The Anterolateral Capsule of the Knee Behaves Like a Sheet of Fibrous Tissue

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Background: The function of the anterolateral capsule of the knee has not been clearly defined. However, the contribution of this region of the capsule to knee stability in comparison with other anterolateral structures can be determined by the relative force that each structure carries during loading of the knee.

Purpose/Hypothesis: The purpose of this study was to determine the forces in the anterolateral structures of the intact and anterior cruciate ligament (ACL)–deficient knee in response to an anterior tibial load and internal tibial torque. It was hypothesized that the anterolateral capsule would not function like a traditional ligament (ie, transmitting forces only along its longitudinal axis).

Study Design: Controlled laboratory study.

Methods: Loads (134-N anterior tibial load and 7-N·m internal tibial torque) were applied continuously during flexion to 7 fresh-frozen cadaveric knees in the intact and ACL-deficient state using a robotic testing system. The lateral collateral ligament (LCL) and the anterolateral capsule were separated from the surrounding tissue and from each other. This was done by performing 3 vertical incisions: lateral to the LCL, medial to the LCL, and lateral to the Gerdy tubercle. Attachments of the LCL and anterolateral capsule were detached from the underlying tissue (ie, meniscus), leaving the insertions and origins intact. The force distribution in the anterolateral capsule, ACL, and LCL was then determined at 30°, 60°, and 90° of knee flexion using the principle of superposition.

Results: In the intact knee, the force in the ACL in response to an anterior tibial load was greater than that in the other structures ($P < .001$). However, in response to an internal tibial torque, no significant differences were found between the ACL, LCL, and forces transmitted between each region of the anterolateral capsule after capsule separation. The anterolateral capsule experienced smaller forces (~50% less) compared with the other structures ($P = .048$). For the ACL-deficient knee in response to an anterior tibial load, the force transmitted between each region of the anterolateral capsule was 434% greater than was the force in the anterolateral capsule ($P < .001$) and 54% greater than the force in the LCL ($P = .036$) at 30° of flexion. In response to an internal tibial torque at 30°, 60°, or 90° of knee flexion, no significant differences were found between the force transmitted between each region of the anterolateral capsule and the LCL. The force in the anterolateral capsule was significantly smaller than that in the other structures at all knee flexion angles for both loading conditions ($P = .004$ for anterior tibial load and $P = .04$ for internal tibial torque).

Conclusion: The anterolateral capsule carries negligible forces in the longitudinal direction, and the forces transmitted between regions of the capsule were similar to the forces carried by the other structures at the knee, suggesting that it does not function as a traditional ligament. Thus, the anterolateral capsule should be considered a sheet of tissue.

Clinical Relevance: Surgical repair techniques for the anterolateral capsule should restore the ability of the tissue to transmit forces between adjacent regions of the capsule rather than along its longitudinal axis.

Keywords: anterolateral capsule; anterior cruciate ligament; biomechanics; in situ forces

The origin of the anterolateral capsule is close to the lateral femoral epicondyle, and its insertion is slightly inferior to the tibial articular surface posterior to the Gerdy tubercle.[2,4,9,20,21] In 1879, the French surgeon Paul Segond was the first to describe the pearly, resistant, fibrous band within this area.[20] Recently, several authors have described different anatomic origins or insertions of the anterolateral ligament.[2,21] Some investigators mention that a thickening of the anterolateral capsule itself may be the reason for these findings.

A recent study evaluating the biomechanical function of the anterolateral structures using robotic technology reported that the anterolateral structures are important stabilizers during internal rotation at higher knee flexion angles.[14] However, individual regions of the capsule were not separated from each other as an intermediate step. The effect of interactive forces between the regions of the

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capsule could have then been quantified,[8] and thus, the experimental protocol did not satisfy the principle of superposition.[17] The principle of superposition requires 3 assumptions: (1) There is no interaction between the structures of interest, (2) the bones are rigid relative to the ligaments, and (3) the positions of the bones are accurately reproduced.

Therefore, the contribution of the anterolateral knee structures to knee stability needs to be clarified. Similar to the menisci, the anterolateral capsule is a secondary stabilizer of anterior translation and rotation of the lateral compartment.[12] Increased internal rotation at 90° of knee flexion and increased anterior translation in knee flexion as well as in extension have been shown to be present in a combined injury to the anterior cruciate ligament (ACL) and the anterolateral structures.[23]

Knowledge of the in situ forces is crucial to determine the contribution of the anterolateral structures to stability after injury, which may prove important for surgical decision making. Furthermore, anatomic reconstruction procedures should replicate these forces. The aim of our study was to determine the in situ forces in the anterolateral capsule structures in response to an anterior tibial load and internal tibial torque in the ACL-intact and ACL-deficient knee using a robotic testing system.[17]

Our hypothesis was that the anterolateral capsule becomes an important restraint during application of an internal tibial torque and anterior tibial load in the ACL-deficient knee. In addition, the anterolateral capsule would not function like a traditional ligament that transmits forces only along its longitudinal axis. It would rather transmit forces between adjacent regions of the capsule perpendicular to its longitudinal axis.

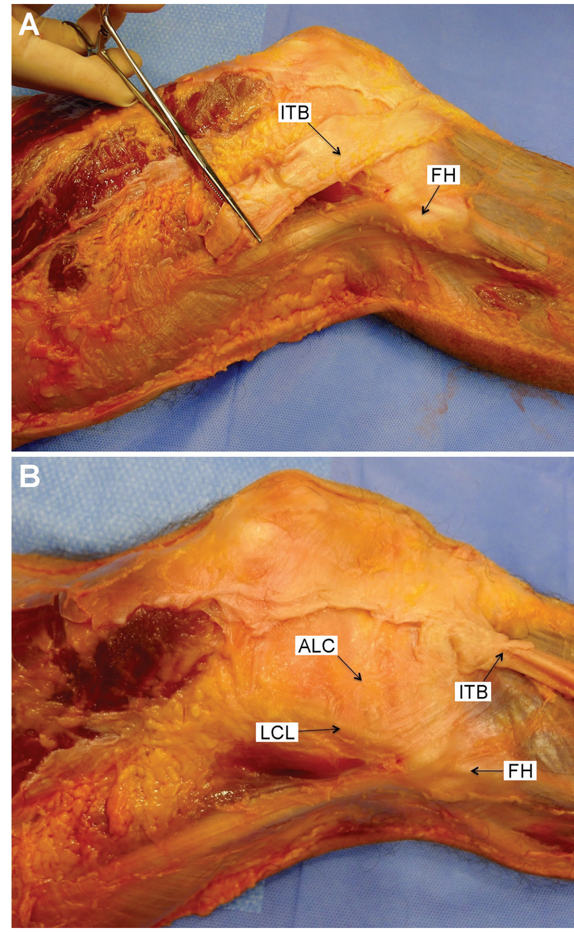

Figure 1. Lateral view on a left knee of a 46-year-old specimen. To enable visualization of the anterolateral structures, the iliotibial band was carefully dissected from the underlying tissue, (A) starting proximally and (B) ending with the transection from the Gerdy tubercle. ALC, anterolateral capsule; FH, fibula head; ITB, iliotibial band; LCL, lateral collateral ligament.

METHODS

After we obtained ethical approval, 7 fresh-frozen human cadaveric knees (mean age, 49 years; range, 46-59 years) were used in this study. Each specimen was examined manually and radiologically before testing to exclude any specimens with ligament or bony abnormalities. Specimens were thawed overnight at room temperature for 24 hours before testing,[22] and the tibia and femur were cut 20 cm from the joint line. The fibula was fixed to the tibia using a bicortical screw to maintain its anatomic position. The skin and musculature were removed, exposing the femoral and tibial shaft and leaving the knee joint intact. During the experimental protocol, the specimen was kept moist with saline.[22]

To enable visualization of the anterolateral structures, the iliotibial band was carefully dissected from the underlying tissue, starting proximal and ending with the transection from the Gerdy tubercle (Figure 1).

A pair of digital calipers (model 147; General Tools & Instruments; accuracy ±0.02 mm, repeatability 0.01 mm) was used to measure the distance between the insertion of the lateral collateral ligament (LCL) and the Gerdy

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tubercle before testing. The thickness of the anterolateral capsule was measured after cutting the structure of interest. The femur and tibia were potted in an epoxy compound (Bondo) and secured within custom-made aluminum clamps. The knee was mounted in a robotic testing system.

The robotic testing system (MJT model FRS2010) consists of a 6 degree of freedom (DOF) manipulator. A universal force-moment sensor (UFS; ATI Delta IP60 model SI-660-60) is used to provide feedback to the controller. Control of the system is accomplished through a LABVIEW Program (Technology Services Inc) designed for knee joint biomechanical testing and uses hybrid velocity impedance control. The position and orientation repeatability of the robotic manipulator is less than ±0.015 mm and $\pm0.01°$ (precision). The measurement uncertainty of the UFS is approximately 1% of full scale (accuracy).

The 6 DOF path of passive flexion-extension of the intact knee joint was first determined from full extension to 120° of knee flexion.[19] Throughout the range of motion, the positions that satisfied the condition of zero forces and moments across the joint were determined as the path of passive flexion-extension.

Two loading conditions were applied to the intact knee while the knee was continuously flexed, and the resulting kinematics were recorded.[1] The 2 loading conditions were (1) an anterior tibial load of 134 N and (2) an internal tibial torque of 7 N·m. After loading the intact knee, the ACL was transected arthroscopically. Next, operating in position-control mode, the robotic manipulator reproduced the previously recorded kinematics of the intact knee under the 2 external loads, while the UFS measured the new forces and moments. By the principle of superposition, the change in the force measured before and after ACL transection represents the in situ force in the ACL.[3,6,10,19] In addition, the 2 external loading conditions were applied to the ACL-deficient knee by the robotic testing system to determine the 6 DOF knee kinematics of the ACL-deficient knee.

The anterolateral capsule and the LCL were then separated from the surrounding tissue and from each other. This was done by performing 3 vertical incisions: lateral to the LCL, medial to the LCL, and lateral to the Gerdy tubercle (Figure 2). Attachments of the LCL and anterolateral capsule were detached from the underlying tissue (ie, meniscus), leaving the insertions and origins intact.

Next, the previously recorded kinematics of the intact knee and of the ACL-deficient knee were replayed, while the UFS measured the new forces and moments. The change in the force measured before and after anterolateral capsule separation represents the forces transmitted between anterolateral capsular regions in the intact and ACL-deficient knees. Subsequently, the anterolateral capsule was cut. Kinematics of the intact knee and of the ACL-deficient knee were replayed again, and the new forces and moments were recorded. The change in the force measured before and after cutting of the anterolateral capsule represents the in situ force of the anterolateral capsule in the intact and ACL-deficient knee. This is the force that is transmitted in a longitudinal direction. Longitudinal is defined by the direction of the vector connecting the points of insertion on the femur and tibia. Finally, the LCL was transected, again the kinematics of the intact and ACL-deficient knees were replayed, and the new forces and moments were recorded. The change in the force measured before and after cutting of the LCL represents the in situ force in the LCL (Table 1).

The in situ forces in each structure were compared using the statistical software package SPSS (version 20.0; SPSS Inc). Since the loading conditions were applied within the same knee specimen, statistical analyses were performed using a repeated 1-factor analysis of variance with multiple contrasts to analyze the variations of the in situ forces at 30°, 60°, and 90° of knee flexion; significance was set at $P < .05$.

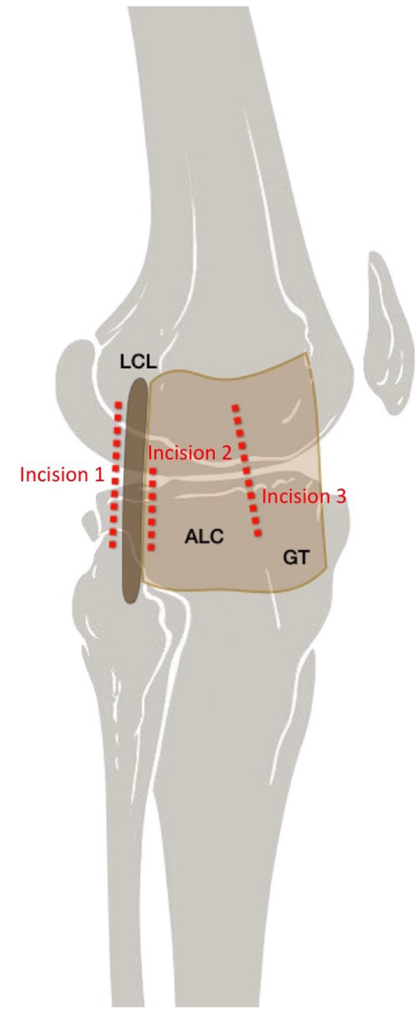

Figure 2. Model of a right human knee (lateral view). The LCL and the anterolateral capsule were separated from the surrounding tissue and from each other. This was done by performing 3 vertical incisions: (1) lateral to the LCL, (2) medial to the LCL, and (3) lateral to the Gerdy tubercle. Attachments of the LCL and anterolateral capsule were detached from the underlying tissue (ie, meniscus), leaving the insertions and origins intact. ALC, anterolateral capsule; GT, Gerdy tubercle; LCL, lateral collateral ligament.

TABLE 1
The Experimental Protocol and Data Acquired[a]

Step of Injury	Loading Conditions/Replays	Data Acquired
Intact	134-N ATL, 7-N·m ITT	Kinematics of intact knee (1)
Cut ACL	Replay 1	Force of ACL in intact knee
	134-N ATL, 7-N·m ITT	Kinematics of ACL-deficient knee (2)
Separate ALC	Replay 1	Force of ALC Sep in intact knee
	Replay 2	Force of ALC Sep in ACL-deficient knee
Cut ALC	Replay 1	Force of ALC in intact knee
	Replay 2	Force of ALC in ACL-deficient knee
Cut LCL	Replay 1	Force of LCL in intact knee
	Replay 2	Force of LCL in ACL-deficient knee

[a]ACL, anterior cruciate ligament; ALC, anterolateral capsule; ALC Sep, anterolateral capsule separation (forces transmitted between capsular regions); ATL, anterior tibial load; ITT, internal tibial torque; LCL, lateral collateral ligament.

RESULTS

Macroscopic evaluation revealed a thickening of the anterolateral capsule with fibers running from the lateral femoral epicondyle to the tibia midway between the Gerdy tubercle and fibula head in 2 out of 7 specimens. The thickness of the anterolateral capsule was 0.34 ± 0.24 cm. The distance between the LCL and the Gerdy tubercle was 2.65 ± 0.54 cm.

In comparison with the intact knee, cutting of the ACL increased anterior tibial translation by 12.4 ± 2.4, 10.8 ± 3.2, and 6.4 ± 2.0 mm and internal rotation by 1.8° ± 1.1°, 1.1° ± 0.5°, and 1.0° ± 0.4° at 30°, 60°, and 90° of knee flexion, respectively.

In response to an anterior tibial load with the knee at 30°, 60°, or 90° of knee flexion, the in situ forces in the ACL were significantly greater than the forces in all other structures ($P < .001$) (Figures 3A and 4A). With the knee at 30° of flexion, the in situ forces in the ACL were 1015%, 457%, and 926% greater than the in situ forces in the LCL, the forces transmitted between each region of the anterolateral capsule, and the in situ forces of the anterolateral capsule, respectively. Similar findings occurred at 60° and 90° of knee flexion.

In response to an internal tibial torque with the intact knee, no significant differences in the forces occurred between the ACL and LCL, as well as in the forces transmitted between each region of the anterolateral capsule (Figures 3B and 4B). The anterolateral capsule experienced significantly smaller forces compared with all other structures tested ($P = .048$).

In the ACL-deficient knee, in response to an anterior tibial load with the knee at 30° of flexion, the forces transmitted between each region of the anterolateral capsule were 434% greater than the in situ forces in the anterolateral capsule ($P < .001$) and 54% greater than the in situ forces in the LCL ($P = .036$) (Figures 3C and 4C). With the knee at 60° and 90° of flexion, there were no significant differences in the forces transmitted between each region of the anterolateral capsule and the forces in the LCL. However, the forces transmitted between each region of the anterolateral capsule were still significantly higher than the forces in the anterolateral capsule ($P = .004$).

In response to an internal tibial torque at 30°, 60°, or 90° of knee flexion, no significant differences were found between the forces transmitted between each region of the anterolateral capsule and the forces in the LCL (Figures 3D and 4D). The anterolateral capsule experienced negligible forces (81%, 74%, and 72% lower than the LCL and 88%, 79%, and 74% lower than the forces transmitted between each region of the anterolateral capsule at 30°, 60°, and 90° of knee flexion, respectively) ($P = .04$).

DISCUSSION

The most important finding of this study was that separation of the anterolateral capsule into its components revealed large forces were transmitted between the anterolateral capsular regions. However, the forces in the longitudinal direction were negligible, suggesting that the anterolateral capsule does not function like a traditional ligament that supports tensile force along its length.

The results are characteristic of a sheet of tissue as has been described for other joints (eg, shoulder).[13] The data suggest that the anterolateral capsule experiences larger in situ forces in response to an anterior tibial load and internal tibial torque in the ACL-deficient knee than in the intact knee. However, the anterolateral capsule transmits larger forces perpendicular to its longitudinal axis than in the longitudinal direction. In the intact knee, the ACL is the primary restraint against anterior tibial load. However, the ACL, LCL, and anterolateral capsule before separation transmitted load in response to internal tibial torque.

To date, ongoing debate exists in the literature on the biomechanical function of anterolateral structures in the human knee. A distinct function of an anterolateral ligament was recently proposed for rotatory and translational stability,[14] sparking a controversial discussion.[7,8,15] Other authors highlighted the importance of other anterolateral structures, such as the iliotibial band.[11,16] Based on our results, a proposed ligament or thickening of the capsule does not have a significant role in knee stability in humans. These findings do not agree with the previous study[14] that suggested the anterolateral ligament is an important stabilizer of internal rotation at higher knee flexion angles. However, contrary to the previous

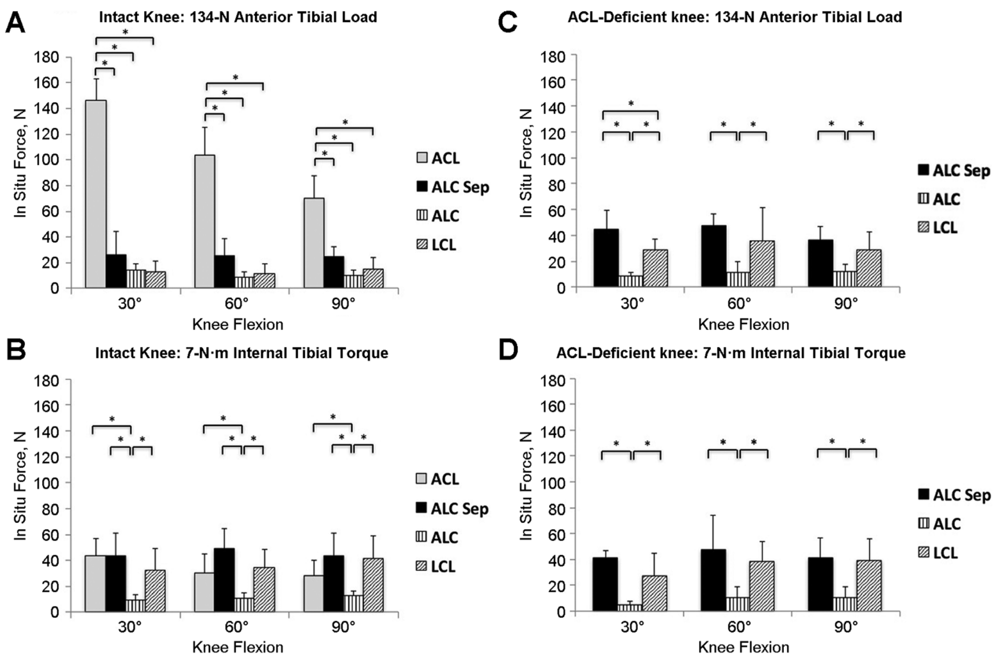

Figure 3. In situ forces in response to (A) a 134-N anterior tibial load and (B) a 7-N·m internal tibial torque in the intact knee and in response to (C) a 134-N anterior tibial load and (D) a 7-N·m internal tibial torque in the ACL-deficient knee. ACL, anterior cruciate ligament; ALC, anterolateral capsule; ALC Sep, anterolateral capsule separation (forces transmitted between anterolateral capsular regions); LCL, lateral collateral ligament. *Statistically significant difference between groups ($P < .05$).

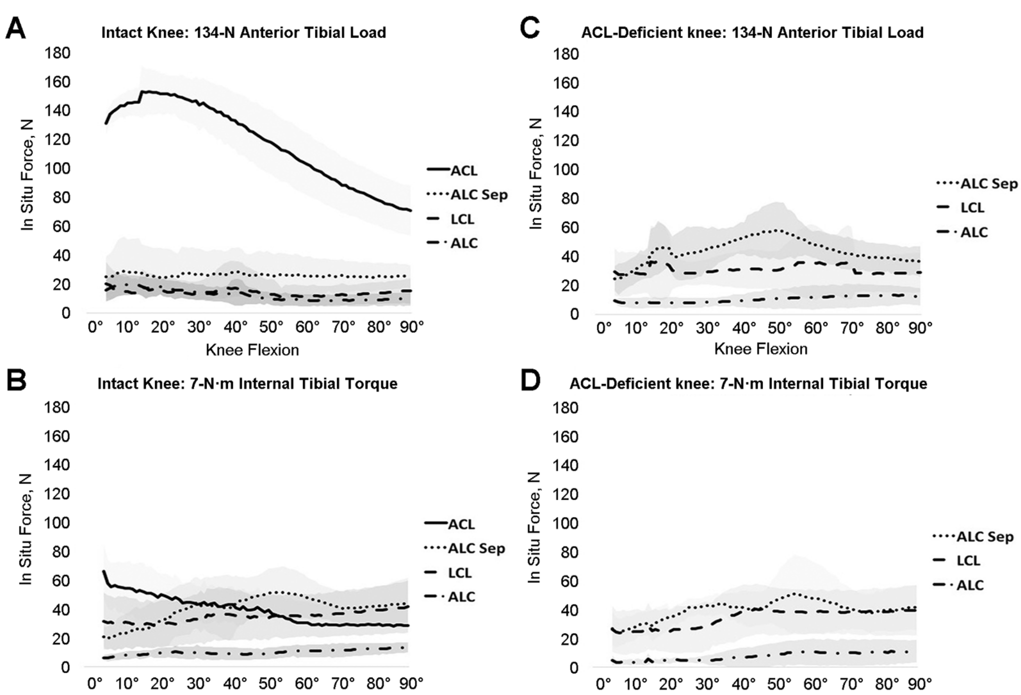

Figure 4. In situ forces in response to (A) a 134-N anterior tibial load and (B) a 7-N·m internal tibial torque in the intact knee and in response to (C) a 134-N anterior tibial load and (D) a 7-N·m internal tibial torque in the ACL-deficient knee. ACL, anterior cruciate ligament; ALC, anterolateral capsule; ALC Sep, anterolateral capsule separation (forces transmitted between anterolateral capsular regions); LCL, lateral collateral ligament. The shaded areas represent the SDs.

study, capsule separation was performed to meet the assumption required for the principle of superposition.[17] The results lead to the assumption that the interaction between the tested structures plays a significant role in knee stability and that the anterolateral capsule should be handled as a sheet of tissue rather than a composition of individual components.

In addition to the present study, different approaches have been applied to evaluate the biomechanical function of the anterolateral structures. Transection of the anterolateral ligament did not increase tibiofemoral translation or rotation, when evaluated by a mechanized pivot shifter in an ACL-deficient cadaveric model.[18] The change in length of the anterolateral capsule was also measured using metal eyelets, a monofilament suture, and a linear variable displacement transducer. The anterolateral ligament was found to be isometric from 0° to 60° of knee flexion, slackened when the knee flexed to 90°, and was lengthened by imposing tibial internal rotation.[4] Another study[24] measured the strain in the anterolateral ligament using polydimethylsiloxane gauges. The authors concluded that the anterolateral ligament was a nonisometric structure that tensions with knee flexion and internal tibial rotation. The inconsistent findings of current studies could be explained by different anatomic descriptions of the anterolateral ligament. On the basis of the present study, it can be speculated that a thickening of the capsule rather than a traditional ligament may be present in variable localizations. In addition, failure to properly use the principle of superposition could result in further differences.

The role of the anterolateral capsule as a restraint to internal tibial torque in the intact knee and anterior tibial load in the ACL-deficient knee may explain the presence of a thickening in the capsular region as has been described in anatomic dissections.[5,16] Instead of a discrete ligamentous structure in the anterolateral capsule, the capsule may tend to thicken in light of tissue remodeling.

The findings of the present study question whether injuries to the anterolateral capsule should be treated at the time of ACL surgery. A reason for persistent rotatory instability after ACL reconstruction may be an injury of the anterolateral capsule. Further research is required to evaluate the healing potential of anterolateral capsule structures and the influence of different reconstruction techniques on joint kinematics. Surgical repair techniques for the anterolateral capsule should restore the ability of the tissue to transmit forces between adjacent regions of the capsule rather than along its longitudinal axis. Treatment algorithms have to be established based on the quantitative data defining normal soft tissue function and the grade of rotatory instability.

One limitation of our study is that only the force in the lateral structures was determined. The in situ forces of the medial and posterior structures, as well as the in situ forces in the menisci, were not assessed. Second, the iliotibial band adds rotational stability to the knee in vivo. However, to visualize the capsule and fulfill the assumptions of the principle of superposition, the iliotibial band had to be removed.

In summary, the ACL is the primary restraint to anterior tibial translation in the intact knee. The anterolateral capsule is a restraint to internal rotation in the intact knee and becomes an important restraint to anterior tibial load in the ACL-deficient knee. However, a proposed ligamentous structure within the anterolateral capsule does not function as a traditional ligament.

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