An Exploratory Analysis of a Treatment-Based Classification Algorithm: Treating Tendinopathy in the High School Setting

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by

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AUTHORIZATION TO SUBMIT

This dissertation of Patti Syvertson, submitted for the degree of Doctor of Athletic Training with a Major in Athletic Training and titled "An Exploratory Analysis of a Treatment-Based Classification Algorithm: Treating Tendinopathy in the High School Setting," has been reviewed in its final form. Permission, as indicated by the signatures and dates 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) highlights the Doctor of Athletic Training (DAT) student's journey through evidence of quality improvements and scholarly advancement. The Plan of Advanced Practice (PoAP) provides the structure and builds the foundation for the DAT student to identify strengths, weaknesses, and barriers while evaluating his or her growth as a clinician. As the student evolves, depth of his or her knowledge base is attained as professional goals and objectives are successfully completed through patient centered care, outcome measures, evidence-based practice, practice-based evidence, and action research. Clinical growth occurs through critical self-reflection of clinical decision-making processes, patient outcomes, focus on *a priori* research, and advanced practice expertise. Finally, in an effort to improve patient care, an exploratory analysis of a treatment-based classification algorithm for treating tendinopathy patients is presented. A thorough literature review on tendon pain coupled with forthcoming manuscripts provides evidence of scholarly advancement and clinical development.

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The choice to enroll in the Doctor of Athletic Training Program at the University of Idaho was a difficult decision both professionally and personally. Fortunately, the journey to becoming an advanced scholarly practitioner was not a journey I would take alone.

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DEDICATION

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CHAPTER 1

NARRATIVE SUMMARY

When the National Athletic Trainers' Association (NATA) was founded in 1950, its organizers sought to strengthen the profession of athletic training through the exchange of ideas and knowledge (Delforge & Behnke, 1999). In 1990, the American Medical Association recognized athletic trainers (ATs) as health care professionals. For the next several years, the NATA identified the essential knowledge, skills, clinical competencies, and curricular development necessary for an entry-level (i.e., professional) athletic-training education (Delforge & Behnke, 1999). Historically, ATs had been prepared through an apprenticeship model, where professors and mentors propagated a belief-based educational philosophy that lacked empirical validity (Myer, Kreiswirth, Kahanov, Martin, 2009). Many ATs accepted the educational model without questioning the theories or practices taught at the entry level. Because there was little to no research component at the entry level, few ATs were trained to challenge the flawed educational system (Myer et al., 2009). This resulted in the perpetuation of traditional patterns of ineffective treatments and behaviors.

Ultimately, the athletic training profession recognized the need to develop its own body of knowledge (Knight & Ingersol, 1998). Because scholarly research communicates the principles of athletic training to other allied health and medical professionals, the need for published research was vital to the advancement of the athletic training profession (Myer et al., 2009). However, few educational programs allowed ATs to pursue terminal degrees that were designed to both train them as scholars and help them to become clinical practitioners who excel in patient care (Nasypany, Seegmiller, Baker, 2013). As the athletic training profession recognized the growing need for scholarly and clinical advancement, the Doctor of

Athletic Training (DAT) was created. Offering a post-professional athletic training program that provides opportunities for the AT to evolve into an advanced scholarly practitioner is a fundamental component of the DAT Program at the University of Idaho.

In the DAT Program, each student is challenged to develop as advanced practitioners while conducting scholarly research. Utilizing a professional practice doctorate (PPD) model, the DAT student performs research that is intended to develop solutions to real-world problems (Willis, Inman, & Valenti, 2010). The DAT requires the creation of a multifaceted capstone product that serves as the student's culminating academic and intellectual experience. This product is known as the Dissertation of Clinical Practice Improvement (DoCPI).

The DoCPI provides evidence of the student's achievements and advanced practice expertise on a chosen topic for his or her applied research, which is founded on principles of action research (AR) (Nasypany et al., 2013). While there is no universally accepted definition for AR, ATs consider AR to be research that is more patient-centered than disease-oriented; a clinician works explicitly *for* patients rather than *on* them (Meyer, 2000). There are advantages of and limitations to AR. Action research is participatory and provides continuous learning opportunities for the clinician to solve local problems, which is meaningful to the clinician's patients; however, making global generalizations using the findings that result from AR is difficult for researchers (E. Koshy, V. Koshy, & Waterman, 2011). A DAT student seeks to use an AR philosophy to study his or her practice and translate research findings directly to patient care.

The collection of patient outcomes in health care is critical to the advancement of an ATs clinical practice, because it shifts the manner in which the effectiveness of patient care is

judged from purely clinician-based to more patient-based (McLeod, Snyder, Parsons, Bay, Michener, & Sauers 2008; Wilkerson & Denegar, 2014). Focusing on patient outcome evidence (POE) versus disease-oriented evidence (DOE) affords the DAT student the opportunity to practice as a patient-centered clinician. Patient-centered care changes the relationship between the clinician and the patient, in that treatment decisions are collaborative in nature rather than clinician-dominant (Epstein, & Street, 2011). When the patient has the opportunity to interact with the clinician, the patient becomes a more active participant in his or her own healthcare (Epstein, & Street, 2011). Requiring a healthcare practitioner to become more mindful, informative, and empathetic to his or her patients' needs is at the core of patient-centered care. The collection of patient outcomes assists each patient in being an active participant in his or her own care.

The DAT student collects patient outcome measures to help understand the severity of each patient's pain as well as his or her disablement, level of function, and quality of life issues (Hurley, Denegar, & Hertel, 2011). The DAT student analyzes patient outcome measure in an effort to guide and assess clinical practice in that specific patient's case, as well as data to reflect on to guide future practice. After patient outcome measures are evaluated and synthesized, the DAT student is then able to use the data retrieved from the measures in *a priori* and *post-hoc* scholarly practice. The information is utilized to improve the clinician's patient care in real-time or after a period of reflection, while also serving as a potential "problem" to study as a future AR topic.

In the DAT student's pursuit of an applicable research topic, a working knowledge of the terms *evidence-based practice* (EBP) and *practice-based evidence* (PBE) is essential. These concepts are introduced early in the DAT Program. Evidence-based practice, which is a

fairly recent paradigm shift (Hurley et al., 2011) for ATs, allows a clinician to combine the best of scientific research with clinical expertise (Sackett, Rosenberg, Gray, Haynes, & Richardson, 1996). The end result is an improved synthesis of current information/knowledge and a method for improving patient care by combining new evidence with expertise in an individual patient case. In short, an EBP clinician considers the best of research evidence in all decision-making processes, but does so with respect to the needs of the patient and the individual skill of the clinician (Hurley et al., 2011).

The shift to a culture of EBP in athletic training has the potential to optimize patient care and facilitate the advancement of ATs as health care professionals (Welch, Hankemeier, Wyant, Hays, Pitney, & Van Lunen, 2014). The 2011 release of the 5th edition of the Athletic Training Education Competencies was intended to assist with the shift in the culture of athletic training from a tradition-based approach to one that was based on the acquisition of evidence (Welch et al., 2014). While ATs have expanded knowledge in this area and are gradually beginning to understand the importance of the EBP paradigm shift, most are not practicing in an evidence-based manner (Welch et al., 2014; Valier, Jennings, Parsons, & Vela, 2014). Lack of time, resources, and/or intellectual understanding of EBP are some of the barriers that prevent ATs from implementing EBP into their clinical settings (Valier et al., 2014).

The DAT curriculum introduces a variety of opportunities for the DAT student to improve his or her understanding of EBP. Learning modalities such as class discussions, blogs, and scholarly research assist each student in identifying and reducing the barriers to EBP that prevent him or her from fully utilizing this method of practice. As the student's depth of knowledge improves and the value of implementing outcome measures becomes

more evident, the student begins to embrace his or her role as an EBP clinician who identifies solutions to both personal and professional barriers and who has improved his or her clinical decision-making skills through applicable scholarly research. An EBP clinician utilizes systematic clinical decision-making that is supported with scholarly research for patient care, as well as the implementation of paradigms and scientific models. The EBP clinician is able to interpret, evaluate, and critically appraise the clinical evidence (Hurley et al., 2011). Not only has the EBP clinician developed scholarship and clinical expertise, the clinician understands the psychosocial needs of the individual patient (Hurley et al., 2011). Ultimately, the process of collecting patient outcomes for the purpose of determining treatment effectiveness, and then translating these findings into practice for clinical decision-making, serves as the cornerstone of EBP (Sauers et al., 2012). To ensure that the DAT student evolves as an EBP clinician, the DAT curriculum follows a multifaceted approach that challenges each student to advance his or her knowledge and clinical practice behavior through the exploration of clinical theories, critical reasoning, scholarly reflection, and classification systems.

The clinical residency component allows the DAT student to move beyond the status of an AT who is developing his or her EBP skills to an advanced clinician who generates PBE. Practice-based evidence is an important complementary paradigm to EBP for developing clinical practices that are relevant and responsive to patient needs. However, according to Swisher (2010), “In the concept of Practice-Based Evidence, the real, messy, complicated world is not controlled” (p. 1). The AT who uses this model conducts purposeful research within his or her clinical practice while integrating best research from the current scientific evidence (Nasypany et al., 2013). Practice-based evidence is critically developed,

refined, and validated before research findings are disseminated to the community of scholars and clinicians (Nasypany et al., 2013). The PBE paradigm is most effective when clinicians in the field collaborate with health care professionals to evaluate and identify best practices in patient care.

One method through which a student might develop PBE is case studies research, or case series research. In this model, the DAT student creates opportunities to identify a local problem individually or coordinates systematic investigations with clinicians at multiple sites. Not only does this allow for several case studies to be linked together demonstrating consistency of clinical findings and patient observations, it also serves to complete the EBP research continuum (Hurley et al., 2011). The DAT student identifies problems in his or her patient care with the goal to develop an *a priori* clinical research design. Such a design will allow the student to study a local problem/phenomenon from an AR perspective. Choosing the research topic early in the DAT Program provides ample time for the DAT student to discover the problem, create a plan, collect research, observe the data, reflect, revise, and repeat the cycle, each step of which leads to the end goal of changing a problem situation for the better (Koshy et al., 2011). Ideally, the AR cycle leads the DAT student into preliminary research, which allows for final adjustments to be made to methods, procedures, and inclusion or exclusion criteria before research is conducted or research findings are analyzed. While in the DAT Program, each student is encouraged by program faculty to identify a local problem and to employ various research methodologies to gain understanding of the key elements of evidence-based research.

In the DAT Program, each student is taught to recognize that case studies/series research does not produce what is considered to be a high level of clinical evidence. Indeed,

the Center for Evidence-Based Medicine (CEBM) places such research at Level 4 (Hurley et al., 2011). However, the research findings can easily translate to clinicians at a local level, where they can serve as preliminary findings that warrant more rigorous investigation of treatment interventions in a variety of clinical settings. While randomized, controlled trials (RCT) are considered the "gold standard" for evidence (Level 1), the research findings rarely translate to the clinical setting (Hurley et al., 2011). When a student is encouraged to improve his or her patient care and is taught a variety of research methodologies, he or she is able to determine which research approach is the best for a particular situation or problem. The student is also prepared to serve as a translation researcher.

Translational research, commonly known as clinical research, is Level 5, the lowest level of evidence (Hurley et al., 2011). Translational research attempts to expedite the development of effective patient care interventions through a combination of bench and field research (Hurley et al., 2011). Bench research or basic science is usually performed on animals in a tightly controlled laboratory setting (Sauers, McLeod, & Bay, 2012). Field research is conducted outside of a laboratory in a natural setting (Hurley et al., 2011). Each DAT student uses the AR philosophy to ensure that clinical treatments and applied research translates directly to the patient or population intended. Action research also helps the student to remember to consider the individual situation of each patient who is treated. While it is important to study a technique or paradigm in practice, it is also important to note that the technique is not being studied in isolation. The performance of any clinical research study also examines the clinician's skill in applying the technique on an (in theory) appropriately classified patient (Nasypany et al., 2013). The DAT student seeks to inform and influence

clinical practice through translational research and local application, which will result in improved patient care (Koshy, et al., 2011).

When I initially read an advertisement for the DAT Program at the University of Idaho, I was excited to discover a terminal degree specific to the athletic training profession. I now realize that if I had not enrolled in the DAT Program, I would have remained an AT, who, like so many others, was reluctant to embrace the EBP competency. Evidence-based practice is an important transformation for the athletic training profession; yet, for reasons discussed previously (e.g., lack of resources, barriers), many ATs are hesitant to participate in this paradigm shift (Sauers et al., 2012). As I continue to advance my knowledge and participate in applied research, I recognize that, with appropriate guidance, ATs can easily contribute to EBP and PBE. Partnerships between scientists, researches, and clinicians need to be formed to raise awareness and educate ATs and the medical community. Through personal experience, I have come to understand that many ATs who are interested in research simply need support and to be taught a structure for patient outcomes collection.

To support learning and development as an advanced scholarly practitioner, the DoCPI demonstrates proficiency as both an EBP and PBE practitioner. As stated in the previous section, the DAT student's DoCPI demonstrates his or her proficiency as both an EBP and a PBE practitioner. The following chapters of my own DoCPI illustrate my progression as a student and clinician and, ultimately, give witness to my development into a scholarly practitioner and researcher.

Chapter 2 of this document contains my Plan of Advanced Practice (PoAP), which has served as a blueprint to guide my development during and well after my completion of the DAT Program. My PoAP describes the circumstances that led me to enroll in the DAT

Program and chronicles my journey through the program, including reflections upon my strengths, and weaknesses, the barriers I have had to overcome, paradigm shifts and the philosophical changes I have gone through as I have evolved in the program. I share my struggles and successes and discuss how they contributed to my development as a scholarly clinician. I discuss novel paradigms and outline professional goals for the continued advancement of my clinical practice. My PoAP will be a reflective piece that I will refer back to frequently as I continue to write new goals and contemplate goals I have achieved.

Another element essential to the development of advanced clinical practice is the collection, assessment, and reflection of patient outcomes during the clinical residency. In Chapter 3 of this DoCPI, I discuss the implementation of new paradigms, patient outcome measures collection, clinical reasoning, and applied research. The information in this chapter demonstrates my growth as I examine positive and negative outcomes identified in my patient care. Ultimately, Chapter 3 serves as evidence of my clinical development and of improvements I have made in my clinical practice through the assessment of patient outcomes. Scholarly and purposeful reflection on patient outcomes is an integral component to the DAT process and is a characteristic of expert practice. As I detail my residency findings in chronological order, I provide rich subjective and objective reflections through patient outcomes, *a priori* designs, and blog entries that were used to evaluate my practice. Preliminary outcomes data presented for my action research topic (effects of an indirect treatment based classification (TBC) algorithm on tendinopathy patients) provide meaningful insight into the development of my patient care and clinical practice.

Chapter 4 of this DoCPI provides evidence of my increased breadth and depth of knowledge in the area of tendon pain and is comprised of a review of literature that contains a

detailed exploration of the epidemiology, etiology, pathoanatomics, physiological changes, classification systems, and treatments related to tendon pain. The efficacy of indirect and direct treatments associated with tendon pain are examined, as are the relationships between the mechanical, vascular, and neural systems and the potential effects of these relationships on tendon pain.

In Chapter 5, I offer the final product of my of my DoCPI, a culmination of my final research which was created out of an AR philosophy. The three articles produced represent the growth and development of a group of DAT students (to which I belonged) who effectively collaborated and communicated from multiple clinical sites as they produced applied scholarly research utilizing a novel TBC algorithm on tendinopathy participants. The final results of my research studies provide evidence of my ability to create, design, implement, analyze, and successfully disseminate scholarly research that is directly applicable to tendinopathy patients and ATs across the country. This, in turn, provides evidence of my development as a research professional. The DoCPI is an opportunity to share my successes and failures as I transform from a novice AT into an advanced scholarly clinician who is working toward advanced practice. As I imagine my life beyond the DAT, I realize that this is merely the beginning of my journey, not the end. Ultimately, the DoCPI highlights the impact of the DAT program on my clinical philosophies, patient care, and development into an advanced practice AT clinician.

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CHAPTER 2

PLAN OF ADVANCED PRACTICE: FINALIZED JULY 18, 2015

The Plan of Advanced Practice (PoAP) is an important component of the DAT curriculum. An advanced practitioner must have an expert knowledge base, complex clinical reasoning skills, and clinical competency to produce the expected quality of patient care. The PoAP provides an opportunity for the DAT student to develop his or her skills and engage in critical self-evaluation through a detailed plan that includes the creation of short and long term goals, the evaluation of patient outcomes, and the development of clinical philosophies, paradigms, and theories. The PoAP is an organized plan that guides the DAT student through his or her journey of advancement. The PoAP has become an integral part of my reflective process and will remain a fluid document that reflects changes as I continue to evolve as an advanced scholarly practitioner.

Rationale for Pursuing the Doctor of Athletic Training

Journey to the DAT

Decades ago, I received a bachelor's degree in exercise science. Kinesiology majors interested in exercise physiology were encouraged to pursue a degree with an emphasis in cardiac rehabilitation or corporate wellness. At the time, I was living in a rural area with few corporate wellness programs or hospitals that offered cardiac care. Fortunately, I secured an internship as a cardiac technologist at the only hospital that offered cardiac rehabilitation. I was also gainfully employed elsewhere as a fitness director, personal trainer, and bartender.

As a personal trainer, clients expected me to know how to "fix" or rehabilitate their injuries. I was unsettled over the fact that clients assumed a personal trainer was a specialist in all things related to the body. Upon completion of my bachelor's degree, I felt that I was not

prepared to advise clients on injury management. Fortuitously, on the day of my graduation, a professor in the Kinesiology program publicized that his graduate assistant (GA) had unexpectedly quit. He inquired about whether I had considered pursuing a master's degree. I claimed that I was not interested in any more schooling; yet, truth be told, I was not satisfied with my educational knowledge base. After a few weeks of enduring the tedium of cardiac care, I contacted the professor. He immediately offered me the GA position in Biomechanics and Structural Kinesiology. I took the position and quit the cardiac internship.

While in the master's degree program, I studied with a classmate enrolled in the athletic training program. I had taken electives in the program and was intrigued by the courses. At the time, I did not believe it was possible for me to complete the athletic training degree, especially with the 1,800 internship hours under the direct supervision of a certified athletic trainer that were required. After talking to the program director, we agreed that I could enroll in the didactic courses concurrently with my master's classes and obtain the bulk of my internship hours after graduation.

Unfortunately, during the second summer of the master's degree program and after a long day at football camp and a longer night of bartending, I was called upon to substitute teach an early morning step aerobics class. While teaching the class, I stepped up on the step while hyperextending my left leg and felt a sharp pain in my low back. When I stepped back on to the floor, my leg was unable to stabilize my body. I had paralysis in my left leg. Several days later, a magnetic resonance image (MRI) indicated that I had herniated three discs in my lumbar region. I spent a year negotiating with the workman's compensation system to schedule a surgery. Eventually, a microdisectomy was performed at one level. After the

surgery, my drop foot disappeared, but my lumbar pain, left hip pain, and intermittent numbness remained.

One year later, while attempting to roll a keg into a bar well, I reinjured my lumbar spine. I was crippled by the injury. The pain was excruciating; basic tasks of daily living were impossible. I withdrew from my master's degree courses and took a leave from work to recover from a second surgery, a spinal fusion. The surgery provided some relief, but it was apparent that my function was limited. After months of grappling with the fact that I had an unsuccessful surgical outcome, I rectified my mindset and decided the outcome of the injury had come from a "higher" place. Obviously, there was a deeper lesson for me to learn. For pain relief, I sought out alternative medicine. I studied with psychics, mindfulness teachers, and Buddhist monks. I did yoga, chanted, meditated, medicated, acupunctured, acupressured, and swam, but none of these activities resolved my pain. Thankfully, prescription medication, biofeedback, and class recordings provided enough pain relief to complete my master's degree work.

After graduation, I completed my athletic training internship hours while employed at a physical therapy (PT) clinic that provided services to a local college and high school. Fortunately, I had an amazing mentor, and the job was a perfect fit. The clinic was affiliated with an orthopedic office where the athletic trainer (AT) performed many of the duties that were normally completed by a physician's assistant. I was exposed to a plethora of interventions and manual therapy techniques. In the morning, I was either in the orthopedics office or the PT clinic; in the afternoons, I dedicated my athletic training services to the college or high school sector. Despite the excellent mentorship I received, I was petrified at

the first football game that I was scheduled to cover alone; fortunately, I discovered my internship hours prepared me well for that moment.

After a few years of dabbling in different athletic arenas, I discovered that I preferred the high school setting and found part-time employment as an AT. My goal was to obtain full-time employment with this high school by developing a sports medicine program for the Allied Heath Care program in the science department. However, a teaching credential was required to teach the sports medicine classes. Consequently, I returned to the university I received my master's degree from to acquire a teaching credential. With this abundance of education, one would think that I would have had the "wealth of knowledge" I so eagerly sought when I first enrolled in college courses; yet the more education I completed, the more frustrated I became by how little I knew. Fortunately, this never impeded my desire to learn.

Despite having made the decision to be a life-long learner, I thought very little about how I was advancing my athletic training career. I had worked with amazing mentors and felt confident with manual therapy techniques and therapeutic exercise prescriptions. I was passionate and believed I held myself to a high standard as an AT. To stay current in the athletic training profession, I attended workshops, seminars, and conferences. As the years passed, however, I found myself pursuing continuing education opportunities outside of the traditional local, state, and national athletic training conferences. I often left the aforementioned conferences feeling frustrated with the topics presented, due to the lack of clinical application. By this time, I was teaching at a local university as an adjunct professor and was employed at a private high/middle school as an full time faculty member, athletic trainer, administrator, and strength and conditioning coach. I had diverse interests, but I found myself drifting away from what originally drew me to the athletic training profession: patient

care. One auspicious day, while perusing the National Athletic Trainers' Association (NATA) website, I read the advertisement for the Doctor of Athletic Training (DAT) program. In that moment, I knew the DAT was the educational opportunity I had been seeking.

Reflection on Clinical Competence

Professional Knowledge

After one week in the DAT program, it was apparent my internship program offered minimal didactic requirements. When the athletic training internship education program ended, the accreditation requirements appeared to provide ATs with a foundational knowledge base that I was lacking. However, I felt the internship program provided practical experience the accredited programs lacked. While I believed I had above average clinical skills, I realized that my patient care was primarily based on a rationale extrapolated from mentors, intuition, and personal experiences. Not only was I performing as an entry level AT, I had become a "knobologist:" a clinician who rarely researched interventions or modalities.

Through the DAT coursework and critical self-reflection, many of my clinical limitations and weaknesses have been addressed. My foundational knowledge has improved. Moreover, reflecting on patient outcomes and analyzing research applicable to my intervention choices has enhanced my clinical reasoning processes. Additionally, conducting research in the clinical setting has provided me with a communication element I was missing in my patient care. With the adolescent patient becoming more inquisitive each year (thanks to Dr. Google), I am prepared to explain my rationale for choosing interventions. I have developed a deeper understanding of the research and the theories related to the paradigms that I have integrated into my clinical practice. Overall, I am more confident and competent with my clinical decision-making, and I actually enjoy educating my patients.

Weaknesses and Barriers

The first summer of the DAT, I quickly discovered that I had marked weaknesses with foundational knowledge and clinical skills. Moreover, I had set up barriers that prevented me from developing as an AT. As far as being able to articulate my clinical, rehabilitation philosophy, or clinical reasoning, I found it surprisingly difficult. I had worked for almost 20 years as a certified AT, yet I was uncertain of my philosophical beliefs. My self-confidence level was further challenged when I realized that the terms *evidence-based practice*, *practice-based evidence*, and *action research* were completely foreign to me. The best way to overcome my weaknesses or barriers to becoming an advanced scholarly practitioner was to identify the areas that required improvement. In the following section, I discuss my weaknesses and barriers as well as simple solutions identified through DAT coursework, self-reflection, or class discussions that have remedied these weaknesses and barriers for the time being. I recognize that these areas will need to be continually evaluated after I have graduated from the DAT program.

- *Foundational Knowledge* - When I enrolled in the DAT, I presumed that I had adequate foundational knowledge. I had been an adjunct professor at a local university, and I trusted teaching core Kinesiology courses was an effective method of expanding my knowledge. Unfortunately, I had performed very little research beyond the textbook. After two years of didactic work, I have developed a much better understanding of the physiological, anatomical, vascular, mechanical, neural, and psychosocial systems. I have also developed a special interest in the mechanisms of pain and mindfulness techniques.

- *Traditional AT* - I define a traditional AT as an AT that rarely questions theories, paradigms, or his or her own philosophical beliefs. Because I valued the mentors in my internship program, most of my rationale for treatment selection or athletic training behaviors identified with a belief-based education. My AT mentors were not researchers. While I recognized and eventually understood that some errors/flaws existed with the way I had been taught, I believed the concepts I had learned were valid. Furthermore, like many traditional ATs, I had morphed into a triage AT. I classified musculoskeletal injuries with a cookie-cutter approach to treatment (e.g., ice, electrical stimulation, therapeutic exercise). Prior to the DAT, I believed my foundational knowledge, clinical reasoning, and assessment skills were sound; however, after one week in the DAT Program, I quickly realized that I was merely "chasing" the pain. My focus on only the pathoanatomics of injury was myopic.
- *Limited Interventions* - Modalities, such as ice, hot packs, ultrasound, electrical stimulation, manual therapy, and therapeutic exercise were my mainstay treatments before I enrolled in the DAT program. Because my clinic time was limited, my patient-care was focused on modalities and therapeutic exercises. I have since remedied this weakness, as the DAT has introduced me to a plethora of paradigms that I discuss in detail in the *Intervention* section of this chapter.
- *Patient care* - Before entering the DAT program, I believed that modalities and therapeutic exercises accelerated the patients' return to activity. For strains, sprains, aches, or pains, I treated the patient from two days to two weeks. If the patient demonstrated minimal improvement under my care, I would refer the patient to an MD to obtain a prescription to attend formal physical therapy. Subsequently, I would

continue to collaborate with the physical therapists and facilitate the patients' return to sport. After two years of DAT coursework, presentations, and research, I have a completely different perspective toward patient care. I rarely send a patient to formal physical therapy. I have implemented a systematic approach with all patient evaluations. Once I classify the patient, I am comfortable choosing a paradigm that will expedite the patient's safe return to sport. Through scholarly reflection, my clinical reasoning is much more deliberate with each patient. I understand that the patient's symptoms may not be the "driver" of the pain; thus, I evaluate the patient much more globally and holistically.

- *Outcome Measures* - Prior to the DAT, my outcomes measures consisted of a 0-10 pain scale. I normally only inquired about the patient's pain level pre-treatment. I did not have any outcomes data to support my belief that I was producing positive patient outcomes. I also did not understand the relevance of patient outcome measures. Upon learning the various outcome measures necessary to validate positive patient outcomes, I was humbled by my lack of knowledge (*Advanced Practice: Areas of Development).*
- *Barriers* - The number of barriers the DAT considered detrimental to the athletic training profession shocked me. One of my first DAT assignments was to identify my potential barriers. Because I adored my former athletic director (AD) and recognized that he, like me, was overwhelmed, I found myself managing the score table for volleyball and basketball games, setting up tournaments, calling officials, and filling water bottles. By taking on duties that were not part of my job description, I became even more stressed out and overwhelmed. The barriers assignment coupled with some

prodding from Dr. Nasypany forced me to come to terms with my truth. After acknowledging the fact that I had created my own reality, I presented my concerns to the school administration regarding my excess duties. I addressed budget issues related to an increase in staffing. Suffice to say, I no longer manage the score table or lead after school strength and conditioning workouts.

- *Roles* - The barriers list also indicated that I was contributing to the negative press ATs often unknowingly perpetuate. For example, despite an annual parent night, where I educated new members of the community regarding my multiple roles, parents still thought that I was either the school nurse or a personal trainer. Few parents understood the difference between an AD, AT, a personal trainer, a nurse, or a strength and conditioning coach. Unfortunately, I realized why the confusion existed: I had contributed to it by taking on the responsibility of each profession. While change takes time, I have made great strides, and I believe I have affected a positive cultural shift. Through bi-monthly parent newsletter articles, annual community presentations, and my involvement in the West Bay Athletic League Sports Medicine group, the community finally understands the difference between an AD, a personal trainer, a strength and conditioning coach, a nurse, and an AT.

Strengths

The DAT Program has afforded me the opportunity to make philosophical shifts that have enhanced my roles as a colleague and a clinician. After decades in the area of fitness and athletic training, I never critically analyzed my athletic training performance. As an educator, I am evaluated annually, but as the lone AT, I had never been formally evaluated. Identifying

and understanding my weaknesses was a critical piece to my scholarly development. Interestingly, this analysis allowed me to identify my true strengths.

- *Collegial Relationships* - I have an excellent working relationship with a wide range of health care professionals, which is extremely important as an AT in a college preparatory middle/high school setting. Because the DAT program challenged me to analyze my habitual patterns and behaviors, I have become much better at active listening, articulating my decision-making processes, and navigating difficult conversations. As an administrator, faculty member, and AT, the community at large respects my candor, humor, and professionalism. I have made a point of communicating my scholarly aptitudes through a variety of mediums (e.g., board presentations). Also, noteworthy, I realize I manage all my roles more proficiently.
- *Patient Rapport* - Because I chose to take on many roles in my current setting, I had limited time for patient care. Often I would rush a patient through an evaluation or treatment without explanation. The requirement to collect patient outcome measures in the DAT transformed my relationships with my patients. Clarifying the reasons I was incorporating the outcome measures into the evaluation process put many of my patients at ease. While it took some time and patience, the patients realized I was individualizing his or her treatment. Eventually, my clinical protocol had become more systematic and methodical; I slowed down and valued the educational component of patient care. Subsequently, as I discussed quality-of-life issues with the patients, conversations seamlessly evolved into educational opportunities to treat the patient more holistically.

- *Interventions* - Initially, the primary reason I entered the DAT Program was to enhance my clinical skills and explore new paradigms. The DAT provided innumerable opportunities for the student to investigate a wide array of treatment options. Research and the integration of new paradigms were invaluable additions to my clinical practice. I am competent and confident with the theories and application of the interventions that I have chosen to focus on, especially those related to the tendinopathy research. More importantly, the variety of treatments options I now use has reduced the amount of patients I refer to formal physical therapy. I have a newfound excitement for patient care and a clinical expertise that has transformed my clinical practice (*Areas of Advanced Practice).*
- *Classification* - Historically, I classified patients based on the site of their pain. I focused purely on special tests and the pathoanatomics of injuries. After creating a treatment based classification (TBC) algorithm with the tendinopathy research, I understand that classifying injuries is imperative to identifying the patient's true dysfunction. Additionally, incorporating global assessments into the evaluation process has made it possible to not only classify patients, but also help both the patient and me be more objective in recognizing physical improvements post-treatment. The addition of a classification system eliminated my shotgun approach and improved patient outcomes (Chapter 3).
- *Adaptable* - Recently, in a teacher evaluation, an administrator wrote that I was "adaptable" to any situation. At first glance, I was not sure if the word was complimentary or not. Eventually, I came to realize that being adaptable was a valuable character trait to possess. After two years in the DAT Program, I realize the

ability to adapt is actually imperative. The ebbs and flows related to a terminal degree are challenging. Because I am open to new challenges and try not to take anything too seriously, I recognize that my ability to adapt to any situation has been key to my success in the DAT. At times, I grew frustrated with the educational process. During those moments, I reflected on my growth both professionally and personally and was grateful that I had an innate ability to adapt and to focus on the "bigger picture."

- *Research* - Prior to the DAT, I inputted research data for another researcher and believed that that would be the extent of my research abilities. Despite the addition of the EBP category for continuing education, I did not understand the implications of the category for ATs until I was in the DAT Program. Evidence-based practice, PBE, and action research (AR) together, form the cornerstone of an advanced scholarly practitioner in the DAT. While the research component of the DAT was difficult to master, I am confident in my abilities to design, conduct, analyze, and disseminate scholarly research (Chapter 5). However, I realize research is truly an art form that develops with repetition. I am excited for the opportunity to continue to advance my development with a concussion research study beginning in Fall 2015 (Table 2).

The following clinical philosophy statements further demonstrate my clinical advancement from the first semester in the DAT didactic portion of the program to my final semester:

> *Fall 2013 - I will begin to make the shift in my clinical practice from a traditional athletic training room to a clinical setting that aligns more with the medical model; the implementation of patient outcomes and evidence-based practice will be an integral component. A valued part of this shift will be educating the entire community in a scholarly fashion. Lastly, I will be respectful and mindful of the variety of pathologies (emotional, physical, spiritual) pertinent to the patient in order to treat him/her more holistically.*

February 2015 - Implementing an evidence-based practice approach guided both my clinical and rehabilitation philosophies. Remaining open minded to new paradigms helped me attain my end goal, which was to reconcile my archaic traditional athletic trainer (AT) philosophy. Making this clinical shift was challenging. Historically, I utilized suboptimal traditional modalities (i.e., heat, cryo-therapy, massage, joint mobilizations, electrical stimulation, ultrasound, proprioceptive neuromuscular facilitation, manual therapy, therapeutic exercise) with no clinical reasoning skills associated with the treatment. While I believed that I had decent results with my patient's *treatments, I did not collect patient outcome measures, nor did I have any data to validate my treatment selection. Fortunately, through scholarly reflection and research, philosophical shifts have occurred. A thoughtful comprehensive evaluation coupled with a classification system guides sound clinical reasoning. The transition transpired when I took the time to perform scholarly research, reflect on patient outcomes, movement screens, postural assessments, and selected interventions. Of course, the over-arching goal of the rehabilitation phase continues to be an expedited, highly functional, safe return to activity. As demonstrated by my recent patient outcomes, utilizing indirect techniques such as, Mulligan, neurodynamics, positional release therapy (PRT), total motion release (TMR), primal reflex release therapy (PRRT), and myokinesthetics (MYK) expedites the rehabilitation phase. While my clinical and rehabilitation philosophical changes have not been fully embraced by the entire patient population, I recognize that the tincture of time is a necessary part of the process. Implementing new paradigms has also enhanced my holistic approach to patient care; I remain respectful and mindful of the variety of pathologies (emotional, physical, spiritual) that may influence a patient's outcome. Actively listening to the patient's history coupled with mindfulness techniques has alleviated much of the anxiety affiliated with injuries incurred by my younger patient population. While many of the chosen interventions I implement have not been well documented in the literature, I remain confident that adhering to evidence based practice while functioning, as a practice based evidence clinician will facilitate exemplary patient care.*

Path to Advanced Practice

Interventions

I now understand that there is much more to a patient's injury than the pathoanatomics. While tissue healing is a component of the rehabilitation process, close examination of the neural, vascular, mechanical, and psychosocial systems allows me to classify patients and help match treatment selection to patients individually. Integrating paradigms introduced in the DAT Program has insured the shift from mediocre patient care to

exemplary patient care. Scholarly exploration, research, and implementation of the following paradigms has been an integral component to the advancement of my clinical practice:

• *Mulligan Concept* (MC) *(Mulligan, 2010)* - The MC is a manual therapy treatment modality 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 was restricted and/or painful (Mulligan, 1993). If MWM produces a pain free, instant, and long lasting (PILL) effect, the mobilization is sustained throughout the entire movement. The success of this manual therapy treatment is attributed to the correction of a positional fault (Mulligan, 1993). MWM has become part of my evaluation process and an intervention I commonly choose to treat joint dysfunctions (Chapter 3)

• *Positional Release Therapy/Strain-counterstrain (PRT) (D'Ambrogio & Roth, 1997*; *Jones, 1981)* - Positional Release Therapy is used to interrupt the pain spasm cycle and treat somatic dysfunctions (Jones, 1981). Positional Release Therapy is an indirect technique that uses tender points (TPs) as a diagnostic guide. After locating the TP, the patient is placed in a position of comfort (POC) as the clinician palpates the affected TP with a sub-threshold pressure (Jones, 1981). If performed correctly, one of the goals of PRT is to reduce irregular muscle spindle activity (Speicher, 2006). Theoretically, PRT benefits the patient by positively affecting several pathophysiological events: neuromuscular hyperirritability, muscular hypertonicity, tissue tension, and inflammation (D'Ambrogio & Roth, 1997). My clinical research algorithm requires a quarter screen assessment of tender points as part of the

evaluation; therefore, as is the case with MC MWMs, I frequently choose this intervention to treat a variety of dysfunctions (Chapter 3).

• *Neurodynamics (Butler, 2000; Shacklock, 2005)* - According to Shacklock (2005), neurodynamic tests have two types of responses: mechanical and physiological. 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. Neural sliders are used to slide a neural structure relative to the surrounding tissues. Sliders produce movement among nerves without tension or compression of the structure. Neural tensioners produce tension in the nervous system (Butler, 2000). A positive neurodynamic response would manifest as change in the patient's symptoms or range of motion. Neurodynamic testing has become part of my evaluation process, too. If the neurodynamic test is positive, I consider using neural sliders or tensioners as my first intervention.

• *Total Motion Release (TMR) (Tom Delanso Baker)* - The philosophy behind TMR is based on the theory that the body works most efficiently when the body is in balance. Total Motion Release is a patient-friendly method of treatment wherein the patient exercises the "good" side to fix the "bad" side. Occasionally, the painful or dysfunctional side is actually restricted somewhere else in the body. To ease the restriction, patients are taught how to balance their own imbalances through a systematic progression. Initially, patients are taught sagittal movements. As the treatment advances, more planes of motion are utilized. The goal of TMR is to empower patients to treat their own restrictions related to injury. I have found the

technique works extremely well with adolescent patients; I teach them how to self-treat aches, pains, and strains. Additionally, I use specific TMR exercises as a warm-up for individuals or sport teams.

• *Primal Reflex Release Technique™ (PRRT*) *(John Iams, 2014)* - The body activates primal reflexes as a response to injury, trauma, and stress. These reflexes may persist and contribute to pain and dysfunction. Many of the PRRT techniques use cutaneous stimulation of either the ipsilateral antagonistic or contralateral agonist muscle groups to accomplish reciprocal inhibition of selected muscle groups. I use the technique alone, or as a reciprocal innervation technique to "reboot" the system or to compliment other treatments.

• *Selected Functional Movement System (SFMA) (Cook, 2010); Myokinesthetics (MYK) (Michael Uriarte, 2014)* - Implementing a movement screen (SFMA) or postural assessment (MYK) has become an integral part of the evaluation process. In the high school setting, time is rarely on my side; thus, I have adopted a system where I treat the patient for three sessions. If, by the third session, the patient has not improved, I integrate either the SFMA or the MYK postural assessment. Each of these assessments has challenged me to be more definitive with my clinical reasoning processes and to identify an appropriate classification system for the patient. If I become too focused on the patient's area of pain versus the "driver" of the pain, the assessments force me to take a step back and reassess the patient more globally.

• *Muscle Energy* (Chevan & Clapis, 2013) - The muscle energy technique is a manual therapy procedure that involves the voluntary contraction of a muscle in a precisely controlled direction at varying levels of intensity against a distinct counterforce

applied by the clinician. I was introduced to muscle energy in 1999, by a physical therapist that primarily treated low back pain and sciatica patients. While I was taught a more simplistic version of muscle energy for low back dysfunction than the Osteopathic Approach (Chevan & Clapis, 2013) or the techniques I researched from Richard Jackson, Diane Lee, or Dr. Nasypany, the basic principles are very similar. For the majority of my low back and hamstring patients, I use muscle energy to clear a pelvic dysfunction then treat.

• *Mindfulness* (Kabat-Zinn & Hanh, 2009) - Breath work and meditation impacts all systems of the body. Controlling the respiratory system provides the opportunity for the patient to take conscious control over their autonomic system, specifically the parasympathetic system (PNS). The PNS is believed to reduce anxiety by stimulating the vagus nerve, thereby increasing neurotransmitters in the brain that help decrease anxiety. Personally, I have used meditation techniques and breath work for decades. Mindfulness practice has recently become an integral part of my patient treatment; the calming result positively affects both the patient and me. Because pain can often be multifaceted and pain perception can be very individualized for each patient, integrating mindfulness has provided opportunities to connect with the patient at a much deeper level (e.g., emotionally, spiritually).

As I research new paradigms, my clinical philosophy continues to evolve. While I only discussed a few paradigms that I was introduced to in the DAT, I am becoming well versed in many treatment paradigms (Tables 1 and 2). Occasionally, my patient (or rather, *parent*) population pleads for traditional modalities. When I take the time to explain the theory and the potential benefits of the "new" paradigm, the majority of the patient's (parents)

recognize that my treatment selection is in the best interest of the patient. There are moments when I become annoyed by these conversations. Fortunately, continuing education opportunities, such as the TMR webinar (November 2014, March 2015) provides perspective. In the webinar, Tom Dalanzo discussed his philosophical approach with reluctant patients. He said that he asks the patient, "If you have pain after two hours, would you rather treat yourself or pay $50.00 to see me?" I had to chuckle when I heard his response. I thought to myself, "Even the 'creators' of the paradigms struggle with patient buy-in." Scholarly research and continuing education will be a critical component of continuing my clinical advancement beyond the DAT (Table 2).

Incorporating the myriad of paradigms introduced in the DAT Program completely changed my rehabilitation philosophy. The following low back rehabilitation philosophy statement from September 2014 demonstrates my development in one area of rehabilitation:

Historically, my primary rehabilitation philosophy for low back pain patients was adopted from a combination of Back School, McKenzie, and Sahrman's principles. Historically, my clinical reasoning was if it's not fractured, I would focus on neutral spine, core training, and centralizing the pain. While I will continue to employ some of these techniques with patient's therapeutic exercise routine, the most important shift philosophically has been integrating a more systematic approach to the patient evaluation and treatment. Inclusion of a more robust classification system based on evidence (i.e., Treatment Based Classification Approach) will guide my examination. Specific outcome measures to low back pain (i.e., modified Owestry) will be included with additional outcomes measures (NRS, DPAS, GRC, PSFS) to improve scholarly reflection and clinical reasoning for the treatment of low back pain. Incorporating interventions such as Positional Release Therapy (PRT), Mulligan (e.g., SNAGs, NAGs), manipulations (HVLA), and muscle energy will enhance patient treatments by effecting chemical, neural, or mechanical dysfunction. If MCID's are not demonstrated in patient outcomes after three treatment sessions, either the SFMA screening or MYK postural assessment will be used to evaluate the patient more globally with the understanding that low back pain is frequently multifaceted. Finally, I will be more cognizant of the biopsychosocial impact on the chronic pain patient. Educating the patient on mindfulness techniques, posture, and emotional freedom techniques will become an integral part of patient care.

Areas of Development

The DAT Program has challenged me both personally and professionally. Clinical reform has been a fundamental component to my development as an advanced practitioner. Change can be a slow, methodical process, and it requires patience, flexibility, and the courage to be uncomfortable. To facilitate paradigm shifts, I needed to immerse myself in didactic coursework, presentations, and scholarly research. Through the DAT curriculum, online courses, workshops, and seminars, my traditional AT philosophy was constantly challenged. As my clinical philosophy became more sophisticated, I reflected back to "the poster" (Nasypany, Seegmiller, & Baker, 2012) that was introduced to our cohort in Summer 2013. While it appeared extremely complicated at the time, I now understand that the process of coupling the "best of what is known from the scientific evidence" (Evidence Based Practice - EBP) with research collected through a systematic inquiry (Practice Based Evidence - PBE) constitutes best practices as an advanced practitioner. Making a concerted effort to collect patient outcomes, develop *a priori* or post hoc research designs, conduct research, and implement "new" paradigms has advanced both my clinical skill set and my clinical reasoning processes. The following are key components that contributed to my scholarly development as a PBE clinician:

- *Patient Outcome Measures* - Patient reported outcome measures are a critical component of validating the benefits of a chosen intervention. Daily treatment sessions include a pre- and post-numeric rating scale (NRS). Additional outcome measures, such as the Disablement and Physical Activity Scale (DPA Scale) and Global Rating of Change (GRC) were distributed to the patient (e.g. first and third visit). If the patient's diagnosis is related to tendon pain, more specific evaluation

forms are provided (e.g., Visa-A, Visa-P). Finally, manual muscle testing and range of motion measurements are documented. Tracking patient outcomes insures quantifiable measures that help guide, validate, and support treatment selection. As I reflect on the implementation of new paradigms in my clinical practice, my patients' outcome measures have been and continue to be invaluable when selecting an appropriate intervention to address the patient's complaints of pain or dysfunction. At the middle/upper school setting, daily patient care can be overwhelming. Reflection on patient outcome measures changed my evaluation protocol to include a more multifaceted approach versus focusing merely on the site of the injury.

• *Action Research* - Action Research (AR) is an approach commonly used for improving conditions and practices in the healthcare environment (Koshy, Koshy, & Waterman, 2011). Clinicians improve patient care through systematic enquiry and research. When I began conducting AR, I started to understand the necessity of developing a hypothesis with patient evaluations. Over time, my proficiency as a clinician was realized more completely through *a priori* research designs coupled with my involvement in the tendinopathy research. A comprehensive literature review on epidemiology, etiology, anatomy, physiology, classification systems, and various treatments for tendon pain has been instrumental in my pursuit to become an advanced scholarly practitioner (Chapter 4).

• *Conducting Research* - After conducting my own research, I understand the necessity of developing valid and reproducible research methods (Chapter 5). I appreciate that while the research design may be valid and reliable, the lack of patient numbers can diminish the potential of transferring findings either locally or globally.

> The TBC algorithm that was developed for the tendinopathy research has forced me to be more deliberate and systematic in my evaluation process; consequently, the AR process guided me in my quest to achieve quality patient care (Chapter 5).

Professional Goals

Insightful reflection, clinical reasoning, and reexamination of my goals have provided me with numerous opportunities to refine my path to becoming an advanced practitioner (Tables 1 and 2). Through the didactic coursework, I have discovered where my weaknesses manifested themselves in my clinical setting, and I was able to create a positive philosophical shift in my clinical practice. Moreover, I am more cognizant of the whole patient, not just the injured site. As David Butler says (Butler, NES), "It is not just physical handling exercises, but also communication exercises." I realize that despite the frenetic energy in the clinic, I can "ground" myself and attend to each patient as a more fully present clinician. Of course, implementing a more mindful approach in patient care is often easier said than done. Providing exemplary patient care will forever be a work in progress (Table 2).

Each day, the DAT Program challenged my traditional AT philosophy and clinical decision-making. With both scholarly reflection and the integration of a more EBP, my practice has developed into a PBE clinic that disseminates knowledge to the athletic training profession both locally and globally. The knowledge I have obtained though the DAT Program has enhanced so many realms, from the classroom to the clinic; however, I realize being an advanced scholarly clinician will continue to be a circuitous journey. The Plan of Advanced Practice has become an authentic reflection that will continue to guide me on my quest to advanced scholarly clinician (Tables 2.1 and 2.2).

Professional Future - Beyond the DAT

With a renewed confidence and competence as an advanced scholarly clinician, I look forward to my future in the athletic training profession. Prior to the DAT, I was not convinced that my passion was in the clinical setting. The DAT Program has encouraged, inspired, and motivated me to give back to the profession. I appreciate that athletic training students need preceptors who are involved in scholarly research and are not stuck in the "traditional" AT mindset. With an advanced knowledge base in both clinical skills and research, the DAT Program has prepared me to be an excellent preceptor. Moreover, I will be facilitating research for a concussion study with Stanford University. The collaborative opportunities with Dr. Ghajar and the concussion research team at Stanford University will contribute to my advancement as a researcher. I acknowledge the fact that part of "giving" back to the profession is continuing my own scholarly research and disseminating my findings for the education of others.

On a personal note, the DAT has reawakened my desire to educate my patients and my community on the benefits of mindfulness (e.g., yoga, meditation, breath work). As an adolescent athlete, I was introduced to the world of yoga, visualization, meditation, and energy work. I have continued to practice and teach some form of "mindfulness" techniques throughout my life. While in the DAT Program, I studied several books and articles on chronic pain, the neurological system, energy work, and mindfulness; it was a seamless transition to become involved in specific research for a patient interested in integrating mindfulness into his rehabilitation. Eventually, my research led to a case study that incorporated a mindfulness component to a patient's rehabilitation protocol. (Syvertson, Baker, Nasypany, forthcoming). Exploring the mindfulness and energetic realm with this

patient provided a greater understanding of how this paradigm benefits patient care and how the stigma surrounding mindfulness has decreased since its presentation as a secular practice. I remain passionate with my own mindfulness practice and look forward to educating patients on mindfulness techniques, while continuing to conduct research in this area.

DAT Accomplishments

Without a definitive plan, goals are extremely hard to attain. The PoAP became my plan that outlined the path to my personal and professional advancement. Through constant reflection and critical self-evaluation, the PoAP forced me to remain focused on my journey as I was able to analyze my growth as a clinician. Subsequently, I was able to effect a change in my clinical practice as I made the progression to advanced scholarly practitioner. While the DAT Program was extremely challenging, overcoming those challenges was incredibly rewarding. The amount of growth and development that I have experienced on this journey cannot be measured by purely objective measures; it can, however, be observed as I continue my advancement as a scholarly clinician well beyond the DAT Program (Tables 2.1 and 2.2).

Justification for Plan of Advanced Practice

The PoAP will remain a fluid document that provides the structure for my continued advancement beyond the DAT. My development in the DAT program was reinforced by consistent self-reflection on the goals and objectives outlined in my PoAP. In my first version of the PoAP, I described what compelled me to pursue a terminal degree; I had begun to design my path to advanced practice. I realized that remaining open to change, philosophically, and professionally, was a necessary requirement to my success in the program. As I addressed my barriers, strengths, and weaknesses as a clinician, I identified specific goals and objectives that would facilitate my development in each of these areas. The

PoAP became an invaluable detailed plan that required continued analysis as I chronicled my personal and professional experiences, goals, and objectives.

I have progressed on my path in the DAT by remaining focused on the essential components of scholarly and clinical development as identified in my PoAP: action research, scholarly reflection, the collection of patient outcomes, evaluations, implementation of new paradigms, theoretical models, and treatment strategies into my clinical practice. My development has been further validated in the publication of scholarly products that have been disseminated to the AT community. As I reflect back to my first day in the DAT Program, I am pleased to acknowledge that I have evolved beyond my expectations both as a clinician and a researcher. As I transition from DAT student to an advanced scholarly representative of the athletic training profession, I am excited to begin the next part of my journey as a preceptor, educator, researcher, and clinician. The PoAP will remain the blueprint for my professional future and will be a critical component of my development well beyond the DAT.

Table 2.1 Assessment of Scholarly Advancement

Scholarly Focus Area	Method of Advancement	Date Completed
Functional Movement Screen/Selected Functional Movement Assessment	NES: Functional Movement Screen (Cook, Kiesel)	August 2013
	NES: Selected Functional Movement Assessment (Rose, Voight)	August 2013
	Read: Movement Book (Cook, 2010)	December 2013
	Reread: Athletic Body in Balance (Cook, 2003)	January 2014
	SFMA Workshop (Plinsky)	March 2014
Mulligan	NES: Upper and Lower Quadrant (Mulligan)	August 2013
	Read: Manual Therapy: Nags, Snags, MWM's, etc. (Mulligan, 2010)	September 2013
	Reread: Jones Strain-Counterstrain (Jones et al., 1995)	December 2013
	Mulligan/Wilk; Diagnosis and Treatment of the Upper and Lower Quadrant Seminar	September 2014
	Mulligan Upper Extremity Workshop	August 2014
	Action Research – Data Collection	March 2015
	Mulligan Concept - Lower Quadrant, Mobilizations with Movement, 'SNAGS', SMWLM' & MORE	TBD
Neurodynamics	Read: The Sensitive Nervous System (Butler, 2000)	July 2014
	Read: Clinical Neurodynamics (Shacklock, 2005)	On-going
	NES: Mobilization of the Nervous System The Mobile Nervous System, Clinical Reasoning and Pain (Butler)	December 2013
	Neurodynamics Presentation UID	July 2014
Positional Release Therapy	Read: Positional Release Therapy Assessment & Treatment of Musculoskeletal Dysfunction (D'Ambrogio & Roth, 1997)	December 2014
	PRT-i Spine/Pelvis Workshop (Speicher)	July 2013
	PRT-i Lower Extremity Workshop	July 2014
	Action Research – Data Collection	March 2015
Primal Reflex Release Therapy	Home Study Course	December 2013
	Advanced Course	April 2014
Myokinesthetics	Upper Extremity Workshop	August 2014
	Postural Screening Workshop	July 2014
	Lower Extremity: Home Study Course	September 2014

Total Motion Release	Level I: Home Study Course	October 2014
	Level II: Home Study Course	March 2015
Muscle Energy	Pelvic Dysfunction Workshop	August 1999
	Research - Diane Lee, Richard Jackson	December 2013
DoCPI: Tendinopathy	Problem Statement, Review of Literature, Methods	January 2015
	Data Collection	April 2015
	Chapter 1	April 2015
	Chapter 2	April 2015
	Chapter 3	April 2015
	Chapter 4	April 2015
	Chapter 5	May 2015
Mindfulness	Mindfulness Training Workshop	May 2014
	Transcendental Meditation Workshop	January 2014
	Audible: The Mindbody Prescription (Sarno)	August 2013
	Audible: Full Catastrophe Living (Kabat-Zinn)	October 2013
	Audible: Adventures in Mindfulness (Kabat-Zinn	December 2013
	Audible: Why Zebras Don't Get Ulcers (Sapolsky)	March 2014
	Audible: Mind Over Medicine (Rankin)	October 2014
	Associative Awareness Technique™	July 2014
NES Videos Advancement of Foundational knowledge	Recognition & Treatment of Shoulder Injuries in the Overhead Athlete (Wilk)	October 2013
	Diagnosis and Treatment of Muscle Imbalances and Associated Movement Impairment Syndromes (Sahrmann)	March 2014
	Myofascial Release vs. Feldenkrais Method - A Comparison and Contrast (Burkart)	January 2014
	Managing Lower Limb Problems (McConnell)	February 2014
	Assessment of the Lumbar Spine (Magee)	April 2014
	Functional Anatomy: A Cadaver Review of the Shoulder, Hip, and Pelvis (Mettler)	May 2014
	Functional Relationships of the Lower Half (Jackson)	April 2015

Scholarly Research	Abstract Submitted (not accepted): WFATT: The Effects of Indirect Tendinopathy Treatment on Patellar Tendon Pain Using a Manual Therapy Algorithm	January 2015
	Article (forthcoming): IJATT: Avulsion Fracture of the Anterior Superior Iliac Spine and the Iliac Crest: A mindfulness approach to rehabilitation	May 2015

Table 2.2 Scholarly Development: Post-DAT

Scholarly Focus Area	Method of Advancement	Date to be Completed
Mulligan	Mulligan Lower Extremity Workshop	TBD
	Mulligan Concept - Lower Quadrant, Mobilizations with Movement, 'SNAGS,' ‘SMWLM' & MORE	TBD
Spinal Manipulation	Total Motion Physical Therapy Seminar	TBD 2016
Primal Reflex Release Techniques	Repeat: Advanced Course	TBD 2015
	Appointment with John Iams	TBD 2016
DoCPI: Tendinopathy	Chapter 1	July 2015
	Chapter 2	July 2015
	Chapter 3	July 2015
	Chapter 4	July 2015
	Chapter 5	August 2015
	Final Draft DoCPI	October 2015
	Defend DoCPI Submit DoCPI to Department Chair Submit Bond Copy of DoCPI to COGS	November 2015 November 2015 November 2015
Mindfulness	Spiritual Direction: Learning to Listen to Your Soul: Caroline Myss	TBD
	Energy Medicine Donna Eden Workshop: San Francisco	TBD
	International Conference on Mindfulness in Rome	June 2016
	Audible: Buddha's Brain: The Practical Neuroscience of Happiness, Love, and Wisdom (Hanson)	August 2015
	Mind Valley: Advanced Creative Visualization	July 2015
	Still Quiet Place: 10-Week Online Practicum for K-12 Educators and Allied Professionals	October - December 2015

Scholarly Research	Submit Abstract WFATT: The Effects of Indirect Tendinopathy Treatment on Tendon Pain Using a Manual Therapy Algorithm (Denied)	January 2015
	Submit Abstract NATA: The Effects of Indirect Tendinopathy Treatment on Tendon Pain Using a Manual Therapy Algorithm	July 2015
	Submit Abstract FWATA: The Effects of Indirect Tendinopathy Treatment on Tendon Pain Using a Manual Therapy Algorithm	TBD
	Presentation: Community Based Concussion Updates with Stanford University Concussion and Brain Performance Center	August 2015
	Submit Manuscripts: JMMT, JAT, JSR, IJATT 1. The Effects of Indirect Tendinopathy Treatment on Patellar Tendon Pain Using a Manual Therapy Algorithm: A pilot study 2. An Exploratory Analysis of a Treatment Based Classification Algorithm to Treat Patellar Tendinopathy: A Case Series 3. Analysis of a Treatment-Based Classification Algorithm to Treat Achilles Tendinopathy: A Case Series 4. An Exploratory Examination of a Treatment Based Classification Algorithm to Treat Lateral Epicondylalgia: A Case Series	TBD
Concussion	The 10th Annual National Summit on Sports Concussion: Sports and Concussion	June 2016
	First Annual Community-Based Concussion Conference: Stanford Concussion and Brain Trauma Center, The Brain Trauma Foundation and the California Concussion Institute	August 2015
	Stanford Concussion and Brain Performance Center: Eye Tracking Study	August 2015 to July 2016

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CHAPTER 3

OUTCOME SUMMARY, RESIDENCY FINDINGS, AND IMPACT

A critical component to the DAT student's scholarly advancement is the clinical residency. The clinical residency is designed to challenge the DAT student to evaluate philosophical beliefs, foundational knowledge, clinical skills, treatment techniques, evaluation protocols, and clinical reasoning processes in an effort to advance clinical practice. The DAT student improves knowledge through didactic instruction, but scholarly reflection, the analysis of patient outcome measures, and action research is the medium the student employs to critically evaluate patient care objectively. The following narrative chronicles the struggles, challenges, and successes that occurred during my clinical residency. Through scholarly research, meaningful self-reflections, journal entries, analyzing patient outcome measures, and applied research, my clinical philosophy has evolved to that of an advanced scholarly practitioner.

Embracing Change

The mission and philosophy of the DAT Program intrigued me. Professionally, I was ready for a challenge. As I researched terminal degrees, the DAT program appeared to provide the practical guidelines I desired to advance my clinical practice. I realized to fully embrace the process, I would need to remain open and willing to critically evaluate my philosophical beliefs and be able to respect the constructive criticism of my attending clinician and my professors. Philosophically, the DAT Program had clinical views, opinions, and paradigms that were contrary to my own. I had been treating the musculoskeletal system and the source of pain for decades and considered myself an "expert" in the rehabilitation of injuries. My primary treatment protocols were traditional modalities (e.g., electrical

stimulation, ultrasound) and therapeutic exercises. Because the health care community respected my ability to evaluate, diagnose, and safely return patients back to sport in a timely manner, I considered myself above average in my profession. I entered the DAT Program because I was interested in exploring new paradigms and refining my clinical skills.

When I instituted a computerized injury tracking system, I believed the patient data I collected was evidence that I was producing positive patient outcomes. I studiously inputted patient data daily and printed out graphs annually for my school's administration and board members. I tracked injury rates and the number of patients I treated annually/seasonally/daily. After one lecture in the DAT Program, I realized the information I gathered from the computerized system was not patient outcome measures. I quickly discovered I had many weaknesses beyond understanding outcome measures that would be exposed while in the DAT Program. I would need to identify the challenges ahead of me to develop as an advanced scholarly practitioner.

Overcoming Challenges

Exposing my weaknesses in the manner required in the DAT Program was not an easy feat. The DAT challenged me to leave my ego at the door and let go of any preconceived notions related to the profession of athletic training. Through didactic instruction, scholarly reflection, research, and insightful discussions, my clinical philosophy shifted to a more patient-oriented evidence (POE) practice. Becoming a patient-centered clinician, I recognized there was more to patient care then disease-oriented evidence (DOE) (e.g. goniometer measurements). In my previous practice as an AT, I was predominantly a DOE practitioner. With a patient-centered approach, the experience of each patient mattered from a quality of life perspective. Patient-centered care focused on creating a more collaborative relationship

between the patient and the clinician (Epstein, & Street, 2011). Patient-centered care resonated with my personal philosophy: to be more mindful, informative, and empathetic to the patient's needs (Epstein, & Street, 2011); however, it was not part of my clinical philosophy (i.e., focused on DOE). Interestingly, because I had been in employed as an AT in the same middle/high school for the past 15 years, creating a philosophical shift in my clinical setting would prove to be more difficult than I had anticipated; as I began to advance as a clinician, I noted my struggles and successes were best illustrated through the analysis of my patient outcomes.

Data Analysis, Results, and Reflections

Fall I 2013

The Summer I immersion in the DAT Program inspired me to eliminate the use of traditional modalities, overcome personal and professional barriers, review foundational knowledge, research and implement new paradigms. Requiring patient appointments would benefit patient care as I would have more time to evaluate and treat patients more effectively. Developing a classification system, adopting a movement screening, and creating a documentation system for the collection of patient outcomes would benefit my clinical reasoning processes. By four weeks into the fall semester, I was overwhelmed with all the changes I was attempting to adopt. I reconsidered my immediate goals and chose to focus my attention on researching and implementing patient outcome measures and "new" paradigms.

The DAT curriculum required the collection of patient outcome measures to assist the DAT student with consistent and comparable patient outcomes regarding pain, disability, and function. The DPA Scale had been validated as an objective psychometric measure (Vela & Denegar, 2010a, 2010b). A meaningful clinical important difference (MCID) is a change of

nine points for an acute injury and six points for chronic injuries. The numeric rating scale (NRS) was established as a valid and reliable scale (Phan, Blome, Fritz, Gerss, Reich, Ebata, Augustin, Szepietowski, Stander, 2012) for pain, with an MCID of two points or 30% reduction (Farrar, Young, LaMourauz, Werth, & Poole, 2001). The outcome measures seemed user friendly. I was familiar with NRS scores because I had used a 0 to 10 scale to assess a patient's worst pain score prior to the DAT. However, collecting and tracking these simple patient outcome measures was much more challenging than I had expected. In the beginning, I attempted to collect outcome measures on every patient. Fortunately, Monday night class discussions, blogs, and scholarly research provided opportunities to discover the "how, who, and when" of collecting patient outcome measures. Despite classmates attempting to assist each other long distance, creating a documentation system to store patient outcomes proved to be difficult to create.

To complicate matters, the fall season brought an inordinate amount of concussions, which were extremely time intensive for me. Consequently, patient care suffered. I began to revert back into old patterns of behaviors. In an effort to save time with patient care, I started to exploit traditional modalities (e.g., ice, electrical stimulation). I documented minimal patient outcomes. As I wrote my patient reflections, I contemplated the clinical goals I proposed in my Plan of Advance Practice (PoAP) (Chapter 2). If I was to become a more advanced practitioner, I had to commit myself to the DAT curriculum and process. I made a more concerted effort to, at the very least, integrate the "new" paradigms introduced during the summer session into my patient care.

While I was not currently using Strain Counter-Strain (Jones, 1981), I was familiar with the method. Positional Release Therapy (PRT) (D'Ambrogio & Roth, 1997; Speicher,

2006) was a similar technique; it was my first exploration into a "new" paradigm. My first PRT patient was a football player who landed on a hyperextended elbow and suffered a forearm flexor strain. The patient's arm was swollen and tender with limited active range of motion (AROM) due to pain. The patient lacked 10 degrees of elbow extension and 30 degrees of wrist extension and was unable to perform manual muscle tests (MMT). The patient rated his pain as 7 out of 10 on the NRS and reported a score of 34 out of 64 on the DPA Scale. The patient's pediatric physician ruled out fracture and the patient returned to the clinic seven days post-injury with minimal swelling present and decreased pain (NRS 6 of 10). The patient's AROM had improved; he was only lacking four degrees of elbow extension and 10 degrees of wrist extension. With a decrease in swelling and pain, I was able to check for tender points (TPs). I located and treated three TPs. Following PRT treatments, the patient's NRS score decreased from 6 out of 10 to a 3 out of 10. The following day, the patient reported that his improvements had remained (NRS 4 of 10). He presented with only two of the three TPs. I treated the remaining TPs with PRT. The treatment completely resolved the patient's pain (NRS 0 of 10). At two weeks post injury, the patient's DPA Scale was 2 of 64 and his AROM was within normal limits (WNL).

The second PRT treatment that I performed on this patient will be forever etched in my memory. After the treatment, the patient's response to being pain free was, "That was magic dude." I was pleased with the changes reported in the patient outcome measures; my confidence level increased after successfully treating a patient with a new technique and tracking the improvement with outcome measures.

Positional Release Therapy became my new intervention for musculotendinous injuries. While I was successfully treating patients with PRT, my clinical reasoning for

choosing the technique was lacking. If PRT did not demonstrate a positive patient outcome, I merely attempted another intervention. During first semester, I had become a "shotgun" therapist; if one treatment did not provide pain relief or increase the patient's function, I randomly selected another intervention. My clinical decision-making was lacking; yet, I continued to investigate new paradigms to develop a better understanding of the theories and reduce my randomized treatments.

I had been introduced to Total Motion Release (TMR) and the SFMA during the summer; however, I did not discover the benefit of utilizing these paradigms in my clinical practice until after I was unable to successfully treat a hamstring injury with PRT. While performing a split, a patient reported she "heard and felt an audible tear." Because the injury had occurred during summer session, she immediately went to see a physician (MD) and was diagnosed with a "muscle pull." The MD prescribed four weeks of formal physical therapy. At six weeks post-injury, the patient started the cross-country season. One week later, the patient was in my clinic complaining of chronic hamstring tendon pain and tightness. The patient's pain on the NRS was 5 of 10 with running. The patient's straight leg raise lacked seven degrees of extension from the ipsilateral side. Manual muscle testing demonstrated equal strength bilaterally. Her DPA Scale was low (18 of 64). I discovered two TPs near the ischial tuberosity and treated with PRT based on that clinical finding. PRT did not provide pain relief or increase function. After three PRT treatments, the patient's post treatment NRS scores remained the same.

As a result of the patient's poor outcomes, I evaluated her low back and pelvis. I detected a slight pelvic upslip on the ipsilateral side of her hamstring pain. Because I was familiar with muscle energy pre-DAT, I chose a muscle energy technique to treat the pelvic

dysfunction. I corrected the pelvic dysfunction, but she demonstrated only a slight change in her pain score (one point decrease on the NRS). At this point, I was frustrated with my lack of clinical reasoning. I submitted a WordPress blog for feedback regarding the patient. My classmates recommended using the SFMA to guide my care. I had performed the SFMA top tier on several adolescent patients, but I was incorporating the SFMA more as a pre/post assessment to determine if the chosen intervention improved the patient's SFMA dysfunction. I decided to try the assessment on the patient. The SFMA indicated functional painful movements in all movement patterns for the patient. Based on patient outcomes and SFMA findings, I was discouraged and unsure how to continue; I applied a hot pack to her hamstring and reviewed the SFMA demonstration on the Northeast Seminar (NES) website to determine if I had missed something.

While watching the SFMA video, I shuffled through some paper work and uncovered the TMR sheet. I had completely forgotten about TMR. I remembered Dr. Nasypany had mentioned in class that he sometimes used TMR for patients with numerous imbalances on the SFMA. Immediately, I had the patient complete the TMR Fab 6 assessment; the seated leg raise demonstrated the largest discrepancy. After completing three sets of ten repetitions with the seated leg raise, the patient reported a full resolution of her pain on the NRS (0 of 10). I reevaluated the patient's SFMA; she was functional normal in all movement patterns except the overhead squat. The patient was elated. After a two mile run at 80% maximum, the patient returned to the clinic and claimed she was "pain-free." Throughout the season, she continued to self-monitor her symptoms with TMR. She periodically returned to the clinic to discuss aches/pains or to confirm she was performing the TMR correctly. She was able to complete her cross-country season without requiring further treatment on her injured hamstring.

In my continued pursuit to reduce my "shot gun" approach, during the summer session, I was introduced to the paradigm Primal Reflex Release Therapy™ (PRRT) theory. I investigated PRRT through an on-line course. The theory for the paradigm was based on treating the neurophysiological system, which is often "ignored or overlooked" (Iams, 1999). I had been functioning as a musculoskeletal clinician who focused on the site of pain. While I understood the concepts of reciprocal innervation, muscle joint receptors, and spinal modulation, I had never considered a "startle" reflex. According to the PRRT theory, reflexes are hard wired into the nervous system to protect the body. When a person experiences a painful or startling event, an over-stimulation of the body's primal reflexes is triggered in an attempt to protect the body. The reflex can manifest itself in a patient as a startle or a withdrawal reflex (Iams, 1999).

My first treatment with PRRT presented with two cross-country runners that I classified with medial tibia stress syndrome (MTSS) as a working diagnosis. Both patients' experienced palpable pain at the distal one-third of the medial aspect of the tibia and experienced an increase in pain with walking (4 of 10 on NRS) and running (8 of 10 on NRS). The patients' DPA Scale scores were very similar (38 and 42 of 64). I performed PRRT on both MTSS patients. After the treatment, one patient reported complete resolution of pain (0 of 10). The other patient was not convinced the treatment provided any relief and reported his pain remained 4 of 10 on the NRS. I performed a second treatment of PRRT on him. After the second treatment, he also reported a complete resolution of pain (0 of 10 NRS). I allowed both patients to return to cross country practice that day. When I returned from football practice, both patients were in the clinic icing their shins. The patients claimed they were pain free during practice, but wanted to be "proactive and keep icing." As I reflected on the PRRT

outcomes, I realized I had a better understanding of the theories and the practical application of the "new" paradigms; however, my patient outcomes were inconsistent. I was reducing my "shot gun" approach, but collecting outcomes measures was necessary to provide evidence that my treatment selections were effective.

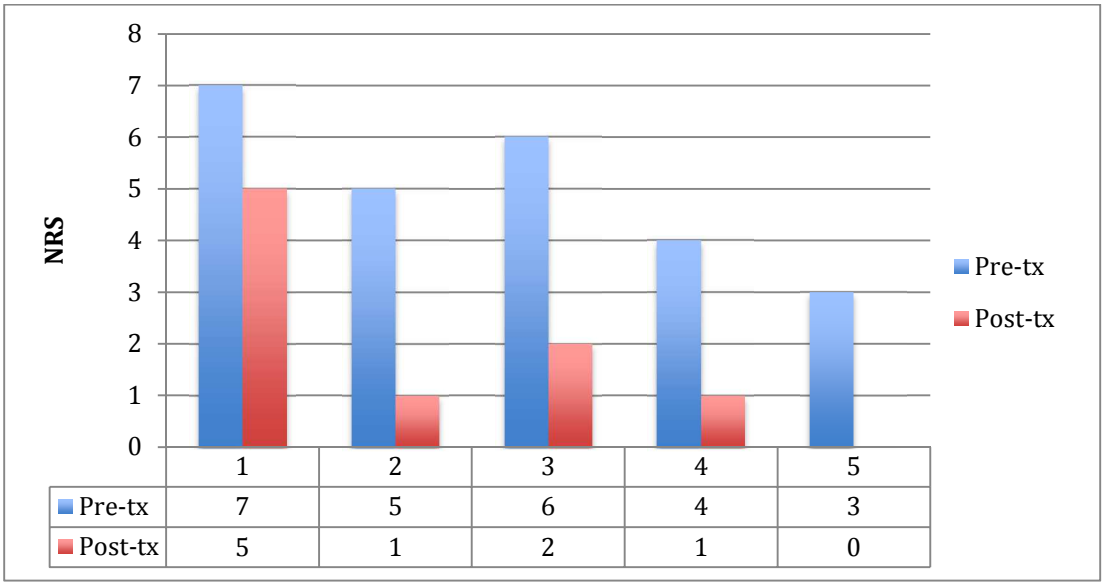
As I continued to research and practice "new" paradigms, I could not have predicted the impact the Mulligan Concept (MC) would have on my patient care. I had watched the NES MC videos multiple times and read the MC book; however, attending a Mulligan/Wilk seminar enhanced my understanding of the theory. With each treatment, Brian Mulligan performed on me; I felt the amount of force that he applied to the joint when he performed the various techniques. I left the seminar more confident of my MC clinical skill set, but still needed practical application and patient outcomes to determine its usefulness in my practice.

My first MC patient #18001 had suffered a lateral ankle sprain (LAS) playing badminton. The patient had already been evaluated in an emergency department (ED) where fracture was ruled out. She was sent home in a boot, crutched, and non-weight bearing (NWB). The physician instructed her to remain NWB for one week; he instructed her to gradually progress to walking; her prognosis was six to eight weeks before return to activity. The patient was devastated. Immediately following the ED visit, I evaluated the patient. The patient's pain level at rest was 7 of 10 (NRS), while her DPA Scale score was 44 of 64. She was unwilling to weight bear. She had palpable pain on the anterior talofibular and calcaneofibular ligaments. She was too swollen and tender to perform stress tests or AROM measurements. While I was concerned about the amount of swelling present, I was confident the MC LAS mobilization with movement (MWM) would decrease her pain and increase her function. After I explained the theory behind the MWM treatment, the patient (and her

mother) agreed to the treatment. The patient was extremely tender at the distal aspect of the fibula. I used a foam pad and found a “pain free” one to two inches proximal on the fibula. I performed three sets of six repetitions of MWM with overpressure. After treatment, her pain decreased to a 4 of 10 (NRS).

The next day, the patient presented with an increase in swelling and ecchymosis in the lateral ankle area and reported a pain score of 5 of 10 (NRS). I completely forgot to apply the MC tape application to maintain the fibular position and wondered if it could have been one of the contributing factors to her increase in symptoms. I treated the same site with MWM, but increased to 3 sets of 10 repetitions with overpressure. After the last set, the patient’s NRS score was 1 of 10. I asked the patient to walk over to the electrical stimulation machine (approximately10 steps). Understandably, the patient was apprehensive to walk, but once she did, she claimed that her ankle was “pain free.” I applied the MC tape application to support the treatment. After three sessions of MWM treatment, the patient reported a complete resolution of pain (NRS 0 of 10) and returned to badminton 13 days post injury. While I had questioned the “positional fault” theory during the summer session, the patient’s positive response to MWM treatment coupled with NRS pain scores that met MCID after each treatment confirmed that MWM was a viable treatment option for LAS patients (Table 3.1).

Table 3.1 Patient #18001 Fall I Pre- and Post- NRS Score - MWM Treatment of LAS



	1	2	3	4	5
Pre-tx	7	5	6	4	3
Post-tx	5	1	2	1	0

Key: tx - treatment

When I reflect back on the paradigms I implemented during Fall I, I was impressed with my ability to reduce pain and improve function with minimal treatments on patients. Prior to the DAT, I had very little experience with other treatment interventions beyond modalities (e.g., electrical stimulation, ice, heat) and therapeutic exercises. I was excited to continue researching and implementing "new" paradigms. The practical experience I obtained with the integration of these interventions shifted my expectation of patient care. I felt that I could expedite my patients' recovery process; yet, I did not have a grasp on the research or the theories associated with the paradigms I was implementing. Also, I was sabotaging a critical component of my clinical practice; I had been inconsistent with the collection of patient outcomes. My documentation system remained haphazard and inconsistent, which meant I had very little evidence to support my assumption that I was producing positive patient outcomes. While my records indicated I performed 188 treatments during the fall season, I collected very little outcome measures on the patients treated. Moreover, I was not classifying patients systematically, which led to poor clinical reasoning skills. I was

advancing my clinical skills with the implementation of new paradigms, but I was missing fundamental components that were essential to improving my patient care.

Spring I 2014

During the Spring I semester, course work required me to focus more on the development of *a priori* research designs, classification systems, and incorporating additional patient outcomes measures (e.g., patient specific functional scale, global rating scale). Additionally, a switch in my research topic from concussion to tendinopathy forced me to reconsider my clinical philosophy and challenged me to demonstrate a scholarly advancement that would eventually progress me to a clinician who understood the significance becoming both EBP and PBE practitioner. Personally, my goals for this semester were to be more diligent in the collection of patient outcome measures and increase my knowledge and applications with the paradigms I had learned and experimented with during Fall 1 semester.

For my *a priori* research design assignment, I was interested in determining if MWM could decrease the number of total treatments on LAS patients. Within 10 days of choosing my topic, I had three patients diagnosed with a LAS by a physician. Two of the patients suffered Grade 2 LAS and one patient was diagnosed with a Grade 3 LAS. In an effort to understand the effects of the MC MWM in my practice, I treated each patient with MC MWM and tape only. The Grade 2 LAS patients (#18030 and #18052) reported decreases in pain on the NRS that met MCID criteria after each session (Table 3.2 and 3.3). Evaluation of the DPA Scale scores revealed similar improvements, further quantifying the patient's positive outcomes during treatment (Table 3.4). Unfortunately, Patient #18057 developed complex regional pain syndrome (CRPS) and this patient did not display improvement on the NRS or DPA Scale. Because of the CRPS development, I stopped treating this patient with MWMs

and altered the care to meet the needs of the patient (Table 3.5). Despite improvements in my collection of patient outcomes and positive results in two of the three cases, I realized my initial attempt with an *a priori* design lacked many components of an effective design as the semester progressed.

Table 3.2 Spring I Patient #18030 Pre- and Post- NRS Scores - MWM Treatment of LAS

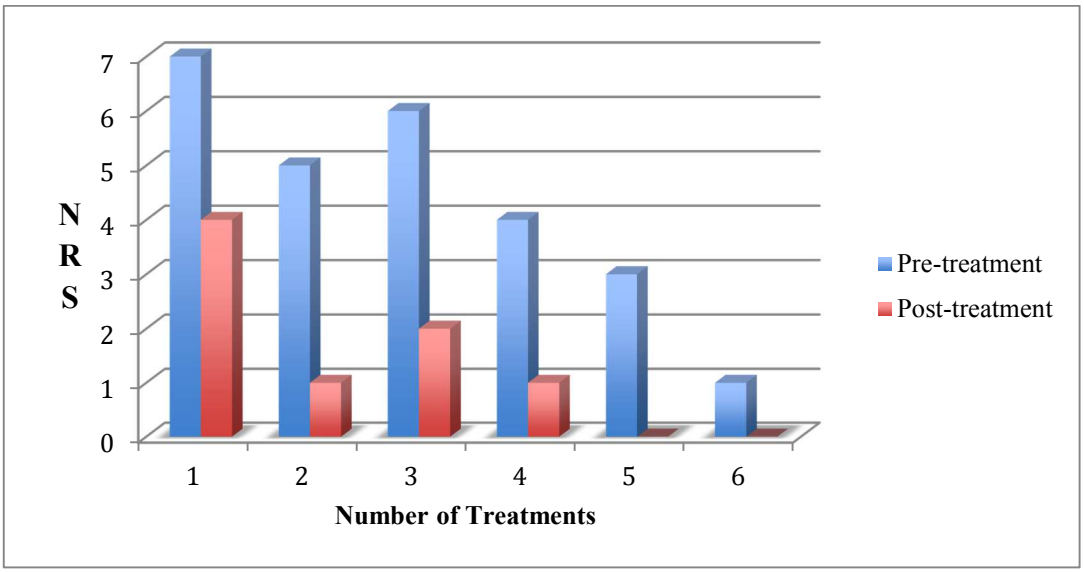


Table 3.3 Spring I Patient #18052 Pre- and Post- NRS Scores - MWM Treatment of LAS

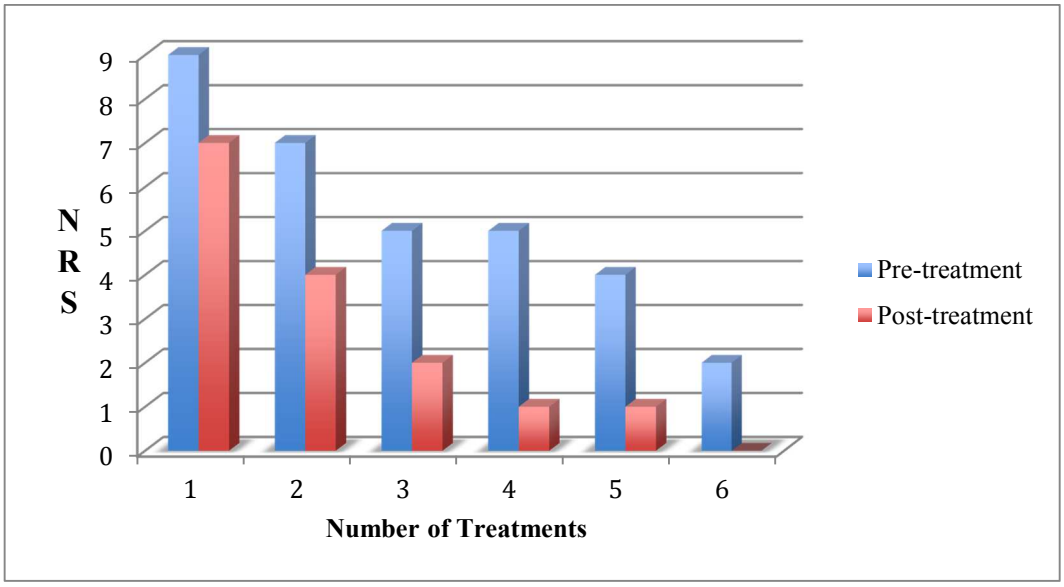
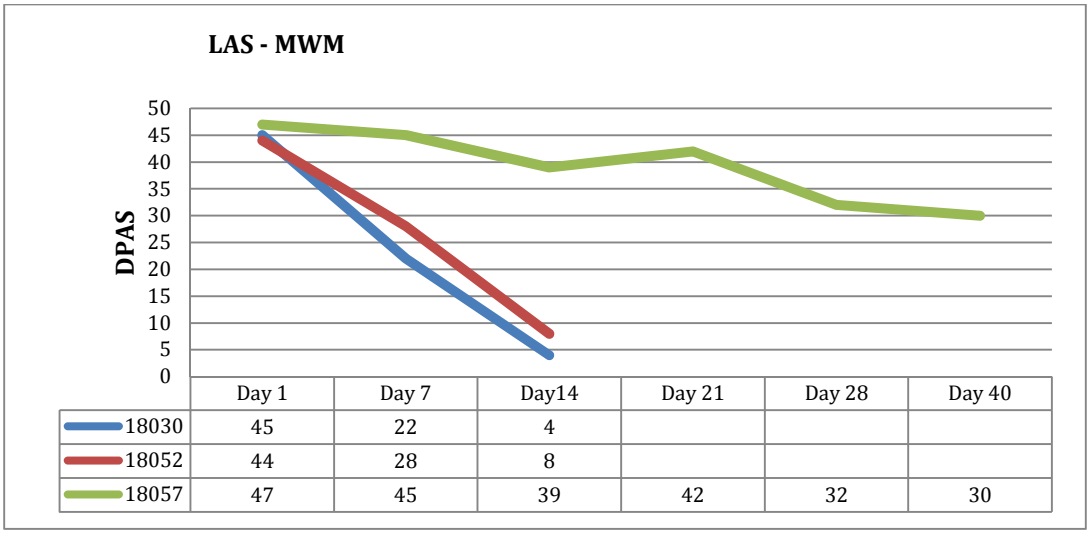
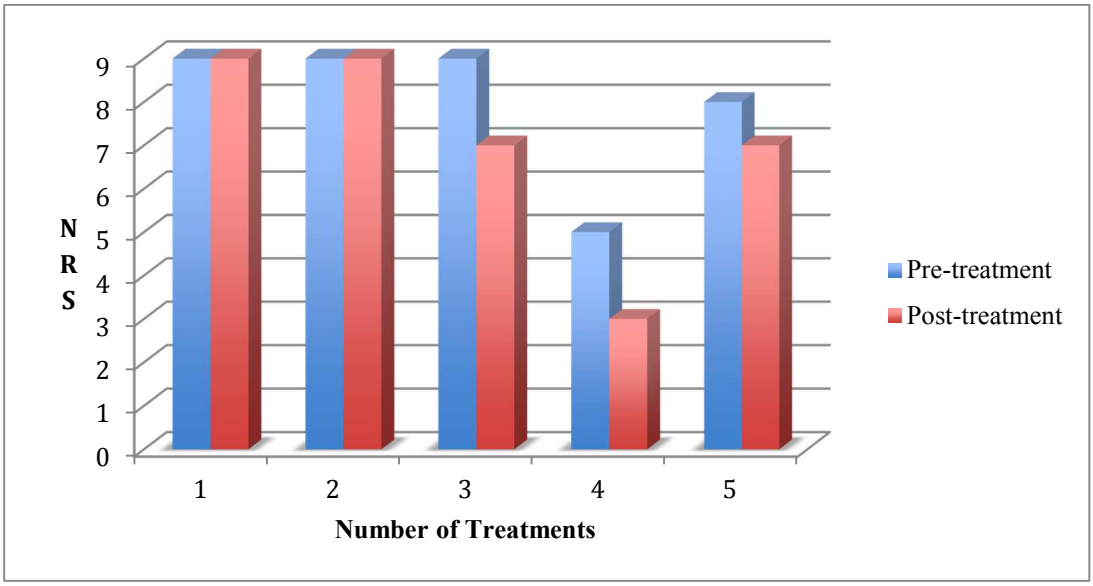


Table 3.4 Spring I Patients #18030, #18052, #18057 DPA Scale Scores - MWM Treatment of LAS



	Day 1	Day 7	Day14	Day 21	Day 28	Day 40
18030	45	22	4			
18052	44	28	8			
18057	47	45	39	42	32	30

Table 3.5 Spring I Patient #18057 Pre- and Post- NRS Scores - MWM Treatment of LAS



The practical experience I obtained focusing on only one treatment was invaluable. I learned that while I had a greater variety of interventions, I could not treat all my patients and expect instant improvements. I also realized that classifying patients was essential, but I was uncertain how to develop a classification system. Fortunately, the development of a

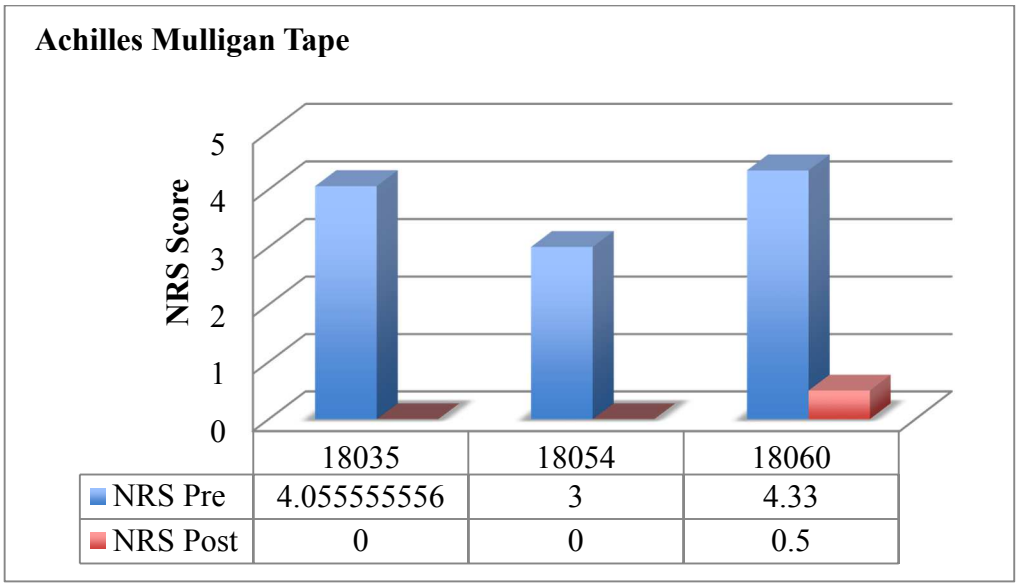
classification system occurred organically with the tendinopathy research. As I critically evaluated classification systems, treatment paradigms, and patient outcomes instruments, my clinical practice and decision-making evolved. I quickly understood the flaws in my LAS *a priori* design. A well thought out *a priori* design has many facets. One of the facets was to determine which outcome measures to administer for the research.

The NRS, DPA Scale, Global Rating of Change (GRC), Nirschl Phase Rating Scale (NPRS), the Patient-Rated Tennis Elbow Evaluation (PRTEE) questionnaire, the Victorian Institute of Sport Assessment (VISA) scales for the patella (VISA-P), and the Achilles (VISA-A) were outcome measures selected for the tendinopathy research. The GRC scale required the patient to assess their current health status, recall their status from the previous treatment, and then calculate the difference between the two time periods (Norman, Stratford, Regehr, 1997). While the validity of the NPRS, VISA, and the PRTEE had been established (Visentini, Khan, Cook, Kiss, Harcourt, Wark, 1998; Nirschl & Ashman, 2003; Rompe, Overend, & MacDermid, 2007), the scales were complicated and proved difficult to administer to the adolescent patient. Similarly, while the patient specific outcomes scale (PSFS) (Stratford, Gill, Westaway, & Binkley, 1995) was a useful questionnaire to quantify activity limitations and measure functional outcomes for patients, the adolescent patient was impatient when asked to choose 3 to 5 activities that were difficult to perform. We decided not to include the PSFS for the tendinopathy research. We believed the aforementioned measurements were sufficient without the PSFS. As we started to establish our methodology for the tendinopathy research that included a detailed evaluation, a classification system, an algorithm, and three indirect treatment protocols, the concept of applied research in the clinical setting became a reality.

The MC, PRT, and neurodynamics were the indirect treatments we chose for tendon pain patients. While I was familiar with MC and PRT, examining the research and discussing the nuances of each treatment with the group advanced my knowledge much faster than if I had been researching the interventions alone. With the incorporation of a more systematic evaluation process, my clinical reason and treatment outcomes would become more meaningful; I no longer haphazardly selected interventions. The only downside I could foresee with applied research in my clinical setting was the strict protocol. Adhering to the research methodology meant that patients who volunteered to participate in the study could theoretically receive four treatment options. Depending on the patient's classification, one treatment was applied for three consecutive sessions. If the treatment did not produce positive patient outcome measures, the patient was reevaluated and classified into another treatment option. The reevaluation process would continue until positive outcome measures were demonstrated, thus, prolonging the patient's return to activity.

By the end of the semester, the tendinopathy research methodology had been completed for piloting. I evaluated and treated three Achilles tendon patients #18035, #18054, and #18060 that met the tendinopathy inclusion criteria. I treated all three patients with the MC taping technique for Achilles tendon pain. Preliminary results demonstrated positive results on the NRS (Table 3.6); however, one-on-one assistance was required with the VISA-A, DPA Scale, and GRC, which meant the scores were not consistently documented; I had to discard the outcome measures. The number of outcome measures and the evaluation criteria required for the tendinopathy research concerned me. I feared the fast-paced middle/high school setting would preclude me from conducting research effectively.

Table 3.6 Spring I Patient #18035, #18054, #18060 Pre- and Post-NRS Scores Initial Treatment – Mulligan Taping of Achilles Tendon



	18035	18054	18060
NRS Pre	4.055555556	3	4.33
NRS Post	0	0	0.5

While I was satisfied with my scholarly advancement with the tendinopathy research, I was disappointed with my patient outcomes collection. I was becoming more confident with both my clinical skills and my patient evaluations. Plus, I had successfully employed a myriad of interventions on patients during spring semester. While I believed my patients' recovery time was less; I had minimal data to support this belief. I collected NRS score pre- and post-treatment every visit, but I continued to struggle with the timing of each of the outcome measures. Occasionally, I collected the DPA Scale every third visit; other times I collected on the patients first and last visit. With the help of an intern, I was able to collect the DPA Scale more consistently. While I believed the patients were experiencing a decrease in disablement, I was surprised when I did not observe a consistent downward trend on the DPA Scale scores. Eventually, I discovered the arbitrary outcomes were attributed to my lack of communication with my intern. I had been subtracting the 16 points from the raw score for every patient. The intern was not subtracting the 16 points. I did not realize the error until I was writing up the

outcomes paper for Spring I. The inconsistent results were due to the fact that I had not thoroughly explained the scoring system to the intern.

At this point, I was ready for summer session. After completing one year in the program, I was concerned over the difficulties I was still experiencing with the collection of patient outcome measures and injury classification. I was pleased with my clinical skills advancement and I was determined to learn from my mistakes.

Fall II 2014

The Summer II semester provided the inspiration and motivation I was seeking. The DAT Program had provided a plethora of opportunities for student advancement. From survey design, to statistical analysis, to scholarly research, to continuing education, the summer overwhelmed and invigorated me. Having the opportunity to learn, teach, discuss, write, and conduct research with my classmates was the applied experience I needed to build my confidence with applied research, practical applications, and theoretical models. Collecting patient outcomes had been one of my biggest challenges. Fortunately, the tendinopathy research provided an effective template for the collection of patient outcomes. The summer semester also allowed my cohort to share a variety of patient outcome collection systems that had accelerated the input of patient's outcome measures and provided an easy conversion for analysis. While the majority of my focus for Fall II was on tendinopathy research, I continued to investigate paradigms introduced during Summer II session (e.g., Myokinesthetics, MC). My personal goals for Fall II were to continue refining my clinical skills, develop and implement an *a priori* research design, defend my research topic, and advance my knowledge in statistics.

During Summer II session, I completed both the upper and lower extremity courses for MYK; I chose this system for the evaluation and treatment of medial tibial stress syndrome (MTSS) as an *a priori* clinical research project for the fall semester. As I identified inclusion and exclusion data, outcome measures, and research questions, I realized I had a much better understanding of the necessary elements an *a priori* design than Spring I.

With cross-country season in session, I quickly found four MTSS patients to test my MYK hypothesis. When the MTSS patient's outcomes scores met MCIDs in the first 2-3 weeks, I was convinced that MYK treatment would prove to be more effective than previous treatments I had used for MTSS. By Week 2, Patients #18400, #18412, and #18415 function scales (PSFS) had met MCIDs (Table 3.7). Similarly, patients' pain scales (NRS) continued to decrease after each treatment (M = 2.4). Interestingly, the patient's DPA Scale scores were not following the same pattern as the other outcome measures (Table 3.8). In fact, not one MTSS patient had met an MCID on the DPA Scale by week two. By weeks three and four, the patients' outcome measures revealed a decrease in function scores (PSFS) and an increase in disablement (Table 3.7 and 3.8). Originally, I did not begin MYK treatments until a physician had ruled out a stress fracture for each patient. While radiographs came up negative, I was apprehensive to allow the patients to return to sport. Based on my evaluation, I believed the patients had a either a stress fracture or a stress reaction. I started MYK treatment because theoretically if the patients suffered from MTSS, MYK could alleviate the pain/symptoms. I advised the cross-country coach to allow the patients to ease back into running. Unfortunately, the patients' physician's indicated a gradual return to running was permissible. The coach ignored my advice and did not change the patients running regimen. When the patient's outcome measures revealed that pain, function, and disability were worse, I was not surprised.

I referred the patients back to the physicians for reevaluation. MRI scans revealed all four patients had suffered a stress fracture of the medial one-third of the tibia.

Table 3.7 Fall II Patients #18400, #18412, #18415, #18420
PSFS Scores - MYK Treatment of MTSS

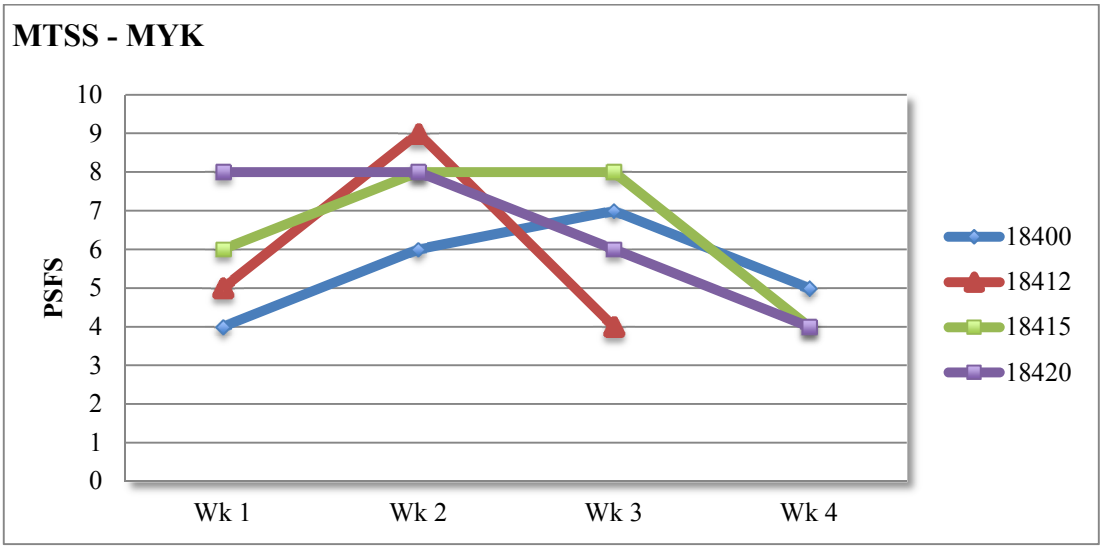
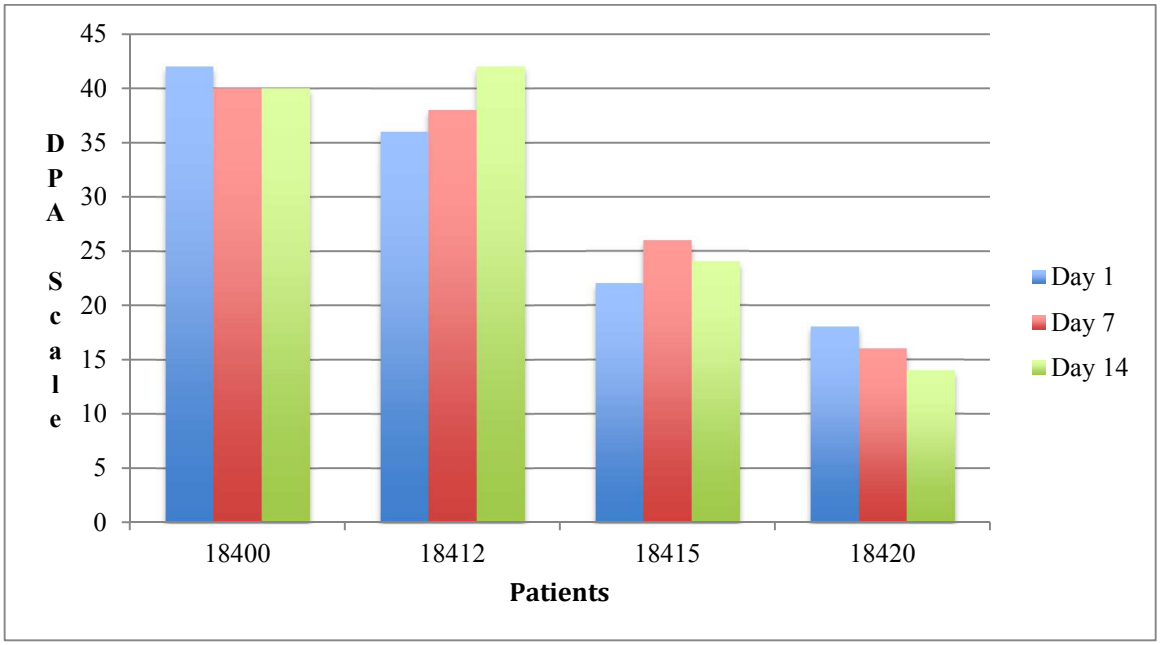


Table 3.8 Fall II Patients #18400, #18412, #18415, #18420
DPA Scale Scores - MYK Treatment of MTSS



As I reflected on the patient's outcome measures, I contemplated the patient's positive outcome measures in the first three weeks of MYK treatment. I could not ignore the fact that I had spent a disproportionate amount of time (for the high school setting) performing MYK on these patients. Perhaps, the personal attention coupled with the massage-like technique of the MYK treatment contributed to the positive patient outcomes during weeks one through three (Adams, White, & Beckett 2010).

While MYK was a time consuming technique, I was becoming more proficient in postural evaluations and better understood the theory affiliated with the treatment. While I was disappointed in my *a priori* research outcomes, I was pleasantly surprised when I chose MYK as a treatment on a chronic low back pain (LBP) patient (#18404). The patient had been suffering with LBP and intermittent radiculopathy for four years. The patient's two magnetic resonance imaging (MRI) tests, approximately one year apart, were negative. She had been enrolled in six-weeks of formal physical therapy for the last two summers with poor results. I had tried modalities, kineseotaping, muscle energy, reactive neuromuscular training, neurodynamics, MC, PRT, and mindfulness training on this patient with minimal improvement. The patient was a soccer player and the type of athlete that rarely complained.

Two weeks into the soccer season, I noticed her running mechanics had completely changed. The patient was much slower and lacked her usual quickness from previous years. After her first game, she stopped by the clinic to wrap her knees and low back in ice. I inquired about her LBP. She admitted that when she played her pain level was 8 of 10 (NRS). We discussed the theory behind MYK. She agreed to try three consecutive treatments.

After a postural assessment, I chose the MYK L5 treatment. The patient's pain score was 6 of 10 at best. Her DPA Scale was 12 of 64, which was low and well within the healthy

normative data. After the first MYK treatment, the patient's NRS scored decreased to 2 of 10. She had met the MCID for pain (NRS) after one treatment. When I saw the patient the next day, her NRS score increased to 4 of 10; however, her GRC score was +5 (quite a bit better). I performed another MYK treatment, which she reported resolved her current pain (0 of 10 on the NRS). After the treatment, she played in a game and her worst pain score returned to 3 of 10 (NRS). I performed a total of five MYK treatments on this patient over 10 days. At discharge, I documented her outcome measures: GRC +7 (a very great deal better), NRS 0 of 10 (playing soccer), DPA Scale 4 of 64. I was pleased with the results of MYK on a chronic LBP patient's pain, disability, and function. The patient had met MCIDs for the NRS and DPA Scale from initial evaluation to discharge. Prior to the DAT, I had not been exposed to MYK. Learning MYK and adding the postural exam as part of the patient evaluation simplified the classification and treatment plan for a variety of injuries.

By mid-Fall of my second year, I was focused on mastering the additions to the evaluation process, utilizing indirect treatments, and perfecting outcome measures collection for the tendinopathy research and the MTSS patients. Subsequently, data collection for my other patients suffered. I could only locate minimal data on non-research patients from the computerized on-line system I utilized: injury, number of treatments, intervention, and treatment results (Table 3.9). I was concerned that my technological ineptitudes would be perceived as a lack of scholarly development with regards to my patient outcomes and dissemination of research. Consequently, to demonstrate that I truly understood how to conduct research and analyze patient outcomes data worthy of scholarly dissemination, I collaborated with a colleague to write an abstract for a poster presentation submission to the 2015 World Congress World Federation of Athletic Training and Therapy (WFATT). While

the Federation declined our submission, the findings were significant and indicated that the treatment-based classification algorithm for tendinopathy patients expedited recovery and produced positive outcomes. The following excerpt was from the patella tendinopathy abstract submitted on January 20, 2015.

> ***Findings****: The participants included 8 females and 5 males with a mean age of 18.76 ± 2.97 years. Paired t-tests were performed for each of the outcome scales to assess the changes from initial exam to discharge. Effect sizes and minimum clinically important differences (MCIDs) were assessed for each scale to determine clinical meaningfulness of the changes. The mean change of 4.14 ± 1.91 on the NRS was significant (p = .000). A Cohen's d value of 2.84 indicated a large effect size and 100% of the patients experienced an MCID on the NRS. The mean change of 14.54 ± 8.32 on the DPA Scale was significant (p = .000). A Cohen's d value of 1.38 indicated a large effect size and 92% of patients (n = 12) met the persistent pain MCID for the DPA Scale. The mean change of -3.46 ± 2.07 on the GRC was also significant (p = .000). A Cohen's d value of 1.76 indicated a large effect size and 100% of the patients experienced an MCID on the GRC. The mean change of -14.00 ± 22.64 on the VISA- P was not statistically significant (p = .082); however, a Cohen's d value of 0.721 indicated a moderate effect size and 60% of the patients experienced an MCID on the VISA-P. At discharge, patients had completed 4.15 ± 2.6 treatment sessions and 85% (n =11) of the patients reported resolution of tendon pain, while 100% scored in the healthy range on the DPA Scale.* ***Conclusions****: Preliminary data collection demonstrated the use of an indirect treatment algorithm that matched patients to specific interventions based on exam findings could produce statistically and clinically significant changes across a variety of outcomes measures for patellar tendinopathy patients. These results were experienced with fewer treatments, within a shorter time period, than is reported in the literature when using pharmaceutical or direct (i.e., eccentric exercise) interventions for treating tendinopathy. The findings from this study support the idea that appropriate patient classification during the evaluation process is a critical component to consider for identifying the best treatment option for patients suffering from patellar tendon pain.*

The preliminarily results for patellar tendinopathy patients using indirect treatments was significant and achieved in fewer treatment sessions than the more direct technique (eccentric exercise) advocated as the gold standard (Alfredson, Pietilä, Jonsson, & Lorentzon, 1998; Cannell, Taunton, Clement, Smith, & Khan, 2001; Fahlström, Jonsson, Lorentzon, & Alfredson, 2003; Öhberg, Lorentzon, Alfredson, 2004; Purdam, Jonsson, Alfredson, Lorentzon, Cook, & Khan, 2004; Jonsson, Alfredson, Sunding, Fahlström, & Cook, 2008;

Dimitrios, Pantelis, & Kalliopi, 2012). Analyzing the pilot data and validating the patella tendinopathy research was statistically significant and clinically meaningful was encouraging. As I analyzed and re-analyzed the statistics for the abstract results section, I integrated the statistical concepts we had learned in our statistics courses. I realized that not only was I capable of conducting research, but I could successfully analyze data, interpret the results, and disseminate the findings. Moreover, compiling data for the abstract inspired me to co-write a manuscript for the preliminary patella tendinopathy abstract. The task provided further evidence of my scholarly advancement.

Fall II was filled with learning moments. While my documentation for the general population outcome measures appeared to be lacking, I was actually quite pleased with my scholarly advancement. The documentation system developed and implemented for the tendinopathy research proved to be effective for a group of clinicians collecting data at multiple sites. Moreover, I was making the appropriate strides in my research efforts and toward my goal of becoming a PBE clinician. I had a much better understanding of each outcome measure and appropriate administration. Upon reflection of outcome measures, I was encouraged by the vast development of my clinical practice from Fall I to Fall II. Conducting research facilitated growth in several areas of my clinical practice. For example, the inclusion of a thorough evaluation improved my consistency with disease-oriented outcomes, such as active range of motion and special orthopedic tests. Similarly, less familiar methods, such as Mulligan Concept and neurodynamic testing, improved and were implemented into all patient evaluations.

Perhaps the most notable change occurred with the integration of a treatment-based classification (TBC) algorithm for tendinopathy patients. Implementing the TBC transferred

over to a more systematic approach with all my patient examinations. The algorithm required me to methodically evaluate and classify patients, which greatly reduced my previous "shot gun" approach. I was confident with my clinical decision-making. I understood the theories associated with the treatments and could explain to my patients that positive effects might not occur during the treatment session; sometimes, a decrease in pain, disability, or an increase in function occurred post-treatment (24 to 72 hours). As I reflected back on these conversations, realistically, I could not rule out the fact that the increase of time, energy, and attention I spent treating these patients could have had a biopsychosocial effect that also contributed to the positive patient outcomes (Kabat-Zinn, 2012).

Spring II 2015

During the Spring II semester, my primary focus was identifying patients to include in the tendinopathy research and collect outcome measures. With a reliable TBC algorithm in place, a better understanding of outcome measures, an improved confidence with indirect manual treatment techniques, and conducting research, I was progressing as a PBE clinician. I discovered an increased competency and confidence in my treatment selections, clinical skill set, and my clinical decision-making processes with all my patients.

For example, producing positive patient outcomes with confirmed meniscal patients seemed impossible two years ago. Coincidentally, my patient care provided me the opportunity to challenge my clinical advancement with the treatment of four meniscal injuries. While patients #18425, #18428, #18431, and #18436 did not have identical injuries, each patient was diagnosed with either a medial or lateral meniscus lesion (Table 3.10). All patients were given the same prognosis: one month NWB with a straight leg brace, formal physical therapy twice a week for six weeks. The recommendation from each physician was

six to twelve weeks of rehabilitation before a return to activity would be considered. Potential treatments for meniscal lesions are the MC "squeeze" and tibial rotation for knee pain techniques. After spending a semester treating tendinopathy patients with MC tibial rotation technique, I was confident in its application, but did not have the same confidence in the "squeeze" technique. I evaluated patient #18436 and discussed my clinical findings and treatment in a WordPress blog published on March 1, 2015:

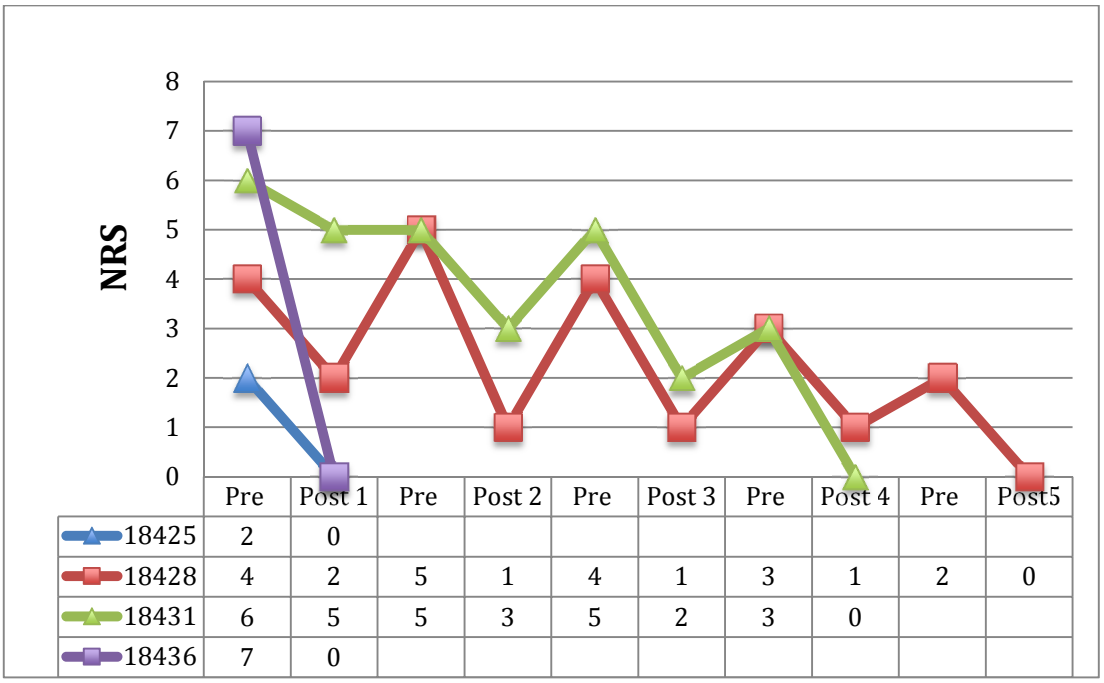
> *As I read Kari's blog and comments last week on special tests, I thought about a patient I had recently treated with a meniscal tear. After performing special tests, the orthopedic diagnosed the patient with a posterior lateral meniscus tear. As always, I was curious if I would find the special tests positive too. (Pre-DAT this was very important to my ego. I guess I needed the "validation" so I could prove to myself that my special test skill set aligned with an MD's.) As Kari's comments implied, Pre-DAT many of us had confirmed pathanatomic injuries with "special" tests. The following case demonstrates a typical outcome post DAT.*
>
> *(Scott, think kinesthetically unaware.:0) Hx; Patient stepped on his left leg getting out of bed. He planted his foot and twisted his knee putting valgus stress on his the joint. Due to "excruciating" pain, the parent's drove the patient to the ED where the ED gave him morphine to reduce the pain, put him in a SL brace and crutched him. I attempted to evaluate the patient the next morning. He was unwilling to bend his knee much less perform "special" tests. Later that day, he was seen by an orthopedic. He was diagnosed with a posterior lateral meniscal tear; he was told to remain in a SL brace for 1 month then begin formal PT. The ortho's prognosis was a full return to sport in 3 mos.*
>
> *Since the patient could not get into formal PT for a week, Day 3 post-injury, the parents and the patient came to the clinic so that I could take some ROM measurements and discuss a rehab program. The patient had minimal swelling on the lateral aspect of joint line. He had pain (NRS 7/10) with palpation on lateral posterior joint line. His AROM was 45 degrees of knee flexion and -10 degrees of extension. He felt that his knee "would not go any further." The standard meniscal special tests were positive. Even though the patient was in a lot of discomfort (NRS Best/Worst/Rest 4/7/4; DPAS 46), the parents and I discussed the Mulligan tibial rotation technique. I asked permission to try a Mulligan MWM. I explained the technique and confirmed that I had some success treating meniscal injuries. The PILL effect occurred with tibial lateral rotation. After 1×10 reps, I asked him to lie on the plinth and perform AROM (flexion/extension). AROM had increased significantly for both flexion/extension (90/-5 degrees). Moreover, when he walked over to the plinth, his pain had decreased (NRS 5/10). I performed two more sets of 10 reps. As I performed the 3rd set of MWMs, his joint shifted ever so slightly; it was accompanied by an audible pop/crunch. The patient wanted to know why his knee popped. (Admittedly, I was paranoid to take my hands off his tibia). I pulled my hands off the tibia and*

encouraged him to perform knee flexion. He confirmed he had no pain. As he walked back over to the plinth to perform AROM exercises, he claimed that his knee pain was gone (NRS 0/10). I retested his AROM. It was equal to the contralateral side. He walked out of my office without the knee brace or crutches.

Day 7, the patient returned to the ortho pain free. He attempted to explain to the MD what had happened during our treatment; to which the MD replied, "It was impossible." The MD sent a note back with the patient. The note said, "Meniscal tear. No activity for 3 mos," Unfortunately, this note meant I was rehabbing a fairly functional adolescent for 3 months. At three weeks, the patient was fully functional. (OK… I use the term "functional" loosely. :0) The PT had seen him once and agreed that he was ready for return to sport. I spoke with the patent's Mom and explained my rationale for returning him to sport. It took a few days to convince the parents that he could safely return to sport. The parent's cancelled follow-up appointments with the MD. The patient has been playing pain free for the past 3 weeks.

Interestingly, I had a similar experience with patient #18425. Although I did not feel the audible pop when I performed tibial rotation, the patient did experience a complete resolution of pain after one treatment. While not all my meniscal patients responded as quickly as patient #18425 or #18436 (Table 3.11), the overall outcomes were positive for the group. I ran paired *t*-tests on the NRS, DPA Scale, and PSFS scores for the meniscal patients to determine the effectiveness of MC tibial rotation mobilization treatment on all four patients from initial exam to discharge. Mean differences from the initial visit to discharge scores and 95% confidence intervals (CIs) were calculated for the NRS, DPA Scale, and PSFS. Cohen's *d* was also computed to determine the effect size, or maximum likelihood of outcome measures. The mean change on the NRS (M =4.75, 95% CI[1.22 to 8.27], $p = .023$) demonstrated a statistically significant change for pain, while a Cohen's *d* value signified a large effect size (.93). The only patient who did not meet a MCID on the NRS after the first treatment was patient #18431. All of the patients were discharged reporting a resolution of pain in five treatments or less (Table 3.11).

Table 3.11 Spring II Patients # 18425, #18428, #18431, #18436 Pre- and Post- Treatment NRS Scores - MC Treatment of Meniscal Injuries



	Pre	Post 1	Pre	Post 2	Pre	Post 3	Pre	Post 4	Pre	Post5
18425	2	0								
18428	4	2	5	1	4	1	3	1	2	0
18431	6	5	5	3	5	2	3	0		
18436	7	0								

Even though the outcome measures were statistically significant, all meniscal lesion patients' met MCIDs for all other outcome measures from initial exam to discharge. The mean change on the DPA Scale (M =16.75, 95% CI[1.75 to 31.75], p = .038) demonstrated a statistically significant change in disablement, while a Cohen's d of 4.10 signified a large effect size (.90). The mean change on the PSFS (M = 3.25, 95% CI[5.96 to .53], p = .032) demonstrated a statistically significant for function, while a Cohen's d of 4.39 signified a large effect size (.91). The mean change on the GRC (M = 6.2, 95% CI[9.44 to 2.56], p = .012) demonstrated a statistically significant change for pain, while a Cohen's d of 6.41 indicated a large effect size (.95).

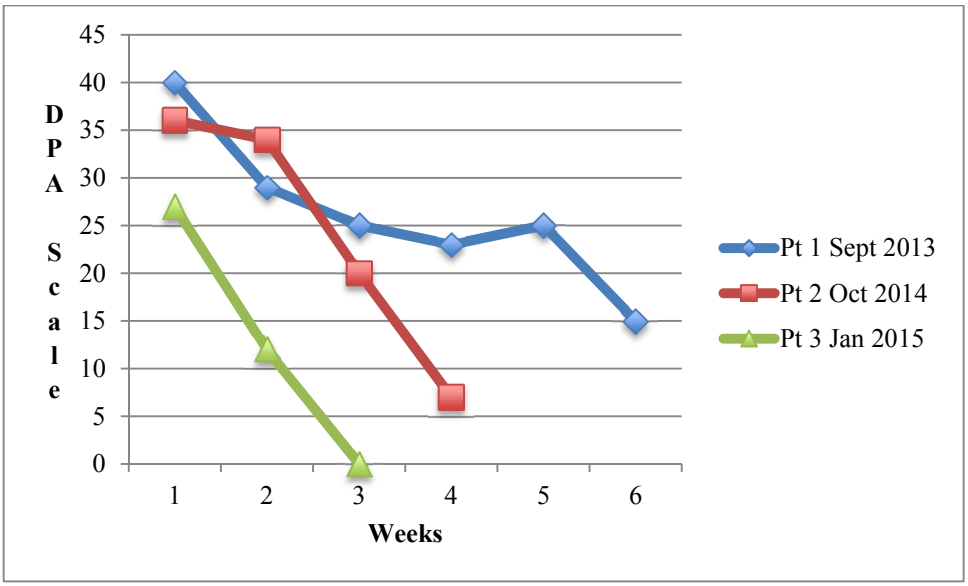
Adding the MC as part of my knee evaluation led me to a more concise classification and treatment plan. I used a matched MC treatment with each of these patients. The statistical analysis demonstrated that the use of the MC tibial rotation mobilization produced significant

improvements in outcome measures from initial evaluation to discharge demonstrating a positive change on both the patient's pain, disability, and function in less than five treatments over 9 ±8 days. I was pleased with my results with my second *a priori* design. My methodology and data collection was much more precise then my first attempt. As I reflected on my clinical development from Fall I to Spring II, I wondered if I would discover a trend indicating that patient's timeline for return to sport had decreased for other injuries.

I examined the records of three lateral ankle sprain (LAS) patients diagnosed with similar Grade 2 LAS injuries during fall 2013, spring 2014, and spring 2015. Each patient had received a similar MD prognosis of an eight to twelve week recovery period. Like the meniscal patients, prior to the DAT summer immersion in 2013, I would have accepted the physician's prognosis. I would have abided by the prescribed rehabilitation and never considered performing a simple mobilization. As I examined my LAS patient outcome measures, I recognized the significance of having quantifiable data to support my clinical reasoning and to determine the value of my interventions.

I compared the three Grade 2 LAS patients. All three patients were discharged well before the eight to twelve week prognosis (Table 3.12). More importantly, each of their discharge time-lines demonstrated a meaningful decrease in return to function. While individual patient differences (e.g., injury severity) is likely partially responsible for the different results between patients, I could not ignore my evolution as a clinician also being a major influence on the more positive patient outcomes I was able to produce over my time in the program.

Table 3.12 Spring II Patients Outcome Comparison Fall 2013 to Spring 2015
DPA Scale Scores – MWM Treatment of LAS



Finally, I collected data on six patients who met the lateral elbow and Achilles tendinopathy inclusion criteria. Based on the indirect TBC algorithm four patients were classified into the MC MWM treatment for lateral elbow (LE) epicondylagia. The most common complaint for the LE patients (N = 4) (31 ±16 years of age) was a decrease in grip strength. The overall outcomes were positive for all four patients. Paired *t*-tests were analyzed on the NRS, DPA Scale, and PRTEE for the LE patients to determine the effectiveness of the MC MWM treatment on LE patients from initial exam to discharge. Mean differences from the initial visit to discharge scores and 95% CIs were calculated for the NRS, DPA Scale, and PSFS. Cohen's *d* was computed to determine the effect size, or maximum likelihood of outcome measures. The mean change on the NRS (M = 7.75 ±.95, 95% CI[6.23 to 9.27], $p = .001$) demonstrated a statistically significance change for pain, while a Cohen's *d* of 18.69 signified a large effect size (.99). The mean change on the DPA Scale (M = 21.50 ±6.95, 95% CI[10.44 to 32.56], $p = .009$), while a Cohen's *d* of 7.14 indicated a large effect size (.96).

The mean change on the PRTEE (M = 77 ±11.19, 95% CI[39.19 to 74.81], p = .002), while a Cohen's d of 11.76 signified a large effect size (.98).

I treated each LE patient with a matched MWM treatment. The statistical analysis demonstrated that the use of lateral elbow MWM produced significant improvements in all outcome measures from initial evaluation to discharge demonstrating a positive change for the patient's pain, disability, and function in 4.25 ±2.06 treatments over 10.50 ±7.55 days. This time line was much less than the original prognosis of six to eight weeks.

I collected data on two Achilles patients (18 and 16 years of age respectively). Based on the indirect TBC algorithm both patients were classified into the MC treatment for Achilles tendinopathy. The overall outcomes measures did not demonstrate a positive change for either patient. I ran paired t-tests on the NRS, DPA Scale, and VISA-A scores for the Achilles patients to determine the effectiveness of MC taping technique on both patients from initial exam to discharge (3 treatments). Mean differences from the initial visit to their reevaluation scores and 95% CIs were calculated for the NRS, DPA Scale, and VISA-A. Cohen's d was also computed to determine the effect size, or maximum likelihood of outcome measures. The patients had not demonstrated a statistically significant change for pain (NRS), disablement (DPA Scale), or function (VISA-A) with the MC taping technique. The mean change on the NRS was (M =1.00 ±1.14, 95% CI[-11.70 to 13.70], p = 0.5) for pain with a medium effect size (.70). The mean change on the DPA Scale was (M = .50 ± .70, 95% CI[6.85 to 5.8], p = 0.5) for disablement with a medium effect size (.70). The mean change on the VISA-A was (M =3.5 ±4.94, 95% CI [40.97 to 47.97], p = 0.5) for function with a medium effect size (.70). Neither patient met discharge criteria; both patients had to be revaluated.

Based on the indirect TBC algorithm, both patients were reclassified into the PRT treatment. The PRT treatment established positive outcome measures for both patients. However, the second treatment results only demonstrated statistically significant for the NRS. I ran the same statistical analysis as the MC taping technique to determine the effectiveness of PRT from reevaluation exam to discharge. The mean change on the NRS (M = 6.5 ±.707, 95% CI[.146 to 12.85], $p = 0.04$) demonstrated a statistically significant change for pain, while a Cohen's *d* signified a large effect size (.98). While the mean change on the DPA Scale (M =24 ±11.31, 95% CI [77.64 to 125.64], $p = 0.205$) met an MCID the analysis was not statistically significant for disablement; yet, a Cohen's *d* indicated a large effect size (.94). The mean score on the VISA-A (M = 27 ±2.82, 95% CI[52.41 to 1.58], $p = 0.047$), demonstrated a statistically significant for function, while a Cohen's *d* signified a large effect size (.95).

While not all tendinopathy patients responded immediately to the first treatment selection, the overall outcomes were positive for both patients from initial exam to discharge, suggesting the TBC algorithm was effective. The mean change on the NRS (M = 6.5 ±.70, (95% CI[.146 to 12.85], $p = 0.04$) demonstrated a statistically significant change for pain, while a Cohen's *d* signified a large effect size (.98). While the mean change on the DPA Scale (M =2.35 ±10.60, 95% CI[71.79 to 118.79], $p = 0.179$) met an MCID the analysis was not statistically significant for disablement; yet, a Cohen's *d* indicated a large effect size (.94). The mean score on the VISA-A (M = 27.5 ±3.53, 95% CI[59.26 to 4.26], $p = 0.05$), demonstrated a statistically significant for function, while a Cohen's *d* signified a large effect size (.95). However, the use of these techniques might be more effective if used in

combination rather than in isolation; especially, if the participant's evaluation presents with multiple factors (e.g., participant responds positively to MWM and has TP's) (Chapter 5).

I treated both Achilles patients first with a matched MC taping techniques as my first treatment. After three treatments, the patients (N = 2) were reevaluated and assigned into the PRT treatment. The lower quarter screen signified the more dominant TP's were on the gastrocnemius, soleus, and the medical aspect of the calcaneus. The statistical analysis demonstrated that the use of PRT produced significant improvements in all outcome measures from initial evaluation to discharge demonstrating a positive change on both patient's pain, disability, and function in less than six treatments over 10 days.

Historically, the gold standard for the treatment of tendon pain has been eccentric exercise (Alfredson et al., 1998; Cannell et al., 2001; Mafi, Lorentzen, & Alfredson, 2001; Fahlström et al., 2003; Öhberg et al., 2004; Purdam, et al., 2004; Cook, & Kahn, 2004; Jonsson et al., 2008; Dimitrios et al., 2012). The use of indirect treatments (e.g. MC, PRT, neurodynamics) could be an effective treatment for the reduction of pain and disability, while expediting a patient's return to function (Baker et al., 2013; Takasaki, Hall, & Jull, 2013; Bisset, Beller, Jull, Brooks, Darnell, & Vicenzino, B. 2006; Speicher & Draper, 2006; Shacklock, 2005; Vicenzino, Paungmali, Buratowski, & Wright, 2001; D'Ambrogio & Roth, 1997; Jones, 1981). A more extensive patient evaluation that classifies patients with a TBC algorithm in conjunction with the collection of patient outcome measures resulted in statistically significant and clinically meaningful evidence with the tendinopathy patients. The research process has enhanced my foundational knowledge and provided a depth of understanding I could not have predicted in my quest to becoming a PBE clinician.

Final Reflection and Impact of Residency

Initially, I was concerned with the curricular expectations with the clinical residency component of the DAT Program; however, I was pleased with the end results. The DAT program challenged me to overcome fears, weaknesses, and barriers. I had become more competent and confident as a clinician. I discovered that researching paradigms, theoretical models, and becoming more proficient with my clinical skill set rewarding and in-line with the goals I sought entering the DAT Program. I was not prepared, however, for the impact the DAT Program would have on my scholarly development.

Prior to the DAT, I had spent decades treating the patient's injured site. I did not assess patients globally. Researching and learning the movement analysis and postural assessments enhanced my clinical reasoning. As I researched the tendinopathy theories related to the neural, vascular, and mechanical system, my foundational knowledge advanced. Through the tendionopathy research, a novel TBC algorithm was created. I started to utilize a TBC system to classify patients and treat according to the source of pain, not merely the painful site or local pathology As I started to understand the intricacies of the somatic system, I appreciated that not all injuries were purely physical; frequently, injuries had a biopsychosocial or energetic element as well.

As I started to research and focus more on the biopsychosocial and energetic aspects in my patient care, I understood the value of adopting a more patient-centered philosophy. Researching and integrating a more holistic approach into my clinical evaluations had a profound effect on my patient population and also on me as a clinician. As I focused more on the biopsychosocial effects (e.g., mindfulness), I discovered I could treat the patient with a greater sense of ease and empathy. Patients were more open to discussing and identifying

potential triggers/stressors that might be contributing to the cyclic nature of acute or chronic injuries. While biopsychosocial research in its infancy, I am fascinated with the topic and look forward to contributing to this body of knowledge (Syvertson, Baker, and Nasypany, forthcoming).

When the EBP category was included as a continuing education requirement for ATs, I was familiar with the term; but I had no knowledge of how much it would impact my clinical practice. Recently, I attended the Far West Athletic Trainers' Association conference and made it a point to attend all of the EBP presentations. As I listened to the presenters, I began to recognize the depth of my knowledge as an EBP clinician. I had pertinent and timely research to share with other AT professionals. I could have easily presented an applicable EBP presentation to the ATs at the conference. The DAT Program had successfully transformed my clinical philosophy to that of an EBP clinician. I realized the EBP knowledge I had been introduced to and researched during the DAT Program was more advanced then the information presented in this forum. I had the knowledge and competence too not only present a user-friendly format of EBP to ATs, I had discovered tangible approaches that had changed my clinical practice to PBE.

After six semesters of didactic coursework, I have evolved as an EBP clinician and am confident with my role as a PBE clinician. As a PBE clinician, conducting research has transformed my clinical practice. I no longer haphazardly evaluate or treat patients. I have implemented a systematic approach that includes a classification system, global assessments, and outcome measures. While it took time and patience to create an effective documentation system, I recognized the value of incorporating multiple outcome measures. Moreover, quantifying patient's improvements proved to be extremely beneficial with my patient

population. Adolescent patients (my main patient population) are very motivated by numbers. In the event a patient was disillusioned with his or her progress, the outcome measures provided the objective data necessary to help the patient identify positive improvements or lack of improvements.

Before entering the DAT, I had minimal confidence in my ability to contribute to the AT profession in a scholarly fashion. At the time, I accepted that my contribution would be inputting patient outcomes for another researcher who understood the research process. I now recognize that not only am I capable of developing and conducting meaningful applied research, my patient population has benefitted from my clinical advancements. Finally, I am excited to have a cohort of advanced scholarly clinicians that are interested in collaborating together beyond the DAT in our pursuit to disseminate research directly applicable to exemplary patient care. While in the DAT Program, my personal and professional growth knew no bounds. In retrospect, I have far surpassed my original goals of learning new paradigms and improving my clinical skill set. The DAT Program successfully challenged me to discard my traditional philosophical AT mind-set and truly function as an advanced scholarly practitioner.

Chart 3.1 Fall II Patient Outcome Measures

Pt ID	Type	# of Tx	Location	Intervention(s)	Tx Result
FALL II					
18400	Acute	3	Tibia	MYK	Negative
18401	Chronic	7	Hamstring	PRT, MC, TMR, AAT, PRRT	Positive
18402	Acute	4	Shoulder	MC, TMR	Positive
18403	Acute	5	Ankle	MC	Positive
18404	Chronic	5	Lumbar spine	MYK	Positive
18405	Acute	1	Meniscus	MC	Positive
18406	Acute	3	Shoulder	PRT	Positive
18407	Chronic	4	Tibia	MYK	Negative
18408	Acute	2	Ankle	MC	Positive
18409	Acute	1	Knee	MC	Positive
18410	Acute	3	Ankle	MC	Positive
18411	Acute	6	Ankle	MC	Positive
18412	Acute	3	Tibia	MYK	Negative
18413	Acute	1	Sacroiliac	ME, RNT	Positive
18414	Acute	3	Thumb	MC	Positive
18415	Chronic	6	Tibia	MYK	Negative
18416	Acute	3	Achilles	MC	Positive
18417	Acute	4	Ankle	MC	Positive
18418	Acute	3	Hamstring	PRT, TMR	Positive
18419	Acute	4	Hip Flexor	PRT, MC	Positive
18420	Acute	4	Tibia	MYK	Negative
18421	Acute	3	Knee	MC	Positive
18422	Acute	2	Wrist	MC	Negative

Key: Pt ID – patient identification; # of tx – number of treatments; MYK – Myokinesthetics; PRT – Positional Release Therapy; Total Motion Release – TMR; AAT – Associative Awareness Technique; PRRT – Primal Reflex Release Therapy; MC – Mulligan Concept; ME – Muscle Energy; RNT – Reactive Neuromuscular Training

Chart 3.2 Spring II Patient Outcome Measures

Pt. ID	Tx #	Location	NRS pre	NRS post	GRC	DPA pre	DPAS post	Intervention	Outcome
SPRING II									
18423	6	Head	4	0	x	x	x	PRT, AAT	Positive
18424	12	Lumbar	8	5	0	48	44	MYK, PRRT, TMR, AAT	Negative
18425	1	Knee	2	0	6	22	2	MC	Positive
18326	2	Head	3	1	x	x	x	AAT	Positive
18427	3	Ankle	4	1	5	32	14	MC, RNT	Positive
18428	5	Knee	4	0	6	26	8	MC	Positive
18429	1	Lumbar	5	0	x	36	18	ME	Neutral
18430	3	Hip	2	0	6	44	26	TMR	Positive
18431	4	Knee	6	0	4	32	24	MC, RNT	Positive
18432	2	Lumbar	4	2	5	38	26	ME, RNT	Neutral
18433	4	Shoulder	8	0	7	x	x	MC, TMR	Positive
18434	1	Head	3	x	x	x	x	PRT	Positive
18435	5	Elbow	6	1	5	48	12	MC, Neuro	Neutral
18436	1	Knee	7	0	6	34	4	MC	Positive
18437	1	Thigh	4	2	x	32	x	PRT	Neutral
18438	3	Ankle	7	1	5	38	10	MC	Positive
18439	2	Head	3	0	6	28	2	PRRT, AAT	Positive
18440	5	Ankle	8	0	6	42	4	MC	Positive
18441	15	Ankle	9	4	0	52	38	MC, MYK, PRRT, AAT	Negative
18442	3	Foot	7	2	4	28	22	PRT	Neutral
18443	3	Hip	5	1	4	24	18	PRT	Positive
18444	4	Ankle	7	0	6	32	12	MC	Positive
18445	2	Tibia	3	0	7	18	4	PRRT	Positive
18446	1	Knee	7	0	7	x	x	MC	Positive

Key: Pt ID – patient identification; # of tx – number of treatments; NRS – Numeric Rating Scale; MYK – Myokinesthetics; PRT – Positional Release Therapy; Selected Functional Movement Assessment – SFMA; Total Motion Release – TMR; AAT – Associative Awareness Technique; PRRT – Primal Reflex Release Therapy; MC – Mulligan Concept; ME – Muscle Energy; RNT – Reactive Neuromuscular Training

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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; Fahlström, 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 to 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 two to five centimeters 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 for the knee. 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 (Fahlström 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; stretching, concentric/eccentric exercises, quick rebounding toe-raises, range of motion, balance, and gait exercises. The control group performed gastroc-soleus 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 (two to six centimeters 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 two to six centimeters 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 five kilogram 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; Fahlström 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

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International Journal Sports Physical Therapy

5.1 Patellar Tendon

Abstract

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

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

Outcomes: Paired *t*-tests, with a 95% confidence intervals, were analyzed on NRS, DPA Scale, and VISA-P to determine the effectiveness of all treatment algorithm from initial exam to discharge. Cohen's *d* was also computed to determine the effect size of each of the aforementioned outcome measures. Descriptive statistics were computed for the GRC at discharge. The mean change for the NRS (M = 4.7, 95% CI[3.57 to 5.82], p < .001), DPA Scale (M = 21.8, 95% CI[12.43 to 31.16], *p* = .001), and VISA-P (M = 22.70, 95% CI[33.71 to 11.68], *p < .001*) were statistically significant. The mean for the GRC (M = 5.3) was clinically meaningful. All of the participants (100%) met discharge criteria.

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

Background and Purpose

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

Though the clinical exam is the accepted standard for tendinopathy diagnosis, varying patient presentations and injury states make it difficult to identify the origin of tendon pain.[9] Previously, tendon pain was thought to be a mechanical overuse injury, which caused

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

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

treatment complications. Upon return to activity, participants who complete the protocol also report a high recurrence rate.[15,26]

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

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

identify and treat these patients. The purpose of this study was to determine if a novel treatment based classification (TBC) algorithm could be used to classify tendon pain participants and what the effects of using the algorithm would be in participants diagnosed with patellar tendinopathy.

Procedures

Case Description

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

Examination

Each participant was examined using a pre-determined clinical evaluation to ensure consistency in patellar tendinopathy diagnosis and classification with the Treatment Based Classification (TBC) Algorithm. Inclusion criteria included: tendon pain before, during, or after patella loading activities; point tenderness over the patellar tendon upon palpation; pain

near patella origin; impaired function; and tendon focal or generalized swelling. Exclusion criteria included: cortisone injection (<six weeks), fluoroquinolones ciprofloxacin use (<12 months), post-operative participants unable to perform the treatment (<eight weeks), wore orthotics, currently healing or suspected fractures, or receiving physical therapy for the tendon of concern. Participants who met the inclusion criteria then completed a thorough history, range of motion (ROM), and special test examination. Special tests performed included: Clark's sign, patellar grind, patellar compression, prone knee bend, slump, a quarter screen for tender points, and the application of the Mulligan Concept technique for the knee (an internal rotation glide followed by an external rotation glide if pain was not resolved during application). Clark's sign, patellar grind, and patellar compression tests were performed to rule out patellar dysfunction as the source of pain. The prone knee bend and slump tests were performed to rule in neurological tension and sliding dysfunctions. The quarter screen was performed to determine the presence of tender points; while the Mulligan Concept Technique was performed last to determine classification into the Mulligan Concept treatment.

Treatment-Based Classification Algorithm

The TBC algorithm consisted of a MC technique, PRT, neurodynamics, and eccentric exercise. If the participant reported a resolution of his or her symptoms when the MC technique was applied during the exam, then the participant was classified as being a responder to the MC treatment. If the application of the MC did not resolve symptoms during the exam and the participant presented with tender points (TP), which could be reduced by moving the participant into a position of comfort (POC), then the participant was classified as being a responder to the PRT treatment. If the application of the MC did not resolve symptoms and a POC could not be identified with PRT, the participant would be classified

into the neurodynamic treatment if a positive neurodynamic test was found during the initial exam. In the case where the participant could not be classified into the MC, PRT, or neurodynamic group, the participant was classified into the eccentric exercise treatment protocol (Figure 5.1a).

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 a, 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 have evidence of effectiveness on tendinopathy patients in other research studies.[13,41,42,43]

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 week follow-up.[42] While few studies have been conducted on the effectiveness of PRT or neurodynamics in treating tendinopathy, Baker et al.[13] were able to re-establish normal, pain free function in a patient with a history of bicep tendinopathy when using PRT, while Matocha et al.[43] were able to alleviate pain and restore function in a patient diagnosed with lateral epicondylalgia using neural sliders and tensioners.

Thus, the TBC algorithm used in this study was designed because the clinicians could observe participant response to potential interventions while in a painful state and to utilize manual therapy techniques that could potentially produce rapid changes. Patient response

enabled the clinician to classify the participant to an intervention that was designed to be matched to their dysfunction. In theory, matching tendinopathy patients to therapies through classification could improve outcomes. In this study, all of the participants were classified as being a responder to either the MC or PRT and were able to meet the pre-established discharge criteria without a single participant needing to be classified into the EE protocol sub-group at any time. The preliminary outcomes associated with the use of the TBC algorithm can be viewed as similar, or potentially superior in certain cases, to the EE protocol outcomes found in the literature.

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

painful experience to produce effective outcomes. Additionally, the use of a TBC algorithm may allow clinicians to identify which participants actually need to participate in an EE protocol or when to add this protocol as an adjunct therapy to provide complete resolution of participant complaints.

While the preliminary results of this case series are important, the limitations of this study must also be noted. Although 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 this TBC algorithm were provided at sub-therapeutic doses (e.g., not treating multiple TPs with PRT, etc.) and the interventions could be more effective if treatment dose was maximized. 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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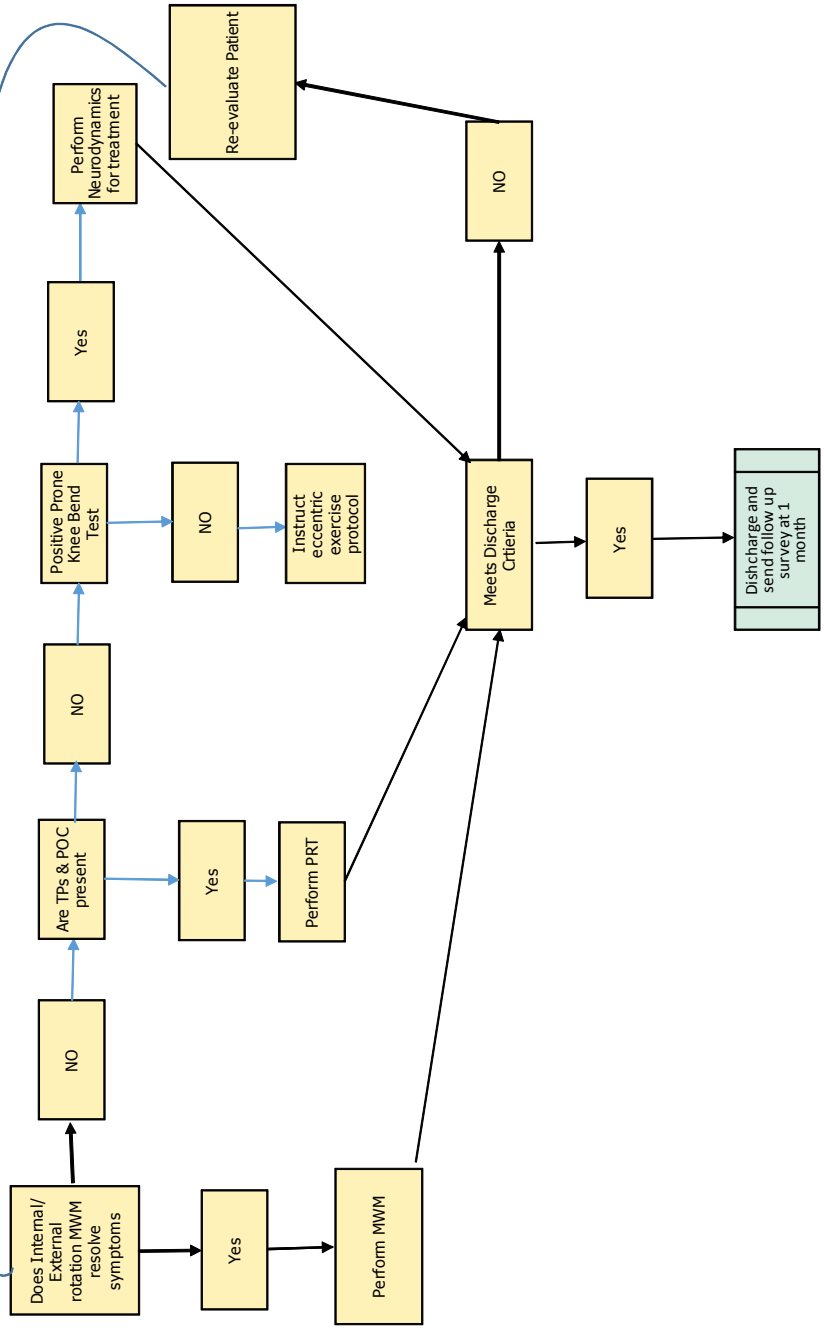

Figure 5.1a: Treatment Classification Algorithm for Patellar Tendinopathy

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

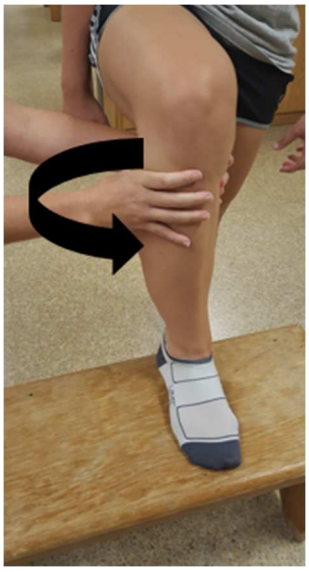

Figure 5.1c: Example of a Positional Release Therapy Technique

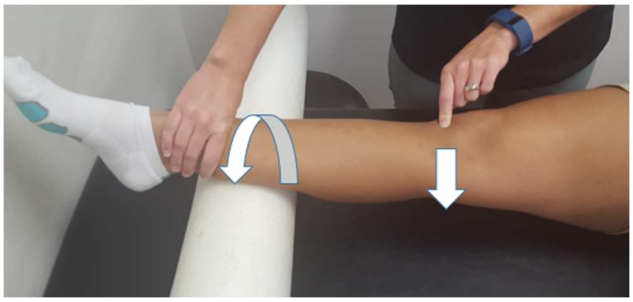

Figure 5.1d: Example of a Neurodynamic Slump Slider Technique

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						

CHAPTER 5

APPLIED CLINICAL RESEARCH

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

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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 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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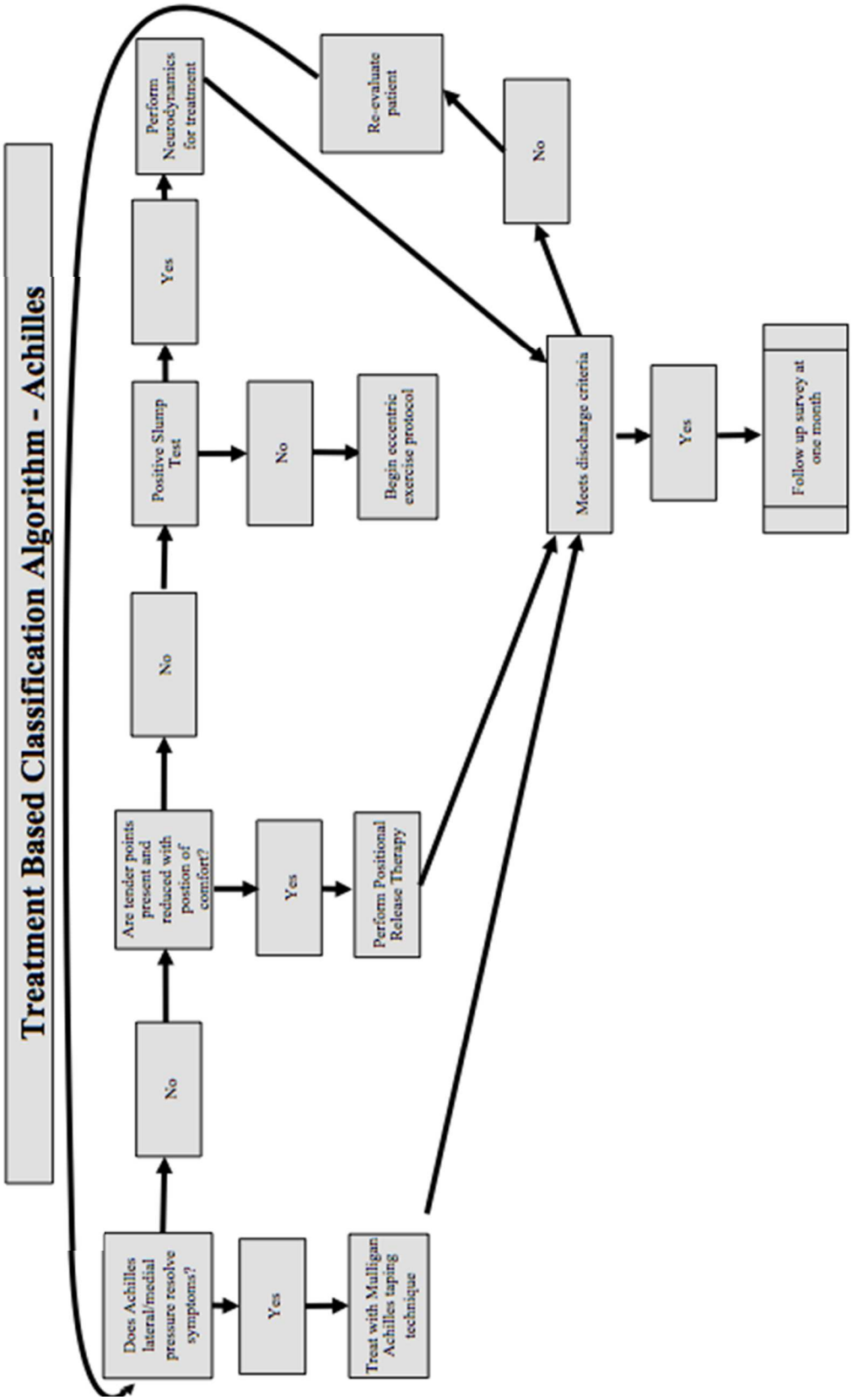

Figure 5.2a: Treatment Based Classification Algorithm for Achilles Tendinopathy

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.

CHAPTER 5

APPLIED CLINICAL RESEACH

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

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International Journal of Athletic Training & Therapy

5.3 Lateral Elbow

Key Points:

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

Abstract

Context: Lateral epicondylalgia is a common condition experienced by both males and females in occupational environments and the sports arena. Appropriate classification and treatment of lateral epicondylalgia 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 literature.[38]

Patient Related Tennis Elbow Evaluation

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

substantial change after six treatments on the PRTEE. The reported change in PRTEE, from the participant who withdrew, was 11 points, a MCID, following three treatments (initial = 56, post = 45).

Global Rating of Change

Analysis of the GRC outcome measure revealed a mean change score (M = 4.85 ±2.31) that met the MCID value prior to discharge and also exceeded the MCID value for "important improvement."[40] Additionally, 33% (two out of six) of participants perceived their change as "a great deal better" in three treatments, while 50% (three out of six) achieved similar or greater changes in six treatments. The participant who did not complete the study reported no change in the GRC (zero).

Nirschl Phase Rating Scale

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

Discussion

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

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

Although no gold standard treatment has been established for LE, due to the effectiveness of EE protocols for patella and Achilles tendinopathy, EE is prescribed for patients presenting with LE.[23-29,44-46] Most EE protocols for LE range from four to twelve weeks and are frequently coupled with other modalities.[44] In a review of 12 research studies on the effectiveness of EE programs for treatment of LE, 60% incorporated adjunct therapies

(e.g., stretching, icing, ultrasound, massage, heat) with EE, and reported a mean of 7.7 weeks of treatment. The frequency of required treatments ranged from three to seven per week.[44] Also, in the aforementioned studies, researchers reported grip strength deficits and functional impairments of the elbow still remaining at discharge.[44] In the current study, without using adjunct therapies, participants were discharged in less time (8.6 days ± 6.18) when compared to EE protocol time frames (four to twelve weeks) and 67% (four out of six) of participants who completed the study reported complete resolution of symptoms in three to nine days (three to six MWM treatments).

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

The results from the current study also compared favorably to other studies conducted on the effectiveness of MWMs for the treatment of LE. In a single case study, a patient received four MWM treatments over a two-week period before reporting full resolution of

pain associated with LE.[25] In another study conducted by Amro et al.,[48] 17 subjects reported a statistically significant change (mean change = 5.3 ± 0.9) in VAS pain rating to a final score of one out of ten following three MWM treatment sessions over four weeks. Additional therapy (i.e., self MWM exercise, Mulligan adjunct taping technique) was included in the MC treatment of LE in previous studies, which is a common recommendation to extend and ensure the long lasting therapeutic effect of MWM treatments.[49] In the current study, without using adjunct taping or prescribing self MWM exercises, 83% (five out of six) of the participants reported resolution of pain and return to function at discharge in six treatments or less. Long-term effects in our study are difficult to conclude due to limited participation (50%, three out of six) in the follow-up survey; however, of these three participants, two reported no change in pain (zero out of ten NRS), while the other reported a NRS rating of one out of ten.

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

Despite the positive outcomes, a number of potential limitations were present in this study. First, the current study had no control group and participants were not randomly assigned. 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

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 5.3c Changes in Patient Reported Outcome Measures

P No.	Age	G	Function (PRTEE) (0-100)			Pain (NRS) (0-10)			Disability (DPAS) (0-64)			Number of Treatments
			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	

P No.=Participant number, G=Gender, M=Mean, SD=Standard deviation, P=P value, Cd=Cohen's d
*Did not complete study

Figure 5.3a Lateral Elbow Treatment Based Classification Algorithm