Knee joint laxity affects muscle activation patterns in the healthy knee

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Abstract:

This study investigated the effects of anterior knee joint laxity on muscle activation patterns prior to and following a lower extremity perturbation. Participants were subjected to a forward and either internal (IR) or external (ER) rotation perturbation of the trunk and thigh on the weight-bearing shank. Pre-activity (%MVIC) before the perturbation, and reflex time (ms) and mean reflex amplitude (%MVIC) following the perturbation were recorded via surface electromyography (sEMG) in the medial and lateral gastrocnemius, hamstring and quadriceps muscles. Twenty-one NCAA DI intercollegiate female athletes with below average anterior knee laxity (3–5 mm) were compared to 21 with above average anterior knee laxity (7–14 mm) as measured by a standard knee arthrometer. Groups differed in reflex timing by muscle (P= 0.013), with females with above average knee laxity ($KT_{(>7mm)}$) demonstrating a 16 ms greater delay in biceps femoris reflex timing compared to females with below average knee laxity (KT(15mm)). Groups also differed in muscle activation amplitude by response, muscle and direction of rotation (i.e. a 4-way interaction; P= 0.027). The magnitude of change from pre to post perturbation was significantly less in $KT_{(>7mm)}$ vs. $KT_{(15mm)}$ for the medial (MG) and lateral (LG) gastrocnemius muscles, primarily due to higher levels of muscle preactivity while awaiting the perturbation (MG = 20% vs. 12% MVIC, P= 0.05; LG = 33% vs. 21% MVIC, P= 0.11). Further, $KT_{(>7mm)}$ demonstrated higher activation levels in the biceps femoris than $KT_{(15mm)}$ (47% vs. 27% MVIC; P= 0.025) regardless of response (pre vs. post perturbation) or direction of rotation. These findings suggest females with increased knee laxity may be less sensitive to joint displacement or loading (delayed reflex), and are more reliant on active control of the gastrocnemius and biceps femoris muscles to potentially compensate for reduced passive joint stability. Keywords: Long latency reflex; Anterior cruciate ligament; Surface electromyography; Proprioception; Sensory

Article:

INTRODUCTION

Females injure their anterior cruciate ligament (ACL) at a rate of two to eight times that of similarly trained males, depending on the age or sport under study [1,2,9,10,15,19,24,28,39]. While many have attempted to identify potential risk factors to explain the higher rate of injuries

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in females, research has failed to conclusively demonstrate the relationship of any one or combination of variables to ACL injury risk. Research advances in the past 5–7 years suggest sex differences in neuromuscular and biomechanical function are compelling factors to explain the increased risk of ACL injury rates in females [16]. But while sex differences in neuromuscular and biomechanical function have been identified, the underlying cause for these differences has not been explored. Further, it is conceded that the extent to which sex-dependent anatomical factors contribute to sex differences in neuromuscular and biomechanical function, or independently contribute to ACL injury risk is unknown [16].

Anterior knee joint laxity is often proposed as an anatomical risk factor in ACL injury, with females having greater joint laxity than males [14,23,30,31]. The implications of increased knee joint laxity on neuromuscular and biomechanical function of the knee joint are not well understood, yet appear to be critical to our understanding of sex-dependent factors that may influence functional joint stability and injury risk.

"Functional joint stability" defines the combined joint stabilization forces required to perform functional activities and is achieved through both static and dynamic stabilization [16]. As a static stabilizer of the knee, the ACL provides the majority (~86%) of passive restraint to anterior translation of the tibia on the femur [8]. Studies have shown that when transitioning from nonweight-bearing to weight-bearing with the knee near full extension (15–30° flexion), there is a natural anterior shift of the tibia on the femur [11,37], that is limited by the intact ACL [6,18].

The presence of mechanoreceptors in the human ACL [32,40] also enables the ligament to contribute to active joint stability, providing sensory feedback about sudden changes in ligament tension or length [4,36]. A significant hamstring reflex arc [12,36] and quadriceps inhibition [36] have been demonstrated at high levels of ACL loading not present at low loads, suggesting the neural reflex response is dependent on the intensity and time course at which the ACL is loaded. However, there is also evidence [20,35] that the cruciate ligaments provide proprioceptive feedback and preparatory muscle stiffening at relatively small tensile loads (5–40 N), through heightened gamma motor neuron activation and muscle spindle sensitivity prior to excessive loading [21]. These findings indicate that under both high and low threshold loading, the ACL plays an important sensory role in regulating and maintaining active muscle stiffness and neuromuscular control of joint stability.

Given the contribution of the ACL to both static and dynamic knee control, it seems plausible that increased joint laxity may compromise functional joint stability. Increased knee laxity may diminish the passive restraint capabilities of the ACL in weight bearing, placing greater demands on active muscle forces to stabilize the knee. Increased knee laxity may also diminish the ability of the neuromuscular system to respond in a timely fashion to stabilize the joint, by allowing greater lengthening and joint displacement before a reflexive force threshold is reached. The ACL-deficient knee has been widely studied in this regard, and has demonstrated diminished proprioception [27], delays in reflex responses to an anterior tibial translation [5,38], and altered reactive neuromuscular control strategies during cutting maneuvers [7] and landing activities [13,26] when compared to uninjured knees. Limited research, however, has examined whether more subtle changes in joint laxity in an otherwise healthy knee would have similar, but perhaps less pronounced deficits.

Rozzi et al. [31] examined conscious joint reposition sense and neuromuscular response characteristics in healthy male and female collegiate basketball and soccer athletes. Compared to males, females had inherently greater knee laxity (mean difference of ~l.25 mm anterior tibial translation), a decreased ability to detect knee extension joint motion, and increased compensatory activity of the lateral hamstring when landing from a jump. The author's theorized from these findings that excessive joint laxity may contribute to diminished joint proprioception, rendering the knee less sensitive to damaging forces. Increased hamstring activity was considered an attempt to actively stabilize the knee, and compensate for the reduced passive stability. We are not aware of any other research that has examined the consequence of non-pathological knee joint laxity on neuromuscular response characteristics in a weight-bearing application.

Hence, our purpose was to examine the effect of anterior knee joint laxity on muscle activation patterns prior to and following a functional, weight-bearing perturbation in healthy female collegiate athletes. We hypothesized that those with greater knee laxity would exhibit longer reflex delays yet increased response amplitude of the hamstrings following the perturbation.

METHODS

Setting and design

All testing was performed in the University's Sports Medicine and Athletic Training Research Laboratory. Participants consisted of 42 healthy NCAA Division I intercollegiate female athletes (19.5 f 1.2 years, 171.7f 7.3 cm, 69.6 f 8.5 kg, 6.8 f 2.9 mm knee laxity), comparing 21 with anterior knee laxity less than 5mm ($KT_{(>5m\ m)}$), to 21 with anterior knee laxity greater than 7 mm ($KT_{(>7mm)}$). Group assignments were based on below and above average knee laxity values that have been previously reported in the literature for healthy female athletes using an applied force of 133 N (approximately 6–7 mm) [30,31]. Healthy was defined as no previous history of knee ligament injury or surgery, no history of connective tissue disorders or diseases, and no lower extremity injury in the past 6 months. Group means ±SD for age, height, weight, knee laxity and other anatomical measures recorded as part of a larger study are listed in Table 1. Anatomical measures are reported to demonstrate the two groups were relatively comparable on other structural factors. Prior to participation in the study, participants signed a written informed consent form approved by the University's Institutional Review Board.

Table 1
Descriptives by group

	$KT_{(<5mm)}$ (n= 21)		$KT_{(>7mm)}$ (n= 21)	
	Mean	(SD)	Mean	(SD)
Age (years)	19.8	(1.3)	19.2	(1.1)
Height (cm)	172.4	(7.5)	171.1	(7.3)
Weight (kg)	69.3	(7.8)	69.9	(9.3)
Anterior knee laxity (mm)	4.3	(0.7)	9.2	(2.0)
Navicular drop (mm)	5.1	(3.6)	7.5	(3.0)
Quadriceps angle (deg)	15.4	(4.2)	15.0	(4.3)
Pelvic inclination (deg)	5.8	(3.4)	7.0	(2.6)
Hip anteversion (deg)	8.0	(5.4)	5.9	(4.5)
Knee recurvatum (deg)	5.1	(2.6)	6.5	(3.4)
Tibial torsion (deg)	25.8	(6.0)	21.5	(7.3)
Femur length (cm)	43.4	(2.8)	42.9	(2.5)
Tibial length (cm)	38.4	(2.5)	37.9	(0.8)

Assessment of knee laxity

Knee laxity was defined as the amount of anterior tibial displacement at 133 N, measured by a KT-2000® knee arthrometer (MEDmetric® Corporation, San Diego, CA). Subjects were positioned as per the manufacturer's guidelines in supine on an examination table with a thigh support placed just proximal to the popliteal fossa so that the subject's knee was in 250 of flexion. In addition to placing their ankles in the manufacturer provided foot cradle, a Velcro strap was placed around the subject's thighs to control rotation of the lower extremity. Once properly positioned, the KT-2000® was then applied to the anterior tibia of the right lower extremity in proper alignment with the subject's joint line as per the manufacturer's instructions. A masonry bubble level (Stanley Works Inc., New Britain, CT) was attached to the body of the instrument to insure the device remained level and that a direct anterior pull was performed for each subject and trial. The average of three trials was recorded as the subject's knee laxity measure. All knee laxity measures were performed by a single, experienced investigator (CRC) with established intratester reliability ($ICC_{2,k}$ = 0.97; SEM = 0.37).

Lower extremity perturbation

To evoke the reflex response in weight bearing, we used a custom-built lower extremity perturbation device that produced a forward and either internal (IR) or external (ER) rotation of the trunk and femur on the weight-bearing tibia (Fig. 1). The design, reliability and validity of this device have been previously reported [33], and the model has been used previously to identify neuromuscular response characteristics in males and females [34]. Participants stood barefoot on their dominant leg, restrained by two kevlar cables

Fig. 1. Lower extremity perturbation device.

attached to a wall mounted cable release mechanism, that was adjustable in height to maintain the cables in a horizontal line of pull across subjects. Participants were asked to place their arms across their chest, lean into the cables allowing them to fully support their body weight, and flex their knee to ~30°. This position was standardized and verified for each participant and trial using a Penny and Giles electrogoniometer (Model XM180; Biometrics Ltd, UK) aligned along with the femur and tibia on the lateral aspect of the thigh to measure knee flexion angle and the Chattecx Balance System (Chattanooga Group, Inc., Hixson, TN) visual training target to consistently place the center of pressure over the midfoot. Subjects were instructed to look forward with their eyes focused straight ahead at the visual training target and, either the left or right cable was released at a time unannounced to cause either an internal or external rotational perturbation. Upon cable release, participants were asked to try and maintain their single leg balance. Ten trials were performed for both internal and external rotation perturbations, with the direction of rotation randomized to minimize anticipatory responses. Participants were given a 30 s rest period between trials, and were instructed to shift their weight to the non-test leg during the rest periods to avoid fatigue.

Electromyographical analysis

To record muscle activity, we used an eight channel Noraxon Myosystem 2000 Surface Electromyogram (EMG) (Noraxon, Scotsdale, AZ), with unit specifications as follows: amplification of 1 mV/V, frequency bandwidth of 16 to 500 Hz, CMRR of 114 dB, input resistance of 1 GOhm, and sampling rate of 1000 Hz. The signal of each muscle was detected with 10 mm bipolar Ag-AgCl surface electrodes (Medicotest Blue Sensor Model No. N-00-S; Ambu Products, Germany), placed over the vastus medialis (VM) and vastus lateralis (VL) (midway between the motor point and distal tendon), medial hamstring (MH) and biceps femoris

(BF) (mid-belly), and medial (MG) and lateral (LG) gastrocnemius (midbelly of the medial and lateral heads) with a center-to-center distance of 2.5 cm. The ground electrode was positioned on the anterior tibia. All electrode placements were confirmed with manual muscle testing and checked for cross talk. We interfaced the EMG and perturbation device with Data Pac 2000 Lab Application Software (Run Technologies, Laguna Hills, CA) to acquire, store, and analyze the EMG data. A voltage signal at the time of trigger release was sent from the lower extremity perturbation device to the computer software to mark the time of stimulus and begin data recording. We recorded muscle activity from 100 ms prior to and 900 ms following cable release using the trigger sweep function.

Prior to collection of the perturbation trials, maximal EMG signals were recorded during maximal voluntary isometric contractions (MVIC) of each muscle group for later normalization of the EMG data. Participants were positioned in an isokinetic dynamometer (KIN-COM II Isokinetic Dynamometer; Chattanooga Group, Inc., Chattanooga, TN) at 300 of knee flexion and asked to complete three 5s maximal effort knee extension (quadriceps) and knee flexion (hamstrings) contractions with the dynamometer locked at 00/s. Normalization of the gastrocnemius muscle was performed by completing three 5s maximal effort, single leg toe raises. MVIC trials were digitally processed using a centered (symmetric) root mean square (RMS) algorithm, with a 100 ms time constant. The peak amplitude (RMS value) identified over the middle 3 s was averaged across the three trials and used to normalize sEMG amplitudes of the perturbation trials.

The EMG signals for the perturbation trials were digitally processed, using a centered (symmetric) root mean square (RMS) algorithm, with a 5 ms time constant. Individual trials for IR and ER were visually inspected and selected if a long latency reflex was identified within 150 ms following cable release, baseline muscle activity was sufficiently quiet and stable to insure an acceptable signal to noise ratio, a readable signal was obtained from all six muscle sites and the signal was free of movement artifact to allow clear interpretation of the signal. If a trial failed to meet any of the above criteria, the event was excluded from further analysis. Using the first five trials to meet these criteria, the signal was averaged to obtain a single representative signal from which to determine muscle onset times and amplitudes. The reliability of this procedure has been previously established [33], and the investigator processing the data (SJS) was blinded to subject and group membership.

Pre-perturbation muscle activity (PreAmp = %MVIC) was defined as the mean signal amplitude for 50 ms prior to the perturbation, normalized to the maximal voluntary isometric contraction for that muscle. Long latency reflex time (RT = ms) was defined as the time delay between the onset of the perturbation and a one (quadriceps), or two (hamstring and gastrocnemius), standard deviation increase in muscle activity above baseline activity (100 ms pretrigger) for 10 ms or longer. A one standard deviation threshold was used for the quadriceps due to its higher baseline activity in maintaining the single leg weight bearing stance [33]. Mean reflex amplitude (RAmp = %MVIC) represented the mean signal amplitude over 150 ms immediately post perturbation, also normalized to the MVIC for each muscle.

Data analysis

Data were analyzed using the SPSS Statistical Software Package version 11.0 (Allegiant Techno logies, Inc.). To compare groups with below average ($KT_{(<5mm)}$) and above average ($KT_{(>7mm)}$) knee joint laxity on reflex timing, we used a mixed model repeated measures ANOVA with one between (KT group) and two within [perturbation (internal, external) and muscle (MG, LG, MH, BF, VM and VL)]. A separate mixed model repeated measures ANOVA with one between (KT group) and three within (perturbation at two levels (internal, external), response amplitude at two levels (Pre_{Amp}, R_{Amp}) and muscle at 6 levels (MG, LG, MH, BF, VM and VL)) compared knee laxity groups on preactivity and reflex response amplitudes for each muscle and for each perturbation condition. Post hoc analyses consisted of repeated contrasts for within effects, and simple main effects testing for significant interactions. Bonferroni corrections were used for multiple comparisons. Alpha was set a priori at $P< 0.05$.

RESULTS

Table 2 lists the means and standard deviations for all dependent measures by group. Groups differed in reflex timing by muscle ($F(5,200)= 2.987$; $P= 0.013$) (Fig. 2), with the biceps femoris demonstrating a 16 ms

Table 2
Means and standard deviations (SD) for reflex timing (RT), preactivity $_{(PREAmp)}$ and reflex amplitude (R_{Amp}) for internal and external rotation perturbations

	Muscle											
	Medial gastrocnemius		Lateral gastrocnemius		Medial hamstring		Biceps femoris		Vastus medialis		Vastus lateralis	
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
RT (ms)												
External rotation												
$KT_{(<5mm)}$	64.9	(6.4)	66.3	(6.7)	66.8	(9.8)	76.0	(12.5)	97.2	(28.0)	105.4	(23.5)
$KT_{(>7mm)}$	62.8	(13.5)	66.6	(11.5)	83.5	(43.3)	96.1	(36.6)	94.6	(19.9)	99.1	(22.6)
Internal rotation												
$KT_{(<5mm)}$	63.1	(10.1)	64.9	(8.1)	64.2	(9.5)	84.0	(26.9)	93.4	(19.8)	99.8	(19.7)
$KT_{(>7mm)}$	62.8	(11.0)	66.1	(11.3)	78.4	(38.7)	96.7	(28.1)	88.9	(12.4)	94.1	(12.6)
PRE_{Amp} *(%MVIC)*												
External rotation												
$KT_{(<5mm)}$	12.2	(04.0)%	21.3	(09.8)%	24.2	(16.1)%	22.5	(13.6)%	36.1	(11.5)%	33.9	(12.9)%
$KT_{(>7mm)}$	19.6	(15.9)%	33.0	(30.5)%	27.6	(18.5)%	43.7	(35.5)%	36.5	(13.4)%	37.9	(15.8)%
Internal rotation												
$KT_{(<5mm)}$	12.3	(03.8)%	21.3	(09.4)%	25.6	(17.0)%	22.5	(13.4)%	42.1	(15.5)%	32.2	(11.3)%
$KT_{(>7mm)}$	19.5	(16.7)%	33.0	(32.0)%	28.6	(20.1)%	43.5	(36.5)%	40.0	(15.8)%	39.4	(17.9)%
R_{Amp} *(%MVIC)*												
External rotation												
$KT_{(<5mm)}$	42.7	(12.9)%	46.1	(13.1)%	37.3	(20.6)%	33.8	(14.9)%	43.9	(13.3)%	32.3	(13.6)%
$KT_{(>7mm)}$	42.3	(16.0)%	48.2	(28.7)%	36.3	(21.8)%	51.3	(37.0)%	40.8	(15.5)%	37.8	(19.9)%
Internal rotation												
$KT_{(<5mm)}$	39.1	(12.9)%	47.2	(16.3)%	40.3	(24.4)%	30.6	(15.3)%	44.3	(13.8)%	33.6	(14.2)%
$KT_{(>7mm)}$	39.9	(16.4)%	49.1	(29.7)%	38.6	(23.7)%	49.4	(37.2)%	44.6	(16.5)%	40.0	(19.7)%

$KT_{(<5mm)}$, below average knee laxity group; $KT_{(>7mm)}$, above average knee laxity group.

greater delay in females with above average knee laxity ($KT(>_{7mm})$) compared to females with below average knee laxity ($KT_{(<5mm)}$). While a similar group difference in the onset of the medial hamstring was found ($KT_{(>7mm)}$ 15 ms > $KT_{(<5mm)}$), the post hoc comparison was not significant. The direction of the rotational perturbation (internal vs. external) had no effect on group ($P= 0:654$), muscle ($P= 0:102$), or muscle by group ($P= 0:824$) for reflex timing.

Groups also differed in muscle activation amplitude by response, muscle and direction of rotation (i.e. 4-way interaction; $F_{(5;200)}$= 2:592; P= 0:027) (Table 3). In order to interpret the 4-way interaction, post hoc analysis consisted of separate ANOVAs for each muscle, followed by simple main effects testing. These analyses revealed that both groups increased muscle activation amplitude from Pre_{Amp} to R_{Amp} in all muscles (each P< 0:001) except the lateral quadriceps (P= 0:953). However, the magnitude of change from Pre_{Amp} to R_{Amp} was significantly less in the $KT_{(>7mm)}$ compared to the $KT_{(<5mm)}$ group in the medial gastrocnemius (P= 0:04) and the lateral gastrocnemius (P= 0:006). While this interaction appeared to be primarily due to higher levels of preactivity in $KT_{(>7mm)}$ compared to $KT_{(<5m\ m)}$, this difference was significant for the medial gastrocnemius (20% vs. 12% MVIC; P= 0:05) (Fig. 3), but not for the lateral gastrocnemius (33% vs. 21% MVIC; P= 0:11), likely due to the greater response variability in the LG for the $KT_{(>7mm)}$ group (See Table 2). Groups also differed in biceps femoris activation, with the above average knee laxity group demonstrating higher activation levels (47% vs. 27%; P= 0:025), regardless of the direction of rotation (IR vs. ER) or response (Pre_{Amp} vs. R_{Amp}). No group differences were noted for the medial hamstring, vastus medialis or vastus lateralis.

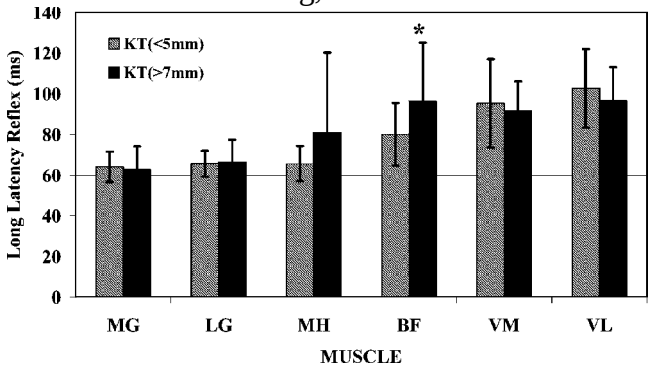

Fig. 2. Differences in long latency reflex times (ms) by muscle (P= 0:013) between KT(<5mm) and KT(>7mm) laxity groups. * Indicates significant differences between groups (P< 0:05). VM, vastus medialis; VL, vastus lateralis; MH, medial hamstrings; BF, biceps femoris; MG and LG, medial and lateral heads gastrocnemius.

Table 3
ANOVA table identifying group differences in muscle activation amplitude by response ($PreA_{mp}$, RA_{mp}), muscle (MG, LG, MH, BF, VM, VL) and rotation (IR, ER)

	df	MS	F	Sig
Within				
Rotation	1	0.097	3.355	0.074
Rotation by KT group	1	0.001	0.512	0.478
Error (rotation)	40	0.002		
Response amplitude	1	3.455	207.8	0.000
Resp amp by KT group	1	0.106	6.404	0.015
Error (resp amp)	40	0.017		
Muscle	5	0.326	3.076	0.011
Muscle by KT group	5	0.225	2.126	0.064
Error (muscle)	200	0.106		
Rotation by resp amp	1	0.003	1.141	0.292
Rot by resp amp × KT group	1	0.002	0.696	0.409
Error (rot by resp amp)	40	0.002		
Rotation by muscle	5	0.015	6.973	0.000
Rot by muscle × KT group	5	0.001	0.372	0.868
Error (rot by muscle)	200	0.002		
Resp amp by muscle	5	0.381	48.447	0.000
Resp amp by muscle × KT group	5	0.016	2.022	0.077
Error (resp amp by muscle)	200	0.008		
Rot by resp amp by muscle	5	0.006	7.036	0.000
Rot by resp amp by muscle × KT group	5	0.002	2.592	0.027
Error (Rot by resp amp × muscle)	200	0.001		
Between				
KT group	1	0.895	2.616	0.114
Error	40	0.342		

Based on our findings of both increased reflex delay and increased activation amplitude in the biceps femoris in those with above average knee laxity, we ran a secondary post-hoc correlation analyses to determine if there was a relationship between increased pre- activity and increased post perturbation reflex timing and amplitude. Pearson product moment correlations revealed no significant relationship between preactivity level and reflex timing (r= 0:233; P= 0:137 (ER); r= 0:221 P= 0:159 (IR)), but did find very high correlations between preactivity and post perturbation reflex amplitude (r= 0:957; P< 0:0001 (ER); r= 0:964 P< 0:0001 (IR)). Further, significant positive correlations were noted between knee joint laxity values and preactivity (r= 0:529; P= 0:001 (ER); r= 0:532; P= 0:001 (IR)), reflex amplitude (r= 0:479; P= 0:001 (ER); r= 0:497; P= 0:001 (IR)), and to a lesser extent reflex timing (r= 0:280; P= 0:072 (ER); r= 0:331; P= 0:032 (IR)) for the biceps femoris. Of interest, strong correlations were also noted between preactivity of the biceps femoris with preactivity of medial (r= 0:685; P< 0:0001) and lateral (r= 0:700; P< 0:0001) gastrocnemius muscles.

DISCUSSION

Our primary findings were that, when compared to those with below average knee laxity, participants with above average knee joint laxity demonstrated increased levels of muscle preactivity in the medial gastrocnemius

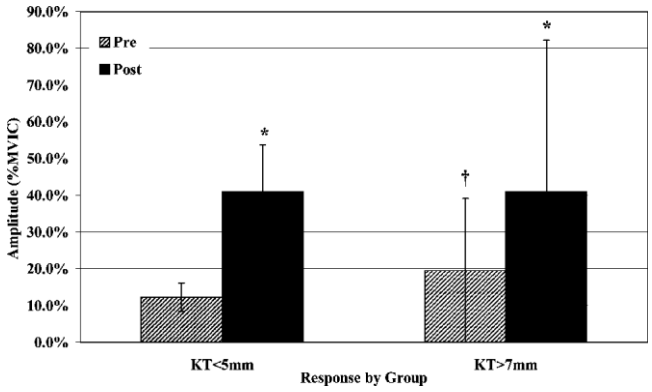

Fig. 3. Differences between $_{KT(<5mm)}$ and $_{KT(>7mm)}$ knee laxity groups in activation amplitude of the medial gastrocnemius by response. * Indicates post-perturbation reflex amplitude (POST) greater than pre-activity (PRE) amplitude, † Indicates KT(>7mm) > KT(<5mm).

and biceps femoris muscles prior to the perturbation, and greater delays in reflex timing and increased reflex amplitude in the biceps femoris following the perturbation. Similar differences in reflex timing for the medial hamstring muscles were also noted between groups, but this was not statistically significant, and was not accompanied by differences in activation amplitude. While greater delays in hamstring reflexes would suggest a proprioceptive deficit, the increased levels of preactivity in the medial gastrocnemius and consistently higher activation levels in biceps femoris (pre and post) would suggest a compensatory strategy to aid in joint stabilization. A lack of significant correlations between muscle preactivity and reflex timing of the biceps femoris suggests that factors other than the level of pre-activity contributed to group differences in reflex timing.

While we were unable to find another published study that evaluated reflex activation patterns in healthy participants with non-pathological knee laxity, our findings are surprisingly consistent with those demonstrated in ACL deficient individuals. Beard et al. [5] and Wojtys et al. [38] compared reflexive hamstring response times in ACL deficient and control subjects using anterior tibial translation tests. Both studies indicated slower reflex times following the anterior tibial translation stimulus in the ACL deficient knees, which they attributed to a loss of proprioception and passive resistance provided by the ACL. The fact that our findings paralleled their results suggests even subtle changes in joint laxity may influence sensory perception and synergistic reflex control of the hamstrings. In support of this theory, Rozzi et al. [31] found females who had significantly greater anterior knee joint laxity compared to males (6.1 vs. 4.8 mm) had a decreased ability to detect joint motion into extension with the knee at 150 of flexion.

Of interest is that our participants demonstrated these reflex delays even though fully weight bearing, which is thought to maximize axial compression, joint congruency and frictional forces to effectively limit joint displacement and ligament tensioning [25]. However, recent evidence by Fleming et al. [11] suggests otherwise. Using an arthroscopically applied strain transducer to the intact ACL, they demonstrated significantly greater ACL strain with weight bearing compared to non-weight bearing with low to moderate anterior shear loads and external and internal torques. Varus–valgus loading only strained the ACL in weight bearing, and strain increased with increasing loads. Hence, it is plausible that weight bearing may actually facilitate the ligaments role in sensory perception of mild to moderate joint displacement and loads, potentially

mediating reflex response characteristics. Further research investigating the sensory role of the ACL in weight bearing is warranted.

Our findings of increased biceps femoris activation amplitude agree with those of Rozzi et al. [31], as well as studies of ACL deficient knees [3,13,22,26]. In our participants, both preactivity and post perturbation reflex amplitude of the biceps femoris showed moderately high positive correlations with knee joint laxity and with preactivity of the medial and lateral gastrocnemius muscles. This suggests the biceps femoris, with aid from the gastrocnemius muscles, may be attempting to compensate for increased joint laxity to enhance knee joint stability. Research has clearly established the contribution of the hamstrings, both singularly and in co-activation with the quadriceps, in maintaining joint stability and ACL protection by preventing or decreasing anterior and rotary displacement of the tibia on the femur [17,29]. At flexed positions, the hamstrings line of pull is primarily parallel to the joint surface and in a more favorable position to counteract anterior, as well as rotary displacement of the tibia. Hence, while delays in hamstring reflexes may compromise anterior and rotary stability, increased preactivity of the gastrocnemius and biceps femoris may sufficiently stiffen the joint and reduce rotary movement through increased muscular co-activation.

The implications of our findings on resultant functional joint stability are as yet unclear, but support the need for future research regarding knee joint laxity as an ACL injury risk factor. A limitation of our study is that we evaluated only neuromuscular response characteristics. The implication of knee joint laxity and altered neuromuscular control strategies on biomechanical function is an important direction for future research. Future studies combining neuromuscular and biomechanical analyses will help determine the impact alterations in neuromuscular control actually have on knee joint motion and forces. Future research should also strive to understand the corollary factors that potentially contribute to permanent (e.g. lower extremity alignment) or transient (e.g. exercise, female sex hormones) increases in knee joint laxity in females, and how these factors may combine to increase the risk of ACL injury. Transient increases in joint laxity resulting from these or other factors in females who already demonstrate inherently greater joint laxity may compound proprioceptive and ligament mediated neuromuscular control deficits, thereby increasing injury risk.

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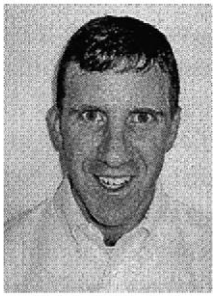

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