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An Investigation of Postural Control in Postoperative Anterior Cruciate Ligament Reconstruction Patients

Mark Hoffman, PhD, ATC*; John Schrader, HSD, ATC†; David Koceja, PhD†

* University of Nevada-Las Vegas, Las Vegas, NV; † Indiana University, Bloomington, IN

Objective: To investigate quadriceps strength and static and dynamic balance in the anterior cruciate ligament (ACL)-reconstructed patient and to compare these findings with an age-matched, injury-free control group.

Design and Setting: A 2 × 2 mixed-design analysis of variance (group × leg) was applied to the static posture, dynamic balance, and strength data. In addition, Pearson product-moment correlations were calculated to determine the strength of the relationships among the dependent measures. All data were collected in the Motor Control Laboratory at Indiana University.

Subjects: The experimental group was composed of 20 individuals who had undergone ACL reconstruction with a patellar tendon autograft. The control group comprised 20 participants with no history of significant orthopaedic injuries to the lower extremities.

Measurements: The dependent variables were sway path linear mean for the static condition, dynamic-phase recovery time after perturbation for the dynamic measure, and quadriceps peak torque for strength.

Results: We found significant differences between the ACL and control groups on the measures of dynamic-phase duration and peak torque. The static sway variable did not show a significant difference.

Conclusions: Evaluation of the postural control system under 2 conditions, static and dynamic, showed differences between the ACL and control groups for the dynamic condition only. These results suggest the presence of independent control mechanisms for the control of static and more dynamic postures. In addition, because there were no differences between the injured and noninjured legs of the ACL group, the theory of a central postural control scheme is supported.

Key Words: balance, posture

Over the past 20 years, both athletes and athletic trainers have witnessed monumental improvements in the treatment of anterior cruciate ligament (ACL) ruptures. Today, an estimated 50000 reconstructions are performed worldwide each year.[1] For an athlete, rupturing the ACL typically results in the end of the competitive season. This contrasts with years past, when rupturing the ACL resulted in the end of a competitive career. Most of the advances responsible for allowing the return to preinjury activity have resulted from improvements in surgical techniques and rehabilitation procedures. Along with these technical and procedural advances have come directed experimental research related to ACL injury. One specific area that continues to flourish and that is being addressed from a variety of perspectives is the study of the neurologic structure and function of the ACL. For example, some reports have detailed the neural network of the ACL,[2–7] while others have focused on the role of the ACL in proprioception[8,9] and postural control.[10]

Although some questions concerning the neural innervation of the knee have been addressed through animal[2,3,11] and human[3–6,12] models, the exact neurologic importance of the ACL remains equivocal. However, researchers generally agree that the ACL does contains mechanoreceptors.[2–6,11,13] Other work in this area has identified a direct pathway from the ACL to the central nervous system via the posterior articular and sciatic nerves.[14] Additional work by Pitman et al[15] revealed a direct connection between the human ACL and the cerebral cortex via the use of evoked potential measurements from the scalp.

A primary reason that ACL neurology has become so intensely studied relates to speculation that sensory information disruption at the knee results in repeated episodes of microtrauma.[2] Specifically, when the central nervous system has decreased sensory information from the knee, there is a decreased ability to adequately stabilize the lower extremity,[16] initiating a repetitive cycle of sensory impairment and microtrauma.[2] In support of this theory, proprioceptive deficits related to passive movement have been found in patients with chronic ACL injuries,[8] and, following ACL rupture, static postural control is decreased.[9,16] However, little is known about the dynamic aspects of postural control after ACL reconstruction.

Address correspondence to Mark Hoffman, PhD, ATC, Department of Kinesiology, University of Nevada-Las Vegas, 4505 Maryland Parkway 453034, Las Vegas, NV 89154-3034. E-mail address: MHOFFMAN@nevada.edu

Although static postural control is a valuable measure of somatosensory integration, a need exists for postural measurements that target the dynamic aspects of postural control. Moreover, authors have expressed concern that measurement of static postural control fails to provide critical information related to factors that might predispose individuals to injury during functional activities.[10,17] The concerns related to static balance have spurred an increased effort to develop a tool for the assessment of the dynamic components of posture and balance. Although static measures of stability are valuable, one possible limitation is their questionable relationship to dynamic balance and function.[18]

The purpose of our study was to investigate quadriceps strength and static and dynamic balance in the ACL-reconstructed patient and to compare these findings with an age-matched, injury-free control group.

METHODS

Subjects

A total of 40 individuals agreed to participate in this study. Twenty healthy individuals who reported no history of significant orthopaedic injury or balance-related disorders served as the control group. Significant orthopaedic injury was defined as an injury with symptoms persisting for longer than 2 weeks. The control group comprised 7 females and 13 males of mean age 24.0 ± 4.07 years, height 175.30 ± 9.21 cm, and weight 75.41 ± 16.22 kg. For the ACL-reconstructed group, mean age was 23.4 ± 5.79 years, height was 172.72 ± 9.65 cm, and weight was 70.91 ± 17.84 kg. The 12 females and 8 males had undergone complete reconstruction of the ACL with a patellar tendon graft in an arthroscopically assisted procedure. In all cases, the surgery was performed within 30 months of testing, and no procedures were more recent than 3 months (mean, 9.52 months). Each subject was functionally stable and was cleared by the surgeon for participation in this study. The ACL participants were additionally screened, via a questionnaire, for any potentially confounding conditions (eg, arthritis or leg length differences). Individuals were excluded if they had sustained a significant injury to either lower extremity other than the ACL rupture or if they had injured any other knee ligaments at the time of the ACL rupture. Individuals who had undergone minor meniscal treatment were included.

Before testing, each participant read and completed a subject informed consent form, as approved by the Committee for the Protection of Human Subjects at Indiana University, and a participant questionnaire requiring demographic and injury information.

Participants were tested on the following protocols: (1) functional leg dominance determination, (2) isokinetic strength test, (3) static balance evaluation, and (4) dynamic balance evaluation.

Leg Dominance

Functional leg dominance was determined with 3 functional tests: ball kick test, step-up test, and balance recovery test. Three trials were conducted for each test. During the ball kick test and the step-up test, the leg used to kick the ball and the leg used to step up was identified as dominant. The balance recovery test consisted of the experimenter's nudging the subject off balance by applying a force on the spine at the midscapular level. The leg the subject used to recover balance was identified as dominant. The leg used as the dominant leg by the participant in 2 of 3 trials for each test was identified as the dominant leg for that test. After the 3 tests were completed, the results were examined, and the dominant leg in 2 of the 3 tests was determined to be the functionally dominant leg for this study. A detailed explanation of these tests can be found in Hoffman et al.[19]

Strength Testing

A Cybex II dynamometer (Lumex, Ronkonkoma, NY) was used for testing the strength of the quadriceps muscles. The dynamometer, interfaced with a personal computer, was configured to measure peak torque generated at 60°/s; 60°/s was chosen as the testing speed because the primary focus of the strength evaluation was determining peak torque. If the focus of this study had been to measure functional strength, a velocity spectrum of faster speeds would have been used. Each participant was seated with the hip and knee flexed to 90°. The joint line of the knee was aligned with the rotational axis of the dynamometer head, and the lower end of the torque arm was secured just superior to the level of the malleoli. Each participant was secured at the thigh, waist, and chest and performed 1 set of 5 repetitions after a warm-up session. The peak value from the 5 repetitions was determined to be the peak torque and was used in the analysis.

Static Balance Evaluation

During the static balance evaluation, the participants were instructed to assume single-leg stance on a Kistler Force Platform (Kistler Instrument Company, Amherst, NY), place hands on hips, and focus on a visual target approximately 1.0 m away placed at eye level. Additionally, the participants were instructed to hold the hip, knee, and ankle of the nonsupport leg at a self-selected angle without allowing the 2 legs to touch. After a verbal signal from the participant indicating the assumption of a comfortable and stable stance, a 20-second trial was recorded. Each participant of the experimental group performed 4 20-second trials on both the involved and uninvolved legs; each participant of the control group performed the same trials on both the dominant and nondominant legs. The center-of-pressure excursions were monitored at a sampling rate of 50 Hz. The dependent variable used for the static evaluation was sway path linear mean. Sway path linear mean is the average distance (mm) traveled per sample interval (20 milliseconds). It was calculated by summing the excursions of the center of pressure between each sample and dividing by the number of samples. A comprehensive explanation of the calculation of this variable has been detailed by Hufschmidt et al.[20]

Dynamic Balance Evaluation

For evaluation of dynamic balance, each participant was tested for 20 seconds in a stance similar to the stance used in the static condition. However, to assess dynamic balance, recovery time from perturbation was measured. At a random point between seconds 8 and 12, an electrical perturbation was delivered to the tibial nerve of the support leg. The stimulation induced an involuntary contraction of the triceps surae, resulting in posterior displacement of the participant's center of gravity over the base of support. This random perturbation forced the participant to make corrective movements with the leg and hips in order to re-establish a stable posture. If a subject moved hands from hips or touched the ground with the nonstance foot, the trial was repeated. A detailed description of this methodology appeared in Hoffman and Koceja.[21]

To produce the perturbation, soleus M-waves were elicited according to procedures outlined by Hugon.[22] Briefly, surface recording electrodes (Ag/AgCl) were placed over the soleus muscle belly bilaterally. A stimulating electrode (1 cm^2) was placed in the popliteal fossa for current delivery, and a dispersal pad (3 cm^2) was placed superior to the patella on the distal thigh. A percutaneous electrical stimulus (1.0-millisecond square-wave pulse) was applied to the posterior tibial nerve in the popliteal fossa to elicit the maximum M-wave. Before testing, the maximum M-wave was established. During each trial, the peak-to-peak M-waves were recorded to assure perturbation consistency.

The intensity of the stimulation used for perturbation on each leg was individually set above the level that elicited a maximum soleus M-wave (eg, 4× motor threshold). Since the maximum M-wave indicated activation of all the alpha motoneurons of the soleus motoneuron pool, consistency of perturbation for each participant was assured.

Computerized scanning and graphing of each trial's sagittal plane center-of-pressure movement allowed separation of the dynamic trials into 3 phases: prestimulation, active, and recovered (Figure 1). The prestimulation phase included all data from the start of the trial to the point of rapid center-of-pressure acceleration (perturbation). The active phase included all data from the point of rapid acceleration of the center of pressure to the point where the participant returned to a level of sway similar to that measured in the prestimulation phase. The recovered phase included the remaining portion of the trial.

A computer program was designed to scan the data and to determine the points used to separate the trials into the 3 phases. The first step in the analysis was to determine the beginning of the active phase, which was easily detected due to the rapid acceleration of the center of pressure from the perturbation. The second step was to establish the threshold level used to determine when the participant had recovered. This was done by determining the sway variability of the prestimulation phase and applying that variability as a threshold to the data from the point of stimulation to the end of the trial. By scanning from the point of perturbation forward, the program determined the point where the participant had recovered from the perturbation.

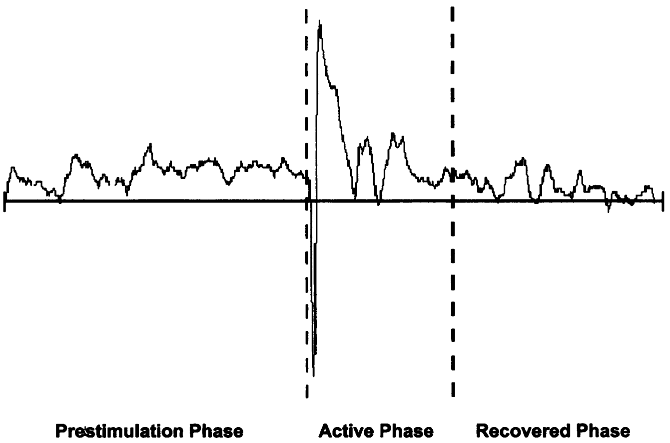

Figure 1. Anterior-posterior center-of-pressure tracing for a dynamic balance trial (subject 14E, trial 3). The vertical axis is center-of-pressure displacement from a relative starting point, and the horizontal axis represents 20 seconds total time. Note the separation of the trial into the 3 phases: prestimulation, active phase, and recovered phase.

Statistical Analysis

Zero-order correlations at a .05 probability level were determined to establish the relationships among the 3 dependent variables (peak torque, sway path linear mean, and dynamic-phase duration). Since none of these correlations were significant, univariate analyses were used to analyze each dependent variable. Specifically, a 2-factor analysis of variance (group × leg) was performed on each dependent variable. All significant interactions were broken down into simple main effects for interpretation.

RESULTS

The zero-order correlations are presented in Table 1. A summary of all group and leg means is presented in Table 2.

Table 1. Dependent Variable Zero-Order Correlations (N = 40)

	Dynamic-Phase Duration	Sway Path Linear Mean	Peak Torque
Dynamic-phase duration	1	0.218 (*P* = .18)	−0.210 (*P* = .19)
Sway path linear mean		1	−0.149 (*P* = .36)
Peak torque			1

Table 2. Summary Table of Group and Leg Means ± SD

	Control			ACL		
Variables	Leg 1*	Leg 2†	Group	Leg 1*	Leg 2†	Group
Strength						
Peak torque (Nm)	249.76 ± 81.86	241.37 ± 79.99	245.49 ± 80.01	144.08 ± 72.27	213.96 ± 70.26	179.02 ± 78.74
Static condition						
Sway path linear mean (mm)	0.86 ± 0.12	0.88 ± 0.12	0.87 ± 0.12	0.86 ± 0.16	0.84 ± 0.12	0.85 ± 0.14
Dynamic condition						
Dynamic-phase duration (s)	2.54 ± 0.73	2.77 ± 0.64	2.65 ± 0.68	3.21 ± 0.87	2.92 ± 0.77	3.06 ± 0.82

* Dominant leg for the control group and involved leg for the ACL group.

† Nondominant leg for the control group and uninvolved leg for the ACL group.

Strength

The analysis of peak torque at 60°/sec demonstrated a significant group-by-leg interaction ($F_{1,38} = 49.56$, $P < .001$) (Figure 2). Investigation of the interaction showed a significant simple main effect for the ACL group ($F_{1,38} = 78.75$, $P < .001$) and a nonsignificant effect between legs in the control group ($F_{1,38} = 1.17$, $P = .286$). Specifically, there was a significant reduction in the peak force generated by the ACL leg when compared with the uninjured leg.

Static Condition

The analysis of the static condition sway path linear mean showed no significant main effects or interaction (Figure 3).

Dynamic Condition

The primary dependent variable for this study, dynamic-phase duration, showed a significant main effect for group ($F_{1,38} = 4.94$, $P = .032$), with the ACL group having longer durations (3.06 seconds versus 2.65 seconds) but no main effect for leg ($F_{1,38} = 0.04$, $P = .840$) (Figure 4).

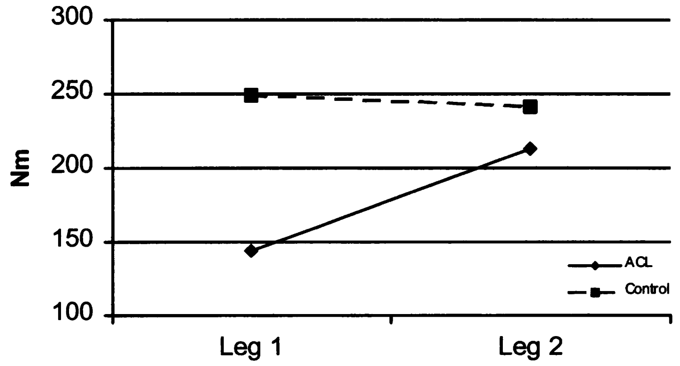

Figure 2. Peak torque. Leg 1 represents the reconstructed leg for the ACL group and the dominant leg for the control group. Leg 2 represents the uninjured leg of the ACL group and the nondominant leg of the control group. The significant interaction was due to reduced peak torque in the ACL group compared with the control group.

DISCUSSION

The methods used to analyze the data in this study allowed us to compare postural sway data from a variety of perspectives. Specifically, they allowed for the separation of the dynamic trials into 3 parts. The first part of the dynamic trials contained static sway data from the beginning of the trial to the onset of the perturbation. The second part of the dynamic trials contained sway data obtained while the participant was responding to the perturbation and therefore was the active, or dynamic, portion of the trial. Finally, the third part of the trial contained sway data that was obtained after the participant had recovered from the perturbation. Breaking the trials into these parts allowed us to compare the 3 phases. Theoretically, before the perturbation (prestimulation phase) and after the point of recovery from the perturbation (recovered phase), the participant was in a relatively quiet single-leg stance. Based on this premise, the data from these 2 parts of the perturbation trials (prestimulation and recovery) could be compared with the static sway trials. Additionally, it was also of interest to determine whether, after the perturbation, the subjects had truly returned to a level of stability that was equal to the static trials. These results showed no differences in the amount of sway in any of these comparisons. These findings provide some level of assurance that the subjects did not

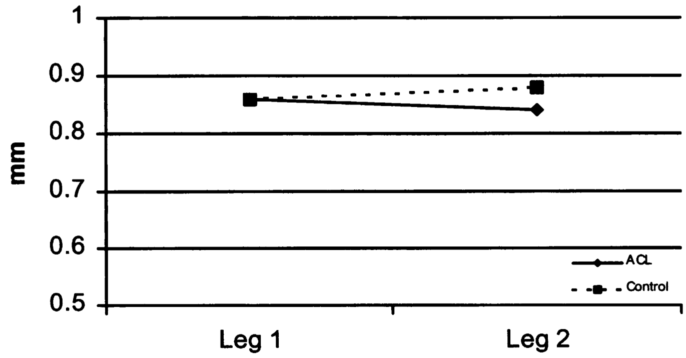

Figure 3. Static condition sway path linear mean. Leg 1 represents the reconstructed leg for the ACL group and the dominant leg for the control group. Leg 2 represents the uninjured leg of the ACL group and the nondominant leg of the control group. The static measures on all legs were remarkably similar.

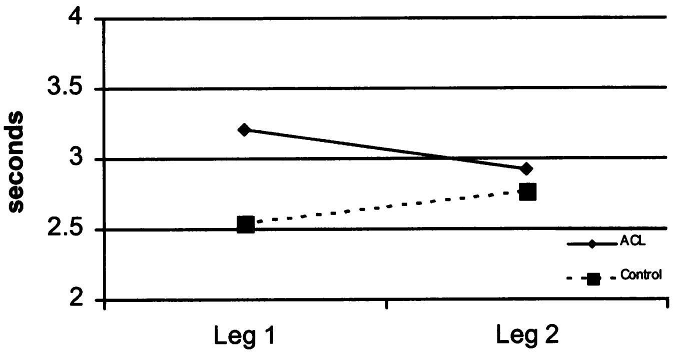

Figure 4. Dynamic-phase duration. Leg 1 represents the reconstructed leg for the ACL group and the dominant leg for the control group. Leg 2 represents the uninjured leg of the ACL group and the nondominant leg of the control group. Although there was a significant leg difference, there was no significant difference between the two legs of the ACL group.

alter their standing strategies before or after being perturbed. From a methodologic standpoint, this is very important in demonstrating that, although the participants were aware that they were going to be perturbed, their prestimulation static sway was not affected.

Two common mechanisms known to alter afferent joint activity (somatosensory information) are direct mechanoreceptor damage[23] and joint effusion.[24] The concept of joint pathology affecting somatosensory input to the central nervous system was first thoroughly outlined by Freeman and Wyke.[23] These authors suggested that injury to the connective tissue of a joint damages the mechanoreceptors of the capsule and ligaments.[23] In addition, they reported that damage to the joint mechanoreceptors alters feedback to the central nervous system for the control of that specific joint.

Other authors report that joint injury to the mechanoreceptors manifests changes in a different way.[24–26] These authors suggested that mechanoreceptor damage disrupts a central postural control mechanism.[24–26] Based on this theory, joint damage to mechanoreceptors should affect the postural control of both the injured and uninjured legs as measured by single-leg tests. Conversely, if postural control changes result directly from mechanoreceptor damage, causing only localized joint impairment, then deficits should be confined to the injured joint or limb, as reported by Freeman and Wyke.[23]

Independent of the exact mechanism of decreased postural control associated with ACL injury, it has been documented that ACL injury negatively affects postural control.[10] As mentioned above, the second mechanism known to affect joint mechanoreceptors is articular joint effusion. Although this mechanism has been studied much less than mechanoreceptor damage, very small amounts of fluid introduced into the knee capsule have resulted in inhibition of the quadriceps muscle[2] as a result of altered afferent activity from increased joint capsule pressure. If more advances in the treatment and rehabilitation of ACL rupture are to occur, the exact mechanism responsible for sensory alterations and detailed neural pathway mapping must be established. Although none of the subjects in this study had a knee effusion, the effects of recent effusions cannot be dismissed.

Many areas of research provide supporting evidence that the ACL contains a vast neurologic supply.[2–6,12] Moreover, research supports the existence of direct connections between neurologic structures of the ACL and the spinal cord, as well as supraspinal areas.[4] In addition, it has been shown that ACL rupture disrupts the postural control system,[10] which receives sensory information from the visual, vestibular, and somatosensory systems. Rupture of the ACL directly affects only the somatosensory weighting of information in the postural control equation.

Our strength findings parallel the reports of others who detail quadriceps strength deficits after ACL reconstruction.[27,28] The peak torque values of the quadriceps indicated strength differences between the reconstructed and the contralateral leg. This indicates that, although there is clear evidence of contralateral neural connections associated with strength,[29] the ACL rupture and reconstruction do not appear to affect force production of the contralateral leg.

The static sway variable of the linear sway path mean showed no difference between the groups or between the legs of the ACL group. This finding is particularly interesting when evaluated in conjunction with the difference between groups on the phase duration of the dynamic balance testing. Our previous study has shown low correlations between static and dynamic measures of postural control.[8] This finding lends support to the idea that static postural control and dynamic balance are governed by different mechanisms.

Participants in the ACL group demonstrated significantly longer phase durations than the participants in the control group, even though no differences between the involved and uninvolved legs of the ACL group were detected. These findings suggest a neurologic crossover effect from the injured to uninjured leg and support the use of a central postural control mechanism. We are not the first to report a decrease in the postural control of the uninvolved leg of patients after ACL rupture. Other authors[30,31] measured the single-leg static postural control of patients with unilateral ACL deficiencies and reported differences between the ACL and control groups, with no difference between the legs of the ACL group. They attributed their findings to a decrease in overall physical activity of the participants in the ACL group.[29,30] We have taken a more theoretical approach to explaining our results.

Our explanation is based on the idea that the central nervous system is a very plastic entity that can make alterations based on functional demands. Simply, when the ACL is ruptured, the involved leg is compromised because a major mechanical structure has been injured. Unilateral ACL rupture results in an asymmetry between the involved and uninvolved legs. The mechanism the body is able to use, which quickly reestablishes symmetry, is to reduce the function of the uninvolved leg. By decreasing the function of the uninvolved leg, the magnitude of the asymmetry is lessened. Although this decreases the overall postural control of the system to some degree, it re-establishes symmetry between the legs of the patient. Similar findings in a related area (functional testing) have shown functional decreases in the uninvolved limb after ACL rupture when compared with a control group.[32] The results suggested that, in activities where the knee was exposed to great levels of stress, the involved leg exhibited a functional profile similar to the uninvolved leg. However, both legs of the ACL group showed decreased functional ability compared with a control group.[31] The authors concluded that a change in the central control of posture had affected both the involved and the uninvolved legs of the ACL group.[31] In addition, these authors suggested that the phenomena of no differences between the involved and uninvolved legs when both legs are actually affected may be problematic for previous studies that were limited to between-leg comparisons of individuals.

The goals of our study were to investigate aspects of static balance, dynamic balance, and quadriceps strength. Our current results did not indicate differences between the groups in the measurement of static balance. Although Friden et al[31] reported decreased static postural control in both the involved and uninvolved legs of participants with ACL ruptures, this result is possibly due to the use of different static variables of static sway measurement. The results of both studies suggest a decrease in the general control of posture after ACL rupture.

In conclusion, participants who had undergone ACL reconstruction demonstrated differences from a control group in both strength and recovery from perturbation without demonstrating differences in a measure of static balance. Although differences were found on both the dynamic balance measure and the strength measure, these variables were not correlated in either the ACL or control groups. The results suggest the disruption of a central control mechanism of posture, since the dynamic balance measure did not show differences between the legs of the ACL group but did show group differences between the ACL and control groups.

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