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Neuromuscular Dynamic Restraint in Women with Anterior Cruciate Ligament Injuries

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The purpose of this study was to identify neuromuscular characteristics related to dynamic restraint in the knee. Observing compensatory changes to these characteristics in women with anterior cruciate ligament injuries provides important information for understanding functional knee stability, injury prevention, and performance. Twelve female subjects with anterior cruciate ligament injuries and 17 female control subjects participated in this study to assess electromyographic activity during landing from a hop and knee perturbation; hamstring muscle stiffness and flexibility; and isokinetic strength. Females with anterior cruciate ligament deficiencies had significantly increased preparatory muscle activity in the lateral hamstring before landing, but no differences in reactive muscle activity during landing or reflex latency after joint perturbation. Females with anterior cruciate ligament deficiencies had significantly less hamstring muscle stiffness and flexibility, but also had greater peak torque and torque development for knee flexion. Lower Lysholm scores were observed in females with anterior cruciate ligament deficiencies but no difference was found in functional performance of the single leg hop test. These neuromuscular characteristics provide a foundation for future research investigating injury prevention and rehabilitation techniques that maximize dynamic restraint through stiffness regulation and the timing of specific muscle activation strategies.

The incidence of noncontact anterior cruciate ligament (ACL) injuries in females has attracted attention to the dynamic restraint mechanism and its role in protecting capsuloligamentous structures from excessive joint loads.[22] The capacity of the dynamic restraint mechanism is dictated by several neuromuscular characteristics including preparatory and reactive muscle activation, muscle stiffness, flexibility, and muscle force production.[2,11,38,45,54] Changes in these compensatory mechanisms may affect performance in those with ACL injuries. Identifying a pattern of aberration in the dynamic restraint mechanism may lead to new prevention and treatments strategies.[45] Those with ACL injuries who return to high levels of physical activity may have the most successful adaptive mechanisms, preventing repeated bouts of functional instability and minimizing serious pathologic sequelae.[42,45,59]

Previous studies have had mixed results related to the most advantageous characteristics of the dynamic restraint mechanism. Some electromyography (EMG) research links preparatory or reactive muscle activation strategies to enhanced dynamic restraint.[2,5,8,9,27,38,45,48,58] Other studies suggest biomechanical factors such as muscle stiffness, flexibility, and strength correlate well with functional performance.[20,38,45,58] Although good clinical research suggests that all of these neuromuscular characteristics are important to dynamic restraint, incomplete data exist regarding the relationship between these characteristics and the functional status of individuals with ACL injuries. Therefore, the purpose of the current study was to investigate the dynamic restraint mechanism by establishing the neuromuscular characteristics of lower extremity muscles in females with ACL injuries and healthy subjects. In addition, the relationships between these neuromuscular characteristics and function were explored. Where moderate to strong correlations were seen, the ability to predict function by assessing these characteristics also was determined.

MATERIALS AND METHODS

Experimental Design

This was a stratified post-test only control group design consisting of 29 female volunteers. The independent variable was the

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condition of the ACL. The dependent variables were muscle activity before and after landing from a hop, as measured by the area of integrated EMG recordings, hamstring latency after joint perturbation, hamstring muscle stiffness, knee flexion and extension force production, hamstring flexibility, and function. Variability of pilot data from EMG studies yielded the most conservative estimate of the sample size for this experimental study. A minimum effect size of $K^2 = 0.20$ between groups and a probability level of $\Delta = 0.05$ required 10 subjects in each group for a statistical power of $p = 0.83$.[17] Pilot experiments confirmed moderate to high test-retest correlation coefficients ranging from 0.80–0.99.[2,4,32,36,44,49,50,53]

Subjects were required to complete a questionnaire and provide consent in accordance with the university's biomedical institutional review board. All subjects completed, in order, the Lysholm functional knee rating scale and tests for muscle force production. The measure of peak torque was required for assessing muscle stiffness. The order of the remaining tests was counterbalanced, including the single leg hop for distance, muscle activity during landing, hamstring latency, muscle stiffness, and flexibility assessment. No reports of knee pain or functional instability occurred during testing.

Subjects

The experimental group (n = 12; age, 25.2 ± 7.3 years) had complete unilateral anterior cruciate ligament tears at least 1 year before (mean time, 33.6 ± 5.2 months) shown by mechanical instability.[33] The criteria for mechanical instability were positive Lachman and pivot shift tests assessed by an orthopaedic surgeon, and anterior laxity greater than 3 mm (ACL-injured with 8.6 ± 2.7 mm; control, 4.9 ± 1.45 mm) (KT 1000 MEDmetric Corp, San Diego, CA) compared with the uninvolved limb.[10,30,33] For inclusion all subjects had a minimum Tegner activity score of 3 (ACL-injured, 5.4 ± 1.83; control, 5.41 ± 1.5).[33,49,50] In addition, the experimental group must have completed a rehabilitation program, and had a range of motion (ROM) of at least 90° flexion and less than 5° flexion contracture.[30] Subjects with disability attributable to secondary meniscal damage or ligamentous injury in excess of Grade I, or those who had reconstructive surgery were excluded.

The control group (n = 17; age, 22.7 ± 4.0 years) consisted of recreationally active subjects between the ages of 18 and 30 years, with no previous history of knee disorder. The dominant limb was used for testing and was determined for each subject by identifying the leg in which they would prefer to kick a ball.

Electromyographic Assessment

Surface EMG data were collected from the vastus medialis, vastus lateralis, medial hamstring, and lateral hamstring muscles during landing from a hop, anterior tibial displacement, and muscle stiffness testing. Multi Bio Sensors self adhesive Ag-AgCl bipolar surface electrodes (Multi Bio Sensors Inc, El Paso, TX) detected myoelectric activity and was processed with the Noraxon Telemyo system (Noraxon USA Inc, Scottsdale, AZ). The location for placing electrodes was identified by bony landmarks and by palpating the midlength of the contractile component during an isometric contraction. The ground electrode was positioned over the proximal tibia. Each electrode measuring 10 mm in diameter was placed 25 mm apart after the skin was shaved, lightly abraded, and cleaned with 70% ethanol solution. The acceptable impedance between paired electrodes for each muscle was less than 2 kΩ and was measured with a standard multimeter. Signals from the muscle leads were passed to a battery-operated eight-channel frequency modulation transmitter worn by the subject. A single-ended amplifier was used (gain 500) with a fourth order Butterworth filter (10–500 Hz) and a common mode rejection ratio of 130 db. A receiver (gain 2, total gain 1000) converted the signal from analog to digital data with an A/D card (Keithley Metrabyte DAS-1000; Keithley Instruments, Inc, Tauton, MA). The signal then was passed to a computer where raw EMG data were sampled at a frequency of 2500 Hz, and analyzed with Myoresearch software (Noraxon USA Inc). Before each test, the myoelectric signal was calibrated with the subject in a relaxed position to establish the baseline EMG activity. Raw EMG data were processed by full-wave rectification and smoothing over a 15-ms moving window.

Muscle Activity during Landing

The subject stood on a 20-cm step, balanced momentarily on the test limb, and hopped to a target (x) placed 30 cm horizontally (Fig 1).[37] The subject did two practice attempts followed by three test trials and the ensemble peak was used for amplitude normalization.[61] Two force sensitive resistors were mounted on the heel and head of the first metatarsal to mark ground contact. The area of integrated EMG preparatory muscle activity was represented by a 150-ms period before landing and reactive muscle activity was described by a 250-ms period after ground contact (Fig 2).[37,58]

Hamstring Latency after Joint Perturbation

Reflexive muscle activity in the hamstrings also was assessed by measuring the onset time after anterior translation of the tibia. A special knee perturbation device was designed, based on previous publications, to impart a 100-N force on the posterior aspect of the tibia while anterior displacement of the tibia and raw EMG data were recorded (Fig 3).[2,24,58] The subject's foot was placed on a scale to monitor weightbearing status (9–13 kg) and secured in 10°–15° ankle dorsiflexion.[58] The knee and hip were placed in 30° flexion with restraints around the anterior thigh.[2,58] Two linear potentiometers (Omega Technology, Stamford, CT) interfaced with the EMG computer were positioned to quantify linear displacement in increments of 0.05 mm. The first linear potentiometer was placed against the inferior pole of the patella and the second was placed on the tibial tubercle. The difference between these two values eliminated error attributable to extraneous movement and established the onset of anterior displacement of the tibia relative to the femur.

A pneumatic cylinder (Tri State Hydraulics, Pittsburgh, PA) attached to a sliding mechanism imparted a 100 ± 1.6-N force to the posterior aspect of the tibia displacing it anteriorly.[2,24,58] Simultaneously, EMG signals from the four thigh muscles and linear potentiometers were passed to the computer for analysis.[58] Two practice trials were done followed by three test trials. A 100-ms period before the onset of tibia translation was analyzed

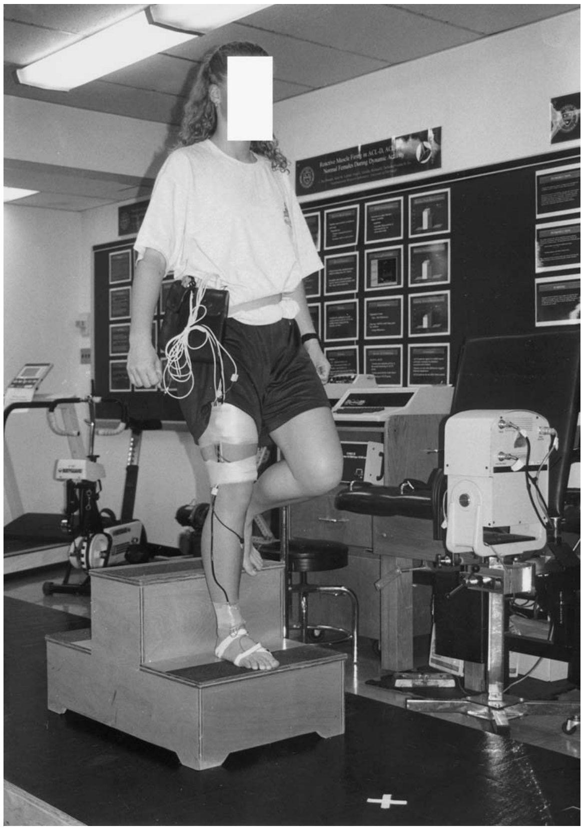

Fig 1. Preparatory and reactive muscle activities were recorded with an EMG telemetry unit while the subject did a landing task.

to establish a mean and standard deviation in microvolts (μV) of the baseline signal.[41] The criteria of 4 standard deviations above the mean baseline amplitude was used to denote the onset of reactive muscle activity and was recorded in milliseconds (Fig 3A).[41]

Hamstring Stiffness Assessment

Muscle stiffness was calculated by modeling the hamstring and lower leg after one-degree-of-freedom mass spring system.[38,57] Subjects would lie prone on a table with the thigh supported in 30° hip flexion (Fig 4). A boot attached to the subject's foot was weighted corresponding with 45% of the subject's peak torque.[38] The subject then was instructed to position the lower leg parallel to the floor (30°-knee flexion) maintaining a constant level of contraction in the hamstring muscles with the aid of the EMG display and verbal cues.[38,47] A force (approximately 100 N) was applied manually to the posterior aspect of the boot initiating small oscillations of the lower leg.[38,47,54] A uniaxial accelerometer (NeuwGhent Technology, LaGrangeville, NY) attached to the boot measured the damped natural frequency of oscillation (Hz) of the lower leg. The frequency was determined from the interval between the first complete sinus cycle displayed as successive maximum amplitude peaks. The change in amplitude between successive peaks, measured in gravitational force (g), was used to calculate the coefficient of damping. With these two values, and knowledge of the mass attached to the lower leg, the stiffness of the hamstring muscles was calculated (Fig 5).[38,53,56] Anthropometric data were used to transform the linear stiffness value of Newton-meters (N-m) into angular equivalents (N-m rad^{-1}).[56] Two practice and three test trials were done.

Muscle Force Production

Isokinetic strength testing was done with the Biodex System 2 Dynamometer (Biodex Medical Inc, Shirley, NY) to assess peak torque and torque at 0.2 seconds of the quadriceps and hamstring muscle groups. For isokinetic strength, a standardized knee position was assumed and testing was done at speeds of 60°/second with torque values automatically adjusted for gravity by the Biodex Advantage Software v. 4.0 (Biodex Medical Inc).[19,44] Warm-up procedures consisted of two submaximal (50% and 75%), and one maximal repetition followed by data collection during five reciprocal maximum repetitions.

Flexibility

Hamstring flexibility was measured with the active knee extension test (Fig 6).[60] The subject was positioned supine on a table with the test limb in 90° hip and knee flexion. The anterior surface of the thigh was placed in contact with a crossbar attached to the table and the axis of a goniometer was positioned at the knee. The subject actively extended the knee until the thigh began to break contact with the crossbar and the degrees from full knee extension were recorded.

Functional Performance and Impairment

Functional performance was assessed by measuring the distance of a single leg hop.[4,26] Subjects stood on their involved or dominant limb, with the opposite limb positioned at 90° hip and knee flexion and arms placed on their hips.[4,26] The subjects were asked to achieve the maximum horizontal distance without swinging the arms or contralateral leg. The horizontal distance (cm) was measured and the mean of three trials was normalized to the subject's stature.[26] The Lysholm knee rating scale contains eight items based on clinical findings seen after knee ligament injury.[36] Each of the eight items was scored based on the frequency of symptoms and the subject's ability to do the specified activity. A sum of the scores represented knee function with a rating of 100 indicating no impairment.[36]

Statistical Analysis

Multiple t tests with Bonferroni correction were used to establish mean group differences between the experimental and control groups on the dependent variables. A $p < 0.05$ was accepted to denote statistical significance.[17] To establish the relationship between the neuromuscular characteristics and function, Pearson product-moment correlation coefficients were calculated.[17] When moderate to strong correlations existed, a stepwise multiple regression analysis was done to determine the degree to which the neuromuscular characteristics can be used to predict function.

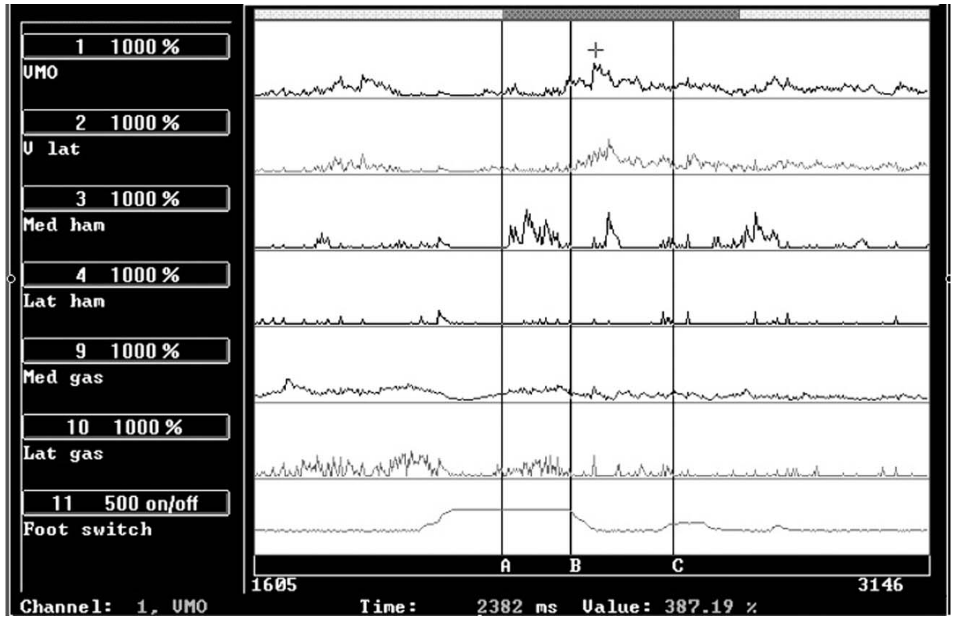

Fig 2. Electromyographic data (channels 1–10) were synchronized with force sensitive resistors (channel 11) to establish preparatory (150 ms between markers A and B) and reactive (250 ms period between markers B and C) muscle activity.

RESULTS

The analysis of integrated EMG data revealed a significant increase in the area of preparatory activity in the lateral hamstring muscles for the ACL-injured group (Table 1) but no differences were identified in the area of reactive muscle activity (Table 2) or hamstring latency after joint perturbation (Table 3). Muscle stiffness testing revealed that the ACL-injured group had significantly lower hamstring muscle stiffness (mean, 2.90 ± 1.11 N-m rad^{-1}/weight) than the control group (mean, 3.84 ± 1.51 N-m rad^{-1}/weight). The ACL-injured group also showed

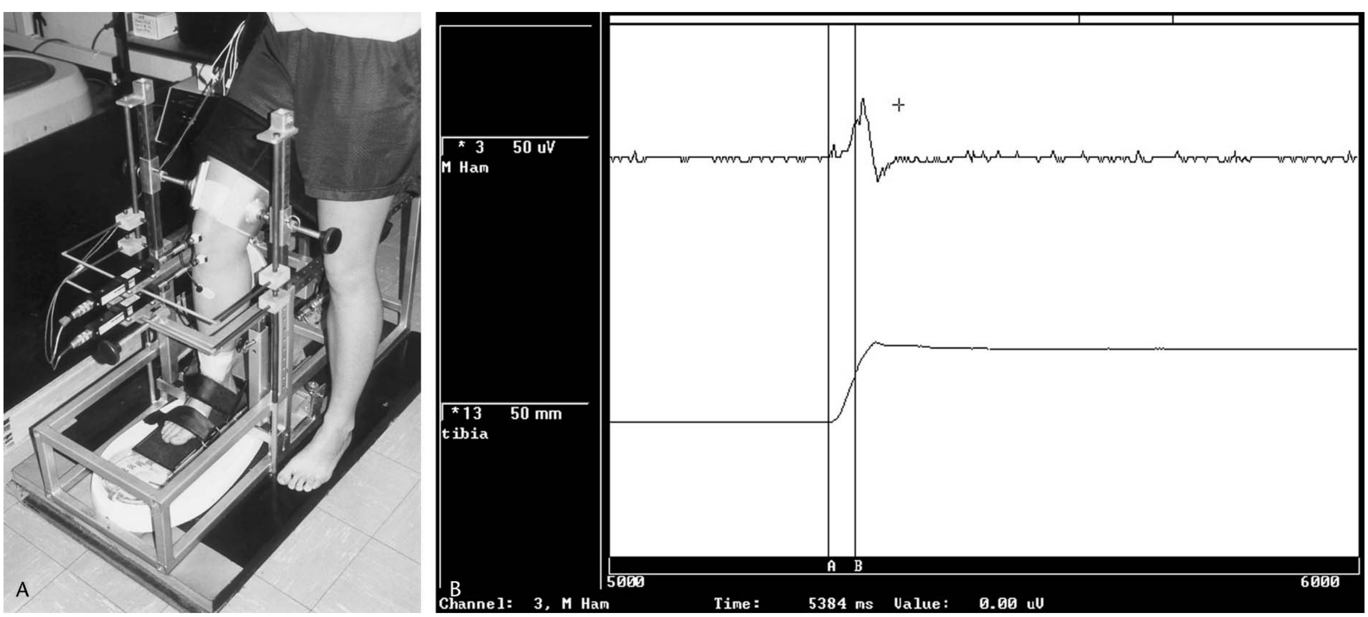

Fig 3A–B. (A) A joint perturbation device induced anterior tibial translation while EMG activity in the hamstring muscles was recorded. (B) A typical EMG recording shows the onset of anterior tibial translation (marker A) measured by a linear potentiometer (channel 13) and the onset of reactive muscle activity (marker B) in the medial hamstring (channel 3) after 35 ms.

Fig 4. The subject was in a prone position for hamstring muscle stiffness testing. Electromyographic activity was synchronized with an accelerometer attached to the weighted boot.

significantly greater peak torque and torque at 0.2 seconds during isokinetic knee flexion (Table 4). Flexibility results revealed that the degrees from active knee extension for the ACL-injured group (mean, 19.52° ± 10.74°) were significantly less than the control group (mean, 13.28° ± 7.59°). The single leg hop functional test did not reveal significant group differences; however, the ACL-injured group scored significantly lower on the Lysholm knee rating scale (Table 5).

Significant positive correlations were revealed between hamstring flexibility and functional performance (hop/height distance) ($r = 0.65$; $p = 0.00$) and between physical activity level (Tegner score) and functional performance ($r = 0.42$; $p = 0.03$). A stepwise multiple regression analysis revealed that 42% of the hop per height distance can be explained by hamstring flexibility ($r^2 = 0.42$; $F = 19.75$; Significant $F = 0.00$), but physical activity level did not significantly increase the predictability of functional performance in the hop test (Significant $T = 0.09$).

DISCUSSION

Preparatory Muscle Activity before Landing

Contemporary theories regarding dynamic joint stabilization emphasize the significance of preactivated muscle

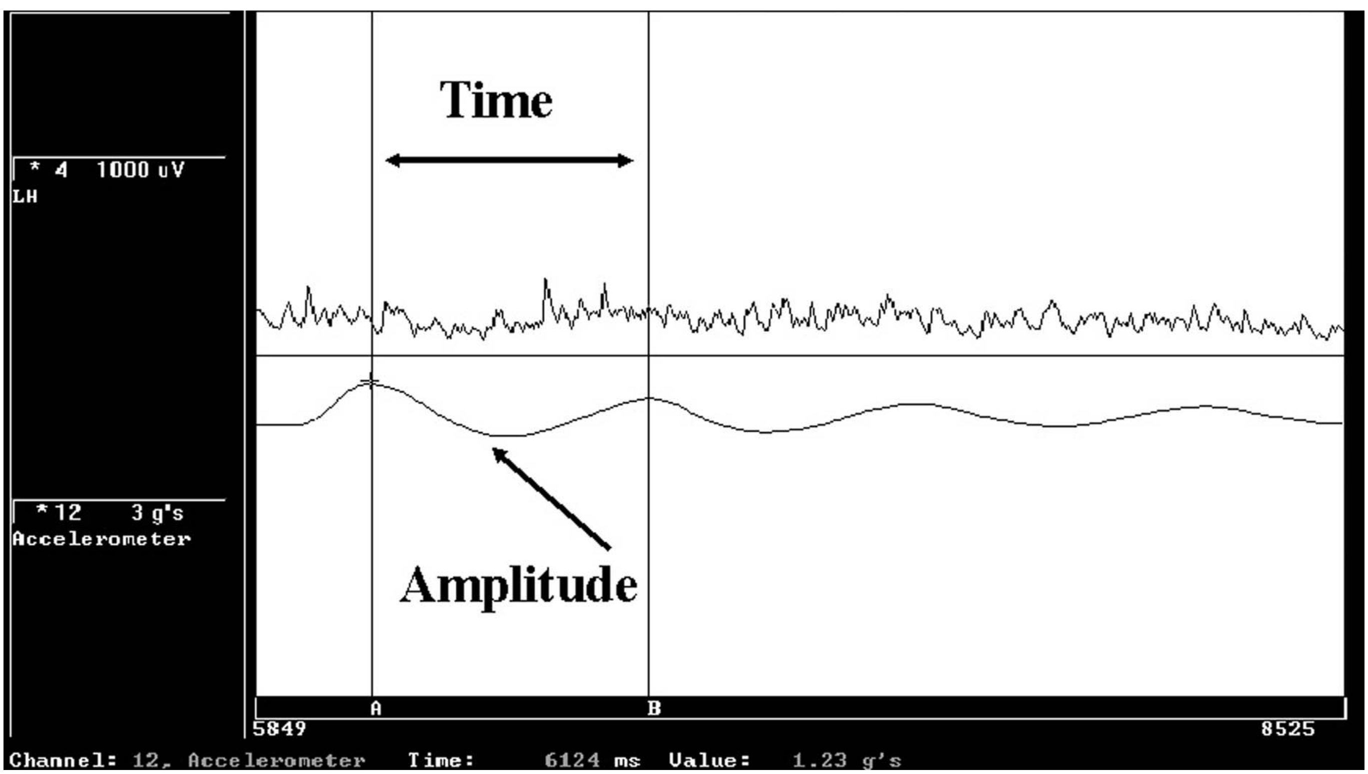

Fig 5. A typical recording included hamstring muscle activity and accelerometer signal synchronized during stiffness testing. The time and amplitude of the first (marker A) and second (marker B) sinusoid peaks was used to calculate hamstring muscle stiffness.

Fig 6. The subject was positioned supine for the hamstring flexibility active knee extension test.

patterns in anticipation of joint loads, but the results are mixed.[7,11,13,16,18,21,37,51] Gauffin and Tropp[16] and McNair and Marshall[37] used a landing task similar to the one used in the current study and did not identify significant side-to-side or between-group differences in subjects with ACL injuries. However, Branch et al[5] observed significant increases (38%) in the area of lateral hamstring activity when subjects with an ACL injury did a cutting maneuver. The current study also identified significant increases in preparatory lateral hamstring muscle activity in subjects with ACL injuries. Branch et al[5] concluded that because episodes of functional instability frequently involve anterior tibial translation and internal rotation, increased activation of the lateral hamstring muscle would provide the most protection for maintaining joint stability. Although there are contradictory reports involving preparatory muscle activation, the evidence supporting preprogrammed, compensatory muscle activation strategies in subjects with ACL injuries is growing.[5,11,18,27]

The increased preparatory hamstring activity may best be explained by the feed-forward process of motor control.[31] Feed-forward processing emphasizes preactivated muscle patterns in anticipation of movements and joint loads.[11,13,18] After ACL injury, sensory feedback may be used to build a new internal model depicting the expected conditions during functional activities.[28] Using this advance information about a task permits subjects with ACL injuries to preprogram muscle activation patterns and compensate for mechanical instability through dynamic restraint. With feed-forward processing, knee stability can be maintained during high speed, functional movements.

Reactive Muscle Activity

Debate exists regarding the capacity of reactive muscle contractions to help with functional joint stability. Timing (latency) and amount of reactive muscular contractions have the potential to assist with dynamic restraint in subjects with ACL injuries.[2,24,45,58]

Previous studies suggest that subjects with ACL injuries may use the feedback process of motor control to minimize anterior tibial translation. This reactive strategy includes increasing hamstring activity and decreasing quadriceps activity.[3,5,6,8,12,16,31,40] However, the results of the current did not reveal significant differences and a quad avoidance strategy was not observed. It is possible that increased preparatory muscle activity in the ACL-injured group eliminated the need for modifications to the reactive muscular contractions. Pretensioning the hamstring muscle with a preparatory contraction may restrict excessive tibial translation and therefore reactive muscle activity remained unchanged.

The timing of reactive muscle contractions is another critical factor if the feedback motor control process is expected to contribute to dynamic restraint.[2,24,45,58] It is suggested that if reactive dynamic stabilization is to be effective at protecting joint structures, a very fast motor response (30–70 ms) is necessary.[2,24,58] Beard and Refshauge[2] reported a significantly longer delay in the reactive hamstring contractions of limbs with ACL injuries (99 ms) than uninvolved limbs (53 ms) and a control group (43

TABLE 1. Area of Preparatory Muscle Activity before Landing

Muscle	Anterior Cruciate-Injured Group Mean (SD)	Control Group Mean (SD)	t Value	p Value
Vastus medialis	4.24 (2.09)	3.68 (1.34)	–0.88	.19
Vastus lateralis	2.55 (1.09)	2.66 (1.15)	0.26	.40
Medial hamstring	4.19 (2.92)	4.86 (2.46)	0.65	.26
Lateral hamstring	7.54 (3.64)	5.54 (2.66)	–1.71	.05
Medial gastrocnemius	35.13 (20.10)	43.44 (25.59)	0.94	.18
Lateral gastrocnemius	4.63 (2.77)	5.23 (3.28)	0.52	.31

Area of integrated electromyography (% ms); SD = standard deviation

TABLE 2. Area of Reactive Muscle Activity after Landing

Muscle	Anterior Cruciate-Injured Group Mean (SD)	Control Group Mean (SD)	t Value	p Value
Vastus medialis	12.95 (2.28)	13.58 (1.93)	0.81	.21
Vastus lateralis	8.80 (3.58)	9.10 (3.99)	0.21	.42
Medial hamstring	7.52 (5.83)	8.34 (5.30)	0.40	.35
Lateral hamstring	13.91 (7.00)	10.77 (4.43)	−1.48	.08
Medial gastrocnemius	26.57 (16.10)	18.52 (13.13)	−1.48	.08
Lateral gastrocnemius	5.40 (3.02)	7.76 (8.05)	0.96	.17

Area of integrated electromyography (% ms); SD = standard deviation

ms). Wojtys and Huston[58] confirmed these results and observed that subjects with ACL injuries had slightly longer delays depending on the time from injury. However, results of the current study concur with those of Jennings and Seedhom,[24] who were unable to reproduce these deficits, reporting onset times of 41 ms in the uninvolved limb and 32 ms in the ACL-injured limb. There are numerous factors that may have contributed to these discrepancies including different test procedures, instrumentation, and subject samples. Another possible explanation is that joint perturbation tests do not elicit reflexive muscular activation originating from the ACL or other capsuloligamentous structures, but a monosynaptic stretch reflex response from muscle spindles in the medial and lateral hamstrings.[2,24] The peripheral site where sensory feedback originates and the neural pathway followed contribute to the response time of muscles. The onset times recorded in this study coincide with the monosynaptic stretch reflex and seem to be unaffected by ACL injury.[15,25,28]

Hamstring Muscle Stiffness

Muscle stiffness was calculated to determine the resistance of the hamstring muscles to stretch. The results identified group differences but numerous factors are involved with regulation of stiffness including joint position, preparatory and reactive muscle activation strategies, and flexibility.[34,37,45,54,55] The complex interaction between these components mediates muscle stiffness and is incorporated into the neuromuscular control strategy best suited for dynamic restraint and functional performance.[1,37]

In vivo measurements of muscle stiffness originally were done to determine how performance was affected by the mechanical properties of the muscle[1,37,38,56] Bach et al[1] suggested that the neuromuscular control apparatus modifies muscle stiffness, depending on the requirements of a task, to optimize these properties. For example, one-legged hopping is better done in resonance with the natural frequency of muscles elastic properties.[1,39] This strategy uses the mechanical properties of muscle to conserve energy and increase the efficiency of movement.[1,39,56] Therefore, individual muscle stiffness seems to be an important determinant in the selection and performance of functional movement strategies. Wilson et al[54] and Rudolph et al[46] suggested that lower stiffness might be advantageous during functional activities because stored elastic energy is used to absorb loads more efficiently. Subjects with ACL injuries in the current study may use lower muscle stiffness to absorb ground reaction forces in tenomuscular rather than capsuloligamentous structures. McNair et al,[38] however, identified a positive correlation between increased hamstring muscle stiffness and increased functional performance in subjects with ACL injuries. A second landing study done by McNair and Marshall[37] revealed that subjects with ACL injuries with greater hamstring muscle activity had lower ground reaction forces and lower hamstring muscle stiffness. Subjects with ACL injuries in the current study also had greater

TABLE 3. Onset Time of Reactive Muscle Activity

Muscle	Anterior Cruciate Injured Group Mean (SD)	Control Group Mean (SD)	t Value	p Value
Medial hamstring (ms)	43.94 (13.90)	38.41 (16.56)	−0.95	.18
Lateral hamstring (ms)	42.54 (14.38)	39.80 (17.70)	−0.44	.33

SD = standard deviation

TABLE 4. Isokinetic Force Production of 60°/second

Strength Measure	Anterior Cruciate-Injured Group Mean (SD)	Control Group Mean (SD)	t Value	p Value
Peak torque				
Flexion (Nm)	87.71 (15.77)	75.23 (12.78)	−2.30	.01
Extension (Nm)	135.53 (21.60)	133.55 (28.43)	−0.20	.42
Torque at .2 seconds				
Flexion (ms)	65.58 (13.60)	52.09 (14.84)	−2.43	.01
Extension (ms)	103.76 (17.36)	104.71 (27.37)	0.10	.46

SD = standard deviation

preparatory hamstring muscle activity, less hamstring muscle stiffness, and were relatively functional when compared with subjects in the control group. This supports research by Rudolph et al[46] and suggests there is an important relationship or interaction among muscle activation timing, stiffness, and function in subjects with ACL injuries. However, additional research with subjects with ACL injuries is needed because it is unclear whether low muscle stiffness is a congenital, predisposing factor to injury or a compensatory adaptation benefiting the dynamic restraint mechanism.

Muscle Force Production

Previous isokinetic assessments have revealed peak torque deficits in the quadriceps and hamstring muscles of subjects with ACL injuries.[14,23,29,33,35,43,47] However, only quadriceps peak torque values are consistently less in the ACL-injured limb, but this was not observed in the current study.[29,33,47] Wojtys and Huston[58] assessed peak torque and body weight ratio in a group of subjects with ACL injuries who were considered to be functioning well, and found normal or increased hamstring muscle strength.[58] This data are consistent with the current results. No previous literature has been identified confirming the improved torque at 0.2 seconds for knee flexion of the ACL-injured group. These results support rapid force production as a characteristic beneficial to dynamic restraint in subjects with ACL injuries. The capacity of the hamstring muscles to generate greater torque in a shorter time would create a posterior force vector resisting excessive anterior translation of the tibia.[33,43,51,58] This quality may be inherent or the result of muscle hypertrophy and recruitment strategies developed during rehabilitation.[52] Subjects with ACL injuries who possess these characteristics may be able to preserve functional stability through dynamic mechanisms, and therefore elect not to have surgical reconstruction.

Flexibility

The premature development of tension from inflexible hamstrings may be beneficial for restricting excessive anterior tibial translation and provide dynamic restraint in the ACL-injured knee. Results of the current study agree with the results of Harner et al[20] who assessed hamstring flexibility and found that females with ACL injuries had significantly less flexible hamstring muscles when compared with a control group. The mechanism responsible for hamstring inflexibility involves excitatory and inhibitory protective reflexes initiated by muscle spindles and golgi tendon organs. Protective reflexes originating from these mechanoreceptors are the primary determinants of maximum muscle length.[19] Neuromuscular adaptations to these reflexive pathways can decrease hamstring muscle flexibility and result in the premature development of ham-

TABLE 5. Functional Performance and Functional Impairment

Test of Function	Anterior Cruciate-Injured Group Mean (SD)	Control Group Mean (SD)	t Value	p Value
Functional performance				
Hop/height (%)	68.58 (12.52)	74.27 (8.30)	1.47	.07
Functional impairment				
Lysholm scale	78.17 (16.38)	95.59 (6.05)	4.04	.00

Hop/height = single leg hop distance divided by body height; SD = standard deviation; Lysholm scale ranges from 0–100 string muscle tension as the knee is extended. This mechanism could resist excessive anterior translation, increase dynamic restraint, and assist with functional stability in the ACL-injured knee.[20]

Functional Performance and Functional Impairment

Performance measures such as the hop for distance are designed to objectively assess functional status with a standardized test. Functional performance for the control group (74.27% ± 8.30%) was similar to previously established normative data (70.48% ± 10.88%) for healthy females and the subjects with ACL injuries were not significantly different (68.58% ± 12.52%) scoring in the normal limits of healthy females.[26] Juris et al[26] also tested subjects with ACL reconstructions who had almost identical scores (67.52% ± 14.22%) as the ACL-injured group in the current study. These results are an indication that the ACL-injured group was able to maintain a normal level of functional performance despite the mechanical instability. The neuromuscular characteristics assessed in the current study may be responsible for enhancing dynamic restraint capabilities, thereby maintaining functional stability and performance in the ACL-injured group.

Functional impairment was assessed with the Lysholm knee rating scale and confirmed that the control group (score, 95) did not experience functional impairment because of a knee injury.[36] The ACL-injured group scored significantly lower (score, 78), which was considered fair.[36] Although the ACL-injured group experienced more symptoms of knee injury, these symptoms did not seem to effect activity level or functional performance.

Relationships Between the Neuromuscular Characteristics and Function

Significant positive correlations were revealed between hamstring flexibility and functional performance, and between activity level and functional performance. The relationship between hamstring flexibility and functional performance may result from the use of stored elastic energy during the single leg hop test.[56] All subjects were observed doing a small counter movement before executing the hop test. This movement can enhance performance by pretensioning the elastic component of the hamstring muscle and eliciting a stretch reflex response.[56] Subjects with more flexible hamstring muscles may be capable of storing and using the elastic energy more effectively as evidenced by increased hop distance.[1,55,56] The correlation between activity level and functional performance may imply that the physically active subjects were familiar with the specific demands of the task and the most successful motor control strategies needed to execute the single leg hop test.

The current study explored dynamic restraint mechanisms by assessing the neuromuscular characteristics of females with ACL injuries and healthy female subjects. Females with ACL injuries showed greater preparatory activity in the lateral hamstring muscles, less hamstring stiffness and flexibility, but no differences in reactive muscle activity. The ACL-injured group also had greater peak torque and torque development for knee flexion. Positive correlations were seen among hamstring flexibility, activity level, and functional performance with 42% of the hop and height distance explained by hamstring flexibility.

Increased preparatory activity suggests that females with ACL injuries use feed-forward processing to pretension muscle thereby enhancing dynamic restraint capabilities. Decreased muscle stiffness in the ACL-injured group most likely involved a complex regulatory strategy to maximize energy absorption. The results of strength testing suggest that subjects with ACL injuries possess adaptations to the hamstring muscle group that permit them to produce greater force in a shorter time, therefore increasing dynamic restraint. Our research also suggests that hamstring inflexibility may contribute to dynamic restraint through adaptive shortening and the protective reflexes that mediate hamstring tension. The compensatory neuromuscular characteristics assessed in this study may be responsible for enhancing the dynamic restraint capabilities and assist with functional stability in females with ACL injuries. These characteristics should be explored to reveal their potential for preventing knee injuries through dynamic restraint.

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References

1. Bach TM, Chapman AE, Calvert TW: Mechanical resonance of the human body during voluntary oscillations about the ankle. J Biomech 16:85–90, 1983.
2. Beard DJ, Refshauge K: Effects of ACL Reconstruction on Proprioception and Neuromuscular Performance. In Lephart SM, Fu FH (eds). Proprioception and Neuromuscular Control in Joint Stability. Champaign, IL, Human Kinetics 213-224, 2000.
3. Berchuck M, Andriacchi TP, Bach BR, Reider B: Gait adaptations by patients who have a deficient anterior cruciate ligament. J Bone Joint Surg 72A:871–877, 1990.
4. Bolgla LA, Keskula DR: Reliability of lower extremity functional performance tests. J Orthop Sports Phys Ther 26:138–142, 1997.
5. Branch T, Hunter R, Donath M: Dynamic EMG analysis of the anterior cruciate ligament deficient legs with and without bracing during cutting. Am J Sports Med 17:35–41, 1989.
6. Brener J: Sensory and Perceptual Determinants of Voluntary Visceral Control. In Schwartz GE, Beatty J (eds). Biofeedback Theory and Research. New York, Academic Press 29-66, 1977.

7. Carlsoo S, Nordstrand A: The coordination of the knee-muscles in some voluntary movements and in the gait in cases with and without knee joint injuries. Acta Chir Scand 134:423–426, 1968.
8. Ciccotti M, Kerlain R, Perry J, Pink M: An electromyographic analysis of the knee during functional activities: I. The normal profile. Am J Sports Med 22:645–650, 1994.
9. Ciccotti M, Kerlain R, Perry J, Pink M: An electromyographic analysis of the knee during functional activities: II. The anterior cruciate ligament-deficient knee and reconstructed profiles. Am J Sports Med 22:651–658, 1994.
10. Daniel DM: Reference, Maintenance and Users Guide for the Knee Ligament Arthrometer. San Diego, CA, Medmetric Corp 30-31, 1993.
11. Dietz V, Noth J, Schmidtbleicher D: Interaction between preactivity and stretch reflex in human triceps brachii during landing from forward falls. J Physiol 311:113–125, 1981.
12. Dunn TG, Gillig SE, Ponser SE, Weil N: The learning process in biofeedback: Is it feed-forward or feedback. Biofeedback Self Reg 11:143–155, 1986.
13. Dyhre-Poulsen P, Simonsen B, Voigt M: Dynamic control of muscle stiffness and H reflex modulation during hopping and jumping in man. J Physiol 437:287–304, 1991.
14. Engstrom B, Gornitzka J, Johansson C, Wredmark T: Knee function after anterior cruciate ligament ruptures treated conservatively. Int Orthop 17:208–213, 1993.
15. Freeman MAR, Wyke B: Articular contributions to limb reflexes. Br J Surg 53:61–69, 1966.
16. 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 20:182–192, 1992.
17. Glass GV, Hopkins KD: Regression and Prediction. Statistical Methods in Education and Psychology. Ed 3. Needham Heig, MA. Simon & Schuster Company 385:111, 170-172, 1996.
18. Greenwood R, Hopkins A: Landing from an unexpected fall and a voluntary step. Brain 99:375–386, 1976.
19. Hamill J, Knutzen FM: Muscular Considerations for Movement. Biomechanical Basis of Human Movement. Philadelphia, PA, Williams & Wilkins 84-89, 1995.
20. Harner CD, Paulos LE, Greenwald AE, Rosenberg TD, Cooley VC: Detailed analysis of patients with bilateral anterior crutiate ligament injuries. Am J Sports Med 22:37–43, 1994.
21. Houk JC, Crago PE, Rymer WZ: Function of the Dynamic Response in Stiffness Regulation: A Predictive Mechanism Provided by Non-linear Feedback. In Taylor A, Prochazka A (eds). Muscle Receptors and Movement. London, Macmillan 299-309, 1981.
22. Ireland ML, Gaudette M, Crook S: ACL injuries in the female athlete. J Sports Rehabil 6:97–110, 1997.
23. Itoh H, Ichihashi N, Maruyama T, Kurosaka M, Hirohata K: Weakness of the thigh muscles in individuals sustaining anterior cruciate ligament injury. Kobe J Med Sci 38:93–107, 1992.
24. Jennings AG, Seedhom BB: Proprioception in the knee and reflex hamstring contraction latency. J Bone Joint Surg 76B:491–494, 1994.
25. Johansson H, Sjolander P, Sojka P: A sensory role for the cruciate ligaments. Clin Orthop 268:161–178, 1991.
26. Juris PM, Phillips EM, Dalpe C, et al: A dynamic test of lower extremity function following anterior cruciate ligament reconstruction and rehabilitation. J Orthop Sports Phys Ther 24:184–191, 1997.
27. Kålund S, Sinkjær T, Arendt-Nielsen A, Simonsen D: Altered timing of hamstring muscle action in anterior cruciate ligament deficient patients. Am J Sports Med 18:245–248, 1990.
28. Kandell ER, Schwartz JH, Jessell TM: The Organization of Movement. In Ghez C, Krakauer J (eds). Principles of Neural Science. Ed 4. Norwalk, CT, Appleton & Lange 653-657, 1996.
29. Kannus P, Latvala K, Jaervinen M: Thigh muscles strength in the anterior cruciate ligament deficient knee: Isokinetic and isometric long term results. J Orthop Sports Phys Ther 9:223–227, 1987.
30. Katz JW, Fingeroth R: The diagnostic accuracy of ruptures of the anterior cruciate ligament comparing the Lachman test, the anterior drawer sign, and the pivot shift test in acute and chronic knee injuries. Am J Sports Med 14:88–91, 1986.
31. La Croix JM: The acquisition of autonomic control through biofeedback: The case against an afferent process and a two process alternative. Psychophysiology 18:573–587, 1981.
32. Leggin BG, Neuman RM, Iannotti JP, Williams GR, Thompson EC: Intrarater and interrater reliability of three isometric dynamometers in assessing shoulder strength. J Shoulder Elbow Surg 5:18–24, 1996.
33. Lephart SM, Perrin DH, Fu FH, et al: Relationship between selected physical characteristics and functional capacity in the anterior cruciate-insufficient athlete. J Orthop Sports Phys Ther 16:174–180, 1992.
34. Lieber RL, Friden J: Neuromuscular Stabilization of the Shoulder Girdle. In Matsen FA (ed). The Shoulder: A Balance of Mobility and Stability. Rosemont, IL, American Academy of Orthopaedic Surgeons 91-106, 1992.
35. Lorentzon R, Elmqvist LG, Sjostrom M, Fagerlund M, Fuglmeyer AR: Thigh musculature in relation to chronic anterior cruciate ligament tear: Muscle size, morphology, and mechanical output before reconstruction. Am J Sports Med 17:423–429, 1989.
36. Lysholm J, Gillquist J: Evaluation of knee ligament surgery results with special emphasis on use of scoring scale. Am J Sports Med 10:150–154, 1982.
37. McNair PJ, Marshall RN: Landing characteristics in subjects with normal and anterior cruciate ligament deficient knee joints. Arch Phys Med Rehabil 75:584–589, 1994.
38. McNair PJ, Wood GA, Marshall RN: Stiffness of the hamstring muscles and its relationship to function in anterior cruciate deficient individuals. Clin Biomech 7:131–173, 1992.
39. Melvill-Jones GM, Watt GD: Observations of the control of stepping and hopping in man. J Physiol 219:709–727, 1971.
40. Merton PA: Speculations on the Servo-Control of Movement. In Wolstenholme GEW (ed). The Spinal Cord, London, Churchill 247-255, 1953.
41. Moffroid MM, Tranowski J, Ricamato A, Henry S: Computer solutions to identify EMG latency of automatic postural reactions. IEEE Eng Med Biol 11:48–51, 1992.
42. Morgan DL: Separation of active and passive components of short-range stiffness of muscle. Am J Physiol 32:45–49, 1977.
43. Noyes FR, Barber SD, Mangine RE: Abnormal lower limb symmetry determined by function hop tests after anterior cruciate ligament rupture. Am J Sports Med 19:513–518, 1991.
44. Perrin DH: Isokinetic Instrumentation. Isokinetic Exercise and Assessment. Champaign, IL, Human Kinetics 25-33, 1993.
45. Raunest J, Sager M, Burgener E: Proprioceptive mechanisms in the cruciate ligaments: An electromyographic study on reflex activity in the thigh muscles. J Trauma Inj Inf Crit Care 41:488–493, 1993.
46. Rudolph KS, Axe MJ, Buchanan TS, et al: Dynamic stability in the anterior cruciate ligament deficient knee. Knee Surg Sports Traumatol Arthrosc 9:62–71, 2001.
47. Shirakura K, Karo K, Udagawa E: Characteristics of the isokinetic performance of patients with injured cruciate ligaments. Am J Sports Med 20:754–758, 1992.
48. 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 15:207–213, 1987.
49. Tegner Y: Lysholm: Rating systems in the evaluation of knee ligament injuries. Clin Orthop 198:43–49, 1985.
50. Tegner Y, Lysholm J, Odensten M, Gillquist J: Evaluation of cruciate ligament injuries: A review. Acta Orthop Scand 59:336–341, 1988.
51. Tibone JE, Antich TJ, Fanton GS, Moynes DR, Perry J: Functional analysis of anterior cruciate ligament instability. Am J Sports Med 14:276–284, 1986.
52. Walla DJ, Albright JP, McAuley E, et al: Hamstring control and the unstable anterior cruciate ligament-deficient knee. Am J Sports Med 13:34–39, 1985.
53. Walshe AD, Wilson GJ, Murphy AJ: The validity and reliability of a test of lower body musculotendinous stiffness. Eur J Appl Physiol 73:332–339, 1996.

54. Wilson GJ, Murphy AJ, Pryor JF: Musculotendinous stiffness: Its relationship to eccentric, isometric, and concentric performance. J Appl Physiol 76:2714–2727, 1994.
55. Wilson GJ, Wood GA, Elliott BC: The relationship between stiffness of the musculature and static flexibility: An alternative explanation for the occurrence of muscular injury. Int J Sports Med 12:403–407, 1991.
56. Wilson GJ, Wood GA, Elliott BC: Optimal stiffness of series elastic component in a stretch-shorten cycle. J Appl Physiol 70:825–833, 1991.
57. Winter DA: Synthesis of Human Movement-Forward Solutions. Biomechanics and Motor Control of Human Movement. Ed 2. New York, J Wiley Sons Inc 150–159, 1990.
58. Wojtys E, Huston L: Neuromuscular performance in normal and anterior cruciate ligament-deficient lower extremities. Am J Sports Med 22:89–104, 1994.
59. 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 67-80, 1994.
60. Worrell TW, Sullivan MK, DeJulia JJ: Reliability of an active-knee-extension test for determining hamstring muscle flexibility. J Sport Rehabil 1:181–187, 1992.
61. Yang JF, Winter DA: Electromyographic amplitude normalization methods: Improving their sensitivity as diagnostic tools in gait analysis. Arch Phys Med Rehabil 65:517–521, 1984.