An In Vitro Robotic Assessment of the Anterolateral Ligament, Part 1

Secondary Role of the Anterolateral Ligament in the Setting of an Anterior Cruciate Ligament Injury

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Background: Recent investigations have described the structural and functional behavior of the anterolateral ligament (ALL) of the knee through pull-apart and isolated sectioning studies. However, the secondary stabilizing role of the ALL in the setting of a complete anterior cruciate ligament (ACL) tear has not been fully defined for common simulated clinical examinations, such as the pivot-shift, anterior drawer, and internal rotation tests.

Hypothesis: Combined sectioning of the ALL and ACL would lead to increased internal rotation and increased axial plane translation during a pivot-shift test when compared with isolated sectioning of the ACL.

Study Design: Controlled laboratory study.

Methods: Ten fresh-frozen human cadaveric knees were subjected to a simulated pivot-shift test with coupled 10-N·m valgus and 5-N·m internal rotation torques from 0° to 60° of knee flexion and a 5-N·m internal rotation torque and an 88-N anterior tibial load, both from 0° to 120° of knee flexion via a 6 degrees of freedom robotic system. Kinematic changes were measured and compared with the intact state for isolated sectioning of the ACL and combined sectioning of the ACL and ALL.

Results: Combined sectioning of the ACL and ALL resulted in a significant increase in axial plane tibial translation during a simulated pivot shift at 0°, 15°, 30°, and 60° of knee flexion and a significant increase in internal rotation at 0°, 15°, 30°, 45°, 60°, 75°, 90°, 105°, and 120° when compared with the intact and ACL-deficient states. Based on the model results, ALL sectioning resulted in an additional 2.1 mm (95% CI, 1.4-2.9 mm; $P < .001$) of axial plane translation during the pivot shift when compared with ACL-only sectioning, when pooling evidence over all flexion angles. Likewise, when subjected to IR torque, the ACL+ALL-deficient state resulted in an additional 3.2° of internal rotation (95% CI, 2.4°-4.1°; $P < .001$) versus the intact state, and the additional sectioning of the ALL increased internal rotation by 2.7° (95% CI, 1.8°-3.6°; $P < .001$) versus the ACL-deficient state.

Conclusion: The results of this study confirm the ALL as an important lateral knee structure that provides rotatory stability to the knee. Specifically, the ALL was a significant secondary stabilizer throughout flexion during an applied internal rotation torque and simulated pivot-shift test in the context of an ACL-deficient knee.

Clinical Relevance: Residual internal rotation and a positive pivot shift after ACL reconstruction may be attributed to ALL injury. For these patients, surgical treatment of an ALL tear may be considered.

Keywords: anterolateral ligament; anterior cruciate ligament; rotational knee instability; pivot shift; Segond fracture

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The anterolateral ligament (ALL) of the knee, also referred to as the midthird lateral capsular ligament and anterolateral femorotibial ligament, has received increased attention in light of its recent anatomic "rediscovery" and its hypothesized stabilizing contributions during the pivot-shift test and internal rotation.[1,3,4,14,24,25,29] Historically, Segond[28] has been credited with the discovery of the ALL in 1879, when he first described a bony avulsion fracture of the proximal lateral tibia. Over time, a "Segond fracture" has been reported to be pathognomonic for a concomitant anterior cruciate ligament (ACL) tear.[8,33,35] Concurrent ALL and ACL tears have been theorized to occur via a common mechanism of injury involving an excessive internal rotation torque.[13]

Recent anatomic and biomechanical investigations have expanded on Segond's initial description by providing

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qualitative and quantitative anatomic descriptions of the ALL in relation to the surrounding lateral knee structures. Anatomic studies have reported that the ALL courses deep to the superficial layer of the iliotibial band from the lateral aspect of the femur to the proximal anterolateral tibia.[3,12,18,34] Quantitatively, the femoral origin has been reported to be 4.7 mm posterior and proximal to the fibular collateral ligament, while the tibial insertion was located 9.5 mm distal to the joint line and approximately midway between the Gerdy tubercle and the anterior margin of the fibular head.[18]

Biomechanically, the failure load of the ALL has been reported to be 175 N, with a stiffness of 20 N/mm, and at failure, a Segond fracture occurred in 30% of tested knees.[18] Parsons et al[26] recently published a biomechanical study on the function of the ALL. They reported that the ALL contributed significantly to internal rotation stability at flexion angles >35° but contributed minimally to anterior tibial translational stability from 0° to 90° of flexion. They hypothesized that unrecognized ALL injuries might be the cause of a positive pivot shift in patients with an intact or reconstructed ACL; however, this was not assessed during testing. Monaco et al[24] reported increased instability (as indicated by an increase from grade 2+ to 3+) in cadaveric specimens during a manually applied pivot-shift test when both the ACL and midthird lateral capsular ligament were injured. The findings of Parsons et al and Monaco et al motivated further investigation into quantifying the ALL's secondary role in providing restraint to the knee.

The purpose of this study was to expand on these studies and determine the secondary stabilizing function of the ALL in the setting of an ACL tear from 0° to 120° of knee flexion via a 6 degrees of freedom robotic system for a simulated clinical examination. It was hypothesized that combined sectioning of the ALL and ACL would lead to increased internal rotation and tibial translation during a pivot-shift test in the knee when compared with an isolated ACL injury.

METHODS

Specimen Preparation

Ten fresh-frozen human cadaveric knees with no prior injury, surgical history, or gross anatomic abnormality (mean age, 49.3 years; range, 41-64 years; all male) were included in this study. Internal review board approval was not necessary to conduct this investigation, because de-identified cadaveric specimens are exempt from review at our institution. All specimens were stored at –20°C and thawed at room temperature for 24 hours before preparation. The tibial, fibular, and femoral diaphyses were cut 20 cm from the joint line. All soft tissues on the tibia, fibula, and femur within 10 cm of the joint were preserved. The remaining soft tissue was removed to expose the tibia, fibula, and femur for potting. The distal end of the tibia and fibula and the proximal end of the femur were potted axially in a custom-made cylinder with poly(methyl methacrylate) (Fricke Dental International, Inc).

Robotic Testing Setup

Knee biomechanics were evaluated with a 6 degrees of freedom robotic system (KUKA KR 60-3, KUKA Robotics), which was previously described and validated for knee joint testing.[9,10] Before the knee was mounted within the robotic system, an anatomic knee joint coordinate system was defined for each knee based on palpable tibial and femoral anatomic landmarks,[1,11,36] as measured with a portable coordinate measuring device (7315 Romer Absolute Arm, Hexagon Metrology; manufacturer-reported point repeatability of 0.025 mm). After collection, the potted tibia and fibula were secured within a custom fixture attached to a universal force/torque sensor (Delta F/T Transducer, ATI Industrial Automation) located at the end effector of the robotic system, and the potted femur was secured within a custom fixture mounted to a stationary pedestal (Figure 1).

Before simulated knee examination, the passive flexion-extension path was determined for each knee from 0° (or full extension) to 120° in 1° increments. While a 10-N axial load was applied to ensure contact between the femoral condyles and tibial plateau, forces and torques in the remaining 5 degrees of freedom were minimized (<5 N and <0.5 N·m, respectively) and knee positions recorded. These initial intact knee flexion angle positions served as starting points during subsequent testing.

Biomechanical Testing

Intact, ACL-deficient, and ACL+ALL-deficient knees were subjected to a simulated pivot-shift test, composed of coupled 10-N·m valgus and 5-N·m internal rotation torques,[5,15,16,23] applied at 0°, 15°, 30°, 45°, and 60° of knee flexion. An 88-N anterior tibial load and a 5-N·m internal rotation torque were additionally applied at 0°, 15°, 30°, 45°, 60°, 75°, 90°, 105°, and 120° of knee flexion. Knee conditions were tested in a consistent order: intact, ACL deficient, and ACL+ALL deficient. The ALL was sectioned last to determine its secondary role in providing stability to the knee in the context of ACL deficiency. At the onset, it was assumed that the ACL and ALL functioned in a codominant relationship, similar to the individual bundles of the posterior cruciate ligament.[19,20] The flexion

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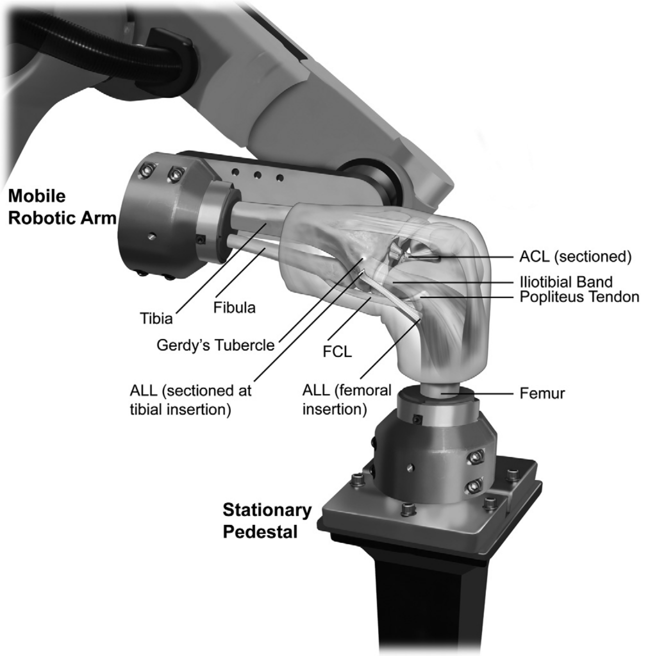

Figure 1. Illustration of a left ACL+ALL-deficient knee mounted in an inverted orientation within the robotic system. Both bundles of the ACL were transected midsubstance. The ALL's tibial insertion was located according to previous anatomic descriptions. At a point midway between the Gerdy tubercle and the anterior margin of the fibular head, a 1-cm transverse incision was made, and soft tissues were carefully dissected down to bone to ensure that the ALL was completely detached. ACL, anterior cruciate ligament; ALL, anterolateral ligament; FCL, fibular collateral ligament.

angle order for testing was randomized to decrease any incremental testing bias.

Identification and Sectioning of the ACL and ALL

Not only were available medical histories screened for confounding injuries and previous surgeries, but specimens were also inspected before testing with the assistance of an arthroscope via a medial parapatellar arthrotomy[9] to assess the integrity of tissues and identify abnormalities (eg, meniscal degradation) that could confound findings of simulated physical examinations.[23] A hockey stick–shaped lateral incision,[32] limited to the skin and subcutaneous tissues, was used to access the lateral extra-articular structures. All surrounding tissues were left intact, including the iliotibial band's attachment at the knee. After inspection, the arthrotomy and lateral incision were closed with a No. 2 polyethylene/polyester suture (FiberWire, Arthrex, Inc).

After the intact knee was tested, the medial arthrotomy was reopened to section the ACL. With the knee positioned in 120° of flexion, a No. 15 blade was used to sharply transect the ACL at its midsubstance, thus preserving the femoral and tibial attachments. Care was taken to preserve the anterior intermeniscal ligament (when present) and the anterior root attachment of the lateral meniscus.[21] The arthrotomy was closed again before simulated examinations were performed on the ACL-deficient knee.

After the ACL-deficient knee was tested, the lateral incision was reopened to section the ALL. The ALL tibial insertion was identified according to previous anatomic descriptions,[3,18,34] midway between the Gerdy tubercle and the anterior margin of the fibular head, approximately 9.5 mm distal to the joint line.[18] At this point, an approximately 1-cm transverse incision was made, and soft tissues were carefully dissected down to bone to ensure that the ALL was completely detached (Figure 1). The lateral incision was closed before performing subsequent testing.

Statistical Analysis

Based on an alpha level of 0.0167 (overall alpha of 0.05 with Bonferroni correction for 3 comparisons), 10 specimens were found to be sufficient to detect a standardized mean difference (d) of 1.22 with 80% power. To assess the 3 conditions—intact, ACL deficient, and ACL+ALL deficient—all pairwise comparisons were made with paired t tests separately at each flexion angle. The Holm method was used to control the familywise error rate for the tests conducted within each flexion angle.

Additionally, multifactorial models were built with data from all flexion angles to characterize the translation and rotation measurements more generally and to detect small but consistent differences among conditions. Flexible linear mixed-effects models were built that incorporated a random intercept for each specimen and allowed a continuous cubic relationship for flexion angle. Model selection was performed via Akaike information criterion, and a compound symmetry correlation structure among conditions was fit. For the measurements of anterior tibial translation during anterior tibial loading and axial translation during simulated pivot shift, analysis was performed on intact-subtracted data, which served to make group variances more equal and hence produce a better model fit. Residual diagnostics were performed and models iterated when assumptions were not met. These models were reported when they added valuable information beyond what was provided by the pairwise t tests. The statistical computing software R (R Foundation for Statistical Computing; with *lme4, ggplot2, rms, reshape*, and *effects* packages) was used for all statistical analyses.[27]

RESULTS

Knee kinematics are reported in Tables 1 and 2. Results for the ACL- and ACL+ALL-deficient states are reported as the mean ± SD from the 2-factor models, and pooled statistics are reported with 95% CIs.

Axial Plane Translation and Internal Rotation During Simulated Pivot Shift

Combined sectioning of the ACL and ALL resulted in significant increases in axial plane translation when compared with isolated sectioning of the ACL at 0°, 15°, 30°,

TABLE 1
Axial Plane Translation and Internal Rotation During a Simulated Pivot-Shift Test: Raw Values, Unadjusted for Flexion Angle[a]

	Axial Plane Translation, mm			Internal Rotation, Degrees		
Flexion Angle	Intact	ACL Deficient	ACL+ALL Deficient	Intact	ACL Deficient	ACL+ALL Deficient
0°	2.0 ± 1.0	5.1 ± 1.9[b,c]	6.3 ± 2.5[b,c]	10.0 ± 3.0	12.1 ± 3.4[b,c]	13.7 ± 3.7[b,c]
15°	2.7 ± 1.5	6.6 ± 3.6[b,c]	8.6 ± 4.3[b,c]	13.9 ± 4.8	14.9 ± 4.7[b,c]	17.2 ± 5.2[b,c]
30°	2.9 ± 1.6	5.6 ± 3.8[b,c]	8.0 ± 5.9[b,c]	16.4 ± 5.3	16.6 ± 5.3[c]	19.8 ± 5.9[b,c]
45°	2.7 ± 1.9	4.4 ± 3.3	6.7 ± 5.9	16.8 ± 5.6	17.0 ± 5.6[c]	20.3 ± 5.9[b,c]
60°	2.5 ± 1.8	3.2 ± 2.2[b,c]	5.9 ± 4.8[b,c]	16.1 ± 5.2	16.2 ± 5.2[c]	19.7 ± 5.3[b,c]

[a] Values presented in mean ± SD. ACL, anterior cruciate ligament; ALL, anterolateral ligament.
[b] Significantly different from intact ($P < .05$ via paired t test with Holm adjustment).
[c] Significantly different between ACL deficient and ACL+ALL deficient.

TABLE 2
Anterior Translation During a Simulated Anterior Drawer and Internal Rotation During a Simulated Internal Rotation Torque: Raw Values, Unadjusted for Flexion Angle[a]

	Anterior Translation, mm			Internal Rotation, Degrees		
Flexion Angle	Intact	ACL Deficient	ACL+ALL Deficient	Intact	ACL Deficient	ACL+ALL Deficient
0°	3.2 ± 0.8	11.0 ± 2.2[b,c]	11.9 ± 2.4[b,c]	9.8 ± 2.8	11.7 ± 3.2[b,c]	13.2 ± 3.4[b,c]
15°	3.5 ± 1.1	13.1 ± 3.2[b]	14.3 ± 3.8[b]	13.4 ± 4.5	14.4 ± 4.5[b,c]	16.7 ± 5.2[b,c]
30°	3.3 ± 0.8	12.3 ± 3.7[b]	13.7 ± 5.1[b]	15.7 ± 5.1	16.2 ± 5.1[b,c]	19.1 ± 5.8[b,c]
45°	3.2 ± 1.0	10.4 ± 4.2[b]	11.7 ± 6.1[b]	16.0 ± 5.4	16.3 ± 5.5[b,c]	19.5 ± 5.8[b,c]
60°	3.0 ± 1.2	8.4 ± 4.0[b]	9.6 ± 5.4[b]	15.4 ± 4.9	15.6 ± 4.9[c]	18.8 ± 5.2[b,c]
75°	2.7 ± 1.1	6.8 ± 3.0[b]	7.7 ± 4.2[b]	14.2 ± 4.5	14.5 ± 4.5[c]	17.2 ± 4.1[b,c]
90°	2.5 ± 1.0	6.1 ± 2.3[b]	7.0 ± 3.4[b]	14.0 ± 4.4	14.1 ± 4.3[c]	17.1 ± 4.4[b,c]
105°	2.4 ± 0.9	6.1 ± 1.5[b]	6.7 ± 2.3[b]	14.5 ± 4.9	14.7 ± 4.8[c]	17.6 ± 4.3[b,c]
120°	2.5 ± 1.0	6.1 ± 1.3[b,c]	6.5 ± 1.7[b,c]	15.2 ± 5.8	15.4 ± 5.6[c]	18.1 ± 4.7[b,c]

[a] Values presented in mean ± SD. ACL, anterior cruciate ligament; ALL, anterolateral ligament.
[b] Significantly different from intact ($P < .05$ via paired t test with Holm adjustment).
[c] Significantly different between ACL deficient and ACL+ALL deficient.

and 60° of flexion during a simulated pivot-shift test (Table 1 and Figure 2). The largest mean increase (2.8 mm) for the ACL+ALL-deficient state versus the ACL-deficient state occurred at 60° of knee flexion. When evidence was pooled across all tested flexion angles, the linear mixed-effects model demonstrated a modeled effect of 2.1 mm (95% CI, 1.4-2.9 mm; $P < .001$) of additional axial plane translation when the ALL was sectioned, as compared with ACL-only sectioning. The ACL- and ACL+ALL-deficient states both had significant increases in axial plane tibial translation when compared with the intact state at 0°, 15°, 30°, and 60° of knee flexion. Maximum translations were observed at 15° of knee flexion, with significant increases observed for the ACL-deficient state (3.9 ± 3.1 mm; $P = .0061$) and the ACL+ALL-deficient state (5.9 ± 3.6 mm; $P = .0016$) as compared with the intact state.

The ACL+ALL-deficient state resulted in significant increases in internal rotation during the simulated pivot shift when compared with the intact and ACL-deficient states at all flexion angles (Table 1 and Figure 2). The largest mean increase (3.5°) for the ACL+ALL-deficient state versus the ACL-deficient state occurred at 60° of knee flexion. Based on the modeling results, the ACL+ALL-deficient state resulted in an additional 3.5° of internal rotation (95% CI, 2.6°-4.4°; $P < .001$) when compared with the intact state, and the additional sectioning of the ALL increased internal rotation by 2.8° (95% CI, 1.8°-3.7°; $P < .001$) when compared with the ACL-deficient state. Isolated sectioning of the ACL resulted in a small nonsignificant ($P = .06$) estimated increase in internal rotation of 0.7° (95% CI, –0.2° to 1.7°) versus the intact state, when pooled across flexion angles. Individual t tests showed a significant increase in internal rotation during simulated pivot shift at 0° ($P < .001$) and 15° ($P = .001$) of knee flexion when compared with the intact state. For the ACL-deficient state, the largest significant increase (2.1° ± 0.9°; $P < .001$) in internal rotation was observed at full extension. For the ACL+ALL-deficient state, the average observed increases were markedly constant throughout flexion, ranging between 3.3° and 3.7°.

Internal Rotation During Internal Rotation Torque

During applied internal rotation torques, the ACL+ALL-deficient state resulted in significant increases in internal

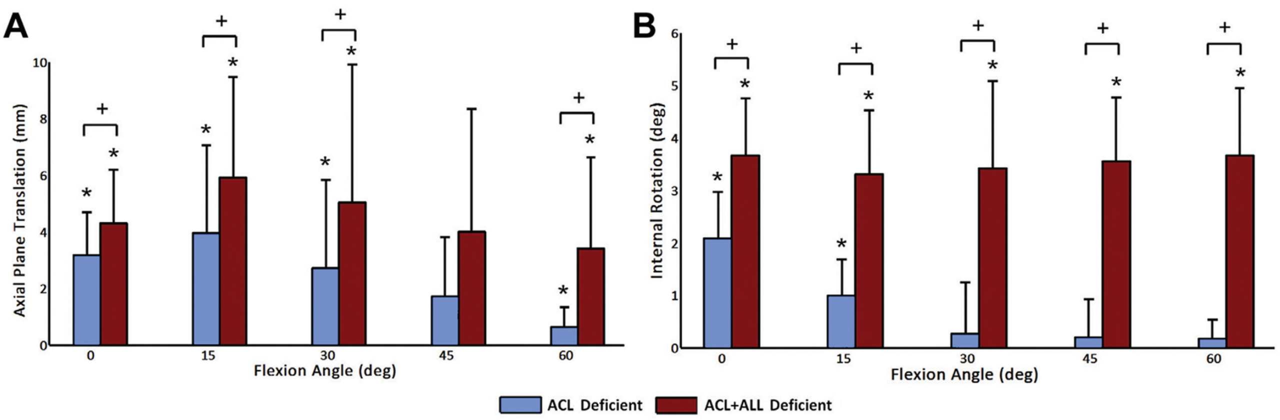

Figure 2. (A) Axial plane translations and (B) internal rotation during simulated pivot shift for the ACL- and ACL+ALL-deficient states, compared with the intact state. ACL, anterior cruciate ligament; ALL, anterolateral ligament. *Significantly different from intact; $^+$significantly different between ACL deficient and ACL+ALL deficient.

rotation when compared with the intact and ACL-deficient states at all flexion angles (Table 2 and Figure 3). Based on the modeling results, the ACL+ALL-deficient state resulted in an additional 3.2° of internal rotation (95% CI, 2.4°-4.1°; *P* < .001) when compared with the intact state, and the additional sectioning of the ALL increased internal rotation by 2.7° (95% CI, 1.8°-3.6°; *P* < .001) when compared with the ACL-deficient state. Isolated sectioning of the ACL resulted in a small nonsignificant (*P* = 0.15) estimated increase in internal rotation of 0.5° (95% CI, –0.3° to 1.4°) versus the intact state when pooled over all flexion angles, while the individual *t* tests demonstrated that the ACL-deficient state resulted in significant increases in internal rotation at 0° to 45° of knee flexion versus the intact state. For the ACL-deficient state, the largest significant increase (2.0° ± 0.9°; *P* < .001) in internal rotation was observed at full extension. The average increase attributed to ACL+ALL deficiency relative to the intact knee was remarkably consistent, ranging between 2.9° and 3.5°.

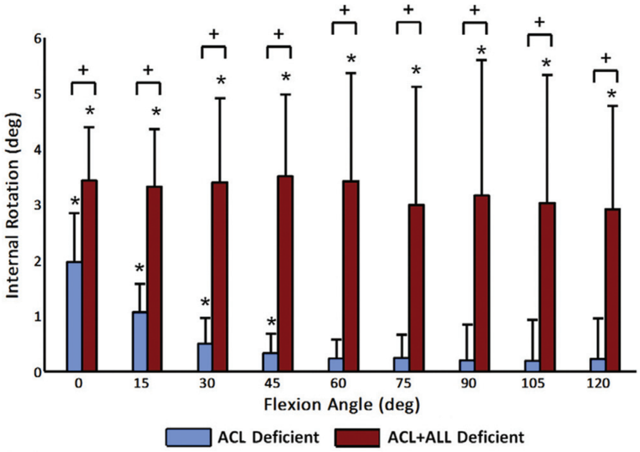

Figure 3. Internal rotation during an applied 5-N·m internal rotation torque for the ACL- and ACL+ALL-deficient states, compared with the intact state. ACL, anterior cruciate ligament; ALL, anterolateral ligament. *Significantly different from intact; $^+$significantly different between ACL deficient and ACL+ALL deficient.

Anterior Tibial Translation During Anterior Tibial Loading

The ACL- and ACL+ALL-deficient states both had significant increases in anterior tibial translation when compared with the intact state from 0° to 120° of knee flexion (Table 2 and Figure 4). The ACL+ALL-deficient state resulted in further significant increases in anterior translation when compared with the ACL-deficient state at 0° and 120° of flexion. Pooling evidence across flexion angles demonstrated that subsequent sectioning of the ALL led to a small but consistent additional increase of 0.9 mm (95% CI, 0.2-1.6 mm; *P* = .011) of anterior tibial translation when compared with the ACL-deficient state. The ACL- and ACL+ALL-deficient states both had a maximum increase in anterior tibial translation versus the intact knee at 15° of knee flexion with significant increases in anterior translation of 9.7 ± 3.0 mm (*P* < .001) for the ACL-deficient state and 10.8 ± 3.5 mm (*P* < .001) for the ACL+ALL-deficient state. At 30° of knee flexion, where the Lachman test is performed clinically, significant increases of 9.0 ± 3.8 mm (*P* < .001) and 10.4 ± 4.9 mm (*P* < .001) of anterior tibial translation versus the intact state were observed for the ACL- and ACL+ALL-deficient states, respectively.

DISCUSSION

The most important finding of this study was that during a simulated pivot-shift test, a combined injury to the ACL and ALL resulted in a significant increase in axial plane translation and internal rotation when compared

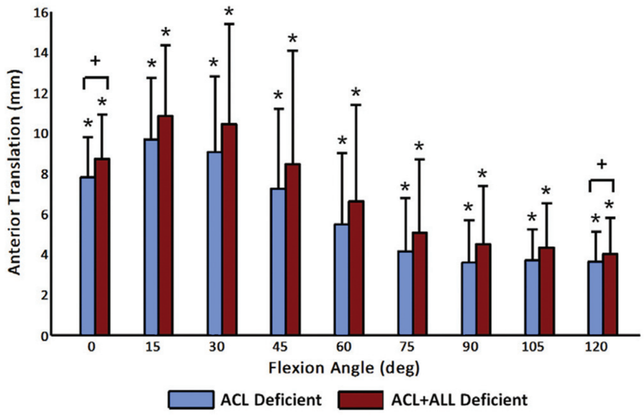

Figure 4. Anterior tibial translations during an applied 88-N anterior tibial load for ACL-deficient and ACL+ALL-deficient states compared with the intact state. ACL, anterior cruciate ligament; ALL, anterolateral ligament. *Significantly different from intact; +significantly different between ACL deficient and ACL+ALL deficient.

with both an intact knee and an ACL-deficient knee. These results confirm that the ALL has a significant secondary role in providing rotatory stability to the knee during the pivot-shift test. When internal rotation was assessed, the isolated ACL-deficient state resulted in small but significant increases in internal rotation at full extension and lower flexion angles (0°-45°); however, the ACL+ALL-deficient state had a significant increase throughout all tested flexion angles (0°-120°) when compared with the intact and ACL-deficient states. In addition to providing rotatory restraint, the ALL had a limited role in restricting anterior tibial translation. This was demonstrated by a small but consistent additional increase in anterior tibial translation after the ALL was sectioned in an already ACL-deficient knee.

The results from the pivot-shift test may help to explain why some select patients have a residual positive pivot-shift test after an ACL reconstruction.[29,31] It has been reported that isolated sectioning of the ACL can result in grade 0, 1+, or 2+ injuries during a pivot-shift test, but it was not until additional sectioning of the ALL that grade 3+ injuries were observed.[24] In the current study, an ACL-deficient knee exhibited a significant increase in axial plane translation, but further sectioning of the ALL resulted in a greater significant increase in translation, particularly near extension (5.9 mm of axial plane translation vs intact at 15° of flexion). This finding corroborates earlier speculations that unrecognized injury to an extra-articular structure such as the ALL could account for some cases of residual rotatory instability after an ACL reconstruction.[3,6,14,25] As a result, we recommend that future studies employ imaging modalities to fully characterize bony anatomy, soft tissue integrity, and other potentially high-risk factors in patients with a residual positive pivot shift after ACL reconstruction.

The rotatory results of the current study are similar to the findings by an earlier biomechanical investigation. Parsons et al[26] reported that the ALL had a greater contribution in preventing internal rotation at higher degrees of flexion, specifically flexion angles >35°. Similarly, the results of this study demonstrated the secondary role of the ALL to be more important in higher degrees of flexion (30°-120°) during isolated internal rotation. In contrast, the largest increase in internal rotation for the ACL-deficient knee occurred at 0° (or full extension), and there was a minimal effect in higher flexion. This finding may be attributed to the ability of the ACL in limiting internal rotation near full extension where it is tight.[7,9] However, sectioning of the ALL concurrent with the ACL resulted in consistently significant total increases in internal rotation throughout the full tested range of flexion (0°-120°) when compared with the intact state. Moreover, the largest increases (3.4°-3.5°) occurred between 30° and 60° of knee flexion. This is consistent with an earlier report that additional sectioning of the midthird lateral capsular ligament resulted in a significant increase in internal rotation (8.7°) at 30° of knee flexion when compared with the intact state.[24] This information may have implications on the proper flexion angle for ALL graft tensioning and fixation during a potential reconstruction procedure. The assessment by Parsons et al[26] was based on in situ force contributions of the ACL, ALL, and lateral collateral ligament during internal rotation. This approach relied on the assumption that the 3 ligaments function independently of one another, which to our knowledge has not been investigated for the ALL. In the current study, we assumed that the ACL and ALL functioned in a synergistic relationship; therefore, we elected to focus on kinematic data rather than force/torque sensor data, which may not be entirely reflective of a synergistic load-sharing relationship. Thus, the results reported by Parsons et al may not be directly comparable with our actual kinematic findings.

Ultimately, the findings of the current study need to be considered in light of the limitations of the biomechanical testing setup. Prior research has reported the iliotibial band to be an important dynamic stabilizer to internal rotation and pivot-shift translation.[2,17] In the absence of this important dynamic stabilizer, the present findings cannot be directly applied to in vivo scenarios. Specifically, we theorize that magnitudes of internal rotatory instability and pivot-shift translation may be smaller in the in vivo ACL+ALL-deficient knee where dynamic stabilizers such as the iliotibial band contribute additional stability.

In addition to providing restraint during internal rotation and the pivot-shift phenomenon, the ALL had a minor role in restricting anterior tibial translation. Parsons et al[26] reported that the ALL had a minimal nonsignificant primary role in preventing anterior tibial translation. Similarly, in our study, there existed a small, consistent, yet significant increase in anterior tibial translation that was attributable to sectioning of the ALL when evidence was pooled over all flexion angles (0° to 120°). This expands on the findings of Monaco et al,[24] who reported significant increases in anterior tibial translation during a more limited flexion range (30°, 60°, and 90° of knee flexion) in ACL+ALL-deficient knees versus the intact state. However, Monaco et al did not observe significant increases between the ACL- and ACL+ALL-deficient states as observed in the present study. By demonstrating the

ALL's secondary role in stability of the knee in the setting of an ACL tear, this study provides the basis for potential clinical and surgical applications. The ALL may be a key to addressing a persistent positive pivot shift after ACL reconstruction for a select group of patients.[29] We theorize that it may also explain some cases of graft failure[29] or why certain patients develop degenerative changes after an ACL reconstruction.[22,30] These surgical scenarios reveal the need for the development of an extra-articular ALL reconstruction technique capable of restoring intact knee kinematics. Future biomechanical and clinical studies are necessary to address these questions.

This study demonstrated the results for complete sectioning of the ACL and ALL. While current clinical examination and magnetic resonance imaging (MRI) techniques are believed to be reliable to document an ACL tear, this is not currently the case with an ALL injury. This is especially true for a chronic case where MRI soft tissue edema has resolved and an injury with residual laxity may have healed sufficiently enough to make it difficult to distinguish a completely healed injury from a partially healed injury on an MRI scan. We caution against widespread ALL repair or reconstruction concurrent with an ACL reconstruction until improved means to objectively diagnose ALL injuries and surgical techniques are developed.

We acknowledge some limitations in the current study. As with any time-zero biomechanics study, this one examined the biomechanics only at the time of injury; in addition, long-term perspectives on how these combined tears might alter other knee structures and increase degenerative changes over time are beyond the scope of the current study. Second, the age range of the donor specimens may not represent the most susceptible population for ACL and ALL injuries. In addition, dynamic muscle stabilizers were not accounted for during testing. Thus, excessive loading of static stabilizers could have resulted in minor elongation of the structures during the testing protocol. While analyzing the role of the ALL as a secondary restraint to tibial translation and rotation, we maintained the testing order for the 3 knee conditions for all specimens. This too could introduce a small amount of increased laxity in the final state (ACL+ALL deficient). However, randomization of the order of tested flexion angles was performed to help reduce any testing bias. Last, knee laxity was assessed with kinematic measurements from simulated clinical examinations. Although these movements may not fully cover the full range of motion and loading that the knee experiences during in vivo activities, they provide an objective means for effectively assessing knee laxity during basic rotational and translational movements.

CONCLUSION

The results of this study further confirm the ALL as an important lateral knee structure that provides rotatory stability to the knee. Specifically, the ALL was a significant stabilizer throughout flexion during an applied internal rotation torque (0°-120°) and simulated pivot-shift test (0°-60°) in the face of concurrent ACL sectioning. In addition, the ALL had a small secondary role in controlling anterior tibial translation when combined with a deficient ACL.

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