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The Biomechanical Function of the Anterolateral Ligament of the Knee

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Abstract

Background—Recent anatomic investigations of the lateral structures of the knee have identified a new ligament, called the anterolateral ligament (ALL). To date, the anterolateral ligament has not been biomechanically tested to determine its function.

Hypothesis—The ALL of the knee will resist internal rotation at high angles of flexion but will not resist anterior drawer forces.

Study Design—Controlled laboratory study.

Methods—Eleven cadaveric knees were subjected to 134 N of anterior drawer at flexion angles between 0° and 90° and separately to 5 N·m of internal rotation at the same flexion angles. The in situ forces of the ALL, anterior cruciate ligament (ACL), and lateral collateral ligament (LCL) were determined by the principle of superposition.

Results—The contribution of the ALL during internal rotation increased significantly with increasing flexion, whereas that of the ACL decreased significantly. At knee flexion angles greater than 30°, the contribution of the ALL exceeded that of the ACL. During anterior drawer, the forces in the ALL were significantly less than the forces in the ACL at all flexion angles ($P < .001$). The forces in the LCL were significantly less than those in either the ACL or the ALL at all flexion angles for both anterior drawer and internal rotation ($P < .001$).

Conclusion—The ALL is an important stabilizer of internal rotation at flexion angles greater than 35°; however, it is minimally loaded during anterior drawer at all flexion angles. The ACL is the primary resister during anterior drawer at all flexion angles and during internal rotation at flexion angles less than 35°.

Clinical Relevance—Damage to the ALL of the knee could result in knee instability at high angles of flexion. It is possible that a positive pivot-shift sign may be observed in some patients with an intact ACL but with damage to the ALL. This work may have implications for extra-articular reconstruction in patients with chronic anterolateral instability.

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Keywords

knee; anterolateral ligament; anterior cruciate ligament; biomechanics; robotics

The lateral structures of the knee are complex. A recent anatomic study by Claes et al[4] described a distinct and consistent structure termed the anterolateral ligament (ALL) of the knee. The ALL has been alluded to in previous work,[3,10,12-15,19,29,32-34,36] but the reports of the insertion sites had not been consistent or well documented before the Claes et al study. The ALL was initially hypothesized to exist as the "pearly band" associated with a Ségond fracture. The Ségond fracture is a small avulsion of the lateral tibia that is considered to be pathognomonic for anterior cruciate ligament (ACL) tears.[3,12-14,17,36] Ségond fractures are associated with an injury pattern similar to that of ACL tears, resulting from combined internal and varus rotations.[14]

The biomechanics of the knee have been studied extensively by use of cadaveric testing with robotics. The components of the mid-third of the lateral capsule,[36] including the iliotibial (IT) band and the lateral collateral ligament (LCL), have been previously investigated.[25,30,37,39] The forces in the LCL were studied by use of cadavers under single degree of freedom translations[25] and rotations.[30] More recently, the effects of IT band tension on the pivotshift maneuver were evaluated.[37] In the report by Claes et al,[4] the authors note that the ALL is its own structure and is not a part of the IT band. The contributions of the ALL have not yet been specifically determined.

The classification of lateral instabilities by Hughston et al[15] showed that acute and chronic anterolateral instability are both associated with damage to the mid-third of the lateral capsule and may be combined with damage to the ACL. Other researchers found that the Ségond fracture is an indicator of major ligamentous damage and is associated with anterolateral instability.[12,36] Terry et al[32] determined that the capsular-osseous deep layer of the iliotibial tract is linked with the ACL when evaluating the grade of knee instability for anterior translation, internal rotation, and varus rotation. Although the importance of the lateral capsular structures has been acknowledged, no controlled laboratory study has tested the range of motion over which the ALL has been isolated as a biomechanically discrete ligament.

Previous researchers have hypothesized that the ALL will experience tension under internal rotation at high angles of flexion due to its anatomic orientation.[4] The purpose of this study was to investigate the biomechanical significance of the ALL under internal rotation and anterior drawer (separately) between 0° and 90° of knee flexion.

Materials and Methods

Institutional Biosafety Committee (IBC) approval was obtained before the study for use of biological tissues. Twelve cadaveric specimens (mean age, 76.3 years; range, 35-92 years) were stored fresh frozen at −30°C until 24 hours before dissection and then were thawed at room temperature. One knee was subsequently excluded because there was no ALL present. All knees were dissected by the same surgeon (A.O.G.) and were examined for deformities or damage to the ACL. The skin and the subcutaneous fat tissue were removed. The IT band was released from the proximal end of the specimen, proximal to the distal femur, and then gently dissected from the underlying tissue until it remained attached at its distal insertion on the Gerdy tubercle. Care was taken to ensure that only the superficial layer of the IT band was reflected, leaving the underlying capsulo-osseous layer intact.[33] The LCL was located by palpating the ligament with the knee in slight varus, and its insertion onto the fibular head was identified. The ligament was then dissected proximally to identify its insertion on the lateral femoral condyle and was freed from the underlying joint capsule and then tagged with sutures that were tied around the ligament. The quantitative descriptions of the ALL insertions by Claes et al[4] were used as a reference to identify the ligament (Figure 1). The tibial and femoral insertions of the ALL were marked with a surgical pen. The insertion on the tibia was posterior to the Gerdy tubercle and anterior to the tip of the fibular head. The insertion on the femur was located near the LCL insertion on the lateral femoral condyle with a confluence of tissue from both the LCL and the ALL as described and illustrated by Claes et al. The remainder of the underlying deep capsule with which the ALL was confluent was left intact so as not to inadvertently damage the ALL before testing. Once the ALL was identified, a standard medial parapatellar incision was used to open the joint capsule and visualize the ACL. The ACL was inspected for ligamentous damage, and this capsulotomy was left open at the time of biomechanical testing. The specimen was stored at 10°C until the day of robotic testing. Specimens were tested between 24 and 72 hours after the dissection.

The robotic testing system consists of a 6 degrees of freedom hexapod (model R2000; Mikrolar) with a supplemental flexion fixture (Newmark Systems Inc) that provides a range of motion up to 120° of knee flexion. The system has the ability to operate in either force control mode or position control mode. Force control mode uses feedback from the load cell (Theta IP65; ATI Industrial Automation) to move the knee to a desired loading condition (ie, for passive flexion, the forces can be minimized). When the system is in position control mode, the robot will move to a desired position and record the forces acting on the knee. The robotic system has a worst-case path repeatability of 0.36 mm under maximum applied loading conditions.[21]

The overall workflow for specimen preparation and robotic testing is shown in Table 1. The femur was cut 20 cm proximal to the joint line and the tibia was cut 15 cm distal to the joint line. The remaining soft tissue was dissected 7 cm from the proximal end of the femur and the distal end of the tibia. The femur and the tibia were then potted in body filler (Bondo) and mounted to the robot by use of 6.35-cm diameter collars. The tibia was mounted to the load cell, and the femur was mounted to the flexion fixture (Figure 2). A joint coordinate system was defined using a 6 degrees of freedom spatial digitizer (model G2LX; eMicroScribe) by marking the long axes of the femur and the tibia, the lateral and medial epicondyles of the femur, and the lateral and medial tibial plateau. The joint coordinate system was optimized by using force control feedback from the load cell to flex the knee from 0° to 90° with minimally applied loads. The knee was then subjected to 10 cycles of passive flexion preconditioning trials. Each knee was also preconditioned once with 150 N of anterior drawer at 30° of flexion and 5 N·m of internal rotation at 30° of flexion.

The primary force control trajectory was created with the knee intact and then later used in position control mode to recreate the same kinematics.[11,26,27,35] An anterior drawer force of 134 N was applied at flexion angles of 0°, 15°, 25°, 35°, 45°, 60°, 75°, and 90°. Then 5 N·m of internal rotation was applied at the same flexion angles. The positions of the robot were recorded at 20 Hz. The knee was then retested in position control mode using the positions from the force control trajectory, and the forces were recorded at 20 Hz.

The ligaments (ACL, LCL, ALL) were then sequentially cut in a block randomized design, and the knee was subsequently tested again using the position trajectory from the force-controlled trial. The ACL and LCL were sectioned in the midportion of each ligament, respectively. When the ALL was sectioned, it was released at the level of the tibial plateau, and the underlying capsule at the location was also incised in a submeniscal fashion, thereby ensuring that the ligament was entirely transected and that the lateral meniscus was not damaged.

A single time point at each loading condition and each flexion angle from the position control trials was used for all 4 cases: (1) intact, (2) the first ligament removed, (3) the second ligament removed, and (4) the third ligament removed. The in situ forces in the ACL, LCL, and ALL were determined by the principle of superposition.[26,35] The force vector at each time point was calculated and subtracted from the previous case. The difference between cases represents the in situ force of the ligament. The percentage force contribution for each ligament was calculated by dividing the magnitude of the force vector by the magnitude of the intact force vector.

For each ligament (ALL, ACL, LCL) and each type of loading (anterior drawer and internal rotation), the mean relative contributions were estimated at each angle of flexion by use of a no-intercept linear regression model with flexion angle as a factor variable. The robust variance estimator from the generalized estimating equations approach was used in the linear modeling to account for possible correlation between observations from the same cadaver.

To assess whether there were general increases or decreases in the percentage of contributions to force and moment over the different angles of flexion, additional linear models were computed similar to the above but a linear term was used as the independent variable rather than a factor. A slope term that was statistically significantly different than zero was interpreted as evidence that the percentage of contributions progressively changed at increasing angles of flexion.

Finally, because of the small number of independent units (11 knees from 8 cadavers), a third set of nonparametric analyses were performed to evaluate the evidence regarding change of force and moment over increasing angle of flexion. In these analyses, slopes of the force and moment contributions for each individual knee were computed using the least squares approach to give a knee-level measure of overall increase or decrease over the range of angles. To reduce the data to independent units, for cadavers that contributed 2 knees, the slopes were averaged to give a cadaveric-level measure. A sign test was then computed to assess whether the median slopes were zero.

Results

The ALL was a distinct and identifiable structure in all but 1 specimen (83% identified), which was excluded from the analysis. In all other specimens, the ALL attached just anterior to the insertion of the LCL on the femoral epicondyle. The tibial attachment was located posterior to the Gerdy tubercle and anterior to the tip of the fibular head on the ridge of the tibial plateau. As described previously, we noted a convergence of the ALL with the insertion of the LCL on the femur proximally, and it was confluent with the anterolateral joint capsule and the deeper lateral meniscus.

The kinematics of the force-controlled trajectory for the intact specimens are reported in Table 2. The anterior tibial translation was a maximum of 8.5 ± 2.5 mm at 35° of flexion. The internal tibial rotation was a maximum of 22.6° ± 6.1° at a flexion angle of 45°.

Tables 3 and 4 present the estimates of the means and 95% CIs for percentage contributions to the anterior drawer force and internal rotation moments, respectively. Figures 3 and 4 show these data graphically. For the anterior drawer forces, the tests for significant change over increasing angles of flexion indicated evidence of significant change for the ACL ($P = .008$), while no significant change was indicated for the ALL ($P = .29$). During internal rotation, both ACL and ALL indicated significant evidence for change in force contribution over increasing angle ($P = .003$ and $P < .001$, respectively). As shown in Table 4 and Figure 4, the changes are in opposite directions: The ACL contribution decreases as flexion angle increases while the ALL contribution increases.

Because the LCL appears to be a uniformly minor contributor to forces and moments at all angles (<5%), tests of change over flexion angle were not computed for the LCL. Values for percentage contributions at a given angle do not sum to 100% because of the contributions of other soft tissue structures not examined here.

After the reduction in data to independent units (see above), the change in ALL contribution to internal rotation was still highly significant ($P = .008$) across the range of knee flexion angles, whereas all other changes were not significant (ACL internal rotation, $P = .29$; ALL anterior drawer, $P > .999$; ACL anterior drawer, $P = .07$).

Discussion

In this study, we confirmed the existence of the ALL through anatomic dissection in all but 1 of 12 specimens and provided evidence that demonstrates its role in resisting internal tibial rotation as knee flexion increases. Unlike the ACL, the ALL did not have a role in resisting anterior tibial drawer at any angle of knee flexion. The LCL was not a primary stabilizer of either anterior drawer or internal rotation at any angle of knee flexion.

The kinematics of the force control trajectory for the intact knee (Table 2) agreed with the findings of previous authors. Zantop et al[38] found a maximum anterior tibial translation of 8.2 ± 1.8 mm with the knee at 30° of flexion with an applied 134 N of anterior drawer force. Similarly, a combined 10 N·m valgus and 4 N·m internal rotation produced a maximum of 24.1° ± 6.5° of internal rotation at a knee flexion angle of 30° in a study by Gabriel et al.[11]

Despite the differences in robotic testing systems and specimen preparation, the trajectory generated during force control in the present study produced kinematics that closely matched prior work.

The findings of this study show that the ALL is a primary stabilizer in internal rotation of the tibia at high knee flexion angles. It is well known that a patient with an intact ACL or a surgically reconstructed ACL may still have a positive pivot-shift sign.[2,5,18,20,21] Also, injuries to the lateral structures of the knee have been shown to produce a positive pivot-shift sign in knees that have no injury to the ACL.[24,31] We speculate that a positive pivot-shift sign in an ACL-intact knee could possibly be explained by unrecognized damage to the ALL. It is also possible that anatomic extra-articular reconstructions, alone or in addition to intra-articular ACL reconstructions of the ALL, may provide additional rotational stability to the unstable knee, especially in the setting of a hyper-lax or revision ACL reconstruction.

Current intra-articular reconstruction of the ACL via either a single-bundle or a double-bundle technique does not eliminate the pivot shift in some patients. For patients with persistent anterolateral rotational knee instability, it is possible that the damage to the ALL has resulted in an additional loss of stability and that extraarticular reconstruction of the ALL in addition to the intra-articular reconstruction of the ACL may be beneficial. Many extra-articular augmentation techniques have been described (eg, MacIntosh,[16] sling and reef,[22] Ellison[9]). Extra-articular reconstruction often involves removing an isolated strip of the IT band that is left attached to the tibia to be used as a graft. The released IT band is then passed deep to the LCL and reattached posterior to the femoral insertion of the LCL (MacIntosh procedure).[16] Several studies have reported little success with extra-articular reconstruction.[5] Amis and Scammell[1] found that supplementing single-bundle ACL reconstruction with the MacIntosh procedure in 10 cadavers did not provide additional knee stability under anterior drawer, internal rotation, or valgus rotation. Draganich and colleagues[7,8,28] explored the effects of extra-articular reconstruction using the Müller anterolateral femorotibial ligament tenodesis alone or in combination with intra-articular reconstruction. They reported that this procedure was effective in constraining anterior drawer and internal tibial rotation from 30° to 90° of knee flexion but that it could also overconstrain internal tibial rotation. They concluded that anterolateral reconstruction could be useful as an adjunctive procedure in appropriate clinical situations. A subsequent study by the same group[6] showed that the surgeon could affect anterior and rotational laxity by adjusting the tension in the tenodesis.

One of the limitations to this study was the evaluation of the “intact” knee kinematics during force control feedback. The trajectory was generated with the skin removed, the IT band reflected, and a medial parapatellar incision through the joint capsule. The dissection of the specimen before the trajectory path had been determined may have altered the kinematics of knee. However, a comparison of the kinematics in the present study with previous work shows that this effect was minimal. Future studies should investigate the effects of anatomic extra-articular reconstruction of the ALL in addition to single-bundle ACL reconstruction.

Conclusion

The results from this study suggest that the ALL is a contributor to rotational knee stability and that the lateral structures of the knee must not be overlooked when determining knee disorders.

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References

1. Amis AA, Scammell BE. Biomechanics of intra-articular and extra-articular reconstruction of the anterior cruciate ligament. J Bone Joint Surg Br. 1993; 75:812–817. [PubMed: 8376447]
2. Bull AM, Amis AA. The pivot-shift phenomenon: a clinical and biomechanical perspective. Knee. 1998; 5:141–158.
3. Campos JC, Chung CB, Lektrakul N, et al. Pathogenesis of the Segond fracture: anatomic and MR imaging evidence of an iliotibial tract or anterior oblique band avulsion. Radiology. 2001; 219:381–386. [PubMed: 11323461]
4. Claes S, Vereecke E, Maes M, et al. Anatomy of the anterolateral ligament of the knee. J Anat. 2013; 223:321–328. [PubMed: 23906341]
5. Dodds AL, Gupte CM, Neyret P, et al. Extra-articular techniques in anterior cruciate ligament reconstruction: a literature review. J Bone Joint Surg Br. 2011; 93:1440–1448. [PubMed: 22058292]
6. Draganich L, Hsieh Y, Reider B. Iliotibial band tenodesis: a new strategy for attachment. Am J Sports Med. 1995; 23:186–195. [PubMed: 7778704]
7. Draganich L, Reider B, Ling M, Samuelson M. An in vitro study of an intraarticular and extraarticular reconstruction in the anterior cruciate ligament deficient knee. Am J Sports Med. 1990; 18:262–266. [PubMed: 2372075]
8. Draganich L, Reider B, Miller P. An in vitro study of the Müller anterolateral femorotibial ligament tenodesis in the anterior cruciate ligament deficient knee. Am J Sports Med. 1989; 17:357–362. [PubMed: 2658630]
9. Ellison AE. Distal iliotibial-band transfer for anterolateral rotatory instability of the knee. J Bone Joint Surg Am. 1979; 61:330–337. [PubMed: 429400]
10. Fairclough J, Hayashi K, Toumi H, et al. The functional anatomy of the iliotibial band during flexion and extension of the knee: implications for understanding iliotibial band syndrome. J Anat. 2006; 208:309–316. [PubMed: 16533314]
11. Gabriel MT, Wong EK, Woo SLY, et al. Distribution of in situ forces in the anterior cruciate ligament in response to rotatory loads. J Orthop Res. 2004; 22:85–89. [PubMed: 14656664]
12. Goldman AB, Pavlov H, Rubenstein D. The Segond fracture of the proximal tibia: a small avulsion that reflects major ligamentous damage. AJR Am J Roentgenol. 1988; 151:1163–1167. [PubMed: 3263770]
13. Haims AH, Medvecky MJ, Pavlovich R, Katz LD. MR imaging of the anatomy of and injuries to the lateral and posterolateral aspects of the knee. AJR Am J Roentgenol. 2003; 180:647–653. [PubMed: 12591668]
14. Hess T, Rupp S, Hopf T, et al. Lateral tibial avulsion fractures and disruptions to the anterior cruciate ligament: a clinical study of their incidence and correlation. Clin Orthop Relat Res. 1994; 303:193–197. [PubMed: 8194233]
15. Hughston JC, Andrews JR, Cross MJ, Moschi A. Classification of knee ligament instabilities, part II: the lateral compartment. J Bone Joint Surg Am. 1976; 58:173–179. [PubMed: 1254620]

16. Ireland J, Trickey EL. Macintosh tenodesis for anterolateral instability of the knee. J Bone Joint Surg Br. 1980; 62:340–345. [PubMed: 7410466]
17. Irvine GB, Dias JJ, Finlay DB. Segond fractures of the lateral tibial condyle: brief report. J Bone Joint Surg Br. 1987; 69:613–614. [PubMed: 3611168]
18. Lane CG, Warren R, Pearle AD. The pivot shift. J Am Acad Orthop Surg. 2008; 16:679–688. [PubMed: 19056917]
19. LaPrade RF. Arthroscopic evaluation of the lateral compartment of knees with grade 3 posterolateral knee complex injuries. Am J Sports Med. 1997; 25:596–602. [PubMed: 9302462]
20. Leitze Z, Losee RE, Jokl P, et al. Implications of the pivot shift in the ACL-deficient knee. Clin Orthop Relat Res. 2005; 436:229–236. [PubMed: 15995446]
21. Lie DTT, Bull AMJ, Amis AA. Persistence of the mini pivot shift after anatomically placed anterior cruciate ligament reconstruction. Clin Orthop Relat Res. 2007; 457:203–209. [PubMed: 17195812]
22. Losee RE, Johnson TR, Southwick WO. Anterior subluxation of the lateral tibial plateau: a diagnostic test and operative repair. J Bone Joint Surg Am. 1978; 60:1015–1030. [PubMed: 721850]
23. Meredick RB, Vance KJ, Appleby D, Lubowitz JH. Outcome of single-bundle versus double-bundle reconstruction of the anterior cruciate ligament: a meta-analysis. Am J Sports Med. 2008; 36:1414–1421. [PubMed: 18508977]
24. Norwood LA, Andrews JR, Meisterling RC, Glancy GL. Acute anterolateral rotatory instability of the knee. J Bone Joint Surg Am. 1979; 61:704–709. [PubMed: 582318]
25. Piziali RL, Seering WP, Nagel DA, Schurman DJ. The function of the primary ligaments of the knee in anterior-posterior and medial-lateral motions. J Biomech. 1980; 13:777–784. [PubMed: 7440592]
26. Rudy TW, Livesay GA, Woo SL, Fu FH. A combined robotic/universal force sensor approach to determine in situ forces of knee ligaments. J Biomech. 1996; 29:1357–1360. [PubMed: 8884481]
27. Sakane M, Fox RJ, Woo SL, et al. In situ forces in the anterior cruciate ligament and its bundles in response to anterior tibial loads. J Orthop Res. 1997; 15:285–293. [PubMed: 9167633]
28. Samuelson M, Draganich L, Zhou X, et al. The effects of knee reconstruction on combined anterior cruciate ligament and anterolateral capsular deficiencies. Am J Sports Med. 1996; 24:492–497. [PubMed: 8827309]
29. Sanchez AR, Sugalski MT, LaPrade RF. Anatomy and biomechanics of the lateral side of the knee. Sports Med Arthrosc. 2006; 14:2–11. [PubMed: 17135939]
30. Seering WP, Piziali RL, Nagel DA, Schurman DJ. The function of the primary ligaments of the knee in varus-valgus and axial rotation. J Biomech. 1980; 13:785–794. [PubMed: 7440593]
31. Tanaka M, Vyas D, Moloney G, et al. What does it take to have a high-grade pivot shift? Knee Surg Sports Traumatol Arthrosc. 2012; 20:737–742. [PubMed: 22222616]
32. Terry GC, Norwood LA, Hughston JC, Caldwell KM. How iliotibial tract injuries of the knee combine with acute anterior cruciate ligament tears to influence abnormal anterior tibial displacement. Am J Sports Med. 1993; 21:55–60. [PubMed: 8427369]
33. Vieira ELC, Vieira EA, da Silva RT, et al. An anatomic study of the iliotibial tract. Arthroscopy. 2007; 23:269–274. [PubMed: 17349469]
34. Vincent JP, Magnussen RA, Gezmez F, et al. The anterolateral ligament of the human knee: an anatomic and histologic study. Knee Surg Sports Traumatol Arthrosc. 2012; 20:147–152. [PubMed: 21717216]
35. Woo SLY, Fox RJ, Sakane M, et al. Biomechanics of the ACL: measurements of in situ force in the ACL and knee kinematics. Knee. 1998; 5:267–288.
36. Woods GW, Stanley RF, Tullos HS. Lateral capsular sign: x-ray clue to a significant knee instability. Am J Sports Med. 1979; 7:27–33. [PubMed: 420385]
37. Yamamoto Y, Hsu WH, Fisk JA, et al. Effect of the iliotibial band on knee biomechanics during a simulated pivot shift test. J Orthop Res. 2006; 24:967–973. [PubMed: 16583447]

38. Zantop T, Herbort M, Raschke MJ, et al. The role of the anteromedial and posterolateral bundles of the anterior cruciate ligament in anterior tibial translation and internal rotation. Am J Sports Med. 2007; 35:223–227. [PubMed: 17158275]

39. Zantop T, Schumacher T, Schanz S, et al. Double-bundle reconstruction cannot restore intact knee kinematics in the ACL/LCL-deficient knee. Arch Orthop Trauma Surg. 2010; 130:1019–1026. [PubMed: 20217104]

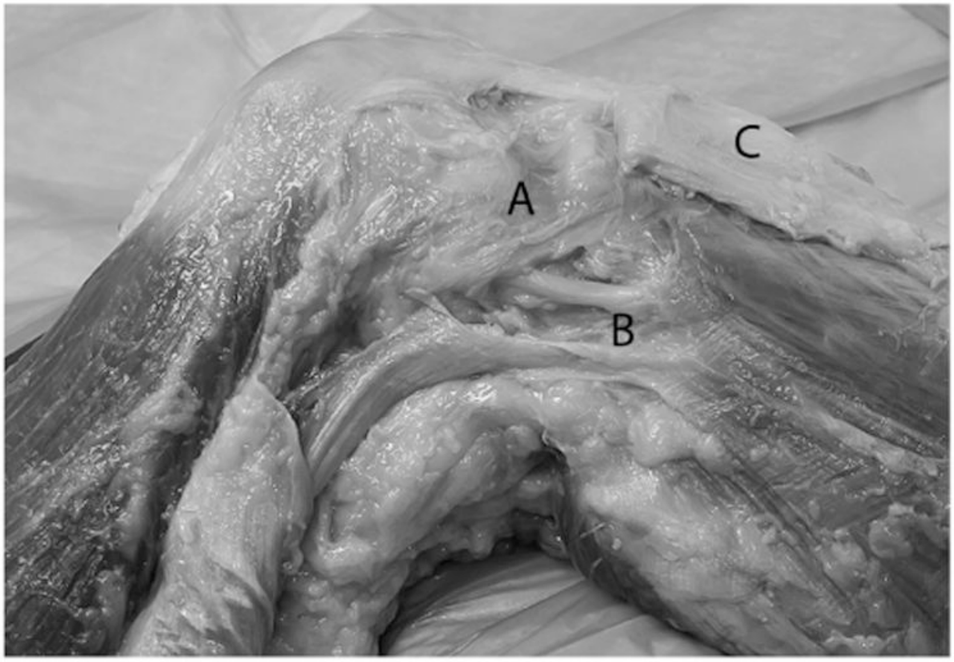

Figure 1.

The lateral structures of a right knee. A, The anterolateral ligament; B, the lateral collateral ligament; C, the iliotibial band reflected.

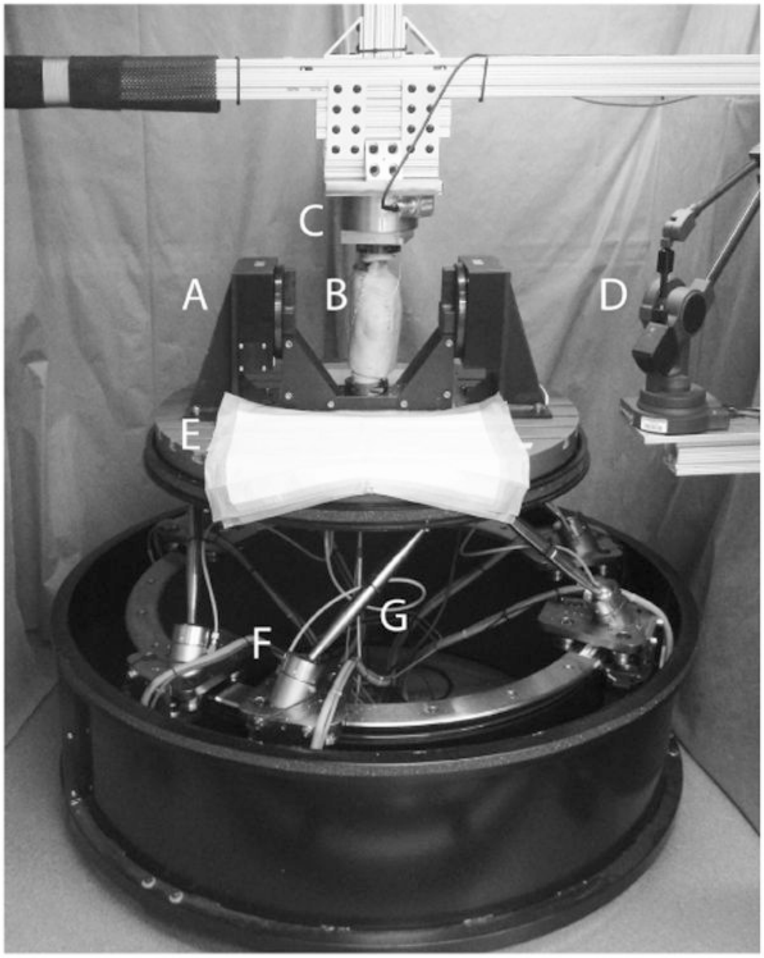

Figure 2.
The robotic testing system with components labeled: A, rotating knee fixture; B, knee specimen mounted with femur downwards; C, load cell; D, MicroScribe for locating anatomic landmarks; E, platform of Rotopod; F, one of the 6 robot trucks; G, one of the 6 robot legs.

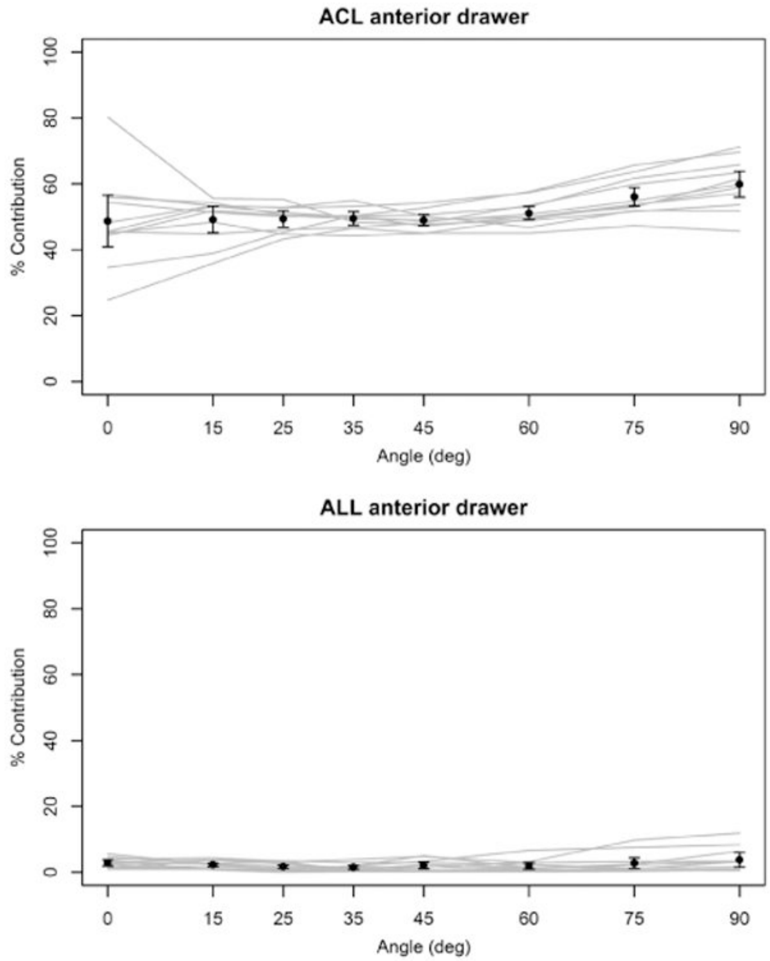

Figure 3.

The in situ force contributions (%) of the anterior cruciate ligament (ACL) and the anterolateral ligament (ALL) averaged for 11 specimens subjected to 134 N of anterior drawer force. The contribution of the lateral collateral ligament was small (see Table 3) and is not shown.

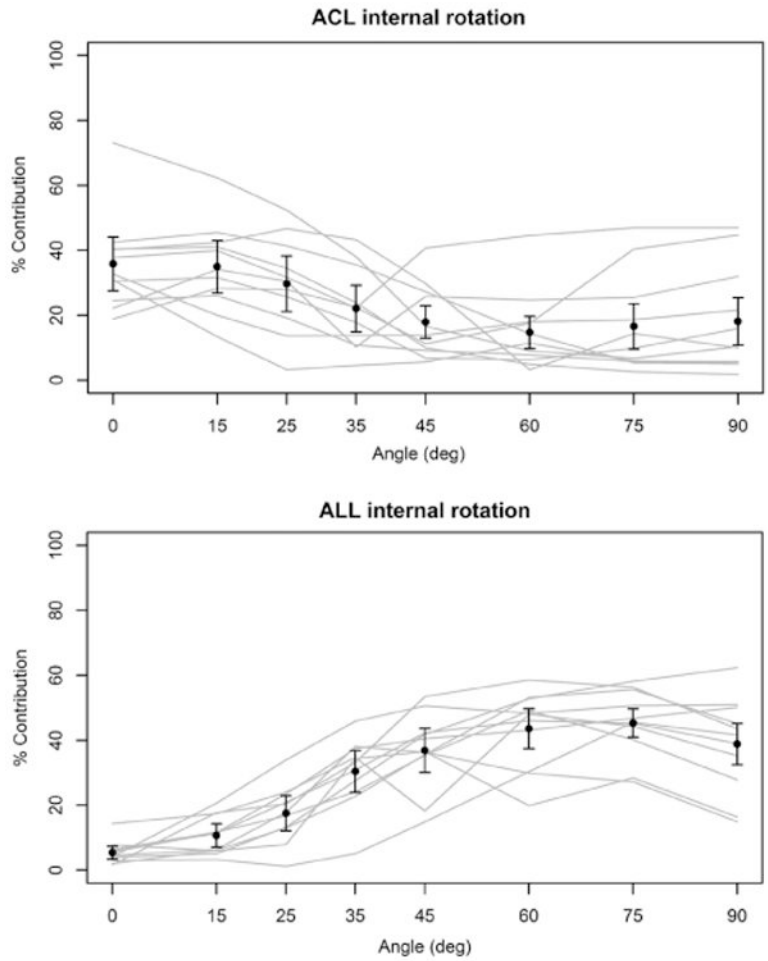

Figure 4.

The in situ force contributions (%) of the anterior cruciate ligament (ACL) and the anterolateral ligament (ALL) averaged for 11 specimens subjected to 5 N·m of internal rotation moment. The contribution of the lateral collateral ligament was small (see Table 3) and is not shown.

Table 1

Workflow for Specimen Preparation and Robotic Testing[a]

Specimen handling:

- Acquired fresh frozen at −30°C
- Thawed at room temperature 24 h before potting
- Tibia and femur cut to length and potted
- Dissection, identification, and tagging of ALL, LCL, and ACL
- Excess soft tissue removed
- Stored at −10°C for maximum of 72 h

Robotic testing:

- Anatomic landmarks established
- Joint coordinate system optimized
- Specimen preconditioned with 10 flexion cycles
- Specimen preconditioned with 1 cycle each of anterior drawer and internal rotation
- Knee tested under force control runs to establish kinematic trajectories:
  - Anterior drawer at 8 flexion angles
  - Internal rotation at 8 flexion angles
- Intact knee tested under position control using previously measured kinematic trajectories
- Knee tested with ligament 1 sectioned (randomly chosen from ALL, LCL, and ACL)
- Knee tested with ligament 2 sectioned (randomly chosen from 2 remaining ligaments)
- Knee tested with remaining ligament sectioned

[a]ACL, anterior cruciate ligament; ALL, anterolateral ligament; LCL, lateral collateral ligament.

Table 2

Kinematics of the Intact Knee: Force-Controlled Trajectory for All Specimens (n = 11)[a]

	Flexion Angle							
	0°	15°	25°	35°	45°	60°	75°	90°
Anterior tibial translation under 134 N of anterior drawer force, mm	5.4 ± 2.0	7.8 ± 2.5	8.3 ± 2.5	8.5 ± 2.5	8.1 ± 2.5	7.6 ± 2.6	7.1 ± 2.4	6.1 ± 2.5
Internal tibial rotation under 5 N·m of internal rotation moment, deg	11.5 ± 2.5	17.9 ± 4.5	20.0 ± 5.3	21.9 ± 6.0	22.6 ± 6.1	22.5 ± 6.1	22.1 ± 5.6	22.0 ± 5.9

[a]Values are reported as mean ± SD.

Table 3

Percentage Contribution to Anterior Drawer Force[a]

	ALL		ACL		LCL	
Angle	Estimate	95% CI	Estimate	95% CI	Estimate	95% CI
0°	3	2-4	49	41-57	4	2-5
15°	2	2-3	49	45-53	4	2-5
25°	2	1-2	49	47-52	2	1-3
35°	1	1-2	49	47-52	1	1-1
45°	2	1-3	49	47-51	1	1-1
60°	2	1-3	51	49-53	1	1-2
75°	3	1-4	56	53-59	1	1-2
90°	4	2-6	60	56-64	2	1-2

[a]ACL, anterior cruciate ligament; ALL, anterolateral ligament; LCL, lateral collateral ligament.

Table 4

Percentage Contribution to Internal Rotation Moment[a]

	ALL		ACL		LCL	
Angle	**Estimate**	**95% CI**	**Estimate**	**95% CI**	**Estimate**	**95% CI**
0°	5	3-7	36	28-44	5	3-7
15°	11	7-14	35	27-43	4	2-6
25°	18	12-23	30	21-38	3	2-5
35°	30	24-37	22	15-29	4	2-5
45°	37	30-44	18	13-23	4	2-5
60°	44	37-50	15	10-20	3	2-5
75°	45	41-50	17	10-24	3	2-5
90°	39	32-45	18	11-25	4	2-5

[a]ACL, anterior cruciate ligament; ALL, anterolateral ligament; LCL, lateral collateral ligament.