Hamstring Activity in the ACL Injured Patient: Injury Implications and Comparison With Quadriceps Activity

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Purpose: To investigate the potential causes of diminished knee extension after acute anterior cruciate ligament (ACL) injury using both surface electromyography (sEMG) analysis of the quadriceps and hamstrings, and gait analysis to assess muscle action and tone. **Methods:** Consecutive patients with an acute ACL tear underwent sEMG and gait analysis within 2 weeks of injury, before ACL reconstruction. Standard motion analysis techniques were used and sEMG data were collected simultaneously with gait data. *T*-tests were used to determine differences between the ACL-deficient and control subjects in knee flexion angles, peak external knee joint moments, and total time that a muscle was activated ("on") during gait. External knee moments were expressed as a percentage of body weight times height. **Results:** Ten patients (mean age 24 ± 4 years) were included at a mean 10.2 days between injury and analysis; 10 uninjured, matched control subjects were included for comparison. There were significant increases in minimum flexion angle at heel strike (5.92 ± 3.39 *v* −3.49 ± 4.55, $P < .001$) and midstance (14.1 ± 6.23 *v* 1.20 ± 4.21, $P < .001$) in the injured limb compared with controls. There were significantly lower maximum external extension moments at heel strike (−0.99 ± 0.46 *v* −2.94 ± 0.60, $P < .001$) and during the second half of stance in the injured limb compared with controls (−0.56 ± 1.14 *v* −3.54 ± 1.31, $P < .001$). The rectus femoris was "on" significantly less during gait in the injured leg compared with controls (49.1 ± 7.76% *v* 61.0 ± 14.8%, $P = .044$). There were no significant differences in hamstring activity "on" time during gait ($P > .05$). **Conclusions:** In patients with acute ACL injury, the ACL-deficient limb does not reach as much extension as controls. Although the rectus femoris is "on" for shorter periods during the gait cycle, there is no difference in hamstring time on during gait. This information may help clinicians better understand muscle function and gait patterns in the acute time period after ACL injury. **Level of Evidence:** Level III, case control study.

Injury to the anterior cruciate ligament (ACL) remains one of the most common knee injuries in the United States, with more than 200,000 ACL reconstructions (ACLRs) performed annually.[1] It is well documented that patients with acute ACL injuries often present with a diminished range of motion (ROM), especially in terminal extension. Regaining terminal extension has historically proven to be paramount to successful outcomes in ACLR with respect to postoperative ROM and function.[2,3] With the loss of the primary restraint to anterior tibial translation as a result of an acute ACL injury, the tibia slides anterior until a secondary soft tissue restraint (collateral ligaments, menisci) provides resistance. To compensate, patients may reflexively inhibit their quadriceps activity to avoid unopposed anterior tibial translation. Reflexive quadriceps inhibition after joint distension, which is almost always present after ACL injury, is thus thought to be one of the main factors responsible for the lack of knee extension incurred after acute ACL injury.[4] Although the lack of terminal knee extension after acute ACL injury has long been thought to be due exclusively to this reflexive inhibition of the quadriceps, it is possible that hamstring spasm may also play a role, because hamstring activation provides an additional restraint to anterior tibial translation.[5-7]

Intraoperative stimulation of the ACL in patients undergoing knee arthroscopy has proven to elicit hamstring reflexes.[8] The current literature, however, is

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limited regarding the possible contribution of this hamstring reflex and/or spasticity to the loss of knee extension after acute ACL injury.[9] Although there are several studies that describe muscle activity of the quadriceps and hamstrings, including their impact on gait, after ACLR, to our knowledge, there is minimal information on the activity of muscle groups in patients with acute ACL injury.

The clinical implications of determining the true cause of the loss of extension in these patients may be relevant. If it is determined that knee extension loss is due to hamstring spasm, direct attention could be focused on hamstring relaxation potentially resulting in earlier recovery of extension and decreased risk of flexion contracture. In turn, this could result in safer and earlier reconstruction of the ACL as well as a quicker return to play and/or work with a reduced loss of time and/or wages. Alternatively, in addition to muscle activity, stiffness (contracture) of the periarticular soft tissues after acute injury can be a factor contributing to the loss of extension, and if this is the case, attention to regaining ROM by focusing rehabilitation modalities on the soft tissues would be helpful.

Therefore, the purpose of this study was to investigate the potential muscle-related causes of diminished knee extension after acute ACL injury using both surface electromyography (sEMG) analysis of the quadriceps and hamstrings, and gait analysis to assess muscle action and tone. The authors hypothesized that the hamstrings would be activated for a longer amount of time in the acutely ACL-deficient limb, accounting for loss of extension during gait. In addition, the authors hypothesized that the ACL-deficient limb would not reach as much extension as the control limb, and that the sagittal plane flexion moment would be greatly reduced in the ACL-deficient limb.

Methods

This study received approval by our university's Institutional Review Board. All patients with an acute ACL injury between ages 18 and 60 years were eligible to participate. Patients were prospectively identified on their presentation to the office by 1 of 4 senior sports medicine surgeons (B.F., B.R.B., N.N.V., B.J.C.) and were invited to participate in sEMG and gait analysis before ACLR. The following inclusion criteria were used: patients between ages 18 and 60 years with physical examination and magnetic resonance imaging documentation of an acute, full thickness ACL tear who presented to the office within 2 weeks of their injury. Exclusion criteria included patients with previous ipsilateral knee ligamentous surgery; evidence of concomitant posterior cruciate ligament, medial or lateral collateral ligament pathology; displaced meniscal tear; evidence of patellofemoral pathology; active ipsilateral hip, contralateral hip, or contralateral knee injury or impairment; and presentation more than 2 weeks after injury. All included patients underwent a comprehensive preoperative physical examination by a senior sports-medicine fellowship trained surgeon, including gait assessment, passive ROM assessment, and stability testing of collateral and cruciate ligaments including Lachman and attempted pivot shift testing. All patients then underwent sEMG and gait analysis within 2 weeks of injury, before ACLR. A group of nonsymptomatic matched control subjects with previously collected gait and sEMG data were chosen from an Institutional Review Board approved data repository. Subjects were chosen to match the age, height, and weight of the acute ACL patients as closely as possible.

Motion Analysis Testing

All testing was carried out in the Motion Analysis Laboratory at our institution. Standard motion analysis techniques were used to obtain knee joint flexion angle and external knee joint moments during walking, using a 12-camera optoelectronic system (Qualysis, Gothenburg, Sweden) at 120 Hz. Gait assessment was performed using a 24-marker modified Helen Hayes marker set. The model combined the Helen Hayes[10] model with our existing 6-marker link model (placed over bilateral bony prominences including the greater trochanter, lateral malleolus, lateral tibial plateau, iliac crest, lateral aspect of the calcaneus, and base of the fifth metatarsal).[11] Three to 5 normal gait trials were obtained for each subject at a self-selected walking speed on a level walkway 10 m in length, equipped with a multicomponent force plate (Bertec, Columbus, OH) embedded in the walkway to capture ground reaction forces. The ground reaction force and motion data were used to calculate lower extremity kinematic and kinetic parameters within The Motion Monitor software (Innovative Sports Training, Chicago, IL). Variables included knee flexion angle and external knee joint moments. External knee moments were expressed as a percentage of body weight times height (%BW $\times$ Ht).

sEMG data were collected simultaneously with the gait analysis data at 1200 Hz during each trial using a TeleMyo transmitter and receiver (model 2400T/2400R: Noraxon, Scottsdale, AZ) from 4 muscle groups: bicep femoris, vastus medialis, semimembranosus (SM)/semitendinosus (ST), and rectus femoris. A self-adhesive dual Ag/AgCl electrode (Noraxon) was placed on the palpated belly of each muscle group in parallel with the muscle fibers at the mid-portion of the muscle. To reduce interelectrode impedance, resistance caused by dead skin cells, skin oil, and moisture, the skin was cleaned using antimicrobial wipes before application. The raw sEMG voltages were filtered (FIR bandpass 20 to 450 Hz), rectified, and smoothed (RMS,

Table 1. Patient and Control Subject Characteristics

	ACL Patients	Control Subjects	*P* (*t*-test)
Age	32.2 ± 11.2 yr (range, 18-47 yr)	34 ± 10.9 yr (range, 21-47 yr)	.720
Sex	6 males, 4 females	6 males, 4 females	–
Height	5.70 ± 0.30 ft (range, 5.17-6.04 ft)	5.61 ± 0.25 m (range, 5.19-5.88 m)	.468
Weight	173 ± 47.5 lb (range, 106-261 lb)	163 ± 21.8 lb (range, 122-187 lb)	.534
Body mass index	25.7 ± 5.13 kg/m^2 (range, 19.3-36.6 kg/m^2)	25.2 ± 2.57 kg/m^2 (range, 20.6-29.7 kg/m^2)	.803
Laterality	8 right, 2 left	n/a	–
Mechanism of injury	• Skiing (n = 3) • Snowboarding (n = 1) • Athletic contact injury (1 basketball, 1 football), • Athletic noncontact injury (1 football, 1 lacrosse, 1 beach volleyball) • Noncontact twisting injury while walking (n = 1)	n/a	–
Preoperative range of motion	Extension: average 2.5° ± 2.6° (range, −1° to 8° short of full extension) Flexion: average 110.5° ± 20.7° (range, 80° to 130°)	n/a	–
Preoperative stability testing	2B Lachman: n = 8 (80%) Unable to assess: n = 2 (due to guarding)	n/a	–
Graft choice	5 patellar tendon autograft 5 patellar tendon allograft	n/a	–

ACL, anterior cruciate ligament; n/a, not applicable.

50 ms window). The percentage of time that a muscle was activated during gait was determined from the processed voltage signals using a method developed for determining the burst activity of rhythmic EMG signals.[12] First, a threshold for on-off activity was calculated from histograms of the processed voltage amplitudes. The voltage amplitude that had a frequency of occurrence 3 standard deviations above the voltage amplitude with the highest frequency of occurrence was defined as the threshold. When the voltage amplitude was greater than the threshold, the muscle was deemed "on," and when the voltage amplitude was less than the threshold, the muscle was considered "off."

Statistical Analysis

The data from the injured leg were compared with the control subjects' leg data. Independent *t*-tests were used to determine differences between the ACL-deficient and control subjects' limb in knee flexion angles, peak external knee joint moments, total time that a muscle was activated ("on") during the gait cycle, and muscle on/off timing. A muscle group, "any quadriceps and any hamstring," was included in the analysis of total time "on" during gait as an indicator of coactivation of the quadriceps and hamstring muscles. Linear regression modeling was used to determine the effect of muscle group total time "on" during the gait cycle on knee flexion angle. After determining which knee flexion angle key points were different between the ACL-deficient and control subjects, linear regression models were created for each subject group. All muscle groups were entered into the regression models as dependent variables and a backward approach was used to eliminate muscle groups that did not contribute to variance in the knee flexion angle (independent variable). Statistical analyses were conducted using SPSS (SPSS, Chicago, IL) with significance determined for *P* values less than .05.

Results

A total of 10 patients (6 males, 4 females) with a mean age of 32.5 ± 11.2 years were included at a mean 10.2 days (range, 4 to 14 days) between injury and motion analysis. The mechanism of injury included skiing (n = 3), snowboarding (n = 1), athletic contact injury (1 basketball, 1 football), athletic noncontact injury (1 football, 1 lacrosse, 1 beach volleyball), and noncontact twisting injury while walking (n = 1). ROM for the 10 patients included extension ranging from −1° to 8° and flexion ranging from 80° to 130° (Table 1). Eight patients (80%) presented with a positive Lachman test (2B), with 2 patients deferring examination due to pain. All 10 patients (100%) presented with magnetic resonance imaging evidence of a complete ACL tear. All patients underwent arthroscopic-assisted ACLR using patellar tendon autograft (n = 5) or allograft (n = 5). Five patients underwent a combined 7 concomitant procedures, including partial medial or lateral meniscectomy (n = 4) and patellar chondroplasty (n = 1), medial meniscal repair (n = 1), and lateral meniscal repair (n = 1). Ten control subjects were chosen for comparison with the acute ACL patients. The 10 patients (6 males, 4 females) did not have

Table 2. Gait Analysis Results

	ACL Patients	Control Subjects	*P* (*t*-test)
Flexion angle, °			
Heel strike flexion angle	5.92 ± 3.39	−3.49 ± 4.55	<.001*
Mid stance maximum flexion angle	19.8 ± 5.12	17.0 ± 5.54	.245
% Gait cycle	17.09 ± 5.83	14.0 ± 1.29	.128
Mid stance minimum flexion angle	14.1 ± 6.23	1.20 ± 4.21	<.001*
% Gait cycle	37.5 ± 6.40	39.4 ± 4.43	.442
Toe off flexion angle	38.2 ± 6.42	38.1 ± 4.68	.976
% Gait cycle	63.2 ± 2.79	60.6 ± 2.35	.037*
Maximum flexion angle during swing	53.0 ± 7.47	62.4 ± 3.37	.002*
% Gait cycle	72.3 ± 3.61	71.5 ± 2.50	.548
Dynamic range of motion	50.2 ± 10.1	69.3 ± 5.80	<.001*
Sagittal plane external moment (Percentage of Body Weight × Height)			
First maximum extension moment	−0.99 ± 0.46	−2.94 ± 0.60	<.001*
% Gait cycle	3.03 ± 1.24	1.36 ± 0.60	.001*
Maximum flexion moment	2.65 ± 1.42	2.49 ± 1.26	.795
% Gait cycle	16.9 ± 4.93	13.3 ± 0.94	.044*
Second maximum extension moment	−0.56 ± 1.14	−3.54 ± 1.31	<.001*
% Gait cycle	45.6 ± 5.42	43.8 ± 2.64	.356
Frontal plane external moment (Percentage of Body Weight × Height)			
Maximum abduction moment	0.17 ± 0.34	1.05 ± 0.30	<.001*
% Gait cycle	5.93 ± 2.54	3.90 ± 1.22	.036*
First maximum adduction moment	−1.52 ± 0.74	−2.75 ± 0.95	.005*
% Gait cycle	21.1 ± 4.80	14.1 ± 2.48	.001*
Second maximum adduction moment	−1.75 ± 0.74	−1.75 ± 0.53	.996
% Gait cycle	48.6 ± 2.34	40.3 ± 11.7	.053
Transverse plane external moment (Percentage of Body Weight × Height)			
Maximum external rotation moment	0.39 ± 0.24	0.17 ± 0.10	.019*
% Gait cycle	14.7 ± 5.38	6.53 ± 4.58	.002*
Maximum internal rotation moment	−0.71 ± 0.35	−0.98 ± 0.26	.063
% Gait cycle	49.5 ± 2.89	44.0 ± 2.36	<.001*

ACL, anterior cruciate ligament.
*Statistically significant.

significantly different age, height, weight, or body mass index compared with the acute ACL patients (Table 1).

Motion analysis data revealed a significantly reduced dynamic ROM during level walking in the injured knee (average 50.2° ± 10.1°) compared with the control subject (69.3° ± 5.80°, $P < .001$) during gait (Table 2).

There was a significant increase in minimum flexion angle at heel strike, increase in minimum flexion angle at midstance, and decrease in maximum flexion angle during swing in the injured leg compared with the control leg, respectively (Fig 1A, Table 2). In the sagittal plane, there was a significantly lower maximum external extension moment at heel strike in the injured leg compared with the control leg, and significantly lower maximum external extension moment during midstance (Fig 1B, Table 2). In the coronal plane, there was a significantly lower maximum external abduction moment in the injured leg compared with the control leg and a significantly lower maximum external adduction moment (Fig 1C, Table 2) during the first half of stance. In the transverse plane, there was a significantly higher maximum external-rotation (ER) moment (Fig 1D, Table 2) in the injured leg compared with the control leg.

The rectus femoris was "on" significantly less during gait in the injured leg compared with the control leg (Fig 2, Table 3). There were no significant differences in hamstring activity "on" time during gait ($P > .05$). The lower limb muscles from the injured leg activated later in the gait cycle than the control leg for the vastus medialis and the biceps femoris, and the injured leg deactivated later during the gait cycle than the control leg for the vastus medialis (Fig 3, Table 4).

Discussion

The principal findings of this study are as follows: (1) the ACL-deficient limb did not reach as much extension at heel strike and midstance, did not reach as much flexion during swing, and had a reduced dynamic ROM during gait compared with the control; (2) in the ACL-deficient limb, the extension moments during heel strike terminal stance were reduced compared with the control, indicating a reduced net activity of the hamstrings in the ACL-deficient limb; (3) the vastus medialis and biceps femoris activated later in the gait cycle in the ACL-deficient versus the control; and (4) the rectus femoris was "on" for shorter periods during the gait cycle in the injured limb. Our primary hypothesis was

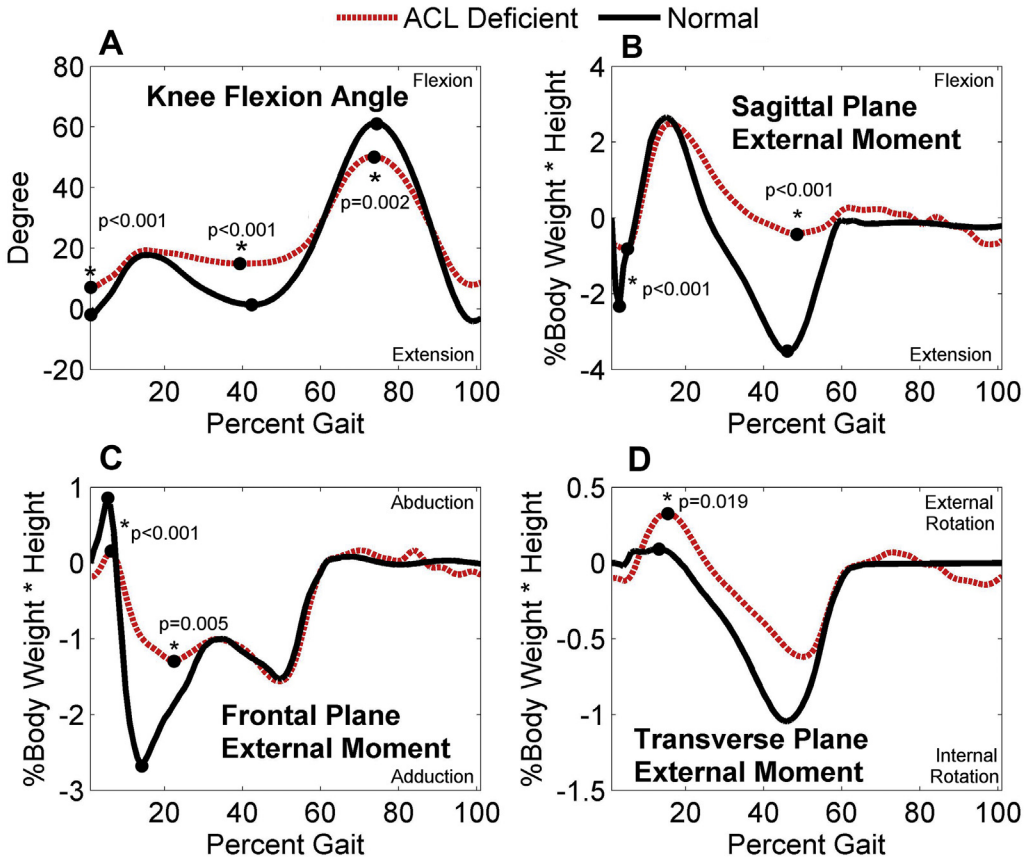

Fig 1. Kinematic and kinetic outcomes showing (A) a significant increase in minimum flexion angle at both heel strike (5.92° *v* −3.49°, $P < .001$) and midstance (14.1° *v* 1.20°, $P < .001$), and a significant decrease in maximum flexion angle during swing (53.0° *v* 62.4°, $P = .002$) in the injured leg compared with the control leg, respectively; (B) a significantly lower maximum external extension moment at heel strike in the injured leg compared with the control leg (−0.99 *v* −2.94 percentage of body weight times height [%BW × Ht], $P < .001$), and lower maximum external extension moment during midstance (−0.56 *v* −3.54 % BW × Ht, $P < .001$); (C) a significantly lower maximum external abduction (0.17 *v* 1.05 %BW × Ht, $P < .001$) and adduction moment (−1.52 *v* −2.75%BW × Ht, $P = .005$) during the first half of stance in the injured leg compared with the control leg; and (D) a significantly higher maximum external external-rotation moment in the injured leg compared with the control leg (−0.39 *v* 0.17 %BW × Ht, $P = .019$). (ACL, anterior cruciate ligament.)

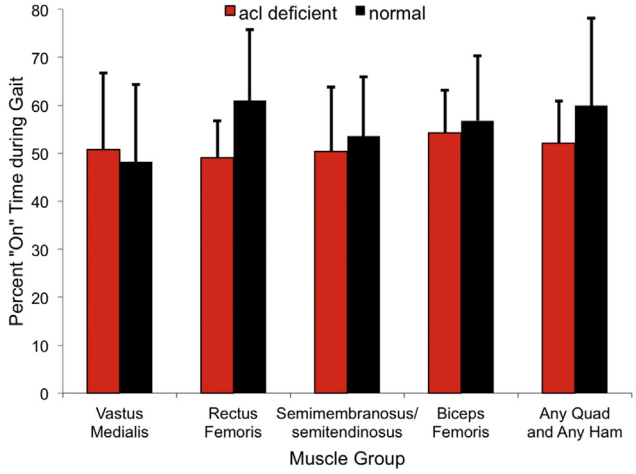

Fig 2. Bar graph showing the total percentage of time that a muscle was "on" during gait. In the injured leg, the rectus femoris was on significantly longer during gait in the control leg (54.8% *v* 49.1%, $P = .044$). (ACL, anterior cruciate ligament; Ham, hamstring; Quad, quadriceps.)

rejected, as the hamstrings did not show hyperactivity based on sEMG data in this study. Clinically, these data may help patients undergo more efficient and focused "prehabilitation" programs within the first 2 weeks after acute ACL injury, which may allow for earlier ACLR and ultimately, earlier return to sport and/or work. Specifically, these findings support "prehabilitation"

Table 3. Surface Electromyography Results: Total Percentage "on" During Gait

	ACL Patients	Control Subjects	*P* (*t*-test)
Biceps femoris	54.3 ± 8.91	56.8 ± 13.5	.642
Semimembranosus/ semitendinosus	50.4 ± 13.4	53.5 ± 12.5	.639
Rectus femoris	49.1 ± 7.76	61.0 ± 14.8	.044*
Vastus medialis	50.7 ± 16.0	48.2 ± 16.2	.745
Any quad. and any ham.	52.0 ± 8.87	59.9 ± 18.2	.257

ACL, anterior cruciate ligament; ham., hamstring; quad., quadriceps.
*Statistically significant.

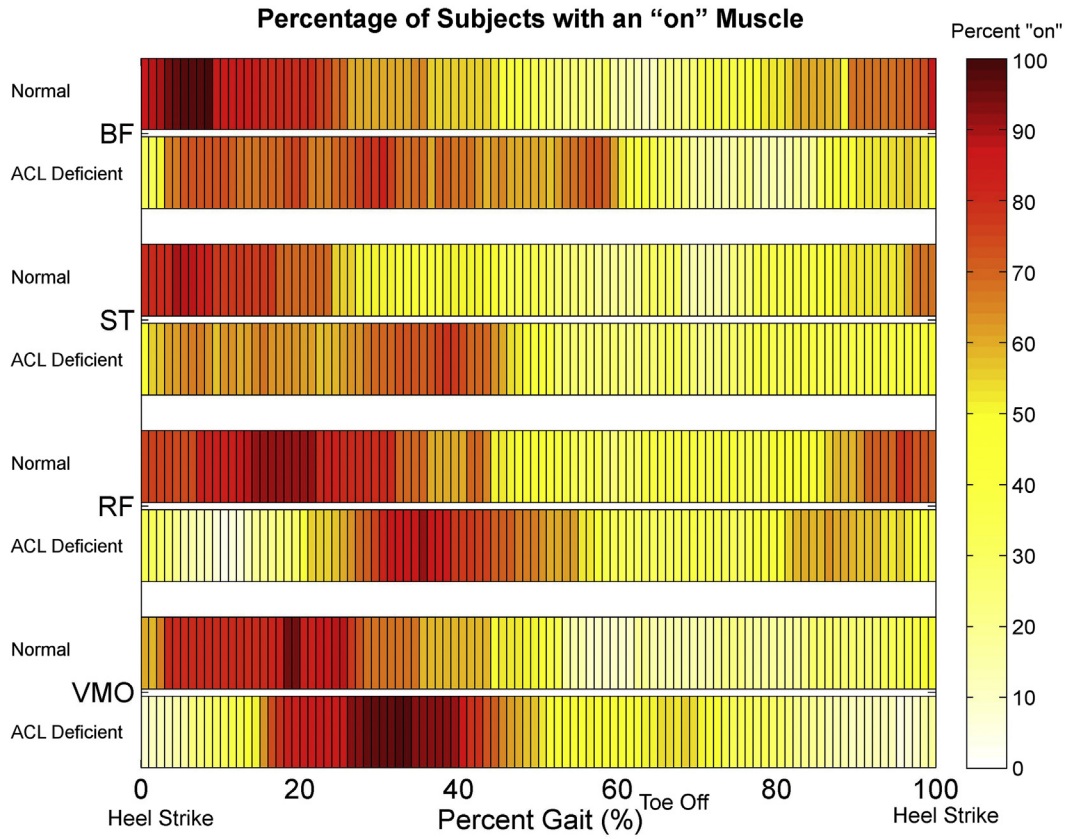

Fig 3. Electromyography average muscle activation patterns during gait. Delays in muscle activation and deactivation can be seen for the injured leg compared with the control subjects. Each rectangle on each horizontal bar represents a time point during the gait cycle. The darker colors represent a higher percentage of muscles "on" at that time in the gait cycle; a lighter (white, light yellow) color means that those particular muscles were not "on" or activated, during that time in the gait cycle. Muscles were vastus medialis (VMO), rectus femoris (RF), semitendinosus (ST)/semimembranosus, and biceps femoris (BF). (ACL, anterior cruciate ligament.)

programs that focus on quadriceps activation and strengthening in an effort to improve regain full extension and improve gait mechanics before ACLR.

In the setting of an ACL injury, during normal ambulation, the tibia moves anterior until a secondary restraint to anterior tibial translation comes into effect. Secondary restraints to anterior tibial translation include the medial collateral ligament, the medial meniscus, and/or cocontraction of the hamstring musculature.[13-16] Contraction of the quadriceps during the stance phase of gait, as the knee is in or near full extension, results in anterior directed forces on the tibia.[13,17] After ACL injury, decreased quadriceps contraction caused by reflexive quadriceps inhibition is thought to reduce the peak knee flexion moment during normal ambulation, essentially compensating for the otherwise minimally resisted anterior tibial translation.[18-29] Clinically, quadriceps musculature atrophy has been noted in patients with acute ACL injuries, further supporting the theory of quadriceps inhibition in these patients.[30,31] Interestingly, multiple authors have noted the difference in patient response to ACL injury based on EMG analysis, identifying "copers" and "noncopers" to the injury, which may influence the interpretation of early studies on quadriceps inhibition in the setting of both acute and chronic ACL insufficiency.[32-38]

When interpreting the findings of the coronal, transverse, and sagittal plane motion analyses, we did note

Table 4. Surface Electromyography Results: On/Off Timing During Gait

	ACL Patients (% gait)	Control Subjects (% gait)	*P* (*t*-test)	Cohen's *d* (effect size)
Biceps femoris				
On	2.22	−14.1	.021*	1.17
Off	50.0	36.8	.138	0.71
Semimembranosus/semitendinosus				
On	−1.33	−18.0	.116	0.76
Off	40.2	30.2	.286	0.51
Rectus femoris				
On	8.11	−6.90	.087	0.83
Off	39.44	42.20	.806	−0.11
Vastus medialis				
On	14.0	−2.00	.020*	1.18
Off	57.8	41.9	.031*	1.08

NOTE. Zero percent gait is heel strike.
ACL, anterior cruciate ligament.
*Statistically significant.

differences between the injured knee and control subjects in all planes. In the sagittal plane, there was a lower maximum ER moment at heel strike in the injured leg compared with the control subject, as well as a lower maximum ER moment during midstance. As the extension moment reflects net activity of the knee flexors, the reduced extension moment is the result of either reduced hamstring activity, or increased quadriceps activity, or some combination of the above. Because of the limitations of the methodology, we cannot compare the maximum activity of a given muscle group, only the timing of activation, and so it is impossible to determine if the hamstrings or quadriceps are the main factor. In the coronal plane, there was a lower maximum external abduction moment and lower external adduction moment in the injured leg compared with control. These findings are more difficult to interpret because of the muscles crossing the knee that function to perform adduction and/or abduction about the knee, including the gracilis, which was not specifically assessed in this study. In the transverse plane, there was a higher maximum ER moment in the injured leg compared with control. These findings are also difficult to interpret clinically, especially given that the knee is subjected to internal/external rotation when in flexion, as opposed to when the knee is extended. The popliteus, SM, and ST contribute to internal rotation, whereas the biceps femoris and sartorius contribute to ER, and so the combined function of these muscles may have contributed to these findings.

As noted by Shelbourne et al.,[2,3] regaining full ROM, and particularly full extension, before ACLR is paramount to avoiding arthrofibrosis postoperatively, and thus, ensuring that all preventable causes of motion loss before surgery are identified and treated is critical. The motivation for the present study, therefore, was to determine if hamstring muscle activity contributes to decreased knee extension after ACL injury, in addition to, or instead of, reflexive quadriceps inhibition or in some cases, a mechanical block to motion (i.e. ligament stump in the notch). In this study, we determined that the extension moments during heel strike and terminal stance were reduced, indicating a reduced net activity of hamstrings in the ACL-deficient limb. The external extension moment is balanced during gait by net internal flexor muscle activity. Importantly, we found that the biceps femoris activated later in the gait cycle in the ACL-deficient limb compared with the control limb, and that the rectus femoris was "on" for shorter periods of time in the ACL-deficient limb compared with the normal limb, which supports prior studies reporting quadriceps inhibition in the setting of ACL insufficiency. Thus, based on the results from the present study, changes in the contraction timings of both hamstrings and quadriceps likely lead to reduced extension moments in the ACL injured limb.

To the authors' knowledge, this is the first study to comment on the EMG and gait analysis of muscle activation after ACL injury in the acute setting, within 2 weeks of injury. Notably, the ACL-deficient limb does not reach as much extension as the control limb, the sagittal plane extension moments were reduced in the injured limb, and the rectus femoris was "on" for shorter periods during the gait cycle in the injured limb. The authors propose that these changes may be in part due to the combination of loss of knee extension with the onset of an antalgic gait pattern acquired in the acute injury setting. Certainly, additional research is needed to further elucidate the extent of hamstring coactivation involvement in the loss of knee extension in the acute (within 2 week) time period after ACL injury.

Limitations

This study had several limitations. It is possible that pain medication may have altered gait patterns, and we were unable to control this. Similarly, the presence of a knee effusion may impact the ability to fully extend the knee, and this may impact our gait analysis results. For the EMG analysis, we were unable to use a maximum voluntary contraction to normalize the data to be able to evaluate the magnitude of muscle activity, because of the perceived inability of subjects to maximally contract the muscle groups in the ACL-deficient limb, and so alternatively only evaluated muscle contraction on-off timing. In addition, there is no evidence to show that the altered muscle activity pattern shown in this study leads to the loss of passive knee extension, and this needs to be further evaluated in additional studies. Finally, we had a small sample size due to the strict inclusion criteria of enrolling patients within 2 weeks of injury but before surgery. We calculated effect sizes for the on-off timing of all muscles and found a large effect for the on timing of the rectus femoris (Cohen's d of 0.83), medium effects for the on and off timing of the SM/ST (Cohen's d of 0.76 and 0.51, respectively), and a medium effect for the off timing of the biceps femoris (Cohen's d of 0.71). Putting the significant differences and medium to large effect sizes together, the off timing of the rectus femoris muscle is the only value that is likely not different between the ACL-deficient and control limb given a larger sample size. Given the strict inclusion criteria of capturing patients within 2 weeks of ACL injury as well as the cost associated with analysis, we did not perform an a priori power analysis, which may subject our dataset to type II errors.

Conclusions

In patients with acute ACL injury, the ACL-deficient limb does not reach as much extension as controls. Although the rectus femoris is "on" for shorter periods during the gait cycle, there is no difference in hamstring

time on during gait. This information may help clinicians better understand muscle function and gait patterns in the acute time period after ACL injury.

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