In vivo biomechanics of cruciate ligament injuries

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CHAPTER

The effect of anterior cruciate ligament deficiency on the in vivo elongation of the medial and lateral collateral ligaments.

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ABSTRACT

Background: Although anterior cruciate ligament deficiency has been shown to lead to joint degeneration, few quantitative data have been reported on its effect on soft tissue structures surrounding the knee joint.

Hypothesis: Anterior cruciate ligament deficiency will alter the deformation of both collateral ligaments during in vivo weightbearing knee function from 0° to 90°.

Study Design: Controlled laboratory study.

Methods: Six patients who had acute anterior cruciate ligament injury in 1 knee with the contralateral side intact participated in this study. Using magnetic resonance and dual orthogonal fluoroscopic imaging techniques, we measured the length of the fiber bundles of the superficial medial collateral ligament, deep medial collateral ligament, and lateral collateral ligament of the 6 patients; the healthy contralateral knee of each patient served as a control.

Results: Anterior cruciate ligament injury caused a significant elongation of the fiber bundles of the superficial and deep medial collateral ligament at every flexion angle. In contrast, the lateral collateral ligament fiber bundles shortened after anterior cruciate ligament injury.

Conclusion: The altered deformations of the collateral ligaments associated with the changes in tibiofemoral joint kinematics after anterior cruciate ligament injury demonstrate that deficiency of 1 of the knee joint structures upsets the in vivo knee homeostasis.

Clinical Relevance: Restoring normal knee kinematics after anterior cruciate ligament reconstruction is critical to restore the normal function of the collateral ligaments.

INTRODUCTION

Rupture of the anterior cruciate ligament (ACL) is a common injury. Up to 77% of patients evaluated with acute traumatic hemarthrosis of the knee are estimated to have an injury of the ACL, often without clinically noticeable abnormal joint laxity.[5,16,26,30] Acute rupture of the ACL is often associated with meniscal tears, chondral damage, and injury to the medial collateral ligament (MCL).[5,8,16,26,30] As the ACL injury becomes more chronic, an increasing incidence of joint swelling, instability, and meniscal tears is found, eventually resulting in progressive osteoarthritis.[2,13,27,28] In combined ACL and MCL injuries, the MCL does not heal as well as it does in isolated MCL injuries.[6,17,25,32,41]

A potential explanation for the poor outcome after ACL injury is that the changes in tibiofemoral joint kinematics associated with ACL injury disturb the normal function of other tissue structures of the knee.[3,12,18,25,36,38] Numerous in vivo studies have demonstrated that the ACL plays an important role in restraining anterior translation and internal rotation of the tibia at low flexion angles.[4,14,24,35] In addition, ACL injury was recently shown to cause a medial translation of the tibia during in vivo weightbearing flexion.[9] Transection of the ACL in cadaveric knees significantly increased the in situ forces in the MCL,[12,18,25,36,38] medial meniscus,[3] and the bony surface[36] under an anterior tibial load. Furthermore, studies using finite-element modeling predicted higher MCL strains after ACL transection under anterior tibial loads.[12]

In this study, we hypothesized that injury to the ACL alters the deformation of the collateral ligaments during an in vivo functional activity. We used magnetic resonance (MR) and dual orthogonal fluoroscopic imaging techniques to analyze the effects of injury to the ACL on the length of the collateral ligaments during in vivo knee flexion from 0° to 90°, with the healthy contralateral knee of each patient serving as a control.

MATERIALS AND METHODS

Six patients (5 men and 1 woman; age range, 19-38 years old; active on a moderate athletic level before injury and with no previous abnormal condition of the knee or lower limb) with complaints of knee laxity were included in the study. The protocol was approved by the Institutional Review Board at our institute. The included patients had diagnosed unilateral ACL injuries documented by clinical examination (8 mm Lachman with no end point and a grade 2 pivot shift measured by the same orthopaedic surgeon) and MR imaging. The patients had minimal associated injuries to other knee ligaments and cartilage and had healthy contralateral knees. Two patients had no significant damage to the menisci, 1 patient had a partial-thickness tear of the lateral meniscus, and the remaining 3 patients had injuries requiring up to 30% removal of the lateral meniscus at time of ACL reconstruction. Subjects had been injured within an average of 4.5 ± 3 months of testing. With the patients

supine and the knee in a relaxed, extended position, both knees were imaged with an MR scanner using a 1.5-tesla magnet (General Electric, Waukesha, Wis) and a fat-suppressed 3D spoiled gradient-recalled sequence. 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. These images were used to create 3D models of the knees in a solid modeling software (Rhinoceros, Robert McNeel and Associates, Seattle, Wash). Each anatomical knee model included the geometry of the femur and tibia, as well as the attachment sites of the superficial medial collateral ligament (SMCL), deep medial collateral ligament (DMCL), and lateral collateral ligament (LCL) (Figure 1). The ligament attachment sites were directly obtained from the MR images with the assistance of anatomical studies.[7,40,42]

After the MR image–based computer models were constructed, both knees of each subject were imaged using 2 orthogonally placed fluoroscopes as the patient performed a

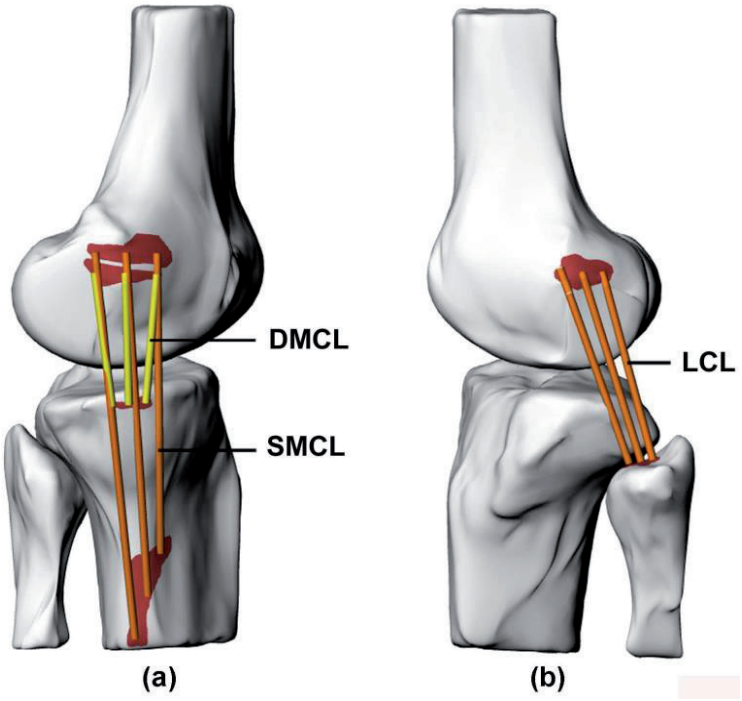

Figure 1. A typical 3D model of the knee created from sagittal plane MR images. (**A**) the attachment sites of the superficial medial collateral ligament (SMCL) and deep medial collateral ligament (DMCL); (**B**) the attachment sites of the lateral collateral ligament (LCL)

quasistatic single-legged lunge at 0°, 15°, 30°, 60°, and 90° of flexion. Flexion angle was verified with a goniometer as subjects stood upright on the platform with the fluoroscopes positioned in the horizontal plane. These images were used to quantify the in vivo knee position at each of the targeted flexion angles. The orthogonal images were imported into a solid modeling software and placed in the orthogonal planes based on the position of the fluoroscopes. The MR image–based knee models were viewed from 2 orthogonal directions corresponding to the views of the fluoroscopes. The models were manually 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 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.

These imaging techniques have been described in detail in previous publications.[9,10,19-23,33] This system has an accuracy within 0.1 mm and 0.1° in determining the position and orientation of regularly shaped solid objects.[23] Recently, the procedure was further validated using a cadaveric knee, and the accuracy of measuring translation was less than 0.1 mm, whereas the repeatability was less than 0.1 mm and 0.3°.[9] The digitized attachment sites of the collateral ligaments from the MR images are not exactly the same for the pair of knees of each subject. To quantify the effect of ACL injury on length change of the

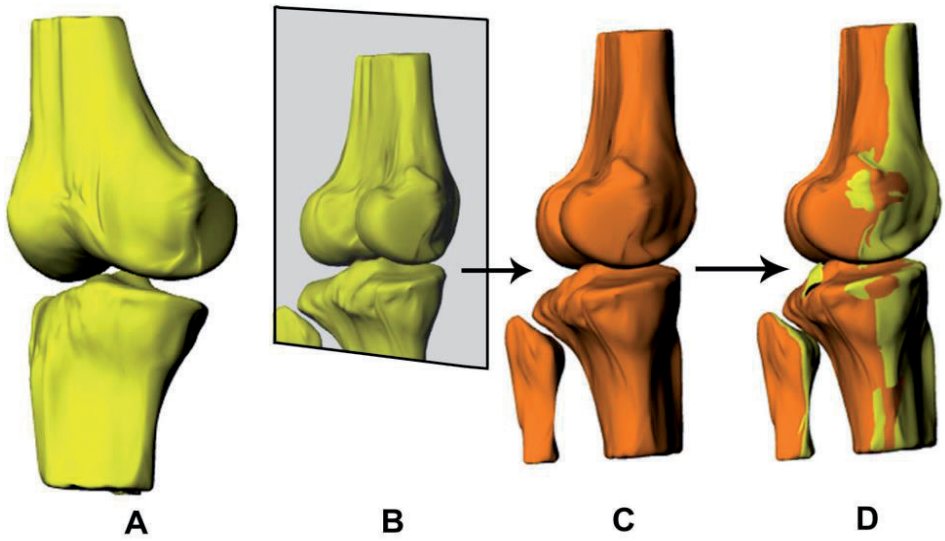

Figure 2. (**A**) a typical 3D model of the right knee; (**B**) the mirrored right knee model; (**C**) the 3D model of the left knee; (**D**) the mirrored right knee model is overlapped with the left knee model.

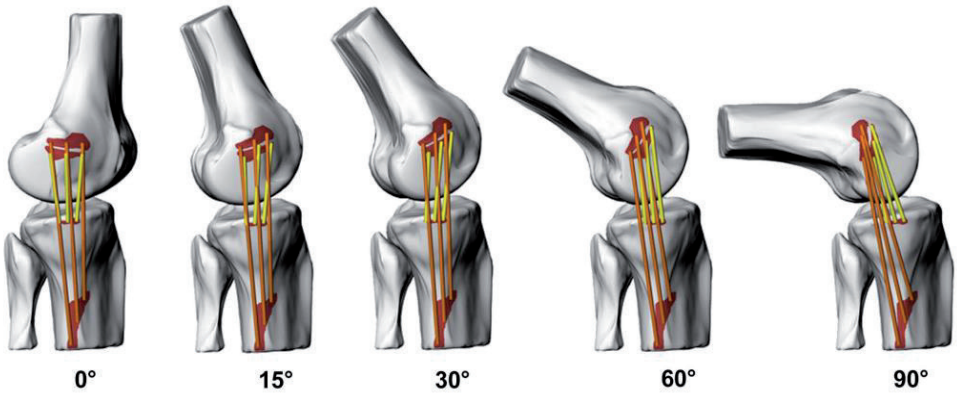

Figure 3. The knee models for a typical subject at 0°, 15°, 30°, 60°, and 90° of flexion during a quasistatic lunge.

collateral ligaments, we chose to use the centroids of the ligament attachment sites of the left knee for measurement of the bundle lengths of the collateral ligaments for both the ACL-injured and intact contralateral knees for each subject. The 3D model of the right knee was mirrored to create a "left" knee model.[9,22] The mirrored right knee was then overlapped with the 3D model of the left knee so that the centroids of the ligament attachment sites could be created on both knees simultaneously. The variability of the measurements due to differences in digitized ligament attachment sites is reduced because the same ligament attachment sites were used for both knees.[9] Figure 2 shows a patient's left knee model and the mirrored right knee.

The knee models for a typical subject at different flexion angles are shown in Figure 3. From the knee models, the relative positions of the SMCL, DMCL, and LCL attachment sites on the femur, tibia, and fibula were determined. The lengths of the collateral ligaments were directly measured from these models at each flexion angle. The insertion areas of each ligament were separated into 3 equal portions, creating 3 equal fiber bundles: the anterior bundle, the middle bundle, and the posterior bundle. The centroids of each portion were calculated. The length of ligaments was defined as the shortest distance between the centroids of the insertion areas. Because the collateral ligaments wrap around the femoral condyles and the tibial plateau, the direct line connecting the area centers was projected on the bony surfaces to create a curved ligament path (Figure 4). The length of this projected curve was measured as ligament length. At each flexion angle, the Wilcoxon signed rank test was used to compare the length of the fiber bundles between the intact and ACL-injured knees. Statistical significance was set at $P < .05$.

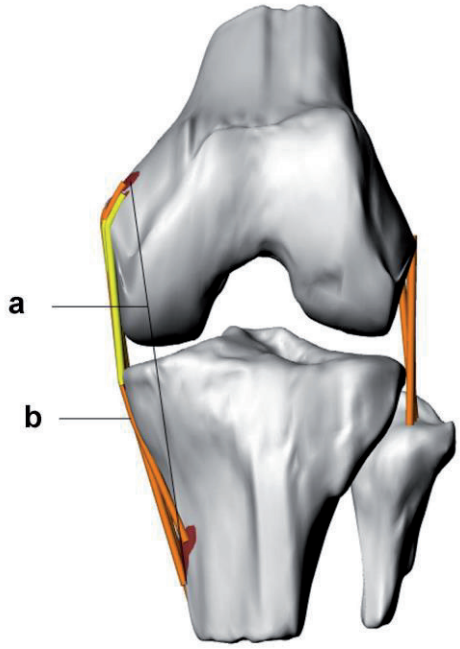

Figure 4. An anterior view of a typical knee model. (**a**) the direct line connecting the ligament area centers; (**b**) the direct line was projected on the bony surfaces to create a curved ligament path.

RESULTS

Superficial Medial Collateral Ligament

The ACL injury caused significant lengthening ($P < .05$) of all the fiber bundles of the SMCL at every target flexion angle (Figure 5). In the intact knee, the length of the anterior bundle at 0° was 78.8 ± 5.8 mm and increased gradually to 86.0 ± 8.2 mm at 90°. In the ACL-injured knee, the anterior bundle measured 80.2 ± 5.9 mm at 0° and 87.0 ± 7.7 mm at 90°. The middle bundle of the intact knee was 91.4 ± 6.1 mm long at 0°, increased slightly to 91.7 ± 6.0 mm at 30°, and then decreased to 89.2 ± 7.1 mm at 90°. A similar pattern of deformation was observed in the ACL-injured knee, but injury to the ACL caused a 1.5% increase in length ($P < .05$) at every flexion angle. In the intact knee, the length of the posterior bundle decreased from 103.3 ± 6.7 mm at 0° to 92.2 ± 6.2 mm at 90°. In the injured knee, the posterior bundle was 104.7 ± 7.0 mm long at 0° and decreased to 94.0 ± 6.4 mm at 90°.

Deep Medial Collateral Ligament

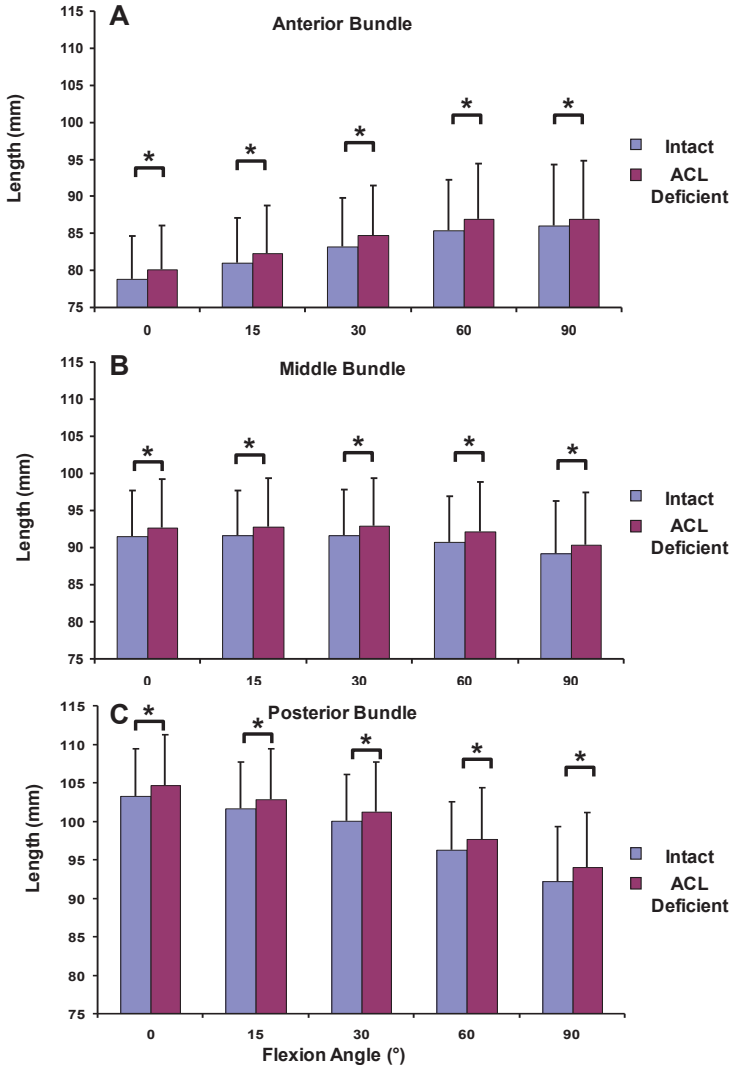

Figure 5. The length of the anterior (**A**), middle (**B**), and posterior (**C**) bundles of the superficial medial collateral ligament (SMCL) as a function of flexion for the intact and ACL-deficient knees. *Statistically significant difference ($P < .05$).

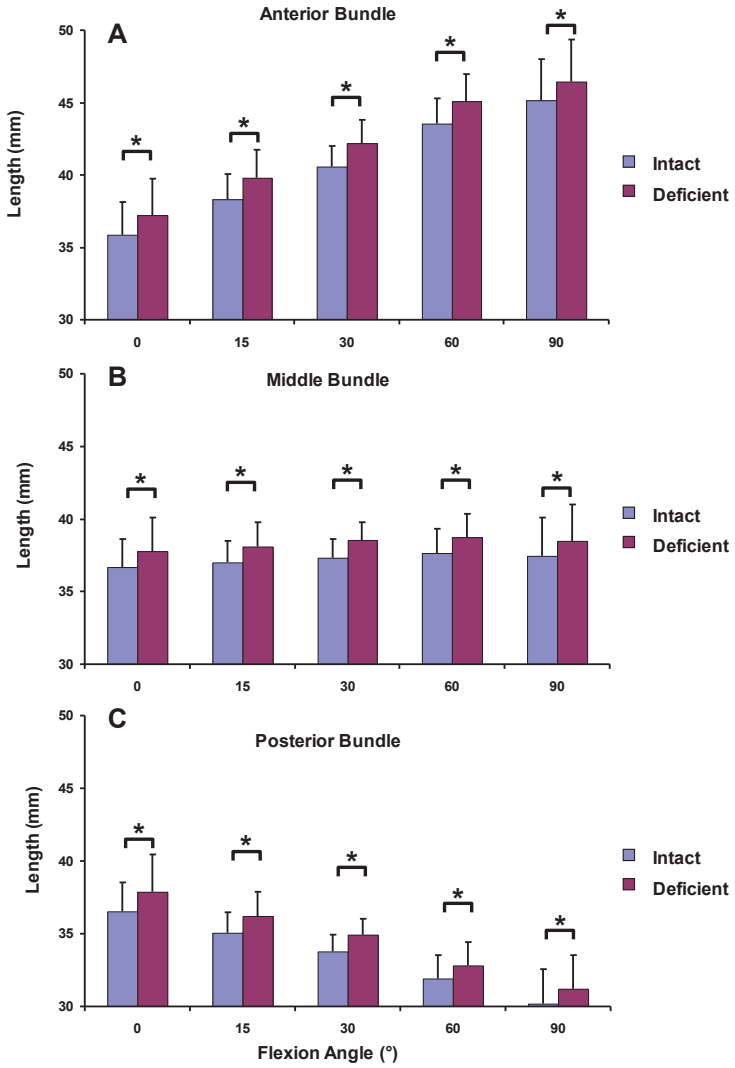

Figure 6. The length of the anterior (**A**), middle (**B**), and posterior (**C**) bundles of the deep medial collateral ligament (DSMCL) as a function of flexion for the intact and ACL-deficient knees. *Statistically significant difference ($P < .05$).

The ACL injury caused significant lengthening ($P < .05$) of all the fiber bundles of the DMCL at every flexion angle (Figure 6). In the intact knee, the length of the anterior bundle at 0° was 35.9 ± 2.2 mm and increased gradually to 45.2 ± 2.8 mm at 90°. In the ACL-injured knee, the anterior bundle measured 37.2 ± 2.5 mm at 0° and 46.5 ± 2.9 mm at 90°. The effect of ACL injury was maximal at 30° of flexion, with a 3.9% increase in length compared to the length of the same bundle in the intact knee. The middle bundle of the intact knee was 36.7 ± 1.9 mm long at 0° and increased slightly to 37.4 ± 2.6 mm at 90°. In the ACL-injured knee, the middle bundle was 37.6 ± 2.3 mm at 0° and 38.5 ± 2.5 mm at 90°. The length of the posterior bundle in the intact knee decreased from 36.5 ± 2.0 mm at 0° to 30.2 ± 2.3 mm at 90°. In the injured knee, the posterior bundle was 37.9 ± 2.5 mm long at 0° and decreased to 31.2 ± 2.2 mm at 90°, a 3.4% increase relative to the healthy ligament.

Lateral Collateral Ligament

A relative shortening of the fiber bundles of the LCL was observed after ACL injury. From 0° to 90° of flexion, ACL injury decreased the length of the anterior bundle significantly ($P < .05$). In the intact knee, the anterior bundle lengthened from 52.2 ± 8 mm at 0° to 54.0 ± 8.8 mm at 30°, and from 50.3 ± 7.8 mm to 52.5 ± 9 mm at 30° in the injured knee. Similar increases in length with further flexion of the intact and injured knee were observed, with statistically significant decreases for the ACL-deficient knee. The ACL injury decreased the length of the bundles of the middle and posterior bundles of the LCL significantly ($P < .05$) at every flexion angle. The length of the middle bundle remained relatively constant along the flexion path in the intact and injured knee. The middle bundle at 0° measured 51.1 ± 7.9 mm and 49.3 ± 7.8 mm in the intact and injured knee, respectively. At 90°, the middle bundle measured 50.4 ± 9.1 mm and 49.2 ± 9.6 mm in the intact and injured knee, respectively. The length of the posterior bundle in the intact knee decreased from 49.7 ± 7.7 mm at 0° to 43.4 ± 9.2 mm at 90°. In the injured knee, the posterior bundle was 47.8 ± 7.8 mm long at 0° and decreased in length to 42.0 ± 9.8 mm at 90°. The maximal significant difference between the intact and injured knee was at 15°, at which the length of the posterior bundle measured 48.2 ± 8.1 mm and 46.2 ± 8.4 mm, respectively.

DISCUSSION

This study quantified the effects of injury to the ACL on the collateral ligaments during an in vivo quasistatic lunge from 0° to 90° of knee flexion. The lengths of the fiber bundles of the SMCL, DMCL, and LCL of 6 patients with unilateral ACL injury were measured using MR and dual orthogonal fluoroscopic imaging techniques, with the healthy contralateral knee of each patient serving as control.

In the healthy contralateral knees, the elongation patterns of the fiber bundles of the collateral ligaments described in our study followed similar trends throughout the flexion path as the elongation patterns previously described in healthy subjects.[33] In the knees with ACL injury, the SMCL fiber bundles of the knee were significantly lengthened. The fiber bundles of the SMCL lengthened around 1.5% compared with the healthy knee. All the fiber bundles of the DMCL were significantly lengthened after ACL injury at every flexion angle. The effect of ACL injury was maximal at 30° of flexion in the anterior bundle of the DMCL, with a 3.9% increase in length compared to the length of the same bundle in the intact knee.

The ACL injury resulted in significant shortening of the LCL bundles. The maximal difference between the intact and injured knee occurred at 15°, where the length of the posterior bundle was 3.9% shorter compared with the length of the same bundle in the intact knee.

The changes in tibiofemoral joint kinematics associated with ACL injury are the key to the altered elongation patterns of the collateral ligaments during in vivo knee flexion. Numerous studies have demonstrated that the ACL plays an important role in restraining anterior translation and internal rotation of the tibia.[4,9,11,14,24,35,37] Andriacchi and Dyrby[4] observed a decrease in the external tibial rotation before heel strike in patients with ACL injury. DeFrate et al[9] found that ACL injury not only caused a statistically significant anterior shift (approximately 3 mm) and internal rotation of the tibia (approximately 2°) at low flexion angles but also caused a medial tibial translation of approximately 1 mm from 15° to 90° of flexion. An ACL injury might also cause an increased valgus rotation compared with the intact knee.[9] These findings might help to explain the increased elongation of the MCLs and the shortened length of the LCL in the ACL-injured knees observed in our study. For example, we observed that the orientation of the SMCL and DMCL is approximately parallel to the tibial shaft at full extension, whereas the LCL passes from anterior proximally to posterior distally, as shown in Figure 1. Therefore, the increased internal tibial rotation associated with ACL injury will elongate the SMCL and DMCL and shorten the LCL.

Previous in vitro studies have examined the effect of injury to the ACL on other tissue structures of the joint.[3,12,15,18,25,36,38,39] For example, transection of the ACL in cadaveric knees significantly increased the in situ forces in the MCL.[12,18,25,36,38] The in situ forces in the MCL increased by 63% at 30° of knee flexion under a 134-N anterior tibial load in the ACL-deficient knee.[18] A finite-element modeling study predicted significantly higher MCL strains after ACL transection under anterior tibial loads.[12] Combined musculoskeletal modeling and computer simulation calculated a peak force in the MCL that was 3 times greater in the ACL-deficient knee than in the ACL-intact knee during walking.[38] Even though a direct comparison of our data and those from the in vitro studies is difficult

because of the differences between in vivo and in vitro studies, they showed similar effects of ACL injury on the function of the MCL.

These data suggest that because of the altered role of the MCL in maintaining knee joint stability, the MCL is at greater risk of secondary injury after isolated ACL injury. This suggestion is supported by the observation that there is an increased incidence of MCL tears in ACL-injured knees compared to uninjured knees.[31] Tissue strain analysis in a rat MCL indicated that necrotic fibroblast damage was induced at ligament strain levels significantly below the structural damage threshold.[34] In our study, up to 3.9% lengthening of the medial collateral fiber bundles occurred in the ACL-injured knee, which might have a negative effect on the biomechanical function of the MCL.

Traumatic rupture of the ACL is frequently associated with injury of the MCL.[29] Numerous animal studies have demonstrated that the mechanical properties of the healing MCL are inferior in a combined ACL and MCL injury as compared with an isolated MCL injury.[6,17,25,32,41] For example, at 6 weeks after ACL and MCL transection in a goat model, the tensile strength of the healing MCL was only 10% of the tensile strength of controls.[1] Excessive loads across the MCL as a result of the combined ACL and MCL injury have been thought to decrease the ability of the MCL to heal. The findings of the present study suggest that there might be higher loads on the MCLs after ACL injury, potentially impeding MCL healing.

It is interesting to note that ACL injury resulted in significant shortening of the LCL bundles, which might indicate that ACL injury alters the biomechanical function of the posterolateral structures. The impact of this finding is currently unclear. Further research is necessary to investigate the effect of ACL injury on the stabilizing structures of the lateral knee compartment, such as the popliteus tendon, the popliteofibular ligament, and the lateral meniscus.

One of the limitations of this study was that we measured the length of the collateral ligament fiber bundles as the distance between the bundle insertion centroids on the tibia and femur. These data cannot be directly related to ligament strains because the reference lengths of the ligaments (zero-load length) are unknown. Furthermore, we only examined the effect of ACL injury during a quasistatic single-legged lunge using a goniometer to control the flexion angle. In the future, the effects of injury to the ACL on the elongation during other in vivo activities such as gait and stair ascent and descent should also be investigated.

In conclusion, injury to the ACL altered the lengths of the collateral ligaments during in vivo knee flexion. The ACL injury caused a significant elongation of the fiber bundles of the SMCL and DMCL at every flexion angle. Throughout most of the range of motion, ACL injury resulted in significant shortening of the LCL bundles. These altered length patterns of the collateral ligaments, associated with the changes in tibiofemoral joint

kinematics after ACL injury, demonstrate that deficiency of the ACL upsets the in vivo knee homeostasis and puts the associated joint environment at greater risk of secondary injury. The altered deformations of the collateral ligaments associated with ACL injury stress the importance for ACL reconstruction techniques to restore knee kinematics in 6 degrees of freedom. Restoring knee kinematics might prevent secondary injury to other tissue structures around the knee joint and improve the healing process of the associated structures in the event of a combined ligament injury.

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