Within Chapter 4 is a literature review that contains information related to the theories of the mechanisms behind the symptoms for meniscus tears and the efficacy of the MC “Squeeze” treatment, itself. Chapter 4 also serves as an introduction to, and background for, the final research study found in Chapter 5. The review of literature includes information regarding meniscus anatomy, meniscus injuries and epidemiology, orthopedic special tests used to assess for meniscal tears, partial meniscectomy data, and an analysis of the Mulligan Concept (MC) and its efficacy in treating patients who present with meniscus tear symptoms. The MC technique specifically outlined in the literature review is the MC “Squeeze” technique. Through researching and critically appraising peer-reviewed studies and manuscripts, I obtained a mastery of the subject matter relating to the MC and the efficacy of interventions used to treat meniscus tears. Based on the outcomes of accepted therapies, I, along with the other members of the multi-site study, determined that a need existed for more conservative treatments that would contribute to positive patient care.

Through a multi-site study, four DAT students, including myself, examined the effect the MC "Squeeze" technique had on patients with meniscus tear symptoms. The preliminary exploration of the technique began in the fall of 2015, with an *a priori* design pilot study. We identified patients who presented with traditional signs and symptoms of meniscal tears, and we treated them using the MC "Squeeze" technique. We collected outcomes for the duration of the semester. Afterward, we analyzed the data and presented the results in a manuscript for the purpose of publication in a peer-reviewed journal. Based on our pilot data and our reflection on those results, we identified inclusion and exclusion criteria and refined our methods for the final research study.

Chapter 5 is entitled, "An Alternative Approach to the Treatment of Meniscal Pathologies: A Randomized, Sham-Controlled Trial of the Mulligan Concept 'Squeeze' Technique." It is a culmination of two years of systematic and rigorous coursework, journeys to Moscow, ID from my clinical practice in Denton, TX, collecting patient outcomes, and numerous manuscript edits. The study was designed to analyze the effects of the MC "Squeeze" technique, as compared to a sham technique, on the symptoms of meniscus tears. The MC was an unknown paradigm to me when I joined the DAT program; now, the RCT study in chapter 5 is the second exploration of the effects of an MC specialized technique for treating meniscus tear symptoms. Given the results of the pilot study and the outcomes of the research team's most recent research, the goal is to provide the medical community with information about a technique that could revolutionize how meniscus tears are managed. Along with the management of the tear itself, the study also includes literary support for diagnostic clinical testing using a battery of special orthopedic tests that are just as accurate as (if not more accurate than) magnetic resonance imaging (MRI).

This DoCPI is representative of the emphasis on the scholarly dissemination of practice-based evidence (PBE) that exists within the UI DAT program. For athletic trainers to be exposed to clinically applicable evidence-based medicine (EBM), and to advance our knowledge, scholarly works need to be produced and shared (Knight & Ingersoll, 1998). Because evidence-based clinical research encourages and fosters scholarship within the profession of athletic training, peer-reviewed journal submissions are highly encouraged in the UI DAT program. All manuscripts in this dissertation will be submitted for publication.

Throughout my student journey, I have wanted to change the perception that other healthcare professionals, patients, and the general public have of me as a medical professional. The previous perception of me as healthcare professional was as a "trainer" who covered practices and games, taped ankles, and handed out ice bags to athletes. As I continue to produce clinically applicable, evidence-based outcomes in a systematic and scholarly way, I will contribute to the development and advancement of athletic training as a profession. And as athletic trainers produce more PBE studies, we will gain more ownership over our body of knowledge.

Collectively, the following chapters, which contain a collection of data and documented evidence of patient outcomes produced in my clinical practice, serve as proof of my development as a clinician. Previously, my athletic training entry-level approach to patient care did not sufficiently and consistently produce quality outcomes; basing my clinical decisions on evidence through outcomes collection, increased theoretical knowledge and understanding in advanced treatment paradigms led to increased positive patient responses to the appropriate interventions and the development of a scholarly practitioner. Sharing my knowledge of advanced paradigms such as the Mulligan Concept, PRRT, and PRT and their effect on my patients with my fellow colleagues also contributed to the advancement of my clinical growth and the growth of the athletic training profession. Educating other athletic trainers on the benefits and necessities of EBP can and will change athletic training for the better as more clinicians implement scholarly and patient-centered care. My journey through the DAT program transformed me into a scholarly practitioner and renewed my passion for the calling that I answered so many years ago.

References

1. Bradley, D., & Clifton-Smith, T. (2005). *Breathe, stretch, and move.* Auckland, New Zealand: Random House.
2. Chaitow, L., Bradley, D., & Gilbert, C. (2014). *Recognizing and treating breathing disorders* (2nd ed.). London: England: Elsevier Health Sciences.
3. Hurley, W.L., Denegar, C.R., & Hartel, J. (2011). *Research methods: A framework for evidence-based clinical practice*. Baltimore, MD: Lippincott Williams & Wilkins.
4. Iams, J. (2005). When reflexes rule: A paradigm in understanding why some patients don't get well. *Advance for Physical Therapists, 16* (3), 41.
5. Iams, J. F. (2012). *Primal reflex release technique home study course*. Retrieved November 17, 2015 from http://www.theprrt.com/online/content/prrthomestudycourse/documents.php.
6. Knight, K.L., & Ingersoll, C.D. (1998). Developing scholarship in athletic training. *Journal of Athletic Training,* 33 (3), 271-274. Retrieved July 8, 2014, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1320437/.
7. Koshy, E., Koshy, V., & Waterman, H. (2011). *Action research in healthcare*. London, England: SAGE Publication Ltd.
8. Meyer, J. (2000). Qualitative research in health care: Using qualitative methods in health related action research. *BMJ: British Medical Journal*, *320*(7228), 178.
9. Neibert, P. J. (2009). Novice to expert practice via postprofessional athletic training education: A grounded theory. *Journal of athletic training, 44*(4), 378-390.
10. Sueki, D.G, Cleland, J.A, & Wainner, R.S. (2013). A regional interdependence model of musculoskeletal dysfunction: Research, mechanisms, and clinical implications. *Journal of Manual and Manipulative Therapy,* 21(2), 90-102.
11. Willis, J., Inman, D., & Valenti, R. (2010). *Completing a professional practice dissertation*. Charlotte, NC: Information Age Publishing.

CHAPTER 2

Using the MyoKinesthetic™ (MYK) System to Treat Bilateral Chronic Knee Pain: A Case Study

By
Valerie Stevenson, Russell Baker, James May, Alan Nasypany

Submitted to The Journal of Chiropractic Medicine for consideration for publication

Key Words:

Chondromalacia, Osteoarthritis, and Manual therapy

Abstract

Context

Chronic knee pain associated with osteoarthritis can take a physical and emotional toll on individuals suffering with the condition. Current treatment recommendations are varied while patients report inconsistent short- and long-term patient outcomes.

Objective

The MyoKinesthetic (MYK™) System is a novel manual therapy treatment system designed to treat postural imbalances that may contribute to both acute and chronic pain. The purpose of this case study was to report the effects of MYK™ on pain, functional ability, and psychosocial well-being of a patient diagnosed with chronic bilateral knee pain associated with osteoarthritis.

Design

Case study

Setting

NCAA athletic collegiate setting

Patient

A 20-year-old female collegiate softball athlete presenting with chronic bilateral knee pain associated with osteoarthritis.

Intervention

The patient received 4 MYK™ treatments, which were based on her postural assessment presentation, over 2 weeks. Treatments 1 through 3 were directed at the S1 nerve root; the fourth treatment was directed at the L4 nerve root.

Main Outcome Measures

Pain level was measured using the Numeric Pain Rating Scale (NRS); patient functional ability was measured using the Patient-Specific Functional Scale (PSFS); and patient impairment, functional limitation, disability, and health-related quality of life was measured through the Disability in the Physically Active (DPA) scale. Pain, function, and quality of life were measured collectively using the Knee injury and Osteoarthritis Outcome Score (KOOS).

Results

The patient experienced clinically significant improvements (MCIDs, MDCs) for all outcome measures, with the exception of the quality-of-life subscale within the KOOS.

Conclusion

The patient in this case study reported decreases in pain and an increase in function during the course of 4 treatments that were administered over the course of 14 days and in accordance to the MYK™ guidelines. Traditional treatment guidelines typically recommend 8 weeks for positive effects to occur. Alternative manual therapy techniques such the MYK™ system may be viable, effective, and efficient treatment options for patients coping with knee OA.

Introduction

The prevalence of osteoarthritis (OA) is widespread across all demographics and is observed in both active and sedentary populations.[1] Moreover, OA of the knee is one of the largest causes of disability on a global scale.[2] Chronic joint pain is the most common symptom of osteoarthritis (OA) and contributes to a wide array of physical and psychosocial disabilities.[3] Distress, dependency, anxiety, depression, and a reduced quality of life are possible side-effects of coping with chronic pain.[4,5]

Causes of chronic knee pain can be obscure and varied, and patients' vague symptoms can lead clinicians to identify a variety of conditions as the potential underlying reason for the pain. Further, a complete and concise diagnosis can be challenging as many structures of the knee are involved as possible culprits for pain.[1] When pain and crepitus are reported with movement along with associated effusion and a popliteal cyst, knee OA is a common diagnosis. Joint space narrowing and valgus and varus deformation of the knee are also common signs of knee OA.[3]

Treatment for OA varies depending on the severity and progression of the disease. Vague diagnoses and a lack of knowledge of the disease's causes and progression often lead to ineffective rehabilitation. Pharmacological treatments designed to treat the symptoms of the disease do not contribute to long-term effective outcomes.[3] Although mobilization with movement (MWM) techniques and a combination of joint mobilization with exercise have both been shown to decrease pain in patients with knee OA,[6,7] the time-commitment for more conservative therapeutic exercise could be extensive and could result is less patient compliance and higher associated medical costs.

The effectiveness of traditional conservative therapy may be lacking because pharmacologic interventions treat the symptoms without addressing the underlying cause of pain, and physical therapy mostly involves addressing range-of-motion (ROM) deficits and muscle strength improvements.[8-10] Addressing ROM deficits and increasing muscle strength of the quadriceps, specifically the Vastus Medialis Oblique (VMO), is a local approach to therapy. The ineffective outcomes of traditional therapy may be a result of not addressing the global and regional interdependent (RI) underlying causes of OA, which are multifaceted and involve more than structures at the knee joint.

The efficacy of MWMs and other joint mobilizations and exercise combinations may lie in the correction of the misalignment or positional fault [6,11,12] of joint surfaces, which are thought to contribute to the development and progression of knee OA.[13] Incorporating a global assessment to determine structural imbalances lead to intervention strategies that address specific local and/or global dysfunctions. Although global and RI interventions may not reverse or cure the disease of OA, correcting regional dysfunctions and imbalances may slow progression of the condition and provide patient psychological wellness by decreasing pain and increasing function. As such, by assessing and treating postural imbalances, the Myokinesthetic (MYK™) System, a novel intervention strategy uses an RI approach to correcting the collective causes contributing to the progression of OA.

The MyoKinesthetic (MYK™) System is a manual therapy technique that decreases and clears nerve nociceptor firing that has occurred due to joint or tissue movement restrictions.[14] Nociceptors are located within joint capsules, muscles, and tendons; they send pain signals to the central nervous system whenever a muscle is in spasm, posture is unbalanced, or there is limited ROM within a joint.[14-15] The targeted outcome of MYK™

treatments is to create bilateral postural balance by treating the neuromuscular system along a specific nerve root, leading to more functional and pain-free movements.[16] Treating specific muscles along the nerve root and balancing posture is theorized to decrease muscle spasm and increase ROM, which quiets nociceptive signals and decreases pain.[14]

Implementation of this system begins by selecting among a list of patient evaluation options. The options could include the following: determining the location of nerve pain and dermatome pathways, identifying painful or weak movements related to specific nerve roots, selecting indicated or named conditions pre-established by the system, performing a postural exam, or measuring range of motion and performing muscle tests on muscles related to a specific nerve root.[16] Based on the results from the evaluation, a specific nerve root level is identified as the nervous system cause of the postural compensation. The clinician treats the patient by massaging/stimulating muscles along the identified nerve root, bilaterally. The clinician moves the joint passively while massaging the involved muscles, and then instructs the patient to move actively while continuing to massage the same muscles.[16] The massage pressure during the treatment is deep enough to stimulate the muscles along the selected nerve root without causing discomfort. The passive movement during the treatment is designed to help clear neuromuscular compensations imbedded in the patient's nervous system. The passive component is followed by an active component to elicit reciprocal inhibition. Active movement helps to re-educate both the muscle in spasm and its antagonist.[16]

The evidence that postural and biomechanical dysfunctions can lead to knee OA,[3,10,12,17] combined with the theoretical design of the MYK™ System, suggests that utilization of the system as an evaluation and intervention paradigm may correct postural dysfunctions[16,18] and improve pain, function, and psychosocial well-being for patients suffering from knee OA. However, there is currently a paucity of evidence regarding the use of the MYK™ System; therefore, the purpose of this case study was to report the effects of the MYK™ System on a patient diagnosed with chronic bilateral knee pain associated with OA.

Case Description

The patient was a 20-year-old collegiate softball pitcher who complained of chronic bilateral knee pain. She did not recall a specific mechanism of injury, but described the pain as constant and gradually worsening over the past 2 years. She reported that her pain was most intense during pitching activities and during weight-bearing cardiovascular conditioning.

An initial clinical orthopedic knee exam was performed to rule out common orthopedic injuries. A visual inspection of the area revealed no edema or ecchymosis, although non-surgical scars were noted. The patient explained those to be the result of sliding during base running. The patient was point tender over the lateral joint line and along the illiotibal band (ITB). Tautness of the ITB was also noted on the exam.

Active ROM for knee flexion (131°) was within normal limits and hyperextension (-4°) was recorded. The patient reported pain at both terminal knee flexion and terminal knee extension. Passive ROM revealed 133° of flexion and -5° of extension as well as pain at the end range. Resistive ROM for flexion was a 3/5 and extension was a 4/5. A Lachman's test and anterior drawer were negative, as were the posterior drawer and valgus and varus stress tests. McMurray's test, Apley's distraction and compression, and Thessaly's at 20° were all positive for a possible meniscus tear. The Clarke's test was also positive, indicating potential patellofemoral dysfunction and the presence of chondromalacia. Because of the possibility of a torn meniscus, the patient was referred to an orthopedic surgeon for further testing and diagnosis.

Magnetic Resonance Imagining (MRI) on the right knee revealed a loose bone fragment in the tendon of the popliteal space, but no meniscus damage, ligament injury, or Baker's cyst was found. The physician diagnosed the patient with chondromalacia and the beginning stages of OA. The diagnosis was based on a combination of the patient's clinical history, physician's clinical exam, MRI findings, and radiographic imagining, which indicated narrowing of the knee joint space and a tibial plateau bone spur (Fig. 1.1). The physician recommended interarticular corticosteroid injections to decrease the patient's pain, and also suggested that she begin a traditional rehabilitation program focused on passive and active stretching of the hamstrings and strengthening of the quadriceps, specifically the VMO. He also recommended the use of 420 mg of naproxen sodium, twice daily or as needed, for 2 weeks. The patient was advised not to squat, lunge, or run up or down stairs.

The patient's therapeutic exercises included 8 weeks of progressive resistive exercises (PREs), quadriceps and hamstring passive stretches, proprioceptive balance training, and standard therapy modalities: cold and heat therapy, ultrasound, and electrical stimulation

(Table 1.1). The patient also began taking the recommend amount of naproxen sodium; however, neither the naproxen sodium, the injection, nor the traditional therapy provided pain relief or an increase in function. The patient's Numeric Pain Rating Scale (NRS) score (mean = 5.22 ± 0.370) and Patient-Specific Functional Scale (PSFS) score (4.2 ± 0.836) did not significantly improve over the 8 weeks of therapy. Due to the ineffectiveness of conservative traditional therapies, MYK™ was considered by the clinician as a possible solution to her pain and dysfunction.

Intervention

Patient-reported outcome measures were collected prior to the MYK™ System's postural assessment. The Numeric Pain Rating Scale (NRS) score, the Patient-Specific Functional Scale (PSFS) score, the Knee Injury and Osteoarthritis Outcome Score (KOOS), and the Disablement in the Physically Active (DPA) scale score were recorded prior the first treatment application to determine baseline scores. Baseline scores prior to the first MYK™ were as follows: DPA (35); NRS (6); and PSFS (5) (Table 1.2). After outcome measures were taken, the MYK™ postural assessment was completed and the patient presented with the following neuromuscular compensations: extended lumbar spine, hip anterior rotation, adduction/upslip, medial rotation, knee extension and external rotation, ankle plantar flexion and inversion, and big toe flexion. Based on the MYK™ guidelines, an S1 nerve root treatment was indicated for the first visit. Each subsequent visit required an additional postural assessment to determine the appropriate nerve root level to treat at follow-up visits.

During the S1 treatment, the joint was treated passively for 8 repetitions, and then the patient was instructed to actively perform the movements for 10 repetitions while the clinician continued to massage the muscle. The treatment was performed bilaterally, irrespective of which side the postural dysfunction was noted on.

The postural assessment was repeated prior to treatment at all follow-up visits. The MYK™ postural assessment on the second and third visits indicated the patient as still needing an S1 treatment; however, on the final visit, the postural assessment revealed postural changes. The majority of the postural imbalances had been resolved and the patient presented with asymmetries in lumbar extension, knee extension, and ankle inversion only. The new postural imbalances indicated that the patient needed an L3 treatment. The L3 treatment was administered in the same fashion as performed in the S1 treatment and patient outcomes were collected.

Outcomes

At the end of each treatment, patient outcome measures were reassessed for current NRS and PSFS activity only. The DPA scale score was collected at intake (baseline), at the 1-week mark, and at discharge; the KOOS was collected at intake (baseline) and discharge only (Table 1.2).

The patient experienced a clinically significant improvement (i.e., MCID) in pain after the first treatment, when her NRS score decreased two points.[19]; however, her PSFS score (5/10) remained the same from pre- to post-treatment. At her second visit, which occurred 3 days after the initial visit, the patient reported further improvement in pain (1/10 on NRS) and improvement in her function (8/10 on the PSFS). Despite the fact that the patient had not received further treatment and denied altering her physical activity or using any other adjunct therapies, the reported improvement on the PSFS exceeded the scale's minimal detectable change (MDC)[20] value.

The second treatment resulted in no immediate changes in pain or function, and the patient did not report any further improvement between the second and third visit. After the end of the third intervention, however, she reported a resolution of her pain and almost full function (9/10 on PSFS). On the patient's fourth and final treatment visit, she arrived with an NRS score of 1 and a PSFS score of 8. After treatment for the L3 nerve root, her pain remained a 1 on the NRS, but her function increased 1 point on her PSFS score.

At discharge, 3 days after her fourth treatment intervention, final outcome measures were collected. The patient's cumulative NRS score was a 1, while her PSFS was a 9. Her discharge scores exceeded previously accepted percent changes seen in similar patient populations. Her pain decreased by 83%, and her function improved 80%. Previous studies measuring the percent change in pain and function following interventions that were aimed at treating knee OA have ranged from 8% to 27% and 10% to 39%, respectively. The patient's DPA scale scores also improved over time and the patient now scored in the healthy normal range.[21] She also exceeded the DPA scale MCID[21] for chronic patients. KOOS scores exceeded the MDC[22] on each of its subsections, with the exception of Quality of Life (QOL), which did not change from intake to discharge (Table 1.2).

Follow-up outcome measures were collected at 16 weeks post discharge. The patient's pain increased (3/10 on the NRS) from discharge to follow-up, and function, as recorded by the PSFS, decreased (4/10). Most subsections scores on the KOOS continued to improve over time. However, pain and sport subsection scores decreased. Although setbacks in pain and sports activity were reported, both of those subsection scores remained higher than scores reported at baseline. The patient's quality of life subsection score, although not changed from baseline to discharge improved 40% from discharge to 16 weeks. The patient's DPA score increased only 2 points, and she remained within the healthy normal patient range (Table 1.2).

Discussion

The patient in this case had undergone 8 weeks of therapeutic exercise with minimal relief. After completing the recommended traditional rehabilitation protocol, her pain level averaged a 5.22 ± 0.370 (out of 10) on the NRS, and her function averaged a 4.2 ± 0.836 (out of 10) on the PSFS. However, when she was treated for 2 weeks using the MYK™ system (a total of 4 treatments), she reported positive outcomes in pain and function (Table 1.2).

Traditional treatment of OA falls under 4 categories: non-pharmacologic (physical therapy), pharmacologic, complementary and alternative, and surgical. Physical therapy for knee OA usually involves active range-of-motion exercises at the knee and hip, strengthening exercises at the knee and hip, stretching of muscles at the knee and hip, and stationary or aquatic cardiovascular exercises.[8,24-26] Strengthening exercises of hip and knee musculature target the quadriceps and, more specifically, the VMO; however, Sharma et. al.[17] reported that developing quadriceps strength does not contribute to a decrease in the progression of OA of the knee. Although the positive effects of physical therapy and exercise[7,23,27] average between 8% and 27% improvement in pain and between 10% and 39% increase in function,[25,26] the duration of the exercise protocols are commonly recommended to be performed 3 times a week over a period of 8 weeks for positive effects to occur.[23] The patient in this case study experienced an 83% decrease in pain and an 80% increase in function over a period of 2 weeks, while similar studies examining the effects of manual therapy and exercise on patients with knee OA reported only 20% to 40% relief of symptoms after 2 to 3 clinical treatments.[8]

Pharmacologic interventions are effective at decreasing pain[28] in short-term time frames, but do not offer long-term, effective solutions.[9] Prior to treatment with MYK™, the patient in this case study received an interarticular injection into her right knee. After the injection, the patient complained of soreness and tightness within the knee joint for 2 days. After the subsequent soreness from the injection had subsided, the patient reported that her pain-level decreased from a 6/10 on the NRS to a 3/10; but the relief from pain only lasted 2 weeks, returning her pre-injection level.

Interarticular injections are more effective than non-steroidal anti-inflammatories (NSAIDS) at addressing pain;[28] however, medications do not correct dysfunctional and abnormally loaded contact areas in the knee, either of which could be the initial, contributing factor for the condition.[10] If normal mechanical arthrokinematics are not restored and balanced in the knee, progression of OA will continue, even though pain perception is decreased with medication.[10] The MyoKinesthetic™ System could potentially correct and balance abnormal joint positioning, leading to decreased pain and increased function.

The patient's MYK™ postural assessment revealed several imbalances involving the hip, knee, ankle, and foot, all of which may have contributed to increases in pain over time, despite therapy, regular NSAID use, and corticosteroid injections. Valgus and varus deformation of the knee, along with misalignment of the hip-knee angle and knee-ankle angle, may have contributed to dysfunctional load distribution of the knee joint on its articulating surfaces.[13] After using the MYK™ interventions, subsequent follow-up postural assessments, which were performed at each patient visit, revealed that prior postural imbalances no longer present.

The clinically significant positive effects of the MYK™ system on this patient were both physical and psychological. Along with the increased physical well-being that comes from a decrease in pain and an increase in function, the patient also improved psychologically as her pain decreased. She began within the chronic pain range of the DPA scale with a score of 35 at her initial visit, but she was discharged within the healthy normal range with a score of 16.[21] The treatment application of the MYK™ system involves exclusive one-on-one patient interaction and contact, beginning with the postural assessment and continuing through the application of the technique. Clinician-patient contact incorporates the components of manual therapy that increase a patient's well-being and may elicit the release of an endorphin that aids in pain relief.[29]

The patient was discharged within the healthy normal limits of the DPA scale [21] and while her KOOS quality-of-life scores did not change from intake to discharge, they did improve over time after discharge. However, all other areas of the KOOS did improve, as did her PSFS score. The sport subscale score on the KOOS was the most improved of all other subsections, as it increased from a 0 to an 80. The substantial improvement on the KOOS sport subsection score corresponds with a high percent (80%) improvement on the PSFS. The patient experienced not only an improvement in sport related activities, but also with her normal daily tasks as her symptoms, pain, and activities of daily living subsection scores improved from baseline to discharge (52% improvement). The patient's percent improvements on her KOOS subscale scores are higher than scores experienced in patients undergoing manual therapy and exercise treatments that have been reported in the literature.[8] Patients who were prescribed manual therapy in conjunction with physical exercise reported a total mean improvement in Western Ontario and McAlester Universities Arthritis Index (WOMAC) scores of 60% on the pain subscale score, 54% on the stiffness subscale score, and 54% improvement on the functional ability scale.[8] The KOOS is an extension of the WOMAC and was designed to measure knee OA symptoms in younger, more active patients than used with the WOMAC. Scores from both of these outcome measures are commonly used collectively in studies accessing effects of interventions on knee OA.[30]

Although this patient experienced set-backs in her pain level and functionality from discharge to the 16 week follow-up, she maintained and exceeded many other discharge scores (Table 1.2). The patient continued unrestricted participation with intercollegiate softball after discharge and follow up outcomes were taken within a few days of beginning preseason training. The decrease in function and increase pain could have been attributed to general delayed onset muscle soreness (DOMS) due to the rigors of returning to intense activity after a 6 week winter break. Deyle et al.[8] observed similar follow-up outcomes between 4 and 8 weeks after discharge on the WOMAC scores of participants treated with manual therapy and exercise. Although this patient's function, as recorded by the PSFS, decreased, KOOS subscale scores for sport and ADLs were well above the level recorded at baseline, supporting the a maintained benefit from the MYK™ treatment. All other subscale scores improved from discharge to the 16 week follow-up, even though the patient played collegiate softball with little restrictions. The Disablement in the Physically Active scale score also remained within the healthy normal limits (Table 1.2).

The case study that is described in this manuscript is the first to examine the effect of MYK™ on knee OA. However, it is a report of outcomes on a single patient. Other, individual factors may influence the positive effects of this technique on patients with a similar presentation. Patients may present with other causes of chronic knee pain that are more progressive than the beginning stages of OA. The patient in this case is a young, physically active athlete; the effects of MYK™ may not be as beneficial on an older, more sedentary population.

Additionally, physical activity improves patient function and decreases pain in patients with OA.[23-27] The patient, in this case study, remained physically active, and only minor adjustments were made to her training, which could have contributed to her positive outcomes. Further research is needed on larger samples of participants, as is an exploration of the effects the technique may have on older and sedentary populations. Because OA is a life-long condition and many patients progressively decline in activity-level due to pain, the short-term outcomes of this study are limiting. The outcomes in this case were collected over a total of 2 weeks, and subsequent follow-up outcomes are needed to determine the long-term efficacy of the treatment.

Conclusion

The patient in this case study experienced clinical significant improvements in pain, function, and psychological well-being by undergoing an MYK™ system treatment protocol. The time from the first MYK™ treatment to discharge was 2 weeks and involved 4 treatments; this is in comparison to the 8-week traditional rehabilitation protocol that the patient was prescribed by her orthopedic doctor, which did not produce significant improvements. While the MYK™ system may not be the solution for all patients with knee OA, it may be a viable treatment option for patients who present with postural asymmetries or who have not consistently found relief from the symptoms of OA with the use of other types of manual therapy, traditional rehabilitation, or pharmacologic intervention.

Tables

Table 1.1: Traditional Rehabilitation Protocol (Performed 3 times per week for 8 weeks)

Mode	Exercise	Prescription
Stretching	Hamstring (supine)	3 x 30 sec
	Quadriceps (standing)	3 x 30 sec
Strengthening	Quadriceps Sets	3 x 25 (5 sec hold)
	Straight Leg Raise (long-sitting)	3 x 15
	Hip Adduction (side-lying)	3 x 15
	Isometric Adduction	3 x (30 sec hold)
	Terminal Knee Ext (supine)	3 x 12
	Terminal Knee Ext (standing)	3 x 12
	Knee Flexion	3 x 15
Balance Training	Baps Board Ankle circumduction (sitting)	50 CW/ 50 CCW
	AirEx Stork Stands (eyes open)	3 x 45 sec

* Moist heat was applied for 15 min prior to stretches

** Ice bags were applied for 20 min after the last strengthen exercise

Table 1.2: Patient-Specific and Region-Specific Outcome Measures

Outcome Measure	Baseline Day 1	Visit 2 Day 4	Visit 3 Day 8	Visit 4 Day 14	†Discharge	†16 week Follow up
DPAS	35	N/A	24**	N/A	16**	18
NRS	6 (pre)	1 (pre)	1 (pre)	1(pre)	1*	3
	4** (post)	1 (post)	0 (post)	1 (post)		
PSFS	5 (pre)	8 (pre)	8 (pre)	8 (pre)	9*	4
	5 (post)	8 (post)	9 (post)	9 (post)		
KOOS						
- Symptoms	25				46.43*	53.75
- Pain	30.75				63.89*	58.50
- ADL	44.25				67.63*	72.25
- Sports	0				80*	30
- QL	31.25				31.25	43.75

*MDC
** MCID
† Non-treatment day

Figures

Figure 1.1: Tibial Plateau Bone Spur

References

1. Sinusas K. Osteoarthritis: diagnosis and treatment. Am Fam Physician. 2012; 85(1):173-181.
2. Cross M, Smith E, Hoy D, Nolte S, Ackerman I, Fransen M, ... Laslett LL. The global burden of hip and knee osteoarthritis: estimates from the Global Burden of Disease 2010 study. *Ann Rheum Dis*. 2014;annrheumdis-2013.
3. Sharma L, Song J, Felson DT, Cahue S, Shamiyeh E, Dunlop DD. The role of knee alignment in disease progression and functional decline in knee osteoarthritis. *JAMA.* 2001;286(2):188- 195.
4. Hootman JM, Helmick CG. Projections of US prevalence of arthritis and associated activity limitations. *Arthritis Rheum.*2006;54:226-229.
5. Woolf AD, Pflegar B. Burden of major musculoskeletal conditions. *Bull World HealthOrgan*.2003;81:646-656.
6. Takasaki H, Hall T, Jull G. Immediate and short-term effects of Mulligan's mobilization with movement on knee pain and disability associated with knee osteoarthritis–a prospective case series. *Physiother Theory Pract.*2013;29(2):87-95.
7. Jansen MJ, Viechtbauer W, Lenssen AF, Hendriks EJ, de Bie RA. Strength training alone, exercise therapy alone, and exercise therapy with passive manual mobilisation each reduce pain and disability in people with knee osteoarthritis: a systematic review. *J. Physiother.*2011;57(1):11-20.
8. 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.*2000:132(3):173-181.
9. Bjordal JM, Klovning A, Ljunggren AE, Slørdal L. Short term efficacy of pharmacotherapeutic interventions in osteoarthritic knee pain: A meta analysis of randomised placebo controlled trials. *Eur J Pain.*2007;11(2):125-138.
10. Brandt KD, Dieppe P, Radin EL. Etiopathogenesis of osteoarthritis. *Rheum Dis Clin North Am.*2008;34(3):531-559.
11. Mulligan BR. Manual Therapy NAGS, SNAGS, MWMS etc. Wellington, New Zealand: Plane View Services, 2004.
12. Vicenzino B, Hall T, Hing W, Rivett D. A new proposed model of the mechaisms of

action of mobilization with movement. In: Vicenzino B, Hall T, Hing W, Rivett D. Mobilisation with movement: The art and the science, pp 75-85. London, England: Churchill Livington, 2011.

13. Tetsworth K, Paley D. Malalignment and degenerative arthropathy. *Orthop Clin North Am.*1994;25:367-377.

14. Marchettini P, Simone DA, Caputi G, Ochoa J. Pain from excitation of identified muscle nociceptors in humans. *Brain Res*.1996; 740(1):109-116.

15. Stein C, Clark JD, Oh U, Vasko MR, Wilcox GL, Overland AC, ... Spencer RH. Peripheral mechanisms of pain and analgesia. *Brain Res Rev*.2009;60(1): 90-113.16.

16. Uriarte M (4th ed.). MyoKinesthetic System Lower Body Training Manual. 2010 Presented: July 21-22, 2016 at The University of Idaho.

17. Sharma L, Dunlop DD, Son J, Hayes KW. Quadriceps strength and osteoarthritis progression in malaligned and lax knees. *Ann Intern Med.*2003;138(8):613-619.

18. Brody K, Baker RT, Nasypany A, May J. The MyoKinesthetic system, part II: treatment of chronic low back pain. *IJATT*.2015;20(5):22-28.

19. Salaffi F, Stancati A, Silvestri CA, Ciapetti A, Grassi W. Minimal clinically important changes in chronic musculoskeletal pain intensity measured on a numerical rating scale. *Eur J Pain*.2004;*8*(4):283–291.

20. Chatman AB, Hyman SP, Neel JM, Binkley JM, Stratford PW, Schonberg A, Stabler M. The patient-specific functional scale: measurement properties in patients with knee dysfunction. *Phys Ther.*1999;7(8*):*820-829.

21. Vela L, Denegar C. The disablement in the physically active scale, part II: the psychometric properties of an outcomes scale for musculoskeletal injuries. *J Athl Train.* 2010*;*45(6):630-641. doi:10.4085/1062-6050-45.6.630.

22. Roos EW, Roos HP, Lohmander LS, Ekdahl C, Beynnon BD. Knee injury and osteoarthritis outcome score (KOOS) – development of a self-administered outcome measure. *J Orthop Sports Phy Ther*.1998;78(2):88-96.

23. Tanaka R, Ozawa J, Kito N, Moriyama H. Efficacy of strengthening or aerobic exercise on pain relief in people with knee osteoarthritis: a systematic review and meta-analysis of randomized controlled trials. *Clin Rehabil*.2013;27(12):1059-1071.

24. Hurley MV. Muscle dysfunction and effective rehabilitation of knee osteoarthritis: what we know and what we need to find out. *Arthritis Care Res.*2003;49(3):444-452.

25. Kovar PA, Allegrante JP, MacKenzie CR, Peterson MG, Gutin B, Charlson ME. Supervised fitness walking in patients with osteoarthritis of the knee: a randomized, controlled trial. *Ann Intern Med.*1992; 116(7): 529-534.

26. Ettinger WH, Burns R, Messier SP, Applegate W, Rejeski WJ, Morgan T…Craven T. A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with knee osteoarthritis: the Fitness Arthritis and Seniors Trial (FAST). *Jama*.1997; 277(1): 25-31.

27. Bennell KL, Hinman RS. A review of the clinical evidence for exercise in osteoarthritis of the hip and knee. *J. Sci. Med. Sport.*2011;14(1):4-9.

28. Bannuru RR, Schmid CH, Kent DM, Vaysbrot, EE, Wong JB, McAlindon TE. Comparative effectiveness of pharmacologic interventions for knee osteoarthritis: a systematic review and network meta-analysis. *Ann Intern Med.*2015;162(1):46-54.

29. Bialosky JE, Bishop MD, Price DD, Robinson ME, George SZ. The mechanisms of manual therapy in the treatment of musculoskeletal pain: a comprehensive model. *Manual Ther.*2009;14:531-538 doi: 10.1016/j.math.2008.09.001.

30. Roos EM, Toksvig-Larsen S. Knee injury and osteoarthritis outcome score (KOOS)-validation and comparison to the WOMAC in total knee replacement. *Health Qual Life Outcomes*.2003;1(1):1.

CHAPTER 3

A Novel Approach to Treating Groin and Hip Flexor Pain
Using Primal Reflex Release Technique:
A Case Series

By
Valerie Stevenson, Alan Nasypany, Russell Baker, James May

Abstract

Study Design

Case Series

Background

Athletes who participate in sports that involve sprinting and multidirectional movements commonly complain of groin/hip flexor pain, in which the nervous system plays a significant role. Patient examinations that fail to include a study of the nervous system's involvement in groin/hip flexor pain may lead to ineffective and lengthy durations of treatments that result in chronic pain. Even after discharge, a patient may suffer from muscle splinting, dysfunctional breathing patterns, and/or continuously excited (also known as "up-related") primal reflexes. Primal Reflex Release Technique (PRRT) is a novel treatment paradigm that was designed to calm primal reflexes from their excitatory state and return the nervous system to its normal status.

Case Description

The 6 patients in this case presented with complaints of acute groin and/or hip flexor pain, sustained during athletic activities. Patients were examined using muscle integrity strength and range-of-motion (ROM) measurements, special orthopedic tests, and breathing and PRRT rib palpation assessments. If, based on the rib palpations, patients were determined to be potential PRRT responders, the technique was performed according to PRRT guidelines. Outcome measures included the Numeric Pain Rating Scale (NPRS), Patient Specific Functional Scale (PSFS), the Global Rating of Change (GRoC) Scale, and the Disability in the Physically Active (DPA) Scale.

Outcomes

All patients demonstrated full resolution of pain, and the change was statistically ($p = 0.001$) and clinically significant for the NRS. All patients returned to optimal function

reported by the PSFS (mean = 9.33 ± .516), and the change was both clinically (MDC) and statistically significant (p = 0.001). All patients exhibited a balanced seated assessment of lateral expansion (SALE), indicating the return of normal breathing functionality. The number of treatments (mean = 1.83 ± 1.16) and the length of time to the resolution of symptoms was minimal (mean = 2.833 ± 2.56 days).

Discussion

By assessing and treating a stressed and sustained excitatory nervous system (exhibited through signs of dysfunctional breathing), patients returned to full activity, without pain, in less than 3 days. After a 2-week follow-up, patients remained functionally pain free. Considering the state of the nervous system in the presentation of musculoskeletal pain and not relegating all treatment options to local muscle structures is beneficial and is needed to determine other pain factors. In this case series, the use of PRRT was effective and efficient in treating patients who complained of hip flexor and/or groin tightness and pain.

Level of Evidence

Level 4

Key Words

Primal Reflexes, Adductor Pain, Breathing Assessment

Introduction/Background

Groin and hip flexor injuries are commonly suffered in sport participation, with higher prevalence among soccer and hockey athletes.[1,2] Because patients who complain of general groin pain also exhibit hip flexor musculature dysfunction, weakness, and pain, the exploration of these injuries in the literature is generally combined.[3,4,5] In a prospective study of 207 athletes who complained of general groin pain, Holmich[2] found iliopsoas-related pain to be the primary clinical entity in 35% of the patients and the secondary entity in 33% of the patients. Thus, 68% of the patient population in the study had groin pain that was accompanied by hip flexor pain, supporting the idea that a patient who complains of general groin pain is likely to report pain in the iliacus, psoas major, and other hip flexor musculature, even if a direct injury did not occur to those specific structures.

The occurrence of groin and hip flexor injuries often results in significant time lost from athletic activity,[1] which is likely partially due to the difficult differential diagnostic procedure associated with this presentation.[4] Patients who complain of general groin pain may experience direct musculoskeletal pain and dysfunction or indirect referred pain pathologies, such as gynecological dysfunction, appendicitis, or sports hernia.[6,7] Referred pain to the groin area most likely is due to the genitofemoral nerve's course through the psoas major, a strong hip flexor.[8] Traditionally, a common mechanism of hip flexor injury is resistive hip flexion or passive hyperextension, usually caused by running, sprinting, or participating in change-of-direction activities.[9,10]

Sprinting during athletic activities is demanding on both the musculoskeletal and respiratory systems.[11] Rapid movement of the diaphragm during inhalation and exhalation results in an excess of carbon dioxide (CO_2) and creates an acidic environment.[11] The body may then respond by interpreting this reaction as respiratory distress, which may trigger dysfunctional diaphragm movement. Normal breathing patterns require input from the sympathetic nervous system[12]; however, if the body anticipates a threat to normal breathing rhythm, it will activate the brain's defense reflex system and signal the primal startle response to initiate an increase in sympathetic nervous system involvement.[13] Receiving constant signals from the sympathetic nervous system could delay a full resolution of symptoms, because the patient remains in a heightened state of neurological stress.[14,15] Patients may also report pain in the hip girdle structures in this scenario, because the psoas major (a primary hip flexor) and quadratus lumborum both run superiorly into the region of the diaphragm.[2]

The neurological stress could also result in a muscle "splinting" mechanism, termed *acute hypertonicity*[14] of the primary and secondary muscles that are responsible for respiration and elicit the nociceptive flexion reflex (NFR). The NFR is a polysynaptic reflex that causes the body to respond to painful stimuli by initiating a withdrawal effect.[16] When the NFR is initiated, it will also activate the body's nociceptive fibers or pain receptors.[16] In the case of a lower extremity injury, such as a groin and/or hip flexor injury, long-lasting protective reflexes that continue to splint the injured area result in the loss of quick, multidirectional movement.[17] Theoretically, if the NFR does not return to its dormant state, chronic pain will be a result (e.g., central sensitization).[18,19]

Determining the correct diagnosis of the primary structures involved in groin and hip flexor pain is critical to the selection of the most appropriate intervention and to the reduction of time lost to injury.[1,20] Because of the possibility of multifaceted origins of groin and hip flexor pain, a multidisciplinary approach to treatment is needed if effective and positive outcomes are to be achieved. Such an approach involves musculoskeletal, neurological, and diaphragm dysfunction assessments. If the state of the nervous system is not assessed and treated (perhaps through calming excited reflexes), local interventions and rehabilitation at the hip region can be extensive in nature and, ultimately, ineffective.

Traditionally, a suspected muscle injury would include rest, ice, compression, and elevation (RICE) for the first 24 to 72 hours.[1,2,4,10] As pain decreases and rehabilitation begins, additional methods are used to return the patient to activity. Some treatment protocols are more effective than others, and many require a significant amount of time and commitment to physical therapy.[2] Passive physical therapy for groin and hip flexor pain consists of manual massage, stretching, and modalities. Historically, such therapy has not contributed to a significant improvement in pain or function.[20] However, an 8- to 12-week active strengthening program, consisting of progressive resistive strengthening exercises, balance training, abdominal strengthening, and eccentric exercises, has been found to produce effective results: Of the patients who completed the active program, 79% returned to their previous non-painful functional level at discharge.[2] Due to the extensive amount of time needed for an active strengthening program to produce positive results, an investigation into an expedited effective treatment technique for treating groin and hip flexor pain is needed. One novel treatment paradigm that has the potential to treat groin and hip flexor pain is Primal Reflex Release Technique (PRRT).

Primal Reflex Release Technique is designed to treat primal reflexes that have been elicited during the startle or withdrawal (nociceptive) response to injury. The treatment technique involves reflexively and reciprocally inhibiting reflexes that are in a constant state of stress and excitement due to injury. The treatment is performed by lightly tapping the facilitated areas' deep tendon reflexes (DTR) for approximately 12 seconds. Tapping these areas stimulates the muscle without causing increased pain.[17,21] The paradigm includes a 1-minute nociceptive exam that evaluates TriggerRegions™. TriggerRegions are areas of hyperesthesia that are sensitive to the smallest amount of pressure. The clinician bilaterally palpates the given areas and determines if the patient responds to touch in any of three ways: a gasp, a groan, or a grimace. Areas that elicit those responses are treated with PRRT and are then reassessed for changes in tightness or tenderness, or for thickened appearance. Individual areas are also assessed independently of the 1-minute nociceptive exam and are treated using specialized PRRT intervention techniques.[17,21]

The following case series was based on the recognition that primal reflexes elicit protective neuromuscular mechanisms after injury and that dysfunctional breathing patterns can result from reflexive muscle splinting of the muscles of respiration causing nervous system dysfunction. The purpose of this case series was to determine the effect of relaxing or down-regulating primal reflexes using PRRT on pain, function, and breathing pattern dysfunction in patients who presented with groin and hip flexor pain.

Case Description

The patients (n = 6) in this case series were all females whose primary complaint was hip flexor and/or general groin area tightness and pain. The patients were all healthy, otherwise, and were physically active in either collegiate or recreational sports at the time of injury (2 = soccer, 1 = gymnastics, 2 = softball, 1 = coaching). Their ages ranged from 18 to 26 years (mean = 21.33 $\pm$ 2.94; Table 2.1). The patients reported various mechanisms of injury during athletic activity; however, all patients described a history of experiencing pain after explosive athletic movements, such as sprinting, jumping, stopping, and changing direction. All injuries were acute in nature and occurred within three days of their initial examination. All patients were evaluated using an extensive injury history and standard hip orthopedic exam (e.g., range of motion (ROM) measurements, strength assessments, special tests). Testing that was done for the express purpose of evaluating adductor and hip flexor musculature involvement included Thomas' test, Ely's test, and resistive adduction and hip flexor muscle break tests. Patients were excluded from this case series if any of the following was suspected during the initial evaluation: fractures, hip pointers, acetabulofemoral joint pathology (e.g., labral tear), or complete muscle rupture. Institutional review board (IRB) approval was given prior to the collection of all patient outcomes, and all patients gave informed, written consent for the sharing of the outcomes of their treatments.

Diaphragm/Breathing Assessments

Patients were classified as PRRT responders based on rib palpation findings acquired during each patient's initial evaluation (Table 2.2). All patients who reported to the researcher's athletic training clinic during the data collection period and who complained of general groin and hip flexor pain were evaluated and treated with PRRT. Rib palpation was performed to assess for rib tenderness and movement restriction in three different areas and to determine the appropriate PRRT treatment to utilize. The three areas were the first and second ribs, the sixth and seventh ribs, and the eleventh and twelfth ribs. Specific palpation locations and directions were determined as instructed in the PRRT paradigm: The first and second ribs were assessed by pressing lightly along the first rib—starting superiorly and moving inferiorly, as well as from the posterior to the anterior of the rib—feeling for tenderness or movement restriction; the sixth and seventh ribs were palpated by lightly strumming along the antero-lateral ribs at 90° to their direction of orientation; the eleventh and twelfth ribs were palpated by strumming superiorly to inferiorly, just lateral to the costo-transverse joint.[17,21] Rib palpations were performed at intake, at the beginning and end of each treatment session, at discharge, and at the 2-week follow-up.

Rib and abdominal movement during breathing were assessed using the seated assessment of lateral expansion (SALE), which is similar to the Modified Manual Assessment of Respiratory Motion (MARM) test.[22] The SALE was performed as the patient was seated comfortably on a plinth, with knees in flexion and hanging from the plinth. The clinician sat behind the patient and placed her hands on the lower lateral rib cage, bilaterally. The hands were open and rested firmly on the ribs without restricting or interrupting the patient's breathing motion. The fifth metacarpal had a horizontal orientation, and the thumbs were approximately vertical. The remaining fingers were below the lower ribs, so the clinician could feel the patient's abdominal expansion.[22,24] The patient was instructed to breathe normally, and an objective assessment of the overall vertical motion relative to the overall lateral motion was recorded as "lateral motion," "vertical motion," or "balanced motion."[22] Diaphragm assessments were performed at intake, at the beginning and ending of each treatment session, at discharge, and at the 2-week follow-up.

Outcomes Scales

The cumulative Numeric Pain Rating Scale (NPRS) was used to assess for the pain level at the current time, the best pain level in the past 24 hours, and the worst pain level in the past 24 hours. The NPRS is an 11-point scale (0-10), where 0 equaled "no pain" and 10 equaled "extreme pain."[24] Current NPRS and the Global Rating of Change (GRoC) Scale scores were taken at the end of each treatment session. The GRoC scale that was used in this case was a 15-point patient-reported perception scale that quantified the extent of improvement or regression experienced by the patient as a result of treatment.[25] The scale begins with -7 (“a very great deal worse”) and ends with 7 (“a very great deal better”).[26]

The Patient Specific Functional Scale (PSFS) was taken to assess the patients’ level of functional activity. Using an 11-point scale (0-10), each patient rated one formerly-functional activity that had become dysfunctional due to injury. A score of 0 represented the patient’s inability to perform the activity; a score of 10 represented a full return to functional activity.[27] The NPRS, PSFS, and GRoC were collected at intake, daily, and at discharge; the GRoC collected at intake was collected immediately after the first treatment intervention.

The Disability in the Physically Active (DPA) scale was taken to assess patient impairment, functional limitation, disability, and health-related quality of life.[28] The DPA scale was collected at intake and discharge, only. All outcome measures were taken at a 2-week follow-up, although only four of the six patients followed up at two weeks post discharge.

Intervention

All rib palpations and treatment sessions were performed with the patient in a supine position. If the patient exhibited first and second rib tenderness, or if the clinician felt any resistance to palpation within the rib space, the patient was instructed to laterally rotate the head to the involved tender side as far as was comfortable and extend the arms straight, so the fingers point toward the toes (Figure 2.1). Next, the patient performed a side-bend to the side where the tenderness was noted (e.g., right side tender: turn head to the right and side bend to the right) and reached down as far as was possible toward the knee (Figure 2.2). The patient returned to the starting position and was instructed to do the same motion as before, but was also told to add a forceful cough with each side-bend repetition (Figure 2.3). Each patient performed three sets of three side-bend/cough repetitions before being reassessed for first and second rib tenderness. If tenderness was reported or resistance was observed, the patient performed three additional sets of three side-bend/cough repetitions and was then reassessed.

If the patient exhibited sixth and seventh rib palpation tenderness on either the right or left side associated with the costo-sternal region, she was instructed to lay in a supine position and flex the arm on the involved side to approximately 100°. The patient was then told to abduct approximately 10° from that position (Figure 2.4). The clinician stood at the involved side and placed a hand on the wrist of the flexed and abducted arm. The clinician then instructed the patient to pull toward the opposite hip in a diagonal fashion (e.g., across the chest) while the clinician resisted the motion (Figure 2.5). The clinician's isometric resistance was provided for five seconds and was then released. The resistance was strong enough that when it was released, the patient was not able to control or stop the horizontal movement to the opposite hip (Figure 2.6). The horizontal arm pull was performed for three sets of three repetitions, and then the sixth and seventh ribs were reassessed for tenderness and movement restriction. If tenderness was reported or movement restriction of the ribs was present, three additional sets of three pulls were performed, and the patient was reassessed again.

If the patient exhibited eleventh and twelfth rib tenderness associated with the PRRT diaphragm-specific reflex release assessment, she was then instructed to turn her head to the uninvolved side and then side bend to the involved side while coughing (e.g., right side tender: turn head to the left and side bend to the right) (Figure 2.7; Figure 2.8). The patient was asked to perform three sets of three repetitions, after which the eleventh and twelfth ribs were reassessed for tenderness and movement restriction. If tenderness was noted, three additional sets of three repetitions were performed.

All treatment interventions were applied in groups of three sets of three repetitions until rib tenderness and/or movement restriction was resolved, at which point the current NRS for groin and hip flexor pain and the PSFS were taken. The number of sets performed by each patient varied during each treatment session: The first session required the most sets (3 ± .894), with Patient 6 requiring the maximum number of 4 sets, and Patient 3 needing the minimum number of 2 sets. If a patient needed additional treatment sessions, 2.33 ± .577 sets were needed for the second treatment session, while the third and fourth treatment session required 3 and 1 set, respectively (Table 2.2).

Outcomes

To evaluate a change in pain and functional scores over time, a one-way repeated measures analysis of variance (ANOVA) was performed using SPSS (SPSS version 23.0; SPSS Inc., Chicago, IL, USA). No significant difference ($F_{2,2}$ = 13.796, Wilk's λ= .068, p = .068, partial η^2 = .932, power = .523) in pain was reported over time from intake (mean = 5.65 ± 1.044) to discharge (mean = .500 ± .289) to the 2-week follow-up (mean = 0). However, the large effect size[29] implied by partial η^2 indicates that over 90% of the positive variability, or improvement in pain, may be attributed to the PRRT treatment intervention. Further, Cohen's *d* effect size calculations indicated a large magnitude of effect of PRRT on pain, with less than 20% of the follow-up scores coinciding with pain scores taken at intake. Although a statistically significant difference was not found in the initial ANOVA, post hoc comparisons were conducted because of the exploratory nature of this study and the risk of a Type II error due to the observed low power (.523); thus, further statistical comparisons were needed to assess any other potential differences across time. Pairwise comparisons revealed a significant difference (mean difference = 5.15 ± .850, p = .027, 95% CI: 1.022, 9.278) between intake and discharge scores, as well as a significant difference between intake and the 2-week follow-up (mean difference = 5.65 ± 1.044, p = .037, 95% CI: .581, 10.719), scores. Moreover, the mean differences between intake and discharge and intake and the 2-week follow-up both exceeded the NRS minimal clinically significant difference (MCID), and were, therefore, clinically significant.[24] No difference was noted from discharge to follow-up (mean difference = .500 ± .289, p = .545, 95% CI: -.902, 1.902), which indicates that patients remained within the same level of pain that they achieved at discharge and did not have a return in symptoms.

A one-way repeated analysis of variance (ANOVA) was conducted to determine a change in functional scores over time. For the PSFS, a significant difference ($F_{1,3}$ = 121.000, Wilk's λ = .024, p = .002, partial η^2 = .976, power = 1) was reported over time from intake (mean = 6.500 ± .577) to discharge (mean = 9.500 ± .577) to the 2-week follow-up (mean = 9.250 ± .500). A large effect size[29] implied by partial η^2 indicated that a significant percentage of the increase in function could have been attributed to PRRT's effects. The Cohen's *d* value (4.76) was also large,[29] indicating a large magnitude of effect of PRRT on function with less than 10% of follow-up scores coinciding with scores reported at intake. Pairwise comparisons revealed specific differences from intake to follow-up (mean difference = -2.75 ± .250, p = .005, 95% CI: -3.964, -1.536) and from intake to discharge (mean difference = -3.00 ± .203, p = .002, 95% CI: -3.000, -1.235); no difference was seen from discharge to follow-up (mean difference = .250 ± .250, p = 1, 95% CI: -.964, 1.464). Mean differences in scores between intake and discharge and between intake and follow-up exceeded the minimal detectable change (MDC)[27] for the PSFS, which added clinical meaningfulness to the statistical differences observed in outcomes scores.

A repeated measures ANOVA was also performed to assess for differences in scores measuring patient impairment, functional limitation, disability, and health-related quality of life as indicated by the DPA Scale. A significant difference ($F_{2,2}$ = 9.332, Wilk's λ = .097, p = .097, partial η^2 = .903, power = .404) was not found for patients' DPA Scale scores. Post hoc comparisons were conducted because of the exploratory nature of this study and the risk of a Type II error due to the observed low power (.404); thus, further statistical comparisons were needed to assess any other potential DPA Score differences across time. Pairwise comparisons revealed significant differences between intake and discharge scores (mean difference = 8.250 ± 1.652, p = .046, 95% CI: .227, 16.273) and between intake scores and follow-up scores (mean difference = 7.250 ± 1.377, p = .040, 95% CI: .563, 13.937). No difference (mean difference = -1.000 ± .707, p = .757, 95% CI: -4.434, 2.434) was observed between discharge scores and follow-up scores. Additionally, all patients were discharged within the normal, healthy ranges of a score that is less than 23 for the DPA scale.[28] The lack of significant differences in DPA Scales scores from the initial ANOVA may be attributed to baseline scores being relatively low compared to the healthy range at intake (i.e., patients 1 and 5 began with scores below the cut-off score of 23) and the small sample size.

The patient-reported perception of the extent of improvement after PRRT treatment, which was measured by the GRoC, also suggests effective treatment; mean scores after the first treatment application (5.00 ± 2.097) to discharge (6.66 ± .516) to the 2-week follow-up (7 ± .000) increased. After the first treatment application, most GRoC scores (67%) were reported as "quite a bit better" or higher. Additionally, at the 2-week follow-up, all of the patients reported a GRoC score coinciding with "a very great deal better", which suggest the patients perceived a large level of improvement over the course of treatment.

Discussion

Treatment and rehabilitation for hip flexor and groin injuries may last 8 to 12 weeks and may not result in positive patient outcomes.[20] In the Holmich et al.[20] study, patients who underwent passive traditional therapy (passive stretching, manual massage, ultrasound, and electrical stimulation) after complaining of adductor pain did not report significant improvements in symptoms and function four weeks after discharge from therapy. The patients in this study had previously received up to 12 weeks of therapy and were questioned 4 weeks after discharge about any return of symptoms; only 4 of the 30 participants reported an absence of adductor pain or dysfunction.[20] When compared with reported outcomes of extensive rehabilitation and treatment protocols, time commitment for patients in this case series was minimal: All patients were treated and discharged in approximately two treatment sessions (mean = 1.83 ± 1.16) that took place over a course of less than three days (mean = 2.833 ± 2.56). After being treated with PRRT, patients reported a complete resolution of pain and return of function at discharge and no return of symptoms at the 2-week follow-up. Half of the patients in this case series needed only one treatment session to experience significant pain improvements. Moreover, all patients reached minimal clinical improvements in their NRS and PSFS scores by discharge, and the scores remained improved within the 2-week follow-up period. The subjective GRoC scale responses in this case series are similar to outcomes from the Holmich et al.[20] study, in that all participants reported feeling either "better" or "much better" at discharge; however, the active rehabilitation program in the Holmich et al. study lasted much longer (median = 18.5 weeks) than the treatment period required to produce resolution in the current cases.

In this case series, the positive patient outcomes from using PRRT may have occurred because the treatment is aimed at treating nervous system dysfunction.[17,21] The reported ineffective and extensive treatments of groin and hip flexor injuries could be the result of failure to reset (or down-regulate) reflexes that remain in a constant state of excitement (or up-regulation) after injury. Not assessing and addressing nervous system involvement could lead to chronic groin and hip flexor pain, and, due to movement compensations developed because of pain, place the patient at risk for further injury to the hip girdle musculature. In chronic groin pain cases, symptoms are often complex and uncharacteristic. After sustaining injury to the groin area, patients have reported areas of pain migrating, over time, to the medial thigh and rectus abdominis.[30] Pain signals referred to those areas could be the result of an active nociceptive reflex contributing to central sensitization, and not necessarily the result of soft tissue damage.

An increased response to various stimuli, such as mechanical pressure, chemical substances, light, sound, cold, heat, and electricity, are all outcomes of the processes involved in central sensitization[31]; thus, patients' sensitivity to rib palpation during the PRRT evaluation could indicate a presence of an over-reactive central nervous system. In addition, assessment of breathing function could also offer insight into the cause of referred groin and hip flexor pain, because sympathetic nerve outflow to skeletal muscles varies during normal respiratory cycles. Sympathetic output decreases during inspiration and then rises to its peak during the end stage of expiration[15]; thus, a disruption in normal breathing patterns would affect the normal influence of the nervous system and could render it dysfunctional.[32] The common mechanism of injury for all patients in this case series involved running or sprinting at their maximum speed, which can cause labored breathing that affects diaphragm movement. Breathing assessments that tested the connection of the psoas major to the diaphragm were performed in these cases, due to recognition of a fascial link of the psoas major and the diaphragm. Fascial connections between the psoas major and the diaphragm occur though the medial arcuate ligament, which is a continuation of the superior psoas fascia that connects superiorly to the diaphragm.[33] Additionally, the mechanism by which abnormal or dysfunctional breathing patterns would affect the psoas major (a hip flexor) is through the association of the nervous system's regulation of intra-abdominal pressure and lumbopelvic postural stability. While the nervous system regulates intra-abdominal pressure and lumbopelvic stability, the diaphragm, pelvic floor, and transverse abdominis maintain those components.[34,35]

The integrated spinal stabilization (ISSS) described by Kolar is made up of the diaphragm, deep cervical flexors and extensors, thoracic extensors, pelvic floor, and all sections of the abdominal and spinal extensors in the lumbar regions.[37] Spinal stability and the multifaceted movements involved in athletic performance are enhanced because of the dual roles of a properly functioning diaphragm.[35,37] Because the psoas major and the diaphragm share a fascial attachment in the lumbar spine,[33] when the ISSS is functional, hip flexor muscles (such as the psoas major) will most likely function optimally, as intended, without affecting other structures.[37] However, if the ISSS becomes dysfunctional, due, perhaps, to a chronically excited nervous system as exhibited through primal reflex initiation, the resulting pull or muscle "splinting" (acute hypertonicity) of the psoas major will affect lumbar spine function.[38] A deficient ISSS may cause increased movement of the other muscles associated with the ISSS, leading to overuse and strain[37]; therefore, in the aforementioned example, the "splinting" mechanism of the psoas major could actually be the tightness and lack of full ROM described by many patients who complain of groin and hip flexor pain.

Anatomical relationships also support the efficacy of PRRT treatments. Internal and external obliques (trunk rotators) merge with the diaphragm and lower ribs and can influence respiration.[22] The diaphragm-reset techniques used on the patients in my case series involved turning the head and coughing while bending laterally. The lateral bend elicits movement of the thoracic spine and is initiated by the obliques. Involved with the act of coughing are the diaphragm and accessory breathing muscles, such as the sternocleidomastoid, subclavius, and omohyoid.[22] The act of turning and coughing while laterally bending reflexively contracts these primary and accessory muscles of respiration and causes the diaphragm to reset and regulate as normal. Theoretically, this corrects a dysfunctional sympathetic nervous system, in the process.

Primal Reflex Release Technique could be used as a primary and as a secondary mode of treatment and rehabilitation for groin and hip flexor pain. PRRT could also be used, initially, to calm nociceptive reflexes and impulses as a singular treatment option. Furthermore, it could be used in conjunction with traditional strengthening and ROM active physical therapy to complement the rehabilitation. To maintain the functioning diaphragm and prevent the patient from experiencing a reoccurrence of pain and loss of function at the groin and hip flexor area, corrective breathing strategies that regulate the sympathetic nervous system of the diaphragm could also be implemented.

By assessing and addressing the nervous system through primal reflex investigation and by correcting breathing patterns, patients in this case series returned to pain-free functional movement. The physiology behind a true muscle tear or strain would not be present in these cases. Had an actual tear occurred, resetting the nervous system through diaphragm regulation would not have resulted in tissue healing over such a short period of time. No other treatments were performed on these patients during the active treatment period, nor were any performed during the 2-week follow-up period. All patients participated in full activities and were not limited in any activities of daily living or sport-related activities.

Several limitations were present within this case series. The clinician administering the treatment was a novice in the uses of PRRT and was trained through an introductory home study course. Currently, six levels are available and offer more advanced techniques as the levels increase. Moreover, the clinician only treated the diaphragm based on rib palpations when other techniques within the paradigm may have led to more improved results. Assessing patients using the PRRT 1-minute nociceptive exam and treating all areas in excitatory stress could have decreased the time to resolution in these patients, as well.

Conclusion

The patients in this case series all presented with groin and/or hip flexor pain, tightness, and dysfunction. All patients were examined to ensure that no other underlying causes of pain were present. Breathing assessments were performed in conjunction with orthopedic special tests, muscle-strength tests, and range-of-motion assessments. Patients were treated using the Primal Reflex Reset Technique, based on their response to rib palpations. All patients in this case series experienced a resolution of pain and returned to optimal functional activity without the use of traditional therapeutic exercise or local treatment to hip muscles. Breathing function also returned to functional status by the end of discharge, and the patients remained pain-free and functional up to two weeks after discharge, with no further intervention or activity restrictions.

Tables

Table 2.1: Intake Outcome Scores/Special Tests

Patient	Age	Sport	DPA	NRS	PSFS	GRC*	Ely's Test	Thomas Test	Tenderness/Restriction	SALE	Intervention
1	20	Soccer	31	7	6	5	-	+	1st rib (**same side)	Vertical	1
2	19	Gymnast	22	2.6	7	6	-	+	6th and 7th ribs (**same side	Vertical	3
3	23	Rec SB	35	6	6	7	-	+	1st rib (**opposite)	Vertical	1
4	18	Softball	28	2.6	4	3	+	+	1st rib (**opposite)	Lateral	1
5	26	Coach	21	4	7	7	+	+	11th and 12th (bilateral)	Balanced	2
6	22	Soccer	25	7	7	2	-	+	1st rib (**same side)	Lateral	1

*After first treatment

** side as hip complaint18

Intervention: 1 – Cough to same side as rib tenderness, 2 – Cough to opposite side of rib tenderness, 3- Horizontal arm pull

Table 2.2: Treatment sets

Patient	1st treatment	2nd treatment	3rd treatment	4th treatment
1	3 sets	2 sets	n/a	n/a
2	3 sets	n/a	n/a	n/a
3	2 sets	n/a	n/a	n/a
4	4 sets	2 sets	n/a	n/a
5	2 sets	n/a	n/a	n/a
6	4 sets	3 sets	3sets	1 set

Table 2.3: Discharge Outcomes Scores/Special Tests

Patient	DPA	NRS	PSFS	GRC*	Ely's Test	Thomas Test	Tenderness	SALE	Days	#Treatments
1	19	1	9	6	-	-	None	Balanced	5	2
2	16	0	10	7	-	-	None	Balanced	1	1
3	25	0	9	7	-	-	None	Balanced	1	1
4	24	0	9	6	-	-	None	Balanced	2	2
5	18	0	9	7	-	-	None	Balanced	1	1
6	20	1	10	7	-	-	None	Balanced	7	4

Table 2.4: Two week follow up Outcome Scores/Special Tests

Patient	DPA	NRS	PSFS	GRC*	Ely's Test	Thomas Test	Tenderness/Restriction	SALE
1	21	0	9	7	-	-	-	Vertical
2	18	0	10	7	-	-	-	Balanced
3	26	0	9	7	-	-	-	Balanced
6	19	0	10	7	-	-	-	Lateral

*Overall from intake to 2-week follow up

Figures

Figure 2.1: Head turn to same side

Figure 2.2: Side Bend

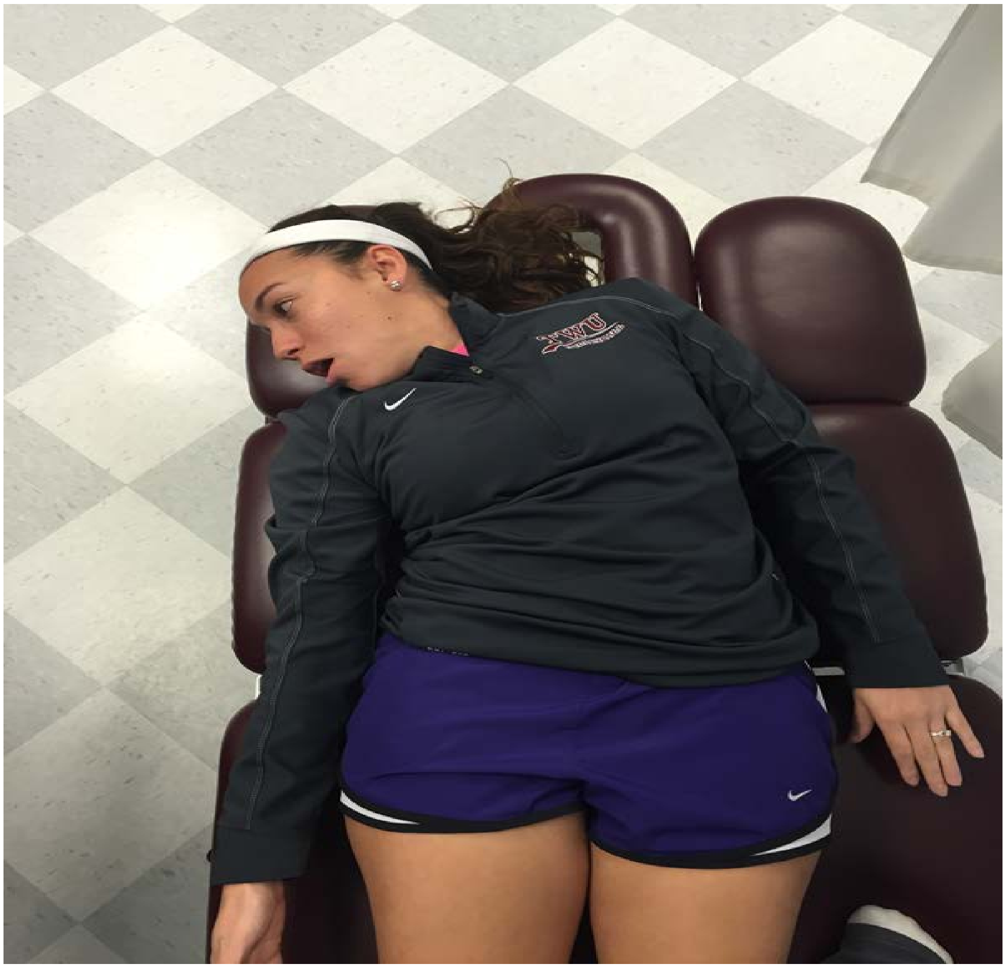


Figure 2.3: Side Bend and Cough

Figure 2.4 Horizontal Arm Raise

Figure 2.5: Clinician Hand Placement

Figure 2.6: Completion of Arm Drop

Figure 2.7: Side bend to opposite side

Figure 2.8: Cough to opposite side

References

1. Anderson K, Strickland, SM, Warren R. Hip and groin injuries in athletes. *Am J Sports Med*. 2001;29:521-533.
2. Nicholas SJ, Tyler TF. Adductor muscle strains in sport. *Sports Med*. 2002;32:339-344.
3. Hölmich, P. Long-standing groin pain in sports, a “clinical entity” approach: a prospective study of 207 patients. *Br J Sports Med*. 2007;41: 247-252.
4. Quinn A. Hip and groin pain: physiotherapy and rehabilitation issues. *Sports Med Open.* 2010;4:93-107.
5. Werner J, Hägglund M, Waldén M, Ekstrand J. UEFA injury study: a prospective study of hip and groin injuries in professional football over seven consecutive seasons. *Br J Sports Med*. 2009;43:1036-1040.
6. Järvinen M, Orava S, Kujala UM. Groin pain (adductor syndrome). *Oper Techn Sport Med*. 1997;*5*(3), 133-137.
7. Hackney RG. The sports hernia: a cause of chronic groin pain. *Br J Sports Med.* 1993;27(1): 58-62.
8. Rab M, Dellon AL. Anatomic variability of the ilioinguinal and genitofemoral nerve: implications for the treatment of groin pain. Plast Reconstr Surg.2001;108(6):1618-1623.
9. Caudill P, Nyland J, Smith C, Yerasimides J, Lach J. Sports hernias: a systematic literature review. *Br J Sports Med.* 2008;42: 954-64.
10. Järvinen TA, Järvinen TL, Kääriäinen M, Kalimo H, Järvinen M. Muscle injuries biology and treatment. *Am J Sports Med.* 2005;33(5): 745-764.
11. Sakamoto A, Naito H, Chow CM. Hyperventilation as a strategy for improved repeated sprint performance. *J Strength Cond Res.* 2014;28(4), 1119-1126.
12. Seals DR, Suwarno NO, Joyner MJ, Iber C, Copeland JG, Dempsey JA. Respiratory modulation of muscle sympathetic nerve activity in intact and lung denervated humans. *Circ Res*. 1993;72:440–454.
13. Lang PJ, Wangelin BC, Bradley MM, Versace F, Davenport PW, Costa VD. Threat of suffocation and defensive reflex activation. *Psychophysiology.* 2011;48(3):393-396.

14. Miller H. The role of autogenic inhibition in the reduction of muscle splinting. *J Can Chiropr Assoc.* 1982;26(1): 21-30.

15. Campbell JN, Meyer RA. Mechanisms of Neuropathic Pain. *Neuron*. 2006;52(1):77-92. doi:10.1016/j.neuron.2006.09.021.

16. Skljarevski V, Ramadan NM. The nociceptive flexion reflex in humans–review article. *Pain.* 2002;96(1): 3-8.

17. Iams JF. When reflexes rule: a new paradigm in understanding why some patients don't get well. *Advance for Physical Therapy and Rehab Medicine.* 2005;16(3):41.

18. Arendt-Nielsen L, Brennum J, Sindrup S, Bak P. Electrophysiological and psychophysical quantification of temporal summation in the human nociceptive system. *Eur J Appl Physiol.* 1994;68(3):266-273.

19. Kiernan BD, Dane JR, Philips LH, Price DD. Hypnotic analgesia reduces RIII nociceptive reflex: further evidence concerning the multifactorial nature of hypnotic analgesia. *Pain.* 1995;60:39–43.

20. Holmich P, Uhrskou P, Ulnits L, Kanstrip I, Nielsen, MB, Bjerg AM, Krogsgaard K. Effectiveness of active physical training as treatment for long-standing adductor-related groin pain in athletes: randomized trial. *Lancet*. 1999;353:439-443. http://doi: 10.1016/SO140-6736(98)03340-6.

21. Iams JF. Primal Reflex Release Technique. What is the primal reflex release technique for pain relief? Published 2012. Retrieved November 1, 2015 from http://www.theprrt.com/what-is-the-primal-reflex-release-technique-for-pain-relief.php.

22. Chaitow L, Bradley D, Gilbert C. Recognizing and Treating Breathing Disorders. London, England: Elsevier Ltd., 2014.

23. Courtney R, Cohen M, Reece J. Comparison of the Manual Assessment of Respiratory Motion (MARM) and the Hi Lo Breathing Assessment in determining a simulated breathing pattern. *Int J Osteopath Med*. 2009;12(3):86-91.

24. Salaffi F, Stancati A, Silvestri CA, Ciapetti A, Grassi W. Minimal clinically important changes in chronic musculoskeletal pain intensity measured on a numerical rating scale. *Eur J of Pain-London.* 2004;8:283–291.

25. Kamper SJ, Maher CG, Mackay G. Global rating of change scales: a review of strengths and weaknesses and considerations for design. *J Man Manip Ther*. 2009;17(3): 163-170.

26. Jaeschke R, Singer J, Guyatt GH. Measurement of health status. Ascertaining the minimal clinically important difference. *Control Clin Trials.* 1989: 407-15.

27. Chatman AB, Hyams SP, Neel JM, Binkley JM, Stratford PW, Schomberg A, Stabler M. The Patient-Specific Functional Scale: measurement properties in patients with knee dysfunction. *Phys Ther.* 1997;77(8):820-829.

28. Vela L, Denegar C. The Disablement in the Physically Active Scale, Part II: The psychometric properties of an outcomes scale for musculoskeletal injuries. *J Athl Train*. 2010;45:630-641. http//doi:10.4085/1062-6050-45.6.630.

29. Cohen J. Statistical Power Analysis for the Behavioral Sciences. New York, NY: Routledge Academic, 1988.

30. Järvinen M, Orava S, Kujala UM. Groin pain (adductor syndrome). *Oper Techn Sports Med*. 1997;*5*(3):133-137.

31. Nijs J, Meeus M, Van Oosterwijck J, Ickmans ., Moorkens G, Hans G, De Clerck LS (2012). In the mind or in the brain? Scientific evidence for central sensitisation in chronic fatigue syndrome. *Eur J Clin Invest*; 42(2): 203-212.

32. Croix CMS, Satoh M, Morgan BJ, Skatrud JB, Dempsey JA. Role of respiratory motor output in within-breath modulation of muscle sympathetic nerve activity in humans. *Cir Res*. 1999; 85(5): 457-469.

33. Bogduk N, Pearcy M, Hadfield G. Anatomy and biomechanics of psoas major. *Clin Biomech.*1992; 7:109-119.

34. Gardner-Morse MG, Stokes IAF. The effects of abdominal muscle co-activation on lumbar spine stability. *Spine.* 1998;23(1): 86-92.

35. Hodges PW, Gandevia SC. Changes in intra-abdominal pressure during postural and respiratory activation of the human diaphragm. *J Appl Physiol*. 2000;89(3):967-976.

36. Kolar P. Facilitation of Agonist-Antagonist Co-activation by Reflex Stimulation Methods In: Craig Liebenson: *Rehabilitation of the Spine – A Practitioner's Manual* (2nd ed.). Lippincott Williams & Wilkins, 2000, 531-565.

37. Hodges PW, Heiinen I, Gandevia SC. Postural activity of the diaphragm is reduced in humans when respiratory demand increases. *J Physiol.* 2001;537(Pt 3): 999-1008.

38. Frank C, Kobesova A, Kolar P. Dynamic neuromuscular stabilization & sports rehabilitation. *Int J Sports Phys Ther*. 2013; 8(1):62-73.

CHAPTER 4

Review of Literature

Introduction

Meniscal lesions are the second most common knee injury in sports (Majewski, Susanne, & Klaus, 2006), and as many as 50% of orthopedic surgeries performed in the United States involve the meniscus (Englund et al., 2010). Tears in the meniscus are more prevalent among males than females, both in adults and adolescents (Drosos & Pozo, 2004; Shieh, Bastrom, Roocroft, Edmonds, & Pennock, 2013), with tears among adolescent populations occurring almost exclusively during sports-related activities (Drosos & Pozo, 2004; Shieh et al., 2013). The current standard of care for treating meniscal tears is surgical intervention. Surgical options for the treatment of meniscal tears include partial meniscectomy, meniscal repair, and meniscus transplant (Brophy & Matava, 2012); when diagnostically indicated (e.g., a tear in the outer vascular zone), arthroscopic surgical repair is generally the first choice due to the salvation of meniscal tissue which delays the onset of osteoarthritis (OA; Getgood & Robertson, 2010). Osteoarthritis of the knee has been associated with meniscal tears, especially in those treated with surgical meniscectomies (Snoeker, Bakker, Kegel, & Lucas, 2013; Englund, 2008).

Patients who undergo any type of meniscal surgery are at a significant risk for requiring a subsequent surgery (Paxton, Stock, & Brophy, 2011). Failure rates of meniscal surgical interventions range from 9% to 49% (Getgood and Robertson, 2010; Hwang & Kwoh, 2014; Katz et al., 2013; Lyman et al., 2013; Nepple, Dunn, & Wright, 2012; Peters & Wirth, 2003; Pujol Barbier, Boisenroult & Beaufils, 2011; Vundelinckx, Vanlauwe, & Bellmans, 2014). Additionally, no difference was found when comparing the outcomes of meniscectomy to those of sham surgery (Sihvonen et al., 2013) or conservative rehabilitation (Herrlin, Hallander, Wange, Wiendenhielm, and Werner, 2007).

The recommendation to exhaust conservative treatment options prior to seeking surgical intervention is commonly reported in the literature (Hwang & Kwoh, 2014; Katz et al., 2012; Herrlin, Hallander, Wange, Wiedenhielm, and Werner, 2007; Bin, Kim, & Shin, 2004). Conservative treatment may involve various manual therapy techniques that are effective in resolving symptoms and increasing function (Englund et al., 1992). To improve the treatment of meniscal pathology it is important to understand that the conservation of meniscal tissue is critical. Research into alternative methods of retaining meniscal tissue post meniscal tears is warranted.

Basic Anatomy and Function of the Meniscus

The medial “C-shaped” meniscus covers 50% of the medial tibial plateau surface area and is wider at the posterior horn than the anterior (Rath & Richmond, 2000). The periphery of the medial meniscus attaches firmly to the joint capsule and to the medial collateral ligament (MCL) at its midsection via the deep medial collateral ligament fibers (Lee & Fu, 2000). The deep medial collateral ligament restricts the medial meniscus from excessive motion (Masouros, McDermott, Amis, & Bull, 2008). The lateral “O-shaped” meniscus accounts for 70% of the surface area on the lateral tibial plateau (Rath & Richmond, 2000). The lateral meniscus is only loosely attached to the joint capsule and has no attachment to the lateral collateral ligament (LCL), allowing for greater mobility during activity (Rath & Richmond, 2000). Also contributing to the mobility of the lateral meniscus are fibers of the popliteal tendon that insert along the lateral meniscus at the posterolateral corner (Rath & Richmond, 2000).

Tibial attachment sites of the medial and lateral menisci exist anteriorly adjacent to the anterior cruciate ligament (ACL) and posteriorly adjacent to the posterior cruciate ligament (PCL; Greis, Bardana, Holmstrom, & Burks, 2002). The anterior horns of the medial and lateral menisci are connected by the transverse ligament (Fox, Bedi, & Rodeo, 2012). The lateral meniscus is supported by two meniscofemoral ligaments: the ligament of Humphry, or anterior meniscofemoral ligament, and the ligament of Wrisberg, or the posterior meniscofemoral ligament (Greis et al., 2002; Poynton, Javadpour, & Finegan, 1997). The occurrence of these ligaments is highly variable.

Microstructure

The meniscus is composed of approximately 70% water and additional dry substance that includes fibrochondryte cells and an extracellular matrix (McDevitt, Cahir A., Webber, 1990; Renstrom & Johnson, 1990). The dry substance is 60-75% collagen (McDevitt, Cahir A., Webber, 1990; Renstrom & Johnson, 1990), 90% of which is type I collagen (McDevitt, Cahir A, Webber, 1990). The concentration of collagen in the meniscus increases from birth until the age of thirty and remains fairly consistent until age of 80, at which point it begins to decline. Elastin and non-collagenous proteins also exist in the meniscus in small quantities (0.6% and 8-13% of the dry substance; McDevitt, Cahir A., Webber, 1990).

The fibers on the surface of the meniscus are organized in a multi-directional mesh-like fashion. The meshed network functions to dissipate shear stress exerted on the surface by the femoral condyles (Greis et al., 2002). Deeper fibers are orientated circumferentially, contributing to the meniscus' ability to withstand weight-bearing loads from the femur. Radial fibers run perpendicular to the circumferential fibers, and both are crimped at rest and elongate under tension (Renstrom & Johnson, 1990). The radial fibers add structural integrity to the meniscus and prevent longitudinal tearing during stress (Renstrom & Johnson, 1990). While the circumferential fibers expand to allow for the dispersal of load, the radial fibers act as ties that prevent excessive expansion.

Vascular Anatomy

The meniscus receives its blood supply from the superior and inferior portions of the medial and lateral genicular arteries via premeniscal capillary plexuses (Arnoczky & Warren, 1982). Radial branches from these plexuses extend into the menisci and travel a short distance toward the center of the joint, ending in terminal capillary loops (Arnoczky & Warren, 1982). The well vascularized periphery is referred to as the "red zone." The narrow transitional region is the "red-white zone," or "pink zone," and the inner most region of the meniscus, which is completely avascular, is the "white zone" (Rodkey, 2000). The depth of vascularity from the periphery ranges from 10-30% in the medial meniscus and 10-25% in the lateral. The lateral meniscus is also avascular at the popliteal hiatus (Arnoczky & Warren, 1982). The zones are useful in describing the location of tears and discussing healing potentials. Tears in the red zone have a potential for healing, while those in the white zone do not (Fox, Bedi, & Rodeo, 2012).

Infants are born with an abundance of blood supply throughout the menisci. Newborn vascularity ranges from 50% (Renstrom & Johnson, 1990) to 100% (Greis et al., 2002). By nine months, the inner portion loses most of its vascularity and continues to diminish until it reaches the reported averages at approximately 10 years of age (Greis et al., 2002). Because the avascular portions of the meniscus depend on diffusion from the synovial fluid for nutrition (Fox, Bedi & Rodeo, 2012; Greis et al., 2002; Renstrom & Johnson, 1990), movement at the knee and weight-bearing activities may aid vascular supply due to mechanical pumping and compression of the menisci (Fox, Bedi & Rodeo, 2012).

Neuroanatomy

The neural supply of the meniscus follows the same path as the vascular anatomy. Local nerve branches have been reported to stem from the posterior and medial articular nerves (Lee & Fu, 2000; Wilson, Legg, & McNeur, 1969). The premeniscal region of the joint capsule is highly innervated, and branches from these nerves extend into the peripheral third of the meniscus as myelinated and unmyelinated free nerve endings. The nerve fibers are more abundant in the anterior and posterior horns of the menisci than they are in the body (Renstrom & Johnson, 1990). Nerve fibers become less dense in the middle third of the meniscus and are absent in the inner third, insertion sites, and at the meniscofemoral ligaments (Lee & Fu, 2000, Wilson et al., 1969). The majority of nerve fibers at the menisci are reported to be mechanoreceptors, providing proprioceptive feedback during extreme end ranges of motion (Fox, 2007; Greis et al., 2002).

Sensory neuromapping, charting areas of the menisci which detect painful versus pain-free sensation, produced similar findings to those previously reported on neural anatomy of the knee (Dye, Vaupel, & Dye, 1998). Mapping of the internal structures of the knee has been conducted without intraarticular anesthesia. Palpation of the peripheral regions of the menisci via arthroscopic probing produced slight to moderate discomfort, while palpation of the inner rims produced only an awareness of the palpation without pain (Dye et al., 1998). Palpation of the synovium, capsule, and retinacula produced the second highest amounts of pain and discomfort (Dye et al., 1998).

Function & Biomechanics

The menisci play a functional role in optimizing articular congruency (Fox, Bedi & Rodeo, 2012; Lee & Fu, 2000; Masouros et al., 2008; Rath & Richmond, 2000; Renstrom & Johnson, 1990); load transmission (Fox Bedi & Rodeo, 2012; Greis et al., 2002; Lee & Fu, 2000; Rath & Richmond, 2000; Renstrom & Johnson, 1990); shock absorption (Fox, Bedi & Rodeo, 2012; Greis et al., 2002; Lee & Fu, 2000; Masouros et al., 2008); stability (Fox, Bedi & Rodeo, 2012; Lee & Fu, 2000; Masouros et al., 2008; McDermott, Masouros, & Amis, 2008; Rath & Richmond, 2000); proprioception (Fox, Bedi & Rodeo, 2012; Greis et al., 2002); joint lubrication (Fox, Bedi & Rodeo, 2012; Lee & Fu, 2000; Rath & Richmond, 2000;

Renstrom & Johnson, 1990); and nutrition (Fox, Bedi & Rodeo, 2012; Lee & Fu, 2000; Rath & Richmond, 2000; Renstrom & Johnson, 1990). Limited evidence exists to support conclusions about the function of the meniscus in joint lubrication and nutrition, but these functions are reported as a secondary effect at the meniscus during weight-bearing activities (Renstrom & Johnson, 1990). Additionally, the existence of mechanoreceptors within the meniscal horns and attachments sites may suggest that the meniscus plays a functional role in joint proprioception (Lee & Fu, 2000; Renstrom & Johnson, 1990).

The biomechanical role of the meniscus is prevalent during weight-bearing activities. On average, the knee joint transmits three times a person's body weight while weight-bearing. The shape of the meniscus allows for better congruency between the articulating surfaces of the flat tibial plateaus and the convex femoral condyles (Masouros et al., 2008). Greater forces are placed on the medial tibial condyles as loads increase (Morrison, 1970), and therefore the meniscus is essential in transmitting and dissipating these forces equally on the tibia. The congruency of the meniscus adds to its role as a secondary stabilizer, especially in resisting anterior translation of the ACL-deficient knee (Renstrom & Johnson, 1990); the meniscus-meniscofemoral ligaments also play a role in the rotational stability of the tibia (Masouros, Bull, & Amis, 2010).

The role of load transmission is critical throughout the entire range of motion at the knee. In full knee extension, the meniscus is centered on the tibial plateau. As the knee flexes, the meniscus moves posteriorly (Masouros et al., 2008; McDermott et al., 2008). The anterior horns have more mobility than do the posterior horns and the lateral meniscus has greater posterior mobility than does the medial meniscus due to its loose peripheral attachment. The greater concavity of the medial tibial condyle may also contribute to the decreased mobility of the medial meniscus (Masouros et al., 2008). Although this posterior translation benefits the load-dispersal capabilities of the meniscus, limited mobility, along with the increased load-bearing responsibility of the medial meniscus, may contribute to the increased prevalence of medial meniscal tears (Fox, Bedi & Rodeo, 2012).

Shock absorption in the meniscus is attributed to its tissue properties. High water content allows for displacement of fluids under pressure, creating a drag force that resists external forces (Masouros et al., 2008; Renstrom & Johnson, 1990). Additionally, the crimped resting state of the circumferential fibers allows for an expansion under hoop stress during weight-bearing activities (Masouros et al., 2008; McDermott et al., 2008).

Meniscal Tears

Meniscal tears commonly result from the compressive forces on the meniscus by the tibia and femur during flexion and rotation of a weight-bearing knee (McDermott, 2006). A tear in young individuals often occurs from a sudden excessive force, while older adults more commonly experience the gradual onset of degenerative tears (McDermott, 2006). Young patients who sustain pathology will recall a specific mechanism of injury 80-90% of the time (Lento & Akuthota, 2000). Classic signs and symptoms of a meniscal tear include: catching, locking, or clicking; joint line pain; and a feeling of “giving out” or instability (Lowery, Farley, Wing, Sterett, & Steadman, 2006). Pain and/or inability to fully squat and a gradual onset of swelling over the first 24 hours following an injury are also commonly reported symptoms (Bower, 2013; McDermott, 2006). Joint-line tenderness has been reported as the most accurate finding in diagnosing meniscal involvement in adolescent patients (Willis, 2006). Common risk factors for sustaining an acute meniscal tear include participation in sports (Snoeker et al., Bakker, Kegel, & Lucas, 2013); chronic tears often occur as a result of persistent kneeling, repetitive squatting, or climbing stairs (Drosos & Pozos, 2003; Snoeker et al., 2013).

Classification of Meniscal Tears

Tears are classified based on their appearance and location. Horizontal tears occur in the mid-substance of the meniscus, separating it into superior and inferior segments. Longitudinal tears occur vertically along the circumferential orientation of the collagen fibers (Jee et al., 2003). A radial, or transverse, tear also occurs vertically and perpendicularly across the circumferential fibers; the disruption of the circumferential collagen fibers will affect the dispersal of weight-bearing loads (Harper, Helms, Lambert, & Higgins, 2005). Oblique, or parrot-beak, tears are a combination of radial and longitudinal tears. A tear of this kind will start in a radial direction at the inner rim and change direction longitudinally as it approaches the periphery (Jee et al., 2003). Bucket-handle tears are longitudinal tears in which the mid portion of the tear has flipped over itself (Jee et al., 2003). Complex tears are those that present with two or more of the previously described classification characteristics (Jee et al., 2003).

Longitudinal and oblique tears are the most viable for surgical repair, so long as they occur in the vascularized periphery. A particular prospective study involving 1,485 meniscal tears found 40% of the tears in the vascular peripheral portion (Metcalf & Barrett, 2014). Of those, 28% were complex tears, and 32% horizontal. Complex tears were more prevalent in patients over the age of 40 (found in 35% of patients) than in younger patients (found in 13%; Metcalf & Barrett, 2004). Tears in the avascular inner rims, as well as radial and complex tears have a lower success rate for surgical repair (Barber-Westin & Noyes, 2014).

Evaluation and Diagnostics

A battery of tests should be used by an experienced practitioner to clinically diagnose meniscal lesions, as no single test is pathognomonic for a meniscus tear (Lowery et al., 2006). The tests, palpations, and history components that have been identified (i.e. inclusion criteria) have a high specificity and high sensitivity, and they have been tested in a battery of tests. Many tests have been identified to detect meniscal tears upon clinical diagnosis. Among these tests include Apley's test, Anderson grind test, McMurray's test, bounce home test, axially loaded pivot shift test, knee compression rotation test, Ege's test, and Thessaly's test (Chivers & Howitt, 2009). In addition to special tests, a detailed history including catching or locking of the knee joint will alert an examiner of a possible meniscal tear (Lowery et al., 2006). The research of Lowery et al., (2006) recommend using the following when assessing patients for suspected meniscal pathology: (a) catching or locking as described by the patient during the history; (b) palpation of joint line tenderness; (c) McMurray's test; (d) pain with hyperextension; and (e) pain with forced flexion.

Additionally, two other tests have been identified and are recommended when assessing meniscal lesions. The first being Thessaly's test at 20 degrees, which is a dynamic weight bearing reproduction of the mechanism of injury. The second is Apley's compression and distraction test which also reproduces the compressive and rotating force involved in the mechanism of injury. Both tests have been studied in a battery, with one or more of the five tests identified by Lowery et al. (2006). Accuracy of Thessaly's test was assessed with joint line tenderness and McMurray's test, indicating that a battery of tests increases the accuracy of physical diagnosis (Konan et al., 2009). Accuracy of Apley's test was assessed with joint line tenderness, pain with forced extension, and the McMurray's test by Kurosaka et al., (1999). The results described in the article concluded physical examination is essential to the diagnosis of meniscal lesions (Kurosaka et al., 1999).

Patient History, Range of Motion, and Palpation

Patient history. One of the most important elements to any diagnosis is taking a detailed history. A few key history components will alert an examiner to meniscal pathology outside of the mechanism of injury (Lowery et al, 2006). Losses of flexion greater than 10 degrees, loss of extension greater than five degrees, crepitus, and/or joint line swelling are common history components of meniscal pathology (Magee, 2008). Catching, locking, or the sensation of catching or locking in the knee has been identified throughout literature as symptoms of meniscal pathology (Lowery et al., 2006). Lowery et al. (2006) investigated the mechanical history component further with an intact ACLs, identifying catching, locking, or the sensation of catching to have a sensitivity of 21% and specificity of 92%. The positive predictive value (PPV) associated with the history component was 74%, and the positive likelihood ratio (PLR) was 3.34 in knees treated surgically (Lowery et al., 2006).

Pain with forced joint movement. Pain associated with forced knee flexion and pain associated with hyperextension were identified by Lowery et al. (2006) as a part of a clinical composite score used to accurately detecting meniscal pathology. Forced knee flexion is performed by having the patient lie supine with examiner on the involved side (Lowery et al., 2006). The patient then actively moves his or her knee into maximum flexion, and the examiner applies an over pressure if pain is not elicited in active movement (Lowery et al., 2006). A positive test is elicited by pain within the joint line in active movement or forced overpressure (Lowery et al., 2006; Fowler & Lubliner, 1989). Lowery et al. (2006) investigated forced knee flexion with intact ACLs, identifying a sensitivity of 47% and specificity of 59%, respectively. The PPV associated with the range of motion (ROM) component was 55%, and the PLR was 1.16 in knees treated surgically (Lowery et al., 2006).

Pain with hyperextension (modified bounce home test) is performed by having the patient lie in the supine position with the examiner on the involved side (Lowey et al., 2006). The examiner cups the heel of the patient’s foot with one hand and the other hand on the knee guiding the knee from flexion into passive extension (Lowery et al., 2006). A positive test is indicated by pain in the joint line of the knee (Magee, 2008; Lowery et al., 2006; Kurosaka et al., 1999; Fowler & Lubliner, 1989). If extension is not complete or a “springy” block is felt, this is thought to be a block from the torn meniscus (Magee, 2008). Lowery et al. (2006) investigated pain with hyperextension with an intact ACL identifying a sensitivity of 33% and specificity of 88%. The PPV associated with the ROM component was 75% and the PLR was 2.59 in knees treated surgically (Lowery et al., 2006).

Palpation. Joint line tenderness is a well-known assessment for meniscal lesions and has a high sensitivity and a low specificity (Malanga et al., 2003; Rose, 2006). Joint line tenderness is assessed by having the patient supine with the examiner on the involved side (Malanga et al., 2003). The patient flexes the affected limb to approximately 90 degrees (Malanga et al., 2003). The medial edge of the medial meniscus is palpated by having the patient internally rotate the tibia, and external rotation allows for improved palpation of the lateral meniscus (Malanga et al., 2003). A positive test is indicated by pain over the palpation site in the joint line (Malanga et al., 2003; Rose, 2006). Joint line tenderness has a high sensitivity in both medial (68%-92%) and lateral (87%-95%) meniscal pathology, but best results are in lateral meniscal tears with only 8% variability between the lowest and highest sensitivity percentage reported (Eren, 2003).

Lowery et al. (2006) investigated joint line tenderness on patients with an intact ACL, identifying a sensitivity of 65% and specificity of 62%. The PPV of joint line tenderness associated with the ROM component was 65%, and the positive likelihood ratio was 1.83 in knees treated surgically. Fowler and Lubliner (1989) identified joint line tenderness with a sensitivity of 86% and a specificity of 29%. Karachalios et al., (2005) report a medial meniscus joint line tenderness sensitivity of 87%, a medial meniscus sensitivity of 87%, a lateral meniscus sensitivity of 78%, a lateral meniscus specificity of 90%, a medial meniscus diagnostic accuracy of 71%, and a lateral meniscus diagnostic accuracy of 78%. Konan et al. (2008) identify this test with a medial meniscus sensitivity of 83%, a medial meniscus specificity of 76%, a lateral meniscus sensitivity of 68%, a lateral meniscus specificity of 97%, a medial meniscus diagnostic accuracy of 81%, a lateral meniscus diagnostic accuracy of 90%, a PPV medial meniscus of 91%, and a PPV lateral meniscus of 87%. Kurosaka et al. (1999) report joint line tenderness to have an overall sensitivity of 55%, overall specificity of 67%, and an overall diagnostic accuracy of 57%. Rose et al. (2006) identify this test with a medial meniscus sensitivity of 92%, a medial meniscus specificity of 78%, a lateral meniscus sensitivity of 95%, a lateral meniscus specificity of 93%, a PPV medial meniscus 73%, and a PPV lateral meniscus of 86%.

ACL assessment. The clinician should rule out ACL involvement prior to assessing a patient for a meniscal tear, so tests used for identifying meniscal pathology will not lead to false positives due to a concurrent injury (Fowler & Lubliner, 1989; Lowery et al., 2006). Lachman's test and the pivot shift test serve as accurate diagnoses of ACL-deficient knees preoperatively, effectively ruling out ACL injuries when these tests are negative (Katz et al., 1986). Katz et al. (1986) identified the pivot shift test and Lachman's test as having a sensitivity of 81.8% individually, the Lachman's test as having a specificity of 98%, and the pivot shift test as having a specificity of 98.4% for all ACL tears (acute and chronic). Twenty studies were included in a 2012 meta-analysis, where the overall sensitivity and specificity (without anesthesia) of the Lachman test was 81% , positive predictive value (PPV) of 88%, negative predictive value (NPV) of 72%, positive likelihood ratio (PLR) of 4.5 and negative likelihood ratio (NLR) of .22 (Eck et al., 2013). The sensitivity of the pivot shift (without sedation) was 28%, specificity 81%, PPV 94%, NPV 30%, PLR 5.35, and NLR 0.30 (Eck et al., 2013). In 2015, Leblanc et al. reaffirmed high sensitivities in both Lachman's test (89% for complete and partial, 96% for complete tears) and pivot shift (79% for complete and partial, 86% for complete tears) during non-sedation evaluation, by conducting a systematic review of 8 studies. Overall, the Lachman's test has the highest sensitivity (without sedation) for diagnosing complete ACL ruptures in clinic but the pivot shift was the most specific (with sedation) (Eck et al., 2013).

Lachman's test. The Lachman's test is performed in the supine position with patient relaxed, examiner on the involved side (Katz et al., 1986). The examiner holds the knee joint in 10 to 20 degrees of flexion in a slight external rotation by stabilizing the distal femur with one hand (the outside hand, when facing a patient's head) and placing the other hand behind the proximal tibia (Katz et al., 1986). The hand on the tibia applies the anterior tibial translation, and force should be applied from the posteriormedial aspect; a negative test is one in which there is steady restraint and an immediate end point is felt (Katz et al., 1986). A positive sign is indicated by a "soft" end feel and the disappearance of the infapatellar tendon slope from tibial translation (Makhmalbaf et al., 2013; Katz et al., 1986). The Lachman's test has many modifications based on examiner hand size or patient limb size, but all positive signs are the same (Makhmalbaf et al., 2013; Katz et al., 1986).

Pivot shift test. The pivot shift test is performed in the supine position with patient relaxed and examiner on the involved side (Malanga et al, 2003). The patient's hip is flexed and abducted about 30 degrees (Malanga et al., 2003). The examiner holds the patient's foot with one hand and places the other at the knee, which is placed in 10 to 20 degrees of flexion. Torque is applied to the tibia while rotating it internally (Malanga et al., 2003). A valgus force is applied to the knee joint, while the leg is flexed to 30 to 40 degrees (Malanga et al., 2003). A positive test is indicated by an anterior subluxation of the lateral tibial plateau under the femoral condyle (Katz et al., 1986; Malanga et al., 2006).

Special Tests for Meniscal Tears

According to Fowler and Lubliner (1989), McMurray's test, Apley's compression and distraction test, and the joint line tenderness test are the most commonly used tests for identifying meniscal pathology. In a 2003 review of orthopedic special tests of the knee, the 3 stated tests plus the bounce home test (forced extension) were examined and identified as reliable tests for the clinical diagnosis of meniscal tears (Malanga et. al., 2003). Thessaly's test is a more recent addition which offers a dynamic element to these well-established tests.

McMurray test. The McMurray's test has been studied by many researchers and its' specificity is reported at various ranges throughout studies. The varying range could be attributed to specific clinician deviations and/or modifications from McMurray's (1928) original methodology, but a positive sign remained the same across all studies reviewed. Modern text books often deviate from McMurray's original work clarifying hand placement, and varying flexion of the knee joint. McMurray's test is performed with the patient in supine with a flexed hip and flexed knee (heel to buttock, if possible) (McMurray, 1928). The examiner on the side of the involved limb places one hand over the joint line with the thumb and middle fingers centered on the joint line to feel for any "popping." The other hand grasps the sole of the foot, and while the patient is relaxed, the examiner has full control over the limb, externally rotating the foot while slowly extending the knee (McMurray, 1928). The examiner checks the medial meniscus with external rotation of the foot while slowly extending the knee, and the lateral meniscus with internal rotation (Hing et al., 2009). The process is repeated several times. A positive test is indicated by a palpable "click" or "pop" in the joint line; pain may be associated, but pain alone is not a positive test (McMurray, 1928; Evans et al., 1993, Hing et al., 2009)).

Lowery et al. (2006) investigated McMurray's test with an intact ACL, identifying a sensitivity of 21% and specificity of 95%. The PPV of McMurray's test associated with the ROM component was 81% and the positive likelihood ratio was 5.00 in knees treated surgically. Evans et al., (1993) stated that McMurray's "thud" is only significant in medial meniscal tears in a prospective study of 104 patients, all of whom received arthroscopy. Accuracy of medial "thud" had a specificity of 98%, sensitivity of 16%, and PPV of 83%; however, lateral pain elicited in internal rotation had a specificity of 94%, sensitivity of 50, and PPV of 29%, illustrating the "thud" was not significant in the lateral joint line, but that pain was indicative of a meniscal tear (Evans, Bell, & Frank, 1993). Kurosaka et al. (1999) identify this test with an overall sensitivity of 37%, overall specificity of 77%, and an overall diagnostic accuracy of 45%. Fowler and Lubliner (1989) identify overall sensitivity as 16% and overall specificity as 95% for McMurray's test. Konan et al. (2008) identify this test with a medial meniscus sensitivity of 50%, a medial meniscus specificity of 77%, a lateral meniscus sensitivity of 65%, a lateral meniscus specificity of 86%, a medial meniscus diagnostic accuracy of 57%, a lateral meniscus diagnostic accuracy of 77%, a PPV medial meniscus of 86%, and a PPV lateral meniscus of 50%. Karachalios et al. (2005) identify this test with a medial meniscus sensitivity of 48%, a medial meniscus specificity of 94%, a lateral meniscus sensitivity of 65%, a lateral meniscus specificity of 86%, a medial meniscus diagnostic accuracy of 78%, and a lateral meniscus diagnostic accuracy of 84%.

Apley's compression and distraction test. Apley's compression and distraction test is normally tested in conjunction with the McMurray test and the joint line tenderness test (Scholten et al., 2001; Meserve et al, 2008; Kurosaka, et al., 1999). In Apley's original research in 1947, he describes the need to recreate the mechanism of injury through compression and rotation during examination. Apley's test is performed by having the patient lie prone, with the knee flexed to 90 degrees and the examiner on the involved side (Apley, 1947). The patient's thigh is stabilized on the table with the examiner's knee (Apley, 1947). The examiner grasps the foot in both hands medially and laterally rotates the tibia, combined with a distraction force (Aply, 1947). The process is then repeated using compression. A positive test is indicated by pain with the compression force and a relief of pain with the distraction force (Magee, 2008; Malanga, et al., 2003).

Kurosaka et al. (1999) identify Apley's test with a sensitivity of 13%, specificity of 90%, and a diagnostic accuracy of 28%. Fowler and Lubliner (1989) identify the overall sensitivity as 16% and specificity as 80%. Karachalios et al., (2005) identify this test with a medial meniscus sensitivity of 41%, a medial meniscus specificity of 93%, a lateral meniscus sensitivity of 41%, a lateral meniscus specificity of 86%, a medial meniscus diagnostic accuracy of 75%, and a lateral meniscus diagnostic accuracy of 82%. All studies were based on the methodology of Apley's original work.

Thessaly's test. Thessaly's test is a dynamic reproduction of load transmission performed at 5 and 20 degrees of flexion. The examiner supports the patient by holding the patient's outstretched arms. The patient stands on a flat surface and flexes the knee to the either 5 or 20 degrees and then internally and externally rotates the knee and body three times (Karachalios et al., 2005). A positive test is indicated by discomfort in the medial or lateral joint line (Karachalios et al., 2005). A feeling of locking or catching may be felt during this test as well, which further supports the diagnosis of a meniscal tear (Karachalios et al; 2005, Harrison et al., 2009) Thessaly's test at 20 degrees has a high specificity (97.7) as well as a high sensitivity (90.3; Harrison et al., 2009.) Thessaly's test has been studied in conjunction with McMurray test, Apley's compression and distraction test, and the joint line tenderness test, and has been identified as superior to all three in a level-one study (Karachalios et al., 2005).

Harrison et al. (2009) identify this test's overall sensitivity as 90%, overall specificity as 98%, overall diagnostic accuracy as 89% and PPV as 99%. Konan et al. (2008) identify this test with a medial meniscus sensitivity of 59%, a medial meniscus specificity of 67%, a lateral meniscus sensitivity of 31%, a lateral meniscus specificity of 95%, a medial meniscus diagnostic accuracy of 61%, a lateral meniscus diagnostic accuracy of 80%, a PPV medial meniscus of 83%, and a PPV lateral meniscus sensitivity of 66%. Karachalios et al. (2005) identify this test with a medial meniscus sensitivity of 89%, a medial meniscus specificity of 97%, a lateral meniscus sensitivity of 92%, a lateral meniscus specificity of 96%, a medial meniscus diagnostic accuracy of 94%, and a lateral meniscus diagnostic accuracy of 96%. All studies followed the original procedures described by Karachalios in 2005.

Clinical Composite Tests

Using a combination of reliable tests is essential in the clinical diagnosis of a meniscal tear. The components of the composite score identified by Lowery et. al., in 2006 are as followed: positive McMurray's test, pain with terminal knee flexion, pain with terminal knee extension, joint line tenderness, and a history of clicking and/or popping. The clinical composite score has a PPV of 92.3%, specificity of 99% and a sensitivity of 11.2% for detecting meniscal tears when all 5 signs are present (Lowery et. al., 2006). The PPV and specificity decrease to 81.8% and 96.1% respectively, while sensitivity increases to 17% when only 4 signs are present (Lowery et. al., 2006). When 3 of the 5 signs are present, the PPV is 76.7%, specificity is 90.2%, and sensitivity is 30.8% (Lowery et. al., 2006); superior or comparable to magnetic resonance imaging (MRI) alone in detecting meniscal pathology (Miller, 1996).

Imaging

Magnetic resonance imaging. Magnetic resonance imaging (MRI) is routinely recommended after a clinical diagnosis of a meniscal tear prior to any surgery discussions with a patient (Miller, 1996). Four major factors are taken into consideration when using MRIs as your only diagnostic tool: (1) image quality affects the recurrence of false positive interpretations; (2) inexperienced scanners; (3) incorrect image parameters yield less than favorable diagnostic accuracy; (4) interpretation issues (Miller, 1996). Structures such as the transverse meniscal ligament, lateral inferior geniculate artery, and the popliteus tendon may replicate the presence of a meniscal tear (Boden et al., 1992; Nikolaou et al., (2008). Meniscal tears and meniscal degeneration have a similar presence on MRIs, leading to false positives (Nikolaou et al., 2008).

MRI compared to clinical exam. Magnetic resonance imaging has been compared to the accuracy of the clinical diagnosis of meniscus tears and has been found to be comparable (Miller, 1996); in some cases, a clinical exam was found to be superior than an MRI (Miller, 1996). The clinical exam using a battery of meniscal specific tests had an accuracy of 80.7%, and MRI had 73.7% accuracy (Miller, 1996). The clinical diagnosis in Miller's study consisted of detailed history, and the assessment of: persistent pain, buckling, locking, effusion, joint line tenderness, and limited function. Muellner et al. (1997) illustrated that clinical diagnoses alone had an accuracy of 89% and 89% in MRI. The clinical diagnostic accuracy in Muellner et al. (1997) study consisted of six tests: joint line tenderness, McMurray's test, Apley's test, Pahyr's test, Steimenn's test and Bohler's test.

In a retrospective analysis of MRI efficacy in detecting internal lesions of the knee, MRI was reported to be slightly better than a clinical exam, but the clinical exam did not include a detailed history and only utilized two special tests (McMurray's and Apley's; Nikolaou et al., 2008). Diagnostic accuracy using clinical exam was reported as 60%, sensitivity as 65%, and specificity as 50%, while the diagnostic accuracy of MRI was reported as 81%, sensitivity as 83%, and specificity as 69% (Nikolaou et al., 2008).

Clinical examination has been determined to have a similar, and in some cases better, diagnostic accuracy than the MRI, concluding that MRI is only necessary in cases lacking a detailed history or one that is confusing (Rose, 2006; Boden et al., 1992; Kurosaka et al., 1999; Lowery et al., 2006; Mohan & Gosal, 2007; Miller, 1996). Surgeons may also advocate for an MRI so as to not appear too aggressive in support of surgery or for financial gains (Muellner et al., 1997). Relying on MRI results in the absence of a proper clinical examination may lead to unnecessary arthroscopic procedures, as it has been well documented that meniscal tears are often found in asymptomatic patients (Troupis et. al., 2014).

Arthroscopy. Arthroscopy is considered the "gold standard" for the detection of meniscal pathology, allowing a surgeon to visually confirm an issue through a scope. Arthroscopy is a demanding procedure and dependent on the surgeon's level of experience; especially in areas that are difficult to view due to overlapping structures or small spaces (Nikolaou et al., 2008). Arthroscopy may not be a desired diagnostic tool because of the risks involved: infection, reaction to general anesthetics, and/or scarring.

Patient Outcomes Scales and Instruments

In addition to the diagnostic assessment of meniscal lesions, the patient should also be assessed with reliable patient-oriented and disease-oriented outcomes. Outcome scales help to monitor and assess the patient's well-being, pain and functionality throughout the course of treatment, allowing the clinician to assess the effectiveness of the chosen treatment. Consideration of the population for which the instrument is intended is an important aspect for the validity of any instrument (Garratt et al., 2004). Accurate outcome measures are the cornerstone in determining effective treatments from non-effective treatments (Roos, et al., 1998). An awareness of how patients perceive their injury through a physical, psychological, and social well-being lens plays a large role in the treatment process. A clinician must be able to determine the need for referral based on psychological components exceeding their scope of practice and when the presence of psychological or social components are hindering the physical healing process (Garratt et al., 2004).

Reliability refers to an instrument's' internal consistency. Validity is whether the instrument measures what it is intended to measure. Responsiveness is whether the instrument is sensitive to changes in health (Garratt, 2004). The following instruments have high reliability, high validity, and high responsiveness.

KOOS

The Knee injury and Osteoarthritis Outcomes Score (KOOS) is a self-administered patient-oriented tool that assesses five dimensions: pain, symptoms, activities of daily living, sport and recreational function, and knee-related quality of life. The KOOS is intended for patients with knee injuries that can result in OA, and has been assessed in men and women from 14 to 79 years of age (Roos & Lohmander, 2003; Roos et al., 1998). The KOOS is a self-explanatory questionnaire that assesses short- and long-term patient relevant outcomes following knee injury, including meniscal pathology. The questionnaire takes about 10 minutes to complete. Each dimension of KOOS is scored separately, and each item is answered on a 5-point Likert scale of 0 to 4; a total score of 100 indicates no symptoms. (Roos et al., 1998) Aggregate scores are not desirable, as the instrument is intended for clinicians to thoroughly assess patients on each component of the KOOS on a regular basis (Roos et al., 1998; Roos & Lohmander, 2003). Each dimension of the KOOS is scored separately, however a composite score ($KOOS_5$) from the average of all five subsections has been used for researcher purposes (Roos & Lohmander, 2003). There are currently no published MCID values for the $KOOS_{5.}$ A total score for the KOOS has not been assessed for validity or reliability; however, reliability for each subsection is as follows: ICC for pain is 0.85-0.93, symptoms are 0.83-0.95, activities of daily living are 0.75-0.91, sports/recreation are 0.61-0.89, and quality of life is 0.83-0.95 (Roos et al., 1998).

PSFS

The Patient Specific Functional Scale (PSFS) is a patient-oriented tool that assesses patients' perceptions of their functional ability and is designed to complement generic or condition specific measurement scales (Chatman et al., 1997). The PSFS should be administered during the history intake at the time of initial assessment. The patient is asked to identify up to five activities, deemed important, that they have difficulty with or are incapable of performing due to injury. The activities are rated by the patient on an 11-point scale, where 0 represents “unable to perform” and 10 represents “able to perform at level before injury.” The tool takes approximately four minutes to complete. The clinician's role is to read instructions and record activities with corresponding ratings and remind patients of activities at follow-up appointments.

The PSFS score is calculated using an average of the ratings associated with each activity given by the patient. The minimum important difference (MID) noted by Abbott and Schmitt (2014) in patients with lower limb injuries was an increase of 2.3 points for a small change, 2.7 for a medium change, and greater than 2.7 for a large change. The reported minimal detectable change (MDC) is a change in 2.5 points when using an individual activity in patients with a lower limb injury (Chatman et al., 1997). The test-retest reliability for the PSFS was found to be excellent and had an ICC of 0.84 (Chatman et al., 1997).

DPA Scale

The Disablement in the Physical Active (DPA) is a patient-oriented scale created to assess disablement across the three interrelated domains of impairment, functional limitation, and disability, as well as health related quality of life (Vela & Denegar, 2010). Responses to the DPA scale range from 1 to 5, where a score of 1 indicates that the patient does not have a problem with the listen item, and a score of 5 indicates that the patient is severely affected by the problem. During the calculation of the patient’s score, 16 points are subtracted from the final score, to make 0 the lowest score and 64 the highest. The 16 points are subtracted because the scale uses a 1-5 interval to rate each item; without the 16-point adjustment a patient with no disablement would score 16 points on the scale rather than 0 (Vela & Deneger, 2010). A normal, healthy range for the DPA is a score of 34 or less, and a score less than or equal to 23 in acute patients indicates that a patient is ready for further functional testing by an athletic trainer or physician (Vela & Denegar, 2010). An MCID is a decrease of 9 points for an acute injury and a decrease of 6 points for a chronic injury (Vela & Denegar, 2010). The DPA scale was found to have a high test-retest reliability with an ICC of 0.943 and high validity for acute (r = -0.751) and chronic (r = -0.714) patients (Vela & Deneger, 2010).

NRS

The numerical rating scale (NRS) for pain has been widely used throughout the medical field and is accepted as a valid patient-oriented scale to assess levels of pain in many patient populations (Krebs et al., 2007). The NRS is a commonly used rating scale in athletic training. The NRS scale is scored on an 11-point scale, where a score of 0 represents no pain, and a score of 10 represents severe pain (Downie et al., 1978). The MCID for the NRS is a decrease of 2 points, or 33% in patients with chronic musculoskeletal pain (Salaffi et. al., 2004). The MID noted by Abbott and Schmitt (2014) was a decrease of 1.5 points for a small change, 3.0 for a medium change, and 3.5 for a large change. The NRS is widely accepted as a valid ($r = 0.90 - 0.92$, $P < 0.5- 0.1$; Good et al., 2001) and reliable (ICC of 1.00) scale (Herr et al., 2004).

Inclinometry

The Clinometer smartphone application has been found to be both valid and reliable when compared to the gold standard goniometry measurements at the shoulder (Werner et al., 2014). Inter-rater reliability was reported to be 0.8 (ICC 2,1; Werner et al., 2014), and validity was reported to be 0.98 at the shoulder in symptomatic patients (Werner et al., 2014). Currently, no studies exist validating the use of the Clinometer smartphone application in the lower extremity.

Goniometry

The goniometric levels of intra-tester and inter-tester reliability have been reported for a universal goniometer when measuring knee joint flexion (ICC of 0.997 and 0.977-0.982) and extension (ICC of 0.972-0.985 and 0.893-0.926). Validity varied from 0.975-0.987 for flexion and 0.390-0.442 for extension (Brosseau et al., 2001).

Treatment

Accurate diagnosis of meniscal lesions is the first step to producing quality outcomes in patients with meniscal tears. However, accurate diagnosis alone does not solve the patient's problem. Following up an accurate diagnosis with the proper course of treatment should be the primary focus of any experienced practitioner.

Currently, there is no general consensus on the proper treatment of meniscal injuries based on sound foundational research (Howell & Handoll, 1996). Previously, clinicians thought that meniscal surgery was necessary to prevent OA after a patient sustained meniscal lesion (Belzer & Cannon, 1993; O'Donoghue, 1980) because of increased contact forces on the articular surfaces of the joint (Belzer & Cannon, 1993). However, a cadaveric study of meniscal tears found that a patients can sustain a tear of up to 90% in either meniscus before joint arthrokinematics are significantly altered as compared to an uninjured knee (Bedi et al., 2010).

There are several surgical treatment options for meniscus injuries, including partial meniscectomy, meniscal repair, and meniscus transplant (Brophy & Matava, 2012). However, a patient's age, activity level, and lifestyle must be considered in addition to the size and location of the meniscal tear (Belzer & Cannon, 1993). Furthermore, Englund et al., (2012) reported that surgery might not be recommended for all meniscal lesions. The researchers found that almost one-third of all meniscal lesions found on an MRI are asymptomatic (Englund et al., 2012). Because surgeries have significant associated risks (Brophy & Matava, 2012), a new trend based on the arthrokinematics of the meniscus, surgery is only necessary if the meniscal tear interferes with normal joint motion is being embraced by researchers (Englund et al., 2012); others believe that conservative therapy should be exhausted first (Hwang & Kwoh, 2014; Katz et al., 2012; Herrlin et al., 2007; Bin, Kim, & Shin, 2004). Finally, some researchers believe partial meniscectomies should be discontinued all together for certain populations, specifically middle-aged patients with degenerative medial meniscal tears (Sihvonen et al., 2013).

Partial Meniscectomy

The most common surgery performed to treat meniscus injury is an arthroscopic partial meniscectomy. Using an arthroscopic procedure, the torn section of the meniscus is removed. The goal is to retain as much intact meniscus as possible to decrease articular forces on the joint. Initially, partial meniscectomy was thought to be indicated regardless of the location of the meniscal lesions (O'Donoghue, 1980). Prevalence of partial meniscectomies has increased significantly over the past five years because of the current clinical philosophy surrounding meniscal injuries (Sihvonen et al., 2013).

In 2004, Bin et al. published a case series on 96 patients with radial tears of the medial meniscus who were treated with a partial meniscectomy after pain persisted following three months of conservative therapy. There was a statistically significant improvement in patients who had less than 50% of the meniscus torn, but no change in patients who had greater than

50% torn. The researchers suggested that partial meniscectomy should be used in patients older than 50 years of age where any portion of the meniscus was torn (Bin et al., 2004). The researchers acknowledged that preserving meniscal tissue was necessary to prevent OA, but older patients were more likely to have OA regardless of meniscal pathology (Bin et al., 2004). Removing damaged meniscal tissue to alleviate mechanical symptoms is the more appropriate option because the articular cartilage was most likely already compromised in the older patients (Bin et al., 2004).

Several years later, Herrlin et al. (2007) contradicted the results of Bin et al. (2004) in a randomized control trial. Herlin et al. (2007) found that there was no significant difference between partial meniscectomy and conservative therapy at eight weeks post-surgery and six months post-surgery and no significant difference in pre- and post-treatment activity level. The researchers suggested that conservative therapy should be exhausted before pursuing surgical options (Herrlin et al., 2007). In 2012, the researchers of another randomized control trial compared the long-term outcomes of conservative therapy to partial meniscectomy, and their results confirmed those of Herrlin et al. (2007): no significant difference in the outcomes existed in 351 patients at six months or 12 months post treatment (Katz et al., 2013).

The Meniscus Repair in Osteoarthritis Research (METEOR) study (Katz et al., 2013), the first large-scale, longitudinal study on partial meniscectomy outcomes in patients with knee comorbidities, was a randomized control trial conducted over seven sites with 351 participants. As stated previously, the researchers found no clinically significant difference between partial meniscectomy and conservative therapy at six and 12-months post treatment. While there was a 30% crossover rate from the physical therapy group to the surgery group, at six months there was no clinically significant difference in the outcomes of the crossover group and the surgery group (Hwang & Kwoh, 2014; Katz et al., 2013).

Finally, in an effort to discontinue the use of partial meniscectomies in middle-aged patients with degenerative medial meniscal tears all together, Sihvonen et al. (2013) conducted a randomized sham study on 146 patients. The researchers found no significant difference between the outcomes of a partial meniscectomy and sham surgery and no significant difference in the patients' ability to identify which surgery they underwent. The researchers also highlighted the fact that since the publication of results of Katz et al. (2013), the use of partial meniscectomies continued to grow exponentially when they should have decreased significantly (Sihvonen et al., 2013).

Over the last decade, evidence is mounting that partial meniscectomies may not lead to improved patient outcomes (Hwang & Kwoh, 2014; Katz et al., 2013; Sihvonen et al., 2013; Herrlin et al., 2007) as once believed (Belzer & Cannon, 1993; O'Donoghue, 1980), and patients also have a significant risk of developing OA in the long term, the exact outcome which the surgical technique intended to prevent (Brophy & Matava, 2012). A Cochrane review of all meniscus surgery studies performed prior to 1996 found an astounding problem: Most of the studies produced only reported surgical outcomes and surgical technique with no control or alternative therapy outcomes, and the ones that did exist were significantly biased and flawed (Howell & Handoll, 1996). While the aforementioned research studies are not without their minor flaws (e.g., small sample sizes, studies conducted on the general population, not controlling for outside treatments (Herrlin et al., 2007; Bin et al., 2004; Hwang & Kwoh, 2014), the results published in these studies account for the level 1 evidence requested by Howell and Handoll (1996).

Meniscal Repair

Meniscus repair is a procedure in which the lesion is sutured, and all of the meniscal tissue is retained; however, meniscal repair is not always indicated. Meniscal repair is only successful when the tear occurs in the small vascular portion of the meniscus (Getgood & Robertson, 2010). Tears in the vascular portion of the meniscus occur in 60.7% of ACL comorbidity patients, but only in about 40% of ACL-intact patients (Metcalf & Barrett, 2014). Currently, several studies have been published where the researchers identify the failure rates of meniscal repair procedures (Lyman et al., 2013; Nepple, Dunn, & Wright, 2012; Pujol Barbier et al., 2011), but published research studies comparing the outcomes of meniscal repair against any other treatment paradigm are limited in quantity.

The statistics on the failure rates of meniscal repair surgery vary greatly. Getgood and Robertson (2010) estimated that meniscal repair surgeries had a 42% failure rate, but only if performed more than three months post-injury. Nepple et al. (2012) concluded that the overall failure rate greater than five years was between 22.3% and 24.3%, and 29% of the failures occurred after two years. In contrast, Pujol et al. (2011) conducted a retrospective cohort study on the failure rates of meniscus repair and subsequent partial menisectomy; the failure rate was 12.3% overall, of which 53% of patients sustained a subsequent lesion equal to, but not greater than, the initial lesion, and 31.3% sustained a smaller subsequent lesion (Pujol et al., 2011). Finally, in patients under 40 years of age, the failure rate was estimated to be 8.9% if the patient sustained a medial meniscal tear and the surgeon performing the procedure participated in more than 24 meniscal repair surgeries per year (Lyman et al., 2013).

While the failure rate is widely disputed, the outcomes of meniscal repair compared to partial meniscectomies are limited in quantity, but clear. Paxton et al. (2011) conducted a systematic review of four studies comparing the outcomes of partial meniscectomies with those of meniscal repair, finding that the latter group had a lower reoperation rate than the former. The meniscal repair groups also had improved disability outcomes compared to the partial meniscectomy group (Paxton et al., 2011). Most researchers are hesitant to refute the efficacy of meniscal repairs, even with a failure rate between 8.9% and 42% (Lyman et al., 2013; Nepple et al., 2012; Pujol Barbier et al., 2011) because more research is needed to corroborate not only the failure rates, but the effect and the efficacy of the treatment and its outcomes as compared to conservative therapy.

Meniscal Transplant

Meniscus transplant is a fairly new development in the treatment of meniscal lesions and was developed through an anatomic cadaveric study (Kohn & Moreno, 1995). Meniscal transplant surgeries were performed as early as 1980, but were and continue to be mainly experimental. As of 2010, only 4,000 procedures total had been performed in the United States (Getgood & Robertson, 2010), which is minuscule compared to partial meniscectomies occurring at the rate of 700,000 per year (Sihvonen et al., 2013).

The meniscus does not have an immune response, so replacement or transplant is fairly uncomplicated, and allograft tissue can either be sutured to meniscal remnants or to posterior and anterior attachments (Getgood & Robertson, 2010). Meniscal lesions must be measured extensively in order to ensure the correct size of the allograft. This can be accomplished through X-ray, bone scan, computerized tomography scan, MRI, and arthroscopy. Allografts, however, have a failure rate of 44% (Peters & Wirth, 2003) to 49% (Vundelinckx, Vanlauwe, & Bellmans, 2014).

In regards to autografts, a multitude of possibilities are being explored for potential tissue donor sites (Makris, Hadidi, & Athanansiou, 2013). Meniscal autografts through growth of meniscal scaffolds from donor tissue are in development (Getgood & Robertson, 2010). There are no reliability or outcomes studies for meniscal autograft transplant because the autografts currently do not resemble or mimic the original meniscus (Makris et al., 2013).

A more recent theory has begun to develop over the last decade that focuses on the surgical treatment of meniscal tears. This theory argues that surgery may not be the quintessential treatment and that conservative therapy treatment paradigms should be investigated further (Hwang & Kwoh, 2014; Katz et al., 2013; Sihvonen et al., 2013; Herrlin et al., 2007) as once assumed (Belzer & Cannon, 1993; O'Donoghue, 1980). Conservative treatment can involve various manual therapy techniques and has been shown to effectively resolve symptoms and increase function (Englund et al., 1992).

The Mulligan Concept

Background

Manual therapy encompasses a wide array of techniques and theories of efficacy (Threlkeld, 1992). The history of these techniques are rooted in the studies and research of well-known scientific scholars and are used for many different musculoskeletal injuries; however, the conservative treatment of symptoms of meniscal tears using the Mulligan Concept (MC) has not been explored. The MC was developed on a mobilization with movement (MWM) theory and principles that involve compression, traction, and/or articulation (joint mobilization) of the restricted or painful joint (Hing, Hall, Rivett, Vicenzino, & Mulligan, 2015; Mulligan, 1993; Mulligan, 2004; Mulligan, 2010; Vicenzino, Hing, Rivett, & Hall, 2011). The MC interventions incorporate a sustained passive joint mobilization during the patient's active movement, which may address and correct pain and discomfort at the knee due to meniscal tears.

The Positional Fault Theory

The MC "Squeeze" technique efficacy in treating meniscal tear symptoms is based primarily in the technique's physiological correction of a theoretical positional fault of the knee joint (Mulligan, 2010). Specifically for the "Squeeze" technique, Brain Mulligan proposes a mechanical lesion called "an abnormal meniscal distortion" should be considered when patients are complaining of meniscus tear symptoms. A typically mechanism of meniscus tears of twisting of the knee while weight bearing could cause the meniscus to distort slightly in towards the periphery of the knee (Mulligan, 2010).

Mulligan's positional fault theory is based in the foundational knowledge of normal arthrokinematics of the joint and the changes that may with injury. Mulligan theorized that minor positional faults occur secondary to injury and cause joint mal-tracking, which leads to pain, stiffness, and/or weakness (Mulligan, 1993; Mulligan, 2004). The changes that occur within the joint are not just limited to the joint surface itself, but also effects connective tissue and all other associated structures within the joint. For example, after a mechanism of injury for meniscus tears occur, meniscal tissue within the joint could cause the joint to become blocked and lose motion thus leading to pain and dysfunction. Gale et al. (1999) also determined that meniscal subluxation is common in knees with OA and is correlated with the severity of joint space narrowing on plain radiographs, thus supporting a faulty mechanical component causing pain and dysfunction. If a meniscus has become dislodged or torn and flaps of the tissue are trapped within the joint, classic meniscus tear symptoms such as knee-joint locking, clicking, pain, and loss of motion could occur, along with other mechanical joint positional dysfunctions.

Although secondary faults due to injury are not typically observed via diagnostic imagining (Mulligan, 1993), evidence of joint positional faults have been reported in both clinical and laboratory settings (Hsieh-Y, Vicenzino, Yang, Hu & Yang, 2002; Hubbard & Hertal, 2008; Hubbard, Hertal & Sherbondy, 2006; Kavanagh, 1999; Fukuhara, Sakamoto, Nakazawa, & Kato, 2012). However, the positional fault theory is not universally accepted and although more evidence continues to be produced, it remains theoretical.

Hsieh-Y et al. (2002) observed a single case study where MRIs were taken of a thumb over a period of three weeks. Imaging was performed before the application of a MWM treatment, and a positional fault was observed. Follow-up imaging was performed immediately after the treatment, and the positional fault was absent; the patient also reported a resolution of symptoms. A three week follow-up MRI revealed a return of the fault in the joint, but the patient did not report a return of the symptoms. Limiting factors in this study were a lack of statistical analysis and the utilization of one patient. Those factors provide low level evidence and an inability to make a definitive statement that all injuries lead to positional faults which MWMs are indicated to correct.

Support for the presence of a positional fault in chronic ankle instability and in acute and subacute ankle sprains is also found in the literature (Berkowitz & Kim, 2004; Hubbard &

Hertal, 2006; Hubbard, Hertal, & Sherbody, 2006; Kavanagh, 1999; Vincenzio, Paungmali, & Teys, 2007). The studies are inconclusive as to whether the positional fault predisposed the participant to injury or if it was caused by the injury, even though significant differences in fibular positioning on the talus was observed in both sub-acute lateral ankle sprain and chronic ankle instability participants as compared to the uninjured ankle and matched controls. Thus, likely supporting positional fault to be the result of injury rather than the cause. The results, however, are promising and suggest that, if these faults exist, treatments such as MWMs would be effective in correcting joint positioning that has been altered due to injury. More research is needed in this area to determine if Mulligan's positional fault theory can be consistently and scientifically accepted.

One possible positional fault mechanism of the menisci within the knee joint could be supported using a physiological rationale similar to the meniscoid in the cervical spine. Hearn and Rivett (2002) explored the biomechanical reasoning for pain relief after a Sustained Natural Apophyseal Glide (SNAG) in the cervical spine. The researchers assessed the role of the meniscoid in zygopohyseal joint dysfunction. The meniscoid in the cervical spine is reminiscent of the mensci in the knee. They both have similar functions and positioning within their respective joints. Hearn and Rivett (2012) discussed the possibility of the meniscoid becoming entrapped between the cervical vertebrae or displaced on the articular surface after the vertebrae returns to the neutral position from an open packed position, much like the meniscus can cause a joint to become mechanically stuck after a patient has been sitting for extended period of time with the knee in an open packed position. The review implicates the possibility that a cervical SNAG could lead to a decrease in pain by separating the facet surfaces and releasing the meniscoid or allowing the trapped segment to return to its normal resting position and normal arthrokinematic function. Also noted is a possibility of stretching adhesions that are secondary to positional faulting of the meniscoid or to the joint capsule in the knee joint, which is attached to the meniscus and may have developed adhesions secondary to meniscal pathology.

Neurophysiological Effects

The body's ascending and descending pathways for pain perception and modulation occur along the same route to the central nervous system (Ossipov, Dusso, & Porreca, 2010). Researchers also theorize the origin of pain associated with meniscal pathology is the result of compression on the peripheral nerve supply on the outer horn of the structure (Renstrom & Johnson, 1990), where joint impingement on the nerve sends noxious signals to the spinal cord and upward to the supraspinal mechanisms of pain perception. Theoretically, chronic pain will continue to exist as long as the tissue of the meniscus is compressed and signals are continually relayed to the brain.

Multiple theories exist to explain how and why joint mobilizations contribute to pain relief in patients with painful and restrictive movement. Melzack and Wall's (1965) classic gate control theory offers insight to a possibility that passive joint movement initiates segmental inhibitory mechanisms that cause spinal mechanisms of pain control to block the noxious signal's pathway to the brain. The peripheral touch stimulated large A-Beta fibers may transmit non-painful contact stimulus faster to the central nervous system (CNS) than smaller noxious transmitting delta fibers (Vicenzino et al., 2011). Initiation of sympathetic nervous system responses were observed after a treatment of MWMs, eliciting similar responses of pain relief to those seen after spinal manipulation (Paungmali, O'Leary, Souvlis, & Vicenzino, 2003). While neurophysiological implications involving CNS hypoalgesia for most MC techniques are accepted, researchers have not concluded the mechanism by which the technique produces the hypoalgesia effect. However, Paungmali et al. (2003) suggest that the hypoalgesic effects of MWMs at the elbow to treat lateral epicondylalgia was not produced by an opioid pain-modulating mechanism and may have resulted from other mechanisms of pain control.

Many studies have been conducted which support the mechanical hypoalgesia component of the MC, but most are case studies or case series with small sample sizes concentrated on the shoulder, elbow, or ankle (Collins, Teys, & Vicenzino, 2004; Paungmali et al., 2003; Slater, Arendt-Nielson, Wright, & Graven, 2006; Teys, Bisset, & Vicenzino, 2008). Studies conducted to explore the hypoalgesic effect in the knee resulting from joint mobilization have typically involved patients with osteoarthritis. While osteoarthritis has been indicated as a secondary joint disease due to meniscal injury (Englund et al., 2009), no studies have measured pain reduction in patients with meniscal pathology exclusively. Despite this, hypoalgesia mechanisms and a physiological component are also suspected to contribute to positive outcomes of the treatment as well.

Psychological Implications

Psychological or psychosocial involvement may also contribute to positive outcomes of the MC "Squeeze" technique; supporting implications of the mechanisms of efficacy of the MC to provide a placebo effect after treatment is completed (Vincezino, Hing, Rivett, & Hall, 2011). The mechanisms by which this may occur lay in musculoskeletal interventions that affect a variety of patient components not directly related to the physical injury itself. The history of both the patient and clinician, in addition to a patient's exposure to pain, healing, and fears about treatment, play a role in how effective the treatment will be for the patient (Bialosky, Bishop, Price, Robinson, & George, 2009; 2011; Vicenzino et al., 2011).

Pain relief has physiological mechanisms by which the placebo and psychological effect takes place. Bialosky, Bishop, George, and Robinson (2011) suggested interpreting and classifying the placebo effect of manual therapy as an active ingredient in pain reduction, while Miller and Kaptchuk (2008) suggested interpreting the placebo effect as 'contextual healing' instead of an unexplained positive reaction to an intervention.

The placebo effect is typically used to determine the efficacy of an indicated therapeutic intervention and disregarded as actively contributing to positive patient outcomes. If the therapeutic intervention does not elicit considerable significant positive outcomes compared to the placebo, the treatment is classified as ineffective (Bialosky et al., 2011). As placebo hypoalgesia relates to MWMs and other treatment interventions, studies support the placebo's relationship to the central nervous system's descending pain inhibitory pathways from the supraspinal structures (Bialosky et al., 2011). Whether or not MWM's hypoalgesic effect is based in actual accepted mechanisms of pain control by correcting biomechanical and physiological faults or by way of the placebo effect is of no difference. If patients are reporting positive outcomes for pain reduction and increases in function, the treatment is successful and indicated for the patient's condition.

Teys et al. (2008) determined during a study on shoulder pain and range of motion that patients receiving a sham treatment gained increases in range of motion and decreases in pain as compared to the control group. While the MWM treatment group had the most significant gains, the study lends credit to both the efficacy of MWMs for the treatment of shoulder pain and restriction and also to the consideration of using a placebo effect as a viable and useful component of manual therapy.

Vicenzino, Paungmali, and Teys (2007) concluded that while the implications and speculations of neurophysiologic involvement elicited from the MWMs is accepted, the actual effect of the technique is much more complex and multifaceted. The implications for other psychological components along with the placebo effect involve diminishing a patient's previous perception that movement at a particular joint is painful. By applying the MWM and instructing the patient to move through the now pain-free range, the previous fearful memory may be eliminated (Vicenzino et al., 2011).

The Mulligan Concept "Squeeze" Technique Procedure.

The basic treatment application for all MWMs incorporates Mulligan's rules and principles for the intervention. Mulligan advocates that his techniques be pain free during the patient's full range of motion. If at any point the movement becomes painful while the glide is applied, the clinician is to stop the movement and adjust the glide. For the treatment to be indicated, the clinician must be able to apply the correct glide to provide the patient with a pain-free range of motion. If pain-free motion is not achieved, the patient may fall within the contraindications of the technique or other principals of the treatment may have not been followed (Mulligan, 1993; Mulligan, 2004; Mulligan, 2010; Vicenzino et al., 2011; Vicenzino, 2011; Hing et al., 2015).

The MC uses the acronym "CROCKS" (contraindications, repetitions, overpressure, communication, knowledge, and skills, subtle movement, sustain, and sense) to serve as a reminder of the general principles for all its intervention ns. If all of these principles are followed, Mulligan suggests that a PILL effect (pain free, instant, long-lasting) will occur for the patient (Hing et al., 2015; Mulligan, 1993; Mulligan, 2004; Mulligan, 2010; Vicenzino et al., 2011).

The technique for the MC "Squeeze" incorporates patient generated open packed positioning of the knee joint, compression of the joint space, and a minor fibio-tibial glide either posterior or anterior dependent upon flexion or extension restrictions. Minimal tibial-femoral rotation may be required if an alteration is needed to provide pain relief (Hing et al., 2015; Mulligan, 1993; Mulligan, 2010). To perform the technique correctly, the patient may be placed in a weight-bearing or supine position. The approach for treating flexion may be done either supine or standing, but treatment for extension can only be done while the patient is supine (Mulligan, 2010; Hing et al., 2015).

The clinician begins the treatment by first testing for restrictive movement and/or local pain during knee flexion or extension, depending on the primary complaint of the patient. If a restriction and/or pain is noted while the patient is supine, the treatment is performed supine; if the restriction and/or pain is noted during a weight-bearing activity, the patient is treated during the weight-bearing activity.

To perform the technique in the supine position, the clinician will begin by palpating the medial and lateral joint line of the knee to locate an area of most tenderness. If tenderness is noted over the postero-medial or medial joint space of the right knee, the clinician will stand at the left side of the patient; however, if tenderness is noted over the lateral joint line, the clinician will stand on the same side as the patient. The clinician will place the medial border of one thumb, reinforced by the other, over the tender joint space and instruct the patient to actively and slowly flex the knee so the joint space will open. When the clinician begins to feel the joint space open beneath the thumbs, a squeeze is applied centrally. While squeezing centrally, the clinician encourages more joint flexion using the ulnar border of the hand that is over the upper end of the tibia. The patient may experience localized discomfort from the overlap grip to tolerance, but the localized discomfort should not be exacerbated with movement. The clinician maintains the squeeze and overpressure for a few seconds, repeat three times, and then reassess motion. This MC "Squeeze" technique, while effective, is uncomfortable due to the pressure caused underneath the clinician's thumb while the squeeze portion of the treatment is performed, but the movement itself should not be painful (Mulligan, 2011). Other MWMs have a pain-free requirement (Mulligan, 1993).

The same technique and hand placement is used for a weight-bearing patient. The clinician will kneel beside the patient and place the his or her thumbs over the joint margin, as indicated for the supine patient. The clinician will then instruct the patient to perform a squat during the movement, at which point the clinician will apply thumb pressure as the joint space is revealed. The patient may feel more comfortable holding on to a table or a chair for support during the weight-bearing alternative. The squeeze is held for a few seconds and then three more repetitions are done before reassessing for pain and motion (Hing et al., 2015; Mulligan, 2010).

The pressure or squeeze from the clinician occurs centrally, from the tender point (as noted in the assessment). The direction of the squeeze is important to mention because of the anatomical movement of the menisci during flexion and extension of the knee, especially if the tender point is located along the lateral joint line. The lateral meniscus is more mobile than the medial meniscus and is pulled anteriorly during knee extension via the patellomensical ligament. During the last few degrees of flexion, the menisofemoral ligament pulls the posterior horn of the lateral meniscus medially and anteriorly (Vedi, Spouse, Williams, Tennant, Hunt, & Gedroyc, 1999). Patients complaining of pain with extension and full flexion may benefit most from the squeeze technique because of the clinician's hand placement and the direction applied in the joint space during active movement.

Efficacy of Treatment of Mobilization with Movement

Hing et al. (2007) conducted a review of all relevant MWM studies and reported significant positive results with the treatment application when compared to placebo or controls. The authors found only one study that did not report notable improvements from applications of MWMs, but this study conducted by Slater et al. (2006) pertained to outcomes of lateral epicondylalgia induced by the research team.

Support exists for the mechanical correction of a theoretical positional fault. In regards to the mechanisms of pain control related to a hypoalgesic effect and psychological theories, Bialosky et al. (2009) suggested a combination of both biomechanical (e.g., positional fault) and neurophysiological (e.g., hypoalgesia) mechanisms are responsible for the efficacy of manual therapy techniques, such as MWMs, for treating musculoskeletal injuries. The MC "Squeeze" technique involves direct pressure on the tender point in the joint space which may incorporate both a mechanical correction of a displaced meniscus and a hypoalgesic effect. By applying direct pressure into the joint line, the potentially displaced tissue could be placed back into its normal anatomical position. Moreover, correcting a potential position fault could lead to a return to functioning arthrokinematics of the joint. The pressure provided by the clinician during the technique also causes minor discomfort to the patient which may elicit peripheral mechanisms of pain control such as endogenous opioids thus, contributing to a decrease in pain.

Conclusion

The MC "Squeeze" technique is a recommended option for conservative therapy of meniscal tears. The manual therapy intervention is designed to treat limited range of motion and localized joint line pain during movement (Mulligan, 2010), which are symptoms often found in the presence of meniscal tears (Lowery et al., 1996). Despite the theorized benefit of this technique with these patients, the authors of this literature review could not identify formal investigations of the efficacy of this treatment. Therefore, research is to examine the effect of the MC "Squeeze" technique in physically active patients who present with clinical symptoms of meniscal tears and meet the criteria for a clinical diagnosis of a meniscal tear.

References

1. American Association of Orthopaedic Medicine. (2013). Cyriax System of Orthopaedic Medicine. Retrieved from: http://http://www.aaomed.org/Cyriax-System-of-Orthopaedic-Medicine on November 29, 2014.
2. Andrews, S., Shrive, N., & Ronsky, J. (2011). The shocking truth about meniscus. *Journal of Biomechanics*, *44*(16), 2737–2740. doi:10.1016/j.jbiomech.2011.08.026.
3. Arnoczky, S. P., & Warren, R. F. (1982). Microvasculature of the human meniscus. *The American Journal of Sports Medicine*, *10*(2), 90–5. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7081532.
4. Berthiaume, M.J., Raynauld, J.P., Martel-Pelletier, J., LaBonte, F., Beaudoin, G., Bloch, D.A., Choquette, D., Haraoui, B., Altman, R.D., Hochberg, M., Meyer, J.M., Cline, G.A. & Pelletier, J.P. (2005). Meniscal tear and extrusion are strongly associated with progression of symptomatic knee osteoarthritis as assessed by quantitative magnetic resonance imaging. *Annals of the Rheumatic Diseases, 64*, 556-563. doi: 10.1136/ard.2004.023796.
5. Bialosky, J.E., Bishop, M.D., Price, D.D., Robinson, M.E., George, S.Z. (2009). The mechanisms of manual therapy in the treatment of musculoskeletal pain: A comprehensive model. *Manual Therapy*, *14* doi: 10.1016/j.math.2008.09.001.
6. Bialosky, J.E., Bishop, M.D., George, S.Z., & Robinson, M.E. (2011). Placebo response to manual therapy: Something out of nothing? *Journal of Manual and Manipulative Therapy*, 19 (1), 11-19. doi: 10.1179/2042618610Y.0000000001.
7. Belzer, J. P., & Cannon, W. D. (1993). Meniscus Tears: Treatment in the Stable and Unstable Knee. *Journal of the American Academy of Orthopaedic Surgeons*, *1*(1), 41–47.
8. Berkowitz, M.J. & Kim, D.H. (2004). Fibular position in relation to lateral ankle instability. *Foot and Ankle International, 25*(5), 318-321.
9. Bin, S.-I., Kim, J.-M., & Shin, S.-J. (2004). Radial tears of the posterior horn of the medial meniscus. *Arthroscopy: The Journal of Arthroscopic & Related Surgery: Official Publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, *20*(4), 373–378. doi:10.1016/j.arthro.2004.01.004.

10. Boden, S. D., Davis, D. O., Dina, T. S., Stoller, D. W., Brown, S. D., Vailas, J. C., & Labropoulos, P. A. (1992). A prospective and blinded investigation of magnetic resonance imaging of the knee. Abnormal findings in asymptomatic subjects. *Clinical Orthopaedics and Related Research*, (282), 177-185.

11. Brophy, R. H., & Matava, M. J. (2012). Surgical options for meniscal replacement. *The Journal of the American Academy of Orthopaedic Surgeons*, *20*(5), 265–272. doi:10.5435/JAAOS-20-05-265.

12. Brosseau, L., Balmer, S., Tousignant, M., O'Sullivan, J. P., Goudreault, C., Goudreault, M., & Gringras, S. (2001). Intra- and intertester reliability and criterion validity of the parallelogram and universal goniometers for measuring maximum active knee flexion and extension of patients with knee restrictions. *Archives Of Physical Medicine And Rehabilitation*, *82*(3), 396-402.

13. Chatman, A. B., Hyams, S. P., Neel, J. M., Binkley, J. M., Stratford, P. W., Schomberg, A., & Stabler, M. (1997). The Patient-Specific Functional Scale: measurement properties in patients with knee dysfunction. *Physical Therapy*, *77*(8), 820-829.

14. Chivers, M., & Howitt, S. (2009). Anatomy and physical examination of the knee menisci: a narrative review of the orthopedic literature. *The Journal of the Canadian Chiropractic Association*, *53*(4), 319-333.

15. Collins, N., Teys, P. & Vicenzino, B. (2004). The Initial Effects of a Mulligan's Mobilization with Movement Technique on Dorsiflexion and Pain in Subacute Ankle Sprains. *Manual Therapy*, *9*(2)' 77-82. doi: 10.1016/S1356-689X(03)00101-2.

16. Deyle, G.D., Henderson, N.E., Matekel, R.L., Ryder, M.G., Garber, M.B., & Allison, S.C. (2000). Effectiveness of Manual Physical Therapy and Exercise in Osteoarthritis of the Knee. *Annals of Internal Medicine*, 132 (3), 173-181, Retrieved from http://annals.org.

17. Dye, S. F., Vaupel, G. L., & Dye, C. C. (1998). Conscious neurosensory mapping of the internal structures of the human knee without intraarticular anesthesia. *The American Journal of Sports Medicine*, *26*(6), 773–777.

18. Eck, C., Bekerom, M., Fu, F., Poolman, R., & Kerkhoffs, G. (2013). Methods to diagnose acute anterior cruciate ligament rupture: a meta-analysis of physical examinations with and without anaesthesia. *Knee Surgery, Sports Traumatology, Arthroscopy*, *21*(8), 1895-1903. doi:10.1007/s00167-012-2250-9.

19. Englund, M., Roemer, F. W., Hayashi, D., Crema, M. D., & Guermazi, A. (2012). Meniscus pathology, osteoarthritis and the treatment controversy. *Nature Reviews Rheumatology*, *8*(7), 412–419. doi:10.1038/nrrheum.2012.69.

20. Englund, M., Guermazi, A. & Lohmander, L.S. (2009). The Meniscus in Knee Osteoarthritis. *Rheumatic Disease Clinics of North America*, *35*(3), 579-590. doi: 10.1016/j.rdc.2009.08.004.

21. Eren, O. T. (2003). The accuracy of joint line tenderness by physical examination in the diagnosis of meniscal tears. *Arthroscopy: The Journal Of Arthroscopic & Related Surgery: Official Publication Of The Arthroscopy Association Of North America And The International Arthroscopy Association*, *19*(8), 850-854.

22. Evans, P. J., Bell, G. D., & Frank, C. (1993). Prospective evaluation of the McMurray test. *The American Journal Of Sports Medicine*, *21*(4), 604-608.

23. Exelby, L. (1996). Peripheral mobilisations with movement. *Manual Therapy 1*, 118-126: Retrieved from: http://kinex.cl/papers/Rodilla/MWMNUEVO.pdf on November 29, 2014.

24. Fowler, P. J., & Lubliner, J. A. (1989). The predictive value of five clinical signs in the evaluation of meniscal pathology. *Arthroscopy: The Journal of Arthroscopic & Related Surgery: Official Publication Of The Arthroscopy Association Of North America And The International Arthroscopy Association*, *5*(3), 184-186.

25. Fox, A. J. S., Bedi, A., & Rodeo, S. A. (2012). The basic science of human knee menisci: structure, composition, and function. *Sports Health*, *4*(4), 340–51. doi:10.1177/1941738111429419.

26. Fox, M. G. (2007). MR imaging of the meniscus: Review, current trends, and clinical implications. *Radiologic Clinics of North America*, *45*(6), 1033–1053. doi:10.1016/j.rcl.2007.08.009.

27. Fukuhara, T., Sakamoto M., Nakazawa R., & Kato K. (2012) Anterior positional fault of the fibula after sub-acute anterior talofibular ligament injury. *Journal of Physical Therapy Science*, 24(1), 115-117. http://doi.org/10.1589/jpts.24.115.

28. Gale, D.R., Chaisson, C.E., Totterman, S.M.S., Schwartz, R.K., Gale, M.E., Felson, D. (1999). Meniscal subluxation: association with osteoarthritis joint space narrowing. *Osteoarthritis and Cartilage*, 7 (6), 526-532. doi: 10.1053/joca.1999.0256.

29. Garratt, A., Brealey, S., & Gillespie, W. (2004). Patient-assessed health instruments for the knee: a structured review. *Rheumatology (Oxford, England)*, *43*(11), 1414-1423.

30. Getgood, A., & Robertson, A. (2010). (v) Meniscal tears, repairs and replacement – a current concepts review. *Orthopaedics and Trauma*, *24*(2), 121–128. doi:10.1016/j.mporth.2010.03.01.

31. Good, M., Stiller, C., Zauszniewski, J.A., Anderson, G.C., Stanton-Hicks, M., Grass, J.A. (2001). Sensation and distress of pain scales: Reliability, validity, and sensitivity. *Journal of Nursing Measurements*, 9(3), 219-238.

32. Greis, P. E., Bardana, D. D., Holmstrom, M. C., & Burks, R. T. (2002). Meniscal injury: I. basic science and evaluation. *The Journal of the American Academy of Orthopaedic Surgeons*, *10*(3), 168–176. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12041938.

33. Harper, K. W., Helms, C. A., Lambert, H. S., & Higgins, L. D. (2005). Radial meniscal tears: Significance, incidence, and MR appearance. *American Journal of Roentgenology*, *185*(6), 1429–34. doi:10.2214/AJR.04.1024.

34. Harrison, B., Abell, B., & Gibson, T. (2009). The Thessaly test for detection of meniscal tears: validation of a new physical examination technique for primary care medicine. *Clinical Journal of Sport Medicine: Official Journal of the Canadian Academy of Sport Medicine*, *19*(1), 9-12. doi:10.1097/JSM.0b013e31818f1689.

35. Hearn, A & Rivett, A. (2002). Cervical SNAGs: a biomechanical analysis. *Manual Therapy*, 7 (2), 71-79, doi: 10.1054/math.2002.0440.

36. Herr, K.A., Spratt, K., Mobily, P.R., Richardson, G. (2004). Pain intensity assessment in older adults: Use of experimental pain to compare psychometric properties and

usability a selected pain scales with younger adults. *The Clinical Journal of Pain.* 20(4), 207-219).

37. Herrlin, S., Hållander, M., Wange, P., Weidenhielm, L., & Werner, S. (2007). Arthroscopic or conservative treatment of degenerative medial meniscal tears: a prospective randomised trial. *Knee Surgery, Sports Traumatology, Arthroscopy*, *15*(4), 393–401. doi:10.1007/s00167-006-0243-2.

38. Hing, W., Bigelow, R. & Bremner, T. (2007). Mulligan's Mobilization with Movement: A Systematic Review. *The Journal of Manual & Manipulative Therapy, 17*(2),39- 66.

39. Hing, W., White, S., Reid, D., & Marshall, R. (2009). Validity of the McMurray's Test and Modified Versions of the Test: A Systematic Literature Review. *The Journal of Manual & Manipulative Therapy*, *17*(1), 22-35.

40. Hing, W., Hall, T., Rivett, D., Vicenzino, B. & Mulligan, B. (2015). The Mulligan Concept of Manual Therapy Textbook of Techniques. Chatswood, Australia: Elsevier.

41. Howe, T., Dawson, L., Syme, G., Duncan, L., & Reid, J. (2012). Evaluation of outcome measures for use in clinical practice for adults with musculoskeletal conditions of the knee: a systematic review. *Manual Therapy*, *17*(2), 100-118. doi:10.1016/j.math.2011.07.002.

42. Howell, J., & Handoll, H. (1996). Surgical treatment for meniscal injuries of the knee in adults. In *Cochrane Database of Systematic Reviews*. John Wiley & Sons, Ltd. Retrieved from http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD001353/.

43. Hsieh C.Y., Vicenzino, B., Yang, C.H., Hu, M.H., Yang, C. (2002). Mulligan's mobilization with movement for the thumb: A single case report using magnetic resonance imaging to evaluate the positional fault hypothesis. *Manual Therapy, 7* (1), 44-49 doi:10.1054/math.2001.0434.

44. Hubbard, T.J. & Hertel, J. (2008). Anterior positional fault of the fibula after sub-acute lateral ankle sprains. *Manual Therapy, 13,* 63-67. doi: 10.1016/j.math.2006.09.008.

45. Hubbard, T.J., Hertel, J., & Sherbondy, P. (2006). Fibular position in individuals with self-reported chronic ankle instability, *Journal of Orthopaedic & Sports Physical Therapy, 36* (1), 3-10.

46. Hwang, Y. G., & Kwoh, C. K. (2014). The METEOR trial: No rush to repair a torn meniscus. *Cleveland Clinic Journal of Medicine*, *81*(4), 226–232. doi:10.3949/ccjm.81a.13075.

47. Jee, W., Mccauley, T. R., Kim, J., Jun, D., Lee, Y., Choi, B., & Choi, K. (2003). Meniscal tear configurations: Categorization with MR imaging. *American Journal of Roentgenology*, (180), 93–97.

48. Johnson, D. L., Swenson, T. M., Livesay, G. A., Aizawa, H., Fu, F. H., & Herner, C. D. (1995). Insertion-Site Anatomy of the Human Menisci: as a Basis for Meniscal Transplantation. *Arthroscopy: The Journal of Arthroscopic and Related Surgery*, *11*(4), 386–394.

49. Karachalios, T., Hantes, M., Zibis, A., Zachos, V., Karantanas, A., & Malizos, K. (2005). Diagnostic accuracy of a new clinical test (the Thessaly test) for early detection of meniscal tears. *The Journal of Bone and Joint Surgery. American Volume*, *87*(5), 955-962.

50. Kavanagh, J. (1999). Is there a positional fault at the inferior tibiofibular joint in patients with acute or chronic ankle sprains compared to normals? *Manual Therapy*, *4*(1), 19-24.

51. Katz, J., & Fingeroth, R. (1986). The diagnostic accuracy of ruptures of the anterior cruciate ligament comparing the Lachman test, the anterior drawer sign, and the pivot shift test in acute and chronic knee injuries. *The American Journal of Sports Medicine*, 88-91.

52. Katz, J. N., Brophy, R. H., Chaisson, C. E., de Chaves, L., Cole, B. J., Dahm, D. L., Losina, E. (2013). Surgery versus Physical Therapy for a Meniscal Tear and Osteoarthritis. *New England Journal of Medicine*, *368*(18), 1675–1684. doi:10.1056/NEJMoa1301408.

53. Kirkley, A., Birmingham, T. B., Litchfield, R. B., Giffin, J. R., Willits, K. R., Wong, C. J., Fowler, P. J. (2008). A Randomized Trial of Arthroscopic Surgery for Osteoarthritis of the Knee. *New England Journal of Medicine*, *359*(11), 1097–1107. doi:10.1056/NEJMoa0708333.

54. Kohn, D., & Moreno, B. (1995). Meniscus insertion anatomy as a basis for meniscus replacement: A morphological cadaveric study. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*, *11*(1), 96–103. doi:10.1016/0749-8063(95)90095-0.

55. Kos, J., Hert, J. & Sevcik, P. (2002). Meniscoids of the intervertebral joints. *Acta Chirurgiae Orthopaedicae et Tramatologiae Cechoslovaca*, *69*(3), 149-157. Retrieved from http://ncbi.nlm.nih.gov on November 27, 2014.

56. Krebs, E. E., Carey, T. S., & Weinberger, M. (2007). Accuracy of the pain numeric rating scale as a screening test in primary care. *Journal Of General Internal Medicine*, *22*(10), 1453-1458.

57. Kurosaka, M., Yagi, M., Yoshiya, S., Muratsu, H., & Mizuno, K. (1999). Efficacy of the axially loaded pivot shift test for the diagnosis of a meniscal tear. *International Orthopaedics*, *23*(5), 271-274.

58. Larsen, E., Jensen, P., & Jensen, P. (1999). Long-term outcome of knee and ankle injuries in elite football, 285–289.

59. Lee, J. M., & Fu, F. H. (2000). The Meniscus: Basic science and clinical applications. *Operative Techniques in Orthopaedics*, *10*(3), 162–168. doi:10.1053/otor.2000.5289.

60. Lento, P. H., & Akuthota, V. (2000). Meniscal injuries: A critical review. *Journal of Back and Musculoskeletal Rehabilitation*, *15*(2), 55–62. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/22388443.

61. Lyman, S., Hidaka, C., Valdez, A. S., Hetsroni, I., Pan, T. J., Do, H., Marx, R. G. (2013). Risk Factors for Meniscectomy After Meniscal Repair. *The American Journal of Sports Medicine*, *41*(12), 2772–2778. doi:10.1177/0363546513503444.

62. Maitland, G. (5th ed). (1986). Vertebral Manipulations. London: Butterworths.

63. Makhmalbaf, H., Moradi, A., Ganji, S., & Omidi-Kashani, F. (2013). Accuracy of lachman and anterior drawer tests for anterior cruciate ligament injuries. *Archives Of Bone And Joint Surgery*, *1*(2), 94-97.

64. Makris, E. A., Hadidi, P., & Athanasiou, K. A. (2011). The knee meniscus: Structure–function, pathophysiology, current repair techniques, and prospects for regeneration. *Biomaterials*, *32*(30), 7411–7431 doi:10.1016/j.biomaterials.2011.06.037.

65. Malanga, G., Andrus, S., Nadler, S., & McLean, J. (2003). Physical examination of the knee: a review of the original test description and scientific validity of common orthopedic tests. *Archives of Physical Medicine and Rehabilitation*, *84*(4), 592-603.

66. Mariani, P., Adriani, E., Maresca, G., & Mazzola, C. (1996). A prospective evaluation of a test for lateral meniscus tears. *Knee Surgery, Sports Traumatology, Arthroscopy: Official Journal of the ESSKA*, *4*(1), 22-26.

67. Mascarenhas, R., Yanke, A., Frank, R., Butty, D., & Cole, B. (2014). Meniscal Allograft Transplantation: Preoperative Assessment, Surgical Considerations, and Clinical Outcomes. *Journal of Knee Surgery*. doi:10.1055/s-0034-1382080.

68. Masouros, S. D., Bull, A. M. J., & Amis, A. A. (2010). (i) Biomechanics of the knee joint. *Orthopaedics and Trauma*, *24*(2), 84–91. doi:10.1016/j.mporth.2010.03.005.

69. Masouros, S. D., McDermott, I. D., Amis, A. A., & Bull, A. M. J. (2008). Biomechanics of the meniscus-meniscal ligament construct of the knee. *Knee Surgery, Sports Traumatology, Arthroscopy: Official Journal of the ESSKA*, *16*(12), 1121–32. doi:10.1007/s00167-008-0616-9.

70. McDermott, I. D., Masouros, S. D., & Amis, A. A. (2008). Biomechanics of the menisci of the knee. *Current Orthopaedics*, *22*(3), 193–201 doi:10.1016/j.cuor.2008.04.005.

71. McDevitt, Cahir A., & Webber, R. J. (1990). The ultrastructure and biochemistry of meniscal cartilage. *Clinical Orthopaedics and Related Research*, (252), 8–18.

72. McMurray, T.P., (1928). The diagnosis of internal derangements of the knee. In: The Robert Jones birthday volume. Humphrey Milford, London, pp 301-306.

73. Melzack, R. & Wall, P.D. (1965). Pain Mechanisms: A New Theory. *Science*, *150*(3699) doi: 10.1126/science.150.3699.

74. Meserve, B., Cleland, J., & Boucher, T. (2008). A meta-analysis examining clinical test utilities for assessing meniscal injury. *Clinical Rehabilitation*, *22*(2), 143-161 doi:10.1177/0269215507080130.

75. Metcalf, M. H., & Barrett, G. R. (2004). Prospective Evaluation of 1485 Meniscal tear patterns in patients with stable knees. *The American Journal of Sports Medicine*, *32*(3), 675–680. doi:10.1177/0095399703258743.

76. Miller, F.G. & Kaptchuk, T.J. (2008). The power of context: reconceptualizing the placebo effect. *Journal of the Royal Society of Medicine, 101*, 222-225 doi: 10.1258/jrsm.2008.070466.

77. Miller, G. K. (1996). A prospective study comparing the accuracy of the clinical diagnosis of meniscus tear with magnetic resonance imaging and its effect on clinical outcome. *Arthroscopy: The Journal of Arthroscopic & Related Surgery: Official Publication Of The Arthroscopy Association Of North America And The International Arthroscopy Association*, *12*(4), 406-413.

78. Mohan, B., & Gosal, H. (2007). Reliability of clinical diagnosis in meniscal tears. *International Orthopaedics*, *31*(1), 57-60.

79. Morrison, J. B. (1970). The mechanics of the knee joint in relation to normal walking. *Journal of Biomechanics*, *3*, 51–61.

80. Moss, P., Sluka, K., & Wright, A. (2007). The Initial Effects of Knee Joint Mobilization on Osteoarthritic Hyperalgesia. *Manual Therapy*, 12(2), 109-118 doi: 10.1016/j.math.2006.02.009.

81. Muellner, T., Weinstabl, R., Schabus, R., Vécsei, V., & Kainberger, F. (1997). The diagnosis of meniscal tears in athletes. A comparison of clinical and magnetic resonance imaging investigations. *The American Journal of Sports Medicine*, *25*(1), 7-12.

82. Mulligan, B. (1993). Manual Therapy Rounds: Mobilisations with Movement (MWM's). *The Journal of Manual & Manipulative Therapy*, *1*(4), 154-156.

83. Mulligan, B.(5th) (2004). Manual Therapy 'NAGS', 'SNAGS', 'MWMS'. Wellington, New Zealand: Plane View Services Ltd.

84. Mulligan, B. (6th) (2010). Manual Therapy NAGS, SNAGS, MWMS etc. Wellington, New Zealand: Plane View Services Ltd.

85. Nikolaou, V., Chronopoulos, E., Savvidou, C., Plessas, S., Giannoudis, P., Efstathopoulos, N., & Papachristou, G. (2008). MRI efficacy in diagnosing internal lesions of the knee: a retrospective analysis. *Journal of Trauma Management & Outcomes*, *2*(1), 4. doi:10.1186/1752-2897-2-4.

86. Nepple, J. J., Dunn, W. R., & Wright, R. W. (2012). Meniscal Repair Outcomes at Greater Than Five Years. *The Journal of Bone and Joint Surgery. American Volume*, *94*(24), 2222–2227. doi:10.2106/JBJS.K.01584.

87. O'Donoghue, D. H. (1980). Meniscectomy Indications and Management. *Physical Therapy*, *60*(12), 1617–1623.

88. Ossipov, M.H., Dussor, G.O. & Porreca, F. (2010). Central Modulation of Pain. *Clinical Journal of Investigation, 120*(11), 3779-3787. doi: 10.1172/JC143766.

89. Paungmali, A., O'Leary, S., Souvlis, T. & Vicenzino, B. (2003). Hypoalgesic and Sympathoexcitatory effects of mobilization with movement for lateral epicondylalgia. *Physical Therapy, 83,* 374-383. Retrived from: http://ptjournal.apta.org on Novemenber 27, 2014.

90. Paungmali, A., O'Leary, S., Souvlis, T., & Vicenzino, B. (2004). Naloxone fails to antagonize initial hypoalgesic effect of manual therapy treatment for lateral epicondylalgia. *Journal of Manipulative and Physiological Therapeutics*, 27(3), 180-185.

91. Paungmali, A., Vicenzino, B. & Smith, M. (2003). Hypoalgesia induced by elbow manipulation in lateral epicondylalgia does not exhibit tolerance. *The Journal of Pain, 4*(8), 448-454. doi: 10.1067/S1526-5900(03)00731-4.

92. Paxton, E. S., Stock, M. V., & Brophy, R. H. (2011). Meniscal Repair Versus Partial Meniscectomy: A Systematic Review Comparing Reoperation Rates and Clinical Outcomes. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*, *27*(9), 1275–1288. doi:10.1016/j.arthro.2011.03.088.

93. Peters, G., & Wirth, C. J. (2003). The current state of meniscal allograft transplantation and replacement. *The Knee*, *10*(1), 19–31. doi:10.1016/S0968-0160(02)00139-4.

94. Pettman, E. (2007). A History of Manipulative Therapy. *The Journal of Manual of Manipulative Therapy, 15*(3), 165-174. Retrieved From: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2565620/pdf/jmmt0015-0165.pdf.

95. Poynton, A. R., Javadpour, S. M., & Finegan, P. J. (1997). The meniscofemoral ligaments of the knee. *The Journal of Bone and Joint Surgery*, *79*(2), 14–17.

96. Prasad, V., Vandross, A., Toomey, C., Cheung, M., Rho, J., Quinn, S., Cifu, A. (2013). A Decade of Reversal: An Analysis of 146 Contradicted Medical Practices. *Mayo Clinic Proceedings*, *88*(8), 790–798. doi:10.1016/j.mayocp.2013.05.012.

97. Pujol, N., Barbier, O., Boisrenoult, P., & Beaufils, P. (2011). Amount of Meniscal Resection After Failed Meniscal Repair. *The American Journal of Sports Medicine, 39*(8), 1648–1652. doi:10.1177/0363546511402661.

98. Rath, E., & Richmond, J. C. (2000). The menisci: Basic science and advances in treatment. *British Journal of Sports Medicine*, *34*(4), 252–257. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1724227&tool=pmcentrez&rendertype=abstract.

99. Renstrom, P., Johnson, R. J. (1990). Anatomy and biomechanics of the menisci. *Clinics in Sports Medicine*, *9*(3), 523–538.

100. Rodkey, W. G. (2000). Basic biology of the meniscus and response to injury. *Instructional Course Lectures*, *49*, 189–193.

101. Roos, E. M., Roos, H. P., Ekdahl, C., & Lohmander, L. S. (1998). Knee injury and Osteoarthritis Outcome Score (KOOS)--validation of a Swedish version. *Scandinavian Journal Of Medicine & Science In Sports*, *8*(6), 439-448.

102. Rose, R. C. (2006). The accuracy of joint line tenderness in the diagnosis of meniscal tears. *The West Indian Medical Journal*, *55*(5), 323-326.

103. Scholten, R., Devillé, W., Opstelten, W., Bijl, D., van der Plas, C., & Bouter, L. (2001). The accuracy of physical diagnostic tests for assessing meniscal lesions of the knee: a meta-analysis. *The Journal of Family Practice*, *50*(11), 938-944.

104. Shultz, S., Nguyen, A.D., Windley, T.C., Kulas, A.S., Botic, T.L. & Bruce, D. (2006). Intratester and intertester reliability of clinical measures of lower extremity anatomic characteristics: Implication for multicenter studies. *Clinical Journal of Sport Medicine*, *16*(2), 155-161.

105. Sihvonen, R., Paavola, M., Malmivaara, A., Itälä, A., Joukainen, A., Nurmi, H., Järvinen, T. L. N. (2013). Arthroscopic Partial Meniscectomy versus Sham Surgery for a Degenerative Meniscal Tear. *New England Journal of Medicine, 369*(26), 2515–2524. doi:10.1056/NEJMoa1305189.

106. Slater, H., Arendt-Nielson, L., Wright, A., & Graven, N. (2006). Effects of a manual therapy technique in experimental lateral epicondylalgia. *Manual Therapy, 11 (2).* 107-117. http://dx.doi.org/10.1016/j.math.2005.04.005.

107. Takasaki, H., Hall, T., & Jull, G. (2013). Immediate and short-term effects of Mulligan's Mobilization with Movement on knee pain and disability associated with knee osteoarthritis: A prospective case series. *Physiotherapy Theory and Practice,* 29(2), 87-95. doi: 10.3109/09592985.2012.702854.

108. Teys, P., Bisset, L., & Vicenzino, B. (2008). The initial effects of a mulligan's mobilization with movement technique on range of movement and pressure pain threshold in pain-limiting shoulders. *Manual Therapy, 13(1),* 3742. http://dx.doi.org/10.1016/j. math. 2006.07.0 11.

109. Threlkeld, J.A. (1992). The effects of manual therapy on connective tissue. *Physical Therapy,* 72 (12), 893-902, Retrieved from: http://ptjournal.apta.org/content/72/12/893.full.pdf.

110. Timotijevic, S., Vukasinovic, Z., & Bascarevic, Z. (2014). Correlation of clinical examination, ultrasound sonography, and magnetic resonance imaging findings with arthroscopic findings in relation to acute and chronic lateral meniscus injuries. *Journal of Orthopaedic Science: Official Journal of the Japanese Orthopaedic Association, 19*(1), 71-76. doi:10.1007/s00776-013-0480-4.

111. Troupis, J. M., Batt, M. J., Pasricha, S. S., & Saddik, D. (2015). Magnetic resonance imaging in knee synovitis: Clinical utility in differentiating asymptomatic and symptomatic meniscal tears. *Journal Of Medical Imaging & Radiation Oncology, 59*(1), 1-6. doi:10.1111/1754-9485.12240.

112. Vedi, V., Spouse, E., Williams, A., Tennant, S.J., Hunt, D.M., & Gedroyc, M.W. (1999). Meniscal movement an in-vivo study using dynamic MRI. *Journal of Bone & Joint Surgery, 81*(1), 37-41.

113. Vela, L. I., & Denegar, C. R. (2010). The Disablement in the Physically Active Scale, part II: the psychometric properties of an outcomes scale for musculoskeletal injuries. *Journal of Athletic Training, 45*(6), 630-641. doi:10.4085/1062-6050-45.6.630.

114. Vicenzino, B. (2011, June). Mulligan's Mobilisation with Movement: The science, the evidence and the art. PowerPoint Presentation presented at the 2nd International Mulligan Concept Conference, Porto, Portugal.

115. Vicenzino, B., Hing, W., Rivett, D. & Hall, T (2011). Mobilisation with Movement The art and the science. Australia: Elsevier Australia.

116. Vicenzino, B., Paungmali, A., & Teys, P. (2007). Mulligan's mobilization-with-movement, positional faults and pain relief: Current concepts from critical review of literature. *Manual Therapy*, 12 (2), 98-108, doi: 10.1016/j.math.2006.07.012.

117. Voloshin, A. S., & Wosk, J. (1983). Shock absorption of meniscectomized and painful knees: a comparative in vivo study. *Journal of Biomedical Engineering*, *5*(2), 157–161.

118. Vundelinckx, B., Vanlauwe, J., & Bellemans, J. (2014). Long-term Subjective, Clinical, and Radiographic Outcome Evaluation of Meniscal Allograft Transplantation in the Knee. *The American Journal of Sports Medicine*, *42*(7), 1592–1599. doi:10.1177/0363546514530092.

119. Wareluk, P., & Szopinski, K. (2012). Value of modern sonography in the assessment of meniscal lesions. *European Journal of Radiology*, *81*(9), 2366-2369. doi:10.1016/j.ejrad.2011.09.013.

120. Werner, B. C., Holzgrefe, R. E., Griffin, J. W., Lyons, M. L., Cosgrove, C. T., Hart, J. M., & Brockmeier, S. F. (2014). Validation of an innovative method of shoulder range-of-motion measurement using a smartphone clinometer application. Journal of Shoulder and Elbow Surgery, 23(11), e275 http://doi.org/10.1016/j.jse.2014.02.030.

121. Willis, R. B. (2006). Meniscal injuries in children and adolescents. *Operative Techniques in Sports Medicine*, *14*(3), 197–202. doi:10.1053/j.otsm.2006.06.003.

122. Wilson, A. S., Legg, P. G., & McNeur, J. C. (1969). Studies on the innervation of the medial meniscus in the human knee joint. *The Anatomical Record*, *165*(4), 485–491.

CHAPTER 5

Innovative Treatment of Clinically Diagnosed Meniscal Tears: A Randomized Sham-Controlled Trial of the Mulligan Concept "Squeeze" Technique

By

Valerie Stevenson, Robinetta Hudson, Amy Richmond, Belinda Sanchez, Russell Baker, James May, Alan Nanypany

Abstract

Background

Meniscal tears are a common injury, often leading to surgery or lengthy conservative treatment. Arthroscopic surgery is currently the gold standard for treatment; however, this option may lead to subsequent surgeries and osteoarthritis prompting a need for alternative treatment options for meniscal tears.

Purpose

To assess the effects of the Mulligan Concept (MC) "Squeeze" technique compared to a sham technique in participants presenting with a clinically diagnosed meniscal tear.

Study Design

A multi-site randomized sham-controlled trial.

Methods

Participants (n=23) were recruited as a sample of convenience in a physically active and sedentary population, ranging from 14-62 (age = 24.91 ± 12.09) years of age, who reported common symptoms of a meniscal tear. Randomization ensured equal distribution of participants into either the MC "Squeeze" technique treatment group or the sham group. A maximum of 6 treatments were applied within a 14-day period for each treatment. Patients were assessed using the Numeric Pain Rating Scale (NRS), Patient Specific Functional Scale (PSFS), the Disablement in the Physically Active (DPA) scale and the Knee Injury Osteoarthritis Outcome Score (KOOS).

Results

All participants in the MC "Squeeze" group met the discharge criteria of ≤ 2 points on the cumulative NRS, ≥ 9 points on the PSFS, and ≤ 34 points on the DPA Scale for chronic and ≤ 23 for acute injuries at the end of the treatment intervention. A significant difference was found on the changes in PSFS scores ($F(1, 21) = 4.40$, $p = .048$, partial eta squared = .17,

observed power = .52) and DPA Scale scores at discharge ($F(1, 21) = 7.46$, $p = .013$, partial eta squared = .27, observed power = .74) between the two groups.

Conclusion

The results indicate the MC "Squeeze" technique had a positive effect on patient function and health-related quality of life over a period of 14 days that was clinically and statistically superior to the sham treatment.

Clinical Relevance

The MC "Squeeze" technique is an effective treatment for reducing symptoms associated with meniscal tears in a patient population meeting the criteria for a clinical diagnosis.

Key Terms

Meniscal Tears, Manual Therapy, Knee Pain, Rehabilitation

What is known about the subject

Arthroscopic surgery is currently the gold standard of care for meniscal tears, performed at a rate of 700,000 surgeries per year and costing patients 4 billion dollars a year. However, partial meniscectomy does not consistently produce the desired positive outcomes intended for meniscal tears. Furthermore, patients who undergo any type of meniscal surgery are at risk for requiring subsequent surgeries.

One theorized cause of the mechanism behind meniscus tears is tissue disruption or derangement. Classic symptoms of meniscus tears may appear due to the meniscal tissue becoming dislodged from its normal anatomical resting place, either acutely or over time in chronic cases. If the meniscus tissue to not relocated to its functional resting place, symptoms persist and function decreases; thus a treatment that is aimed at mobilizing the tissue towards the center of the knee joint could result in relief from symptoms.

What this study adds to existing knowledge

To our knowledge, this is the first multi-site, randomized sham-control trial which examined the effects of the MC "Squeeze" technique on participants presenting with clinically diagnosed meniscal tears. By detailing the effects of the MC "Squeeze" Technique, the outcomes of this study lend further support that the technique is effective at resolving the symptoms of meniscus tears.

Introduction and Background

The incidence of lower body injury, especially knee injuries, has grown[45,59] due to increased participation in recreational sports[24,53] and intercollegiate athletic competition.[27] Meniscal tears commonly occur as a result of sport participation[45] and, in a 10 year epidemiologic study on the occurrence of knee injuries, researchers found meniscus tears were the second most common knee injury.[45] Meniscal injuries are not only common in the young, athletic population; 35% of adults over the age of 50 experience degenerative tears.[34]

Injuries to the meniscus are often the result of compressive forces placed on the meniscus by the tibia and femur during flexion and rotation during weight bearing.[46] A meniscal tear can affect critical functions of the meniscus, such as joint congruency, load transmission, and shock absorption[22,38] leading to the classic signs and symptoms of a meniscal tear: catching, locking, or clicking; joint line pain; and a feeling of "giving out" or instability.[39] Despite the importance of the meniscus tissue for function, incidental findings of asymptomatic tears on magnetic resonance imaging (MRI) are relatively common,[40,62,73,70] suggesting the presence of a meniscal tear does not directly correlate to knee disability. In theory, patients with meniscal tears may not seek medical treatment if physical symptoms that would indicate injury or pathology are not being experienced. Therefore, the presence of meniscal lesions on MRI findings may not equate to the pathology being the root cause of dysfunction.[62,73]

When a meniscus tear is diagnosed, treatment options are typically categorized as surgical, involving partial meniscectomy or meniscal repair, or non-surgical, which is defined as conservative therapy.[47] Arthroscopic surgery currently remains the proposed gold standard for treatment of meniscal tears. Arthroscopic partial meniscectomy (APM) is often a more attractive surgical option for patients due to shorter post-surgery rehabilitation time-lines.[20] An APM occurs in as many as 61 per 100,000 meniscal tears[23] and approximately one-third of patients who exhaust conservative care will go on to have a meniscectomy to decrease pain and increase function.[47] Although patients elect to have APM more often, the APM procedure has inconsistent results for alleviating the symptoms of meniscal tears[54,50,41,34,63] and 50% of patients who undergo APM develop knee OA symptoms confirmed by radiographic images years after surgery.[16,17,19,20] Furthermore, the severity of symptoms and the extent of cartilage damage seen on imaging in patients who underwent APM is worse than the damage observed in cases of degenerative meniscus tears.[16,17,19,20]

Preservation of the meniscus through arthroscopic surgical repair is considered the most ideal option;[20] however, failure rates have been reported as high as 42% following those procedures[23] and the risk for subsequent surgeries is as high as 20%.[52] Consequently, patients who undergo any type of meniscal surgery are at risk for requiring subsequent surgeries,[52] which suggests clinicians should exhaust conservative care options for meniscus tears before pursuing surgical options.[26]

Recommendations for conservative therapy for meniscus tears commonly includes active exercises focused on increasing range of motion (ROM) and muscle strength while improving balance and flexibility.[26,47] Although conservative therapy protocols are recommended as an alternative to surgery,[26,34,30] lengthy timelines[47] and poor outcomes[26,34,30] may make those protocols less appealing to patients. Time commitment for conservative care has been reported to be between 8 and 10 weeks with patients performing therapeutic exercises 3 times a week or more[47] and no significant difference was found between the immediate and long-term outcomes of partial meniscectomy and conservative therapy in middle aged patients with degenerative medial meniscal tears,[26,34,30] Because reported outcomes of surgery and conservative care are similar and have inconsistent results,[26,34,30] there is a need for research into non-operative alternative treatment methods for treating the symptoms of meniscal tears.

The Mulligan Concept (MC) is a manual therapy paradigm with specific techniques theorized to address the symptoms associated with meniscal tears.[49] One of those techniques, the MC “Squeeze” technique, is designed to treat range of motion deficits and pain localized to the joint line of the knee during movement.[49] Such symptoms are often reported in the presence of meniscal tears due to altered joint mechanics and function caused secondarily by the disruption of meniscal tissue.[4] If meniscal tissue is dislodged or subluxed from its normal anatomical position after a tear, the disrupted tissue may cause increased pressure on the highly innervated periphery of the meniscus tissue and result in the commonly reported symptoms.[55,38,71,15] Conceivably, to alleviate the pain and dysfunction resulting from the tissue disruption, the abnormal pressure on the periphery of the meniscus and the pain-sensitive anterior capsular structures need to be resolved. Within the MC, it has been proposed that relocating the tissue towards the midline of the joint would reduce pain because the periphery of the menisci would no longer send pain signals.[49] The MC “Squeeze” technique may produce this benefit through the application of a therapeutic pressure to the meniscus.[49] Pressure is applied through a “squeezing” force on the meniscus at the most tender/swollen point along the joint line while the patient actively flexes and extends their knee to mobilize the tissue back to its normal anatomical position.[49]

The MC “Squeeze” technique has produced favorable patient outcomes for clinically classified meniscal tears in anecdotal reports and published a priori case studies.[5,29] In these reports, patients reported positive changes in pain, function, disability, and psychosocial well-being on patient reported outcome measures; however, the small sample size and lack of comparison groups necessitates the need for further investigation to determine the effectiveness of the MC “Squeeze” technique. Therefore, the purpose of this study was to assess the effects of the MC Squeeze technique compared to a sham technique in participants presenting with a clinically diagnosed meniscal tear.

Methods

Study Design

The present study was a multi-site randomized sham-controlled trial, designed to be conducted across four clinics with four clinician-researchers providing treatment. Clinical experience among the clinician-researchers ranged from 3-10 years (mean = 6.5 ± 2.89 years), but each had equal experience and training in the MC. Prior to beginning this study, the clinicians all completed two accredited MC courses together and had one year of experience in applying the MC in patient care. Additionally, a training session was conducted in-person with the four clinician-researchers to review methods prior to commencing the study. The training involved the review of all inclusion/exclusion orthopedic tests and dependent variables, and the verification of MC "Squeeze" technique application by a certified MC teacher with over 20 years of experience within the MC.

The Institutional Review Boards at the four clinical sites approved the application of treatment and collection of medical information from the participants in this study. Participant recruitment took place between October 2015 and March 2016. Participants signed written informed consent acknowledging possible publication of de-identified outcomes, and consent/assent forms were collected from all minors participating in this study.

Participant Selection

Participants were recruited as a sample of convenience of physically active and sedentary participants, ranging from 14-62 years of age. Any participant who reported any of the common symptoms of a meniscal tear with various mechanisms of injury or onset of symptoms (i.e., acute and chronic) was considered for participation in this study at each clinical site. Participants were screened by the clinician-researchers using an extensive medical history, common knee orthopedic tests, muscle/strength integrity, and range of motion (ROM) assessments.

Inclusion criterion were a positive finding in a minimum of three of the following: McMurray's test, pain with maximal knee flexion, pain with maximal knee extension, joint line tenderness, and a history of clicking and/or popping.[39] The preceding inclusion criteria ware formed according to the clinical composite score (CCS) developed by Lowery et al.[39] (Table 3.1). When three of the signs were present, the CCS had a specificity of 90.2% and a positive prediction value (PPV) of 76.7%;[39] in comparison, an MRI has a specificity of 69-93.3%[10,51] and a PPV of 80.4-83.2%[10] for meniscal tears. Participants were also required to present with a positive finding in a minimum of one of the following orthopedic tests: Apley's compression and distraction (specificity = 90%);[31] and Thessaly's performed at 20 degrees of knee flexion (specificity = 96-97%).[37] Exclusion criteria were the presence of knee comorbidities, such as anterior cruciate ligament (ACL) tears, knee contusion, fracture, knee dislocation, other knee ligament instability, and non-mechanical causes of pain (e.g., hyperalgesia).

Randomization

An a priori randomization was designed to ensure equal distribution of participants into either the MC "Squeeze" technique treatment group or the sham group. Participant numbers were randomly generated prior to the commencement of the study and assigned prior to clinical exam. Each clinician-researcher was assigned a set of participant numbers consisting of an equal distribution of participants to treatment groups. If a participant was disqualified based on the results of their clinical exam, the participant number was assigned to the next eligible participant.

Outcome Measures

Patient outcome measures were collected to track participant progress and treatment effects. Patient outcomes included the Numeric Pain Rating Scale (NRS), the Patient Specific Functional Scale (PSFS), the Disability in the Physically Active (DPA) Scale, and the Knee injury and Osteoarthritis Outcome Score (KOOS). Cumulative NRS and PSFS were collected at intake, daily pretreatment, and 24-hours after the final treatment. Current NRS and PSFS scores were also collected daily after each treatment intervention. The DPA Scale and KOOS were only collected at intake and 24-hours after the final treatment.

Numeric Rating Scale (NRS)

Participant reported level of pain was measured using the NRS. The NRS is a patient-oriented scale used among various patient populations.[35] The NRS is scored on an 11-point scale, with 0 representing no pain and 10 representing severe pain.[11] Cumulative NRS is calculated as an average of the current, best, and worst pain scores over the past 24 hours. The reported minimal clinically important difference (MCID) for the NRS is a decrease of 2 points or 33%.[61]

Patient Specific Functional Scale (PSFS)

Participant function was measured using the PSFS. The PSFS is a patient-oriented tool that assesses the patient's perception of their current functional ability.[64] The participant is asked to list up to three activities which are affected by their injury and rate their perceived ability to perform each activity on a scale from 0 (unable to perform the activity) to 10 (able to perform the activity at the same level as before the injury occurred). For this study, each participant was asked to identify the single activity most affected by his or her knee injury and rate it using the PSFS 11-point scale. The same activity was used to assess PSFS throughout the duration of the study. The reported minimal detectable change (MDC) is a change in 2.5 points when using an individual activity in participants with a lower limb injury.[8]

Disablement in the Physically Active (DPA) Scale

Participant physical impairment, functional limitation, disability, and health-related quality of life[68] were measured using the DPA Scale. The DPA Scale is a questionnaire in which responses are based on a scale ranging from 1 (no problem) to 5 (severe problem) across 16 items; 16 points are subtracted from the total to create a total possible score range from 0 to 64 points.[68] A normal, healthy range has been observed to be a score of less than 35, and a score of 23 or less has been observed in participants deemed ready to return to full participation after injury by an athletic trainer or physician.[68] The MCID is a decrease of 9 points for an acute injury and 6 points for a chronic injury.[68]

Knee Injury Osteoarthritis and Outcome Score (KOOS)

The KOOS is a questionnaire designed for patients suffering from a knee pathology often associated with osteoarthritis, including ACL tears, meniscal tears, and chondral lesions. The tool includes questions regarding pain, symptoms, and functional limitations in activities of daily living and sport/recreation, as well as quality of life. Responses within each dimension are based on a scale ranging from 0 to 4; a total score of 100 would indicate no symptoms.[58] The MCID for each subsection is a change of 8-10 points.[58] However, an MCID value has not been established for $KOOS_{5,}$[58] which is a composite score of all five subsection scores.

Treatment Interventions

Treatment and participant position began in the same position that elicited knee symptoms during assessment, which was either supine/non-weight bearing (NWB), partial weight bearing (PWB), or full weight bearing (FWB)[49] for both treatment options.

Mulligan Concept "Squeeze" Intervention

The clinicians placed themselves in a position of biomechanical advantage based on each participant's individual treatment position. The participant actively placed the involved knee in approximately 90 degrees of flexion (allowing access to the joint line) or to the participant's pain-free limit of flexion in NWB. The clinician then placed the medial border of one thumb (i.e., the contact thumb) on the site of maximum pain and/or joint line edema (i.e., joint line tenderness), while the other thumb (i.e., the mobilizing thumb) was used to apply a force through the first thumb in an overlapping manner (Figure 3.1). Next, the participant extended their knee through their pain-free range, while the clinician maintained contact force with thumbs, releasing the force as the joint space closed in maximal knee extension (Figure 3.2). The participant then performed active knee flexion as the clinician continued to apply a "squeezing" force towards the center of the joint until maximal knee flexion was reached (Figure 3.3). The clinician held the pressure at the joint line for two seconds as the participant applied overpressure by pulling their tibia with both hands to their end range of knee flexion (Figure 3.3). If a participant could not grasp their tibia, they were given a strap to assist them into flexion (Figure 3.4). The participants returned to their end-range of knee extension, while the clinician released the force as the joint space closed. The participants were allowed to experience localized discomfort from the overlap grip, but the localized discomfort was not exacerbated with movement.

When participants were restricted in flexion, they were asked to perform active knee flexion only (Figure 3.3). Participants, who were restricted in extension, were asked to perform active knee extension only (Figure 3.2). Participants, who were restricted in both flexion and extension, were asked to perform knee flexion first, followed by knee extension. The treatment consisted of three sets of 10 repetitions with a minimum of 30 seconds of rest between each set. As the participants progressed towards full weight bearing, the participant position during treatment application also progressed from supine to partial weight bearing (Figure 3.5) to full weight bearing (Figure 3.6). Each participant was monitored for any increase in pain throughout the technique in accordance with MC treatment principles.

Sham Intervention

The “sham” treatment followed the same protocol as the MC “Squeeze” group (i.e., flexion/extension movement pattern was consistent) with the exception of the hand placement and the force. The hand placement for the sham treatment consisted of the same overlap grip of the thumbs, but the clinician applied the “squeeze” a ½ inch below the point of maximal joint line tenderness (Figure 3.7, 3.8). To provide consistent force using the sham treatment across treatment applications and participants, the clinician used only enough force to blanch the nail bed of the reinforcing thumb when applying the “sham” treatment.

Treatment Application Protocol

The protocol consisted of a maximum of 6 treatments within a 14-day period. Treatment applications were separated by a minimum of 24 hours and a maximum of 72 hours between each treatment session. If participants reached discharge criteria prior to the sixth treatment, they could be discharged successfully from the study prior to completing all 6 treatments; a minimum of 24 hours was required after the last treatment to assess a participant for discharge. Participants were not restricted from any activities of daily living and were allowed to participate as tolerated (based on clinical presentation and clinician assessment) in any specific sport activities throughout the duration of this study.

Discharge Criteria

The discharge criteria for both treatment groups included: a PSFS score of nine or higher for the reported patient-specific activity, a cumulative NRS score of two or less (with no greater than a one on current pain), and a DPA Scale score of 34 or less for persistent/chronic injuries and 23 or less for acute injuries. Participants were discharged from the study once they reached the predetermined criteria and maintained the outcomes a minimum of 24 hours post treatment.

Data Analysis

Descriptive statistics (mean ± SD) were calculated for all participant demographics. Using NRS, PSFS, DPA, and KOOS scores from a pilot study, an a priori power analysis using G power determined a minimum of 16 participants would be required for this study. A series of one-way analyses of variance (ANOVAs) was performed on the NRS and PSFS scores due to the variance in baseline scores between each group (i.e., linearity and homogeneity of regression did not exist). A series of one-way analyses of covariance (ANCOVAs), with baseline scores as the covariate, was performed on DPA Scale and $KOOS_5$ scores. Patient outcomes on NRS and PSFS were used to assess the effect of each intervention after a single treatment, and NRS, PSFS, DPA, and $KOOS_5$ were used to assess the effect of each treatment intervention after final treatment. Mean differences, ± standard deviation (SD), were calculated with statistical significance set at $p \leq 0.05$, confidence intervals (CI) at 95%, and partial eta squared values: small = 0.02, medium = 0.13, and large = 0.26.[9] All data analyses were performed using Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL, USA) version 23.0.

Results

Participant Demographics

Twenty-eight participants (males = 14, females = 14) qualified for this study. Five participants elected to withdraw prior to reaching discharge criteria in the allotted 14-day period. Two participants withdrew due to the time constraints of the study (MC “Squeeze” group = 1, sham group = 1), two sustained additional injuries (sham = 2), and the last did not offer a reason (sham = 1). The remaining 23 participants (age = 24.91 ± 12.09, males = 11, females = 12) were included in the final data analysis. The MC “Squeeze” group was composed of 12 participants (acute = 6, chronic = 6) and the sham group was composed of 11 participants (acute =3, chronic = 8). Participants were generally healthy (i.e., no general medical or orthopedic comorbidities) with a mean BMI of 28.48 ± 5.35, from both athletic and general populations (MC “Squeeze” BMI = 25.98 ± 5.62, Sham BMI = 26.35 ± 5.17; Table 3.2). The results of each participant’s clinical exam are presented in Table 5.3.

Numeric Rating Scale Outcomes

A univariate ANOVA was used to assess the change in current pain between the MC “Squeeze” and sham groups immediately after the first treatment. No significant difference was found ($F(1, 21) = .006$, $p = .938$, partial eta squared = .000, observed power = .051) between the two groups. The MC “Squeeze” group reported a mean reduction on current NRS of 1.56 ± 1.01 after a single treatment, while the sham group reported a mean reduction of 1.30 ± 1.51.

A univariate ANOVA revealed no significant difference in cumulative pain scores between the MC “Squeeze” and sham groups after the final treatment ($F(1,21) = 1.70$, $p = .21$, partial eta squared = .075, observed power = .24) (Table 5.1). However, the MC “Squeeze” group reported a mean reduction on cumulative NRS of 2.19 ± 1.00 effectively meeting the MCID of 2 points for NRS,[61] while the sham group only reported a mean reduction of 1.24 ± 2.31 (Table 3.4). All 12 (100%) participants in the MC “Squeeze” group met the discharge criteria of ≤ cumulative 2 points on NRS at the end of the treatment intervention, while only 4 (36%) of the 11 sham participants met the discharge criteria for NRS.

Patient Specific Functional Scale Outcomes

A univariate ANOVA was used to assess the change in PSFS scores between the MC “Squeeze” and the sham groups immediately after the first treatment. A significant difference was found ($F(1, 21) = 4.40$, $p = .048$, partial eta squared = .17, observed power = .52) between the two groups. The MC “Squeeze” group reported a mean improvement of function on PSFS of 1.58 ± 2.69 after a single treatment application, while the sham group reported a mean reduction of .46 ± 1.86. Four (33%) participants in the MC “Squeeze” group reported an MDC on the PSFS after the first treatment while no participants in the sham group reported clinically meaningful improvements in function.

A univariate ANOVA revealed a significant difference in the change in PSFS scores between the MC “Squeeze” and the sham groups after the final treatment ($F(1, 21) = 41.92$, $p < .001$, partial eta squared = .67, observed power = .10) (Table 3.4). After the final treatment, the MC “Squeeze” group reported a mean change on PSFS of 5.83 ± 1.85, twice the MDC of 2.5 for PSFS,[8] while the sham group only reported a mean change of .55 ± 2.07 (Table 3.4). All 12 (100%) participants in the MC “Squeeze” group reported a PSFS score equal or greater than 9 points after final treatment, while only 4 (36%) of the 11 sham participants reported equivalent PSFS scores, and produced a moderate effect size.[9]

Disablement in the Physically Active Scale Outcomes

A univariate ANCOVA, with baseline scores set as the covariate ($p < .001$), revealed a significant difference in DPA Scale scores between the MC “Squeeze” and sham groups after the final treatment ($F(1, 21) = 7.46$, $p = .013$, partial eta squared = .27, observed power = .74) (Table 3.4). The mean difference in DPA Scale scores between the two groups was 8.78 ($p = .013$, 95% CI: -15.48, -2.08). After the final treatment, the MC “Squeeze” group reported a mean DPA Scale score of 9.00 ± 8.12, 14 points below the accepted “return to play” score of 23,[68] while the sham group reported a mean score of 18.55 ± 14.05 (Table 5.4). The mean change for the MC “Squeeze” group was 14.92 ± 7.68, more than twice the mean change of the sham group (mean change = 6.36 ± 8.15) (Table 3.4).

Knee Injury Osteoarthritis and Outcome Scores

A univariate ANCOVA, with baseline scores set as the covariate ($p < .001$), did not reveal a significant difference in $KOOS_5$ scores between the MC "Squeeze" and sham groups after the final treatment ($F(1, 21) = 2.11$, $p = .16$, partial eta squared = .095, observed power = .28) (Table 3.4). The mean difference in $KOOS_5$ scores between the two groups was 6.23 ($p = .16$, 95% CI: -2.73, 15.19). However, after final treatment, the MC "Squeeze" group reported a mean $KOOS_5$ score of 79.32 ± 15.23, while the sham group only reported a mean score of 69.84 ± 13.69 (Table 3.4). The mean change for the MC "Squeeze" group was 13.82 ± 10.94, more than the mean change of the sham group (mean change = 9.07 ± 11.13) (Table 5.4). Five (42%) of the 12 participants in the MC "Squeeze" group reported $KOOS_5$ scores of ≥ 80/100 points by the end of the treatment intervention, while only 2 (18%) of the 11 sham participants reported equivalent scores.

Discussion

Participants among both treatment groups in this randomized sham-controlled study experienced positive effects, but the results suggest the improvements reported by the MC "Squeeze" group were superior overall. All 12 participants in the MC group met discharge criteria within the 14-day, 6 treatment restriction; whereas only 4 sham participants ($n = 11$) met discharge criteria within the research timeframe. Additionally, 42% (n=5) of the MC "Squeeze" participants displayed a full resolution of positive findings on a clinical exam; 58% (n=7) continued to display up to two positive findings, despite self-reporting as asymptomatic (Table 3.3). In comparison, none of the sham participants displayed a full resolution of positive findings on a clinical exam (Table 3.3).

A significant difference was not found between groups on the NRS; both groups reported a decrease in pain immediately after the first treatment and over the course of treatment. However, there is a possibility of a type II error occurring in the interpretation of this analysis. The analysis of change in pain scores yielded a low power (0.051) immediately after the first treatment and a low power (0.24) from intake to discharge. The lack of significant difference between the groups on the NRS at any point during the study may be attributed to higher intake scores and more variability in pain for the sham group. Lower mean NRS scores at intake for the MC "Squeeze" group afforded less room for improvement compared to the sham group during the course of treatment; thus, a "floor/ceiling" effect for the MC group may have limited the ability to detect a statistically significant difference between groups. A notable clinical difference, however was found between groups; after the first treatment, 50% of participants in the MC "Squeeze" group reported an MCID on the NRS, while only 36% of participants in the sham group reported equivalent results. Furthermore, 100% of the MC "Squeeze" group reported NRS scores of 1 or less at the completion of the study, as opposed to only 36% of the sham group.

Analysis of the PSFS scores revealed a statistically significant difference between the two groups, immediately after the first treatment and over the course of treatment, in favor of the MC "Squeeze" group. In addition, the MC "Squeeze" group experienced clinically significant improvements (i.e., MDC) immediately after the first treatment and over the course of treatment on the PSFS. It is possible the sham group experienced a "floor/ceiling" effect due to a smaller window for improvement with mean PSFS scores at baseline of 6.45 ± 1.57 as compared to the MC "Squeeze" group's mean baseline scores of 3.67 ± 1.72; however, further consideration of the outcomes suggests the MC group experienced superior outcomes to the sham group. For example, none of the sham patients reported an MDC on the PSFS after the first treatment, whereas 33% of the MC "Squeeze" group did. Moreover, 100% of the participants in the MC "Squeeze" group reported a PSFS score of 9 or better over the course of treatment as compared to just 36% of the sham. Thus, the differences between the MC "Squeeze" group and the sham group suggest the MC "Squeeze" technique may have had advantageous effects in alleviating the functional activity symptoms associated with clinically diagnosed meniscal tears compared to the sham intervention. In addition to improving functional activity, the MC "Squeeze" treatment also improved the group's perception of their disability as reported in their DPA Scale scores. A statistically significant difference was found between the MC "Squeeze" group and the sham group over the course of treatment. The MC "Squeeze" group reported lower scores on the DPA Scale, with 100% of participants reporting scores of less than 23 points by the end of the treatment intervention. In contrast, only 55% of the sham participants reported scores of less than 23 points. A score below 23 is clinically relevant for the participants in this study because it is indicative of normative values reported after discharge from treatment for an acute injury and would also fall within the published normal, healthy range (0-34 points) for uninjured people.[68]

A statistically significant difference between groups was not found on the $KOOS_5$. The lack of significant difference between the MC "Squeeze" and sham groups could be due to the $KOOS_5$ inquiring about symptoms within the past week. The timeframe of this study was two weeks and the $KOOS_5$ was administered within 24 to 72 hours of the participants reporting being symptom-free or completing the 6 treatment sessions. Although a number of participants were asymptomatic (e.g., pain resolved, etc.) at the time of $KOOS_5$ administration, it is a possible that participants may have still been symptomatic within the week the final KOOS questionnaire was completed, which may have led to depressed scores. It is also worth noting that there was a moderate effect size and a low power for the $KOOS_5$ analysis; thus, it is possible a Type II error is being committed by accepting that there is no difference between groups.

One potential reason for the positive effects experienced by the MC "Squeeze" group is the treatment's theorized effect on the meniscal tissue.[48,49] After meniscal injury, meniscal tissue can become dislodged from its normal anatomical position,[55,38,71,15] defined as meniscal derangement.[60] Tissue derangement has been theorized to contribute to approximately 42% of all knee pain.[43] In the presence of tissue derangement at the knee, pressure may be placed on the highly innervated joint line structures.[55,38,71,15] Hypothetically, the MC "Squeeze" technique repositions the deranged meniscal tissue into its normal anatomical position and therefore alleviates the symptoms commonly associated with meniscal tears.[48,49] However, these ideas remain purely theoretical, as there is a paucity of research available on the tissue derangement model in the extremities.[60]

The positive effects experienced by the sham group also cannot be ignored. Approximately 36% of the sham group experienced symptom improvement that qualified those patients for discharge from the study. Additionally, the majority of the sham group experienced some positive effects on most outcome instruments. The positive effects in the sham group could be attributed to the resemblance of our sham treatment to the repeated directional preference movements in the Mechanical Diagnosis and Therapy (MDT) paradigm. The MDT paradigm involves the classification of patients according to how their symptoms respond to repetitive or sustained unidirectional movements, the most common of which is a "derangement syndrome."[25,44,14,43,60,2] Derangement is defined as an anatomical disturbance in the normal resting position of a joint.[3,25,44,43,60] Patients with a reducible derangement will present a directional preference during the MDT evaluation.[3,25,44,43,60] While the MDT evaluation method was not followed in this study, it was possible that sham participants experienced improvements, or even complete abolishment of symptoms, due to the "sham" treatment resulting in applied repeated motion in a directional preference. Patients classified with a knee derangement have experienced significantly better outcomes in pain and function when compared to a control group.[60]

The positive effects achieved by the sham group could also be attributed to the psychological mechanisms of the placebo effect. The magnitude of the placebo effect depends largely on patient expectation.[21,33,67] The participants in this case series were blinded to the intervention that they received. As a result, patient outcomes may have improved based on the participant's expectation of being randomized into the treatment group. The positive effects reported by our sham participants are comparable to other placebo-controlled studies in which participants are told they will either receive a treatment or a placebo and results in small, but significant improvements in pain with small effects sizes.[28] Additionally, the sham participants that reached discharge criteria is not a new phenomenon; the placebo effect has been attributed to up to 50% of patients reaching discharge criteria, particularly in manual therapy.[6] While placebos may not alter the pathophysiology, they can alleviate symptoms (e.g. patient-reported pain).[33] Different types of manual therapies or therapeutic touch elicit various mechanisms of pain control associated with Central Nervous System (CNS) descending pain modulation including, but not limited to, an increase in β-endorphins, serotonin meditation, increases in dopamine production and oxytocin mediation.[69] Therefore, the placebo effect could explain why some participants experienced improvements in symptoms but most participants did not experience the significant improvements in functional activity and disability reported by the MC "Squeeze" group.

One limitation of this study was the inclusion of a relatively small sample size for generalization across all patient populations suffering from meniscal tears. Power was calculated based on pilot data of a 5-participant sample and, although the minimum sample size (n = 16) was surpassed in this study, a larger sample size including a more diverse patient population would allow for greater generalization to clinical practice. A larger sample size is also likely necessary in this study due to the number of scales used and is evident in the low power, but moderate effect size noted on certain outcomes measures (e.g., $KOOS_5$). Specifically regarding the KOOS, there was a limitation in study design because the final data collection was 24 hours post symptom resolution and/or sixth treatment intervention and the scale requires patients to analyze symptoms over the past week when symptoms may have still been present. Therefore, a true analysis of improvement on the KOOS may not have occurred with the study design.

Other limitations included difficulty determining a true sham/placebo (i.e., sham was similar to MDT) treatment in manual therapy, a lack of clinician blinding, a lack of arthroscopy for the confirmation of meniscal tears, and not controlling for each participants' activity during the course of treatment. Additionally, in participant recruitment of an injured population within the confines of the researcher's individual clinics, equal numbers of acute and chronic patients could not be obtained or equally distributed with the a priori randomization (Table 3.2). Lastly, the MC guidelines recommend applying an internal rotation accessory glide of the tibia when treating patients with general knee pain, and to then progress to medial/lateral glides of the tibia, to provide the greatest reduction in symptoms.[49] Thus, results reported in this study may have been further improved by determining which MC technique was best for each individual participant or through utilizing multiple interventions within the MC.

Future research on the effects of the MC "Squeeze" technique should include sub-classification of participants (e.g., acute versus chronic mechanism, etc.) prior to randomization. Because most of the participants included in this study were younger athletic patients with BMIs below the obesity level, additional research assessing older, sedentary individuals with higher BMIs would be advantageous because chronic degenerative meniscus tears are typically observed in populations who are older, sedentary, and overweight.[23,72] Additionally, the MC paradigm includes various other treatments for knee pain in addition to the "Squeeze" technique and contains recommendations to attempt multiple treatment interventions to match the patient to an intervention that abolishes pain during treatment as opposed to limiting rehabilitation to one technique for all patients.[47,48] Therefore, future research on the effects of the MC in the treatment of meniscal tears should be conducted by following the complete MC treatment guidelines and utilizing the full treatment paradigm; it will also be useful to compare the MC to traditional conservative rehabilitation protocols as opposed to a sham intervention. Researchers should also wait a week after the final treatment to collect the KOOS outcomes measure, as it is designed to capture patient symptoms over the course of a week. Finally, future research should include follow-up data (short-term and long term), identifying the time frames improvements are maintained following a return to sport or activities of daily living.

Conclusion

The results in this study indicate the MC "Squeeze" technique had a positive effect on patient function over a period of 14 days that was, in general, clinically and statistically superior to the sham treatment. While participants in both groups experienced a decrease in pain, only the MC "Squeeze" group reported a significant increase in functional activity and decrease in disability. The results in this study indicate that the MC "Squeeze" technique is an effective treatment for reducing symptoms associated with meniscal tears in a patient population meeting the criteria for a clinical diagnosis.

Tables

Table 3.1. Positive Findings for the Clinical Composite Score Proposed by Lowery et al. (2009) for the Detection of Meniscal Tears.

	5 Positive Findings	**4 Positive Findings**	**3 Positive Findings**
Sensitivity (%)	11.2%	16.86%	30.8%
Specificity (%)	99%	96.1%	90.2%
PLR	11.45%	4.29%	3.15%
PPV	92.3%	81.8%	76.7%

Note: PLR = Positive Likelihood Ratio; PPV = Positive Predictive Value

Table 3.2. Participant demographic data for the MC "Squeeze" and sham group

Participant ID #	Gender	Age	Sport/Activity	BMI	Onset (Duration of Symptoms)	Joint Line Point of Treatment
101	Male	45	Football Coach	35.6 BMI	Chronic	Medial
102	Male	23	Football	32.8 BMI	Chronic	Medial
103	Female	53	General Population	24.0 BMI	Chronic	Lateral
104	Male	22	Soccer	24.3BMI	Chronic	Medial
105	Male	20	Baseball	32.5 BMI	Acute	Medial
106	Male	21	Track & Field	23.6 BMI	Acute	Lateral
107	Male	14	Basketball	18.5 BMI	Acute	Medial
108	Female	18	Dance	29.9 BMI	Chronic	Lateral
109	Female	21	ROTC	24.0 BMI	Acute	Medial
110	Female	25	Swim Coach	26.8 BMI	Acute	Medial
111	Female	20	Basketball	21.30BMI	Chronic	Medial
112	Male	16	Soccer	18.5 BMI	Acute	Lateral
113*	Male	33	Football/Track Coach	23.0 BMI	Chronic	Lateral
114*	Male	19	Baseball	25.7 BMI	Chronic	Lateral
115*	Female	20	Soccer	24.4 BMI	Chronic	Medial
116*	Female	19	Cross Country	20.4 BMI	Acute	Medial
117*	Male	23	Football	31.0 BMI	Acute	Medial
118*	Female	19	ROTC	24.1 BMI	Acute	Lateral
119*	Female	18	Recreational Basketball	21.3 BMI	Chronic	Medial
120*	Female	21	General Population	35.2 BMI	Chronic	Medial
121*	Female	62	General Population	30.4 BMI	Chronic	Posterior Lateral
122*	Male	23	General Population	33 BMI	Chronic	Lateral
123*	Female	18	Recreational Basketball	21.3 BMI	Chronic	Medial

*= Sham Treatment Group

Table 3.3. Signs and symptoms present among all participants at intake and discharge/after the 6 treatments.

Sign/Symptoms	**MC ‘Squeeze” Group (n=12)**		**Sham Group (n=11)**	
	Intake	*Final Treatment*	*Intake*	*Final Treatment*
	n (%)	n (%)	n (%)	n (%)
History of Popping/Clicking	10 (83.33)	2 (16.67)	9 (81.82)	9 (81.81)
JLT	12 (100)	4 (33.33)	11 (100)	8 (72.73)
Pain in TKE	6 (50)	0 (0)	6 (54.55)	6 (54.55)
Pain in TKF	11 (91.17)	0 (0)	10 (90.90)	6 (54.55)
Positive McMurray’s Test	11 (91.17)	2 (16.67)	10 (90.90)	8 (72.73)
Positive Thessaly’s Test	10 (83.33)	0 (0)	11 (100)	6 (54.55)
Positive Apley’s Test	5 (41.67)	0 (0)	2 (18.18)	2 (18.18)
Edema	0 (0)	0 (0)	1 (9.09)	1 (9.09)
NWB/PWB	3 (25)	0 (0)	1 (9.09)	0 (0)

MC = Mulligan Concept; JLT = joint line tenderness; TKE = terminal knee extension; NWB = non weight-bearing; PWB = partial weight-bearing

Table 3.4. Analysis of variance (ANOVA) in outcome measures from intake to final treatment between groups

Outcomes	**MC 'Squeeze" Group M (±SD)**		**Sham Group M (±SD)**		p	Effect Size	Power
	Intake	*Final Treatment*	*Intake*	*Final Treatment*			
NRS (Avg)	2.64 (±.89)	0.44 (±.44)	3.67 (±2.50)	2.42 (±1.96)	.206	.075	.238
PSFS	3.67 (±1.72)	9.50 (±1.85)	6.45 (±1.57)	7.00 (±2.07)	.000*	.666*	1.00*
‡DPA	23.92 (±10.05)	9.00 (±8.12)	24.91 (±11.96)	18.55 (±14.05)	.013*	.272*	.739
‡KOOS$_5$	65.50 (±12.26)	79.32 (±15.22)	60.76 ±18.32)	69.84 (±13.69)	.162	.095	.282

MC = Mulligan Concept; NRS = Numeric Rating Scale for pain; Avg = average; PSFS = Patient Specific Functional Scale; DPA = Disablement in the Physically Active Scale; KOOS$_5$ = Knee injury and Osteoarthritis Outcome Score (composite score)

‡ANCOVA with baseline scores extracted as covariates

*Notes statistical significance, large effect size, and high power

Figures

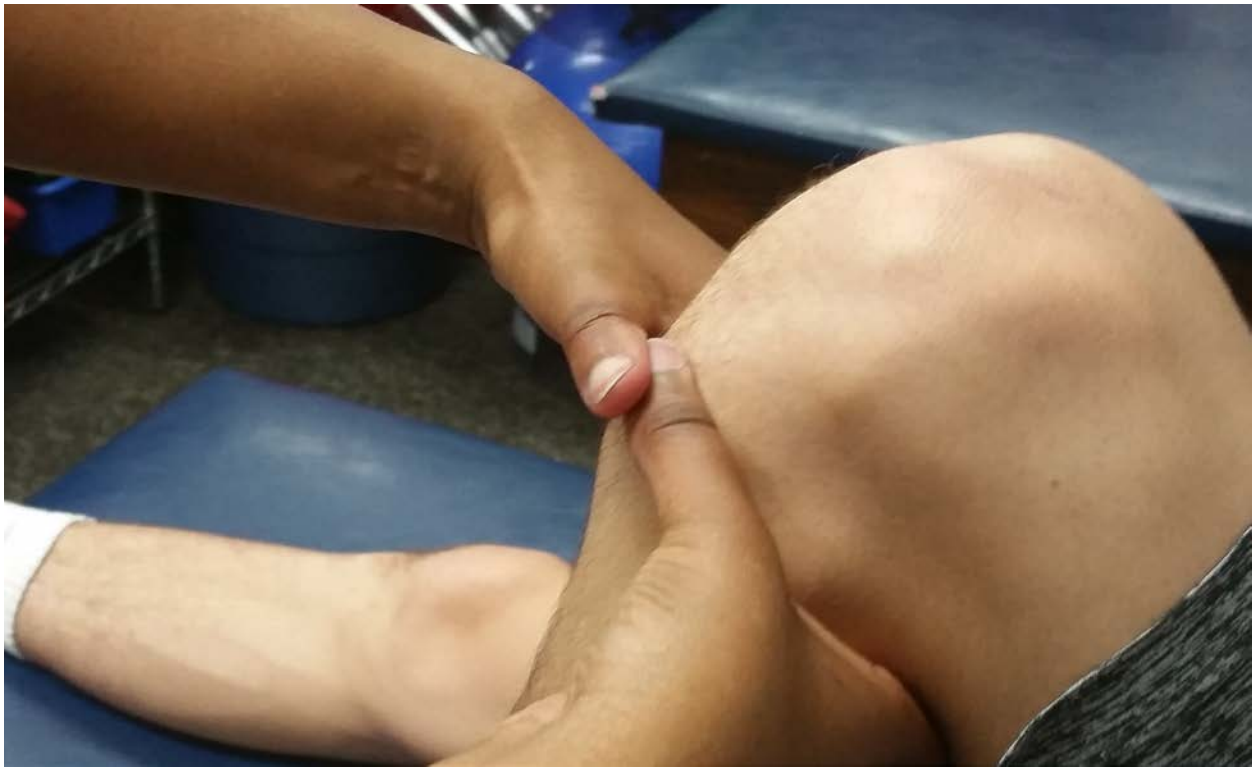


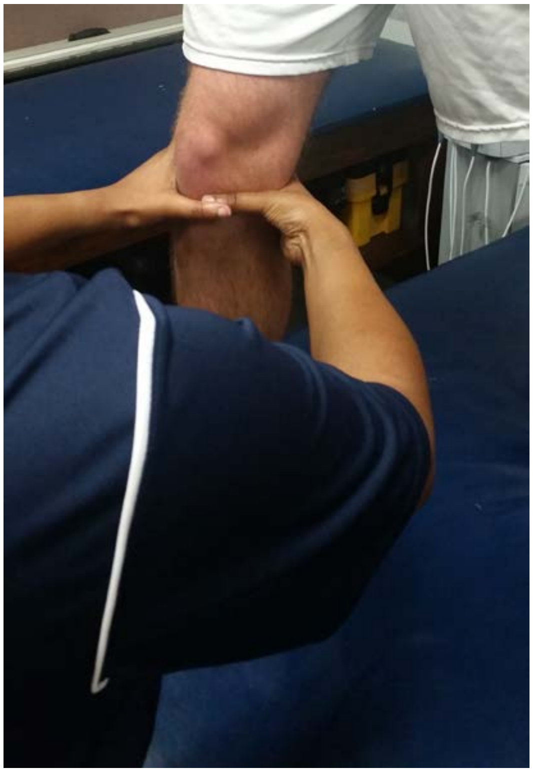
Figure 3.1: Starting hand placement showing the overlap thumb grip

.

Figure 3.2: Clinician hand placement in NWB (supine) for the MC “Squeeze” technique treatment in full knee extension. Clinician alleviates pressure on joint line

Figure 3.3: Clinician hand placement in NWB (supine) for the MC "Squeeze" technique treatment in full knee flexion. Over-pressure is provided by the participant.

Figure 3.4: Clinician hand placement in NWB (supine) for the MC “Squeeze” technique treatment in full knee flexion. Participant uses a strap to assist in providing over-pressure.

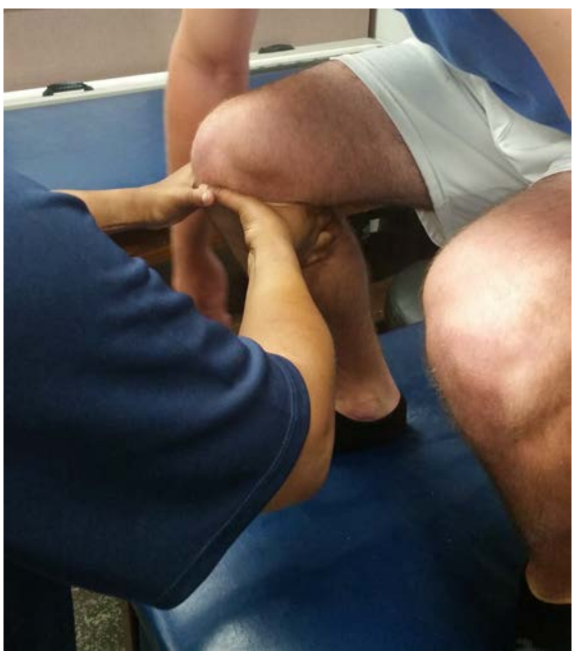


A.

B.

Figure 3.5: Clinician hand placement in PWB (lunge) starting (A) and ending position (B) for the MC "Squeeze" technique

A.



B.

Figure 3.6: Clinician hand placement in FWB (squat) starting (A) and ending position (B) for the MC "Squeeze" technique

Figure 3.7: Clinician hand placement in NWB (supine) for the sham treatment in full knee flexion. Clinician applies overlap thumb grip ½ inch inferior to the reported joint-line tenderness.

Notes: Blue line indicates joint line. Arrow indicates ½ inferior to joint line

Figure 3.8: Clinician hand placement in NWB (supine) for the sham treatment in full knee extension. Clinician applies overlap thumb grip ½ inch inferior to the reported joint-line tenderness.
Notes: Blue line indicates joint line. Arrow indicates ½ inferior to joint line

References

1. Acebes C, Romero F, Contreras M, Mahillo, Herrero-Beaumont G. Dynamic ultrasound assessment of medial meniscal subluxation in knee osteoarthritis. Rheumatol. 2013;52(8): 1443-1447. http://dx.doi.org/10.1093/rheumatology/ket110.
2. Apeldoorn AT, Helvoirt H, Meihuizen H, Tempelman H, Vandeput D, Knol DL, … Ostelo RW. The influence of centralization and directional preference on spinal control in patients with nonspecific low back pain. J Ortho Sports Phys Ther. 2016;46(4): 258–269. http://doi.org/10.2519/jospt.2016.6158.
3. Aytona MC, Dudley K. Rapid resolution of chronic shoulder pain classified as derangement using the McKenzie method: A case series. J Man Manip Ther. 2013;21(4): 207–212. http://doi.org/10.1179/2042618613Y.0000000034.
4. Bedi A, Kelly NH, Baad M, Fox AJS, Brophy RH, Warren RF, Maher SA. Dynamic contact mechanics of the medial meniscus as a function of radial tear, repair, and partial meniscectomy. J Bone Joint Surg Br. 2010;92(6): 1398-1408 http://doi.org/10.2106/JBJS.I.00539.
5. Brody K, Baker R, Nasypany A, Seegmiller J. Meniscal lesions: The physical examination and evidence for conservative treatment. Int J Athl Ther Train. 2015;20(5): 35-38.
6. Chaibi A, Benth JS, Russell MB. Validation of placebo in a manual therapy randomized controlled trial. Sci Rep. 2015;5(11774): 1-8 http://dx.doi.org/10.1038/srep11774.
7. Chakravarty E, Hubert H, Lingala V, Zatarain E, Fries J. Long distance running and knee osteoarthritis. Am J Prev Med. 2008;35(2): 133-138. doi:10.1016/j.amepre.2008.03.032.
8. Chatman AB, Hyams SP, Neel JM, Binkley JM, Stratford PW, Schomberg A, Stabler M. The patient-specific functional scale: Measurement properties in patients with knee dysfunction. Phys Ther. 1999;77(8): 820-829.
9. Cohen J. Statistical power analysis and research results. Am Educ Res J. 1973;10(3): 225. http://dx.doi.org/10.2307/1161884.
10. Crawford R, Walley G, Bridgman S, Maffulli N. Magnetic resonance imaging versus arthroscopy in the diagnosis of knee pathology, concentrating on meniscal lesions and

ACL tears: A systematic review. Brit Med J. 2007;84(1): 5–23. http://doi.org/10.1093/bmb/ldm022.

11. Downie WW, Leatham PA, Rhind VM, Wright V, Branco JA, Anderson JA. Studies with pain rating scales. Ann Rheum Dis. 1978;37(4): 378-381.
12. Drawer S. Propensity for osteoarthritis and lower limb joint pain in retired professional soccer players. Brit J Sports Med. 2001;35(6): 402-408. doi:10.1136/bjsm.35.6.402.
13. Drosos GI, Pozo JL. The causes and mechanisms of meniscal injuries in the sporting and non-sporting environment in an unselected population. Knee. 2004;11(2): 143-149. http://doi.org/10.1016/S0968-0160.
14. Dunsford A, Kumar S, Clarke S. Integrating evidence into practice: Use of Mckenzie-based treatment for mechanical low back pain. J Multidisc Healthc. 2011;4: 393–402 http://doi.org/10.2147/JMDH.S24733.
15. Dye SF, Vaupel G, Dye CC. Conscious neurosensory mapping of the internal structures of the human knee without intraarticular anesthesia. Am J Sports Med. 1998;26(6): 773-777.
16. Englund M, Roos EM, Roos, HP, Lohmander LS. Patient-relevant outcomes fourteen years after meniscetomy. Influnce of type of meniscal tear and size of resection. Rheumatology. 2001;40: 631-639.
17. Englund M, Roos EM, Lohmander LS. Impact of type of meniscal tear on radiographic and symptomatic knee osteoarthritis: A 16-year follow-up of meniscetomy with matched controls. Arthritis Rheum. 2003;48: 2178-2187.
18. Englund M, Lohmander LS. Risk factors for symptomatic knee osteoarthritis fifteen to twenty-two years after meniscectomy. Arthritis Rheum. 2004;50(9): 2811-2819 http://doi.org/10.1002/art.20489.
19. Englund M, Paradowski, P, Lohmander LS. Radiographic hand osteoarthritis is associated with radiographic knee osteoarthis after meniscetomy. Arthritis Rheum. 2004;50: 469-475.
20. Englund M, Guermazi A, Lohmander LS. The Meniscus in knee osteoarthritis. Rheum Dis Clin North Am. 2009;35(3): 579-590 doi: 10.1016/j.rdc.2009.08.004.

21. Ernst E. Does spinal manipulation have specific treatment effects? J Fam Pract. 2000;17: 554-556.

22. Fox AJS, Bedi A, Rodeo SA. The basic science of human knee menisci: Structure, composition, and function. Sports Health. 2012;4(4): 340-351 http://doi.org/10.1177/1941738111429419.

23. Getgood A, Robertson A. (v) Meniscal tears, repairs and replacement – A current concepts review. Orthop Trauma. 2010;24(2): 121-128 doi:10.1016/j.mporth.2010.03.01.

24. Hanney WJ, Kolber MJ, Pabian P, Cheatham SW, Schoenfeld BJ, Salamh PA. Endurance times of the thoracolumbar musculature: Reference values for female recreational resistance training participants. J Strength Cond Res. 2015;30(2): 588-594.

25. Hefford, C. McKenize classification of mechanical spinal pain: Profile of syndromes and directions of preference. Man Ther. 2008;13: 75–81 http://doi.org/10.1016/j.math.2006.08.005.

26. Herrlin S, Hållander M, Wange P, Weidenhielm L, Werner S. Arthroscopic or conservative treatment of degenerative medial meniscal tears: a prospective randomised trial. Knee Surg Sports Traumatol Arthrosc. 2007;15(4): 393–401 doi:10.1007/s00167-006-0243-2.

27. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: Summary and recommendations for injury prevention initiatives. J Athl Train. 2007;42(2): 311-319.

28. Hrobjartsson A, Gotzsche PC. Is the placebo powerless? An analysis of clinical trials comparing placebo with no treatment. N Engl J Med. 2001;44: 1594-1602.

29. Hudson R, Richmond A, Sanchez B, Stevenson V, Baker R, May J, ...Reordan D. An alternative approach to the treatment of meniscal pathologies: a case report analysis of the Mulligan Concept “Squeeze” technique. Int J Sports Phys. 2016; 11 (4). 1-11.

30. Hwang YG, Kwoh CK. The METEOR trial: No rush to repair a torn meniscus. Cleve Clin J Med. 2014;81(4): 226-232 doi:10.3949/ccjm.81a.13075.

31. Karachalios T, Hantes M, Zibis A, Zachos V, Karantanas A, Malizos K. Diagnostic accuracy of a new clinical test (the Thessaly test) for early detection of meniscal tears. J Bone Joint Surg Am. 2005;87(5): 955-962.

32. Kaptchuk TJ. The placebo effect in alternative medicine: can the performance of a healing ritual have clinical significance? Ann Intern Med. 2002;136: 817-825.

33. Kaptchuk T, Miller F. Placebo effects in medicine. N Engl J Med. 2015;373(1): 8-9 http://dx.doi.org/10.1056/nejmp1504023.

34. Katz JN, Brophy RH, Chaisson CE, de Chaves L, Cole BJ, Dahm DL, Losina E. Surgery versus physical therapy for a meniscal tear and osteoarthritis. N Engl J Med. 2013;368(18): 1675-1684. doi:10.1056/NEJMoa1301408.

35. Krebs EE, Carey TS, Weinberger M. Accuracy of the pain numeric rating scale as a screening test in primary care. J Gen Intern Med. 2007;22(10): 1453-1458.

36. Kujala UM, Kettunen J, Paananen H, Aalto T, Battie MC, Impivaara O, . . . Sarna S. Knee osteoarthritis in former runners, soccer players, weight lifters, and shooters. Arthritis Rheum. 1995; 38(4): 539-546. doi:10.1002/art.1780380413.

37. Kurosaka M, Yagi M, Yoshiya S, Muratsu H, Mizuno K. Efficacy of the axially loaded pivot shift test for the diagnosis of a meniscal tear. Int Orthop. 1999; 23(5): 271-274.

38. Lee JM, Fu FH. The Meniscus: Basic science and clincal applications. Oper Tech Orthop. 2000; 10(3): 162-168. http://doi.org/10.1053/otor.2000.5289

39. Lowery DJ, Farley TD, Wing DW, Sterett WI, Steadman RJ. A clinical composite score accurately detects meniscal pathology. Arthroscopy. 2006; 22(11): 1174-1179 http://doi.org/10.1016/j.arthro.2006.06.014.

40. Ludman CN, Hough DO, Cooper TG, Gottschalk A. Silent meniscal abnormalities in athletes: magnetic resonance imaging of asymptomatic competitive gymnasts. Br J Sports Med. 1999; 33(6): 414-416.

41. Lyman S, Hidaka C, Valdez AS, Hetsroni I, Pan TJ, Do H, … Marx RG. Risk factors for meniscectomy after meniscal repair. Am J Sports Med. 2013; 41(12): 2772–2778 http://doi.org/10.1177/0363546513503444.

42. May S, Aina A. Centralization and directional preference: A systematic review. Man Ther. 2012; 17(6): 497–506. http://doi.org/10.1016/j.math.2012.05.003.

43. May S, Rosedale R. A survey of the McKenzie classification system in the extremities: Prevalence of mechanical syndromes and preferred loading strategies. Phys Ther. 2012; 92(9): 1175-1186. http://dx.doi.org/10.2522/ptj.20110371.

44. May S, Ross J. The McKenzie classification system in the extremities: A reliability study using Mckenzie assessment forms and experienced clinicians. J Manipulative and Physiol Ther. 2009; 32(7): 556–563. http://doi.org/10.1016/j.jmpt.2009.08.007.

45. Majewski M, Susanne H, Klaus S. Epidemiology of athletic knee injuries: A 10-year study. Knee, 2006; 13(3): 184-188.

46. McDermott ID. (ii) Meniscal tears. Current Orthop. 2006; 20(2): 85-94 http://doi.org/10.1016/j.cuor.2006.02.010.

47. Mordecai SC, Al-Hadithy N, Ware HE, Gupte CM. Treatment of meniscal tears: An evidence based approach. World J Orthop. 2014; 5(3): 233-241 doi:10.5312/wjo.v5.i3.233.

48. Mulligan, B. Manual therapy rounds: Mobilisations with movement (MWM's). J Man Manip Ther. 1993; 1(4): 154-156.

49. Mulligan BR. Manual therapy NAGS, SNAGS, MWMs etc. (6th ed.). Wellington, New Zealand: Plan View Services Ltd; 2010.

50. Nepple JJ, Dunn WR, Wright RW. Meniscal repair outcomes at greater than five years. J Bone Joint Surg Am. 2012; 94(24): 2222–2227 http://doi.org/10.2106/JBJS.K.01584.

51. Nikolaou V, Chronopoulos E, Savvidou C, Plessas S, Giannoudis P, Efstathopoulos N, Papachristou G. MRI efficacy in diagnosing internal lesions of the knee: A retrospective analysis. J Trauma Manag Outcomes. 2008; 2(1): 4. doi:10.1186/1752-2897-2-4

52. Paxton ES, Stock MV, Brophy RH. Meniscal repair versus partial meniscectomy: A systematic review comparing reoperation rates and clinical outcomes. Arthroscopy. 2011; 27(9): 1275-1288.

53. Physical Activity Council. The Physical Activity Council's annual study tracking sports, fitness, and sports recreation participation in the US: http://www.physicalactivitycouncil.com/pdfs/current.pdf. Published 2016. Accessed May 1, 2016.

54. Pujol N, Barbier O, Boisrenoult P, Beaufils P. Amount of meniscal resection after failed meniscal repair. Am J Sports Med. 2011; 39(8): 1648–1652 http://doi.org/10.1177/0363546511402661.

55. Renstrom P, Johnson RJ. Anatomy and biomechanics of the menisci. Clin SPorts Med. 1990; 9(3): 523-538.

56. Roemer F, Jarraya M, Niu J, Silva J, Frobell R, Guermazi A. Increased risk for radiographic osteoarthritis features in young active athletes: A cross-sectional matched case control study. Osteoarthr and Cartil. 2015; 23(2): 239-243 doi:10.1016/j.joca.2014.11.011.

57. Roos H, Lindberg H, Gardsell P, Lohmander L, Wingstrand H. The prevalence of gonarthrosis and its relation to meniscectomy in former soccer players. Am J Sports Med. 1994; 22(2): 219-222. doi:10.1177/036354659402200211.

58. Roos EM, Roos HP, Ekdahl C, Lohmander LS. Knee injury and osteoarthritis outcome score (KOOS) - Validation of a Swedish version. Scand J Med Sci Sports; 1998; 8(6): 439-448.

59. Roos KG, Marshall SW, Kerr ZY, Golightly YM, Kucera KL, Myers JB, ... Comstock RD. Epidemiology of overuse injuries in collegiate and high school athletics in the United States. Am J Sports Med. 2015; 42(7): 1790-1797. 0363546515580790.

60. Rosedale R, Rastogi R, May S, Chesworth BM, Filice F, Willis S, … Robbins SM. Efficacy of exercise intervention as determined by the McKenzie System of Mechanical Diagnosis and Therapy for knee osteoarthritis: A randomized controlled trial. J Orthop and Sports Phys Ther. 2014; 44(3): 173–181 http://doi.org/10.2519/jospt.2014.4791.

61. Salaffi F, Stancati A, Silvestri CA, Ciapetti A, Grassi W. Minimal clinically important changes in chronic musculoskeletal pain intensity measured on a numerical rating scale. Eur J Pain. 2004; 8(4): 283–291.

62. Shellock FG, Hiller WDB, Ainge GR, Brown DW, Dierenfield L. Knees of Ironman triathletes: Magnetic resonance imaging assessment of older (> 35 years old) competitors. J Magn Reson Imaging. 2003; 17(1): 122-130.

63. Sihvonen R, Paavola M, Malmivaara A, Itälä A, Joukainen A, Nurmi H, . . . Järvinen TL. Arthroscopic partial meniscectomy versus sham surgery for a degenerative meniscal tear. N Engl J Med. 2013; 369(26): 2515-2524.

64. Stratford P, Gill C, Westaway M, Binkley J. Assessing disability and change on individual patients: A report of a patient specific measure. Physiother Can. 1995; 47(4): 258-263.

65. Takeda H, Nakagawa T, Nakamura K, Engebretsen L. Prevention and management of knee osteoarthritis and knee cartilage injury in sports. Br. J. Sports Med. 2011; 45(4): 304-309. doi:10.1136/bjsm.2010.082321.

66. Turner A. Long term health impact of playing professional football in the United Kingdom. Br. J. Sports Med. 2000; 34(5): 332-336. doi:10.1136/bjsm.34.5.332.

67. 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; 145: 36-44.

68. Vela LI, Denegar CR. The disablement in the physically active scale, part II: The psychometric properties of an outcomes scale for musculoskeletal injuries. J Athl Train. 2010; 45(6): 630-641. doi:10.4085/1062-6050-45.6.630.

69. Vigotsky AD, Bruhns RP. The role of descending modulation in manual therapy and its analgesic implications: a narrative review. Pain Res Treat. 2015;2015:292805. doi:10.1155/2015/292805.

70. Walczak BE, McCulloch PC, Kang RW, Zelazny A, Tedeschi F, Cole BJ. Abnormal findings on knee magnetic resonance imaging in a asymptomatic NBA players. Am J Knee Surg. 2008; 21(1): 27-33.

71. Wilson AS, Legg PG, McNeur JC. Studies on the innervation of the medial meniscus in the human knee joint. Anat Rec. 1969; 165(4): 485-491.

72. Yeh PC, Starkey C, Lombardo S, Vitti G, Kharrazi FD. Epidemiology of isolated meniscal injury and its effect on performance in athletes from the National Basketball Association. Am J Sports Med. 2012; 40(3): 589-594 http://doi.org/10.1177/03635465114286.

73. Zanetti M, Pfirmann CWA, Schmid MR, Romero J, Seifert B, Hodler J. Patients with

suspected meniscus tears: Prevalence of abnormalities seen on MRI of 100 symptomatic and contralateral asymptomatic knees. Am J Roentgenol. 2003;181: 635-64.1.

APPENDIX A

Plan of Advanced Practice: Developed November 20, 2015

Introduction

A famous quote by one of my favorite authors states “Oh the places you will go” (Seuss, 1990). I think about my future as a practicing athletic training often. I wonder if I would like to continue working in the setting that has been so rewarding for over 10 years or am I ready to transition my current setting into a practice that is more patient-centered, scholarly and balanced. To decide where I would like for this road to lead, composing of plan of action for advancing my practice is paramount. The plan of action, including goals for the future requires that I honestly reflect on where I am now as an athletic trainer. Where do I excel and where are my deficits? How will I address both my strengths and my weaknesses? My professional objective is to improve my weaknesses and reinforce my strengths by reflecting on where I began and how I arrived at my current destination. Composing an evolving and progressive Plan of Advanced Practice (PoAP) will assist me in answering these questions.

My journey to advance practice began by developing my current competences through education instruction and past professional experiences. Those competences include an above average ability to evaluate lower extremity injuries, an understanding of functional anatomy which leads to global treatment interventions based on regional interdependence, implementing effective prevention and health promotion strategies, using advanced therapeutic rehabilitation paradigms and an ability to act in emergency situations according to protocol and apply advanced life saving techniques. Now that the foundation is laid, my goals for the future will reinforce my current abilities and lead to further goal planning and adjustments. I will begin discussing my plan for advance practice by elaborating on my clinical background prior to beginning the Doctorate of Athletic Training (DAT) program.

Current Clinical Competence

Education Background

My educational background began with at non-accredited internship program at the University of Memphis as an undergraduate and proceeded to an accredited athletic training program at the University of Arkansas during my Master’s education. I began in athletic training as a student during the time that the Commission on Accreditation of Athletic

Training Education (CAATE) was being established and discussions about transitioning the profession from the apprenticeship routes to accredited formal educational degrees were trending in the athletic training community. The rationale behind this push for accreditation was to develop consistent education and competencies in athletic training. For the profession to grow in as an allied health profession, basic guidelines for entry-level athletic trainers needed to be established. The profession needed on objective manner of establishing that graduates were prepared to succeed in patient care once they reached completion of their degree programs. I did not decide to begin an accredited program immediately. I had invested three years into my internship program, and I did not want to transfer to an accredited program and lose that valuable time. I decided I would continue with my apprenticeship education and apply to an accredited professional Master's athletic training program before graduation.

My experiences during my professional Master's education at the University of Arkansas were a mix of excitement and confusion. Prior to enrollment in the program, I had a certain amount of autonomy with my 'athletes' at the University of Memphis. I was allowed to perform evaluations and make decisions regarding the course of treatment and rehabilitation. I made return to play decisions independently of the head athletic trainer because I was trusted to make informed decisions based on the patient's presentation. I traveled with the team without a preceptor and only consulted with a certified AT when I encountered an issue I could not treat.

At the University of Arkansas, I was forbidden to make any decisions without the guidance and approval of my preceptors. Although I would attend every practice, game, and clinic hours I was only allowed to implement treatments decided upon by my preceptor. I performed only the orthopedic special tests that I was cleared on through my course check off sheets and then the certified AT would perform the evaluation gain and begin the patient's course of treatment.

During a formal education in the professional Master's AT program, I quickly learned that many of the methods taught in my undergraduate education were not based in evidence and I did not possess a basic understanding of clinical reasoning and decision-making. I realized I should not have had so much autonomy without proper training during my undergraduate apprenticeship and those types of situations were what CAATE was fighting petitioning to prevent. If I had entered the professional workplace after my Bachelor's of Science (BS), I would not have been as prepared to be a practicing clinician. I would have taken all of the anecdotal evidence learned as an intern and used it in my patient care and would not have been aware of the clinical pearls I learned while in graduate school. Although I received an excellent education at the University of Arkansas and it helped prepare me to be a better clinician, I was not completely devoid of training and knowledge from my undergraduate education. My clinical decision-making had its foundation in my apprenticeship training and it was in that time period my athletic training philosophy began to develop.

Because of my educational background, I was confident working independently and using formal didactic teachings in my practice, however after I began to practice athletic training professionally, my approach to patient care relied on treating the local area of complaint exclusively due to a lack of thorough and global evaluations. After the evaluation, I would apply electrical stimulation or therapeutic ultrasound with the manufacture's pre-settings along with cryogenic modalities during the acute phase of injury and thermogenic modalities in a chronic state of injury. When I established the patient was prepared for rehabilitation, I would instruct the patient to begin flexibility and progressive resistive exercise (PRE's) programs for the duration of the injury. I used this formula of clinical practice for the next seven years, but made some efforts to improve my clinical skills.

I attended introductory manual therapy classes and I used those techniques with unremarkable success. I enrolled in the Graston Technique® Instrument Assisted Soft Tissue Mobilization (IASTM) basic course with the intension of following up with the advanced courses but I have not to date. I also participated in a myofasical release continuing education course given by a local hospital. Although I learned the new techniques, I was not using them as the evidence indicated because I did not read the literature or collect patient outcomes to determine best practice with those techniques. I became frustrated with athletic training and my patient care because my interventions were not successful. I had accepted that my career and practice were not going to change and I began to plan for a shift in my career placement. I was not physically or mentally present on most days in the clinic and I relegated myself to using physical therapy software to print out exercises and instructions so I would not engage in individual interventions with my patients. I did, however, began to shift my clinical practice, my outlook and my attitude after learning how using evidence based interventions and outcome measures would lead to more productive and successful patient care.

Injury Evaluation

My athletic training knowledge has increased and diversified over the 15 years I have been a part of the profession, either as a student or a professional. My evaluation and treatment skills of knee, lower leg and ankle injuries have increased exponentially over evaluation skills of other body regions. My basic assessment knowledge of this region has been reinforced due to repetition and advanced continuing education. My patient population over the last 10 years has included female soccer athletes who are at higher risk for lower body injury due to the nature of their sport and in an effort to apply the most effective treatments, I have attended continuing education courses relating to the evaluation of the knee and ankle at my local hospital and at educational conferences and symposiums. I have sought out the advice and counsel of numerous orthopedic surgeons on strategies to best determine the source of pain and dysfunction in the lower extremity.

I also use Brian Mulligan's positional fault theory as a basis and guideline of determining the cause of pain and dysfunction after injury. Brain Mulligan theorized that after injury occurs a miniature positional fault may be present within the joint caused secondarily to injury. The fault is not typically seen on imaging, however studies on lateral ankle sprains have supported the theory that the fibula can become retched forward on the talus after an inversion sprain and remain in the position causing chronic pain and dysfunction. The positional fault theory can be applied to all joints in the body and I use subacute applications of the Mulligan Concept's Mobilization with Movement to determine if in fact a positional fault could be corrected with the technique, thus using the MC as both an evaluation and a treatment method especially in the lower extremity. Based on my most recent exploration and advice of other healthcare professionals, I have also begun to use regional interdependence (RI) evaluations and treatments in my patient population, thus adding to my knowledge of evaluating other regions in the body as well.

Functional Anatomy

My current foundational knowledge as it relates to overall basic functional anatomy is solid. I am fortunate that three years ago, I began teaching a functional anatomy course for pre-occupational therapy students. The course involves teaching functional anatomy and neurophysiology. Before I began teaching, my knowledge of basic anatomy was fair and I knew enough to perform basic patient orthopedic evaluations. Although I knew the basics, I was not aware how much I was lacking in advanced anatomy knowledge until I began to gather information for class lectures. I quickly realized, to teach the information, I needed to become more advanced in my knowledge of the material. Each semester I am challenged by my students regarding human anatomy and how our structures interact in both functional and dysfunctional movement. My students compel me to think outside of my comfort level and see the anatomy from their perspective.

I view functional anatomy in 3 dimensional layers both when I am teaching and when I am performing an evaluation. I begin with the skin and visualize the layers of the fascia system and the complex system of muscles and nerves that are intertwined underneath. Next, I envision musculoskeletal attachments and bone to bone connections at the joints. When I see the joint connections, I also see osteokinematic and arthokinematic movement that is occurring during activity. Recognizing the body as a system of links and pivots aids my interpretation of RI evaluations and treatments and contributes to my teaching philosophy. If a student does not understand RI, I have to modify my explanation so they will see the relationship. An adjustment in my teaching requires a higher degree of understanding beyond my previous novice level while attaining post-professional certifications have contributed to an advancement in not only teaching but also in my patient care.

Prevention and Health Promotion

I became a Certified Strength and Conditioning Specialist (CSCS) through the National Strength and Conditioning Association (NSCA) four years ago. Maintaining this certification through continuing education has improved my ability to implement prevention and health promotion strategies as well as advance my rehabilitation exercise prescription. My knowledge of the human body's physiological response to strength and conditioning prior to this certification was at a moderate level as my undergraduate degree was in Exercise and Sports Science, however my approach to patient rehabilitation was not well rounded. Strengthening, joint range of motion mobilization and exercises and proprioception retraining was used repeatedly in my patient care with mediocre outcomes until about two years ago. I did not implore any strategies for maintaining patient conditioning and general health during long term rehabilitation. After making the decision to sit for my CSCS certification, I learned more about human physiology and the physical needs of the body while it is going through healing phases. Many of the principles I previously used in my practice were partly ineffective because I did not have firm grasp on body function and how maintaining cardiovascular conditioning and proper nutrition contribute to recovery from injury as well. I had previously overlooked overall health maintenance during post surgical rehabilitation and other injuries that required the patient to be out of activity for an extended period. I now educate patients on knowledge as it relates to proper nutrition, periodization training and cardiovascular conditioning to promote a balanced healthy recovery from their long-term injuries.

Global Assessment and Treatment

To complement my knowledge of human physiology and RI, becoming certified in The Selective Functional Movement Assessment (SFMA) added another layer to my capability to appropriately treat patients from a global perspective. The SFMA reveals dysfunctions in the body by examining a patient's ability to perform the assessment's required movements (Cook, Burton, Kiesel, Rose & Bryant, 2010). Based on the patient's dysfunctional movement patterns, I am able to identify specific components that need addressing and discern if the patient's issue is a joint mobility issue, a tissue extensibility issue, or a stability motor control issue. Results of the breakout flowcharts in the assessment assist me in choosing the appropriate treatment paradigm and subsequent corrective exercise protocol that addresses the dysfunction and not just pain. Based on my training and education to date, I typically implement the Mulligan Concept (MC) or Mobilizations with Movement (MWM) to treat joint mobility dysfunction, Graston IASTM or positional release technique (PRT) to treat tissue extensibility dysfunction, and Reactive Neuromuscular Training (RNT) to treat stability motor control dysfunction..

Professional and Personal Strengths and Weaknesses

Professional and Personal Strengths

My strengths in athletic training are both varied and diverse. Professionally and personally I approach patient care from a physical and psychological standpoint to contribute to whole patient well being. Incorporating a psychosocial model in my patient care requires my full presence and consistent patience. Through analyzing patient oriented outcome measures such as the Disablement in the Physically Active (DPA) scale and actively inquiring and listening to all aspects of my patient's lives, I am able to recognize the need for psychosocial interventions such as Trauma Releasing Exercises (TRE) and Reflexerice. I interact with my patients in a manner that is conducive to providing a safe space for all forms of healing to occur both by my patients and myself. My focus on personal well-being and positive energy building also contributes to an all encompassing progressive environment for patients to recovery from both acute and chronic pain. My role as an athletic trainer includes being an example for my patients by reinforcing a healthy and productive lifestyle. If I am happier, positive, and present my patients will see me as an example of how to set their intentions to happiness within the physical and psychological realm.

Being present in my patient care also contributes to my seamless flexibility in the clinic. Patient injuries are fluid in their presentations and require a daily reassessment and adjustment in treatment based on my observation that day. A lateral ankle sprain may respond positively to the MC lateral ankle MWM on the first and second day, however on the third day, the patient may complain of pain, tightness or dysfunction in another area. In this scenario, the MC lateral ankle MWM may not be the indicated treatment although it was the one that I planned to apply. Critical thinking and refocusing on another area of complaint has to be continuous and expected of me.

At a moment's notice, an emergency situation may arise in my setting. My days are unpredictable and I make an active effort to refocus my attention to the situation that currently requires the highest priority. While handling some acute care situations I am able to critically think through a potentially life threating occurrence and either treat or refer the patient as needed and then return to my regular patient load in the clinic without allowing the event to derail my intentions in the clinic. After treating patients throughout the day, adjusting interventions as needed, and prioritizing my clinical responsibilities, I transition into administrative healthcare administration needs and arrange all of those components into detailed documentation.

I am very organized in my clinic, which leads to productive healthcare administration and professional development. Patient records are kept up to date and patient dictations are done nightly after the last patient interaction. I am able to remain attentive and present by setting aside time in the evening to dictate notes from the day because I am completely focused on the task of analyzing a patient's response to treatment in their outcome measures.

Subsequent follow ups are more productive and conductive to healing when a plan is in place; although the patient may present with different signs and symptoms the follow day and adjustments are needed.

A summary of my professional and personal strengths can be seen below:

- Whole patient well being approach to patient care through incorporating both physical and psychosocial components into daily clinical practice
- Organized and deliberate healthcare administration
- Flexible and welcoming to change
- Hardworking and contributor to a teamwork environment in the workplace
- Solid and effective personal coping mechanisms when managing multi projects and patient cases

Professional and Personal Weaknesses

Recognizing my strengths in clinical practice is rewarding and useful, but for my practice to continue to advance, critical evaluation and reflection on my weaknesses is needed as well. Effective and efficient hip, pelvic girdle, and sacroiliac joint (SIJ) evaluations are key weaknesses in my clinical practice. I learned basic hip and pelvic girdle evaluation in my professional Master's AT program, but I have failed to advance my ability to assess and treat pelvic girdle dysfunction. I am able to perform a pelvic girdle evaluation using a variety of special test and by observing patient reported symptoms, but usually differential diagnosis is a struggle. Many underlying pathologies exist at the hip and pelvis and pelvic dysfunction is most likely a direct cause of joint or tissue dysfunction from another region of the body.

I have treated soccer patients regularly throughout my career and they have a very high incident of hip related pathology due to the nature of the sport (Falvey, Frankly-Miller & McCrory, 2009). Many of those injuries include soft tissue involvement such as hamstring complex pain and or restriction, anterior hip flexor pain and dysfunction and SIJ/piriformis syndrome presentations. I am ineffective in treating those injuries in part because my initial evaluation does not reveal enough pertinent information to begin an indicated intervention. Because of my insecurity of evaluating and treating hips, I typically refer my hip and low back patients to physical therapy clinics. To address this weakness, I have enrolled in MedBridge® continuing education courses online to begin developing my advanced knowledge of hip evaluations and identifying key differential diagnosis. From this point, I will continue to practice those tests on more patients and find other hands-on seminars where I can learn more closely with experts in the area.

Within my lack of advanced hip evaluation skills also lies my inability to use more evidence-based practice. Although I have incorporated more evidence-based medicine into my clinical practice by collecting patient outcomes and studying pertinent literature, I lack depth in practicing based on supported and peer-reviewed studies. If I consult the literature more often and begin to characterize and classify my patients, I will begin to see better patient outcomes. For instance many of the special tests that are used to assess SIJ dysfunction such as thigh thrusts, SIJ distraction, sacral thrust, and SIJ compression tests are not very useful individually, however Laslett et al. (2005) found that a battery of those tests used to assess SIJ and pelvic dysfunction have a higher sensitivity and specify for determining SIJ pathology. More exploration of the literature on all conditions and treatments will increase evidence-based medicine implementation in my practice.

Exhausting the literature to determine best practice in my patient care has also made me more aware of my lack of knowledge and deficiencies in professional scholarship. I do not have an extensive background in manuscript writing. Before joining the DAT, I had not composed a full publishable manuscript. My professional Master's program mandated that I compose a case study and while the document was acceptable for department requirements, it contained little to no statistical analysis and was void of scholarly discussion and implications. Within my manuscript composition currently, I am indecisive regarding statistical analysis and how best to identify statistical test that should be done a priori. Once I have completed my data collection, I am unsure how best to present my outcomes in a concise and meaningful way. Disseminating patient outcomes in my practice is very important to advancing my practice and enhancing my professional scholarship. My solution to strengthening this weakness is repetition in writing. The more I collect data, compose manuscripts, and consult with scholars that have published multiple studies, the better I will become. I will be open to critical feedback and edits and use the advice of experienced writers to my advantage.

When I reflection on my professional weakness, I tend to also struggle personally. Focusing on my shortcomings can become overwhelming and this can affect my patient care on some occasions. When I fail to evaluate and treat patients effectively, I fall into a systematic negative mind-set where I will not celebrate my successes because I am fixated on my inadequacies. When I cannot edit a manuscript so that it is concise and well written, but also contains all pertinent information, I can begin to elicit negative self-talk. In these moments, I rely on my coping mechanisms to return to positive, free flowing energy and I am able to reset my focus towards improvement without negativity. A summary of my professional and personal weaknesses can be seen below:

- Inadequate hip girdle and sacroiliac evaluation and treatment skills
- Lack of evidence-based medicine within my daily practice
- Professional scholarship in manuscript composition lacks depth and concise meaningful information
- Statistical analysis and recognition of useful information a priori is minimal
- Negative self talk and focus on weaknesses can overshadow strengths and lead to poor patient care

Professional Practice Goals/ Future Goals

Specialization in Clinical Practice

Specialization is not very common in athletic training; however a study conducted by Neibert (2009) concluded that post professional graduates felt specialization through our educational programs is crucial to the advancement of athletic training. Healthcare providers specialize as a means to focus their energy within a particular discipline of medicine and become an expert in that specialty. The National Strength and Conditioning Association also offers specialized certificate programs for training women, the elderly, and other specialized subsets of clients (http://nsca.com) and if other healthcare providers are advancing their patient care by specializing, athletic trainers should also participate in obtaining specialties. Current education standards for athletic training are consistent with other healthcare professions which contribute to the possibility of specialization more so (Perrin, 2007). While it may not be feasible for athletic trainers to specialize in body regions such as shoulders, knees, and feet, it may be possible to concentrate our knowledge on a particular population and become leading experts in that population. The marketability of athletic trainers will improve along with greater public acceptance for us as advanced health care practitioners.

Despite relatively few separate gender athletic departments, the number of females participating in collegiate athletics as a result of Title IX compliance laws has continued

(Grappendorf & Lough, 2006). Female athletes have unique medical circumstances and needs. We would be remised if we ignored the emotional and psychosocial components and concerns of athletic female patients. In my practice, I have addressed disordered eating patterns, pregnancy, other reproductive system involvement, and the unhappy triad which are all unique and specific to female athletes. Athletic training clinics with a mission to comply with Title IX mandates and an aspiration to address the best interests of the female athlete, may consider hiring an athletic trainer that has chosen to specialize and become an expert in the female athletic patient.

Cultivating and honing my knowledge and experience as it relates to an active female population would allow for that population to benefit from a specialist such as myself that has made it a point to study their unique needs in patient care. Many physical and psychosocial benefits are possible because my patients are aware that I have spent most of my professional career caring for female patient while continuing to advance my practice through recognition and studying of their population.

Educator Goals

Athletic training encompasses a variety of areas for advancement in my career and clinical practice. The level of education required to practice as an entry-level athletic trainer has been a topic of discussion for years. Arguments have been made on both sides to either continue with a bachelor's entry-level degree or progress to an advanced pre-professional Master's education, however during the Fall of 2015 the strategic alliance that include CAATE, the athletic training Board of Certification, the National Athletic Training Association (NATA), and NATA foundation decided to change the athletic training degree to a Master's degree with a specific implementation deadline of no less than seven years. I believe for our profession to progress, education of future athletic trainers must be scholarly, thorough, and based in evidence. I plan to contribute to the growth and develop of athletic training by continuing to improve my skills as an educator in order to provide the tools necessary for entry-level AT's to further advance their clinical practice.

Since beginning the DAT program, some of my professional goals have evolved. Becoming full time faculty as a future career goal has changed as my desire to teach has not diminished completely, but I will remain in my role as an adjunct professor while continuing to primarily focus on advancing my patient care. My teaching approach engages my students by providing them with all the important components of a concept and then allowing them to critically think how to put that information into action. For example, I teach orthopedic special tests separate from anatomy and also separate from mechanisms of injury to allow my students the opportunity to have 'ah ha' moments when they are able to make the connections on their own. I am very hands-on in my classroom setting and I am open to learning from my students while they are learning from me.

Teaching is very enjoyable and I appreciate being able to introduce students to a career that has been so rewarding for me. My goal is to enhance my students' learning experience by developing my own clinical practice knowledge even further. They will benefit from the experiences I have with new and innovative treatments paradigms through the DAT and through continuing education post DAT. More students will be able to perform the Mulligan Concept and understand the guiding principles within the paradigm; students will be able to learn the indications for applying positional release technique and how to combine paradigms for the most effective protocols. Therefore, I will continue to enrich my student's educational journey through a variety of teaching tactics involving sharing evidence found in my patient care outcomes along with the work of other scholars. In the next five years, I plan to increase the number of my students who sit for and pass the Board of Certification for Athletic Training and/or the Texas State Licenser Exam test. Because I am not currently employed at an institution with an accredited undergraduate athletic training program, as long as I am employed at Texas Woman's University, I will empower and prepare more students to go on to professional Master's athletic training programs at other institutions after they successfully graduate. As the educational requirements are currently in transition so will my goals in ensuring I lead my students to the correct path of professional certification.

Clinical philosophy goals

I have been a pain chaser my entire professional career. My approach has been to assess for symptoms at the area of complaint and begin my treatment regimen at that location. Going forward I have decided to strength my rehabilitation and clinical approach to pain by pursuing the driver and treating dysfunction from an advanced RI standpoint using techniques such as the Myokinesthetic System postural assessment and the SFMA.

To strengthen my weakness of hip and SIJ treatment and to reach my goal of changing my clinical philosophy, I will begin by studying the literature, doing global patient assessments during orthopedic evaluations and classifying my patients based on which interventions work best. Using a treatment based classification (TBC) system will advance my knowledge, experience and outcomes on my SIJ patients. Collecting both region specific and patient specific outcomes will also provide the data necessary for analysis of the efficacy of my plan. For pelvic girdle pain, SIJ dysfunction and associated low back pain, I have developed a RI plan of action that includes assessing for breathing dysfunction associated with a dysfunctional diaphragm. Because of the physiological location and collection of the diaphragm to other regions of the body, clearing breathing first will most likely add to the effectiveness of other subsequent techniques. To assess a successful completion of this goal, all patient evaluation forms will now include either a postural exam or an SFMA top-tier assessment. All of my patients will be re-evaluated as needed via these RI assessments and treated accordingly.

Along with using an RI physical approach to patient care, maintaining a balance between physical and psychological healing is crucial for my future as an advanced athletic trainer. To enhance patient healing and foster an environment of patient-centered care, I will continue to use clinic scheduling so I allot for personalized and thorough interactions with each patient.

I plan to increase my understanding and use of advanced skills in integrating a psychological component into daily practice. Pain perception and pain modulation are both regulated within the central nervous system (Ossipov, Dussor & Porreca, 2010), so incorporating psychological techniques such as Emotional Freedom Technique (EFT), Trager Approach, and Trauma and Tension Releasing techniques (TRE) into daily musculoskeletal rehabilitation is logical. I am aware of the emotional connection my patients have with their athletic activities. I have noticed while working with my female population that negative emotions in academics or personal relationships affect rehabilitation and athletic performance. If I can seamlessly work psychological interventions into physiological healing, I believe I will improve my patient outcomes and decrease time for return to play.

Although I have improved markedly in treating from a psychosocial stance, I intend to continue to improve recognizing when such an intervention is warranted and not ignore yellow or red flags. I will implement administration of patient outcome measure that asses for psychological involvement such as the DPA scale and the Yellow Flags Questionnaire.

Understanding central sensitization and psychosocial pain presentation will provide the tools to implement interventions that are balanced and meaningful in more than one dimension.

Assessing and treating breathing dysfunction in my daily practice is one strategy I will implore to treat patients both physically and psychologically. Regular practice of determining respiration dysfunction as it relates to injury presentation will add an element of rounded patient care. Assessing breathing as it relates to other pathology in the body has been the most transcendent clinical practice adjustment recently. Chaitow's *Recognizing and Treating Breathing* details why treating breathing is so pivotal. To observe breathing from a global perspective, physiology of the diaphragm is a major component. The diaphragm connects to the entire body via the fascia system and when the fascia system becomes dysfunctional due to a dysfunctional diaphragm, it pulls and distorts all of the structures it is connected to. Pain may translate from an acute to a chronic state if the diaphragm is not reset and released via techniques such a PRRT. John Iam's explains in his teaching of PRRT that the startle response is initiated at the first contact of injury and a deep breath or hyperventilation ensures immediately afterwards. The dysfunction will continue if breathing is not assessed and reset thus relieving the patient of both physical and psychological pain. To reach the goal of assessing breathing daily, the Hi Lo breathing assessment and the Modified Manual Assessment of Respiratory Motion Test (MARMs) have been added to all evaluation forms and will assessed during all patient evaluations.

Professional Scholarship

Disseminating evidence of my advanced practice will foster an environment for other AT's to begin practicing evidence-based medicine as well. To reach as many healthcare professionals as possible, I plan to annually submit at least one manuscript to a peer-reviewed journal. During my matriculation through the DAT, I have identified numerous topics of interest for study and publication and have begun to collect data on those topics. The Mulligan Concept "Squeeze" Technique and its effect on meniscal tear symptoms has been a topic of interest for over a year. Pilot data collected during the Fall of 2014 and the Spring of 2015 has supported the efficacy of the technique with all participants in the study reaching statically and clinically significant differences in pain and function. Large effect sizes were noted and minimal clinical important differences (MCID) or minimal detectable changes (MDC) were observed in pain and function. Journal publication for this study has been rejected to date, but edits are in progress and resubmission is planned before the end of May 2016.

Based on the results of the pilot study, a follow up study comparing the MC "Squeeze" technique to a sham technique is currently in data collection until December 2015 with data analysis beginning in January of 2016. Once this data has been analyzed and the manuscript composed, submission for publication will follow. Other current ongoing studies include a survey to assess meniscus tear evaluation and treatment habits of athletic trainers and a clinometer app validity study. Data collection for the long term patient follow study up on the MC "Squeeze" Technique will begin in the Spring of 2016 pending Institutional Review Board (IRB) approval and the effects of the MC internal rotation, external rotation, posterior translation and anterior translation on knee pain related to meniscus tears are in discussions for implementation.

In addition to the studies connected to the meniscus pilot data, I have collected patient outcome data on the effect of correcting breathing dysfunction on anterior hip flexor pain and dysfunction. By assessing breathing on patients complaining of anterior hip flexor pain using the MARM test and a battery of rib tenderness tests, I was able to treat respiration and reassess the MARMs along with pain and function of the hip flexor. All patients had a resolution of pain and full return of function by discharge and I will disseminate this evidence to provide other healthcare professional with an alternative treatment for a condition very prevalent in sports. My goal is to have the suggested edits for this study by the end of May 2016 and submission to a peer-reviewed journal by December of 2016. I will continue to explore breathing dysfunction as it relates to other musculoskeletal injuries and share my findings with the athletic training community.

Once these studies have reached completion and data analysis reveals meaningfulness of data, I plan to present my papers and other trending topics at professional conferences at least once a year and conduct learning labs to demonstrate the proper administration of these techniques and applications for clinical practice. Along with presentation at conferences, I will facilitate conducting continuing education courses within my local district by hosting advanced courses on site in our clinic. By hosting these courses, athletic trainers will have the opportunity to come together to discuss evidence-based medicine within a think tank of likeminded professions interested in advancing their practice; much like I have experienced in continuing education courses at the University of Idaho in the past two summers.

To elaborate on goals in relations to patient care, it is important that I mention my current setting and my desire to transition my clinic into a more patient centered clinic. I have spoken with my supervisor on numerous occasions on ideas I have about enriching the culture of our clinic and improving patient outcomes. My entire career has involved working in a traditional athletic model and while I have been very content and happy in this setting, I have noticed how my professional and personal priories have evolved. I am at a point in my life and career where my family is more important and cultivating personal goals outside of my career are also essential. I would like to see a more organized and deliberate approach to patient care that involves collection of outcomes and evidence based practice being the norm. I have proposed that we implement as system where we use patient specific and region specific outcome measure within our online injury tracking software to make for a more seamless transition. We use the SOAP notes application in the program and input information into out tracking software daily so having those readily available would be a reminder to collect outcomes on patients to determine efficacy of treatment. My goal is to begin implementation of those outcomes into injury tracking software by the beginning of August 2016 when our patients return to campus for the beginning of another school year. Along with using those outcome measures, I have proposed we set our intentions on tracking injuries and their connection to indicated treatment paradigms so that we can begin to also practice in a treatment based classification (TBC) approach. Categorization and classification of patients based on treatments will also begin in August 2016.

Justification for Planning for Advancement

Why develop a plan? Benjamin Franklin once said, if we fail to plan, we are planning to fail. Developing a plan provides a means to make as many adjustments as necessary throughout my career. As my career develops academically, professionally and personally, modifications will need to be made to my goals and plans. ‘Life’ happens and when it does, I will be flexible enough to make a true reflection of where I am in my career and decide if I want to continue down the path I am on at that time. If someone asked me eight years ago when I began my athletic training career at Texas Woman’s University if I would ever want to make such drastic changes in my work setting, I would have scoffed at the idea and responded with never in a million years. Only eight of those million years have passed and my desire to incorporate a different approach to my practice is unfolding along with the potential of either leaving the athletic model or working with my supervisor to transition my current setting to a more medical model patient-centered setting. I believe in mental imagery and positive self-speak to create energy in my life which brings about the events upon which I focus. I will continue to concentrate and visualize my goals for the future because I know if I see them written and they are tangible items, my motivation will be strengthened when times are difficult.

Spending time developing this plan has also guided me to thinking more about my future and how I would like it to appear. Now I have determined where I would like to go, but as in any other trip, I needed to determine the best vehicle for transportation and seek a road map for help along the way. My PoAP is my roadmap and continuing education is the vehicle of choice at this time. The journey is exciting and at times frightening, but worth every emotion spent to arrive at my destination.

Plan/Checklist for Obtaining My Professional Goals

Plan of Advanced Practice Primary Focus Areas

- ➢ Advancing clinical proficiency through advanced continuing education classes
- ➢ Recognizing and treating breathing dysfunction in my daily practice
- ➢ Increasing proficiency in the Mulligan Concept for the treatment of Low Back Pain
- ➢ Developing a plan/algorithms for indications of using the Myokinesthetic System in my patient population
- ➢ Treating Post-Concussive Symptoms with techniques other than prescribed rest
- ➢ Explore Primal Reflex Reset Technique (PRRT) diversity in treating upper, lower, chronic, acute and breathing dysfunction
- ➢ Advance professional scholarship and action research
- ➢ Discover and develop specialization within patient population

Advancement of Continuing Education

- Enroll in progressive treatment and theory techniques seminars; Focus on evidence based effective techniques
- Advanced courses for evaluation of musculoskeletal injuries
- Seek the counsel and advice of known experts
  - ✓ Positional Release Techniques Upper Quarter Course
    - o Completed July, 2014
  - ✓ Mulligan Concept Mobilization with Movement Upper Quarter Course
    - o Completed July, 2014
  - ✓ Mulligan Concept Mobilization with Movement Lower Quarter Course
    - o Completed July, 2015
  - ☐ Mulligan Concept Mobilization with Movement Advanced Course
    - o Target: August, 2016
  - ✓ Selective Movement Functional Assessment Certification
    - o Completed Dec., 2014
  - ✓ Primal Reflex Release Technique (PPRT) Home Study
    - o Purchased August, 2014
  - ☐ Primal Reflex Release Technique (PRRT) Advanced Home Study Course
    - o Target: Jan. 2016

- Primal Reflex Release Technique (PRRT) Live Seminar Basic Course
  - Target: Jan. 2017
- Primal Reflex Release Technique (PRRT) Live Seminar Advanced Course
  - Target: Jan. 2018
- ✓ Total Motion Release Technique progression through high school level and beyond
  - Completed May, 2015
- ✓ Myokinesthetic System Lower Body Course
  - Completed July, 2015
- Myokinesthetic System Upper Body Course
  - Target: July 2016
- Hip and Pelvic Girdle Basic and Advanced Evaluation Techniques
  - Target: Fall 2016
- Cervical, Thoracic, and Lumber Spine Basic and Advanced Evaluation Techniques
  - Target: Spring 2017

Recognizing and Treating Breathing Dysfunction

- ✓ Read Chaitow, et al., 2013 Recognizing and Treating Breathing Dysfunction (
  - Completed May, 2015
- Take 'Spine Health and Trunk Stability: Influence of the Thorax & Respiration Home Study Course
  - Target: December 2015
- ✓ Collect patient-centered and region specific outcomes to determine how best to implement breathing treatments
  - Began Aug. 2015
- ✓ Adjust breathing assessments and treatments based on outcomes research
  - Began Aug. 2015
- Develop deeper foundational knowledge, theory, and principals of the variety of breathing interventions
  - Ongoing

Proficiency in the Mulligan Concept for the treatment of low back pain

- ✓ Attend both upper and lower quarter Mulligan Concept basic courses
  - Completed July 2014 Upper

    - Completed July 2015 Lower
- ☐ Exhaust resources and literature relating to the Mulligan Concept and low back pain
    - Ongoing
- ☐ Consult with Mulligan Concept experts regularly to ensure proper technique
    - Ongoing
- ✓ Use the Mulligan Concept as indicated in all low back pain patients in sub-therapeutic doses to determine progression or course of action
    - Began Sept. 2015
- ✓ Collect patient outcomes to analyze for improvement of the technique and patient response
    - Began Sept. 2015

Develop a plan/algorithms for using The Myokinesthetic System (MYK)

- ✓ Identify patient who would best respond to MYK
    - Began Oct. 2015
- ✓ Proficiency in identifying and correcting postural dysfunction with MYK System
    - Began Oct. 2015
- ☐ Develop deeper foundational knowledge, theory, and principals behind the efficacy of the technique
    - Ongoing

Actively treat post-concussion symptoms

- ✓ Determine techniques that are indicated to treat classic post-concussion symptoms
    - Mulligan Concept headache SNAGS
    - Mulligan Concept cerviogenic dizziness SNAG
    - Primal Reflex Release Technique hair pull
    - Breathing Assessments
- ☐ Collect patient outcomes to determine timing of when to implement the techniques and the efficacy of each
    - Ongoing

Examine Primal Reflex Release Technique's diversity

- ✓ Incorporate PRRT into my daily practice
    - Began Aug, 2015

- ✓ Collect patient outcomes to determine best practice in my patient population
    - o Began Aug 2015
- ✓ Purchase Basic Home Study
    - o Completed July, 2014
- ✓ View all Reflex Speed Pain Release Videos
    - o Completed Oct. 2015
- ☐ Purchase Advanced Home Study
    - o Target: Jan, 2016
- ☐ Attend Live Seminar Classes (Basic and Advanced)
    - o Target: Jan, 2017 Basic
    - o Target: Jan, 2018, Advanced
- ☐ Exhaust all pertinent manuals, videos, and instructional manuals pertaining to the principals, guidelines, and theories behind the technique
    - o Ongoing

Advance Professional Scholarship and Action Research

- ✓ Maintain patient outcomes for use in analysis for disseminating of evidence in my clinical practice
    - o Began Aug, 2014
- ✓ Compose scholarly manuscripts for submission to peer-reviewed journals
    - o First submission, Oct, 2015
- ☐ Disseminate evidence of clinical practice outcomes and action research
    - o Edit manuscript, submit for publication and present "The Effect of Treating Breathing Dysfunction on Anterior Hip Flexor Pain"
        - ▪ Target: Sept. 2016
    - o Edit manuscript, submit for publication and present "The Effect of the Myokinesthetic System on chronic knee pain associated with osteoarthritis"
        - ▪ Target: Sept. 2016
    - o Resubmit manuscript for publication and present pilot data "The Mulligan Concept "Squeeze" Technique: A Case Series on an Alternative Approach to the treatment of Meniscal Pathologies"
        - ▪ Target: May 2016

- o Complete manuscript, submit for publication and present dissertation study "An Alternative Approach to the Treatment of Meniscal Pathologies: A Randomized Shame-Controlled Trial of the Mulligan Concept "Squeeze" Technique"
  - ▪ Target: July 2016
- o Complete analysis and composition of Reliability Study on the Clinometer App manuscript
  - ▪ Target: July 2016
- o Complete analysis and composition of the Survey of athletic trainers' treatment of meniscal tears manuscript
  - ▪ Target Dec. 2015
- o Complete long term follow up study of meniscus patients treated with the Mulligan "Squeeze" technique
  - ▪ 3 months
  - ▪ 6 months
  - ▪ 12 months

Develop specialization and job setting evolution

- Specialize in an area of rehabilitation of musculoskeletal injuries and incorporate psychological components within physiological approach
- Continue working exclusively with athletic/active female population

✓ Collect outcomes on active female population
- o Began Aug, 2014

✓ Consult with other athletic trainers in medical model athletic training clinics
- o Began Aug, 2014

□ Consult with current administration about adjusting job description and responsibilities to include more attention to rehabilitation and advanced patient care
- o May 2016

□ Incorporate outcomes collection within injury tracking software
- o August 2016

□ Develop Treatment Based Classification within my patient population
- o August 2016

References

1. Chaitow, L., Bradley, D., & Gilbert, C. (2nd ed). (2014). *Recognizing and Treating Breathing Disorders.* London, England: Elsevier Health Sciences.
2. Cook, G., Burton, L., Kiesel, K., Rose, G. & Bryant, M. (2010). *Movement: Functional Movement Systems: Screening, Assessment, Corrective Strategies.* Aptos, CA: On Target Publications.
3. Falvey, E.C., Frankly-Miller,A. & McCrory, P.R. (2009). The groin triangle: a patho-anatomical approach to the diagnosis of chronic groin pain in athletes. *British Journal of Sports Medicine, 43,* 213-220. doi: 10.1136/bjsm.2007.042259.
4. Grappendorf, H. & Lough, N. (2006). An endangererd species: characteristics and perspectives from female NCAA division I athletic directors of both separate and merged athletic department. *The Sport Management and Related Topics Journal, 2*(2), 6-20. Retrieved from: http://www.thesmartjournal.com.
5. Iams, J. F. (2012). Primal Reflex Release Technique Home Study Course. Retrieved from: www.theprrt.com.
6. Laslett, M., Aprill, C. N., McDonald, B., & Young, S. B. (2005). Diagnosis of sacroiliac joint pain: validity of individual provocation tests and composites of tests. *Manual therapy, 10*(3), 207-218.
7. Mulligan, B. (6th) (2010). Manual Therapy NAGS, SNAGS, MWMS etc. Wellington, New Zealand: Plane View Services Ltd.
8. Neibert, P.J. (2009). Novice to expert practice via postprofessional athletic training education: a grounded theory. *Journal of Athletic Training, 44*(4), 378-390. Retrieved from: http://www.nata.org/jat.
9. Perrin, D.H. (2007). Athletic Training: from physical education to allied health. *Quest, 59*(1), 111-123. Retrieved from: http://libres.uncg.edu.
10. Ossipov, M.H., Dussor, G.O. & Porreca, F. (2010). Central modulation of pain. *Journal of Clinical Investigation, 120*(11), 3779-3787. doi: 10.1172/JCI43766.
11. Seuss, D. (1990). *Oh, the Places you'll go!* New York: Random House.