Effect of ACL Transection on Internal Tibial Rotation in an in Vitro Simulated Pivot Landing

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Background: The amount of resistance provided by the ACL (anterior cruciate ligament) to axial tibial rotation remains controversial. The goal of this study was to test the primary hypotheses that ACL transection would not significantly affect tibial rotation under the large impulsive loads associated with a simulated pivot landing but would increase anterior tibial translation.

Methods: Twelve cadaveric knees (mean age of donors [and standard deviation] at the time of death, 65.0 ± 10.5 years) were mounted in a custom testing apparatus to simulate a single-leg pivot landing. A compound impulsive load was applied to the distal part of the tibia with compression (~ 800 N), flexion moment (~ 40 N-m), and axial tibial torque (~ 17 N-m) in the presence of five trans-knee muscle forces. A differential variable reluctance transducer mounted on the antero-medial aspect of the ACL measured relative strain. With the knee initially in 15° of flexion, and after five combined compression and flexion moment (baseline) loading trials, six trials were conducted with the addition of either internal or external tibial torque (internal or external loading), and then six baseline trials were performed. The ACL was then sectioned, six baseline trials were repeated, and then six trials of either the internal or the external loading condition, whichever had initially resulted in the larger relative ACL strain, were carried out. Tibiofemoral kinematics were measured optoelectronically. The results were analyzed with a nonparametric Wilcoxon signed-rank test.

Results: Following ACL transection, the increase in the normalized internal tibial rotation was significant but small ($0.7^\circ/\text{N-m} \pm 0.3^\circ/\text{N-m}$ to $0.8^\circ/\text{N-m} \pm 0.3^\circ/\text{N-m}$, $p = 0.012$), while anterior tibial translation increased significantly (3.8 ± 2.9 to 7.0 ± 2.9 mm, $p = 0.017$).

Conclusions: ACL transection leads to a small increase in internal tibial rotation, equivalent to a 13% decrease in the dynamic rotational resistance, under the large forces associated with a simulated pivot landing, but it leads to a significant increase in anterior tibial translation.

Clinical Relevance: An ACL reconstruction that restores both ligament orientation and stiffness will provide major resistance to anterior tibial translation while providing minor resistance to axial tibial rotation.

Over 200,000 anterior cruciate ligament (ACL) injuries are treated with surgical reconstruction annually in the United States¹. Despite surgical treatment, the development of posttraumatic degenerative disease, perhaps as a result of impaired knee joint kinematics, has been reported². To better restore knee joint kinematics and reduce the risk of degenerative changes of the knee joint, alternative surgical techniques have been introduced, including a double-bundle reconstruction and a single-bundle reconstruction with more oblique ACL graft orientation in the sagittal and coronal planes³. The goal of both techniques is to improve knee joint kinematics in axial tibial rotation in addition to

anterior tibial translation. While it is well documented that the ACL plays a primary role in restraining anterior tibial translation, its role in limiting axial tibial rotation remains controversial⁴.

The results of previous in vivo and in vitro studies regarding the role of the ACL in limiting axial tibial rotation are not in agreement⁵⁻¹⁰. Recently, Markolf et al. reported an increase in internal tibial rotation ($7.3^\circ \pm 3.4^\circ$ and $4.0^\circ \pm 2.8^\circ$ at a 0° and 30° knee flexion angle, respectively) under 5 N-m of internal tibial torque during a passive knee extension test performed after removal of the ACL⁵. Similarly, Lipke et al. found that sectioning of the ACL caused less than a 5° increase

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in internal tibial rotation, which was significant when the cadaveric knee specimens were quasi-statically tested under 10 N-m of axial tibial torque in the presence of the quadriceps muscle force⁶. An in vivo study by DeFratre et al. showed that ACL deficiency resulted in a significant increase in both anterior tibial translation (~3 mm) and internal tibial rotation (~2°) at a low knee flexion angle during a quasi-static lunge¹¹. In contrast, in an in vitro study in which 5 N-m of internal or external tibial torque was quasi-statically applied to quadriceps-stabilized knee specimens and the knee flexion angle was varied from 0° to 60°, Lane et al. found that the increases in internal tibial rotation (<4°) were insignificant under both weight-bearing (89 N) and non-weight-bearing conditions⁷. Similarly, Wroble et al. reported that the 2° ~ 3° increase in internal tibial rotation that they found was too small to be clinically important despite its statistical significance⁹. These differing observations regarding the functional implications of ACL deficiency in terms of knee axial rotational resistance were quantified with use of quasi-static biomechanical outcome measurements, usually in the absence of muscle forces or under modest test loads. Snyder-Mackler et al. demonstrated that passive joint laxity is poorly correlated with the patient's functional or dynamic ability¹². Given how common large dynamic knee loads are in vivo¹³, it seemed worthwhile to investigate how ACL transection affects axial tibial rotation in the presence of muscle forces and under the large dynamic loads associated with a pivot landing involving an internal tibial torque.

The anterior tibial translation that is coupled with tibial rotation has been measured in order to determine the degree of "rotational resistance" provided by the ACL under axial tibial torques¹⁴⁻¹⁶. Kanamori et al. observed significant increases in both internal tibial rotation and coupled anterior tibial translation in response to either 10 N-m of internal tibial torque, or a combined torque of 10 N-m of internal tibial torque and 10 N-m of valgus moment¹⁴. Although the increase in internal tibial rotation was significant, the magnitude of the internal tibial rotation was <3° at low flexion angles. Diermann et al. obtained similar results when they employed a robotic methodology to investigate the effect of ACL deficiency under a combined torque consisting of 10 N-m of valgus moment and 4 N-m of internal tibial torque¹⁷. Thus, they concluded that the anterior tibial translation, rather than the internal tibial rotation, should be evaluated for objective assessments of the contribution provided by the ACL to resistance of a combined rotatory load. It is worth noting that these data appear to be contradictory because internal tibial rotation can cause ACL strain, but the increase in internal tibial rotation following ACL transection is not as large as might be expected. The goal of this study, therefore, was to examine the effect of ACL transection on axial tibial rotation and anterior tibial translation in cadaveric knees placed under compound impulsive loading in the presence of simulated muscle forces. The primary hypothesis was that transecting the ACL would not increase tibial axial rotation but would increase anterior tibial translation.

Materials and Methods

Specimen Procurement and Preparation

Twelve unembalmed cadaveric limbs from eight female donors (mean age [and standard deviation] at the time of death, 65.0 ± 10.5 years; mean body weight, 655.0 ± 147.2 N), with four contributing two limbs, were acquired from the University of Michigan Anatomical Donations Program. All lower extremities were visually checked for scars, other indications of surgery, and severe malalignments or deformities. Prior to specimen procurement, frontal plane digital images of axially compressed and fully extended knees were obtained to determine the natural alignment angles. With use of the methods of Withrow et al.¹⁸⁻²⁰, the lower extremities were cut ~8 in (~20 cm) proximal and distal to the knee joint in order to standardize specimen length. The specimens were then dissected, with the ligamentous knee structures and the tendons of the quadriceps, medial hamstring, lateral hamstring, and medial and lateral gastrocnemius muscles left intact. Each end of the specimen was potted in polymethylmethacrylate. The frontal plane alignment of the knee specimens was reproduced on the basis of the frontal plane digital images. The dissected specimens were kept frozen at -20°C until they were needed and were thawed at room temperature for at least twelve hours before testing commenced.

Knee Testing Apparatus

The testing apparatus developed by Withrow et al.¹⁸⁻²⁰ was used to simulate the position of a single extremity as it strikes the ground while a person lands on one leg during a jump or pivot maneuver. The simulated quadriceps and medial and lateral gastrocnemius muscles were represented by elastic structures (~2-mm-diameter woven nylon cord with a tensile stiffness of ~2 kN/cm) pretensioned to 180 and 70 N, respectively¹⁸. Two custom-made constant force springs (each pretensioned to 70 N) were used to represent the medial and lateral hamstring muscle forces. Each simulated muscle-tendon unit was connected from the femur or tibia to the relevant tendons via cryoclamps through the anatomical lines of action, thereby representing its in vivo dynamic resistance to sudden stretch. In all trials, an initial knee flexion angle of 15° was maintained. A jump-landing ground reaction force was simulated by dropping a weight onto the distal end of the tibia of an inverted knee specimen so as to generate a 2 × BW (where BW is each donor's postmortem body weight) impulsive force. The weight drop was initiated in a standardized manner via a quick-release mechanism. A customized shock absorber (ACE Controls, Farmington Hills, Michigan) was attached in series at the distal end of the drop weight so that the ground reaction force peaked at 50 msec. In a new departure from the original apparatus developed by Withrow et al., the linear momentum of the weight at impact was transformed into the combination of an axial compressive force and an impulsive axial torque component (peaking at 70 msec) by a specially designed torsional transformer device mounted in series with the distal part of the tibia (Fig. 1). The device consisted of two circular plates between which three inclined palls are located, the angle of the

Fig. 1
Schematic diagram showing how a weight (W) dropped from a fixed height onto the torsional transformer device (T) applies impulsive compression and axial torque to the distal part of the tibia of an inverted knee specimen. The two load transducers (L) measure the three components of force and moment applied to the knee specimen in the presence of quadriceps (Q), hamstring (H), and gastrocnemius (G) forces, while a miniature displacement transducer (ϵ) implanted on the surface of the anteromedial bundle of the ACL measures strain.

palls to the vertical being adjustable and preset to give the desired gain between the applied compressive force and the rotational torque delivered to the specimen. The peak axial torque was adjusted to reach a nominal value between 10 and 25 N-m and was recorded.

As in the apparatus developed by Withrow et al.¹⁸⁻²⁰, two six-degrees-of-freedom load cells (AMTI, Watertown, Massachusetts) measured the three-dimensional tibial forces and moments delivered to the construct as well as the three-dimensional femoral reaction forces and moments. A 3-mm differential variable reluctance transducer (MicroStrain, Burlington, Vermont) was mounted on the anteromedial bundle of

the ACL to record ACL relative strain. In order to standardize the location of the differential variable reluctance transducer on the anteromedial bundle of the ACL, the transducer was placed at the first quartile of the ACL length measured from the tibial attachment site of the ACL.

Five single-degree-of-freedom load cells (Transducer Techniques, Temecula, California) measured simulated muscle tensions. Impulsive forces, the five muscle forces, and ACL strain data were recorded at 2 kHz with use of a 16-bit analog-to-digital converter board, while tibiofemoral kinematics, which were defined in accordance with the work by Grood and Suntay²¹, were recorded at 400 Hz with use of an Optotrak Certus system (Northern Digital, Waterloo, Ontario, Canada).

Testing Protocol (Table I)

The baseline loading condition (Base1, Base2, and Base3) was designed to simulate a drop landing in which the impulsive ground reaction force provides the compressive force on the knee joint and induces the knee flexion, thereby causing sudden stretch of the quadriceps muscle-tendon unit. In addition, the stretch of the quadriceps muscle-tendon unit resulted in the posterior femoral displacement, thereby straining the ACL. For the axially directed loading condition (Int1, Ext1, Int2, and Ext2), either internal or external tibial torque was added to the baseline loading condition.

During the first five preconditioning trials, the height of the weight drop was varied to find the drop height that best simulated a $2 \times \text{BW}$ impulsive ground reaction force for the baseline loading condition. That drop height was then maintained throughout all trials to apply the same kinetic energy to the knee specimens. After the five pre-baseline trials (Base1), three blocks of six trials were run on each ACL-intact specimen in a Base1-B-C-Base2 design, where the blocks B and C were randomized to be either an internally directed (Int1) or an externally directed (Ext1) axial tibial torque combined with a standard compression force and a flexion moment, followed by the post-baseline trial block (Base2). The ACL was then transected. Two blocks of six trials were then repeated on each ACL-deficient specimen in a Base3-D design, where Base3 was the baseline trial for the ACL-deficient specimens and D (either Int2 or Ext2) was applied in the direction of the axial tibial torque that had resulted in the larger ACL relative strain in the B and C conditions. The first trial for each configuration was considered a preconditioning trial, and the last five trials were used to calculate the biomechanical variables.

Statistical Analysis

The peak internal tibial rotation for each of the ten specimens that fulfilled the Int2 condition during the internally directed trials was normalized by the peak internal tibial torque. This was done for the last five trials under each loading condition (i.e., Int1 and Int2), and the values were averaged for each specimen. The primary hypotheses that were tested were (1) the normalized peak internal tibial rotation for the ACL-deficient state would be no different from that for the ACL-intact state, and (2) the anterior tibial translation would

significantly increase following ACL transection. A nonparametric Wilcoxon signed-rank test was used to test the hypotheses. An alpha level of 0.05 was chosen for the level of significance.

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Results

Ten of the twelve specimens exhibited larger peak ACL relative strain under the internal tibial axial torque than under the external tibial torque. According to the protocol, these ten were therefore retested, after ACL transection, under

the combination of the standard compression force, flexion moment, and internal tibial torque in the testing condition, D, in order to test the primary hypotheses. The remaining two specimens were tested under external tibial torque. Sample temporal behaviors from a single specimen and trial are shown in Figure 2.

There were no differences between two baseline loading conditions (Base1 and Base2) for the ACL-intact state, thereby confirming that the knee specimens were not damaged during the testing.

Effect of ACL Transection on Internal Tibial Rotation

Under the baseline loading conditions, in the absence of any applied axial tibial torque, the internal tibial rotations were

(a) Sample Plots for Baseline Trials

(b) Sample Plots for Internally-directed Trials

Fig. 2

Sample temporal behavior of the applied impulsive compressive force, resulting quadriceps and hamstring force, internal tibial torque, knee flexion angle, anterior tibial translation, internal tibial rotation, and ACL relative strain from a single loading trial under the baseline (a) and internally directed loading (b) conditions for one specimen. Measurements have been normalized to their peak values (see keys).

TABLE I Testing Protocol

Protocol*	Loading Type
ACL-intact condition	
Base1	Compression + flexion moment
Int1†	Compression + flexion moment + internal tibial torque
Ext1†	Compression + flexion moment + external tibial torque
Base2	Compression + flexion moment
ACL-deficient condition	
Base3	Compression + flexion moment
Int2 or Ext2‡	Compression + flexion moment + internal or external tibial torque

*See text for abbreviations. †The sequence of the Int1 and Ext1 tests was randomized. ‡The direction of the axial tibial torque was chosen on the basis of whether the Int1 or Ext1 resulted in the larger ACL strain.

$1.6^\circ \pm 1.2^\circ$ and $2.5^\circ \pm 1.5^\circ$ for the ACL-intact (Base1) and ACL-deficient (Base3) conditions, respectively (Fig. 3). The internal tibial torque induced internal tibial rotations of $11.6^\circ \pm 2.8^\circ$ and $12.1^\circ \pm 3.0^\circ$ for the ACL-intact (Int1) and ACL-deficient (Int2) conditions, respectively (Fig. 3).

In the test of the first primary hypothesis, the increase in the normalized internal tibial rotation caused by ACL

transection was significant under the compound loading with internal tibial torque (normalized internal rotation, $0.7^\circ/\text{N-m} \pm 0.3^\circ/\text{N-m}$ and $0.8^\circ/\text{N-m} \pm 0.3^\circ/\text{N-m}$ for the ACL-intact [Int1] and ACL-deficient [Int2] states, respectively; $p = 0.012$) (Fig. 3). These findings correspond to a 13% decrease in the dynamic rotational resistance in the ACL-deficient state compared with that in the ACL-intact state.

Effect of ACL Transection on Anterior Tibial Translation

In the absence of any applied axial tibial torque, the anterior tibial translations of the center of the tibial plateau were 1.2 ± 1.1 mm and 3.9 ± 2.1 mm for the ACL-intact (Base1) and ACL-deficient (Base3) conditions, respectively ($p = 0.009$) (Fig. 4). In the presence of the internal tibial torque, the anterior tibial translation of the center of the tibial plateau was increased from 3.8 ± 2.9 mm for the ACL-intact condition (Int1) to 7.0 ± 2.9 mm for the ACL-deficient condition (Int2) ($p = 0.017$) (Fig. 4). These findings supported the second primary hypothesis.

In order to investigate the portion of the anterior tibial translation caused by the internal tibial torque, the increments due to the addition of internal tibial torque to the baseline loading condition were calculated by subtracting the anterior tibial translation for the baseline conditions (Base1 or Base3) from the corresponding value for the internally directed loading conditions (Int1 or Int2). After ACL transection, these increments were 2.6 ± 2.0 mm and 3.0 ± 1.5 mm for the ACL-

Fig. 3

a: Mean peak internal tibial rotation (and standard deviation, represented by the error bars) under the baseline and internally directed loading conditions for the ACL-intact (Base1 and Int1) and ACL-deficient (Base3 and Int2) states. ACL transection resulted in an increase of 0.9° and 0.5° in internal tibial rotation for the baseline and internally directed loading conditions, respectively. b: Normalized internal tibial rotation in the presence of the internal tibial torque for the ACL-intact (Int1) and ACL-deficient (Int2) conditions. The asterisk indicates a significant difference (see text).

TABLE II Peak Impulsive Compressive Force, Internal Tibial Torque, and Quadriceps Force for All ACL-Intact and ACL-Deficient Knees Loaded Under Internal Tibial Torque*

	Impulsive Compressive Force (N)	Internal Tibial Torque (N-m)	Quadriceps Force (N)
ACL-intact (Int1)	866.1 ± 101.0	17.1 ± 3.9	1055 ± 226
ACL-deficient (Int2)	777.7 ± 140.6	16.6 ± 4.7	1108 ± 163

*The values are given as the mean and standard deviation.

intact and ACL-deficient states, respectively (compare $\Delta 1$ with $\Delta 2$ in Fig. 4).

Summary of Flexion Angle, Impulsive Compressive Force, Quadriceps Force, and Internal Tibial Torque

Table II summarizes the mean peak values (and standard deviations) of knee flexion angle, impulsive compressive force, quadriceps force, and internal tibial torque over the test conditions. In the absence of any applied axial tibial torque, the peak knee flexion angles were $5.0^\circ \pm 1.3^\circ$ and $5.3^\circ \pm 1.4^\circ$ for the ACL-intact (Base1) and ACL-deficient (Base3) conditions, respectively. In the presence of the internal tibial torque, the peak knee flexion angles were $5.0^\circ \pm 1.3^\circ$ and $4.9^\circ \pm 3.0^\circ$ for the ACL-intact (Int1) and ACL-deficient (Int2)

conditions, respectively. The peak impulsive compressive forces under the internally directed tibial torque were $\sim 1.3 \times \text{BW}$ and $\sim 1.2 \times \text{BW}$ for the ACL-intact (Int1) and ACL-deficient (Int2) conditions, respectively, whereas the corresponding values in the absence of any applied axial tibial torque were $1299.3 \pm 217.6 \text{ N}$ ($\sim 1.7 \times \text{BW}$ to $\sim 2.3 \times \text{BW}$) and $1206.8 \pm 126.5 \text{ N}$ ($\sim 1.6 \times \text{BW}$ to $\sim 2.0 \times \text{BW}$) for the ACL-intact (Base1) and ACL-deficient (Base3) conditions, respectively. On the other hand, the resulting quadriceps forces were $1040.9 \pm 235.0 \text{ N}$ and $1079.4 \pm 182.8 \text{ N}$ for the ACL-intact (Base1) and ACL-deficient (Base3) conditions, respectively; these values were similar to the corresponding values under the internally directed loading conditions (Table II).

Fig. 4

Mean values (and standard deviations) for anterior tibial translation (left) and relative strain in the anteromedial bundle of the ACL (AM-ACL) (right) across all specimens. The addition of the internal tibial torque to the baseline loading condition increased the relative strain in the anteromedial bundle of the ACL by 108% (right). This increase in the relative strain in the anteromedial bundle of the ACL is consistent with the increase in the anterior tibial translation (compare Base1 and Int1, at left). Following the ACL transection, the anterior tibial translation significantly increased under both the baseline and internally directed loading conditions. The asterisks indicate a significant difference (see text).

Discussion

This study was designed to evaluate the contribution of the ACL to resistance to axial rotation of the tibia under dynamic loading conditions similar to a pivot landing maneuver involving internal tibial axial rotation. Authors of previous *in vitro*⁵⁻⁹ and *in vivo*¹⁰ studies have used static or quasi-static loading conditions employing low force levels. Those studies are relevant because clinical examinations are often performed quasi-statically to measure the degree of passive knee laxity. However, Snyder-Mackler et al.¹² demonstrated that passive joint laxity is poorly correlated with the patient's functional or dynamic ability. Because daily mobility tasks and sports maneuvers induce large dynamic loads on the knee^{22,23}, we investigated the effect of ACL transection on knee kinematics under combinations of impulsive loading conditions (compressive force, flexion moment, and axial tibial torque) with simulated muscle-tendon unit forces. In our baseline testing, the dynamic loading caused the increase in the knee flexion angle to stretch the quadriceps muscle-tendon unit and increase its force, thereby causing the femur to translate posteriorly relative to the tibia so as to increase ACL strain. Then, internal tibial torque was added to the baseline loading configuration. Overall, the ACL played a secondary role by providing 13% of the total dynamic rotational resistance to the applied internal tibial torque. Although the internal tibial rotation was increased as a result of the ACL deficiency, the increase is probably too small to be clinically relevant.

The ACL transection resulted in a significant increase in the normalized internal tibial rotation. However, the increase in the internal tibial rotation was small (a 0.5° difference between the ACL-intact [Int1] and ACL-deficient [Int2] states; Fig. 3). Previous studies of smaller quasi-static forces in the absence of muscle forces, or in the presence of low-level muscle forces, have demonstrated larger increases in internal tibial rotation following ACL transection. For example, Markolf et al.⁵ reported the largest increase in internal tibial rotation ($7.3^\circ \pm 3.4^\circ$ and $4.0^\circ \pm 2.8^\circ$ at 0° and 30° of knee flexion, respectively) under 5 N-m of internal tibial torque during a passive knee extension test. Similarly, Lipke et al.⁶ and Lane et al.⁷ found a ~4° to ~5° increase in internal tibial rotation. Other studies showed a less than or slightly larger than 3° increase in internal tibial rotation after ACL transection^{8,9,14,17,24,25}. The most plausible explanation for the larger values of internal tibial rotation in those studies is the absence of either large muscle forces or large compressive force. Previous studies demonstrated that axial compressive load induced by either external load or quadriceps muscle contraction reduces the total anterior-posterior translation of the tibia relative to the femur^{26,27}. Wojtyś et al. demonstrated more directly that the internal tibial rotation in the presence of active muscle contractions was smaller than that in the passive muscle state²⁸. The large trans-knee external and muscular compressive forces acting in the present study may have resulted in better conformity of the femoral condyle, tibial plateau, and meniscus, leading to increased resistance to internal tibial torque. In addition, a large quadriceps force translates the femur poste-

riorly, thereby tightening the peripheral ligaments to make them more effective in resisting internal tibial torque. Wroble et al. showed that cutting the ACL produced only a small, but significant, increase in internal tibial rotation, but that subsequent sectioning of the anterolateral structures produced a significant increase in internal tibial rotation⁹. They concluded that internal tibial rotation is restrained more by the lateral extra-articular structures than by the ACL. Thus, our results imply that the increase in internal tibial rotation due to ACL insufficiency is typically small during a pivot landing.

The contribution by the ACL to the dynamic rotational resistance was estimated by calculating the difference in the reciprocal of the normalized internal tibial rotation between the ACL-intact (Int1) and ACL-deficient (Int2) states. In our pivot landing scenario, the mean contribution by the ACL corresponds to 13% of the dynamic rotational resistance for the ACL-intact knees. This is a modest contribution when compared with the finding, by Butler et al., that the ACL resists 86% of anterior tibial force⁴. This minor contribution of the ACL to the dynamic rotational resistance can be explained by the functional anatomy—i.e., the fact that, because the attachment site of the ACL on the tibial plateau is near the longitudinal axis of the knee joint^{29,30}, the moment arm is too short to be effective in resisting internal tibial torque³¹.

As shown in Figure 4, the addition of the internal tibial torque to the baseline loading condition increased the anterior tibial translation, thereby increasing the strain of the ACL by a factor of two. However, despite the resulting stretch on the ACL, which would have contributed to the dynamic rotational resistance, the increase in the internal tibial rotation was small (compare Int1 with Int2 in Fig. 3, *a*). Authors of previous quasi-static studies have observed coupled anterior tibial translation under an isolated internal tibial torque, or under a combined internal tibial torque and valgus torque^{17,32}. In a comparison of ACL-deficient knees (those with ACL transection) with ACL-intact knees, Diermann et al.¹⁷ found no significant increase in internal tibial rotation but a significant increase in anterior tibial translation under a combination of 10 N-m of valgus moment and 4 N-m of internal tibial torque. Our results corroborate their findings that anterior tibial translation was the primary variable altered following ACL transection when the knee was subjected to the combined rotatory load. These findings appear to be contradictory because internal tibial rotation can cause coupled anterior tibial translation, thereby straining the ACL, but the contribution of the induced ACL force with regard to limiting internal tibial rotation is minor. However, the findings can be reconciled by considering that the attachment site of the ACL on the tibial plateau is close to the longitudinal axis, resulting in a short moment arm. Hence, an internal tibial torque primarily loads the ACL in its role as a primary restraint to anterior tibial translation. Furthermore, these results imply that the decrease in the dynamic rotational resistance due to the ACL insufficiency could be reduced even more if ACL reconstruction were to restore physiological anterior tibial translation.

The strength of this in vitro study is that we were able to compare kinematics between ACL-intact and ACL-deficient knees while simulating a realistic jump landing scenario under highly controlled conditions that would be hard to achieve in vivo. Pflum et al. predicted the ACL force during drop landings using their computer model²³. In their study, the drop landing data, including kinematics, muscle electromyographic (EMG) data, and ground reaction force, were collected from a healthy male athlete. As shown in Figure 2, *a*, in the present study, the ground reaction force and simulated muscle forces for the baseline loading condition are consistent with the ground reaction force and in vivo muscle EMG pattern in the study by Pflum et al.²³. In our study, a pivot landing scenario was simulated by adding the impulsive axial tibial torque to the baseline loading condition. During an in vivo pivot landing, the magnitude and direction of the axial tibial torque applied to the knee joint depends on the body's axial angular momentum while landing, thereby making this pivot landing scenario realistic.

There are several limitations to our study. First, only one initial knee flexion angle (15°) was tested because Li et al.³² reported the ACL strain to be highest with the knee in 15° of flexion. More recently, in maximum jump landings, initial mean knee flexion angles (and standard deviations) of 11° ± 3° at toe contact and 28° ± 7° at heel contact have been measured³³. Finally, Olsen et al.¹³ used injury videotapes to estimate the knee flexion angle at injury to be 16°. Therefore, the initial 15° knee flexion angle used in our study seems reasonable, but other angles could be investigated in the future. A second limitation of our study is that the change of knee flexion angle under the impulsive test load was smaller than those in some in vivo studies. For example, Pflum et al.²³ reported that the change in knee flexion angle in the first 70 msec was ~35°. Our studies showed an average change in knee flexion angle of ~5° in the first 70 msec. This is consistent with the findings of Russell et al.³⁴, who reported a change in the knee flexion angle, during a single-leg drop landing, of <10° over the initial 70 msec. Hence, the change in knee flexion angle in our study appears reasonable.

A third limitation is that the peak impulsive compressive force for the internally directed trials was slightly higher than 1.2 times body weight, whereas the corresponding value for the baseline loading conditions was about two times body weight. This decrease in the peak impulsive compressive force resulted from the custom torsional transformer device (T in Fig. 1). However, by maintaining the height of the drop weight at the same level across all trials, we applied the same kinetic energy to the knee specimen under all test conditions. Withrow et al.¹⁸ showed that the ACL relative strain was highly correlated with the quadriceps force, not the impulsive compressive force. Dikeman³⁵ and DeMorat et al.³⁶ also found that an increase in quadriceps force increased ACL strain. These results suggest that the increase in quadriceps force dominates the increase in ACL strain more than does the increase in impulsive compressive force. In our testing, under all loading conditions, the quadriceps forces were maintained at the same force level,

thereby providing a good opportunity to investigate the effect of the different loading directions and the ACL status on the knee joint kinematics.

A fourth limitation of the study is that the effect of the iliotibial band was not simulated even though it reduces the anterior tibial translation of the lateral tibial plateau, thereby limiting the internal tibial rotation and the coupled anterior tibial translation under a rotatory load. Yamamoto et al. investigated the effect of the iliotibial band force on the coupled anterior tibial translation, internal tibial rotation, and in situ ACL force in response to a combined 10 N-m of valgus moment and 5 N-m of internal tibial torque³⁷. They reported that all three measurements were generally decreased as the iliotibial band force increased. However, the effect of the iliotibial band force level on internal tibial rotation was not significant. Additionally, the coupled anterior tibial translation was significantly affected by the iliotibial band force only at high degrees of knee flexion (45° and 60°) under the highest iliotibial band force (88 N). In our study, the initial knee flexion angle was maintained at 15° for all trials and the changes in knee flexion angle were <10°. The results reported by Yamamoto et al. suggest that the iliotibial band force can be ignored in the case of our low initial knee flexion angle and small change in knee flexion angle.

Finally, our study was limited by the absence of a complete set of knees tested under the external tibial torque: only two knees were tested under the external tibial torque following the ACL transection because we were concerned about deterioration of the knee specimens due to too many trials.

In conclusion, our results show that transection of the ACL leads to a small increase in internal tibial rotation, equivalent to a 13% decrease in the dynamic rotational resistance, under the large forces associated with a simulated jump landing but results in a significant increase in anterior tibial translation. ■

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