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Anastasios D. Georgoulis
University of Ioannina

Anastasios Papadonikolakis
University of Ioannina

Christos D. Papageorgiou
University of Ioannina

Argyris Mitsou
University of Athens

Nicholas Stergiou
University of Nebraska at Omaha, nstergiou@unomaha.edu

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Three-dimensional tibiofemoral kinematics of the anterior cruciate deficient and reconstructed knee during walking. (MS#5922)

Anastasios D. Georgoulis, M.D., PhD, Anastasios Papadonikolakis, M.D., Christos D. Papageorgiou M.D., PhD, Argyris Mitsou, M.D., PhD* and Nicholas Stergiou, PhD**.

Orthopaedic Sports Medicine Center of Ioannina

Department of Orthopaedic Surgery, University of Ioannina, Medical School,

Ioannina, Greece

*Department of Orthopaedic Surgery, University of Athens, Medical School, Athens, Greece

**HPER Biomechanics Lab., University of Nebraska at Omaha, Omaha, NE, USA

Address correspondence and reprint requests:

Anastasios D. Georgoulis, M.D., PhD

Department of Orthopaedic Surgery, University of Ioannina, Ioannina, Greece
Meth. Anthrakitou 1,

45221 Ioannina, Greece

Tel: +30-651-64980, 75674, Fax.: +30-651-64980

e-mail:grakos@otenet.gr

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Abstract

~~Anterior cruciate ligament (ACL) deficiency has been associated with abnormal knee biomechanics and a high incidence of further knee pathology. An understanding of the in vivo kinematics of the ACL deficient and ACL reconstructed knee is important to determine the adaptations and pathological motions of the ACL deficient knee as well as for evaluating the functional outcome of the ACL reconstruction.~~ (reviewer #1). Using a three-dimensional optoelectronic gait analysis system, ~~which allows a quantitative three-dimensional description of the human gait~~ we examined the impact of ACL deficiency and reconstruction in knee joint kinematics. Forty-four subjects (13 ACL deficient, 21 ACL reconstructed and 10 control) were examined during walking. The normal patterns of knee flexion-extension, abduction-adduction and internal-external rotation during the gait cycle were maintained in all subjects. A significant difference in tibial rotation angle during the initial swing phase was found when the ACL deficient subjects were compared to the reconstructed ($p<0.008$) and the control subjects ($p<0.007$). The ACL deficient patients rotated the tibia internally during the initial swing phase, while the mean tibial rotation in the ACL reconstructed and normal knees was external. A significant decrease was also found for midthigh circumference ($p<0.001$) at the ACL deficient limb relative to the contralateral limb. ~~This decrease reflects possible weakness of the quadriceps and hamstrings, which could be associated with poor muscle rotational stabilizing effect on the knee~~. (reviewer #1)

~~Our study indicates that the different pattern of tibial rotation in the ACL deficient knees, which is observed even during the low stress activity of walking, could be a mechanism of further knee pathology~~. It was concluded that ACL deficient patients experience repeated episodes of knee rotational instability during walking, while ACL reconstruction results in patterns of tibial rotation that are closer to normal. However, further investigation is required to clarify if the observed abnormal pattern of tibial rotation is part of the re-injury events that occur after the rupture of the ACL and if it can eventually lead to further degeneration of the knee. ~~ACL reconstruction was effective at restoring the normal knee joint rotation pattern during walking~~. ~~Our findings provide further data for the importance of the ACL reconstruction~~

even in patients with a low level of activities. In addition, we provide further evidence for the need of an appropriate muscle strengthening rehabilitation program for ACL deficient and reconstructed patients. (reviewer #2)

Introduction

Anterior cruciate ligament (ACL) rupture is a frequent ligamentous injury of the knee joint. This injury can lead to increased anterior translation of the tibia and knee instability[18]. It has also been suggested that some ACL deficient patients develop functional adaptations like the quandriceps avoidance gait pattern to prevent excessive anterior translation of the tibia[4]. Unfortunately, these adaptations fail to protect the ACL deficient knee from future pathology. The literature [14;18;21]has indicated that ACL deficiency is associated with a high incidence of meniscal or chondral injury as well as osteoarthritis of the knee[7;10]. Probably, pathological knee joint motions that occur during daily activities could be associated with the failure of the above mentioned adaptations to prevent future knee pathology following an ACL rupture. Therefore, it is very important to understand the gait adaptations of the ACL deficient knee, the in vivo kinematics of the knee joint and the influence of ACL reconstruction on them.

Using gait analysis several biomechanical studies have evaluated the changes in the gait patterns of the normal and ACL deficient knees[1;4;11;16;20;24;26]. Wexler et al[26] reported that ACL deficient patients walk with increased knee extension angles during terminal stance when time post injury was above 7.5 years. They attributed this finding in lower magnitudes of external flexion moments in the ACL deficient patients when compared with healthy controls. Devita et al[11] reported that the sagittal knee kinematics showed excellent recovery at 6 months after ACL reconstruction and following accelerated rehabilitation. Berchuck et al[4] reported the most accepted functional adaptation in the ACL deficient patients, the so called "quadriceps avoidance gait pattern".In this adaptation, the midstance knee flexion moment is an extension moment rather a flexion moment. This phenomenon has been explained as decrease in quadriceps activity, which along with the potential quadriceps strength deficit can reduce the anterior drawer.

The above studies emphasized in their evaluation movements that occur in the sagittal plane (flexion-extension). Thus, our knowledge about tibial abduction-adduction and internal-external rotation

in normal, ACL deficient and ACL reconstructed subjects is limited[22]. The objective of our study was to examine the impact of ACL deficiency and reconstruction in knee joint kinematics during walking. A three-dimensional optoelectronic gait analysis system was utilized to analyze the lower limb kinematics during walking. We tested the hypothesis that the three-dimensional kinematics of the ACL deficient knees are changed even during low stress activities like walking. We further hypothesized that the 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 (3 females and 10 males, mean age 26 ± 5 yrs, mean weight 76 ± 7 kgr, mean height 1.75 ± 0.04 m) with ACL deficient knees and 21 patients (2 females and 19 males, ages 25 ± 4 yrs, mean weight 69.11 ± 7.89 kgr, mean height 1.73 ± 0.06 m) with ACL reconstructed knees were evaluated. Ten healthy subjects matched for age, height and weight (2 females and 8 males, mean age 24.7 ± 3.7 yrs, mean weight 62.1 ± 12.38 kgr, mean height 1.71 ± 0.05 m) without history of lower limb pathology were selected as the control group. The ACL deficient group consisted of patients with complete ACL rupture but no previous knee or lower limb pathology. The rupture was diagnosed clinically and confirmed using imaging studies as well as during arthroscopy. The operated patients underwent arthroscopically assisted ACL reconstruction using an autogenous bone-patella-tendon-bone autograft (BPTB)[13;15]. No clinical evidence of knee pain was found in these subjects and all of them had resumed their daily living activities.

The gait evaluation was performed in the ACL deficient patients 7.6 ± 4.3 weeks after the injury, while the ACL reconstructed patients were evaluated 30 ± 16.9 weeks after the operation. Kinematic data were collected using a Peak Performance real time motion analysis system with 6 infra-red light cameras (CO, USA). Data collection was performed at the sampling rate of 50 Hz and a Butterworth filter was used to decrease digitizing error with a cut-off frequency of 6 Hz. Reflective markers were placed on both

lower limbs, on the lateral side of the foot at the head of the second metatarsal, on the heel and on surface locations over the lateral malleolus, lateral side of the calf at the level of maximum calf circumference, lateral femoral condyle, lateral side of midthigh circumference, right anterior superior iliac spine (ASIS), left ASIS and sacrum according to the model developed by Vaughan et al[25]

The testing protocol was the same for all groups. The subjects were asked to walk at a self-selected pace on a 10 meters walkway. To increase the accuracy of the measurements none of the subjects knew when data were actually recorded. All gait mean variables were calculated by averaging twelve strides from six trials/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 ANOVAs were performed on the subject means on the above parameters. For tests that resulted in a significant F-ratio ($p<0.05$), post-hoc analysis was performed using the Tukey multiple comparison test. For the midthigh circumference, paired t-tests were utilized between 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 (Figure 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 to 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 ± 8.66^0 of internal rotation, while in the ACL reconstructed and control group the mean value was 0.3 ± 9.9^0 of external rotation and 3.6 ± 6.22^0 of external rotation, respectively. The tibial rotation angle during swing reached its maximum value during the initial swing phase in all groups (Figure 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 ($p<0.001$) and 1.5 ± 0.86 cm ($p<0.001$) for the ACL reconstructed and the ACL deficient group.

Discussion

The objective of this study was to examine the impact of ACL deficiency and ACL reconstruction in the knee joint three-dimensional kinematics during walking. For the data collection we utilized an optoelectronic gait analysis system, which provides data for the three-dimensional positions of body segments. The system allows assessing the kinematics of the ACL deficient knee and the effectiveness of the 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[11;23] have reported significant decrease in flexion during the terminal stance phase, in our study the differences in terminal knee flexion did not reach significance. Berchuck et al[4] have indicated that ACL deficient patients tended to walk with a reduced knee flexion angle during the stance phase of gait. Therefore, it is possible that only during the late post-injury time the terminal knee flexion angle is significantly reduced[26]. It is possible that as the nervous system adapts to the injury, the knee flexion angles can decrease resulting in lower demand on the quadriceps. Arms et al[3] reported that simulated isometric quadriceps 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 tibia translation in ACL deficient knees. Nevertheless, having the knee in a more extended position may not allow the hamstrings to prevent abnormal anterior tibial translation and rotation[8]. Thus, a more extended knee places the structure in an unstable position and possibly forces a neuromuscular decision in preference of activation between rectus femoris and hamstrings.

The lack of significant findings regarding knee flexion extension indicates that examining isolated the sagittal kinematics is not comprehensive in evaluating all the neuromuscular adaptations that occur during walking after ACL rupture and reconstruction. This is in agreement with other studies that have also found no significant differences in knee flexion extension[1;5;20;24].

Regarding tibial rotation, in all groups the tibia reached its maximum degree of rotation during the initial swing phase of the gait cycle. Large standard deviations for the tibial rotation were found in all groups, which probably reflect between subject variability in knee mobility. This is probably due to the variable degree of laxity of the ligaments and the different anatomical 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 to the maximum tibial rotation angle of the ACL reconstructed and control groups. In the control group, the mean rotation of the tibia was external during the initial swing phase, while in the ACL deficient subjects this rotation was internal.

On ACL deficient rehabilitated patients, Ciccicoti et al[6] reported a significant increase in rectus femoris muscle activity. This increased activity, which probably results in increased internal tibial rotation because normally and during knee extension, the extension moment produced by activation of the quadriceps results in tibial anterior translation and internal rotation. ANASTASH, auth thv protash dev thv katalabaivw. Eite kave thv duo protaseis, eite 3avayrapse thv yiati ta agglika edw eivai akatalabistika. Increased tibial anterior translation and internal rotation result in a pivoting phenomenon. The increased combined motion of tibia anterior translation and internal rotation in ACL deficient knees has also been reported by Grood et al [19]. Furthermore and in the present study, increased tibial internal rotation reached a significant level only during swing. This is possibly due to the hamstrings inactivity and the increased activity in rectus femoris and gastrocnemius musculature in late stance and early swing period[12;17]. Andriacchi et al[2] has also reported increased tibial internal rotation in ACL deficient knees during the entire gait cycle.

However, the authors cannot be sure that these gait adaptations are long or short-term anomalies. If this adaptation is also a long-term anomaly then it is likely that these repeated episodes of rotational instability increase the risk of further knee pathology. Wexler et al[26] examined how patients compensate for loss of anterior cruciate ligament function during time. They supported the hypothesis that there is a subconscious reprogramming of the locomotive process that protects the knee from excessive anterior

translation of the tibia. The changes in gait showed that the reprogramming process is adaptable. They also hypothesized that these changes may develop as the secondary restraints (medial meniscus) to anterior translation of the proximal tibia begin to stretch out. In addition, it has been proposed that adaptations that occur in ACL deficient patients are the result of earlier experiences following the loss of the ACL[1].If so, then the increased internal rotation found during gait in the presentACL deficient group can be considered as repeated episodes of instability, which occur during the early post-injury period, 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 group. Given the similar pattern of tibial rotation between the ACL reconstructed and 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 the knee kinematics to the normal levels found in the intact knee[21]. Thus, it is possible that the ACL reconstruction can contribute beneficially to the protection of the knee from future degenerative changes such as damage to the menisci[21] even in patients with a low level of activities due to protection against repeated episodes of rotational instability. However, this study can not provide any evidence that ACL deficient patients with a low level of activity who do not sustain re-injury events have greater risk of degenerative changes that those who undergo an ACL reconstruction.

A significant decrease of midthigh circumference ($p < 0.001$) at 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 3-12 weeks before they were studied. Furthermore, these differences could be related to the abnormal tibial rotation seen in the ACL deficient patients.

Czerniecki et al[9] have investigated the rotational stabilizing effect of the knee musculature during walking and jogging. They have shown that there was not a significant correlation during walking between the extent of tibial rotation (walking speed 84 m/min -1.4 m/sec) and the isometric or

isokinetic strength of the quadriceps and hamstrings. However, this correlation was significant at higher ambulation speeds (slow jogging pace - ambulation speed 132m/min or 2.2m/sec and moderate jogging pace - ambulation speed 156 m/min or 2.6m/sec). In our study the mean ambulation speed was below the mean walking speed (1.4 m/sec) reported by Czerniecki et al[9] (Table 1). Probably, during higher demand activities (like jogging and running) the abnormal pattern of tibial rotation found in the present ACL deficient patients may no longer exist. According to Czerniecki et al., this may be due to the fact that the knee rotational stabilizing effect of the quadriceps and hamstrings is significant[9]. However, this issue needs further investigation.

In summary, the present study indicated that examining the knee joint function only in the sagittal plane is not comprehensive in evaluating the complexity of the ACL biomechanics during walking. Furthermore, this study demonstrates that the ACL deficient patients have a tendency to rotate the tibia internally during the initial swing phase, while the ACL reconstructed and normal subjects rotate the tibia externally. Nevertheless, further studies are needed to evaluate the patterns of tibial rotation during activities that include greater rotational forces applied on the ACL deficient and ACL reconstructed knees, as well as to prospectively assess the effect of strengthening programs during rehabilitation in both deficient and ACL reconstructed individuals. In addition, future studies should delineate if the repeated episodes of knee rotational instability found in our study, along with other re-injury events that occur during daily living activities, contribute to the reprogramming of the locomotor process so that protective adaptations like the "quadriceps avoidance" gait pattern may develop.

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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.
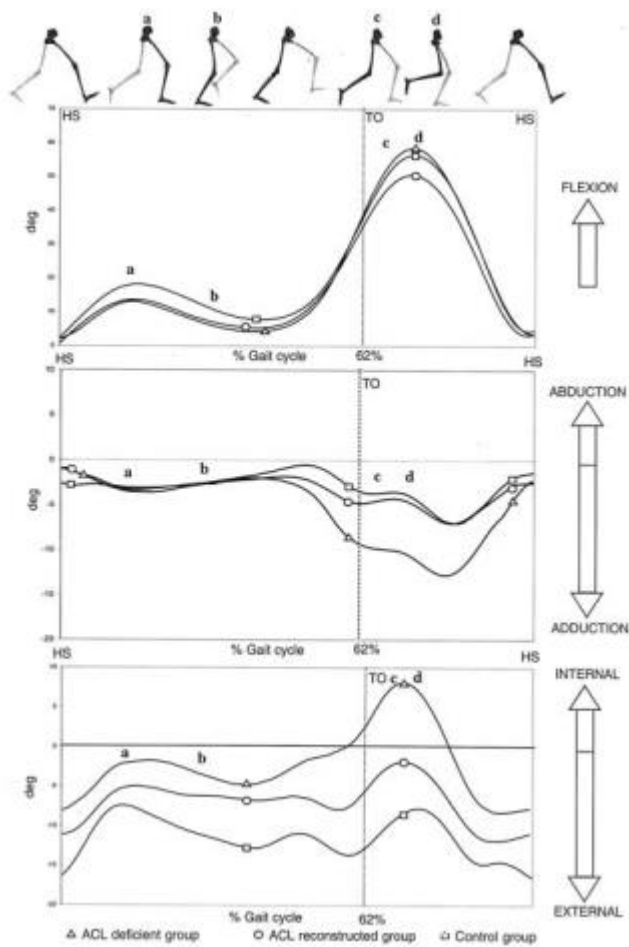
Table 1. Group means and standard deviations for all dependent variables examined.

Dependent variables (mean ± SD)	ACL deficient (n=13)	ACL reconstructed (n=21)	Control (n=10)	p value
knee flexion at toe off (^{0})	32.65 ± 3.33	34.12 ± 3.68	33.78 ± 2.87	p>0.05
max knee flexion during swing(^{0})	59.08 ± 7.71	58.54 ± 3.94	57.89 ± 3.59	p>0.05
knee flexion at heel strike(^{0})	3.41 ± 4.72	1.23 ± 6.33	2.3 ± 3.3	p>0.05
max knee flexion during loading response(^{0})	15.64 ± 5.99	13.41 ± 9.97	14.52 ± 5.81	p>0.05
max tibial abd(+)-add(-) during gait cycle(^{0})	2.12 ± 6.54	1.72 ± 4.10	0.99 ± 3.09	p>0.05
max tibial rot. (+)int, (-)ext during gait cycle(^{0})	9.6 ± 8.66	-0.3 ± 9.9*	-3.6 ± 6.22**	p<0.05
Cadence (steps/min)	109.18 ± 6.49	109.71 ± 7.01	109.32 ± 5.67	p>0.05
Averaged gait velocity (m/sec)	1.31 ± 0.33	1.28 ± 0.19	1.18 ± 0.2	p>0.05

Significant differences (* p=0.008, **p=0.003)