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Three-Dimensional Tibiofemoral Kinematics of the Anterior Cruciate Ligament-Deficient and Reconstructed Knee during Walking*

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Background: It is possible that gait abnormalities may play a role in the pathogenesis of meniscal or chondral injury as well as osteoarthritis of the knee in patients with anterior cruciate ligament deficiency.
Hypothesis: The three-dimensional kinematics of anterior cruciate ligament-deficient knees are changed even during low-stress activities, such as walking, but can be restored by reconstruction.
Study Design: Case control study.
Methods: Using a three-dimensional optoelectronic gait analysis system, we examined 13 patients with anterior cruciate ligament-deficient knees, 21 patients with anterior cruciate ligament-reconstructed knees, and 10 control subjects with uninjured knees during walking.
Results: Normal patterns of knee flexion-extension, abduction-adduction, and internal-external rotation during the gait cycle were maintained by all subjects. A significant difference in tibial rotation angle during the initial swing phase was found in anterior cruciate ligament-deficient knees compared with reconstructed and control knees. The patients with anterior cruciate ligament-deficient knees rotated the tibia internally during the initial swing phase, whereas the others rotated externally.
Conclusions: Patients with anterior cruciate ligament-deficient knees experienced repeated episodes of rotational instability during walking, whereas patients with reconstruction experienced tibial rotation that is closer to normal.
Clinical Relevance: Repeated episodes of knee rotational instability may play a role in the development of pathologic knee conditions.

Anterior cruciate ligament rupture is a frequent injury of the knee joint that can lead to increased anterior translation of the tibia and knee instability.[19] It has also been suggested that some patients with ACL-deficient knees develop functional adaptations like the "quadriceps avoidance gait pattern" to prevent excessive anterior tibial translation.[5] Unfortunately, such adaptations fail to protect the ACL-deficient knee from developing pathologic conditions. Reports in the literature have indicated that ACL deficiency is associated with a high incidence of meniscal or chondral injury as well as osteoarthritis of the knee.[8,11,15,19,22] Although these changes have been most clearly related to the discrete giving-way episodes that are known to occur in the ACL-deficient knee, it is possible that gait abnormalities may also play a role in their pathogenesis. Therefore, it is very important to understand the

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gait adaptations of the ACL-deficient knee, the in vivo kinematics of the knee joint, and the influence of ACL reconstruction on these failures.

Using gait analysis, several investigators have conducted biomechanical studies to evaluate the changes in gait patterns of ACL-deficient knees compared with uninjured knees.[1,5,12,17,21,25,27] Wexler et al.[27] reported on patients with ACL-deficient knees more than 7.5 years after injury and found that these patients walked with increased knee extension angles during the terminal stance. The authors attributed this finding to lower magnitudes of external flexion moments in the ACL-deficient knees as compared with healthy controls. DeVita et al.[12] reported that sagittal knee kinematics showed excellent recovery at 6 months after ACL reconstruction and after accelerated rehabilitation. Berchuck et al.[5] reported on the most-documented functional adaptation of ACL-deficient knees, the quadriceps avoidance gait pattern. In this adaptation, the midstance knee flexion moment is an extension moment rather than a flexion moment. This phenomenon has been attributed to a decrease in quadriceps muscle activity, which, along with a potential quadriceps muscle strength deficit, can reduce the anterior drawer motion.

In their evaluations, the authors of these studies emphasized movements that occur in the sagittal plane (flexion-extension). Thus, our knowledge about tibial abduction-adduction and internal-external rotation in uninjured, ACL-deficient, and ACL-reconstructed knees is limited.[23] The objective of our study was to examine the effect of ACL deficiency and reconstruction on knee joint kinematics during walking. A three-dimensional optoelectronic gait analysis system was used to analyze lower limb kinematics during walking. We tested the hypothesis that the three-dimensional kinematics of ACL-deficient knees is changed even during low-stress activities such as walking. We further hypothesized that ACL reconstruction can restore normal knee joint kinematics.

MATERIALS AND METHODS

Forty-four subjects were analyzed during walking at a freely selected pace. Thirteen patients with ACL-deficient knees (3 women and 10 men with a mean age of 26 ± 5 years, a mean weight of 76 ± 7 kg, and a mean height of 1.75 ± 0.04 meters) and 21 patients with ACL-reconstructed knees (2 women and 19 men with a mean age of 25 ± 4 years, mean weight of 69.11 ± 7.89 kg, and a mean height of 1.73 ± 0.06 meters) were evaluated. Ten healthy subjects matched for age, height, and weight (two women and eight men with a mean age of 24.7 ± 3.7 years, a mean weight of 62.1 ± 12.38 kg, and a mean height of 1.71 ± 0.05 meters) without a history of lower limb pathologic conditions were selected as the control group. The ACL-deficient group consisted of patients who had complete ACL rupture but no previous knee or lower limb pathologic conditions. Patients who had anterior knee pain or who had injuries to the articular surface, collateral ligaments, or the PCL were excluded from the study. However, 6 of the 21 patients with ACL-reconstructed knees had had an associated medial meniscal injury. A partial meniscectomy had been performed in two of the six patients, but no more than 25% of the meniscus had been removed. In the remaining four patients, the torn meniscus had been sutured.

The ACL ruptures were diagnosed clinically and confirmed with MRI as well as by arthroscopic examination in both groups. The operated patients underwent arthroscopically assisted ACL reconstruction with use of an autogenous bone-patellar tendon-bone autograft.[14,16] The effectiveness of the ACL reconstruction in reducing the anterior displacement of the tibia was evaluated with KT-1000 arthrometer (Medmetric Corp., San Diego, California) measurements. Patients with ACL-reconstructed knees who had a side-to-side difference in anterior tibial displacement of more than 3 mm or who had loss of knee motion were excluded from the study. An additional inclusion criterion was that walking was pain-free. All patients with ACL-deficient and reconstructed knees had resumed activities of daily living.

The gait evaluation of the patients with ACL-deficient knees was performed 7.6 ± 4.3 weeks after their injury, and the patients with ACL-reconstructed knees were evaluated 30 ± 16.9 weeks after their operation. Kinematic data were collected from a Peak Performance real-time motion analysis system with six infrared light cameras (Peak Performance Technologies, Inc., Englewood, Colorado). Data collection was performed at the sampling rate of 50 Hz, and a Butterworth filter was used to decrease digitizing error with a cutoff frequency of 6 Hz. Reflective markers were placed on both lower limbs according to the model developed by Vaughan[26]: on the lateral side of the foot at the head of the second metatarsal joint, on the heel, and on surface locations over the lateral malleolus, the lateral side of the calf at the level of maximum calf circumference, the lateral femoral condyle, the lateral side of the midthigh circumference, the right and left anterior superior iliac spine, and the sacrum.

The testing protocol was the same for all groups. The subjects were asked to walk at a self-selected pace on a 10-meter walkway. To increase the accuracy of the measurements, we kept the subjects from knowing when data were actually recorded. All gait mean variables were calculated by averaging 12 strides from 6 trials or walks. In each trial, two consecutive strides from each side were recorded. On the basis of the recommendations of Bates et al.,[4] 12 strides were considered an adequate sample to provide a representative mean for each subject/condition.

The variables examined in the present study were the knee flexion at toe-off, maximum knee flexion during swing, knee flexion at heel-strike, maximum knee flexion during loading response (midstance), maximum tibial internal-external rotation during the gait cycle, maximum tibial adduction-abduction during the gait cycle, cadence, and average gait velocity. The midthigh circumference was also evaluated for the affected and unaffected limbs for the ACL-deficient and reconstructed groups. In addition, the differences between the two limbs were estimated and averaged. One-way analyses of variance were performed on the subject means on the listed parameters.

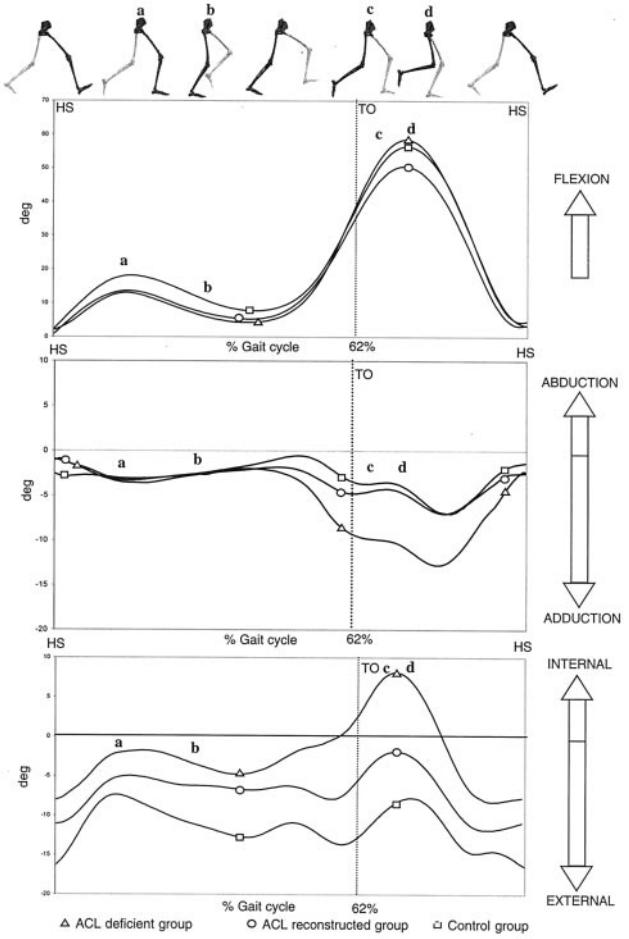

Figure 1. Group mean ensemble curves for knee flexion-extension (top panel), tibial abduction-adduction (middle panel), and tibial internal-external rotation (bottom panel). a, loading response; b, midstance; c, initial swing; d, midswing; TO, toe-off; HS, heel-strike.

For tests that resulted in a significant F ratio ($P < 0.05$), post hoc analysis was performed by using the Tukey multiple comparisons procedure. For the midthigh circumference, paired t-tests were used to compare the affected and unaffected limbs for the ACL-deficient and reconstructed groups.

RESULTS

In all subjects, the configurations of the knee flexion-extension, abduction-adduction, and internal-external rotation angle curves were maintained (Fig. 1). Furthermore, the examination of discrete events from these curves revealed no significant differences between the three groups, except maximum tibial rotation during the gait cycle (Table 1).

Specifically, a significant difference was found in maximum tibial rotation angle in the ACL-deficient group when compared with the ACL-reconstructed ($P < 0.008$) and the control group ($P < 0.003$). The mean value of this variable in the ACL-deficient group was 9.6° ± 8.66° of internal rotation. In the ACL-reconstructed and control groups, the mean value was 0.3° ± 9.9° of external rotation and 3.6° ± 6.22° of external rotation, respectively. The tibial rotation angle during swing reached its maximum value during the initial swing phase in all groups (Fig. 1).

Furthermore, significant differences were found for midthigh circumference between the affected and the unaffected limbs for the ACL-deficient and the ACL-reconstructed groups. The mean difference for the midthigh circumference was 1.43 ± 0.98 cm ($P < 0.001$) for the ACL-reconstructed group and 1.5 ± 0.86 cm ($P < 0.001$) for the ACL-deficient group.

DISCUSSION

The objective of this study was to examine the effect of ACL deficiency and ACL reconstruction on the three-dimensional kinematics of the knee joint during walking. For data collection, we used an optoelectronic gait analysis system, which provided data for the three-dimensional

TABLE 1
Group Means and Standard Deviations for All Dependent Variables Examined

Dependent variables (mean ± SD)	Group			P value
	ACL deficient (N = 13)	ACL reconstructed (N = 21)	Control (N = 10)	
Knee flexion at toe-off (deg)	32.65 ± 3.33	34.12 ± 3.68	33.78 ± 2.87	>0.05
Maximum knee flexion during swing (deg)	59.08 ± 7.71	58.54 ± 3.94	57.89 ± 3.59	>0.05
Knee flexion at heel-strike (deg)	3.41 ± 4.72	1.23 ± 6.33	2.3 ± 3.3	>0.05
Maximum knee flexion during loading response (deg)	15.64 ± 5.99	13.41 ± 9.97	14.52 ± 5.81	>0.05
Maximum tibial abduction (+) adduction (−) during gait cycle (deg)	−2.12 ± 6.54	−1.72 ± 4.10	−0.99 ± 3.09	>0.05
Maximum tibial rotation internal (+) external (−) during gait cycle (deg)	9.6 ± 8.66	−0.3 ± 9.9[a]	−3.6 ± 6.22[b]	<0.05
Cadence (steps/min)	109.18 ± 6.49	109.71 ± 7.01	109.32 ± 5.67	>0.05
Averaged gait velocity (m/sec)	1.31 ± 0.33	1.28 ± 0.19	1.18 ± 0.2	>0.05

[a] Significantly different at $P = 0.008$.
[b] Significantly different at $P = 0.003$.

positions of body segments. The system allowed for assessment of the kinematics of the ACL-deficient knees and the effectiveness of reconstruction in restoring normal angular tibiofemoral kinematics.

No statistically significant differences were found in the sagittal-plane knee kinematics during the gait cycle. Although other investigators have reported a significant decrease in knee flexion during the terminal stance phase,[12,24] in our study the differences in terminal knee flexion did not reach significance. Berchuck et al.[5] reported that patients with ACL-deficient knees tended to walk with a reduced knee flexion angle during the stance phase of gait. However, it is possible that the terminal knee flexion angle is significantly reduced only after a substantial amount of time has passed.[27]

It is possible that, as the nervous system adapts to the injury, the knee flexion angles can decrease, resulting in a lower demand on the quadriceps muscles. Arms et al.[3] reported that simulated isometric quadriceps muscle contraction increased anteromedial ACL strain significantly above the normal resting level through the first 45° of knee flexion ($P < 0.005$). Thus, low knee flexion angles during the stance phase can be a protective mechanism against excessive anterior tibial translation in ACL-deficient knees. Nevertheless, the hamstring muscles may not be able to prevent abnormal anterior tibial translation and rotation when the knee is in a more extended position.[9] Thus, a more extended knee places the structure in an unstable position and possibly forces a neuromuscular decision in preference of activation between the rectus femoris and hamstring muscles.

The lack of significant findings regarding knee flexion-extension indicates that examination of sagittal kinematics in isolation does not provide a comprehensive evaluation of all the neuromuscular adaptations that occur during walking after ACL rupture and reconstruction. This conclusion is in agreement with that of other studies that have also found no significant differences in knee flexion-extension.[1,6,21,25]

The tibia reached its maximum degree of rotation during the initial swing phase of the gait cycle, and large standard deviations for tibial rotation were found in all groups, which probably reflected between-subject variability in knee mobility. This finding is probably due to the variable degree of laxity of the ligaments and the different anatomic configuration of the knee between the subjects. However, a significant difference was found in the maximum tibial rotation angle of the ACL-deficient group when compared with the maximum tibial rotation angle of the ACL-reconstructed and control groups. Among the control group patients, the mean rotation of the tibia was external during the initial swing phase, whereas in the ACL-deficient subjects, this rotation was internal.

Ciccotti et al.[7] reported a significant increase in rectus femoris muscle activity among patients with ACL-deficient knees who had undergone rehabilitation. Normally, and during knee extension, the extension moment produced by activation of the quadriceps muscles results in tibial anterior translation and internal rotation. Thus, the increased activity of the rectus femoris muscle probably results in increased internal tibial rotation. Increased tibial anterior translation and internal rotation characterize the pivot shift phenomenon. This increased combined motion of tibial anterior translation and internal rotation in ACL-deficient knees has also been reported by Noyes and Grood.[20] Furthermore, in the present study, increased tibial internal rotation reached a significant level only during the swing phase. This finding is possibly due to the inactivity of the hamstring muscles and the increased activity in the rectus femoris and gastrocnemius muscles in the late stance and early swing period.[13,18] Andriacchi et al.[2] have also reported increased tibial internal rotation in ACL-deficient knees during the entire gait cycle.

However, we cannot be sure whether these gait adaptations are long- or short-term anomalies. If this adaptation is also a long-term anomaly, then it is possible that this pattern of internal rotation during the swing phase increases the risk of further knee damage. Wexler et al.[27] examined how patients compensate for loss of ACL function over time. They supported the hypothesis that there is a subconscious reprogramming of the locomotive process that protects the knee from excessive anterior tibial translation. The changes in gait showed that the reprogramming process is adaptable. They also hypothesized that these changes may develop as the secondary restraints to anterior translation of the proximal tibia (the medial meniscus) begin to stretch out. In addition, it has been proposed that adaptations that occur in patients with ACL-deficient knees are the result of earlier experiences after the loss of the ACL.[1] If so, then the increased internal rotation found during gait among our ACL-deficient group may be analogous to repeated episodes of instability that occur during the early period after injury and may play a role in the development of gait adaptations or be part of the reprogramming process.

No significant differences in tibial rotation were found between the ACL-reconstructed and the control groups. Given the similar pattern of tibial rotation between the ACL-reconstructed and the healthy control subjects, the ACL reconstruction was effective in reducing increased tibial rotation. This finding is also in agreement with findings from cadaveric studies in which the ACL reconstruction was effective in restoring knee kinematics to the normal levels found in the intact knee.[22] Thus, it is possible that ACL reconstruction can help to protect the knee from future degenerative changes, such as damage to the menisci, even in patients with a low level of activity, through protection against repeated episodes of rotational instability. However, this study cannot provide any evidence that patients with ACL-deficient knees whose level of activity is low and who do not sustain reinjury events have a greater risk of degenerative changes than those who undergo an ACL reconstruction.

A significant decrease in midthigh circumference of the injured and reconstructed limb relative to the contralateral limb was found for the ACL-deficient and the ACL-reconstructed group. The difference in thigh circumference is not surprising in a group of patients who were injured only 3 to 12 weeks before they were studied.

Czerniecki et al.[10] have investigated the rotational sta-

bilizing effect of the knee musculature during walking and jogging. They have shown that there is not a significant correlation during walking between the extent of tibial rotation and the isometric or isokinetic strength of the quadriceps and hamstring muscles (walking speed 84 m/min to 1.4 m/sec). However, this correlation was significant at higher ambulation speeds (slow jogging pace, ambulation speed of 132 m/min or 2.2 m/sec, and moderate jogging pace, ambulation speed of 156 m/min or 2.6 m/sec). In our study, the mean ambulation speed was below the mean walking speed (1.4 m/sec) reported by Czerniecki et al.[10] (Table 1). During higher-demand activities (like jogging and running), the abnormal pattern of tibial rotation found in our ACL-deficient patients may no longer exist. According to Czerniecki et al., this change may be due to the fact that the rotational stabilizing effect on the knee of the quadriceps and hamstring muscles is significant at high ambulation speeds. However, this issue needs further investigation.

In summary, the results of the present study indicated that examining knee joint function only in the sagittal plane is not sufficient for a comprehensive evaluation of the complexity of ACL biomechanics during walking. Furthermore, we demonstrated that patients with an ACL-deficient knee had a tendency to rotate the tibia internally during the initial swing phase, whereas uninjured subjects and patients with ACL-reconstructed knees rotated the tibia externally. Nevertheless, further studies are needed to evaluate the patterns of tibial rotation during activities that include greater rotational forces applied to the ACL-deficient and ACL-reconstructed knees. The effect of strengthening programs during rehabilitation in both ACL-deficient and ACL-reconstructed knees should also be prospectively assessed. The role of compensatory mechanisms that may be taking place at the femur is an additional issue that needs further investigation.

Future studies should delineate whether the repeated episodes of knee rotational instability found in our study, along with other reinjury events that occur during activities of daily living, contribute to the reprogramming of the locomotor process so that protective adaptations like the quadriceps avoidance gait pattern may develop.

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