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In vivo biomechanics of cruciate ligament injuries

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CHAPTER

The effect of anterior cruciate ligament deficiency and reconstruction on the patellofemoral joint.

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ABSTRACT

Background: Little is known about the impact of anterior cruciate ligament deficiency and reconstruction on the patellofemoral joint.

Hypothesis: Anterior cruciate ligament deficiency changes the patellofemoral joint biomechanics. Reconstruction of the ligament does not restore the altered patellofemoral joint function.

Study Design: Controlled laboratory study.

Methods: Eight patients with an acute anterior cruciate ligament injury in 1 knee and the contralateral side intact were included in the study. Magnetic resonance and dual-orthogonal fluoroscopic imaging techniques were used to compare the patellofemoral joint function during a single-leg lunge between the intact, the anterior cruciate ligament-injured, and the anterior cruciate ligament-reconstructed knee. Data on the patellar tendon apparent elongation and orientation, patellar tracking, and patellofemoral cartilage contact location were collected preoperatively and at 6 months after reconstruction.

Results: Anterior cruciate ligament deficiency caused a significant apparent elongation and change in orientation of the patellar tendon. It decreased the flexion and increased the valgus rotation and tilt of the patella. Anterior cruciate ligament injury caused a proximal and lateral shift in patellofemoral cartilage contact location. Anterior cruciate ligament reconstruction reduced the abnormal apparent elongation but not the orientation of the patellar tendon, and it restored the patellar flexion and proximal shift in contact. The abnormal patellar rotation, tilt, and lateral shift in cartilage contact persisted after reconstruction.

Conclusion: The altered function of the patellar tendon in anterior cruciate ligament deficiency resulted in an altered patellar tracking and patellofemoral cartilage contact. Persistent changes in patellofemoral joint function after anterior cruciate ligament reconstruction imply that reconstruction of the anterior cruciate ligament does not restore the normal function of the patellofemoral joint.

Clinical Relevance: The abnormal kinematics of the patellofemoral joint might predispose the patellofemoral cartilage to degenerative changes associated with anterior cruciate ligament deficiency, even if the ligament is reconstructed in a way that restores anteroposterior knee laxity.

INTRODUCTION

Patients with ACL deficiency often develop alterations in quadriceps muscle performance with weakness and atrophy^{5,7,38,41} and degeneration of the patellofemoral joint (PFJ) cartilage.³² In patients who have undergone ACL reconstruction with a bone–patellar tendon–bone (BPTB) graft, some of the most prevalent complications are persistent patellar irritability,³⁶ patellofemoral pain,^{1,4,10,15-17,30,36} and quadriceps weakness.^{15,36} Yet little research has focused on the PFJ in ACL deficiency, and there is even less on the PFJ after ACL reconstruction. Hsieh et al^{20,21} hypothesized that patellofemoral kinematics and contact characteristics are different in the ACL-deficient knee, causing patellofemoral problems. Excision of the ACL in cadaveric knees resulted in an increased lateral shift and tilt of the patella²⁰ and decreased patellofemoral contact area and pressure.²¹ Reconstruction of the ACL restored the abnormal biomechanics to normal levels in both in vitro studies.

To our knowledge, no data have been reported on the PFJ function in ACL deficiency under in vivo weightbearing conditions. Furthermore, the effect of ACL reconstruction on the patellofemoral function under weightbearing conditions is unknown.

The objective of this study was to investigate the effects of ACL deficiency and reconstruction on the 3-dimensional (3D) behavior of the patellar tendon, the patellofemoral kinematics, and the contact characteristics of the PFJ during an in vivo weightbearing activity. In this study, we hypothesized that ACL deficiency changes the 3D behavior of the patellar tendon, the patellar tracking, and subsequently the cartilage contact location of the PFJ. Furthermore, we hypothesized that contemporary ACL reconstruction does not restore the normal PFJ function, although anteroposterior laxity of the knee under anterior tibial loads is restored.

MATERIALS AND METHODS

Subject Recruitment and Exclusion Criteria

Eight patients (6 men and 2 women; age range, 19-38 years) were included in the study.^{12,25,34,39} The included patients were diagnosed with an acute, isolated ACL injury, documented by clinical examination (8-mm Lachman test with no end point and a grade 2 pivot-shift test measured by the same orthopaedic surgeon) and MRI and had no injuries to the contralateral knee. Four patients had an injury of the left knee, and 4 had an injury of the right knee. Subjects had been injured within a mean 4.5 ± 3 months of testing. Injury to other ligaments, distinguishable cartilage lesions, and injury to the underlying bone were reasons for exclusion from the study. However, patients with minimal meniscal injury were allowed in this study because patients with an isolated ACL injury and absolutely no damage to the meniscus are relatively rare, and it is difficult to precisely quantify the extent to which the meniscus is damaged without arthroscopic examination. However, the status

of the meniscus was documented during subsequent arthroscopic reconstruction of the ACL. If removal of more than 50% of the medial or lateral meniscus was required during reconstruction, the patient was excluded. Three patients had no significant damage to the meniscus, 1 patient had a partial-thickness tear of the lateral meniscus, and the remaining 4 patients had injuries requiring partial removal of the lateral meniscus (10%, 15%, 30%, and 40% removal of the lateral meniscus).^{12,25,34,39}

The purpose of the present study was explained in detail to all of the subjects at the time of recruitment. Each subject signed a consent form that had been approved by our institutional review board.

The ACL Reconstruction Technique

The subjects underwent arthroscopic surgical reconstruction of the ACL of the injured knee. All surgeries were performed by 1 orthopaedic surgeon. A diagnostic arthroscopy was performed before graft placement. During surgery, the status of each patient's menisci was documented. Reconstruction was performed in a standard fashion using a central 10-mm BPTB autograft. A 10-mm tibial tunnel was drilled using a 55° guide (Linvatec-Conmed, Largo, Fla) centered at a point 7 mm anterior to the PCL on the downslope of the medial tibial spine. A 10-mm femoral tunnel was drilled using a 6-mm femoral offset guide (Arthrex, Naples, Fla) centered at the 10:30 position for right knees (1:30 for left). The graft was passed in retrograde fashion, and the femoral and tibial bone blocks were secured with titanium interference screws (Guardsman, Linvatec-Conmed). The femoral screw length was 25 mm and was placed with the knee in maximal flexion. The tibial screw length was 30 mm. The graft was fully tensioned with the knee in full extension. Screw diameter was determined based on graft-tunnel fit. Examination confirmed that there was no notch impingement, and cycling of the knee revealed less than 2 mm of graft motion in all cases. The anterior laxity of the reconstructed knee as measured with the KT-1000 arthrometer was similar to that of the intact contralateral knee.³⁴

The MRI Scan and 3D Knee Model

Before the ACL reconstruction, both knees were imaged with a magnetic resonance (MR) scanner using a 1.5-T magnet (General Electric, Waukesha, Wis) and a fat-suppressed 3D spoiled gradient-recalled sequence.^{13,24} The patients were lying horizontally with the knee in a relaxed, extended position. The MR scans spanned the medial and lateral extremes of the knee and were used to generate parallel sagittal plane images (resolution 512 × 512 pixels) with a field of view of 16 × 16 cm and a spacing of 1 mm. For each knee, the MR scanning time was approximately 12 minutes. Approximately 120 sagittal plane images were obtained for each knee. The MRIs were used to create 3D models of the knees in a solid modeling software (Rhinoceros, Robert McNeel and Associates, Seattle, Wash) using a protocol established in our laboratory.^{13,23} The contours of the bone and cartilage surfaces of the femur, tibia, and patella were digitized within each image. The digitized spatial data

(x, y, and z coordinates) were then linked using B-spline curves to reproduce the contours of the femur, tibia, and patella. Bone and cartilage surfaces of the femur, tibia, and patella were created from the contours with the use of nonuniform rational B-splines. In addition to the bone and cartilage surface contours, the attachment areas of the patellar tendon on the tibial tubercle and the apex of the patella were delineated.

Dual-Orthogonal Fluoroscopic Imaging of the Knee During a Weightbearing Activity

After the MRI-based computer models were constructed, both knees of each subject were simultaneously imaged using 2 orthogonally placed fluoroscopes as the patient performed a single-leg quasi-static lunge at 0°, 15°, 30°, 60°, and 90° of flexion. Flexion angle of the knee was monitored using a handheld goniometer. The subject kept the knee stable for 1 second at each target flexion angle, so that the fluoroscopes captured the knee position, and then flexed the knee to the next target position. At each selected flexion angle, the subject supported his or her body weight on the leg being scanned, while the other leg was used to help balance the body. Data were collected preoperatively and at 6 months after single-bundle ACL reconstruction. These images were used to quantify the in vivo knee position at each of the targeted flexion angles.

Measurement of In Vivo 6 Degrees of Freedom Knee Kinematics Using Image-Matching Technique²⁸

The orthogonal images were imported into a solid modeling software and placed in the orthogonal planes based on the position of the fluoroscopes during the imaging of the patient. The contours of the femur, tibia, and patella were outlined on each fluoroscopic image. The 3D MRI-based model of the patient was then imported into the software and viewed from the 2 orthogonal directions corresponding to the orthogonal fluoroscopic setup used to acquire the images. The models were independently manipulated in 6 degrees of freedom inside the software until the projections of the models matched the outlines of the images. When the projections matched the outlines of the images taken during in vivo knee

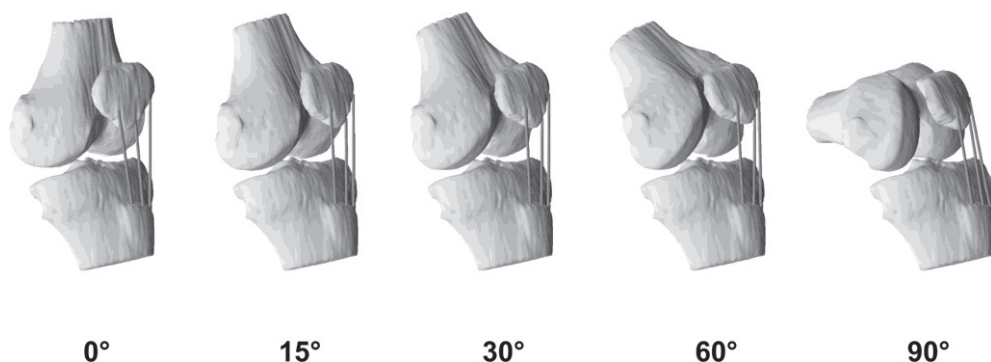


Figure 1. The knee models for a typical subject at 0°, 15°, 30°, 60°, and 90° of flexion.

flexion, the model reproduced the in vivo position of the knee. A series of knee models that reproduce knee positions at all target flexion angles re-created the in vivo knee flexion from full extension to 90° of flexion (Figure 1).

Magnetic resonance and dual-orthogonal fluoroscopic imaging techniques have been described in detail in previous publications. This system has an accuracy of less than 0.1 mm in measuring tibiofemoral joint kinematics.^{12,28} The procedure was further validated for measuring the patellofemoral kinematics.³³ The methodology has an error of less than 0.1 ± 0.2 mm in measuring patellar shift and $0.2^\circ \pm 0.1^\circ$ in patellar tilt.³³

Description of the Biomechanical Function of the Patellar Tendon

The kinematics of the patellar tendon were measured from the series of bone models representing the kinematics of the knee.¹¹ The attachment sites of the patellar tendon on the patella and tibial tubercle were divided into thirds: a medial portion, a central portion, and a lateral portion. The apparent elongation of each portion of the patellar tendon was defined as the length of the line connecting the attachment sites on the patellar apex and tibial tubercle. The sagittal plane angle was defined as the angle formed between the long axis of the tibia and the projection of the patellar tendon on the sagittal plane of the tibia (Figure

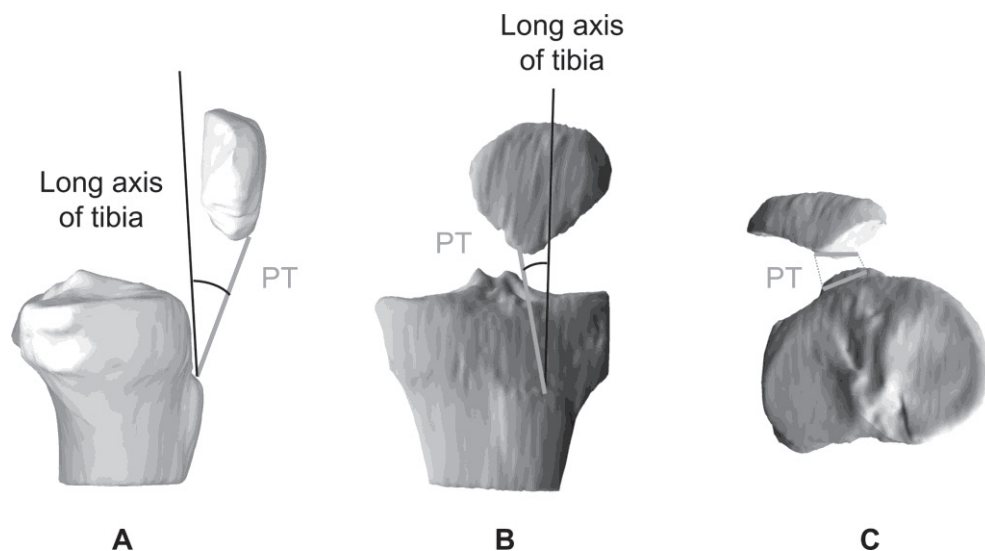


Figure 2. The sagittal plane angle (A), measured in the sagittal plane between the patellar tendon (PT) and the long axis of the tibia; the coronal plane angle (B), measured in the coronal plane between the PT and the long axis of the tibia; and twist (C), measured in the transversal plane between the patellar and tibial attachment of the PT.

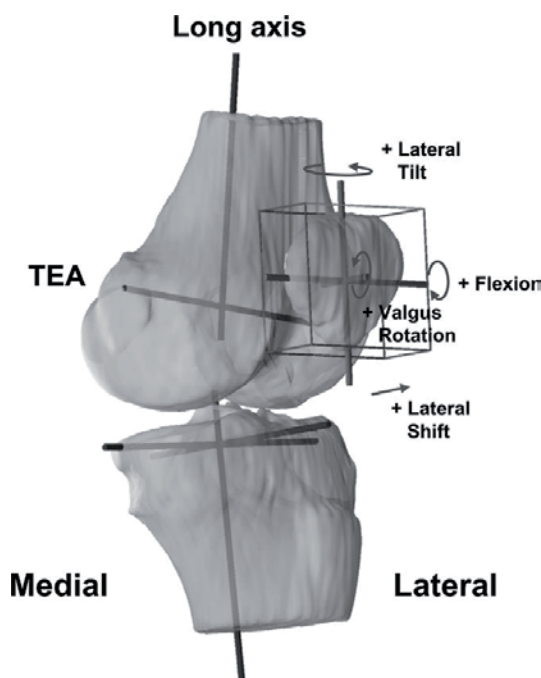


Figure 3. Coordinate systems used to quantify the patella tracking. The femoral coordinate system consisted of the transepicondylar axis (TEA) and the long axis intersecting at the center of the knee joint (midpoint of TEA). A cuboid was enclosed around the patella to determine the patellar center. The patellar coordinate system consisted of the proximodistal, anteroposterior, and mediolateral axes. Patellar flexion, shift, tilt, and rotation are considered positive as shown in the figure.

2A). A positive sagittal plane angle corresponded to an anterior orientation of the patellar tendon (patellar attachment anterior to the tibial attachment) relative to the long axis of the tibia, and negative values correspond to a posterior orientation. The coronal plane angle was defined as the angle between the long axis of the tibia and the projection of the patellar tendon on the coronal plane of the tibia (Figure 2B). Positive coronal plane angles corresponded to a medial orientation of the patellar tendon (patellar attachment medial to the tibial attachment) relative to the long axis of the tibia, whereas negative values corresponded to a lateral orientation. In this fashion, the kinematics of the patellar tendon were quantified for each subject as a function of flexion. Twist of the patellar tendon was defined as the angle measured in the transversal plane between the patellar and tibial attachment sites (Figure 2C).

Description of Patellar Tracking

After reproducing the *in vivo* knee positions along the flexion path, the patellar tracking was measured from the series of knee models.³³ A joint coordinate system¹⁹ was established for each knee to describe the motion of the patella. Two axes were drawn on the femur: the long axis along the posterior femoral shaft surface in sagittal plane and the transepicondylar axis (TEA) connecting the epicondyle extremes of the medial and lateral femoral condyles.³¹ The knee center was defined as the midpoint of the TEA. An axis parallel to the posterior wall of the tibial shaft was defined as the long axis of the tibia. The flexion angle of the knee was defined as the angle between the long axes of the femur and tibia in the

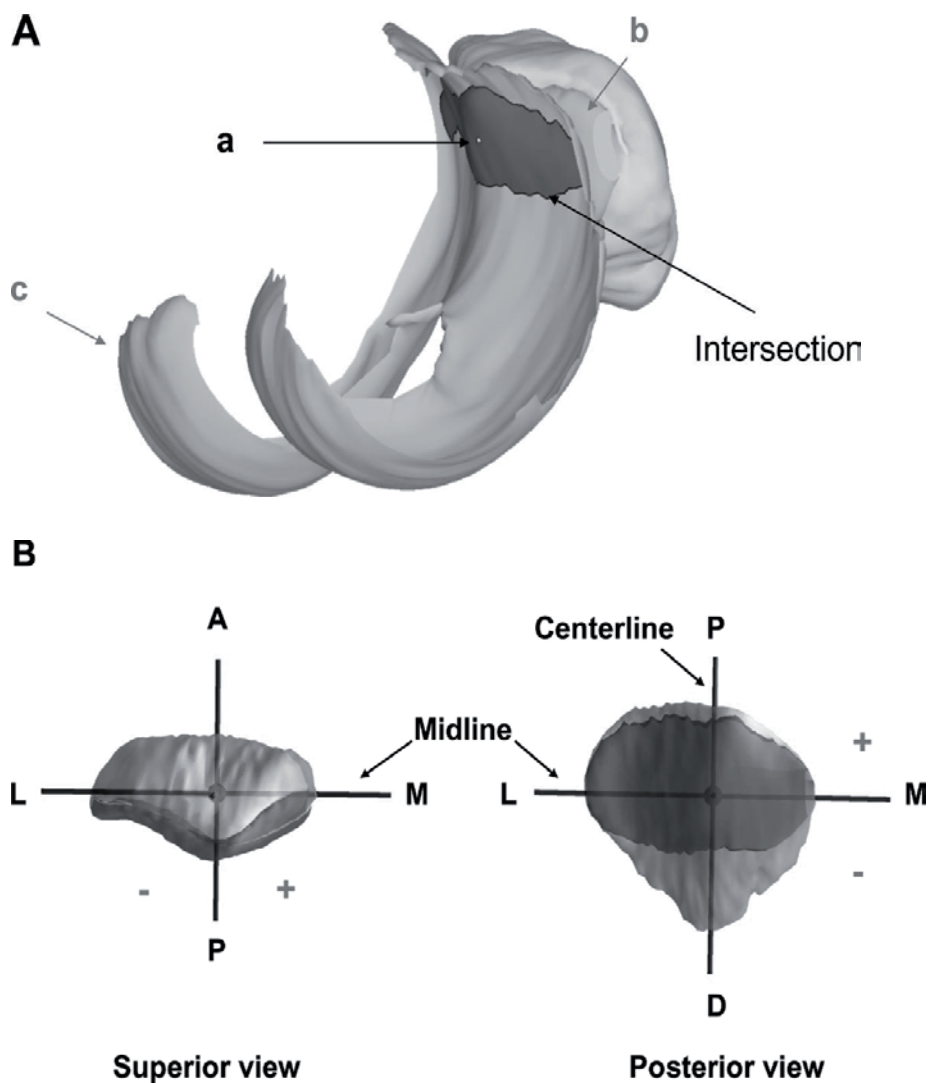


Figure 4. **A**, the centroid (a) of the intersection of the patellar (b) and femoral (c) cartilage was used to determine the patellofemoral contact locations. **B**, the coordinate system on the patellar cartilage surface for patellofemoral cartilage contact analysis. The proximal (P)–distal (D) axis was called the centerline. The medial (M)–lateral (L) axis was called the midline. Contact proximal to the midline and medial to the centerline was positive. A, anterior.

sagittal plane. To reduce the variability in creating patellar coordinate systems, a cuboid was used to enclose the patella so that it touched the proximodistal, anteroposterior, and mediolateral borders of the patella.^{26,33} The center of the cuboid was defined as the origin of the patella. The long axis of the patella was defined as the line along the superior-inferior direction.

Patellar flexion was defined as the rotation of the patella about the TEA of the femur (Figure 3).⁸ Patellar shift was defined as the medial or lateral movement of the center of the patella along the TEA of the femur (Figure 3). A positive shift corresponded to the lateral movement of the patellar center with respect to the knee center along the TEA of the femur. Patellar tilt was defined as the rotation of the patella about its long axis, where lateral tilt followed the direction of external femoral rotation (Figure 3). Patellar rotation is the rotation of the patella about the anteroposterior axis of the patella, where valgus rotation follows the direction of valgus rotation in tibiofemoral motion (Figure 3), that is, an outward angulation of the distal segment of the patella. In this fashion, the patellar tracking was quantified for each subject as a function of flexion of the knee.

Description of Patellofemoral Cartilage Contact Points

The contact points on the patellar cartilage were calculated by finding the centroid of the intersection of the patellar and femoral cartilage layers.^{13,22,25} From the series of models used to reproduce knee motion, the relative positions of the cartilage layers on the femur and patella were determined. The overlap of the 2 cartilage layers was used to approximate the cartilage contact area (Figure 4A). The solid modeling software automatically outlined the intersection of the patellar and femoral cartilage layers and calculated the centroid of the enclosed area. The centroid of this contact area was defined as the contact point. To describe the motion of the cartilage contact points, a coordinate system was created on the surface of the patella (Figure 4B). The center of the vertical ridge of the patella was the origin of the coordinate system. In this coordinate system, the proximodistal axis was called the centerline, and the mediolateral axis was called the midline. In the proximodistal direction, the contact point was positive if it was proximal to the midline and negative if it was distal to the midline. In the mediolateral direction, a contact point was positive if it was on the medial side of the centerline and negative if it was on the lateral side of the centerline.

Statistical Methods

A 1-way repeated-measures analysis of variance was used to compare the biomechanical function of the patellar tendon (apparent elongation, sagittal plane angle, coronal plane angle, and twist), the patellar tracking (patellar flexion, shift, tilt, and rotation), and the position of the patellofemoral contact points on the patellar cartilage of the ACL-deficient, ACL-reconstructed, and intact (contralateral) knees. The Student-Newman-Keuls post hoc

test was performed to isolate statistically significant differences between groups. The level of significance was set at $P < .05$.

RESULTS

Biomechanical Function of the Patellar Tendon

In the healthy knee, all 3 portions of the patellar tendon deformed similarly with flexion. The apparent elongation of the lateral, central, and medial portions of the patellar tendon increased from 64.9 ± 10.1 mm, 53.7 ± 6.9 mm, and 62.2 ± 7.9 mm, respectively, at 0° to 70.8 ± 8.6 mm, 57.9 ± 7.2 mm, and 66.6 ± 8.2 mm, respectively, at 30° of flexion, after which no marked change in apparent elongation was observed in the portions (Figure 5A). Anterior cruciate ligament deficiency caused an apparent elongation of all 3 portions of the patellar tendon, which was significant at most flexion angles. The effect of ACL deficiency on the apparent elongation of the patellar tendon was greatest at 0° of flexion in the lateral portion; an increase of 8.1% was noticed, compared to the healthy knee ($P < .05$). Anterior cruciate ligament reconstruction decreased the abnormal apparent elongation of the patellar tendon in all 3 portions to levels not significantly different from those of the intact knee.

In the sagittal plane (Figure 5B), the angle between the patellar tendon and the long axis of the tibia decreased with flexion from $21.8^\circ \pm 4.7^\circ$, $25.1^\circ \pm 5.6^\circ$, and $23.4^\circ \pm 4.8^\circ$ at 0° of flexion to $-4.7^\circ \pm 4.0^\circ$, $-2.3^\circ \pm 4.0^\circ$, and $-0.5^\circ \pm 3.4^\circ$ at 90° of flexion in the lateral, central, and medial portions, respectively, of the healthy patellar tendon. In the flexion range from 0° to 30° , ACL deficiency decreased the sagittal plane angle by approximately 3° . The greatest decrease in sagittal plane angle occurred at 0° in the central portion of the patellar tendon: from $25.1^\circ \pm 5.6^\circ$ to $20.8^\circ \pm 5.1^\circ$. At 0° of knee flexion, ACL reconstruction did not significantly change the sagittal plane angle, compared to the ACL-deficient knee. In the lateral and central portions, the sagittal plane angle remained significantly decreased in the ACL-reconstructed knee compared with the intact knee at 0° of knee flexion ($P < .05$). The ACL reconstruction reduced the abnormal sagittal plane angles in all the patellar tendon portions at 15° and 30° of knee flexion to levels not significantly different from those of the intact knee. At 60° of knee flexion, ACL reconstruction significantly increased the sagittal plane angle in the medial and central portion compared to the healthy knee; at 90° of knee flexion, ACL reconstruction significantly increased the sagittal plane angle in all the patellar tendon portions.

In the coronal plane (Figure 5C), the 3 portions of the healthy patellar tendon exhibited a similar trend of gradually decreasing coronal plane angles with flexion: from $9.0^\circ \pm 1.8^\circ$, $13.2^\circ \pm 3.4^\circ$, and $16.0^\circ \pm 4.6^\circ$ at 0° of flexion to $1.3^\circ \pm 2.8^\circ$, $2.3^\circ \pm 3.1^\circ$, and $5.0^\circ \pm 3.5^\circ$ at 90° of flexion in the lateral, central, and medial portions, respectively. In the ACL-deficient knee, the patellar tendon showed a similar trend of a gradually decreasing coronal plane

angle with flexion, but the coronal plane angle was less deviated to the medial side compared to the healthy knee, with a maximal difference of 7.6° at 0° of flexion in the central portion of the patellar tendon. The coronal plane angle in the ACL-deficient knee decreased from $3.0^\circ \pm 3.2^\circ$, $5.6^\circ \pm 6.4^\circ$, and $9.1^\circ \pm 5.4^\circ$ at 0° of flexion to $-3.0^\circ \pm 4.0^\circ$, $-1.6^\circ \pm 4.2^\circ$, and $2.6^\circ \pm 4.5^\circ$ at 90° of flexion in the lateral, central, and medial portions, respectively. The decrease in the coronal plane angle after ACL deficiency was significant at all angles and in all 3 portions of the patellar tendon, except at 90° of knee flexion in the central portion and between 30° and 90° in the medial portion. The ACL reconstruction was unable to restore the normal coronal plane angle. The coronal plane angle in the ACL-reconstructed knee varied from $-0.4^\circ \pm 4.8^\circ$, $5.4^\circ \pm 5.8^\circ$, and $10.4^\circ \pm 4.9^\circ$ at 0° of flexion to $-5.8^\circ \pm 4.2^\circ$, $-2.6^\circ \pm 4.9^\circ$, and $2.4^\circ \pm 4.1^\circ$ at 90° of flexion in the lateral, central, and medial portions, respectively.

The healthy patellar tendon demonstrated a gradually increasing external rotation of its patellar attachment site relative to the tibial attachment site with flexion (Figure 5D): from $0.6^\circ \pm 4.7^\circ$ at 0° of flexion to $-8.6^\circ \pm 6.5^\circ$ at 90° of flexion. In the ACL-deficient knee, the patellar attachment site was about 3° more externally twisted relative to the tibial attachment site compared to the healthy knee: from $-3.8^\circ \pm 6.4^\circ$ at 0° of flexion to $-11.6^\circ \pm 7.9^\circ$ at 90° of flexion. The ACL reconstruction did not significantly reduce the increased external twist of the patellar tendon that was observed in the ACL-deficient knee: from $-4.5^\circ \pm 4.5^\circ$ at 0° of flexion to $-13.5^\circ \pm 8.3^\circ$ at 90° of flexion.

Patellar Tracking

Anterior cruciate ligament injury significantly decreased the flexion angle between the patella and femur at 0° and 15° of flexion (Figure 6A) ($P < .05$). In the healthy knee, the patellar flexion angle measured $9.7^\circ \pm 5.6^\circ$ at 0° of knee flexion and increased to $16.2^\circ \pm 6.9^\circ$ at 15° of knee flexion. In ACL-deficient knees, patellar flexion at 0° was $3.2^\circ \pm 4.4^\circ$ and increased to $9.1^\circ \pm 3.2^\circ$ at 15° of knee flexion. On average, ACL deficiency reduced the patellar flexion angle by 6.6° throughout the measured range of motion. Anterior cruciate ligament reconstruction reduced the abnormal patellar flexion. At 0° of knee flexion, the patellar flexion was $7.5^\circ \pm 5.8^\circ$ and increased to $13.6^\circ \pm 5.7^\circ$ at 15° of knee flexion in ACL-reconstructed knees. On average, patellar flexion remained 3.1° less than

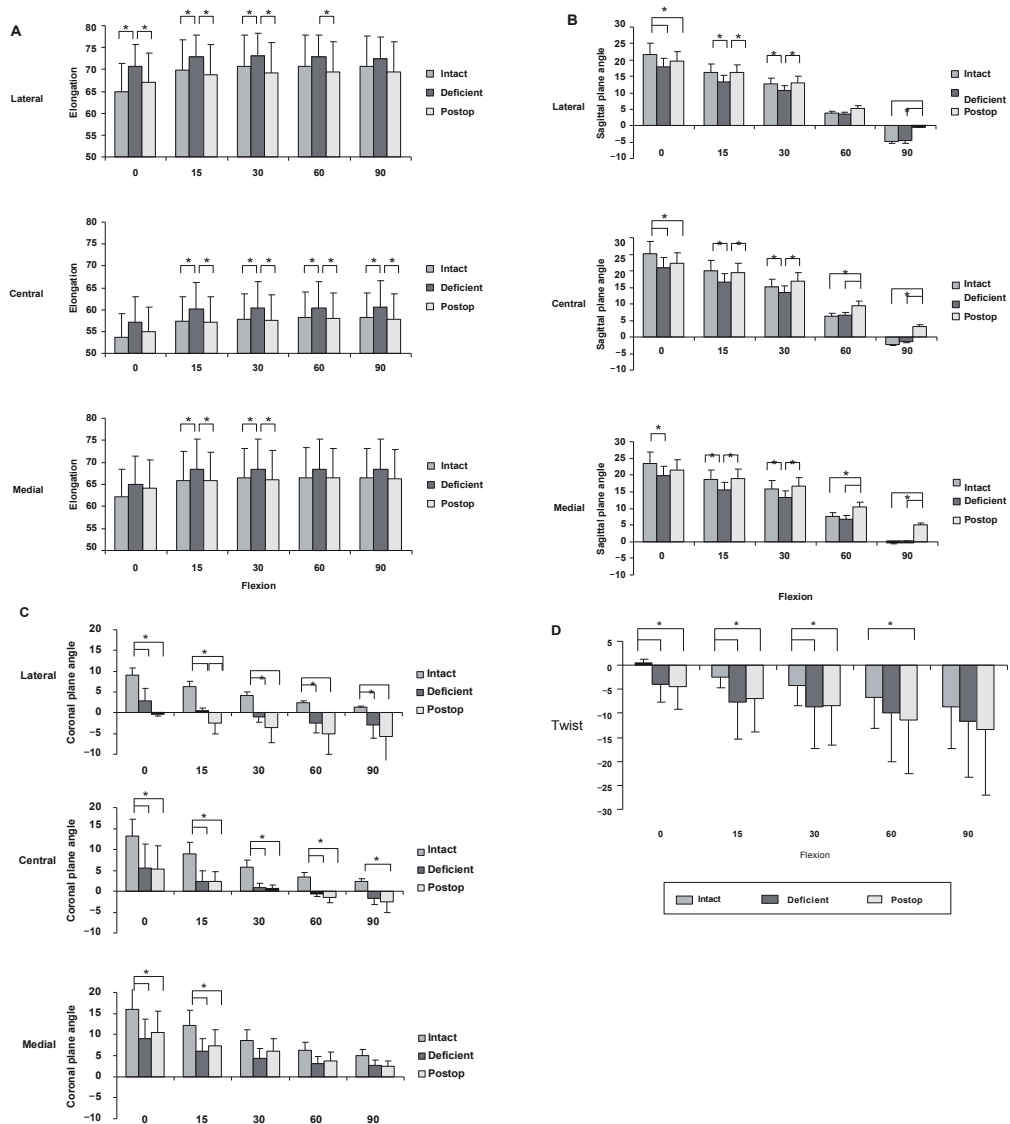


Figure 5. The biomechanical function of the patellar tendon. **A**, apparent elongation; **B**, sagittal plane angle; **C**, coronal plane angle; and **D**, twist of the patellar tendon as a function of knee flexion angle. Central, central portion of the patellar tendon; Deficient, ACL-deficient knee; Intact, intact knee; Lateral, lateral portion of the patellar tendon; Medial, medial portion of the patellar tendon; Postop, postoperative ACL-reconstructed knee. Mean $\pm$ SD; * $P < .05$.

that of the control knee; however, this residual decrease in flexion was not significantly different from the intact or ACL-deficient knee.

The patella in ACL-deficient knees shifted significantly more laterally at 0° of knee flexion, and reconstruction of the ACL did not reduce the abnormal shift (Figure 6B). At 0° of flexion, the patella was 7.2 ± 4.6 mm, 8.9 ± 4.2 mm, and 9.2 ± 3.9 mm lateral to the knee center along the TEA of the femur in the healthy, ACL-deficient, and ACL-reconstructed knees, respectively.

Anterior cruciate ligament deficiency significantly changed the patellar rotation at 15° of knee flexion, and ACL reconstruction was unable to restore the values to normal (Figure 6C). In the ACL-deficient knee, the patella was approximately 2° more valgusly rotated. Anterior cruciate ligament reconstruction was not only unable to restore the normal rotation pattern of the patella but actually further increased the valgus rotation between 0° and 30° of knee flexion.

The effect of ACL deficiency was more pronounced on the patellar tilt (Figure 6D). Between 0° and 60° of knee flexion, ACL deficiency increased the lateral tilt of the patella by nearly 5°. The maximum effect of ACL deficiency occurred at 0° of knee flexion; the lateral tilt increased from $2.2^\circ \pm 6.0^\circ$ to $7.4^\circ \pm 3.4^\circ$ after ACL deficiency. Reconstruction of the ACL did not have an effect on the abnormal tilt of the patella, as there was no significant difference in tilt detected between the ACL-deficient and reconstructed knees. At 0° of knee flexion, the patella was $7.0^\circ \pm 3.5^\circ$ tilted in the ACL-reconstructed knees.

Patellofemoral Cartilage Contact Points

We did not observe any contact between the femoral and patellar cartilage in the healthy, ACL-deficient, and ACL-reconstructed knees at 0° of knee flexion. Articular cartilage contact in the healthy knee moved from 5.3 ± 3.6 mm distal of the midline at 15° of knee flexion to 4.8 ± 4.4 mm proximal of the midline at 90° of knee flexion (Figure 7A) and was located along the vertical ridge (centerline) of the patellar surface (Figure 7B).

Rupture of the ACL caused a significant proximal and lateral shift of the cartilage contact between 15° and 90° of flexion. On average, the contact point location in the ACL-deficient knees was 4.8 mm more proximal than in the healthy knee joint (Figure 7A). The maximum effect was observed at 15° of flexion, at which ACL deficiency caused a 5.0 ± 3.9 mm proximal shift. The effect of ACL deficiency was more pronounced in the mediolateral direction (Figure 7B). A lateral shift of 6.5 ± 2.4 mm minimum (at 15° of flexion) to 7.2 ± 2.9 mm maximum (at 60° of flexion) was measured along the midline.

Reconstruction of the ACL reduced the abnormal contact location in the proximodistal direction, but a significant proximal shift remained compared with the intact knee, except at 90° (Figure 7A). Between 15° and 60° of knee flexion, a significant residual 2.5-mm

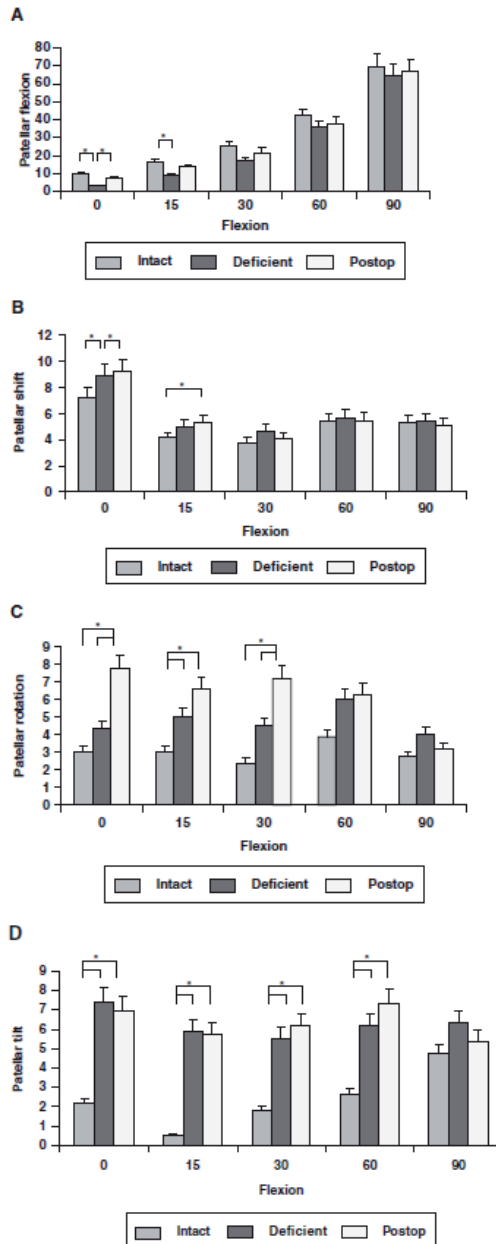


Figure 6. Patellar tracking. **A**, flexion; **B**, shift; **C**, rotation; and **D**, tilt of the patella as a function of knee flexion angle. Deficient, ACL-deficient knee; Intact, intact knee; Postop, postoperative ACL-reconstructed knee. Mean $\pm$ SD; * $P < .05$.

proximal shift remained after ACL reconstruction. In the mediolateral direction, ACL reconstruction did not restore normal contact kinematics (Figure 7B). A lateral shift of minimum 6.5 ± 2.2 mm (at 15° of flexion) to maximum 8.1 ± 1.9 mm (at 30° of flexion) persisted along the midline.

DISCUSSION

We used a combined MR and dual-orthogonal fluoroscopic imaging technique to compare the 3D behavior of the patellar tendon, the patellofemoral kinematics, and the contact characteristics of the PFJ between the intact, ACL- injured, and ACL-reconstructed knees. Data were collected preoperatively and at 6 months after single-bundle ACL reconstruction with a BPTB autograft. The anterior laxity of the reconstructed knees as measured with the

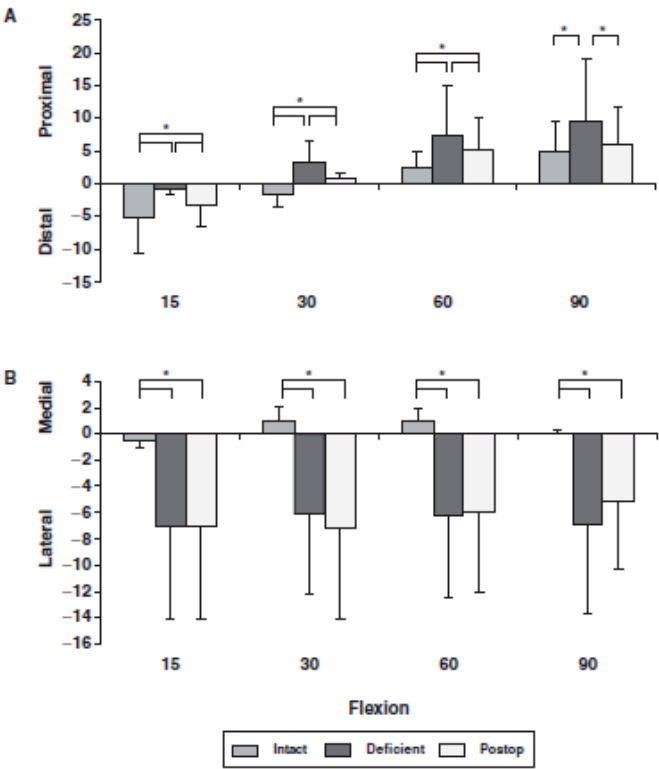


Figure 7. Patellofemoral cartilage contact points. Cartilage contact in the (A) proximodistal and (B) mediolateral direction as a function of knee flexion angle. Deficient, ACL-deficient knee; Intact, intact knee; Postop, postoperative ACL-reconstructed knee. Mean $\pm$ SD; $*P < .05$.

KT-1000 arthrometer was similar to that of the intact contralateral knee.

Anterior cruciate ligament injury caused an increase in the apparent elongation of the patellar tendon. The increased apparent elongation of the patellar tendon in ACL-deficient knees could be responsible for the quadriceps muscle weakness associated with ACL injury. The patellar tendon length was found to influence considerably the mechanical behavior of the patellar articulation.⁴⁰ An increased length of the patellar tendon, an essential element of the knee extensor mechanism, implies an increase in quadriceps slack length, thus decreasing the quadriceps mechanical advantage.¹⁴

We found that ACL injury caused a decreased sagittal and coronal plane angle and an increased external twist. This altered orientation of the patellar tendon is understandable because the PFJ is not an isolated unit in the knee. Instead, a kinematic coupling exists between the patellofemoral and the tibiofemoral articulations, connected by the patellar tendon.²⁶ In the tibiofemoral joint of ACL-deficient patients, an increased anterior translation and internal rotation of the tibia^{2,18,29,35} as well as an increased medial tibial translation¹² are observed. The increased anteroposterior tibial translation could explain the decreased sagittal plane angle, as the anterior tibial translation moves the tibial attachment of the patellar tendon forward, bringing the patellar tendon more parallel to the tibial shaft in the sagittal plane. Similarly, an increased medial tibial translation in ACL deficiency¹² will move the tibial attachment of the patellar tendon more medial relative to its patellar attachment, effectively reducing the angle between the patellar tendon and the tibia in the coronal plane. Finally, the increased internal rotation of the tibia that is observed in ACL deficiency could explain the increased twist of the patella that we found.

The abnormal orientation of the patellar tendon after ACL injury implies an alteration in patellar tracking, as the patellar tendon links the tibiofemoral joint and the PFJ. This investigation has demonstrated that ACL injury decreased the flexion and increased the valgus rotation and lateral tilt of the patella. Furthermore, the patella in ACL-deficient knees shifted significantly more laterally at 0° of knee flexion. These findings are consistent with the *in vitro* study results obtained by Hsieh et al,²⁰ in which they showed that excision of the ACL resulted in increases in lateral patellar tilt and in lateral patellar shift.²⁰ An alteration in patellar tracking after ACL injury would be expected to lead to changes in patellofemoral articular contact biomechanics.

Because of the high congruency of the PFJ, small changes in patellar tracking were expected to result in major changes in patellofemoral contact characteristics. Indeed, we found that rupture of the ACL caused a significant proximal and lateral shift of the articular cartilage contact on the patellar cartilage surface. A proximal shift in articular cartilage contact could be explained by the decreased patellar flexion. The lateral patellar shift and increased lateral tilt of the patella could be responsible for the large lateral shift in cartilage contact and may help to explain the onset of anterior knee pain in patients with ACL

deficiency. It is interesting to note that the lateral shift in cartilage contact was more pronounced than was the proximal shift. This finding can be attributed to the geometry of the femoral trochlear groove and articular surface of the patella. Study of our MRIs showed that the vertical ridge of the patella had thicker cartilage than that of the medial and lateral aspect of the patellar cartilage surface. In the healthy knee, cartilage contact occurred along this vertical ridge of the patellar cartilage surface. This is consistent with the cartilage contact characteristics of the tibiofemoral joint, in which it was found that cartilage is up to 50% thicker in regions where cartilage-to-cartilage contact is present.^{6,27} Healthy cartilage adapts to mechanical stimuli^{3,37} and ultimately becomes dependent on the maintenance of the mechanical stimulus for normal tissue function.⁹ The thicker cartilage within the cartilage-to-cartilage contact area may result in a reduced contact stress, as was demonstrated by a 3D finite element analysis suggesting that thicker cartilage bears a lower peak contact stress than does thinner cartilage under the same loading conditions.²⁴ A subtle change in patellar tracking will create a large shift in cartilage contact, moving the articular loading away from the vertical ridge toward the lateral aspect of the patellar cartilage. This abnormal loading of unconditioned PFJ chondrocytes might predispose the patellofemoral cartilage to degenerative changes associated with ACL injury. Future studies will follow up the patients to examine the correlation between cartilage degeneration and changes in PFJ contact kinematics.

Anterior cruciate ligament reconstruction reduced the abnormal apparent elongation and sagittal plane angle of the patellar tendon. Furthermore, ACL reconstruction restored the patellar flexion and proximal shift in contact. These findings suggest that there might be an improvement in anteroposterior tibiofemoral stability after reconstruction. In our previous study of tibiofemoral kinematics after ACL reconstruction that included the patients of this study,³⁴ the anterior laxity of the reconstructed knee as measured with the arthrometer was similar to that of the intact contralateral knee. Once the anteroposterior knee stability is improved, the tibial tubercle moves more posteriorly relative to the patellar apex, returning the sagittal plane angle to a level similar to that of the healthy knee. A restored sagittal plane angle will reduce the abnormal patellar flexion, as the inferior pole of the patella is no longer pulled anteriorly relative to the femur. Once the patellar flexion is normalized, the cartilage contact in proximodistal direction returns to normal.

However, the abnormal coronal plane angle and twist of the patellar tendon persisted after ACL reconstruction. As the patellar tendon links the tibiofemoral joint and the PFJ, it was not surprising to find that the persistent abnormal coronal plane angle and twist of the patellar tendon that were observed in ACL-reconstructed knees resulted in abnormal patellar rotation and tilt after the reconstruction. Consequently, the lateral shift in cartilage contact that was detected in ACL-deficient knees did not improve after ACL reconstruction. These findings demonstrate that the surgical reconstruction of the ACL does not restore the rotational stability of the knee, as the coronal plane angle and twist of the patellar tendon

are likely a function of tibiofemoral rotation. This persistent abnormal motion of the PFJ might predispose the patellofemoral cartilage to degenerative changes and may explain the onset of patellofemoral pain after reconstruction. Our observations differ somewhat from the study by Hsieh et al,²¹ which demonstrated that intra-articular reconstruction of the ACL completely returned the normal PFJ contact characteristics. The difference between the data might be owing to the differences between the in vitro and in vivo loading conditions applied in the studies. During the in vivo knee function, the quadriceps and hamstring forces may be higher and more complicated than are the simulated muscle loads.

Our study has several limitations. We measured only 1 functional activity, namely a single-leg lunge, using a goniometer to control the flexion angle. Other in vivo activities such as walking, running, and stair climbing should be considered in future studies. This study evaluated the PFJ only at 6 months after surgery. In the future, patients should be followed up at various time intervals to investigate the change in kinematics over time. This might also provide insight into the relationship between altered joint behavior and joint degeneration. In addition, this study approximated the function of the patellar tendon using 3 straight lines. However, these lines did not penetrate into the tibia, and the differences in deformations between the 3 regions were relatively small, indicating a relatively uniform deformation of the patellar tendon. In addition, the unstrained (reference) length of the patellar tendon was not known, so it is difficult to quantify the strains experienced by the patellar tendon from these data. Furthermore, this study did not measure the ground reaction force. Future studies should incorporate a load cell into the system, so that the moment applied to the joint might be estimated. The cartilage contact position was determined as the centroid of the intersection area formed by the tibial and femoral cartilage surfaces. Cartilage deformation was not considered during calculation of the contact point. We included some patients who had partial tears of the lateral meniscus. Therefore, the explanation of our data should be based on the patient condition as indicated in the Materials and Methods section. Eight patients were investigated in this study. A larger patient population should be included in future studies, so that the effects of various combined meniscal injuries and ACL reconstruction on the PFJ function can be investigated.

In conclusion, we found that the altered tibiofemoral joint kinematics associated with ACL injury changed the apparent elongation and orientation of the patellar tendon. The disturbed function of the patellar tendon corresponded to an altered patellar tracking and patellofemoral cartilage contact. Anterior cruciate ligament reconstruction reduced the abnormal apparent elongation and abnormal orientation of the patellar tendon in the sagittal plane, as well as the abnormal patellar flexion and the superior shift in cartilage contact. These findings imply that ACL reconstruction improved the PFJ function. However, the abnormal orientation in the coronal plane and twist of the patellar tendon, as well as the abnormal patellar rotation, tilt, and lateral shift in PFJ cartilage contact, demonstrate that

ACL reconstruction did not fully restore the rotational stability of the knee. This abnormal loading of the PFJ may predispose the patellofemoral cartilage to degenerative changes associated with ACL injury and might help to explain the onset of patellofemoral pain after reconstruction, even if the ACL is reconstructed in a way that restores the clinical anteroposterior stability of the knee.

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