Novel Approaches to Measurement of Passive Torque and Stiffness in the Hamstrings

By

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ABSTRACT

Recent evidence related to passive force enhancement (PFE, Herzog & Leonard, 2002) and the calculation of passive stiffness (Nordez et al., 2006) show that the measurement of passive torque may need to be revisited. The purpose of this study was to compare a novel assessment of passive resistance to the traditional assessment. Mathematical models were used to determine model goodness of fit between experimental and traditional conditions and these models were used to predict maximal passive stiffness (MPS).

Peak passive resistance was significantly lower ($p < .001$) after a 1-minute 3-minute, and 5-minute rest period and resetting to resting length in comparison to the traditional measurement. Among the experimental conditions, 1-minute was significantly lower than 3-minutes of rest and resetting to resting length ($p = .039$), while the other comparisons were not significantly different. These results show that passive resistance is lower in the novel assessment that considers PFE compared to the traditional methodology.

In the second-order polynomial model, root mean squared error (RMSE) was significantly lower ($p = .011$) after a 1-minute rest period, a 3-minute rest period ($p = 0.23$), and a 5-minute rest period and resetting to resting length ($p = .028$), compared to the traditional measurement. In the fourth-order polynomial model, RMSE was significantly lower ($p < .001$) after a 1-minute, a 3-minute ($p = .002$), and a 5-minute rest period and resetting to resting length ($p = .004$), compared to the traditional measurement. In the exponential model, RMSE was significantly lower ($p < .001$) after a 1-minute, a 3-minute ($p < .001$), and a 5-minute rest period and resetting to resting length

($p < .001$), compared to the traditional measurement. Comparisons among the experimental conditions in each model were not significantly different.

When observed MPS was compared to predicted MPS, all comparisons among each testing condition and each mathematical model were significantly different ($p < .001$). These results show that PFE is likely to not be involved in the experimental conditions as they show a better fit to each mathematical model. Further, no one model appeared to predict MPS better than the others.

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CHAPTER I: DISSERTATION INTRODUCTION

Muscle force production can be classified as active or passive. Active movements involve nerve impulses that stimulate excitation-contraction coupling, while passive force generation occurs through the lengthening of structural elastic elements within and around the muscle (Freundt & Linke 2019). In clinical settings the measurement of passive force is generally converted to torque, as movements occur about an axis within the body. The use of passive torque is a standard outcome in studies using flexibility interventions. In such investigations, electromyography (EMG) is used to ensure the muscle being tested is in a passive state, denoted by minimal or negligible muscle activity (Gajdosik et al., 1999b; Gajdosik et al., 1990; Gajdosik et al., 1999a). Further, passive torque can be used to calculate stiffness (Nordez et al., 2006). Though there have been several mathematical models used in such calculations, no single model being shown to be superior to the others (Nordez et al., 2006).

However, with the emergence of new evidence surrounding the passive elements within muscle, the methodologies used to measure passive torque, and then calculate stiffness may need to be revisited. Specifically, the Winding Filament Hypothesis suggests that the elastic myofilament titin may have a greater contribution to passive force production than previously thought (Nishikawa et al., 2012). This contribution has been shown to be long lasting (>25 seconds, Herzog & Leonard, 2002), will persist after muscular deactivation (Joumaa et al., 2007), and until the muscle is returning to its resting length (Herzog & Leonard, 2002). To the author's knowledge, there does not appear to be a methodology of assessing passive torque that adequately accounts for the contribution of titin during passive movements. Further, if a new methodology is utilized

and is shown to result in better passive torque values, then it would be appropriate to revisit the mathematical models used to calculate stiffness.

Title: Novel Approach to Measurement of Passive Resistance in the Hamstrings

Purpose of Study 1

The purpose of the first study is to compare a methodology that is traditionally used to assess passive torque to a novel methodology that considers the role of passive force enhancement within muscle.

Research Questions for Study 1

1. What is the difference in passive resistance values between the traditional method of assessment and the novel methodology?
2. What is the difference in passive resistance values among the experimental conditions, as it relates to rest time?

Delimitations

1. The study will target healthy male participants.
2. Participants will be excluded from the study if they had a lower limb injury in the past three months, and if they are currently ill.
3. Extension movement speed will be controlled at 5°/sec.
4. EMG activity will be continuously monitored to ensure the hamstrings are at a resting state.
5. Experimental conditions will be randomized among each participant, for each of the two trials that will be collected.
6. Each participant will perform two trials at each condition.

Limitations

1. The participants were only male.
2. There was a small sample size (n = 10) and trials per condition (number of trials = 2) used.
3. The rationale for the study was theoretical, and specific alterations to titin were not able to be measured with the current methodology.

Basic Assumptions

1. Participants will remain relaxed and in a resting muscular state during the entire data collection process.

Significance of Study 1

This study will investigate a novel methodology for assessing passive torque in the hamstrings. This study will help better understand the passive component of muscle as it relates to force production. Practitioners and researchers will be able to replicate this new methodology with the traditional method to elucidate any differences.

Title: Calculation of Passive Torque and Stiffness Using Different Mathematical Models

Purpose of Study 2

The purpose of the second study is to fit multiple mathematical models to passive torque-angle data to elucidate whether the nature of the passive torque curve is different in each of the experimental situations.

Research Questions for Study 2

1. Which mathematical model shows the best fit to the experimental data?
2. What is the difference in passive stiffness calculations among the varying mathematical models?

Delimitations

1. Three mathematical models that have been used in the literature will be used in the current study to identify the best model fit.
2. Sum of squares due to error (SEE) and root mean squared error (RMSE) will be used to measure model fit.

Limitations

1. There was a small sample size (n = 10) and trials per condition (number of trials = 2) used.
2. The second order polynomial model and the exponential model have not been used in passive conditions in clinical settings.
3. The data points used were observed and not extrapolated, reducing the amount of data points used in each mathematical model prediction.
4. There is no clear parameter used to indicate the validity of a specific model.

Basic Assumptions

1. SEE and RMSE will correctly measure goodness of fit of each mathematical model to the experimental data.

Significance of Study 2

This study will investigate the use of different mathematical models in calculating passive stiffness from torque-angle data. This will provide practitioners and researchers with insight into which model would be best suited to calculating passive stiffness near the end range of motion.

CHAPTER II: REVIEW OF LITERATURE

Muscle Structure

The muscle-tendon unit is the anatomical structure that permits movement of the body (Gajdosik, 2001). The muscular system can be divided into the following levels of organization: organ (whole muscle), tissue (fascicles), cellular (myofibers), microscopic (myofibrils and sarcomeres), and molecular (actin and myosin). Within the molecular level there are also non-contractile elements that are involved in the production of movement. Further, there is connective tissue that surrounds and connects these levels. The epimysium is the outermost layer of connective tissue that surrounds the entire muscle (Powers et al., 2021). Beneath the epimysium is the perimysium, which separates bundles of muscle fibers (Powers et al., 2021). Even deeper is the endomysium, that surrounds each individual muscle fiber in the bundle (Powers et al., 2021). Just under the endomysium is a layer of connective tissue called the basement membrane (Powers et al., 2021). While the classification of these divisions seems to imply clear separation of these structures, it needs to be noted that they are all interconnected. The endomysium also connects adjacent muscle fibers, while joining with the perimysium (Borg & Caulfield, 1980; Rowe, 1981). The perimysium connects to the epimysium, that encapsulates the entire muscle (Borg & Caulfield, 1980; Rowe, 1981). The muscle belly consists of sarcomeres which shorten, producing movement of the skeletal system. During passive lengthening, the muscle belly is the primary contributor to force production across the length-tension curve (Stolov & Weilepp, 1966; Halar et al., 1978; Tardieu et al., 1982).

Passive Force

There are several structures within the muscle that contribute to passive tension. These include stretching links between actin and myosin, stretching non-contractile proteins, and connective tissues surrounding the muscle, with the first two constituting the series elastic component (SEC), and the third composing the parallel elastic component (PEC) (Gajdosik, 2001). Passive tension has been attributed to the level of stiffness in the PEC of muscle and these forces increase exponentially as the muscle lengthens (McHugh et al., 1998). Borg and Caulfield (1980) noted that the perimysium is likely the most important connective tissue during passive resistance due to its abundance and arrangement. Specifically, McHugh et al. (1998) mentioned that 79% of the variance in hip flexion ROM could be explained by the resistance in the PEC of the hamstrings.

These non-contractile filamentous components of the PEC can be divided into two subunits, the endosarcometric and exosarcometric cytoskeletons (Gajdosik, 2001). The primary structure within the endosarcometric cytoskeleton to resist passive lengthening is titin (Wang et al., 1993), which attaches from the M line region to the Z disc of the sarcomere (Gajdosik, 2001). The primary structure of the exosarcometric cytoskeleton is desmin (Wang & Ramirez-Mitchell, 1983), which connects Z discs horizontally and connects Z discs to organelles, apart from the t-tubules (Tokuyasu et al., 1983). Desmin is considered part of the exosarcometric cytoskeleton as it also connects Z discs to one another outside of the sarcomere (Wang et al., 1993; Wang & Ramirez-Mitchell, 1983).

Another noncontractile protein with multiple roles is nebulin. One of the primary roles of nebulin is regulating the length of actin. Nebulin deficient mice have been shown to die within two weeks of birth due to muscle weakness, that was linked to a reduction in

the length of actin (Bang et al., 2006; Witt et al., 2006). In a clinical human trial, an individual expressing a nebulin mutation also showed shorter actin lengths (Ottenheijm et al., 2009). Additionally, nebulin is involved in the regulation process of muscle contraction by assisting in sarcoplasmic reticulum calcium handling (Ottenheijm et al., 2008) and increasing actin-myosin binding (Bang et al., 2009; Chandra et al., 2009; Ochala et al., 2011; Ottenheijm & Granzier, 2010). Nebulin also assists desmin in the regulation of Z disc structure and maintaining the lateral position of the sarcomere (Bang et al., 2006; Tonino et al., 2010).

Muscle Contraction

Historical Perspective

Before 1950, muscle contraction and force production were explained by alterations undergone by myosin (Herzog, 2014b). It was thought that upon muscle activation via calcium release in the sarcoplasm; that the structure of myosin transformed from a helical to a coil-like structure, thus shortening the sarcomere, and producing force (Herzog, 2014b). However, in 1954, Andrew and Hugh Huxley independently suggested that myosin did not shorten, but that myosin and actin slid past one another, a process termed the sliding-filament theory (Huxley & Niedergerke, 1954; Huxley & Hanson, 1954). As such, it was suggested that myosin contained projections, termed cross-bridges, that interacted with and pulled actin past myosin. This addition to the sliding-filament theory was called the cross-bridge theory (Huxley, 1957). Though there were several modifications made to the model in a few short years (Huxley, 1969; Huxley & Simmons, 1971), it has since been used to explain muscle contraction. While the cross-bridge theory explains concentric and isometric force production well, this model

inadequately explains force production during eccentric contraction (Pollack, 1990; Woledge et al., 1985). As such, Tom McMahom referred to eccentric muscle contractions as the “dark side of the force-velocity curve” (Lindstedt et al., 2001).

Hypothesized Mechanisms of Eccentric Muscle Contraction

Eccentric muscle contractions have several distinct characteristics, including the ability to generate high forces with minimal energy requirements and the presence of residual force enhancement, an elevated steady-state force after lengthening compared to a purely isometric contraction (Edman et al., 1982). The high force production of eccentric contractions at low energy cost was first depicted in 1952. During an experiment, two stationary bikes were positioned opposite one another, sharing a single chain. A larger male participant pedaled in one direction, performing concentric contractions, while a smaller female resisted the pedaling in the opposite direction, resulting in eccentric contractions. It was observed that the smaller participant was able to generate higher forces than the larger participant, and at a much lower oxygen consumption (Abbot et al., 1952). According to the National Strength and Conditioning Association (NSCA), the SEC, specifically the tendon, plays the predominant role in force production following an eccentric action (Haff & Triplett, 2015). However, evidence is mounting that the PEC, composed of the structures previously mentioned, is responsible for the increased force production.

Residual force enhancement is the phenomena in which isometric force is greater following active lengthening of a muscle, compared to forces produced at the same length before stretching (Edman et al., 1982), and has been shown to occur at all levels of the muscular structure (Herzog et al., 2012). There have been several explanations for

residual force enhancement following eccentric contraction, including increased force production by cross-bridges, sarcomere length non-uniformities, and the use of elastic components within the muscle, upon activation. However, the first two rationales have lacking and contradicting support, while the third has compelling evidence for its use.

Increased Cross-Bridge Force

Measurement of cross-bridge force production is a task that has yet to be accomplished and is unlikely to be accomplished in the near future (Hessel et al., 2017). Thus, indirect measures must be made via alterations in stiffness compared to force (Herzog et al., 2014a). The cross-bridge theory suggests that eccentric force production can be explained by an increase in the number of attached cross-bridges or by an increase in the force produced by each cross-bridge, or both (Herzog, 2014b). If there is an increase in the number of attached cross-bridges, then there should be a corresponding increase in muscle stiffness (Ford et al., 1981). However, muscle stiffness has been shown to be either minimally increased or not increased at all in the active lengthening state compared to a reference isometric contraction (Sugi & Tsuchiya, 1988; Rassier & Herzog, 2005). Further, myofibrils have been shown to experience force enhancement lasting minutes (Leonard et al., 2010). In this instance, if the increased force was explained by an increase in cross-bridge force it would require cross-bridges to remain attached for minutes. Therefore, for the increased force to be explained by an increased cross-bridge force, they must stay attached for minutes, which is unlikely (Herzog, 2014b). for cross-bridges alone to be the explanation, they would need to extend nearly three times their resting length to produce such forces (Leonard et al., 2010).

Sarcomere Non-Uniformity

For sarcomere non-uniformities to account for residual force enhancement, there are several predictions being made: 1) sarcomere lengths will vary during active lengthening, while not varying during an isometric contraction, 2) force enhancement will only be seen on the descending limb of the force-length curve, 3) active lengthening force cannot exceed isometric force, and 4) force enhancement cannot occur in an individual sarcomere. Contrary to these predictions, evidence indicates that sarcomere lengths are most uniform during active lengthening (Edman et al., 1982; Joumaa et al., 2008a), the occurrence of force enhancement during the ascending limb of the force-length curve is well documented (Abbott & Aubert, 1952; Morgan et al., 2000; Peterson et al., 2004), force production can exceed the isometric value at all muscular levels (Abbott & Aubert, 1952; Schachar et al., 2002; Lee & Herzog, 2008; Joumaa et al., 2008a; Leonard et al., 2010), and force enhancement can occur in individual sarcomeres (Leonard et al., 2010).

Elastic Structures

The last explanation available to describe residual force enhancement is the recruitment of elastic components within the muscle, that upon activation, increase in stiffness. This was first suggested in 1976, as single muscle fibers were shown to shorten faster after active lengthening (Edman et al., 1976). In 2002, it was shown that force enhancement continued for several seconds after deactivation when preceded by muscle stretch (Herzog & Leonard, 2002). This was termed passive force enhancement (Herzog & Leonard, 2002) and was seen in individual myofibrils and muscle fibers (Joumaa et al., 2008b; Lee et al., 2007).

Specifically, in cat soleus muscle preparations, passive force enhancement was shown to range from 8.3% to 83.7%, with larger increases seen in response to longer stretch lengths (Herzog & Leonard, 2002). Rassier et al. (2003) showed passive force enhancement to occur in a variety of testing conditions utilizing frog tibialis anterior and lumbrical single muscle fibers. In all rabbit psoas myofibrils tested, Joumaa et al. (2007) showed passive force enhancement to range from 86% to 145% of force produced during passive stretch. Additionally, it should be noted that passive force enhancement persisted until the preparation was shortened to its resting length (Herzog & Leonard, 2002) and was most likely to occur when the initial length was near the muscle fiber's optimum length, corresponding to the plateau region in the force-length relationship (Rassier et al., 2003). To account for this passive enhancement of force, it was suggested that a structural element was responsible (Joumaa et al., 2008a), mainly titin (Linke et al., 1998).

Titin

Introduced in 2012, the winding filament hypothesis introduces a molecular explanation for titin contributing to active force production following muscle lengthening (Nishikawa et al., 2012). In the I-band of a sarcomere, titin is composed of "springs", consisting of proximal and distal tandem Ig domains, separated by a PEVK segment (Linke et al., 1998; Gautel & Goulding, 1996). During passive lengthening at short sarcomere lengths, the Ig domains elongate, resulting in a minimal increase in tension (Wang et al., 1991; Granzier & Labeit, 2004). When the sarcomere is stretched further, the PEVK segment is lengthened, providing a sharp increase in tension (Gautel & Goulding, 1996; Trombitas et al., 1998).

During muscle activation, the N2A region of titin, which separates the proximal and distal tandem Ig domains, binds to actin (Monroy et al., 2011). Specifically, in mice experiencing activated soleus muscle unloading, the force-displacement curve is shifted to shorter sarcomere lengths, with titin being approximately 2.5 times stiffer and 10% shorter, compared to resting soleus muscle (Monroy et al., 2011). However, in mutant mice missing N2A impairment, there was no change in titin characteristics during muscle activation (Monroy et al., 2011).

Joumaa et al. (2008b) investigated the potential interaction effect between calcium and troponin C on passive force production. Specifically, when comparing intact versus myofibrils with troponin C removed, passive forces were the same, indicating that troponin C has little effect on force production in a passive state (Joumaa et al., 2008b). When examining passive force enhancement, the intact fibers showed greater passive force in the presence of calcium, compared to a relaxed state. This force enhancement ranged from 25-30% compared to the force produced during the relaxed state (Joumaa et al., 2008b). After removing titin, force generation fell to ~20% of that seen in intact fibers, and the differences between forces in the passive and activated conditions was removed (Joumaa et al., 2008b). Further, when calcium concentrations were increased, the force curve was shifted upward (Joumaa et al., 2008b). Taken together, these data indicate that during calcium influx, indicating muscle activation, titin binds to actin, resulting in a shorter and stiffer spring mechanism (Nishikawa et al., 2012).

It has been shown that to maintain proper cross-bridge structure, actin must rotate as the myosin head move the thin filament (Morgan, 1977). According to the winding filament hypothesis, in the presence of calcium, while the N2A region of titin is bound to

actin, rotation of the thin filament will cause titin to wind onto actin (Nishikawa et al., 2012). This winding results in the PEVK segment changing length and stiffness, with the formed elastic energy being stored and then used during active shortening (Nishikawa et al., 2012).

Tensile Properties

Passive and active force generation can be differentiated as force generated due to muscle stretching without being activated by nerve impulses and force generated due to excitation-contraction coupling, respectively (Freundt & Linke 2019). During passive movement, muscles exhibit behavior that is reflective of their viscoelastic properties (Taylor et al., 1990), which includes the rate of stretch (viscous) and applied load (elastic). Passive stiffness refers to the viscous property of muscle and is defined as the length-tension relationship of a muscle that is passively moved through a range of motion (Gajdosik, 1997; Chesworth & Vandervoort, 1989) or as the change in passive force, across the change in muscle length (Gajdosik, 2001).

The initial length, or initial passive resistance, of the muscle can be defined as the length at which passive force increases from a negligible value to a detectable one (Gajdosik, 2001). As the muscle is lengthened further, passive force will increase until a maximum passive resistance is reached, which corresponds with the muscle's maximal length (Gajdosik, 2001). To limit the stretch reflex responses, passive stiffness is usually measured with a slow constant stretch (Gajdosik, 2001). Further, and based on structures previously mentioned, passive resistance and passive stiffness are dependent on the mass of muscle tissue (Gajdosik, 2001).

Research attributing this claim has shown that passive compliance, the reciprocal of passive stiffness, is negatively associated to the volume of the arm (Wiegner & Watts, 1986), indicating that as arm volume increases, passive compliance decreases. Further, passive stiffness increases with improvements in muscle strength (Kubo et al., 2002). Specifically, Kubo et al. (2002) found that 8-weeks of isotonic resistance training increased triceps sure muscle volume and stiffness. Additionally, the flexibility index, defined as the passive torque-angle curve from 15° to 25° of dorsiflexion, was significantly higher after the resistance training intervention. It was also noted by the authors that passive torque was higher at all joint angles measured, consisting of 5, 10, 15, 20, and 25°, through a 0° to 25° dorsiflexion ROM (Kubo et al., 2002). As such, it has been suggested that longer and stronger muscle will result in an increased level of passive stiffness (Gajdosik, 2001).

Methodological Considerations

The measurement of passive torque *in vivo* has predominately been used to assess muscular stretch tolerance as part of investigations related to flexibility. Further, such modifications in stretch tolerance are observed through increases in joint range of motion. However, to investigate the material properties of muscle, joint range of motion and speed must be controlled, to limit the contribution of the individual tolerance to a stretch. As such, the following section will present methodological considerations that are important when investigating material muscular properties through measuring passive torque.

Passive Force/Torque

LaRoche and Connolly (2006) constructed a custom device to measure resistance to stretch and maximum voluntary hamstring torque. Participants were positioned supine, and the device utilized a Cybex II isokinetic dynamometer with the resistance arm connected to a load cell, that was used to measure force. The protocol was designed to examine the differences in passive torque in response to static or ballistic stretching programs employed over a four-week period. Though a test re-test design was not used, this assessment device was able to detect significant changes throughout the testing protocol.

Magnusson et al. (1995a) utilized a different assessment technique to measure passive resistance to stretch in the hamstring muscle group. The equipment utilized consisted of a KinCom dynamometer (Kinetic Communicator, Chattecx Corp, Chattanooga, TN, USA) with a modified thigh pad and internal load cell used to measure resistance. To test the reliability of this new technique, Magnusson et al. (1995a) used a test re-test design in which a second stretch was administered 60 minutes after the first. Participants were in a seated position, with their left thigh positioned at 30° from horizontal by resting on the modified thigh pad. The thigh was positioned in this manner to prevent participants from reaching full knee extension during the stretching protocol. Since the end range of motion of the stretch was below complete knee extension, significant contribution of the joint capsule was eliminated (Magnusson et al., 1995a). Therefore, the measured resistance was attributed to the muscle (Magnusson et al., 1995a). Initial and final resistance were found to have correlation coefficients of $r = 0.99$ and $r = 0.98$, with a coefficient of variation of 6.5% and 9.1%, respectively.

Reid and McNair (2011) used a similar methodology when investigating passive torque in older adults with and without osteoarthritis. However, in comparison to the hip angle of 30° used by Magnusson et al. (1995a), Reid and McNair (2011) created a hip angle of 25°. Additionally, knee extension speed was set at 10°/sec for Reid and McNair (2011), compared to 5°/sec for Magnusson et al. (1995a). Further, the starting position for knee extension was set at 80° of knee flexion and maximum voluntary isometric contractions were obtained at 60° of knee flexion for Reid and McNair (2011), though these specifics were not mentioned by Magnusson et al. (1995a). It was noted by Reid and McNair (2011) that the specific methodology used had been shown to be reliable in a previous study (Reid & McNair, 2004). Though it was not noted by either group of authors, the differing sample populations could have affected these differences, with Magnusson et al. (1995a) utilizing young male participants (29.5 ± 4.4 years), and Reid and McNair (2011) utilizing older adults (68.7 ± 4.8 years) with and without knee osteoarthritis.

Due to the viscoelastic properties of human muscle, torque-angle curves during the dynamic loading phase of a stretch (i.e., the eccentric phase of a movement), can be divided into three regions: the initial toe region, a transition period, and a linear portion (Magnusson, 1998). When examining these portions of the eccentric phase before the initiation of a static stretch, Magnusson et al. (Magnusson et al., 1995a; Magnusson et al., 1996b) found the largest coefficient of variation (20-28%) in stiffness and energy to occur in the initial toe region, while the coefficient of variation in the transition and linear phases were much less (5.8% and 14.5%, respectively) (r = 0.91-0.99), between test-retest measurements. For their purposes, Magnusson et al. (Magnusson et al., 1995a;

Magnusson et al., 1996b) defined stiffness and energy as the change in torque across the change in position, and as the area under the curve, respectively. As such, Magnusson (1998) noted that in future studies they would use final stiffness and energy as measures of the passive muscle properties of the dynamic loading phase (eccentric movement).

EMG

In humans, and from a whole muscle perspective, a passive muscle state can be defined as when there is minimal or negligible electromyography (EMG) activity recorded by surface electrodes (Gajdosik et al., 1999b; Gajdosik et al., 1990; Gajdosik et al., 1999a). The end point of a maximal stretch is defined as the point where EMG activity begins to increase, as stretch-induced involuntary muscle activity increases near a muscle's end range of motion (Gajdosik et al., 1999b; Gajdosik et al., 1990). Practically speaking, it is also beneficial to define the end point of a stretch as the point at which the participant feels discomfort or pain. A participant's verbal identification of the end point of a stretch has been used alone (LaRoche & Connolly, 2006; McHugh et al., 1992; Magnusson et al., 1995a; Magnusson et al., 1996b) and in conjunction with increase EMG activity (Gajdosik et al., 1999b; Gajdosik et al., 1990; Gajdosik et al., 1999a). Due to the potential for EMG to change across a passive movement, it can be beneficial to divide the movement, and thus EMG readings, into segments.

As such, Magnusson et al. (1995a) utilized integrated EMG (IEMG) as part of their assessment protocol, as previously mentioned. However, the authors did not specify which hamstring muscle groups were utilized, only that “EMG activity of the human hamstring muscle group” (Magnusson et al., 1995a) was assessed. Specifically, Magnusson et al. (1995a) employed a 90-second static stretch, in which IEMG

measurements were divided into seven 5-second segments. For analysis, initial 5-second IEMG, final 5-second IEMG and delta IEMG (the difference between final and initial 5-second EMG) were used. The correlation coefficients for initial and final IEMG were r = 0.41 and r = 0.19, respectively. Therefore, and taken together, during a passive movement, force (resistance) and EMG measurements of specific muscles should be divided into segments. This will allow and ensure that minimal muscle activity is maintained near end ranges of motion, leading to a more accurate view of the passive properties being examined. Further, and in combination with participant identification, this will allow an accurate end range of motion to be identified.

Calculating Torque and Stiffness

In the calculation of torque, several mathematical models have been proposed and utilized, depending on the situation. For example, an exponential model has been used, and shown to be effective in isolated muscle fibers (Goubel & Lensel-Corbil, 1998; Sten-Knudsen, 1953). Whereas, a second-order polynomial model and a fourth-order polynomial model have been used, with the fourth-order polynomial model to the most commonly used in the literature to assess stiffness in clinical situations (Magnusson et al., 1996a; Magnusson., 1998; Magnusson et al., 1998; Riemann et al., 2001). However, to this point, no one model has been shown to be superior to the others. Nordez et al. (2006) calculated torque and three stiffness indices before and after a stretching intervention using an exponential, a second-order, and a fourth-order polynomial mathematical model. Though the second-order polynomial model showed to be a better fit at lower torque levels, there was no difference in the models at the largest range of motion test (45°) (Nordez et al., 2006).

From the calculation of torque, a stiffness index can be calculated to give further insight into the measure. However, such an index has only been used during active stiffness conditions (Cornu et al., 2001; Lambertz et al., 2001), and not passive. A stiffness index can be beneficial as it provided information regarding the range of motion the torque measure was calculated at. This information is valuable in clinical settings, as one value is able to convey the same information regarding the elastic behavior of muscle (Nordez et al., 2006), as compared to multiple. This then lowers the information burden on practitioners and eases the tracking of measures.

Activity Level and Passive Torque

Passive force (or torque) is the change in passive force across a change in muscle length (Gajdosik, 2001) and is dependent on the muscle viscoelastic properties (Taylor et al., 1990). When examining changes in passive torque through a range of motion, the mechanism response for the change must be considered. Specifically, these mechanisms can be related modifications in the tolerance to stretch (Magnusson et al., 1996a) or to an alteration in the mechanical properties of a muscle (Taylor et al., 1990). These alterations can be induced via flexibility training and resistance training, respectively.

Flexibility Training

Flexibility interventions are a common modality utilized when assessing modifications in passive torque. In response to a single held stretch, passive torque was shown to decrease by approximately 30%, with the decline lasting roughly 45 seconds (Magnusson et al., 1995a). However, these effects are short lived, as resistance to stretch was returned to baseline after 45 minutes (Magnusson et al., 1995b) and 60 minutes (Magnusson et al., 1996b). Nevertheless, muscles are generally stretched more than once

in a clinical or sporting environment (Magnusson, 1998). As such, passive torque was shown to be reduced after five, 90-second static stretches, separated by a 30-second rest (Magnusson, 1998). As was seen after a single stretch, the passive torque decrements were eliminated during a 60-minute rest period (Magnusson, 1998).

In response to proprioceptive neuromuscular facilitation (PNF) stretching, passive torque was shown to be increased (Magnusson et al., 1996a). Yet, there was an associated increase in joint range of motion, while during a fixed angle stretch, and passive torque was not different between the PNF and static stretching techniques (Magnusson et al., 1996a). Therefore, the increased passive torque was likely attributable to the additional range of motion, and not the stretching technique, per se.

However, passive torque was not affected by 3-weeks of stretch training when a constant angle stretching assessment was used (Magnusson et al., 1996c). Passive torque was only increased when the assessment involved a variable angle, allowing for a greater range of motion to be achieved. In addition to the previously mentioned studies, it has been concluded that the increase in passive torque was due to an increase in stretch tolerance, and not an alteration in muscular properties (Magnusson, 1998). In contrast, LaRoche and Connolly (2006) showed that ROM and peak passive torque were increased in a stretching group, after unaccustomed eccentric exercise, compared to a control group. Specifically, four weeks of static stretching resulted in an increased ROM and peak passive torque after the eccentric exercise, whereas a ballistic stretching group showed no differences compared to the control group (LaRoche & Connolly, 2006). As previously mentioned, the increased values were likely attributable to an increased stretch tolerance, and specifically a mitigation of decrements to stretch tolerance after

unaccustomed eccentric exercise (LaRoche & Connolly, 2006). It was also mentioned by the authors that structured resistance training would likely result in a superior protective effect to the unaccustomed exercise, compared to stretching (LaRoche & Connolly, 2006).

Resistance Training

It has been suggested that the muscle-tendon unit can absorb more energy in response to strength training (Garrett, 1990). It has also been suggested that isometric strength training can lead to improvement in the material properties of muscle (Magnusson, 1998). Specifically, Klinge at al. (1997) found that during a constant angle stretch, energy, stiffness and passive torque were increased after 13 weeks of hamstring isometric strength training on one side and isometric and flexibility training on the other. Additionally, isometric strength was shown to increase approximately 43% after both training protocols, while the viscoelastic stress relaxation response was unchanged. Although, with 6 to 12-weeks of isometric strength training leading to an increase in CSA (Jones & Rutherford, 1987; Narici et al., 1989; Davies et al., 1988; Garfinkel & Cafarelli, 1992), it was suggested that muscle hypertrophy could have occurred and led to an increase in stiffness (Magnusson, 1998). Similarly, Kubo et al. (2002) showed that isotonic resistance training alone and isotonic training and stretching together both led to increased triceps surae stiffness. However, the flexibility index, defined as the passive torque-angle curve, was statistically higher in the resistance training group, but not the resistance training and stretching group. It was also noted by the authors that passive torque was higher at all joint angles measured in the resistance training group, but not the resistance training and stretching group (Kubo et al., 2002). Taken together, research has

shown passive torque to increase in response to isometric and isotonic resistance training, while stretching could lead to a diminished response.

Further, passive tension has been shown to increase after eccentric contractions in animals (Whitehead et al., 2001) and is considered to occur in humans in response to eccentric exercise (Chleboun et al., 1995; Howell et al., 1993; Jones et al., 1997). Passive stiffness has been shown to increase with a concomitant decrease in joint angle at the elbow in response to eccentric exercise (Clarkson et al., 1992; Cleak & Eston, 1992). Similar inferences have been made as muscle hardness has been shown to increase due to eccentric exercise (Murayama et al., 2000), alluding to changes in muscle stiffness.

Several explanations have been postulated for these observations. First, passive tension is thought to increase in response to eccentric exercise due to swelling occurring in response to muscle damage (Howell et al., 1993; Leger & Milner, 2000). Although Whitehead et al. (2001) showed that the time course for peak passive tension was immediately after exercise, whereas swelling did not markedly increase until 24 hours after exercise. Additionally, it was noted that the 2% increase in muscle volume due to swelling was unlikely to result in the 40% observed increase in passive tension (Whitehead et al., 2001).

Yet another explanation for the sustained passive tension is related to increased resting muscle fiber calcium levels (Hoang et al., 2007), which has been shown in mice (Balnave et al., 1997). Specifically, it is suggested that sarcomere damage results in uncontrolled calcium release, leading to contracture (Whitehead et al., 2001), and thus increased tension. Further, Howell et al. (1993) suggested that injured muscles result in an exaggerated calcium release upon stretch activation, which increases passive stiffness.

Due to the nature and structure of titin, as previously described, it is conceivable that the PEVK segment is affected due to this increased calcium release (Hoang et al., 2007), ultimately resulting in increased passive force. In contrast, Mahieu et al. (2008) showed plantar flexion passive torque to decrease, while dorsiflexion ROM increased in response to eccentric exercise. These findings have been suggested to be due to structural (Magnusson et al., 1996c), and not stretch tolerance adaptations (Mahieu et al., 2008). Specifically, Lynn and Morgan (Öhberg & Alfedson, 2004) showed muscle fiber compliance to be increased in rats due to the addition of sarcomeres in series, after completing eccentric exercise.

Conclusions

Passive resistance/torque and stiffness are common measures used when investigating the passive properties of muscle. Specifically, they are commonly used in flexibility studies, with results attributed to stretch tolerance alterations. However, new evidence has emerged suggesting that the passive structural elements within muscle may have a greater contribution than previously thought. As such, no apparent study has investigated a proper methodology that considers these passive elements when assessing passive resistance and stiffness in a clinical setting. Further, if a novel methodology is found, then it would also be appropriate to investigate different mathematical models used in the calculation of torque and stiffness to elucidate the best model to use.

CHAPTER III

NOVEL APPROACHES TO MEASUREMENT OF PASSIVE RESISTANCE IN THE HAMSTRINGS

Introduction

Residual force enhancement (RFE) is a phenomenon in which there is an increase in isometric steady-state force after an eccentric contraction as compared to the isometric steady-state force of isometric-only contraction at the corresponding length of activation (Herzog et al., 2016). This increased steady-state force has been observed to remain in deactivated muscle and has been termed passive force enhancement (Herzog & Leonard, 2002). Passive force enhancement (PFE) has been observed in preparations across the several levels of muscle hierarchy (Herzog & Leonard, 2002; Joumaa et al., 2007; Rassier et al., 2003). Due to its proportionality to PFE, RFE has been suggested to be associated with passive structures, such as titin, within muscle (Edman et al., 1982; Herzog et al., 2002). While the contribution of passive structural elements to muscle function is well known, recent discoveries point to an even greater contribution to muscle function (Nishikawa et al., 2012; Herzog et al., 2002; Herzog et al., 2016; Hessel et al., 2017).

Passive resistance and stiffness are measures that have been used to indicate the amount of passive resistive force a muscle exerts as it is stretched (LaRoche & Conolly, 2006) and may provide information on the passive tensile properties of the musculotendinous complex, including the passive structures that are believed to contribute to PFE. The measurement of passive resistance *in vivo* has predominately been used to assess muscular stretch compliance as part of investigations related to flexibility (Magnusson et al., 1995; Reid & McNair, 2011). Such modifications in stretch

compliance are observed through increases in joint range of motion. Methodologies of these studies included controlling range of motion and speed while monitoring muscle activation, through electromyography (EMG), during passive stretch to ensure the musculature is not acticated during the movement. Specifically, EMG was limited to minimal or negligible levels (Gajdosik et al., 1999b; Gajdosik et al., 1990; Gajdosik et al., 1999a).

While efforts have been made to ensure that passive resistance and stiffness measures are predominately quantifying structural elements of muscle, recent revelations around residual and PFE may challenge previous methodological controls. For example, Herzog and Leonard (2002) noted PFE continued after deactivation for well beyond times set in the study. They noted that PFE remained until the muscle tissue was returned to its initial length. These new findings may confound traditional approaches to measuring passive resistance and stiffness and studies should explore whether the traditional passive resistance and stiffness approach is affected by PFE. This has led us to conduct small pilot testing with observations showing that varying rest times before passive tension measurements resulted in lower values. Specifically, these laboratory observations showed a rest time of 3 minutes to be superior to rest times of 0-, 1-, and 5-minutes. Therefore, the purpose of this study was to compare traditional passive resistance and stiffness methodology by methodologies that considers PFE. It is hypothesized that (i) the methodologies considering PFE will result in lower passive resistance values, and (ii) that the 3-minute rest period and resetting to resting length will result in the overall lowest passive resistance values.

Methods

Study Design

To investigate passive resistance and stiffness methodologies as it relates to PFE, a randomized test design was utilized. Assessments were randomized between the collection of passive resistance according to traditional methods, and those that consider PFE. For the traditional methods, the right knee was passively extended, as described below, with no prior control of the muscle state. For the passive resistance measures considering PFE, one repetition of knee extension and knee flexion was completed before the collection of data. The initial repetition was used to bring the hamstring muscle to resting length, to account for PFE (Herzog & Leonard, 2002). Additionally, a 1-, 3-, and 5-minute rest period was utilized before the initial repetition in the assessment of passive resistance considering PFE. Dependent-samples *t*-tests were used to compare resistance measures across testing conditions.

Participants

A power analysis was conducted in G*Power 3.1 (Universitat Kiel, Germany) for a repeated measures one-way ANOVA, with 4 repeated measures, an alpha level of 0.05, power of 0.80, a medium effect size ($f^2 = 0.25$) (Cohen, 1988), and correlations between the reproducible measures of passive torque equal to 0.98 (Magnusson et al., 1995), indicated a sample size of 4 participants was required for this study. The sample population consisted of ten healthy males (age: 25.30 ± 4.72 yrs; height: 172.15 ± 9.50 cm; weight: 76.83 ± 13.75 kg). Participants were excluded from the study if they had a lower limb injury in the past 3 months, or if they had an acute illness. Inclusion criteria were 18–35-year-old apparently healthy males. After the participants were informed of the benefits and possible risks of the protocol, all participants read and signed a written

informed consent prior to participating. Participants were recruited from the university via word of mouth. The Institutional Review Board at Middle Tennessee State University approved this study prior to data collection.

Instrumentation

For the purposes of this study, we used the following terms and definitions, as described previously by Norman et al. (manuscript in preparation):

- $EMG_{initial}$: low-level EMG magnitudes in a relaxed state, with no voluntary muscle activity
- EMG_{active}: highest EMG magnitudes during a 3-second maximal voluntary isometric contraction (MVIC)
- Passive $Resistance_{initial}$: passive force (N) detected by the load cell of the dynamometer before the initiation of the movement
- Passive $Resistance_{final}$: passive force (N) detected by the load cell of the dynamometer at the end of the movement
- Passive Torque: resistance to stretch during the movement, without prior muscle activation. Torque was calculated as described by Norman et al. (manuscript in preparation).
- Passive $Torque_{control}$: passive torque value after knee extension, with no prior modification to the muscle state before measurement.
- Final Range of Motion (ROM_{final}): The range of motion used for testing was determined as follows. After properly positioned (described below), each participant was instructed to extend their knee as far as they are able. Once this

maximal ROM was reached, the ROM_{final} was determined as 5° below the participant's determined maximal ROM and 60° (Moffroid et al., 1969; Yoon et al., 1991) of total knee flexion range of motion. Range of motion was determined in this way to ensure that the hamstring musculature was at its resting length before the onset of the passive movement used for data collection. Additionally, to further ensure the hamstring was positioned at its resting length before the onset of a testing movement, one repetition of the passive movement, described below, was completed.

Muscle activity was measured using the Trigno wireless electromyographic (EMG) system (Delsys, Natick MA) during passive torque and resistance testing. The system contained Trigno Flex EMG sensors that were placed directly on the skin surface over the mid-belly of the biceps femoris and semitendinosus. Prior to placing EMG sensors, hair was shaved with a safety razor, exfoliated with Redux paste, and cleaned with isopropyl alcohol to reduce signal impedance. The underside of the sensors was attached to the skin with double-sided adhesive tape and then the outside of the sensor was further secured with adhesive stretch tape. Location and procedures for placement of sensors was implemented in accordance with the SENIAM project guidelines (SENIAM, 2022).

To measure hamstring resistance values, a custom resistive torque apparatus was used on the participants' right leg. Participants were in a seated position on a Biodex isokinetic dynamometer system 3 (Shirely, NY) and secured via the manufacturer's guidelines. The participants' leg was positioned with the hip positioned with 30° from horizontal and the lever arm was placed 2cm proximal to the malleolus (Magnusson et

al., 1995). Additionally, a load cell (Transducer Techniques, Temecula, Calif), was attached to the resistance arm of the dynamometer to record passive resistance.

Procedure

During the collection of passive resistance, participants were instructed to relax all leg muscles and allow the apparatus to move the testing leg into knee extension at a speed of 5°/sec (Magnusson et al., 1995) and 10°/sec during knee flexion. To ensure the hamstring musculature was not activated during the movement, EMG readings were collected during testing to ensure only $EMG_{initial}$ values were present. This procedure was completed on the right leg for two repetitions across four conditions, for a total of eight repetitions. The four conditions were as follows: passive $torque_{control}$, passive torque after 1-minute + resetting repetition, 3-minutes + resetting repetition, and 5-minutes of a rest period + resetting repetition. During the collection of passive $torque_{control}$, the knee was extended as mentioned above. Before testing, a trial repetition was completed familiarize the participant with the movement. After the rest period of each of the experimental conditions, one repetition of knee extension and knee flexion movements were completed prior to the measurement of passive resistance. This initial repetition, prior to data collection, ensured that the hamstring musculature was at resting length before the onset of data collection. The passive $torque_{control}$ condition was collected first for each participant, while the other three conditions were randomized. The participants were then given a 3-minute break in which they were permitted to engage in normal activities of daily living. After this break period, the above-mentioned procedures were completed again, for the second repetition of each condition.

EMG was recorded and monitored for the duration of the assessment. Specifically, EMG was used to ensure no muscle activity was present during the recording of passive resistance$_{initial}$, passive resistance$_{final}$, and passive torque.

The participants completed three MVICs with their hamstring musculature. During each MVIC, the participants were instructed to maximally activate their hamstring musculature for a three second period. A one-minute rest period was given between the three repetitions. The EMG data collected during the MVICs was used for normalization purposes during data analysis.

Statistical Approach

A repeated-measures one-way ANOVA was used to detect differences in peak passive resistance between the traditional resistance measurement and the experimental conditions. Post hoc paired-samples *t*-tests were used to identify differences between the traditional measurement and each experimental condition. An alpha level of .05 was used for all analyses. Hedges' *g* was used for post hoc effect size to correct for small sample size, with values of 0.2, 0.5, and 0.8 indicating small, medium, and large effect sizes, respectively.

Results

Mauchly's test indicated that the assumptions of sphericity had been violated, $X^2(5) = 26.75$, $p < .001$, therefore Greenhouse-Geisser corrected tests are reported ($p < .001$). The results showed that resistance was significantly different among testing conditions, $F(1.19, 10.67) = 50.95$, $p < .001$, $\eta^2 = 0.85$.

Results of the post hoc paired-samples *t*-tests comparing peak passive resistance across testing conditions are displayed in Table 1. Peak passive resistance was

significantly lower ($p < .001$) after a 1-minute ($M = 71.9$, $SD = 18.1$), 3-minute ($M = 74.2$, $SD = 20.3$), and 5-minute ($M = 74.9$, $SD = 18.8$) rest period with resting length repetition compared to the traditional measurement ($M = 112.7$, $SD = 26.3$). Among the experimental conditions, 1-minute was significantly lower than 3-minutes of rest and resetting to resting length ($p = .039$), while the other comparisons were not significantly different. Normalized mean muscle activity during each condition is displayed in Table 2.

Table 1.

Post Hoc Comparisons of Passive Resistance

	Resistance (N·m) M(SD)	*t*	*p*	Mean Difference	95% CI	Hedges' *g*
Pair 1						
Traditional	112.7(26.3)	8.09	.001*	-40.85	[1.18, 3.70]	2.45
1-minute	71.9(18.1)					
Pair 2						
Traditional	112.7(26.3)	7.27	.001*	-38.50	[1.07, 3.50]	2.30
3-minute	74.2(20.3)					
Pair 3						
Traditional	112.7(26.3)	6.83	.001*	-37.80	[.942, 3.16]	2.07
5-minute	74.9(18.8)					
Pair 4						
1-minute	71.9(18.1)	-2.41	.039	-2.35	[-1.40, -.035]	-.762
3-minute	74.2(20.3)					
Pair 5						
1-minute	71.9(18.1)	-1.90	.090	-3.05	[-1.21, .086]	-.576
5-minute	74.9(18.8)					
Pair 6						
3-minute	74.2(20.3)	-.446	.666	-.700	[-.728, .465]	-.135
5-minute	74.9(18.8)					

Note. 1-, 3-, and 5-minute denotes the rest period taken prior to the normalizing repetition that the starting position was at 60° range of motion.
*denotes significance of $p < .001$ in comparison to the traditional assessment

Table 2.

Normalized Mean Muscle Activity

	Biceps Femoris M(SD)	Semitendinosus M(SD)
Traditional	.042(.029)	.019(.012)
1-minute	.044(.031)	.017(.014)
3-minute	.045(.037)	.018(.023)
5-minute	.041(.026)	.016(.008)

Note. 1-, 3-, and 5-minute denotes the rest period taken prior to the normalizing repetition that the starting position was at 60° range of motion

Discussion

The purpose of the current study was to compare traditional passive resistance and stiffness methodology by methodologies that considers PFE. The results show that passive resistance is reduced in the experimental conditions, compared to the traditional method of assessment. Specifically, the experimental conditions consisted of a rest period followed by an initial passive extension and flexion movement. The passive flexion movement utilized in the current study continued beyond a standard range of motion that corresponds to muscle resting length. This resting length, or optimal length, was defined as the point where the muscle was able to produce the most force (Moffroid et al., 1969; Yoon et al., 1991). The results showed a significant difference in resistance values among the 1- and 3-minute rest experimental conditions, but no differences between 1- and 5-minutes, and 3- and 5-minutes. Therefore, returning the muscle to its resting length had the largest effect on passive resistance, with no effect of rest time beyond 1-minute.

A plausible explanation for the current results is that PFE contributed to the traditional measure of passive resistance. PFE has been shown to occur across the muscle hierarchy (Herzog & Leonard, 2002; Joumaa et al., 2007; Rassier et al., 2003), is long lasting (>25 seconds; Herzog & Leonard, 2002), and persists after muscle deactivation (Joumaa et al., 2007). Due to its presence in deactivated muscle, it has been suggested that the main component responsible for PFE is titin, a passive myofilament within muscle. According to the Winding Filament Hypothesis, titin interacts with actin in the presence of calcium to increase force production during active lengthening (Nishikawa et al., 2012). This interaction involves the N2A region of titin binding to actin. Due to the helical structure of actin, as the myofilament translates, it also rotates (Morgan, 1977). This actin rotation winds the PEVK segment of titin onto actin producing a large increase

in force production. Leonard and Herzog (2010) showed force increases of up to four times in activated and lengthened myofibrils compared to nonactivated preparations. Further, the stretching lengths used were beyond cross-bridge overlap, eliminating cross-bridge contribution. Similarly, in the current study, passive resistance was 1.57 times higher during the traditional assessment compared to the 1-minute rest condition. In vitro investigation of titin's role in force production in rabbit psoas myofibrils demonstrated that when titin was knocked out by use of a trypsin solution, there was no difference in force production between the activated and non-activated preparations (Leonard & Herzog, 2010). It was concluded that this titin-actin interaction in the presence of calcium was responsible for the large increased force production (Leonard & Herzog, 2010). Based on the current literature of the winding filament hypothesis and our results, it is possible titin-actin interaction was reduced or eliminated as a result of positioning the muscle to resting length in each of the experimental conditions.

Herzog and Leonard (2002) were among the first to show that PFE will persist until the muscle is returned to this initial length. Specifically, they showed that PFE persisted in cat soleus muscle preparations until they were returned to their initial lengths, defined as 10mm on the ascending limb of the force-length curve. Muscle damage was excluded as a possible explanation, as only muscles that produced the same (± 1N) isometric contraction forces before and after stretching were retained for analysis (Herzog & Leonard, 2002).

The current results shed new light on the methodologies used to assess passive resistance. Though previous research has utilized minimal EMG activity as a proxy for a resting muscle's state, EMG does not account for the passive structures within muscle.

The PEVK segment of titin could still be bound to actin even though minimal EMG activity is present. This could result in larger forces produced than what would be registered if the N2A region of titin was not attached the actin. As mentioned previously, this PFE will persist until the muscle is returned to its resting length (Herzog & Leonard, 2002). Therefore, what previous research denotes as passive resistance due to minimal EMG activity, could also be measuring a muscular state that includes this PFE. To the author's knowledge this is the first study to ensure that the muscle within question begins assessment at its resting length, in conjunction with minimal EMG activity. As such, trials meeting both criteria showed lower passive resistance measures compared to trials only ensuring minimal EMG activity. This brings into question the methodologies previously utilized to assess passive resistance. Based on the evidence that has mounted regarding the presence of PFE, methodologies assessing passive muscular states should account for this phenomenon.

As this study implemented a novel assessment of passive resistance, there are several limitations that should be noted. First, this was a novel clinical assessment with a strong theoretical foundation. As such the direct rationale for the study as related to the characteristics and functions of titin within muscle were not measured. Second, the study involved a small (n = 10) sample size of male participants, and two trials per testing conditions. Future studies should attempt to reproduce the current results, as well as expand the sample population to more diverse groups. As the current study focused on the hamstring muscle group, the practicality of assessing other muscle groups in a similar fashion is also of interest. Lastly, as the current methodology was shown to result in superior passive resistance values, this novel methodology should be expanded to

compare passive resistance to the traditional methodology as well as to an “activated” or enhanced-passive condition.

In conclusion, the current novel methodology was shown to result in lower passive resistance values compared to the traditional assessment. It is suggested that the novel assessment reduced titin-actin interaction, which may be present in the traditional assessment. Therefore, a likely rationale for the current study is that titin can change its structure and force producing capabilities, and these changes are able to be measured in a clinical setting. If such modifications are detected in a resting state, it is interesting to theorize how other possible alterations to titin in response to prior activation, or in response to eccentric training, may be detected or monitored with the novel methodology. If titin can be strengthened during eccentric training in conjunction with other muscular properties, it could be expected that PFE would be increased after a period of training compared to baseline.

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CHAPTER IV

CALCULATION OF PASSIVE TORQUE AND STIFFNESS USING DIFFERENT MATHEMATICAL MODELS

Introduction

Passive force enhancement (PFE) is a phenomenon referring to the presence of an increased eccentric steady-state force, when compared to an isometric condition (Herzog & Leonard, 2002). This has been shown to occur at varying levels of the muscular structure (Herzog & Leonard, 2002; Joumaa et al., 2007; Rassier et al., 2003) and will persist after deactivation for a length of time, and until the muscle was returned to its resting length (Herzog & Leonard, 2002). It has been known that structural elastic elements, mainly titin, are responsible for this increased force production, however, new evidence has suggested that the titin contribution may be greater than previously thought (Nishikawa et al., 2012; Herzog & Leonard 2002; Herzog et al., 2016; Hessel et al., 2017).

Passive torque is the resistive force produced by a muscle during lengthening, through a range of motion (LaRoche & Connolly, 2006). Muscle stiffness has been defined as the relationship between muscular resistive force and the muscle length during stretch (Gajdosik, 2001), or as the slope of the torque-angle curve (Nordez et al., 2006). Passive torque and stiffness are predominantly used as measures in studies investigating stretch tolerance (Magnusson et al., 1995; Reid & McNair, 2011), and are therefore, used to measure the tensile properties of the musculotendinous complex. Further, these measures have been used as indirect assessments of PFE, with modifications conceivably being attributed to the passive structural elements within muscle. In such studies

investigating stretch tolerance or flexibility, methodologies are used to monitor muscle activity via electromyographic (EMG) sensors, control speed, and range of motion. However, under new revelations, these methodologies may need to be revisited. Specifically, Herzog and Leonard (2002) showed that PFE persisted well beyond times outlined in the study and continued until the muscle was returned to its resting length. Therefore, the contribution of titin could be causing unexpected increases in what are expected to be passive measures.

In the calculation of stiffness, few studies have focused on the passive state, as much of the interest has been around active stiffness. Additionally, there is uncertainty around which mathematical model should be utilized. For example, a second-order polynomial model has been used when investigating active elastic stiffness (Cornu et al., 2001; Lambertz et al., 2001), an exponential model has been used in isolated muscle preparations (Goubel & Lensel-Corbeil, 1998; Sten-Knudsen, 1953), while the most commonly used is a fourth-order polynomial model (Magnusson et al., 1996; Magnusson et al., 1998; Magnusson, 1998; Riemann et al., 2001). Nordez et al. (2006) conducted a study investigating the effects of an exponential model, a second-order polynomial, and a fourth-order polynomial model had on passive stiffness calculations. As such, mathematical models may be used when investigating passive stiffness near end ranges of motion, as “no one model can be recommended exclusive of other” (Nordez et al., 2006). Therefore, the purpose of this study was to fit multiple mathematical models to passive torque-angle data to elucidate whether the nature of the passive torque curve was different in each of the experimental situations.

Methods

Study Design

To investigate passive torque and stiffness methodologies as it relates to PFE, a randomized test design was utilized. Assessments were randomized between the collection of passive torque according to traditional methods, and those that consider PFE. For the traditional methods, the right knee was passively extended, as described below, with no prior control of the muscle state. For the passive torque measures considering PFE, one repetition of knee extension and knee flexion was completed before the collection of data. This initial repetition was to ensure the hamstring muscle was at its resting length, to account for the effects of PFE. Additionally, a 1-, 3-, and 5-minute rest period were utilized before the initial repetition in the assessment of passive torque considering PFE. The torque-angle data of the traditional, 1-min, 3-min, and 5-min trials used and each of the mathematical models (2^{nd} order polynomial, 4^{th} order polynomial, and exponential) were applied to calculate a goodness-of-fit index (GFI). The sum of squares error (SSE) and the root mean squared error (RMSE) of the GFI were used for comparing models across each of the trials.

Participants

A power analysis was conducted in G*Power 3.1 (Universitat Kiel, Germany) for a repeated measures one-way ANOVA, with 4 repeated measures, an alpha level of 0.05, power of 0.80, a medium effect size ($f^2 = 0.25$) (Cohen, 1988), and correlations between the reproducible measures of passive torque equal to 0.98 (Magnusson et al., 1995), indicated a sample size of 4 participants was required for this study. The sample population consisted of ten healthy males (age: 25.30 ± 4.72 yrs; height: 172.15 ± 9.50 cm; weight: 76.83 ± 13.75 kg). Participants were excluded from the study if they had a

lower limb injury in the past 3 months, or if they had an acute illness. Inclusion criteria were 18–35-year-old apparently healthy males. After the participants were informed of the benefits and possible risks of the protocol, all participants read and signed a written informed consent prior to participating. Participants were recruited from the university via word of mouth. The Institutional Review Board at Middle Tennessee State University approved this study prior to data collection.

Instrumentation

For the purposes of this study, we used the following terms and definitions, as described previously by Norman et al. (manuscript in preparation):

- $EMG_{initial}$: low-level EMG magnitudes in a relaxed state, with no voluntary muscle activity
- EMG_{active}: highest EMG magnitudes during a 3-second maximal voluntary isometric contraction (MVIC)
- Passive $Resistance_{initial}$: passive force (N) detected by the load cell of the dynamometer before the initiation of the movement
- Passive $Resistance_{final}$: passive force (N) detected by the load cell of the dynamometer at the end of the movement
- Passive Torque: resistance to stretch during the movement, without prior muscle activation. Torque was calculated as described by Norman et al. (manuscript in preparation).
- Passive $Torque_{control}$: passive torque value after knee extension, with no prior modification to the muscle state before measurement.

- Final Range of Motion (ROM_{final}): The range of motion used for testing was determined as follows. After properly positioned (described below), each participant was instructed to extend their knee as far as they are able. Once this maximal ROM was reached, the ROM_{final} was determined as 5° below the participant's determined maximal ROM and 60° (Moffroid et al., 1969; Yoon et al., 1991) of total knee flexion range of motion. Range of motion was determined in this way to ensure that the hamstring musculature was at its resting length before the onset of the passive movement used for data collection. Additionally, to further ensure the hamstring was positioned at its resting length before the onset of a testing movement, one repetition of the passive movement, described below, was completed.

Muscle activity was measured using the Trigno wireless electromyographic (EMG) system (Delsys, Natick MA) during passive torque and resistance testing. The system contained Trigno Flex EMG sensors that were placed directly on the skin surface over the mid-belly of the biceps femoris and semitendinosus. Prior to placing EMG sensors, hair was shaved with a safety razor, exfoliated with Redux paste, and cleaned with isopropyl alcohol to reduce signal impedance. The underside of the sensors was attached to the skin with double-sided adhesive tape and then the outside of the sensor was further secured with adhesive stretch tape. Location and procedures for placement of sensors was implemented in accordance with the SENIAM project guidelines (SENIAM, 2022).

To measure hamstring resistance values, a custom resistive torque apparatus was used on the participants' right leg. Participants were in a seated position on a Biodex

isokinetic dynamometer system 3 (Shirely, NY) and secured via the manufacturer's guidelines. The participants' leg was positioned with the hip positioned with 30° from horizontal and the lever arm was placed 2cm proximal to the malleolus (Magnusson et al., 1995). Additionally, a load cell (Transducer Techniques, Temecula, Calif), was attached to the resistance arm of the dynamometer to record passive resistance.

Procedure

During the collection of passive resistance, participants were instructed to relax all leg muscles and allow the apparatus to move the testing leg into knee extension at a speed of 5°/sec (Magnusson et al., 1995) and 10°/sec during knee flexion. To ensure the hamstring musculature was not activated during the movement, EMG readings were collected during testing to ensure only $EMG_{initial}$ values were present. This procedure was completed on the right leg for two repetitions across four conditions, for a total of eight repetitions. The four conditions were as follows: passive $torque_{control}$, passive torque after 1-minute + resetting repetition, 3-minutes + resetting repetition, and 5-minutes of a rest period + resetting repetition. During the collection of passive $torque_{control}$, the knee was extended as mentioned above. Before testing, a trial repetition was completed familiarize the participant with the movement. After the rest period of each of the experimental conditions, one repetition of knee extension and knee flexion movements were completed prior to the measurement of passive resistance. This initial repetition, prior to data collection, ensured that the hamstring musculature was at resting length before the onset of data collection. The passive $torque_{control}$ condition was collected first for each participant, while the other three conditions were randomized. The participants were then given a 3-minute break in which they were permitted to engage in normal activities of

daily living. After this break period, the above-mentioned procedures were completed again, for the second repetition of each condition.

EMG was recorded and monitored for the duration of the assessment. Specifically, EMG was used to ensure no muscle activity was present during the recording of passive resistance$_{initial}$, passive resistance$_{final}$, and passive torque.

The participants completed three MVICs with their hamstring musculature. During each MVIC, the participants were instructed to maximally activate their hamstring musculature for a three second period. A one-minute rest period was given between the three repetitions. The EMG data collected during the MVICs was used for normalization purposes during data analysis.

Data Analysis

Passive torque and range of motion data were processed and compared to second-order polynomial, fourth-order polynomial, and exponential models through Curve Fitting Application in MatLab software (version R2022A). Sum of squares due to error (SEE) and root mean squared error (RMSE) were extracted from MatLab for comparative statistical analysis between the models at each testing condition for each participant. For example, for participant 1, SEE and RMSE were calculated for a second-order polynomial model in MatLab for the traditional assessment, 1-minute rest period and resting to resting length, 3-minute rest period and resting to resting length, and 5-minute rest period and resting to resting length. The same process was completed for the fourth-order polynomial model and the exponential model. Further, the previous steps were replicated across the other 9 participants.

Statistical Approach

A repeated-measures two-way ANOVA was used to analyze whether there was a difference between the SSE and the RMSE for each testing condition as calculated by a second-order polynomial model, a fourth-order polynomial model, and an exponential model. The overall model for SSE, $F(1.01, 9.09) = 21.65$, $p = .001$, $\eta^2 = .706$, and RMSE, $F(1.30, 11.71) = 50.12$, $p < .001$, $\eta^2 = .848$, were both significant. RMSE values will be reported as the effect size was larger. Post hoc paired-samples *t*-tests were used to identify differences between RMSE for the traditional measurement and each experimental condition. Further, comparisons were made to identify differences in the observed maximal passive torque collected and predicted maximal passive torque calculated by each mathematical model. An alpha level of .05 was used for all analyses. Hedges' *g* was used for post hoc effect size to correct for small sample size, with values of 0.2, 0.5, and 0.8 indicating small, medium, and large effect sizes, respectively.

Results

Mauchly's test indicated that the assumptions of sphericity had been violated, $X^2(20) = 87.33$, $p < .001$, therefore Greenhouse-Geisser corrected tests are reported ($p < .001$). The results showed that RMSE was significantly different among testing conditions, $F(1.30, 11.71) = 50.12$, $p < .001$, $\eta^2 = .848$.

Results of the post hoc paired-samples *t*-tests comparing RMSE in each testing condition for the second-order polynomial model, fourth-order polynomial model, and exponential model are displayed in Table 1, Table 2, and Table 3, respectively. In the second-order polynomial model, RMSE was significantly lower ($p = .011$) after a 1-minute rest period ($M = .450$, $SD = .226$) and resetting to resting length, a 3-minute rest period ($M = .461$, $SD = .271$) and resetting to resting length ($p = 0.23$), and a 5-minute

rest period (M = .476, SD = .272) and resetting to resting length (p = .028), compared to the traditional measurement (M = .723, SD = .275). All comparisons among the experimental conditions were not significantly different.

In the fourth-order polynomial model, RMSE was significantly lower (p < .001) after a 1-minute rest period (M = .141, SD = .056) and resetting to resting length, a 3-minute rest period (M = .154, SD = .062) and resetting to resting length (p = .002), and a 5-minute rest period (M = .158, SD = .078) and resetting to resting length (p = .004), compared to the traditional measurement (M = .243, SD = .100). All comparisons among the experimental conditions were not significantly different.

In the exponential model, RMSE was significantly lower (p < .001) after a 1-minute rest period (M = 1.28, SD = .540) and resetting to resting length, a 3-minute rest period (M = 1.28, SD = .504) and resetting to resting length (p < .001), and a 5-minute rest period (M = 1.30, SD = .500) and resetting to resting length (p < .001), compared to the traditional measurement (M = 3.06, SD = .985). All comparisons among the experimental conditions were not significantly different.

Results of the post hoc paired-samples *t*-tests comparing observed maximal passive torque to predicted maximal passive torque calculated by each mathematical model for the traditional assessment and each experimental condition (1-, 3-, and 5-minue rest period plus a normalizing repetition) found in Table 4, Table 5, Table 6, and Table 7 respectively. All comparisons among each testing condition and each mathematical model were significantly different (p < .001). Normalized mean muscle activity during each condition is displayed in Table 8.

Table 1.

Post Hoc Comparisons of RMSE for a Second-Order Polynomial Model

	RMSE M(SD)	*t*	*p*	Mean Difference	95% CI	Hedges' g
Pair 1						
Traditional	.723(.275)	3.17	.011	.272	[.206, 1.679]	0.959
1-minute	.450(.226)					
Pair 2						
Traditional	.723(.275)	2.74	.023	.261	[.110, 1.517]	0.83
3-minute	.461(.271)					
Pair 3						
Traditional	.723(.275)	2.61	.028	.247	[.081, 1.468]	0.79
5-minute	.476(.272)					
Pair 4						
1-minute	.450(.226)	-.699	.502	-.011	[-.807, .395]	-.212
3-minute	.461(.271)					
Pair 5						
1-minute	.450(.226)	-1.05	.322	-.026	[-.920, .302]	-.317
5-minute	.476(.272)					
Pair 6						
3-minute	.461(.271)	-.717	.492	-.015	[-.813, .390]	-.217
5-minute	.476(.272)					

Note. 1-, 3-, and 5-minute denotes the rest period taken prior to the normalizing repetition that the starting position was at 60° range of motion.

Table 2.

Post Hoc Comparisons of RMSE for a Fourth-Order Polynomial Model

	RMSE M(SD)	*t*	*p*	Mean Difference	95% CI	Hedges' g
Pair 1						
Traditional	.243(.100)	4.81	.001*	.102	[.551, 2.33]	1.46
1-minute	.141(.056)					
Pair 2						
Traditional	.243(.100)	4.20	.002	.090	[.426, 2.08]	1.27
3-minute	.154(.062)					
Pair 3						
Traditional	.243(.100)	3.87	.005	.085	[.357, 1.95]	1.17
5-minute	.158(.078)					
Pair 4						
1-minute	.141(.056)	-1.74	.117	-.013	[-1.15, .127]	-.525
3-minute	.154(.062)					
Pair 5						
1-minute	.141(.056)	-1.57	.151	-.017	[-1.10, .168]	-.475
5-minute	.158(.078)					
Pair 6						
3-minute	.154(.062)	-.557	.591	-.005	[-.763, .434]	-.169
5-minute	.158(.078)					

Note. 1-, 3-, and 5-minute denotes the rest period taken prior to the normalizing repetition that the starting position was at 60° range of motion.

*denotes significance of $p < .001$ in comparison to the traditional assessment

Table 3.

Post Hoc Comparisons of RMSE for an Exponential Model

	RMSE M(SD)	*t*	*p*	Mean Difference	95% CI	Hedges' g
Pair 1						
Traditional	3.06(.985)	7.63	.001*	1.79	[1.09, 3.50]	2.31
1-minute	1.28(.540)					
Pair 2						
Traditional	3.06(.985)	7.18	.001*	1.78	[1.01, 3.31]	2.18
3-minute	1.28(.504)					
Pair 3						
Traditional	3.06(.985)	7.37	.001*	1.76	[1.04, 3.39]	2.23
5-minute	1.30(.500)					
Pair 4						
1-minute	1.28(.540)	-.077	.940	-.003	[-.616, .571]	-.023
3-minute	1.28(.504)					
Pair 5						
1-minute	1.28(.540)	-.747	.474	-.025	[-.823, .382]	-.226
5-minute	1.30(.500)					
Pair 6						
3-minute	1.28(.504)	-.399	.699	-.022	[-.714, .478]	-.121
5-minute	1.30(.500)					

Note. 1-, 3-, and 5-minute denotes the rest period taken prior to the normalizing repetition that the starting position was at 60° range of motion.

*denotes significance of $p < .001$ in comparison to the traditional assessment

Table 4.

Post Hoc Comparisons of Predicted Max Passive Torque vs Observed Max Passive Torque for Traditional Assessment

	Torque (N·m) M(SD)	*t*	*p*	Mean Difference	95% CI	Hedges' g
Pair 1						
Observed	50.44(10.82)	8.31	.001*	.722	[1.22, 3.79]	2.52
Poly2	49.71(10.80)					
Pair 2						
Observed	50.44(10.82)	7.67	.001*	.243	[1.10, 3.52]	2.32
Poly4	50.20(10.82)					
Pair 3						
Observed	50.44(10.82)	9.84	.001*	3.06	[1.49, 4.44]	2.98
Exp	47.37(10.65)					

Note. Observed = collected passive torque, Poly2 = predicted passive torque from second-order polynomial model, Poly4 = predicted passive torque from fourth-order polynomial model, Exp = predicted passive torque from an exponential model

*denotes significance of $p < .001$ in comparison to the observed assessment

Table 5.

Post Hoc Comparisons of Predicted Max Passive Torque vs Observed Max Passive Torque for 1-minute Rest + Normalizing Repetition Assessment

	Torque (N·m) M(SD)	*t*	*p*	Mean Difference	95% CI	Hedges' g
Pair 1						
Observed	27.84(7.57)	6.29	.001*	.450	[.840, 2.94]	1.91
Poly2	27.39(7.56)					
Pair 2						
Observed	27.84(7.57)	7.99	.001*	.141	[1.16, 3.66]	2.42
Poly4	27.70(7.58)					
Pair 3						
Observed	27.84(7.57)	7.49	.001*	1.28	[1.06, 3.43]	2.26
Exp	26.57(7.34)					

Note. Observed = collected passive torque, Poly2 = predicted passive torque from second-order polynomial model, Poly4 = predicted passive torque from fourth-order polynomial model, Exp = predicted passive torque from an exponential model

*denotes significance of $p < .001$ in comparison to the observed assessment

Table 6.

Post Hoc Comparisons of Predicted Max Passive Torque vs Observed Max Passive Torque for 3-minute Rest + Normalizing Repetition Assessment

	Torque (N·m) M(SD)	*t*	*p*	Mean Difference	95% CI	Hedges' g
Pair 1						
Observed	28.75(8.33)	5.38	.001*	.461	[.663, 2.56]	1.63
Poly2	28.29(8.32)					
Pair 2						
Observed	28.75(8.33)	7.82	.001*	.154	[1.13, 3.58]	2.37
Poly4	28.60(8.35)					
Pair 3						
Observed	28.75(8.33)	8.03	.001*	1.28	[1.16, 3.67]	2.43
Exp	27.47(8.12)					

Note. Observed = collected passive torque, Poly2 = predicted passive torque from second-order polynomial model, Poly4 = predicted passive torque from fourth-order polynomial model, Exp = predicted passive torque from an exponential model

*denotes significance of $p < .001$ in comparison to the observed assessment

Table 7.

Post Hoc Comparisons of Predicted Max Passive Torque vs Observed Max Passive Torque for 5-minute Rest + Normalizing Repetition Assessment

	Torque (N·m) M(SD)	*t*	*p*	Mean Difference	95% CI	Hedges' g
Pair 1						
Observed	29.13(8.42)	5.52	.001*	.476	[.691, 2.62]	1.75
Poly2	28.65(8.35)					
Pair 2						
Observed	29.13(8.42)	6.46	.001*	.158	[.871, 3.01]	1.96
Poly4	29.97(8.43)					
Pair 3						
Observed	29.13(8.42)	8.24	.001*	1.30	[1.20, 3.76]	2.50
Exp	27.83(8.26)					

Note. Observed = collected passive torque, Poly2 = predicted passive torque from second-order polynomial model, Poly4 = predicted passive torque from fourth-order polynomial model, Exp = predicted passive torque from an exponential model

*denotes significance of $p < .001$ in comparison to the observed assessment

Table 8.

Normalized Mean Muscle Activity

	Biceps Femoris M(SD)
Traditional	.042(.029)
1-minute	.044(.031)
3-minute	.045(.037)
5-minute	.041(.026)

Note. 1-, 3-, and 5-minute denotes the rest period taken prior to the normalizing repetition ensuring that the starting position was at 60° range of motion

Discussion

The first part of the results of the current study show that RMSE values were significantly lower in the experimental conditions compared to the traditional assessment. Comparisons among the experimental conditions were not significantly different. These findings show that the novel assessment of passive torque exhibit a better fit to the three mathematical models than the traditional assessment. This could indicate that PFE was present during the traditional assessment, and that when the muscle was returned to its resting length, as shown by Herzog and Leonard (2002), a more appropriate passive state was achieved during the experimental conditions. Therefore, it is likely that PFE was not contributing to force production during the experimental conditions. Both interpretations can be seen as there were differences between the traditional assessment and each experimental condition, but no differences between the experimental conditions in which the common methodological consideration was returning the hamstring to its resting length.

According to the Winding Filament Hypothesis put forward by Nishikawa et al. (2012), the N2A segment of titin binds to actin in a calcium dependent manner. This binding of titin to actin shortens and stiffens its free length. This phenomenon was shown in *mdm* mice (Monroy et al., 2011), characterized by the deletion of the N2A region of titin (Garvey et al., 2002). In this investigation, wild type mice exhibited a shift in the force-length curve to shorter muscle lengths and a stiffening of titin during muscle activation (Monroy et al., 2011). However, in these *mdm* mice, there was no change in the stiffness of titin (Monroy et al., 2011). This could indicate that titin may be attached to actin in the traditional assessment, therefore, affecting how force is produced. As there was a better fit among the experimental conditions, this could indicate that the novel methodology was able to unwind titin from actin giving a better representation of the passive force curve in the hamstrings.

Length-tension relationship diagrams often include an active portion, intended to represent the tension produced from contractile coupling between actin and myosin, and a passive portion representing the parallel and series elastic components of the musculoskeletal complex. The passive portion of the length-tension relationship often is represented as slowly introducing minimal force. As the musculotendinous tissue lengthens there is a notable exponential rise in force, producing a curvilinear graph. It should be noted that the passive portion graphs were most often created from obtained disarticulated, deactivated muscle. When the torque curves were processed for this study, the curves for the experimental sessions (1-min, 3-min, and 5-min) were noticeably curvilinear and the traditional sessions were noticeably more linear. It is interesting that a main purpose of the experimental session was to unwind (reset) the influence of titin in

the torque measure. Additionally, the mathematical models used in this study are most often used to extrapolate curvilinear relationships rather than linear relationships. Given that the current study consistently found (in all three models) that the traditional session had significantly higher RMSE values than the experimental sessions and that nature of the curves were observable different, it is likely that PFE played a role in the passive torque curve of the traditional session.

The second part of the results show that the predicted maximal passive torque values calculated by the three mathematical models was significantly different than the observed maximal passive torque. These findings are in line with those of Nordez et al. (2006), who showed significant differences at their maximal range of motion of testing. However, though the values were statistically different, the actual differences for all models, specifically the fourth-order polynomial model were likely to be practically insignificant.

There are several limitations of the current study that should be noted. First, the study involved a small (n = 10) sample size, and two trials per testing condition. Second, the second-order polynomial and the exponential mathematical models have not been used in passive assessment in the clinical setting. Third, the data points used during the fitting of the mathematical models were collected and not extrapolated, therefore, potentially reducing the amount of data points used in the fitting process. Lastly, there is no clear parameter that should be used in the assessment of the validity of a specific mathematical model. As such, future studies should continue to fit torque-angle data to different mathematical models, as well as experimenting with different measures of model fit, to elucidate the best method to measure model fit and predict passive torque.

Chapter IV References

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CHAPTER V: OVERALL CONCLUSIONS

This dissertation focused on a methodology to assess passive torque and stiffness in the hamstrings and to use this information to fit different mathematical models calculating stiffness indices. Study one implemented a novel methodology to assess passive torque in the hamstrings in comparison to a traditional methodology. Study two examined three mathematical models used to calculate torque/stiffness.

Study one required healthy males to perform a total of eight passive knee extension movements across four conditions. The first condition was a traditional method used to assess passive torque. The other three conditions consisted of a 1-, 3-, and 5-minute rest period, followed by a normalizing repetition to ensure the hamstring was at its resting length before the collection of passive torque data. Our hypotheses were partially supported as the experimental conditions results in lower passive torque values compared to the traditional assessment. However, there was no different between the experimental conditions beyond a 1-minute rest period, as it relates to modifying rest time prior to assessment.

Study two required fitting the torque-angle data collected during study one to three mathematical models that have been used in the literature to calculate passive torque and stiffness. The experimental data from each of the four testing conditions were fit to a second-order polynomial model, a fourth-order polynomial model, and an exponential model, and a goodness-of-fit index (GFI) was calculated. The root mean squared error (RMSE) of the GFI was used for comparison of models across each trial. Further, passive stiffness was calculated for each model and compared to the experimental data at the largest range of motion.

Our first hypothesis was partially supported, as RMSE values were all significantly lower in each experimental condition compared to the traditional. The fourth-order polynomial model showed the overall lowest RMSE. However, this difference would unlikely be practically significant. Our second hypothesis was rejected, as the differences between the predicted and observed passive stiffness were significantly different for each model across each condition. In line with the results of the first hypothesis, though the differences between observed and calculated passive stiffness were significantly different, the practicality of the differences were unlikely to be meaningful.

Due to emerging evidence regarding residual and passive force enhancement, methodologies used to assess passive torque from a clinical perspective have been viewed in a new light. Specifically, Herzog and Leonard (2002) showed that passive force enhancement persisted longer than expected and continued until the muscle was returned to its resting length. Further, Nishikawa et al. (2012) have put forward a hypothesis detailing how titin may function. Based on the results of the current studies, the novel methodology used to assess passive resistance was shown to be superior to the traditional assessment. However, though the methodology showed lower passive resistance values, no one mathematical model was shown to predict passive stiffness better than the rest. These results are in line with previous research conducted by Nordez et al. (2006). Therefore, it is suggested that the novel assessment of passive resistance be considered instead of the traditional methodology. It is of interest to use the novel methodology under differing situations to continue to assess it validity and reliability.

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