An Exploratory Analysis of a Novel Treatment-Based Classification Algorithm for Treating Tendinopathy

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

Janet McMurray

Major Professor: Russell Baker, DAT

Committee Members: Alan Nasypany, EdD; Don Reordan, MPT; Marsha Rutland, ScD

Department Administrator: Phillip W. Scruggs, PhD

January 2016

Authorization to Submit Dissertation

This dissertation of Janet McMurray, submitted for the degree of Doctor of Athletic Training with a Major in Athletic Training and titled "An Exploratory Analysis of a Novel Treatment-Based Classification Algorithm for Treating Tendinopathy" 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, DAT

Committee Members: _____ Date: _____
Alan Nasypany, EdD

_____ Date: _____
Don Reordan, PT

_____ Date: _____
Marsha Rutland, ScD

Department Administrator: _____ Date: _____
Phillip W. Scruggs, PhD

Abstract

A Dissertation of Clinical Practice Improvement (DoCPI) is a comprehensive document detailing the evolution of a scholarly practitioner and progression towards advanced practice. The DoCPI will include a Plan of Advanced Practice (PoAP) which discloses the development of the clinician and provides the outline for attaining advanced practice in chosen areas of focus. The PoAP details professional philosophies, personal clinical reflection, and patient care. The DoCPI will also include a summary and analysis of collected patient care outcomes from clinical practice and residency findings, depicting improvement in clinical practice, development of advanced clinical skill, and advancement of clinical reasoning and competence. The inclusion of a literature review on etiology, classification, causative factors, and common treatments for tendon disorders serves to demonstrate foundational knowledge of a focus area. Finally, evidence of advanced clinical practice and scholarly development is provided included research products.

Acknowledgements

Though only my name appears as the author of this dissertation, there are many people who have contributed to its production and helped it come to life. I am pleased and humbled to acknowledge them here.

Dr. Russell Baker, my committee chair, a dedicated advanced scholarly practitioner: thank you for your guidance and instruction. His insightful comments and constructive criticisms were thought-provoking and helped me focus. He set high standards for my research, personal practice, and quality of intellectual friendship. He introduced me to action inquiry—a discipline which extends beyond this project—and his writing has inspired me steadfastly through this dissertation process. Thank you for providing me with the confidence to stay the course as I began my journey to becoming an advanced scholarly practitioner.

Other thanks to Dr. Alan Nasypany, committee member, for his knowledge and courage to challenge the status quo. I am grateful for your vision and appreciate your innovative spirit. I appreciate the challenge to improve personally and professionally.

I'd like to thank Dr. Marsha Rutland, committee member, for her constant support and encouragement. Her support helped me overcome setbacks, stay focused, and complete the process. I deeply appreciate the professional and personal friendship we have cultivated.

Don Reordan, committee member, thank you for your work and dedication to the Mulligan Concept; your guidance in my research of the Mulligan Concept was greatly appreciated.

I am indebted to the members of the tendinopathy group with whom I have interacted during the course of my journey. I greatly value their friendship and commitment. I am thankful for the pursuit of deeper learning with the group.

To my colleagues and friends at McMurry University, thank you for your continued support and advice. Most particularly the Kinesiology faculty members, who gave me space and the time to go about my work while encouraging me, I am highly appreciative.

Dedication

To my sisters, Dawn and Terry, for what we have shared. Terry I am sorry you were unable to see me through the end. Dawn, for knowing who I am and where I came from, I thank you for continuous love.

To my children, Zach, Kendall, Sam and Megan, you are my life. Zach, I miss you every day. Kendall, thank you for your ready smile and laughter, I welcomed the relief during stressful times. Sam, I thank you for the patience shown and your availability. My struggles with computers and linguistics were lessened with your expertise; my work improved immeasurably with your help. Megan, I thank you for believing in me, when I did not believe in myself. I deeply appreciate your loving spirit. I am profoundly thankful for your love and proud to call you my children.

To my husband, David, my most important source of support and strength. Your friendship, humor, counsel, and encouragement have sustained me as I never imagined. None of this would have been possible without your enduring love.

I love you!

Table of Contents

List of Tables

List of Figures

CHAPTER 1: Narrative Summary

Narrative Summary

Traditionally, doctoral degree programs prepared students for careers as academic scholars, researchers, and university professors. The relevance of this model is now being questioned, however, as fewer students are seeking this path (Willis, Inman, & Valenti, 2010). New forms of doctorate programs are emerging because of the increased demands for a broader range of professional knowledge, skill and expertise (Altbach, Reisberg, & Rumbley, 2009). The professional practice doctorate, as opposed to the traditional academic doctorate, emphasizes augmenting clinical skills and theory with research and is designed to prepare students as learned clinical professionals rather than pure academics. A salient component of most professional practice doctoral programs is the professional practice dissertation (PPD), which underscores equally the importance of field experience, coursework, and research. The PPD is more practical in nature with a focus on developing solutions to real world problems in professional clinical patient practice (Willis et al., 2010).

Recognizing the shortcomings of traditional academic doctoral degrees for athletic trainers, the University of Idaho (UI) athletic training (AT) faculty created a professional practice Doctor of Athletic Training (DAT) degree to develop scholarly advanced AT professionals by structuring a program to facilitate development of research skills, AT advanced clinical practice, and expertise that the profession demands. The multidisciplinary approach of didactic coursework, reflective practice, and clinical research provides clinicians with the necessary resources to pursue an individualized area of advanced practice (Nasypany, Seegmiller, & Baker, 2013). The Dissertation of Clinical Practice Improvement (DoCPI), the PPD in the DAT, is the culmination of scholarly work for the DAT student.

The DoCPI is designed to develop scholarly advanced practitioners through the integration of several key components which include: the students' plan to increase foundational knowledge and clinical skills (i.e., Plan of Advanced Practice [PoAP]), reflection on, and analysis of, clinical practice outcomes data, a literature review, and completion of an applied clinical research project and manuscript.

One of the foundational components of the DoCPI is the PoAP (Chapter 2). Unique to each clinician, the PoAP is created after identifying clinical care weaknesses and strengths, most often based on clinical knowledge in patient care (e.g., evaluation, diagnosis, treatment, research-based evidence). Critical self-appraisal and clinical interest help determine the specific focus area a clinician pursues in his or her advanced practice. Further factors which may influence this decision are clinical environment and resources, patient population, and local clinical problems. A clinician establishes time-sensitive goals to address clinical weaknesses and advance expertise in the desired clinical research area. Frequently, the PoAP is reviewed and revised throughout the DAT program attendance to account for goal prioritization and achievement. These periodic reviews are essential for identifying any ongoing or remaining weaknesses needing attention, while also allowing for refinement of a developed plan for continued professional development. The structure and utility of the PoAP provides guidance and accountability for the DAT student in their educational, scholarly, and clinical practice goals.

Though some may share similar strengths or weaknesses, each DAT student has been impacted by their own life and clinical experiences. Critical self-reflection provides valuable insight required to develop the PoAP and direct the clinician to advanced practice. As a stakeholder in the PoAP, the student is motivated to conceive and complete the stated goals.

Though the accomplishment of professional goals is satisfying, every student must understand the important role improved patient care plays in advancing AT.

Improvement and advancement in patient care begins by utilizing and analyzing patient outcomes collected in clinical practice (Chapter 3). In the UI DAT program, students are instructed to incorporate numerous outcome measures into their clinical practice to determine the effectiveness of various treatment interventions in his or her patient population. The anticipated effectiveness of treatment interventions is based on the best available research evidence for that therapy, if any exists. Patient responses to the clinical interventions are collected using global and specific outcome measures. Through outcome measure data analysis, and ongoing reflection, a clinician may be able to identify who will benefit from a particular intervention within their practice, therefore improving clinical practice and patient care. A predicted patient response to specific interventions provides the basis for production of practice-based evidence (PBE). There is a demand for practice-based evidence, as it is conducted in real world settings to solve real clinical problems, unlike basic science research (Nasypany, May, & Krzyzanowics, 2014). Clinicians producing PBE can generate general applicable knowledge and clinically relevant research questions, thereby closing the gap between basic science research and field research (Nasypany, May, & Krzyzanowics, 2014).

Currently, the medical community endorses evidence-based practice (EBP), which is the utilization of the current best evidence available in making decisions about patient care (Sackett, Rosenberg, Gray, Haynes, & Richardson, 1996). Though utilization of EBP is a critical component in improving patient care, there are often roadblocks to its acquisition and implementation. Clinicians may have difficulty locating and remaining current with

published clinical research findings. Applying knowledge from the basic sciences can also be problematic, as it takes an 0average of seventeen years for clinical research to be fully integrated into daily practice (Green, 2008). Even when evidence is widespread, clinicians may still make decisions based on habit rather than published evidence (Balas, & Boren, 2000). Though EBP continues to be deficient in most athletic training settings, the DAT faculty addresses concerns surrounding EBP while providing the necessary guidance to initiate change.

In the UI DAT program, students learn applied clinical research is not the exclusive province of academia. Students are encouraged to integrate EBP and PBE by utilizing an action research approach to advance knowledge in their clinical practice. Action research (AR) is intended as a collective and collaborative inquiry between researcher and participant and requires the researcher to begin with a local problem to analyze (Koshy, Koshy, & Waterman, 2011). Once the problem has been identified, a plan of action is developed and implemented to improve the issue. Observation of the effects of the action and reflection on outcomes provides the basis for further research planning (Koshy, Koshy, & Waterman, 2011).

Utilizing these action research principles, DAT students learn how to solve local clinical problems (e.g. treatment for low-back pain) by first identifying a specific patient care issue that requires additional investigation for it to be thoroughly understood. After a careful review and analysis of the current published literature is conducted to gain a thorough appreciation for the problematic patient issue (Chapter 4), students develop a research proposal and methods to investigate the specific patient problem within their clinical practice. The research proposal investigation then includes the collection and

analysis of patient outcome data. Though the analysis of patient outcome data may initially lead to the development of a proposed solution, further refinement and revisions to the research methodology may be required for relevant change to be demonstrated. Final proposed solutions to the clinical problem are shaped jointly by patient responses, treatment outcomes analysis, clinical reasoning, and thoughtful practice reflection. The resulting solutions benefit both the patient and the DAT student. As a stakeholder in the process, the patient is the recipient of a meaningful solution. The hands-on AR research experience augments the student's existing knowledge, allows for the dissemination of resulting findings, propagates professional knowledge, all while providing evidence of the DAT student's progress towards advanced practice and the ability to demonstrate scholarship.

The element of reflective practice is instrumental to understanding the role of action research in clinical practice and is a fundamental concept developed in the UI DAT program. In the literature, reflective practice is synonymous with critical thinking, problem solving and learning when and what questions to ask (Wright, 2002: Fisher, 1990). Students in the DAT are encouraged and taught during the degree program to reflect on their evaluations methods, treatment selections, intervention applications, and the results of each intervention. The reflection, accomplished often in documentation of patient care and weekly journal entries, enables students to analyze their clinical-decision processes, which leads to improved clinical practice. The rationales for clinical decisions were particularly evident during weekly discussions between DAT students and faculty when various perspectives on singular patient cases and outcomes were presented. The reflections also allow students to realize clinical reflection's importance in directing clinical decisions, improving patient care, and as a component of advanced practice.

I experienced profound change and growth in several areas in my efforts to become an advanced scholarly practitioner. My depth of knowledge increased through the UI DAT academic coursework and clinical experiences, however, my greatest improvement came in my clinical reasoning. I had not advanced my clinical reasoning skills prior to acceptance in the DAT program.

Sound clinical reasoning is a complicated practice, not based merely on decision-making and judgment alone. Advanced clinical reasoning—developed in stages through both academic and educational experiences—is characterized by the transition from biomedical knowledge and deductive reasoning process exercised by novice clinicians to the forward reasoning and clinical knowledge evidenced by expert clinicians (Wainwright, Shepard, Harman, & Stephens, 2010). Prior to the DAT program, my clinical reasoning process was underdeveloped, firmly grounded in the novice stage. I routinely made clinical decisions out of habit, with little regard to the patient experience or input. In the UI DAT program, engaging in didactic coursework and clinical experiences guides students through the stages to become an advanced practice clinician exhibiting advanced clinical reasoning and inclusive of patient perspectives. On-going data collection and analysis directed my clinical reasoning processes and I began to more readily recognize patterns associated with specific conditions. With my growing insight of clinical conditions, I was able to determine the most appropriate interventions for my patients by integrating both my expertise and experiences.

I further discovered my patient care improved when I took a patient-centered approach in my clinical practice. Originally, I built my clinical practice with the belief I should be the expert, directing all aspects of patient healthcare; I did not consider the patient

or their feed-back as integral components to improving care. The utilization of patient-oriented outcomes was instrumental in producing positive clinical care outcomes. The patients became active participants in their healing and rehabilitative process, realizing the care they received was patient-centered and I ensured their input was valued.

Through DAT coursework, I expanded my knowledge base and clinical skills while learning the importance of meaningful change in patient care. I conducted research in my clinical setting utilizing an action research philosophy and was encouraged to disseminate research results through professional publications, furthering my development as a scholar. My development continued each semester and additional evidence of my clinical growth and progress towards advanced practice AT clinician is delineated in my DoCPI.

The DoCPI documents and affirms my professional journey towards advanced practice as an AT clinician. Created after extensive personal reflection and critical analysis of my professional history, my PoAP is a guide with specific goals for continued scholarly growth and clinical practice development. My PoAP (Chapter 2) begins with an analysis and summary of my professional experiences and clinical work as an AT. My professional growth as a DAT student is illustrated in my PoAP, and goals associated with becoming an advanced practitioner are described. The inclusion of certain professional patient care philosophies provides perspective and insight into my evolving clinical practice. My clinical focus area, the development of diverse manual therapy techniques for acute and chronic injuries, was chosen after reflective appraisal of my professional strengths, weaknesses, and clinical interests. I outlined specific measures and educational experiences intended to bolster my competence and advance my expertise in manual therapies. Finally, future goals

are listed to establish my continuing course toward advanced practice after finishing the UI DAT program.

Chapter 3 contains elements central to the clinical and action research component of the UI DAT degree requirements: an outcomes summary, final residency findings, and the impact of those findings on my clinical practice. Through analysis of these outcome measures, evidence is provided of my progression toward advanced clinical practice and skill development. The personal struggle to find and apply meaning to clinical practice through examining patient outcomes is underscored in the accompanying critical reflection in the chapter. Growth in my clinical and decision-making skills is also demonstrated each semester and subsequently recorded in this chapter.

Lastly, Chapters 4 and 5 present my group research project while enrolled in the DAT. Chapter 4 is a review of the current literature on tendinopathy and its impact on both the general and athletic populations. The group researched the etiology, classification, causative factors, and common treatments of tendon disorders. The literature review was undertaken to understand the clinical context and extent of this problem and to devise a research plan to propose new methods to address the condition. Additionally, Chapter 4 includes our investigation of indirect clinical interventions as proposed treatments for tendon pain.

Chapter 5 contains the group original applied research with a focus on the local problem of tendon pain as seen across several patient populations. The description of the algorithm utilized in the treatment of patellar, Achilles, and lateral elbow tendinopathies is included, with the analyzed patient outcomes data collected during the course of the

research. The results are discussed with the intent of advancing current knowledge on the topic and benefiting clinicians' future practice.

The AT profession is evolving quickly as the scope of clinical practice expands. Due to this evolution, a growing number of athletic trainers are seeking a path to increase clinical knowledge and skills rather than a traditional academic doctoral degree; I am one of those clinicians. In most post-professional programs, the identification and training of expert or advanced practice clinicians is a missing component. The University of Idaho was the first to offer a progressive model with an advanced doctorate degree in athletic training with a patient care focus. The program framework has criteria emphasizing reflective practice to improve foundational knowledge and clinical practice in both the educational and patient care environments while participating in clinical research. Each requisite component for advancement is cataloged in my DoCPI and provides evidence of advanced clinical practice. With the guidance and the achievement provided by my work to achieve the educational and clinical goals outlined in the document, my own journey to advanced practice will be realized.

CHAPTER 2: Plan of Advanced Practice

Plan of Advanced Practice: Finalized July 30, 2015

The Plan of Advanced Practice (PoAP) provides the framework for my evolutionary journey in athletic training toward gaining more professional knowledge and skills as I work to become an advanced practitioner. A detailed plan was constructed after performing a careful analysis and inventory of my current clinical competence, professional strengths, weaknesses, and professional goals. The plan will remain fluid with an element of continual reflection on my educational and professional experiences. As I advance my clinical practice, revisions will be noted in my philosophies and patient care; my PoAP will mark my progress and help shape my future.

Current Clinical Competence

Reflection on professional experience and development. The decision to major in pre-veterinary medicine at Lubbock Christian University for my baccalaureate studies was based on an interest in science and healing; marriage and children delayed my formal educational journey. Though I did not complete my degree, I continued to take courses, focusing on core courses required by most universities. The courses, despite lacking academic rigor, were engaging and ultimately fueled my interest in the human body and movement. That same interest led me to the Cooper Clinic in Dallas, Texas, where they emphasized preventive health care. During a one week stay at the clinic, I was introduced to the concept of wellness and made the decision to finish my academic studies. I wanted to enter a profession where I could educate people about the disciplines of nutrition, physical activity, and wellness.

I began the next chapter of my educational journey at McMurry University, a small university in Abilene, Texas which the strongest science and physical education departments in the area. I enrolled, intending to focus on nutrition and wellness. The Physical Education Departmental Head encouraged me to seek acceptance into the athletic training program; although I did not have any knowledge or understanding of athletic training, I did as he asked and was accepted. The athletic training program was an apprenticeship model following the Texas state licensure curriculum. Course work was limited, and lab hours were spent completing duties the head athletic trainer deemed appropriate. Emphasis was placed on establishing rapport with coaches and athletes rather than building foundational knowledge.

The apprenticeship program was considered very "hands-on," which meant athletic training students covered most of the practices and games with little or no supervision. Educational competencies were not established nor required to pass the lab. We rarely practiced evaluations, and assessments of musculoskeletal injuries and palpation skills were not developed. Strategies or rationale for treatment interventions were not discussed as most treatment interventions were similar. A strong work ethic was prized over knowledge or skills.

As I spent time around other athletic trainers, licensed practitioners, and students, I began to doubt the strength of my learning experiences, particularly when watching other professionals perform evaluations and assessments on athletes. Their evaluations were more deliberate, systematic, and heavily focused on anatomy and special tests while mine were based on the location of pain and the specific anatomical tissue producing the pain. I completed the apprenticeship program in three years, graduated, and passed Texas state

board licensure in 1996. In 2002, I secured the assistant athletic training job at McMurry University; however, my excitement was tempered with apprehension as national certification was required for the position.

When I sat for my national certification exam, it was clear I needed more knowledge. I passed two sections of the national certification exam with ease and failed the practical portion. My failure clearly demonstrated the flaw of the hands-on approach of my apprenticeship program: the lack of required competencies. I was surprised by the number of questions relating to anatomy and muscle and special testing that had not been covered in our apprenticeship curriculum. I sat for the practical national certification exam three times before passing. The experience shaped my intent to further my clinical knowledge with post-graduate studies.

I received a master's degree in education from Hardin-Simmons University in 2002 with the hope this educational experience would, in time, assist me in achieving a greater level of clinical knowledge; unfortunately, my new degree did not help meet my expectations. The degree included an interdisciplinary approach to education with emphasis placed on outdoor adventure education and sport and recreation management. Only two courses in the curriculum were relevant to my discipline: gross anatomy and biomechanics. The biomechanics course was taught by an athletic trainer with a good working knowledge of the subject, and our assignments were application-based with a focus on movement assessment. I took gross anatomy with the first year physical therapy students; my schedule allowed me to attend the class and participate in the lab. Both were good learning experiences, and I felt I had increased my knowledge base with completion of the courses.

After several years, I was named the Head Athletic Trainer at McMurry University. Soon after, a discontent—born out of my yearning to become a more knowledgeable athletic trainer and frustration and dissatisfaction with my clinical practice—pushed me for further change and growth. A peer provided me with information regarding the University of Idaho Doctorate of Athletic Training (DAT) program with a curriculum based on aspects of clinical practice; I felt I found what I was looking for in an educational program for athletic trainers. Previously, I had not found a PhD program directly applicable to my work and hoped the DAT would challenge me while providing the means to acquire knowledge and skills needed to improve my clinical practice.

Reflection on current knowledge. When I applied to the DAT program, I felt certain I was not a novice as an athletic trainer due to my years of practicing in the profession. The number of years practicing does not automatically confer expertise; however, it did provide more opportunities for valuable experiences both in and out of the clinic. I was not an expert in any one area; although, I felt confident in my rehabilitation knowledge and skills. I had begun to explore more non-traditional methods to treat patients, discovering instrument assisted soft tissue mobilization (IASTM), (e.g. Graston®). After attending two Graston® workshops, I utilized this treatment clinically. After I attended a course in movement dysfunction and assessment, I decided to implement some changes in my practice based on this experience. However, at the end of day one in the DAT program, I was challenged to see my clinical practice and patient care in another light.

Feelings of personal and professional inadequacy surfaced after conducting a critical analysis on a personal and professional clinical practice level. I was not confident in most clinical aspects of athletic training; I was unsure of my foundational knowledge and

questioned my clinical actions and decisions. Upon reflection, I was not doing much correctly, and this fostered an uncomfortable, disconcerting experience. Therefore, I began to review anatomy and foundational knowledge via textbooks, Northeast Seminar videos to reinforce my knowledge base and learn new material.

At the end of the first DAT summer session, I knew I was standing on the cusp of change thanks to an introduction to new concepts and an altered perspective on patient care. I felt rejuvenated yet anxious to return to clinical practice. After learning so much in a short time, I felt eager to implement everything discussed during the summer in the clinic. I wanted to make a difference in my patients' care by decreasing their pain and increasing their functional status while collecting measurable outcomes. The results were only partially successful. Each stumble gave me pause to study, review, and reflect on the reasons why I was not succeeding as I intended; each challenge bolstered my technique while sharpening my clinical reasoning.

Reflection of strengths. Throughout the last year and a half in the DAT, I have become more confident as a practitioner in my clinical practice. The exposure to mindfulness in the DAT has facilitated a greater understanding of my patients' experiences and allowed me to become more attentive to their needs. This insight, coupled with collecting outcome measures, sharpened my clinical reasoning. I found myself better able to determine the best approach in my patient care as I became a more considerate, thoughtful practitioner. With this patient-centered approach, I found patient care improved as patients became more engaged in their treatment. Interestingly, the athletic training students noticed this change and also became more involved. The students' reactions made me keenly aware of their interest to my work in the clinic.

This renewed interest in my clinic has also led to more autonomy within my program. My professional role requires that I oversee the McMurry University athletic training program; this position creates further opportunities to educate the athletic training students. I may have missed or skipped some valuable teaching opportunities prior to the DAT, but now I am more passionate about my role as an educator and diligently educated the students. I have demonstrated new techniques while articulating the rationale for choosing specific interventions, and student questions have led me to better understand the concept myself. The patients I treated contributed to my deeper understanding of clinical reasoning as they questioned my decisions related to their treatment. Consequently, my explanations had to be clear and valid. A few patients were Kinesiology professors and possessed a depth of knowledge other patients and students did not have. Therefore, my understanding of each paradigm had to be thorough to effectively communicate with them on a professional level. My diligence has paid dividends, yielding a greater depth of clinical knowledge while enhancing my communication with patients to effectively counsel and educate them.

I continue to be open-minded with regard to my education and my willingness to learn and use new concepts have not wavered. Even if I lack confidence in certain roles or tasks, I feel compelled to try. The DAT brought to my attention a number of unfamiliar paradigms, and I believe there are many more to discover on my own. There is much more to learn, and I accept the challenge to implement and understand each new concept.

Specific strengths.

- Educator
    - I am dedicated in my role of educating athletic training students on new concepts.

    - I educate my patients on new concepts utilized for their care.
    - I educate other professionals on new concepts I have learned.
    - I have advanced my knowledge and skills as a scholarly practitioner.

- Clinician

    - I am cognizant of being present during patient care.
    - I treat every patient as a unique individual.
    - I am an effective communicator.
    - I have advanced my manual therapy skills to an intermediate level and am working on transitioning to expert with a plan for improvement.
    - I possess advanced IASTM clinical reasoning and skills.
    - I keep an open mind to new concepts and seek out new ideas to implement in my clinical practice.
    - I accept being challenged to improve my clinical practice.
    - I enjoy learning.
    - I have become competent in working in multiple clinical paradigms.

Reflection on weaknesses. Early in the DAT, I identified two critical areas of weakness: clinical reasoning and foundational knowledge. I have improved in both of these areas while in the program—though my decision-making and reasoning requires further examination as I progress as a clinical professional. I need to strengthen my foundational knowledge in neuroscience (e.g. neuroanatomy, neurophysiology) so I may better educate my patients concerning their pain experience and make the most appropriate clinical decisions regarding intervention choices. Despite improvements as a writer and disseminator of knowledge, I continue to struggle with the level of professional scholarship expected of an advanced practitioner.

Occasionally, I have found myself engaging in old behaviors, particularly during patient assessments. My tendency was to have a diagnosis in mind before I completed the

assessment, which in clinical reasoning terms is known as backwards reasoning (Patel, Arocha, & Zhang, 2004). I would like to blame this behavior on a shortage of time, but seldom was time a factor. The behavior was closely linked to my propensity for quick resolutions so I could move on to the next order of business and, to some extent, my underdeveloped clinical reasoning. I am keenly aware of the importance I place on the end product; however, with continuous reflection, I expect to disrupt and mitigate this line of thinking.

Additionally, many of my clinical decisions in the past were not based on best practice as I was unaware conceptually of evidence based practice (EBP). My decisions were founded on personal educational and practical experiences as I relied on anecdotal evidence to guide my treatment and intervention choices. My approach to clinical practice is changing: I now base more of my clinical decisions on the best available evidence found in current literature and patient responses to treatment. An important component in improving all clinical decisions is increasing foundational knowledge. Though I made progress in improving my foundational knowledge, my understanding of neuroscience is still lacking.

While I feel confident that more time dedicated to studying neuroscience will help shore up any gaps in knowledge, I do concede the nervous system is quite complex and pain processing in the human body is difficult to comprehend. I have read sections of *The Sensitive Nervous System and Explain Pain* by David Butler in an attempt to glean additional knowledge on the basics of neuroscience. With each new concept or revised theory, another layer of understanding is added with respect to central and peripheral sensitization and the precipitating pain effects after injury. More study is needed by the clinician to understand the role of these concepts in patient care. The influence of neural

components on injury cannot be understated and must be understood order to improve patient care. Possessing a deeper knowledge of pain mechanisms will improve my patient care and allow me to be more adept in choosing interventions and educating my patients on their pain.

My endeavor to improve as a research professional continues and encompasses several areas. Although, my writing has improved each semester, and I had a manuscript accepted for peer-reviewed publication, I continue to labor with writing mechanics, organization, and grammar usage (e.g., tenses). Additionally, I need to become more competent analyzing statistics and interpreting data, particularly when I review research articles.

Specific areas I would like to improve.

- I will continue to engage in reflection specifically related to making clinical decisions.
- I will incorporate more EBP into my clinical practice.
- I will improve my knowledge of EBP, particularly what is relevant to treatment interventions.
- I will improve my knowledge and application of MyoKinesthetic System in patient care.
- I will continue my education on manual therapy to improve my knowledge and skill.
- I will complete the remaining Mulligan Concept courses.
- I will improve my knowledge of pain processing and neuroanatomy.
- I will elevate my writing skills to reflect a scientific writing style ready for publication.
- I will improve critical appraisal of research, notably statistical analysis.

Goals for professional practice. Initially, I pursued an advanced degree to move from the clinic to the classroom; however, the introduction to novel treatment interventions has prompted me to continue to practice athletic training in the clinic. I value greatly my role as a mentor to the students in the athletic training program and my staff. My presence in the clinic allows for more discussion of new paradigms, rationale for use, and the importance of reflective practices. The time spent in clinical practice facilitates my clinical reasoning, improves my manual therapy skills, and provides greater opportunities for me to affect change in patient care, while also educating students.

Clinically, my goal is to pursue advanced practice in manual therapies to treat musculoskeletal conditions. These techniques are part of my current research, and I have become more adept at applying the Mulligan Concept, Positional Release Therapy (PRT) and neurodynamics as a result of my research efforts. The immediate effect of pain reduction and increased function utilizing these techniques piques my interest. I want to explore the various paradigms, build on my knowledge of each, and attempt to discover those techniques yielding the most direct, positive effects on particular conditions. The exploration includes the desire to produce research to determine the most appropriate manual therapies for various conditions, based on collecting and analyzing patient outcome responses. I believe this research would provide invaluable data and help direct further pursuits. I would further like to search out new paradigms, while deepening my understanding of the interventions I presently utilize to continue to improve my effectiveness as a clinician. My overall goal is to build a mastery of a very board, diverse base of manual therapies with more astute technical skill.

Finally, I want to expand my scholarship role in athletic training. My first submission to International Journal of Athletic Therapy & Training (IJATT) has been accepted for publication; I plan to submit other research for publication. Presently, I have two case studies I am working to complete: the first deals with treating tendinopathy with a Mulligan Concept Mobilization with Movement at the elbow; the second covers the utilization of neurodynamics to treat medial tibial stress syndrome. Additionally, I have been approached by my attending clinician to present my tendinopathy research findings upon completion of my degree and work with her on future research projects. I will actively pursue avenues where I may present my research and may continue to work as a scholarly professional.

Athletic Training Philosophies

These philosophies are a comprehensive set reflecting my personal and professional values, and each aspect of my practice is influenced and governed by them. My philosophies guide my practice and how I treat patients, students, and peers. In the future, these philosophies, coupled with continued learning opportunities, will be used alongside the attributes of an advanced practitioner. I will continue to seek out new theories and treatment techniques, record and analyze outcomes and participate in reflective practice. If I fail to follow this path, my patient care declines and I fall back into old behaviors.

Patient care philosophy. My philosophy of care represents more than a viewpoint: it is a commitment. I will conduct myself in a fair, trustworthy manner and uphold professional and ethical standards. Each patient is treated with compassion. I am respectful, empathetic, understanding and attentive to their diverse needs.

Developing a caring relationship is fundamental to improving my patients' quality of life. Collaboration empowers the patient, enabling them to make sound, critical decisions that lead to a higher overall quality of health care.

Rehabilitation philosophy. My rehabilitation philosophy is patient-centered. The rehabilitation process is not based on protocols, time frames, or anatomy alone, but overall patient quality of life. I focus on patients' personal preferences related to their lifestyle to produce positive outcomes. I incorporate an integrated approach in evaluations, focusing on the whole person and not just pathology. Pain reduction with restoration of function guides my decision making process. Emphasis is placed on utilizing manual therapy techniques, as they help me establish a connection with my patients. Patient-centered and evidence-based outcomes are important measurements of therapy effectiveness. Communication and follow-up is also vital to accomplishing the overall treatment goals of returning my patients to their daily lives with optimal functional abilities.

My rehabilitation philosophy reflects my belief in wellness, influenced by my earlier experiences with the Cooper Clinic and the necessity of total well-being with prevention as the cornerstone. No body system can be overlooked in evaluation as each may contribute to pain and loss of function. Rehabilitative decisions are guided by thorough assessments, outcome measures, and patient feedback. Outcome measures can improve patient care by producing opportunities to understand patient-based evidence. Open communication provides an effective way to educate my patients as I assist them in achieving their goals. Implementing this philosophy on an ongoing basis will require me to research literature and attend seminars to stay current on rehabilitation trends.

Spine/Low-back philosophy. My low-back rehabilitation philosophy is aimed at restoring movement, eliminating pain, and returning my patient to their daily living activities. Each patient is recognized as a unique individual with specific needs. A comprehensive, holistic approach serves as the foundation of spinal or low-back rehabilitation interventions. A systematic evaluation of musculoskeletal, myofascial, and neural components while the patient is non-weight and weight bearing are used to determine the full extent of a patient's condition and his or her expectations for treatment. Particular attention is paid to the influence of physical, psychological, and social factors on low-back pain. This enhances the evaluation process and help screens for potential medical or psychosocial issues that may require referral.

I realize the limitations of the pathoanatomical model when determining a diagnosis and underlying causes of a clinical condition, and consider my patient's asymmetry in their human structure and movement. My movement assessment is patterned after Janda's and Cook's approach. I consider the role postural and muscular balance has on function, patterns of movement and contributions of the central nervous system on motor control. The intervention choice is based on my clinical reasoning associated with examination findings but may change depending on patient improvement. I prefer to use non-painful manual therapy interventions, but understand some treatments will not be as comfortable for all as I would desire. Tracking patient response to chosen interventions can help identify specific patients who respond to certain interventions and may guide future clinical decisions. Successfully treating patients with low-back pain requires considering both physical and psychosocial factors, and educating patients is key for producing positive, long lasting results.

Teaching philosophy. Education must encompass more than transmission of knowledge. It should foster inquiry and reasoning skills to allow students with diverse methods of learning to acquire essential knowledge. Varied didactic approaches should be utilized to effectively teach a concept while also engaging students. A passionate teacher shapes the learning environment, cultivates interest, and deepens understanding of the subject matter. Fear from intimidation cannot reside in a classroom because it needs to be an open and comfortable space where every student feels welcome and free to explore, ask questions; comments should be embraced and encouraged to develop passion, creativity, and fun. The learning environment should combine both structure and freedom, so a hands-on approach can be used for applied knowledge in labs or workshops to reinforce students' transition from textbook readers to learned individuals. Mentoring a student is vital to fostering and celebrating this transition in and out of the classroom.

My teaching philosophy is student-centered and relationship-based, focused on learning with undergraduate research, scholarly, and creative activities. As a professor, I endeavor to infuse pedagogy with my passion for athletic training. I seek to impart current, factual information relevant to athletic training while striving to discover the students' proclivity for learning. Each student's learning style is important to his or her success, so I constantly search for motivational strategies. I encourage my students to question while giving direction to their thoughts. Application of knowledge in the clinical setting is necessary to be a successful healthcare provider, so critical thinking skills are developed and tested in that environment. The educational experience should encompass all aspects of learning, empowering students and preparing them to deal with complexity, diversity, and change.

Justification of the Plan of Advanced Practice

My primary goal is to become an advanced AT practitioner. Since beginning the DAT program, I have experienced many changes, both personally and professionally. I understand the importance of integrating academic work with clinical work to improve patient care. I have learned the difference between evidence-based practice and practice-based evidence and how to utilize both in my practice. Conceptually, I comprehend the influence of pain on injury and the necessity to acknowledge this fact. I possess a confidence previously unknown, allowing me to become a better educator and professional.

To determine the impact I envision my advanced status will have on my practice, I must continually analyze my strengths, weaknesses, and interests. I understand as I continue to grow and my practice evolves, my goals will change. I will strive to improve skills and expand my knowledge base to avoid stagnating in my clinical practice. I will endeavor to improve both my strengths and weaknesses while identifying new areas requiring attention; a comprehensive approach will best help me prepare to meet these stated goals.

My PoAP will guide my progress and is crucial key for assessment and accountability. My behaviors will reflect my plan as I meet my personal and professional goals. The PoAP has been a personal catalyst for change. I identified and implemented methods to increase my knowledge in my interest and focus areas. My patient care has been positively impacted by my enhanced knowledge and diverse clinical skills; I can now provide comprehensive healthcare to my patients. My students have also benefitted. As I introduced various treatment paradigms along with EBP, the athletic training students began to understand the importance of outcome measures and reflection when making clinical decisions. I have relished the decisions and opportunities to explore my chosen focus areas

of interest while sharing this information with my colleagues. Although I have acknowledged my weaknesses and understand the present barriers, I cannot foresee what lies ahead. The fluidity of my plan encourages constant reassessment of my goals reducing the likelihood to stray from my course. I am prepared for the future as I continue to work towards advanced clinical practice.

Additionally, my plan advances the dissemination of knowledge within the profession while furnishing, to an external audience, an example of what the future of athletic training will look like. The DAT curriculum supplies the overall means to achieve expertise and advanced practice, while the PoAP supplies the individual way. I look forward to my new clinical practice guided by my PoAP.

Plan of Advanced Practice: Areas of Focus Tables

1. Develop diverse manual therapy knowledge and skills for the treatment of musculoskeletal conditions.
2. Increase EBP in clinical practice.
3. Increase neuroscience knowledge.
4. Develop as a scholarly professional.

Table 2.1.
Manual Therapy Knowledge and Skills

Focus Area 1: Develop Diverse Manual Therapy Knowledge/Skills			
Method	**Description**	**Completed**	**Completion Date**
Graston M1, Advanced Determine other courses to take over the next 2-5 years Collect and analyze outcomes	Upper/Lower Quadrant	X	Fall 2011
PRT Collect and analyze outcomes	Spine & Pelvis	X	July 2013
	UE	X	July 2014
	LE		Spring 2016
Mulligan Concept Completion of advanced and follow-up courses in 1-3 years Take Certified Mulligan Practitioner Exam in 5-8 years Collect and analyze outcomes	UE	X	December 2013/ July 2014
	LE		Fall 2016
Myokinesthetics Utilize in practice to determine effectiveness Collect and analyze outcomes	UE	X	July 2014
	LE		Summer 2015
TMR TMR Collect and analyze outcomes	I & II III	X	Fall 2015 Summer 2015

Table 2.1.
Manual Therapy Knowledge and Skills (Continued)

Focus Area 1: Develop Diverse Manual Therapy Knowledge/Skills (Continued)			
Method	**Description**	**Completed**	**Completion Date**
PRRT Live Training Seminar 2-5 years	Home Study	X	Summer 2014
Neurokinetic Therapy		X	Summer 2015
NES Sahrmann Movement Systems Impairment Approach		X	Fall 2014
NES Feldenkrais Method		X	July 2014
McKenzie Institute Courses	Part A and B		2016-2017
DNS	Basic Course “A”		Spring 2016

Table 2.2.
Increase EBP in Clinical Practice

Focus Area 2: Increase EBP in Clinical Practice			
EBP	**Description**	**Completed**	**Completion Date**
EBP fundamentals: Continued reading and research of best practices in clinical practice in current literature	Glynn-Clinical Prediction Rules		Purchase Fall 2015
	Denegar-EB outcomes instruments in sports med Hamson-Utley et al. Using clinical orthopedic exam (CORE) to facilitate evidence-based practice in the orthopaedic evaluation Sackett-EBM, what it is and isn't Haynes Guyatt-Clinical expertise in the era of evidence-based medicine and patient care Benecuik et al.-Clinical prediction rules for physical therapy interventions: a systematic review		Ongoing
AT Level 1 & 2	NATA (online)	X	July 2014
Assessment	Evidence-based examination of the lumbar spine 3.5 contact hours Movement dysfunction: an evidence-based overview 2.0 contact hours	X	Summer 2015
Treatment	Evidence-based treatment of the hip 3.0 contact hours	X	Summer 2015
Clinical Decision Rules-Diagnosis	Utilization of OAR Utilization of Kuhn's tests-SLAP lesion Canadian cervical spine rules Medial collateral ligament pathology Meniscal pathology SI joint pain Subacromial impingement		Ongoing

Table 2.2.
Increase EBP in Clinical Practice (Continued)

Focus Area 2: Increase EBP in Clinical Practice			
EBP	**Description**	**Completed**	**Completion Date**
Clinical Decision Rules-Intervention	Cervical manipulation for neck pain Cervicothoracic manipulation for shoulder pain Manipulation for patellofemoral pain syndrome Mechanical traction for low back pain Patellar taping for patellofemoral pain syndrome Stabilization for low back pain Thoracic manipulation for neck pain		Ongoing
Outcomes Assessment- Generic Region specific Dimension specific Continue to evaluate patient-based outcomes assessment tools for reliability, validity, and appropriateness	Disability of physically active scale, global rating of change, patient specific functional scale Incorporation of Oswestry low back pain disability questionnaire, Disabilities of the arm, shoulder and hand scoring system, lower extremity functional scale, Fear-avoidance beliefs questionnaire, McGill pain questionnaire		Ongoing
Reflection on clinical practice	Reflection on chosen interventions and patient responses, how practice has changed and improved by adopting a more EB approach		Ongoing

Table 2.3.
Increase Neuroscience Knowledge

Focus Area 3: Increase Neuroscience Knowledge			
Author / Contributor	**Title**	**Completed**	**Completion Date**
David Butler and G. Moseley	The Sensitive Nervous System Explain Pain Neurodynamic Techniques Mobilisation of the Nervous System The Graded Motor Imagery Handbook		1-2 years
	Research and determine appropriate books for reading		Ongoing
Barb Hoogenboom	Neurodevelopmental Sequencing	X	Spring 2015
Medbridge	The Neuroscience of Sprains, Strains, Pain and Sport Performance	X	Summer 2015

Table 2.3.
Increase Neuroscience Knowledge (Continued)

Focus Area 3: Increase Neuroscience Knowledge (Continued)			
Author / Contributor	**Title**	**Completed**	**Completion Date**
American Academy of Neurology	NeuroLearn, NeuroSAE®	X	Summer 2015
NOI	TBD - research courses related to interest		5-10 years
Dermo Neuro Modulation	TBD - research courses related to interest		5-7 years
Coursera	TBD - research for courses related to neuroscience		3-5 years

Table 2.4.
Develop Scholarship

Focus Area 4: Develop Scholarship			
Institute	**Description**	**Completed**	**Date Completed**
IJATT, IJSPT, JAT, ACSM	Submit Tendinopathy Research- pilot data		Six months following completion of dissertation
	Dissertation findings separate studies, elbow, Achilles, and patella		Submit 3 month follow-up and 12 month follow-up within 2 year time frame
	Submit case study on MTSS		Submit within a year of completion of dissertation
NWATA regional convention	Return to Play Presentation	X	Spring 2015
NATA convention	Present at Next Available Convention		On-going
University of Idaho	Submit Manuscript	X	Spring 2015
University of Idaho	Conduct Action Research		On-going

Table 2.4.
Develop Scholarship (Continued)

Focus Area 4: Develop Scholarship			
Institute	**Description**	**Completed**	**Date Completed**
Coursera	Audit Statistic Class		Fall 2015
	Take technical writing course		Within the next 1-2 years
TAPHRED	Submit Presentation		Fall 2015
ACSM	Submit Presentation		Next 1-2 years
CATS	Submit Abstract		Next 1-5 years
IJATT, IJSPT, JAT, ACSM	Dissemination of results from collected outcomes on manual therapy techniques-plan to submit for publication once a year		Next 1-10 years

Table 2.4.
Develop Scholarship (Continued)

Focus Area 4: Develop Scholarship			
Institute	**Description**	**Completed**	**Date Completed**
Various	Engage in Research with Colleagues		On-going
Various	Work with tendinopathy group to continue research and publishing tendinopathy research		Next 1-5 years
Various	Network with scholars who share similar interests (i.e. manual therapy) to continue research for presentations/publications		Next 3-5 years

CHAPTER 3: Outcome Summary, Residency Findings, and Impact

Outcome Summary, Residency Findings, and Impact

My clinical residency and evolution in patient care is highlighted in this summary. Through utilization of novel interventions and analysis of patient outcomes, the depiction of my patient care emerges, providing the reader an opportunity to critically appraise my progression and maturation in clinical practice. Clinical practice has given me the opportunity to be involved in my own research with the intention to improve my practice, addressing issues which have arisen within my practice. My outcome summary offers a description and an explanation for my professional learning as I reflect honestly and critically on my clinical practice.

General Analysis of Growth

Prior to entrance in the DAT, my patient care centered on traditional treatments practiced in athletic training. I followed standard treatment protocols while treating patients similarly, focusing on managing symptoms—most particularly pain. I relied heavily on improving muscular strength and flexibility as measures of my success during rehabilitation. My clinical decisions were based on time and numbers; specifically, how much time I needed to treat the number of patients present in the clinic. I was not entirely convinced of the effectiveness of this approach; intuitively, I understood the need for improvement in my patient care and clinical-decision skills.

I found a way to quantify my effectiveness through the DAT: the DAT challenges traditional patient care in athletic training. Each aspect of patient care and clinical reasoning is reviewed, analyzed, and improved upon. To improve my patient care, I included the use of novel interventions and the implementation of outcome measures in our clinical practice,

part of a larger behavior change which needed to occur. I was a novice in many of these areas, continually questioning my knowledge and skills; changing my practice and thought process caused me considerable anxiety. I struggled to comprehend the theoretical basis of each new paradigm, and my attempts to employ new techniques occurred without well-designed structure during evaluations.

My initial introduction to outcome measures occurred during the summer of 2013, framed as question, posed to my cohort: "Do you believe you are a good athletic trainer; how do you know?" I could not effectively articulate an answer to the question. I had little to substantiate my expertise and no measurable proof. These questions began the dialogue to discuss outcome measures and their importance in patient care. Prior to this experience, the nearest I came to tracking a patient outcome was documentation on return to play. The process of collecting patient outcomes appeared straightforward: with an investment of time, I would have evidence of my prowess in athletic training. Instructions were provided on collecting patient outcomes. Though enthusiastic, I went into fall of 2013 without a plan. I did not completely grasp data collection or analysis conceptually; consequently, the first two semesters of collecting patient outcomes fell short of DAT expectations. Initially, my outcome measure collection was sporadic, and my system of data collection contained little structure; therefore, it was difficult to derive meaning from the few early outcomes I did collect. Though failure is an unpleasant experience, it provides valuable information. The recognition of this shortfall was critical in my development of improving my clinical practice. Although my outcome collection lacked consistency, it did not detract from my success as I progressed through the DAT program.

The evolution of my patient care has been slow, albeit steady. New opportunities arose with each patient as I was challenged to measure clinical outcomes, perform examinations, and determine a treatment plan. With each small success, my confidence grew— whether improvement in my technique or deeper contemplation of my clinical decision. The improvements justified the need for continued change and structure in my practice. The introduction to new paradigms provided a vehicle to change patient care while the addition of new tools to my clinical toolbox swayed me away from utilizing ineffectual traditional treatments. I was no longer content with interventions which produced pain without substantive results. Although my clinical reasoning was challenged more frequently as I increased time in study and reflection, I began to notice a difference in my practice. I became more systematic in my evaluations, using an a priori approach and began to see patterns in injury presentations. Through critical assessment, I began to construct a correlation between intervention choice and outcome data collection. I am pleased with my progress, though I have more to learn. With continued exposure to new ideas, concepts, and techniques, my advancement towards a becoming a scholarly practitioner will be realized.

I was not unfamiliar with all the intervention strategies introduced by the DAT. I had experience with Instrument Assisted Soft Tissue Mobilization (IASTM) and the Selective Functional Movement Assessment (SFMA). Though I attended courses for both techniques before acceptance in the DAT, I soon realized attendance did not automatically confer knowledge or understanding of a particular paradigm, even those I was familiar with. To truly advance my practice, I need to study my current practice, while gaining new knowledge and exploring new paradigms in clinical practice. Through this process, I would

increase my depth of knowledge of current treatment interventions as I learned new approaches to treat and improve patient care.

Primary Treatment Paradigms, Patient Outcomes, and Reflection of Changes

Instrument assisted soft tissue mobilization. Numerous pathological conditions which respond to IASTM have been identified and are typically either chronic or acute soft tissue dysfunctions (e.g. carpal tunnel syndrome, ankle sprains)(Burke, Buchberger, Carey-Loghmani, Dougherty, Greco, & Dishman, 2007; Melham, Sevier, Malnofski, Wilson, & Helfst, 1998). Most commonly, IASTM is utilized in treating chronic inflammatory conditions and pain associated with muscular and joint dysfunction from repetitive activity and postural imbalances (Hammer, 2008; Aspegren, Hyde, & Miller, 2007; Sevier, Gehlsen, Wilson, & Stover, 1995). Instrument interventions can also be applied to a tissue on slack, stretch, or in conjunction with motion (Carey, 2001; Hammer, & Pfefer, 2005, Baker, Nasypany & Seegmiller, 2013). I had utilized IASTM in my clinical practice as my intervention of choice for several years. I relied on this intervention to reduce patient pain, normalize range of motion, and improve movement function. Generally, my patients responded well to IASTM, indicating good anecdotal results, though I did not have any patient outcomes to support these findings. I had also neglected to examine the theoretical basis for IASTM as I readily accepted findings presented at the IASTM courses I attended. Therefore, I utilized IASTM somewhat indiscriminately for numerous conditions without conscious clinical choice. If asked, I could not explain my rationale when I chose IASTM. It is difficult to progress in the DAT without understanding theoretical basis and rationale for your choice of interventions. The importance of those factors drove me to investigate IASTM research studies and its utilization as a treatment intervention more thoroughly. I

started taking a closer look at my cases, attempting to discover true causative factors as I considered treatment choices. Also, I began to question the utilization of IASTM in my clinical practice and wondered if another technique, alone or in combination with IASTM, would produce better results. I incorporated other treatments into my clinical practice, relying on IASTM less. Although I continued to trust the effectiveness of IASTM, I realized without the understanding of the intervention or the outcome measures to support its utilization, I could not maintain this belief.

Initially, when utilizing IASTM, I treated patients while in a static position. I focused on treating soft tissue in a particular area attempting to discover myofascial restrictions. Apparent advantages of IASTM are the manner in which the instruments augment the feel of soft tissue restrictions, providing vibratory feedback and the positive effects of mechanical load to promote tissue change through cellular responses (Hammer, 2008). As my skills improved, I began to apply IASTM in a progressive fashion, transitioning from static positioning to motion. While reviewing the literature on treating soft tissue dysfunctions, I discovered the importance of movement and mechanical load in resolving these dysfunctions. I concluded IASTM with motion—or in conjunction with exercise—would produce better outcomes. Also, the mechanical load provided by IASTM, particularly for tendon disorders, would re-initiate the inflammatory process so proper healing could occur (Hammer, 2008). A common misconception regarding soft tissue mobilization, which includes IASTM, is the idea that these treatments can permanently affect tissue length. The force applied during most soft tissue mobilizations is not likely to produce the required compression, shear, or tension forces to deform fascial tissue; therefore, the improvements seen in tissue extensibility are more likely a result of neural responses to the treatment

(Chaudhry, Schleip, Zhiming, Bukiet, Maney, & Findley, 2008; Schleip, 2003). Possessing a greater depth of knowledge, I chose IASTM as my initial intervention only when I felt it was most appropriate, most often combining IASTM with other interventions. Each time I examined the rationale for my intervention choice, striving to improve my clinical-decision making process. Questioning my clinical decisions better directed my choices and enhanced my knowledge, demonstrating my growth as a clinician while advancing my clinical practice. My development as a clinician using IASTM is best illustrated by exploring my application and assessment of my use of the technique in practice.

I treated four cases utilizing IASTM during my initial semester in the DAT without collecting outcomes. Each patient presented with chronic knee pain having experienced this pain over the course of six weeks to several years. My intervention choice was guided by findings of limited ankle dorsiflexion during the SFMA. Limitations in flexibility of the gastrocnemius/soleus that restrict ankle dorsiflexion have been reported in individuals with patellofemoral pain (Marcum, Bell, Boling, Lewek, & Padua, 2012). My thought process was focused on treating tissue extensibility issues based on chronicity of the complaints. I reasoned, if I could improve flexibility in the gastrocnemius/soleus complex with IASTM, then I could increase ankle dorsiflexion and reduce knee pain. I applied the treatment to patients who were lying prone and focused on applying IASTM to the dorsum and plantar aspect of the ankle, the Achilles tendon, several centimeters above the medial and lateral malleoli, and the anterior and posterior compartments of the leg. Only one patient responded to this treatment: a track athlete who experienced Achilles and knee pain for over four years. Her improvement, however, only occurred in range of motion (ROM) at the ankle that was noted during squat retest of the SFMA; she did not report any meaningful improvement in

her pain. The other three patients did not demonstrate improvement in pain or ROM. Despite multiple treatments over multiple weeks, I was not able to generate meaningful results for these patients using IASTM.

While IASTM is recommended for treatment of tissue extensibility dysfunction (Baker, Nasypany, & Seegmiller, 2013) the effectiveness of this treatment is likely based on sound patient classification. The examination I performed, and my clinical reasoning at the time, could have resulted in an incorrect diagnosis or classification of these patients. Poor classification may have resulted in poor resolution of range of motion (ROM) deficiencies at the ankle and pain reduction at the knee; however, it is also possible that IASTM was simply ineffective in these cases. As I reflected on my patient care, it became apparent that improved patient classification, clinical reasoning, and patient outcomes collection would be necessary to assess the use of IASTM in my practice.

The following semester, I treated one patient, a cross-country runner, with IASTM. The patient reported tightness and pain in his hamstring muscle group while sprinting, and I classified him with apparent hamstring tightness. His SFMA screen, which I was more diligent in applying, led me to identify dysfunction for the upper extremities only; however, in my novice development of the regional interdependence philosophy, I still focused on his lower extremity. My examination was marginally more involved this semester as I also considered the effect of somatic dysfunction (i.e., tender points), but did not find any during my exam. My physical exam was still limited at this time and I utilized no other assessment methods (e.g., neural exam), and moved to treating this patient with a vaguely improved classification. I did, however, do a better job of consistently collecting pain ratings for this

patient pre- and post-treatment, but still lacked in-depth collection of patient outcomes. He rated his pain as 6/10 on the Numerical Rating Scale (NRS), pre-treatment.

I also had now begun to consider other paradigms that could produce efficient outcomes and benefit my clinical decision-making. For this patient, I now thought his underlying issue could be a motor control dysfunction, as opposed to a true tissue extensibility dysfunction (TED), and I began treating with reactive neuromuscular training (RNT). Following two sets of 10 repetitions, the patient ran slowly while focusing on his stride length. Upon completion of running, the patient indicated his restriction was still noticeable with his pain unimproved. The minimal results produced by RNT resulted in a shift in my diagnostic classification back to a TED. As a result, my treatment shifted to an IASTM intervention. My treatment was focused on releasing his restriction after moving from a non-weight bearing to weight-bearing position. I applied IASTM initially over his hamstring and adductors with the patient on a plinth in a prone position, and his knee in full extension. I treated the patient in this static position for two minutes, and then requested the patient flex and extend his knee during the remainder of the IASTM treatment (i.e., three minutes). I had the patient stand and assume a long-stride running position and treated his hamstring with IASTM for two minutes in this position.

Following the two IASTM treatments, the patient returned to activity and he did not report experiencing any pain while jogging or sprinting. As a result of his improvement, no other treatments were performed for this patient. My clinical decision for choosing IASTM was based on the patient response to RNT, which was something I could not have previously utilized in practice prior to the DAT.

While this was improvement, my outcomes indicated the need for further advancement as this patient was treated frequently with IASTM during the track season, suggesting an underlying cause I had been unable to identify.

In my final semester of the DAT, I treated one patient with IASTM only. By this time, I had established a systematic evaluation which directed my intervention choices and improved my clinical decision process. Patient #16304, a baseball pitcher who presented with a first metatarsophalangeal hyperflexion condition, similar to turf-toe, was treated with IASTM-only after my classification. Utilizing IASTM, I was able to reduce the patient-reported pain after the first treatment by two points on the NRS, which was a clinically significant change. A clinically significant change on the NRS is identified by a change of two points (Pool, OStelo, Hoving, Bouter, & de Vet, 2007). Also, this patient's condition was completely resolved with eight treatments as shown in Table 3.1. A review of the literature regarding similar injuries to this area indicates that these results exceeded what has typically been reported in the literature, both in number of treatments and time of discharge. Typically, these injuries are treated over two to four weeks with patients being treated three or four times per week until discharge or resolution of symptoms (McCormick,& Anderson, 2009; Anderson, 2002). Also, three to five days rest is recommended before engaging in activity (McCormick, & Anderson, 2009; Anderson, 2002). The patient did not stop practicing or playing during treatment of his condition.

Table 3.1.
IASTM Intervention for Metatarsophalangeal Injury (Patient 16304)

IASTM Intervention for Metatarsophalangeal Injury (Paitent 16304)						
Technique IASTM	Treatment 1		Treatment 5		Treatment 8	
NRS-Best	3		1		0	
NRS-Worst	7		4		0	
NRS-Rest	2		0		0	
DPAS	40		30		16	
PSFS	2/10 Pushoff	6/10 Running	6/10 Pushoff	10/10 Running	10/10 Pushoff	10/10 Running
FADI	104		55			
GRC	N/A		5			

My progression in the DAT produced a progression in my use of IASTM. Following the completion of my IASTM workshops, I treated all conditions using this modality. During my first several semesters in the DAT, I continued to use IASTM as my default intervention—particularly when I was unable to quickly reduce pain and restore function in my patients. I was comfortable with IASTM and felt my technique was sound; therefore, I relied heavily on this intervention. With the introduction to new treatment paradigms, I began to modify my use of IASTM as I practiced new techniques. Observing the immediate and longer-lasting effects produced by multiple interventions in my outcome measures findings, I found other treatment paradigms more effective, beneficial, and less painful than IASTM. Although several patients requested IASTM, my ability to produce an immediate positive outcomes utilizing another intervention, without unpleasant side-effects (i.e., bruising, soreness), was welcomed. Though I relied on IASTM less, I began to better understand the usefulness of IASTM when conditions were properly classified. Utilizing a treatment screening method to evaluate my patients clarified the involvement of particular anatomical systems (e.g., neural, fascial), providing a means to ensure improved classification of conditions. With proper classification, I could more accurately conclude which conditions would respond most favorably to IASTM treatment intervention. The acquisition of this knowledge helped improve my outcomes when I did utilize IASTM. I continue to think IASTM was an effective treatment method; although, I now realize the importance of analyzing outcomes to determine if my treatment selection was appropriate.

Though IASTM has been an effective treatment selection in my practice, I have not fully studied the combination of this treatment intervention with others. Combining IASTM with another treatment may provide additional benefits not yet known. Most importantly, I

need to continue to discover the most appropriate IASTM treatment parameters for various conditions. Due to the exploration of my application and assessment of IASTM, moving forward, my choice of IASTM as a treatment intervention will be based on sound clinical reasoning and not anecdotal evidence or clinical habits.

Selective functional movement assessment. The SFMA provides a systematic method of assessing movement and dysfunction and is designed to identify the underlying issue causing symptoms or pain (Cook, 2010). Purportedly, if a clinician does not discover the true cause of a patient's symptoms, an incorrect diagnosis is made, resulting in less-than-ideal treatment choices (Cook, 2010). My initial exposure to assessing movement dysfunction came during a one-hour continuing education workshop. The concept intrigued me; I hoped to discover a new way to understand and treat my patients' conditions. I sought out the speaker for more information, and an invitation was extended to attend a SFMA course.

Following the completion of this course, a group of physical therapists and I came together to assess a group of athletes utilizing the SFMA. Eager to practice my skills, I found the SFMA more challenging and time-consuming in the field than during the workshop. Though I labored to complete all the necessary components required for a successful assessment, I saw the importance of the SFMA. Returning to my clinic, however, I seldom assessed movement dysfunction in my patients. Despite recognizing the importance of movement assessment, I only periodically attempted to implement SFMA with limited success.

The inclusion of the SFMA in the DAT presented me with another opportunity to become proficient in assessing movement dysfunction. The necessity of implementing the

SFMA in my clinical practice grounded me; I could no longer be partially committed to utilizing the SFMA in my clinical practice. I knew I had to become competent in all components of the SFMA. I would have to demonstrate proficiency in the basic assessment and corresponding breakouts while exhibiting knowledge in diagnosis and treatment. The incorporation of the SFMA in my clinical practice directed my clinical reasoning. The use of this systematic method during evaluation limited my proclivity for shortcuts. I had to spend additional time describing and explaining movement dysfunctions to my patients while reflecting on my findings. The breakouts were still a struggle for me; however, when I incorporated the SFMA in its entirety, I increased my skill and understanding.

When I initially utilized Selective Functional Movement Assessment, I was unable to completely resolve my patients' chief complaints. Frequently, I would complete a partial SFMA without further investigation, and my tendency was to utilize only the breakouts which were most familiar. For example, in the fall of 2013, I screened four patients using the SFMA; each time, the overhead squat seemed to be the most dysfunctional movement pattern. I used the overhead squat as my default, as I was most accustomed and comfortable with portions of the subsequent breakout. My usual finding was limited ankle dorsiflexion, and I would search no further for tissue extensibility, stability/motor control, or joint dysfunctions. Though I did find limitations in ankle dorsiflexion for each of the four patients, no further issues were investigated and no other breakouts were performed; therefore, it is not surprising my intervention choice was not successful in resolving the patients' complaints.

The inconsistent use and poor application of the SFMA continued into the spring of 2014. I only utilized the SFMA on one patient who presented with left mid-back tightness

without pain. The SFMA indicated dysfunctions in upper extremity patterns, rotation, forward flexion, and overhead-squat, with forward flexion and rotation the most dysfunctional movement patterns. I did not breakout the dysfunctions; instead, I chose Total Motion Release (TMR) therapy and RNT to address his flexion and rotation issues. Retesting rotation and forward flexion with TMR and RNT treatment resulted in mild improvement in his ease of movement, though the patient stated he still felt restricted. Two additional sets of TMR and RNT produced a mild improvement in the restriction and I decided to continue with this therapy. The patient received eight additional treatments at irregular intervals during the course of six weeks with minimal improvement, indicating I had not discovered the underlying cause of his restriction. With additional findings provided by the SFMA breakouts and improved understanding of the new paradigms, I could have chosen a more appropriate treatment to help resolve the patient's condition in a timely manner.

In the fall of 2014, I screened three patients using the SFMA, each demonstrating the greatest dysfunction during the overhead squat. Eliminating the overhead portion, each patient could perform the squat, and I returned to reassess the upper extremity. Slight limitation in internal rotation was seen when compared to the non-involved side in all patients, and internal rotation was the only noted dysfunction. After placing each patient in an unloaded position, internal rotation limitations were still seen; therefore, I choose MWM and Primal Reflex Release Technique (PRRT) as my treatment interventions. The patients responded positively to the treatments (Table 3.2) and I was able to reduce their pain after the first treatment by an average of 3.33 points, a clinically significant change. Without completing the full breakout, vital information may have been missed as Patient #16210 did

return to the clinic a month later experiencing recurring pain. I attributed her pain to her position change on the volleyball court. An additional three treatments, over the span of a week, alleviated her pain. The patient required no further treatments during the remainder of her playing season. Although I did not complete the full breakout, I more closely followed the breakout sequence for the dysfunctional movement pattern. The addition of the partial breakout sequence directed my treatment choice which led to improved patient outcomes. Following this experience I realized the necessity of completing the SFMA and accompanying breakouts in their entirety.

The best representation of my development using the SFMA involves a patient who was a baseball player. In spring of 2014, the player sought me out, complaining of hamstring tightness and low-back pain. He stated this was an ongoing problem he frequently experienced. Previously, his treatment regimen had included stretching and chiropractic adjustments with limited success. After my initial evaluation, I had the patient perform the SFMA top tier movements. His greatest movement dysfunction occurred when performing the overhead squat, unassisted and assisted. His ankle dorsiflexion was not limited, so I moved to using a RNT treatment as an evaluative technique to determine if his dysfunction was due to a stability-motor control issue without completing the full SFMA breakouts. The RNT treatment technique did provide some relief; therefore, I continued to treat him using RNT and prescribed several core exercises. This was routine, as I often advised patients to integrate body core exercises into their fitness routine. Although the patient improved somewhat following his treatment, recurrent pain and tightness hindered his ability to practice. He was unable to participate in several games during the season due to this condition.

The next spring, the aforementioned patient sought me out, complaining of the identical pain and tightness from the previous year. Without reflecting on the case, I prescribed the same treatment protocol. On the second day of treatment, however, I stopped to reflect on my rationale for choosing this treatment. Each semester my goals were similar: systematically collect outcome measures, conduct methodical evaluations, engage in reflective practice, and stay true to paradigm procedures. Thoughtfully, I realized I was not meeting these goals, and my behavior was hampering my clinical reasoning and negatively impacting my patient care. I made a change. I informed the player I needed to reassess his condition, and this would require a more extensive and thorough evaluation. I began with a breathing assessment followed by the slump test; his breathing was dysfunctional and the slump test was positive. I completed the assessment utilizing the top tier SFMA. No change was noted in his top tier movement assessment. Again, his greatest movement dysfunction was found performing the overhead squat; however, this time I continued through the appropriate breakouts.

The breakouts indicated a weight-bearing stability-motor control dysfunction. With these patient evaluation findings, I designed a comprehensive rehabilitation program: breathing reset, rolling patterns, neural sliders, and corrective exercises. The breathing reset and neural sliders brought immediate relief to the patient. Initially, the patient rated his pain at rest as 2/10 and 6/10 during activity on the NRS pain scale. At times, when sprinting, his pain would reach a seven or eight. The patient most often described his pain as sharp and localized just lateral to L-4 on his right side. Following the first treatment intervention, his pain was reduced to 3/10 and within two days, he ceased to experience pain or tightness. Within four days, he returned to practice. He labored to roll, particularly prone to supine

upper body, though he made good progress in a week's time. The progress was also noted in his outcome scores. The Disablement of the Physically Active (DPA) scale score he reported decreased from 49 to 16, while his score on the Patient Specific Functional Scale (PSFS) rose from 2/10 during activity to 6/10. I employed SFMA corrective exercises; however, reverse patterning for the squat proved more effective in correcting this patient's stability-motor control dysfunction. Following Gray Cook's recommended progression; the patient could execute an overhead squat without assistance in less than two weeks. Visibly, his movement was smooth, and he exhibited control squatting and rising. His stability and movement improved rapidly.

Within three weeks, I was able to put him on a maintenance program. He came to understand the connection between disordered breathing and low-back pain. Consequently, he reset his breathing once every one to two weeks during his rehabilitative routine. With the exception of the four days missed during initial evaluation and treatment the patient participated in every practice the remainder of the season. Infrequently, the patient would remark he felt some tightness in his low-back or hamstring, which would resolve with his breathing reset. With an improved evaluation process, I expected the patient to improve. The speed of his recovery, however, surprised me because this patient had experienced low-back pain in the past year. Low-back pain has a high recurrence rate; most people (66-84%) experience a low-back pain episode within a year after initial occurrence (Chevan & Clapis, 2013). Also, with each recurrence, patients exhibited increased pain and a higher level of disability (Carey, Evans, Hadler, Kalsbeek, McLaughlin, & Fryer, 1995). For patients with impaired joint mobility, motor function, and muscle performance, recovery times have been estimated to occur within one to six months (American Physical Therapy Association,

2001). With a thorough examination, however, I was able to match the interventions to the patient's specific dysfunctions, resolving the patient's issue while exceeding the standard recovery timeframe for a low-back pain episode recurrence.

The aforementioned case is an example of how proper utilization of the SFMA directed my clinical decisions and treatment choices, thereby resolving the patient's painful condition. Utilizing the SFMA provided me with the necessary information to correctly identify the patient's underlying cause. With a clear understanding of the patient's issue, I was able to design a treatment program to address all aspects related to his condition. Previously, during my first semesters when using SFMA, I would circumvent steps particularly if I felt the accompanying breakouts were too arduous or time-consuming. What I soon realized was avoiding the accompanying breakouts resulted in poor treatment choices, thereby extending treatment time and the resolution process. Each semester as I became more confident, I completed more of the accompanying breakouts. The additional information provided by the breakouts enabled me to properly identify underlying causes thereby I choose more appropriate intervention measures to address the condition. Consequently, I had fewer patients with conditions I could not resolve and fewer return with similar complaints following treatments.

Table 3.2.
Upper Extremity Conditions Screened Using SFMA

Upper Extremity Conditions Screened Using SFMA						
Patient ID	Condition Treated	Number of Treatments	Treatment Duration	NRS Pre-Intervention	NRS Post-Intervention	Condition Resolved
16210	Left Shoulder Positional Fault	7	2 Treatments a Week for 3 Weeks	3	0	Patient Returned Month Later with Recurring Pain
16211	Right Shoulder Positional Fault	4	2 Treatments a Week for 2 Weeks	4	0	Yes
16212	Right Shoulder Positional Fault	5	2 Treatments a Week for 3 Weeks	3	0	Yes

Mulligan concept. The Mulligan Concept is a manual therapy concept of mobilization with movement to correct suspected positional faults in joints created by injury. The mobilization techniques have been developed to align joint-tracking with subtle biomechanical movements (Mulligan, 2010). Indications for use of the Mulligan Concept MWM include a loss of joint movement, pain associated with movement, or pain associated with specific functional activities. Correct application of mobilization with movement results in a pain-free, immediate, long lasting, (PILL) effect for the patient (Mulligan, 2010). Previously, when I saw patients presenting with restricted or painful joint movements, I would assess them for flexibility and muscular imbalances. If a sizable difference in muscular balance was evident, I had patients begin corrective exercise therapy, incorporating both strengthening and stretching exercises. If no significant disparity was noted, I utilized IASTM. The quick resolution of joint dysfunction utilizing the Mulligan Concept caused me to reconsider my rationale of treatment choice.

My initial case utilizing the Mulligan Concept occurred when a female volleyball player suffered a grade three lateral ankle sprain the first day of practice, mid-August 2013. I began treating this injury with ice and compression immediately after the occurrence. Based on Ottawa ankle rule findings, I sent her to an orthopedic doctor to rule out a fracture. Her X-rays were negative for a fracture; she was instructed by the orthopedic physician not to bear weight on the ankle for six weeks, using crutches and a walking boot during ambulation. The following day, I attempted the MWM for lateral ankle sprains. I discontinued my attempt when the patient exclaimed it was much too painful.

Though her pain rating at that time was 8/10 during rest and 9/10 on crutches, I believe my technique was more problematic than her pain because I was a novice utilizing this intervention technique.

I saw this patient every day, working to reduce her edema and control her pain with compression, IASTM, and gentle ankle-pumping exercises. The administration of those interventions did help reduce her edema and pain; however, I was not satisfied with her gains, particularly regarding her edema reduction as she was 16 days post-injury. Her pain subsided to 3/10 at rest, 4/10 on crutches at week three and I again attempted the lateral ankle MWM. The technique was non-painful, and the patient was able to complete three sets of 10 repetitions. Following the intervention, the patient reported her pain was 1/10. I applied the adjunct taping technique to ensure the mobilization held. The patient reported to the clinic the following day, significantly improved. Her edema was noticeably absent, pain reduction held at 1/10 at rest and on crutches, and considerable increases in Range of Motion (ROM) (+8° in dorsiflexion and +9° in plantarflexion) were found. From that day forward, she progressed rapidly. I continued the adjunct tape application for two more weeks. In total, I treated her with MWM on four different occasions, each time applying the adjunct taping technique following the mobilization.

The patient returned to full weight-bearing activities after 12 weeks of treatment, which exceeded the physician's expected time frame for return of six months. Due to the unknown timeline of ankle ligament healing, there is no well-established time frame for return to play regarding grade 3 ankle sprains, though the percentage of re-injury and chronic instability is large (Hubbard, & Hicks-Little, 2008). The patient did not exhibit any signs or symptoms of instability at the ankle when returning and was able to participate in

the spring season without difficulties. When compared to findings recorded in the literature, approximately 3% - 31% of patients experiencing lateral ankle sprains still exhibit ankle instability at six months, and early immobilization for six to eight weeks is recommended following the injury to combat future instability (Cetti, Christensen, & Corfitzen, 1984; Lamb, Marsh, Hutton, Nakash, & Cook, 2009).

Although I was unable to perform the MWM successfully initially, the positive results experienced through my first case made me eager to attempt this intervention again. I had several more opportunities to treat lateral ankle sprains with MWM, some more successful than others due in part to improvement in my technical skills and reflection on my patient care. For example, after reviewing patient cases, I noticed a pattern that the length of time between injury and application of the MWM appeared to have an effect on outcomes. Similarly, I found it was more difficult to produce a PILL effect for those patients I did not see immediately following their injury. Patients (#16219, #16220, #16301, and #16303) who were not seen immediately (i.e. 3-14 days post-injury) received more treatments and were discharged later than patients I mobilized immediately following their injury. Results are found in Table 3.3. Despite not having immediate access to treat all of my lateral ankle sprain patients with the lateral ankle sprain MWM, the utilization of the intervention still produced a clinically significant decrease in all patients' pain (Pool, et al., 2007), dropping on average over four points on the NRS, following the initial mobilization. Discharge pain scores were collected for Patient #16300, 16301, and #16303 with all reporting pain as zero on the NRS.

Table 3.3.
MWM Treatment of Lateral Ankle Sprains

MWM Treatment of Lateral Ankle Sprains							
Patient Number	Semester of Treatment	Severity	Attempts for PILL	NRS Pre-Intervention	NRS Post-Intervention	Number of Treatments	Discharged After
16219	Fall 2014	Grade 1+	3	7	2	7	3 weeks
16220	Fall 2014	Grade 1	3	5	1	5	3 weeks
16221	Fall 2014	Grade 1+	2	8	3	5	2.5 weeks
16222	Fall 2014	Grade 1	1	7	2	4	2 weeks
16300	Spring 2015	Grade 1+	1	5	0	2	3 days
16301	Spring 2015	Grade 1+	2	2	0	3	2 weeks
16303	Spring 2015	Grade 1+	2	5	2	4	2.5 weeks

My most successful case utilizing this MWM involved a baseball player who suffered a lateral ankle sprain during practice. I was not present at the end of practice when this injury occurred, so I saw the patient the next morning. Moderate edema was present over the anteriolateral aspect of the ankle. The patient reported a five on the NRS, with walking and stepping in the shower the two most painful activities. During the exam, he was still tender to palpation over the lateral ankle and lacked 7° in passive dorsiflexion, 8° in active dorsiflexion, and 9° in active and passive plantarflexion. Initial treatment included lateral MWW with overpressure combined with adjunct tape application. Following treatment, the patient reported a resolution of his pain and upon ROM measurement, an increase of 12° in active dorsiflexion and 43° in plantarflexion. Upon standing, the patient still did not report experiencing any pain. The treatment was repeated once more that week. The patient was pain free in weight-bearing, and I added proprioceptive rehabilitation exercises the next day after initial treatment with the MWM. Proprioceptive and balance training appear to improve functional ankle balance (Mattacola & Dwyer, 2002), and I wanted to ensure the patient did not develop balance deficiencies leading to compensatory movements, especially due to his quick return to practice. My patient's perception of his injury—particularly in regards to healing—led to my decision to incorporate some proprioceptive exercises into his rehabilitation routine. The patient was discharged and returned to practice three days after his initial injury and had no further issues the remainder of the season.

I have also had the opportunity to utilize the Sustained Natural Apophyseal Glides (SNAGS) technique from the Mulligan Concept. The application of the technique, to my surprise, provided relief to a couple of patients suffering from back pain. Prior to the DAT, I

was uncomfortable treating patients with low-back pain and based my treatments on the patient's hamstring flexibility, core strength, and biomechanics related to resistance training. As such, normally I would treat these types of conditions with IASTM alone or in combination with core exercises and counsel the patients on their resistance lifting techniques; however, my application of this strategy had produced limited success. Even if the patients followed my recommendations to decrease load during resistance training and incorporate stability exercises into their routine, they often returned once every seven to 10 days requiring IASTM treatments to manage their pain. Approximately 70% of my low-back pain patients required continued treatment for the remainder of their season to continue to practice and play. The patients who continued their activities without modifications were most often seen in the clinic on a daily basis for treatments. I did not expect to be able to change back pain in any meaningful way from one visit, nor did I expect to be able to quickly discharge back pain patients pain free. The instantaneous effect of the SNAG mobilization, even as a novice using the technique, caused me to reflect on my previous intervention choices and reconsider physiological effects of manual therapy.

The first patient I treated with the SNAG mobilization was a men's basketball player who presented with low-back pain. He stated he experienced low-back pain periodically and blamed improper, heavy resistance training as the cause. He rated his pain as a six with activity and as a two at rest. The patient was unable to complete forward flexion without pain, thus restricting his range of motion and limiting his participation during practice. Another athletic trainer had him riding a stationary bike and foam rolling before practice, though this treatment failed to produce a positive outcome according to the patient. The patient had dealt with the pain for over two weeks when I first evaluated him. He exhibited

muscular tension and guarding, particularly the multifidus, which I surmised was contributing to his pain. Seated, his pain diminished slightly, though no change was noted in his range of motion. I performed the Mulligan Concept SNAG three times at the site of pain: L-3 on the lumbar spine. Upon completion, the patient was pain-free and able to forward flex without restriction. He returned to practice that afternoon. I did not see this patient again for this condition, although he did return infrequently with reports of low-back pain or tightness.

The second case of my utilization of a SNAG involved a patient who was a track participant complaining of pain during back extension, particularly when high jumping in practice and at meets. Pain rating for this patient was a 5/10 NRS score. He only experienced pain while high jumping. During examination, the patient was unable to fully perform back extension while standing or seated. Right erector spinae exhibited muscular hypertrophy, with increased muscular tension. With the patient seated on a plinth, I performed Mulligan Concept SNAGs three times at T-9 level on the thoracic spine while the patient extended his back. Following the treatment, the patient was able to perform back extension, seated and standing, without pain. The patient practiced that afternoon without experiencing problems. I was unable to see the patient again due to scheduling conflicts, though I was informed the patient infrequently returned to the clinic presenting with pain during back extension. Neither of my patient evaluations was extensive; I had not collected enough information to determine the underlying cause or the most appropriate intervention dose. Notwithstanding, my inadequate examination, the Mulligan Concept technique was indicated as an effective treatment. Even as a novice with limited experience, I was able to provide immediate benefit, albeit short-term, which was evidence of my growing skill.

Previously, my treatment of LBP involved multiple treatment sessions in combination with extensive exercise. I now possessed a skill which enabled me to provide immediate relief altering my view of LBP and my previous treatment choices.

My prior treatment successes with LBP patients led to an interest to attend an upper extremity Mulligan Concept course. Upon completion of the course, both my skill level and patient outcomes improved, particularly when treating conditions related to the shoulder. Interestingly, patients' presenting with acute shoulder injuries treated prior to my course attendance required additional treatments to discharge compared to those I treated after attending the course (Table 3.4).

As I became more proficient using the Mulligan Concept techniques I realized the significance of patient response to the mobilizations. A positive response to a Mulligan Concept technique suggested the patient could be successfully treated with the same technique, and due to this response, I began to incorporate the Mulligan Concept into my evaluations. Also, I began to use the Mulligan Concept as a rehabilitative exercise, most notably after reviewing the outcomes following a staff member's treatment. The young man was diagnosed with full thickness labrum tear in his left shoulder and partial rotator cuff tear in his right shoulder. He underwent surgery to repair these structures and was not pleased with the results. He reported a pain level of 6/10 and his ROM was severely limited, particularly in his left shoulder. The incorporation of Mulligan MWM for internal rotation increased range of motion from 34° to 55° in three visits, and his pain was reduced from a six to a two on the NRS. He maintained and exceeded these range of motion gains and began performing some weight-lifting exercises which previously had caused pain. Prior to this experience, I seldom considered patient response to an intervention as particularly

significant when determining treatment choice or designing rehabilitation exercises. Realizing the implication of patient response, I was able to modify and improve my evaluation methods while enhancing the rehabilitative process for my patients. I became more aware of my screening methods and the importance of matching interventions with specific dysfunctions. Additionally, I found rehabilitation can take many forms; it does not necessarily need to include traditional strengthening exercises.

Table 3.4.
Acute and Chronic Shoulder Conditions Treated with Mulligan Concept

Acute and Chronic Shoulder Conditions Treated with Mulligan Concept						
Patient Number	Diagnosis	Condition	Pre-NRS	Post-NRS	Change in DPAS	Number of Treatments
16210	Positional Fault	Acute	3	0	8	7
16211	Positional Fault	Acute	4	0	4	4
16212	Positional Fault	Acute	3	0	8	5
16213	SMCD	Chronic	6	2	19	2
16214	SMCD	Chronic	5	1	21	3
16215	SMCD	Chronic	6	2	33	2
16309	Positional Fault & SMCD	Chronic	4	2	16	4

Positional release therapy (PRT). My introduction to PRT occurred during the summer of 2013. I attended the PRT Spine and Pelvis workshop, taught by PRT Institute, held at the University of Idaho. I was unfamiliar with this treatment paradigm and found my skill level was poor when attempting to locate tender points. I seldom experienced muscular fasciculation when initially applying PRT. My confidence level waned with each attempt to find suspected tender points though I understood the importance of the treatment paradigm. I knew with practice I would improve (as I had done with other techniques), so I began utilizing this paradigm in my clinical practice.

I treated several patients utilizing PRT in the fall of 2013. Based on my patient outcomes, I was able to produce positive outcomes with PRT when treating patients presenting with acute hamstring strain or experiencing pain and restriction of movement in upper trapezius. Although I found some success with PRT, I seldom committed to a true PRT exam to scan for tender points before initiating PRT, did not classify my patients well, and failed to collect outcomes effectively. Therefore, I was able to derive little meaning from my use of PRT during the first fall. In the spring of 2014, I made some improvements in the application of PRT, but reflection of my practice indicated very little true growth as a clinician using PRT. For example, I treated two patients presenting with apparent hamstring tightness, so I performed a scan for tender points prior to PRT application. My reasoning was still lacking at this time as I decided to treat with PRT despite not locating any tender points. Neither patient responded to PRT treatment, so I discontinued the treatment; I should have realized the treatment would be unsuccessful due to the lack of tender points. My lack of growth utilizing PRT was understandable as I had not spent the appropriate amount of time practicing and refining my skill. Additionally, I failed to perform full scans and

abandoned the technique too quickly when I was unable to resolve a patient's complaint quickly.

I was able to complete another PRTi workshop, the Upper Quarter, held at the University of Idaho in the summer of 2014. By taking a second course, I felt that I was better able to locate tender points and had a better understanding of PRT at a conceptual level. During the practice sessions, I was able to position my patient correctly and, in several instances, I was able to find a fasciculation. I began to feel more confident and sure of my skill level. To continue my clinical growth with PRT, however, I needed to test my improved knowledge and skill in clinical practice.

I had an opportunity to assess my skill and knowledge in the fall of 2014. A faculty member sought me out regarding treatments for his lateral elbow pain. The professor's condition was a focus of a tendinopathy group study, which I was a member. We had designed a treatment-based algorithm, which included PRT, to treat specific tendinopathies. I felt the patient's condition provided an opportunity for me to test the algorithm while I worked on developing my skills. Following the algorithm, patient response indicated PRT as the recommended treatment. The patient had been experiencing pain for over six weeks and rated his pain as 0/10 at rest and 3/10 during activity (i.e. full extension) on the NRS. During the scan, three tender points were located: subscapularis, lateral extensors, and brachioradialis. The patient indicated the lateral extensors as his most painful (3/10 on the NRS) tender point. I treated each tender point, beginning with the lateral extensors. Following treatment, the patient fully extended his elbow and experienced reduced pain. I treated this patient a total of six times based on the treatment algorithm.

Table 3.5.
PRT Treatment of Lateral Elbow Pain

PRT Treatment of Lateral Elbow Pain					
Treatment Number	NRS Rest	NRS Activity	DPAS	PRTEE	GRC
1	0/10	3/10	35	22	N/A
3	0/10	2/10	29	17	0
6	0/10	1/10	23	12	2

Although not entirely successful treating this patient with PRT, I still found a useful learning lesson as this patient experienced improvement with PRT. To this point, this patient had been my most successful utilization of PRT—which I attributed to my improved skill level along with better injury classification. Despite the progress, I knew I still wanted to improve using PRT. In this case, my application of PRT produced a MCID on the DPAS following the third treatment, while a MCID on the NRS and GRC did not occur until the sixth and final treatment. While my clinical outcomes were not as substantial as I would have preferred, I was able to produce a clinically meaningful change (i.e., 50% reduction of pain initially on the NRS) utilizing PRT, a marked improvement from earlier attempts. I did not meet the 70% pain reduction recommended by D'Ambrogio and Roth (1997), though other factors may have contributed to this lower reduction in pain. The patient's tender points lacked pain severity and the patient continued to engage in gardening and other activities following treatment, which is not recommended (D'Ambrogio & Roth, 1997). With continued practice, my skill and understanding had improved.

Combining clinical techniques. The ability to effectively use various treatment paradigms enables clinicians to discover the most effective treatments for a patient's condition. Although some patients find relief under one treatment paradigm, few conditions affect physiological systems singularly. Therefore, it is crucial to understand treatment philosophies to be able to effectively determine the most suitable treatment option for each individual case. Each patient must be examined and treated on an individual basis, with the best evidence and matched-treatment being applied in the context of that specific situation. Incorporating a systematic treatment-based approach to evaluations and treatment could provide information needed for proper classification and intervention choice, be it one or several. Understanding the nuances of each treatment philosophy is one of the guiding principles of the DAT.

Initially, during my first semester in the DAT, I attempted to treat patients using only one treatment technique. Although I had some favorable outcomes, I had difficulty understanding why the treatment was successful as I relied on clinical experience with little clinical reasoning. Later in the program, I began to combine treatments—again due in part to my underdeveloped clinical reasoning. The combinations most often occurred when I could not quickly reduce pain in my patients with one treatment intervention. As my clinical reasoning and evaluation process developed, I had a better understanding of my patient, their condition, and which intervention or interventions would most effectively treat their condition.

An early example of this occurred when I combined PRT with MWM for a tennis player diagnosed with a triangular fibrocartilage tear in the fall of 2013. The patient reported to the clinic with a pain rating of 6/10 during activity and 0/10 at rest. He taped his wrist for

support during practice and play, stating the tape provided some pain relief. I initiated a MWM, and following the treatment, the patient rated his pain 2/10. He returned in a week, requesting another MWM because the treatment provided longer pain relief than the standard tape application to his wrist. Again, I initiated a MWM and reduced his pain to 2/10. On his third visit, I scanned for tender points on his forearm and found two. Following PRT treatment, the patient's reported pain was a 1/10. The treatment held for two weeks when the patient returned to the clinic. Unfortunately, my patient outcomes for this case were incomplete, as was my evaluation, so it is difficult to draw large conclusions from this case. The immediate post-treatment benefit for a triangular fibrocartilage tear, however, provides evidence of my development in matching treatments based on patient presentation or response to treatment.

As I progressed through the DAT, I became more adept in this process. In spring of 2015, for example, I treated a patient with an acute muscle strain and found the combination of PRT, PRRT, and exercise effective in reducing pain and muscle tension. The patient (#16305) reported to the clinic three days after injuring his hamstring in a baseball game. He reported his pain as a 2/10 at rest and a 6/10 while walking on NRS. The patient was clearly apprehensive during the exam and stated this was his third hamstring injury. Previously, he had been diagnosed with a grade one plus strain as a senior in high school and grade two strain as a sophomore in junior college.

During his exam, the SFMA screen led me to find a stability, motor control dysfunction at the hip. Additional testing included a PRT scan, slump test and traction straight leg MWM. The Slump test was negative and he did not respond to the traction straight leg MWM. While performing the lower quarter PRT screen, I located a two tender

points, middle sacroiliac and gemeilli on his left side, each with a pain rating of four. Due to exam findings, I initiated PRT as his treatment and produced a 75% decrease in his pain ratings for each tender point. Following PRT treatment the patient walked for several minutes and forward flexed. He stated his pain was barely noticeable, though he felt "something" during forward flexion. I believed the patient's prior history with injury contributed to his feeling and surmised it was related to his increased muscular tension. I initiated PRRT, utilizing percussion technique on his quadriceps tendons to inhibit the hamstring muscle group. Following this treatment, the patient reported minimal pain (i.e., <1) without tightness or feelings of restriction, while walking or during forward flexion. With his pain and restriction diminished, I felt the patient could begin rehabilitation.

The patient was hesitant to begin rehabilitation, indicating his prior rehabilitative process was painful, difficult to complete and time consuming. The exercises I prescribed did not cause pain or discomfort; therefore, I increased his rehabilitation workload during the next two weeks. I treated this patient eight times over the course of three weeks, with complete pain resolution occurring after the third treatment session. I could not locate any tender points following his fourth treatment and discontinued PRT as a result; however PRRT was still utilized prior to each exercise session. The patient progressed through practice during the next several weeks, returning to full practice and play at the end of week three, surpassing his previous return from injury by three weeks. The patient completed the remainder of the season without re-injury.

Although the patient experienced an acute injury, I believe the underlying cause of his hamstring strains was never addressed in previous cases, indicating this was actually a chronic condition. The combination of PRT and PRRT treatment principles helped treat his

immediate symptoms, providing the relief he needed to complete his corrective exercises. Though another treatment intervention other than PRRT may have provided the same benefit, my clinical outcomes indicate my treatment choice was effective and I had more objective data to support my clinical reasoning in the treatment choice. With improved clinical reasoning and treatment selections, my patient care was much improved from my initial semester, and continued to improve as I progressed through the DAT program. The change is best illustrated by evaluating the differences in patient care found in the fall of 2014 and spring of 2015 semesters. The most notable change was in my evaluation methods and selection of treatment interventions. As I modified my evaluation based on patient response to treatments, I choose more effective interventions. My improved patient care is reflected in my outcomes.

Fall 2014. I collected patient outcome measures for a total of 16 patients in fall of 2014, and while my data collection was not as extensive or consistent as needed, my collection was much improved from previous semesters (Table 3.6). An MCID was produced on the NRS, DPAS, and GRC all patients (n=16); however, I still discovered several areas requiring improvement. I reviewed pain scores from pre- to post-treatment and upon discharge, though I did not compare pain scores from initial treatment to subsequent treatments. I discharged patients, on average, following 3.4 treatments, though 50% returned within a three week time period with a similar complaint which I theorized demonstrated either poor injury classification, intervention choice, or both. My patients presenting with chronic conditions needed fewer treatments, a mean of 2.0, than my acute patients, a mean of 4.8; though 75% of my patients with chronic conditions returned to the clinic within a three week time period, compared to 25% of patients with acute injuries. Most often, I

treated chronic conditions using multiple interventions; however, I was seldom attentive to patient response to chosen interventions.

My evaluation methods for chronic conditions were also insufficient and I struggled to treat patients presenting with LBP and MTSS. I did not incorporate the specific anatomical outcome measure for any of the patients presenting with chronic conditions. The addition of another outcome measure would have provided pertinent information to help determine patient progress. I was equally effective in resolving patient conditions when I choose the Mulligan Concept alone or combined with another treatment intervention (i.e. 25%). My success related to choosing Mulligan Concept alone or in combination indicates an element of chance was involved and not clinical reasoning.

My data collection, in general, for the fall 2014 semester was inconsistent. I collected outcome measures sporadically, instead of following a standard time frame (e.g., one week, two weeks). Collecting similar outcomes at similar times would have allowed me to gauge treatment effectiveness, particularly for comparable conditions. I found without a comprehensive evaluation and proper screening method, my intervention choice was likely to produce only short-term effects for chronic conditions. The inclusion of specific anatomical outcome measures would have provided a boarder picture of my patient, their condition, and treatment effectiveness. With a more discriminating eye, I could determine the most appropriate treatment based on patient response to interventions and improve my patient outcomes. Having identified these issues, however, I was prepared to improve my patient care by implementing solutions to these problems during the next semester.

Table 3.6.
Global Patient Outcomes - Fall 2014

Global Outcomes – Fall 2014						
Patient Number	**Pain Location**	**Injury Classification**	**Injury Type**	**Therapeutic Intervention(s)**	**Number of Treatments**	**Outcome**
16210	Shoulder	Positional Flt	Acute	MWM/PRRT	7	Positive
16211	Shoulder	Positional Flt	Acute	MWM/PRRT	4	Positive
16212	Shoulder	Positional Flt	Acute	MWM/PRRT	5	Neutral
16213	Shoulder	SMCD	Chronic	MWM/RNT/TMR	2	Positive
16214	Shoulder	SMCD	Chronic	MWM/RNT/TMR	3	Positive
16215	Shoulder	SMCD	Chronic	MWM/RNT/TMR	2	Neutral
16216	Lower leg	MTSS	Chronic	MWM/RNT	3	Negative

Table 3.6.
Global Patient Outcomes - Fall 2014 (Continued)

Global Outcomes – Fall 2014 (Continued)						
Patient Number	**Pain Location**	**Injury Classification**	**Injury Type**	**Therapeutic Intervention(s)**	**Number of Treatments**	**Outcome**
16217	Lower leg	MTSS	Chronic	IASTM	2	Negative
16218	Wrist	Positional Flt	Chronic	MWM/PRT	1	Neutral
16219	Ankle	Positional Flt	Acute	MWM	7	Positive
16220	Ankle	Positional Flt	Acute	MWM	5	Positive
16221	Ankle	Positional Flt	Acute	MWM	5	Positive
16222	Ankle	Positional Flt	Acute	MWM	4	Positive
16223	Low back	SMCD	Chronic	IASTM, Muscle Energy, Exercise	1	Negative
16224	Low back	SMCD	Chronic	Muscle Energy	2	Negative
16225	Low back	SI Dysfunction	Acute	Muscle Energy	2	Neutral

Spring 2015. With an improved evaluation system, clinical reasoning, improved understanding of various paradigms, and more effective use of outcomes measures, my greatest clinical progress was noted in the spring of 2015. Prior to spring of 2015, if I successfully treated a patient, I would apply the same treatment technique for a similar condition without determining if it was truly warranted in the new case. I discontinued this practice in spring of 2015 and improvement was noted in most areas of my patient care.

I collected patient outcome (Table 3.7) for a total of 10 patients in spring of 2015. Again, an MCID was produced for the NRS and DPA Scale for all patients (n=10). I was able to reduce patient pain 50% from initial pre- to post-intervention, an increase of 25% from the previous semester. I believe this reduction was a product of my improved screening and injury classification process.

During this semester, I tracked pain scores following subsequent treatments. Comparing the two pain measures was helpful, especially when reviewing patient progress and treatment selection. Similar to the pain measure, DPA scores were collected at regular intervals, not upon discharge only. The collection of the additional DPA score was essential in tracking patient improvement. A change occurred in the number of treatments required for patient presenting with chronic conditions. Patients presenting with chronic conditions required more treatments, mean of six than acute, mean of four, with 10% of acute conditions going unresolved and the mean number of patient treatments increased from 3.4 to 4.7. Although the visits increased, unlike previous semesters, patients did not return with the same complaint. I successfully treated 90% of my patients regardless of the various interventions chosen to treat patients' conditions, indicating my injury classification and treatment selection had improved. I base the improvements seen in my outcome measures on

the utilization of my evaluation algorithm along with increased knowledge and confidence in my skills.

Table 3.7.
Global Patient Outcomes - Spring 2015

Global Outcomes – Spring 2015						
Patient Number	Pain Location	Injury Classification	Injury Type	Therapeutic Intervention(s)	Number of Treatments	Outcome
16300	Ankle	Positional Flt	Acute	MWM	2	Positive
16301	Ankle	Positional Flt	Acute	MWM	3	Positive
16302	Lower Leg	MTSS	Acute	ND-Sliders	3	Positive
16303	Ankle	Positional Flt	Acute	MWM	4	Positive
16304	Great Toe	Tendinalgia	Acute	IASTM	8	Positive
16305	Hamstring	SMCD	Chronic	PRT, PRRT, Exercise	8	Positive
16306	Quadriceps	Strain	Acute	PRT, PRRT, IASTM	4	Positive
16307	Low Back & Hamstring	SMCD	Chronic	Breathing/ND Exercise	6	Positive
16308	Calf	Strain	Acute	PRT, IASTM, TMR	5	Negative
16309	Shoulder	SMCD/Positional Flt	Chronic	MWM, RNT	4	Positive

Analysis of Tendon Outcomes

Tendinopathy. Tendon disorders are a common condition seen in sports and the general population (Fredberg & Stengaard-Pedersen, 2008). Exact etiology of tendon disorders is unknown and tendon disorders are often misclassified (Scott, Docking, Vicenzino, Alfredson, Zwerver, Lundgreen, Finlay, Pollock, Cook, Fearon, Purdam, Hoens, Rees, Goetz, & Danielson, 2013: Kaux, Forthomme, Goff, Crieland & Croisier, 2011). Numerous strategies have been employed in the treatment of tendon disorders with limited success in symptom resolution (Cook, Khan, Harecourt, Grant & Young, 1997; Scott & Ashe, 2006; Grigg, Wearing, & Seathers, 2008; Rees, Stride, & Scott, 2012). Presently, the utilization of manual therapy techniques to resolve tendon disorders is under investigation.

During the past two years, I had incorporated manual therapy techniques into my clinical practice though not as a treatment for tendon disorders. Given that I followed standard treatment protocols, the success I had treating tendon disorders mirrored those in the current literature: short-term pain relief followed by re-occurrence of pain and disability. Prior to research conducted in DAT, I would not have considered using manual therapy to treat tendon disorders. Examining current literature, I discovered how mechanical loading and chronicity impacts musculoskeletal conditions, specifically tendon disorders. Changes in both the peripheral and central nervous system occur, and motor control is altered (Ljung, Alfredson, & Forsgren, 2004; Khan, Cook, Malfulli, & Kannus, 2000; Amro, Diener, Bdair, Hameda, Shalabi, & Ilyyan, 2010). Acknowledging various manual treatment interventions affect multiple physiological systems in the body, among those are the neural and motor systems (Vicenzino, Paungmali, Buratowski, & Wright, (2001). It is understandable how manual therapy interventions could be effective in the treatment of tendon disorders.

Achilles. I treated a total of three patients presenting with Achilles pain. For each patient, a complete medical history, and a full evaluation were performed prior to treatment classification. The evaluation included the documentation of active, passive, and resisted range of motion for the painful area, special anatomical tests, and a mobilization with movement (MWM), and tape application for the Achilles. Treatment classification was based on an algorithm assessing patient response to effect of joint repositioning (Mulligan, 1993), number and sensitivity of tender points (D'Ambrogio & Roth, 1997), or the involvement of the nervous system through neurodynamic tests (Shacklock, 2005). If joint repositioning (i.e. MWM) created a pain-free, immediate, long-lasting (PILL) effect, the patient received a Mulligan MWM for treatment. If the MWM did not produce a PILL effect, a quarter-screen associated with the body part assessed the patient for tender points. If tender points were present, the patient received positional release therapy (PRT). If no tender points were present, the patient was progressed to neurodynamic testing. Positive results from neurodynamic testing indicated the patient received neurodynamic (ND) treatment. A patient who did not meet the above criteria would be placed in an eccentric exercise (EE) program. A total of three treatments were allowed for each intervention. Following these three treatments, if the patient met discharge criteria, the patient was discharged. If the patient did not meet the discharge criteria, he or she was reassessed to determine appropriate treatment following the treatment classification algorithm, and three more treatments would ensue. Patients were discharged following another three treatments if discharge criteria were met. Patients not discharged after six treatment interventions were placed in an EE program.

All patients presenting with Achilles tendon pain responded favorably to the Mulligan Concept MWM; consequently, each was treated utilizing this intervention. No Mulligan MWM exists for the treatment of Achilles tendon pain, only a specific application of tape to the area. In the treatment of Achilles tendinopathy, utilizing the tape application, I was able to reduce patient reported pain on the NRS by an average of three points after the initial treatment, and 5.8 points upon discharge—which occurred in only three treatments for each patient. The reduction in pain was significant for the patient as defined by the MCID. A reduction of two points on the NRS has been identified as a clinically significant change (Farrar, Young, La Moreaux, Werth & Poole, 2001; Salaffi, Stancali, Ciapetti & Grassi, 2004). Comparing initial pain scores to discharge pain scores using a paired t-test, I found statistically significant changes in pain ($p=.003$), with a mean change of 5.8 ± 6.33. Comparing initial DPA Scale scores to discharge DPA Scale scores using a paired t-test, I found statistically significant changes on the DPA Scale ($p=.022$) with a mean change of 11.66 ± 3.06. The patients' passive range of motion (PROM) and active range of motion (AROM) improved, following initial treatment, falling within normal limits (Kapandji, 1987: Greene & Heckman, 1994) while deficits in resisted range of motion (RROM) were resolved (Table 3.8 and Table 3.9). The improvements in range of motion (ROM) were maintained in subsequent treatments. Additionally, all patients were discharged after three treatments. A review of the literature regarding the treatment of Achilles tendon disorders indicates these results exceeded what has been normally reported in the literature both in number of treatments and length of time until discharge (Cook, et al., 1997). Generally, treatments for tendon disorders are ineffective and not long-lasting (Scott & Ashe, 2006; Grigg et al., 2008; Rees et al., 2012). The most effective treatment requires engaging in a

rehabilitative EE program two times a day, seven days a week for 12 weeks or until resolution of symptoms (Fahlstrom, Jonsson, Lorentzon, & Alfredson, 2003; Ohberg & Alfredson, 2004; Jonsson, Alfredson, Sunding, Fahlstrom & Cook, 2008).

Table 3.8.
Dorsiflexion ROM

Dorsiflexion ROM																		
Patient	**AROM**						**PROM**						**RROM**					
	1st Treatment		2nd Treatment		3rd Treatment		1st Treatment		2nd Treatment		3rd Treatment		1st Treatment		2nd Treatment		3rd Treatment	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
16330	4°	10°	10°	13°	12°	15°	5°	12°	10°	14°	13°	17°	4	5	5	5	5	5
16332	3°	10°	11°	14°	11°	15°	4°	11°	11°	15°	13°	16°	4	5	5	5	5	5
16333	4°	11°	12°	14°	13°	15°	6°	12°	12°	16°	12°	15°	5	5	5	5	5	5

Table 3.9.
Plantarflexion ROM

Plantarflexion ROM																		
Patient	**AROM**						**PROM**						**RROM**					
	1st Treatment		2nd Treatment		3rd Treatment		1st Treatment		2nd Treatment		3rd Treatment		1st Treatment		2nd Treatment		3rd Treatment	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
16330	43°	51°	52°	60°	58°	64°	45°	55°	56°	64°	60°	66°	4	5	5	5	5	5
16332	42°	50°	53°	61°	59°	64°	45°	54°	54°	63°	62°	66°	4	5	5	5	5	5
16333	44°	52°	52°	61°	60°	63°	46°	56°	55°	64°	62°	65°	5	5	5	5	5	5

The average length of time patients had experienced pain and disability was six weeks, and only one patient had experienced pain for less than three weeks, suggesting the patients' condition was sub-acute or chronic. Similarities were found in activity levels. All of the patients continued to participate in activities without modification while denying a specific event precipitating their painful condition. No obvious signs of inflammation were present though all patients exhibited decreased range of motion passively and actively. Two of the Achilles patients presented with greater deficits in RROM; both of the patients engaged in running as their fitness activity and logged considerable miles. The other Achilles patient participated in martial arts training, averaging one to two classes a week and did not exhibit RROM deficits. Though similarities and differences existed for all patients, each responded favorably to the Mulligan Concept with resolution of their symptoms.

Lateral Elbow. A total of three patients presenting with lateral elbow pain were treated. A complete medical history and a full evaluation were performed prior to treatment classification for each patient. The evaluation included the documentation of active, passive, and resisted range of motion for the painful area, special anatomical tests, and a mobilization with movement (MWM). Treatment classification was based on an algorithm assessing patient response to effect of joint repositioning (Mulligan, 1993), number and sensitivity of tender points (D'Ambrogio & Roth, 1997), or the involvement of the nervous system through neurodynamic tests (Shacklock, 2005). If joint repositioning (i.e., MWM) created a pain-free, immediate, long-lasting (PILL) effect, the patient received a Mulligan MWM for treatment. If the MWM did not produce a PILL effect, a quarter-screen associated with the body part assessed the patient for tender points. If tender points were present, the patient received positional release therapy (PRT). If no tender points were present, the patient was

progressed to neurodynamic testing. Positive results from neurodynamic testing indicated the patient received neurodynamic (ND) treatment. A patient who did not meet the above criteria would be placed in an eccentric exercise (EE) program. A total of three treatments were allowed for each intervention. Following these three treatments, if the patient met discharge criteria, the patient was discharged. If the patient did not meet the discharge criteria, he/she was reassessed to determine appropriate treatment, following the treatment classification algorithm and three more treatments would ensue. Patients were discharged following another three treatments if discharge criteria were met. Patients not discharged after six treatment interventions were placed in an EE program.

A favorable response to MWM for patient classification was recorded for all three of my patients presenting with lateral elbow pain. Utilizing the Mulligan Concept for tendon pain at the lateral elbow, however, produced truly successful results for only one patient. The successful case resulted in my ability to reduce his pain by four points following initial treatment and six points upon discharge, in only three treatments. Improvements in pain, PROM, AROM, and RROM were noted after the initial treatment but were not maintained until after the second treatment (Table 3.10). The patient was discharged after three treatments, again exceeding the standard time frame for resolution of this condition (Fahlstrom et al., 2003; Ohberg & Alfredson, 2004: Jonsson et al., 2008).

Table 3.10.
Lateral Elbow Tendinopathy – Spring 2015

Lateral Elbow Tendinopathy – Spring 2015						
Patient 16331	Initial Treatment		Second Treatment		Third Treatment	
	Prior	Post	Prior	Post	Prior	Post
NRS	6	2	4	0	0	0
AROM-ext	0	-3	0	-3	-3	-5
PROM-ext	-3	-5	-3	-6	-5	-7
AROM-flex	140	143	140	144	145	149
PROM-flex	142	146	141	147	145	152
RROM	4	5	4	5	5	5

Two cases involving elbow pain were not resolved with MWM. In the first case, the patient reported post-treatment resolution of pain on the NRS after initial treatment though gains were not maintained. The patient received three treatments, declined reclassification and discontinued the study. The second case involved a patient who reported altered pain location after initial treatment; the patient no longer reported pain on the tendon. As a result, the patient no longer qualified for the tendon study (i.e., exclusionary criterion: altered pain location) and was not a fit for treatment with this Mulligan Concept technique. In the unresolved patient case, the patient had experienced lateral elbow pain over two years, and engaged in numerous manual tasks. The combination of these causative factors may have played a role in the poor resolution of this condition (Shiri, Viikari-Juntura, Varonen, & Heliovaara, 2006). The implementation of home MWM exercises, in addition to clinic care, may have helped resolve this condition as the patient responded favorably to the MWM each time in the clinic.

The average length of time patients had experienced pain and disability was 48 weeks, with no patient experiencing pain less than 12 weeks, indicating all patients were experiencing a chronic condition. Unlike Achilles patients, the patients experiencing elbow pain had all modified their activities (i.e., resistance training, gardening, and carpentry) due to pain. Chronicity may have had an influence on activity modification. Analogous to patients experiencing Achilles pain, the patients experiencing elbow pain responded favorably to the Mulligan MWM, although complete resolution of symptoms did not occur for all patients.

Discussion. Few research studies exist exploring the effects of indirect treatments or treatment-based classification for tendinopathies, particularly for the methods we chose on patellar, Achilles, and lateral elbow tendinopathy. As a group, we felt the indirect treatments chosen were the most appropriate and the algorithm can help determine the most applicable treatment. We also recognize there are other beneficial interventions techniques not included into the algorithm. Further research is warranted to establish the most appropriate interventions and algorithm for treatment of tendinopathy.

Table 3.11.
Achilles Tendinopathy Patient Data

Achilles Tendinopathy Patient Data													
Patient ID	Tendon	Treatment	Pre-Ant	Post-Ant	Dif	Pre-NRS	Post-NRS	Change Dif	Pre-DPA	Post-DPA	Change Dif	Tr Xs	Total Days
16330	Achilles	Mulligan Concept	34	66	-32	6	0	6	29	20	9	3	3
16332	Achilles	Mulligan Concept	23	129	-106	6	0	6	31	16	15	3	5
16333	Achilles	Mulligan Concept	53	100	-47	7	0	7	28	17	11	3	6

Table 3.12.
Lateral Elbow Tendinopathy Patient Data

Lateral Elbow Tendinopathy Patient Data													
Patient ID	Tendon	Treatment	Pre-Ant	Post-Ant	Dif	Pre-NRS	Post-NRS	Change Dif	Pre-DPA	Post-DPA	Change Dif	Tr Xs	Total Days
16331	Lateral Elbow Extensors	Mulligan Concept	37	21	16	6	0	6	30	21	9	3	5

My Struggles in Patient Care

What I believed a straightforward process—collecting, analyzing, and understanding patient outcome measures—proved more than a difficult endeavor. I was in my third semester before the collection of my outcome measures met DAT standards. Initially, the clinical implementation of my outcome measures was haphazard; consequently, it was difficult to derive meaning from those early attempts at collection or to attest to treatment effects. The incorporation of novel treatment interventions was inconsistent, and frequently, my treatment applications were imprecise. In my first three semesters, I often failed to follow-up with my patients; therefore, modest data exists to substantiate my treatment selections. My greatest challenge involved my clinical reasoning. If I was unable to resolve a patient's complaint quickly, I abandoned the treatment and shifted between treatment paradigms without thought. I struggled with uncertainty as often as when my intervention strategy worked as when it did not. I did not always understand why my patients' pain was resolved, and the immediate changes experienced by patients created confusion within me. Determining the next best step was problematic, and I repeatedly questioned my clinical reasoning and decisions, attempting to understand the process.

I also struggled with the lack of interest shown by colleagues, particularly when they witnessed change in my patient care approach. Some would ask questions, though I felt more out of curiosity than interest in learning new techniques. I have additional responsibilities due to my position, and my time in the clinic is limited; therefore, conflicts arose particularly during patient follow-ups. My treatment protocol was rarely followed, creating uncertainty between patients and staff, which often resulted in missed appointments, reducing quality patient care. Additionally, personnel changes in the

department led to staffing shortages, which created discord. Overall, there was a lack in communication, and my morale was negatively impacted. Though not everyone shared my interest, I continued to pursue change and improvement in my practice.

Residency Impact

The impact of my residency has been felt both clinically and academically. I have been able to introduce various interventions to my staff and the athletic training students. The students were receptive to learning new treatments and worked diligently to improve their technique. Furthermore, I requested the students to research the rationale and utilization of outcome measures in clinical practice. As students began understanding the importance of utilizing outcome measures in clinical practice, numerous discussions took place—most related to clinical reasoning and treatment effectiveness. Also, a few students expressed interest in engaging in practice-based evidence research.

Academically, I began to change course content. I included more evidence-based material in certain courses while requiring the students to research efficacy of traditional athletic training practices in others. The topics of clinical reasoning, action research, and reflective analysis of clinical practice were all discussed in select courses. In addition, the departmental chair has expressed interest in including additional course offerings (e.g., manual therapy) for athletic training students. The most significant contribution of my residency involved altering the apprenticeship athletic training model currently in place. Several discussions have taken place with the departmental chair and other administrators. Athletic training program assessment and feasibility studies have occurred, and the university president is supportive of changing the program model. The goal is to establish an athletic training program centered on DAT principles.

Conclusion

As I have grown and progressed in the DAT, my patient care has changed and improved. Initially, I was uncertain of my skills and knowledge and struggled to implement the necessary changes in my clinical practice. With each passing semester, as I collected, analyzed, and reflected on patient outcome measures, my understanding increased. I was able to make the necessary adjustments to continue to improve my patient care and strengthen my knowledge. Moving forward, I will continue to collect and analyze outcome measures to ensure my continued growth and improvement as an advanced AT clinician.

CHAPTER 4: Review of Literature

Tendon Pain

Tendon pain has been identified in both active and inactive populations (Alfredson & Cook, 2007; Alfredson & Lorentzon, 2002; Kvist, 1994; Scott & Ashe, 2006). Despite an improved understanding of the pathophysiology of tendon injury, appropriate interventions to address pain reduction and return to function remain elusive (Grigg, Wearing, & Smeathers, 2009; Rees, Stride, & Scott, 2013; Scott & Ashe, 2006). Sites commonly involved with tendon pain include the lateral elbow, patella, and Achilles tendon (Alfredson & Lorentzon, 2002). A critical review is required to address the etiology, pathoanatomics, inflammatory responses, classifications, and interventions in order to address the appropriate treatment for tendon pain.

Numerous terms describing tendon pain are ambiguous and have often been misunderstood throughout the healthcare community. *Tendinitis*, a term that is commonly used to classify tendon pain, is currently defined as an inflamed tendon associated with inflammatory mediators (Khan, Cook, Kannus, Maffulli, & Bonar, 2002; Mayor, 2012; Maffulli, Wong, & Almekinders, 2003). *Tendinosis* is often associated with a degenerative process without clinical symptoms (Ferretti, Conteduca, Camerucci, & Morelli, 2002; Kaux, Forthomme, Goff, Crielaard, & Croisier, 2011; Khan, et al., 2002; Mayor, 2012; Sharma & Maffulli, 2008). *Tendinopathy* is characterized by pain in the tendon and impaired performance (Kaux et al., 2011; Khan, et al., 2002; Mayor, 2012; Sharma & Maffulli, 2008). *Tendinalgia* is defined as tendon pain with causative factors outside the pathology of the tendon (Baker, Riper, Nasypany, & Seegmiller, 2014). The confusion in definitions and classifications could potentially hinder determining appropriate treatment interventions.

Historically, tendon pain was characterized as an acute inflammatory process and was treated with rest, ice, and nonsteroidal anti-inflammatory drugs (NSAIDS) (Abbott, Patla, & Jensen, 2001; Alfredson, 2005; Andres & Murrell, 2008; Jelinsky, Lake, Archambault, & Soslowsky, 2008; Teys, Bisset, Collins, Coombes, & Vicenzino, 2013; Vicenzino, Paungmali, Buratowski, & Wright, 2001). The current gold standard for treating tendon pain and dysfunction is eccentric exercises, which is time consuming and painful (Alfredson, Pietilä, Jonsson, & Lorentzon, 1998; Cannell, Taunton, Clement, Smith, & Khan, 2001; Dimitrios, Pantelis, & Kalliopi, 2011; Fahlstrom, Jonsson, Lorentzon, & Alfredson, 2003; Jonsson, Alfredson, Sunding, Fahlström, & Cook, 2008; Mafi, Lorentzon, & Alfredson, 2001; Ohberg & Alfredson, 2004; Purdam, Jonsson, Alfredson, Lorentzon, Cook, & Khan, 2004; Svernlöv, Hultgren, & Adolfsson, 2012; Young, Cook, Purdam, Kiss, & Alfredson, 2005). Manual therapy interventions to treat tendinopathy have included the Mulligan Concept (Abbott et al., 2001; Teys et al., 2013; Vicenzino et al., 2001) and positional release therapy (PRT) (Baker et al., 2013). According to Rees, Wilson, and Woodman (2006), tendon pain can be attributed to neural inflammation; therefore, neurodynamics should be investigated as a treatment for tendon pain. To date, no optimal management technique has been established using MWM, PRT, or neurodynamics to treat tendon pain.

Epidemiology of tendinopathy. Overuse injuries frequently occur among the working and athletic populations (Fredberg & Stengaard-Pedersen, 2008). Injuries sustained by these populations are typically related to chronic tendon disorders and account for a high number of referrals to rheumatologists and orthopedic surgeons (Bamji, Dieppe, Haslock, & Shipley, 1990). Incidences of overuse injuries appear to be on the rise due to the following

factors: society has elevated the importance of sport and athletics, movements toward gender equality have sanctioned greater numbers of women in sport, leisure time has allowed for increased youth involvement in sport, and longevity has spurred continued engagement in work and activity beyond the traditional retirement age (Oeppen & Vaupel, 2002; Poser, 2011).

Numbers regarding the prevalence of tendon injuries in work and sport are largely unknown due to differences in terminology, etiology, and reporting. Tendon problems are often classified based on supposed cause (e.g., overuse injury, cumulative trauma disorder, or repetitive strain injury) or improper classification (Rees et al., 2006). Epidemiological surveys indicate more than half of all injuries fit one of these descriptors (Almekinders, 1998).

Although there are no accurate figures specifically relating to tendon disorders, several studies have been published on the frequency of tendon problems in the general population and in athletics (Forde, Punnett, & Wegman, 2005; James, Bates, & Osternig, 1978; Kujala, Sarna, & Kaprio, 2005). Forde et al. (2005) found the prevalence of musculoskeletal disorders fell between 2% and 65% for workers involved in physical labor. The prevalence of musculoskeletal disorders increases with age and employment duration; people who have worked for 25 to 35 years are more likely to develop tendinopathy (Forde et al., 2005). Riley (2008) reported, in the working population, 30% of consultations with a general practitioner were related to musculoskeletal complaints, while Bamji et al. (1990) stated soft tissue ailments comprised up to 43% of new rheumatology patient referrals. Kujala et al. (2005) also reported the lifetime cumulative incidence of Achilles tendinopathy to be 5.9% among sedentary individuals.

In sport, 50% of elite endurance athletes experience Achilles tendinopathy (Kujala et al., 2005) while chronic tendon problems represent nearly 30% of all running-related injuries (James et al., 1978), 9 to 40% of injuries reported among tennis players (Gruchow & Pelletier, 1979; Maffulli et al., 2003; Scott & Ashe, 2006), and 71% of injuries among elite swimmers (Scott & Ashe, 2006). A review of studies on the frequency of sport and exercise-related tendinopathies at the second International Scientific Tendinopathy Symposium (ISTS) (2012), however, found that most of the studies that provided the aforementioned data were focused on selected populations and that tendon injuries were often ill-defined (Scott et al., 2013). Kaux et al. (2011) found the description and definition of dissimilar musculoskeletal disorders differs between medical specialists and the general population. In addition, many people continue to work or participate in their chosen activity despite their chronic overuse injury or tendon pain. The continuation of activity indicates many epidemiological studies do not include tendinopathies in the incidence rate as new injuries that cause lost time from work or sports (Scott et al., 2013). Given these limitations, it is difficult to determine the true onset and etiology of tendon pain.

Etiology of tendinopathy. The exact etiology of tendinopathy is not fully understood, but it appears to be a multifactorial process. A multitude of intrinsic and extrinsic factors, either alone or in combination, have been suggested as contributors to the development of tendon injuries (Fredberg & Stengaard-Pedersen, 2008; Jarvinen, Kannus, Maffulli, & Khan, 2005; Nirschl & Ashman, 2003; Scott & Ashe, 2006). While the range of probable risk factors is large and various, the mechanical behavior of tendons and their response to loading are currently thought to be the most important factors in tendon

pathology and pain (Kaux et al., 2011). Other contributing factors may be vascular or neural changes (Alfredson, 2005; Rees et al., 2006).

The mechanical behavior of the tendon depends on its location and function (Rees et al., 2006). Tendons from different sites have differences in structure and composition and are subjected to varying levels of mechanical loads. For example, the Achilles tendon can withstand greater tensile forces than that of the tibialis anterior, mostly due to the larger surface area and the architecture of the Achilles tendon (Maganaris, 2002; Wang, 2005).

The mechanical stress on the tendon also depends on the level of muscle contraction and the tendon's size (Wang, Iosifidis, & Fu, 2006). The greater the cross-sectional area of the muscle, the greater the force it produces and the larger the stress on the tendon. Mechanical load appears to explain how tendon damage can become progressively worse over time; however, it does not address why certain areas of some tendons are more prone to degenerative changes, nor does it explain the pain associated with tendinopathy.

Alterations in tendon vascularity and neural adaptations are currently under investigation as contributing factors for tendon pain. The disruption of the vascular system is thought to cause tendon degeneration with certain tendons, such as the Achilles, being more susceptible to a vascular compromise (Fenwick, Hazleman, & Riley, 2002). Alfredson (2005) hypothesized that neovessels and accompanying nerves are a potential origin of the pain in tendinopathy.

Determination of the source and cause of tendon pain continues to undergo examination. Various theories have been proposed to explain the pain mechanisms. Likely, a combination of several theories more accurately addresses tendinopathy etiology than any

one theory on its own (Rees et al., 2006). Understanding tendon anatomy may provide answers to tendinopathy etiology and subsequent pain mechanisms.

Anatomy of the Tendon

Gross anatomy. Tendons are transitional anatomical structures that transmit forces between muscle and bone, turning those forces into movement. The myotendinous junction (MTJ) is the site where the muscle and tendon join, while the osteotendinous junction (OTJ) is the region where the tendon connects to the bone (Jozsa & Kannus, 1997). The MTJ transmits force that is generated within the intracellular contractile proteins of a muscle fiber into the extracellular connective tissue protein of the tendon, and the OTJ transmits force from the viscoelastic tendon to the rigid bone (Jozsa & Kannus, 1997). Tendon appearance varies according to the health of the tendon. Healthy tendons are white in color and can vary in shape and size from one person to another and depending on the location in the body and what type of force is needed to produce specific movements (Khan, Cook, Bonar, Harcourt, & Astrom, 1999). Shorter, broader tendons produce more power, whereas longer tendons are used in fine motor movement (Jozsa & Kannus, 1997).

Tendons are composed of a dry mass that consists of 65 to 75% collagen fibers and 2% elastin embedded into proteoglycans and a water matrix (Jozsa & Kannus, 1997; Scott, Alfredson, & Forsgren, 2008). Collagen makes up 25-30% of the human body's protein content and is formed by a triple helix of the amino acids proline, glycine, and hydroxproline (Lodish et al., 2000). The collagen fiber is the basic unit of the tendon and is made up of collagen fibrils. The fibrils, produced by tenocytes and tenoblasts, band together to form collagen fibers. The extracellular matrix (ECM) is composed of the collagen, fibroblasts, specialized fibroblasts (tenocytes), proteoglycans (PG), glycoproteins, and

water. The ECM functions as an interactive matrix that provides structural and mechanical support. With its adhesive properties, the ECM modulates cell growth and communicates with a cell's biomechanical and mechanical changes within the matrix (Birch, Thorpe, & Rumian, 2013; Ross & Pawlina, 2006).

The architecture of collagen fibers contributes to a tendon's gross primary functions: to transmit tensile loads, to stabilize joints, to protect muscles, and to absorb shock. There are 16 types of collagen fibers. Type 1 collagen (60% dry weight [dw]) constitutes the majority of the healthy tendon and has an enormous amount of tensile strength. Small amounts of types III, IV, V, VI, XII, and XIV fibers are also present in the tendon along with the PG (0.5% dw) decorin, versican, and lumican and the glycoproteins (5% dw) tenascin, cartilage oligomeric matrix protein (COMP), and elastin. The insertion of the tendon includes all of the properties of the midsubstance coupled with collagen (types II, IV), aggrecan, and biglycan (Lodish et al., 2000; Riley, 2008). The role of collagen is to provide strength, while the proteoglycans provide structural support and tissue hydration for the collagen. The synthesis of a tendon occurs as fibroblasts respond to mechanical loading (Rees, Wolman, & Wilson, 2009; Schulze-Tanzil et al., 2011; Scott & Ashe, 2006). The tissues surrounding and supporting a tendon help decrease friction associated with tendon movement. Tendons, including the Achilles, patellar, and the common extensor tendon, are not protected by a synovial sheath, but are enclosed by a paratenon.

The Achilles tendon is the strongest and broadest tendon in the body. The gastrocnemius and soleus muscles join at the MTJ and continue to form the Achilles tendon that inserts into the OTJ at the posterior aspect of the calcaneus. The tendon's broad, flat origin and narrow, round insertion are designed to withstand a great amount of force,

including sprinting, jumping, and pivoting (Bains & Porter, 2006; Jozsa & Kannus, 1997). The Achilles tendon length ranges from 11 to 26 centimeters (cm), most of which is the gastrocnemius portion of the tendon (Bains & Porter, 2006; Jozsa & Kannus, 1997). Before inserting onto the mid-posterior aspect of the calcaneus, the tendon can rotate 90 degrees so that the posterior fibers become lateral, lateral fibers become anterior, anterior fibers become medial, and medial fibers become posterior (Bains & Porter, 2006; Jozsa & Kannus, 1997). Tendon rotation allows stress produced by movement to be dissipated (Jozsa & Kannus, 1997). The pain site most commonly associated with Achilles tendinopathy is the distal tendon, typically 2-5 cm proximal to the insertion into the calcaneus (Baines & Porter, 2006).

The patellar tendon is a continuation of the quadriceps tendon; it originates on the apex and the bordering margins of the patella. The rough depression on its posterior surface inserts on the tibial tuberosity (Bains & Porter, 2006; Jozsa & Kannus, 1997; Khan, Maffulli, Coleman, Cook, & Taunton, 1998; Tan & Chan, 2008). The patellar tendon is a strong, flat band, about 8 cm in length, used in the extension of the knee (Baines & Porter, 2006). The posterior aspect of the patellar tendon is separated from the knee's joint capsule by the infrapatellar fat pad and from the tibia by a bursa (Baines & Porter, 2006). Patellar tendinopathy, also known as jumper's knee, is typically associated with sports that require explosive quadriceps activation such as basketball and volleyball (Baines & Porter, 2006). The pain site that is associated with patellar tendinopathy is the lower pole of the patella; however, mid-tendon pain is not uncommon (Baines & Porter, 2006).

The structure associated with lateral elbow pain is the common extensor tendon (CET) (Donaldson, 2013). The CET is comprised of the extensor carpi radialis brevis

tendon, with some involvement from the extensor digitorum communis (Donaldson, 2013; Scott & Ashe, 2006). The CET originates on the lateral humeral epicondyle and acts on the dorsal aspect of the forearm and wrist producing an extension movement (Tosti, Jennings, & Sewards, 2013). The articular origin of the CET is where pain and degeneration has been often identified (Donaldson, 2013). Pain onset is usually gradual and can radiate down the forearm and lead to a decrease in grip strength (Donaldson, 2013).

Vascular system and neurochemical responses of tendons. Until the nineteenth century, it was believed that tendons were avascular. Recently, it was determined that tendons are more metabolically active than previously thought as demonstrated through circulatory responses and collagen turnover changes related to activity (Alfredson, 2005). Compared with many other tissues in the adult body, however, the tendon does have a low blood supply. The Achilles and patellar tendon receive their blood flow from the paratenon, as well as the OTJ and MTJ (Tan & Chan, 2008). Though tendon blood supply comes from various sources, portions of the tendon may not receive adequate circulation (Jozsa & Kannus, 1997). Arner, Lindholm, and Orell (1959) demonstrated that between 2 and 6 cm proximal to the Achilles tendon insertion is an area of avascularity. The avascularity may be the reason more degeneration and ruptures occur in the middle aspect of the Achilles tendon. The distal attachment of the patellar tendon to the tibial tuberosity also includes an avascular zone. One difference between the Achilles tendon and patellar tendon is that the area where most degeneration occurs in the patellar tendon is not at the avascular site. Instead, degeneration typically occurs at the patellar tendon's proximal attachment, which is richly vascularized from the inferior half of the patella (Khan et al., 1998).

Due to the small amount of soft tissue surrounding the lateral epicondyle, the CET blood supply is considered poor (Jozsa & Kannus, 1997). The blood supply to the muscles connecting to the epicondyle is sufficient, but the tendon fibers attached to the periosteum of the epicondyle are considered avascular (Jozsa & Kannus, 1997). A lack of blood supply decreases the recovery rate from usage. An increase in age also decreases the amount of nutrients that are available to the lateral elbow region, thus decreasing the recovery rate further (Jozsa & Kannus, 1997).

The neural system also influences blood supply to the muscles. Nerves are exposed to stresses such as tension and compression during movement. An increase in tension forces will reduce blood flow at 8% elongation and will cease blood flow at 15% elongation. A failure threshold for compression stresses occurs at 30-50 mmHg. These compressive forces will impair blood flow (Shacklock, 2005,1995). Axoplasm, the cytoplasm for peripheral nerves, is 5% thicker than water. Decrease in axoplasm flow can result in unproductive neurons. The unproductive neurons may affect the axon, cell body, or target cells (Butler, 2000).

Ion channels are gated to open, allowing ions to flow through, or to close, preventing the passage of ions, based on several mechanisms. Some ion channels respond to electrical current (voltage gated), others respond to chemicals (ligand gated), and still others respond to stretch or pressure (mechanically gated). Ion channels are made in the dorsal root ganglion and are sent to unmyelinated sections of nerves via axoplasm to the axolemma. A majority of ion channels are found unevenly distributed in the cell body, axon, hillock, dendrites, terminals, and nodes of Ranvier. If a nerve loses myelination due to injury, more ion channels may be formed. The increase of ion channels creates abnormal impulse

generating sites (AIGS). Abnormal impulse generating sites are often associated with an atypical pain response. Fortunately, ion channels only live one to two days, providing an opportunity to alter nociceptive pain via the periphery (Butler, 2000).

Inflammation of the tendon. Whether tendon pain originates from cellular, biochemical, or neurochemical adaptations has not been well established. Classifying tendon inflammation based on histology has also been problematic (Scott & Ashe, 2006). When a tendon endures repetitive mechanical force, it may result in cumulative microtrauma. Macroscopically, injured tendon fibers are no longer white. The tendon has a yellow-brown appearance and disorganized structure, known as mucoid degeneration (Ackermann, Domeij-Arverud, Leclerc, Amoudrouz, & Nader, 2013; Khan et al., 1999). When viewed microscopically, collagen fibrils are disorganized, torn, and separated (Rio et al., 2013; Scott & Ashe, 2006). When a disruption of the tendon occurs, the acute inflammatory phase is initiated. Platelets and leukocytes migrate to the tendon and produce cytokines, tumor necrosis factor-x (TNF-x), and the interleukins (IL) IL-1ß, IL-6, IL-8, and IL-10. Tenocytes increase production of cyclo-oxygenase (COX)-2, prostaglandins (PGE1, PGE2), IL-6, and IL-1ß. Cytokines have both pro and anti-inflammatory actions. In the acute stage of tendon injury, blood coagulates as leukocytes, and platelets create an inflammatory response (Ackermann & Renström, 2012). The migration of inflammatory products is followed with cytokine production. As a result, TNF-x reduces type I collagen while increasing the production of IL-1ß, IL-6, IL-8, and IL-10. Interleukin-1ß promotes prostaglandin E2 (PGE2) production, IL-6 acts as an anti-inflammatory on TNF-ß and IL-1ß, and IL-10 reduces the synthesis of TNF-x and IL-2 (Ackermann & Renstrom, 2012).

Repetitive loading creates microruptures of the collagen fibers producing inflammatory mediators (e.g., PGE2, macrophages, mast cells, and B and T lymphocytes) that infiltrate the injured structure, setting off a cascade of pro-inflammatory cytokines (i.e., IL-18, IL-15, IL16) and transforming growth factor (TGF-ß) (Ackermann & Renstrom, 2012). If the tendon endures repeated tensile, compressive, or shear forces, a chronic injury may develop. Researchers have demonstrated that a load to the Achilles tendon during exercise revealed an increase in inflammatory mediators (Wang et al., 2003; Yang, Crawford, & Wang, 2004). Both PGE_2 and thromboxane B_2 increased during exercise and continued to increase after a recovery period of 60 minutes. Almekinders, Banes, and Ballenger (1993) reported repetitive mechanical loading of human fibroblasts increased the production of PGE_2. Tendon fibroblasts produced PGE_2 in response to repetitive mechanical loading; consequently, repeated exposure of tendons to PGE_2 could result in degenerative changes to tendons. Khan and Wang (2005) revealed that repetitive exposure of a tendon to PGE_2 caused disorganization of the collagen matrix as well as decreased diameter of collagen fibers. A study by September et al. (2009) investigated specific genetic markers associated with tendinopathy in two different populations. Researchers demonstrated that both the genetic markers COL5A1 and B*sf*UI RFLP were associated with Achilles tendinopathy in both populations. Tendon injury has been demonstrated to occur from repetitive microtrauma. While research relating to the role of genetics and its relationship to tendon pain is in its infancy, the role of specific genotypes associated with tendon pathology should not be ignored.

In the pathological tendon, researchers found an increase in messenger RNA (mRNA) and collagen fiber types I and III in the tendon matrix. Increases in glutamate and

its receptor N-methyl-D-aspartate (NMDARI) were also associated with the mRNA increase (Alfredson, Ljung, Thorsen, & Lorentzon, 2000; Riley, Cox, Harrall, Clements, & Hazleman, 2001). The importance of the neurotransmitter glutamate as a mediator of pain in the human central nervous system has been identified (Alfredson et al., 2000; Alfredson & Cook, 2007; Dickenson, Chapman, & Green, 1997). Alfredson et al. (2000) used microdialysis to compare extensor carpi radialis brevis (ECRB) tendinosis patients with a control group. The results indicated higher concentrations of the excitatory neurotransmitter glutamate and the cytokines IL-6, IL-8, and IL-10 in the tendinosis patients.

Interestingly, the researchers found no significant differences in mean concentration of PGE_2 between the control and the tendinosis group (Alfredson & Lorentzon, 2000); yet, a common opinion exists that there is involvement of a chemical inflammation with all tendon injuries (Alfredson, 2005; Kvist, 1994; Leadbetter, 1992; Shrier, Matheson, Grodon, Kohl, & Harold, 1996).

Researchers have also examined whether tendon inflammation is biochemical or neurogenic. Nerve fibers are located in the periosteum, synovium, fat pad, and peritendinous connective tissue (Fredberg & Stengaard-Pedersen, 2008). Healthy tendons are aneuronal, but repetitive loading increases sensory nerve fibers alongside the blood vessels, which increases exogenous pain. Prostanoids influence chemical inflammation and immune responses, and their administration reproduces the major signs of inflammation, including peripherally and centrally augmented pain sensitivity (Fredberg & Stengaard-Pedersen, 2008; Solomon, Fretzin, & Dewald, 1968). Peripherally, physiological pain occurs with the activation of nociceptors reacting to inflammation. Centrally, pathophysiological pain is related to functional changes in the nervous system (Fredberg & Stengaard-Pedersen, 2008;

Rio et al., 2013). Alfredson and Cook (2007) created an algorithm for managing Achilles tendinopathy and described "four cornerstones" of histology: 1) increase in cell numbers and cellular activation, 2) increase in ground substance, 3) collagen disorganization, and 4) neovascularization. While these components were not considered to be inflammatory conditions, the presence of the neuropeptide substance P (SP) and calcitonin gene-related peptide (CGRP) indicated an inflammatory component (Fredberg & Stengaard-Pedersen, 2008). The upregulation of SP impacts TGF-ß, which contributes to fibrosis, hypercellularity, hypervascularization, and tenocyte changes (Ackermann & Renström, 2012; Hoffmann, Hoeck, Deters, Werner-Martini, & Schmidt, 2010). Microscopic findings indicate vascular ingrowth, tenocyte death (necrosis and apoptosis), and proliferation (hypercellularity). The abnormal and degenerated extracellular matrix, accompanied with sprouting and ingrowth of nociceptive nerves has been implicated in the generation of neurogenic inflammation (pain, edema, and fibrosis) (Scott & Ashe, 2006). Whether neovascularization contributes to neoangiogenesis is difficult to determine; however, ultrasound (US) studies do suggest that neovessel formation could be responsible for the pain in chronic tendinopathy (Alfredson, Ohberg, & Forsgren, 2003; Rees et al., 2013).

Researchers have theorized that neovessals are accompanied by neural 'sprouting'. The neoinnervation may be responsible for the pain associated with tendinopathy (Alfredson et al., 2003; Rees et al., 2013). Alfredson, Ohberg and Forsgren (2003) studied 28 patients with chronic painful mid-portion Achilles tendinosis who were injected with a local anesthesia in the area with neovessels outside the tendon. The injection resulted in a pain-free tendon loaded motion. The researchers hypothesized that the neovessels and

accompanying nerves were responsible for the pain in the area with tendinosis (Alfredson, 2005).

The increased sensitization experienced with tendinopathy could be also related to an upregulation of the nervous system's response to a stimulus, thereby inducing pain into a structure that has already healed. Either allodynia or hyperalgesia might play a major role in the functional capacity of tendinopathy patients (Rio et al., 2013). Hyperalgesia occurs when a stimulus that usually evokes pain evokes more pain than normal, whereas allodynia occurs when a stimulus that usually does not evoke pain evokes pain (Rio et al., 2013). At the central level there are many reactions to injury. Normal response to injury involves A delta and C fibers excreting excitatory chemicals such as glutamate and amino acids. With the abundance of chemicals being excreted, the wide dynamic range (WDR) creates more ion channels. The inhibitory, which is located within the spinal cord, and the brain release inhibitory chemicals to balance the excitatory chemicals from the A delta and C fibers. Pain usually subsides and chemicals return to allostasis as healing occurs. However, if A delta and C fibers continue to release excitatory chemicals, one of three things will happen: 1) the inhibitory will die of amino acid toxicity, 2) WDR will begin sprouting dendrites, creating inappropriate synapses, or 3) non-specific neurons waiting for direction become easy to fire. At this point, the tissues have healed but the pain continues due to the brain's view of the central mechanisms (Butler, 2000, 2014).

Classifications of Tendinopathy

Tendon pathology terminology. Researchers have been unable to establish well-defined causative factors of tendon pain due to a lack of evidence from histological markers. Clinically, the diagnosis has been difficult to determine based solely on signs and symptoms

of patients with perceived tendinopathy. The medical community has used the terms tendonitis, tendinosis and tendinopathy interchangeably as diagnostic classifications for patients with tendon pain (Alfredson & Lorentzon, 2002; Rees et al., 2006). More recently, tendinalgia has been proposed as a classification term, but is not, yet, commonly used by clinicians to classify tendon pain throughout the body (Baker et al., 2014). Lateral epicondylalgia, a location-specific type of tendinalgia, is commonly used as the diagnostic term for tendon pain at the lateral epicondyle of the humerus (Donaldson, 2013).

Tendinitis implies inflammation of the tendon as the origin of tendon pain (Rees et al., 2006). The term *tendinitis* has been removed from vernacular, for the most part, due to researchers demonstrating a lack of inflammatory markers present on histopathological exams (Rees et al., 2006). *Tendinosis* is degeneration of the tendon, which is due to a failed healing process (Tan & Chan, 2008). Tendinosis does not correlate with clinical signs and symptoms (Alfredson & Lorentzon, 2002), but imaging may detect deformities associated with or without pain (Alfredson & Cook, 2007). *Tendinopathy* is described as a pathological state causing pain and stiffness (Rees et al., 2006) and is a term more commonly used for diagnoses in research (Rees et al., 2013; Scott et al., 2013). The concern with this term is that it implies that the patient's pain and cause of dysfunction is local pathology to the tendon, which may not be the case (Kaux et al., 2011; Khan et al., 1999). Tendinalgia denotes that the main clinical symptom is pain, and its multiple causes, many unrelated to local tendon inflammation or degeneration, could result in pain presentation at a tendon (Baker et al., 2014). Due to the vast possible causative factors of tendon pain, *tendinalgia* has been recommended as a more accurate term for diagnosis and classification of all tendon

pain (Baker et al., 2014). Information provided by a study performed by Astrom (1998) illustrates the complexity involved in tendinopathy terminology.

Astrom (1998) performed a retrospective study to analyze Achilles tendinopathy to determine a clinical classification for Achilles tendon ruptures. During 342 Achilles tendinopathy operations, 78 (23%) of the tendons had a partial rupture, 168 (49%) had tendinosis, and 96 (28%) did not appear to have a pathology (Astrom, 1998). The aforementioned study is an example of why clinicians and researchers have not established common terminology in the diagnosis and classification of tendinopathy.

Clinical diagnosis and classification of tendinopathy. According to Rio et al. (2013), the diagnosis of tendinopathy requires clinical symptoms. The clinical signs and symptoms reported by researchers that may appear in a subject with tendinopathy include tendon pain, dysfunction of the tendon (Khan et al., 1999; Rio et al., 2013), decrease in performance in association with tendon swelling, morning stiffness (Alfredson, 2005; Alfredson & Lorentzon, 2002; Khan et al., 1999), palpable crepitus (Alfredson, 2005; Alfredson & Lorentzon, 2002;Fredberg & Stengaard-Pedersen, 2008), and localized swelling (Khan et al., 1999). The diagnosis of tendinopathy is mainly based on patient complaints (Cook, Khan, & Purdam, 2001; Maffulli et al., 2003). Experienced examiners may have problems reproducing the results of a clinical examination based on simple tests (Maffulli et al., 2003). The patient will seldom complain of pain at rest and during low tendon loading activities (Rio et al., 2013). The most common complaint is point tenderness and pain during high tendon loading activities, such as jumping (Alfredson, 2005).

Rating scales have been introduced in the literature to assist clinicians in classifying tendon dysfunctions. Through his research, Blazina established three phases of

tendinopathy. In phase one, the patient would present with pain after activity only with no functional impairment. In phase two, the patient would have pain during and after activity and continue with no functional impairment. In phase three, the patient would present with functional impairment along with an increase in pain during and after activity for longer periods of time (Blazina, Kerlan, Jobe, Carter, & Carlson, 1973).

Nirschl and Ashman's phase rating scale (Kaux et al., 2011; Nirschl & Ashman, 2003) focused on pathological stages and phases of pain. There are four pathological stages: 1) temporary irritation, 2) permanent tendinosis with less than 50% tendon cross section, 3) permanent tendinosis with more than 50% tendon cross section, and, 4) partial or total rupture of tendon. The seven phases of pain are: 1) mild pain after exercise activity for less than 24 hours, 2) pain after exercise activity for more than 48 hours, and pain resolves with warm up, 3) pain with exercise activity, but the pain does not alter activity, 4) pain with exercise activity, and the pain does alter activity, 5) pain caused by heavy activities of daily living, 6) intermittent pain at rest that does not disturb sleep, and pain caused by light activities of daily living, and 7) constant rest pain and pain that disturbs sleep (Kaux et al., 2011; Nirschl & Ashman, 2003). Nirschl and Ashman (2003) proposed that pathological stages three and four generally require surgical intervention, and pain phases four and five with a pain rating of five or greater correlate with stages two and three pathology. Pain phase six and seven, with a persistent pain rating of five or greater, correlate with pathologic stages three and four.

Several researchers have suggested a timeline for tendinopathy where *acute* is equal to two weeks or less, *subacute* is two to six weeks, and *chronic* is more than six weeks (Tan & Chan, 2008). More recently, others have suggested a different timeline: zero to six weeks

would be characterized as acute, six to twelve weeks would be subacute, and three months or longer would be considered chronic (Kaux et al., 2011).

Tendon pathology as a continuum. Cook and Purdam (2008) proposed a theory of tendinopathy as a continuum that consists of three stages: reactive tendinopathy, tendon dysrepair, and degenerative tendinopathy. Patients may transition between the stages of the continuum, and not necessarily in a progressive manner. Based on the continuum, physiological responses in an acute phase of tendinopathy may return to normal if given the appropriate opportunity. The theory of reactive tendinopathy suggests a short-term adaptive response in the tendon, which results in thickening of the tendon. Patients that presented with reactive tendinopathy usually had a sudden increase in physical activity (Cook & Purdam, 2008).

Researchers and clinicians continue to use terms interchangeably for diagnosis and classification of patients with tendon pain, which could be misleading (Scott et al., 2012). Additionally, many clinicians continue to diagnose based on reported signs and symptoms from patients without the use of diagnostic testing (Cook et al., 2000; Maffulli et al., 2003), which may lead to misdiagnosis. The inappropriate diagnosis and classification could hinder effectiveness of chosen treatments due to causative factors not being addressed for specific patients or situations.

Conservative Treatment of Tendinopathy

The goal of most rehabilitation programs is to decrease pain and restore function. Determining appropriate treatment interventions for tendinopathy remains elusive due to its unknown etiology. Classic, conservative treatments for tendinopathy include a combination of rest, ice, non-steroidal anti-inflammatory drugs (NSAIDS), passive physical therapy,

orthotics, corrections of malalignment, stretching, and corticosteroid injections (Alfredson, 2005; Glaser, Poddar, Tweed, & Webb, 2008; Kaux et al., 2011). Multiple approaches to treat tendinopathy have been attempted with varying success (Cook, Khan, Harcourt, Grant, & Young, 1997). In recent years, eccentric exercises have produced good clinical results (Alfredson et al., 1998; Alfredson & Cook, 2007; Dimitrios et al., 2011; Fahlstrom et al., 2003; Jonsson et al., 2008; Jonsson & Alfredson, 2005; Mafi et al., 2001; Morrissey et al., 2011). Even with its success, eccentric exercises are painful and patient compliance can be difficult. Other treatment options used to decrease pain in tendinopathy patients that are not commonly researched include mobilization with movement (MWM) (Abbott et al., 2001; Case & Desantis, 2006; Djordjevic, Vukicevic, Katunac, Jovic, & Katunac, 2012; Takasaki, Hall, & Jull, 2013; Teys et al., 2013; Teys, Bisset, & Vicenzino, 2008; Vicenzino et al., 2001) and positional release therapy (PRT) (Baker et al., 2014; Howell, Cabell, Chila, & Eland, 2006). To date, no investigators have determined the most effective method for treating tendinopathy.

Common conservative treatments.

Rest. Tendon overload is thought to be one of the causes of tendinopathy (Rees et al., 2006). Although the biological effects of relative rest are not well known, rest has been suggested as an initial treatment for tendon overload (Alfredson, 2005; Jelinsky et al., 2008). Jelinsky et al. (2008) demonstrated that two weeks of rest is sufficient to recover from two to four weeks of overuse. Although this study was performed on rats, it provides a foundational understanding to the potential biological effects of rest on human tendons.

Translating animal studies to practice and experiencing similar results may be difficult for many reasons. In addition to physiological differences between participants,

many laboratory studies use time frames that are not often seen in clinical practice. For instance, the majority of patients who experience tendon pain report symptoms longer than four weeks duration, while many of the laboratory studies are focusing on a true acute inflammatory condition.

Knobloch et al. (2007) compared relative rest and cryotherapy to an eccentric training program on patients who had Achilles tendinopathy. After twelve weeks of relative rest and cryotherapy, no significant change in the visual analog scale (VAS) was found, whereas patients who participated in the eccentric protocol saw a significant decrease in the VAS. Alfredson et al. (1998) also reported patients who were in a relative rest group experienced no significant change in clinical symptoms. While relative rest is sometimes prescribed, its clinical outcomes are inconclusive at this time (Alfredson et al., 1998; Jelinsky et al., 2008; Knobloch, Spies, Busch, & Vogt, 2007).

Non-steroidal anti-inflammatory drugs and corticosteroids. Non-steroidal anti-inflammatory drugs (NSAIDs) are widely used in sport and the general population (Magra & Maffulli, 2006; Paoloni, Milne, Orchard, & Hamilton, 2009). The rationale for NSAIDs use is based on the drugs' ability to reduce inflammation. Non-steroidal anti-inflammatory drugs inhibit cyclooxygenase (COX) production. Cyclooxygenase regulates cyroprotection, platelet aggregation, vascular hemostatis, and renal blood flow (COX-1), and promotes the inflammatory mediators and cytokines (COX-2) (Chen & Dragoo, 2013). The inhibition of COX-1 and COX-2 blocks the release of prostaglandins, which then interrupts the metabolic cascading inflammatory response (Hashimoto, Nobuhara, & Hamada, 2003). Researchers, however, have demonstrated that few, if any, inflammatory markers are present in the chronic stage of tendinopathy (Hashimoto et al., 2003). If inflammatory markers are not

present, the use of NSAIDs would not be indicated. Non-steroidal anti-inflammatory drugs are frequently recommended for the initial treatment of symptomatic tendinopathy; however, NSAID use remains controversial in either the acute or chronic stage (Rees et al., 2006; Wang et al., 2006).

Studies have been conducted to assess the short-term effects of NSAIDs use, but research on the long-term effects of NSAIDS on tendinopathy is lacking. Andres and Murrell (2008) reviewed seventeen placebo-controlled studies of NSAIDs use in the treatment of chronic tendon problems. The NSAIDs group experienced greater short-term (i.e., 7 to14 days) pain relief than the placebo group in 14 of the studies, but did not demonstrate any improvement in the other three studies (Andres & Murrell, 2008). Andres and Murrell (2008) reported that patients with greater symptom duration or severity were less likely to experience a favorable response to NSAID use. Additionally, while favorable pain results were identified in the majority of the studies, no long-term follow-ups were made; therefore, the complete resolutions of the tendon problems were not recorded. Although the lasting effects of NSAID use on tendinopathy are unknown, long-term use of NSAIDs does appear to increase the risk of gastrointestinal, cardiovascular, and renal side effects (Andres & Murrell, 2008; Kaux et al., 2011).

While NSAIDs appear to be effective for short-term pain control, there is potential for the effectiveness to result in negative consequences. Researchers have suggested that the analgesic effect may cause patients to ignore symptoms, which could result in further damage to the effected tendon (Kaux et al., 2011). While NSAIDs are used to reduce inflammation, researchers have established that NSAID use also results in the inhibition of tendon regeneration, (Marsolais, Cote, Frenette, & Côté, 2003) glycosaminoglycan

synthesis, and cell proliferation (Riley et al., 2001). Currently, it is not clear if NSAID use actually alters the healing of the tendon (Rees et al., 2006). Although NSAIDs appear to reduce pain, there is no biological evidence of their effectiveness in treating tendinopathy.

Corticosteroids are also used to treat tendon pathologies; however, the benefits appear to be short-lived. Smidt et al. (2002) found significantly better outcomes when treating lateral epicondylitis with cortisone injections compared to physical therapy and a wait-and-see policy at a six-week follow-up. Long-term follow-ups, however, indicated that physical therapy, coupled with the wait-and-see policy, had greater success on the outcomes measures than the injection group, which experienced a high recurrence rate.

Similarly, Bisset et al. (2006) compared corticosteroid injection (two if necessary), physiotherapy (manipulation and exercise), and wait-and-see methods in subjects with tennis elbow. The randomized control trial resulted in favorable short-term outcomes for corticosteroid injections at six weeks compared to the wait-and-see method. Conversely, the corticosteroid injection group had the highest recurrence rate among the three groups with the least favorable outcomes at fifty-two weeks. Physiotherapy was superior to both the corticosteroid injection and the wait–and-see groups at six weeks, but it was not significantly different from the wait-and-see group at fifty-two weeks (Bisset et al., 2006). Other researchers have produced similar short-term and long-term outcomes when using corticosteroids to treat tendinopathy patients (Hay et al., 1999; Smidt et al., 2002), which supports a potential need to decrease the use of corticosteroids to treat tendon pain.

Negative consequences with the use of certain medication with tendon patients. Corticosteroid use has been associated with a risk of negative long-term effects (Ackermann & Renström, 2012). Researchers have demonstrated that local corticosteroid injection has

reduced tendon strength in animals (Orchard & Kountouris, 2011). Researchers have also reported several incidents of Achilles tendon rupture following a corticosteroid injection in patients (Andres & Murrell, 2008), but the percentage of complications decreased when the injection occurred under fluoroscopic guidance around the tendon and not within the substance of the tendon (Gill, Gelbke, Mattson, Anderson, & Hurwitz, 2004). Although corticosteroids may provide initial short term pain relief, a practitioner and patient must be cautious of the possible negative effects when deciding on a tendinopathy treatment.

Moreover, when treating tendinopathy patients, clinicians must be aware of patients who are taking fluoroquinolones. Fluoroquinolones are broad-spectrum antibiotics used to treat a variety of illnesses (e.g., urinary tract infections) and include ciprofloxacin (Cipro), gemifloxacin (Factive), levofloxacin (Levaquin), moxifloxacin (Avelox), norfloxacin (Noroxin), and ofloxacin (Floxin) (Andersson & MacGowan, 2003; Ivanov & Budanov, 2006). The use of these medications may have negative effects on tendons (e.g., increased risk of tendinitis, increased risk of tendon rupture) and may exacerbate the symptoms of the neurological disorder myasthenia gravis (Lewis & Cook, 2014). The highest occurrence of these adverse effects has been reported at the Achilles tendon, but adverse effects have also occurred at other tendons throughout the body. The overall frequency of fluoroquinolone-associated Achilles tendon rupture in patients treated with ciprofloxacin or levofloxacin has been estimated at 17 per 100,000 treatments (three times the rate in people without fluoroquinolone exposure) (Khaliq & Zhanel, 2005; Owens & Ambrose, 2005). Fluoroquinolone-associated tendinopathy symptoms have occurred as early as two hours after initial exposure to the medication and as late as six months after the medication was discontinued (Hall, Finnoff, & Smith, 2011).

Younger patients typically experience good recovery following these reactions, but permanent disability is possible, especially in the geriatric population (Kim, 2010). Risk is substantially elevated in the elderly and in those with recent exposure to topical or systemic corticosteroid therapy. Simultaneous use of corticosteroids is present in almost one-third of quinolone-associated tendon rupture (Khaliq & Zhanel, 2005). Other risk factors include: patients with kidney, heart, or lung transplants, patients that participate in strenuous physical activity during or immediately after treatment, patients with renal failure, or patients with previous tendon disorders, such as rheumatoid arthritis. Some experts have advised avoidance of fluoroquinolones in athletes (Sode, Obel, Hallas, & Lassen, 2007).

Eccentric exercise. Stanish, Rubinovich, and Curwin (1985) were pioneers in developing an eccentric exercise (EE) training protocol to treat tendinitis. Stanish et al.'s technique consisted of three tenets: 1) increasing the length of the muscle with static stretching decreased the strain on the tendon during movement, 2) progressively increasing a load on a tendon increased tensile strength, and 3) increasing the speed of contraction increased the force developed. The program was continued until the patient no longer had pain or the pain did not affect activities of daily living (Stanish, Curwin, & Rubinovich, 1985).

Alfredson, et al. (1998) also studied the effects of EE on tendinopathy and created a similar protocol to Stanish (1985). The Alfredson protocol consisted of performing EE two times a day, seven days a week, for twelve weeks. Patients performed the exercises with the knee straight and the knee bent. The load was progressed as pain decreased. The difference in the Alfredson protocol compared to the Stanish protocol is that the speed of the movement was slower and a set period of twelve weeks of exercise was performed.

Additionally, patients were required to exercise into pain when utilizing Alfredson's protocol, whereas Stanish required no pain or minimal discomfort from his patients.

Several researchers have demonstrated promising results using eccentric exercises to treat tendinopathy (Alfredson et al., 1998; Cannell et al., 2001; Dimitrios et al., 2011; Fahlström, Jonsson, Lorentzon, & Alfredson, 2003; Jonsson et al., 2008; Mafi et al., 2001; Ohberg & Alfredson, 2004; Purdam et al., 2004; Svernlöv et al., 2012; Young et al., 2005). While the EE protocol is time intensive and requires the patient to exercise through pain, there are minimal adverse affects. The overall trend suggests a positive effect with EE protocols established for Achilles, patellar tendon, and common extensor tendons.

Eccentric exercise – achilles. Positive results were found using EE on chronic mid-portion Achilles tendon pain but were not found in patients with insertional Achilles tendon pain (Fahlstrom et al., 2003). Jonsson et al. (2008) evaluated EE calf training with and without dorsiflexion in 27 patients who had insertional Achilles tendinopathy. The researchers hypothesized the previous studies poor results of EE for insertional Achilles tendinopathy, compared to mid-portion tendinopathy, could be attributed to possible impingement that occurs between the tendon, bursa, and bone when the ankle goes into dorsiflexion. Upon completion of the modified EE training protocol, 67% of the patients were satisfied with their results and had a significant reduction in VAS scores. While the remainder of the patients did experience improvement in their reported pain levels, the reported changes were not statistically significant and these patients were unable to return to previous activity levels. The researchers theorized the promising results for treating insertional Achilles tendinopathy occurred because the movement avoided dorsiflexion, which limited the impingement between the tendon, bursa, and bone (Jonsson et al., 2008).

To study the effects of a slightly modified eccentric protocol for Achilles pain, Silbernagel et al. (2001) progressed an experimental group through three different phases over the course of twelve weeks. The phases including stretching, concentric/eccentric exercises, quick rebounding toe-raises, range of motion, balance, and gait exercises. The control group performed gastroc-soleous complex stretching and two-legged concentric and eccentric toe-raises. Although the experimental group had significant decreases in pain (not seen in the control group) at the twelve-week and six-month follow-up, it was difficult to determine what the true cause of pain reduction was due to multiple treatment methods.

Eccentric exercises vs. concentric exercises – achilles. Other researchers have investigated the effects of EE on Achilles tendinopathy (2-6 cm above the insertion) in comparison to concentric exercises. Mafi et al. (2001) compared EE training to concentric exercise (CE) training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. Patients in the EE group followed the Alfredson et al. (1998) protocol (Mafi et al., 2001). At the end of twelve weeks, 82% (18/22) of the patients who were in the EE group reported satisfaction and resumed their previous activity levels, while 36% (8/22) of the patients in the CE group reported satisfaction. Possible explanations for the improved results with EE include lengthening the muscle-tendon unit and putting decreased load on the tendon during motion, or metabolic changes, both of which cause alterations in pain perception. Mafi et al. (2001) concluded that EE yielded good short-term results in patients who have Achilles tendinosis 2-6 cm above the insertion.

Morrissey et al. (2011) compared Achilles tendon stiffness after EE and CE protocols. The EE group performed exercises according to the Alfredson protocol for six weeks, while the CE group performed a matched intensity heel raise. Morrissey et al. (2011)

demonstrated a significant decrease in Achilles tendon stiffness in the EE group. The CE group did not have any significant observed changes, and a difference in jump height was not observed in either group. The researchers concluded that EE training could decrease tendon stiffness and contribute therapeutic benefits to patients with Achilles tendinopathy (Morrissey et al., 2011).

Eccentric exercise – patellar. Researchers have also investigated the effects of EE on patellar tendinopathy. Cannell et al. (2001) compared the effectiveness of a drop squat program with a progressive training leg extension/curl program on 19 patients with jumper's knee (i.e., patellar tendinitis). The patients were randomly assigned to one of the respective groups after clinical evaluation and testing was performed at baseline (i.e., VAS pain measurement and isokinetic quadriceps and hamstring strength). During the first two weeks, the patients in both groups were treated with ice, anti-inflammatory medication, and relative rest. After the two weeks of common conservative treatments, each group began their respective exercise program. The drop squat protocol consisted of three sets of twenty drops performed once each day, five days per week. When the patient was able to perform three sets of twenty easily, the weight was progressed. Patients in the leg extension and leg curl group slowly performed three sets of ten leg extension and leg curl lifts once each day, five days per week. Once patients in either group were pain free, they performed a graduated return to run protocol. A significant reduction in pain for both groups was observed; however, between groups pain reduction was not significant. The researchers concluded that a drop squat program was safe and as effective as a leg extension and hamstring curl program in reducing painful patellar tendinopathy. Although the investigators observed significant results, future studies with larger sample sizes are needed (Cannell et al., 2001).

Young et al. (2005) studied the effects of treating patellar tendinopathy using two different EE protocols: a flat surface and a decline protocol. Patients who suffered from pain in the proximal tendon participated in the non-randomized pilot study. The flat surface group (N=9) performed three sets of fifteen repetitions twice daily for twelve weeks. The remaining eight patients performed the same protocol on a twenty-five degree decline surface. Patients in both the incline and flat surface group increased their workload by 5kg increments as the exercises became pain-free. Both groups had improved significantly in both outcome measures (VISA-P and VAS) at twelve weeks and twelve months, but there was not a significant difference between groups for either outcome measure.

Eccentric exercises - lateral elbow. While evidence indicates that EE can be beneficial for Achilles and infrapatellar tendinopathy, few researchers have addressed the benefits of EE on lateral epicondylalgia. Martinez-Silvestrini et al. (2005) randomly assigned 94 patients with lateral epicondylalgia into one of three groups: stretching, CE strengthening with stretching, and EE strengthening with stretching. Each patient was assessed for pain function and strength of the lateral elbow and measurements were repeated after six weeks of respective treatment. While all three groups demonstrated significant improvement over the six week period, there was not a statistically significant difference in improvement between groups. Each treatment group was provided with ice, time to stretch, and education, which might explain the lack of any significant difference between treatment groups. Additionally, according to Alfredson et al. (1998), EE protocol should be performed for twelve weeks instead of six weeks. The use of a shorter time period and the lack of continued follow-up examinations may account for the results being similar between treatment groups. Finally, the use of a control group (i.e., no treatment) would have helped

to determine if the treatment options produced better outcomes than natural recovery from rest. Comparison to a control group would have provided additional evidence to support treatment effectiveness, while also potentially identifying if additional time (i.e., longer treatment periods, long-term follow-up) was needed to determine effectiveness of the interventions.

Novel Treatments

Mulligan concept - mobilization with movement. The Mulligan concept is a manual therapy treatment designed to treat musculoskeletal pain and decreased range of motion. Mobilization with movement (MWM) is a technique where a pain-free joint glide is applied parallel to the treatment plane while the patient performs an active movement that has been restricted and/or painful (Mulligan, 1993). The mobilization is sustained through the entire movement. The success of this manual therapy treatment was attributed to the correction of a positional fault (Mulligan, 1993). Brian Mulligan suggested that if the application of MWM during the assessment produces a **p**ain-free, **i**nstant result, and **l**ong **l**asting (PILL) response, the use of MWM as a treatment method is indicated (Mulligan, 2010). There is significant evidence that pain decreases with the application of MWM in various peripheral joints (Abbott et al., 2001; Case & Desantis, 2006; Djordjevic et al., 2012; Takasaki et al., 2013; Teys et al., 2013, 2008; Vicenzino et al., 2001).

Mobilization with movement – knee. Limited research is available regarding the effects of MWM on pathology in the knee, particularly patellar tendinopathy. Takasaki, Hall, and Jull (2013) investigated immediate and short-term effects of MWM on patients with osteoarthritis (OA). Significant improvements in VAS were observed at baseline and at all assessment points in each task. The investigators were not able to determine the exact

mechanism for pain reduction as both biomechanical and neurophysiological mechanisms could have been involved. Although this study was performed on patients with OA, further investigation into the effects of MWM on patellar tendinopathy are warranted.

Mobilization with movement - lateral elbow. Clinical implications for using MWM to treat lateral epicondylalgia include pain at the lateral aspect of the elbow, pain with gripping, pain gripping with resisted wrist or finger extension (Abbott et al., 2001). The method for a lateral-glide MWM includes moving the ulna/radius laterally while the other hand stabilizes the distal end of the humerus. If repositioning the ulna on the distal end of the humerus decreases the pain, then MWM is an appropriate treatment. Brian Mulligan recommended three sets of ten repetitions or more to alleviate all the pain when gripping (Mulligan, 2010).

Several researchers have produced significant clinical results when treating lateral epicondylalgia with MWM (Abbott et al., 2001; Fernández-Carnero, Fernández-de-las-Peñas, Cleland, Fern, & Fern, 2009; Pagorek, 2009; Paungmali, Vicenzino, & Smith, 2003; Vicenzino et al., 2001). Abbott et al. (2001) observed increases in pain-free grip strength (PFGS) after MWM. However, the researchers only measured immediate effects of the treatment and no long-term follow-up was tracked. Vicenzino et al. (2001) demonstrated that after three treatment sessions, patients with lateral epicondylalgia had a significant increase in PFGS compared to the placebo. Paungmali et al. (2003) found similar results. Vicenzino et al. (2001) and Paungmali et al. (2003) demonstrated that after only three treatments, rapid hypoalgesic effects occurred only in the group treated with MWM and not the placebo. Pain pressure threshold also increased in both groups that received MWM treatment.

Slater et al. (2006) investigated whether the lateral glide MWM would activate mechanisms associated with analgesia in experimentally induced lateral epicondylalgia. Delayed onset muscle soreness (DOMS) and hypertonic saline solution were induced prior to lateral glide MWM treatment intervention. The MWM failed to reduce pain symptoms. The researchers stated the lateral glide MWM does not activate mechanisms associated with analgesia. Comparisons between experimentally induced lateral epicondylalgia and actual symptomatic lateral epicondylalgia are difficult to make due to the differences in etiology. Mobilization with movement is indicated to treat a positional fault (Mulligan, 1993), and it is unlikely that saline-induced pain and DOMS creates the same physiological mechanism of injury that would respond to an MWM. In addition, the pathology of experimentally-induced lateral epicondylalgia and tendinopathy are not the same. Delayed onset muscle soreness is thought to be due to microtrauma and disruption of the Z-bands; however, its origin is unknown (Yu, Liu, Carlsson, Thornell, 2013). In contrast, researchers have exhibited that lateral epicondylalgia has the presence of free nerve endings, substance P, and glutamate (Donaldson, 2013). The lack of pain reduction post MWM treatment in saline-induced lateral elbow pain is not surprising.

Mulligan techniques - taping. If an MWM produces the PILL response, taping techniques can be utilized in order to replicate the mobilization and assist with providing pain reduction outside treatment (Mulligan, 2010; Vicenzino, 2003). Limited research is available that addresses the use of MWM and a Mulligan taping technique on the elbow, knee, or Achilles tendons. Amro, Diener, Bdair, Hameda, Shalabi, and Illyyan (2010) studied the effects of Mulligan mobilization and taping on patients who had lateral epicondylalgia. The experimental group received a combination of traditional therapy

(thermal heat, massage, and ultrasound) and three sets of twelve lateral glides of MWM in conjunction with a taping technique. The control group received the traditional treatment only. Both groups were seen three times a week for four weeks. At the end of four weeks, the researchers discovered that both groups demonstrated significant improvements in VAS, patient-rated tennis elbow evaluation (PRTEE), and maximum grip strength; however, the VAS and PRTEE improved significantly greater in the experimental group compared to the control group. The researchers believed that the addition of Mulligan techniques to traditional treatment led to better outcomes (Amro et al., 2010). Further research is necessary to study the effects of the Mulligan techniques on tendon pain in the elbow, Achilles, and patellar tendons.

Positional release therapy. Positional release therapy (PRT), also called "Strain-Counterstrain" or "Counterstrain," (Jones, 1981) is used to interrupt the pain spasm cycle and to treat somatic dysfunctions. Korr (1975) believed that the sensitivity of the monosynaptic stretch reflex played a role in restricted range of motion. Positional release therapy is an indirect technique that uses tender points (TPs) as a diagnostic guide. After locating a TP, the clinician treats the affected tissue by holding the TP with a sub-threshold pressure while placing the patient into a position of comfort (POC) (Jones, 1981). Treatment is applied to the most active tender points first, followed by the next most active tender points, and so on. Proximal or medial points are treated before distal or lateral points (D'Ambrogio & Roth, 1997). If done correctly, PRT should reduce irregular muscle spindle activity (Speicher & Draper, 2006). Theoretically, PRT benefits the patient by positively affecting several pathophysiological events: neuromuscular hyperirritability, muscular hypertonicity, tissue tension, and inflammation. Positional release therapy reduces pain by

restoring the local tissue environment through increased oxygen and a decrease in inflammatory metabolites (D'Ambrogio & Roth, 1997). While PRT is thought to play a role in the treatment of somatic dysfunction, minimal research has been conducted on its effectiveness in treating Achilles tendinopathy, lateral epicondylalgia, and infrapatellar tendinopathy.

According to Baker et al. (2014), PRT was effective in alleviating pain and dysfunction in a swimmer who presented with a history of bicipital tendinitis. The patient presented with pain and thickening over the long head of the biceps tendon. Tender points were found in the biceps long head (BLH), supraspinatus (SSL), and pectoralis minor (PMI). The patient received PRT to the tender points at the conclusion of swim practice. Positional release therapy was applied the next day, and the patient was discharged on the fourth day as asymptomatic. Baker et al. (2014) concluded that use of PRT was an appropriate treatment intervention and produced clinically significant results. Further research is needed to determine the effectiveness of PRT in tendon disorders in various stages of the reactive tendinopathy continuum.

Neurodynamics. According to Apelby-Albrecht et al. (2013), Kenneally was the first to investigate the three major "upper limb tension tests." Shacklock and Butler now utilize four tests for what is known as the "upper limb neurodynamic test" (Apelby-Albrecht et al., 2013). While performing neurodynamic tests, there are two types of movements: sensitizing and differentiating. Sensitizing movements involve placing stress on both musculoskeletal structures and the neurological system. Differentiating movements only involve placing stress on the neurological system. Coppieters et al. (2008) wanted to determine if the movements in the slump and straight leg raise (SLR) tests were sensitizing

movements. In this study the researchers injected patients with hypertonic saline in either the anterior tibialis or soleus. The foot and ankle were placed in a splint while electromyography (EMG) was connected to the muscle injected with the saline solution. Electromyography was utilized to determine if there was musculoskeletal stress placed on the structures during testing. An increase in perception of pain was not reported. According to the researchers, the movements involved in the slump and SLR tests are sensitizing movements. Coppieters et al. (2008), however, went on to explain that the movements had no musculoskeletal involvement and solely placed stress on the nervous system. According to Shacklock (2005), this would mean they are differentiating movements. Terminology was not used correctly in this study and could potentially mislead the reader.

Neurodynamic tests have both mechanical and physiological responses. Mechanical responses include neural movement, tension, intraneural pressure changes, alterations of cross sectional shape, and viscoelastic function. Physiological responses include alterations in intraneural blood flow, impulse traffic, and axonal transport. Neurodynamic tests may also cause sympathetic activation (Shacklock, 2005).

A neurodynamic response will result in a change in symptoms or range of motion, or a palpable resistance with differentiating movements. A musculoskeletal response will result in no change in any of the aforementioned assessments (Shacklock, 2005). According to Shacklock (2005), there are three responses to a neurodynamic test: normal, overt abnormal, or covert abnormal. A normal response would result in the patient feeling a pulling sensation, a neurological response not associated with a condition. An overt abnormal response is an obvious neural cause, such as tingling or numbness. A covert abnormal response is more difficult to classify as either a neurological or a musculoskeletal response

to testing. Testing may need to be re-examined with musculoskeletal components. A positive neurodynamic test will include the reproduction of symptoms, the observance of differences when compared bilaterally, and the symptoms will increase with structural differentiation (Shacklock, 2005).

Neurodynamic tests indicate two types of dysfunctions, sliding and tension dysfunctions. A sliding dysfunction is indicated if a patient's symptoms decreased as the patient was pushed further into the test. A tension dysfunction is indicated if a patient's symptoms increased as the patient was pushed further into the test. Based on clinical findings, the clinician determines the appropriate intervention using Shacklock's classification system (Shacklock, 2005).

Table 4.1.
Shacklock's Classification System

Shacklock's Classification System

Level	Characteristics
Zero	Psychosocial and psychological issues, severe pain due to physical problems, neurodynamic tests are contraindicated
One	Limited examination, to evoke symptoms, neurological deficit would be present, musculoskeletal and neurodynamics tests separate, differentiating movement would be applied first
Two	Standard exam, take nerve through full available range of motion, musculoskeletal and neurodynamics tests separate, avoid excessive pain and neurological symptoms
Three	Apply sensitizing maneuver but not always using differentiating movements, start sequence locally, use of innervated tissues, symptoms result of neuromusculoskeletal dysfunction, place in symptomatic position or movement

Nee, Yang, Liang, Tseng, and Coppieters (2010) applied the upper limb neurodynamic test (ULNT) to the median nerves of seven cadavers in order to determine the amount of tension on the nerve. Prior to this test, researchers proposed that the sequence of the movements would place more strain on certain parts of the nerve; however, after performing three movement sequences, Nee et al. (2010) found the tension on the nerve was the same. Researchers did find higher strain for longer duration in the distal to proximal movement (Nee et al., 2010).

Castellote-Caballero et al. (2013) split 28 patients into two groups: control and neurodynamics. Both groups complained of hamstring tightness. The neurodynamics group performed a slump slider for sixty seconds five times a day for three days. Researchers found a significant increase in range of motion (ROM) among the patients in the neurodynamics group. Castellote-Caballero et al. (2013) findings could substantiate the

argument that many of the common injuries or complaints seen on a daily basis may have a neurological component.

Boyd & Villa (2012) had 40 healthy patients with no positive neurodynamic test. Each subject underwent two SLR tests. First, each subject performed the SLR test in a brace, holding the proper foot position. Second, each subject performed the SLR test manually, with the clinician holding the foot in the proper position. A significant difference was found between the two methods. Researchers were able to state with 95% confidence that 90% of the general population have inter-limb differences no greater than 10.9 degrees for plantarflexion and SLR 9.4 degrees for dorsiflexion and SLR.

Apelby-Albrecht et al. (2013) utilized 51 patients with cervical radiculopathy and compared the ULNT to a standard test. When all 4 ULNT were performed, there was 88.2% accuracy with a sensitivity of .97. Radial alone was the least accurate, with 52.9% accuracy. Ulnar was the highest individual test, with a specificity of .87. Using all four tests should be used to rule in cervical radiculopathy, and the ulnar test, alone, can help rule it out.

Coppieters & Butler (2008) examined two cadavers and used digital vernier calipers, transducers, and computers to track the changes in the nerve as sliders and tensioners were applied. The researchers wanted to determine if sliders slide and tensioners place tension on the nerves. The use of the computer was unique in the fact the clinician could see the angles and positions of the limb while performing the test. Seeing the computer in relation to the movements helped the clinician take the limb through the same motions and positions each time. Sliders produced 0.8% strain of the median nerve whereas; tensioners produced 6.8% strain of the median nerve. Coppieters & Butler (2008) concluded sliders do slide and tensioners do create tension.

Villafane, Pillastrini, Borboni (2013) reported on a case of peroneal nerve paralysis where neurodynamic mobilizations were performed for the sciatic nerve along with spinal and fibular head mobilizations. Manual muscle tests improved from one out of five to five out of five with the exception of the anterior tibialis, which only improved to four out of five. Pain pressure threshold increased from 1.5 to 5.75 kg/cm^2. The patient was pain free at follow-up three months post interventions, but it is difficult to determine which intervention helped the patient.

Neurodynamics, specifically sliders, on acute injuries, should be performed with few repetitions more frequently (Butler, 2014). Patients could perform four repetitions every hour or for a specific amount of time, such as two minutes, instead of a set number of repetitions. Sliders should be stopped just prior to pain. Chronic pain-state patients, with relevant physical dysfunction should ease into the pain followed by a release of the mechanism without a long duration hold. Repetitions should be performed less often than in the acute injury with a higher number of repetitions (i.e., 4-8 repetitions). Currently, literature does not exist expressing a link between neurodynamics and tendinopathy. Mechanical, vascular and neural theories have been proposed for tendinopathy (Rees et al., 2006), which indicate the potential appropriate use for neurodynamics.

Conclusion

The current gold standard for treating tendon pain and dysfunction is EE (Cannell et al., 2001; Curwin, 1998; Knobloch et al., 2007; Mafi et al., 2001; Martinez-Silvestrini et al., 2005; Morrissey et al., 2011; Purdam et al., 2004; Silbernagel, Thomee, Thomee, & Karlsson, 2011). Though EE has been extensively researched, the protocol is time consuming, painful, and can result in patient compliance issues (Alfredson et al., 1998;

Cannell et al., 2001; Dimitrios et al., 2011; Fahlstrom et al., 2003; Jonsson et al., 2008; Mafi et al., 2001; Ohberg & Alfredson, 2004; Purdam et al., 2004; Svernlöv et al., 2012; Young et al., 2005) and does not account for all of the examples of recovery. Scott et al. (2013) suggested that all treatments should be individualized based on specific patient presentations. General unimodal treatment plans are likely to be unsuccessful in the treatment of tendinopathy, especially for athletic populations (Scott et al., 2012).

Several researchers have demonstrated positive outcomes treating tendon pain using indirect treatments, such as MWM (Abbott et al., 2001; Fernández-Carnero et al., 2009; Paungmali et al., 2003; Takasaki et al., 2013; Vicenzino et al., 2001) and PRT (Baker et al., 2013), which would not appear to produce benefits under the same model as EE. Although research studies do not exist to validate the claim, researchers have suggested tendon pain may be due to neurogenic inflammation (Rees et al., 2006), which could indicate the use of neurodynamic treatment. Accordingly, the importance of classification of tendinopathy cannot be understated as appropriate classification is potentially vital to determining the best patient-match interventions. Upon delineating the appropriate classification, a clinician can then apply the treatment most likely to benefit the patient (Lewis, 2009). The purpose of this study is to determine if indirect treatment for patients with apparent tendinopathy will positively affect short- and long-term outcomes.

References

Abbott, J. H., Patla, C. E., & Jensen, R. H. (2001). The initial effects of an elbow mobilization with movement technique on grip strength in subjects with lateral epicondylalgia. *Manual Therapy*, *6*(3), 163–169.

Ackermann, P. W., Domeij-Arverud, E., Leclerc, P., Amoudrouz, P., & Nader, G. A. (2013). Anti-inflammatory cytokine profile in early human tendon repair. *Knee Surgery, Sports Traumatology, Arthroscopy : Official Journal of the ESSKA*, *21*(8), 1801–1806. doi:10.1007/s00167-012-2197-x

Ackermann, P. W., & Renström, P. (2012). Tendinopathy in sport. *Sports Health*, *4*(3), 193–201. doi:10.1177/1941738112440957

Alfredson, H. (2005). The chronic painful Achilles and patellar tendon: research on basic biology and treatment. *Scandinavian Journal of Medicine & Science in Sports*, *15*(4), 252–9. doi:10.1111/j.1600-0838.2005.00466.x

Alfredson, H., & Cook, J. (2007). A treatment algorithm for managing Achilles tendinopathy: new treatment options. *British Journal of Sports Medicine*, *41*(4), 211–6. doi:10.1136/bjsm.2007.035543

Alfredson, H., Ljung, B. O., Thorsen, K., & Lorentzon, R. (2000). In vivo investigation of ECRB tendons with microdialysis technique--no signs of inflammation but high amounts of glutamate in tennis elbow. *Acta Orthopaedica Scandinavica*, *71*(5), 475–479. doi:10.1080/000164700317381162

Alfredson, H., & Lorentzon, R. (2002). Chronic tendon pain: no signs of chemical inflammation but high concentrations of the neurotransmitter glutamate. Implications

for treatment? *Current Drug Targets*, *3*(1), 43–54. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11899264

Alfredson, H., Ohberg, L., & Forsgren, S. (2003). Is vasculo-neural ingrowth the cause of pain in chronic Achilles tendinosus? An investigation using ultrasonography and colour Doppler, immunohistochemistry and diagnostic injections. *Knee Surgery Sports Trauma/Arthroscopy*,*11*(5), 334–8. doi:10.1007/s00167-003-0391-6

Alfredson, H., Pietilä, T., Jonsson, P., & Lorentzon, R. (1998). Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *The American Journal of Sports Medicine*, *26*(3), 360–6. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9617396

Almekinders, L. C. (1998). Tendinitis and other chronic Tendinopathies. *Journal American* Academy of Orthopaedic Surgeons, 6, 157–14.

Altbach, P., Reisberg, L., & Rumbley, L. (2009). Trends in global higher education:tracking an academic revolution. *UNESCO* World Conference on Higher Education

Andersson, M., & MacGowan, A. (2003). Developement of the quinolones. *Journal of* Antimicrobial Chemotherapy, 51(Suppl.S1), 1–11.

Andres, B. M., & Murrell, G. a C. (2008). Treatment of tendinopathy: what works, what does not, and what is on the horizon. *Clinical Orthopaedics and Related Research*, *466*(7), 1539–54. doi:10.1007/s11999-008-0260-1

Apelby-Albrecht, M., Andersson, L., Kleiva, I. W., Kvåle, K., Skillgate, E., Josephson, A., & (2013). Concordance of Upper Limb Neurodynamic Tests With Medical Examination and Magnetic Resonance Imaging in Patients with Cervical

Radiculopathy: A Diagnostic Cohort Study. *Journal of Manipulative and Physiological Therapeutics*, *36*(9), 626–632.

Arner, O., Lindholm, A., & Orell, S. (1959). Histologic changes in subcutaneous rupture of the Achilles tendon; a study of 74 cases. *Acta Chirurgica Scandinavica*, *116*(5-6), 484–490.

Astrom, M. (1998). Partial rupture in chronic achilles tendinopathy. *Acta Orthopaedica Scandinavica*, *69*(4), 404–407.

Bains, B. S., & Porter, K. (2006). Lower limb tendinopathy in athletes. *Trauma*, *8*(4), 213–224. doi:10.1177/1460408606078110

Baker, R. T., Nasypany, A., Seegmiller, J. G., Baker, J. G., Baptist, A. T. C. C., Kaltenborn, F., & Maitland, G. (2013). The Mulligan Concept : Mobilizations With Movement. *Manual Theapy*, *18*(January), 30–35.

Baker, R. T., Riper, M. V., Nasypany, A. M., & Seegmiller, J. G. (2014). Evaluation and Treatment of Apparent Reactive Tendinopathy of the Biceps Brachii. *International Journal of Athletic Therapy & Training*, *19*, 14–21.

Balas, E., & Boren, S. (2000). Managing clinical knowledge for health care improvement. Yearbook of Medical Informatics 65-70.

Bamji, A., Dieppe, P. A., Haslock, D, I., & Shipley, M. E. (1990). What do rheumatologist do? A pilot study. *British Journal of Rheumatology*, *29*, 295–2998.

Birch, H. L., Thorpe, C. T., & Rumian, A. P. (2013). Specialisation of extracellular matrix for function in tendons and ligaments Corresponding author : *3*(1), 12–22.

Bisset, L., Beller, E., Jull, G., Brooks, P., Darnell, R., & Vicenzino, B. (2006). Mobilisation with movement and exercise, corticosteroid injection, or wait and see for tennis elbow: randomised trial. *BMJ (Clinical Research Ed.)*, *333*(7575), 939.

Blazina, M. E., Kerlan, R. K., Jobe, F. W., Carter, V. S., & Carlson, G. J. (1973). Jumpers Knee. *Orthopedic Clinics of North America*, *4*(3), 665–678.

Butler, D. (2000). *The Sensitive Nervous System*. Australia: Noigroup Publications.

Butler, D. (2014). Mobilization of the Nervous System – The Mobile Nervous System Clinical Reasoning and Pain. Retrieved January 1, 2014, from NES.com

Cannell, L. J., Taunton, J. E., Clement, D. B., Smith, C., & Khan, K. M. (2001). A randomised clinical trial of the efficacy of drop squats or leg extension/leg curl exercises to treat clinically diagnosed jumper's knee in athletes : pilot study. *British Journal of Sports Medicine*, *35*, 60–64.

Case, I. A., & Desantis, L. (2006). Use of Mobilization with Movement in the Treatment of a Patient, *14*(2).

Chen, M. R., & Dragoo, J. L. (2013). The effect of nonsteroidal anti-inflammatory drugs on tissue healing. *Knee Surgery, Sports Traumatology, Arthroscopy : Official Journal of the ESSKA*, *21*(3), 540–9. doi:10.1007/s00167-012-2095-2

Cook, J. L., Khan, K. M., & Purdam, C. R. (2001). Conservative treatment of patellar tendinopathy. *Physical Therapy in Sport*, *2*(2), 54–65. doi:10.1054/ptsp.2001.0069

Cook, L., Khan, K. M., Harcourt, P. R., Grant, M., & Young, D. A. (1997). A cross sectional study of 100 athletes with jumper's knee managed conservatively and surgically. *British Journal of Sports Medicine*, *31*, 332–336.

Curwin, L. (1998). The aetiology and treatment of tendinitis. Oxford of textbook of sports medicine. (2nd edition.). Oxford University Press.

D'Ambrogio, K., & Roth, G. (1997). Positional Release Therapy, Assesment & Treatment of Musculoskeletal Dysfunction. St. Louis: Mosby.

Dickenson, A. H., Chapman, V., & Green, G. M. (1997). The pharmacology of excitatory and inhibitory amino acid-mediated events in the transmission and modulation of pain in the spinal cord. *General Pharmacology*, *28*(5), 633–638.

Dimitrios, S., Pantelis, M., & Kalliopi, S. (2011). Comparing the effects of eccentric training with eccentric training and static stretching exercises in the treatment of patellar tendinopathy. A controlled clinical trial. *Clinical Rehabilitation*, *26*(5), 423–30. doi:10.1177/0269215511411114

Djordjevic, O. C., Vukicevic, D., Katunac, L., Jovic, S., & Katunac L, J. S. D. O. V. D. (2012). Mobilization with movement and kinesiotaping compared with a supervised exercise program for painful shoulder: results of a clinical trial. *Journal Of Manipulative And Physiological Therapeutics*, *35*(6), 454–463. doi:10.1016/j.jmpt.2012.07.006

Donaldson, O., Vannet, N., Gosens, T., & Kulkarni, R. (2013). Tendinopathies around the elbow part 1: lateral elbow tendinopathy. *Shoulder & Elbow*, *5*(4), 239-250.

Fahlstrom, M., Jonsson, P., Lorentzon, R., & Alfredson, H. (2003). Chronic Achilles tendon pain treated with eccentric calf-muscle training. *Knee Surgery, Sports Traumatology, Arthroscopy : Official Journal of the ESSKA*, *11*(5), 327–333. doi:10.1007/s00167-003-0418-z

Fenwick, S. a, Hazleman, B. L., & Riley, G. P. (2002). The vasculartureand its role in the damaged and healing tendon. *Arthritis Research*, *4*(4), 252–260. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=128932&tool=pmcentrez&rendertype=abstract

Fernández-Carnero, J., Fernández-de-las-Peñas, C., Cleland, J. A., Fern, J., & Fern, C. (2009). Mulligan's Mobilization with Movement and Muscle Trigger Point Dry Needling for the Management of Chronic Lateral Epicondylalgia: A Case Report. *Journal of Musculoskeletal Pain*, *17*(4), 409–415. doi:10.3109/10582450903284802

Ferretti, A., Conteduca, F., Camerucci, E., & Morelli, F. (2002). Patellar tendinosis: a follow-up study of surgical treatment. *The Journal of Bone and Joint Surgery. American Volume*, *84-A*(12), 2179–85.

Fisher, R. (1990). Teaching children to think. London: Nelson Thorns Ltd.

Forde, M. S., Punnett, L., & Wegman, D. H. (2005). Prevalence of musculoskeletal disorders in union iron workers. *Journal of Occupational and Environmental Hygiene*, *2*, 203–212.

Fredberg, U., & Stengaard-Pedersen, K. (2008). Chronic tendinopathy tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. *Scandinavian Journal of Medicine & Science in Sports*, *18*(1), 3–15. doi:10.1111/j.1600-0838.2007.00746.x

Gill, S. S., Gelbke, M. K., Mattson, S. L., Anderson, M. W., & Hurwitz, S. R. (2004). Fluoroscopically Guided Low-Volume Peritendinous Corticosteroid Injection for Achilles Tendinopathy. *The Journal of Bone & Joint Surgery*, *86*(4), 802–806. Retrieved from http://jbjs.org/content/86/4/802.abstract

Glaser, T., Poddar, S., Tweed, B., & Webb, C. W. (2008). Clinical inquiries. What's the best way to treat Achilles tendonopathy? *The Journal of Family Practice, 57*(4), 261–263.

Green, L. (2008). Making research relevant: if it is an evidence-based practice, where's the practice-based evidence? *Family Practice* 25(supp 1), 20-24.

Grigg, N. L., Wearing, S. C., & Smeathers, J. E. (2009). Eccentric calf muscle exercise produces a greater acute reduction in Achilles tendon thickness than concentric exercise. *British Journal of Sports Medicine*, *43*(4), 280–3. doi:10.1136/bjsm.2008.053165

Gruchow, H. W., & Pelletier, D. (1979). An epidemiologic study of tennis elbow: incidence, recurence, and effectiveness of prevention strategies. *American Journal of Sports Medicine*, *7*, 234–238.

Hall, M. M., Finnoff, J. T., & Smith, J. (2011). Musculoskeletal complications of fluoroquinolones: guidelines and precautions for usage in the athletic population. *PM&R*, *3*(2), 132–142.

Hashimoto, T., Nobuhara, K., & Hamada, T. (2003). Pathologic Evidence of Degeneration as a Primary Cause of Rotator Cuff Tear. *Clinical Orthopaedics and Related Research*, *415*. Retrieved from http://journals.lww.com/corr/Fulltext/2003/10000/Pathologic_Evidence_of_Degeneration_as_a_Primary.15.aspx

Hay, E. M., Paterson, S. M., Lewis, M., Hosie, G., & Croft, P. (1999). Pragmatic randomised controlled trial of local corticosteroid injection and naproxen for treatment of lateral epicondylitis of elbow in primary care. *Bmj*, *319*(7215), 964-968.

Hoffmann, P., Hoeck, K., Deters, S., Werner-Martini, I., & Schmidt, W. E. (2010). Substance P and calcitonin gene related peptide induce TGF-alpha expression in epithelial cells via mast cells and fibroblasts. *Regulatory Peptides*, *161*(1-3), 33–37. doi:10.1016/j.regpep.2009.12.016

Howell, J. N., Cabell, K. S., Chila, A. G., & Eland, D. C. (2006). Stretch reflex and Hoffmann reflex responses to osteopathic manipulative treatment in subjects with Achilles tendinitis. *The Journal of the American Osteopathic Association*, *106*(9), 537–45. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17079523

Ivanov, D., & Budanov, S. (2006). Ciprofloxacin and antibacterial therapy of respiratory tract infections. *Antibiot Khimioter*, *51*(5), 29–37.

James, S. L., Bates, B. T., & Osternig, L. R. (1978). Injuries to runners. *American Journal of Sports Medicine*, *6*, 40–50.

Jarvinen, T. A. H., Kannus, P., Maffulli, N., & Khan, K. M. (2005). Achilles tendon disorders: etiology and epidemiology. *Foot and Ankle Clinics*, *10*(2), 255–266. doi:10.1016/j.fcl.2005.01.013

Jelinsky, S. A, Lake, S. P., Archambault, J. M., & Soslowsky, L. J. (2008). Gene expression in rat supraspinatus tendon recovers from overuse with rest. *Clinical Orthopaedics and Related Research*, *466*(7), 1612–7. doi:10.1007/s11999-008-0270-zJones, L. (1981). *Strain and Counterstrain*. The American Academy of Osteopathy.

Jonsson, P., & Alfredson, H. (2005). Superior results with eccentric compared to concentric quadriceps training in patients with jumper's knee: a prospective randomised study. *British Journal of Sports Medicine*, *39*(11), 847–50. doi:10.1136/bjsm.2005.018630

Jonsson, P., Alfredson, H., Sunding, K., Fahlström, M., & Cook, J. (2008). New regimen for eccentric calf-muscle training in patients with chronic insertional Achilles tendinopathy: results of a pilot study. *British Journal of Sports Medicine*, *42*(9), 746–9. doi:10.1136/bjsm.2007.039545

Jozsa, L., & Kannus, P. (1997). *Human Tendons. Anatomy, Physiology, and Pathology.* Champign,IL: Human Kinetics.

Kaux, J. F., Forthomme, B., Goff, C. Le, Crielaard, J. M., & Croisier, J. L. (2011). Current Opinions on Tendinopathy. *Journal of Sports Science & Medicine*, *10*(2), 238–253. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3761855&tool=pmcentrez&rendertype=abstract

Khaliq, Y., & Zhanel, G. G. (2005). Musculoskeletal injury associated with fluoroquinolone antibiotics. *Clinics in Plastic Surgery*, *32*(4), 495–502.

Khan, K. M., Cook, J. L., Bonar, F., Harcourt, P., & Astrom, M. (1999). Histopathology of common tendinopathies. Update and implications for clinical management. *Sports Medicine (Auckland, N.Z.)*, *27*(6), 393–408. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10418074

Khan, K. M., Cook, J. L., Kannus, P., Maffulli, N., & Bonar, S. F. (2002). Time to abandon the "tendinitis" myth: Painful, overuse tendon conditions have a non-inflammatory pathology. *BMJ: British Medical Journal*, *324*(7338), 626.

Khan, K. M., Maffulli, N., Coleman, B. D., Cook, J. L., & Taunton, J. E. (1998). Patellar tendinopathy: some aspects of basic science and clinical management. *British Journal of Sports Medicine*, *32*(4), 346–55. Retrieved from

http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1756121&tool=pmcentrez&rendertype=abstract

Kim, G. K. (2010). The risk of fluoroquinolone-induced tendinopathy and tendon rupture: what does the clinician need to know? *The Journal of Clinical and Aesthetic Dermatology*, *3*(4), 49.

Knobloch, K., Spies, M., Busch, K. H., & Vogt, P. M. (2007). Sclerosing therapy and eccentric training in flexor carpi radialis tendinopathy in a tennis player. *British Journal of Sports Medicine, 41*(12), 920–1. doi:10.1136/bjsm.2007.036558

Koshy, E., Koshy, V., & Waterman, H. (2011). *Action research in healthcare.* Thousand Oaks, CA: SAGE Publications Inc.

Kujala, U. M., Sarna, S., & Kaprio, J. (2005). Cumulative incidence of Achilles tendon rupture and tendinopathy in male former elite athletes. *Clinic Journal of Sport Medicine*, *15*, 133–135.

Kvist, M. (1994). Achilles tendon injuries in athletes. *Sports Medicine (Auckland, N.Z.)*, *18*(3), 173–201.

Leadbetter, W. B. (1992). Cell matrix response to tendon injury. *Clinics in Sports Medicine, 3*, 533– 578.

Lewis, T., & Cook, J. (2014). Fluoroquinolones and tendinopathy: a guide for athletes and sports clinicians and a systematic review of the literature. *Journal of Athletic Training*, *49*(3), 422–427. doi:10.4085/1062-6050-49.2.09

Lodish, H., Berk, A., Zipursky, S. L., Matsudaira, P., Baltimore, D., & Darnell, J. (2000). *Molecular Cell Biology, 4th Edition* (4th ed.). New York, N.Y.: W. H. Freeman.

Maffulli, N., Wong, J., & Almekinders, L. C. (2003). Types and epidemiology of tendinopathy. *Clinics in Sports Medicine*, *22*(4), 675–692.

Mafi, N., Lorentzon, R., & Alfredson, H. (2001). Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. *Knee Surgery, Sports Traumatology, Arthroscopy*, *9*(1), 42–47. doi:10.1007/s001670000148

Maganaris, C. N. (2002). Tensile properties of in vivo human tendinous tissue.*Journal of biomechanics*, *35*(8), 1019-1027.

Magra, M., & Maffulli, N. (2006). Nonsteroidal antiinflammatory drugs in tendinopathy: friend or foe. *Clinical Journal of Sport Medicine : Official Journal of the Canadian Academy of Sport Medicine*, *16*(1), 1–3. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16377967

Marsolais, D., Cote, C., Frenette, J. J., & Côté, C. H. (2003). Nonsteroidal Anti-inflammatory Drug Reduces Neutrophil and Machrophage Accumulation but Does Not Improve Tendon Regeneration. *Laboratory Investigation; a Journal of Technical Methods and Pathology*, *83*(7), 991. doi:10.1097/01.LAB.0000078688.07696.AC

Martinez-Silvestrini, J. A, Newcomer, K. L., Gay, R. E., Schaefer, M. P., Kortebein, P., & Arendt, K. W. (2005). Chronic lateral epicondylitis: comparative effectiveness of a home exercise program including stretching alone versus stretching supplemented with eccentric or concentric strengthening. *Journal of Hand Therapy : Official Journal of the American Society of Hand Therapists*, *18*(4), 411–9, quiz 420. doi:10.1197/j.jht.2005.07.007

Mayor, R. B. (2012). Treatment of athletic tendonopathy. *Connecticut medicine,76*(8), 471-475.

Morrissey, D., Roskilly, A., Twycross-Lewis, R., Isinkaye, T., Screen, H., Woledge, R., & Bader, D. (2011). The effect of eccentric and concentric calf muscle training on Achilles tendon stiffness. *Clinical Rehabilitation*, *25*(3), 238–47. doi:10.1177/0269215510382600

Mulligan, B. (1993). Mobilisations with movement (MWM's). *Journal of Manual & Manipulative Therapy*, *1*(4), 154–156.

Mulligan, B. R. (2010). *Manual Therapy NAGS, SNGAS, MWM etc.* (6th ed.). Wellington, New Zealand: Plane View Services Ltd.

Nasypany, A.M., Seegmiller, J.G., & Baker, R. T. (2013). A model for developing scholarly advanced practice athletic trainers in post-professional education programs. Athletic Trainers' Educator Conference. Dallas, TX.

Nasypany, A., May, J., & Krzyzanowics, R. (Clinicians) (2014). *Nuts and Bolts: A Practical* Guide to Collecting Patient Outcomes. [DVD]. Available from https://www.neseminars.com/products/.

Nee, R. J., Yang, C. H., Liang, C. C., Tseng, G. F., & Coppieters, M. W. (2010). Impact of order of movement on nerve strain and longitudinal excursion: a biomechanical study with implications for neurodynamic test sequencing.*Manual therapy*, *15*(4), 376-381.

Nirschl, R. P., & Ashman, E. S. (2003). Elbow tendinopathy: tennis elbow. *Clinics in Sports Medicine*, *22*(4), 813–836. doi:10.1016/S0278-5919(03)00051-6

Oeppen, J., & Vaupel, J. W. (2002). Demography. Broken limits to life expectancy. *Science (New York, N.Y.), 296*(5570), 1029–1031. doi:10.1126/science.1069675

Ohberg, L., & Alfredson, H. (2004). Effects on neovascularisation behind the good results with eccentric training in chronic mid-portion Achilles tendinosis? *Knee Surgery, Sports Traumatology, Arthroscopy : Official Journal of the ESSKA*, *12*(5), 465–70. doi:10.1007/s00167-004-0494-8

Orchard, J., & Kountouris, A. (2011). The management of tennis elbow. *BMJ*, *342*. Retrieved from http://www.bmj.com/content/342/bmj.d2687.abstract

Owens, R. C., & Ambrose, P. G. (2005). Antimicrobial safety: focus on fluoroquinolones. *Clinical Infectious Diseases, 41*(Supplement 2), S144–S157.

Pagorek, S. (2009). Effects of Manual Mobilization with movement on Pain and Strength in Adults With Chronic Lateral Epicondylitis. *Journal of Sports Rehabilitation*, *18*(3), 448–457.

Paoloni, J. A, Milne, C., Orchard, J., & Hamilton, B. (2009). Non-steroidal anti-inflammatory drugs in sports medicine: guidelines for practical but sensible use. *British Journal of Sports Medicine, 43*(11), 863–5. doi:10.1136/bjsm.2009.059980

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

Poser, S. (2011). Leisure time and techonology. *History online*. Retrieved from ieg-ego.eu/en/threads

Patel, V. & Groen, G. (1986). Knowledge based solutions strategies in medical reasoning. Cognitive Science 10(1), 91-116.

Purdam, C. R., Jonsson, P., Alfredson, H., Lorentzon, R., Cook, J. L., & Khan, K. M. (2004). A pilot study of the eccentric decline squat in the management of painful chronic patellar tendinopathy. *British Journal of Sports Medicine, 38*(4), 395–7. doi:10.1136/bjsm.2003.000053

Rees, J. D., Stride, M., & Scott, A. (2013). Tendons - time to revisit inflammation. *British Journal of Sports Medicine*, 1–7. doi:10.1136/bjsports-2012-091957

Rees, J. D., Wilson, a M., & Wolman, R. L. (2006). Current concepts in the management of tendon disorders. *Rheumatology (Oxford, England), 45*(5), 508–21. doi:10.1093/rheumatology/kel046

Rees, J. D., Wolman, R. L., & Wilson, A. (2009). Eccentric exercises; why do they work, what are the problems and how can we improve them? *British Journal of Sports Medicine*, *43*(4), 242–6. doi:10.1136/bjsm.2008.052910

Riley, G. (2008). Tendinopathy--from basic science to treatment. *Nature Clinical Practice. Rheumatology*, *4*(2), 82–9. doi:10.1038/ncprheum0700

Riley, G., Cox, M., Harrall, R. L., Clements, S., & Hazleman, B. L. (2001). Inhibition of tendon cell proliferation and matrix glycosaminoglycan synthesis by non-steroidal anti-inflammatory drugs in vitro. *Journal of Hand Surgery (Edinburgh, Scotland), 26*(3), 224–8. doi:10.1054/jhsb.2001.0560

Rio, E., Moseley, L., Purdam, C., Samiric, T., Kidgell, D., Pearce, A. J., … Cook, J. (2013). The pain of tendinopathy: Physiological or pathophysiological? *Sports Medicine*, *44*(1), 9–23. doi:10.1007/s40279-013-0096-z

Ross, M. H., & Pawlina, W. (2006). *Histology*. Lippincott Williams & Wilkins, 2006. 148-166.

Sackett, D., Rosenberg, W., Gray, J., Haynes, R., & Richardson, W. (1996). Evidence based medicine: What it is and what it isn't. *Bmj, 312*(7023), 71-72.

Schulze-Tanzil, G., Al-Sadi, O., Wiegand, E., Ertel, W., Busch, C., Kohl, B., & Pufe, T. (2011). The role of pro-inflammatory and immunoregulatory cytokines in tendon healing and rupture: new insights. *Scandinavian Journal of Medicine & Science in Sports*, *21*(3), 337–351. doi:10.1111/j.1600-0838.2010.01265.x

Scott, A., Alfredson, H., & Forsgren, S. (2008). VGluT2 expression in painful Achilles and patellar tendinosis: evidence of local glutamate release by tenocytes. *Journal of Orthopaedic Research*, *26*(685-692).

Scott, A., Docking, S., Vicenzino, B., Alfredson, H., Murphy, R. J., Carr, A. J., … Danielson, P. (2013). Sports and exercise-related tendinopathies: a review of selected topical issues by participants of the second International Scientific Tendinopathy Symposium (ISTS) Vancouver 2012. *British Journal of Sports Medicine*, *47*(9), 536–44. doi:10.1136/bjsports-2013-092329

Scott, & Ashe, M. (2006). Common tendinopathies in the upper and lower extremities. *Current Sports Medicine Reports*, *5*(5), 233–41. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16934204

Shacklock, M. (1995). Neurodynamics. *Physiotherapy*, *81*, 9–16.

Shacklock, M. (2005). *Clinical Neurodynamics*. London: Elsevier Inc., 4-181.

Sharma, P., & Maffulli, N. (2008). Tendinopathy and tendon injury: the future. *Disability and Rehabilitation*, *30*(20-22), 1733–45. doi:10.1080/09638280701788274

Shrier, Matheson, Grodon, Kohl, & Harold. (1996). Are corticosteroid injection useful or harmful? *Clinic Journal of Sport Medicine*, *6*(4).

Silbernagel, K., Thomee, R., Thomee, P., & Karlsson, J. (2001). Eccentric overload training for patients with chronic Achilles tendon pain–a randomised controlled study with reliability testing of the evaluation methods.*Scandinavian journal of medicine & science in sports*, *11*(4), 197-206.

Smidt, N., van der Windt, D. A. W. M., Assendelft, W. J. J., Devillé, W. L. J. M., Korthals-de Bos, I. B. C., & Bouter, L. M. (2002). Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. *The Lancet*, *359*(9307), 657–662.

Sode, J., Obel, N., Hallas, J., & Lassen, A. (2007). Use of fluroquinolone and risk of Achilles tendon rupture: a population-based cohort study. *European Journal of Clinical Pharmacology*, *63*(5), 499–503.

Solomon, Fretzin, & Dewald. (1968). The epidermal nervous syndrom. *Archives of Dermatology*, *97*(3), 273–285.

Speicher, T., & Draper, D. (2006). Top 10 Positional-Release Therapy Techniques to Break the Chain of Pain: Part 1, *11*(5), 60–62.

Stanish, W., Curwin, S., & Rubinovich, M. (1985). Tendinitis: the analysis of treatment for running. *Clinics in Sports Medicine*, *4*(4), 593–609.

Svernlöv, B., Hultgren, E., & Adolfsson, L. (2012). Medial epicondylalgia (golfer's elbow) treated by eccentric exercise. *Shoulder & Elbow*, *4*(1), 50–55. doi:10.1111/j.1758-5740.2011.00152.x

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:10.3109/09593985.2012.702854

Tan, S. C., & Chan, O. (2008). Achilles and patellar tendinopathy: current understanding of pathophysiology and management. *Disability and Rehabilitation*, *30*(20-22), 1608–15. doi:10.1080/09638280701792268

Teys, P., Bisset, L., Collins, N., Coombes, B., & Vicenzino, B. (2013). One-week time course of the effects of Mulligan's Mobilisation with Movement and taping in painful shoulders. *Manual Therapy*, *18*(5), 372–7. doi:10.1016/j.math.2013.01.001

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-limited shoulders. *Manual Therapy*, *13*(1), 37–42. doi:10.1016/j.math.2006.07.011

Tosti, R., Jennings, J., & Sewards, J. M. (2013). Lateral epicondylitis of the elbow. *The American Journal of Medicine*, *126*(4), 357.e1–6. doi:10.1016/j.amjmed.2012.09.018

Vicenzino, B. (2003). Lateral epicondylalgia: a musculoskeletal physiotherapy perspective. *Manual Therapy*, *8*(2), 66–79. doi:10.1016/S1356-689X(02)00157-1

Vicenzino, B., Paungmali, a, Buratowski, S., & Wright, A. (2001). Specific manipulative therapy treatment for chronic lateral epicondylalgia produces uniquely characteristic hypoalgesia. *Manual Therapy*, *6*(4), 205–12. doi:10.1054/math.2001.0411

Villafañe, J. H., Pillastrini, P., & Borboni, A. (2013). Manual therapy and neurodynamic mobilization in a patient with peroneal nerve paralysis: A case report. *Journal of Chiropractic Medicine*, *12*(3), 176–181. doi:10.1016/j.jcm.2013.10.007

Wainwright, S.F., Shepard, K.F., Harman, L.B., & Stephens, J. (2010). Novice and experienced physical therapist clinicians: a comparison of how reflection is used to inform the clinical decision-making process. *Phys Ther 90*(1), 75-88.

Willis, J., Inman, D., & Valenti, R. (2010). *Completing a professional practice dissertation: A guide for doctoral students and faculty.* Charlotte, NC: Age Publishing, Inc.

Wang, C. J., Wang, F. S., Yang, K. D., Weng, L. H., Hsu, C. C., Huang, C. S., & Yang, L. C. (2003). Shock wave therapy induces neovascularization at the tendon-bone junction. A study in rabbits. *Journal of Orthopaedic Research : Official Publication of the Orthopaedic Research Society*, *21*(6), 984–9. doi:10.1016/S0736-0266(03)00104-9

Wang, J. (2005). Mechanobiology of tendon. *Journal of Biomechanics*. 1563-1582.

Wang, J. H.-C., Iosifidis, M. I., & Fu, F. H. (2006). Biomechanical basis for tendinopathy. *Clinical Orthopaedics and Related Research*, *443*, 320–332. doi:10.1097/01.blo.0000195927.81845.46

Wright, I. (2002). Is that right? Critical thinking and the social world of the young learner. Ontario, Canada: Pippin Publishing Corporation.

Yang, G., Crawford, R. C., & Wang, J. H. (2004). Proliferation and collagen production of human patellar tendon fibroblasts in response to cyclic uniaxial stretching in serum-free conditions. *Journal of biomechanics*, *37*(10), 1543-1550.

Young, M. a, Cook, J. L., Purdam, C. R., Kiss, Z. S., & Alfredson, H. (2005). Eccentric decline squat protocol offers superior results at 12 months compared with traditional eccentric protocol for patellar tendinopathy in volleyball players. *British Journal of Sports Medicine*, *39*(2), 102–5. doi:10.1136/bjsm.2003.010587

Yu, J. G., Liu, J. X., Carlsson, L., Thornell, L. E., & Stal, P. S. (2013). Re-evaluation of sarcolemma injury and muscle swelling in human skeletal muscles after eccentric exercise. *PLoS One, 8*(4), e62056.

CHAPTER 5: APPLIED CLINICAL RESEARCH

An Exploratory Analysis of a Treatment-Based Classification Algorithm to Treat Patellar Tendinopathy: A Case Series

Authors: Monica Matocha, Emily Dietz, Janet McMurray, Patti Syvertson

Submitted for consideration to the International Journal Sports Physical Therapy

5.1 Patellar Tendon

Abstract.

Background and Purpose. The general and athletic populations commonly experience patellar tendon pain. The current gold standard treatment for patellar tendinopathy is a 12 week eccentric exercise protocol. The present research study was designed to determine the effects of a treatment based classification (TBC) algorithm utilizing indirect treatment techniques in patellar tendinopathy participants.

Case Description. Ten participants were evaluated and included in this study. Each participant underwent a thorough evaluation process which included: participant history, range of motion measurements, orthopedic tests, a scan for tender points, neurodynamic tests, and a local Mulligan Concept technique to determine diagnosis, study inclusion, and treatment classification. Outcome measures were collected to establish baseline scores and assess participant improvement. The measures included: the Disablement in the Physically Active Scale (DPA Scale), Numerical Rating Scale (NRS), Victorian Institute of Sports Assessment for the Patellar Tendon (VISA-P), Global Rating of Change (GRC), Nirschl Phase Rating Scale, and Blazina Knee Scale.

Outcomes. Paired *t*-tests, with a 95% confidence intervals, were analyzed on NRS, DPA Scale, and VISA-P to determine the effectiveness of all treatment algorithm from

initial exam to discharge. Cohen's *d* was also computed to determine the effect size of each of the aforementioned outcome measures. Descriptive statistics were computed for the GRC at discharge. The mean change for the NRS (M = 4.7, 95% CI[3.57 to 5.82], p < .001), DPA Scale (M = 21.8, 95% CI[12.43 to 31.16], *p* = .001), and VISA-P (M = 22.70, 95% CI[33.71 to 11.68], *p* < *.001*) were statistically significant. The mean for the GRC (M = 5.3) was clinically meaningful. All of the participants (100%) met discharge criteria.

Discussion. The results of this case series demonstrated an increase in function and decrease in pain for participants with patellar tendinopathy within three office visits when utilizing a TBC algorithm.

Background and Purpose. Tendon related pathologies comprise of 30 to 45% of sport related injuries[1] and frequently cause impairment in the general population.[2] Patellar tendinopathy accounts for 7 to 40% of tendon related pathologies in sport.[3] Patellar tendinopathy is characterized clinically by tendon pain, tendon dysfunction,[4,5] decreased performance in association with tendon swelling, morning stiffness,[4,6,7] palpable crepitus,[3,6,7] and localized swelling.[4] Pain over a tendon is the key clinical diagnostic criteria used by clinicians to diagnose tendinopathy.[8] The use of advanced diagnostic imaging/testing (e.g., diagnostic ultrasound) is not common clinically, but is necessary to determine the exact physical state of the tendon.[5]

Though the clinical exam is the accepted standard for tendinopathy diagnosis, varying patient presentations and injury states make it difficult to identify the origin of tendon pain.[9] Previously, tendon pain was thought to be a mechanical overuse injury, which caused inflammation in the tendon, and was classified as a *tendinitis.*[10] Due to a lack of inflammatory markers being present during histological tests, the term *tendinopathy* has

generally become the preferred diagnostic term for tendon pain,[2,11] while *tendinosis* is utilized for a degenerative tendon diagnosed using diagnostic imaging.[2] As tendon pathology research has elucidated other causative factors for the presentation of tendon pain (e.g., mechanical, neural, vascular),[10] other researchers have proposed the use of the terms *reactive tendinopathy*[12] and *tendinalgia*[13] when classifying a patient with tendon pain. The use of the term tendinalgia would allow clinicians to acknowledge the patient complaint of pain at the site of a tendon without predetermining a state of tissue pathology.[13]

The risk of using terminology focused on a specific causative factor is that it may lead to treatments that are not optimal for a specific patient or situation.[9] Due to the previous acceptance of an inflammatory condition being present when diagnosed as tendinitis, most interventions have been aimed at treating the inflammatory process; however, most of these strategies do not produce effective long-term results (i.e., improvement past six weeks).[14-16] Commonly used conservative treatments for patellar tendinopathy include: rest, nonsteroidal anti-inflammatory drugs (NSAIDs), stretching, eccentric exercises, and corticosteroid injections.[2,7,17] The current treatment gold standard is the use of eccentric exercises. The Alfredson et al. protocol[18-24] has become the foundation of most eccentric exercise protocols with participants performing the exercises two times a day, seven days a week, for 12 weeks. For many patients, however, compliance is difficult due to the length of the treatment, muscle soreness, and/or the pain experienced with treatment.[18,23,25] Other concerns with the protocol, such as tendon rupture rates, are not well understood as researchers do not always report treatment complications. Upon return to activity, participants who complete the protocol also report a high recurrence rate.[15,26]

Another treatment option is to utilize manual therapies theorized to address the different causative factors of tendon pain; however, few research studies have been conducted to assess the effectiveness of manual therapy for the treatment of patellar tendinopathy. While there are a variety of manual therapy options that have been proposed to treat this disorder, clinicians could theoretically address the causative factors by applying the Mulligan Concept (MC), Positional Release Therapy (PRT), and/or neurodynamics in these cases. The MC techniques for knee dysfunction are based on applying a pain free glide (mobilization) to the joint while the patient actively moves into a position that was painful prior to the glide being applied.[27] Positional Release Therapy (PRT) is theorized to restore the muscle or tendon to normal function by increasing oxygen and decreasing inflammatory metabolites.[28] Neurodynamics is the movement of the nervous system on other body structures to determine if a sliding or tension dysfunction is present in the peripheral nervous system.[29] The use of these techniques in isolation, or combination, might better target the individual differences in patient presentation.

The use of manual therapies and tendon classification have been proposed as a means to improve the treatment of tendon pain[12,13] due to the high rate of tendon pathology recurrence[15,26] and patient non-compliance.[23,25] Researchers have proposed that many patients classified with tendinopathy may not actually have a true tissue pathology that must be addressed with tissue remodeling[13] and that classifying patients based on their response to sub-therapeutic doses of intervention techniques may improve patient outcomes.[9,13] Thus, it is important for clinicians to consider alternative examination and treatment strategies to better identify and treat these patients. The purpose of this study was to determine if a novel treatment based classification (TBC) algorithm could be used to classify tendon pain

participants and what the effects of using the algorithm would be in participants diagnosed with patellar tendinopathy.

Procedures.

Case Description. A convenience sample of participants diagnosed with patellar tendinopathy at four clinical sites across the United States of America participated in the study. The University of Idaho Institutional Review Board approved the research project. All participants signed an informed consent form; if the participant was under the age of 18 years old, the legal guardian signed the informed consent and assent was provided by the minor. During the evaluation period, a total of 10 participants (seven females, three males, mean age = 19.6 ± 1.07, mean symptom duration = 2.14 years with a range of one week to six years) presented for possible inclusion in the study. All of the potential participants were diagnosed with patellar tendinopathy according to the inclusion criteria, agreed to participate in the study, and completed the study through discharge. All participants reported with patellar tendon pain, increased pain and stiffness in the morning and after sitting for long periods of time with a decrease in symptoms after warm up of physical activity.

Examination. Each participant was examined using a pre-determined clinical evaluation to ensure consistency in patellar tendinopathy diagnosis and classification with the Treatment Based Classification (TBC) Algorithm. Inclusion criteria included: tendon pain before, during, or after patella loading activities; point tenderness over the patellar tendon upon palpation; pain near patella origin; impaired function; and tendon focal or generalized swelling. Exclusion criteria included: cortisone injection (<six weeks), fluoroquinolones ciprofloxacin use (<12 months), post-operative participants unable to perform the treatment (<eight weeks), wore orthotics, currently healing or suspected

fractures, or receiving physical therapy for the tendon of concern. Participants who met the inclusion criteria then completed a thorough history, range of motion (ROM), and special test examination. Special tests performed included: Clark's sign, patellar grind, patellar compression, prone knee bend, slump, a quarter screen for tender points, and the application of the Mulligan Concept technique for the knee (an internal rotation glide followed by an external rotation glide if pain was not resolved during application). Clark's sign, patellar grind, and patellar compression tests were performed to rule out patellar dysfunction as the source of pain. The prone knee bend and slump tests were performed to rule in neurological tension and sliding dysfunctions. The quarter screen was performed to determine the presence of tender points; while the Mulligan Concept Technique was performed last to determine classification into the Mulligan Concept treatment.

Treatment-Based Classification Algorithm. The TBC algorithm consisted of a MC technique, PRT, neurodynamics, and eccentric exercise. If the participant reported a resolution of his or her symptoms when the MC technique was applied during the exam, then the participant was classified as being a responder to the MC treatment. If the application of the MC did not resolve symptoms during the exam and the participant presented with tender points (TP), which could be reduced by moving the participant into a position of comfort (POC), then the participant was classified as being a responder to the PRT treatment. If the application of the MC did not resolve symptoms and a POC could not be identified with PRT, the participant would be classified into the neurodynamic treatment if a positive neurodynamic test was found during the initial exam. In the case where the participant could not be classified into the MC, PRT, or neurodynamic group, the participant was classified into the eccentric exercise treatment protocol (Figure 5.1).

Once the clinician determined the appropriate treatment classification, the participant underwent three treatments within 10 days. The participant was re-assessed to determine if discharge criteria had been met at the conclusion of the third visit. Discharge criteria included: phase 1 on the Nirschl Phase Rating Scale, phase 1 on the Blazina Knee Scale, and met MCID for Global Rating of Change (GRC), and Disablement of the Physically Active Scale (DPA Scale). Additionally, participants had to report a worst pain score equal to or less than two out of ten on the Numerical Rating Scale (NRS) during the discharge evaluation. If the participant was not discharged, a re-evaluation using the TBC algorithm was conducted to determine the participant's treatment classification for the next three visits. The participant was only able to be re-classified into the initial treatment classification if the participant demonstrated enough improvement to meet 50% of each discharge outcome criteria; if not, the participant was classified into the eccentric exercise treatment. Following discharge, each participant was sent a one month follow up survey to re-assess participant progress. The NRS was the only outcome measure reported during the administration of the survey. If the participant reported a zero out of ten on the NRS, the survey was complete and no additional information was requested.

Outcome Measures. Disablement and global participant outcome measures were utilized in this study to determine participant perceptions of their condition and recovery. The six outcome measures utilized in this study were the: NRS, GRC, DPA Scale, VISA-P, Nirschl Phase Rating Scale, and the Blazina Knee Scale.

The NRS is a rating scale a clinician can utilize to determine a participant's perception of his or her pain from zero, no pain, to ten, worst pain imaginable.[30] Each participant of this case series was asked to rate his or her pain at best, worst, and rest before

and after each treatment. The recorded NRS scores represent the participant's reported worst pain. The participant was also asked to rate his or her pain while the clinician performed a quarter screen for tender points (TPs). The GRC was utilized to determine participant's perception of his or her improvement or deterioration over time.[31] The GRC was reported at every third visit for each participant. The minimal clinical important difference (MCID) has been established at two points for both the NRS[32] and GRC.[31]

The DPA Scale was developed to determine the participant's perception of how his or her injury has effected disablement.[33] A participant reported his or her perception on a scale of one, no problem, to five, severe, on 16 questions across multiple domains: pain, motion, muscular function, stability, changing directions, daily actions, maintaining positions, skill performance, overall fitness, participation in activities, and well-being. The rating for each item on the scale is summed and 16 points are subtracted to produce a final score that ranges from zero to 64 points. The DPA Scale was administered upon the first visit, third visit, and every third visit after until discharged. The MCID has been established for the DPA Scale as nine points for acute injuries and six points for chronic injuries.[33] The healthy range for the DPA Scale has been established to be between zero and 34 points.[33]

The VISA-P was created to determine functional impairment in a participant with patellar tendon pain.[34] The participant recorded responses to questions regarding his or her function on a numerical scale from zero, unable to perform, to ten, fully functional. All responses were then summed and recorded on a scale from zero, no function, to 100, fully functional. Each participant recorded VISA-P score upon the first visit, third visit, and discharge visit. Currently, a MCID has not been established for VISA-P.

The Nirschl Phase Rating Scale and Blazina Knee Scale were both developed to help classify participant symptoms. The Nirschl Phase Rating Scale was created for all tendon pain participants,[2,35] whereas the Blazina Knee Scale was created to determine dysfunction specifically for participants with patellar tendon pain.[36] All participants reported his or her symptoms in accordance to both scales upon the first visit, third visit, and discharge visit. Currently, the Nirschl Phase Rating Scale, and Blazina Knee Scale do not have an established method for evaluating patient improvement on the scales.

Intervention. If the participant was classified into the MC treatment subgroup, the participant was treated with the Mobilization with Movement (MWM) (internal or external rotation glide) to resolve the participant's pain complaint. The internal or external rotation glide was applied by having the participant perform a movement that exacerbated the chief complaint. Once the painful movement was established, the clinician gently placed her hands just below the tibiofemoral joint line, around the tibia and fibula and applied the appropriate glide to the tibia in association to the femur (Figure 5.2). Simultaneously, the participant performed the previously established painful movement. The MWM was performed through three sets of 10 repetitions of pain-free movement.

If the participant was classified into the PRT treatment subgroup, the dominant TP was monitored while the participant was passively moved into a POC. The POC was defined as a position resulting in the resolution of pain (zero out of 10 on the NRS) during palpation of the TP. If a POC was achieved, the participant received PRT for the dominant TP only. The dominant TP was treated while the clinician maintained the POC (Figure 5.3). The POC was held for a minimum of 30 seconds, and a maximum of 90 seconds. The participant was then returned to the normal anatomical position while the clinician continued monitoring the

TP. The TP was reassessed by determining pain to palpation (using the NRS) in the normal anatomical position. If the participant still reported tenderness to palpation of the TP after one set of treatment, the clinician repeated the treatment; if the patient reported resolution of pain to palpation, the treatment was concluded for that session. A patient could receive a maximum of three treatment sets per visit.

If the participant was classified into the neurodynamic treatment subgroup, the participant was instructed on the proper technique to perform a general neural slider in the slump position (Figure 5.4). As the participant released tension at the head (head moved from cervical flexion to neutral), tension was increased at the ankle (foot moved from plantarflexion to dorsiflexion). Each participant completed three sets of 10 repetitions, through a slow and controlled movement.

If the participant was classified into EE treatment subgroup, the participant completed a monitored EE protocol two times a day, seven days a week for 12 weeks. Participants completed one set of 15 repetitions of a single leg squat on a 25 degree decline board for each session. The participant was instructed to keep the trunk in the upright position, slowly flexing the knee to 90 degrees and returning to the starting position with the uninjured leg. The participant was then instructed to squat into pain during the eccentric portion (knee flexion). When the participant's pain decreased to two of ten on the NRS while performing EE, an external load was added.

Statistical Analysis. All data was analyzed using SPSS version 23.0 (SPSS Inc., Chicago, IL, USA). Paired *t*-tests were performed on the NRS, DPA Scale, and VISA-P to determine the effects of classifying and treating participants with this novel TBC algorithm for patellar tendinopathy. Mean differences from the initial visit scores and 95% confidence

intervals (CIs) were calculated for the NRS, DPA Scale, and VISA-P for discharge. Cohen's *d* was also computed to determine the effect size, or maximum likelihood, of each of the aforementioned outcome measures. For Cohen's *d* an effect size of 0.2 to 0.3 was considered a "small" effect, around 0.5 a "medium" effect and 0.8 to infinity, a "large" effect.[37] Descriptive statistics were performed on the GRC scores reported at discharge.

Outcomes. During the initial examination, all participants were classified into a manual therapy treatment sub-group (Mulligan Concept = nine, PRT = one) (Figure 5.1). All participants were successfully treated through discharge with the initial treatment classification and no participants met the criteria for classification into the EE subgroup at any point of time during treatment. The number of treatments each participant received was three over a mean of 4.8 ±1.4 days to discharge (Table 5.1).

Numerical Rating Scale. The use of the TBC algorithm resulted in a significant mean change in pain from initial visit to discharge, M = 4.7 ± 1.64 (95% CI[3.57 to 5.82], $p < .001$) with a large effect size (Cohen's $d = 2.41$) (Table 5.1a). The mean difference in pain scores from initial visit to discharge, as well as the lower boundary CI, exceeded the MCID of "much better" for the NRS.[26] The mean change was accomplished in just three visits over 4.8 ±1.4 days. At discharge, 60% of participants (6/10) reported a complete resolution of their pain. The remaining 40% of participants (4/10) reported their "worst" pain as a one (20%; 2/10) or two (20%; 1/10) on the NRS. One month post discharge data demonstrated that all participants who completed the follow-up survey (n = 2) continued to experience a resolution of pain with full return to activity.

Disablement in the Physically Active Scale. Statistically significant changes on the DPA Scale from initial evaluation to discharge were recorded M = 21.8 ± 12.3130, (95%

CI[12.43 to 31.16], $p = .001$), with a large effect size (Cohen's $d = 1.98$) when using the TBC algorithm (Table 5.1). The mean change from initial visit to discharge, as well as the lower boundary of the CI, exceeded the MCID for acute conditions, a reduction of nine points or greater, which is greater than the MCID for chronic conditions (six points).[33] All of the participants (100%) met MCID for both acute and chronic conditions prior to discharge, as well as being discharged within the healthy range (zero to 34 points).[33] Published data for DPA Scale scores for return to activity for chronic conditions does not exist; however, the reported mean for participants who returned from acute injury is M = 8.82 ± 6.71 (R = 0 – 23 points). All of the participants (100%) in this case series were discharged below the reported mean score for returning to activity after an acute injury (M = 8.5 ± 9.11; R = 0 - 22). Consequently, participants in this case series perceived less disablement than has been reported in the previous literature on the DPA Scale.[33]

Victorian Institute Sport Assessment – Patella. The use of the TBC algorithm resulted in a significant increase in scores on the Victorian Institute of Sport Assessment-Patella outcome measure from initial exam to discharge (M = 22.70 ± 16.07, 95% CI[33.71 to 11.68], $p < .001$), with a large effect size (Cohen's $d = 1.37$) (Table 5.1). Of greater clinical importance, 80% (8/10) of the participants reported a VISA-P score for "completely recovered" within three days of initiating treatment.

Global Rating of Change. A clinically meaningful increase on GRC scores from initial visit to discharge was reported (M= 5.7 ± 2.11) (Table 5.1). The GRC scale ranges from -7 (a very great deal worse) to +7 (a very great deal better).[31] All (100%) of the participants exceeded a MCID for the GRC scale (≥ 2) upon discharge.[31] More clinically relevant, 50% (5/10) of participants reported a +7 (a very great deal better), 10% (1/10)

reported a +6 (a great deal better), and 40% (4/10) reported a +4 (moderately better) at discharge.[31]

Nirschl Phase Rating Scale. During initial evaluations, 30% (3/10) of participants reported a phase three on the Nirschl Phase Rating Scale meaning "pain that is present during activity without causing activity modification", 40% (4/10) reported a phase five "pain that is present during all activities and occurs with activities of daily living", and 30% (3/10) reported a phase six "intermittent rest pain that does not disturb sleep".[35] All participants (100%, 10/10) reported a phase one ("mild stiffness or soreness after activity with resolution of symptoms within 24 hours") on the Nirschl Phase Rating Scale prior to discharge. More clinically relevant, 60% (6/10) of the participants did not feel a phase one rating on the Nirschl Phase Rating Scale was applicable due to their experience of full resolution of symptoms.

Blazina Knee Scale. During the initial evaluation, 50% (5/10) of participants reported a phase two on the Blazina Knee Scale "pain/discomfort during and after activity with the subject still able to perform at a satisfactory level (does not interfere with participation)";[36] while the other 50% (5/10) of participants reported a phase three "pain during and after activity with more prolonged, with subject having progressively increasing difficulty in performing at a satisfactory level (interferes with competition)".[36] All of the participants (100%) reported a phase one on the Blazina Knee Scale prior to discharge ("pain after activity only"). More clinically relevant, 60% (6/10) of participants did not feel a phase one rating on the Blazina Knee Scale was applicable due to their experience of full resolution of symptoms.

Discussion. Currently, eccentric exercise is the gold standard treatment for patellar tendinopathy. Several researchers have demonstrated positive results with the use of a 12 week protocol.[18-21,23-25,37,38] Jonsson and Alfredson[39] compared an eccentric exercise group to a concentric exercise group for the treatment of "jumper's knee" and reported nine out of 10 participants were "satisfied" and discharged with a mean Visual Analogue Scale (VAS) of 23 out of 100 and a VISA-P score of 83 points with the use of a 12 week eccentric exercise protocol. Similarly, Purdam et al.[24] reported a mean VAS score of 28.5 points at discharge for participants who performed eccentric exercises on a decline board, compared to a mean VAS score of 72 points at discharge for participants who performed traditional squat eccentric exercises for 12 weeks. In these studies, however, not all participants reported being "satisfied" at discharge (10%,[39] 25%[24]). The participants, who did report being "satisfied" did not, on average, experience a full resolution of pain at discharge after 12 weeks of therapy.[24,39]

Although EE has been found to produce beneficial results when the protocol is completed, there are still concerns over the effectiveness of the protocols for all patients and a lack of a clear understanding of the mechanism of action. Thus, there is a need to determine if tendinopathy participants should be screened prior to using an EE protocol in a one-size fits all model.[8,9] The lack of a screening process for identifying patients likely to respond to EE and the extended time required for patients to become symptom free has created a need for improved assessment methods.[8,9] One potential solution to improve tendinopathy outcomes is the use of a TBC system or more novel manual therapy techniques. Lewis[9] has suggested a series of four mechanical techniques, or a combination of interventions, to be used as a TBC system to produce improved patient classification and

treatment outcomes for patients with rotator cuff tendinopathy. The manual therapies used in the TBC algorithm in this study also have evidence of effectiveness in tendinopathy patients reported by researchers in other studies.

Researchers have found promising results with the use of the MC when treating lateral epicondylalgia.[40-42] Bisset et al.[42] observed favorable outcomes for the use of MC mobilizations in combination with exercises over corticosteroids and a wait and see method. Although corticosteroid injections were statistically better than either of the other groups initially, 72% (47/65) of the corticosteroid injection group had a higher recurrence rate. The mobilization and exercise group had superior results to both the wait and see and the corticosteroid injection groups at six and 52 week follow-up.[42] While few studies have been conducted on the effectiveness of PRT or neurodynamics in treating tendinopathy, Baker et al.[13] were able to re-establish normal, pain free function in a patient with a history of bicep tendinopathy when using PRT, while Matocha et al.[43] were able to alleviate pain and restore function in a patient diagnosed with lateral epicondylalgia using neural sliders and tensioners.

Thus, the TBC algorithm used in this study was designed because the clinicians could observe participant response to potential interventions while in a painful state and to utilize manual therapy techniques that could potentially produce rapid changes. Patient response enabled the clinician to classify the participant to an intervention that was designed to be matched to their dysfunction. In theory, matching tendinopathy patients to therapies through classification could improve outcomes. In this study, all of the participants were classified as being a responder to either the MC or PRT and were able to meet the pre-established discharge criteria without a single participant needing to be classified into the EE

protocol sub-group at any time. The preliminary outcomes associated with the use of the TBC algorithm can be viewed as similar, or potentially superior in certain cases, to the EE protocol outcomes found in the literature.

For example, an NRS score of two out of 10 was utilized as discharge criteria because participants are often deemed to have successful outcomes at the conclusion of tendinopathy studies examining EE protocols with a reported pain equal to or greater than a NRS score of two out of 10 in the literature.[24,39,44] In the current study, utilizing the TBC algorithm allowed participants to be discharged with a lower mean NRS (M = .6 points) compared to mean VAS scores of 23 points[39] to 28.5 points[24] in fewer visits over less time than the previously discussed EE protocol studies. Similarly, the mean change in the VISA-P for the current study (M = 22.70 ± 16.07, $p < .001$) was statistically significant from initial visit (M = 53.6 ± 16.58) to discharge (M = 76.3 ± 18.36), as were the changes in the Jonsson and Alfredson[39] study (initial mean M = 41.1 ± 17.9 to discharge mean M = 83.3 ± 23.4; mean change M = 42.2). While significant changes were found in both studies, the changes in VISA-P score in the current study were achieved in less time. All participants were discharged at a faster rate (three visits, within 4.8 ± 1.4 days) when compared to participants that completed a 12 week EE protocol (168 treatments over 84 days).[24,39] Thus, a TBC algorithm may be more effective at matching participants to appropriate treatments that do not require extended therapy or a painful experience to produce effective outcomes. Additionally, the use of a TBC algorithm may allow clinicians to identify which participants actually need to participate in an EE protocol or when to add this protocol as an adjunct therapy to provide complete resolution of participant complaints.

While the preliminary results of this case series are important, the limitations of this study must also be noted. Although clinicians made attempts to decrease the influence of bias, a bias could have been created because the clinician and participants were not blinded to the treatment or collection of outcome measures. Additionally, a control or placebo group was not used in the study. The lack of control group and long term follow-up made it difficult to definitively determine if the outcomes were the result of treatment or the natural course of healing; however, a number of participants had chronic symptoms unlikely to have spontaneously healed over the treatment period. The lack of comparison group made it difficult to determine if one treatment intervention was superior to another within the TBC algorithm, but the purpose of the study was not to identify the "best" intervention. Instead, the focus was on determining the effectiveness of classifying patients using sub-therapeutic doses on indirect manual therapies. Additionally, it could be argued the treatments provided as part of this TBC algorithm were provided at sub-therapeutic doses (e.g., not treating multiple TPs with PRT, etc.) and the interventions could be more effective if treatment dose was maximized. There are also several MC techniques available for use in the treatment of patella tendon pain and recommendations to alter force and volume of application to sustain the effects of the treatment; as only one technique was used in the current study, it is possible that fully utilizing the Mulligan Concept could further improve patient response. Finally, a sample of participants may not have fully represented patellar tendinopathy patients and those who volunteered may have been motivated to improve.

As this study is an initial examination of a TBC algorithm for patellar tendon pain, it is possible that altering the order or adding other treatment paradigms may be appropriate to maximize the effectiveness of the TBC algorithm. The results of this study do provide

support for the utilization of a TBC algorithm for patellar tendinopathy patients because all 10 participants experienced statistically and clinically significant improvements in pain and function in three visits. Future research should compare this TBC algorithm with a control or placebo group and utilize long-term follow-up with the participants. Forthcoming research should also include diagnostic imaging or histological exams, which would benefit the understanding of the physiological changes in the tendon following treatment utilizing the TBC algorithm.

References

1. Murtaugh, B., Ihm J. Eccentric Training for the Treatment of Tendinopathies. *Curr Sports Med Rep*. 2013;12(3):175–182.

2. Kaux J-F, Forthomme B, Goff C Le, Crielaard J-M, Croisier J-L. Current Opinions on Tendinopathy. *J Sports Sci Med*. 2011;10(2):238–253.

3. Fredberg U, Stengaard-Pedersen K. Chronic tendinopathy tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. *Scand J Med Sci Sports*. 2008;18(1):3–15. doi:10.1111/j.1600-0838.2007.00746.x.

4. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common tendinopathies. Update and implications for clinical management. *Sports Med*. 1999;27(6):393–408. Available at: http://www.ncbi.nlm.nih.gov/pubmed/10418074.

5. Rio E, Moseley L, Purdam C, et al. The pain of tendinopathy: Physiological or pathophysiological? *Sport Med*. 2013;44(1):9–23. doi:10.1007/s40279-013-0096-z.

6. Alfredson H, Lorentzon R. Chronic tendon pain: no signs of chemical inflammation but high concentrations of the neurotransmitter glutamate. Implications for treatment? *Curr Drug Targets*. 2002;3(1):43–54.Available at:http://www.ncbi.nlm.nih.gov/pubmed/11899264.

7. Alfredson H. The chronic painful Achilles and patellar tendon: research on basic biology and treatment. *Scand J Med Sci Sports*. 2005;15(4):252–9. doi:10.1111/j.1600-0838.2005.00466.x.

8. Scott A, Docking S, Vicenzino B, et al. Sports and exercise-related tendinopathies: a review of selected topical issues by participants of the second International Scientific Tendinopathy Symposium (ISTS) Vancouver 2012. *Br J Sports Med*. 2013;47(9):536–44. doi:10.1136/bjsports-2013-092329.

9. Lewis J. Rotator cuff tendinopathy/subacromial impingement syndrome: Is it time for a new method of assessment? *Br J Sport Med*. 2008. Available at: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=18838403\n(null).

10. Rees JD, Wilson a M, Wolman RL. Current concepts in the management of tendon disorders. *Rheumatology (Oxford)*. 2006;45(5):508–21. doi:10.1093/rheumatology/kel046.

11. Mayor RB. Treatment of athletic tendonopathy. *Conn Med*. 2012;76(8):471–5.

12. Cook JL, Purdam CR. Is tendon pathology a continuum ? A pathology model to explain the clinical presentation of load-induced tendinopathy. *Br J Sports Med*. 2008;43:409–416. doi:10.1136/bjsm.2008.051193.

13. Baker RT, Riper MV, Nasypany AM, Seegmiller JG. Evaluation and Treatment of Apparent Reactive Tendinopathy of the Biceps Brachii. *Int J Athl Ther Train*. 2014;19(July):14–21.

14. Hay EM, Paterson SM, Lewis M, et al. Pragmatic Randomised Controlled Trial of Local Corticosteroid Injection and Naproxen for Treatment of Lateral Epicondylitis of Elbow in Primary Care Committee (JISC) in the UK . This content is also freely available on PubMed Central. controlled trial. 1999;319(7215):964–968.

15. Smidt N, van der Windt D a WM, Assendelft WJJ, Devillé WLJM, Korthals-de Bos IBC, Bouter LM. Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. *Lancet*. 2002;359(9307):657–62. doi:10.1016/S0140-6736(02)07811-X.

16. Andres BM, Murrell G a C. Treatment of tendinopathy: what works, what does not, and what is on the horizon. *Clin Orthop Relat Res*. 2008;466(7):1539–54. doi:10.1007/s11999-008-0260-1.

17. Glaser T, Poddar S, Tweed B, Webb CW. Clinical inquiries. What's the best way to treat Achilles tendonopathy? *J Fam Pract*. 2008;57(4):261–263.

18. Alfredson H, Pietilä T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med*. 1998;26(3):360–6. Available at: http://www.ncbi.nlm.nih.gov/pubmed/9617396.

19. Mafi N, Lorentzon R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. *Knee Surgery, Sport Traumatol Arthrosc*. 2001;9(1):42–47. doi:10.1007/s001670000148.

20. Fahlström M, Jonsson P, Lorentzon R, Alfredson H. Chronic Achilles tendon pain treated with eccentric calf-muscle training. *Knee Surg Sports Traumatol Arthrosc*. 2003;11(5):327–33. doi:10.1007/s00167-003-0418-z.

21. Jonsson P, Alfredson H, Sunding K, Fahlström M, Cook J. New regimen for eccentric calf-muscle training in patients with chronic insertional Achilles tendinopathy:

results of a pilot study. *Br J Sports Med*. 2008;42(9):746–9. doi:10.1136/bjsm.2007.039545.

22. Morrissey D, Roskilly A, Twycross-Lewis R, et al. The effect of eccentric and concentric calf muscle training on Achilles tendon stiffness. *Clin Rehabil*. 2011;25(3):238–47. doi:10.1177/0269215510382600.

23. Dimitrios S, Pantelis M, Kalliopi S. Comparing the effects of eccentric training with eccentric training and static stretching exercises in the treatment of patellar tendinopathy. A controlled clinical trial. *Clin Rehabil*. 2011;26(5):423–30. doi:10.1177/0269215511411114.

24. Purdam CR, Jonsson P, Alfredson H, Lorentzon R, Cook JL, Khan KM. A pilot study of the eccentric decline squat in the management of painful chronic patellar tendinopathy. *Br J Sports Med*. 2004;38(4):395–7. doi:10.1136/bjsm.2003.000053.

25. Svernlöv B, Hultgren E, Adolfsson L. Medial epicondylalgia (golfer's elbow) treated by eccentric exercise. *Shoulder Elb*. 2012;4(1):50–55. doi:10.1111/j.1758-5740.2011.00152.x.

26. Bisset L, Beller E, Jull G, Brooks P, Darnell R, Vicenzino B. Mobilisation with movement and exercise, corticosteroid injection, or wait and see for tennis elbow: randomised trial. *Br Med J*. 2006;333(7575):939–941.

27. Mulligan BR. *Manual Therapy NAGS, SNGAS, MWM etc.* 6th ed. Wellington, New Zealand: Plane View Services Ltd; 2010.

28. D'Ambrogio K, Roth G. Positional Release Therapy, Assesment & Treatment of Musculoskeletal Dysfunction. St. Louis: Mosby; 1997.

29. Shacklock M. *Clinical Neurodynamics*. London: Elsevier Inc.; 2005. Available at: http://www.elsevierhealth.co.uk/physiotherapy/clinical-neurodynamics-paperback/9780750654562/\nhttp://www.amazon.co.uk/Clinical-Neurodynamics-System-Neuromusculoskeletal-Treatment/dp/0750654562.

30. McCaffery M, Beebe A. *Pain: Clinical Manual for Nursing Practice.* St. Louis, MO: C. V. Mosby; 1989.

31. Kamper S, Maher C, MacKay G. Global Rating of Change scales. *Aust J Physiother*. 2009;55(4):289. doi:10.1016/S0004-9514(09)70015-7.

32. Farrar JT, Young JPJ, LaMoreaux L, Werth JL, Poole RM. Clinical importance of changes in chronic pain intensity measured on an 11-point numerical pain rating scale. *Pain*. 2001;94(2):149–158.

33. 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):630–41. doi:10.4085/1062-6050-45.6.630.

34. Tanner SM, Dainty KN, Marx RG, Kirkley A. Knee-specific quality-of-life instruments: which ones measure symptoms and disabilities most important to patients? *Am J Sports Med*. 2007;35(9):1450–1458. doi:10.1177/0363546507301883.

35. Nirschl RP, Ashman ES. Elbow tendinopathy: tennis elbow. *Clin Sports Med*. 2003;22(4):813–836. doi:10.1016/S0278-5919(03)00051-6.

36. Blazina ME, Kerlan RK, Jobe FW, Carter VS, Carlson GJ. Jumpers Knee. *Orthop Clin North Am*. 1973;4(3):665–678.

37. Ohberg L, Alfredson H. Effects on neovascularisation behind the good results with eccentric training in chronic mid-portion Achilles tendinosis? *Knee Surg Sports Traumatol Arthrosc*. 2004;12(5):465–70. doi:10.1007/s00167-004-0494-8.

38. Cannell LJ, Taunton JE, Clement DB, Smith C, Khan KM. A randomised clinical trial of the efficacy of drop squats or leg extension / leg curl exercises to treat clinically diagnosed jumper ' s knee in athletes : pilot study. *Br J Sports Med*. 2001;35:60–64.

39. Jonsson P, Alfredson H. Sperior results with eccentric compared to concentric quadriceps training in patients with jumper's knee: A prospective randomised study. *Br J Sports Med*. 2005;39:847-850.

40. Vicenzino, B., & Wright, A. Effects of a novel manipulative physiotherapy technique on tennis elbow: a single case study. Man Ther. 1995;1:30-35.

41. Amro, A., Diener, I., Bdair, W., Hameda, I., Shalabi, A., & Ilyyan, D. The effects of Mulligan mobilization with movement and taping technique on pain, grip strength, and function in patients with lateral epicondylitis. Hong Kong Physiotherapy Journal. 2010; 28: 19-23.

42. Bisset L., Beller E., Jull, G., Brooks, P., Darnell, R., & Vicenzino, B. Mobilisation with movement and exercise, corticosteroid injection, or wait and see for tennis elbow: randomised trial. *British Medical Journal.* 2006;333: 939–941

43. Matocha MA, Baker RT, Nasypany AM, Seegmiller JG. Effects of Neuromobilization on Tendinopathy: Part II. *Interatnional J Athl Ther Train*. 2015;20(2):41–47.

44. Phan N, Blome C, Fritz F, Reich A, Ebata T, Augustin M, Szepietowski C, Stander S.Assessment of pruritus intensity: Prospective study on validity and reliability of the

visual analogue scale, numerical rating scale and verbal rating scale in 471 patients with chronic pruritus. *Acta dermato-venereo.* 2012;92(5):502-507.

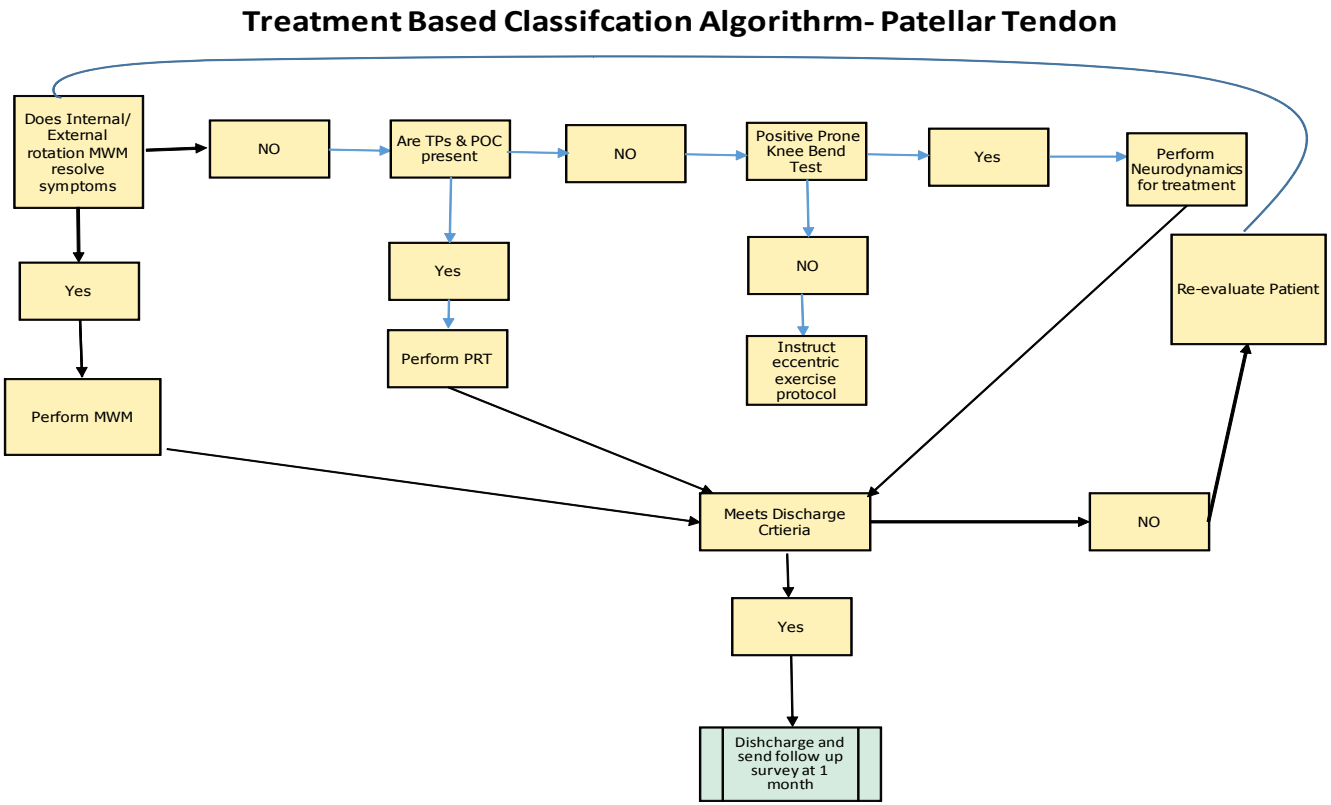

Figure 5.1.
Treatment Classification Algorithm for Patellar Tendinopathy

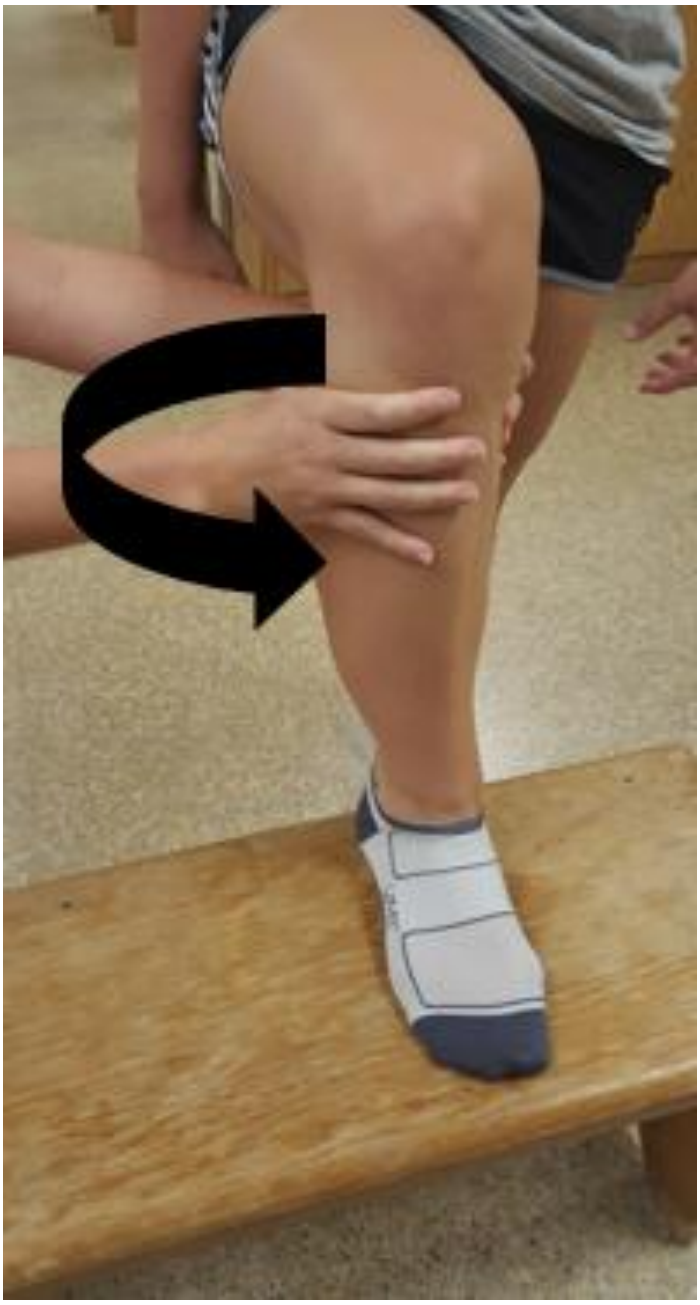

Figure 5.2.
Examples of Hand Placement during the Mobilization with MovementTtechnique.

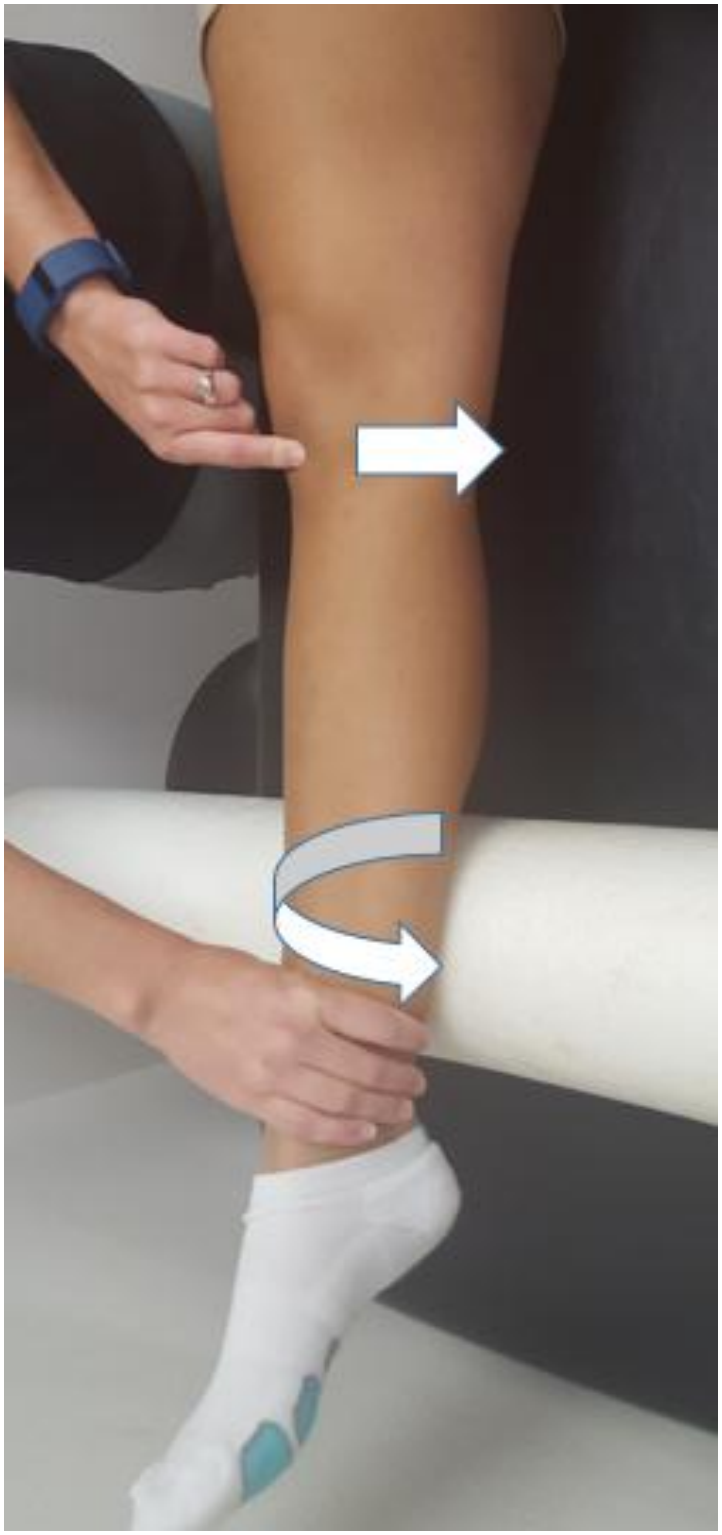

Figure 5.3.
Examples of Positional Release Therapy Position of Comfort

Figure 5.4.
Examples of Neurodynamic Slump Slider Position

Table 5.1.
Results of Patellar Tendinopathy TBC Algorithm

Results of Patellar Tendinopathy TBC Algorithm						
Outcome Measure	**Intake Score**	**Discharge Score**	**Mean Change**	**95% CIs**	**Sig (2 tailed)**	**Effect Size (Cohen's *d*)**
NRS	5.3 ± 1.94	.6 ± .84	4.78 ± 1.64	3.5160, 6.0395	.000	2.41
DPA Scale	30.3 ± 11.02	8.5 ± 9.12	19.89 ± 12.31	10.4242, 29.3535	.001	1.98
VISA-P	53.6 ± 16.58	76.3 ± 18.36	-23.56 ± 16.71	-35.9088, -11.2023	.002	1.37
NRS: Numeric Rating Scale; DPA Scale: Disablement in the Physically Active Scale; VISA-P: Victorian Institute of Sport Assessment for the Patellar Tendon						

A Treatment-Based Classification Algorithm to Treat Achilles Tendinopathy: An Exploratory Case Series

Authors: Patti Syvertson, Emily Dietz, Monica Matocha, Janet McMurray

Submitted for consideration to the Journal of Sports Rehabilitation

5.2 Achilles Tendon

Abstract.

Context. Achilles tendinopathy is relatively common in both the general and athletic populations. The current gold standard for the treatment of Achilles tendinopathy is eccentric exercise, which can be painful and time consuming. Indirect treatment approaches have been proposed to treat tendinopathy patients in fewer treatments and without provoking pain; however, research in this area is lacking.

Objective. The purpose of this study was to determine the effectiveness of utilizing a Treatment Based Classification (TBC) algorithm as a strategy for classifying and treating patients diagnosed with Achilles tendinopathy.

Participants. Eleven subjects (mean age 28.0 ±15.37 years) diagnosed with Achilles tendinopathy.

Design. Prospective case series.

Setting. Participants were evaluated, diagnosed, and treated at multiple clinics.

Main outcome measures. Numeric Rating Scale (NRS), Disablement in the Physically Active Scale (DPA Scale), Victorian Institute of Sport Assessment–Achilles (VISA-A), Global Rating of Change (GRC), and Nirschl Phase Rating Scale were recorded to establish baseline scores and evaluate participant progress.

Results. A RM-ANOVA was conducted to analyze NRS scores from initial to discharge and at 1 month follow up. Paired *t*-tests were analyzed to determine the effectiveness of utilizing a TBC algorithm from initial exam to discharge on the DPA Scale and VISA-A. Descriptive statistics were evaluated to determine outcomes as reported on the GRC.

Conclusion. The results of this case series provides evidence that utilizing a TBC algorithm can improve function, while decreasing pain and disability in Achilles tendinopathy participants.

Keywords*:* Achilles tendinopathy, treatment-based classification, outcome measures, manual therapy

Introduction. Achilles tendinopathy commonly affects elite and recreational athletes, as well as the sedentary individual.[1–5] The pathology is most prevalent in men ages 35 to 45 years old who are involved in running and jumping activities; [2] however, increases in high level sports participation in adolescence has produced an increased risk for prevalence in the younger population.[6] Self-reported musculoskeletal injury for the heterogeneous population has become so high, ranging from 2 to 65% that tendinopathy has been called "a nemesis for patients and physicians."[7]

Various theories have been proposed to explain the etiology of Achilles tendon pain. The origin of tendon pathology appears to be a multifactorial process;[7–12] a combination of intrinsic and extrinsic factors could be working together or alone.[8–10,13] Extrinsic factors (e.g., excessive load, training errors)[9] and intrinsic factors (e.g., age, sex, genetics, tendon vascularity) are predisposing factors related to pathophysiological effects.[6] These intrinsic and extrinsic factors are thought to be present in 60 to 80% of tendon pain patients.[9]

To better understand the etiology of the pathology, researchers have conducted histopathological studies investigating the role of physical activity in tendon pathology.[1] Mechanical and vascular factors have been theorized as potential contributors in tendon pain.[3] While researchers recognize mechanical loading is a causative factor for tendinopathy, the exact loading conditions are poorly defined or understood.[7] Theoretically, mechanical loading causes the collagen fibrils to become disorganized and torn resulting in an increase in tenocyte production, creating a cascade of inflammatory events associated with an abnormal increase of collagen III.[4,11,12] Paradoxically, there is a subgroup of people afflicted by Achilles tendinopathy with sedentary lifestyles who have no discernible history of mechanical overload, suggesting mechanical loading may not be the only cause.[14] The

vascular theory examines the disruption of the vascular system, which may be more susceptible in the Achilles, causing tendon degeneration.[15] Vascular compromise can create an apoxic state or a hypovascular areas that results in ischemic pain.[16,17] Vascular alterations due to stress or tension on neural structures may alter blood flow to the nerve resulting in an influx of pain chemicals, such as the neurotransmitter glutamate and substance P.[1,18] Recently, these neural effects have been recognized as potential factors in tendon pain.[1,17,18]

Due to the difficulties in identifying the exact causation of Achilles tendinopathy, treatment and management remains controversial.[19] Historically, conservative treatment strategies included a combination of rest, ice, non-steroidal anti-inflammatory drugs (NSAIDs), physical therapy, orthotics, corrections of malalignment, stretching, and corticosteroid injections.[16,20] The introduction of eccentric loading training programs revolutionized the treatment of Achilles tendinopathy.[21,22] Researchers reported good short-term and long-term clinical results with mid-portion Achilles pain using eccentric exercises (EE);[1,23–25] however, participants complained of pain and muscle soreness, which often resulted in poor patient compliance.[26] Furthermore, loading the tendon had to be carefully managed by the clinician to assure the patient's treatment was not exacerbating the pathological state (e.g., matrix reorganization, collagen) or pain.[25] Although EE were believed to initiate tissue regeneration, research has not conclusively established the changes are due to mechanical loading.[5,21]

Considering the multifactorial nature of tendon pathology and the inconsistent results with traditional treatments,[25] the implementation of a treatment based classification (TBC) algorithm utilizing indirect manual therapy treatments is timely. While researchers have not come to a definitive conclusion on the origin of tendon pain or a mechanism of recovery,[27]

the addition of a TBC algorithm would provide a treatment strategy for clinicians to assist with the classification of tendon pain while matching patients to an appropriate manual therapy treatment.[27–29] While there is no definitive research on indirect manual therapy treatment techniques, such as the Mulligan Concept (MC) or Positional Release Therapy (PRT) for the treatment of Achilles tendinopathy, researchers have reported positive outcomes using manual therapies to address causative factors of tendon pain.[28–35] The purpose of this study was to determine the effectiveness of utilizing a TBC algorithm as a strategy for classifying and treating patients diagnosed with Achilles tendinopathy.

Methods.

Design. Utilizing a novel TBC algorithm, Achilles tendinopathy participants were classified into a corresponding treatment group (i.e., MC, PRT, neurodymanics, or EE) (Figure 5.5). Multiple outcome measures were used to determine the effect of the TBC algorithm on pain, disability, and function: Numeric Rating Scale (NRS), Disablement in the Physically Active Scale (DPA Scale), Victorian Institute of Sport Assessment–Achilles (VISA-A), Global Rating of Change (GRC), and the Nirschl Phase Rating Scale (Table 5.2).

Participants. A convenience sample of 11 participants (seven females, four males, mean age = 28 ±15.37 years) volunteered at four clinical sites across the United States. All participants read and signed an informed consent form. If participants were under the age of 18, the parent or legal guardian signed an informed consent and the minor signed an assent form. The University of Idaho Institutional Review Board approved the study.

Procedures. The evaluation process was pre-determined by the clinicians to ensure consistency of procedures and classification using the TBC algorithm. Participants were selected based on a preset inclusion and exclusion criteria (Table 5.3). The initial clinical

evaluation included a participant history, a standard physical examination, and specific orthopedic testing: 1) the Thompson test to rule out Achilles dysfunction, 2) a sub-therapeutic application of MC technique to determine a reduction in participant symptoms, 3) a lower quarter screen to detect the presence of tender points, and 4) a slump test to rule in neurological dysfunctions.

The MC technique required the participant to be kneeling on a chair or lying prone on a plinth. The clinician applied lateral pressure to the Achilles while the participant performed plantar flexion. If the movement exacerbated the pain, the clinician applied the same technique using medial pressure to the Achilles tendon. If the participant reported a resolution of symptoms during application of this technique, then the participant was classified in the MC treatment sub-group. If the participant symptoms did not resolve with the MC, the TBC algorithm was progressed to consider the PRT sub-group. The participant was assigned to the PRT treatment sub-group if tender points were detected when the participant underwent the quarter screen and a position of comfort (POC) was found. If the participant was not assigned to the MC or PRT group, the results of the slump test were considered; a positive slump test at this point would lead to the participant being classified into the neurodynamic treatment sub-group. In the case where the participant could not be classified into the MC, PRT, or neurodynamic sub-groups, the participant was then classified into the eccentric exercise subgroup (Figure 5.5).

When the treatment classification process was completed, the participant underwent the designated treatment (Table 5.4). The participant was treated for three visits in a period of 10 days. At the conclusion of the third visit, the participant was assessed to determine if discharge criteria had been met. The discharge criteria included a worst pain score equal to,

or less than two out of ten on the NRS, a minimal clinical important difference (MCID) change score for the GRC and DPA Scale, and a report of phase 1 on the Nirschl Phase Rating Scale.

If the participant did not meet discharge criteria by the third treatment visit, the participant was re-evaluated. If the participant met 50% of the MCID for the DPA Scale, GRC, and NRS, they were classified into the same treatment subgroup if the treatment was still appropriate, as determined by following the steps of the TBC algorithm. If the participant's improvement did not meet 50% MCID on each of the outcome measures and if the TBC algorithm classified the participant into another treatment subgroup, the participant was re-classified into another subgroup. The participant was assigned to the eccentric exercise subgroup if the participant could not be classified into the first three treatment subgroups.

Participants completed outcome measures at predetermined intervals. The NRS pre and post scores were documented every visit. The DPA Scale, VISA-A, and the Nirschl Phase Rating Scale were documented during the initial visit, every third visit, and at discharge. The GRC was reported every third visit and at discharge. A one-month follow-up survey was emailed to collect participant's post-discharge NRS scores.

Statistical Analysis. All data was analyzed using SPSS version 23.0 (SPSS Inc., Chicago, IL, USA). A RM-ANOVA was analyzed for the initial, discharge, and one-month follow-up on NRS scores. Paired *t*-tests were performed on the DPA Scale and VISA-A to determine the effectiveness of classifying and treating participants utilizing the TBC algorithm for Achilles tendinopathy. Mean differences from the initial visit scores and 95% confidence intervals (CIs) were calculated for the DPA Scale and VISA-A for discharge.

Cohen's *d* was computed to determine the effect size for the DPA Scale and VISA-A; an effect size of 0.2 to 0.3 was considered a "small" effect, a 0.5 was a "medium" effect, and 0.8 to infinity, a "large" effect.[36] Descriptive statistics were analyzed to determine the outcomes as reported on the GRC.

Results. The results of the RM-ANOVA of the NRS scores indicated a significant effect of time on the use of indirect treatments (Wilks' Lambda = .005, F (2, 3) = 317.250, $p < .001$, $\eta^2 = .995$). Follow-up comparison indicated a significant mean change in pain from initial visit to discharge (M = 6.40, 95% CI [5.43 to 7.37], $p < .001$), and initial to one month follow up (M = 6.04, 95% CI [4.01 to 8.06], $p = .001$).

The significant improvement in NRS scores indicated that indirect treatments of the associated subgroup classification had a positive effect on the participant's pain score. The change in mean scores from discharge and one-month follow up (M = -.360, 95% CI [-1.78, 1.06], $p = 1.00$) was not significant, indicating the improvement was maintained at one-month follow up.

All 11 participants (100%) exceeded the MCID for the NRS[37] after the first treatment. The mean change in pain from the initial worst pain score to discharge, and initial to one month follow up, continued to exceed the MCID for the NRS.[37] More clinically relevant, 81% (n = 9) of the participants reported a full resolution of pain at discharge. The remaining 19% (n = 2) reported a "worst" pain NRS score of one or two respectively. Five participants responded to the one-month follow up survey after returning to full activity. Of the five participants, four reported a pain score of zero on the NRS at one-month post discharge; one participant reported a worst pain score as a two out of ten on the NRS, which

was a six point decrease from initial NRS and a two point increase from the discharge NRS score.

The TBC algorithm resulted in a significant mean change in disablement as measured on the DPA Scale, from initial evaluation to discharge (M = 16.61 ±11.71, 95% CI [9.53 to 23.69], p = .001), with a large effect size (Cohen's d = 3.01). The mean change from initial visit to discharge, as well as the lower boundary of the confidence interval, exceeded the MCID for disablement[38] in six treatment sessions or less. More clinically relevant, all 11 participants (100%) met an MCID for acute (nine points) or chronic pain (six points)[38] prior to discharge. Initial DPA Scale scores (M = 29.45) were within the healthy range score (R = zero to 34 points); mean discharge scores (M = 9.81, R = 0 to 20 points) were similar to the reported DPA Scale normative values (M = 8.82 ± 6.71, R = 0 to 23 points) for acute injuries upon return to participation.[38]

An examination of the change in VISA-A scores revealed a significant improvement in patient scores from initial exam to discharge (M = 35.15 ±28.81, 95% CI [17.73 to 52.56], p = .001), with a large effect size (Cohen's d = 3.66). Of greater clinical relevance, six of the 11 participants were in the excellent range (90 points), while the remaining five participants reported scores that were in the healthy range for the VISA-A.[34,39] While an MCID for the VISA-A has been established for insertional Achilles tendinopathy (6.5 points),[39] one has not been established for mid-portion Achilles tendinopathy. The participants in this study (n = 11) were diagnosed with mid-portion tendinopathy, but all still met the insertional Achilles MCID at discharge.

The GRC mean score at discharge was M = 4.3 ± 2.66. The mean GRC scores at discharge did not meet the value for "important improvement"[33] because one participant did

not report a change meeting this value at discharge. However, 91% (n = 10) of the participants reported a MCID for the GRC scale and were discharged within three treatment sessions.

During the initial exam, the Nirschl Phase Rating Scale for participants ranged from phase 5 (pain that is present during all activities and occurs with activities of daily living) to phase 2 (stiffness or mild soreness before activity that is relieved by warm-up). Specifically, 27% (n = 3) reported a phase 5, 36% (n = 4) reported a phase 4 (pain with activity that causes modification), 18% (n = 2) reported phase 3 (stiffness or mild soreness before specific sport or occupational activity), and 18% (n = 2) reported a phase 2.[10,13] At discharge, all participants (100%) reported a phase one on the Nirschl Phase Rating Scale.

Discussion. Historically, diagnosing tendinopathy involved a thorough history, a physical examination, and special tests to isolate the injured structure. The use of this methodology and the reliance on traditional diagnostic testing is changing.[12,17,27] Lewis[27] developed a unique method of assessment for rotator cuff tendinopathy patients using a shoulder symptom modification procedure that identified either one, or a series of techniques to reduce symptoms and match the patient to treatment interventions. The method of assessment Lewis[27] implemented constituted a TBC system for tendinopathy patients. Research on TBC systems for tendinopathy, however, is lacking and the majority of research on TBC systems has been focused on low back pain patients.[28,29]

The positive outcomes experienced from the use of a novel TBC algorithm in this case series supports the theory that TBC systems could be an effective evaluation and treatment strategy to improve patient-outcomes in Achilles tendinopathy patients. The utilization of multiple outcome measures to fully assess patient progress while implementing

the TBC algorithm was important to determine the effect of the algorithm. In our study, Achilles tendinopathy participants reported statistically and clinically significant improvements in pain, disability, and function at discharge after being treated with the treatment associated with their sub-grouping classification, without a single participant being assigned to the EE sub-group. Moreover, the participants who received indirect manual therapy techniques matched to their sub-group were discharged in less time (5.58 ±2.31 days) when compared to the gold standard 12 week EE protocol found in the literature.[2,16,21,31,35]

Additionally, the use of the TBC algorithm also produced comparable changes across other outcomes when compared to EE outcomes reported in the literature. Roos et al.[26] conducted a comparison study on Achilles tendinopathy participants that were assigned to a control, a splint, or an EE group for six weeks. Participants in the EE group reported significant improvement in pain and function at six weeks, as measured by the Foot and Ankle Outcome Score (FAOS) scale (27% compared with baseline, $p = 0.007$) and one year (42%, $p = 0.001$). While the FAOS and VISA-A are not identical, both scales rate pain and function. The scales use a similar rating system (0, worst to 100, best) and are specific and applicable to the foot and ankle region. In a comparison of the EE FAOS scores[26] with VISA-A outcome measures reported in our study, the FAOS scores decreased 26 points from baseline in 52 weeks, while the VISA-A scores in our study decreased 35 points in approximately six days (5.58 ±2.31).

Fahlström et al.[23] studied a large number of patients (n = 78) with chronic Achilles mid portion tendinosis pain and patients (n = 30) with insertional Achilles tendon pain. All patients were treated with a 12-week EE program. Visual Analogue Scale (VAS) scores

(0mm, no pain, to 100mm, severe pain) reported by patients with mid-portion Achilles tendon pain decreased significantly (initial M = 66.8 ±19.4; discharge [12 week] M = 10.2 ± 13.7). Similarly, in a study by Mafi et al.,[40] eccentric and concentric training protocols were compared; 82% of the EE patients were satisfied after 12 weeks of EE versus 26% of patients treated with the concentric protocol. Overall, VAS satisfaction scores for the EE patients significantly decreased (baseline M = 69; post-treatment M = 12). With a high correlation between the VAS and NRS (e.g., 35mm VAS score corresponds to NRS score of 3.5 to 4.7 out of 10)[41] our initial to discharge mean NRS scores decreased from a mean of 5.86 out of 10 to a mean less than one (M = .27), suggesting that utilizing a TBC algorithm to classify and match participants to appropriate treatment decreased NRS scores in equal, to improved, rates than the gold standard EE protocol.

Currently, there is no known research on the use of indirect manual therapy treatment techniques found in our TBC algorithm for Achilles tendinopathy. However, in an article by Vicenzino,[42] the author discussed MC mobilizations and taping techniques for the treatment of lateral epicondylalgia. Vicenzino[42] suggested specific taping techniques could replicate a manual glide force, thereby, reducing the patient's pain outside of the clinic. More recently, in a case study utilizing PRT, researchers demonstrated positive outcomes for pain and function on a swimmer with a history of bicep tendinopathy.[43] In our study, all of the participants were classified into either the MC or PRT treatment sub-group and reported positive outcomes. The TBC algorithm allowed the clinician to match the participant to an intervention that corresponded with the participant's dysfunction. In theory, matching Achilles tendinopathy patients to treatments with the use of a TBC algorithm could improve outcomes.

While the preliminary results are clinically meaningful, there were limitations to this study. Clinicians attempted to decrease the risk of bias, though a bias could have been introduced because the clinician and the participant were not blinded to the treatment or the collection of outcome measures. The lack of a control or long term follow-up made it difficult to differentiate if the participant's improvement was from the intervention or the natural course of healing; however, it would be unlikely that chronic pain patient's symptoms would have spontaneously resolved within the time frame of the treatments reported in this study. Although, several MC applications exist for the treatment of Achilles tendon pain with suggested force and application adjustments to resolve symptoms; only one was utilized in the present study. Therefore fully following MC recommendations the patient response to the treatment may have improved. Finally, the sample of participants may not have fully represented Achilles tendinopathy patients; participants who volunteered may have been motivated to improve.

Given the positive outcomes associated with the TBC algorithm, future research is warranted. Future studies should include a comparison of the TBC algorithm with a control or placebo group. Comparing the individual components of the TBC algorithm may also elucidate which interventions are the most effective and may guide the development of a more effective order of the sub-groups. Additionally, long-term follow-up results are needed to assess the full effectiveness of the TBC algorithm. Finally, the inclusion of histopathological exams or diagnostic imaging following the use of the TBC algorithm would provide invaluable insight into the physiological changes of the Achilles tendon through the course of treatment to help clarify the mechanism of action of the intervention.

Conclusion. In this case series, promising short-term clinical results with a small group of participants diagnosed with Achilles tendinopathy were found. Our findings were statistically significant and clinically meaningful and support future research utilizing a TBC algorithm to classify and match participants to appropriate indirect manual therapy treatment techniques for the management of Achilles tendinopathy.

References

1. Alfredson H, Cook J. A treatment algorithm for managing Achilles tendinopathy: new treatment options. *Br J Sports Med*. 2007;41(4):211-216. doi:10.1136/bjsm.2007.035543.
2. Grigg NL, Wearing SC, Smeathers JE. Eccentric calf muscle exercise produces a greater acute reduction in Achilles tendon thickness than concentric exercise. *Br J Sports Med*. 2009. doi:10.1136/bjsm.2008.053165.
3. Rees JD, Wilson a M, Wolman RL. Current concepts in the management of tendon disorders. *Rheumatology (Oxford)*. 2006. doi:10.1093/rheumatology/kel046.
4. Scott, Ashe M. Common tendinopathies in the upper and lower extremities. *Curr Sports Med Rep*. 2006.
5. Morrissey D, Roskilly A, Twycross-Lewis R, et al. The effect of eccentric and concentric calf muscle training on Achilles tendon stiffness. *Clin Rehabil*. 2011. doi:10.1177/0269215510382600.
6. Maffulli N, Wong J, Almekinders LC. Types and epidemiology of tendinopathy. *Clin Sports Med*. 2003.
7. Wang JHC, Iosifidis MI, Fu FH. Biomechanical basis for tendinopathy. *Clin Orthop Relat Res*. 2006. doi:10.1097/01.blo.0000195927.81845.46.
8. Fredberg U, Stengaard-Pedersen K. Chronic tendinopathy tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. *Scand J Med Sci Sports*. 2008. doi:10.1111/j.1600-0838.2007.00746.x.
9. Jarvinen TAH, Kannus P, Maffulli N, Khan KM. Achilles tendon disorders: etiology and

epidemiology. *Foot Ankle Clin*. 2005. doi:10.1016/j.fcl.2005.01.013.

10. Nirschl RP, Ashman ES. Elbow tendinopathy: tennis elbow. *Clin Sports Med*. 2003. doi:10.1016/S0278-5919(03)00051-6.
11. Khan KM, Maffulli N, Coleman BD, Cook JL, Taunton JE. Patellar tendinopathy: some aspects of basic science and clinical management. *Br J Sports Med*. 1998.
12. Rio E, Moseley L, Purdam C, et al. The pain of tendinopathy: Physiological or pathophysiological? *Sport Med*. 2013. doi:10.1007/s40279-013-0096-z.
13. Kaux JF, Forthomme B, Goff C LE, Crielaard JM, Croisier JL. Current Opinions on Tendinopathy. *J Sports Sci Med*. 2011.
14. Scott A, Huisman E, Khan K. Conservative treatment of chronic Achilles tendinopathy. *Can Med Assoc J* . 2011. doi:10.1503/cmaj.101680.
15. Fenwick SA, Hazleman BL, Riley GP. The vasculartureand its role in the damaged and healing tendon. *Arthritis Res*. 2002.
16. Alfredson H. The chronic painful Achilles and patellar tendon: research on basic biology and treatment. *Scand J Med Sci Sports*. 2005;15(4):252-259. doi:10.1111/j.1600-0838.2005.00466.x.
17. Matocha MA, Baker RT, Nasypany AM, Seegmiller JG. Effects of Neuromobilization on Tendiopathy:Part I. *Interatnional J Athl Ther Train*. 2015.
18. Alfredson H, Ljung BO, Thorsen K, Lorentzon R. In vivo investigation of ECRB tendons with microdialysis technique--no signs of inflammation but high amounts of glutamate in tennis elbow. *Acta Orthop Scand*. 2000. doi:10.1080/000164700317381162.
19. Kearney R, Costa ML. Insertional Achilles tendinopathy management: a systematic

review. *Foot Ankle Int* . 2010. doi:10.3113/FAI.2010.0689.Glaser T, Poddar S, Tweed B, Webb CW. Clinical inquiries. What's the best way to treat Achilles tendonopathy? *J Fam Pract*. 2008.

20. Alfredson H, Pietilä T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med*. 1998.

21. Stanish W, Curwin S, Rubinovich M. Tendinitis: the analysis of treatment for running. *Clin Sports Med*. 1985.

22. Fahlstrom M, Jonsson P, Lorentzon R, Alfredson H. Chronic Achilles tendon pain treated with eccentric calf-muscle training. *Knee Surg Sports Traumatol Arthrosc*. 2003. doi:10.1007/s00167-003-0418-z.

23. Jonsson P, Alfredson H. Superior results with eccentric compared to concentric quadriceps training in patients with jumper's knee: a prospective randomised study. *Br J Sports Med*. 2005. doi:10.1136/bjsm.2005.018630.

24. Scott A, Docking S, Vicenzino B, et al. Sports and exercise-related tendinopathies: a review of selected topical issues by participants of the second International Scientific Tendinopathy Symposium (ISTS) Vancouver 2012. *Br J Sports Med*. 2013. doi:10.1136/bjsports-2013-092329.

25. Roos EM, Engström M, Lagerquist A, Söderberg B. Clinical improvement after 6 weeks of eccentric exercise in patients with mid-portion Achilles tendinopathy – a randomized trial with 1-year follow-up. *Scand J Med Sci Sports*. 2004. doi:10.1111/j.1600-0838.2004.378.x.

26. Lewis J. Rotator cuff tendinopathy/subacromial impingement syndrome: Is it time for a

new method of assessment? *Br J Sport Med*. 2008.

27. Delitto A, Erhard RE, Bowling RW. A treatment-based classification approach to low back syndrome: identifying and staging patients for conservative treatment. *Phys Ther*. 1995;75(6):470-479.

28. Stanton TR, Fritz JM, Hancock MJ, et al. Evaluation of a treatment-based classification algorithm for low back pain: a cross-sectional study. *Phys Ther* . 2011;91 (4):496-509.

29. D'Ambrogio K, Roth G. *Positional Release Therapy, Assesment & Treatment of Musculoskeletal Dysfunction*. St. Louis: Mosby; 1997

30. Dimitrios S, Pantelis M, Kalliopi S. Comparing the effects of eccentric training with eccentric training and static stretching exercises in the treatment of patellar tendinopathy. A controlled clinical trial. *Clin Rehabil*. 2011. doi:10.1177/0269215511411114.

31. Jones LH, Kusunose R, Goering E. *Jones Strain-Counterstrain*.; 1995.

32. Kamper S, Maher C, MacKay G. Global rating of change scales. *Aust J Physiother*. 2009;55(4):289. doi:10.1016/S0004-9514(09)70015-7.

33. Robinson JM, Cook JL, Purdam C, et al. The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy. *Br J Sports Med*. 2001.

34. Wasielewski NJ, Kotsko KM. Does eccentric exercise reduce pain and improve strength in physically active adults with symptomatic lower extremity tendinosis? A systematic review. *J Athl Train*. 2007.

35. Vincent W, Weir J. *Statistics in Kinesiology*. Human Kinetics; 1994.

https://books.google.com.br/books?id=J7RaNTMsul4C.

36. Farrar JT, Young JPJ, LaMoreaux L, Werth JL, Poole RM. Clinical importance of changes in chronic pain intensity measured on an 11-point numerical pain rating scale. *Pain*. 2001.

37. 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

38. McCormack J, Underwood F, Slaven E, Cappaert T. The minimum clinically important difference on the VISA-A and LEFS for patients with insertional Achilles tendinopathy. *Int J Sports Phys Ther*. 2015;10(5):639-644.

39. Mafi N, Lorentzon R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. *Knee Surgery, Sport Traumatol Arthrosc*. 2001. doi:10.1007/s001670000148.

40. Phan NQ, Blome C, Fritz F, et al. Assessment of pruritus intensity: prospective study on validity and reliability of the visual analogue scale, numerical rating scale and verbal rating scale in 471 patients with chronic pruritus. *Acta Derm Venereol*. 2012;92(5):502-507. doi:10.2340/00015555-1246.

41. Vicenzino B. Lateral epicondylalgia: a musculoskeletal physiotherapy perspective. *Man Ther*. 2003. doi:10.1016/S1356-689X(02)00157-1.

42. Baker RT, Riper M Van, Medical S, Nasypany AM, Seegmiller JG. Evaluation and treatment of apparent reactive tendinopathy of the biceps brachii. *Int J Athl Ther Train*. 2014.

43. Butler D. *The Sensitive Nervous System*.; 2000.

44. Butler D, Moseley L. *Explain Pain*.; 2003.

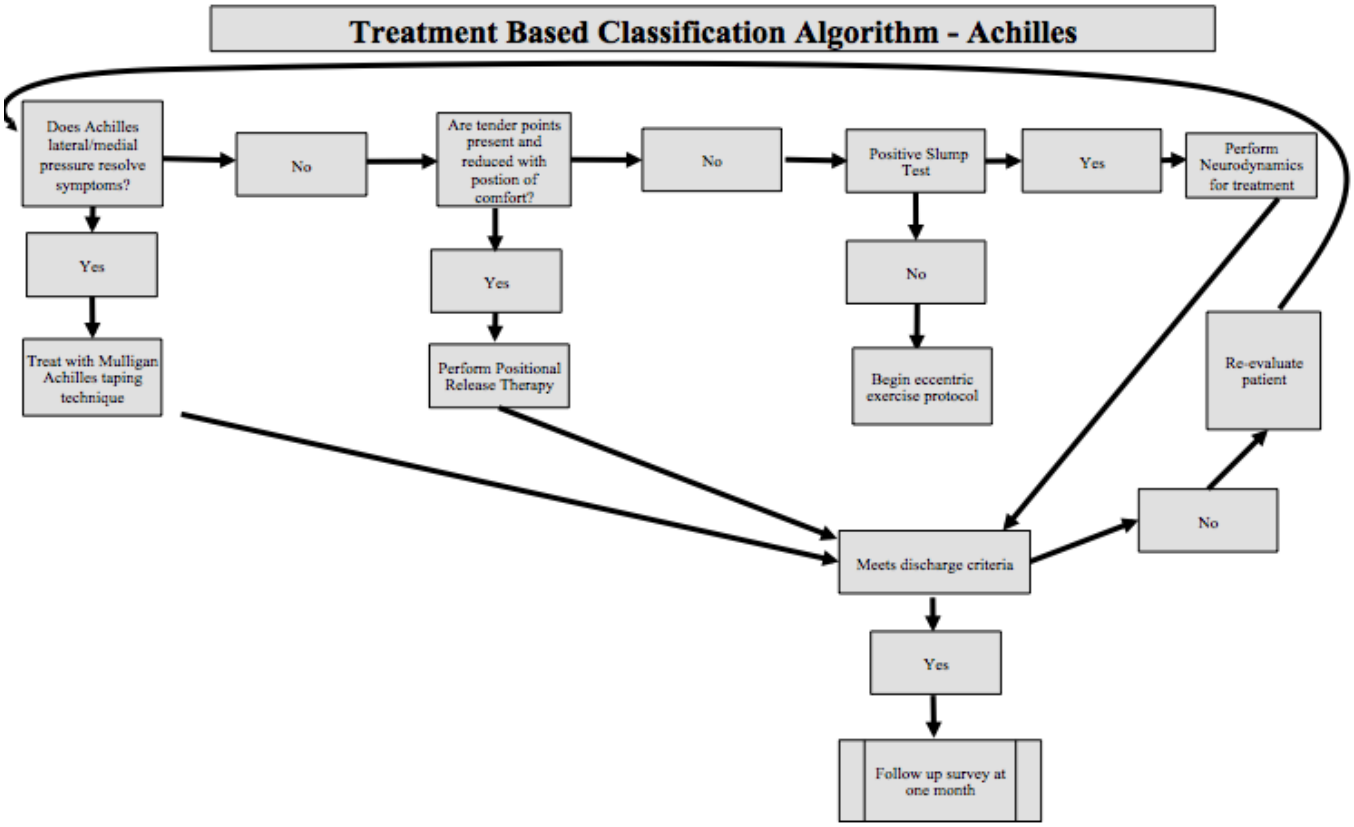

Figure 5.5.
Treatment Based Classification Algorithm for Achilles Tendinopathy

Table 5.2.
Description of Outcome Measures

Description of Outcome Measures		
Outcome measure	**Construct**	**Description**
Numeric Rating Scale (NRS)[37]	Pain	The NRS is an 11-point pain rating scale ranging from 0 (no pain) to 10 (worst pain imaginable). Each participant identified their rating of pain at best, worst, and rest at the beginning and end of each treatment. The MCID has been established at 2 points or a reduction of 30% for the NRS.
Disablement in the Physical Active (DPA Scale)[38]	Disability	The DPA Scale is a 16 item rating scale correlated with the participant's impairment, functional limitations, disability, and quality of life issues. Each item was rated on a scale of 1 (no problem) to 5 (severe) with a maximum score of 64 points and minimum score of 0. MCID has been established at nine for acute injuries and six for chronic injuries.

Table 5.2.
Description of Outcome Measures(Continued)

Description of Outcome Measures (Continued)		
Global Rating of Change Scale (GRC)[33]	Efficacy of treatment	The GRC establishes the effectiveness of treatments by documenting the participant's improvement or deterioration over time. The GRC is a 15-point scale. A score of -7 indicates a great deal worse and +7 suggests a great deal better. A score of 0 represents no change from initial injury. The MCID for the GRC has been established at 2 points.
Nirschl Phase Rating Scale (NPRS)[10,13]	Tendon Pain	The NPRS was developed to classify symptoms and help determine prognosis by separating activities of daily living from sports performance. The NPRS is a 7-phase pain scale. Phase 1 (mild pain) Phase 7 (intense pain). No MCID has been established.
Victorian Institute of Sport Assessment-Achilles (VISA-A)[34]	Pain, function, perceived effect on physical activity	The VISA-A evaluates three domains: pain, function, and the effect on physical activity specific to Achilles tendinopathy. The VISA-A is 0-100 point questionnaire. The lower the score the more symptoms and the greater limitation of physical activity. An MCID for insertional Achilles tendinopathy has been established at 6.5 points.[39]

Table 5.3.
Inclusion and Exclusion Criteria

Inclusion and Exclusion Criteria	
Inclusion Criteria	**Exclusion Criteria**
• Participant 14 years or older • Tendon pain or dysfunction with weight bearing activities • Tendon pain before, during, or after loading activities • Palpable tendon pain 2 – 6 cm above the insertion of the calcaneus • Tendon pain in the morning • Tendon pain or stiffness that resolves with activity	• Cortisone injection < 6 weeks • Use of fluoroquinolones or ciprofloxacin (<12 months) • Post-operative instructions excluding from treatment • Current healing of or a suspected fracture • Current dislocation • Enrolled in physical therapy for Achilles tendon pain • Participant is pregnant

Table 5.4.
Description of Treatment Techniques

Description of Treatment Techniques	
Mulligan Achilles Taping Technique[36]	• Participant kneeled on a chair or was prone on a plinth. • Two-inch leukotape was applied to the medial aspect of the tendon. Tape was wrapped around the lateral ankle and secured on the anterior/medial distal tibia. • Tape was secured with another piece of tape. • Note: If participant responded to medial pressure in evaluation the tape would be wrapped accordingly (lateral to medial).
Positional Release Therapy[30,32]	• Proximal tender point eliciting the greatest discomfort was treated first. • Participant was placed in a position of comfort (POC). • Clinician held the affected tender point at a sub-threshold pressure for approximately 90 seconds or until tissue pulsation stopped • Clinicians slowly returned participant back to normal position. • If the participant reported pain after one set of treatment, the clinician returned the participant to the POC while monitoring the TP for full resolution of pain for a maximum of three treatment sets held for 30-90 seconds at the TP.

Table 5.4.
Description of Treatment Techniques(Continued)

Description of Treatment Techniques (Continued)	
Neurodynamics[44,45]	• A neurodynamic slider or tensioner was performed. • A neural slider was chosen as the neurodynamic movement (neural tensioners often aggravate the nerve). • A slider was performed if the participant experienced a release in neural symptoms when the head moved from cervical flexion to neutral during the slump test. • Participant is seated. Participant slumped forward (cervical and trunk flexion) then performed knee extension; as the foot was actively dorsiflexed, the participant moved into cervical neutral. As the participant moved the foot into plantarflexion, the cervical spine was simultaneously moved into flexion. • Sliders were performed eight to 10 reps, five to eight times/day.
Eccentric Exercise[21]	• Participant was plantar flexed ankle. • Heel was slowly lowered. • Uninjured foot was used to raise the heel back to the starting position (no concentric contraction). • Exercises performed seven days a week, twice a day, three sets of 10 to 15 repetitions, for up to12 weeks. • Exercises were performed with knee straight and knee bent.

An Exploratory Examination of a Treatment Based Classification Algorithm to Treat Lateral Epicondylalgia: A Case Series

Authors: Janet McMurray, Emily Dietz, Monica Matocha, Patti Syvertson

Submitted for consideration to the International Journal of Athletic Training & Therapy

5.3 Lateral Elbow

Key Points:

- Utilizing a treatment-based classification algorithm may improve patient classification.
- Utilizing a treatment-based classification algorithm may improve treatment selection.
- Matched indirect intervention techniques may be more effective than traditional treatments in resolving lateral elbow tendon pain.

Abstract.

Context. Lateral epicondylalgia is a common condition experienced by both males and females in occupational environments and the sports arena. Appropriate classification and treatment of lateral epicondylalgia is still under investigation; as a result, traditional treatment methods are often ineffective and tendon pathology classification remains unclear. Though not commonly researched, other novel treatment options have been linked to pain reduction and functional restoration in tendinopathy patients.

Objective. The purpose of this study was to explore the effectiveness of a novel treatment-based classification (TBC) algorithm on participants with lateral epicondylalgia.

Participants. A total of 8 participants (female = 1, males = 7; mean age 42.43 ± 18.58 years) with a primary complaint of lateral elbow pain were evaluated and included in this case series.

Interventions. Each participant underwent a thorough initial evaluation process which included: medical history, range of motion measurements, orthopedic tests, a scan for tender points, neurodynamic tests, and Mulligan Concept techniques to determine diagnosis, study inclusion, and treatment classification. Data for pain intensity, disability, and function utilizing the Disablement in the Physically Active (DPA) Scale, Numeric Rating Scale (NRS), Nirschl Phase Rating Scale, Global Rating of Change (GRC), and Patient Rated Tennis Elbow Evaluation (PRTEE) were collected to determine the efficacy of the TBC.

Main Outcome Measures. Paired *t*-tests were conducted on DPA Scale and PRTEE scores and descriptive statistics were conducted on the GRC. RM-ANOVA was conducted on the NRS scores.

Result. Statistically significant and clinically meaningful improvements were found for pain, disability, and function from the initial evaluation to discharge.

Discussion. The results of this case series provide evidence of the effectiveness of the TBC algorithm for participants with lateral epicondylalgia.

Introduction. Lateral elbow pain associated with gripping and manipulation of the hand is commonly diagnosed as tennis elbow or lateral epicondylalgia (LE). Lateral epicondylalgia is reported to affect 1-3% of the general population[1] and represents four to seven cases per 1000 patients seen in general practice.[2,3] The diagnosis is viewed as challenging to treat and patients are prone to recurrent episodes.[1-3] The common clinical signs attributed to LE are pain during direct palpation over the lateral extensor tendons and associated pain and weakness during grip strength testing.[4] An LE diagnosis is often based on a history of occupation- or activity-related pain at the lateral elbow, with symptoms that are reproduced with resisted supination or wrist flexion, particularly with the arm in full

extension.[5] Despite the prevalence of the condition, the underlying etiology of LE is not readily understood and dissimilar patient presentations, varying injury states and multiple pathology labels for tendon pathologies have created difficulties in classifying tendon pain and contribute to ineffective treatment selections.[4-6]

Historically, tendon pain was considered an inflammatory response to overload or overuse.[6-9] While mechanical load is still considered a factor in tendon pain, more recent evidence from microscopic and histological analysis has identified an absence of inflammatory cells. As a result of these findings, theories of causative factors for tendon pain have been revised and indicate chronic tendon pain is often non-inflammatory in nature .[7-9] Due to the close association between neural structures, microvasculature, and neurochemicals, neovessel ingrowth has recently received increased attention as a source of tendon pain. [6,10,11] Increasingly, researchers have suggested tendon pain is an integration of several interrelated components (i.e., neural, vascular, and tendon structure),[6,10-11] but this improved understanding of tendon pathology has not led to an enhancement in identifying the origin of tendon pain to guide patient classification and clinical treatment.[4,6,12]

Currently, a gold standard for LE treatment has not been established. Common conservative methods include pharmacotherapy, electrophysical therapy, and eccentric exercise (EE).[1,5,6,] Researchers conducting studies on the efficacy of these treatments report unsatisfactory results for improving function, pain reduction, and high recurrence rates.[6,13-19] No specific (EE) protocols have been clearly delineated for LE, but an emphasis is on the role of isolated eccentric strengthening exercises, mirroring the use of these exercises for lower limb tendinopathies.[20-22] Researchers have discovered early evidence of positive initial

effects utilizing manipulative therapy techniques for pain relief and restoration of function for patients experiencing tendon pain.[23-29]

The lack of evidence supporting common conservative treatment methods, combined with evidence of effectiveness for less-used manual therapy techniques, has led to the proposal of utilizing treatment-based classification (TBC) strategies for tendon pathology. Researchers have proposed patients diagnosed with a tendon disorder may not have an actual tissue pathology requiring tissue remodeling and would benefit from an alteration in the clinical assessment strategy.[30-32] Modifying assessment methods based on the various tendon pain components and patient response to intervention may assist the clinician in matching individual patients to more effective treatment approaches.[6,33] The purpose of this study was to explore the effectiveness of a novel TBC algorithm on participants diagnosed with lateral epicondylalgia.

Methods.

Participants. A convenience sample of participants diagnosed with LE at four different athletic training clinical sites participated in the study. During the study period, eight participants (one female, seven males, mean age 42.43 ± 18.58) were examined, met initial inclusion/exclusion criteria (Table 5.5), and were enrolled in the study. Each participant (n = 8) signed an informed consent form. If the participant was under the age of 18 years old, the legal guardian signed the informed consent, and the minor signed an assent form. The University of Idaho Institutional Review Board approved the research project.

During the study, two participants were removed after initial exam and treatment: one participant experienced altered pain location and no longer met inclusion criteria; the second participant used additional self-treatment (therapeutic tape) outside of the treatment

protocol of the study. A third participant declined reclassification following three treatments and withdrew from the study. Although the participant voluntarily dropped out, outcome measures were generated for one round of treatment (i.e., 3 visits). Outcome measures taken following the last treatment were used as discharge measures and included in the final data analysis. The remaining five participants (female = one, males = four, mean age 37.6 ± 18.90) completed the study from initial exam to discharge.

Instrumentation. The outcome measures utilized in this study included: Nirschl Phase Rating Scale, Numeric Rating Scale (NRS), Disablement of the Physically Active Scale (DPA Scale), Patient Rated Tennis Elbow Evaluation (PRTEE), and Global Rating of Change (GRC) (Table 5.6). Outcome measurements, with exception of GRC, were obtained at the initial evaluation, third treatment session, and at discharge. The NRS scores were recorded pre- and post-treatment during each session. Outcome measures determined participant perceptions of their condition, progression of treatment, and recovery.

Procedures. Each participant was examined using a standardized clinical evaluation to ensure consistency in LE diagnosis and classification through the TBC algorithm among all researchers. The initial evaluation included the participant's medical history, range of motion testing, and special tests which included: Cozen's, Mill's, varus stress, upper limb neurodynamic tests (ULNT), upper quarter screen for tender points, and Mulligan Concept Mobilization with Movement (MWM) at the elbow (lateral glide with elbow flexion/extension followed by medial glide if pain was unresolved after initial application).

Treatment-Based Classification Algorithm. The evaluative component of TBC algorithm (Figure 5.6) consisted of the Mulligan Concept lateral or medial glide MWM, upper quarter screen for tender points, and ULNT. Participant response to these evaluation

strategies determined placement into the treatment sub-groups of the TBC algorithm: Mulligan Concept MWM, Positional Release Therapy (PRT), neurodynamic treatment, or EE. Any participant who did not respond to one of the indirect manual therapy treatments during the initial exam was placed in the EE sub-group. Following treatment classification into a sub-group, the participant received three treatment sessions utilizing the matched treatment associated with that sub-group.

Initially, the clinician scanned for tender points using a quarter screen based on the teachings of D'Ambrosio & Roth[34] during the physical exam. The TP severity was determined using participant reported pain to palpation on the NRS.[34] Participant neurologic response was assessed utilizing ULNT during the physical exam. A single sub-therapeutic application of the Mulligan Concept MWM was used as a special test to determine if its use resolved the participant's symptoms during elbow flexion and extension. If the evaluation application of the Mulligan MWM resolved the participant's complaints, the participant was classified into the MWM sub-group. If MWM did not resolve participant symptoms, the clinician reviewed the TPs identified during the exam. If TPs were identified during the upper quarter screen and a position of comfort (POC) (i.e., placing the particular muscle into an ideal position which reduced tender point irritability) could be found for the dominant TP, the participant was classified into the PRT sub-group. If no symptom resolution occurred with MWM or PRT, the participant's response to ULNT was considered. Participants with a positive ULNT were then placed in the neurodynamic sub-group. If the participant could not be classified into the MWM, PRT, or neurodynamic treatment sub-group, the participant was classified into an EE sub-group and followed a lateral elbow EE protocol.

Following the third treatment, the participant was reassessed and discharged if the following discharge criteria were met: a report of phase 1 on the Nirschl Phase Rating Scale, met minimal clinically important difference (MCID) values for the GRC (two points),[40] DPA Scale (nine points acute, six points chronic),[38] and reported a worst pain score equal to, or less than, two out of ten on the NRS. If criteria were not met, the participant was re-evaluated using the TBC algorithm to determine the treatment classification for the next three visits. The participant was returned to their initial treatment sub-group for three more treatment sessions if the participant had met 50% of the MCID score value for each discharge outcome measure (NRS, GRC, DPA Scale, and PRTEE) and was appropriate based on re-evaluation findings. If the participant could not be re-classified into a manual therapy sub-group, the participant began the EE protocol. Following discharge, each participant was sent a one-month follow-up survey to determine changes in pain post discharge.

Treatment Post-Classification. If the participant was classified into the Mulligan Concept MWM sub-group, the participant was treated with the MWM for LE. Brian Mulligan recommended a lateral glide MWM initially; however, a medial glide was applied if the lateral glide did not produce improvement.[35] To perform the MWM, the participant was supine on a plinth with the elbow extended and supinated. The distal humerus was stabilized laterally with one hand as the proximal ulna was glided laterally and painlessly with the other hand. In the study treatment protocol, the lateral glide was applied and sustained during for sets of ten repetitions with pain-free elbow flexion/extension.

If the participant was classified into the PRT subgroup, the most painful TP was treated. The participant was passively moved into a POC while the TP was held. The TP was

held until the pulsation under the skin felt by the clinician dissipated, and the participant reported TP pain was rated a zero out of ten on the NRS.

If the participant was classified into the neurodynamic treatment subgroup, the participant performed neural sliders. The participant was instructed on the proper technique to perform a neural slider, which consisted of cervical lateral flexion movement simultaneously with elbow flexion and extension movements. While moving the head into cervical lateral flexion, the elbow was extended. As the elbow began to flex, the cervical spine was returned to neutral. Slow, controlled movements were performed for three sets of ten repetitions.

If the participant was classified into EE subgroup, the participant was treated with a standardized EE protocol. During EE treatment, the participant was seated next to a plinth with the effected elbow fully extended on the plinth, forearm pronated, wrist in full extension, and hand hanging over the edge of the plinth. The participant would flex their wrist slowly while counting to 30. The participant returned to the starting extended position with the help of the other hand and was instructed to continue with the exercise despite mild pain. If pain rated higher than a seven out of ten on the NRS during EE, the participant ceased the exercise. The EE protocol required the completion of three sets of 12 repetitions twice a day for up to 12 weeks.

Statistical Analysis. All data was analyzed using SPSS version 23.0 (SPSS Inc., Chicago, IL, USA). A Repeated Measures ANOVA (RM-ANOVA) was performed for initial, discharge, and one-month follow-up for NRS scores only. Paired *t*-tests were performed on DPA Scale and PRTEE, while descriptive statistics were computed for the GRC, to determine the effectiveness of the TBC algorithm from initial exam to discharge.

Mean differences from the initial visit scores and 95% confidence intervals (CIs) were calculated for the NRS, DPA Scale, and PRTEE for discharge. Cohen's *d* was also computed to determine the effect size, or maximum likelihood, of each outcome measure. For Cohen's *d*, an effect size of 0.2 to 0.3 was considered a "small" effect, around 0.5 a "medium" effect, and 0.8 to infinity, a "large" effect.[36]

Results. The six participants who participated in the study responded positively to a MWM at the elbow during initial exam and were classified into the Mulligan Concept MWM sub-group utilizing the TBC algorithm to discharge. The majority of participants reported chronic elbow pain (n = 4), with an average duration of symptoms of 59 weeks (SD = 60.56). The remaining participants (n =2), reported acute elbow pain, with average duration of symptoms of six days (SD = 2.24). No participants, including those who withdrew and were excluded, met the EE group criteria during initial exam or through completion of the TBC algorithm methodology through discharge from the study.

Of the six participants, three were discharged on the third visit, two were discharged on the sixth visit, and one participant voluntarily withdrew from the study. The participant who withdrew reported improvements on all outcome measures except the GRC after three treatment visits, but declined to further participate in the study. Of the remaining five participants, two received two rounds (i.e., 6 treatments) of treatment (i.e., MWM), while three participants received one round (i.e., three treatments) of treatment. Participants received a mean of 3.8 ± 1.57 treatments over a mean number of 8.6 ± 6.18 days until discharge. The discharge criteria of phase 1 on the Nirschl Phase Rating Scale, MCID for the GRC, and DPA Scale and worst pain score equal to or less than two out of ten on the NRS were met by all five participants who completed the study. (Table 5.7)

Numeric Rating Scale. Utilization of the TBC algorithm resulted in statistically significant improvements in pain; however, the results of the RM-ANOVA did not indicate a significant time effect, Wilks' Lambda = .004, F (2,1) = 127.00, p =.063, η^2 = .996. The mean changes in NRS scores from initial visit to discharge were statistically significant (M = 7.33, 95% CI[.588, 14.079] p =.042). The initial to one month follow up was also statistically significant (M = 7.00, 95% CI[2.58, 11.416], p = .020). A large effect size (.99) indicated that 99% of the variance in NRS scores could be explained by TBC algorithm use. The change in mean scores from discharge and one-month follow-up M = .333, (95% CI[2.883, 2.216] p = 1.00) was not statistically significant and indicated the improvements were maintained at one-month follow-up.

The mean difference from the initial visit to discharge (i.e., three or six treatments), and initial to one-month follow-up exceeded the MCID for the NRS prior to discharge for participants completing the study.[37] Following the initial treatment visit, 67% (four out of six) of the participants reported a MCID on NRS (initial M = 7.6 points, post-treatment M = 2.3 points). More clinically relevant, 67% (four out of six) of participants at discharge reported a resolution of their pain within three to six treatments. The remaining participant reported a NRS of one out of ten upon discharge and during follow-up survey. The participant who withdrew reported a worst pain score of five out of ten after three treatment visits, a decrease of three points from his initial NRS pain score of eight out of ten.

Disablement in the Physically Active Scale. The use of the TBC algorithm resulted in improved participant reported disablement. Statistically significant changes on the DPA Scale (M = 16.8, 95% CI[7.32 to 26.34] p = .006), with a large effect size (Cohen's d = 2.15) were found from initial evaluation to discharge. Although all participants (100%) met MCID

prior to discharge, 83% (five out of six) of the participants exceeded the MCID for acute conditions (nine points), which is greater than the MCID for chronic conditions (six points).[38] A MCID (six points) was reported by the participant who withdrew with his DPA Scale score dropping from a 37 to a 31 following three treatments.

Return to activity scores on the DPA Scale has not been established for persistent injury; however, a mean score (8.82 ± 6.71) for physically active patients returning from acute injury has been reported in the literature.[38] The mean DPA Scale score at discharge (M = 16.5 ± 10.03) for the current study at discharge was above the mean for acute return to play injuries previously reported; however, the current range of DPA Scale scores (R = 2 - 21) at discharge, was within the acute return to activity range (R = 0 - 23) and the uninjured range (R = 0 - 34) previously reported in the literature.[38]

Patient-Related Tennis Elbow Evaluation. The use of the TBC algorithm resulted in a significant decrease in PRTEE scores from initial exam to discharge with a mean change of M= 42.5 (95% CI[17.17 to 67.82], p = .008), and a large effect size (Cohen's d = 2.53). The mean change in instrument scores from initial exam to discharge exceeded the MCID value[39] for the PRTEE (7 points or 22% of baseline score [12.76]). At discharge, 50% of the participants reported a substantial change (11 points or 37% of baseline score [21.46]) following three treatments and 33% (2 out of 6) reported substantial change after six treatments on the PRTEE. The reported change in PRTEE, from the participant who withdrew, was 11 points, a MCID, following three treatments (initial = 56, post = 45).

Global Rating of Change. Analysis of the GRC outcome measure revealed a mean change score (M = 4.85 ±2.31) that met the MCID value prior to discharge and also exceeded the MCID value for "important improvement."[40] Additionally, 33% (two out of

six) of participants perceived their change as "a great deal better" in three treatments, while 50% (three out of six) achieved similar or greater changes in six treatments. The participant who did not complete the study reported no change in the GRC (zero).

Nirschl Phase Rating Scale. Initial Nirschl Phase Ratings[41] ranged from phase four to phase seven during the initial exam. Phase seven (constant rest pain that disturbs sleep) was reported by 33% (two out of six) of participants, while phase five (pain that is present during all activities and occurs with activities of daily living) was reported by 33% (two out of six) of participants and phase four (pain with activity that causes modification) was reported by 33% (two out of six) of participants. All participants (100%) who completed the study (n = 5) reported a phase one (pain after activity, which resolves within 24 hours) on the Nirschl Phase Rating Scale at discharge. The one participant who withdrew from the study reported a phase four on the Nirschl Phase Rating Scale at discontinuation of study, which was an improvement from the phase five score reported during the initial evaluation.

Discussion. Researchers recognize the complexity of tendinopathy and the inadequacies of the present method of assessment.[6,33] Currently, a clinical diagnosis of the state of tendon injury cannot be made utilizing present clinical evaluation methods.[6,33] Thus, a new model of clinical assessment for tendinopathy has been proposed using participant response to assessment methods to guide treatment selection.[33] Researchers utilizing treatment-based systems to classify and treat individuals with low-back pain have demonstrated improvement in pain and function, verifying the importance of properly matching treatments to individual symptom presentation.[42,43] Therefore, incorporating a TBC algorithm into assessment methodology may improve the classification and treatment

of LE, while indirect treatment interventions may prove to be more effective than traditional treatments when participants are matched to a particular treatment intervention.

The positive results observed in this study support the inclusion of a TBC system as an evaluation and treatment strategy for patients with LE. In the present study, classification and treatment utilizing the TBC algorithm produced effective pain resolution and improvements in function as measured by the various outcome measures. Moreover, those participants who received manual therapy techniques matched to their sub-group were discharged in less time compared to conservative treatment methods previously reported in the literature.[5,6,44-46]

Although no gold standard treatment has been established for LE, due to the effectiveness of EE protocols for patella and Achilles tendinopathy, EE is prescribed for patients presenting with LE.[23-29,44-46] Most EE protocols for LE range from four to twelve weeks and are frequently coupled with other modalities.[44] In a review of 12 research studies on the effectiveness of EE programs for treatment of LE, 60% incorporated adjunct therapies (e.g., stretching, icing, ultrasound, massage, heat) with EE, and reported a mean of 7.7 weeks of treatment. The frequency of required treatments ranged from three to seven per week.[44] Also, in the aforementioned studies, researchers reported grip strength deficits and functional impairments of the elbow still remaining at discharge.[44] In the current study, without using adjunct therapies, participants were discharged in less time (8.6 days ± 6.18) when compared to EE protocol time frames (four to twelve weeks) and 67% (four out of six) of participants who completed the study reported complete resolution of symptoms in three to nine days (three to six MWM treatments).

Within the tendinopathy literature, it is common for patients to be discharged without full resolution of pain following EE protocols.[44] Wen et al.[45] reported a VAS discharge score of 27 out of 100 following a four week EE program, while Martinez-Silvestrini et al.[46] reported a VAS discharge score of 24 out of 100 following a six week EE and stretching program. A score of 10mm on a 100mm scale is interpreted as no pain, and a score of 10 to 25mm on a 100mm scale is used as an acceptable status for patients with inflammatory or degenerative disorders.[47] In our study, 67% of the participants (four of six) achieved a MCID for pain after one treatment (initial M = 7.6 points, post-treatment M = 2.3 points, mean change 5.3 points on NRS), while the mean NRS discharge pain score was less than one (.2 out of 10). The preliminary results suggests utilization of a TBC algorithm to match and classify participants to an appropriate treatment may decrease NRS scores in an equal , or superior, fashion to the results of EE protocols found in the literature.[44-46]

The results from the current study also compared favorably to other studies conducted on the effectiveness of MWMs for the treatment of LE. In a single case study, a patient received four MWM treatments over a two-week period before reporting full resolution of pain associated with LE.[25] In another study conducted by Amro et al.,[48] 17 subjects reported a statistically significant change (mean change = 5.3 ± 0.9) in VAS pain rating to a final score of one out of ten following three MWM treatment sessions over four weeks. Additional therapy (i.e., self MWM exercise, Mulligan adjunct taping technique) was included in the MC treatment of LE in previous studies, which is a common recommendation to extend and ensure the long lasting therapeutic effect of MWM treatments.[49]In the current study, without using adjunct taping or prescribing self MWM exercises, 83% (five out of six) of the participants reported resolution of pain and return to

function at discharge in six treatments or less. Long term effects in our study are difficult to conclude due to limited participation (50%, three out of six) in the follow-up survey; however, of these three participants, two reported no change in pain (zero out of ten NRS), while the other reported a NRS rating of one out of ten.

As demonstrated in this case series, the use of a TBC algorithm may be more effective than traditional evaluation and treatment strategies for matching participants to specific interventions to optimize pain resolution and functional restoration. Although this study is only an initial exploration of a TBC algorithm for LE, its use may allow clinicians to identify participants who will respond to matched manual therapy interventions versus those who need an EE protocol. Additionally, it may be possible to alter the sequence of the TBC algorithm treatments or include additional treatment paradigms to further expand the effectiveness of the TBC algorithm for more diverse participant presentations of LE.

Despite the positive outcomes, a number of potential limitations were present in this study. First, the current study had no control group and participants were not randomly assigned. Although attempts were made to decrease bias, bias may have been introduced as the participants and primary researchers were not blinded to the procedures or the collection of outcome measures.. The lack of a comparison group limits generalization of the findings and the value of the TBC as a whole cannot be determined because every participant was classified into the MC subgroup. Additionally, participants were discharged who were not pain-free based on standards found in current literature (two out of ten on the NRS)[14,44-46]. There are several MC techniques available for the treatment of LE, along with further recommendations to adjust direction of force or volume of application to sustain treatment effects. Without fully following the recommendations and principles of the Mulligan

Concept, it is not known if these patients would have reached full resolution of their pain with further treatment. In the current study, long-term treatment effects cannot be definitively determined due to the small sample size that completed the follow-up survey and the limited duration (i.e., one month) of the follow-up period. Lastly, the sampling of participants may not have been fully representative of LE patients, and those who entered the study may have felt compelled to improve.

While this is the initial examination of the TBC algorithm for LE, further studies are needed to confirm the positive outcomes reported in this case series compared to other interventions. Revising the order of interventions or adding other treatment paradigms may enhance the effectiveness of the TBC algorithm. Future research should include comparison of a control group to the TBC algorithm, while also considering other intervention strategies to optimize participant sub-grouping. Although positive findings for pain were reported at one-month follow-up, subsequent studies should be conducted to investigate the long-term effects of using the TBC algorithm. Additionally, it may be beneficial to include diagnostic imaging or histological exams to determine tendon physiological response to treatment using the TBC algorithm.

References

1. Hong, Q., Durand, M., & Loisel, P. Treatment of lateral epicondylitis; where is the evidence. *Joint, Bone, Spine.* 2004;71:369-373.
2. Hamilton, P. The prevalence of humeral epicondylitis a survey in general practice. *J R Coll Gen Pract.* 1986; 36:464-465.
3. Smidt, N., Lewis, M., van der Windt, D., Hay, E., Bouter, L., & Croft, P. Lateral epicondylitis in general practice: course and prognostic indicators of outcome. *The Journal of Rheumatology*. 2006; 33(10): 2053-2059.
4. Vicenzino, B. Lateral epicondylalgia: a musculoskeletal physiotherapy perspective. *Manual Therapy.* 2003; 8(2): 66-79.
5. Johnson, G., Cadwallader, K., Scheffel, S., & Epperly, T. Treatment of lateral epicondylitis. *Am Fam Physician.* 2007; 76(6): 843-848.
6. Coombes, B., Bisset, L., & Vicenzino, B. A new integrative model of lateral epicondylalgia. *Br J Sports Med.* 2009; 43: 252-258.
7. Nirschl, R., Elbow tendinosis/tennis elbow. *Clin Sports Med.* 1992; 11: 851-870.
8. Alfredson, H., Ljung, B., Thorsen, K., & Lorentzon, R. In vivo investigation of ECRB tendons with microdialysis technique-no signs of inflammation but high amounts of glutamate in tennis elbow. *Acta Orthop Scand.* 2000; 71: 475-479.
9. Fredberg, U., & Stengaard-Pedersen, K. Chronic tendionopathy tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. *Scand J Med Sci Sports.* 2008; 18: 3-15.

10. Uchio, Y., Ochi, M., Ryoke, K., & Lorentzon, R. Expression of neuropeptides and cytokines at the exstensor carpi radialis brevis muscle origin. *J Shoulder Elbow Surg.* 2002; 11: 570-575.

11. Ljung, B., Alfredson, H., & Forsgren, S. Neurokinin 1-receptors and sensory neuropeptides in tendon insertions at the medial and lateral epicondyles of the humerus. Studies on tennis elbow and medial epicondylalgia. *J Orthoo Res.* 2004; 22: 321-327.

12. Wang, J., Iosifidis., M. & Fu, F. Biomechanical basis for tendinopathy. *Clinical Ortho & Related Res.* 2005; 443: 320-332.

13. Assendelft, W., Hay, E., Adshead, R., & Bouter, L. Corticosteroid injections for lateral epicondylitis: a systematic overview. *Br J Gen Pract.* 1996; 46: 209-216.

14. Smidt, N., Assendelft, W., van der Windt, D., Hay, E,. Buchbinder, R., & Bouter, L. Corticosteroid injections for lateral epicondylitis: a systematic review. *Pain*, 2000; 96: 23-40.

15. Boddeker, I., & Haake, M. Extracorporeal shockwave therapy in treatment of epicondylitis humeri radialis. A current overview. *Orthopade (German).* 2000; 29: 463-469.

16. Bisset, L., Paungmali, A., Vicenzino, B., & Beller, E. A systematic review and meta-analysis of clinical trials and physical interventions for lateral epicondylalgia. *Br J Sports Med.* 2005; 39: 41-422.

17. Green, S., Buchbinder, R. Barnsley, L., Hall, S., White, M., Smidt, N., & Assendelft, W. *Cochrane Database Syst Rev.* 2001. DOI: 10.1002/14651858.CD003686.

18. Smidt, N., van der Windt, D., Assendelft, W., Deville, W., Korthals-de Bos, I., & Bouter, L. Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomized controlled trial. *Lancet.* 2002; 359: 657-662.

19. Buchbinder, R., Green, S., Youd, J., Assendelft, J., Barnsley, L., & Smidt, N. Systematic review of the efficacy and safety of shock wave therapy for lateral elbow pain. *J Rheumatol.* 2005; 33: 1351-1363.

20. Stasinopoulos, D., Stasinopoulow, K., & Johnson, M. An exercise programme for the management of lateral elbow tendinopathy. *Br J Sports Med.* 2005; 39: 944.947.

21. Fedorczyk, J. Tennis elbow: blending basic science with clinical practice. *J Hand Ther.* 2006; 19: 146-153.

22. Croisier, J., Foidart-Dessalle, M,. Tinant, F., Crielaard, J., & Forthomme, B. An isokinetic eccentric programme for the management of chronic lateral epicondylar tendinopathy. *Br J Sports Med.* 2007; 41: 269-275.

23. Abbott, J., Patla, C., & Jensen, R. The initial effects of an elbow mobilization with movement technique on grip strength in subjects with lateral epicondylalgia. *Man Ther.* 2001; 6: 163-169.

24. Vicenzino, B., Paungmali, A., Buratowski, S., & Wright, A. Specific manipulative therapy treatment for chronic lateral epicondylalgia produces uniquely characteristic hypoalgesia. *Man Ther.* 2001; 6: 205-212.

25. Vicenzino, B., & Wright, A. Effects of a novel manipulative physiotherapy technique on tennis elbow: a single case study. *Man Ther.* 1995; 1: 30-35.

26. Vicenzino, B. Physiotherapy for tennis elbow. *Evid Based Med.* 2007; 12: 37-38.

27. Vicenzino, B., Collins, D., & Wright, A. The initial effects of a cervical spine manipulative physiotherapy treatment on the pain and dysfunction of lateral epicondylalgia. *Pain.* 1996; 68: 69-74.

28. Baker, R., Riper, M., Nasypany, A., & Seegmiller, J. Evaluation and treatment of apparent reactive tendinopathy of the biceps brachii. *Int J Athl Ther Train.* 2014; 19(7) 14-21.

29. Matocha, M., Baker, R., Nasypany, A., & Seegmiller, J. Effects of neuromobilization on tendinopathy. Part II. *IJATT.* 2015; 20(2): 41-47.

30. Alfredson, H. The chronic painful Achilles and patellar tendon: research on basic biology and treatment. *Scand J Med Sci Sports.* 2005; 15(4): 252-259.

31. Rees, J., Wilson, M., & Wolman, L. Current concepts in the management of tendon disorders. *Rheumatology (Oxford).* 2006; 45(5): 508-521.

32. Kaux J-F, Forthomme, B., Geoff, C., Crielaard, J., & Croisier, J. Current opinions on tendinopathy. *J Sports Sci Med.* 2011; 10(2): 238-253.

33. Lewis, J. Rotator cuff tendionopathy/subacromial impingement syndrome: is it time for a new method of assessment? *Br J Sports Med.* 2009; 43: 259-264.

34. D'Ambrogio, K., & Roth, G. *Positional Release Therapy Assessment and Treatment of Musculoskeletal Dysfunction.* 1997. St Louis, MO: Mosby.

35. Mulligan, B. *Manual therapy—'NAGS', 'SNAGS', 'MWMS', etc*. 6th ed. Plane View Services Ltd. Wellington, New Zealand.

36. Vincent, W., & Weir, J. *Statistics in kinesiology.* 2012. Champaign, IL: Human Kinetics.

37. Farrar, J., Young, J., LaMoreaux, L., Werth, J., & Poole, R. Clinical importance of changes in chronic pain intensity measured on an 11-point numerical pain rating scale. *Pain.* 2001; 94(2): 149-158.

38. Vela, L., & Denegar, C. The Disablement of the Physically Active Scale, part II: The psychometric properties of an outcome scale for musculoskeletal injuries. *Journal of Athletic Training.* 2010; 45(6): 630-641.

39. Poltawski, L., & Watson, T. Measuring clinically important change with the patient-related tennis elbow evaluation. *J Hand Therapy.* 2011; 3: 52-57.

40. Kamper, S., Maher, C., & 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.

41. Nirschl, R., & Ashman, E. Elbow tendinopathy: tennis elbow. *Clin Sports Med.* 2003; 22(4): 813-836.

42. Brody, K., Baker, R., Nasypany, A., & May, J. The MyoKinesthetic System. Part II. Treatment of chronic low back pain. *IJATT*. 2015; 20(5): 22-28.

43. Delitto, A., Erhard, R., & Bowling, R. A treatment-based classification approach to low back syndrome: Identifying and staging participants for conservative treatment. *Physical Therapy*. 1995; 75(6): 470-485.

44. Cullinane, F., Boocock, M., & Trevelyan, F. Is eccentric exercise an effective treatment for lateral epicondylitis? A systematic review. *Clinical Rehabilitation.* 2014; 28(1): 3-19.

45. Wen, D., Schultz, B., Schaal, B., Graham, S., & Kim, B. Eccentric strengthening for chronic lateral epicondylosis: a prospective randomized study. *Sports Health.* 2011; 3(6): 500-503.

46. Martinez-Silvestrini, J., Newcomer, K., Gay, R., Schaefer, M., Kortecein, P., & Arendt, K. Chronic lateral epicondylitis: comparative effectiveness of a home exercise program including stretching alone versus stretching supplemented with eccentric or concentric strengthening. *J Hand Ther.* 2005; 18:411-420.

47. Mannion, A., Balague, F, Pellise, F., & Cedraschi, C. Pain measurement in patients with low back pain. *Nature Clinical Practice Rheumatology*; 2007; 3(11): 610-618.

48. Amro, A., Diener, I., Bdair, W., Hameda, I., Shalabi, A., & Ilyyan, D. The effects of Mulligan mobilization with movement and taping technique on pain, grip strength, and function in patients with lateral epicondylitis. *Hong Kong Physiotherapy Journal.* 2010; 28: 19-23.

49. Hing, W., Hall, T., Rivett, D,. Vicenzino, B., & Mulligan, B. *The Mulligan Concept of manual therapy: textbook of techniques.* 2015. Elsevier, Australia: Churchill Livingstone.

50. Nirschl, R., & Sobel, J. Conservative treatment of tennis elbow. *Phys Sports Med.* 1981; 9: 43-54.

51. Childs, J., Piva S., & Fritz, J. (2005). Responsiveness of numeric pain rating scale in patients with low back pain. *Spine*, 30(11), 1331-1334.

52. Williamson, A., & Hoggart, B. (2005). Pain: A review of three commonly used pain rating scales. *Journal of Cliinical Nursing*, 14, 798-804.

53. Rompe, J., Overend, T., & MacDermid, J. (2007). Validation of the patient-rated tennis elbow evaluation questionnaire. *J Hand Therapy* 20(1), 3-10.

Table 5.5.
Description of Inclusion & Exclusion Criteria

Description of Inclusion & Exclusion Criteria	
Inclusion Criteria	**Exclusion Criteria**
Must present with pain on the tendon	Receiving a cortisone injection < 6 weeks
Experiencing pain or dysfunction associated with activities	Post-operative instructions excluding from treatment
Morning stiffness	Current healing of or suspected fracture
Pain that subsides after warming up	Current dislocation
Previous diagnosis of tendinopathy	Pregnant
Treated unsuccessfully with conventional treatments (rest, NSAIDS, physical therapy).	Taken fluoroquinolones-ciprofloxacin < 12 months
Pain present with tendon loading at beginning of exercise, subsides with continued activity, can progress to pain during activity - required to stop, impaired function, tendon focal or generalized swelling	Previous history of cervical surgery
	Currently enrolled in physical therapy program

Table 5.6.
Description of Outcome Measures

Description of Outcome Measures		
Outcome measure	**Construct**	**Description**
Nirschl Phase Rating Scale[46]	Pain	Seven phase classification system designed to delineate the significance of pain. Phase 0 (no pain or soreness) to Phase 7 (pain disrupts sleep on a consistent basis. Pain is aching in nature and intensifies with activity).Utilized to assess severity, function, and ability to play sports and participate in daily living activities in patients with tendon pain. No MCID has been established for this scale.
Numeric Rating Scale (NRS)[37,47,48]	Pain Intensity	11-point scale, with 0 representing no pain and 10 indicative of extreme pain. The NRS scores were recorded before and after each treatment and worst score was used for reporting. The minimal clinically important difference (MCID) was a decrease of 2 points. When compared to other pain rating scales, the NRS was valid, reliable, and more sensitive than the verbal rating scale

Table 5.6.
Description of Outcome Measures (Continued)

Description of Outcome Measures		
Disablement in the Physically Active (DPA) Scale [38]	Disability	16-item questionnaire related to the following items: impairment, functional limitation, disability, and quality of life. Each statement was rated by the patient on a scale of 1 (no problem) to 5 (severe), with a maximum score of 64 and minimum score of 0. The MCID was a decrease of 9 points for acute injuries and 6 points for chronic injuries. The DPA Scale is a valid, reliable, and responsive instrument for global assessment of disability.
Patient Rated Tennis Elbow Evaluation[39,49]	Disability and Pain	The PRTEE questionnaire is designed to assess chronic lateral elbow tendinopathy. Pain and function are based on average arm symptoms over the past week on a 0 to 10 point scale in two categories. Worst pain subscale may add to 50 points. Specific and usual activities may add to 100 points. Total point may be 100, indicating extreme pain and disability. The MCID for the total PRTEE score has been established as 22% of the baseline score. The PRTEE was administered before initial treatment and at the third visit for each participant. The PRTEE is a reliable, reproducible, and sensitive instrument for assessment of chronic lateral elbow tendinopathy.

Table 5.7.
Changes in Patient Reported Outcome Measures

Changes in Patient Reported Outcome Measures												
Patient Number			**Function (PRTEE) (0-100)**			**Pain (NRS) (0-10)**			**Disability (DPAS) (0-64)**			**Number of Treatments**
	Age	**Gender**	**Pre**	**Post**	**Change**	**Pre**	**Post**	**Change**	**Pre**	**Post**	**Change**	
1	62	M	37	21	16	6	0	6	30	21	9	3
2	36	F	81	14	67	9	0	9	40	9	31	6
3	56	M	65	4	61	7	0	7	20	2	18	3
4	18	M	41	0	41	7	0	7	32	17	15	2
5	16	M	68	9	59	9	1	8	41	19	22	6
6*	44	M	56	45	11	8	5	3	37	31	6	3
Mean	38.6		58.0	15.5	42.5	7.7	1.0	7.3	33.3	16.5	16.8	3.8
SD	17.4		15.3	14.8	22.0	1.1	1.8	1.9	7.16	9.16	8.27	1.6
P value					.01			.04			.01	
Cohen's d					3.85						4.06	

SD=Standard deviation
*Did not complete study

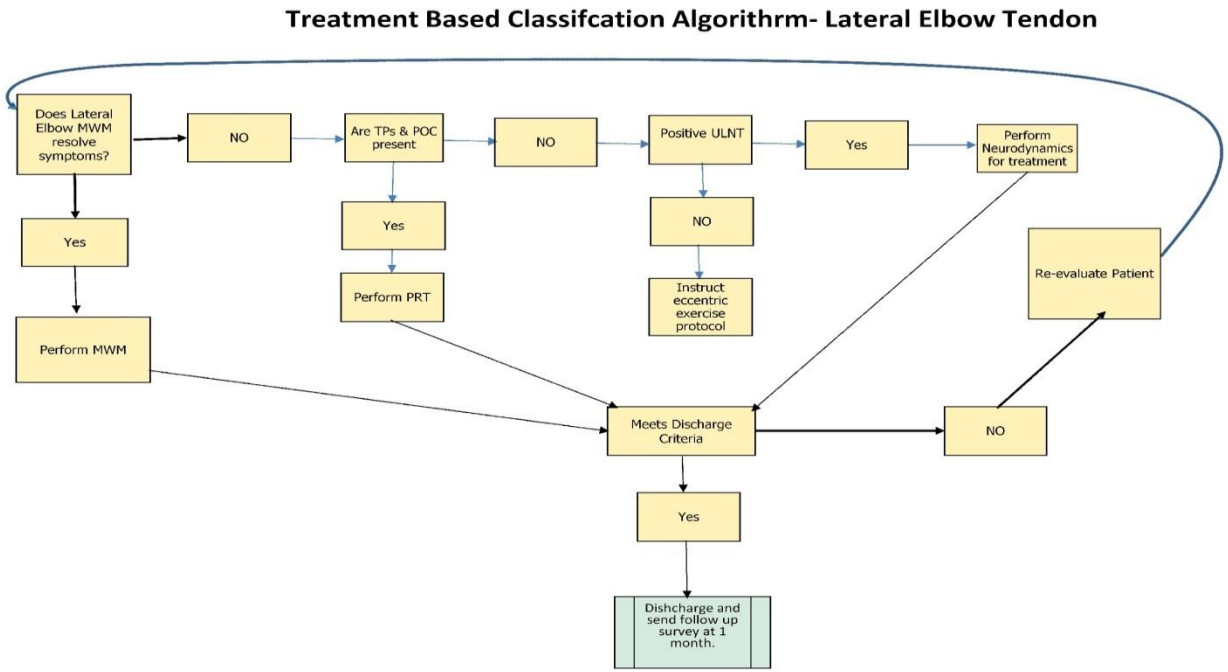

Figure 5.6.
Lateral Elbow Treatment-Based Classification Algorithm