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Reactive Muscle Firing of Anterior Cruciate Ligament-Injured Females During Functional Activities

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Objective: The high incidence of noncontact anterior cruciate ligament (ACL) injuries in females has attracted research to investigate the capacity of muscles to reflexively protect the knee joint from capsuloligamentous injury. Numerous reflex pathways link mechanoreceptors in the ACL with contractile fibers in the quadriceps and hamstring muscles. Loads placed on the ACL modify reactive muscle activity through the feedback process of neuromuscular control and are critical for dynamic muscular stabilization. Noncontact ACL injuries may be the result of aberrations in reactive muscle firing patterns. Therefore, compensatory muscle activation strategies must be employed if functional stability is to be restored after injury or surgical reconstruction. The purpose of our study was to compare the amplitude of reactive muscle activity in females with ACL-deficient (ACLD), ACL-reconstructed (ACLR), and control knees during functional activities.

Design and Setting: Female volunteer subjects were stratified into groups based on the status of their ACLs. Each subject performed 4 functional activities, bilaterally, during a single test session.

Subjects: Twenty-four female subjects participated in this study (ACLD = 6, ACLR = 12, control = 6).

Measurements: Integrated electromyographic (IEMG) data were collected with surface electrodes from the vastus medialis, vastus lateralis, medial hamstring, and lateral hamstring during downhill walking (15°, 0.92 m/s), level running (2.08 m/s), and hopping and landing from a jump (20.3 cm). IEMG was normalized to the mean amplitude of 3 to 6 consecutive test repetitions. The mean area and peak IEMG of a 250-millisecond period after ground contact was used to represent reactive muscle activity. Side-to-side differences were determined using dependent *t* tests, and group differences were determined using a one-way analysis of variance.

Results: During running, the ACLD group demonstrated significantly greater area and peak IEMG activity in the medial hamstring in comparison with the ACLR group and greater peak activity in the lateral hamstring when compared with the control group. The ACLD group also demonstrated greater peak activity in the vastus medialis and a smaller area of IEMG activity in the lateral hamstring than the control group during running. During landing, the ACLD group demonstrated significantly less area of IEMG activity in the vastus lateralis when compared with the control group. No significant differences were identified between the ACLR and control groups, nor were side-to side differences revealed.

Conclusions: Our results suggest that adaptations occur in the reactive muscle activity of ACLD females during functional activities. Strategies to minimize the anterior tibial translation in response to joint loading included increased hamstring activity and quadriceps inhibition. The reactive muscle activity exhibited in ACLD subjects is presumably an attempt to regain functional stability through the dynamic restraint mechanism. The absence of side-to-side differences suggests that these adaptations occur bilaterally after ACL injury.

Key Words: reflex, muscle activity

Contemporary research regarding the disproportionate number of noncontact anterior cruciate ligament (ACL) injuries in females is beginning to focus on the role of the hamstring and quadriceps muscles in providing dynamic muscular stabilization.[1-6] Thigh muscle activation provides dynamic restraint by absorbing the high joint loads generated during athletic activities. Although capsuloligamentous structures like the ACL provide some static restraint capabilities, their primary role is to guide the adjacent skeletal segments during joint motion.[7] Capsuloligamentous structures also possess neurosensory qualities that can modify thigh muscle activity through various reflex pathways.[8] It is suggested that reactive muscle activity must occur with sufficient magnitude in a window of 30 to 70 milliseconds after the onset of joint loading in order to effectively protect the ACL.[5,6] Failure of the dynamic restraint mechanism to control joint forces may expose the ACL to excessive forces and contribute to the incidence of noncontact ACL injuries in females. In addition, adaptations must occur to the dynamic restraint mechanism after injury if functional joint stability is to be maintained.

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Research to establish the role of reactive muscle activity is valuable to clinicians and athletes for the prevention of injury or restoration of knee joint function through the dynamic restraint mechanism.

The feedback process of neuromuscular control is responsible for regulating reactive muscle activity.[9] Mechanoreceptors located within capsuloligamentous and tenomuscular structures detect joint loads and initiate reactive muscle activity through several neural pathways. These neural pathways can involve fast monosynaptic stretch reflexes from muscle spindles, polysynaptic reflexes from capsuloligamentous mechanoreceptors, and longer cortical pathways involving the brain stem and cerebral cortex.[9–11] The effects of ACL injury on these reflex pathways remain unclear. Variations in the reactive muscle activation strategies of females with ACL-deficient knees (ACLD) may result from deafferentated ACL mechanoreceptors and interruption of the reflex pathways or from subconscious attempts to regain functional joint stability through the dynamic restraint mechanism. Surgical reconstruction of the ACL (ACLR) may restore its mechanical function in the knee, but the issue of graft reinnervation and its effect on reactive muscle activity remain undetermined.[11,12] Furthermore, numerous rehabilitation exercises are designed to improve neuromuscular control, but research is still attempting to establish "typical" muscle activation patterns in uninjured, ACLD, and ACLR populations.[2,4,6]

Electromyographic (EMG) equipment assesses reactive muscle activity by measuring the efferent (motor) response of muscles to joint loading.[13] Previous research has examined the sequence and timing of muscle activity; however, until recent technologic advances, the analysis and quantification of EMG amplitude during functional activities (running) were tedious and less reliable.[14] Superimposing the EMG activity of multiple trials into one representative ensemble pattern increases the reliability of EMG amplitude measures and provides additional information about the dynamic restraint mechanism during functional activities.[14]

Limited research using this method has focused on an isolated period of reactive muscle activity during functional activities. Branch et al[2] compared a sample of ACLD subjects with uninjured subjects and found a decrease in the area of quadriceps activity and an increase in the area of medial hamstring activity during the stance phase of a cutting maneuver. These changes in the ACLD group can be considered reactive because they occurred after ground contact while the joint was loaded. Branch et al[2] concluded that the increased hamstring activity was a protective mechanism to minimize anterior translation of the tibia. The concomitant decrease in quadriceps activity during the same period may indicate reflexive inhibition.[1] Excessive quadriceps activity could exacerbate anterior tibial translation; thus, lower levels of activity would be beneficial to the dynamic restraint mechanism and functional joint stability.[1,2] Our study assessed a 250-millisecond period of reactive muscle activity in response to joint loading for the purpose of establishing differences in measures of EMG amplitude among ACLD, ACLR, and uninjured females.

METHODS

Subject Characteristics

Twenty-four female volunteers (mean age = 29.4 ± 10.4 years; mean height = 168 ± 10.7 cm; mean weight = 61.2 ± 6 kg) were stratified based on the condition of their ACLs. The 2 experimental groups comprised 18 subjects (ACLD = 6, ACLR = 12) who had suffered complete unilateral ACL tears diagnosed by an orthopaedic surgeon. All ACLR subjects had received bone-patellar tendon-bone grafts, had completed a rehabilitation program, and had attempted to return to their previous levels of activity.[15] Testing was conducted between 6 and 30 months after surgery. The experimental groups averaged 6.8 ± 1.5 on the Tegner Activity Score and 92.9 ± 5.4 on the Lysholm Knee Scoring Scale.[16,17] Subjects with disability due to secondary meniscal damage or ligamentous injury in excess of grade I were excluded.

The control group consisted of recreationally active female subjects with no previous history of knee pathology. Subjects with any systemic disease or metabolic disorder that might interfere with sensory input or motor function, or both, were excluded. The same investigators performed all testing procedures for all subjects. The dominant limb was used for the control group and was determined for each subject by identifying the leg she would use to kick a ball. All subjects were required to complete a questionnaire and provide consent before participating, in accordance with the University of Pittsburgh's Biomedical Institutional Review Board.

EMG Assessment

EMG data collection. The area and peak reactive muscle activity were collected bilaterally from 4 muscles: vastus medialis (VM), vastus lateralis (VL), medial hamstring (MH), and lateral hamstring (LH) (Figure 1). Each subject performed 4 functional activities: downhill walking (15°, 0.92 m/s), running (2.08 m/s), hopping (self-paced), and landing from a jump (20.3 cm) (Figure 2).

The side being tested and the order of functional activities were randomized. Self-adhesive Ag/AgCl bipolar surface electrodes (Multi Bio Sensors, Inc, El Paso, TX) detected myoelectric activity that was processed with the Noraxon Telemyo system (Noraxon USA, Inc, Scottsdale, AZ). Electrodes measured 10 mm in diameter and were placed 25 mm apart after the skin was shaved, lightly abraded, and cleaned with a 70% ethanol solution.[13] Electrode location was based on recommendations by Basmajian and DeLuca,[13] and the acceptable impedance between electrodes was less than 2 kΩ. Two force-sensitive resistors (FSRs) were secured to the head of the first metatarsal and the heel of the leg being tested (Figure 3).

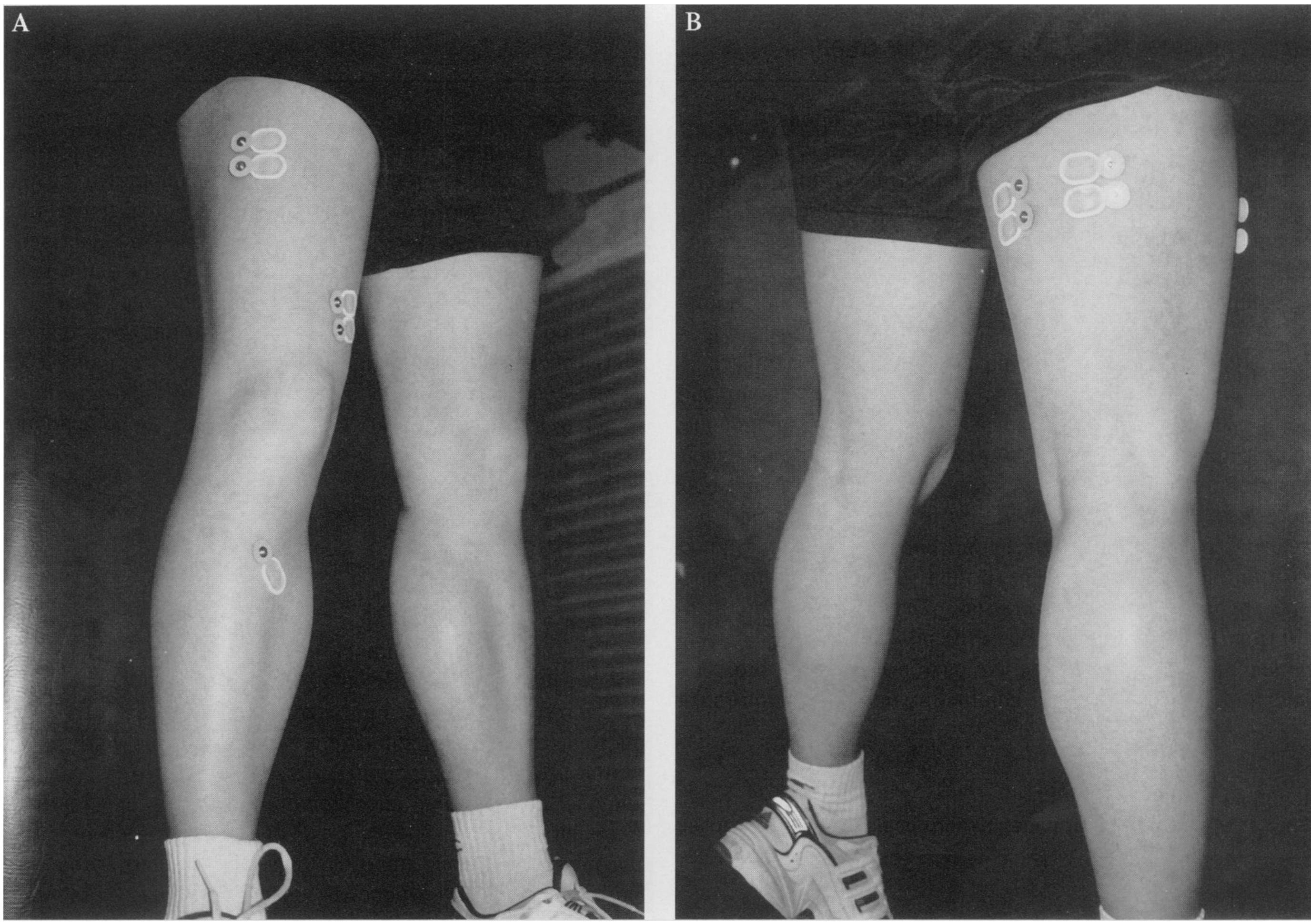

Figure 1. A, Electrode placement for the VM and VL and ground electrode over the proximal tibia. B, Electrode placement for the MH and LH muscles.

The FSRs were used to indicate the ground contact phase for each cycle of motion during the functional activities. Signals from the 4 muscles and FSRs were passed to a battery-operated 8-channel FM transmitter worn by the subject. A single-ended amplifier was used (gain 500) with Butterworth low-pass (15 Hz) and high-pass (500 Hz) filters and a common mode rejection ratio of 130 db. A receiver (gain 500, total gain 1000) converted the signal from analog to digital data with an A/D card (Keithley Metrabyte DAS-1000, Keithley Instruments, Inc, Taunton, MA). The signal then passed to a computer, where raw EMG data were sampled with a frequency of 2500 Hz and further analyzed with Myoresearch software (Noraxon). Before each test, the signal was calibrated with the patient in a relaxed position in order to establish the baseline amplitude of EMG activity.

EMG integration and normalization. All analyses were performed on integrated EMG (IEMG) data, which are expressed in μV·milliseconds. The raw signal was full-wave rectified and averaged over a 15-millisecond moving window. With a sampling rate of 1000 Hz, this equals 15 data points that slide one data point at a time. EMG data integration requires 16 milliseconds of processing time; however, the EMG channels are synchronized with the FSRs to adjust for this delay.

To normalize IEMG data for time, a linear envelope was established based on signals from the FSRs. Markers were placed defining a 250-millisecond period after ground contact (foot strike) during each cycle of motion. Therefore, the beginning of the linear envelope was indicated by the subject's initial contact with the ground, and the end was marked as a point 250 milliseconds after ground contact. This period of time was recorded to permit the collection of reflexive and voluntary muscle activity in response to joint loading.[6,9] Several (3 to 6) consecutive cycles of motion were then combined, using Myoresearch software, to construct a profile of the reactive muscle activity during the linear envelope. This is referred to as an ensemble-averaged profile (Figure 4).[14]

Amplitude normalization was performed to reduce the physiologic population variability.[14] The research of Yang and Winter[14] compared several amplitude normalization procedures in an attempt to reduce the large intersubject variability associated with EMG data and improve reliability. It was observed that the sensitivity of EMG testing could be increased

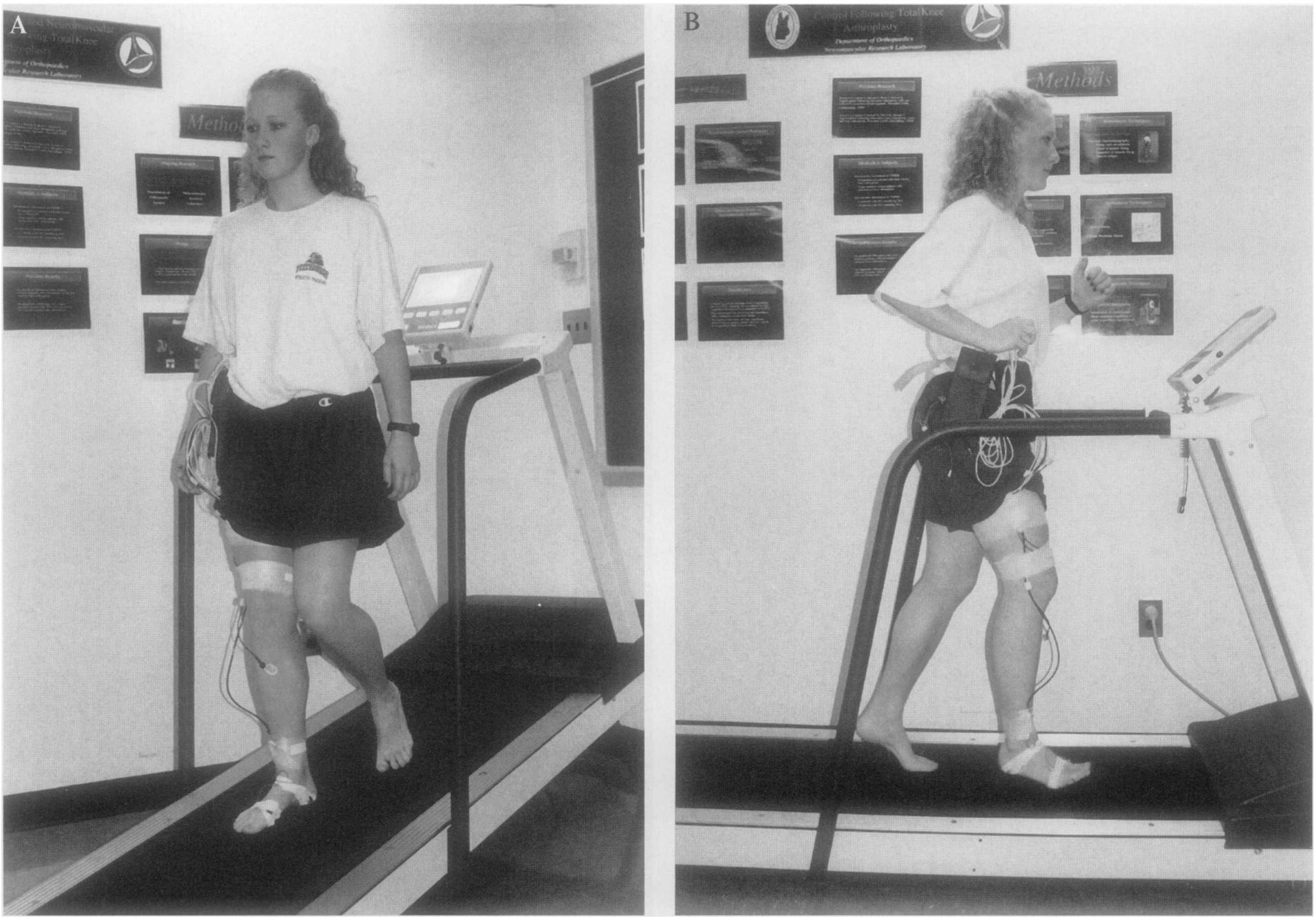

Figure 2. Subject performing 4 functional activities: downhill walking (15°, 0.92 m/s) (A); running (2.08 m/s) (B); hopping (self-paced) (C); and landing from a jump (20.3 cm) (D).

if the average pattern of EMG was constructed for specific functional activities (ensemble-averaged profile) and the mean or peak amplitude that occurs during this pattern was selected to normalize the amplitude of muscle activity. When compared with normalization procedures using a maximum voluntary contraction, 50% maximum voluntary contraction, and EMG per unit of force, the ensemble mean or peak amplitude provides the least intersubject variability during the dynamic types of muscle activation patterns occurring with functional activities.

Based on the results of Yang and Winter,[14] the amplitude of reactive muscle activity for each subject was normalized to the mean amplitude of the ensemble profile for each functional activity. Amplitude normalization converted the IEMG data (μV·milliseconds) into a value that represents a percentage of the ensemble mean (%·milliseconds). Myoresearch software was used to calculate this value. The area and peak IEMG values were used to represent the amplitude of reactive muscle activity (Figure 5).

Statistical Analysis

The independent variable was the condition of the ACL. The dependent variables were measures of reactive muscle activation (area and peak IEMG). Multiple paired t tests were used to establish differences between the involved and uninvolved limbs. A one-way analysis of variance was used to determine differences among the 3 groups. Tukey post hoc analysis was performed when significant differences were established. A probability level of $P < .05$ was accepted to denote statistical significance.

RESULTS

During running, the ACLD group demonstrated significantly greater area and peak IEMG activity in the MH in comparison with the ACLR group and greater peak activity in the LH when compared with the control group. The ACLD group also demonstrated greater peak activity in the VM and less area of IEMG activity in the LH than the control group during running. During landing, the ACLD group demonstrated significantly less area of IEMG activity in the VL when compared with the control group (Table). No significant differences were identified between the ACLR and the control groups, nor were side-to-side differences revealed within the 3 groups.

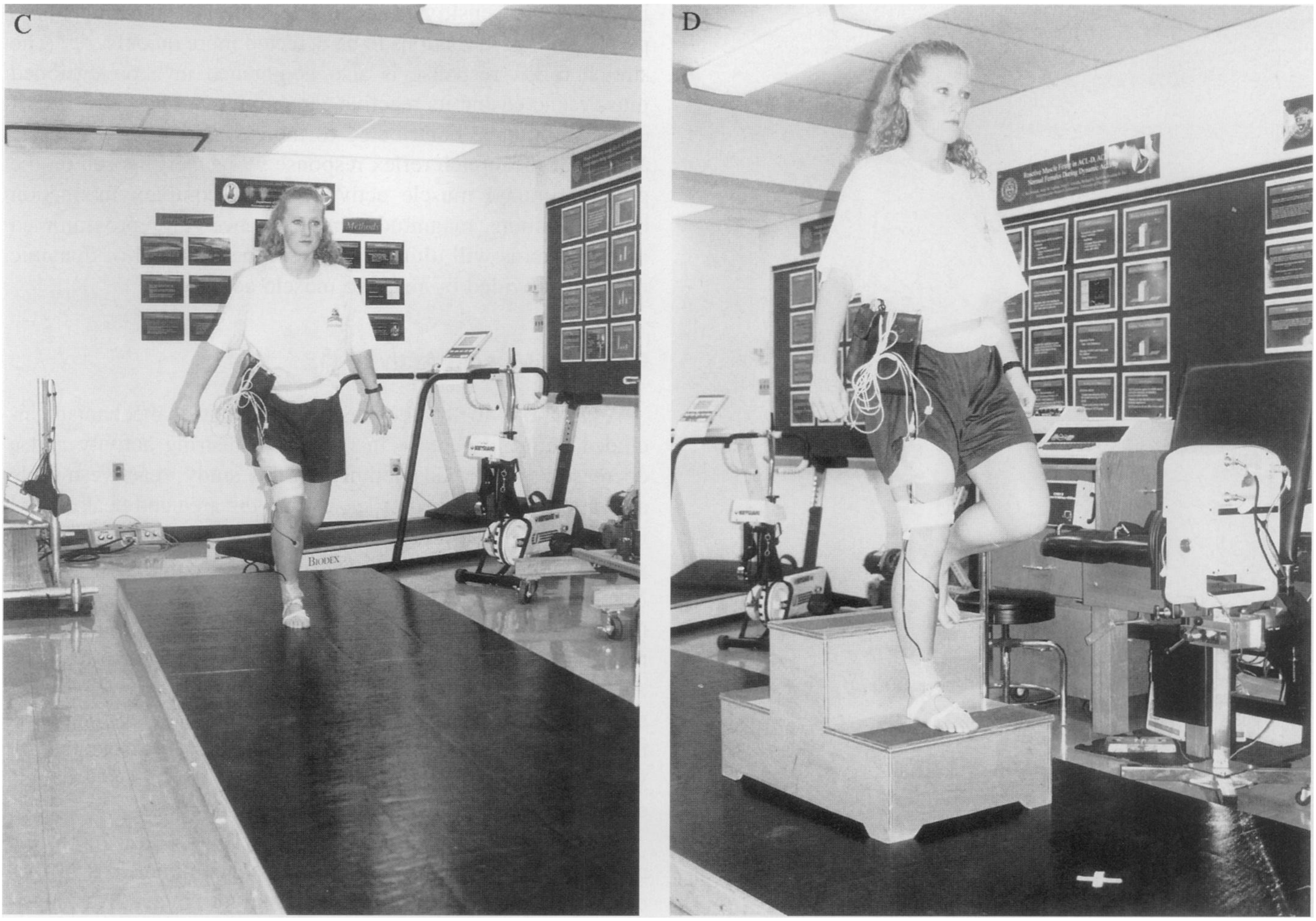

Figure 2. Continued.

DISCUSSION

The objective of our research study was to examine the role of the thigh musculature in providing dynamic restraint to the knees of female subjects. Our results suggest that, during some functional activities, ACLD females adopt reactive muscle firing strategies that are beneficial to the dynamic restraint mechanism and maintenance of functional joint stability. These strategies minimize anterior tibial translation in response to joint loading and include increased hamstring activation and quadriceps inhibition. The magnitude of muscle activation in ACLR subjects, however, did not differ significantly from uninjured females. This suggests that surgical reconstruction restores the mechanical function of the ACL, which is then recognized by the central nervous system and results in muscle activation patterns similar to healthy individuals. The possibility of graft reinnervation is controversial; however, regrowth of free nerve endings would provide proprioceptive feedback and could influence muscle activation patterns.[12,18] Although this would support similarities in the muscle activation strategies of ACLR and healthy females, continued research is needed. The absence of side-to-side differences, specifically in ACLD subjects, suggests that neuromuscular adaptations after ACL injury occur bilaterally. Our study also supports the use of EMG amplitude normalization with ensemble means as a method for minimizing variability between subjects and identifying significant group differences in the amplitude of muscle activity during functional activities.

Feedback Motor Control

The feedback mechanism of motor control is characterized by numerous reflex pathways from joint and muscle receptors that reflexively coordinate muscle activity during the performance of a task.[19–21] Muscle activity elicited through reflex pathways is again receiving attention for its role in the dynamic restraint mechanism. However, controversy still exists regarding the capacity of reactive muscle activity to contribute to functional joint stability in the ACLD subject.[5,6]

Several factors mediate the level of dynamic restraint provided by reactive muscle activity. These include the timing and magnitude of reactive muscle activity, as well as the existence of preprogrammed motor commands.[6,22] The peripheral site where sensory feedback originates and the neural pathways it follows both contribute to the response time of

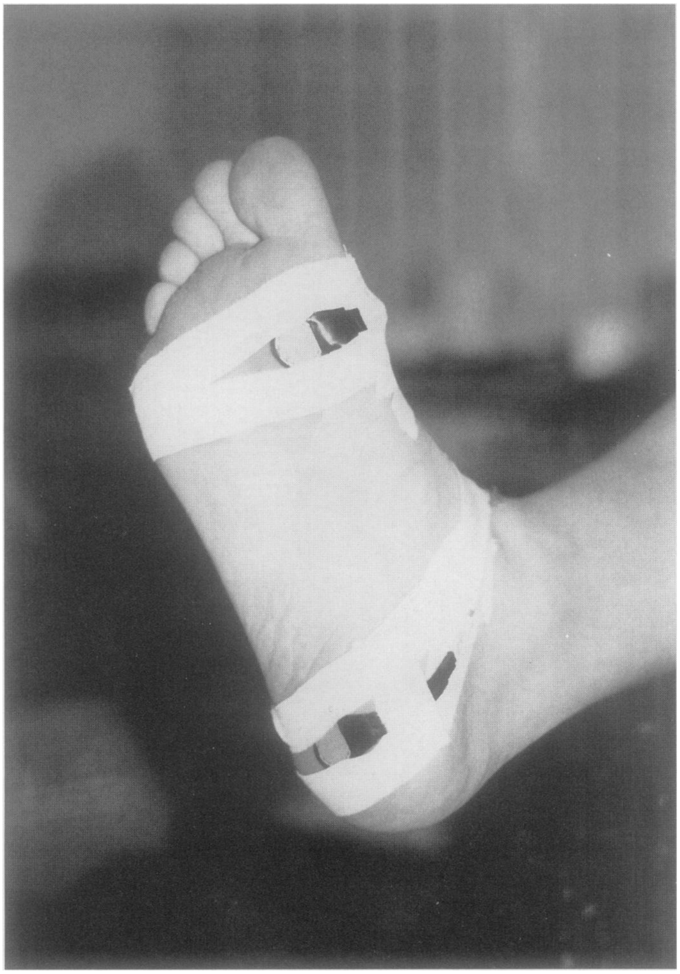

Figure 3. Two force-sensitive resistors (FSRs) secured to the head of the first metatarsal and heel mark ground and air phases of the functional activities.

muscles. Signals that originate in muscle spindles act directly on the motor nerves (stretch reflex), whereas signals from other mechanoreceptors must pass through a series of synapses before stimulating a motor response.[9] The reactive muscle activity observed in this study occurred quickly (<250 milliseconds) and appears to offer adaptations beneficial for dynamic restraint and the maintenance of functional stability in ACLD knees. Muscle spindles also elicit vigorous muscular contractions when compared with the relatively weak activity induced by loads placed on capsule and ligamentous mechanoreceptors.[4] The ACLD subjects exhibited more reactive muscle activity despite ACL injury, emphasizing the importance of muscle spindle receptors rather than capsuloligamentous mechanoreceptors in modifying reactive muscle activity.

Preparatory muscle activity is a confounding variable that may have influenced the timing and magnitude of reactive muscle firing. Joint loads incurred during these functional activities may have been anticipated based on previous experiences.[10,23] It has been established that the level of preparatory muscle activity, in anticipation of joint loading, will influence reactive muscle activation strategies. Muscle preactivation increases the sensitivity of muscle spindles, allowing unexpected joint perturbations to be detected more quickly.[23,24] The stretch reflex response is also heightened in a pretensioned muscle, increasing its reactive capabilities.[24] This sequence of events may have contributed to increases in reactive muscle activity if the stretch reflex response was superimposed on the preprogrammed muscle activity.[23] The complex interaction between timing, magnitude, and existence of preprogrammed motor patterns will ultimately determine the level of dynamic restraint provided by reactive muscle activity.

Reactive Muscle Activity

Adaptations to the feedback motor control mechanism included both an increase in reactive hamstring activity and a decrease in quadriceps activity. In this study, reactive muscle activation was assessed by measuring the area and peak IEMG in the lower extremity for a 250-millisecond period after ground contact during several functional activities. McNair and Marshall[3] conducted a similar investigation, simultaneously recording 128 milliseconds of EMG activity and ground reaction forces during a landing task. They demonstrated that ACLD subjects with greater hamstring muscle activation were able to reduce stress on the knee during landing. This supports the results of our study, and, although reactive muscle activity was observed in all of the subjects, only ACLD subjects exhibited increased hamstring muscle activation.

We concluded that reactive muscle activity is affected by the absence of sensory information and concomitant loss of mechanical restraint normally provided by the intact ACL. Various other structures containing peripheral mechanical receptors may have provided sufficient sensory information to mediate reactive muscle activity and, therefore, compensate for the reflexive influence normally projected by ACL afferents. Modifying the amplitude of reactive muscle activity is an adaptation to increase hamstring muscle stiffness and resist excessive anterior translation of the tibia in the ACLD knee.

Quadriceps Avoidance

The results of our study also confirmed the existence of what Berchuck et al[1] referred to as a "quadriceps-avoidance" gait adaptation in ACLD subjects. Previous research using motion analysis has revealed that, during walking, jogging, and running, ACLD subjects alter their gait to minimize anterior translation by inhibiting quadriceps muscle activation. Although Berchuck et al[1] did not record EMG data, this finding was attributed to excessive shear forces created by quadriceps activation. Gauffin and Tropp[25] were able to document similar results by measuring muscle activation patterns during a one-leg hop. Although hamstring activity was unremarkable, the ACLD group had significantly less quadriceps activity than the control group.

One issue arising from the results of this study contradicts the theory of a quadriceps-avoidance mechanism. A signifi-

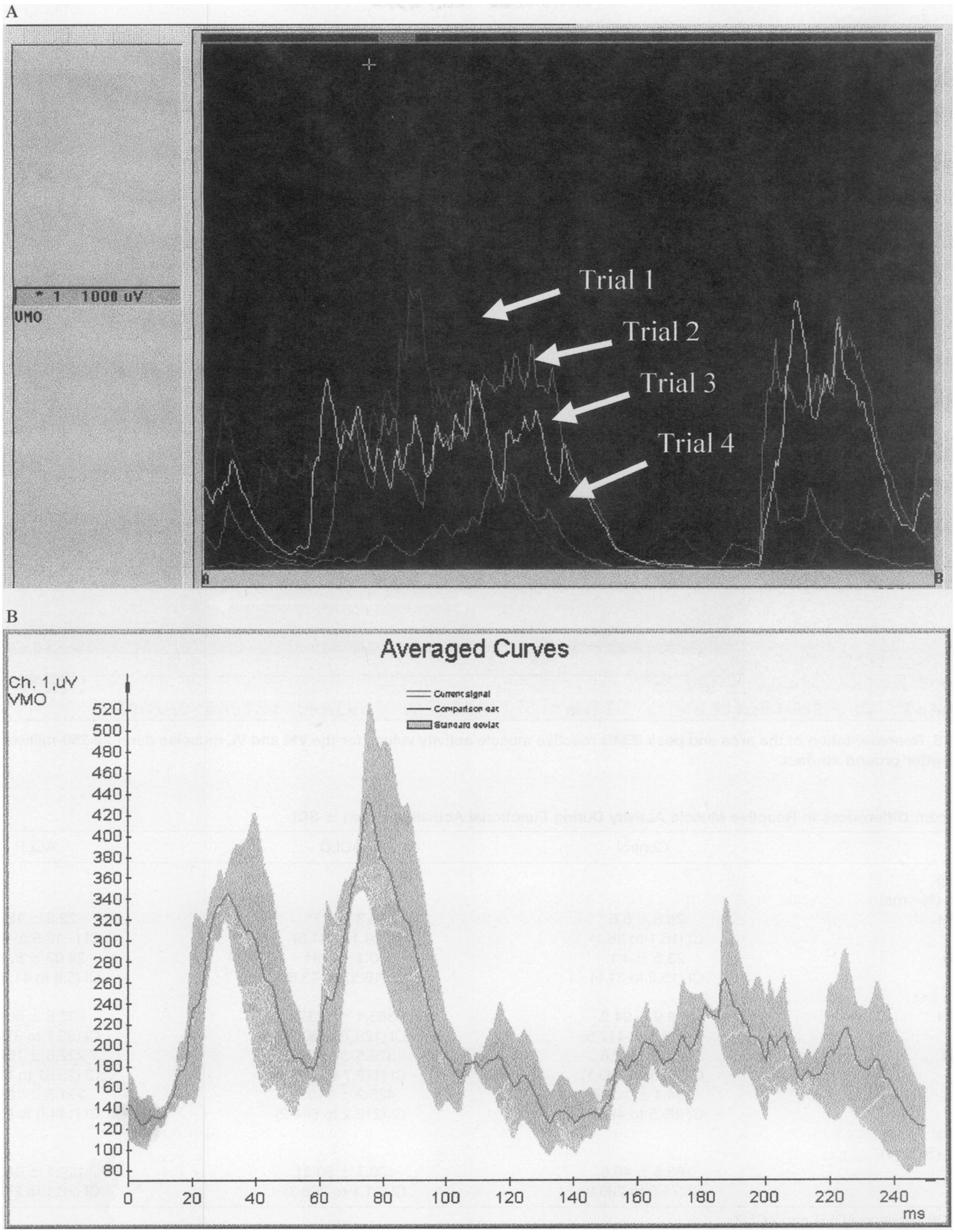

Figure 4. A, VM muscle activity over 4 consecutive cycles of motion are combined to construct a profile of the reactive muscle activity during the 250-millisecond linear envelope. B, The pattern of muscle activity in the VM constructed from consecutive cycles of motion is referred to as the ensemble averaged profile.

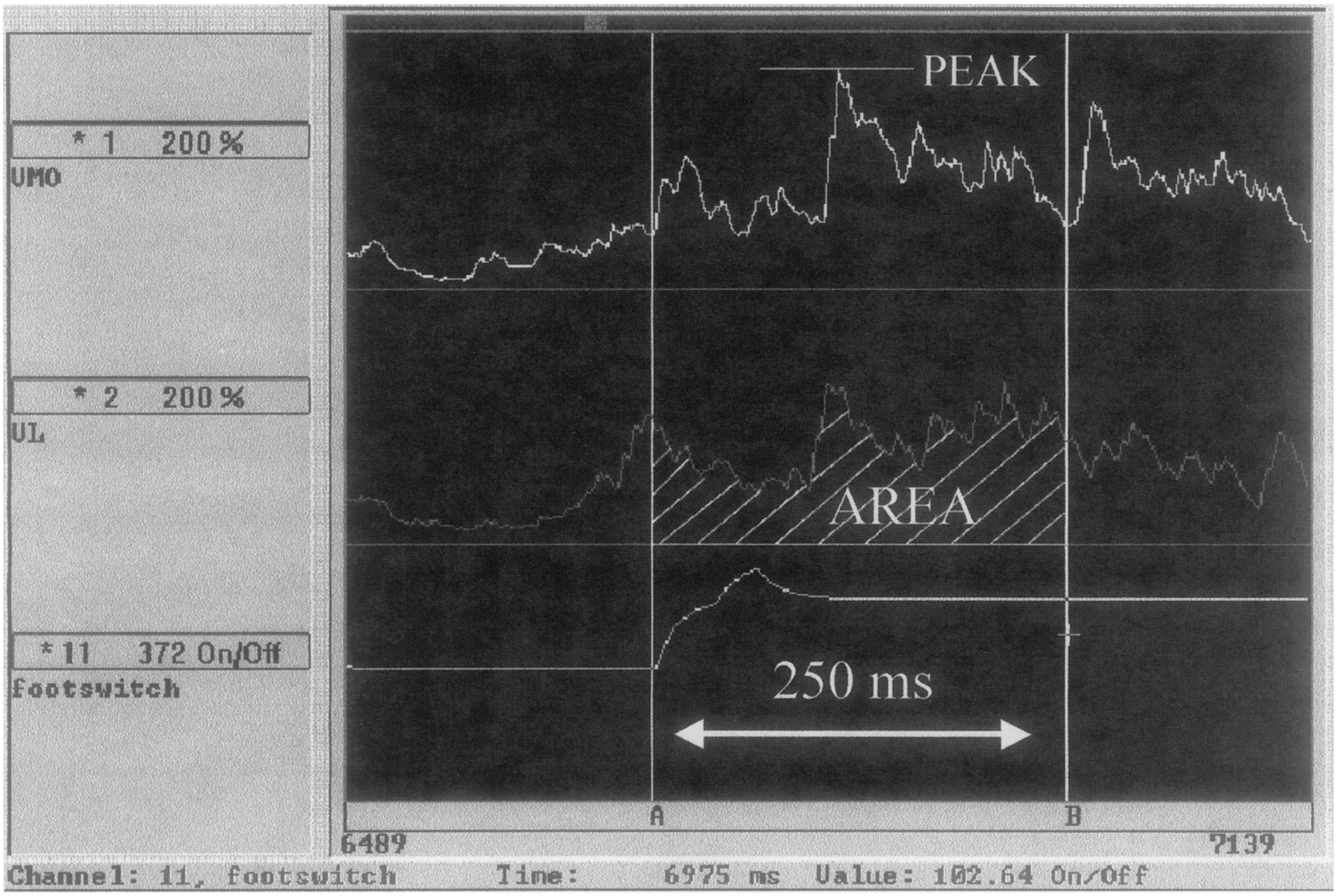

Figure 5. Representation of the area and peak IEMG reactive muscle activity values for the VM and VL muscles during a 250-millisecond period after ground contact.

Significant Differences in Reactive Muscle Activity During Functional Activities (Mean ± SD)

	Control	ACLD	ACLR
Running			
Area (% · ms)			
MH	26.6 ± 5.6	30.3 ± 5.7*	22.5 ± 18.4
	CI (15.1 to 38.1)	CI (19.1 to 41.5)	CI (−13.6 to 58.5)
LH	23.5 ± 4.1	30.1 ± 6.9†	24.02 ± 8.8
	CI (15.5 to 31.5)	CI (16.57 to 43.6)	CI (6.8 to 41.3)
Peak (%)			
MH	232.9 ± 94.2	365.4 ± 123.3*	205.8 ± 86.8
	CI (48.3 to 417.5)	CI (123.7 to 607.1)	CI (35.7 to 375.9)
LH	186.6 ± 52.8	379.5 ± 105.5†	222.8 ± 100.6
	CI (83.1 to 290.1)	CI (172.7 to 586.3)	CI (25.57 to 419.9)
VM	294.1 ± 104.4	428.2 ± 110.2†	391.5 ± 126.3
	CI (89.5 to 498.7)	CI (212.2 to 644.2)	CI (144.0 to 639.0)
Landing			
Area (% · ms)			
VL	168.8 ± 49.6	109.7 ± 50.3†	149.1 ± 64.8
	CI (71.6 to 266.0)	CI (11.1 to 208.3)	CI (22.1 to 276.1)

* $P < .05$ between ACLD and ACLR.
† $P < .05$ between ACLD and control.
CI, Confidence interval (mean ± 1.96 SD).

cantly greater peak VM activity was observed in ACLD subjects when compared with the control subjects. This would appear to exacerbate anterior translation but may also be interpreted as a mechanism to control knee joint deceleration through coactivation of the VM and hamstring muscles.[26] The net effect of coactivation may increase overall joint stiffness and assist with dynamic restraint.[4] Although further research is warranted, the results of our study agree with previous literature suggesting that ACLD subjects adopt reactive muscle activation strategies to minimize anterior tibial translation in response to joint loading.

CONCLUSIONS

Our results suggest that adaptations occur to the feedback mechanism of motor control in ACLD females. Significant differences were identified in the reactive muscle activation strategies by measuring the area and peak IEMG activity in the thigh musculature during functional activities. Strategies to minimize anterior tibial translation in response to joint loading included increased hamstring activity and quadriceps inhibition during a window of 250 milliseconds. The reactive muscle activity exhibited in ACLD subjects is presumably an attempt to regain functional stability through the dynamic restraint mechanism. However, ACLR subjects did not demonstrate significant differences when compared with uninjured subjects. The absence of side-to-side differences suggests that adaptations to the reflex pathways occur bilaterally in ACL-injured females. This study may serve as a platform for future research with larger samples including the effects of time, sex, and various surgical or rehabilitation strategies.

REFERENCES

1. Berchuck M, Andriacchi TP, Bach BR, and B Reider. Gait adaptations by patients who have a deficient anterior cruciate ligament. *J Bone Joint Surg Am.* 1990;72:871–877.
2. Branch TP, Hunter R, Donath M. Dynamic EMG analysis of the anterior cruciate deficient legs with and without bracing during cutting. *Am J Sports Med.* 1989;17:35–41.
3. McNair PJ, Marshall RN. Landing characteristics in subjects with normal and anterior cruciate ligament deficient knee joints. *Arch Phys Med Rehabil.* 1994;75:584–589.
4. Solomonow M, Baratta R, Zhou BH, et al. The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. *Am J Sports Med.* 1987;15:207–213.
5. Beard DJ, Kyberd PJ, Fergusson CM, Dodd CAF. Proprioception after rupture of the anterior cruciate ligament: an indication of the need for surgery? *J Bone Joint Surg Br.* 1993;75:311–315.
6. Wojtys EM, Huston LJ. Neuromuscular performance in normal and anterior cruciate ligament-deficient lower extremities. *Am J Sports Med.* 1994;22:89–104.
7. Woo SL, Sofranko RAZ, Jamison JP. Biomechanics of knee ligaments relating to sports medicine. In Fu FF, Stone DA, eds. *Sports Injuries: Mechanism, Prevention, Treatment.* Baltimore, MD: Williams & Wilkins; 1994:67–80.
8. Freeman MAR, Wyke B. Articular contributions to limb reflexes: the effects of partial neurectomy of the knee-joint on postural reflexes. *Brit J Surg.* 1966;53:61–68.
9. Kandell ER, Schwartz JH, Jessell TM. *Principles of Neural Science.* 3rd ed. Norwalk, CT: Appleton Lange; 1996:535–537, 619.
10. Dunn TG, Gilli SE, Ponser SE, Weil N. The learning process in biofeedback: is it feed-forward or feedback? *Biofeedback Self-Regul.* 1986;11:143–155.
11. Johansson H, Sjölander P, Sojka P. A sensory role for the cruciate ligaments. *Clin Orthop.* 1991;268:161–178.
12. Barrack RL, Lund PJ, Munn BG, Wink C, Happel L. Evidence of reinnervation of free patellar tendon autograft used for anterior cruciate ligament reconstruction. *Am J Sports Med.* 1997;25:196–202.
13. Basmajian JV, DeLuca C. *Muscles Alive: Their Functions Revealed in Electromyography.* 5th ed. Baltimore, MD: Williams & Wilkins; 1985.
14. Yang JF, Winter DA. Electromyographic amplitude normalization methods: improving their sensitivity as diagnostic tools in gait analysis. *Arch Phys Med Rehabil.* 1984;65:517–521.
15. Lephart SM, Perrin DH, Fu FH, Gieck JH, McCue FC, Irrgang JJ. Relationship between selected physical characteristics and functional capacity in the anterior cruciate-insufficient athlete. *J Orthop Sports Phys Ther.* 1992;16:174–180.
16. Lysholm J, Gillquist J. Evaluation of knee ligament surgery results with special emphasis on use of scoring scale. *Am J Sports Med.* 1982;10:150–154.
17. Tegner Y, Lysholm J, Odensten M, Gillquist J. Evaluation of cruciate ligament injuries: a review. *Acta Orthop Scand.* 1988;59:336–341.
18. Barrack RL, Skinner HB, Brunet ME, Cook SD. Joint laxity and proprioception in the knee. *Physician Sportsmed.* 1983;11(6):130–135.
19. Brener J. Sensory and perceptual determinants of voluntary visceral control. In: Schwartz GE, Beatty J, eds. *Biofeedback: Theory and Research.* New York, NY: Academic Press; 1977:29–66.
20. Lacroix JM. The acquisition of autonomic control through biofeedback: the case against an afferent process and a two-process alternative. *Psychophysiology.* 1981;18:573–587.
21. Merton PA. Speculations on the servo-control of movement. In: Wolstenholme GEW, ed. *The Spinal Cord.* London, England: Churchill; 1953: 247–255.
22. Konradsen L, Voigt M, Hojsgaard C. Ankle inversion injuries: the role of the dynamic defense mechanism. *Am J Sports Med.* 1997;25:54–58.
23. Dietz V, Noth J, Schmidtbleicher D. Interaction between pre-activity and stretch reflex in human triceps brachii during landing from forward falls. *J Physiol (Lond).* 1981;311:113–125.
24. Dyhre-Poulsen P, Simonsen EB, Voigt M. Dynamic control of muscle stiffness and H reflex modulation during hopping and jumping in man. *J Physiol (Lond).* 1991;437:287–304.
25. Gauffin H, Tropp H. Altered movement and muscular activation patterns during the one-legged jump in patients with an old anterior cruciate ligament rupture. *Am J Sports Med.* 1992;20:182–192.
26. Ireland ML, Gaudette M, Crook S. ACL injuries in the female athlete. *J Sport Rehabil.* 1997;6:97–110.