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Examining Altered Neural Activation In Patients With Patellar Tendinopathy: A Preliminary Study

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Examining Altered Neural Activation In Patients With Patellar Tendinopathy: A Preliminary Study

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Examining Altered Neural Activation In Patients With Patellar Tendinopathy: A Preliminary Study

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ABSTRACT

Examining altered neural activation in patients with patellar tendinopathy: a preliminary study

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CONTEXT: Patellar tendinopathy (PT) is a common injury that can affect 15-45% of all athletes, with nearly 50% of these athletes reporting no longer being able participate in sport due to the severity of the injury. One of the consequences of PT leading to physical inactivity is muscular weakness, particularly in the quadriceps, which may be attributed to arthrogenic muscle inhibition (AMI). **OBJECTIVE:** To examine the extent of quadriceps AMI in individuals suffering from PT and secondly, to compare subjective reports of pain and dysfunction with objective measurements of PT diagnosis. **DESIGN:** Case-control. **SETTING:** Laboratory **PARTICIPANTS:** Six participants with diagnosed PT (<80 on Victorian Institute of Sport Assessment - Patella (VISA-p) and six healthy participants (VISA-p>80) matched to the PT group based on age, mass, height, and gender. **INTERVENTION:** Each participant completed one test session, which included patient-oriented outcome questionnaires, diagnostic ultrasound (US), Hoffman's Reflex (H-Reflex), superimposed burst to assess central activation ratio (CAR), and a standardized jump landing test. Neuromuscular control during the jump landing was evaluated using the Landing Error Scoring System (LESS) and electromyography (EMG) of the vastus medialis and lateralis. **MAIN OUTCOME MEASURES:** H:M ratio, CAR, LESS score, percent maximum muscle activation during CAR, and percent stance muscle activation during the pre-loading and loading phases of jump landing were assessed between groups using independent-t tests (α<0.05). **RESULTS:** With respect to CAR, the PT group (95.13%±3.46) compared to the healthy group (98.54%±0.60) exhibited a significant loss of

volitional muscle activation (P=.04). There were no differences between groups for H:M, LESS score, or muscle activity (P>0.05). The PT group was then divided into two sub-groups: Subjective (participants that were diagnosed with PT via subjective reports alone)(n=4) and Objective (participants that were diagnosed via subjective reports and US)(n=2). The Objective group demonstrated lower CAR, H-Reflex, muscle activity, and a higher LESS score than both the Subjective PT group and the healthy group. **CONCLUSION:** This study was the first to demonstrate deficits in the PT population using measures of peripheral sources of AMI. Further research needs to be conducted on the extent of these deficits and whether AMI induced from PT is associated with an increased risk of lower extremity injury.

Key Words: neuromuscular dysfunction, cortical alterations, peripheral inhibition

Chapter I: Review of Literature

Patellar Tendinopathy

Knee injuries represent 15.2% of all high school injuries and can cause time losses for athletes ranging from a few days to being career ending. [1] These injuries can include damage to bone, ligament, cartilage, or tendon. Knee injuries are common in lower extremity sports with patellar tendinopathy (PT) being one of the most prevalent. [1] While some knee injuries are more severe their incidence rates are low compared to a knee injury like patellar tendinopathy, which can affect up to 50% of athletes in at the elite volleyball level. [2] Patellar tendinopathy can have long-term effects on the ability for athletes to continue with participation in sport. [3,4]

Etiology and Prevalence

PT is very common and can occur in 15-45% of all athletes. [1,5-7] The prevalence of PT in elite male volleyball players has been reported to be 45%. [2] PT does not only affect volleyball athletes as prevalence in basketball can approach 30%. [6] The prevalence of PT can vary from 14.4% to 2.5% in non-elite athletes, with males being almost twice as likely than females to have the condition. [5,7] Since PT is common in jumping sports at all levels of athletic participation, it is important for clinicians to have an understanding of PT etiology.

The first step in understanding why PT so commonly affects jumping athletes is to recognize what PT is and the role of the patellar tendon during activity. Tendinopathy is a painful, chronic; overuse injury that is associated with degeneration and disorganization with no evidence of inflammation within the tendon. [5,6] There are two main classifications of patellar tendon injury; the first is inflammatory enthestis, which is described as inflammation at the site where tendon intersects with bone and the second is tendinopathy. [8] Patellar tendinopathy

typically presents as pain at the deep proximal portion of the tendon just inferior to the patella that may or may not be accompanied by edema.[9] Patients may also present with pain during knee extension exercises like squatting and jumping. Not only can PT produce pain but it has been demonstrated to cause concentric quadriceps weakness in female fencers.[10] The main purpose of tendon is to transfer force from a muscle to a bone thereby causing joint movement.[6] Other functions of tendon include energy storage and release in addition to protection of the muscle from injury.[6] The patellar tendon must transmit a high volume of force form the quadriceps to the tibia in a jumping sport.

Although the etiology of PT still is not completely understood, a few possible explanations for this pathology have been suggested by the literature. PT is very common in sports that include repetitive jumping and landing like basketball and volleyball.[5] Since tendon tissue transfers force between muscle and bone, the PT is responsible for absorbing high loads and converting this energy to mechanical work that can be utilized during dynamic activities, such as jumping. Given the unique role of the tendon during high-loading activities, a high volume of repetitive jumping is a risk factor.[6,9,11-15] Some other possible causes of PT include a stiff landing technique, high jump performance, large waist girth, male gender, and high training volume.[5] Each of these factors point towards tendinopathy developing when greater strain is present. Jumping higher and having a large waist girth are two traits most often attributed to males and may play a role in the high gender incidence rates.[5] The premise behind these two ideas are that landing from a higher height and increased weight will cause more eccentric strain during landing. Having a stiff landing technique is believed to cause increased tendon strain because less energy is absorbed through supportive musculature that utilizes a limited range of motion, thus transferring more force to the tendon.[5] A new perspective on PT in volleyball may

come in the form of genetics, where researchers have found that volleyball players who are carriers of the BMP4 and FGF3 genes may be predisposed to developing PT. [2]

The prognosis for untreated PT is poor. Gisslen et al. [16] showed through a three-year observation that patellar tendon condition did not change if training intensity remained the same. The study followed 22 high school volleyball players, 11 males and 11 females, through three years of school. Each player had both of his or her patellar tendons examined by ultrasound (US), and power doppler (PD For participants who had clinically and sonographically diagnosed PT,symptoms remain as long as intense training or competition was present. The results of this study show that without rest PT will be a persistent problem for the jumping athlete.

In another study that included a measurement for the duration of PT, van der Worp et al. [17] found that the median length of time a patient suffered from PT was 24 months, however duration of symptoms ranged from 1-219 months. Participants in the study were invited via email to fill out an online questionnaire regarding their demographics, current knee injury status, sport participation, and occupation. PT was assessed using the VISA-P. Men were twice as likely to have PT, and those with a physically demanding job in addition to playing sports regularly were at greater risk for developing PT. When untreated PT lasted an average of 24 months. Both of these studies demonstrate that untreated PT does not resolve without treatment and a decrease in sport or work intensity. For the clinician, these studies help to outline why PT should be taken seriously, especially in males that participate in sport at a high intensity.

Bisseling et al. [18] further illustrated how landing mechanics can both influence and be influenced by PT. This study assigned 89 male participants into one of three experimental groups; 1) a control group with no previous or current PT, 2) a group with a history of PT but no current symptoms, and 3) a group that had PT at the time of the trials. Participants were asked to

step off of boxes that were 30cm, 50cm, and 70cm high five times per box height landing one foot on a force plate. The results of the study showed that the group with recent PT tended to avoid patellar tendon loading during landing through lowering the velocity and rate of range of motion chance of the ankle and knee, while participants with a history of PT tended to land much more stiffly than their counterparts. These data not only suggest that PT can influence the alteration of landing mechanics in the short term but that a stiff landing could either be a potential cause of PT or a long term side effect of the disorder.

Signs and Symptoms

The signs and symptoms of PT are considerably better recognized and understood than the etiology. One of the more common symptoms is pain over the proximal tendon during loading activities like jumping and squatting. [6] A second indicator is pain on palpation of the proximal tendon as it attaches to the inferior patella. [19] Although not as easy to assess clinically, a study of patients with PT by Souza et al. [20] found that they display greater hip extensor activation and decreased knee extensor activation when compared to asymptomatic controls during a hopping task. The experiment compared seven men with history of PT to an equal control group without history of PT. All participants were evaluated for their kinematics and kinetics and asked to hop in place for 20 successful hops on a force plate. Even though the PT group did not report painful hopping, they demonstrated a higher ratio of hip joint to knee joint effort than did the control group. This study suggests that those suffering from PT will attempt to reduce knee joint loading by using hip extensors to absorb landing forces, which shifts loading from the knee to the hip. This shift in loading could possibly be due to a weakness, neuromuscular dysfunction of the quadriceps muscle, or as a conscious effort to decrease pain.

Signs and symptoms are important when making the determination of whether a patient is suffering from PT, however the use of imaging devices is necessary in determining the extent of tendon degeneration, disorganization, and vascularization. Magnetic resonance imaging (MRI), ultrasound (US), and power doppler (PD) are all imaging tools used in the diagnosis of PT. [6] All three instruments are non-invasive but US is the most widely used imaging for PT. US can be used to show tendon thickness, structural changes, and neovascularization. [21] In addition, US has a higher spatial resolution than MRI, resulting in a more detailed picture from a portable and affordable unit. [21] Finally, although US has a similar specificity to MRI (.82) US has a higher sensitivity (.87) than MRI (.57). [8] However, the US is not perfect and one of its biggest drawbacks is that sonographic tendon abnormalities often appear in the absence of clinical symptoms of PT, [22,23] as 10%-18% of tendons that are abnormal on US never become painful. [22]

Some common abnormalities detected via US are tendon thickening, hypoechoicity of the tendon, increased collagen bundle disorganization at the proximal tendon, and greater cross sectional area. [22,24,25] While US is useful for visualizing structural abnormalities, both power and color doppler are useful in determining vascularity within a tendon and have been able to show an association between pain during activity and the amount of vascularization in the tendon. [16] This association is most likely due to an increase in pain causing chemotaxic factors being carried to the injured area. One drawback of doppler is that only high flow rates, like those sometimes present in PT, can be detected. [16] The MRI is the least used of the three because of high costs and inability for differentiation between inflammation and degeneration in the tendon.[24]

The Victorian Institute of Sport Assessment Scale – Patella (VISA-P) is a patient reported outcomes questionnaire designed for individuals suffering from PT and is the most

widely used assessment for PT.[26] The form consists of eight items that are split into two sections. The first section consists of six parts that look to identify pain levels during activities of daily living and functional tests, while the second section has two parts that target the patient's sport participation level.[26] The questionnaire is scored on a scale of 0-100 with 100 being a patient that exhibits no signs or symptoms of PT and zero being the bottom of the scale, which is the equivalent of total disability. A recent study[26] has shown that this scale is able to detect and represent changes in the patient's condition. What this means for the clinician is that he or she will have an accurate idea of how their patient is or is not responding to treatment. The VISA-P has also been shown to be accurate in both the online and written formats. It should be noted that the questionnaire was not designed to be used in research with control subjects that are not suffering from PT, and using the questionnaire in such a way could alter its results.[27]

Arthrogenic Muscle Inhibition

Arthrogenic muscle inhibition (AMI) is often referred to as an ongoing reflex response to joint injury that affects the periarticular musculature.[28,29] AMI is hypothesized to be the body's mechanism to protect the injured joint and prevent further painful stimuli.[30] AMI is caused by a variety of peripheral and central mechanisms that result in either the loss of the ability to volitionally activate alpha motor neurons or a decrease in the alpha motor neuron's firing rate.[29] This decreases the ability of periarticular muscle to voluntarily and reflexively contract, which can lead to atrophy and poor functional ability.[30] AMI has been detected in the musculature surrounding the knee joint following injury, primarily in the quadriceps muscle group.[28,31] Interestingly it has been reported that anterior knee pain seems to cause a greater magnitude of inhibition than does ligamentous injury.[32]

Peripheral Mechanisms

The neurologic pathways involved in AMI are complex, which is one of the key reasons they are still not completely understood. However, AMI is thought to occur due to peripheral and central inhibition or a combination of the two. In the peripheral pathway either effusion, mechanoreceptor damage, and/or pain can cause altered afferent signaling from what would be considered normal. For example, increased afferent activity is what takes place when a joint becomes effused.[31] The effusion within the joint space causes a rise in group II articular afferents discharge from the joint capsule due to the excessive fluid built up and activated mechanoreceptors of the joint capsule. An example of altered afferent activity would be in the case of destroyed mechanoreceptors from the loss of a ligament.[31] Normally, joint mechanoreceptors relay information to periarticular musculature, but once they become damaged the mechanoreceptors can no longer complete this function. Both increased and deceased afferent signaling can cause presynaptic inhibition of the musculature surrounding the joint. In the patellar tendinopathy population it is most likely that pain at the site of injury would cause peripheral AMI due to altered afferent signaling of 1a fibers.

Central Mechanisms

Central mechanisms of AMI include pre-synaptic, cortical, and spinal mechanisms. At the pre-synaptic level inhibitory interneurons release GABA, which decreases the ability of the neuron to generate an action potential by causing chlorine to change the electrical gradient surrounding the neuron. The change in this gradient reduces the amount of calcium entering the pre-synaptic terminal and inhibits proper muscular activity.[30] To examine a second mechanism of centrally originating AMI, Lepley et al.[33] demonstrated joint injury can cause alterations in cortical excitability, which can act to modulate efferent signaling at the alpha motor neuron level.

Rio et al [34] conducted similar work in which it was found that chronic PT can influence cortical changes also.

AMI caused by the spinal pathway can originate from gamma motor neuron loop dysfunction, reciprocal inhibition of type 1a afferent fibers, non-reciprocal inhibition of type 1b fibers, and recurrent inhibition via Renshaw cells [35]. In the gamma loop afferent signals from within the injured joint are transmitted to gamma motor neurons (GMN). These afferent signals into the GMN inhibit a signaling process between the GMN and intrafusal fibers within the muscle spindle [34]. This alters the shortening reaction of intrafusal fibers within the muscle spindles and results in a decreased capacity for muscle contraction. [36] Along with the decrease in contractibility, signals from 1a afferent to high threshold motor units are inhibited, which results in decreased force output. [30] Inhibition of type 1a afferent fibers is caused by firing of motor neurons in the agonist muscle group. This firing stops the antagonist group from working correctly because both the agonist and antagonist groups cannot contract completely simultaneously. Type 1b inhibition is caused by afferent signaling arising from the golgi tendon organ to inhibit the agonist muscle group and activate the antagonist group. Renshaw cells work in the spinal cord and cause inhibition of the target muscle group by decreasing the action potential that is received by the alpha motor neuron. [30]

Quantifying AMI

Whether to detect the presence of AMI or to quantify the amount of inhibition a muscle is experiencing, researchers commonly utilize either the superimposed burst or interpolated twitch technique. [37] One of the most often used equations to calculate muscle activation, which can be used with either testing method, is the central activation ratio (CAR). The formula is derived for

the patients maximum voluntary isometric contraction (MVIC) by their MVIC plus a stimulated force measured in torque. [38]

$$CAR = (MVIC) / (MVIC + stimulated\ force)$$

The force of contraction is measured on a dynamometer, which can quantify torque for each MVIC. [37] The stimulated force described in the CAR equation is the amount of torque caused by a superimposed electrical burst. This process will be described in detail later in this paper. CAR is the percentage of complete muscle activation that is occurring. For example, a patient demonstrating a CAR of 1.00 would mean that he or she had complete voluntary muscle activation, while a CAR of .8 would signify that the muscle was activated to 80% of its complete potential. [39]

CAR measurements are used to determine the amount of voluntary activation possible for the specific muscle. Roberts at al. [37] determined possible confounding variables that can influence quadriceps CAR. The included variables were patient positioning, verbal cueing from the clinician, synergistic muscle activation, and antagonist co-contraction. The purpose of measuring CAR is to determine the amount of activation in a specific muscle. It is important that only one muscle, or muscle group, be targeted without the use of synergists Synergistic muscle activity includes trunk and arm muscle contractions to stabilize the upper body. An example of antagonist muscles are the hamstring group co-contracting when the quadriceps group is activated. Avoiding hamstring group activation is important because these muscles decrease quadriceps activation force. If the patient is properly positioned and verbally cued on isolating

the quadriceps, then synergist and antagonist muscle activation can be avoided. The study went on to show that less synergistic muscle involvement can decrease CAR.

The superimposed burst (SIB) method is used to obtain the CAR to determine the total possible torque that all of the motor units for a specific muscle could create in an ideal situation. A SIB delivers electrical impulses that are able to increase the total number of motor units being recruited, which increases torque beyond a patient's MVIC. [37]. The SIB can be delivered to a muscle in a single, doublet, or train series of pulses. [40] While each method can be used to identifyAMI the pulse trains or burst methods have been found to be more sensitive to central activation failure (CAF) than the single or double methods. [38] The SIB method has been shown to reliably measure central activation of the quadriceps. [37] Even though the method is reliable there can be up to a 5% measurement error with the SIB technique. [41] There are different methods demonstrated for performing SIB and two have been utilized for the quadriceps. The possibilities include the vastus and rectus configurations and these can be set up with either self-adhesive or carbon impregnated electrodes. Both configurations and electrode types have been shown to be interchangeable, which demonstrates the versatility of the SIB method. [42] One final measure of versatility for the SIB method is that it can be used in the percent activation equation with success rates similar to that of CAR. Researchers have been able to demonstrate that when SIB is used with a doublet pulse configuration the percent activation equation correlate well to CAR using SIB with a train of pulses. [40]

Although the use of a superimposed burst technique to calculate CAR is effective at quantifying AMI, one if the drawbacks is that the method cannot determine where AMI is being generated from. The Hoffmann Reflex (H-Reflex) is a peripheral nerve stimulation technique that has been used as an accessory to CAR because of the ability to assess the role that the

spinal-reflex loop may play in the development of AMI. [43] An H-Reflex in a peripheral nerve is an electrically induced equivalent to the muscular stretch reflex, and looks to account for levels of inhibition discussed earlier (presynaptic, postsynaptic). To record H-Reflex an electrical stimulation is delivered to a peripheral nerve, which causes an afferent signal discharge of the 1a nerve fiber to the spinal cord, and the efferent "reflex" is detected using electromyography (EMG) in muscles innervated by the stimulated nerve. [43] The resulting increase in electrical activity within the muscle is represented as a wave on the computer and is considered to the "H-reflex". In subjects with AMI a decrease in peak-to-peak amplitude of the "H-reflex" or an increased stimulus voltage needed to elicit comparable peak-to-peak amplitude when compared to a healthy or control limb and normalized to muscle response would denote an inability for the 1a afferent nerve to effectively transmit electrical impulses to the alpha motor neuron, resulting in an inability to reflexively contract the muscle.

Many confounding variables exist when eliciting the H-Reflex during testing, therefore the process must be performed in a controlled environment. As stated above, presynaptic inhibition of afferent sensory signaling can greatly affect the amplitude of the H-reflex. Changes in light, sound, noise, or body position are just a few of disturbances that can cause presynaptic inhibition. [43] This inhibition occurs because any external stimuli require afferent signaling to reach the spinal cord, as does the H-reflex stimulus. Any additional stimuli can dilute the experimental stimulus for eliciting the H-reflex, which will result in decreased peak-to-peak amplitude as detected though EMG assessment. [44]

In addition to the H-reflex, another variable called the M-Wave is also elicited during H-reflex testing. As the electrical stimulus is increased, the higher threshold efferent limb of the stimulated nerve (i.e. the alpha motor neuron) is directly depolarized. Once the alpha motor

neuron fibers are directly depolarized, the EMG can detect the electrical activity from this event, the M-Wave.[43] Since the M-Wave represents direct depolarization of the alpha motor neuron there is no influence from the spinal cord. Thus, the M-Wave represents the maximal capacity of the alpha motor neuron excitability, while the H-reflex represents the ability of afferent stimulus to evoke efferent spinal cord excitability. These two waves are represented as a ratio of the maximum peak-to-peak H-reflex amplitude compared to the maximum peak-to-peak M-Wave amplitude, H-reflex $_{Max}$: M-Wave $_{Max}$. The comparison of the waves allows researchers to begin to make inferences about the source of AMI. A decreased peak-to-peak H-Wave amplitude with no associated decrease in peak-to-peak M-Wave amplitude is believed to show that the spinal cord, which is not involved with the M-Wave, is influencing efferent signaling to the muscle being tested. Normalizing H-relfex to muscle response allows the researcher to determine if alterations in the H-reflex are also present during muscle response.

Quadriceps AMI

Quadriceps AMI is a common consequence of a variety of knee injuries. In particular, it has been estimated that quadriceps strength deficits in patients with ACL ruptures range between 8%-45%. Some injuries that have been linked to quadriceps AMI are primary ACL injuries, anterior knee pain and osteoarthritis.[29,30,37] Previously discussed in less detail, ACL injuries damage mechanoreceptors in the ligament itself, which can cause abnormal afferent information to be sent to the CNS and PNS and result in muscle inhibition.[30] This leads to an inability of the patient to properly control the quadriceps muscle, potentially leading to biomechanical alterations during gait and athletic participation, which could be problematic for active individuals.[37]

Quadriceps weakness can have major effects for not only the athletic population but also the general population that suffers from AMI. The quadriceps act eccentrically during gait, and a weakened muscle with decreased contractility could adversely affect the gait cycle. [32] Quadriceps weakness can also cause decreased dynamic joint stability, which could lead to decreased functional ability and eventually tibiofemoral osteoarthritis (OA). [31,39]

Palmieri-Smith and Thomas [30] have described some of the changes which take place in the knee joint when the ipsilateral quadriceps is experiencing weakness and how this may effect articular cartilage. First, the load placed upon the knee joint increases as quadriceps strength decreases. The authors believe this was due to the quadriceps acting as a shock absorber for the knee. When the quadriceps muscles are weak they absorb less force, and more force is transmitted to the knee joint and consequently the articular cartilage. Second, altered joint kinematics due to muscular inhibition also lead to new stresses to the articular cartilage which were not previously present. This increased stress on articular cartilage is a possible cause for OA.

Manal and Snyder-Mackler [45] looked to identify quadriceps AMI that may be present after patellar contusions. In 16 participants with patellar contusion, only 1/3 of patients experienced quadriceps muscle AMI with the clinical diagnosis of AMI only being made by determining %MVIC. Patient reported outcomes for those diagnosed with AMI did not show any deficits in daily function. The results of this study show that AMI is possible with a relatively benign injury, however, without the use of laboratory equipment AMI could easily go unnoticed.

Once AMI is detected, or thought to be detected, it must be quantified. Kuenze et al. [41] examined 22 participants with a history of ACL reconstruction in an attempt to determine return to participation criteria for individuals with quadriceps AMI. The study found that 3.00Nm/Kg of

normalized quadriceps strength was a good indicator of positive patient reported outcomes in regards to knee function, as was CAR symmetry of greater than 99.2 %. These data provide clinicians will a baseline for which to compare an individual suspected to be suffering from AMI.

Although AMI has been linked to ACL and other major knee joint injuries, it has yet to be shown as a result of PT. Some evidence does exist that PT can cause concentric quadriceps weakness in female fencers. [10] Though concentric weakness could signal that AMI is present, it alone is not a definitive sign. As in ACL injury, CAR and associated testing must be done to determine if the muscular weakness is due to AMI and where this AMI may be originating from.

Movement Assessment

The Landing Error Scoring System (LESS) is a clinical movement assessment tool, which was designed to help identify individuals at risk for ACL injury, but can also be used to help assess neuromuscular control during functional movement. The LESS requires participants to jump off of a 30cm high box onto a force plate set at 50% of the participant's height from the front of the box while being recorded by video cameras in the frontal and sagittal planes (Figure 1). [46] To complete the trial the patient must successfully perform the jump, landing, and second jump three times (Figure 2).

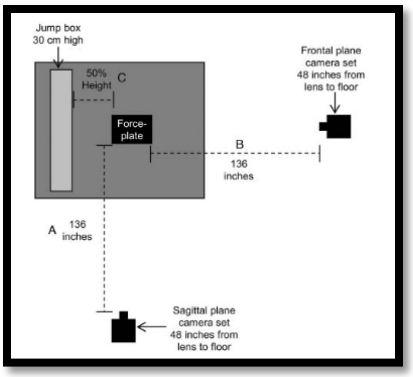


Figure 1. [46]



Figure 2. [46]

After the patient has been filmed a clinician reviews the videos and grades the participant. There are 22 error types that the LESS grades that range from knee valgus at initial contact to joint stiffness. [46] A high score on the LESS correlates to poor biomechanical landing abilities. A poor score greater than 5 is considered predictive of ACL injury. [47] The LESS has been shown to; 1) have good validity when compared to motion analysis, 2) have good interrater ($ICC_{2,k}$ – 0.84 and SEM – 0.71) and intrarater ($ICC_{2,1}$ – 0.91 and SEM – 0.42) reliability, and 3) identify at risk individuals. [46,48] This makes the LESS a clinically applicable test in the fields of overall lower extremity injury including ACL research, rehabilitation, and prevention strategies, although the LESS has not been specifically studied in the PT population.

Future Directions

The purpose of this study is to identify AMI and the mechanisms leading to poor neural control. If this study identifies altered neuromuscular outcomes future research will seek to identify ways to target pathways that lead to AMI or poor neural control. One potential treatment is cryotherapy, which is a common modality in the clinical setting that might have further application in the treatment of AMI. [29] A second possible clinical approach to treating AMI is through motor neuron treatments, like neuromuscular electrical stimulation [35,49,50]. This is thought to work is by directly stimulating the affected motor neurons, causing a contraction to occur in inhibited muscle fibers. [31] A third clinical approach to treating AMI is through addressing the PT itself by using eccentrics to load the tendon [51-53]

Conclusion

Patella tendinopthy is common, especially in athletes that participate in jumping sports. In athletes with PT, the physical and mechanical properties of pathological tendon differ from

normal. The changes in tendon properties, which are evident in PT, have been shown to decrease concentric quadriceps strength. Decreases in quadriceps strength have also been evaluated in participants with ACL injury and in those with OA. A component of the quadriceps strength impairment in both of these conditions is related to diminished neuromuscular control. Alterations in neuromuscular control after joint injury is often referred to as AMI. CAR is one of the primary methods used to assess the magnitude of AMI but does not identify the pathways underlying the deficit. To help identify exactly where the source of the AMI is originating from other measures must be utilized. A combination of H-Reflex testing, to evaluate spinal cord influence, EMG, to determine alterations in dynamic muscular activity, and LESS score, a functional means of evaluating neuromuscular control were all used to make a complete picture of why AMI is present. Quantifying the effects of quadriceps AMI induced by PT have not been extensively investigated and has not been quantified in terms of non-functional, CAR and H-Reflex, and functional, EMG and LESS score, measures. Therefore, the purpose of this investigation is to quantify AMI that is potentially present as a result of PT using the CAR, and then to identify the pathways that are leading to the AMI using H-Relex, EMG, and LESS score.

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Chapter II: Introduction

Patellar tendinopathy (PT) is a degeneration of the patellar tendon that can impair athletic performance, quality of life, and neuromuscular control that may lead to future injury. [1] PT is particularly prevalent in jumping sports, such as volleyball, where it can affect close to 50% of elite male athletes in the sport. [2] Untreated PT can become a source of painful physical activity participation that can influence musculature around the knee by causing generalized weakness. [3] Consequently, nearly 33% of athletes that have chronic PT take longer than six months to return to play. [4,5]

Muscle weakness resulting from joint damage is often referred to as arthrogenic muscle inhibition (AMI). [6,7] AMI is thought to be a mechanism of the body to protect a joint following injury and is commonly seen in the quadriceps muscle group after anterior cruciate ligament (ACL) injury. [8] Two pathways are known to influence AMI, central and peripheral, each varying in ways of inhibiting muscle activity. [6-10] Peripherally induced AMI has been shown to be present in both experimental and injury models in conjunction with persistent pain, which is possible in patients with PT. [11] Losses in quadriceps strength and altered cortical excitability have been documented in patients with PT, [3,12] but it is unknown if AMI might result from peripheral or spinal mechanisms specifically in patients with PT.

AMI can be quantified in many ways, which includes measuring the central activation ratio (CAR) and Hoffman's reflex (H-Reflex). CAR identifies the presence of AMI, through detecting a drop in volitional muscle activation that results from either a decrease in motor neuron firing rate or the number of motor units being recruited [13], but cannot pinpoint the source of the dysfunction (ie. either central or peripheral). While Hoffmann-Reflex (H-reflex) is the normalization of spinal cord reflexivity to maximal muscle response (H:M ratio) in order to

quantify peripherally originating AMI. H-Reflex is a method of stimulating 1a afferent fibers in the muscle spindle and recording the electrical activity that occurs in the target muscle following stimulation [9]. Since the afferent fibers must travel through the spinal cord, any alteration in resultant electrical activity from what is considered normal is attributed to spinal cord influence.

Electromyography (EMG) is used during testing of the H-Reflex to detect electrical stimulus that is leaving the spinal cord or can be used to support other measures by showing how a patient is engaging various muscles during physical activity. This is accomplished through recording the electrical activity between two conduction points, commonly surface electrodes, which takes place during depolarization while a muscle is at work. Alterations in the quality of muscular activation may also influence movement control during functional activities. Certain movements during sport-specific tasks, such as a jump landing, have been associated with lower extremity musculoskeletal injury risk, and such, are important to understand. The Landing Error Scoring System (LESS) was designed as a specific clinical movement assessment tool for the jump-landing task. Together these measures allow for the quantification of AMI and the identification of possible spinal cord involvement, along with the ability to understand muscular activation throughout physical activity and the role this may play in altered mechanics during a jump landing.

Thus, the purpose of this study was to evaluate the presence and origin of AMI in individuals suffering from PT using CAR, EMG, and H-Reflex along with clinically applicable measures, such as the LESS and subjective patient-oriented outcome scales. Our hypothesis is that arthrogenic muscle inhibition will be present in patients with PT, leading to a decrease in CAR, muscle activity, H:M ratio, and LESS score.

Chapter III: Methods

Participants

12 males (n=6) and females (n=6) between the ages of 13-40, were recruited to participate in this research study. Six participants were self-reported as having PT, with symptomatic PT quantified as score of < 80 on the Victorian Institute of Sport Assessment - Patella (VISA-P). Exclusionary criteria for PT participants were a history of lower extremity surgery, any lower extremity injury in the previous six months (other than PT), or having a heart related condition. Six other participants served as healthy controls, scoring between a 95 and 100 on the VISA-P. To be included in the control group, participants needed to be both healthy and physically active at the time of the trial, with no previous history of lower extremity surgery, current knee pain or have a heart related condition. Each participant in the PT group was gender-matched to a control participant that most closely resembled him or her in height and mass. All participants completed informed consent forms, which were approved by the University of Connecticut's Institutional Review Board.

Quadriceps Activation Testing

To quantify defects in volitional muscle activation and determine whether AMI was present, superimposed burst (SIB) testing [14] was performed on a Biodex dynamometer (System 4, Biodex Medical Systems, Inc, Shirley, NY). [15] Each participant was positioned in the Biodex so that his or her hips and knees were both flexed to 90 degrees and the torso was flexed to 90 degrees. [7] Two 7x13cm self adhesive stimulating electrodes were placed on the participant's thigh in space that had already been cleaned with isopropyl alcohol and was shaved free of hair. The superior electrode was placed over the vastus lateralis so that its medial border was in line with the anterior superior iliac spine at the level of the greater femoral trochanter. The inferior

electrode was placed over the vastus medialis so that the lateral border aligned with the midpoint of the patella at a height of 1.5 inches above the superior pole of the patella.

Once the participants were properly positioned, a series of three introductory practice trials were conducted. For the practice trials the participants were instructed to extend their knee at 25, 50, and 75% of their perceived maximal exertion. At the same time as contraction, a stimulus of corresponding percentage of the maximal 150 volts was delivered to the participant's quadriceps. A custom written program (LabVIEW Version 8.5; National Instruments Corporation, Austin, TX) delivered each stimulus as a 100ms train of 10 stimuli, at 100 pps, with a pulse duration of 0.6ms, and a 0.01ms pulse delay [16]. After the introductory practice trials were complete the participant was instructed to complete three MVIC trials, or until the researcher determined that the participant was putting forth full effort, without any electrical stimulation. Finally, three MVIC trials were completed along with electrical stimulation equal to 100% of the 150 volt maximum. The participants were given visual feedback and verbal encouragement during each trial and a 60 second recovery session between each trial. The entire procedure was then completed on the contralateral limb and the order of tested limbs was counterbalanced. The lowest of the three trials was used in data analysis. A Grass S48 dual-output square-pulse stimulator and SIU8T isolation unit (Grass-Telefactor, West Warwick, RI) was used in the application of the superimposed bursts.

Additionally, to quantify alterations in muscle activity prior to the burst, surface electromyographical electrodes (EMG, Desktop DTS, myoMuscle, Scottsdale AZ, USA) were secured over the muscle bellies of the vastus lateralis and medialis according to the technique described by Delagi et al. [17] and were sampled (1500Hz) and synchronized (Analog Input System, Noraxon Inc. Scottsdale AZ, USA) with the torque from the dynamometer. EMG data

data was collected from the onset of muscle contraction was defined at 7.5Nm [18] to assure that baseline movement had been exceeded until 100 msec prior to the superimposed burst.

Hoffmann reflex testing

To measure difference in spinal-reflexive excitability H-Reflex testing [9] was performed using an electromyography (EMG) and stimulation unit (STM100A, BIOPAC Systems, Inc.). Participants were positioned supine on a portable treatment table with their arms placed at their side, head resting in neutral on a pillow, and knees bent in about 10-15 degrees of flexion and supported by a half bolster. Two 10mm, pre-gelled Ag-AgCl EMG electrodes (EL503, BIOPAC Systems, Inc.) were placed 1.75 cm apart in cleaned and shaven sites over the medial vastus medialis muscle belly. EMG signals were band-pass filtered from 10 to 50 Hz and collected at 1024HZ with a common-mode-rejection ratio of 110 dB. A 2mm shielded disc stimulating electrode (EL2524S, BIOPAC Systems Inc) was positioned over the femoral nerve and secured with hypoallergenic tape and a 7x13cm self adhesive electrode was positioned over the hamstring and used as a dispersive electrode. A 1ms square wave stimulus was produced using the BIOPAC stimulator module and a 200 volt maximum stimulus adaptor (STMISOC, BIOPAC Systems Inc.) and was delivered to the femoral nerve.

The participants maintained constant head, eye, and hand position during testing by focusing on a small circle on the ceiling. Once each participant was settled, the stimulus was applied in 2 volt increments until a maximal H-Reflex was obtained. After the first maximal reflex was obtained, three more were elicited using the same voltage setting. Next, the voltage was increased until a maximal muscle response was obtained, and followed by three concurrent trials of eliciting maximal muscle response. The average of the three maximal H-Reflexes were

normalized to the average of the three maximal muscle responses for analysis. This procedure was then repeated for the contralateral limb and the order of tested limbs was counterbalanced.

Sonographic Assessment

Sonographic measurements were taken using an ultrasound unit (Philips, 12-5L linear transducer). Participants were supine with the knee flexed between 80-90 degrees in order to relax the quadriceps muscles. [19] A clinician trained in the use of US for studying patellar tendon abnormalities obtained both longitudinal and transverse images of the patellar tendon. The abnormalities studied were tendon thickening, hypochoic regions, and areas of neovascularization. Tendons with one or more abnormality were considered to be objectively defined PT. Abnormality was used to break the PT group into two subgroups, where participants with tendon abnormality were defined as objective PT and subjects without abnormality were defined as subjective PT. These images were transferred to an external had drive so that could be examined later for structural abnormalities. The US examiner was blinded to participant groupings.

Movement Assessment

Following the CAR trials, participants completed at least one familiarization trial followed by three trials of a standardized jump-landing task. Participants were required to jump forward a distance of half their height from a 30cm high box and land with each foot on a separate non-conductive force plate (model 4060-NC; Bertec Corporation, Columbus, OH), which sampled landing data at 1500 Hz. Immediately following landing, participants jumped for maximal height in one fluid motion. Trials were repeated if the participants' feet did not leave the box at the same time, the participants did not immediately jump after landing, or if they

jumped too far or too short. Each test was recorded by two video cameras, one in the sagittal plane and one in the frontal plane. A single rater, blinded to group, evaluated the trials using the Landing Error Scoring System (LESS), which is a valid and reliable clinical movement screening tool. [20] The EMG system and electrode placement was not changed between CAR and jump landing trials, but the system was synced with both force plates so that muscle activity could be aligned in time during jump landing.

Data Reduction and Analyses

EMG data collected during isokinetic testing and jump landings were analyzed to determine if differences in the average muscle activity during these tasks existed in participants with PT. To do this, the raw EMG data were subsequently processed within the Noraxon software (MR3, myoMuscle, Scottsdale Arizona, USA). Specifically, raw EMG signals were band-pass filtered 6 to 1,000 Hz, rectified and then processed using a root-mean-square algorithm with a 50-millisecond moving window. EMG collected during the isokinetic and jump landing tasks were then normalized to the peak muscle activity that was recorded from either task. Using this normalization technique, all normalized root-mean-square data were at or below 100% of muscle activity. [21] EMG data for the isokinetic testing were analyzed between the onset of muscle contraction to 100msec prior to the burst. EMG during the landing task was then analyzed during two phases of activity: pre-loading (100 milliseconds prior to ground contact) and loading (ground contract to take-off) [22]. Ground contact was defined as a force exceeding 10 Nm, while take off was defined as a force below 10 Nm [22].

Descriptive statistics are presented as means +/- SD. Statistical analyses were performed using SPSS (Version 22). Independent t-tests were used to assess group differences between PT and control participants for CAR, H:M ratio, LESS score, and average muscle amplitude during

both CAR, and the pre-loading and loading phases of jump landing. Assumptions of equal variance and normal distribution were ensured with no abnormalities. Descriptive statistics were used to discuss differences in sonographic assessment. Significance was set *a priori* at an alpha level of < 0.05 and 95% confidence interval (CI), reported as lower bound (LB) and upper bound (UB), around the mean differences was calculated. Each dependent variable was considered independent constructs thus Family Wise Error was not accounted in this analysis.

Chapter IV: Results

Demographics

No significant difference in age, gender, or mass existed between groups (see table 1, $p>0.05$). The PT group was found to be significantly taller than the healthy group (see table 1, $p<0.05$).

Table 1: Group Demographics (Mean ± SD)

Demographic	Group	n	Mean	95% CI: LB	95% CI: UB
Age (years)	PT	6	20.83±0.98	-5.59	1.59
	Healthy	6	22.83±3.82		
Gender	PT	6	0.50±0.55	-0.71	0.71
	Healthy	6	0.50±0.55		
Height (cm)	PT	6	177.80±6.81*	0.32	14.87
	Healthy	6	170.20±4.18		
Weight (kg)	PT	6	74.00±8.67	-7.21	15.87
	Healthy	6	69.67±9.27		

*Indicates significance ($p<0.05$)
95% CI indicates difference between groups

VISA-P

All subjects in the PT group scored under an 80 on the VISA-P (62.67 ± 17.38), while all healthy subjects scored at or above a 99 (99.83 ± .41). Both groups met the criteria defined by the VISA-P for their respective groups.

US

Two of the six subjects in the PT group were classified as having PT through diagnostic imaging, with one of the 2 being diagnosed with bilateral tendon involevment. The remaining four subjects had no visible PT present in the affected limb (see Figure 1). All six of the healthy subjects were shown to have no visual evidence of PT in either limb.

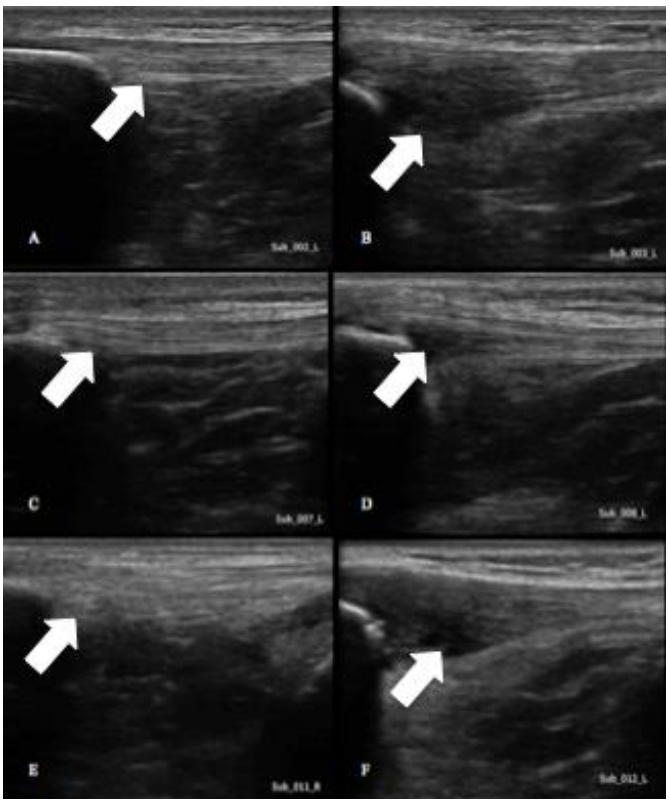


Figure 1. Demonstrates the tendons from the involved limbs for the PT group. A, C, D, and E refer to participants with subjectively reported PT, normal tendons (Denoted by arrows). Tendons B and F refer to diagnostic (objective) PT as shown by their increased width and areas of hypochoic legions (Denoted by arrows).

CAR

I observed a significant deficit in volitional muscle activation in the involved limb in patients with patellar tendinopathy as compared to the healthy group ($F = 10.279$; $p = .04$; Cohen $d = 1.37$; 95% CI: -6.60, -0.21, see table 2).

Table 2. Central Activation Ratio (Mean ± SD)

Limb	Group	n	Mean	95% CI: LB	95% CI: UB
Involved	PT	6	95.13±3.46*	-6.60	-0.21
	Healthy	6	98.54±0.60		
Non-Involved	PT	6	96.97±2.21	-3.26	1.25
	Healthy	6	97.98±1.12		

*Indicates significance (p<0.05)
95% CI indicates difference between groups

Muscle Activity

No significant differences were found between the PT and healthy groups for vastus medialis or vastus lateralis activity during SIB, pre-loading, or loading (see tables 3-5, $p>0.05$).

Table 3. EMG: Percent maximal activation during CAR (Mean ± SD)

Limb: Muscle	Group	n	Mean	95% CI: LB	95% CI: UB
Involved: Vastus Lateralis	PT	6	15.67±19.89	-27.85	14.37
	Healthy	6	22.36±11.97		
Involved: Vastus Medialis	PT	6	23.32±15.14	-15.92	20.26
	Healthy	6	21.15±12.89		
Non-Involved: Vastus Lateralis	PT	6	20.43±20.78	-19.09	26.66
	Healthy	6	16.65±14.15		
Non-Involved: Vastus Medialis	PT	6	35.15±29.62	-6.06	49.45
	Healthy	6	13.46±7.32		

*Indicates significance (p<0.05)
95% CI indicates difference between groups

Table 4. EMG: Percent maximal activation during pre-loading (Mean ± SD)

Limb: Muscle	Group	n	Mean	95% CI:LB	95% CI: UB
Involved: Vastus Lateralis	PT	6	7.93±5.34	-7.53	4.49
	Healthy	6	9.45±3.90		
Involved: Vastus Medialis	PT	6	9.06±3.29	-5.25	5.34
	Healthy	6	9.01±4.80		
Non-Involved: Vastus Lateralis	PT	6	8.56±4.24	-20.81	11.13
	Healthy	6	13.40±17.03		
Non-Involved: Vastus Lateralis	PT	6	9.46±4.56	-8.03	6.53
	Healthy	6	10.21±6.57		

*Indicates significance (p<0.05)
95% CI indicates difference between groups

Table 5. EMG: Percent maximal activation during loading (Mean ± SD)

Limb: Muscle	Group	n	Mean	95% CI: LB	95% CI: UB
Involved: Vastus Lateralis	PT	6	29.23±9.83	-28.51	2.22
	Healthy	6	42.37±14.05		
Involved: Vastus Medialis	PT	6	43.17±6.79	-8.09	24.56
	Healthy	6	34.93±16.61		
Non-Involved: Vastus Lateralis	PT	6	31.56±11.13	-8.99	19.64
	Healthy	6	26.23±11.13		
Non-Involved: Vastus Lateralis	PT	6	38.00±15.33	-9.80	27.87
	Healthy	6	28.97±13.92		

*Indicates significance (p<0.05)
95% CI indicates difference between groups

Spinal Reflexive

No significant difference was found between the PT and healthy groups in H:M ratio (F = 2.665; p = 0.82; 95% CI: -0.20, 0.25, see table 6).

Table 6. H:M Ratio (Mean ± SD)

Limb	Group	n	Mean	95% CI: LB	95% CI: UB
Involved	PT	6	0.22±0.23	-.20	.25
	Healthy	6	0.20±.09		
Non-Involved	PT	6	0.30±.22	-.56	.22
	Healthy	4	0.47±.32		

*Indicates significance (p<0.05)
95% CI indicates difference between groups

LESS Score

No significant difference was found between the PT and healthy groups in average LESS score (F = .260; p = .66; 95% CI: -1.51, 2.29, see table 7).

Table 7. Average LESS Score (Mean ± SD)

LESS Score	Group	n	Mean	95% CI: LB	95% CI: UB
Average	PT	6	4.94±1.64	-1.51	2.29
	Healthy	6	4.56±1.29		

*Indicates significance (p<0.05)
95% CI indicates difference between groups

Sub-Group Analysis

To more accurately report CAR, H:M, muscle activity, and LESS the PT group was divided into two sub-groups in addition to the PT group as a whole. First, a Subjective group, diagnosed with PT based off subjective questionnaires, and second, an Objective group, diagnosed with PT based off a combination of subjective reports and positive identification of tendon abnormality via US, groups. The purpose of this division was to identify the differences that appeared between subjects with and without tendon abnormality and to demonstrate varying levels of severity within the PT group. All sub-group analyses were done descriptively due to the small sample sizes.

Sub-Group CAR

The objective PT group demonstrated a lower CAR than both the subjective PT and healthy groups (see table 8).

Table 8. Central Activation Ratio with sub-groups (Mean ± SD)

Limb	Group	n	Mean
Involved	Objective PT	2	93.66±0.82
	Subjective PT	4	99.00±0.62
	Healthy	6	98.54±0.60
Non-Involved	Objective PT	2	94.91±2.76
	Subjective PT	4	98.00±1.15
	Healthy	6	97.98±1.12

Sub-Group EMG

The objective PT group exhibited lower vastus lateralis and medialis activation during SIB, pre-loading, and loading trials in both the involved and non-involved limbs when compared to both the subjective PT and healthy groups (see tables 9-11)

Table 9. EMG: Percent maximal activation during CAR with sub-groups (Mean ± SD)

Limb: Muscle	Group	n	Mean
Vastus Lateralis: Involved	Objective PT	2	4.57±2.84
	Subjective PT	4	21.14±23.11
	Healthy	6	22.36±11.97
Vastus Medialis: Involved	Objective PT	2	13.03±4.24
	Subjective PT	4	28.47±16.43
	Healthy	6	21.15±12.89
Vastus Lateralis:Non- Involved	Objective PT	2	8.44±7.44
	Subjective PT	4	26.42±23.62
	Healthy	6	16.65±14.15
Vastus Medialis: Non-Involved	Objective PT	2	7.46±1.13
	Subjective PT	4	49.00±26.36
	Healthy	6	13.46±7.32

Table 10. EMG: Percent maximal activation during pre-loading with sub-groups (Mean ± SD)

Limb: Muscle	Group	n	Mean
Vastus Lateralis: Involved	Objective PT	2	2.95±1.97
	Subjective PT	4	10.43±4.61
	Healthy	6	9.45±3.90
Vastus Medialis: Involved	Objective PT	2	6.05±2.45
	Subjective PT	4	10.56±2.64
	Healthy	6	9.01±4.80
Vastus Lateralis: Non-Involved	Objective PT	2	6.15±4.91
	Subjective PT	4	9.77±4.02
	Healthy	6	13.40±17.03
Vastus Lateralis: Non-Involved	Objective PT	2	6.64±2.08
	Subjective PT	4	10.88±5.03
	Healthy	6	10.21±6.57

Table 11. EMG: Percent maximal activation during loading with sub-groups (Mean ± SD)

Limb: Muscle	Group	n	Mean
Vastus Lateralis: Involved	Objective PT	2	18.70±2.92
	Subjective PT	4	34.49±5.75
	Healthy	6	42.37±14.05
Vastus Medialis: Involved	Objective PT	2	38.62±4.69
	Subjective PT	4	45.44±6.98
	Healthy	6	34.93±16.61
Vastus Lateralis: Non-Involved	Objective PT	2	20.83±0.189
	Subjective PT	4	36.92±9.56
	Healthy	6	26.23±11.13
Vastus Lateralis: Non-Involved	Objective PT	2	22.47±2.03
	Subjective PT	4	45.77±12.21
	Healthy	6	28.97±13.92

Sub-Group Spinal Reflexive

The objective PT group was found to demonstrate a lower H:M ratio in both the involved and non-involved limbs when compared to both the subjective PT and healthy groups (see table 11).

Table 12. H:M Ratio with sub-groups (Mean ± SD)

Limb	Group	n	Mean
Involved	Objective PT	2	0.07±0.05
	Subjective PT	4	0.29±0.26
	Healthy	6	0.20±0.09
Non-Involved	Objective PT	2	0.14±0.04
	Subjective PT	4	0.38±0.24
	Healthy	4	0.47±0.32

Sub-Group LESS Score

The objective PT group were found to higher LESS scores than the subjective PT and healthy groups (see table 13).

Table 13. Average LESS Score with sub-groups (Mean ± SD)

LESS Score	Group	n	Mean
Average	Objective PT	2	6.67±0.47
	Subjective PT	4	4.08±1.20
	Healthy	6	4.56±1.29

Chapter V: Discussion

This research study was the first to quantify AMI originating from peripheral sources in the PT population. Prior research has evaluated cortically driven neuromuscular changes in chronic PT cases [12] and deficits in strength exhibited by PT subjects [3] but neither previous study

defined the amount of quadriceps inhibition present in the condition. The current study found evidence of the presence of AMI resulting from peripheral mechanisms at the spinal cord level in subjects with PT. Further, it was found that those subjects demonstrating objective PT, as confirmed by US, also demonstrated alterations in muscle activity and movement control, possibly resulting from the increased tendon abnormality.

Identifying Patellar Tendinopathy

The VISA-P is a means of determining the severity of PT based on subjective patient reports of pain and dysfunction. From the results of this study, it is reasonable to suggest that the VISA-P can also detect neuromuscular dysfunction since the PT group demonstrated neural alterations, but further investigation would be needed. All subjects met their group's VISA-P inclusion criteria and were placed in the appropriate group. Although, there was variability within the PT group as scores ranged from 30-79 respectively. This wide range demonstrated the need for objective testing of tendon injury severity in addition to solely subjective reports of pain.

As the abnormality and pain do not always occur together it was expected that there would be discrepancies between subjective and objective reporting, which has been previously demonstrated. [19,23] Only two of the six subjects in the PT group had tendon abnormalities upon investigation by diagnostic US imaging (Fig. 1), while the remaining four PT participants did not show any evidence of PT through imaging. This is important clinically because individually both subjective and objective reporting measures can be used to detect PT, but combining both measures may be the most cost effective and efficient method.

Quantifying Arthogenic Muscle Inhibition

This study was the first to quantify AMI in PT patients using both CAR and H-Reflex. Together these techniques allow for the identification of altered volitional muscle activation, which is originating either from a decrease in motor neuron firing rate or the number of available motor units, and an evaluation of the quality of afferent signaling from the muscle spindle reaching the spinal cord. This is important because simply identifying the presence of AMI does not describe the underlying source or mechanism. When looking at CAR, the PT group demonstrated a decrease in volitional muscle activation as compared to the healthy group (Table 2). In particular, those individuals with tendon abnormalities on diagnostic imaging showed an even larger decrease in volitional muscle activation when compared to both the remainder of the PT group and healthy individuals (Table 8). These differences in CAR within the PT group may have been caused by variations in the origin of AMI. Based on the data it appeared that the subjective PT group may have been suffering from centrally driven AMI due to the decrease in volitional muscle activation even with a lack of detectable peripheral mechanisms. Conversely, the objective group may have been suffering from a combination of peripheral and central mechanisms of AMI due to the chronic nature of their injuries. This thinking is derived from previous literature showing chronic PT can cause cortical level changes [12],and my data that demonstrated a decrease in H:M ratio. The observed decrease in volitional muscle activation, for either group, did not reach levels seen in more serious injuries, like ACL rupture, which was expected. PT does not exhibit similar levels of joint damage, swelling, or pain as are seen in major ligament rupture. A second reason could be that even the two subjects with tendon abnormality and a decreased CAR were still fully functional in sport.

Unlike CAR, no difference in H:M appeared between the PT group and healthy individuals (Table 6). Although, once the PT group was through US the objective PT group demonstrated a lower H:M in both the involved and uninvolved limbs (Table 12). These data suggest that the spinal cord is modifying afferent signaling from the periphery and could be a result of dysfunction in the gamma loop, Renshaw cells or type 1a. Spinally induced AMI is not caused by peripheral mechanisms, such as pain or effusion, which can be modulated by reducing the amount of afferent signaling. In these peripheral models AMI can be reduced by simply decreasing the amount of joint effusion or by reducing the amount of pain felt. This could be why there was a difference between the two PT sub-groups in regards to H:M. The objective group was likely suffering from peripherally driven AMI due to altered afferent feedback resulting from tendon abnormality, while in both sub-groups centrally driven AMI resulting from their chronic PT was likely caused a reduction in CAR as compared to the healthy group.

Functional Impact

As was previously reported, the results of this study show that PT can lead to AMI that is likely originating from peripheral and central mechanisms, but neither CAR nor H-Reflex is a measure of functionality during physical activity. It is important to understand how AMI in the PT population translates into alterations in muscular activity and neuromuscular control during functional activities.

Neuromuscular control during functional activities, such as a jump landing, is important to evaluate due to its association with injury risk. [24] Previous work has linked AMI to functional deficits in neuromuscular control that may explain the elevated risk of subsequent injury in individuals after knee joint injury, but this work has been largely isolated to patients after ACL injury. Palmeiri-Smith et al [25] found that patients with AMI generally demonstrate altered

kinematics, but this research has not been thoroughly reproduced in the PT population. This is one of the first studies to score jump landings in the PT population. While there were no statistically significant differences between groups in LESS scores (Table 7) the objective PT group demonstrated higher LESS scores than either other group (Table 13). Furthermore, the mean scores for the objective PT group were above six, which has been shown to be predictive of ACL injury in a youth population [26] and may suggest greater neuromuscular compromise in these individuals. These findings support the critical need for clinicians to evaluate movement control in patients recovering from knee joint injuries.

One possibility for the objective PT group's increased scores was altered muscular activity prior to and during landing. The objective PT group had generalized decreased activity in the vastus medialis and lateralis that was not seen in the either the subjective or healthy control groups and a lower percent max activation during the loading phase of jump landing in the vastus lateralis. Conversely, the PT group demonstrated activation that was greater than or equal to healthy individuals in all other trials (Table 5), but these data are not meant to suggest that individuals with PT demonstrate normal levels of quadriceps activation. Once the PT group was separated into subgroups it became apparent that those with diagnostically identified tendon abnormality demonstrated decreased muscle activation during the SIB trials (Table 9) and the pre-loading (Table 10) and loading phases (Table 11) of jump landing for both the vastus lateralis and medialis muscle groups. This suggests that the objective PT group might have been compensating with higher activation in a different muscle group that was not quantified in this study through the use of EMG, or the group had an overall decrease in quadriceps muscle activation, even on muscles not analyzed. The latter is supported through the objective PT group's decrease in CAR and increased LESS score. This could be evidence that AMI

originating from central mechanisms can have an effect on muscular activity that is significant enough to alter landing kinematics. Further investigation is warranted into the source of this decreased activity and whether PT induced AMI is associated with injury risk. Clinically, landing mechanics may be an area of observation that is added to PT evaluations.

Limitations

Overall, the limitations of this study were disparity between levels of dysfunction in the PT group and its small sample size. One possibility that could explain why not all subjects demonstrated neuromuscular dysfunction could be due to the disparities in the origin of the PT. Peripheral mechanisms were shown to be affected in the two objective PT subjects through their decreased H:M. Other subjects were not as acutely injured, which may explain why they did not have alterations in H-Reflex outcomes. For these subjects cortical measures may have been more appropriate to assess neuromuscular alterations. Cortical alterations have been demonstrated in various ACL injury models [27,28] and previous literature suggests that chronic PT can cause changes in cortical pathways, which caused neuromuscular dysfunction. [12] Further, research into long term injury outcomes in subjects with previous ACL injury has shown that these cortical alterations can evolve over time as a response to injury. [9] A larger sample of more severely injured participants would be needed to accurately assess the amount of dysfunction potentially caused by PT.

Future Direction

Future research needs to evaluate the effects of PT on neuromuscular function in a larger sample consisting of acute and chronic PT of varying injury level. Further, this research should aim to evaluate both peripheral and cortical pathways over time to see how the course of the

injury may cause changes in neuromuscular outcomes. Finally, a protocol that includes a treatment program would be necessary to observe how both peripheral and cortical pathways evolve as tendon health improves. This rehabilitation plan should include ways to treat the various underlying causes of AMI present in PT cases.

Conclusions

PT can lead to neuromuscular dysfunction in the quadriceps during varying levels of activity. Individuals with PT exhibit quadriceps AMI that seems to partially stem from peripheral inhibition at the spinal cord level. These individuals with objectively quantified PT also demonstrate lower muscle activity during voluntary isometric contractions and the phases of jump landing, which may be the source of their increased LESS scores. The topic of quadriceps AMI caused by PT is novel and future research is needed to account for the small sample size and relatively healthy PT population. An investigation into PT detrimental enough to cause a cessation of athletic participation is needed to determine to what extent PT can influence neuromuscular dysfunction and the origin of AMI in this population. Clinically, this research presents data that subjects with PT could present with AMI of varying levels corresponding with the amount of tendon abnormality. Rehabilitation practices should focus on not only the tendon itself but also the quadriceps musculature, which are commonly used to combat AMI seen after ligamentous injury. [7,29-31]

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