THE EFFECTS OF A MANUAL THERAPY TREATMENT BASED CLASSIFICATION ALGORITHM ON TENDINOPATHY: A DISSERTATION OF CLINICAL PRACTICE IMPROVEMENT

A Dissertation
Presented in Partial Fulfillment of the Requirements for the
Degree of Doctor of Athletic Training
with a
Major in Athletic Training
in the
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by

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AUTHORIZATION TO SUBMIT DISSERTATION

This dissertation of Monica Matocha, submitted for the degree of Doctor of Athletic Training with a Major in Athletic Training and titled "THE EFFECTS OF A MANUAL THERAPY TREATMENT BASED CLASSIFICATION ALGORITHM ON TENDINOPATHY: A DISSERTATION OF CLINICAL PRACTICE IMPROVEMENT," has been reviewed in its final form. Permission, as indicated by the signatures and dates given below, is now granted to submit final copies to the College of Graduate Studies for approval.

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ABSTRACT

The Dissertation of Clinical Practice Improvement (DoCPI) was designed to document each Doctorate of Athletic Training (DAT) student's individual journey to scholarly advanced practice. Self-reflection brought forth the means to identify strengths and weaknesses of one's clinical practice. Through reflection of patient care outcomes, students were able to identify trends specific to their clinic and determine personal growth as a clinician. The use of a treatment based-classification system to determine the most appropriate treatment for each patient was the leading trend that emerged in my clinic. The DoCPI concluded with a clinical based research project designed apriori to solve a local clinical problem; particularly, this research project was designed to restore function and decrease pain in patellar, Achilles, and lateral elbow tendinopathy patients.

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DEDICATION

To my husband, Matthew, thank you for your support and dedication to helping me finish my DAT degree. I was only able to complete the DAT program because of your support.

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CHAPTER 1

Narrative Summary

Professional practice doctoral (PPD) programs provide practicing professionals with necessary preparedness for their careers (Willis, Inman, & Valenti, 2010). Although each PPD program is unique, there are many aspects that remain the same across disciplines, including: coursework that prepares students for work in the field; faculty comprised of practicing professionals; an integration of coursework, fieldwork, research; and dissertations completed "in the field" (Willis et al., 2010). Professional practice doctoral programs require students to complete either a dissertation or a capstone project, either of which is intended to demonstrate improvement in fieldwork (Willis et al., 2010).

The Doctor of Athletic Training (DAT) program, which is a PPD program, is the first academic doctorate in athletic training with a focus on clinical practice and patient care. The program is intended for the student who aims to become a scholarly practitioner through personal, clinical, and professional growth. A significant portion of this growth, as well as the individual's overall experience in the DAT program, is documented in the Dissertation of Clinical Practice Improvement (DoCPI).

The DAT program is designed to teach each student how to advance his or her clinical practice in a way that is relevant and personal. The program is designed to increase the student's breadth of knowledge of techniques/paradigms that most athletic trainers do not utilize in practice, while also increasing the student's depth of knowledge by allowing for more focused study in his or her area of advanced practice. As part of this process, the student applies newly acquired knowledge to his or her clinical practice using an action research philosophy, or a philosophy that combines the collection of patient outcomes with critical

reflection and an analysis of patient care to address local problems in clinical practice. As the student gains a more informed perspective of his or her knowledge and of the efficacy of using a particular treatment paradigm, a decision can be made on how to utilize and study this technique in daily practice. Each student will have a unique journey, due to individual clinical situations (e.g., local problems, patient population) and the student's experiences using the new paradigms.

Ultimately, the success of a clinician or paradigm is decided by the patient outcomes produced in clinical practice. A clinician establishes practice-based evidence (PBE) by analyzing and reflecting on patient outcomes. In general, however, the medical community has embraced the evidence-based practice (EBP) trend wherein a clinician makes decisions based on the best possible research available in combination with clinical expertise and individual patient needs (Hurley, Denegar, & Hertel, 2011). Some clinicians have begun to utilize an "evidence only" practice (Krzyzanowics, May, & Nasypany, June 2014), thereby removing potential positive interventions from their practice. By generating PBE, a clinician is able to determine whether or not the interventions he or she is choosing for patients are appropriate. Analyzing and reflecting on patient outcomes also allows a clinician to use evidence from their practice to determine which areas of practice need improvement. When needed improvement is identified, the clinician can begin an action research philosophy to produce more efficient practice. When PBE is coupled with EBP, a clinician utilizes the clinical expertise established from known clinical outcomes, in combination with published literature and individual patient needs, to provide more optimal care in clinical practice.

The need for generating PBE results from the gap between basic science research and its applicability for individual practicing clinicians. According to Green (2008), "it takes 17 years to turn 14 percent of original research to the benefit of patient care." The gap between laboratory studies to patient care can be attributed to the length of time it takes to prepare and complete a laboratory study to the acceptance and print of the analyzed results. Throughout the process of laboratory research, advances in techniques occur and clinicians are not able to utilize potentially beneficial interventions for their patients if the clinicians wait for laboratory scientist to generate results. Increasing translational research would assist in closing the gap between laboratory research and clinicians, as would provide incentives for clinicians, placing more emphasis on external validity, and performing more action research. Basic science research is not aimed to solve a local problem, as action research is; therefore, a clinician could use the action research philosophy to produce meaningful PBE for his or her practice. The completion of additional action research could potentially decrease the time between original research being conducted to the benefits of a patient. As noted earlier, action research combines critical reflection with an analysis of patient care and the collection of patient outcomes. Critical reflection allows a clinician to examine his or her practice to determine whether or not it is "satisfactory" (McNiff, 2013). If a particular part of the clinician's practice is deemed "unsatisfactory," the clinician then has the knowledge and ability to create a new action research study to improve clinical practice (McNiff, 2013).

Action research can increase external validity by bringing research to the clinic. In doing so, this allows the clinician/researcher to avoid the extremely strict barriers of laboratory-setting research while assessing authentic patients from backgrounds that, in their variety, better represent the patient population seen by clinicians (Green, 2008). Increasing

emphasis on external validity for research grants and journal publication could increase interest from researchers to conduct research closer to reality of clinician needs (Green, 2008). Incentives to clinicians could also potentially encourage them to participate in scholarly activities that promote EBP continuing education (Green, 2008).

The DAT program faculty encourages each student/clinician to use an action research philosophy to guide clinical examination of their local problems and become scholarly practitioners by disseminating outcome findings. Clinician or disease-oriented outcomes are collected to provide more information about a patient's injury (e.g., blood pressure), while patient-oriented outcomes are directed toward the interest of the patient (e.g., pain, severity of symptoms) (Hurley, Denegar, & Hertel, 2011). Because of my desire to obtain a better understanding of my patients' perceptions of their symptoms and treatment responses, I chose to focus on the collection of patient-oriented outcomes.

To determine the effectiveness of my clinical practice and the benefit I provided my patients, I began collecting and reflecting on outcomes from all of my patients. During the DAT program, I learned several intervention techniques/paradigms that I had not previously been exposed to and I lacked knowledge on my effectiveness utilizing these new skills in clinical practice. Collecting outcomes became a vital part of determining the effectiveness of my application of the following new interventions: Instrument Assisted Soft Tissue Mobilization (IASTM), Mulligan Concept, Positional Release Therapy (PRT), Reactive Neuromuscular Training (RNT), Selective Functional Movement Assessment (SFMA), Total Motion Release (TMR) and Myokinesthetics (MYK). I was also able to incorporate neurodynamics, a long-held interest of mine that involves the movement of the nervous system on the musculoskeletal system, into my clinical practice at a deeper, more advanced

level than had previously been possible as a result of studying my use of the technique. The utilization of the aforementioned techniques improved my clinical practice and helped me progress toward becoming an advanced practitioner.

An advanced practitioner is defined as a clinician with a minimum of five years of scholarly practice who has a depth and breadth of knowledge in all athletic training domains, incorporates PBE and EBP into their individual practice, and has a focus in athletic training of a smaller area outlined in the clinician's Plan of Advanced Practice (PoAP) (Nasypany, Seegmiller, Baker, July 2013). Upon starting the DAT program, I was encouraged to create a PoAP to guide my continued clinical practice and educational endeavors. The PoAP provides direction for continued development on the path to expert practice. In addition to outlining continued professional development in clinical practice, the PoAP also includes delineation of research interests, patient care philosophies, teaching philosophies, and professional goals. As noted in Chapter 2, my PoAP is a fluid document that will change over the next 5 to 10 years as I meet my goals and create new goals.

One of the goals I have set in my own practice is to consistently collect and analyze patient outcomes to continually improve patient care and identify trends to articulate in my scholarly practice. Chapter 3 is an overview of the patient outcomes I collected during my time in the DAT program and it includes a semester-to-semester reflection on my growth as a clinician. As a result of this analysis process, I was able to identify a local problem (tendinopathy) and work with my dissertation group to develop a treatment-based classification algorithm to help determine the appropriate treatment for patients with tendinopathy.

To gain a deeper understanding of tendinopathy and our treatment algorithm, the dissertation group conducted a thorough literature review. The literature reviews conducted to increase my depth and breadth of knowledge in this applied research area. As a result, we gained a new perspective on an injury that ails much of the athletic and general populations and were able to design a study to assess the effectiveness of our proposed treatment algorithm to address the local problem. The literature review on tendinopathy can be found in Chapter 4.

The group dissertation followed an action research philosophy, rather than utilizing a true action research design. Action research is designed to bring research closer to the patient in regards to patient care; therefore changing the design of the research can be completed as needed. The tendinopathy research followed this philosophy in the initial design; however, for consistency in the algorithm and research methodology across four clinicians in four clinics across the United States, more traditional methods had to be blended into our applied research design. The altered design allowed for a meaningful collaborative research project that had both internal and external validity, while creating a research network of multiple clinicians in the DAT who had an interest in addressing a similar local problem. In Chapter 5, I present the methods of the algorithm and the results of our studies on patellar, Achilles, and lateral elbow tendinopathies.

The DAT program, as a PPD for athletic trainers, has begun to advance the athletic training profession by training expert clinicians who do not distinguish between serving as a clinician and a scholar. Expert clinicians are found to utilize professional experience, reflection and scholarly practice in their clinical decision making (Wainwright, Shepard, Harman, & Stephens, 2010; 2011), which are hallmark characteristics of DAT students and

are presented in the DoCPI. The DoCPI serves as evidence of a plan to continue to grow as an athletic trainer (Chapter 2), analyze practice (Chapter 3), and demonstrate scholarly practice (Chapters 4 and 5). The DoCPI serves as evidence for my development as an athletic trainer with advanced skills as a researcher and clinician working towards advanced practice.

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CHAPTER 2

PLAN OF ADVANCED PRACTICE: ESTABLISHED AUGUST 2015

Current Clinical Competence

Reflection on Professional Experience and Development

In 2005, I graduated from Texas Lutheran University, a small private school, with a Bachelor of Science degree in athletic training. Upon graduation, I decided to work toward a master's degree while also working full-time for a physical therapy clinic that contracted me to an area high school. Soon after I began working for the clinic, however, I decided my employment situation was not allowing me the time I needed to complete my degree within the two-year time limit I had set for myself. Therefore, I made a decision to quit my job, and I applied for a graduate assistant position at Texas State University (TXST). I received the assistantship, which gave me the opportunity to work with the TXST softball team while also working to complete my Master of Education degree in two years.

After graduating from TXST, I was faced with choosing between two job opportunities: one at the high school level and one at the NCAA Division II level. For many reasons, I chose to work at the high school. I met some amazing athletic trainers when I worked at the high school; however, there were several times I regretted my decision to accept the job. The setting left me feeling unchallenged and frustrated—mostly because I felt I was missing out on something. Due to a lack of knowledge, I felt incapable of properly helping my patients. After searching for three years and having several discussions with family members and mentors, I decided the DAT program at the University of Idaho offered the academic and clinical opportunities I had been looking for to advance my practice. Although I wanted to explore the educational aspect of athletic training, I was not ready to

give up on clinical practice. The DAT seemed to have everything I was interested in: a challenging academic and clinical program, an appealing location in Idaho, and a curriculum I could apply as a clinician.

Much to my satisfaction, the DAT program has proven to be as worthwhile as I had hoped it would be. The program has led me to job opportunities at Texas State University that have been highly beneficial to me; I now have the necessary time I have needed to improve my patient care. The DAT was the new beginning and challenge that I had been looking for, both academically and clinically.

Reflection on Current Knowledge

When I first applied to the DAT, I was certain that I was, at the very least, a novice in my profession. I did not consider myself to be an expert on any level; however, I felt that I had more knowledge than other graduating professionals in my field. I was confident in my evaluation skills and in my ability to multitask. Because I had worked at a high school for five years prior to my enrollment in the DAT, I knew how to successfully perform an evaluation and administer rehabilitation on two or more patients at the same time —a skill that I believed offered some proof of my abilities as a clinician. When classes began, however, I learned that many expert clinicians spend more individual time with patients than do novice clinicians (Resnik & Jensen, 2003; Wainwright, Shepard, Harman, & Stephens, 2010). The aforementioned helped me to realize that I did not manage my time with my patients as an expert would—a fact that led me to understand that I needed to reevaluate my perceptions of what was required of an advanced practice clinician.

When I analyzed my practice with a critical eye, I determined that I was not competent in many clinical aspects of my profession. I had been doing almost everything wrong. My original Plan of Advance Practice (PoAP) consisted mainly of areas where, upon examination, I realized were some of my weaknesses as a clinician: foundational knowledge, patient education/communication, writing, and appropriate evaluations (Appendix A, Fall 2013 PoAP). My analysis of my practice helped to remind me of the importance of properly evaluating and classifying a patient's injury. When such work is accomplished, it can lead a clinician to make better decisions about patient treatment. I was also reminded that the foundational knowledge I had learned in my undergraduate education was still important and should not be forgotten. My previous foundational knowledge was the base upon which I could expand both the depth and breadth of my future knowledge.

To regain strength and confidence in the content I had already learned, I watched Northeast Seminar videos on anatomy, utilized neuroanatomy websites, reviewed basic knowledge via textbooks, and taught basic anatomy and physiology courses. The challenge to explore interventions throughout the DAT program helped to foster my foundational knowledge and increased my breadth of knowledge regarding new treatment paradigms. Before my first month-long summer session had ended—a session in which I had learned more than I had during several years of clinical work—I had gained a new mindset regarding patient care. I was rejuvenated and ready to try the new concepts I had learned. Everything changed, however, on my way home to Texas from Idaho. I was offered a position as a lecturer at TXST and would no longer have direct access to patients. As a result, I would no longer be able to collect patient data in the way I had planned. Perhaps more discouraging was

the fact that taking this position at TXST would render the dissertation topic that I had chosen impossible.

Despite having to search for patients and ask permission to see specific individuals, I was eager to try the new concepts I had learned thus far in the DAT program. I wanted to prove that I could help patients by decreasing their pain and increasing their function in less time than was previously thought possible. After only three months, I changed attending clinicians due to unforeseen circumstances. The change could not have been more advantageous for me. My new attending clinician was interested in learning more about the concepts I was interested in, while still challenging me on the knowledge and skills I had already attained. The challenge helped me to become more confident in my decision-making skills, and it has helped me develop confidence in my ability to explain certain concepts. The challenges my attending clinician and I have encountered have sparked conversations amongst faculty and staff and have led me to guest lecture for a graduate manual therapy class at TXST on neurodynamics and PRT.

Having regained my confidence in both my knowledge and practice, I narrowed the areas in which I wished to become an advanced practitioner: *tendinopathy* and *pain*. As I was introduced to more interventions targeting pain in the DAT program, I found that I was more interested in pain itself. Understanding the brain and nervous system is part of understanding the pain process and I realized neurodynamics was not always the best intervention choice for many patients who were in pain. For example, I explored MYK for low back pain patients and for patients with radiculopathy.

As I continue to comprehend more aspects of the pain experience, I would like to educate other clinicians on pain and the pain process. In conjunction with educating clinicians on pain, I want to be able to explain why patients with apparent tendinopathy have tendon pain. Some researchers have found pain chemicals, such as glutamate, in painful tendons (Alfredson & Lorentzon, 2002). Being able to explain and understand why a patient has pain, such as an increase in pain chemicals delivered by an increase in neural activity, should lead to better intervention decisions than my pre-DAT intervention choices of rest, ice, electrical stimulation, non-steroidal anti-inflammatory drugs, and stretching.

Reflection on Strengths

During my first year-and-a-half in the DAT program, I developed confidence in my clinical practice and in myself once more. Prior to the DAT program, I tried multitasking by treating and performing rehabilitation with two or more patients and never systematically reflected on patient care. Learning to be present with patients and taking the time to truly listen to their complaints, along with collecting outcome measures, has helped me to improve my intervention decision-making skills. My new job at TXST allowed me time to reflect on each patient case (e.g., outcome measures) prior to the patient's return visit, to determine the next appropriate step in treatment. Due to patient reflection, my patient care improved and I was able to discharge patients in fewer visits each semester (Chapter 3).

I have become diligent in educating athletic training students and patients in the concepts I have learned, including neurodynamics, chronic pain patient education, PRT, MYK, and the Mulligan Concept. I am the most comfortable with neurodynamics, the Mulligan Concept and patient education; this is likely due to more experience in these areas, along with the simplicity of the Mulligan Concept. Myokinesthetics^{TM} is the newest paradigm

to me; however, I have become confident in a short period of time (six months) with the postural assessment and treatments. I utilize PRT the least frequently, mainly due to the simplicity of the other interventions. I am confident, however, after two courses in PRT and studying my patient outcomes to assess my effectiveness using it, that I am performing it correctly. Although most of the interventions are relatively new to me, with my education and knowledge, I am able to effectively educate students, clinicians, and patients on the paradigms. I believe it is vitally important for all parties to understand the technique I employ, along with my rationale for utilizing that technique. The athletic training students are eager to receive and understand new information, and their curiosity encourages me to make sure I thoroughly understand the concepts that influence the treatments I use on my patients. Many of my patients are pursuing undergraduate degrees and hope to study physical therapy someday. Naturally, these patients are just as curious as the athletic training students. As a result of working with both athletic training students and patients, I have developed the ability to articulate my decision-making processes and to explain why I choose particular interventions. Patient knowledge can aid in the effectiveness of the intervention chosen (Street, Makoul, Arora, & Epstein, 2009); therefore, the desire to increase my patients' knowledge about their injuries was an important development for my practice. For example, I had a patient with unpredictable pain who had seen several clinicians and physicians prior to our appointment. The patient did not believe that I would be able to help him. After validating the patient's pain and educating the patient on what I was going to do and why, the patient walked out of the clinic, on the first day, pain free.

I have not shied away from using an intervention simply because I do not feel confident in being able to explain the theory behind it or the reported mechanism of action. For example, the use of MYK has allowed me to discharge patients with back pain and/or radiating pain within three visits. At this time, however, I do not fully understand how the intervention is helping me produce these outcomes. Although I feel that I need to read more about MYK and find a way to better articulate what it is to both professionals and patients, I continue to use the concept. I utilize MYK, despite my limited knowledge with the technique, because it poses little to no risk to patients and has produced positive outcomes in my practice (Chapter 3).

To grow with the rapidly changing knowledge in medicine, I have, and will continue to, keep an open mind regarding new concepts or concepts I have yet to learn. I have come to realize that there will always be something to learn, and there will always be ways for me to challenge myself. These challenges will drive me intellectually to become a better educator and clinician, which, in turn, will benefit the profession, students, clinicians, and patients.

Specific Strengths

1. Educator
    a. I am effective at educating athletic training students on new concepts in patient care.
    b. I educate patients on the interventions being utilized for their care.
2. Scholar
    a. I collect, analyze, and reflect upon outcome measures in my patient care to assess my effectiveness, provide evidence for practice, and generate new clinical questions.

b. I am a developing scholar who has presented at conferences (Appendix B), published in peer-reviewed journals (Matocha, Baker, Nasypany, & Seegmiller, 2015a, b), serve as a journal reviewer, and will continue to conduct research to better patient care.

3. Clinician
    a. I treat every patient as an individual and attempt to incorporate their needs into the application of complete evidence-based practice (EBP).
    b. I am present during patient evaluations and interventions.
    c. I keep an open mind to new concepts and will test concepts/theories in practice.
    d. I have the skill set to research new concepts to incorporate into my evidence based practice (EBP) and practice based evidence (PBE).
    e. I perform administrative duties promptly and attentively.
    f. I am able to overcome adversity and utilize resources to reach goals to succeed.
4. Service
    a. I am a mentor in the Bobcat Bond Program for undergraduate and graduate students at TXST.

Reflection on Weaknesses

In my first year-and-a-half in the DAT program, many of the aspects of my education and practice that I considered weaknesses became strengths. These newfound strengths, which continue to grow, make me a better clinician and professional. However, I still struggle with

the following: 1) formal written scholarship, 2) understanding pain, and 3) comprehending many aspects of neuroanatomy.

Additionally, there are several aspects of scholarship that I continue to attempt to improve upon, with professional writing being one of them. I have made great strides in my professional writing, as evidenced by the acceptance and publication of a two-part article in the *International Journal of Athletic Therapy and Training*. Although I feel that my writing has seen improvement since I began the DAT program, I continue to struggle in some areas (e.g., tenses). Along with the professional writing component of scholarship, I have also struggled with critically appraising articles, which includes analyzing statistics and interpreting data sets.

The knowledge of causative factors of pain, as it relates to my patients, will most likely be an aspect of my scholarship that I will continue to consider a weakness since so much about the brain remains unknown—including the relationship and control over pain. However, I will continue to research and endeavor to understand the basics of brain function, central sensitization, peripheral sensitization, and cascading effects during an injury. I want to be an expert on pain and be able to educate others on pain and how it affects patients. In pursuing this goal, I need to fully comprehend all aspects of pain and strengthen my overall knowledge of neuroanatomy and the brain. In so doing, I will be better able to educate patients about their pain and make clinical decisions regarding intervention choices.

Specific Weaknesses:

1. Educator
    a. Curriculum
        i. There is a lack of neuroanatomy and pain science content in curriculum at TXST for both graduate and undergraduate students.
        ii. There is a lack of rehabilitative paradigms (e.g. Mulligan concept, neurodynamics, MYK) taught in both the graduate and undergraduate athletic training programs at TXST.
        iii. There is a lack of ability to critically appraise the literature by both the graduate and undergraduate students at TXST University.
2. Scholar
    a. Writing: I am not consistent in the use of scientific writing style. .
    b. Data Analyses: I am not fluent in comprehension of statistical analysis, nor understanding the ramifications of the analyses.
    c. I struggle to consider all possible variables when designing a priori studies.
3. Clinician
    a. Personal Education
        i. Pain comprehension: Pain science is continually advancing. I need to be able to fully comprehend pain science to aid my decision making for patients.
        ii. Neuroanatomy: I need to continue my education on neuroanatomy to improve my comprehension of the intricacies of the brain.

b. Practicing Clinician

i. I need to continue to collect outcomes on patients, analyze the outcomes, reflect and change my practice as deemed necessary.

ii. I will continue to look for new interventions and will continue to have an open mind to new options.

4. Service

a. Professional Service

i. I am currently not serving my profession as a volunteer for NATA educational committees.

ii. I am currently not serving my profession as a volunteer for SWATA committees.

iii. There are not courses (e.g. Mulligan Concept, MYK) held at TXST for clinicians. I am not a NATA abstract reviewer.

b. University Service

i. I am currently not a volunteer for committees in either the Health and Human Performances Department and for the University.

ii. The DAT is not recognized as a terminal degree at TXST, therefore, anyone who holds the degree cannot be considered to teach at the graduate level or serve on thesis committees.

Goals for Professional Practice

I believe every person should set goals for their lives, both personally and professionally. Setting goals is how one shapes their future and designates a path for guidance. Because I have served in many roles in the athletic training profession, I have set

several professional goals that I plan to achieve within the next 10 years. Reaching these goals will allow me to reassess and reflect upon my impact on the profession and my journey to becoming a scholarly practitioner. The goals that I have set are discussed in detail in the following paragraphs and in Table 1.

Educator Goals

Being an educator for my profession comes with responsibility for both the students and the professional. The students are the future of our profession and will most likely see many of the changes we are currently attempting to make (e.g., professional master's degree program, etc.). I believe it is my job, as an educator, to enlighten our students and help open their minds to new ideas and concepts.

To open students' minds to new knowledge and new interventions, there must be a curriculum change at TXST within the next five years. I will work to change the curriculum for both graduate and undergraduate programs to include more neuroanatomy and pain science. Through my own research, I have found that much is still being learned about these subjects; however, what is known is vital to understanding a patient and his or her needs. In conjunction with the new curriculum that I will work to create, I will also introduce other interventions (e.g., Mulligan Concept, MYK, neurodynamics) into the curriculum. I believe that once a clinician has an understanding of pain, it is important to empower him or her with the opportunity to choose from several options to create a patient-specific plan of care. Only after the aforementioned curriculum changes are made will I have met my goal of introducing students to new knowledge and interventions.

I will start a journal club at TXST for both graduate and undergraduate students by December 2015. Although it will not be mandatory for students to belong to the club, the opportunity to participate in it will help students to examine the literature more critically. The aim of the journal club will be to effectively appraise one article per week. Such appraisals will include analyses of methodology, variables, treatment effectiveness, reliability, clinical relevance, and statistics. With this deeper understanding of what they read, the students can then form their own opinions on translating the new knowledge to practice. A better understanding of pain science and potential interventions, such as are frequently discussed in medical journals and other scientific publications, will better prepare students to form their own clinical philosophies. The journal club will need to consist of at least ten members per semester (a period of five months) to be successful. Having at least ten members per semester will allow more viewpoints and ensure accountability throughout the group.

Scholarship Goals

I will become more effective in my scientific writing and will be able to produce well-organized writings, requiring fewer drafts, within the next ten years. I will have achieved my writing goal when I can submit an article for publication that has no consistent errors and can do so in within a concise, specified timeframe and in fewer drafts. As mentioned before, I will create a journal club for the purpose of appraising journal articles. Reviewing other writers' work will not only assist me in my own writing, but it will also deepen my comprehension of research (e.g., methodology, data analysis). Despite my difficulties with scholarly writing, I am now a peer reviewer for the *Journal of Athletic Training and Allied Health: The Official Journal of Ohio Athletic Trainers' Association*.

Within the next six to 12 months, I will have yet another opportunity to expand my scholarship, as I will submit several articles for publication. These articles include a case study on tendinopathy and MWM at the knee, a case study on MYK™ and knee pain, and our dissertation pilot data. I will also submit dissertation findings and follow-up papers within the next 18 months. I will work to maintain relationships with the tendinopathy group members to continue our line of research and will strive to produce one presentation or publication every three years. Along with these connections, I will create a network of scholars and researchers with similar interests and will make an effort to produce one to two presentations or publications annually.

In addition, I will share my knowledge by guest-lecturing in a graduate manual therapy class on Myokinesthetics™ in the spring of 2016. I will also submit an abstract, on an annual basis, to speak at the Southwest Athletic Trainers' Annual Meeting and Clinical Symposium. I have committed to being a speaker at the athletic training student competency workshop in January 2016 and hope to speak annually. I also aim to host several intervention courses, such as MYK™, at TXST over the next couple of years.

Expanding my knowledge on pain science will lead me to two of my ultimate goals: 1) educating other clinicians on pain science and 2) putting patients in charge of their health. Within ten years, I will present at NATA on pain science and its importance in choosing interventions. I will also present on how important it is to educate patients in pain science so the patients can become their own health care providers. My patient outcomes will be presented to represent the potential outcomes associated with the topic.

Clinical Goals

Education

There are several courses I will attend to further my knowledge in pain science and in the available interventions that I listed previously. I had the privilege of being present for Lorimere Moseley's presentation at the San Diego Pain Summit (SDPS) in February 2015, where he spoke on the basics of pain and the new theories he is currently researching. Also at the SDPS, I was able to listen to Diane Jacobs explain her version of pain and give a brief preview to the intervention she uses: dermoneuromodulation. Jacobs is offering a course on dermoneuromodulation in August 2016 that I plan to attend. Minimally, the combination of these courses, which I will complete within the next 20 years, will help with the depth and breadth of my pain science knowledge.

I have currently planned to attend six courses within the next seven years that will help me to expand my knowledge of potential interventions for patients. These courses include some of the following: lower extremity Mulligan concept, MYK^{TM} certification, and TMR Level III. The certification courses will not limit me to using only those interventions discussed in the courses; instead, they will expand my knowledge base and introduce me to all the interventions have to offer. More interventions will arise as I continue to keep an open mind in my life-long learning process.

In Practice

While I continue my education, I will also continue to collect and analyze patient outcomes, reflect on my practice, and change my methods as data and reflection indicate. I will also work with TXST University AT clinicians to collect outcomes, find new clinical areas that need improvement, and find an alternative approach to those problems. I will

continually look for new interventions to learn about and utilize with specific patients. I will also use evidence based practice to help guide me in the interventions I choose; however, I will also continually analyze and reflect on my outcomes to determine if other interventions are appropriate. I will work to discharge neurological, tendinopathy, and back pain patients within two to three visits throughout the next five years, with the last visit focusing solely on the patient demonstrating how they can care for themselves in the case of a flare-up.

Service Goals

Professional

In an effort to do a better job at serving my profession, I have applied as a volunteer for the Post-Professional Education Committee for District 6. I have also filled out volunteer forms for both NATA and SWATA. I plan to host continuing education courses at TXST within the next five years. I recently became a peer reviewer for the *Journal of Sports Medicine and Allied Health: The Official Journal of Ohio Athletic Trainers' Association*; however, I am also aiming to become a NATA abstract reviewer within the next two years. I am currently a mentor on an app, called *Wannabe*, for young students who are trying to determine what they want to be when they grow up. I will take advantage of more opportunities as they arise and as often as is possible.

University Level

I am currently a mentor for the Bobcat Bond Program at TXST, and I will continue my involvement with mentoring both graduate and undergraduate students while I reside at TXST. I will volunteer for committees in the university and especially in the Health and Human Performance Department. I have tried to volunteer for committees without success; however, there will be an opportunity, and I will take it.

Athletic Training Philosophies

Throughout the DAT, I was encouraged to develop my philosophies as they pertain to athletic training. Prior to the DAT, I had formulated one philosophy: my teaching philosophy. The need to reflect on my practice required me to begin shaping my philosophy into new ones that are focused on patient care, rehabilitation, and lower back pain. These new philosophies guide my practice in everything I do, and have grown with me throughout my tenure in the DAT program. I cannot fathom how I was a clinician without having a single clinical philosophy prior to the DAT program.

Patient Care Philosophy

As a clinician, my ultimate goal is to get patients better, quickly and safely. Through collecting outcomes, analyzing the outcomes, and reflecting, I will be able to determine what each patient needs and will alter my plan as needed for each patient. I will utilize EBP to encourage my intervention options. Treating the whole patient as an individual is important to understanding their pain and their needs. I will allow all patients the time they need to express their pain and concerns during each of their visits. I will educate each patient on treatment and rehabilitation prior to performing any intervention. I will continue to attend academic courses to learn more about new techniques and theories. I will go beyond the basic level of knowledge required by the BOC and Texas state legislature by engaging in meaningful conferences and discussions. I will uphold the ethics of the BOC and Texas state legislature.

Rehabilitation Philosophy

As a clinician, my goal is to assist patients along their road to a pain-free life. To accomplish this, I try to incorporate current research with a manual approach. Although I have had success with my intervention choices, understanding how a certain technique affects a

patient is as important as the outcome itself. Manual therapy has moved to the forefront of the interventions I prefer to use in practice, since these techniques allow me to connect more effectively with patients. When I utilize a hands-on approach and attempt to find similarities between my patients and myself (in order to connect with them), the patients are better able to feel the quality of care they are being given. Manual therapy provides me with the connection to patients and the time to explain to them how and why they are experiencing pain, which helps them to understand their involvement in the pain process. Many times this will give the patients a sense of control over their pain, which they may have or not have had before.

To help guide my decision-making process, I have chosen to incorporate the Mulligan Concept, a tender point evaluation, and neurodynamic screening into my manual therapy focused evaluations. However, if a patient presents with back pain, the patient undergoes a MYK™ postural assessment, and the appropriate nerve root is treated. My decision-making process has been driven by my patient outcomes collection, which led me to categorize patients rather than diagnose them—which was something I did before I entered the DAT program. I also discontinued the use of SFMA during my evaluation process due to my Spring 2014 outcomes assessment (Chapter 3), in which I found that my intervention choices did not change with the addition of the SFMA in my evaluation. Choosing treatments based on the classification system led me to fewer visits before discharge, and I did not feel the need to continue its use. Rather, I began to decide on an appropriate intervention based on patient classification through an algorithm I created and assess based on my patient outcomes.

Once I choose an intervention, I teach the patient either how to perform the technique on their own, or how to continue "grooving the patterns" so they do not revert to previous dysfunctional patterns. As a result of improved patient classification and education, my

patients leave the clinic knowing how to help themselves through these manual therapy paradigms/concepts. The algorithm and classifications are guided by evidence from my practice and continue to remain fluid. As evidence emerges, the algorithm and classifications will be adapted to better serve my patients. My overall goal as a clinician is to have patients that do not rely solely on me, the clinician, to make them better; instead, they can rely on themselves.

Low Back Pain Philosophy

Low back pain ails more patients globally than any other reported condition (Hoy et al., 2014). I utilized the Mulligan Concept after my initial introduction to the technique; however, many of the patients I treated had reoccurrences. Due to reoccurrences and the introduction to MYK™, I now perform a postural assessment on all back pain patients.

If a patient has a reoccurrence after the use of MYK™, I will perform the TMR™ Fab 6 ™. Once a direction and movement are identified, I can treat those movements and directions according to the TMR ™ guidelines. I will continue to recheck the original complaint of the patient throughout the TMR ™ treatment, to ensure improvement. Total Motion Release™ will also be utilized as a home exercise program, so the patient can demonstrate independency.

Patient outcomes will continue to guide low back pain patient treatments. If a patient does not have a decrease in pain and an increase in function after one MYK™ treatment, I will reevaluate the patient to ensure the appropriate nerve root was treated. I will then decide whether to use another MYK™ treatment or to incorporate TMR ™ into the patient's treatment regime.

Research Philosophy

My focus is to conduct patient-centered research. I believe if the patient perceives an improvement in their ailment, the patient can better manage their own condition. I utilize an action research philosophy to solve local problems in my patient care, with a focus on determining the effectiveness of interventions chosen for patients who present with tendinopathy. Action research is central to my philosophy due to the uniqueness of each individual patient and the ability to bring research to the clinic. Being able to conduct research in the clinic is vital to improving the external validity of the result and utilizing an action research philosophy allows me to design research that can be altered to meet the needs of each individual patient.

Currently, I have an interest in researching treatment-based classification (TBC) algorithms to improve patient outcomes. Effective TBC can guide a clinician in making clinical decisions about interventions that would meet the needs of each individual patient and improve the efficacy of treatment. I also have interest in patients who present with neurological symptoms and chronic pain. The causative factors of symptoms in patients with either condition are often difficult for clinicians to find and address. Pain science and patient education can help patients take charge of their symptoms and have knowledge of what to do in the case of a recurrence. I am interested in determining the use of neurodynamics, MYK^{TM}, and patient education in patient with both conditions and exploring which type of patient, if any, responds best to the different interventions.

Teaching Philosophy

My goals, both as a professor in the classroom and as a practitioner in the clinic, are to educate both students and patients. Students must be provided with the opportunity to develop the skills that are necessary for them to become competent clinicians and educators, themselves. Patient education is important, because patients who understand their role in managing their pain or dysfunction have better outcomes.

Along with teaching required course content, an educator must help students gain a desire to further educate themselves. The ability to instill within students an eagerness to learn and to pursue self-education is of the utmost importance. If students no longer ask questions, or if they lose their desire to understand why something occurs, then their skills and knowledge levels cease to grow. As a result, mediocrity prevails where excellence was once possible

To provide students with the skills and knowledge to become effective clinicians, it is important to meet the individual needs of the students. The ability to explain complex concepts in a simple manner can be a daunting task when one considers the many and varied methods of content delivery through which students might learn most effectively. For example, certain students may learn best through reading a textbook, while others need a verbal explanation or a hands-on demonstration. To provide for the needs of students, an educator must have exceptional knowledge of the course content he or she teaches. If students find that they are personally having a difficult time mastering the content being taught, the students may lose confidence and feel that the information being presented is too complex for them to grasp. The students may then choose not to put forth an effort to learn in the future. Should this occur, the professions in which they are interested may lose talented individuals.

Regardless of the level of difficulty of a class, if the content being taught is presented in a way that is enjoyable to students, the content is more likely to be understood. I try to use several teaching techniques in both the classroom and clinic. For example, I use models and tell stories or metaphors while having the information on a power point or handout for the students or patients. Rarely will an entire class be interested in everything that is presented throughout the course, but if students (and patients) are stimulated and become engrossed in the content being taught, then learning will take place more readily and be more enjoyable.

The ability to learn and to continue to gain knowledge throughout one's lifetime is vital to the success of an individual's career. A person who continually asks "why" will become a much more successful individual than someone who is satisfied with his or her current knowledge. Educators who interact with students during lectures and in labs help to ensure their students' connection to the information being taught. In turn, this helps students invest in their own education. Information continually changes over time due to new research or expanded views; thus, it is important for students to continue to strive to expand their knowledge base even after graduation.

Formal education is the foundation upon which a career is built; yet it is only the base. In order for a practitioner to continue to be successful, he or she must continue to build upon his or her educational base, making it stronger and larger. Also important in education is the level of excitement with which information is presented. If it is obvious that a professor lacks interest in what is being taught, his or her students will also lack interest. While not all students will be engrossed in every course they take, a professor that displays a genuine love for the course he or she teaches will engage students more so than a professor who displays disinterest. I have, in my past, taken several classes in which I had little interest; however, my

professors showed love for these subjects, which, in turn, helped me to gain interest in the course material. Had I not noticed this, I would have had a difficult time caring about what was being taught.

To my own satisfaction, I have been able to watch the students I have taught learn and begin to understand the skills associated with athletic training. When students are asked to demonstrate skills or answer difficult questions and the "light bulb" clicks on, the pride and confidence that builds inside of them shows. These moments in class or on the field make all the extra time that is spent with students outside of class (perhaps creating models students will more readily understand) extremely worthwhile. I have also seen patients that have figured out how to manage their own pain or dysfunction. My heart fills when I pass them in the hall and they explain how much better they are feeling and that they know that if something happens, they will be able to take care of it themselves.

Athletic training is a very hands-on profession, and I believe it is important to provide an education that incorporates a hands-on approach. Following up a difficult classroom lecture with a laboratory exercise facilitates the learning experiences and allows students to put to use the concepts they have been taught, which boosts confidence and insures interest in the course. Such a method of teaching will also help the educator to identify which concepts students are struggling to comprehend. In an all-lecture situation, students can fall into the trap of merely memorizing facts instead of storing them away for long-term use. However, the utilization of information taught in class helps to ensure that students are actually learning the course content.

Student and patient repetition is also important in developing a strong education. Such repetition ensures that what may seem like irrelevant information is learned instead of easily forgotten. Anatomy, for example, is incredibly important in athletic training, to both students and to patients. Without repetition, wherein the educator or clinician who has delivered information over a substantial period of time then frequently reviews that information, students and patients could easily forget important concepts.

Keeping an open mind to new information is important, since each class of students and every patient is different from previous students and patients, and certain teaching strategies will work for some people but not for others. The ability of an educator to adapt to situations as they arise is necessary to his or her students' and patients' educations. Again, course content will continue to change and be updated, so it is important that professors and clinicians remember that they are also still students, just as I am.

Justification of the Plan of Advanced Practice

Since the beginning of my enrollment in the DAT program, I have changed tremendously, both as a person and as a clinician. I have learned what it means to be a clinician who is up to date on current literature, who utilizes EBP, and who can use knowledge of the human body to decrease patients' pain immediately. I have grown to believe in myself. Balancing academics with my current profession and family has been difficult, but I have grown significantly in all three roles.

I understand that throughout my continued growth, my goals will change, my weaknesses will become strengths, and I will discover new weaknesses. I will endeavor to improve in my identified strengths and weaknesses to continue to improve personally and as a clinician. The previously presented goals provide a direction through which I can achieve my

educational, scholarly, clinical, and service ambitions and will help ensure my continued progress in my profession. There will always be an abundance of work ahead of me, but I look forward to conquering each challenge, one step at a time. Throughout the past two years, my knowledge and thought processes have changed for the better. I have become more confident in my knowledge, and most of my weaknesses that I identified in Fall 2013 (Appendix A) have become strengths (Appendix C, Fall 2014 PoAP).

Table 1: Goals for Scholarly Advanced Practice

AREA	GOAL	GOAL DATE	COMPLETION DATE
Educator	Guest Lecture – PRT / Neurodynamics • Assess lecture and update content yearly. • Offer this guest lecture annually.	Spring 2014.	Spring 2014
Education	Graduate DAT	December 2015	In progress
Educator	Journal Club – Texas State University	Missed Deadline – Expanding. New goal will be December 2016.	In progress
Educator	TXST Curriculum 1. Pain science education Increase rehabilitative	1. August 2020 August 2020	In progress

	paradigms (MC, TMR, etc.)		
Educator	Administrative: Become a clinical or education coordinator of a CAATE accredited program	August 2025	In progress
Scholar	Present at SWATA/NATA	Annually	Rejected Summer 2014. Develop topic for Fall 2015 and re-submit. Goal to produce an abstract yearly for review for District or National presentation.
Scholar	Submit for Publication – Effects of Neurodynamics on Tendinopathy Parts 1 & 2	Spring 2014.	Completed and submitted Fall 2014. Completed for publication Spring 2015.
Scholar	Submit for Publication: 1. MC on knee pain	1. October 2015	1-4. In progress 5. Will analyze and submit

	2. Dissertation Pilot Data 3. Dissertation Findings 4. MYK™ on knee pain 5. 3 month & 12 month follow up	2. August 2015 3. December 2015 4. January 2016 5. December 2016	for publication by December 2016
Scholar	Data Analysis Read an article 1 time and know if appropriate statistics were computed	December 2015	In progress
Scholar	ATS Competency Workshop	February 2016 Accepted speaking for February 2016, topic to be determined. Generate topic for annual	February 2016

		presentation.	
Scholar	Work with TXST clinicians to collect outcomes, identify clinical aspects that need improvement and create a plan for improvement.	Spring 2016	In progress
Scholar	Proficient at a priori designs	Spring 2016	In progress
Scholar	Maintain tendinopathy group and line of research. • Produce a professional presentation/publication once every 3 years	Fall 2019	Every 3 years
Scholar	Create network of scholars with similar interests to create research line • Produce 1 to 2	Annually	In progress

	professional presentations/publications annually		
Scholar	Writing Write publishable work within 4 weeks with no more than 2 drafts	August 2025	In progress
Clinician	PRRT Home Study Course	Fall 2013	Fall 2013
Clinician	Mulligan Concept Courses 1. Intro Course 2. Upper Quadrant 3. Lower Quadrant • Evaluate patient outcomes yearly to determine need to re-take course.	1. Fall 2013 2. Summer 2014 3. Fall 2015	1. Fall 2014 2. Summer 2014 3. In Progress

Clinician	Northeast Seminar Anatomy Complete	Fall 2014	Fall 2014
Clinician	PRT Training 1. Spine & Pelvis Course 2. Upper Extremity 3. Lower Extremity 4. Certification	1. July 2013 2. July 2014 3 & 4. Spring 2018	1. July 2013 2. July 2014 3 & 4. Evaluate the need for Lower Extremity Course and Certification over next 3 years.
Clinician	Myokinesthetics™ Training 1. Upper (Cervical) 2. Lower (Lumbar) 3. Certification Course	1. July 2014 2. August 2014 3. Utilize in practice and determine effectiveness.	1. July 2014 2. August 2014 3. To determine by Fall 2018

		Evaluate the need for Certification course	
Clinician	TMR™ Training 1. Level I 2. Level II 3. Level III	1. March 2015 2. April 2015 3. October 2015	1. March 2015 2. April 2015 3. In Progress
Clinician	Pain Summit – San Diego	February 2015	February 2015
Clinician	Dermoneuromodulation Course	August 2016	In progress to establish another course offered.
Clinician	NOI Group 1. Explain Pain Course 2. Neuromobilization 3. Mobilization of Neuroimmune System	1. May 2017 2. May 2019 3. May 2022	1. In progress 2 & 3. As courses are offered in the United States

Clinician	Research Emotional Coding	Spring 2017	In progress
Clinician	Research Breathing	Spring 2017	In progress. Determine if an appropriate breathing course, such as DNS A, is available for my con. Ed by May 2016.
Service	DAT – Terminal Degree at TXST	December 2016	In Progress
Service	Serve on TXST University/ Health and Human Performance committee	Fall 2015	In progress
Service	Bobcat Bond Mentor	Fall 2014	Fall 2014 Spring 2015 Registered for Fall 2015
Service	Reviewer of the J*ournal of Sports Medicine and Allied*	Spring 2015	Spring 2015

	Health: The Official Journal of the Ohio Athletic Trainers' Association		
Service	Volunteer for NATA/SWATA committees	Spring 2016	In progress Applied for PEC and PPEC NATA committees and not accepted.
Service	Host courses for clinicians • e.g., MC, MYK™	Spring 2020	In progress
Service	NATA abstract reviewer	Spring 2017	In progress

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CHAPTER 3

OUTCOMES SUMMARY, RESIDENCY FINDINGS, AND IMPACT

Embracing Change

Throughout the DAT program, I collected, analyzed, and reflected on patient outcomes from my clinical practice. In the beginning, I was uncertain of exactly what would come from my outcomes and what, if anything would actually change as a result of this process. My patient outcomes have become something I analyze and reflect upon on a daily basis for many reasons (e.g., patient discharge rates, classifications). As a result of my improved use of patient outcomes, I have become a clinician who could self-assess my practice and make changes to benefit all of the stakeholders associated with my patient care.

The growth in my practice, combined with my improved understanding of patient outcomes, allowed me to reflect on my practice in novel ways that led to my ability to create solutions for the problems I identified. I not only realized my patient care prior to the beginning to the DAT was less effective than I had perceived, I realized my first semester outcomes in the DAT were not as positive as I thought they were upon first review. Although my perception at the time was that patients were getting better faster than had previously occurred in my practice, I realized that my patient care was not as effective as I had expected. The new perspective and knowledge being developed each semester constantly provided a new lens with which to view my progress. The data collected from my patient care served as the driver to alter my daily clinical practice. During the Spring 2014 semester, I began categorizing patients into treatment groups and treated based upon those categorizations. My patients started being discharged with fewer visits after this recognition. As a result, it became a need for me to collect, analyze, reflect on, and grow from patient outcomes to develop into an advanced practitioner.

Identifying and Overcoming Barriers

There were several barriers to collecting patient outcomes that I had to overcome. Leaving the DAT after the first summer session, I thought I had an idea of the barriers I would encounter; however, changing jobs just hours after leaving Idaho left me uncertain of the obstacles that would lie ahead of me. The first barrier I had to overcome was an absence of a patient population. My new position was as a lecturer without a clinical assignment. Once I spoke with the basketball AT, I was granted consent to work with men's basketball patients. Shortly thereafter (two months), there was an unforeseen circumstance which forced me to find a new attending clinician (AC). The change in ACs highly benefitted me, as my new AC was extremely supportive of me and the DAT process. She also helped me to expand my patient population beyond Men's basketball and encouraged me to share my knowledge with other clinicians and students.

After securing a patient population during Fall I, I was faced with figuring out which patient outcome measures to collect and when to collect each data point. The challenge was difficult and it took many trials to begin to comprehend which outcome measure to administer and at what time intervals. Having multiple discussions on global outcome measures (e.g., Numerical Rating Scale [NRS], Disablement of the Physically Active Scale [DPA Scale]) and appraising the literature on the intervals at which the instrument was designed to be implemented helped my decision making. Anatomical outcome measures (e.g., Victorian Institute of Sport Assessment – Patella, Victorian Institute of Sport Assessment - Achilles) were implemented in my second year, during the Fall II semester, as I reflected upon my previous semester case study and outcome measures (Matocha, Baker, Nasypany, Seegmiller,

2015a, 2015b). During this time, I also began to utilize several different anatomical outcome measures that were dependent on the patient presentation.

After deciding upon which outcome measures to administer, I discovered a new barrier of having patients not report for a discharge visit. Several patients would either not show up or would send me an email stating they were better and did not need another visit with me, which led to me not obtaining discharge scores for my outcome measures. Discussions with other clinicians led me to setting each consecutive appointment prior to the patient leaving the clinic and explaining the importance of each visit, regardless of how the patient perceived their improvement.

Another barrier I had to overcome included patients receiving treatments from other clinicians outside of my care. Many of my patients I only treated once per week and another clinician provided care the other six days of the week. I had to either change my schedule or emphasize the importance of other clinicians not treating my patients so the outcome measures were a true reflection of my intervention choices. I decided to change my schedule to ensure treatments were performed by me on a daily basis until discharge. If there was an instance where I could not see a patient on consecutive days, I explained to the other clinician the importance of what I was doing and why treatment should be delayed until the next session under my care. I also ensured that I would see them the following day.

Data Analysis, Results, and Reflections

Fall I

When I entered the DAT program, analyzing and reflecting on clinical findings was a new concept to me. Upon analyzing my first set of outcomes, I was both impressed and disappointed in myself. I felt as though I had finally started a journey to becoming a better

clinician; however, my results were not what I had hoped for once I reflected on my outcomes at a deeper level.

Without all data present or the perspective necessary to consider the full spectrum of my patient outcomes, my analysis of my effectiveness was misleading. I consistently administered the NRS and the DPA Scale to my patients (n=11) through the Fall I semester of the DAT program. I was able to reduce patient pain by a mean change of 3.09 points on the NRS in a mean of 1.5 visits. At first glance, this was impressive to me. However, this set of data does not depict the two patients who continued to have pain, never achieving a 0 out of 10 (NRS) (Figure 3.1; Patients 15504, 15505); nor, did it depict the number of patients who presented with pain at their follow-up visit (Figure 3.2; Patients 15501, 15502, 15509, 15510, 15511). The two patients who never reported a resolution of their pain (0 out of 10 on the NRS) did not continue follow up visits with me after two or three visits. Of the five patients who reported pain during the follow up visit, four were discharged with a reported NRS value of 1 or lower, while the remaining patient discontinued treatment after three more visits without resolution of the pain complaint (4 out of 10 on the NRS).

Figure 3.1: NRS for Patients Never Achieving 0 out of 10

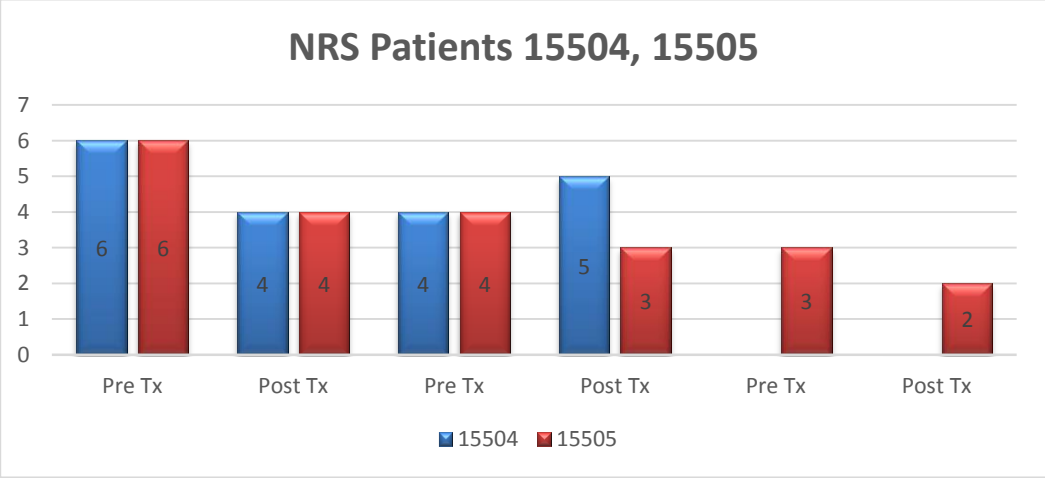
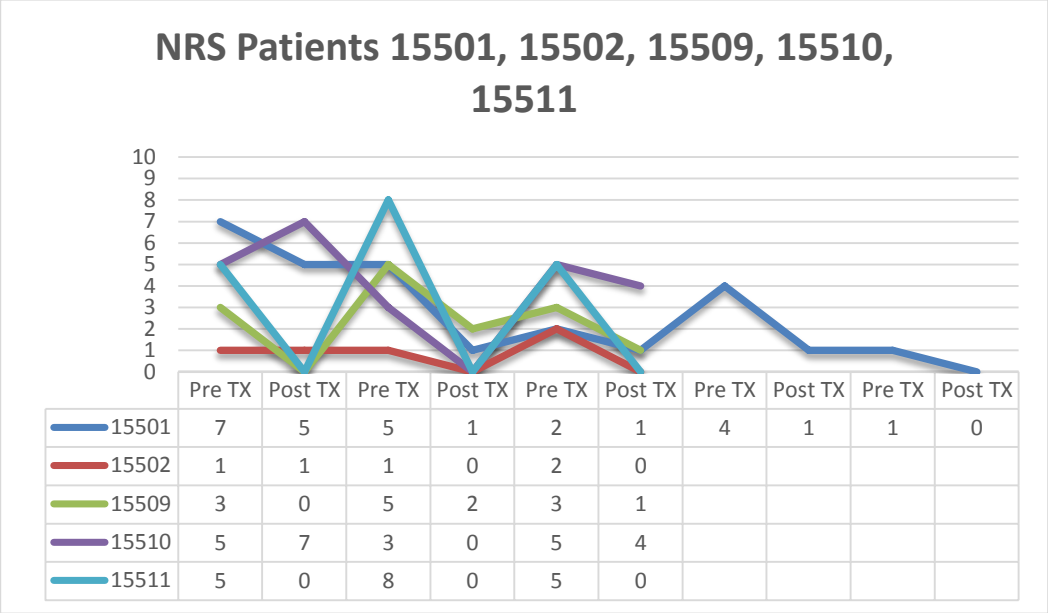


Figure 3.2: NRS for Patients Returned with Pain at Follow Up



	Pre TX	Post TX	Pre TX	Post TX	Pre TX	Post TX	Pre TX	Post TX	Pre TX	Post TX
15501	7	5	5	1	2	1	4	1	1	0
15502	1	1	1	0	2	0				
15509	3	0	5	2	3	1				
15510	5	7	3	0	5	4				
15511	5	0	8	0	5	0				

The DPA Scale mean difference from the first to second administration, one week apart, was 6.27 points, while the mean DPA Scale difference from initial visit to discharge was 12.18 points. On average, the DPA Scale MCID was met for chronic injuries (6 points) between the first and second administration, while the MCID for both chronic and acute injuries (9 points) were met from initial visit to discharge. Further reflection on my data revealed inconsistency in the number of patient visits per week (e.g., one patient had one visit per week and others had two to three visits per week). Due to this inconsistency, it was difficult to fully assess the value of my results and it became clear that I needed to become more consistent with the administration of the DPA Scale to assess the effectiveness of my chosen interventions. After speaking with one of the creators of the DPA Scale, I understood that it was not designed to be administered every week; instead it was designed to be administered when change was expected. The expected change would be different for each patient and I would need to take an a priori approach to collecting patient outcomes utilizing this scale.

Further critical reflection also led to the realization I had been seeing patients well after they had met discharge criteria. For example, some patients reported a resolution of their pain complaint on the NRS, but I continued to provide treatment. The continuation of treatments resulted in a mean of 8.45 office visits per patient. I had to question what I was providing treatment for and why I was not changing my intervention choices as the patient progressed. Ultimately, I believed it was because I was failing to consider what outcome measure collection and analysis should entail. After reflecting on the data, I decided I needed to not only track the NRS, DPA Scale, and office visits to discharge, but to also administer other global outcome measures (e.g., Global Rating of Change, Patient Specific Functional Scale) throughout the treatment process. I also realized I needed a larger sample size and a more planned approach to effectively asses my practice to improve my clinical practice. As a result of my struggles with data collection, I was not able to fully assess trends in my patient care during the Fall I semester. While I found evidence of improved patient care, I was not able to determine the true effectiveness of my intervention choices.

A new component to my practice during the Fall I semester was the Selective Functional Movement Assessment (SFMA). While I did not formally select the SFMA as one of my focus areas until the Spring I semester, I used the SFMA during the Fall I semester to guide me in identifying a patient's source of the pain. In theory, using the SFMA allowed me to address a patient's pain through a suitable intervention aimed at the source dysfunction. By using the SFMA, I was able to sub-classify my patients: one patient had a Stability and/or Motor Control Deficit (SMCD), one patient had a Joint Mobility Disorder (JMD), five patients had Tissue Extensibility Disorders (TEDs), and four had a JMD and/or TED. Of the 11 patients, three continued to need treatment beyond the suggested SFMA protocol and six

were not discharged within three visits. I initially treated SMCDs with the 4x4 matrix, JMDs with the Mulligan Concept, and TEDs with Positional Release Therapy (PRT) and/or Instrument Assisted Soft Tissue Mobilization (IASTM).

When the SFMA protocol did not alleviate symptoms in patients within three visits, I turned to other interventions I had recently learned in the DAT program. I felt as though I needed to try as many of the new interventions as possible to gain a foundational understanding on how to best utilize each intervention in my practice. Although I used the Mulligan Concept, exercise, and PRT the most, I did not do a good job of tracking NRS or DPA Scale changes through discharge. Thus, I was unable to know if my interventions were effective.

Overall, as a result of my struggles in data collection, I was not able to fully assess trends in my patient care. While I found evidence of improved patient care, I was not able to determine the true effectiveness of my intervention choices. The reflective process through the semester led to a plan to be more definitive in selecting interventions (e.g., treatment-based classification), to consistently collect outcomes to assess when I should use specific interventions, and to determine how often to collect specific outcomes.

Spring I

I treated and collected data on 20 patients for Spring I, but was most interested in analyzing the outcome measures for four new patients because I was the only clinician to provide treatment in these four cases. The other patients (n = 16) were treated by me one to two times per week, but also received treatments from other clinicians between our visits. As such, I felt it more appropriate to reflect on and analyze the outcomes from these patients as

two separate groups of data. I could also begin to analyze the difference between the groups to initiate a cursory determination of differences in the outcomes between the two groups.

One of my primary goals for Spring I semester was to produce MCIDs with each treatment session. Patients were expected to have change scores on each instrument that were equal to or greater than the MCID value published for each of the respective outcome measures for each treatment session. The MCID values were met for all patients (n = 4) on all outcome measures (i.e., DPA Scale, NRS, Patient Specific Functional Scale [PSFS], and the Global Rating of Change [GRC]) within a mean of two visits when I was the sole treatment provider (Tables 3.1-3.4; Patients 15512 – 15515).

Table 3.1 Patient 15512

15512	PRE INTERVENTION	POST 1ST INTERV.	POST 2ND INTERV.	POST 3RD INTERV.
NRS	5	4	4	**0***
DPAS	38	-	**26***	9
GRC	-	1	**5***	7
PSFS	5	-	**7***	9

*DENOTES MCID

Table 3.2: Patient 15513

15513	PRE INTERVENTION	POST 1ST INTERV.	POST 2ND INTERV.	POST 3RD INTERV.	POST 4TH INTERV.
NRS	7	**5***	1	1	2
DPAS	32	-	58	**20***	-
GRC	-	7	-7	**5***	6
PSFS	6	**-**	**2***	6	4

*DENOTES MCID

Table 3.3: Patient 15514

15514	PRE INTERVENTION	POST 1ST INTERV.	POST 2ND INTERV.	POST 3RD INTERV.	POST 4TH INTERV.	POST 5TH INTERV.	POST 6TH INTERV.
NRS	5	4	**2***	1	1	0	0
DPAS	25	-	23	24	27	**14***	4
GRC	-	1	**3***	4	5	5	6
PSFS	5	-	**7***	8	7	8	9

*DENOTES MCID

Table 3.4: Patient 15515

15515	PRE INTERVENTION	POST 1ST INTERV.	POST 2ND INTERV.	POST 3RD INTERV.	POST 4TH INTERV.	POST 5TH INTERV.
NRS	5	**3***	1	1	0	0
DPAS	22	-	**12***	10	6	8
GRC	-	1	2	**4***	7	7
PSFS	7	-	8	8	**9***	7

*DENOTES MCID

Patients who were treated by multiple clinicians (n= 16) required an average 1.13 more visits to meet discharge criteria. The reported mean difference of 1.13 visits only accounts for the number of visits with me as the treating clinician; these patients also received an average of four more visits by the other treating clinician prior to discharge. With the addition of the other treatment sessions, these patients received an additional average of 5.13 visits more to meet the discharge criteria.

In regards to changes of pain on the NRS, patients treated in my care only reported a mean pain change of five points, while those receiving care from multiple clinicians reported a mean change of 5.31 points on the NRS. Paired *t*-test statistics were computed to compare initial and discharge scores for both groups. The mean change in DPA Scale scores were 20.05 points for both groups; 17.75 points for patients treated solely by me and 20.63 points for patients treated by multiple clinicians (Figure 3.3). The mean change in PSFS was reported at -3.75 points for both groups; -.75 points for patients treated solely by me and -4.5

points for patients treated by multiple clinicians (Figure 3.4). The potential difference between groups may be due to the number of patients in each group (n=4 versus n=16), the number of interventions, varied skill of multiple clinicians providing treatment, and individual differences in patients and/or pathologies.

Figure 3.3: DPA Scale Mean Between Groups

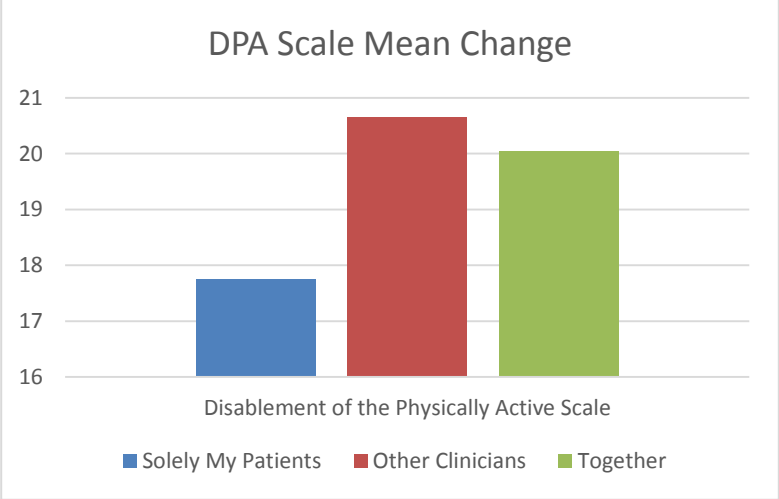
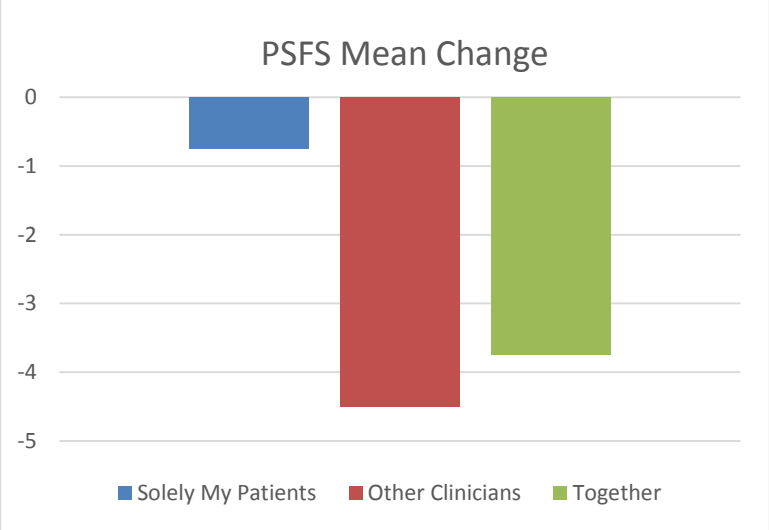


Figure 3.4: PSFS Mean Change



I was able to consistently collect outcome measures for all patients at the same time intervals. I also found that intervention classification was a key component to determining the appropriate treatment during the Spring I semester. The reflection on outcome measures and classification evaluation drove my intervention decision-making process. The resulting, improved structure of my outcome measure collection allowed me to glean the necessary data to publish a two-part article series on a case study in the *International Journal of Athletic Therapy & Training* (Matocha, Baker, Nasypany, & Seegmiller, 2015a, 2015b).

To further my development from an evaluation standpoint, I decided to compare my use of the SFMA with my standard musculoskeletal evaluation during the semester. At this time, I had not realized my standard evaluation and diagnostic process had evolved into a treatment-based classification system during the previous semester. A general overall assessment of my diagnoses and treatment led to the belief that the SFMA results did not change my intervention decisions for treatment in a manner that was more beneficial than my evolved standard evaluation. However, upon further reflection, I have determined that with the limited number of patients I treated during the Spring I semester (n=4), the decision to discontinue the use of SFMA was premature. As I analyzed data to compare Fall I and Spring I semesters, there was not enough data to definitively conclude that the SFMA did not aid in my evaluation process.

After my analysis of and reflection on my Spring I outcomes, I realized that I needed to continue to be exposed to more intervention options if I were to expand the potential for other paradigms to be included in my patient classifications. I decided that throughout the next semester (Fall II), I would focus more on tendinopathy patients and the use of a

treatment-based classification (TBC) algorithm to help my dissertation group determine the best TBC algorithm for our research.

The paradigm that I chose to focus on through the summer and entering Fall II was the MyoKinesthetic™ System. MyoKinesthetics™ is a treatment-based classification system that a clinician utilizes to identify the nerve root thought to be the causative factor of the patient's symptoms. The clinician then stimulates the muscles associated with the identified nerve root to correct an asymmetric posture, which is thought to then correct the causative factor of the patient's symptoms (Uriarte, 2006; Brody et al., 2015). MyoKinesthetics™ (MYK™) is a relatively new paradigm designed to help reduce symptoms in almost all patients, regardless of a patient's chief complaint (Uriarte, 2006; Brody et al., 2015). The classification system is discussed in greater detail later in this chapter.

Fall II

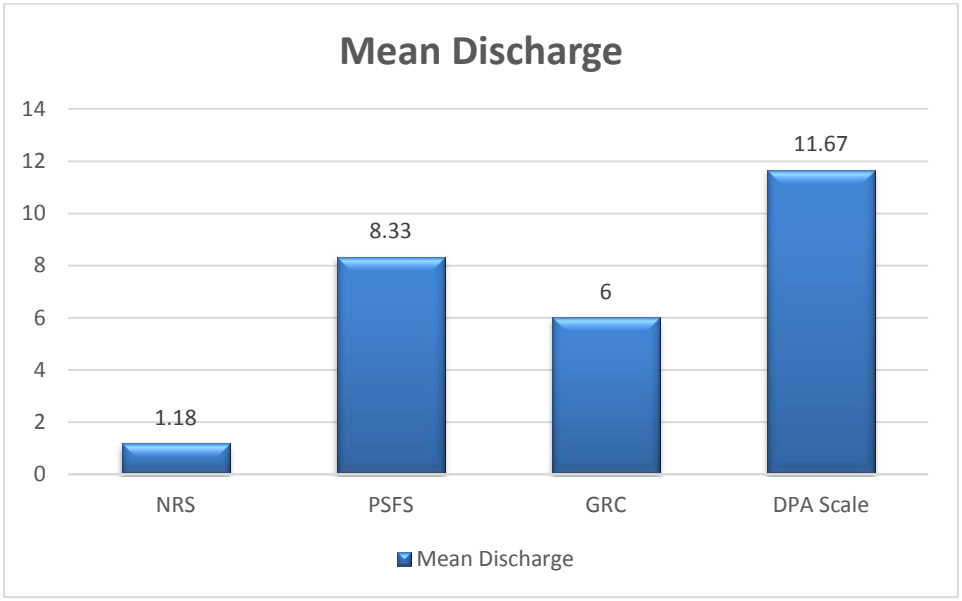
At the beginning of the Fall II semester, I set a goal to increase the function and decrease the pain of my focus group (tendinopathy patients) within three office visits. I evaluated 18 patients during the semester, 6 of which were tendinopathy patients. These patients (n=18) reported a mean change on the NRS of 2.95 out of 10, with a discharge mean of 1 out of 10. Ideally, I would like patients to be discharged with zero out of ten on the NRS; however, when assessing all data present for the patient as an individual, I believe it was acceptable to discharge those individuals with a reported one to two out of ten on the NRS due to other outcome measure deeming a safe return to play and the patient's desire to return to sport. The DPA Scale score at discharge was a mean of 10.28 points, which was within the healthy limit, and the total mean change was 16.22 points. The PSFS score was 4.72±.57 during the initial visit and 8.39±.63 at discharge. The patients perceived their function,

according to the PSFS, to have doubled from their initial visit to discharge. Although a ten is considered fully functional, and was my goal for each patient, most of my patients were content and delighted with the results they felt because they were able to return to participation at an acceptable level according to the patient. The reported GRC at discharge was a mean of +4.78, which is not impressive to me. While the change was positive (i.e., the patient perceived the treatment to improve their status), I was striving for my patients to be the best they can and able to function at an acceptable level in their perspective.

In order to gain a more comprehensive picture of patient improvement, I decided to add an anatomical outcome measure specific to each patient's chief complaint of symptoms or pain. The anatomical outcome measures, which were selected based on patient complaint, were all based on a 100 point maximum score. The mean change (-7.33±4.24) was not significant with a reported initial mean of 39.78 points and a discharge mean of 47.11 points. The addition of these anatomical outcome measures, however, added another functional component for me to assess patient improvement and treatment efficacy. I initially thought the results would be similar to PSFS or GRC; however, the specific anatomical outcome measure results were enlightening because it was not what I had expected. The information provided me with a more depth of understanding as to exactly what the patient was struggling with on a functional daily level. The difference between their anatomical outcome measure and PSFS is that the anatomical outcome measure was a questionnaire where patients were able to rate their pain and function during common daily activities; contrary to the PSFS, where the patient reported on only three activities of the patient's choosing with a high score of ten. There were two tendinopathy patients who reported lower anatomical scores at discharge than at their initial visit, despite their other outcome measure scores improving to meet discharge

criteria and the patients stating they were “feeling much better”. Tendinopathy patients during the Fall II semester were discharged with a mean of 1.18 out of 10 on the NRS, a mean of 11.67 points on the DPA Scale, a mean of 8.33 out of 10 on the PSFS, a mean of 6 on a 7 points scale on the GRC, and a mean of 57.67 points out of 100 points on the respective anatomical outcome measure. The tendinopathy patient results were positive when compared to the other patients throughout the Fall II semester; however, their anatomical scores were not what I had hoped. I do not feel that I did a good job of reflecting on the anatomical outcome measure from one visit to the next. The end of semester Fall II reflection was when I realized how poorly I ensured the function of my patients through the anatomical outcome measure.

Figure 3.5: Tendinopathy Discharge Means (NRS, PSFS, GRC)



Because MyoKinesthetics™ was a new treatment paradigm for me and there was no published research to guide my use of it, I decided to use it on all patients who presented with back pain, radiating pain, shoulder pain, and medial tibial stress syndrome (MTSS). MyoKinesthetics™ is a treatment-based classification system that a clinician utilizes to

identify the nerve root thought to be the causative factor of the patient's symptoms. The clinician then stimulates the muscles associated with the identified nerve root to correct an asymmetric posture, which is thought to then correct the causative factor of the patient's symptoms (Uriarte, 2006; Brody et al., 2015). Although the realization that this was an incorrect practice, it was important to my education; my error made it impossible for me to accurately assess the effectiveness of the MYK^{TM} System in my clinical practice. Once I had reviewed MYK^{TM} protocol and had begun to comply with its precepts, patients were discharged within an average of 2.7 visits. My Fall II goal of discharging all of my patients was met (details found in Table 3.6). I was able to discharge five patients (four patellar tendons, one Achilles tendon) within three office visits with the use of the Mulligan Concept (n=4) and PRT (n=1). All five patients presented with a gradual onset of pain and a duration ranging from three months to nine years. In accordance to the algorithm that had been established, patients met discharge criteria within three office visits. Of the four patellar tendon pain patients, two reported a decrease in pain and perceived disablement according to the NRS and DPA Scale; however, each reported a decrease in function according to the anatomical functional outcome measure (VISA-P).

Treatment-based classification systems continued to evolve my patient care throughout the Fall II semester. The addition of MYK^{TM} to my practice helped me to treat LBP patients with improvement of outcome measures, and confidence. The tendinopathy TBC algorithm continued to expand and develop into a more finalized, but fluid evaluation (e.g., order of evaluation) to ensure one paradigm would not affect the evaluation of other potential intervention choices. Following the algorithm allowed me to discharge patients within the three-office-visit goal I had set. Some patients were discharged within 2 visits,

without recurrence of symptoms. The continued success encouraged me to continue to set new goals and better myself as a clinician. I wanted to continue the implementation of the MYK™ system and discharge patients within the three visits. I also wanted to continue the use of the algorithm, analyze outcomes, and reflect on those outcomes to determine the effectiveness of my chosen treatment interventions on patellar tendinopathy, lateral elbow tendinopathy, and Achilles tendinopathy. While continuing to develop my practice, I began to identify the areas of advanced clinical practice I wanted to focus on.

Reflection on Advancement in Focus Areas

Throughout the DAT program, as I refined my efforts to classify patients and assess the effectiveness of my intervention choices, I continued to develop as an advanced practitioner. I learned that while assessing improvement during each semester by analyzing patient outcomes is important, it is also valuable to reflect on my global development across my focus areas. I selected four focus areas to develop as an advanced practice clinician during the DAT Program: neurodynamics, MYK™, Mulligan Concept, and tendinopathy. The study of each area has provided me with insight into my practice and has led to its advancement.

Neurodynamics

Neurodynamics is movement of the nervous system in relation to other structures (Shacklock, 1995). My use of neurodynamics began prior to starting the DAT program. In an effort to determine a new intervention that would decrease neurological symptoms in my own upper extremities, I reviewed the literature that was available and found a plethora of information on pain and neurodynamics.

Prior to entering the DAT program, I successfully treated myself, followed by other patients, using neurodynamics. I utilized the treatment on patients who presented with the following differential diagnoses: patellar tendinopathy and radiating upper limb pain with an unknown causative factor. As I began to expand my knowledge, I began to try the technique on other patients; many left the clinic in which I worked pain free, after the first visit. As a result of this success, I decided to study the use of neurodynamics on patients with patellar tendon pain. These patients were student athletes with whom I worked in my pre-DAT employment, which was in a previous high school setting.

I applied neurodynamic treatments on three patellar tendinopathy patients and continued to allow the "textbook" treatments (e.g., ultrasound, stretching, ice) on three other patellar tendon pain patients. Unfortunately, I did not have the knowledge about collecting outcomes that I have now; I simply observed the outcomes as the patients described their symptoms. The three patients upon whom I utilized neurodynamics for patellar tendinopathy reported a 0 out of 10 on the NRS within two visits, without a recurrence of patellar tendinopathy within one year. The other three patients continued daily treatment for a minimum of three weeks, with one being evaluated by a physician for their pain five weeks later. The patient who pursued a physician's evaluation was diagnosed with patellar tendinitis and was prescribed rest and a non-steroidal anti-inflammatory drug (NSAID). The results of my observations led me to pursue further knowledge of the neurodynamic treatment paradigm.

After beginning my journey through the DAT program, I decided to add the collection of and reflection on patient outcome measures to my study of neurodynamics. Doing so allowed me to determine if I was really making the best intervention decisions for my

patients. For example, a patient with lateral elbow pain presented seven months post injury with two diagnoses of lateral epicondylalgia. The patient had received two steroid injections and had performed the "textbook" treatments (e.g., rest, stretch, ice) through physical therapy sessions. Due to scheduling conflicts, I was only able to see the patient one time per week for six weeks, at which time she was discharged. The patient was discharged after meeting MCIDs on all outcome measures collected and continued to be pain free one year later (Table 3.3, ID:15514).

I evaluated a patient who was clinically diagnosed with a sprain to the anterior tibiofibular ligament (Table 3.5, ID: 15570). The patient's description of the mechanism of injury, which occurred five months prior to my evaluation, was consistent with a high ankle sprain. The patient reported sharp pain, 4 out of 10 on the NRS, located posterior from the lateral malleoli to the lateral side of the foot. The patient also reported a score of 34 on the DPA Scale and 4 out of 10 on the PSFS at initial assessment. My exam led to the decision to treat with neurodynamics. I instructed the patient in performing general neural sliders. The initial treatment resolved the patient's pain complaint (0 out of 10 on the NRS) prior to the end of the first office visit. Upon follow-up, one-month after the initial treatment, the patient reported full resolution of symptoms (0 out of 10 on the NRS, 2 points on the DPA scale, 10 out of 10 on the PSFS, and +7 on the GRC).

Table 3.5: Patient Outcomes for ID: 15570

Measurement	Initial	One Month Follow Up
NRS	4	0
DPA Scale	34	2
PSFS	4	10
GRC	-	+7

The reflection on these cases and the improvement on outcome measures reinforced the importance of considering neurological symptoms and neurodynamic testing as part of my exam process. Additionally, these two case examples were reminders that when a patient continues to report pain and dysfunction after the tissue has had appropriate time to heal, I must be aware of all possible sources of dysfunction.

Prior to collecting and reflecting on outcome measures, I would have used neurodynamics on any patient with tingling, numbness, or radiating pain. Despite the positive outcomes I found when using neurodynamics in these cases, I also learned through my successful cases how to evaluate what I deemed to be effective use of neurodynamics. When combined with my new knowledge of other paradigms, I found there were times or cases when other treatment paradigms could alleviate pain and symptoms faster in my patients who presented with neurological symptoms. As a result of studying my use of neurodynamics, I had a better understanding of when and how to use the technique in my practice, while also being more prepared to integrate neurodynamics with other paradigms to benefit my patients.

MyoKinesthetic™ System

MyoKinesthetics™ is a treatment paradigm that attempts to correct posture through the stimulation of a muscle while the muscle is in a stretched position. The correction of posture is thought to clear impingement of the peripheral nervous system (Uriarte, 2006; Brody et al., 2015). By performing a postural assessment, the clinician can determine the nerve root responsible for the compensatory posture. The treatment is then designed to correct the compensatory posture by stimulating all muscles associated with that specific nerve. Dr. Uriarte (2006) suggests the first and second treatment be on consecutive days, with the third treatment within one week of the second treatment.

Prior to the DAT program, I was timid when assessing and treating patients with back pain. If a patient presented with radiculopathy, I would treat with neurodynamics irrespective of individual patient case differences. If a patient presented with general low back pain, I did not have a guiding principle or intervention choice in my patient care. After learning MYK™, I felt I had found a paradigm to help my patients in these situations, but I also knew that I needed to improve my understanding of the technique.

When I began using MYK™, I had some patients who responded well and met MCIDs on all outcome measures between the first and second visit; however, others (n=3) tended to continue to have pain past the three treatment goal I had set during the Fall II semester (Table 3.6). Another DAT student mentioned the recommendation that the first and second treatment should be on consecutive days, which I had not been following in the patients who had not improved as expected. Patients who were treated in accordance to Dr. Uriarte's protocol had met MCIDs on all outcome measures and were discharged within a mean of 2.7 visits (range of 2-3 visits); while the patients I did not follow Dr. Uriarte's

protocol for, met MCIDs on all outcome measures and were discharged within a mean of 3.16 visits (range of 3-4 visits). Although the ranges are two to three visits and three to four visits, the majority of patients that were discharged without following protocol were seen for four visits with a recurrence rate of 66.7%; whereas, the patients that were discharged using the protocol were discharged consistently between two and three visits with a .14% recurrence rate. The patients that were seen on two consecutive days reported a decrease in pain and an increase in function faster than the patients that did not receive treatment on two consecutive days. The results meant that patients were able to increase participation in activity sooner than those that I did not follow the protocol. Reflecting on my outcomes provided evidence that I needed to follow the protocol as previously determined by Dr. Uriarte.

After adjusting my schedule to ensure patients would receive the first and second MYK™ treatment on consecutive days, as per Dr. Uriarte's protocol, I successfully discharged my patients within the three office visit goal I had set. My use of MYK™ included patients presenting with a variety of conditions (e.g., general low back pain, extremity pain with an unknown causative factor); the patients (n = 6) treated in accordance with MYK™ all met MCID criteria for their outcome measures and were discharged within three office visits (Table 3.6). As a result of using the MYK™ System and assessing my outcome measures in the treatment of low back pain, I have been able to become more comfortable and effective when treating this condition. As a result of my success in this area, all of my back pain and radiculopathy patients now complete a MYK™ postural assessment and treatment. I am confident in this treatment and hope to be able to contribute to research on the technique.

Table 3.6: MYK™ Assessment & Outcome Measures

ID	Chief Complaint	Postural Assessment	Protocol Vs. No Protocol	Total Visits	NRS Discharge	DPA Scale Discharge	PSFS Discharge
15516	SHOULDER PAIN	C5	No Protocol	4	2	15	8
15517	SHOULDER PAIN	L5	No Protocol	2	1	6	9
15518	KNEE PAIN	S1	No Protocol	4	2	20	9
15519	SI JOINT PAIN	C5	Protocol	3	0	8	10
15571	GENERAL LBP	S1	Protocol	2	0	2	10
15572	B KNEE PAIN	C5	Protocol	3	1	3	10
15573	GENERAL LBP / RADIATING INTO LEGS	C5	Protocol	2	2	14	7
15574	KNEE PAIN	S1	Protocol	3	0	8	9
15575	CERVICAL PAIN	C5	Protocol	2	0	4	10
15576	SHOULDER PAIN	C5	Protocol	3	0	8	9

Mulligan Concept

The Mulligan Concept for mobilization with movement theory is that a positional fault is present in the symptomatic body part and is corrected when a mobilization is applied with movement (Mulligan, 1993). The intent is to reestablish a pain free movement within a joint that was previously painful (Mulligan, 2010). The MWM, which is based on the Mulligan Concept, combines passive mobilizations with active movement in a pain free state.

During Spring I, I began to utilize this technique, not only as a treatment, but as part of my evaluation. If a patient became pain free with the application of the Mulligan Concept during my exam, then I decided to use it as a treatment. When reflecting on my practice, during Summer II, I realized I had begun using the Mulligan Concept as a foundational piece of a treatment-based classification evaluation in my practice. My patient outcomes continued

to improve (e.g., lower recurrence rate) with the use of the Mulligan Concept as an intervention and as a base for the TBC algorithm. Patients reported a resolution of symptoms, often after two to three visits. I found that I should not give up on a glide if it is not pain free during the first movement. Repositioning of the clinician hands, patient position, or amount of force on the glide can all alter the effects of the Mulligan Concept. I have grown more comfortable with applying a glide to a patient that may not be the "textbook" glide, and have found the glides to eliminate patient symptoms within one visit. I also found that if a patient presented with a positive neurodynamic test during the TBC algorithm, but was classified as being a responder to the Mulligan Concept and was treated using the Mulligan Concept, the neurodynamic test was negative upon retest. Due to this finding, I decided it was necessary for the Mulligan Concept to be tested last during the evaluation process. I realized that eliminating treatment options through my evaluation helped me to make better decisions for my patient care.

Tendinopathy

Tendinopathy is a problem with patients in my current place of employment. In Fall I, I was asked to help find a solution. To do so, I needed to help my fellow clinicians to decrease pain and increase their patients' function. Currently, researchers suggest eccentric exercise as the gold standard for patients with tendinopathy (Dimitrios, Pantelis, & Kalliopi, 2011; Svernlov, Hultgren, & Adolfsson, 2012). Unfortunately, the eccentric exercise protocol requires 12 weeks of painful exercise 2 times a day, 7 days a week (Alfredson, 1998; Stanish, Rubinovich, & Curwin, 1985), which often results in noncompliance from patients. Because of the noncompliance and long term protocol, I wanted to help patients back to activity, pain free and functional within three visits. The algorithm decided upon by the tendinopathy group

helped to classify patients into a treatment algorithm, which then determined which treatment the patient would receive.

I began the DAT and my new job at TXST, essentially at the same time. I did not perform prior treatments on the same patients; however, through taking a thorough history, I was able to determine that most patients had performed eccentric exercise and received ice, electrical stimulation, stretching, and non-steroidal anti-inflammatory drugs (NSAIDs), with many patients experiencing a continual loss of function and increase in pain. I maintained the habit of completing neurodynamic tests on patients with tendon pain after I began the DAT program. Doing so has allowed me to determine if a neural slider or tensioner would be an appropriate treatment. Through my growth as a clinician, I have established other intervention paradigms that may also be beneficial to patients. These paradigms may alleviate pain and improve function in a shorter time period than the recommended 12 week eccentric exercise protocol or my previous outcomes using neurodynamics alone.

As I was introduced to more paradigms through the DAT program, I began to also take additional courses (e.g., Mulligan Concept Introduction Course) outside of the curriculum to reinforce the concepts taught in the program. The introduction to the new paradigms allowed me to explore options in evaluating and treating tendon pain patients. To incorporate the intervention paradigms into my evaluation process, I began to integrate their concepts during my treatment-based classification evaluation.

Throughout the first three semesters in the DAT program, my tendon pain patient care changed dramatically. I initially followed the eccentric exercise protocol combined with traditional intervention strategies (e.g., rest, ice, stretching, electrical stimulation) for treatment, if the patient's neurodynamic test was negative. I did not know of alternative

interventions for patients with tendon pain. Now, I am the clinician whom other clinicians call upon when they are unable to alleviate a patient's tendon pain. Although it may not be the best or 100 percent effective, I now have alternative interventions I can utilize in patients with tendon pain and would expect to see results within the first three visits, if not immediately.

The TBC algorithm began by me trying to eliminate intervention options for a patient with lateral elbow pain at the common extensor tendon, whom had tried tradition tendinopathy treatments without success. Once I realized what I had done, I decided to try the TBC on other patients. The TBC algorithm was not initially performed in a specific order or to target only three intervention choices. As a result of continual reflection on more evaluations, I was able to determine the three most commonly used interventions in my clinic. When I noticed the Mulligan Concept alleviating patient symptoms with a sub therapeutic dose, I realized that the Mulligan Concept should be performed as the last part of the evaluation in an attempt to not bias the other tests.

Individual Tendinopathy Dissertation Data

Treatment-based classification became instrumental to my evaluation process throughout the duration of the DAT program. When I was asked to help find a solution to the growing number of tendinopathy patients, I wanted to try a treatment-based classification evaluation to determine if I could help to decrease pain and increase function in a shorter period of time than the recommended 12 week eccentric exercise protocol. The tendinopathy group to which I belonged followed a treatment-based manual therapy algorithm to determine which treatment the patient would receive. Each patient was screened for neurodynamics, tender points and a pain free, long lasting effect with the use of the Mulligan Concept. If the patient reported a pain free movement with the utilization of the Mulligan Concept, the patient

was treated with the Mulligan Concept; however, if they did not and they reported tender points, the patient was treated with PRT. If the patient had neither a pain free movement nor tender points, and had a positive neurodynamic test, the patient was treated with neurodynamics. In the case where a patient did not fit into any of the manual therapy treatment categories, the patient was placed into a respective eccentric exercise group (e.g., Patellar, Achilles).

Patellar

During my study of manual therapy algorithm, I treated nine patients who met the inclusion criteria for a patellar tendinopathy diagnosis. The patients (7 females and 2 males) ranged from 19 to 20 years of age and had a mean symptoms duration of 4.16 years (range = 5 days to 6 years). The most common complaints in these patients were: pain, increased pain with exercise, decreased pain after warming up, increased pain in the mornings, and impaired functional abilities. Using the algorithm, these patients were all classified into one of the indirect treatment sub-classifications: the Mulligan Concept (n=7), PRT (n=1), and neurodynamics (n=0) treatment sub-classifications; no patient needed to be classified into the eccentric exercise group on initial exam.

Prior to my enrollment in the DAT program, I would have classified these patients as having "patellar tendinitis," and would have treated with rest, ice, and potentially neurodynamics (depending on neurodynamic test findings). I would have expected the course of treatment to take at least one week for symptom resolution. Using this algorithm, each patient was classified with a "tendinalgia" and was discharged at the end of their third visit within eight days (range = 3 days to 8 days). To assess the effectiveness of the algorithm, paired *t* tests with a confidence interval of 95% were used to compare initial and post-

discharge scores on the following outcome measures: NRS, DPA scale, and the Victorian Institute of Sport Assessment for the Patellar tendon (VISA-P). Descriptive statistics were computed for the GRC. The change in pain on the NRS was statistically significant (p=.000) from initial evaluation to discharge, with a mean change of 5.0 ±1.02 points. All patients reported their pain as less than 2 out of 10 on the NRS at discharge. The change on the DPA Scale was statistically significant (p=.001) with a mean change of 23.22 ± 10.02 points. The change on the VISA-P was also statistically significant (p=.003) with a mean change of -22.78 ± -12.72 points. All Cohen's d effect sizes were computed as large effects for the aforementioned outcome measures (NRS d=3.79, DPA Scale d=1.78, VISA-P d=1.4). The GRC had a mean of 5.11 ± 2.96 at discharge.

Young et al. (2005) found statistical significance between two eccentric exercise groups after 12 weeks of treatment. The researcher's inclusion criteria were similar to the patient population in my dissertation study. Similarities include: patient age, pain with activity, and a VISA score of less than 80. There were two differences, Young et al. (2005) required an abnormal ultrasound of the patellar tendon and pain had to be present during palpation of only the proximal patellar tendon. In contrast, location within the patellar tendon was not specific to one location for my patients. Young et al. (2005) reported no significant difference between the two groups through a series of follow-ups.

Similarly, Cannell et al. (2001) found a statistically significant reduction in pain between two exercise groups through a variable amount of time. The age range for patients was 15 to 55 years old, with a multitude of athletic capabilities. As was the case with my patients, none of their patients wore orthotics. The presence of orthotics was an exclusion criterion. The inclusion criteria included: a diagnosis by a sports medicine physician, anterior

knee pain point tender at the inferior pole of the patella for more than four weeks, sports related knee pain, and no other knee symptoms. Similarly, my patients had anterior knee pain, had with a previous diagnosis of patellar tendinopathy, and almost all were present for longer than four weeks (symptoms not longer than four weeks n =1). The intervention time was determined by the patient's pain, however, all patients were treated longer than three weeks in the Canell et al. (2001) study.

Although patients were similar between both studies and my patients, I was able to decrease pain and increase function in my patients in a shorter amount of time than was taken in the Young and Cannell studies. With the use of the TBC algorithm, patients were able to return to full function without pain within (at the most) eight days. The TBC algorithm, in its current form, may not be 100 percent effective for all patients; however, its use can reduce symptoms in patellar tendon patients when patients are classified appropriately.

Achilles

During my study of the manual therapy algorithm, I treated two patients who met the inclusion criteria for an Achilles tendinopathy diagnosis. The patients (two females) ranged from 20 to 21 years of age and had a mean symptoms duration of 3.5 years (range = 3 to 4 years). The most common complaints in these patients were: pain, increase pain with exercise, especially stairs, decrease in pain after warming up, increase pain in the mornings, and impaired functional abilities. Using the algorithm, these patients were classified into one of the indirect treatment sub-classification: the Mulligan Concept (n=2), PRT (n=0), and neurodynamics (n=0) treatment sub-classifications; no patient needed to be classified into the eccentric exercise sub-classification during the initial examination. Prior to the DAT, I would have classified these patients as "Achilles tendinitis", would have treated with rest, ice,

eccentric exercise, and stretching. I would have expected the course of treatment to take at least 12 weeks for symptom resolution. Using this algorithm, each patient was classified with a "tendinalgia" and was discharged at the end of their third visit within four days (range = 3 to 4 days). To assess the effectiveness of the algorithm, paired *t* tests with a 95% Confidence Interval were used to compare initial and post-discharge scores on the following outcome measures: NRS, DPA scale, and Victorian Institute of Sports Assessment for the Achilles (VISA-A). Cohen's *d* was also computed to determine the effect size of each outcome measure. Descriptive statistics were computed for the GRC.

All outcome measures were not significant, which was most likely due to the low number of patients (n=2). The change in pain on the NRS was not statistically significant (p=.07) from initial evaluation to discharge with a mean change of 4.5 ± 6.8 points; however, the effect size (d=6.34) was large. Both patients reported below one out of 10 on the NRS at discharge. The change on the DPA Scale was not statistically significant (p=.215) from initial evaluation to discharge with a mean change of 18.50 ± 82.59 points and a mean discharge DPA Scale score of 10.5±7.5 points with a large effect size (d=2.01). The change on the VISA-A was also not statistically significant (p=.500) from initial evaluation to discharge with a mean change of -24 ± 256.94 points with a moderate effect size (d=-.71). The GRC mean was 4.5 ± 1.5 points.

Morrissey et al. (2011) determined Achilles tendon stiffness could be decreased with only 6 weeks of Alfredson's (1998) eccentric exercise program. Participants in the study ranged from 18 to 40 years of age and were recognized as recreational athletes with Achilles tendon pain with no previous history of Achilles pathology or joint injuries. Morrissey et al. (2011) had several more patients than I did, however, patient population was similar in

activity level and potentially age. The use of the TBC algorithm to determine the appropriate manual therapy led to a discharge in three visits within four days, as compared to six weeks, seven days a week, two times a day. Patients were also compliant for three to four days of their pain free intervention. Therefore, manual therapy may be an alternative intervention to return patients to function and decrease pain in a shorter time period.

Discussion

There has not been an abundance of research performed on the effects of the indirect methods the group chose on patellar, Achilles and lateral elbow tendinopathy. I strongly believe the indirect treatments we decided upon were appropriate and believe the current algorithm is useful in determining an appropriate treatment for patients with tendon pain. I also recognize that there are other potential useful techniques that were not included into the algorithm. The use of the algorithm has led to decreased pain and increased function in my patients with tendinalgia in a shorter time than identified in previous literature. The next step, after determining the effectiveness of the treatment based algorithm, is to compare the algorithm directly with an eccentric exercise group as a comparison group.

Final Reflection and Impact of Residency

As was evidenced through my semester-by semester reflection on my residency and through the analysis of my outcome measures, continued data collection will help to advance my clinical practice. Patient outcomes will also provide me, my patients, and my employer with evidence of my strengths and worth as a clinician to my patients. I will be up to date with the most current research to help implement current EBP and PBE into my practice. As more research on new techniques emerges, I may need to alter my evaluation methods, treatments, and outcome measures. During times of professional collaboration, collected outcomes can

help to ensure that all parties have similar goals and expectations as they work to progress and improve their clinical practice.

As I continue to collect data and to perform research on tendinopathy, I must evaluate my strengths and weaknesses and, when weaknesses are identified, I will remain willing to request help from others in my areas of weakness. Not only will other clinicians help me in my weaknesses, they, like me, will collect much needed data that will help researchers to find alternative approaches to treating tendinopathy across several clinical settings across the United States. Each clinician will have their own goals, but the goal as a group will be to advance the treatment of tendinopathy and compare a treatment-based classification algorithm with the eccentric exercise protocol.

My patient care has evolved and will continue to evolve as I continue to grow as a clinician. I was begging for patients when I first started the DAT program; now, I have clinicians and patients seeking me out for treatment. Each semester, I have collected, analyzed, and reflected on patient outcome measures and have made adjustments as necessary. To ensure that I am making appropriate improvements and continuing to grow along with the medical world, I will repeat the process at least every six months. With my continual improvement in collecting outcome measures, as has been shown throughout this chapter, I have improved as a clinician. I will continue to set goals to improve my patient care, as were outlined in Chapter 2.

Table 3.7: Fall I Patient Outcomes

ID	Chief Complaint	TX	Age	Sex	Total Visits	TX Result
15501	Foot P!	MC, PRRT	19	M	20	Negative
15502	Hamstring P!	4x4 Matrix, MC, PRT, TMR	25	M	16	Positive
15503	Cervical Radiculopathy	ND, MC, Exercises	20	M	20	Positive
15504	Ankle P! 1 YR Post surgery	MC	19	F	3	Negative
15505	Cervcal Pain	MC, PRT, TMR, PRRT	23	F	7	Positive
15506	Patellar Tendinalgia	PRT	19	F	3	Positive
15507	Patellar Tendinalgia	PRT	20	F	3	Negative
15508	Achilles Tendinalgia	PRT, PRRT, IASTM	19	F	2	Positive
15509	Patellar Tendinalgia	PRT	19	F	5	Positive
15510	Patellar Tendinalgia	MC	22	F	7	Positive
15511	Patellar Tendinalgia	PRT	20	M	3	Positive

Table 3.8: Spring I Patient Outcomes

ID	Chief Complaint	TX	Age	Sex	Total Visits	TX Result
15512	LAS	RICE, Massage	20	M	3	Positive
15513	Medial Foot P!	Supine Neural Sliders	21	M	5	Negative
15514	Elbow Tendinalgia	Neural Sliders, Tensioners	20	F	5	Positive
15515	Proximal Hamstring P!	Neural Sliders, TMR	21	M	8	Positive
15521	Lateral Knee P!	PRT	19	F	3	Positive
15522	Lateral Meniscus Tear	MC	19	F	4	Positive
15523	Hand P!	Skin Stretch	24	F	5	Positive
15524	Cervical Radiculopathy	MC	22	M	5	Positive
15525	Hamstring Strain	MC	20	M	4	Positive
15526	Prox. Bicep Tendon P!	PRT	21	F	3	Positive
15527	Hamstring Strain	Neural Sliders	19	F	4	Positive
15528	Patellar Tendinalgia	PRT	20	M	4	Positive
15529	Carpal Tunnel Syndrome	Neural Sliders	48	F	2	Positive
15530	Foot P!	Skin Stretch	22	F	4	Negative
15531	Forearm Compartment Syndrome	Neural Sliders	19	F	3	Positive
15532	Patella Subluxation	PRRT, Decrease Latency	18	F	5	Neutral
15333	Gen. Low Back Pain	MC	20	F	6	Positive
15534	Shoulder P!	MC	21	F	5	Positive
15535	Hamstring Strain	PRT	20	F	3	Positive
15536	Hamstring Strain	Neural Sliders	20	F	4	Positive

Table 3.9: Fall II Patient Outcomes

ID	Chief Complaint	TX	Age	Sex	Total Visits	TX Result
15516	Shoulder P!	MYK	23	F	4	Negative
15517	Shoulder P!	MYK	23	M	2	Neutral
15518	Knee P!	MYK	22	F	4	Positive
15519	SI Joint P!	MYK	19	F	3	Positive
15520	Shoulder P!	MC	55	M	4	Neutral
15570	Ankle P!	ND	21	F	1	Positive
15571	General LBP	MYK	38	F	2	Positive
15572	B Knee P!	MYK	32	M	3	Positive
15573	General LBP / Radiate Legs	MYK	20	F	3	Positive
15574	Knee P!	MYK	23	F	3	Positive
15575	Cervical P!	MYK	36	F	2	Positive
15576	Shoulder P!	MYK	20	F	3	Positive
15540P1	Patellar Tendinalgia	MC	19	F	3	Positive
15546A1	Achilles Tendinalgia	MC, PRT	22	M	9	Positive
15547A1	Achilles Tendinalgia	MC	21	F	2	Positive
15548P1	Patellar Tendinalgia	PRT	19	M	3	Positive
15549P1	Patellar Tendinalgia	MC	19	M	3	Positive
15550P1	Patellar Tendinalgia	MC	18	F	3	Negative

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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. The researchers developed a technique that 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

Level	**Shacklock's Classification System** ***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.

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CHAPTER 5

APPLIED CLINICAL RESEARCH

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

by: Monica Matocha, Emily Dietz, Janet McMurray, & Patti Syvertson (Being submitted to International Journal of 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 5.1a).

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 collect follow-up scores on the NRS post-discharge.

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 5.1b). 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 5.1c). 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 5.1d). 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 without exceeding seven out of ten on the NRS during the eccentric portion (knee flexion). If the participant's pain decreased to less than or equal to two out of ten on the NRS while performing EE, an external load was added to increase the difficulty of the exercise.

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 5.1a). 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 5.1a).

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 that took place within 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%; 2/10) 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 5.1a). 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 5.1a). 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 5.1a). 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 on tendinopathy patients in other research studies.[13,41-43]

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 weeks.[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 many attempts were made to decrease the risk 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 the 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. 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.

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Figure 5.1a: Treatment Classification Algorithm for Patellar Tendinopathy

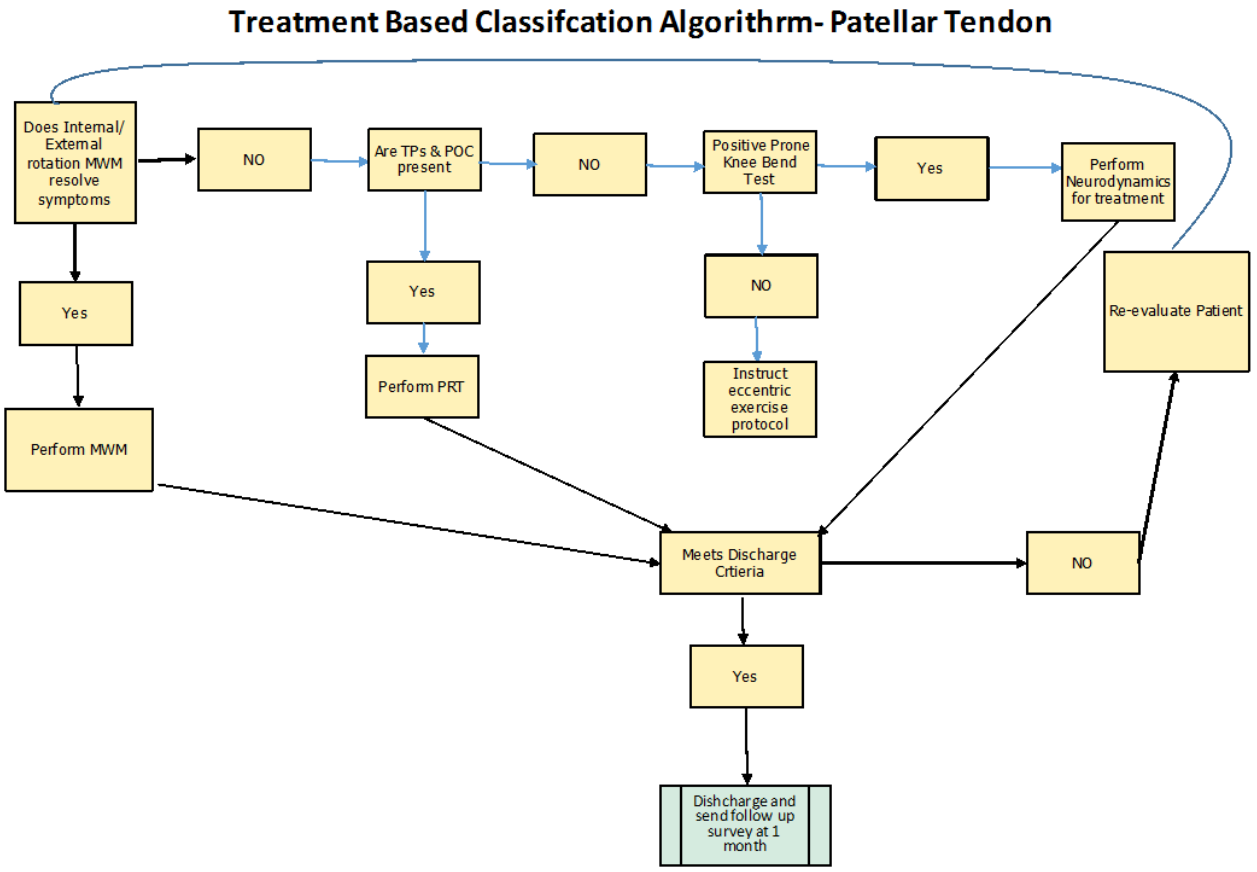

Figure 5.1b: Example of the Mobilization with Movement technique.

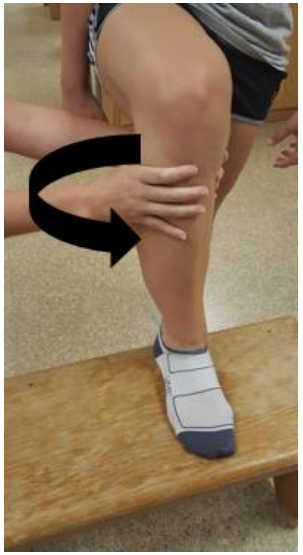

Figure 5.1c: Example of Positional Release Therapy Position of Comfort

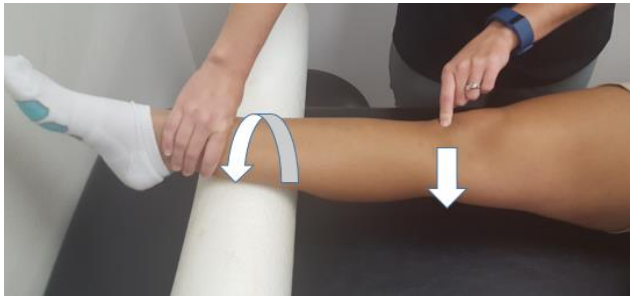

Figure 5.1d: Example of Neurodynamic Slider Technique

Table 5.1a: 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

by: Patti Syvertson, Emily Dietz, Monica Matocha, Janet McMurray (being submitted to Journal of Sport 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 5.2a). 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 5.2a).

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 5.2b). 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 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 5.1a).

When the treatment classification process was completed, the participant underwent the designated treatment (Table 5.2c). 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.

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.

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.

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. 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. 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.

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Figure 5.2a: Treatment Based Classification Algorithm for Achilles Tendinopathy

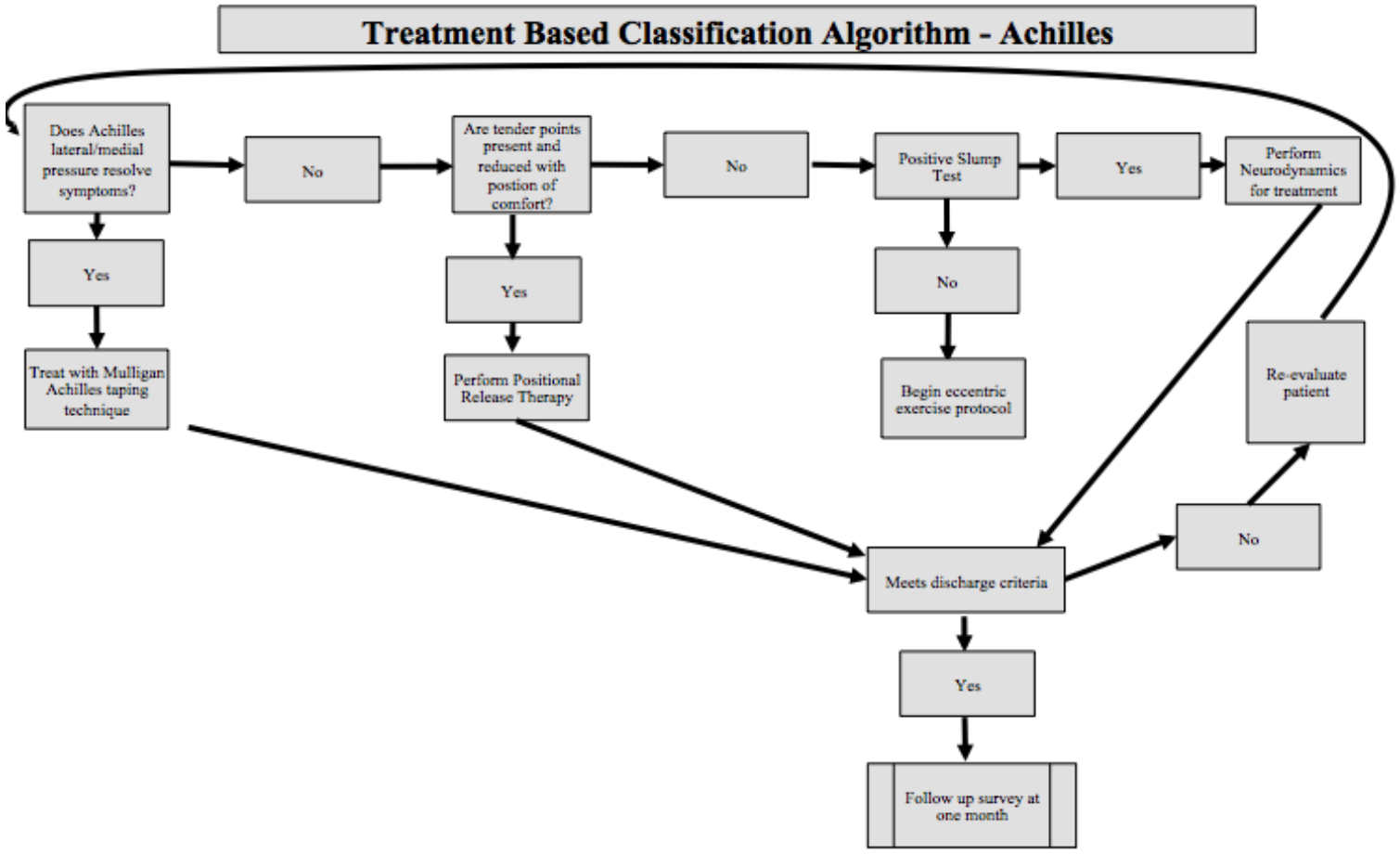

Table 5.2a Description of Outcome Measures

Table 5.2a *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.
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 5.2b Inclusion and Exclusion Criteria

Table 5.2b *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 5.2c Description of Treatment Techniques

Table 5.2c 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.
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.

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

by: Janet McMurray, Emily Dietz, Monica Matocha, Patti Syvertson (being submitted to International Journal of Athletic Therapy and Training)

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 has been inconclusive. 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. ***Results:*** 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 chronic tendon pain have been revised and indicate 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 5.3a), 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 5.3b). 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 5.3a) 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 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 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). 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. 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 5.3c)

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 ltierature.[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. Additionally, the participants and primary researchers were not blinded to the procedures or the collection of outcome measure; therefore, bias may have been introduced.

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] so 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.

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Table 5.3a 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

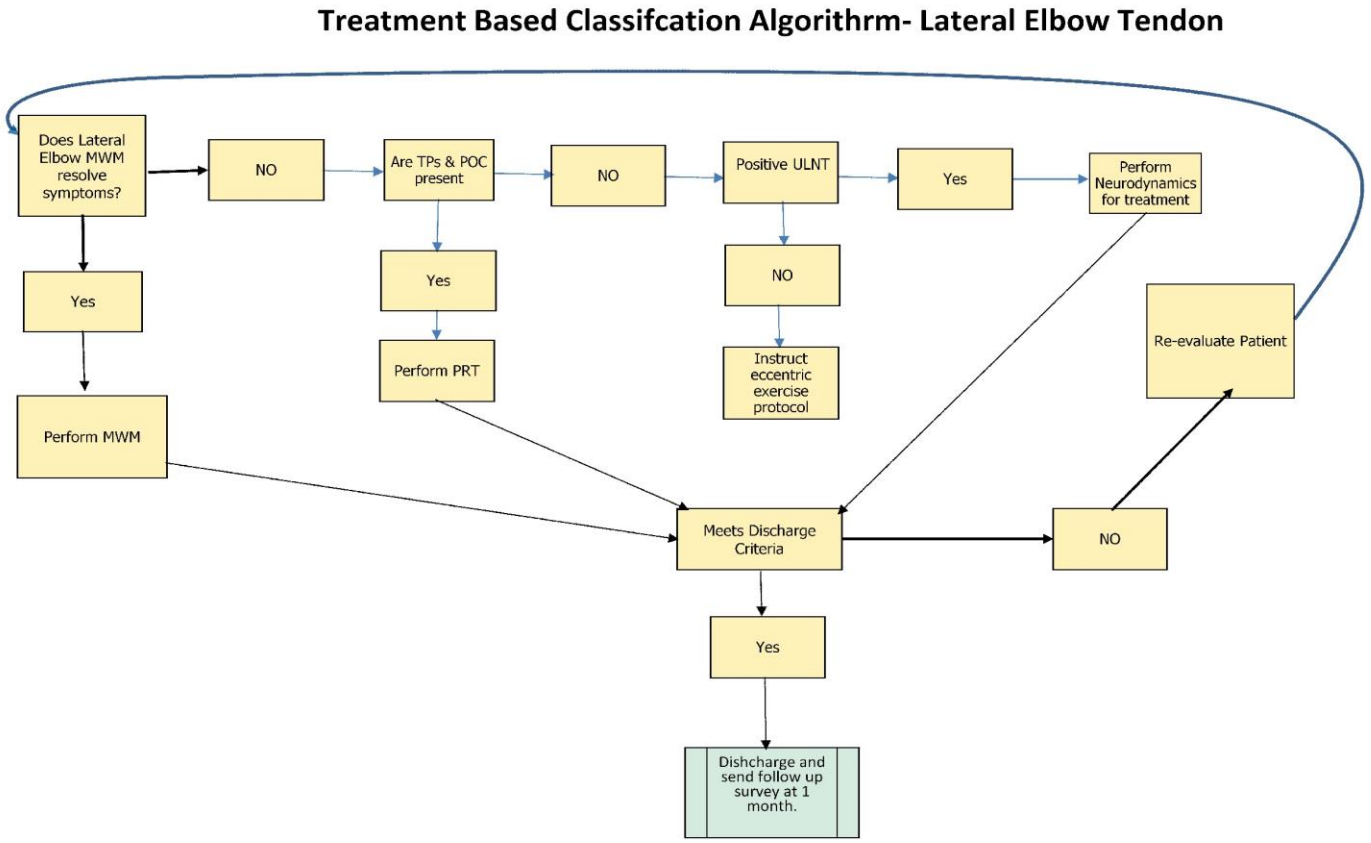
Table 5.3b Description of Outcome Measures

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
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 5.3c Changes in Patient Reported Outcome Measures

P			Function (PRTEE) (0-100)			Pain (NRS) (0-10)			Disability (DPAS) (0-64)			Number of Treatments
No.	Age	G	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change	No.
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
M	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					.01			.04			.01	
Cd					3.85						4.06	

Figure 5.3a Lateral Elbow Treatment Based Classification Algorithm



APPENDIX A

FALL 2013 PoAP

CURRENT

After critically analyzing myself I established I am not confident in many clinical aspects of my profession. I have reflected on my clinical practice more in the past three months than I had in my first six years of practice. I identified only two areas as strengths whereas I identified four areas of weakness.

Throughout the past two years I began to research pain. Only then did I start to realize how much knowledge I was lacking. My research of pain lead me to new interventions to think about such as neurodynamics. I have just begun researching and feel this is still a weakness. Reading research is not my only inconsistency. I also do not feel I understand some of the articles I read, partly due to the lack of knowledge of statistical analysis. I am also guilty of not being critical of the methods described in a research article.

Publishing an article is something I always looked at as prestigious and never thought I would be able to do it. I did not feel I was smart enough or would ever have enough knowledge of a subject to actually publish something important that others would want to read. Lack of foundational knowledge is the reason for this deficiency of confidence. To me foundational knowledge is what every athletic trainer should know such as anatomy and physiology. Included under anatomy would be neuroanatomy and the brain.

The research I did on pain and neurodynamics gave me more knowledge and a new tool but I still struggle with patient education. There have been many times I know why I am performing an intervention but cannot explain it to the patient. I have trouble putting my

knowledge into terms the patient can understand. Patient education can make a difference in outcomes. Past experiences where I was able to properly educate the patient, the outcomes were better than other patients.

Patient education turmoil only comes after the treatment and intervention decision making battle. Trying to decide when to do which intervention has been difficult. I have been a fantastic second guesser since my new education in new interventions. Was it the correct decision? Did I perform it right? What did I miss?

Administration has been one of the two things I am actually confident in. Keeping track of patient paperwork and writing everything down has helped to reflect on my practice. This helped in my decision making process for the next visit. After second guessing myself on the decision making process, I do feel I have been able to come up with appropriate rehabilitation exercises. I have been able to use my imagination to come up with exercises relevant to the patient.

GOALS

My first goal is to obtain foundational knowledge so that I can move forward in my advance practice. I will find an effective intervention for tendinopathy that relieves pain long term within two to three visits. This information will be shared by published articles along with at least one speaking engagement at a conference within the next two years. As I finish the DAT on time, I will continue to define my advanced practice in effective treatment of tendinopathy. I strongly believe these attributes will help in the recognition of the DAT as a terminal degree by Texas State University, my current employer.

OBTAINING GOALS

To gain foundational knowledge I will finish the videos in Northeast seminar on anatomy of the different body parts. I will also go back over anatomy and physiology books to make sure I understand physiology as well as the anatomy. I have to know this information if I am going to have an advanced practice in tendinopathy treatment.

In order to find an effective treatment for tendinopathy I need to be educated on many interventions. I am attending a Mulligan course this coming weekend to start my education in interventions. I will purchase the home course for PRRT by December and will attend a PRT course for the lower extremity by March. Having a better understanding of each intervention will help me in my decision making process. Continuing my research in tendinopathy may open opportunities for more interventions to pursue at another time. This of course means I will understand how to read research articles more effectively. Through reading our assigned readings for DAT class and getting extra help with statistical analysis will help me to better understand the literature. This will also help me in my scholarly writing.

Sharing the information I find with others is important to me. I have already begun the process of trying to become a better writer. When I submit to publish an article, I want it perfect. I am in contact with the writing center of my current employer who has agreed to help me. It makes sense to me when I read the articles on scholarly writing or the helpful tips from Dr. Baker but I struggle implementing them. I know I have a problem with tenses. I am not consistent writing in past, present or future tense. This, among several other writing challenges, I am going to conquer.

I will submit to speak of my findings for two conferences by summer 2015. This will also mean I will finish my dissertation, with my group, and will graduate on the expected date in May 2015.

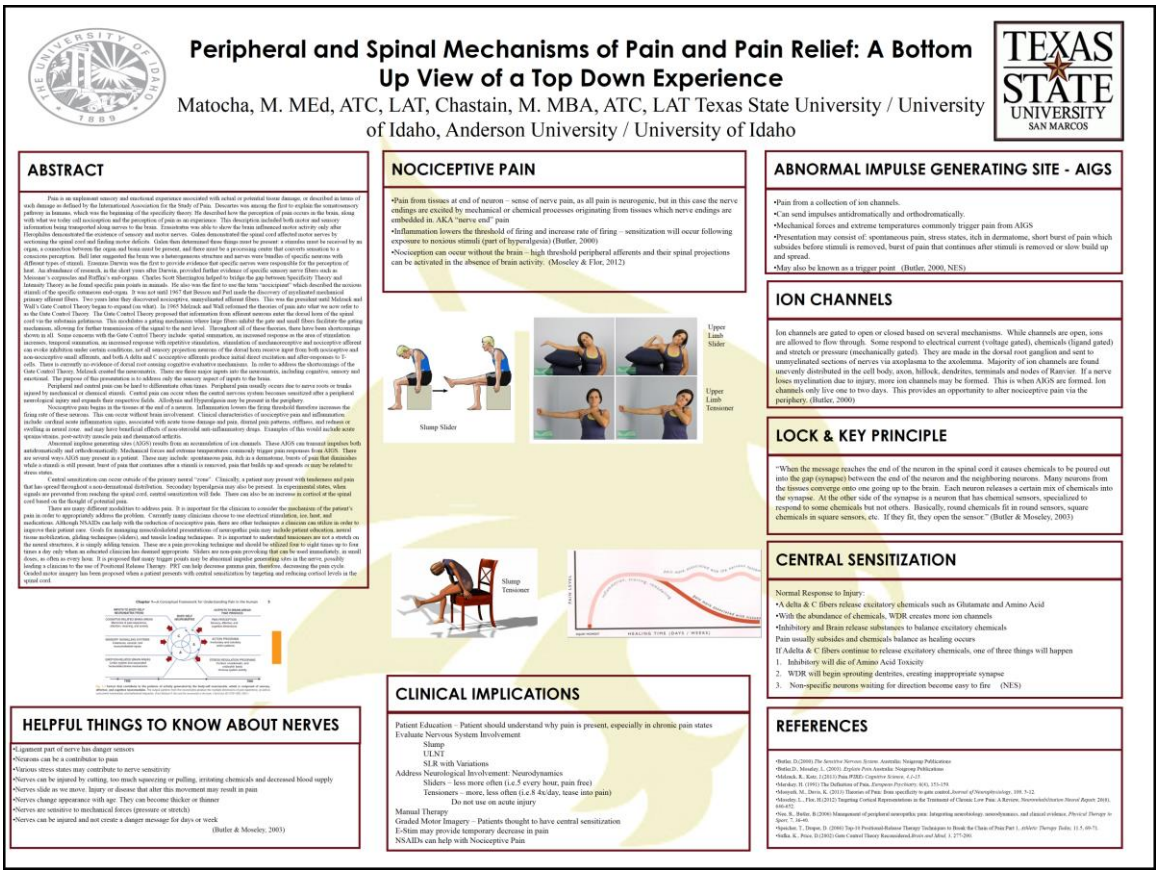
By next summer, 2014, I will report to the University of Idaho with the news of the DAT being recognized as a terminal degree by Texas State University. I have currently spoken to Luzita Vela and Rod Harter who both are willing to help me propose to the department why the DAT should be recognized as a terminal degree.

CONCLUSION

The dates above give me a deadline to strive to achieve my goals. Not only does it supply a timeline, but also gives me direction on how to achieve them. I will become stronger in both my weak and strong areas I identified earlier in order to achieve my goals on time. There is an abundance of work ahead of me and I look forward to conquering it all one step at a time.

APPENDIX B

POSTER PRESENTATION



Peripheral and Spinal Mechanisms of Pain and Pain Relief: A Bottom Up View of a Top Down Experience

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ABSTRACT

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage as defined by the International Association for the Study of Pain. Descartes was among the first to explain the somatosensory pathway in humans, which was the beginning of the specificity theory. He described how the perception of pain occurs in the brain, along with what we today call nociception and the perception of pain as an experience. This description included both active and sensory information being transported along nerves to the brain. Erasistratus was able to show the brain influenced motor activity only after Herophilus demonstrated the existence of sensory and motor nerves. Galen demonstrated the spinal cord affected motor nerves by sectioning the spinal cord and finding motor deficits. Galen then determined three things must be present: a stimulus must be received by an organ, a connection between the organ and brain must be present, and there must be a processing center that converts sensation to a conscious perception. Bell later suggested the brain was a heterogeneous structure and nerves were bundles of specific neurons with different types of stimuli. Erasmus Darwin was the first to provide evidence that specific nerves were responsible for the perception of heat. An abundance of research, in the short years after Darwin, provided further evidence of specific sensory nerve fibers such as Meissner's corpuscles and Ruffini's end-organs. Charles Scott Sherrington helped to bridge the gap between Specificity Theory and Intensity Theory as he found specific pain points in animals. He also was the first to use the term "nociception" which described the noxious stimuli of the specific cutaneous end-organs. It was not until 1967 that Bessou and Perl made the discovery of myelinated mechanical primary afferent fibers. Two years later they discovered nociceptive, unmyelinated afferent fibers. This was the precedent until Melzack and Wall's Gate Control Theory began to expand (as what). In 1965 Melzack and Wall reframed the theories of pain into what we now refer to as the Gate Control Theory. The Gate Control Theory proposed that information from afferent neurons enter the dorsal horn of the spinal cord via the substania gelatinosa. This modulates a gating mechanism where large fibers inhibit the gate and small fibers facilitate the gating mechanism, allowing for further transmission of the signal to the next level. Throughout all of these theories, there have been shortcomings shown in all. Some concerns with the Gate Control Theory include: spatial summation, an increased response as the area of stimulation increases, temporal summation, an increased response with repetitive stimulation, stimulation of mechanoreceptive and nociceptive afferent can evoke inhibition under certain conditions, not all sensory projection neurons of the dorsal horn receive input from both nociceptive and non-nociceptive small afferents, and both A delta and C nociceptive afferents produce initial direct excitation and after-responses to T-cells. There is currently no evidence of dorsal root ensuing cognitive evaluative mechanisms. In order to address the shortcomings of the Gate Control Theory, Melzack created the neuromatrix. There are three major inputs into the neuromatrix, including cognitive, sensory and emotional. The purpose of this presentation is to address only the sensory aspect of inputs to the brain.

Peripheral and central pain can be hard to differentiate often times. Peripheral pain usually occurs due to nerve roots or trunks injured by mechanical or chemical stimuli. Central pain can occur when the central nervous system becomes sensitized after a peripheral neurological injury and expands their respective fields. Allodynias and Hyperalgesias may be present in the periphery.

Nociceptive pain begins in the tissues at the end of a neuron. Inflammation lowers the firing threshold therefore increases the firing rate of these neurons. This can occur without brain involvement. Clinical characteristics of nociceptive pain and inflammation include: cardinal acute inflammation signs, associated with acute tissue damage and pain, dinstal pain patterns, stiffness, and redness or swelling in neural zone, and may have beneficial effects of non-steroidal anti-inflammatory drugs. Examples of this would include acute sprains/strains, post-activity muscle pain and rheumatoid arthritis.

Abnormal impluse generating sites (AIGS) results from an accumulation of ion channels. These AIGS can transact impulses both autoknomatically and orthochromatically. Mechanical forces and extreme temperatures commonly trigger pain responses from AIGS. There are several ways AIGS may present in a patient. These may include: spontaneous pain, itch as a dermatome, bursts of pain that diminishes while a stumli is still present, burst of pain that continues after a stimuli is removed, pain that builds up and spreads or may be related to stress states.

Central sensitization can occur outside of the primary neural "zone". Clinically, a patient may present with tenderness and pain that has spread throughout a non-dermational distribution. Secondary hyperalgesia may also be present. In experimental states, when signals are prevented from reaching the spinal cord, central sensitization will fade. There can also be an increase in cortisol at the spinal cord based on the thought of potential pain.

There are many different modalities to address pain. It is important for the clinician to consider the mechanism of the patient's pain in order to appropriately address the problem. Currently many clinicians choose to use electrical stimulation, ice, heat, and medications. Although NSAIDs can help with the reduction of nociceptive pain, there are other techniques a clinician can utilize in order to improve their patient care. Goals for managing musculoskeletal presentations of neuropathic pain may include patient education, neural tissue mobilization, gliding techniques (sliders), and tensile loading techniques. It is important to understand tensioners are not a stretch on the neural structures, it is simply sliding tension. These are a pain provoking technique and should be utilized four to eight times up to four times a day only when an educated clinicians has deemed appropriate. Sliders are non-pain provoking that can be used immediately, in small doses, as often as every hour. It is proposed that many trigger points may be abnormal impulse generating sites in the nerve, possibly leading a clinicians to the use of Positional Release Therapy. PRT can help decrease gamma gain, therefore, decreasing the pain cycle. Guided motor imagery has been proposed when a patient presents with central sensitization by targeting and reducing cortisol levels at the spinal cord.

NOCICEPTIVE PAIN

- Pain from tissues at end of neuron – sense of nerve pain, as all pain is neurogenic, but in this case the nerve endings are excited by mechanical or chemical processes originating from tissues which nerve endings are embedded in. AKA "nerve end" pain
- Inflammation lowers the threshold of firing and increase rate of firing – sensitization will occur following exposure to noxious stimuli (part of hyperalgesia) (Butler, 2000)
- Nociception can occur without the brain – high threshold peripheral afferents and their spinal projections can be activated in the absence of brain activity. (Moseley & Flor, 2012)

ABNORMAL IMPULSE GENERATING SITE - AIGS

- Pain from a collection of ion channels.
- Can send impulses antidromatically and orthodromatically.
- Mechanical forces and extreme temperatures commonly trigger pain from AIGS
- Presentation may consist of: spontaneous pain, stress states, itch in dermatome, short burst of pain which subsides before stimuli is removed, burst of pain that continues after stimuli is removed or slow build up and spread.
- May also be known as a trigger point (Butler, 2000, NES)

ION CHANNELS

Ion channels are gated to open or closed based on several mechanisms. While channels are open, ions are allowed to flow through. Some respond to electrical current (voltage gated), chemicals (ligand gated) and stretch or pressure (mechanically gated). They are made in the dorsal root ganglion and sent to unmyelinated sections of nerves via axoplasma to the axolemma. 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. This is when AIGs are formed. Ion channels only live one to two days. This provides an opportunity to alter nociceptive pain via the periphery. (Butler, 2000)

LOCK & KEY PRINCIPLE

"When the message reaches the end of the neuron in the spinal cord it causes chemicals to be poured out into the gap (synapse) between the end of the neuron and the neighboring neurons. Many neurons from the tissues converge onto one going up to the brain. Each neuron releases a certain mix of chemicals into the synapse. At the other side of the synapse is a neuron that has chemical sensors, specialized to respond to some chemicals but not others. Basically, round chemicals fit in round sensors, square chemicals in square sensors, etc. If they fit, they open the sensor." (Butler & Moseley, 2003)

CENTRAL SENSITIZATION

Normal Response to Injury:
- A delta & C fibers release excitatory chemicals such as Glutamate and Amino Acid
- With the abundance of chemicals, WDR creates more ion channels
- Inhibitory and Brain release substances to balance excitatory chemicals

Pain usually subsides and chemicals balance as healing occurs

If Adelta & C fibers continue to release excitatory chemicals, one of three things will happen
1. Inhibitory will die of Amino Acid Toxicity
2. WDR will begin sprouting dentrites, creating inappropriate synapse
3. Non-specific neurons waiting for direction become easy to fire (NES)

HELPFUL THINGS TO KNOW ABOUT NERVES

- Ligament part of nerve has danger sensors
- Neurons can be a contributor to pain
- Various stress states may contribute to nerve sensitivity
- Nerves can be injured by cutting, too much squeezing or pulling, irritating chemicals and decreased blood supply
- Nerves slide as we move, injury or disease that alter this movement may result in pain
- Nerves change appearance with age. They can become thicker or thinner
- Nerves are sensitive to mechanical forces (pressure or stretch)
- Nerves can be injured and not create a danger message for days or week

(Butler & Moseley, 2003)

CLINICAL IMPLICATIONS

Patient Education – Patient should understand why pain is present, especially in chronic pain states
Evaluate Nervous System Involvement
- Slump
- ULNT
- SLR with Variations

Address Neurological Involvement: Neurodynamics
- Sliders – less more often (i.e.5 every hour, pain free)
- Tensioners – more, less often (i.e.8 4x/day, tease into pain)
- Do not use on acute injury

Manual Therapy
Graded Motor Imagery – Patients thought to have central sensitization
E-Stim may provide temporary decrease in pain
NSAIDs can help with Nociceptive Pain

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APPENDIX C

FALL 2014 PoAP

Reflection on Professional Experience and Development

In 2005 I graduated from Texas Lutheran University, a small private school, with a Bachelor's of Science degree in athletic training. Upon graduation, I decided to work towards a master's degree while also working full time for a physical therapy clinic that contracted me to an area high school. Soon, however, I decided that my employment was not allowing me the time for study that I needed in order to complete my degree within the two-year time limit that I had set for myself. Therefore, I made a decision to quit my job and applied, instead, for a graduate assistant position at Texas State University. I received the assistantship, which gave me the opportunity to work with the Texas State University softball team while also working to complete my Master's of Education degree in two years.

After graduating from Texas State University, I had to make a hard life decision—one that I often re-visit: I had the choice of working at either a division II university or at a high school. I chose working at a high school for two main reasons: 1) because it promised to be a new experience, and 2) because I needed the substantially higher pay. While I met some amazing athletic trainers when I worked at the high school, there were several times I regretted my decision. The setting left me feeling unchallenged and frustrated—mostly because I felt I was not doing everything I could to help patients. After searching for three years and having several discussions with family and mentors, I decided the Doctor of Athletic Training (DAT) program at the University of Idaho was what I was looking for. Although I wanted to explore the teaching side of athletic training, I was not ready to give up on the clinical aspect. The DAT seemed to have everything I was looking for: challenge,

location, and a curriculum I could apply as a clinician. Pursuing the DAT has also led me to job opportunities, at Texas State University, which have allowed me more time for improvement of patient care. Finding the DAT was the new beginning that I had been looking for.

Reflection on Current Knowledge

When I first applied to the DAT, I was certain that I was more than a novice in my profession. I did not consider myself to be an expert on any level; however, I felt that I had more knowledge than graduating professionals. I was confident in my evaluation skills and my ability to multitask. Having worked at a high school the previous five years, I had learned to successfully perform an evaluation and administer rehabilitation to two or more patients at the same time. I had researched neurodynamics, began use of the treatment clinically, and presented on the concept at the Southwest Athletic Trainers' Association student symposium. Despite my confidence on day one of the DAT, I had to reorganize my thought process. I was challenged to critically analyze myself and my practice within the first week.

After critically analyzing myself and my practice, I established I was not confident in many clinical aspects of my profession. Upon my reflection, I realized that I had been doing, most everything wrong. I was reminded how valuable a proper evaluation is and being present in that moment with the patient was just as important. I was quickly reminded that the foundational knowledge I learned in undergraduate school was still important and should not be forgotten. To regain strength and confidence in this area, I watched Northeast Seminar videos on anatomy and reviewed basic knowledge via textbooks throughout the next year.

Prior to leaving my first summer session, I had a new mindset on patient care. I had learned more in that one month than I had in several years. I was rejuvenated and ready to try the new concepts I had learned. However, everything changed on my way home from Idaho. I was offered a position as a lecturer at Texas State University and would no longer have direct access to patients. My plans of who I would collect data on, along with my potential dissertation topic no longer existed.

Despite having to search for patients and ask permission to see specific patients, I was eager to try the new concepts and collect outcomes. I wanted to prove I could help patients by decreasing their pain and increasing their function in less time than previously thought. My original attending clinician was not interested in what I was doing. Although he told me I could see his patients, he had no interest in what I was doing or why it was working. After only three months, I changed attending clinicians due to some unforeseen circumstances. The change could not have been more advantageous for me. My new attending clinician is interested in learning more about the concepts and challenges me on my knowledge and skills. The challenge has helped me become more confident in my decision making skills along with confidence in explaining the concepts. The challenges sparked conversations amongst faculty and staff, which led me to guest lecturing on neurodynamics and positional release therapy (PRT) for a graduate manual therapy class at Texas State University in March 2014.

Reflection of Strengths

Throughout the course of the first year and a half of the DAT, I became confident in both my clinical practice and myself again. Learning to be present with patients and taking the time to truly listen to their complaints, along with collecting outcome measures, has helped in my intervention decision making skills. The start of my job at Texas State University allowed

me time to reflect on each patient case (i.e. outcome measures) prior to their return visit, in order to determine the next appropriate step.

I have become diligent in educating athletic training students and patients on the new concepts I learn. I believe it is vitally important for both parties to understand the technique, along with my rationale. The athletic training students thrive for new information and to understand new concepts. Their curiosity has forced me to thoroughly understand the concepts I utilize on my patients. Many of my patients are in school, in hopes to get into physical therapy school one day. Those patients are just as curious as the athletic training students and also help engage me in their interventions and challenge me on why I make certain decisions. I have been able to articulate to both the professional and patient both my decision making process and the interventions of choice.

I have not shied away from an intervention simply because I do not feel confident in my explanations. Myokinesthetics, for example, is a new concept that I have grown fond of over the past three months for patients with back pain and or radiating pain. Although I feel I need to read more and find a way to better articulate the concept to both the professional and patient, I continue to use the concept. I have kept an open mind to new concepts and will continue to keep an open mind for more concepts I have yet to learn. The realization is that there will always be something to learn and ways to challenge myself.

Specific Strengths

5. I am diligent in educating athletic training students on new concepts.
6. I am diligent in educated patients on the interventions being utilized for their care
7. I treat every patient as an individual.

8. I am present during every evaluation and intervention.
9. I keep an open mind to new concepts.
10. I continually research new concepts to incorporate into my evidence based practice (EBP) and practice based evidence (PBE).
11. I take outcome measures on the majority of patients.
12. I perform administrative duties promptly and attentively.

Reflection of Weaknesses

Throughout the first year and a half of the DAT, there were many weaknesses that became strengths. The new found strengths continue to grow to make me a better clinician and professional. However, I struggled with three aspects on a continual basis: 1) scholarship, 2) understanding of pain, and 3) neuroanatomy.

There are several aspects of scholarship I continue attempting to improve upon, writing being one of them. I have made great strides with my writing with the acceptance of a two part article, for publication in the *International Journal of Athletic Therapy and Training*. Although I feel that my writing has improved from the start of the DAT, there continue to be aspects with which I struggle (i.e. tenses). Along with the writing aspect of scholarship, I also have struggled with critically appraising articles, including: analyzing statistics and inferring the interpretation of a set of data.

The knowledge of pain, as it relates to my patients, will most likely be a topic that will continue to be a weakness due to the vast majority of uncertainty of the brain. I have researched to understand the basics of the brain, central sensitization, peripheral sensitization, and the cascading effects during an injury. Those are just the beginning of what I want to understand about pain and where it comes from. I also feel that I need to strengthen my

neuroanatomy knowledge throughout the body. Both aspects will help me educate patients on their pain, along with help in making clinical decisions on intervention choices.

Specific areas I would like to improve:

5. Improve my writing skills to involve more of a scientific writing style without errors.
6. Comprehend analysis of statistics along with the meanings of the analysis.
7. Improve my understanding of pain, as it relates to my patients along with neuroanatomy.

Goals for Professional Practice

I would like to better serve my profession as a leader. Volunteering for SWATA committees is where I plan to start. I will then begin to infiltrate the National Athletic Trainers' Association (NATA) by volunteering on committees.

To improve my scholarship, I am starting a journal club at Texas State University, beginning January 2015. The journal club will be for graduate students who have taken methodology and will be in a statistics course. The design of the club will be to critically appraise one article per week, including methodology and statistics. Continually analyzing journal articles will aid in my writing and statistics knowledge by repetitive exposure.

Scholarship will also be expanded in my near future as I plan to submit for publication in the next four months on a case study of tendinopathy and Mulligan Mobilization with Movement at the knee. I have also written a proposal to submit to SWATA for a presentation for this coming conference in July 2015. I will also further my scholarship by guest lecturing in a graduate manual therapy class on Myokinesthetics in the spring of 2015 with a hopeful interest to host a Myokinesthetics course at Texas State University by December 2016.

I am continuing my education on pain in February 2015, as I am going to attend the San Diego Pain Summit, where Lorimere Moseley is the keynote speaker. Another interesting speaker to me is Diane Jacobs, on the dermo-neuro-modulation. I look forward to sharing the information from the conference with fellow athletic trainers. I will continue to read articles and books on pain, the brain, and neuroanatomy.

Athletic Training Philosophies

Patient Care Philosophy

I will implement new techniques into my everyday practice. I will educate the patient on treatment and rehabilitation prior to performing the intervention. I will attend courses to learn more about new techniques and theories. I will advance my knowledge and record outcomes to become a scholarly advance practitioner. I will go beyond the basic level of knowledge required by the BOC and Texas state legislature. I will uphold the ethics of the BOC and Texas state legislature.

Rehabilitation Philosophy

As a clinician my goal is to assist patients in their road to a pain free life. To accomplish this I try to integrate current research with a manual approach. Although I have had success with my intervention choices, understanding the intricacies of how a certain technique affects a patient is equally as important as the outcome itself. Manual therapy has moved to the forefront of my interventions as it allows me to connect with the patients and provide an interactive experience. It allows me the opportunity to explain pain to the patient so they have an understanding and awareness of their involvement in the pain process. Many times this will give the patient a perspective of control, which many have not had before.

I start my evaluations with SFMA, as long as the injury is not acute and the patient is able to stand. I also incorporate the Mulligan Concept into my evaluations to help guide my decision making. Once I decide on an appropriate intervention I teach the patient one of two things: how to perform the technique on their own or how to continue "grooving the patterns" so they do not revert to their old patterns. All of my patients leave the clinic knowing how to help them self.

Low Back Pain Philosophy

Final Version Coming Soon…

Teaching Philosophy

My goals as a professor in the classroom and in the clinic are to educate students and provide them with the necessary skills to become competent clinicians and educators themselves. Along with teaching the required course content, it is important that students gain a desire to further educate themselves and always ask "why". Being able to instill students with an eagerness to learn and pursue self-education is most important. If a student no longer questions or loses their desire to understand why something occurs then their skill and knowledge level will cease to grow, leaving possible excellence left at mediocrity.

In order to provide students with the skills and knowledge to become effective clinicians it is important to meet the individual needs of the students. Making difficult concepts appear simple can often be a daunting task as students require different methods of content delivery. Certain concepts may take several different didactic approaches of the same point to effectively explain a more difficult skill. For example, certain students may learn best through reading a textbook while others need a verbal explanation and yet others may need to see the concept worked out or they themselves need to use what is being taught. In

order to provide many needed different views of individual skills and concepts, exceptional knowledge of the course content is required. Without an in depth knowledge of the course content certain students could possibly be left without a strong understanding of the information being taught.

If students are having a difficult time mastering the content being taught, they may lose confidence in themselves and feel that the information being presented is too difficult for them to grasp. The student may then not put forth the effort to learn in the future. Should this occur, the profession in which they are interested may be losing a talented individual. Not only that, but their drive for further education may possibly be lost as well. If the class and the content being taught are enjoyable to the student, despite the level of difficulty, a student is more likely to succeed. Being able to take concepts that are more challenging and perhaps having students draw a diagram or demonstrate the skill or concept will not only show another perspective but also break up the monotony of lecture and allow the students to relax and enjoy the moment. Should a struggling student be chosen to demonstrate, being allowed to walk through the task with the help of the class or teacher will provide feedback that will allow the student to grasp the idea with less difficulty. Rarely will an entire class be interested in everything being presented but if the students are being stimulated to engross themselves in the content, learning will be more fun and less difficult.

The ability to learn and continue gaining knowledge throughout their lifetime is extremely important to the success of an individual's career. A person that continually asks "why" will become a much more successful individual than someone who is satisfied with their current knowledge. Instilling the drive to gain further knowledge is important to the success of a career. Interacting with students in lectures and labs will help ensure that a

student feels connected to the information and help them invest in their education. Very rare is it that information does not change due to new research or expanded views thus it is important students never settle only for what they learned in class but continually strive to expand their knowledge. Formal education is only a base upon which a career is built. In order for a person to continually be successful it is important that this base be strong and that they continually build on top of it making it stronger and larger.

Also important in education is the level of excitement in which information is presented. If a professor has no interest in what is being taught, students will also show no interest. While not all students will be engrossed in every course they take, a professor that shows a genuine love for the course will engage students more so than a professor uninterested in the course they are teaching. I have in my past taken several classes in which I had no interest, however, my professors showed their love of the subject. Had I not noticed that, I would have had a difficult time caring about what was being taught.

During the classes I have taught, being able to watch the students learn and begin to understand the skills associated with athletic training brings about great joy. When a student is asked to demonstrate a skill or answer a difficult question and the "light bulb" clicks, the pride and confidence that builds inside of them shows through. These moments in class or on the field make all the work of spending a little extra time with students outside of class or creating a model so students will understand extremely worthwhile.

Athletic training is a very hands-on field and I believe it is very important to provide an education that incorporates a hands-on approach. Following up a difficult classroom lecture with a hands-on laboratory exercise will allow the students to put to use the concepts learned and will facilitate their learning experience. Allowing students to use the skills they

have learned will boost confidence ensuring their interest in the course. Not only will it facilitate learning experiences but it will also help identify what areas students are struggling to comprehend. In an all lecture situation students can fall into the trap of merely memorizing important facts versus storing it away for long term use. Using the information learned will help to ensure that students are actually learning the course content.

Repetition is also important in developing a strong education, which ensures that what may seem like irrelevant information is learned instead of being easily forgotten. Anatomy for example, is incredibly important in athletic training. However, those who study anatomy know that learning the vast amount of muscles, bones, ligaments, and other components of human body can be quite a difficult task. Without repetition this could easily become knowledge forgotten; instead it is learned over a period of time and continually reviewed so that it is learned.

Keeping an open mind is important, as each class of students is different and certain teaching strategies will work for some but not for others. Being able to adapt to situations placed in front of you as the professor is important and necessary for a quality education. Course content will continually change and be updated so it is important that as a professor you remember you are also still a student.

Justification of the PoAP

Since the beginning of the DAT, I have changed tremendously both as a person and clinician. I have learned what it means to be a clinician who is up to date on current literature, and who can use knowledge of the human body to decrease pain immediately. I have grown to believe in myself. Balancing academics with my current profession and family has been difficult, but I have grown significantly in all three roles.

I understand that throughout my continued growth, my goals will change, weaknesses will become strengths and new weaknesses will be discovered. I will endeavor to improve in my identified strengths and weaknesses in order to continue to make improvements to myself and my clinical practice.

The above dates provide both a timeline and a direction for achieving my goals. The structure will help ensure my continued progress. There will always be an abundance of work ahead of me, and I look forward to each challenge and conquering it all, one step at a time.