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Clinical Measures Predicting Knee Extensor Muscle Activation During a Maximal Voluntary Isometric Contraction

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Abstract

Strength of a muscle group is determined by its physiological cross-sectional area and one's ability to activate the muscle group. Injury and disuse are associated with a decreased ability to maximally activate a muscle group. Clinical orthopedic measures such as manual muscle testing may provide an indication of muscle strength but no direct insight into the extent a muscle group is being activated. The objective of our studies was to determine the standard clinical orthopedic measures and/or medical history that can best predict muscle activation during a maximal voluntary isometric contraction (MVIC) of the knee extensors. Forty young, healthy subjects completed a pilot study (Study A), which included a medical history assessment and a comprehensive battery of lower-extremity orthopedic tests. The relation of these measures to MVIC knee extensor activation measured via the interpolated-twitch technique was determined. Subjects (n=108) completed a follow-up study (Study B) which further analyzed the best predictors determined from Study A, i.e., hip flexibility, myofascial trigger points (MTrP), and medical history. MTrPs in the knee flexors, hip pain/stiffness, and a medical history of lower-extremity injuries were determined by stepwise regression to be the most predictive measures for knee extensor MVIC activation. Mean knee extensor activation was 5-7% higher when MTrPs were present in the knee flexors than when they were absent ($p \leq 0.006$). Subjects with a history of hip pain/stiffness had, on average, 9.5% higher activations than did subjects with no history of such injury ($p<0.001$). Subjects with a history of lower-extremity injury had, on average, 4% higher activations than did subjects with no history of such injury ($p=0.038$). These three measures were however, relatively poor predictors, explaining less than 20% of the between-subject and -leg variance in activation. In conclusion, none of the collected medical history nor clinical orthopedic measures that were tested can adequately predict knee extensor activation during a MVIC in young, healthy subjects.

Keywords : Interpolated twitch technique; quadriceps; myofascial trigger point; strength

Introduction

Strength is determined not only by the anatomical properties of muscle (e.g., number of muscle fibers, muscle fiber diameter, fiber pennation angle) but also by its neurophysiological properties (e.g., motor unit recruitment, rate coding, motor unit synchronization). Several studies have shown that the deficits in knee extensor strength found after knee injuries are attributable more to decreased knee extensor muscle activation than to muscle atrophy due to disuse [1-3]. The inability to maximally activate the knee extensors in the absence of frank tissue damage can alter athletic performance, which often relies on optimal contraction of this lower-extremity muscle group [1].

The interpolated twitch and central activation ratio methods are considered to reliably assess the percent of a muscle group's mass that is activated during a voluntary contraction [4, 5]. Interestingly, the knee extensors have, on average, been observed to have a relatively low activation during a maximal voluntary isometric contraction (MVIC) compared to other muscles or muscle groups, including the biceps, brachialis, adductor pollicis, tibialis anterior, and ankle plantarflexors [5]. Activation of the knee extensor during a MVIC in otherwise healthy individuals has been reported on average to be in the 85-95% range, while the MVIC activation for other muscle groups exceeds 95%. We have reported a similar mean value for the knee extensors (i.e., 86%) in apparently healthy young adults but individual values in the study ranged greatly from 64 to 99% [6]. The lower knee extensor activation levels observed in healthy individuals may not affect typical activities of daily living, but they could limit athletic performance and/or affect injury prevention.

Knowing knee extensor activation during a MVIC could be useful clinical data for optimizing athletic performance, determining an athlete's ability to return to sport, and design of strengthening and rehabilitation programs. While it is possible to assess activation by performing the interpolated twitch and central activation ratio methods in the research laboratory, the techniques are not practical to perform in a clinical setting or on the training field because they require expertise and training not possessed by most clinicians, and utilize equipment not commonly found in the clinic. Also, the techniques are often

considered uncomfortable by some subjects because of the stimulation of afferents, including those for pain, during muscle electrical stimulation. There are some clinical measures such as manual muscle testing and dynamometry that can provide a clinician with an assessment of strength but yield no direct insight into how much of a muscle is being activated during a contraction.

A means of predicting knee extensor activation without employing the interpolated twitch or central activation ratio method would be highly desirable. Clinical orthopedic measures combined with an athlete's medical history may be able to estimate MVIC knee extensor activation sufficiently enough for the clinician to evaluate and/or treat his/her patient. It would be ideal if the assessment could be easily performed in the clinic or on the training field. The objective of the studies reported here was to determine which clinical orthopedic measures and/or medical history could be used to estimate knee extensor muscle activation during a MVIC in an apparently healthy person.

Methods

Experimental approach to the problem

A pilot study, Study A, was conducted using medical history and an extensive battery of musculoskeletal clinical tests of both lower extremities to determine whether any or a combination thereof could predict knee extensor activation during a MVIC. A comprehensive follow-up study, Study B, was conducted employing more subjects and more extensive testing of the measures found from Study A to be predictive of muscle activation during a MVIC.

Subjects

Study A and B subjects were in the age range of 18-40 years and were recruited from the university and surrounding community. Potential subjects were screened by independent investigators and excluded from participation if they had a history of fibromyalgia or neuropathy, any significant lower-extremity injury (i.e., pain reported with a change in normal function lasting longer than 5 consecutive days) or surgery within the last year, and any known allergy to nickel in order to prevent a reaction to a component of the stimulation electrodes. Prior to testing, subjects in both studies completed a detailed medical history questionnaire that included physical characteristics as well as current and past medical history of any general medical or orthopedic condition. Table 1 lists the general categories of assessments used in the medical history questionnaire. Any admissions of prior or current injury and/or stiffness were clarified as to the affected side and when the injury occurred. An injury or complaint was considered current if it was present within the past week, and subjects were excluded if the injury met the exclusion criteria previously mentioned. Subjects in Study A consisted of 16 males and 24 females with a mean (±SD) age of 27.0 (±5.7) years. Subjects completing Study B consisted of 44males and 64 females with a mean age of 26.1 (±4.0) years. Three subjects did not complete Study B due to discomfort from the muscle electrical stimulation during the interpolated twitch technique and all data for these subjects was excluded from

Table 1: Medical history form for studies A and B.

Question:	**Response:**		
Demographics:			
Age	____ years		
Sex	Male or Female		
Weight	____ pounds / _____ kilograms		
Height	___ feet, _____ inches / _____ inches		
General Medical History	**Yes**	**No**	**Past or Current**
Anemia			
Arthritis			
Asthma/allergies			
Cancer/tumors			
Diabetes			
High cholesterol			
Hypertension			
Migraines			
Smoking history			
Stomach/intestinal problems			
Thyroid disorder			
Orthopedic Medical History	**Yes**	**No**	**Past or Current**
Back pain/stiffness			
Spine congenital deformity			
Degenerative disc disease of the spine			
Herniated disc of the spine			
SI joint disorder			
Scoliosis			
Spine fracture			
Pelvis or hip dislocation			
Groin strain			
Hip pain or stiffness			
Hip surgery			
Hip fracture			
Thigh muscle strain			
Thigh fracture			
Thigh pain			
Anterior cruciate ligament tear or repair			
Posterior cruciate ligament tear or repair			
Medial collateral ligament tear or repair			
Lateral collateral ligament tear or repair			
Iliotibial band syndrome			
Lower leg pain or stiffness			
Lower leg muscle strain			
Ankle pain or stiffness			
Ankle or foot tendinitis			

analysis. These studies were approved by the Georgia State University institutional review board and all subjects provided written informed consent.

Procedures

Study A (pilot study)

Table 2 provides a detailed listing of the 19 clinical orthopedic tests performed in Study A. Two independent investigators, blinded to the subject's history, examined each subject bilaterally for anterior and posterior cruciate ligament laxity, lower-extremity muscle tightness, and bony alignment. A knee laxity testing device(MED metric model KT 1000, San Diego, CA) was used to assess knee ligament laxity [7, 8]. Flexibility measurements of the lower extremities were performed using a standard goniometer, and included hip internal and external rotation measurements taken seated and prone [9], knee flexor (i.e., hamstrings) muscle length measured supine [9, 10], and hip joint flexibility when measured during maximal extension using the Modified Thomas Test [10, 11]. The Q-angle measurement of the knee, which gives an estimate of the line of pull of the vastus medialis, was measured standing using a standard goniometer [9]. Measurements from the two investigators were averaged.

Table 2: Clinical tests and measures performed in Study A.

Clinical Test or Measure	Clarification of Assessment
Knee ligament laxity– ACL ligament	KT 1000 test procedure with apparatus attached to knee while investigator pulls anterior on apparatus for ACL and pushes posterior for PCL
Knee ligament laxity– PCL ligament	
Hip internal rotation (seated)	Standard goniometer for measuring joint angle
Hip internal rotation (prone)	
Hip external rotation (seated)	
Hip external rotation (prone)	
Knee flexors muscle length	Subject positioned supine in 90° hip flexion and 90° knee flexion, then knee extended and angle measured with standard goniometer
Modified Thomas Test for hip joint flexibility during maximal hip extension	Subject positioned on edge of table supine with bilateral hip and knees maximally flexed, posterior pelvic tilt maintained while lowering one leg and hip angle measured when unable to maintain posterior pelvic tilt
Q angle standing – line of pull on patella	Angle formed with line bisecting the patella vertically and line from mid-patella to anterior superior iliac spine
Active MTrP in rectus femoris	Muscle palpated for both presence (yes/no) and total number
Latent MTrP in rectus femoris	
Active MTrP in vastus medialis	
Latent MTrP in vastus medialis	
Active MTrP in vastus lateralis	
Latent MTrP in vastus lateralis	
Active MTrP in lateral knee flexors	
Latent MTrP in lateral knee flexors	
Active MTrP in medial knee flexors	
Latent MTrP in medial knee flexors	

Abbreviations: ACL, anterior cruciate ligament; PCL, posterior cruciate ligament; MTrP, myofascial trigger point

The presence of active and/or latent myofascial trigger points (MTrP) in the bilateral knee extensors and flexors was determined independently by two investigators, with a MTrP considered to exist only if both investigators agreed on its presence. These two investigators were different from the investigators measuring lower extremity ligament laxity, muscle flexibility and bony alignment. Each individual muscle within the knee extensors and flexors was palpated, using trigger point palpation as outlined by Travell and Simons [12]. The first step was to explain the process of palpation and the numeric pain scale to the subject [13]. If the subject experienced pain equal to or greater than 4 out of 10 with palpation, a MTrP was considered to be identified. If palpation elicited a familiar pain for the patient, the MTrP was recorded as active; if the pain was unfamiliar, the MTrP was recorded as latent.

To assess knee extensor activation, the interpolated twitch technique was performed as we have described previously [6, 14]. The participant was seated in a KinCom III dynamometer (Chattecx, Chattanooga, TN) in a semi-reclined position with 110° of hip flexion and 70° of knee flexion. Two 7x10-cm adhesive electrodes (UniPatch 620SS, Wabasha, MN) were placed on the skin overlying the thigh, one over the distal vastus medialis muscle and the other over the proximal vastus lateralis muscle near the anteroinferior iliac spine of the ilium. The electrodes were connected to a constant-current stimulator (Digitimer model DS7AH, Hertfordshire, England) that was controlled using a 667-MHz Pentium computer, an A/D- and D/A-interface board (Keithley Instruments model KPCI-3108, Cleveland, OH), and custom-written software created with Test Point version 7.0 (Capital Equipment Co., Billerica, MA). The software and interface board also sampled the torque output signal from the KinCom III dynamometer at 5 kHz.

To determine the stimulation current needed for the interpolated-twitch contractions, a series of electrically-stimulated isometric contractions of the knee extensors was performed with the current being increased on successive stimulations. Each stimulation consisted of a paired-pulse stimulation, that is, two 0.2-ms pulses separated by 10 ms. Stimulator current was initially set to 100 mA and increased by 20 mA on each successive stimulation. Progressively stronger stimulations were delivered once every 20 s until the peak contraction torque reached a plateau and then showed a decline on two successive stimulations. The current eliciting the highest peak torque on the plateau of the torque–current curve was used for the remainder of the test session.

For the interpolated-twitch contractions, the participant was instructed to perform a 3-s MVIC of his or her knee extensor muscles. Auditory cues elicited by the custom-written software were used to signal the participant to start and stop the contraction. At 2.5 s into the MVIC, the muscle group was

stimulated with a paired-pulse stimulation, and the increase in torque over the MVIC level (i.e., interpolated-twitch torque [ITT]) was measured. At 2 and 4 s after the end of the MVIC, the paired-pulse stimulation was administered to relaxed muscle to determine peak electrically-evoked torque (EET); the average value for the two stimulations was used in the data analyses. The percentage muscle activation during MVIC was calculated as 100% x [1 – (ITT/EET)]. During the test session for each leg, six interpolated-twitch contractions were performed, with 1 min of rest between contractions. Of the six interpolated-twitch contractions, the three best attempts were determined and their data averaged together for use in subsequent analyses. The three best attempts were considered the three trials with the highest voluntary torques that also showed less than 10% variation across the plateau of the voluntary torque–time curve.

Test-retest reliability was assessed on all continuous measures in the study using 12 of the 40 subjects. These subjects were retested one week after their initial test session and intraclass correlations were calculated. The median intraclass correlation was 0.89, with the lowest being 0.84 for the hip joint flexibility assessment. It should be noted that these two values are above the reliability coefficient of 0.75 which Portney et al [15] states as good reliability.

Study B (comprehensive follow-up study)

Prior to initiation of the testing session, subjects completed a 5-minute warm-up on a Monarkcycle ergometer at a resistance eliciting a heart rate equivalent to ~60% of their estimated maximal heart rate; exercise at this percentage of maximal heart rate is roughly equivalent to 50% of maximal aerobic capacity.

Table 3 provides a detailed listing of the 27 clinical tests performed in Study B. These tests were performed bilaterally to assess hip joint flexibility when measured during maximal extension and the presence of MTrP in the knee extensors

Table 3: Clinical tests and measures performed in Study B.

Clinical Test or Measure	**Clarification of Assessment**
Modified Thomas Test for hip joint flexibility during maximal extension with knee flexed 80°	ImageJ software utilized to analyze sagittal image and calculate the hip joint angle. The psoas muscle was considered tight if the angle was greater than 0°.
Modified Thomas Test for hip joint flexibility during maximal extension with knee extended to 0°	ImageJ software utilized to analyze sagittal image and calculate the hip joint angle. The rectus femoris was considered tight if the difference between the MTT with the knee extended at 0° and knee flexed at 80° was greater than 0°.
Ilitiotibial Band tightness assessed via the Modified Thomas Test for hip joint flexibility during maximal extension with the knee flexed 80°	If the subject's lower extremity abducted in the sagittal plane during the test then the IT Band was considered tight.
Active MTrP in the vastus medialis	Presence of at least one MTrP was recorded in the muscle region
Latent MTrP in the vastus medialis	
Active MTrP in the distal 1/3 rectus femoris	
Latent MTrP in the distal 1/3 rectus femoris	
Active MTrP in the middle 1/3 rectus femoris	
Latent MTrP in the middle 1/3 rectus femoris	
Active MTrP in the proximal 1/3 rectus femoris	
Latent MTrP in the proximal 1/3 rectus femoris	
Active MTrP in the distal 1/3 vastus lateralis	
Latent MTrP in the distal 1/3 vastus lateralis	
Active MTrP in the middle 1/3 vastus lateralis	
Latent MTrP in the middle 1/3 vastus lateralis	
Active MTrP in the proximal 1/3 vastus lateralis	
Latent MTrP in the proximal 1/3 vastus lateralis	
Active MTrP in the distal 1/3 medial knee flexors (semimembranous and semitendinous)	
Latent MTrP in the distal 1/3 medial knee flexors (semimembranous and semitendinous)	
Active MTrP in the middle 1/3 medial knee flexors (semimembranous and semitendinous)	
Latent MTrP in the middle 1/3 medial knee flexors (semimembranous and semitendinous)	
Active MTrP in the distal 1/3 lateral knee flexors (biceps femoris)	
Latent MTrP in the distal 1/3 lateral knee flexors (biceps femoris)	
Active MTrP in the middle 1/3 lateral knee flexors (biceps femoris)	
Latent MTrP in the middle 1/3 lateral knee flexors (biceps femoris)	
Active MTrP in the proximal knee flexors (semimembranous, semitendinous, biceps femoris)	
Latent MTrP in the proximal knee flexors (semimembranous, semitendinous, biceps femoris)	
Abbreviations: MTT, Modified Thomas Test; IT Band, iliotibial band; MTrP, myofascial trigger point	

and flexors. These tests were identified in Study A as the best clinical test predictors of knee extensor muscle activation. Two independent investigators assessed hip joint flexibility. Hip joint flexibility into extension was assessed using the Modified Thomas Test, further modified as described by Kendall [10], by holding the ipsilateral knee at two angles, i.e., 80° and 0°of knee flexion. This clinical test gives the examiner an indication of the relative lengths of the psoas, rectus femoris and iliotibial band muscles. Reflective markers were placed on the subject's greater trochanter and lateral epicondyle of the knee. While maintaining the contralateral lower extremity in full hip and knee flexion and the ipsilateral knee at 80°, the ipsilateral lower extremity was lowered into maximal hip extension and a digital photograph was taken from the sagittal plane, capturing the markers' placement. The angle between the horizontal and the line bisecting the two markers was measured usingImageJ software (National Institutes of Health, Bethesda, MD) and was considered to be the maximum extension angle for the hip. The test was repeated with the ipsilateral knee fully extended. This testing position minimizes rectus femoris muscle length from limiting hip joint flexibility. If the measured angle was greater than 0° (i.e., leg above the horizontal), then the psoas was considered tight. Iliotibial band tightness was noted if the lower extremity abducted in the frontal plane past the neutral position when the fully extended ipsilateral knee was lowered. Rectus femoris was assumed to limit hip flexibility if the maximum hip extension angle was greater when the ipsilateral knee was maintained at 80° as compared to when the knee was fully extended.

Palpation for the presence of active and latent MTrP was done by two different independent investigators using the technique described in Study A with an additional analysis of the data by muscle regions. Muscle palpation was done in this order: the vastus medialis, rectus femoris (divided into proximal, middle, and distal segments), vastus lateralis (divided into proximal, middle and distal segments), biceps femoris (divided into distal and middle segments), semimembranous and semitendinous(divided into distal and middle segments)and the proximal knee flexors (Figure 1). The two testers compared results, discussed any discrepancies present, and if necessary, re-palpated to address the discrepancies. Both the order of testers and which lower extremity was assessed first were randomly assigned for each subject. After all clinical tests, knee extensor activation during a MVIC was assessed for both legs as described for Study A.

Statistical Analyses

To determine the relationship of knee extensor percent muscle activation to the subject characteristics, medical history and clinical test measures, Pearson correlations and independent t-tests, or Mann-Whitney tests where appropriate, were used for assessing continuous and dichotomous nominal predictor variables, respectively. Stepwise regression was used to determine the best combination of variables for predicting muscle activation. Variables permitted in the regression model included age, weight, orthopedic medical history for the lower extremity, and all 27 clinical tests in Study B. All statistical tests

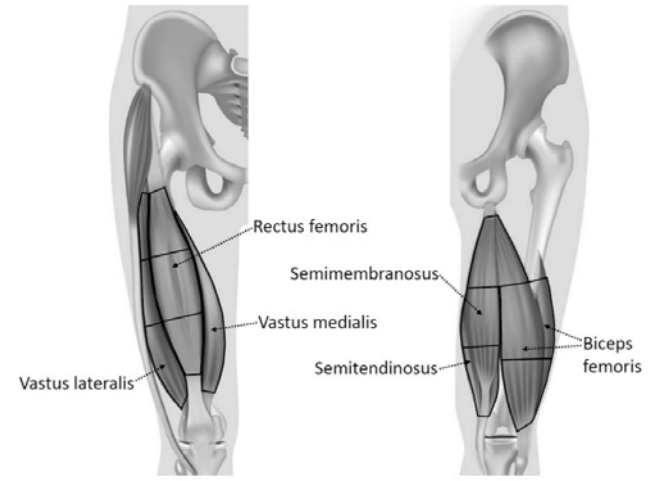

Figure 1: Regions of knee extensor and knee flexor muscle groups as divided for myofascial trigger point palpation in Study B.

were performed using SPSS (ver. 18). An αlevel of 0.05 was used for all tests. Adjustments to the α level were not made for the repeated use of independent t-tests (or Mann-Whitney tests) and correlations because of the exploratory nature of those analyses.

Results

Study A (pilot study)

For the 40 subjects and 80 legs examined in Study A, the median knee extensor activation during a MVIC was 83.3%, with individual values ranging from 56 to 100%. For all clinical measures in Table 2, hip flexibility, as assessed by the Modified Thomas Test, was the only clinical measure by itself that was significantly correlated with knee extensor activation during a MVIC. This correlation was present for both ipsilateral and contralateral lower extremities($r = 0.24$-0.25, $p \leq 0.032$). Here, a positive correlation means that greater knee extensor activation was associated with poorer hip flexibility (i.e., thigh could not be lowered to the horizontalor below during the Modified Thomas Test).

As determined by stepwise regression, the best combination of clinical predictors in the pilot study for knee extensor activation included four variables: contralateral Modified Thomas Test angle, presence of active MTrP in the ipsilateral knee flexors, presence of any type of MTrP in the ipsilateral knee extensors, and history of anterior cruciate ligament (ACL) tear or repair ($R^2 = 0.224$, $p=0.001$) (Table 4). The positive coefficient for presence of active MTrP in the knee flexors indicates that knee extensor activation was higher if the subject had active MTrP in the knee flexors. The negative coefficients for both presence of any MTrP in the knee extensors and history of ACL tear/reconstruction indicates that knee extensor activation was lower for when MTrP were present in the knee extensors and when the subject had a history of ACL tear or reconstruction.

Study B (comprehensive follow-up study)

For the 108 subjects and 216 legs examined in the more

Table 4: Study A – Stepwise regression analysis showing 4 models of clinical predictors for knee extensor muscle activation.

Model	Standardized Coefficient, Beta	R^2	P value
Model #1			
1. Contralateral MTT angle	0.245	0.060	0.029
Model #2			
1. Contralateral MTT angle	0.274	0.121	0.007
2. Ipsilateral Active Knee Flexor MTrP	0.250		
Model #3			
1. Contralateral MTT angle	0.274	0.177	0.002
2. Ipsilateral Active Knee Flexor MTrP	0.293		
3. Ipsilateral Knee Extensor MTrP	-0.241		
Model #4			
1. Contralateral MTT angle	0.310	0.224	0.001
2. Ipsilateral Active Knee Flexor MTrP	0.280		
3. Ipsilateral Knee Extensor MTrP	-0.224		
4. ACL Tear or Repair	-0.221		

Abbreviations: MTT, Modified Thomas Test; MTrP, myofascial trigger point; ACL, anterior cruciate ligament

comprehensive follow-up study (Study B), the median knee extensor activation was 86.5% with individual values ranging from 39 to 100%. For all clinical measures considered in Table 3, the presence of latent MTrP in the proximal portion of the ipsilateral knee flexors or in the distal lateral portions of the ipsilateral or contralateral knee flexors were each associated with higher knee extensor activation (Table 5). The mean knee extensor activations were 5.2-6.6% higher when MTrPs were present in any of these three regions than when they were absent ($p \leq 0.006$). The number of regions in the lateral knee flexors with MTrP was positively but weakly correlated with activation in both the ipsilateral and contralateral knee extensors ($r=0.15$-0.19; $p<0.05$) (Table 6). The number of regions in the proximal knee flexors with MTrP was also positively correlated with activation in both ipsilateral and contralateral knee extensors ($r=0.12$-0.14; $p<0.05$). The number of regions in the knee extensors with MTrP, active or latent, was not significantly associated with knee extensor activation.

Several dichotomous medical history variables were associated with knee extensor activation during a MVIC (Table 5). Subjects with a history of any lower-extremity injury had on average a 3.9% higher knee extensor activation than those without previous injury ($p=0.038$). To further delineate the type of lower-extremity injury history associated with higher activations, it was found that subjects with a history of lower-leg pain or muscle strain had 10.3-11.5% higher knee extensor activations than those without ($p \leq 0.002$). Interestingly, the number of prior or current lower-extremity injuries was positively correlated with knee extensor activation ($r=0.14$-0.17; $p<0.05$) (Table 6).

As determined by stepwise regression, the best combination of clinical measures and/or medical history for predicting knee extensor activation included four variables: the number of ipsilateral lower-extremity injuries, the number of regions in the ipsilateral knee flexors with latent MTrP, a history of ipsilateral hip pain, and the presence of MTrP in the ipsilateral knee flexor muscles ($R^2=0.118$, $p<0.001$) (Table 7). The positive coefficients for the first three variables in the model indicate that knee extensor activation was higher in subjects who had a greater number of lower-extremity injuries, a greater number of regions of the lateral knee flexor muscles with latent MTrP, and a history of hip pain. The fourth variable in the model (i.e., a presence of MTrP in the knee flexor muscles) had a negative coefficient, which would as least partially offset the second variable in the model (i.e., number of regions of the lateral knee flexor muscles with latent MTrP). Interestingly, unlike in Study A, history of an ACL tear or reconstruction had no effect on knee extensor activation either individually or in combination with other variables.

Discussion

The objective of our studies was to identify commonly-used clinical orthopedic measures that could be used to predict the extent of knee extensor activation during a MVIC. In our pilot study (Study A), we screened a number of clinical measures and in Study B, we followed up with a more comprehensive examination of the more promising measures using a larger and more varied sample. MTrPs in the knee flexors, hip pain/stiffness, and a medical history of lower-extremity injuries were the most predictive measures for knee extensor activation during a MVIC. However, these measures, though statistically significant, were weak predictors and accounted for less than 20% of the between-subject and -leg variance in activation.

Stepwise regression in both studies revealed that the presence of MTrP in the knee flexors was predictive of higher knee extensor muscle activation. The effect of MTrP on muscle function has not been widely studied. There is evidence that the presence of MTrP can cause dysfunction within the muscle it resides by altering the motor pattern, i.e., recruitment and timing of muscle activation, possibly leading to a decrease in strength [16-19]. It has also been shown that the presence of latent MTrP can cause an increase in intramuscular EMG activity and alter the synergistic muscle firing pattern during movement [20]. These

Table 5: Study B – Analysis of dichotomous nominal variables as potential predicators of knee extensor muscle activation.

Nominal Variable (presence vs. absence)	% Muscle Activation (Mean ± SD)
Hip flexibility:	
Ipsilateral ITB tightness	82.3±13.3 vs 84.5±12.9
Ipsilateral rectus femoris tightness	83.4±12.7 vs 82.7±14.9
Ipsilateral psoas tightness	82.9±13.7 vs 83.8±12.3
Myofascial Trigger Points:	
Latent MTrP in ipsilateral distal lateral knee flexors	88.9±8.8 vs 82.3±13.5 †
Latent MTrP in contralateral distal lateral knee flexors	87.7±8.4 vs 82.5±13.7 †
Latent MTrP in ipsilateral proximal knee flexors	89.5±4.7 vs 82.9±13.4 *
MTrP in the knee extensor muscles	83.8±13.2 vs 82.7±13.2
MTrP in the knee flexor muscles	84.1±13.6 vs 82.9±12.9
Medical History:	
History of lower-extremity injury	84.6±12.4 vs 80.7±14.3 *
History of ipsilateral hip injury	86.8±12.8 vs 82.1±13.1 *
History of ipsilateral hip pain and stiffness	92.0±6.6 vs 82.5±13.3 †
History of contralateral hip pain and stiffness	89.6±8.5 vs 82.7±13.4 †
History of ipsilateral knee injury	85.9±11.9 vs 81.9±13.6 *
History of ipsilateral lower leg pain	94.5±3.0 vs 83.0±13.2 †
History of ipsilateral lower leg muscle strain	93.2±5.8 vs 82.9±13.2†
History of ipsilateral ankle pain	91.1±7.1 vs 82.2±13.4 †
History of ipsilateral ACL tear/repair	83.2±12.1 vs 83.3±13.2
History of back pain and stiffness	85.8±14.5 vs 82.7±12.9
Current ipsilateral knee pain/stiffness	86.6±11.1 vs 82.8±13.4
Gender (male vs. female)	84.5±11.6 vs 81.4±15.1

Variables in the table are variables identified from Study A.as being of interest.
Abbreviations: ITB, iliotibial band; MTrP, myofascial trigger point; ACL, anterior cruciate ligament
* denotes significant at p<.05
† denotes significant at p<.01

Table 6: Study B – Pearson correlations of continuous variables with knee extensor muscle activation.

Continuous Variable	Correlation with Ipsilateral Percent Muscle Activation	Correlation with Contralateral Percent Muscle Activation
Modified Thomas Test:		
MTT – hip angle with knee at 80° flexion	-0.002	0.013
MTT – hip angle with knee at 0° flexion	0.031	0.040
Myofascial Trigger Point:		
Number of regions of rectus femoris with MTrP	0.092	0.050
Number of regions in vastus lateralis with MTrP	0.031	0.006
Number of regions in vastus medialis with MTrP	0.062	0.097
Number of regions in lateral knee flexors with MTrP	0.193*	0.153*
Number of regions in medial knee flexors with MTrP	-0.024	-0.046
Number of regions in proximal knee flexors wthMTrP	0.115*	0.139*
Total MTrP in knee extensor	0.062	0.050
Total MTrP in knee flexors	0.109	0.082
Total MTrP in knee extensors and flexors	0.103	0.079
Medical history:		
Number of prior LE injuries	0.167*	0.140*
Number of current LE injuries	0.159*	0.139*
Age	0.145*	
Height	-0.025	
Weight	-0.071	

Abbreviations: MTT, modified Thomas test; MTrP, myofascial trigger point; LE, lower extremity
* denotes correlation is significant at p<.05

Table 7: Study B – Stepwise regression analysis showing 4 models of clinical predictors for knee extensor muscle activation.

Model	Standardized Coefficient, Beta	R^2	P value
Model #1		0.043	0.002
1. Total # LE injuries on the ipsilateral side	0.245		
Model #2		0.068	0.001
1. Total # LE injuries on the ipsilateral side	0.178		
2. Total # regions with latent MTrP in the ipsilateral lateral knee flexors	0.160		
Model #3		0.094	<0.001
1. Total # LE injuries on the ipsilateral side	0.132		
2. Total # regions with latent MTrP in the ipsilateral lateral knee flexors	0.171		
3. History of ipsilateral hip pain and stiffness	0.168		
Model #4		0.118	<0.001
1. Total # LE injuries on the ipsilateral side	0.155		
2. Total # regions with latent MTrP in the ipsilateral lateral knee flexors	0.284		
3. History of ipsilateral hip pain and stiffness	0.165		
4. Presence of MTrP in the ipsilateral knee flexor muscles	-0.194		

Abbreviations: #, number; LE, lower extremity; MTrP, myofascial trigger point

studies do not however help explain our observations, which imply that MTrPs can affect muscles in which they do not reside. We are unaware of any evidence for MTrP having effects, much less beneficial effects, on antagonistic muscle function. The knee flexor MTrPs appear to have an excitatory effect on the knee extensors analogous to how activation of a Golgi tendon organ (GTO) has an excitatory effect on antagonist muscles. This plus the previous observation by Lucas and colleagues [17, 18] of MTrP having inhibitory effects on the muscle in which they reside is consistent with the action of a reflex loop similar to a GTO-mediated one. It is interesting that Study A did find active MTrPs in the knee extensors to be predictive of decreased knee extensor activation which is consistent with the findings of Lucas et al [18] as well as our hypothesized GTO-like reflex loop. However, the Study A finding was not confirmed in Study B even though 60% of the lower extremities tested had MTrPs in the knee extensors. In retrospect, we wished that we had assessed muscle activation in both the knee flexors as well as the knee extensors. Doing this combined with the MTrP assessment in both muscle groups could have added clarity to our reflex loop hypothesis.

Unlike the research showing that lower-extremity pain and injury can cause a decrease in muscle activation and strength [1, 2, 21] we found a history of lower-extremity injury and hip pain/stiffness to be predictive of higher knee extensor activation. We do not believe the injury/pain relationship with knee extensor activation is causal in nature. We instead speculate that athletic individuals are more likely to have accrued injuries over their years of training and that these individuals are also likely to have higher knee extensor activations again because of their years of training. Unfortunately, we did not assess the subjects' past and present training regimens nor their level of athleticism. We can only state that we had a young, active and generally healthy subject pool.

Having an ACL tear and/or reconstruction has been shown to lead to an inhibition of the knee extensors and to decrease strength and muscle activation for up to 2 years post injury [1,21-25]. Our study's findings are not in congruence with this observation, i.e., ACL tear/reconstruction was not associated with knee extensor activation during a MVIC. Mean percent activations for subjects with and without an ACL injury history were nearly identical (i.e., 83.2% vs. 83.3%).However, only 8 (i.e., 3.7%) of the lower extremities studied in Study B had an ACL reconstruction.

We were puzzled by the lack of a relationship found between hip flexibility and knee extensor activation in Study B, whereas one was found in Study A. This might be explained by the fact that different testers were used in the two studies. Also, during the Modified Thomas Test, the knee was held at a constant 80 degrees of knee flexion in Study B whereas in Study A, the knee was flexed but not held at a specific angle.

In retrospect, we wished we had examined and evaluated the lumbar spine, specifically the L3/4 segments as these provide neural input to the knee extensors and could possibly affect muscle activation as shown by Grindstaff [26]. However, our medical history questionnaire did have several questions on low back injury and dysfunction and none were identified as predictors of knee extensor activation. While some functional movements and screening exams (i.e., vertical leap) are used to predict performance and/or injury in college and professional

athletes [27-29], we did not assess any functional movements in our studies. It would be interesting to see if such movements or screening exams are related to the degree of knee extensor activation during a MVIC.

Conclusions

We identified MTrPs in the knee flexors, hip pain/stiffness, and a medical history of lower-extremity injuries to be statistically significant predictors of knee extensor activation during the performance of a MVIC. However, these are weak predictors and will not be useful clinically for estimating knee extensor activation. Measures not used in our studies that warrant evaluation for predicting activation include those assessing the lumbar spine and/or functional movements as assessed during athlete screening exams. At the present time, we do not see a viable alternative that can estimate knee extensor activation during a MVIC in lieu of using the interpolated twitch or central activation ratio techniques.

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References

1. Hart JM, Pietrosimone B, Hertel J, Ingersoll CD. Quadriceps activation following knee injuries: a systematic review. J Athl Train. 2010;45(1):87-97. doi: 10.4085/1062-6050-45.1.87.
2. Meier W, Mizner RL, Marcus RL, Dibble LE, Peters C, Lastayo PC. Total knee arthroplasty: muscle impairments, functional limitations, and recommended rehabilitation approaches. J Orthop Sports Phys Ther. 2008;38(5):246-256. doi: 10.2519/jospt.2008.2715.
3. Mizner RL, Petterson SC, Stevens JE, Vandenborne K, Snyder-Mackler L. Early quadriceps strength loss after total knee arthroplasty. The contributions of muscle atrophy and failure of voluntary muscle activation. J Bone Joint Surg Am. 2005;87(5):1047-1053. doi: 10.2106/JBJS.D.01992.
4. Behm DG, St-Pierre DM, Perez D. Muscle inactivation: assessment of interpolated twitch technique. J Appl Physiol (1985). 1996;81(5):2267-2273.
5. Shield A, Zhou S. Assessing voluntary muscle activation with the twitch interpolation technique. Sports Med. 2004;34(4):253-267.
6. Park ND, Maresca RD, McKibans KI, Morgan DR, Allen TS,Warren GL. Caffeines enhancement of maximal voluntary strength and activation in uninjured but not injured muscle. Int J Sport Nutr Exerc Metab. 2008;18(6):639-652.
7. Daniel DM, Stone ML, Sachs R, Malcom L. Instrumented measurement of anterior knee laxity in patients with acute anterior cruciate ligament disruption. Am J Sports Med. 1985;13(6):401-407.
8. Lubowitz JH, Bernardini BJ, Reid JB, 3rd. Current concepts review: comprehensive physical examination for instability of the knee. Am J Sports Med. 2008;36(3):577-594. doi: 10.1177/0363546507312641.
9. Norkin CC, White DJ. Measurement of joint motion: a guide to goniometry. F.A. Davis. 2009.
10. Kendall FP, McCreary EK, Provance PG, Rodgers MM, Romani WA. Muscles testing and function with posture and pain 5th edition. 5th edition ed. Baltimore. MD: Lippincott Williams & Wilkins. 2010.
11. Peeler JD, Anderson JE. Reliability limits of the modified Thomas test for assessing rectus femoris muscle flexibility about the knee joint. J Athl Train. 2008;43(5):470-476. doi: 10.4085/1062-6050-43.5.470.
12. Simons DG, Travell JG, Simons LS. Myofascial pain and dysfunction: the trigger point manual. 2nd ed. Baltimore, MD: Lippincott Williams & Wilkins; 1999.
13. Hartrick CT, Kovan JP, Shapiro S. The numeric rating scale for clinical pain measurement: a ratio measure. Pain Pract. 2003;3(4):310-316. doi: 10.1111/j.1530-7085.2003.03034.x.
14. Cureton KJ, Warren GL, Millard-Stafford ML, Wingo JE, Trilk J, Buyckx M. Caffeinated sports drink: ergogenic effects and possible mechanisms. Int J Sport Nutr Exerc Metab. 2007;17(1):35-55. PubMed PMID: 17460332.
15. Portney LG, Watkins MP. Foundations of clinical research: applications to practice. Upper Saddle River, NJ: Pearson Education, Inc; 2009.
16. Celik D, Yeldan I. The relationship between latent trigger point and muscle strength in healthy subjects: a double-blind study. J Back Musculoskelet Rehabil. 2011;24(4):251-256. doi: 10.3233/BMR-2011-0302.
17. Lucas KR, Polus BI, Rich PA. Latent myofascial trigger points: their effects on muscle activation and movement efficiency. J Bodyw Move Ther. 2004;8:160-166.
18. Lucas KR, Rich PA, Polus BI. Muscle activation patterns in the scapular positioning muscles during loaded scapular plane elevation: the effects of Latent Myofascial Trigger Points. Clin Biomech (Bristol, Avon). 2010;25(8):765-770. doi: 10.1016/j.clinbiomech.2010.05.006.
19. Roach S, Sorenson E, Headley B, San Juan JG. Prevalence of myofascial trigger points in the hip in patellofemoral pain. Arch Phys Med Rehabil. 2013;94(3):522-526. doi: 10.1016/j.apmr.2012.10.022.
20. Ge HY, Monterde S, Graven-Nielsen T, Arendt-Nielsen L. Latent myofascial trigger points are associated with an increased intramuscular electromyographic activity during synergistic muscle activation. J Pain. 2014;15(2):181-187. doi: 10.1016/j.jpain.2013.10.009.
21. Otzel DM, Chow JW, Tillman MD. Long-term deficits in quadriceps strength and activation following anterior cruciate ligament reconstruction. Phys Ther Sport. 2015;16(1):22-28. doi: 10.1016/j.ptsp.2014.02.003.
22. Chmielewski TL, Stackhouse S, Axe MJ, Snyder-Mackler L. A prospective analysis of incidence and severity of quadriceps inhibition in a consecutive sample of 100 patients with complete acute anterior cruciate ligament rupture. J Orthop Res. 2004;22(5):925-930. doi: 10.1016/j.orthres.2004.01.007.
23. Greenberg EM, Greenberg ET, Ganley TJ, Lawrence JT. Strength and functional performance recovery after anterior cruciate ligament reconstruction in preadolescent athletes. Sports Health. 2014;6(4):309-312. doi: 10.1177/1941738114537594.
24. Palmieri-Smith RM, Thomas AC, Wojtys EM. Maximizing quadriceps strength after ACL reconstruction. Clin Sports Med. 2008;27(3):405-424, vii-ix. doi: 10.1016/j.csm.2008.02.001.
25. Urbach D, Nebelung W, Becker R, Awiszus F. Effects of reconstruction

of the anterior cruciate ligament on voluntary activation of quadriceps femoris a prospective twitch interpolation study. J Bone Joint Surg Br. 2001;83(8):1104-1110.

26. Grindstaff TL, Jackson KR, Garrison JC, Diduch DR, Ingersoll CD. Decreased quadriceps activation measured hours prior to a noncontact anterior cruciate ligament tear. J Orthop Sports Phys Ther. 2008;38(8):508-516. doi: 10.2519/jospt.2008.2761.

27. Davis DS, Barnette BJ, Kiger JT, Mirasola JJ, Young SM. Physical characteristics that predict functional performance in Division I college football players. J Strength Cond Res. 2004;18(1):115-120.

28. Kiesel K, Plisky PJ, Voight ML. Can Serious Injury in Professional Football be Predicted by a Preseason Functional Movement Screen. N Am J Sports Phys Ther. 2007;2(3):147-158.

29. Robbins DW. Positional physical characteristics of players drafted into the National Football League. J Strength Cond Res. 2011;25(10):2661-2667. doi: 10.1519/JSC.0b013e318208ae3f.