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Specificity of muscle action after anterior cruciate ligament injury

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Abstract

Neuromuscular control is believed to be a critical factor in dynamic knee stability. The purpose of this study was to evaluate voluntary muscle control in anterior cruciate ligament deficient (ACL-D) and uninjured people. Twenty athletes of similar age participated in this study. Subjects performed a target-matching protocol that required them to produce isometric moments about the knee with fine control in flexion, extension, varus, and valgus (i.e., loads were generated in the plane perpendicular to the long axis of the shank). Electromyographic data were collected from 10 muscles that span the knee. A specificity index was calculated for each muscle to describe how fine-tuned (specific) its muscle activity pattern was with respect to its principal direction of action in the load plane. Diminished specificity of muscle action was observed in 8 of 10 muscles in the ACL-D subjects' involved knees when compared with the activity patterns from their uninvolved knees and those from the uninjured subjects' knees. The vastus lateralis muscle was especially affected. Increased and more global co-contraction was also observed in the ACL-D limbs. The alterations in muscle firing patterns observed in this study are consistent with diminished neuromuscular control.

Keywords: Neuromuscular control; Anterior cruciate ligament; Knee stability; EMG

Introduction

Neuromuscular function is a critical factor in dynamic knee stability [16,18]. The neuromuscular function of people with anterior cruciate ligament deficient (ACL-D) knees has been evaluated using a variety of different methods, including: joint repositioning tasks [3], muscle response testing [19], and biomechanical testing during functional activities, such as gait [13, 15,16]. Altered patterns of muscle recruitment and changes in movement patterns have been common findings, especially among non-copers (i.e., people with ACL-D knees who do poorly without surgical intervention). These altered patterns have been theorized to represent compensatory strategies for pathological knee motion [13,15,16]. The predominant pathological responses and movements involve the quadriceps femoris muscles, which are preferentially affected by ACL rupture.

The ability to produce force in a controlled fashion through finely tuned muscle activity is referred to as neuromuscular control. Evaluating neuromuscular function in functional tasks such as walking, running and jumping is challenging because it is difficult to uncouple all of the biomechanical and neuromuscular events taking place during these movements. Compensatory muscle activity associated with pathological knee motion (excessive anterior translation or rotation) or other factors may mask the voluntary neuromuscular control strategies being used. Examining neuromuscular function in more "basic" tasks, such as controlling force production in an isometric preparation, may provide insight into the voluntary muscle control strategies people use after ACL injury.

Buchanan and Lloyd [4] used a target-matching protocol to evaluate the muscle activation strategies that uninjured people use to counteract isometric flexion–extension and varus–valgus loads at the knee joint. They evaluated the activity patterns of 10 muscles that span

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the knee by plotting electromyographic (EMG) data in polar co-ordinates and also described the principal load direction of each muscle [4]. This approach is an established method of studying neuromuscular control strategies [4,5,8,11]. Examining the muscle activation patterns of people with ACL-D knees using a protocol similar to the one described by Buchanan and Lloyd [4] may provide meaningful insight into the effect(s) that ACL injury has on neuromuscular function. Furthermore, it may help further delineate how neuromuscular function differs in non-copers and uninjured people. This knowledge may have very important implications for the treatment of ACL injuries and the development of rehabilitation programs that address neuromuscular function following this injury.

The purpose of this study was to evaluate the neuromuscular control strategies of people with ACL-D knees by assessing their specificity of muscle action with circular statistics methods. We hypothesized that the muscles surrounding ACL-D subjects' involved knees would have diminished specificity of muscle action when their activity patterns were compared to those from the muscles of their uninvolved knees and the knees of uninjured subjects. The quadriceps femoris muscles were expected to be most affected because ACL injury is known to have a profound impact on this muscle group.

Methods

Twenty subjects participated in this study. Ten subjects (mean age = 23 ± 6 years) had uninjured knees and 10 subjects (mean age = 21 ± 7 years) had sustained isolated unilateral ACL ruptures within the prior six months (mean = 2.5 ± 1.7 months, range = 0.5–6 months). Anterior cruciate ligament ruptures were confirmed by magnetic resonance imaging and side-to-side differences in knee laxity of ⩾ 3 mm on manual maximum KT1000 (MEDmetric, San Diego, CA, USA) tests. All of the ACL-D subjects enrolled in this study were determined to be non-copers using a screening examination that differentiates people that may be able to cope with ACL injuries from those who cannot [10]. Subjects who exhibited impairments upon physical examination were not screened until their impairments had resolved. A board-certified sports physical therapist evaluated the knees of all subjects to ensure that they had near normal range of motion, no noticeable gait abnormalities when their walking patterns were observed in the clinical setting, and no more than a trace effusion (graded according to the University of Delaware Physical Therapy Clinic's established procedure; a trace effusion is defined as a "just-identifiable" effusion that is ascertained by comparing a patient's knees using visual inspection and/or palpation). In addition, a maximal voluntary isometric quadriceps strength test was performed at 90° of flexion using the burst-superimposition electric stimulation method to ensure that the ACL-D subjects had adequate strength to perform the functional tests in the screening examination. Subjects with noteworthy weakness (involved extensor force that was <70% of the force measured when testing the contralateral knee) and those displaying >5% deficits in central activation (indicating inhibition) were required to repeat the testing at a later date. Subjects received physical therapy treatment until all functional impairments had resolved and they were ready to be screened. Exclusion criteria for participation in this study included a previous ACL injury, concomitant ligament pathology, fractures, greater than a trace knee joint effusion, an abnormal gait pattern, and the presence of hip or ankle pathology. There were eight males and two females in each group and all subjects were regular participants in athletic activities that involve quick changes of direction and/or jumping, such as football, basketball, and soccer. Informed consent for participation was obtained from all subjects and the study was approved by the University of Delaware Human Subjects Review board.

Surface or indwelling wire electrodes were used to collect EMG data from 10 muscles that surround the knee. The activity of the rectus femoris, vastus medialis, vastus lateralis, semitendinosus, biceps femoris, and the medial and lateral gastrocnemius was sampled with bipolar surface electrodes with a 2 cm interelectrode distance (Norotrode 20, Myotronics-Noromed, Inc., Tukwila, WA, USA). The activity of the tensor fascia lata, sartorius, and gracilis was sampled with intramuscular electrodes (50 μm stainless-steel wires with the distal 3 mm removed) that were inserted approximately 2 cm apart with 27 gauge injection needles. Electrode placement sites were standardized according to the recommendations of Perotto [14] and prepared by shaving hair from the region and cleaning the skin with alcohol prep pads. The sampling frequency was 500 Hz. The EMG signals were passed through a differential pre-amplifier with a gain of 1000 and a two-pole 30–10,000 Hz bandpass filter. The EMG signals were further conditioned using a filter system (Frequency Devices, Haverhill, MA, USA) that amplified (gain = 1–20) and lowpass filtered (eight-pole Butterworth at a cut-off frequency of 250 Hz) the signals as needed. EMG data were later rectified and averaged off-line.

Subjects were seated on a small platform so that their weight was transferred through the ischial tuberosities and the thighs remained unloaded (Fig. 1). Hip flexion was standardized at 90°. A fiberglass cylinder cast was made around the distal shank of the leg and then rigidly clamped to a six-axis load cell (F/T 150/600, ATI Industrial Automation, Apex, NC, USA). Isometric force information was recorded while subjects attempted to move their legs while attached to the load cell.

Each subject performed a series of maximal voluntary isometric contractions (MVIC) at 70° of knee flexion that were used to obtain a maximum EMG value for each muscle and a peak force/moment value for each load direction ($\pm Fx$, $\pm Fy$, $\pm Fz$, $\pm Mx$, $\pm My$, $\pm Mz$). The EMG values recorded during testing were normalized by representing them as a percentage of the maximum EMG values recorded during the MVIC trials. The peak force/moment data were used to standardize the force required for cursor movement during testing, which was set to 30% of the smaller of the peak varus or valgus loads that were produced during the MVIC trials. The overall mean knee moment required was 15.1 ± 5.2 N m. The load cell was zeroed prior to the MVIC trials and testing at each knee flexion angle, which ensured that the force/moment baselines were constant across tests and subjects.

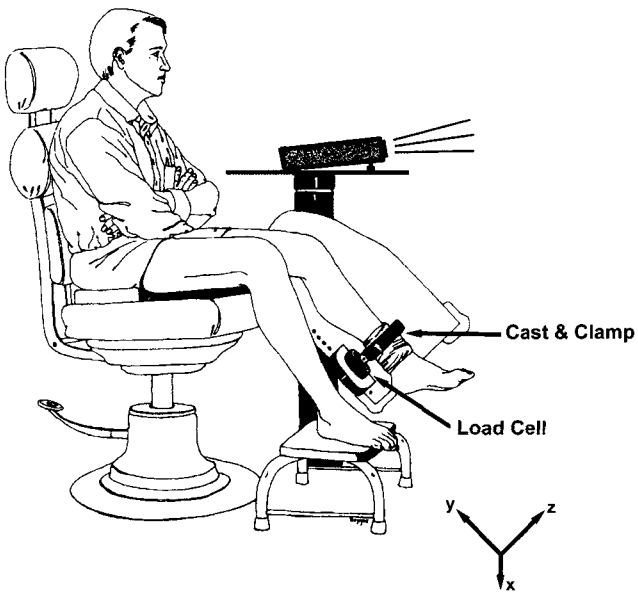

Fig. 1. Subject positioning in the experimental protocol.

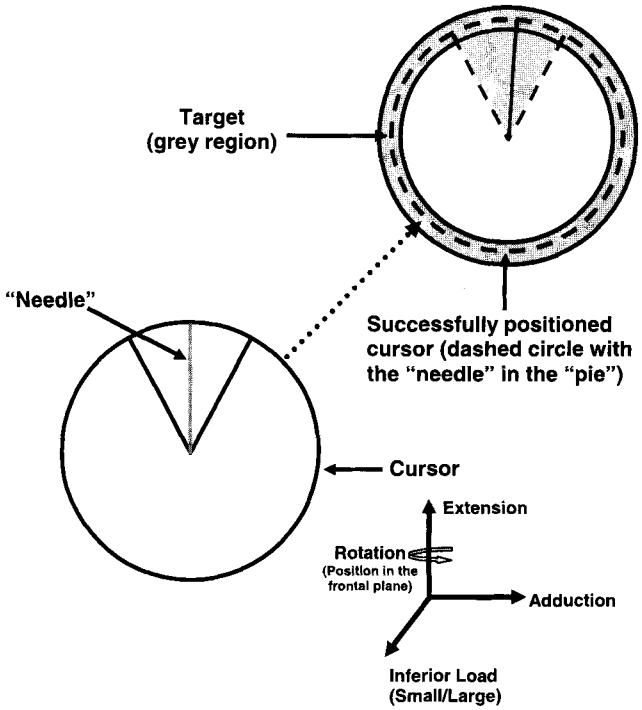

Fig. 2. Depiction of the cursor, target, and successful positioning of the cursor over the target.

Similarly, five 1-s EMG rest trials were recorded prior to testing at each knee flexion angle to obtain baseline values for each muscle. These baseline values were processed in the same manner as the other EMG data and were subtracted from EMG signals in the normalization process.

The experimental task required subjects to position a circular cursor over a narrow target (Fig. 2) and keep it there for 1 s. Cursor movement occurred in response to isometric loads that the subjects produced against the load cell. The cursor moved with four degrees-of-freedom: (1) knee extension/flexion moved the cursor upward/downward, (2) varus (hip adduction) and valgus (hip abduction) loads moved the cursor to the left or right, (3) inferior/superior loads (along the long axis of the shank) made the cursor smaller/larger, and (4) abduction/adduction of the distal shank moved the "needle" in the cursor clockwise or counter-clockwise. To successfully position the cursor over the target, subjects were required to produce loads in the flexion–extension–varus–valgus plane (perpendicular to the long axis of the shank), while minimizing loads along the long axis of the shank (keeping the cursor at the appropriate size). They also had to maintain the knee in a neutral position in the frontal plane (minimize the moment about the z-axis of the load cell; see Fig. 1 for load cell axes), which was depicted by keeping the "needle" within the pie of the cursor (Fig. 2).

Targets appeared one-at-a-time in random order at 18 different locations in the flexion–extension–varus–valgus plane (every 20° around the circumference of a circle). Three knee flexion angles (50°, 70°, and 90°) were selected for the experiment based upon our goal to minimize reactive muscle activity (recruitment of musculature in an attempt to compensate for pathologic knee motion) and our experience with the experimental protocol [4]. Subjects performed 72 trials (four trials appearing randomly at each of the 18 targets) at each knee flexion angle, bilaterally. Thus, a total of 216 trials were performed with each limb. In addition, 25 practice trials were performed prior to testing each knee in order to minimize the effects of learning. The order of testing for the three knee flexion angles and the right and left limbs (tested on separate days) was randomized to control for effects of order. The presence of fatigue was minimized with the following methods: (1) each subject performed the experimental protocol at their own rate, (2) a rest period was provided between testing at different knee angles, (3) subjects were allowed to rest as needed during the testing.

The activity pattern of each muscle was evaluated by plotting the mean normalized EMG amplitude recorded at each target in polar co-ordinates. When EMG values are plotted in polar co-ordinates, each EMG value can be represented as a vector ($\underline{\mathrm{EMG}_i}$). The orientation of this vector corresponds with the target location that the EMG value was recorded at and its magnitude corresponds with the normalized EMG amplitude. Circular statistics methods were used to describe the principal direction of action for each muscle, as well as, the degree of focus associated with each activity pattern [1,8,9]. The principal direction of action for each muscle was defined by the orientation of its resultant EMG vector ($R_{\underline{\mathrm{EMG}}}$), which is determined by summing the EMG vectors in the 18 target directions:

$$R_{\underline{\mathrm{EMG}_i}} = \sum_{i=1}^{18} \underline{\mathrm{EMG}_i}. \quad (1)$$

To describe how focused the activity pattern of each muscle was with respect to its principal direction of action, a specificity index was calculated using the following formula:

$$\text{Specificity index} = \frac{|R_{\underline{\mathrm{EMG}_i}}|}{\sum_{i=1}^{18} |\underline{\mathrm{EMG}_i}|}. \quad (2)$$

The resultant specificity index is a scalar between 0 and 1. An index of 0.0 indicates that a muscle was equally active in all target directions (totally non-specific; depicted by a circular activity pattern), whereas an index of 1.0 indicates that the muscle was active at only one target (totally specific; depicted by an activity pattern that is a single radius). The specificity index used in this study is synonymous with "the index of spatial EMG focus" reported by others [8,17].

Co-contraction was assessed by plotting the activity patterns of the rectus femoris, vastus medialis, vastus lateralis, semitendinosus, and biceps femoris on the same polar plot and then identifying overlap in the activity patterns of the quadriceps and hamstrings muscles. The outer boundary of the region of overlap was delineated by selecting the greater of the semitendinosus and biceps femoris EMG magnitudes at each extension target and the greatest of the rectus femoris, vastus medialis and vastus lateralis EMG magnitudes at each flexion target. The magnitude of co-contraction between the quadriceps femoris and hamstrings muscles was determined by calculating the area of the region of overlap. Co-contraction was further evaluated by plotting the activity patterns of all 10 muscles at representative flexion (200°, 280°, and 340°) and extension (60° and 100°) targets. This approach provided a more global assessment of co-contraction and revealed the neuromuscular control strategies employed at these targets.

Descriptive statistics were performed for each variable. A two factor (group–limb, angle) multivariate analysis of variance with each muscle as a dependent variable was used to test for significant differences in mean specificity indices across group–limb categories (uninjured, ACL-D involved, ACL-D uninvolved) and test angles. The results for one knee of each uninjured subject was used in the analysis. The knee selected corresponded to the involved knee of the ACL-D subject that he or she was matched with. Statistical significance was set at $p < 0.05$. Bonferroni multiple comparison tests were used to identify the source of the main effects.

Results

The specificity indices for nearly all of the tested muscles surrounding the ACL-D subjects' involved knees were lower than the indices calculated for the respective muscles of their uninvolved knees and the uninjured subjects' knees (Fig. 3). Five muscles: the semitendinosus, rectus femoris, tensor fascia lata, vastus lateralis, and lateral gastrocnemius, had significant main effects for group–limb combination. Post hoc tests confirmed that the results of the ACL-D involved limbs were different from those from the uninjured and/or ACL-D

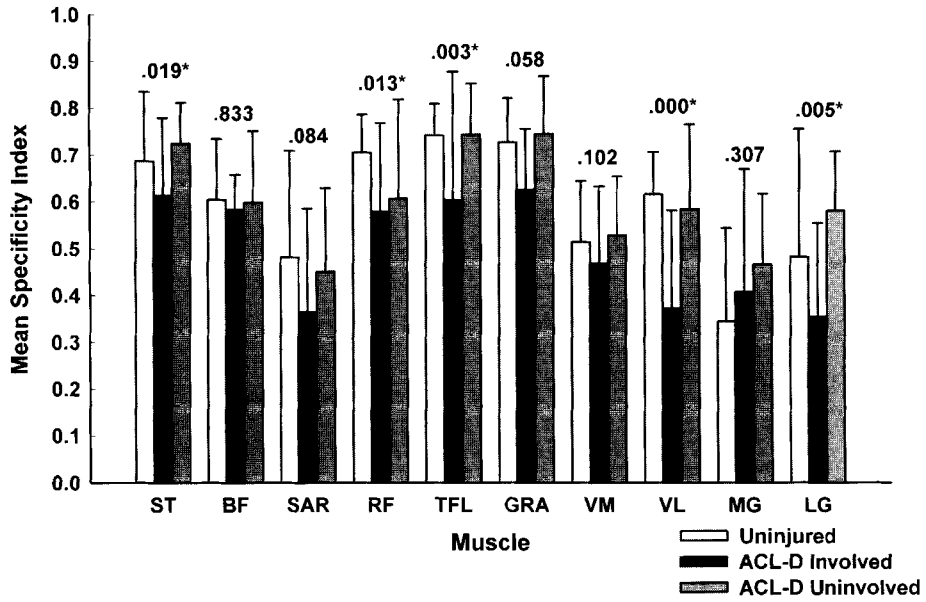

Fig. 3. Mean specificity indices for the three group–limb combinations at 90° of knee flexion. Error bars indicate the standard deviation. Values displayed above the bar charts for each muscle are p-values. Asterisks identify muscles with statistical differences. Abbreviations: ST = semitendinosus, BF = biceps femoris, SAR = sartorius, RF = rectus femoris, TFL = tensor fascia lata, GRA = gracilis, VM = vastus medialis, VL = vastus lateralis, MG = medial gastrocnemius, LG = lateral gastrocnemius.

uninvolved limbs. The sartorius, gracilis, and vastus medialis had noteworthy trends towards statistical significance but insufficient statistical power. The observed powers for these muscles were 0.497, 0.559, and 0.462, respectively. Specificity indices for the biceps femoris and medial gastrocnemius muscles were not significantly different across group–limb combinations. As hypothesized, no significant differences were observed in the results of the uninjured and ACL-D uninvolved limbs.

The diminished specificity of muscle action described in the ACL-D subjects' specificity indices is also apparent when the corresponding muscle activity patterns are depicted graphically with polar plots. For example, when the quadriceps muscle activity patterns of the three group–limb combinations are plotted in polar coordinates, the plots for the ACL-D subjects' involved limbs are characterized as containing greater area and having increased activity during flexion trials (Fig. 4). The lack of a significant difference in biceps femoris specificity indices is also nicely displayed in the similarity of the three group's biceps femoris polar plots (Fig. 4).

The magnitude of co-contraction observed in the ACL-D subjects' involved limbs was 4.5 times that of the uninjured subjects' limbs and 5.7 times greater than the magnitude of co-contraction observed in their own uninvolved limbs (Fig. 5). Most of this co-contraction was attributable to quadriceps activity during flexion; however, the lateral gastrocnemius, gracilis, and the tensor fascia lata also demonstrated more muscle activity during flexion trials when compared with the firing patterns of the other group–limb combinations (Fig. 6).

The sartorius ($p = 0.002$) and rectus femoris ($p = 0.037$) were the only muscles with significant main effects for angle. This finding is not surprising because both of these muscles are biarticular—acting at both the knee and the hip joints. Post hoc tests revealed that the specificity of muscle action of the sartorius was different at each knee flexion angle, whereas the results of the rectus femoris only differed at 50° and 90°. There were no significant group–limb × angle interactions.

Discussion

The purpose of this study was to evaluate the neuromuscular control strategies of people with ACL-D knees by assessing their specificity of muscle action. Our hypothesis that the muscles surrounding ACL-D subjects' involved knees would have diminished specificity of muscle action when their activity patterns were compared with those from the muscles of their uninvolved knees and the knees of uninjured subjects was supported by the data. The quadriceps femoris muscles were affected to a greater degree than other muscles that cross the knee, confirming our second hypothesis.

Diminished specificity of muscle action was observed in 8 of 10 muscles in the ACL-D subjects' involved knees. With the exception of the sartorius and rectus femoris muscles, which are biarticular—acting at the knee and the hip joints, there were no differences in specificity of muscle action results across knee flexion angles. The ACL-D subjects activated the quadriceps femoris muscles of their involved knees at nearly every

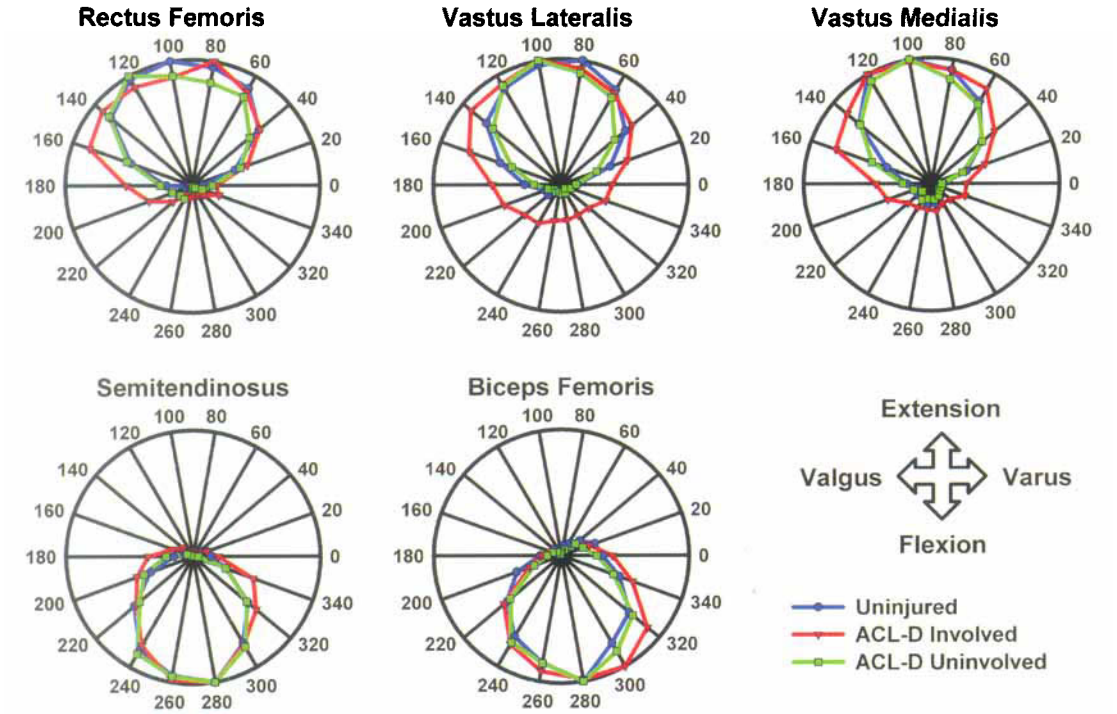

Fig. 4. EMG polar plots for the quadriceps and hamstrings muscles of the three group–limb combinations at 90° of knee flexion. The EMG values of each muscle are normalized so they range from 0 to 1 with 1 being the maximum amplitude recorded for that muscle. This figure demonstrates the diminished specificity muscle action observed in the quadriceps muscles of the ACL-D subjects' involved knees (reflected by the greater area within the red plots).

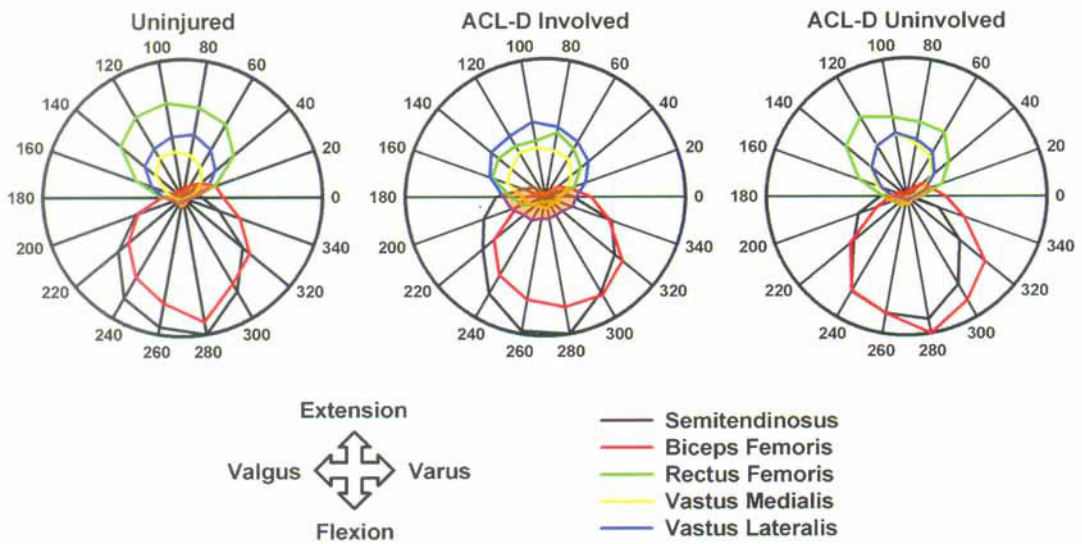

Fig. 5. Co-contraction of the quadriceps and hamstrings muscles of each group-limb combination at 50° of knee flexion. Co-contraction is depicted with orange shading.

target, suggesting that they had difficulty in regulating force production. If left unchecked, this poor quadriceps muscle control may promote the giving-way episodes that commonly occur in ACL-D non-copers. In addition to the quadriceps, they recruited the activity of muscles with principal directions of action that encompass the flexion–extension–varus–valgus plane when performing flexion trials with their involved knees. The likely result of this global co-contraction is increased knee joint stiffness. The ACL-D subjects apparently used this joint stiffening strategy because they had difficulty positioning and maintaining the cursor over the targets. Recruiting muscles that act in each of the principal load directions stiffened the joint enough for them to successfully complete the experimental task. While the excessive co-contraction observed in the involved limbs of the

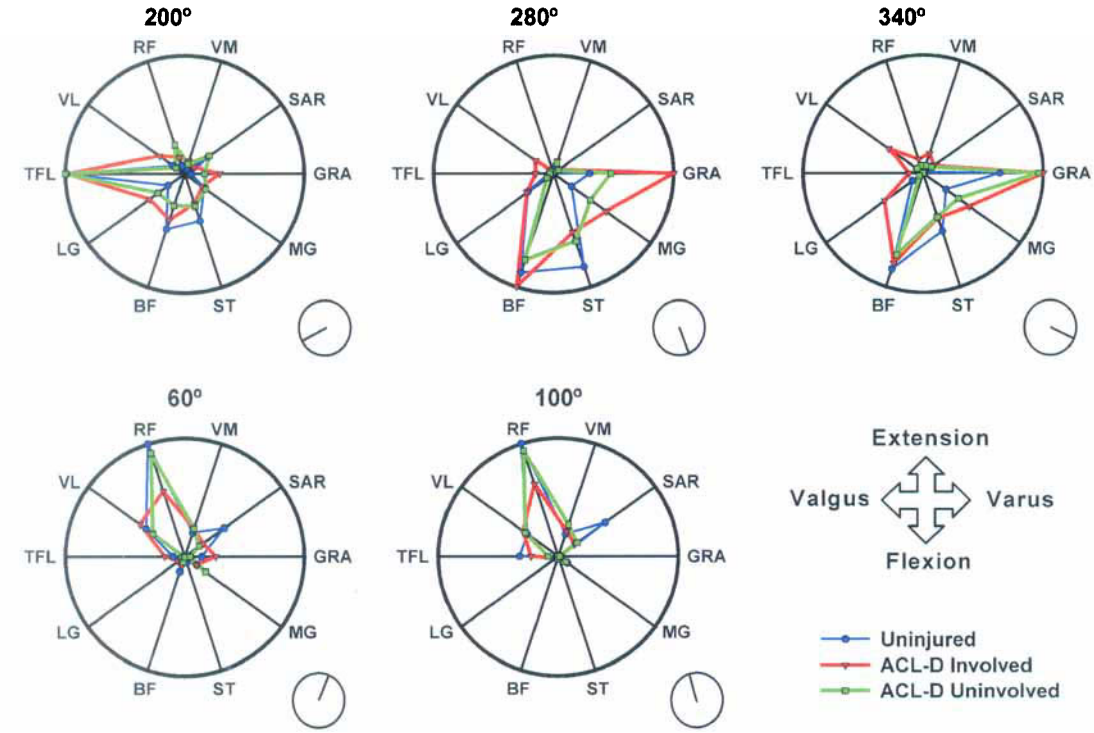

Fig. 6. Activity profiles of each muscle at various flexion and extension targets at 50° of knee flexion. The target direction is identified by the angle (°) at which the data were recorded and by the diagram at the lower right-hand corner of each plot. In these plots, 0° corresponds to 3 PM on a typical clock. Target angles increased in the counter-clockwise direction in increments of 20°. Note the more global activity patterns of the ACL-D subjects' involved knees during flexion. Abbreviations: ST = semitendinosus, BF = biceps femoris, SAR = sartorius, RF = rectus femoris, TFL = tensor fascia lata, GRA = gracilis, VM = vastus medialis, VL = vastus lateralis, MG = medial gastrocnemius, LG = lateral gastrocnemius.

ACL-D subjects appears to have been useful in the task at hand, one must wonder what effect such co-contraction has on the health of the articular cartilage of these subjects' knees (assuming that it is present during normal daily function).

Some readers may be surprised that most of the altered neuromuscular activity in this study was observed during knee flexion trials. They may have expected the opposite, hamstrings activity in extension trials. This thinking is rooted in the idea that the hamstrings act to stabilize the knee and prevent pathological knee motion. Because we carefully controlled factors that may promote reactive muscle activity (e.g. anterior tibial translation was minimized by performing the testing between 50° and 90° of knee flexion and by using low standardized loads) [2,12], we are confident that the observed stiffening strategy was the direct result of poor neuromuscular control and not a compensation strategy for knee instability. In fact, our results suggest that at least some of the reactive muscle activity often observed in dynamic studies may actually be a compensatory mechanism for poor neuromuscular control.

The biceps femoris and medial gastrocnemius muscles did not display the diminished specificity of muscle action that was observed in the other muscles. Rudolph et al. [16] found that lateral hamstrings and medial gastrocnemius of non-copers displayed altered muscle activity patterns during gait when they compared the results of ACL-D non-copers, ACL-D copers, and uninjured controls. Rudolph et al. [16] interpreted these results to be indicative of a knee stiffening strategy during gait. It is plausible that the ACL-D non-copers in our study had regularly been activating their biceps femoris and medial gastrocnemius to provide knee stability during functional tasks (e.g. gait), which may have enabled them to maintain normal specificity of muscle action in these muscles.

Our isometric protocol required subjects to produce relatively low loads (about 15 N m) in many different directions (18 target locations). Previous researchers have demonstrated that when the load requirements used in experiments like ours are increased or decreased, the resultant EMG polar plots scale with load, but the patterns remain similar [8,11]. This is important for two reasons: (1) it allowed us to set the load used in the study at an appropriate level for the involved subjects without adversely affecting the results, and (2) it suggests that in functional conditions where the loads are much higher, the altered activity patterns observed in this study may apply unhealthy loads to the knee joint. The drawback of this protocol is that the link between our static testing and functional activity is currently unknown. Consequently, the degree to which we can extrapolate our findings to normal movement is unclear. However, the

similarities between our findings and the results of some gait studies suggest that there is a link between the two [7,13,16]. For example, it is probably not a coincidence that the vastus lateralis, lateral hamstrings, and medial gastrocnemius were found to be the three muscles of interest in Rudolph et al.'s [16] gait study and also determined to be the muscles with the poorest control and most normal control, respectively, in the present study. We acknowledge that linking our results and the results of gait studies is purely speculative until specificity of muscle action and movement patterns are evaluated in the same subjects.

One of the strengths of this study is its strict inclusion criteria. The subjects in each group were matched by age and activity-level. All subjects were regular participants in high-level activities involving quick changes of direction or had been immediately prior to injury. The involved group was strictly defined: all were non-copers who sustained isolated, first-time ACL ruptures within six months of enrollment. Furthermore, enrollment was not allowed until knee impairments (e.g. joint effusions and motion deficits) had resolved. The control associated with these inclusion criteria is very important because it minimizes many of the extraneous factors that often lead to conflicting results in studies like this one.

In summary, the results of this study indicate that the activity patterns of the muscles surrounding non-copers' ACL-D knees are less specific to their principal directions of action than those of the muscles surrounding both their uninvolved knees and the muscles of people with uninjured knees. This diminished neuromuscular control was observed in nearly every muscle at each test angle. The vastus lateralis muscle was most profoundly affected. A knee stiffening strategy was observed when the ACL-D subjects performed flexion trials with their involved limb. Our results suggest that this knee stiffening is the direct result of poor neuromuscular control. The results of this study have important implications for the treatment of people who sustain ACL injuries, as well as, future research.

Acknowledgements

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